Philosophie

THE EXTINCTION OF LIFE

An Inquiry into the Metaphysics of Disease

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Short abstract

For a long time on, philosophers have been trying to analyze what disease is. The significance of the topic of disease lies mostly in its ubiquity and in the historical relationships between philosophy and medicine. This work is interested in the metaphysics of disease; it defends an original and general theory of disease called "essentialism about disease", according to which, roughly, disease is the destructive process of the essence of a processual part of a living being. Filling in a gap in the literature, we start with methodological considerations; Part I argues that a species/kind is a whole to be defined with respect to its essential parts viz. a genus and a differentia. After having more fine-grainedly distinguished between "disease" and cognates (like "disease kind", "symptom", "etiopathology", or "illness", etc.) in Part II, we provide, in Part III, a neutral framework for thinking the debate around disease, where we distinguish between the group "axiologism about disease", "malfunctionalism about disease" and "hybridism about disease"; these three groups are distinguished with respect to the emphasis the theories of disease they include put on one or both of the main intuitions that we have about disease, which are, respectively, the intuition (1) that disease is a certain negative value viz. a lethal value, and the intuition (2) that disease is a specific biological malfunction. We situate essentialism about disease within the group hybridism about disease, by taking coherently into account both intuitions (1) and (2) through the unifying idea of disease as the destruction of a processual part of a living being. Finally, by investigating the direct consequences of essentialism about disease with several potential counter-examples (like genetic diseases) and controversial cases (like highest-order mental diseases, or aging), we show that essentialism about disease is currently the most encompassing general theory of disease.

Extended Abstract

Any reader who is reading this sentence has already been diseased at least once in his life. Diseases are ubiquitous; indeed, the scope of our concept of disease intuitively includes cases as diverse as phenylketonuria (PKU) and (lung) cancer; but it seems to also exclude cases like drapetomania (i.e. the tendency of African-American slaves to flee from their servitude), while other cases are more controversial such as vices or aging; beyond its ubiquity and unclarity, the concept of disease plays a role in the historical relationships between philosophy and medicine.

Which justified criteria are there for distinguishing between diseases and non- (or pseudo-) diseases? What is the nature of disease? From the Hippocratic theory of disease as disturbance (or imbalance) between the four humors (or temperaments) to the Bio-Statistical Theory of disease (and health) as a biological process of a part of a living being deviating from its normal biological function, which is a statistically typical contribution of this part (within a reference class) to the inclusive fitness of the overall organism, through other various theories of disease, philosophers have been trying for a long time already to thoroughly analyze this elusive and complex concept that is DISEASE. By focusing on the metaphysics of disease, this PhD dissertation adds its own original contribution to this huge pile; it defends the general theory of disease called "essentialism about disease", according to which, roughly, disease is the destructive process of the essence of a processual part of a living being.

Filling in a certain gap in the literature, Part I of this dissertation argues, first, for a certain meta-philosophical position (applicable to all the suitable entities), according to which to define the predicate *D* is to find out those conditions necessarily equivalent to the sentence "*x* is *D*", where those conditions are the intensional or constitutive (or essential) parts (independent from each other) of a numerically distinct whole, which is *D*. Second, we defend the correspondence principle, according to which defining the predicate e.g. "_is a disease" is equivalent to defining the concept DISEASE or disease as another specific entity. Third, we implement this meta-philosophy through a certain understanding of the genus-species hierarchy, according to which a (natural) species or kind (e.g. a disease kind) is a whole numerically distinct from its constitutive parts (independent from each other), which are a genus (proximum) (e.g. a disease) and a differentia. Fourth, alongside with a very permissive neo-Aristotelian ontology leaving room for many irreducible entities, where not every existing entity can be constitutively defined, we defend that a genus, as a fundamental entity, is a constitutive part of a species, while a differentia, as being apart from our ontology, merely reduces to either a kind or a genus.

Part II of the present PhD dissertation defends the genuine status of disease by investigating those concepts directly related to DISEASE and easily confused with in the literature. First, we argue for a definition of disease kinds, according to which a disease kind is a disease (i.e. the genus) plus a negative cause related to a living being and a negative effect related to a living being (i.e. the differentia). Second, we defend that a negative effect of a disease (i.e. a symptom) is a disease (i.e. the genus) plus a directly temporally succeeding negative entity related to a living being (i.e. the differentia). Third, we argue that

an etiopathology is a disease (i.e. the genus) plus a directly temporally preceding negative entity related to a living being (i.e. the differentia). Fourth, we argue that disease is not to be confused with higher-level cases of disease like illness, which is merely the feeling of a disease; sickness, which is a suffering; disability, which is the feeling of suffering; or harm, which is a strong suffering.

Part III of this PhD dissertation argues for a constitutive definition of disease following our meta-philosophy. First, we provide a neutral framework for thinking the debate around disease; we can distinguish between three groups of theories of disease: axiologism about disease, malfunctionalism about disease, and hybridism about disease; these three groups of theories of disease are distinguished with respect to the emphasis the theories of disease they include put on one or both of the two (main) intuitions that we have about what disease is, where, respectively, the first main intuition (1) is that e.g. "_is cancerous" is a specific lethal value (like death) attributed to "cell growth", and the second main intuition (2) is that "cell growth is cancerous" means that cell growth is biologically malfunctioning in a specific way. Second, we briefly illustrate and reject on independent grounds the most well-known theories of disease belonging to these three groups. Third, essentialism about disease, according to which, strictly speaking, disease is objectively analyzed as a negative process of a part of a living being (i.e. the genus), and is the destruction of a positive state p until the negative state $\neg p$ (i.e. the differentia), is situated within hybridism about disease, by taking coherently into account intuitions (1) and (2), as two sides of the same coin, through the unifying idea of disease as the destruction of the essence of a processual part of a living being. The central argument for essentialism about disease is as follows:

(1) what is diseased is a highly fine-grainedly differentiated processual part of a living being;
(2) a processual part of a living being is a healthy process;
(3) as something good, a processual part of a living being's function is its essence;
(4) a negative process is the destruction of a positive process;
(5) disease, as a certain negative process (i.e. here a certain lethal value), destroys a certain positive process i.e. a healthy process;
(6) disease, as a certain negative process (i.e. here a certain biological malfunction), destroys a certain positive process i.e. a processual part of a living being's function;

∴ disease is the destructive process of the essence of a processual part of a living being.

With the use of two detailed illustrations (as a requirement that a kind must be necessarily realized), PKU is the disease destroying in a specific way the function or the essence of phenylalanine hydroxylation, which is a healthy process, i.e. converting the amino acid phenylalanine into the amino acid tyrosine by making tyrosine decrease (or, the change from the state where tyrosine is not present to the state where tyrosine is present); (lung) cancer is the disease destroying in a specific way the essence of cell growth (of the lung's tissues) i.e. dividing into two daughter cells and grouping them (i.e. the change from the state of non-two daughter cells divided and grouped to the state of two daughter cells divided and grouped) through an uncontrolled cell proliferation.

Finally, to provide a most comprehensive theory of disease, we show how essentialism about disease takes into consideration genetic diseases i.e. as fundamental diseases, as well as other potential counter-examples; moreover, we show how essentialism about disease sheds new light on some controversial diseases like vices i.e. by taking them as higher-level mental diseases, or aging taken as the mere passing through the ages.

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ADA	Americans with Disabilities Acts	217
APA	American Psychiatric Association	131
ATC	Anatomic Therapeutic Chemical	272
BMI	Body Mass Index	205
BST	Bio-Statistical Theory	48
CVD	Cardio-Vascular Disease	129
Def_1	Definition 1	13
Def_2	Definition 2	13
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DSM	Diagnostic and Statistical Manual of Mental Disorders	95
EBM	Evidence-Based Medicine	151
ETC	Epistemic Theory of Causality	150
HPC	Homeostatic Property Cluster	83
HPV	Human Papillomavirus	128
ICD	International Classification of Diseases	155

LL	Leibniz's Law	31
MCP	Maximal Compatibility Principle	60
ME	Mereological Essentialism	18
MF	Mereological Fallacy	129
MI	Mereological Infinitism	112
MWF	Mereological Well-Foundedness	112
PAH	Phenylalanine Hydroxylase	241
PAH-tion	Phenylalanine Hydroxylation	241
PII_1	Principle of the Identity of Indiscernibles	30
PII_2	Principle of the Indiscernibility of Identicals	30
PKU	Phenylketonuria	241
PTSD	Post-Traumatic Stress Disorder	83
RCT	Randomized Controlled Trial	151
RDoC	Research Domain Criteria	155
RME	Reverse Mereological Essentialism	20
RWT	Russo-Williamson Thesis	150
SNOMED	Systematized Nomenclature of Medicine	155

STD	Sexually Transmitted Disease	51
WCP	Weak Conservation Principle	26
WSP	Weak Supplementation Principle	17

1

INTRODUCTION

What is disease? This (apparently) very simple question needs for sure a book-length study (if not more) like the present PhD dissertation to be addressed. Indeed, is e.g. every biological defect a disease? What about mental diseases? Do they exist as genuine diseases? Can we literally talk about vices as higher-level diseases? What about controversial (and historical) cases? Is suffering real or only in one's mind? Are there as many specific diseases as there are persons and other living beings on earth? Do/Can we create diseases? Is disease a certain value? Is it a specific lethal value? Is disease a certain biological malfunction? What is the relationship between life, death and disease? What about illness, sickness, harm or disability? What is their relationship with disease? Is disease a process or a state (as a condition)? Is disease not merely a certain feeling? What is the difference between disease and symptoms or the causes of disease? Is disease the absence of health, or health the absence of disease? Or even, how to start an inquiry into disease?

Typically, those are only some of the questions, amidst many more, that immediately pop up to our mind, as soon as we seriously start thinking about the question of what disease is in all its generality. Many have already ventured on this difficult and huge path, which is to provide an analysis of what disease is. Why go there? Or, more generally, why to start an inquiry into *disease*?

1 Introduction

The importance of the topic of disease is - at least (and orthogonal to any pre-conception of disease) - four-fold:

1. it is obvious that we are unclear with what disease is;
2. the topic of disease (in)directly intersects with other topics worth to be examined like health, life, death, etc.; or even, more generally, with the discipline of medical ethics (cf. Ch. 11).
3. the use of the term "disease" is ubiquitous both in the scientific discourse and in daily life;
4. the notion of disease plays a pivotal role in the history of philosophy and its complex relationship to medicine, most notably through the figure of the "philosophical physician", where the best physician is also at the same time a philosopher, for a physician must use philosophical thought to obtain bodily knowledge and get the best remedies; as Galen (Opt. Med. 60-61; tr. Singer, 1997) famously states it:

> What grounds are then left for any doctor who wishes to be trained in the art in a way worthy of Hippocrates not to be a philosopher? He must be practised in logical theory in order to discover the nature of the body, the differences between diseases, and the indications as to treatment; he must despise money and cultivate temperance in order to stay the course. He must, therefore, know all the parts of philosophy: the logical, the physical, and the ethical. […] If, then, philosophy is necessary to doctors with regard both to preliminary learning and to subsequent training, clearly all true doctors must also be philosophers.

This is not a work in the history of philosophy and/or medicine; this is a work on the concept of disease. We could have decided to focus, instead, only on a limited range of diseases; however legitimate this purpose is (and also sufficient for a PhD dissertation), points (1)-(4) above encourage us more to take part in the bigger and relevant task of providing a *general* analysis of what disease is.

The subtitle of the present PhD dissertation is "an inquiry into the metaphysics of disease". If points (1)-(4) are good reasons to begin especially an analysis into the nature of disease i.e. its metaphysics, without denying, of course, the legitimate status of e.g. an *ethical* or *epistemological* inquiry into disease, we can find two additional reasons to focus on the *metaphysics* of disease (however plausibly or correctly understood), rather than its ethics or epistemology:

1. doing a metaphysical inquiry establishes itself as the first obvious or fundamental choice to make, when one wants to start an analysis around a complex notion (like the one of disease), for metaphysics is about being as such (cf. van Inwagen, 2014);
2. doing a metaphysical inquiry dwelves on the idea that it is somehow what comes *first* i.e. here before that other (more) specific analyses around a certain complex concept can fruitfully take place.

From the Hippocratic theory of disease as disturbance (or imbalance) between the four humors (or temperaments) to the Bio-Statistical Theory (BST) of disease (and health) as a biological process of a part of a living being deviating from its normal biological function, which is a statistically typical contribution of this part (within a reference class) to the inclusive fitness of the overall organism (cf. Boorse, 1977; 1997), through other various theories of disease (cf. Ch. 8; also Pellet, 2016; 2018), philosophers have been trying (unsuccessfully until now) for a long time already to thoroughly analyze this elusive and complex concept that is DISEASE.

With its proper methodology, the present PhD dissertation is a contribution on the concept of disease adding to an already huge pile, but constantly worth be increased in our search for truth – moreover, the topic of disease has never been of more actuality than today. We hope to provide the reader with an original and novel contribution to the debate around the concept of disease filling in the gaps of the present literature, most notably through having a proper and clear methodology, more fine-grained distinctions between "disease" and cognates (cf. Ch. 4-5-6-7), a neutral framework for thinking the debate around disease (cf. Ch. 8), the unification of two main intuitions about disease coherently taken into account into a single theory of disease (cf. Box 8.1-8.2), a theory of disease avoiding the same flaws of the other most discussed theories of disease (cf. Sec. 8.2.1-8.2.2-8.2.3), and a more encompassing theory of disease – i.e., in a nutshell, with a theory of disease, we hope so, approaching truth at the closest possible (at time *t*) (cf. Exc. 3.8).

The theory of disease called "essentialism about disease" is defended in this PhD dissertation: roughly, *x* is a disease, iff *x* is the destructive process of the essence of a processual part of a living being (cf. Box 9.1; also 9.3).

The central argument for this bold conclusion is as follows:

(1) what is diseased (or the disease host) is a highly fine-grainedly differentiated processual part of a living being (cf. Sec. 9.2.1; 9.3.2);

(2) a processual part of a living being is a healthy process (cf. Sec. 9.3.3);

(3) as something good, a processual part of a living being's function is its essence (cf. Sec. 9.4.1);
(4) a negative process is the destruction of a positive process (cf. Sec. 9.3.1);
(5) disease, as a certain negative process (i.e. here a certain lethal value), destroys a certain positive process i.e. a healthy process (cf. Sec. 9.3.4);
(6) disease, as a certain negative process (i.e. here a certain biological malfunction), destroys a certain positive process i.e. a processual part of a living being's function (cf. Sec. 9.4.1);
∴ disease is the destructive process of the essence of a processual part of a living being.

The overall structure of the present PhD dissertation is highly basic and is meant to be mainly *positive*: it includes three parts with nine chapters (with their own sections, sub-sections, and sub-sub-sections) both somehow self-contained and all forming a harmonious whole – in the image of the mereology defended in this PhD dissertation. Relatedly, so as to find out a compromise between detailedness and reading clarity, we have added at the end of the first sections, where the explained notions in question occur, *excursuses* – in line here with an old scholastic tradition - as (pretty big) digressions or complementary and general notions necessary for a better understanding of the overall PhD dissertation (at least, for the main line of argumentation), while maintaining local caveats or terminological notes, which are more contextual or less important for the main line of argumentation.
About the contents of this PhD dissertation, we have tried to make explicit as far as possible all the basic assumptions that we make throughout the entire dissertation: e.g. on what is meant by "objective" (cf. Exc. 2.7), "natural" (cf. Exc. 2.6), or even "entity" (cf. Sec. 2.1.2) or "process" (cf. Sec. 9.2.1), etc. Though no expert knowledge is required to understand this PhD dissertation, a minimal background especially in (philosophical) logic and medicine is expected from the reader.
Although philosophy of medicine or, more specifically, the metaphysics of disease (and health) has given rise itself to an important debate around how to correctly approach the question of what disease (and health) is (cf. e.g. Lemoine, 2013; 2015) – the reason for this certainly lies in the old intertwinement between medicine and philosophy -, current theories of disease are very often decoupled from methodological considerations. Filling in this gap, Part I of the present thesis argues for a certain meta-philosophical position. First, in Ch. 2, we defend the meta-philosophical position (meant to apply to all the suitable entities) that to define the predicate *D* (e.g. "_is a disease") is to find out those conditions necessarily (modally speaking) equivalent to the sentence "*x* is *D*", where those conditions are the *intensional* or *constitutive* (or *essential*)

parts (independent from each other) of a numerically distinct whole, which is *D* (cf. Def_4). Second, we defend the *correspondence principle*, according to which defining the predicate e.g. "_is a disease" is equivalent to defining the concept DISEASE or disease as another specific existing entity.

In an effort to implement this abstract metaphilosophy, in Ch. 3, we argue, first, that disease is ontologically and definitionally prior to disease kinds, in so far as disease is the *genus* under which a disease kind as a *species* falls; more generally, we understand a (natural or artefactual) species or *kind* as a whole numerically distinct from its genus (proximum) and differentia, which are its constitutive or essential parts (independent from each other) (cf. Box 3.1). Second, although we argue for a very *permissive neo-Aristotelian ontology* leaving room for many different specific irreducible entities highly fine-grainedly (and non-vaguely) differentiated like (natural and artefactual) kinds and sets (cf. Exc. 3.5; 3.9) – where kinds must still be necessarily (indirectly) empirically realized -, not all of these entities can be constitutively defined (the other entities are trivial ones): e.g. a genus, as a fundamental entity, is a constitutive part of a species (cf. Box 3.1.1); furthermore, as being apart from our ontology, a differentia reduces to merely either a kind or a genus (cf. Box 3.1.2).

If disease and disease kinds are good examples of our understanding of the genus-species hierarchy, before asking ourselves about how disease (taken for granted as a *non-trivial entity*) can be constitutively defined, we may wonder whether disease is not *reducible* to another entity. Part II defends the genuine status of disease through an investigation of those concepts directly related to DISEASE and easily confused with in the literature. In Ch. 4, we develop a theory of *disease kinds*, according to which a disease kind is constituted by a disease (i.e. the genus) plus a negative cause related to a living being and a negative effect related to a living being (i.e. the differentia) (cf. Box 4.2).

Ch. 5 and 6 are devoted to analyzing more precisely, and respectively, what a negative effect of disease and a negative cause of disease are. In Ch. 5, we argue, first, that a negative effect or sign of a disease (or *symptom*) is a disease (i.e. the genus) *plus* a directly temporally succeeding negative entity related to a living being (i.e. the differentia) (cf. Box 5.2) i.e. that e.g., plausibly speaking, Parkinson-plus syndrome is a symptom, for it is constituted by Parkinson's disease plus Pick's disease as an additional directly temporally following entity. Second, we defend the idea that felt and unfelt symptoms are *specific* symptoms themselves.

Ch. 6 is about the causes of disease or the *etiopathological agent* (or etiopathology). First, we defend the idea that an etiopathology is a disease (i.e. the genus) *plus* a directly temporally preceding entity related to a living being (i.e. the differentia) (cf. Box 6.2) (however plausibly

or correctly understood): e.g HIV-AIDS is an etiopathology, for it is constituted by a single whole AIDS plus HIV infection directly temporally preceding it. Second, we relate our own theory of medical causes to the general *mechanistic* theory of causation developed by Stuart Glennan (1996; 2002), according to which, roughly, an event *c* situated at the non-fundamental level causes an event *e* situated at the non-fundamental level, iff there is a(n) (underlying) mechanism *m*, which connects *c* to *e*.

Ch. 7 is devoted to what we take to be specific *higher-level* diseases (possibly reducible to other ones, and only prima facie defined here), which are still necessarily (indirectly) realized by somatic diseases (cf. Exc. 7.2): *illness* is the feeling of a disease, where feeling *x* is a mere attention mechanism directed towards *x* (cf. Box 7.1); being *sick* of *x* is suffering from *x* (cf. Box 7.2); *disability* is the feeling of suffering (cf. Box 7.3); *harm* is a strong suffering (cf. Box 7.4); all these specific diseases are *objectively* analyzed.

Once we are clear that disease is *not* a disease kind nor a symptom nor an etiopathological agent nor a bad feeling (as an illness) nor a sickness nor a disability nor a harm – all entities, which are sometimes used interchangeably with disease in the literature (cf. e.g. Sec. 4.2.7), or fail to be neatly distinguished from disease (cf. e.g. Ch. 7) -, Part III of this PhD dissertation is devoted to provide a constitutive definition of disease following our meta-philosophy. In Ch. 8, we provide, first, a framework for thinking the debate around disease; we can distinguish between three groups of theories of disease: *axiologism* about disease, *malfunctionalism* about disease, and *hybridism* about disease; these three groups of theories of disease are distinguished with respect to the emphasis the theories of disease they include put on one or both of the two (main) intuitions that we have about what disease is, where the first main intuition (1) is that e.g. "_is cancerous" is a specific *lethal value* (like death) attributed to "cell growth" (cf. Box 8.1), and the second main intuition (2) is that "cell growth is cancerous" means that cell growth is *biologically malfunctioning* in a specific way (cf. Box 8.2). Axiologism about disease is a group of theories of disease, which put the emphasis on intuition (1), while somehow explaining away intuition (2); malfunctionalism about disease contains theories of disease seriously accounting for intuition (2), while explaining away intuition (1); hybridism about disease includes theories of disease seriously based on both intuitions (1) and (2). This way to present *neutrally* those three groups of theories of disease avoids the common flaw, in contemporary literature, which is to start with the assumption that a theory of disease belonging to axiologism about disease shall necessarily take disease (as a value) to be *subjective*, while disease shall be necessarily *objective* (or natural) in the group malfunctionalism about disease. Second, we briefly illustrate and reject on independent grounds the most well-known theories of disease belonging to our

three groups, amongst which Nordenfelt (1995; 2000)'s subjectivist theory, according to which, disease is the unpleasant state preventing a subject to accomplish his vital goals, which make the overall organism happy in the sense of having a minimally decent life (cf. Sec. 8.2.1) – the theory is rejected on basis of e.g. its reliance on axiological subjectivism and its too broad condition of what counts as a specific lethal value; Boorse (1977; 1997)'s BST of disease, according to which disease as a value-free entity is *x*'s biologically malfunctioning is *x*'s deviating from its normal biological functioning i.e. a statistically typical contribution of *x* (within a reference class) to the inclusive fitness of the organism of which *x* is a part (cf. Sec. 8.2.2) – the theory is rejected on basis of e.g. the arbitrariness behind the choice of the reference class; and Wakefield (1992)'s harm-malfunction theory that disease is both a harm and a certain biological malfunction (as a naturally selected effect) (cf. Sec. 8.2.3) – the theory is rejected on basis of e.g. the lack of internal coherence between intuitions (1) and (2).

Ch. 9, with two detailed illustrations i.e. phenylketonuria (PKU) and (lung) cancer, defends an original *hybrid* theory of disease called "essentialism about disease", according to which, roughly, disease is the destructive process of the essence of a processual part of a living being (cf. Box 9.1); more precisely, disease is a negative process of a part of a living being (i.e. the genus), and is the destruction of a positive state *p* until the negative state ¬*p* (i.e. the differentia) (cf. Box 9.3); in a word, to be *diseased* is for a part of a living being to be *deceasing*: e.g. PKU is the disease destroying in a specific way the essence of the process of phenylalanine hydroxylation (PAH-tion) i.e. converting the amino acid phenylalanine into the amino acid tyrosine by making tyrosine *decrease*; (lung) cancer is a specific way of destroying the essence of cell growth (of the lung's tissues) i.e. dividing into two daughter cells and grouping them by making it *hyper*function i.e. through an uncontrolled cell proliferation.

Going back to the main line of argumentation put above, with premise (1), along with a permissive neo-Aristotelian ontology, the disease host is, first, a highly *fine-grainedly* differentiated kind like PAH-tion or cell growth; second, the disease host is a *part* of a living being, for, as being another lethal value, *death* is intuitively distinguished from disease with respect to its specific host: only a whole *living* being can be said dead/dying, while only a (highly fine-grainedly diffentiated) part of it can be said diseased (cf. Exc. 9.1); third, the disease host is a (certain) *process*; indeed, as being a thing happening, disease (and its host) is intuitively a certain process, where a process is prima facie the *generation* of a *state* (¬)*x* at time t_{+1} from the contradictory state (¬)*x* at time t_0 (cf. Box 9.2): e.g. PAH-tion is a process, for it is the change from the *state* where tyrosine is *not* present to the *state* where tyrosine is present, to the contrary of PKU; cell growth is a process, because cell growth is the change from the state

of non-two daughter cells divided and grouped to the state of two daughter cells divided and grouped, to the contrary of cancer.

With premise (2), a processual part of a living being is a healthy process. Indeed, as a vital value (like life), health is *objectively* analyzed as the essence of a processual *part* of a *living* being (e.g. a healthy PAH-tion or cell growth just is an existing PAH-tion or cell growth (with its essence)), where life is the value grounding the existence of a certain bearer (e.g. a living human being just is an existing human being) through its having a specific essence like homeostasis, self-reproduction, or metabolism (cf. Box 9.4-9.5).

With premise (3), e.g. how PAH-tion or cell growth works – as a process, it is the change from the state where tyrosine is not present to the state where tyrosine is present, or the change from the state of non-two daughter cells divided and grouped to the state of two daughter cells divided and grouped -, which is its *essence*, is also at the same time intuitively its (biological) *function*, where PAH-tion or cell growth is a processual part of a living being, or a *healthy* process.

Premise (4) is the general idea that a negative entity is the destruction/negation of a positive one i.e. that a negative entity is still an *existent* entity, which can still very well be part of our meta-philosophy; more specifically, a negative process is the destruction of a positive process.

Along premise (5), if disease is taken as a specific *negative* value i.e. a specific *lethal* value, then, as a negative entity viz., more precisely, a negative process, it destroys a certain positive value, which can be intuitively taken here as the one of *health* i.e. the essence of a processual part of a living being; in other words, disease is the absence of health – not the opposite.

As the last premise, premise (6) is the idea that, if disease is a certain biological malfunction, then, as a negative process, it destroys the corresponding positive process, which is here – not a mere function -, but the function of something *good* viz. a processual part of a *living* being, which is its essence.

Essentialism about disease takes into account *coherently* both intuitions (1) and (2), in so far as intutions (1) and (2) are taken as two sides of the same coin (cf. Fig. 9.4), as we can see thanks to premises (5) and (6). The originality behind our hybrid theory of disease precisely lies in our showing the *inseparability* between intuitions (1) and (2) (to the contrary of e.g. Wakefield (1992)'s own hybrid theory of disease) i.e. that, though useful as a template for thinking about disease or for classifying *globally* even our own theory of disease meaningfully said prima facie "hybrid" (for, its starting point is still a serious analysis of both intuitions (1) and (2)), the presented framework must be eventually rejected.

As a last chapter of the present PhD dissertation, Ch. 10 considers the *direct consequences* of essentialism about disease (and our meta-philosophy). First, potential counter-examples

attacking the necessity condition of essentialism about disease are examined – most notably, that essences, (actual) processes, and objectivity are not necessary features: as *fundamental* diseases, we argue that *genetic diseases* merely destroy genetic processes, where a genetic process is not constitutively defined, but teleologically i.e. as the coding for a trait (cf. Sec. 10.1.1); other potential counter-illustrations are subsequently examined like *inherited vs. acquired diseases*, *being born without* e.g. well-functioning legs, *cultural diseases* and *latent diseases* (cf. Sec. 10.1.2-10.1.5); other potential counter-examples attack the sufficiency condition, amidst which chronic diseases, imagination-related diseases, deformities, and artefactual diseases (cf. Sec. 10.1.6-10.1.9). Second, we show how essentialism about disease sheds new light on some controversial cases of disease: vices are taken as genuine diseases viz. bad intentions destroying positive intentions (or the virtue) to do something (cf. Sec. 10.2.1); the same is true for the case of bad personality/character traits or temperaments, which destroy good personality traits consisting in having continuous positive mental states/processes (cf. Sec. 10.2.2). To rule out any threat of trivialization, we exclude *incurable diseases* as genuine diseases, for a disease is not essentially a certain attitude consisting in curing (but which cannot be so) (cf. Sec. 10.3.1); ditto for "aging", which merely refers to the passing through the ages i.e. passing through time (cf. Sec. 10.3.2). Though most of the theories of disease obviously have to make a choice between generality and biological detailedness, they still fail to positively show their universalizability (cf. e.g. Ch. 8 for our objections against those theories); by its capacity to take into account non-standard cases of disease, we hope to show that essentialism about disease is currently the most satisfying and encompassing general account of what disease is.

I

THE (META)PHILOSOPHY OF DISEASE

2

A DEFINITION OF A DEFINITION OF DISEASE

Ch. 2 of the present PhD thesis is about the very topic of interest of this dissertation: disease. Any philosophical investigation into disease should be clear, from the outset, about what it is about i.e. what we are talking about, when one says that this dissertation aims at defining disease. In other words, Ch. 2 is devoted to tell some words about what is here meant by "defining disease".

Ch. 2 can be said to be a philosophical investigation or a conceptual analysis into what it is to define some concept C viz. DISEASE; since this metaphilosophy is meant to be universally applicable (at least, to the suitable entities), Ch. 3-10 strictly begin just to apply this philosophical methodology to the specific concept of disease and related ones.

In Sec. 2.1, I argue that to give a definition of some predicate *D* or concept D viz. DISEASE here, is to give in a specific way necessary and sufficient conditions for the sentence "*x* is *D*" to be necessarily (modally speaking) true i.e. as intensional parts of a whole.

In Sec. 2.2, I argue that to give a definition of disease is to give the sense of the word "disease", or to give an analysis of the concept DISEASE, or to find the essence of disease. More precisely, Sec. 2.2 is meant to be a more accurate analysis of what we are analyzing, when we decide to provide a definition of disease: what are the different ways to refer to the term "disease" or the concept of disease? an instance of the type disease ("a disease"), as instances of the type disease ("diseases"), or as the type disease itself ("disease"); or, analyzing disease, is it analyzing a disease, disease, or diseases?

2.1 What it is to Define Disease: The Relationship

The present PhD dissertation is about what disease is.

Sec. 2.1 is devoted to present, in a very general step-by-step way (with the case of disease as a mere example here), what it is to truly *define* e.g. disease. I argue that to define some predicate *D* is to give necessary and sufficient conditions for the sentence "*x* is *D*" to be necessarily (modally speaking) true, where those necessary and sufficient conditions are to be taken as intensional parts of a whole.

Sec. 2.1 is divided into three further sections: in Sec. 2.1.1, a first modal account of a definition is provided.

However, insufficiency problems are pointed out, in Sec. 2.1.2, while a second – amended – attempt is made by taking into account a mereological structure.

Another insufficiency problem is, in turn, also revealed, in Sec. 2.1.3, where I eventually provide what I take to be the most promising version of an account of what a definition is.

2.1.1 First Attempt: A Modal Account

The expression "defining disease" can have very different meanings to many different people. For some, it may mean "finding out how the concept of disease is defined by the (natural) scientists using this very concept" (cf. Exc. 2.1); for others, it may mean "finding out how the concept of disease is embedded into a socio-historical situation".

It is common, in the history of analytic philosophy (cf. Burge, 1993 for the traditional view on definitions), to answer the question "What is *D*?" viz. here "What does the predicate "_is a disease" mean[1]?" by providing a definition stating all the individually necessary and jointly sufficient conditions for a definiendum to be true with the following logical biconditional form (for a full-fledged defense of that claim, cf. Ch. 3):

[1] Caveat on "_is diseased" vs. "_is a disease": note that we have to be cautious, from the beginning, not to presuppose that there is an *x* such that *x has* the property of being diseased (indeed, against *being diseased* as an accidental property, cf. Sec. 9.3.1). That is why, we prefer starting with the direct question of what disease – rather than the property of being diseased – is.

(Def$_1$) Def (D, F, …) iff $Dx \leftrightarrow (Fx \,\&\, \ldots)$[2], where D, F, … stand for predicates, x for a free variable, and Def (D, F, …) for a definitional sentence linking D and F, …

Def$_1$ is the very first – non-modal – brush as an account of what a definition (as a sentence) is (for the correspondence principle between predicates, concepts and other specific ontological units, cf. Sec. 2.2.1). Indeed, despite the presence of a biconditional relationship, Def$_1$ is highly insufficient: e.g. the sentence "x is a disease, iff x is a value and etc." satisfies Def$_1$, but is clearly not a definition of disease.

To rule out those cases of accidental (or extrinsic) predicates attributed to the predicate "_is a disease", a new proposal can be made as following by using modal logic (on, more generally, *intensional* logic, cf. Fitting, 2015):

(Def$_2$) Def (D, F, …) iff $\Box\, (Dx \leftrightarrow (Fx \,\&\, \ldots))$[3].

Def$_2$ can be read as following (for the same starting point as a definition, but not with predicates, cf. Rosen, 2015): let us suppose that Dx is a place-holder here for the sentence "x is a disease" and Fx, etc., stand for the sentences (or a sentence as a *set*) equivalent to Dx (or, the sentence(s) having all the strictly necessary and jointly sufficient conditions for Dx to be true), then to define the declarative sentence "x is a disease" amounts to saying *ex hypothesi* that it is necessary that x has the predicate "_is a disease", iff x has the predicates F, etc.

In that sense, Def$_2$ rules out the above counter-example satisfying Def$_1$, for there is a possible world in which x is a disease and is not a value (and etc.).

[2] In so far as we are trying to *define* a definition, the same logical equivalence relation drawn on the right side of Def$_1$ also reflexively applies to the left side of Def$_1$ (cf. Rosen, 2015).
However, with all the amendments to Def$_1$ that shall be shortly made (cf. Sec. 2.1.2 et sqq.), applying the same ones to the left side of the amended versions of Def$_1$ would become quickly unintelligible. That is why, I maintain in these cases "Def (D, F, …) iff".

[3] Caveat on Def$_2$: why not resorting here to Mackie (1974)'s famous INUS-conditions (putting aside their link to regularity theories of causation in the present context) to analyze *constitution* (cf. Kaiser, 2018b) – rather than using higher-order logic? The main issue with these INUS-conditions – be they used, actually, for analyzing causation or constitution – is that they unfortunately have (exclusive) disjunctions; and (exclusive) disjunctions, however (often arbitrarily and/or vaguely) constrained the disjuncts can be (cf. e.g. Baumgartner, 2008), are peculiarly hard to handle with in metaphysics (at least, for non-trivial entities) (cf. Walter, 2002), even though, of course, sustaining to causal/constitutive antirealism (or rather, antiobjectivism) remains a possibility (for further objections against disjunctive analyses of kinds, cf. Sec. 3.2.2).
Note, moreover, that INUS-conditions seem, thus, not well suited for analyzing causation/constitution in a *monistic* way. Worse, extended versions of INUS-conditions - adding to disjunctive causes/constituents disjunctive *effects*/*constituted* entities - imply that no *single* whole (taken as a constituted entity) can ever be, actually, picked out to be given a causal/constitutive analysis (even in a weak – but certainly not strong -pluralistic way).

In a nutshell, defining disease is finding out what the declarative sentence "*x* is a disease" is necessarily equivalent to.

2.1 Excursus: Science vs. Philosophy

We can *plausibly* defend that, when we talk about (natural) *scientists*, we are not including here among them philosophers.

However, if (meta)philosophy is also concerned about the study of, amidst others, certain *natural* phenomena like disease, health, life, death, etc. (as it should) – which other sciences like biology do not *properly* study, but still with the same *methodology* broadly understood viz. rational inquiry (on so-called (strong and weak) *liberal naturalism*, cf. e.g. Bennett & Hacker, 2003; De Caro & Voltolini, 2010; Hornsby, 1997; McDowell, 1994; Macarthur, 2017; 2019; Paul, 2012; Stroud, 1996) -, then (meta)philosophy is to be *strictly* counted as a specific (meta)science itself in the *Geisteswissenschaften* (or, strictly, human (meta)*sciences* or humanities) (vs. e.g. Arts) – either by contrast with *Naturwissenschaften* taken as the *biological* (restrictedly taken) or *physical* sciences, or not by contrast with (*Natur*)*wissenschaften* taken as the sciences *tout court* or *natural* sciences (but in the *wide* sense of "nature") here (cf. Exc. 2.6 for our uses of "nature"; Dilthey, 1883 [1989] on this famous distinction).

For, philosophy can be arguably said to investigate its own specific range of *natural* (*narrowly* taken) but also *artefactual* phenomena - just like, actually, other specific (natural) sciences (e.g. AI) -, the very notion of science included.

We can consistently use both (meta)philosophy as *distinct* from the (natural) (meta)sciences (in its plausible or loose usage), and as *continuous* with the (natural) (meta)sciences (in its strict or correct usage), as Tim Williamson (2018, p. 141; cf. also Feser, 2019 on the point that naturalism, properly understood, should not dismiss, actually, traditional metaphysics as unscientific) says in an almost sibylline way: "[p]hilosophy is a science in its own right […]".

Thence, the thesis known as (weak or strong) *liberal* naturalism should be more properly called "(weak or strong) naturalism" *tout court* (in a wide sense), although we can still plausibly use the label "liberal naturalism" on grounds of situating our own position within a certain debate or framework; for, naturalism *tout court* is the thesis, according to which, roughly, only the entities (plausibly speaking) found out or postulated (in an objectivist/subjectivist vein), by the (natural) sciences (through a certain methodology viz. rational inquiry) exist, and philosophy is arguably one of those (natural) sciences (*pace* Dicken, 2015); that much is also applied *reflexively* i.e. to the very thesis of (liberal) naturalism, of course – if it is taken as an entity investigated by philosophy itself (on the famous objection against naturalism as being internally incoherent or self-defeating, cf. Feser, 2019; for tentative answers, Forrest, 2000; Petersen, 2014).

2.1.2 Second Attempt: A Mereological Account

Albeit Def_2 is able to solve the specific insufficiency problem we have pointed out (cf. Sec. 2.1.1), other problems can be further pointed out (by following largely here Rosen, 2015). $Def_{1-(2)}$ is insufficient in other ways.

Indeed, first of all, any account of definition as logical equivalence is threatened by the famous Euthyphro dilemma, where Euthyphro is asked "Is the pious (*τὸ ὅσιον*) loved by the gods because it is pious, or is it pious because it is loved by the gods?" (Plat. *Euthyphr*. 10a) i.e. that the dilemma is here that it is inconsistent to both sustain the view that a definition consists in a logical equivalence and to account for the intuition (or pre-theoretical judgment, or insight into, or understanding of, an a priori truth) (cf. Ludwig, 2010 on that point and on specific intuitions like physical or philosophical intuitions; also Nimtz, 2010 for intuitions as beliefs; *contra* Bealer, 1998 for intuitions as intellectual seemings, and Cappelen, 2012) that, in a definition, there must be a *unique direction of an explanation* (i.e. that the elements of the definiens, analysans or here explanans (phenomenon) explains the definiendum, the analysandum or here explanandum (phenomenon) – but not vice versa). In a word, how to consistently maintain both the symmetry of logical equivalence and the *a*symmetry of an explanation? Or, how to avoid *definitional circles*?

To follow somehow the intuition that to define *x* has to do with finding out what *x* consists of/in or what *constitutes x*, or what *x* is made of (however understood here) i.e. with providing a constitutive (or metaphysical) explanation, we can answer the above question as following: the link between the predicate *D* and the predicates *F* (and *G*) is a link between a predicate

expressing a whole (or composite) (as a real entity) and predicates expressing the (im)proper parts (or components) of the whole[4, 5].

[4] Two terminological notes on parthood: (i) to me, only the notion of what is commonly called "proper part" *properly* captures the true meaning of "part" (or "component") (however *plausibly* understood i.e. e.g. as an *extensional* part, or how it is to be *correctly* understood i.e. as a *non-extensional* part like an intensional part).
Even though the literature (as an entry, cf. Varzi, 2016) distinguishes between cases of proper parthood and cases of improper parthood or parthood *tout court*, for sake of textual readability, except in cases where I specifically distinguish between proper parthood and parthood or related mereological relations or where the distinction does matter, I shall use throughout the PhD dissertation the word "part" (or "component") where I should, strictly speaking, use the word "*proper* part".
(ii) Moreover, the word "part" (or "component") *simpliciter*, when properly understood, refers, actually, to an *essential*, *constitutive* or *intensional* part (on "part" as polysemous, cf. Wallace, 2019).
However, the term "part" (or "component") can, of course, still be used under its *plausible* meaning to refer to an extensional part. I shall use "part" *tout court* under its correct meaning to refer to, indeed, an *intensional* part – except in the cases where its plausible meaning is used, in other easily confusing cases, or in cases where we do not pronounce on the correctness of the use of "part"; in these circumstances (which are plethoric), the distinction between intensional and extensional parts is made either explicit or somehow contextually clear.
[5] Five terminological notes (with caveats) on definitions, essences and intensions: (i) telling what it is to *define* something (i.e. defining a definition) is not the same, of course, as telling what *constitutes* something, for the relationship of constitution has nothing to do unto itself with the relationship of definition, but has to do (arguably) with mereological relationships.
However, for the sake of simplicity here, if not otherwise explicitly precisified, I shall use throughout the PhD dissertation interchangeably "_is defined as_" and "_is constituted by_", or, more generally, the difference between *definition* and *constitution* can be safely avoided, although we have to be aware that we are committing here a *mereological fallacy* (MF) (cf. Sec. 4.1.2), in so far as we can – either properly or prima facie - define a definition along a *constitutive* line.
In other words, finding out the elements of the *definiens* of *x* and finding out the *constituents* of *x* are two very different tasks, although "defining *x*" *correctly* means "finding out the intensional/constitutive parts of *x*" (with other, of course, *plausible* or admissible usages of "definition" ("essence" or even "intension") like in our talk about extensional *definitions*), for the relationship of constitution is here analyzed as a specific mereological relationship viz. based on Def_3 (cf. Koslicki, 2008): that *x* is a constituent of *y* does *not* imply that *x* is an element of the definiens of *y*, but that *x* is an element of the definiens of *y* implies that *x* is a constituent of *y*.
Though it is intuitive that any theory of constitution should say something about its link to *mereological* notions like (im)proper parthood, not everyone has to agree that constitution should be given an analysis as e.g. (intensional or extensional) (im)proper parthood (cf. Paul, 2010; Rea, 1995; Wasserman, 2017 for the more specific case of *material* constitution and its famous puzzles), or even that the notion of constitution has anything to do with the notion of definition.
Our metaphilosophical framework based on the constitution relationship takes, thus, the constitution relationship as a *specific* proper parthood relationship (understood itself in a certain way) (cf. Koslicki, 2008; 2018 for the same point).
(iii) It is also true that the predicate "_is defined as_" or "_is constituted by_" is sometimes used as such, in this PhD dissertation – except, of course, in easily misleading cases -, to refer to, strictly, what the predicate "_is *partly* defined as_" or "_is *partly* constituted by_" refers to.
If not otherwise explicitly specified or made implicitly clear in a certain context (e.g. giving an explanation of *x* as giving the *causes* of *x*, but without necessarily thereby defining *x*), the term "*definition*" (or "essence") (as understood here) and "(constitutive) *explanation*" can be used interchangeably.
(iv) Like many other *action* terms (e.g. "offense", "simulation", etc.) "definition" (or "essence") can (plausibly if not correctly) refer in a *polysemic* way viz. here to the *relationship* between a definiendum and a definiens, as well as to the very definiens *or* definiendum, or the (intentional) (linguistic but non-performative) *act* of defining *x*: e.g. talk about the *definition* of "disease" is, strictly speaking, talk about the *definiens* of "disease", or talk about *giving* (or enunciating) the definition of "disease" (on the polysemy of "simulation", cf. Pellet, mss.a). We largely follow here this polysemy.
(v) Strictly speaking, since intensionality is usually attributed to predicates, an intensional part is a predicate referring to a part of a specific whole and being part of the intension of the predicate referring to a specific whole. For the sake of simplicity and due to our correspondence principle (cf. Sec. 2.2.1), we can interchangeably use (and mix up) "intensional part" with "constitutive part" as well as "(specific) essential part" (or "part" *tout court*).

A second major problem facing $Def_{1\text{-}(2)}$ is that it fails to rule out *trivial* (or empty) definitions like e.g. *reflexive* ones (e.g. the sentence (or, more precisely, the declarative sentence) "a subject has the predicate of being a disease" is (necessarily) equivalent to the sentence "a subject has the predicate of being a disease (and a disease)" satisfies $Def_{1\text{-}(2)}$) or *monotonous* ones (e.g. the sentence "a subject has the predicate of being a square" is (necessarily) equivalent to the sentence "a subject has the predicate of being an equilateral rectangle or of being a disease" satisfies $Def_{1\text{-}(2)}$, as well as *spurious* definitions (e.g. the sentence "a subject has the predicate of being green" is (necessarily) equivalent to the sentence "a subject has the predicates of being grue and bleen" satisfies $Def_{1\text{-}(2)}$ (cf. Goodman, 1983)).

A way out here to this latter major problem is to talk about *intensional* (*constitutive*, or *essential*) parts of a whole analyzed in a certain way viz. as those necessary properties, which are specifically both *natural* (as existent) and *non-trivial* i.e. not logically following from a certain whole's necessary attributes (cf. Della Rocca, 1996; also Gorman, 2005; Lewis, 1986; Wildman, 2016; on trivially essential properties, Fine, 1994a; on constitutive vs. *consequential* essence, Fine, 1994b).

Thus, we obtain the following new attempt:

(Def_3) Def $(D, F, \ldots)$ iff $\Box\ (Dx \leftrightarrow (Fx\ \&\ \ldots))\ \&\ (F \frown D)\ \&\ (\ldots \frown D))$, where $\frown$ strictly stands for "_is an intensional (proper) part of_" (as defined here).

Had we to apply Def_3 to the notion of disease, then Def_3 says that to define the declarative sentence "*x* is a disease" is to find out the declarative sentence to which it is necessarily equivalent, where this specific sentence is constituted by predicates expressing the parts of a whole i.e. all the intensional parts of a whole, to which the predicate "_is a disease" is attributed. As such, Def_3 solves our above insufficiency problems through the notion of (intensional) parthood (taken in a certain way), for it is able to exclude both definitional circles and trivial (and spurious) definitions.

It is of an utmost importance to also note that Def_3 follows the the so-called Weak Supplementation Principle (henceforth WSP), according to which

(WSP) one single (im)proper part of a (*complete*) whole is necessarily supplemented by (at least) a second (non-overlapping) one.

For, Def_3 says that the definiens is a sum/set/bundle of *Fx* & ... For Def_3, a whole has an intensional (proper) part *y* of *x* supplemented by (or in conjunction with) another intensional (proper) part *z* of *x*.

The ontological version related to our idea here (cf. Sec. 2.2.1 for a correspondence principle between intension and constitution (or, essence)) is well-known and has been called "Mereological Essentialism" (hereby ME) – albeit ME is compatible, of course, with other *plausible* notions of essence (cf. Chisholm, 1975; Merricks, 1999; for criticisms on ME, cf. e.g. Olson, 2006; van Inwagen, 1981; Wiggins, 2016) –, according to which

(ME) a whole *x* has (some or all) its (im)proper part(s) *y* (& *z* & etc.) essentially (and nothing else essentially) (however understood)[6].

Although one can argue for a logical equivalence relationship between a whole and its constitutive parts, *one of the intensional parts* of a whole can be said (ontologically) prior (or more basic, fundamental, primitive or foundational) than its whole by following the idea of an *asymmetrical* relationship between a whole and one of its intensional parts (while an *anti*symmetric (rather than *a*symmetric) relationship would hold between a whole and one of its (*im*)*proper* parts)[7].

[6] Three terminological notes (with caveats) on ME: (i) traditionally, ME is most often understood in the *stronger* – barely plausible and little defended today - version that a whole has *all and only* its *proper* part(s) essentially (however plausibly or correctly understood) (cf. Moore, 2015; Wallace, 2014 on that).
However, as such, ME *tout court* should remain silent on this version and be also compatible both with the *weaker* idea that only (under given conditions or desiderata) *some* (im)proper parts of a whole are truly essential to it (e.g. candles or plants in a house are intuitively not essential to it), and with cases of *im*proper parthood, for one might very well argue that a whole has (some or all) its improper parts essentially, in the sense that e.g. its intension (or essence) would be its (absolute) extension (understood in a *reductive* sense here): e.g. a Tinkertoy house is essentially nothing else than, or nothing – *not* in the strict or numerical sense (except for a single unsupplemented improper part), but in the sense of a loose identity like partial identity - over and above, Tinkertoys) (cf. Thomson, 1983), although such an attempt obviously suffers from reflexivity. Thus, along ME, a whole has essentially some *or* all of its improper *or* proper parts.
Only under exceptional circumstances, we can very well split ME in one or other restricted version (i.e. strong or weak).
(ii) Weak or softened ME may sound almost a truism: of course, a whole has – at least – some parts essentially. However, it is not the case, for the exact *reverse* relationship has also been said, rather, to hold.
(iii) It goes without saying that, unless otherwise made contextually explicit or in situations where misunderstandings could easily occur, ME is used in accordance with our own notion of essence; ditto for the reverse relationship.

[7] Three terminological notes (with caveats) on fundamentality (foundationality, basicness (strictly taken), grounding (strictly speaking), primacy or primitiveness): (i) "fundamentality" seems to be an umbrella term referring in a two-fold way: (i) to a specific mereological relationship viz. a constitutive or ontological *priority* relationship (vs. a derivative or emergent relationship) (plausibly if not correctly understood): e.g. a genus as being more fundamental than a species (cf. Box 3.1.1); a disease as being more fundamental than a disease kind (cf. Box 4.2); etc.;
or (ii) to a relationship of specificity or *grounding* (restrictedly taken) (on absolute extensionality, cf. Exc. 3.2) (plausibly if not correctly understood): e.g. disease as being more fundamental than a kind/species; lung cancer as being more fundamental than cancer; etc.

In that sense, there is a unique direction of a constitutive explanation between the predicate *D* and the predicates *F* and etc. or a definitional (and ontological) priority of the parts expressed by the predicates *F* and etc. over a whole expressed by the predicate *D*: the predicates *F* and etc. are both used for defining *D*, but not vice versa.

Of course, one should be aware that there are other *plausible* specific ontological and definitional priority relationships: e.g. it may be argued that the *im*proper parthood relationship is one of them (*pace* Correia, 2017), where the improper parthood relationship is to be interpreted as a *strict*, absolute, total or numerical (synchronic or diachronic) identity or sameness relationship (under the proviso that talk about a single unsupplemented improper *part* makes sense) or as a *loose* identity relationship: e.g. a *many-one* or *partial* identity (Armstrong, 1978; Baxter, 1988). One could describe such an attempt in the following (here) extensionalist way:

> [a] Tinkertoy house is made of Tinkertoys. And surely a Tinkertoy house is made only of Tinkertoys: surely it has no additional ingredients, over and above the Tinkertoys it is made of. (Perhaps there is such an entity as 'house-shape'. Even if there is, it certainly is not literally part of any Tinkertoy house.) (Thomson, 1983, p. 201)

In other words, a Tinkertoy house would be nothing over and above the different Tinkertoys composing it (as improper parts).

Against ME, it can be argued that there is a priority of the whole over its (im)proper parts. The thesis of the priority of the whole over its (im)proper parts is a very well-known and controversial Aristotelian thesis that we may label "Reverse Mereological Essentialism" (henceforth RME) (cf. Inman, 2017; cf. also Koslicki, 2008) according to which

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(ii) There is intuitively a third *plausible* way – inconsistent with way (ii) -, in which "fundamentality " refers i.e. to (iii) a *generality* relationship (vs. specificity): e.g. (the ontological category of) kind/species as being more fundamental than (the one of) disease; cancer as being more fundamental than lung cancer; etc.

Way (iii) to understand fundamentality, as we shall shortly see (cf. Sec. 3.3.1), is not a genuine relationship – the reverse relationship is genuine, instead, i.e. from generality to specificity, properly taken (cf. way (ii)), but which is not to be confused with way (i) i.e. the relationship of ontological priority, strictly taken.

In the following, the notion of fundamentality (or e.g. basicness) is used in either the correct way (i) or (ii) or the plausible way (iii), albeit we have to be aware that, with way (iii), "fundamentality" does not refer to a genuine relationship.

Although the context usually makes clear enough in which way (i), (ii) or (iii) we are referring with "fundamentality", if it is not the case, then it is made somehow explicit.

(iii) Finally, fundamentality, *correctly* taken, does not concern intuitively other existing entities than species/kinds – at least, as one of the relata -, for those other entities have no definitional structure, properly taken, without that preventing to plausibly speak of such entities as being somehow more *indirect* than other ones, or maybe even as being *ultimate* (plausibly taken i.e. not necessarily associated to fundamentality, strictly speaking), of course.

(RME) (some or all) (im)proper part(s) *y* (& *z* & etc.) of a whole *x* is/are essentially (a(n)) (im)proper part(s) of the whole *x* (and nothing else essentially) (however understood)[8].

> [F]or example, in a circumstance which we would ordinarily describe as "Joe accidentally cut off his hand at noon", the object (hand, *in one sense*) that is attached to Joe's body until noon is not numerically identical to the isolated object (hand, *in a distinct sense*) that is not part of any living body […]. (Koslicki, 2008, p. 112 n. 26; my emphasis)

In other words, the illustration of the severed hand being a hand "in name alone", by contrast with a hand of a living being, used ad nauseam in (neo-)Aristotelian scholarship, clearly shows that it is essential for a hand of a living being to be a(n) (im)proper part of the whole (a living being), which it is a(n) (im)proper part of (under the hypothesis that a *severed* hand is not any more a(n) (im)proper part of the whole in question[9]).

RME suffers from a central difficulty. This difficulty has to do with the implausibility of RME itself, for, according to RME, a part of a whole cannot exist separately from the whole of which

[8] Note that ME and RME can be read both at a concrete or token level or at an abstract or type level.
However, since we are taking here ME and RME to be related to a certain metaphilosophy, where we, as philosophers, are typically interested in *kinds* (cf. Ch. 3), ME and RME are read (or understood) here, actually, both at the abstract level or, more precisely, at the level of *kinds*.

[9] Two terminological notes (with caveats) on scattered objects (cf. also Exc. 2.2): (i) the above assumption is sound, for token intensional parts are to be (intuitively) *spatially contained* in a token (positive or negative) whole (cf. Exc. 5.1); the notion of spatial containment allows us to deny the possibility for a token whole – be it *positive* or *negative* - to have a token *scattered* (detached, disaggregated, isolated, or, properly speaking, dis*located* or dis*lodged*) intensional part i.e. a token (positive or negative) whole, whose one token intensional part does *not* occupy a part of the space-time region occupied by the token whole.
Indeed, we may argue that the exact (or most extended possible) space-time region occupied by a token whole is fixed by the *nature* of a whole, or spatial containment is understood as a relationship between parts of a space-time region and a space-time region, that a whole – more precisely a substrate or *substratum* (or a *medium*) - needs such that its nature be correctly tokenized or instantiated.
(ii) Resorting to some (concrete) glue, structure (taken in a certain way), manner of arrangement, shape, compresence, principle of unification, organization, interdependence, substratum (as e.g. a bare particular/entity) or (substantial) *form* (under a certain plausible understanding) (Ari. *Met*. Δ.25, 1023b12-25; cf. also Dumsday, 2016)'s own distinction between a whole which is a form, and a whole which has as a constitutive part a form i.e. a whole which is partly a form; also e.g. De Haan, 2018; Jaworski, 2016) binding together (or unifying), *as (token) intensional parts themselves*, all the other (token) intensional parts of a (token) (positive or negative) whole does not help solve *this* issue here (*contra* Hommen, 2019; Koslicki, 2008; 2018; on the famous topic of compresence and its problems, cf. e.g. Austin, 2008), for such (token) *formal* or *structural* (plausibly speaking) components (or parts) may be said to be, actually, mere (token) *relational* properties, which do not prevent the relata in question to be possibly, thus, *scattered*: e.g. a (token) specific chemical bond between a (token) H_2 and a token O (for a related point, cf. Paolini Paoletti, 2018; on (said) microstructural essentialism about water, Hoefer & Martí, 2019). Indeed, if binding together (token) intensional parts just is making them enter into a certain relationship, then this very relationship may hold with relata deemed scattered.

it is a part, or, more simply put, it is essential for a part to exist that the (*complete*) whole of which it is a part *already* or *synchronically* exists[10, 11].

That much implies that there would not be any emergent (or derivative) properties (or no coming into being of a whole), in the weak sense that a world would have always been there (e.g. since the very onset of the earth system for the actual world) at the same hierarchical level (of complexity), since a complete whole would be ontologically prior to one of its intensional parts (or, a whole would be already *completely* there temporally before the existence of one of its intensional parts)[12]; the thesis of the ontological priority of the whole over (one of) its parts is also known as "priority monism" (Schaffer, 2010; Trogdon, 2017).

The main problem at stake here with such a construal is that it goes against our description, picture or conception – minimally taken i.e. vs. e.g. *conceptualizations*, strictly understood - of what a whole is.

Indeed, if we seriously follow RME, then a whole need *not necessarily* its (im)proper parts to exist – RME does not pronounce on a definition of a whole -, but the (im)proper parts need their whole to exist; but, what is a whole, if it is not (minimally and uncontroversially) something having (im)proper part(s)?

However, we can answer here that a more charitable reading of RME is possible, where a certain definition of a whole as needing (im)proper parts to exist holds, although this would result, of course, in a *circular* definition between a whole and its (im)proper (with respect to RME).

Moreover, one could answer that that much is also applied to the very (conceptual) enterprise into seeking to define a *single* (simple or complex – however plausibly or correctly understood)

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[10] This is true under the proviso that the asymmetry between a *complete* whole and *one* of its (at least two) intensional parts is also intuitively understood as a *temporal* asymmetry: the existence of an intensional part *x* of a *complete* whole *y* temporally precedes the existence of the whole *y*.

Temporal synchronicity is only possible in this situation, when a (complete) whole has a single (proper) intensional part or (an) *improper* part(s) (since the (strict or loose) identity relation is symmetric) – if the notion of a single unsupplemented (im)proper part really makes sense (for a complete whole).

It is important not to confuse here between the relationship (of temporal asymmetry) of a whole with one of its intensional parts and the (*synchronic*) relationship of the *coming into being* of a whole with the gathering together (generation or *genesis*, or *assembly*) of its intensional parts.

[11] Terminological note on spatio-temporal proximity: *spatio*-temporal proximity (or asymmetry) is a relationship whose relata can be only *tokens*, for tokens exist in *space*-time (or have a *spatio*-temporal location), while *abstract* objects (e.g. kinds or types), at the global level, do not exist in space, but it may be argued that they exist (in the world/universe – which is not to be considered, thus, as the totality of space), actually, in *time* only (against the Platonist view of abstract objects as *eternal* or time-less, cf. Sec. 3.2.6; for *non-spatiality* as a criterion of abstractness, Rosen, 2017).

[12] This line of thought somehow parallels the usual criticism in certain evolutionary explanations by natural selection that natural selection does not explain the *appearance* of novel characteristics of organisms but presupposes that they are already there, and, thus, that nothing would prevent us to sustain to a "top-down" mereological picture of a world according to which parts depend for their existence on their whole – and not inversely.

entity[13] *x* (e.g. disease, disease kind, etc.), concept C (e.g. DISEASE, DISEASE KIND, etc.) or word "w" *tout court* (e.g. "disease", "disease kind", etc.) (cf. Sec. 2.2.1 for the justification of this parallel).

Indeed, if we agree that to define some entity *x* is to find out *x*'s essence, then RME tells us that to find out *x*'s essence is to find out the conditions for *x* to be a(n) (im)proper part of *y*.

However, this goes contrary to common sense thinking about what it is to define some entity *x*, concept C or word "*w*".

Indeed, when we are trying to define what *x* is, we are typically trying to find out (the set of) what *x consists* of/in or what *x* is *constituted* of, and these notions are usually taken to be closely akin to mereological notions like the (im)proper parthood relationship (Koslicki, 2008)[14].

[13] Terminological note on entities: I am using the umbrella term "(specific) entity" (or "thing", "sortal", "object", "item", "phenomenon", "taxon", "unit(y)", "substance" (highly deflationarily taken) – *contra* a huge tradition associating a substance either merely with a *state*, because of the mistaken idea that substances (as having an essence) can only be states, while processes are said not to be essentially definable (Seibt, 2017), or with an ontologically simple or independent existent entity (cf. Lowe, 2006; vs. Dumsday, 2014) -, "being" or even "(ontological) category" or "ontological form" itself or "system" (for complex kinds, etc.)) interchangeably with the word "(specific) kind/species", "whole" (or "composite (object)"), "part", "genus", "set", "element", "substratum (or medium)/subject", "*x*", "qua object", or even "property/predicate" or "word/term/notion", etc., depending on the context – thus, with any specific existing (abstract or concrete) entity (vs. e.g. a *nominalist* ontology i.e. an ontology of concrete entities only), which is described with notions understood or used either plausibly or correctly (e.g. "constitution", "essence", "intension", etc.) -, except in cases where an explicit distinction between *objects* (or *entities*) and (*real*) *kinds* or *sets*, amongst others, does matter i.e. where an object "[…] is not a[n *ontological*] category [i.e. a kind, a set, etc.]: it is the node of the universal taxonomic tree" (Simons, 2004, p. 268; tr. Simons) i.e. where it is not something existent.

With this latter distinction, I broadly follow the Austrian tradition (especially Bolzano (1837 [1931]) and Meinong 1904)) (cf. Simons, 2004 on that), which separates an *object* from an *entity* or, minimally understood, a *sortal* (viz., in our terminology, a kind/species, set, etc.) i.e. what is (the most broadly) possible or necessary.

In such cases, thus, strictly speaking, an object does not belong to a genus-*species* hierarchy, or it is not a set, or etc.; as Meinong (1904, p. 12; personal translation) puts it (for the case of a genus-species hierarchy only), "[a]n object is to be properly defined, such that it lacks a genus as well as a differentia" ["Gegenstand ist, formgerecht zu definieren, dazu fehlt es an genus wie an differentia"].

[14] This general point can be illustrated with the famous Bio-Statistical Theory (BST) of health (and disease) (Boorse, 1977; 1997; 2014). For Boorse (1977), a normal function of a part within the members of a reference class is fixed by the contribution of the part to the survival and reproduction of a whole. That much just amounts to RME, for the supporter of the BST is interested in showing how a functional part essentially contributes to a whole.

Furthermore, note that, if RME is excluded, then this also precludes one to adopt a so-called *organizational approach* to disease (Holm, 2014; Montévil & Mossio, 2015; Moreno & Mossio, 2015; Mossio & Moreno, 2010; Mossio et al., 2009; also Etxeberria, 2016; Foot, 2001; Thompson, 2008) whose basic idea is that "[…] functions are interpreted as specific causal effects of a part or trait that contributes to a complex web of mutual interactions, which, in turn, maintain the [whole] organization […]" (Saborido & Moreno, 2015, p. 87) i.e. the idea of an essential contribution of parts to a whole, which is clearly based on RME.

We can, thus, distinguish here, more precisely, between *constitutive* explanations (based on a certain understanding of ME) and teleological ones or teleological relations (based on a certain understanding of RME): e.g. the (biological) function of the heart process is to pump blood because pumping blood is *essential* to the heart process (i.e. a (partly) *constitutive explanation*), although the heart process *necessarily* has or serves a purpose or *telos*: to circulate blood (i.e. a (partly) *teleological explanation* or, more strictly speaking here, a teleological relationship) (cf. Exc. 2.3; on the requirement of relative extensionality, Sec. 3.3.1; on teleology as a necessitation relationship, Henry, 2008); or a genetic process (essentially) codes for a functional molecule (i.e. a *teleological explanation*) (on the notorious difficulty of defining a gene, cf. Fox Keller, 2000; Weber, 2005).

Another disadvantage of RME is that RME has the following counter-intuitive consequence: if e.g. my finger is essentially a(n) (im)proper part of my whole organism, and if my whole organism is essentially a(n) (im)proper part of the whole cosmos, then – under the proviso that transitivity should hold – my finger is essentially a(n) (im)proper part of the whole cosmos.

Thus, if something bad (or good) happens to my finger (or my nails), then it has some repercussion on the whole cosmos, but the following joke is usually made in this context: since when a scratch on my finger (or, the presence of my nails) could e.g. destroy the whole cosmos (or, save my life) (on chaos theory or the so-called butterfly *effect* – in the case where the relationship between intensional parts and its whole is *plausibly* seen as *causal*, of course -, cf. Bishop, 2015)[15]?

However, what about the definition of a(n) (im)proper part itself? One may still answer that a(n) (im)proper part is (minimally and uncontroversially) defined as something somehow related to a whole i.e. that a(n) (im)proper part actually needs its whole to exist.

Thus, it seems not true that we always define entities, concepts or words by spelling out what they consist of or what they are constituted of – the very notion of (im)proper part seems to prove it.

Nevertheless, this answer can be withstood, for we can argue that, in this case, for *x* to be a(n) (im)proper part of *y* is for *x* to necessarily have a relative extension (on my definition of a genus as a *constitutive* part of a species, cf. Box 3.1.1).

Indeed, one can argue that, even though ANIMAL can be said to express a part – or, more precisely in our terminology, an essential part - of a whole expressed by HUMAN BEING, it does not imply that being an animal is itself *essentially* a part of a human being; being an animal is essential to being a human being, but being essential to being a human being is not itself essential to being an animal, *pace* RME. In a nutshell, one can say that being an animal is *necessarily* essential to being a human being.

However, this answer confounds what a(n) (im)proper part is with which *wholes* themselves (e.g. an animal) can be *indirectly* judged as (im)proper parts.

[15] Two caveats on this joke: (i) this joke also applies to e.g. theories of biological functions whose bearer is good (or, biological functions *tout court*, when the context is clear enough) defined with respect to e.g. inclusive fitness, reproduction or overall capacities (Boorse, 1975; 1977; Foot, 2001; Thompson, 2008) or self-maintenance (McLaughlin, 2001; cf. also Woodford, 2016) or organizational closure; or theories of (specific) values defined with respect to another higher-level entity like a subject's overall (un)happiness (Nordenfelt, 1995; 2000; cf. also Venkatapuram, 2013), (bad) flourishing, (dis)pleasure, (negative) desire, (bad) preference (Hausman, 2015), or etc.

(ii) Cannot the very same joke be made in the context of ME – which would, thus, undermine it as a point against RME? Actually, the idea that from something happening at the highest level there is directly (under the proviso that transitivity holds) some lowest-level repercussion is much more plausible (and also widespread) than the idea that from some teeny tiny (lowest-level) process there is directly something huge happening at the highest level.

Thus, like for the case of genus (cf. Sec. 3.3.1), we have here to bite the bullet: a definition of a part – and not of *wholes* indirectly falling under it – does not follow our meta(-)philosophical framework (based on a certain understanding of ME – and not on RME), or the concept of *part* (in all its generality) should be taken as being apart from our metaphilosophy or, more precisely, our ontology of *kinds*/*species* as specific abstract entities, but still within a certain (*meta*)*ontology* or *Weltanschauung* (or worldview) (cf. Ch. 3 on that)[16].

2.2 Excursus: Scattered Objects

How does Def_3 apply to *scattered objects* (or *heaps*)? Do they not exist as specific *positive* kinds or wholes (cf. Cartwright, 1975)? What about positive objects whose nature is seemingly (or prima facie) to be scattered: e.g. an archipelago? We can perfectly well argue that e.g. an archipelago is still a specific *positive* kind or *whole*, for an archipelago may be defined such that some of its (token) intensional parts (viz. islands) are *spread* (or remote), but without meaning by this that a token archipelago can have token detached, separated, fractionated, *scattered* or dislocated intensional parts, for it is arguably part of what an archipelago is to have spread - but not scattered - intensional parts. Thus, no specific *positive* (token) kind is ever *essentially* scattered (or *dis*located).

From this, some philosophers argue that there are not, indeed, such things as heaps or scattered objects (cf. Koslicki, 2008).

[16] Three terminological notes on meta(-…-)philosophy, (meta-…-)metaphysics and (meta-…-)ontology: (i) *strictly* speaking, I take meta(meta…)philosophy as the very general philosophical inquiry into the nature of (meta…)philosophy (which may or may not coincide with (meta-…-)ontology), while, roughly in line here with the neo-Aristotelian tradition (Lowe, 2006; Tahko, 2015), I take ontology as the examination of the most fundamental (or general) reality (e.g. kinds/species, genera, substrata, etc.), and *meta*(meta…)ontology (or formal ontology, properly taken) as an inquiry into the (meta…)ontology of the most fundamental (or general) reality or the *ontological form* (on the Husserlian roots here, cf. Smith & Mulligan, 1983); (meta-…-)metaphysics concerns, thus, more specific aspects of reality, but still of such a generality that (meta-…-)metaphysics *tout court* does not conflate with other more specific or *specialized* - thus, also metaphysical somehow - branches like physics, (meta-…-)epistemology, (meta-…-)ethics, (meta-…-)aesthetics, etc. (Chalmers et al., 2009; Correia, 2005; Fine, 2012; *contra* Gracia, 1999): e.g. the (meta…)metaphysics of causation, mind, disease, health, life, or death (*tout court*) is intuitively more general (in a certain sense) than the (meta…)epistemology (or the (meta…)metaphysics *of knowledge*) of causation, mind, disease, health, life, or death.
Note also that, at an even higher level, we are used to contrast e.g. ontic (or alethic) modalities with more specific modalities like deontic or epistemic ones (cf. Meixner, 2006; also Kment, 2014).
The boundaries between all these notions are, of course, still very vague (cf. e.g. Fine, 2012) – indeed, all those terms are sometimes (*plausibly*) used interchangeably and shall be used so also in some places of the PhD dissertation -, but we do not have to bother with this pending issue for the present purpose.
(ii) Depending on the (implicit or explicit) context, our use of the term "(neo-Aristotelian) meta-philosophy (or (meta)ontology)" shall refer throughout this PhD dissertation, when *properly* understood, either to our higher-level (neo-Aristotelian) metaphilosophy presented here, or to the more specific neo-Aristotelian meta-philosophy based on our understanding of the genus-species relationship (cf. Sec. 3.1.1).
(iii) Note, finally, that, if I am right that epistemology is (indirectly) subordinated to metaphysics (cf. note (i)), then epistemology is taken as a discipline, which still (indirectly) constrains metaphysics.

However, our point does not imply that scattered objects cannot be considered specific *negative* kinds (e.g. apoptotic or necrotic DNA *fragmentation* (*degradation* or *deterioration*) – plausibly taken as biochemical hallmarks of *apoptosis* or *necrosis*; brain layer *decortication*; *dismemberment*; etc.) i.e. that scattered objects may be envisaged as destroyed *positive* objects (in a certain way) (e.g. cells, brain layers, members, or even tissues), while still having token wholes whose intensional parts are spatially contained in it.

A scattered (or, more generally, a negative) whole *has* not *its own* intensional parts *scattered* – otherwise, it would be itself, thus, destroyed, for scattering scattered objects is also a way of *destroying negative* wholes (by making them *positive*) (on the *value neutrality* of generation and destruction/privation/scattering, cf. Exc. 9.3) -, but has intensional parts of *another positive* whole for which they are scattered: e.g. while *de*composing/scattering/detaching/*dis*aggregating x into $\neg x$, we are still at the same time, actually, *composing*/attaching some*thing* (whose tokens have token intensional parts spatially contained in it) viz. $\neg x$.

Indeed, the destruction of a *positive* entity is arguably logically equivalent to the generation of a *negative* one understood as a specific kind itself (cf. Exc. 2.3). Thus, we may argue that a 10-pound cat named "Tibbles" being deprived of its tail *is*, actually, *being* (or becomes) a specific *negative* entity itself called "non-Tibbles" constituted by the deprivation of a tail (and, diachronically, of Tib i.e. Tibbles minus its tail).

Note that that much can be used as a good argument in favor of the view that lower-level negative entities like diseases are *negations* of *positive* ones (cf. Sec. 9.3.1).

The dichotomy Tibbles vs. non-Tibbles is arguably to be put along, more generally, many more cases like good or correct (biological) functions (understand: functions whose bearer is good) vs. *dys*/*mal*functions, ease vs. *dis*ease, symbiosis vs. *dys*biosis, good or correct (mental, etc.) representations vs. *mis*representations, good or correct descriptions vs. *mis*descriptions, a belief vs. a *false*/erroneous belief (or, a dis/misbelief), etc., where good or correct (biological) functions, ease, symbiosis, good or correct representations, good or correct descriptions, a belief, etc., are here said to be specific positive entities or, at least - if we claim that some are not things which can be evaluated (e.g. symbiosis) -, entities wearing somehow the trousers, although e.g. a (biological) *mal*function may still be arguably said a (certain) *correct*, normal or ordinary (biological) function *tout court*, albeit not a *good* one i.e. whose bearer is good.

2.3 Excursus: Teleological Explanations and the Weak Conservation Principle (WCP)

Our *deflationary* understanding of a teleological (or teleonomic) explanation (or teleology/teleonomy) based on RME (cf. Mayr, 1988), albeit teleological explanations are not correct (but only plausible), strictly speaking, *explanations* except for the most fundamental entities (cf. Sec. 3.3.1), for correct explanations are only *constitutive* (*pace* Foot, 2001; Thompson, 2008 - or even, more generally speaking, the mere belief in *perfection* or optimality (or maybe essences themselves) - does not require to posit (for *natural* teleological explanations, of course) the more-than-controversial (to say the least) existence of an *intelligent designer* (or just an intentional agent) (with Foot, 2001 here).

The famous Aristotelian principle that "[…] [n]ature never creates anything without a purpose, but always what is best in view of the possibilities allowed by the essence of each kind of animal […]" (Ari. *IA*. 2, 704b17; tr. Forster, 1937, p. 487) i.e. that nature (in the narrow sense here) does nothing in vain but always what is the best can be maintained (in the context, of course, of (positive) natural *teleological* explanations or relationships (if the principle is strongly taken) or of talk about *optimality*, generally speaking (if the principle is weakly taken)), however, for this principle has been convincingly interpreted by various prominent scholars as *not* implying the existence of an intelligent designer, a Cosmic Nature (to be put on a par with Plato's Demiurge) or a watchmaker (Paley, 1809) – others prefer talking about, rather, *teleonomic* explanations or teleonomy (Bartlett, 2017) -, on the grounds that "[…] Aristotle's personification of nature can only be metaphorical" i.e. as not requiring, indeed, the existence of an intelligent designer (Henry, 2013, p. 230; cf. also Lennox, 2001; Leunissen, 2010; *pace* Baertschi, 2005 for an interpretation of this principle in line with *natural theology* or creationism).

Note, furthermore, that, first, subscribing to this Aristotelian principle is compatible with acknowledging the existence of *bad* entities, in so far as the famous problem of evil (or *theodicy*) does not arise in a context where we reject, of course, the existence of an intelligent designer, but admit the one of some (natural) *mechanisms* (cf. Ayala, 1998) – be they (plausibly or correctly taken) e.g. a natural selection mechanism (cf. e.g. Boorse, 1977) or ontogeny (cf. e.g. Krohs, 2009; 2011) or even a necessitation relationship -, at the basis of a (positive) natural teleology (as assigning this idea to Charles Darwin himself, cf. Lennox, 1993) or, more generally, optimality (Baertschi, 2005).

Second, along a Weak Conservation Principle (WCP) (mostly interpreted in a restricted sense viz. to natural entities) – to be contrasted with the *strong* (Aristotelian) conservation principle that "the corruption of one thing [e.g. a tree] is [the *same* as] the generation of another [e.g. ashes]" (Ari. *GC*. I.3, 318a24-318a25; my emphasis; personal tr.) i.e. that the corruption of *x* is identical with the generation of *y* (cf. Cohen, 2012) -,

(WCP) bad entities are necessarily present i.e., so to say, in equilibrium with positive ones, or they are necessarily (but not essentially) implied by positive ones (and vice versa)

– or, they *neutralize* each other; we have, thus, to deal with them as such; the point here is *not* that a host would not be better off without e.g. diseases (for these are necessarily present), but only that these are not part of what this host (as something *good*) is.
That is why, we cannot subscribe to a view of natural selection (narrowly taken as a positive mechanism) as progressively *eliminating* bad entities (Galton, 1904; Spencer, 1898; cf. also Rolston III, 1992).
Note that, in this context, Nietzsche (1878, §224; italics original; tr. Hollingdale, 1996) has virulently defended the WCP along the following line:

> [w]herever progress is to ensue, deviating natures are of greatest importance. Every progress of the whole must be preceded by a partial weakening. The strongest natures *retain* the type, the weaker ones help to *advance* it. Something similar also happens in the individual. There is rarely a degeneration, a truncation, or even a vice or any physical or moral loss without an advantage somewhere else. In a warlike and restless clan, for example, the sicklier man may have occasion to be alone, and may therefore become quieter and wiser; the one-eyed man will have *one* eye the stronger; the blind man will see deeper inwardly, and certainly hear better.

In other words, something bad (e.g. blindness) is always accompanied by something good (e.g. hearing), and vice versa, or e.g. a specific drug has the disadvantage/drawback to have such-and-such adverse effects as negative side effects or contraindications (plausibly taken): e.g. isotretinoin, a drug against acne, if used during pregnancy, may imply birth defects; etc.
Albeit we might argue that positivity is more fundamental than negativity (cf. Sec. 9.3.1), if the WCP is correct, then we just can never get rid of negativity (and positivity).
With the WCP, generally speaking, positivity and negativity somehow stand in equilibrium, or one comes with a benefit and the other with a cost (or rather, *compensate* or adjust each other).
Two major advantages coming with a subscription to the WCP are the following: first, the WCP can easily take into account *dual-aspect entities* i.e. entities said to have both a positive side and a negative one: e.g. there is no reason not to take perfectionism as some *positive* psychological kind having as (a) *negative* consequence(s) e.g. self-depreciation or depression, etc.
Second, the WCP can explain in a certain way how it *seems* prima facie that one and the same thing can be positive or negative dependently on the context, without resorting, actually, to contextualist or perspectivist solutions i.e. by maintaining that e.g. disease is always bad or health always good (cf. Sec. 9.2.1 for examples).
A few remarks are here in order: first, one should not confuse the WCP with the idea that some positive/negative entity is (also necessarily) part of another positive/negative entity itself, respectively (cf. Exc. 3.2): e.g. working too much may increase the probability to get a stroke.
Second, the WCP does not mean that one should want, be attracted to, or cultivate, such-and-such negative e.g. affective states/processes because of the *positive* consequences they necessarily have.

Third, however, with the WCP, why is it the case that one should intuitively strive to be good (or virtuous), rather than the contrary? Is it the case, actually? As a very big question in itself, one can merely highlight here, for our purpose, that positivity is arguably more fundamental than negativity (cf. Exc. 9.3); they are, thus, the fundaments of entities, generally speaking.
Fourth, the WCP does not imply that a diseased person should be resigned to her situation, for, though negativity shall be always necessarily present, it does not mean that a *specific* disease is not worth being cured.
Fifth, the WCP (when restrictedly applied to natural entities) implies, thus, that not everything in nature (in a narrow sense) is *good*, to the contrary of a common 18th century libertine thinking trying to justify crimes by claiming that they are along the law of nature (cf. e.g. Sade, 1795, who also sometimes argues that there is in nature (in a narrow sense) nothing good or bad).
However, an objection against the WCP comes as follows: if negative and positive entities stand necessarily in equilibrium (alongside the WCP), then how can we be e.g. healthier than diseased (or inversely)? Can we?
While it is true that a healthy *x* stands always in equilibrium with a diseased *y*, what is contingent is precisely *what* is healthy or diseased: that e.g. a human being has a cancer rather than a heart murmur.

2.1.3 Third Attempt: A Constitutive and Intransitive Account

A last insufficiency problem we can point out for $Def_{1\text{-}(2)\text{-}(3)}$ is that, due to its reliance on the logical equivalence relationship, $Def_{1\text{-}(2)\text{-}(3)}$ is *transitive*.
More specifically in the case of Def_3, the parthood relationship is also commonly said to be *transitive* (but, for counterexamples to the transitivity of parthood, cf. Koslicki, 2008; Varzi, 2016). That much seems also the case for this more specific relationship that is the intensional parthood relationship: e.g. if one of the intensional parts (e.g. the differentia) of a human being were rationality, and if one of the intensional parts (e.g. the genus here) of rationality were conformity to reasons, then one would say that conformity to reasons is an intensional part of a human being.
However, one may be keen to argue that, when one gives a definition, transitivity should not hold, for one would intuitively say that, with transitivity, one is just shifting the definiendum at hand for another one (or, changing the topic of interest for another one), and one risks leaving the door open to *reductionism* (cf. Exc. 2.5).

Indeed, when I say, following Def_3, that a human being is partly (correctly) defined as rational, and that rationality is partly (correctly) defined as conformity to reasons, the fact remains that conformity to reasons partly (correctly) defines *directly* what it is to be *rational* – and not partly what it is to be a *human being*, or does so only partly *indirectly*[17]; by following more or less the same line, Kit Fine (1994b) also usefully distinguishes in this context between, respectively, *immediate* and *mediate* essence.

From this, one can easily reply that transitive relationships, of course, do not rule out the idea that one relatum is more *direct* than another – otherwise, few if any relationships would be transitive.

Nevertheless, this point can be resisted, for e.g. the relationship "_being a brother of_" is transitive, and we fail to see in what sense one brother can be said more direct than another one. $Def_{1\text{-}(2)\text{-}(3)}$ does not guarantee per se that transitivity does not hold, if one follows the orthodoxy that the equivalence relationship – and, more specifically for Def_3, the (intensional) parthood relationship - is transitive. Thus, by making the intensional parthood relationship *intransitive*[18], a new proposal has to be formalized (and explicitated) as follows:

(Def_4) Def $(D, F, \ldots)$ iff $\Box\ x\ (Dx \leftrightarrow (Fx\ \&\ \ldots))\ \&\ (F \curvearrowright D)\ \&\ (\ldots \curvearrowright D))$, where $\curvearrowright$ strictly stands for "_is a(n) (direct) intensional (proper) part of_" (as defined here).

To illustrate Def_4, we can use the famous example of the statue of clay (taken as an artefactual kind here): the statue of clay (as a whole) would be defined as something numerically distinct from its constituents, where the constituents are just (intensional) parts of the statue: e.g. clay is, of course, one of the constituents of a statue of clay, in addition to further constituents like

[17] Two caveats (with terminological notes) on intransitivity in definitions: (i) note that the mere fact of constraining the definitional relationship with intransitivity does not imply, *as such*, nor is implied by, a permissive neo-Aristotelian ontology.
Indeed, one can very well hold that definitions are intransitive, while subscribing to a minimal Quinean ontology; or one can also have a permissive neo-Aristotelian ontology without subscribing to intransitivity in definitions.
(ii) Indirect intensional/constitutive parts still bring, of course, *indirect* metaphysical constraints on a certain definition (on other constraints, cf. Sec. 3.3.1).
In the Aristotelian idiom, such indirect constituents are often called "*propria*" ("*ἴδια*"), which refer to non-essential properties flowing from a kind's essence (strictly taken). Those properties can be, in our jargon, either indirect essential properties or (in)direct necessary properties (e.g. for disease to be necessarily a specific disease).
Albeit talk about constitutive essence is redundant (if we agree that only a (said) constitutive essence is an essence *simpliciter*) and may be avoided in clear contexts, such talk is unavoidable in other contexts, exactly like we might have to talk, in a certain context, about (in)direct constitution (while constitution, when properly taken, can only be direct), or about accidental/extrinsic change (while true change can be only substantial), etc.

[18] That much is not an *ad hoc* manoeuver, for we are just here refining our previous definition of intensionality or essentiality based on Def_3.

what makes something a statue (*tout court*) or something statue-wise (for a closely related account of, more specifically, material constitution, cf. Koslicki, 2008; 2018);
furthermore, an element of the definiens of the clay shall not be, through Def_4, also an element of the definiens of the statue of clay, or shall be only *indirectly* so.
A major advantage with requiring intransitivity in definitions is that it allows us, in the biological context, to take into account without problem *genetic chimerism*, where a chimera is a *single* organism said to have *multiple* genotypes.
Indeed, first, the issue of what an organism (as a whole) is, at the general level, cannot be transitivized - which is to be differentiated from a *reduction* - to the issue of what the genotype of an organism is, for an organism only ultimately depends on its genotype;
second, it is in no way essential, strictly speaking, to a chimera to have *multiple* genotypes, where genes are to be treated as ultimate *absolute realizers* here – which can be multiple, *unlike* indirect or ultimate constituents (on my notion of gene(s) (processes) as ultimate constituents or realizers, cf. Sec. 10.1.1) -, albeit it is to be noticed that, contrarily, an absolute realizer, when correctly defined, cannot directly (or, at a same level) fall under multiple absolutely realized properties (if also correctly taken).
Indeed, the requirement of intransitivity in definitions implies the rejection of the very contentious thesis of multiple *realization* - a misnomer, in the present context - understood *here*, in our jargon, as multiple indirect *constitutivity*, actually i.e. the idea that e.g. a human being, pain or a chimera could be indirectly or ultimately constituted in multiple ways, for there is always only *one* correct way for something to be constituted by something else (e.g. a token human being with respect to indirect constituents like tooth enamel, etc.), strictly speaking (on the multiple interpretations of the multiple realization thesis, cf. Bickle, 2020).
Moreover, there is no way to (indirectly) intensionally *reduce* the issue of what it is to be e.g. a human being to the issue of, amidst others, what it is to be rational, or human intelligence to a certain genetic code, if one holds the thesis that a whole is (*numerically*, thus *qualitatively* (plausibly taken), by the Principle of the Identity of Indiscernibles (PII_1)[19]) distinct - and vice

[19] The PII_1 is usually formalized as follows:

(PII_1) $\forall F\,(Fx \leftrightarrow Fy) \rightarrow x{=}y$.

Thus, by *modus tollens*, if x is numerically distinct from y, then x is qualitatively distinct from y (plausibly taken). The converse of the PII is called the "Principle of the Indiscernibility of Identicals" (henceforth PII_2), and is formalized as such:

(PII_2) $x{=}y \rightarrow \forall F(Fx \leftrightarrow Fy)$.

versa - from (the sum of) its intensional part(s) (cf. Exc. 2.4), or that only the relationship of *proper* parthood can *correctly* capture the relationship between a whole and its (intensional or extensional) part(s), where both of the relata do exist as *irreducible* units (on *stronger* notions of irreducibility – but, most notably contrasted to *epistemic* reductionism (cf. Exc. 2.5) -, where e.g. irreducibility has to do with the impossibility for something moral to be *defined* in entirely non-moral terms, cf. Wedgwood, 1999).

2.4 Excursus: Organic Unities and Systems

The thesis of the numerical distinctness of a whole from its parts is arguably similar (but for the case of *values* only, especially aesthetic and ethical ones) to (a certain – *weak* – understanding of) the famous theory of *organic unities* (more properly, *wholes*) concisely stated by Moore (1903, p. 28; my emphasis) – which goes back, actually, to Plato (*Phaedrus* 264c) and Aristotle (*Poet*.) - as follows: "[t]he value of a whole must *not* be assumed to be the *same* as the sum of the values of its parts" i.e. that e.g. the value of disease is not numerically identical to the sum of the values of its parts (on organic unities and criticisms, cf., amongst others, Allen, 2003; Carlson, 2001; 2020; Hurka, 1998; Zimmerman, 1999).

Note, however, that the notion of organic unity (or rather, whole) has been historically more *strongly understood* (and *used*) than here viz. as a *Gestalt whole* in the Romantic era, where one insists, not about the mere numerical distinctness of a whole from the sum of its parts, but about the interdependence, integration, intertwinement or organization between *parts* making an organic (or Gestalt) whole: e.g. parts of a PhD dissertation as being dependent to each other.

Nevertheless, to remain fully consistent with our taking a whole as being *merely* (something) numerically distinct from a conjunction/bundle of parts – and not as being nothing else than something *opposite* to a sum/conjunction viz. an organized and integrated system (or a *network*), as a strong interpretation of the notion of organic whole takes it -, organizational properties of a whole can be argued to reduce to *relational* properties merely (*conjunctively* and *independently*) added to the other parts of a whole (on the network approach to mental diseases (especially psychopathology), cf. Borsboom, 2017; Borsboom et al., 2018).

Relatedly, the notion of *system* (e.g. "cardio-vascular system") may be *deflationarily* understood as merely referring to a *complex* entity i.e., here, a specific entity defined, depending on the context, as e.g. a specific kind with a differentia including many elements or relational properties (cf. Box 3.1), or as a set including many elements, etc. Thence, "system" *simpliciter* does not refer, strictly, to an organized

The conjunction of the PII_1 and PII_2 is commonly called "Leibniz's Law" (LL) (Forrest, 2010). LL may be largely followed throughout this PhD dissertation.

and integrated entity (in the strong sense) (*pace* Gross, 2011; Moghaddam-Taaheri, 2011 in the context of disease).

2.5 Excursus: Reductionism and Eliminativism

We can usefully distinguish, thanks to our distinction between (in)direct (absolute) intensionality/extensionality (cf. Exc. 3.2), between five varieties of (*ontological*) reductionism (at the level of *kinds* as well as of *types* and *tokens*): (i) (in)direct intensional reductionism; (ii) (in)direct *absolute* intensional reductionism (as modeled on the relationship of *absolute* extensionality); (iii) (in)direct *relative* intensional reductionism (as modeled on the relationship of *relative* extensionality); (iv) (in)direct absolute extensional reductionism; (v) (in)direct relative extensional reductionism.

Variety (i) is very likely the most intuitive and common one: if e.g. "human being" is strictly (i.e. intensionally) defined as a rational animal, then a human being is nothing else than a rational animal; and if "rationality" is strictly defined as conformity to reasons, then a human being is *indirectly* nothing else than conformity to reasons plus what defines animality (on so-called *Darwinian reductionism* i.e. the (intensional) reduction of entities investigated by non-molecular biology to entities studied by molecular biology, cf. Rosenberg, 2006).

Variety (ii) is very common too: if a human being is nothing else than a rational animal (as a specific *whole*), then it is indirectly reducible to what a *rational animal* (as a specific *whole* itself – and not as *intensional parts*, like for variety (i)) is: e.g. animal and etc.; variety (ii) is peculiarly used in contexts where one shows that a certain definition of a concept is reducible to a definition of another concept.

Variety (iii) is not much common: if e.g. "disease kind" has for intension a disease plus a negative cause and a negative effect (cf. Box 4.2), then a disease kind is reducible to all the different specific *wholes* that its constitutive parts can *form* – except the *absolute* one (cf. variety (ii)): e.g. a disease plus a negative cause, a disease plus a negative effect, a negative cause-effect relationship; variety (iii) should not be confused with variety (i).

Variety (iv) is, however, quite usual and intuitive: if e.g. "disease" has for absolute realizer(s) (among others, or at least) "mental disease", then disease is reducible to its absolute realizer(s); and, if "mental disease" is absolutely realized by (among others) "physical disease", then disease is indirectly nothing over and above (among others, or at least) a physical disease.

Variety (v) is the least common version of reductionism: if e.g. "disease" has for relative realizer(s) (among others, or at least) "disease kind", then "disease" is reducible to (among others, or at least) "disease kind"; and if a disease is indirectly (among others) a disease super/subkind, then disease is (indirectly) reducible to (among others, or at least) a disease super/subkind.

Varieties (i)-(v) of *reductionism* should be distinguished from *eliminativism*, where the difference between reductionism and eliminativism has to do, not with the common idea that "_is reduced to_"

would be a *two-place* predicate (or, reduction is always the reduction *of* a thing *to* another thing), while "_is eliminated" would be a *monadic* predicate (or, elimination just is the elimination *of* a thing or a reduction till *nothingness*), but with the neo-Aristotelian principle called the "WCP" (on that principle, cf. Exc. 2.3), where, in the traditional square of opposition, elimination is the process of *contradiction*, while the sense of reduction behind varieties (i)-(v) of reductionism is somehow *based* (however understood) on what is implied by it i.e. its subaltern (Strawson, 1952) – but without meaning by this that a reduction per se is implied by an elimination.

To elaborate further (or to dig deeper) on this, eliminativism may be said itself a specific case of reductionism viz. a reductionism to/from nothingness, while varieties (i)-(v) of reductionism are other specific reductionisms (implied by eliminativism).

We reject, in this PhD dissertation, all varieties (i)-(v) of (ontological) (kind/type/token) reductionism (as well as eliminativism – except for *negative* entities seen as negations/eliminations/destructions) (cf. Sec. 9.3.1 on negative entities), for we reject both intransitivity in definitions and extensional definitions (as *correct* definitions), and our metaphilosophical framework is based on the idea that, roughly, the relationship between elements of the definiens and a definiendum is a specific, respectively, part-whole relationship (cf. Def_4) (as well as on a permissive neo-Aristotelian ontology).

Note that medical reductionism is often contrasted in a pretty misfortunate way to, generally, *holism* (Fuller, 2017; Stegenga, 2018a), for holism is a metaphysical position, while reductionism is understood in this context as the (highly) weak thesis that a disease is *explained* by its lower-level parts. In that sense, it is a specific *epistemic* – but not ontological - viz. a specific explanatory reductionism (Kaiser, 2015; Sarkar, 1998; Wimsatt, 2007; cf. also Crowther, 2018 for other specific explanatory reductionisms).

Do we have to subscribe to medical (explanatory) reductionism? Indeed, many philosophers would claim that our way of analyzing definitions (or explanations) as an irreflexive (and asymmetric) constitutive relationship is clearly *reductive*.

Without that implying any *ontological* reductionism or eliminativism, we can very well defend that, indeed, in the above weak sense, our meta-philosophical framework is *explanatorily* reductionist.

However, note that medical reductionism – especially when mistakenly associated to the (bio)medical model (cf. Exc. 7.1) - is also sometimes (mis)understood as a metaphysical position viz. reductive physicalism (Stegenga, 2018a; cf. also Wedgwood, 1999 for a mismatch between the *ontologica*l thesis of supervenience and *epistemic* reductionism).

Throughout this PhD dissertation, I make explicit the different varieties (i)-(v) of reductionism (or eliminativism) – which are about kinds/species, of course - we are talking about, only in the context where it cannot be easily, of course, inferred, lest there be a useless terminological heaviness.

Moreover, except in contexts where the distinction does matter, "reductionism" (or "eliminativism") is to be read as, of course, "*ontological* reductionism (or eliminativism)".

2.2 What it is to Define Disease: The Relata

Once we are clear about what it is to *define* disease (cf. Sec. 2.1), we have still to tell some words about what it is to define *disease*. Sec. 2.2 is devoted to this point.
Sec. 2.2 is divided into two further sections: Sec. 2.2.1 investigates whether to give a definition of disease is to give the sense of the word "disease", to give an analysis of the concept DISEASE, or to find out the essence of disease.
Sec. 2.2.2 investigates whether to provide a definition of disease is to provide a definition of *a* disease or of disease*s*.

2.2.1 "Disease", DISEASE, Disease

If one says that, necessarily, the declarative *sentence* e.g. "*a* is Socrates" is (minimally) equivalent to the declarative *sentence* "*a* is a human being called "Socrates"" (cf. Sec. 2.1), then one seems to be committed to the Aristotelian view that definitions are declarative sentences, *formulae* (*Met*. Z.10, 1034b20-22), or, more specifically, *propositions* or statements.
More precisely, it resorts from Sec. 2.1 that a definition is, strictly speaking, the declarative sentence or, more specifically, proposition that a declarative sentence viz. the definiendum is necessarily equivalent to (an)other declarative sentence(s) viz. (the elements of) the definiens. Thus, a definition is a definition of a declarative sentence viz., in our case, "*x* is a disease".
However, it is justified to talk about definitions of, fundamentally (or generally), *predicates* (e.g. "_is a disease"), rather than of whole declarative sentences (e.g. "*x* is a disease"), to the extent that, if a declarative sentence is minimally constituted by a subject *x* and its predicate, then the subject *x* is taken here as fundamental (or general) or not (essentially) definable - at least along Def $_4$[20].

[20] Would we further define *x* in our case here, then one would not avoid a certain regress: the declarative sentence "*x* has the predicate "_is a subject *x*"" is necessarily equivalent to... i.e. that one would not avoid talking about a certain subject as being itself actually a predicate of another more general or fundamental *subject*.
All this should not be interpreted as implying that a *specific* subject is not, as such, further (constitutively) analyzable, but that an analysis of a specific subject will necessarily make use of the very notion we try to analyze (exactly like we still philosophize, even when we try to define what philosophy is), thus, that a certain subject will be ipso facto interpreted as actually a *predicate* of another subject.

That much means that an enterprise of defining disease actually amounts to (i) an enterprise of defining the word "disease" or the predicate "_is a disease" with other words or predicates, and defining "disease" is giving the meaning or intension of "disease", or what "disease" rigidly designates (on *rigid designators*, cf. Kripke, 1980; LaPorte, 2013; 2016; on the relationship between rigid designation and essentialism, Inan, 2008; Klima, 2002; Sullivan, 2005).

However, contrariwise, we would like to intuitively say that defining "disease" actually amounts to (ii) defining the *concept* of disease (or DISEASE *tout court*) with other concepts. In other words, a philosophical enterprise of trying to define "disease" is to be a conceptual analysis of DISEASE.

Moreover, one could also add that defining disease is not defining the word "disease" or the concept of disease, but (iii) the (specific) *property* of being a disease (or disease understood as a(n) (specific) abstract entity) with other (specific) properties[21].

In other words, defining disease here amounts to give a real definition of disease, and giving a real definition of disease is giving the constitution of, fundamentally (or generally), the property *being a disease*[22]. In that sense, a definition is here a *fact*: e.g. a disease's being *F* and etc.

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[21] Two caveats (with terminological note) on concepts, words and entities: (i) if we argue that, actually, definitions (or essences) of concepts and words are *not* to be situated outside our own metaphilosophical framework (and our treatment of cases outside it) (cf. Sec. 3.3), then we shall say that concepts and words (definitions or essences) are themselves *specific* (maybe *artefactual* or, more specifically, *conventional*) entities viz. concepts and words (definitions or essences) i.e. that conceptual and semantic definitions are still specific existing entities *tout court* (cf. Lalumera, 2010 on concepts as specific functional kinds).

Thus, we cannot, strictly speaking, distinguish (at least, at the same level) between words (or predicates), concepts and entities (or properties) *simpliciter*, for e.g. giving the intension of a certain word (e.g. "disease") just is, actually, finding out the *specific essence* of a specific (maybe artefactual) *entity* viz. a word (e.g. "disease"), where that much should not be confused with finding out the *specific* essence of another *specific* entity viz. what is neither a word nor a concept (or, what is just a *natural entity* (narrowly taken) (cf. Exc. 2.6) – not necessarily a natural *kind* – with a *plausible* reading of "essence"): e.g. here disease.

Thence, this distinction should be, rather, between words, concepts and *specific* entities (viz. what is neither a word nor a concept, or rather, just a *natural* entity, were we to argue that words and concepts (definitions or essences) are two specific *artefactual* entities), where words, concepts and specific entities (maybe just natural entities, or, instead, *specific natural* entities (narrowly taken), were we to argue that words and concepts (definitions or essences) are, actually, two specific natural entities too) all absolutely fall under "entity" *simpliciter*.

(ii) However, for sake of textual readability, if not otherwise explicitly stated or made somehow contextually clear (e.g. through talk, in misleading cases, about *specific* parts (viz. e.g. *intensional* parts), etc.), talk about essence (essential properties, definition, etc.) *tout court* is to be taken throughout this PhD dissertation as talk about, actually, the *specific* essence of a *specific* entity viz. what is neither a word nor a concept (or, perhaps what is just a *natural* entity) – contrasted here with talk about e.g. the intension of a word.

Nevertheless, in line with our *correspondence principle*, talk about e.g. *intensional* parts may be exchanged (and mixed up) with talk about (specific) *essential* parts (although this should not be done in misleading cases).

Note that note (ii) also applies to other related notions like "whole", "kind/species", "constitution", etc.

[22] Three caveats (with terminological notes) on real definitions (or real essence): (i) note that the now flourishing idea that there might exist real definitions (cf. Rosen, 2015 as a contemporary illustration) i.e., in this context, definitions whose definiendum and constitutive parts taken as *specific* wholes themselves are (specific) ontological units, actually has a rather long history in analytic philosophy, as is shown in Beaney, 2014 and Williams, 1936.

(ii) However, *contra* Rosen, 2015, we *merely* oppose *real* definitions (or real essence, or even classifications or taxonomies) (e.g. a human being as a rational animal), following the Aristotelian tradition or Scholasticism, to so-called *nominal* definitions (and nominal essence) (or, *strictly* taken, *descriptions* or characterizations) i.e. (specific) prima facie, loose, rough, neutral or *plausible* (i.e. *partly* (in)correct/(un)*real* (in the sense here), or completely

Amidst the three options (i), (ii) and (iii) about what the relata and the relation "*x* is a definition of *y*" are, do we have to pick out one of these? We can argue that our philosophical enterprise is both about (i) "disease", (ii) DISEASE and (iii) disease, by sustaining a *correspondence principle* between "disease" (as a certain natural kind term), DISEASE (as a specific conceptual kind), and disease (as a specific natural kind) (cf. Exc. 2.7), or, even more globally, between words, concepts and specific ontological units[23].

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*in*correct) definitions (e.g. a human being as a featherless biped) (on nominal definitions both generally and in the Aristotelian tradition, cf. Demoss & Devereux, 1988; Modrak, 2010) – and not to *linguistic* (or stipulative – pending a theory of words) or *conceptual* definitions (which we take just as *real* as definitions whose definiendum and constitutive parts are specific wholes themselves).
The difference between nominal and real definitions may be also given as being the difference between, respectively, what *seems* (the case) and what *is* (*real*ly) (the case).
Thus, giving a nominal definition should not be taken as the enterprise of giving the *meaning* of a word, for talk about *nominal* definitions is misleading.
(iii) If not otherwise explicitly specified or made contextually clear, terms like "definition" ("constitution", "intension" or "essence") *tout court* generally refer, throughout this PhD dissertation, to, more properly speaking, a *real* definition (or a *real* essence, etc.) (as understood here) of *specific entities* or *words* or *concepts* (vs. note (i), thus).
In the cases where a misunderstanding could easily occur, we may, rather, more carefully talk about (explicitly) a *strict*, *proper*, *correct* (vs. *literal*, which is merely opposed to *metaphorical* or analogical) or *real* definition (or essence), or about our *understanding* of a definition (or essence) – which is meant to be the (most possible) correct one, of course.

[23] Two caveats on the correspondence principle: (i) the correspondence principle does not imply the rejection of the famous use-mention distinction, for, although the word e.g. "disease" is said, with the correspondence principle, to *correspond* to the *entity* disease, "disease" and disease remain different: the former is a specific word, while the latter is a(nother) specific entity.
(ii) The crux to note here is that our correspondence principle does not imply the rejection of (a well-understood version of) what is sometimes called "applied ontology" (cf. Arp et al., 2015), "formal ontology" (cf. Basic Formal Ontology, 2017; Smith & Mulligan, 1983; as an example, Scheuermann et al., 2009; Smith et al., 2004) or "bio(medical)-ontology" (cf. Jansen & Smith, 2008; National Center for Biomedical Ontology, 2020).
I avoid using, however, the term "formal ontology" and its cognates, for we can argue that either there is nothing new or original in using the term "formal ontology" by contrast with "ontology" *tout court* – (i) a formal ontology can be said to go back to at least Porphyrian trees, as Munn and Smith (2008) take it -, or (ii) (another version of) "formal ontology" is associated with a different philosophical program to which we do not subscribe (with our mere correspondence principle): e.g. it is often claimed that a formal ontology is also a way to *represent* reality "[…] in formats understandable to both *computers* and to human beings" (Arp et al., 2015, p. xiii; my italics).
However, if a formal *ontology* is version (ii) i.e. a theory meant to represent reality in a computational way, on the grounds that it is the most useful way for knowing it, then we can raise the objection that it confuses (*fundamental*) ontology with epistemology (or non-fundamental ontology): indeed, why should the *epistemological* idea that knowledge is to be usefully stored (standardized and organized) on computers, such that to make this knowledge both unified and accessible to the greatest number of people, play any role in (directly) grounding a(n) (fundamental) *ontology* – albeit epistemology, of course, plays a role in (indirectly) constraining an ontology, but especially in the search for a more thorough and coherent picture?
Lastly, we can object that, under version (ii), formal ontology is, actually, an ontology not so much about the (very) fundamental (or general) ontological categories in the world than about how we can best *represent* (or depict) those (very) fundamental categories (viz. through computations) – which is a very different task from analyzing, defining or even, strictly taken, *describing* those very categories.
Thus, we can worry that the now burgeoning literature on so-called formal ontologies either is (under version (i)) a mere continuation of a huge philosophical tradition (with the use of formal tools meant to *match* reality as close as possible, which could be very well, arguably, computable), or is (under version (ii)) a philosophical program building databases, for it builds up a(n) (meta)ontology from an epistemology (i.e. that it *makes* reality match epistemic needs viz. computability), as well as it mixes up what reality is with representations of it, or is reducible to an ontology of representations of the fundamental categories – an ontology on which we remain agnostic throughout this PhD dissertation on grounds of irrelevance for our purpose.

According to this correspondence principle, indeed, what "disease" means or what DISEASE is matches what the specific entity referred to by "disease" or DISEASE is (on the related idea of a correspondence between knowledge of conceptual truths and of (specific) metaphysical necessities, cf. Nimtz, 2012).

In other words, in order to avoid choosing between the options (i), (ii) or (iii), we can subscribe to the general principle that to define what the property of e.g. being a disease is just is to define what the meaning of "disease" or what the concept of disease is, for our predicates and concepts fix the ontological unit, which we are talking about, albeit we do not somehow read off ontological distinctions from the structure of language or our concepts (Burge, 1993; Thomasson, 2012; *pace* Jones, 2016a) – they merely *correspond* to each other.

Any inquiry into the metaphysics of disease is, thus, a conceptual or semantic analysis of DISEASE or "disease". In other words, we can define what it is to be diseased or access to the essence of disease through our *concept* of disease or the *term* "disease".

To conclude Sec. 2.2.1 on a note of terminology, unless I am specifically talking about words or concepts, I shall, throughout the PhD dissertation, for sake of reading clarity, talk about disease *simpliciter*, since, if the correspondence principle is correct, then defining disease is defining "disease" or DISEASE[24].

The basic purpose of the present PhD dissertation is to find out the essence of disease or to define the property of being a disease, and this purpose can be achieved only be means of a conceptual and semantic analysis into DISEASE and "disease".

2.6 Excursus: On the Different Senses of "Nature"

[24] Two terminological notes or caveats on terrestrial vs. extraterrestrial diseases: (i) it goes without saying that the term "disease" does not merely refer to *terrestrial* diseases – which are, thus, *specific* diseases *simpliciter* -, but also to (possibly existing) *extra*terrestrial diseases, although our illustrations of diseases more directly fall, of course, under terrestrial diseases.
The same note applies to other related notions like "life", "death", "health", (micro/macro)organism", "disease kind", "symptom", "etiopathology", etc.
(ii) The fact that there are only terrestrial disease(s) (kinds) known at time *t* and that this may be only one possible specific disease(s) (kinds) amongst others does not have any incidence on how we are to define disease (kinds), or it does not somehow distort our definition of disease (kinds), for a definition of disease (kinds) is still meant to be universal (within kinds, of course), and it is sufficient that there are – at least – e.g. terrestrial disease(s) (kinds) to (absolutely) realize the definition in question.
Indeed, a definition of disease (kinds) shall not be (necessarily) made in accordance with, or congenial to, terrestrial disease(s) (kinds) only, supposedly because of the fact that terrestrial disease(s) (kinds) are the only specific disease(s) (kinds) known at time *t*: a definition of disease (kinds) is, rather, made to be universal (*tout court*); it has (necessarily) such-and-such (absolute) realizer, where it just happens unfortunately that *terrestrial* disease(s) (kinds) may be the only one known at time *t* (on the related so-called *n=1 problem* (for definitions of life), cf. Cleland, 2012).

The notion of nature (or naturalness) is highly ambiguous; indeed, "nature" seems to *plausibly* refer to (at least) six different entities: to, when used *widely*, (i) mere reality like in the context of artefactual kinds/entities as being still in the natural world; to, when used *narrowly*, a certain portion of reality viz. (ii) a natural entity by contradistinction with an *artefactual* one (for more on this, cf. Exc. 2.7); or (iii) a natural entity as a good one vs. a *bad* entity like in the context where a disease is said counter-natural or unnatural (Boorse, 1975; 1977; King, 1945); or (iv) a natural entity (but still with norms of correction, of course) vs. a *normative* (or (said) non-descriptive) entity (as taken, mostly, in meta-ethical debates) (cf. e.g. Zangwill, 2018); or (v) a natural entity vs. a higher-level entity studied by the *Geisteswissenschaften* (e.g. in discussions, in moral and political philosophy, around the human state of nature, or perhaps even in the debate around nature vs. nurture, or in talk about natural selection, or so-called second nature) (McDowell, 1998); or (vi) the intension/essence/nature of *x* vs. the *de*naturation of *x*.

In this PhD dissertation, when used under its plausible meaning, the term "nature" is used either widely (sense (i)) or narrowly (senses (ii)-(vi)). It should be contextually clear to which sense exactly we are implicitly referring, or which senses we are mixing up; in easily misleading cases, the exact sense of "nature" we are talking about is somehow indicated.

If not otherwise explicitly specified, talk about *natural kinds*/species (or even, more generally, entities) is used restrictedly (or, under its *correct* usage) - though this may appear as somewhat arbitrary, but we mean to situate our talk about natural kinds/entities to the traditional debate - i.e. under, of course, the narrow sense (ii) of "nature" above -, albeit "natural kind" does not carry by itself, actually, any specific pre-conception of naturalness (i.e. not necessarily by opposition with e.g. an *artefactual* kind).

A major advantage with focusing on the narrow distinction between natural and *artefactual* kinds/entities – rather than another one – is that our definition of artifactuality (and of naturalness), as we shall shortly see, is so much encompassing that the distinction between naturalness and artifactuality is able to take into consideration the other narrow distinctions.

Thus, focusing on the distinction between naturalness and artifactuality is the best starting point for providing a general definition of natural kinds/entities. In that sense, we can conjecture that the opposition between naturalness and artifactuality rightly captures the correct meaning of "naturalness".

2.2.2 Disease, a Disease, Diseases

To be clear, when I refer to disease, I more precisely refer to the *kind* disease by distinction here with the *type* disease, or a (or one) *token* or an instance of the type disease, in so far as one thinks that tokens are *particulars* taken as concrete non-repeatable entities (on the notion of

type and its distinction to kinds, cf. Sec. 3.2.6; Marcus, 2009), and by distinction with *a* (or *one*) *specific* disease like lung cancer (still situated at a kind/type level). If one says that we are interested in the meaning of "disease" or in the concept DISEASE or in the nature of disease, then one intuitively says that we are indirectly interested in the *kind* disease, by distinction with a type, a token, or even a *specific* disease.

However, philosophers usually like talking about what e.g. *an* explanation is, what *a* mechanism is, what *a* human being is (cf. Sec. 2.2.1), or even what *a* horse is; but, are they talking about what it is to be a token explanation (of the type explanation), etc. i.e. what it is for something to count as *one* explanation? or, directly about the *type* explanation, etc.? Or, are they talking about, rather, what it is for something to be a *specific* explanation (of the kind explanation), etc.? or, directly about the *kind* explanation, etc.?

According to me, these philosophers merely (plausibly) differentiate either the type by means of one of its tokens, in so far as there is no type without one token, or the kind by means of one of its specific kinds (e.g. "*a* disease like cancer is…") (for the distinction between *disease kinds* and *specific diseases*, cf. Sec. 3.1.2), to the extent that we can argue that there is no kind without one specific kind (cf. Sec. 3.3.1), where this relation should not be confused with the type-token relationship (e.g. "Waverley" from Sir Walter Scott is a *specific* (or a certain) novel (i.e. a novel itself, or a kind of novel), which has itself multiple different *instances* or *tokens* in the world (for my interpretation of the determinable-determinate relationship, cf. Sec. 3.2.3).

In both senses, talk about *a* disease is, so to say, symptomatic of an impossibility to talk about disease without talking at the same time either about a *token* disease or a *specific* disease (on the requirement of absolute/relative extensionality, cf. Sec. 3.1.2; 3.3.1), but with the idea that we still want to refer here either to the type disease or to the kind disease, respectively.

To disambiguate all this, since, in the literature about disease, one also finds philosophical essays to defining what *a* disease is, meaning by this that they really try to define either what the *type* disease is or what the *kind* disease is – where the distinction and the relationship between kinds and types is usually not made clear -, I shall more restrictedly use "*a* disease" to directly refer only the *kind* disease (since we are interested in this PhD dissertation in kinds – not in types (cf. Sec. 3.2.6)), even if it would be clearer to only talk about disease *tout court* to refer to the kind disease, which I also sometimes do (e.g. "disease is a certain negative value"). When we have to talk about the type disease, then I shall just use this very expression; when we have to talk about instances of disease, then I shall use "a token disease"[25]; when it is

[25] Three caveats about tokens here: (i) note that, if we talk about *a* token, aren't we referring, thus, to a *type* called "token"? This would result in a contradiction. By its very nature, *a* token is a concrete non-repeatable entity – and

precisely about a specific disease, I shall just use "a specific disease" (cf. Sec. 3.1.2), or other common locutions used in this case like "a *certain* disease", "the disease *in question*/*at issue*/*at stake*", "*such-and-such* disease", "a *peculiar*/*unique* (vs. *particular*/individual) disease", "the *respective* disease", "*this* disease", "(some/any/a/the) disease *x*", "*one* (specific) disease", "the *single*/*singular* (vs. individual) disease", "*different* (specific) diseases", "a *case* of disease", "a disease *case study*", "a *typical*/*special* (vs. a *species*/kind of) disease", etc.; when the disease in question is explicitly (and nearby) mentioned (e.g. "a disease *like* lung cancer", "the disease called "lung cancer", etc.), we can both use the redundant (but clear) phrasing e.g. "a/one *specific* disease *like* lung cancer", such that to emphasize that we are talking here about a specific disease and not about disease *simpliciter*, as well as the shorter phrasing e.g. "a disease *like* lung cancer", in so far as it is obvious in such an explicit context that we are talking about a *specific* disease – and not about the kind disease[26].

If one distinguishes between the kind disease, the type disease, a token disease and a specific disease, then one also has to distinguish between disease and *diseases*. The word "diseases" seems to have four different meanings.

First, when one talks about diseases, one may want to refer, strictly speaking, to the set of instances (or tokens) of (the type) disease i.e. *n token* diseases (two token diseases, three token diseases, etc.) – by contrast with referring to a single instance of the type disease.

Second, "diseases" may seem to refer to what I shall shortly call, properly speaking, "disease kinds" (cf. Sec. 3.1.1); e.g. we are used to understand a question such as "Which diseases are there?" as the question "Which disease *kinds* are there?".

However, since we strictly distinguish between a disease and a disease kind (cf. Sec. 3.1.2), we have to be careful in not confusing (the set of) diseases (e.g. diseases like lung cancer, cardiac

not itself a type plus an instantiation in a *token*. To disambiguate this, we could speak of *one* token, rather than *a* token or tokens, but both can be admitted, since a token, by its very nature, is non-repeatable. The same also applies to the notion of a *specific* e.g. disease.

(ii) We have to be careful in distinguishing between talk about e.g. a token *disease* and a token *of the type* disease. Talk about a token disease is to be put along the same line as talk about the kind disease, the type disease, etc., by contradistinction with talk about a *kind of* disease (cf. Sec. 3.1.1), etc. In other words, with "token disease", "disease" is used as a token itself, while, with "token of the type disease", "disease" falls under "type".

(iii) The relationship between a type or *universal* and a token or *particular* called "instantiation" (however plausibly or correctly understood here) should be properly distinguished from the relationship between *generality* (or globality) and *specificity* called "exemplification".

[26] Two terminological notes about the kind disease here: (i) the relationship obtaining between the kind disease and specific disease(s) can very well also obtain, of course, between a *token* disease (e.g. a token cancer) and (a) token(s) specific disease(s) (e.g. a token lung cancer). If so, then we would say that this relationship, as a specific kind, is itself tokenized. The same applies to other relationships like the intensional one, which, when explicitly precisified, can be tokenized.

(ii) As being a specific kind itself, a relationship holding between e.g. a disease kind and specific ones is, thus, a *specific* relationship, but, as long as the relata are made explicit, it is obvious that we are talking about e.g. a *specific* absolute extensionality relationship between "cancer" and "lung cancer".

arrest, etc.) with (the set of) kinds (or, more generally, *sorts*) of disease (cf. Sec. 3.1.2), although one can venture to argue that the word "diseases" can be used in its absolute/relative extensional sense[27]: e.g. whilst bacterial diseases can be said specific disease kinds themselves, or lung cancer a specific disease (*tout court*) – absolutely speaking -, disease kinds can be also said specific diseases (*tout court*) – relatively speaking.

Third, exactly like when one uses an instance of "disease" to refer to the type disease, the plural "diseases" may be in turn used to refer to the type disease. Philosophers are used to talk about defining e.g. what explanations are, etc. It is obvious that what they mean by this is not that they are talking about defining the set of instances of e.g. the type explanation, but about defining the type explanation (plausibly) differentiated here by means of the set of its instances.

Fourth, again, exactly like when we use a specific disease to refer to the kind disease, the plural "diseases" may be also used to refer to the kind disease, where the kind disease is (plausibly) differentiated by means of the set of the specific diseases.

Since, in the literature (cf. *passim*), one commonly finds philosophical papers talking about defining diseases in the sense of defining the kind/type disease, I shall innocuously shift from talking about the *kind* disease to talking about a disease or diseases (thus, in its above fourth meaning); only when I specifically talk about the set of instances of disease, about specific diseases or about the type disease, then I shall use, respectively, "instances of disease (or, token diseases)", "specific/certain/peculiar/etc. diseases", and "the type disease", even if it would be clearer, again, to only talk about disease *tout court* to refer to the kind disease. However, to follow the prevailing view here, I shall interchangeably use both "a disease", "diseases" as well as "disease" to refer to the *kind* (in our view) disease (cf. Box 3.1), except, again, in the cases where specific diseases are explicitly mentioned i.e. when it is clear that, even with the phrasing "diseases like…", we are talking about *specific* diseases.

To be fully consistent, the same also applies to the main notions related to disease and discussed throughout this PhD dissertation like constitution, disease kinds, species/kind, cause, effect, (disease) host, genus, differentia, etc. (cf. Sec. 2.1; Ch. 3 et sqq.).

2.7 Excursus: A Minimal Account of Natural and Artefactual Kinds (or Entities): Objections and Replies

[27] Indeed, "diseases" can also be used in its *relative* extensional sense here. In that sense, "diseases" would truly refer to disease kinds (cf. Sec. 3.1.2 for relative extensionality); if "diseases" can truly refer to what is in its relative extension, then it refers to minimally e.g. disease kinds alone (and, maximally, of *other* kinds), whose element of the definiens is "diseases".

If a disease kind is defined as a disease (i.e. the genus) plus an independently defined differentia (cf. Box 4.1), then we would like to say that a natural kind is to be defined, in the same vein, as something natural (i.e. the genus) plus a differentia.

However, it is hard to understand in what sense a natural kind could be really something *more* than what is natural – nor could it be defined as a kind of a certain kind (viz. natural), for "natural kind" is still intuitively in the *absolute* extension of "kind".

Moreover, a simple definition of kind/species as a genus plus a differentia absolutely applies to cases of, as commonly acknowledged (Bird & Tobin, 2017; Krohs & Kroes, 2009), both specific uncontroversial *natural* kinds (e.g. water, tiger, etc.) and (more) controversial ones (e.g. beauty, truth, pain, gender, disease, etc.), as well as specific uncontroversial *artefactual* kinds (e.g. a Turing machine, a car, any *engineered* object, etc.) and (more) controversial ones (e.g. University, money, Newton's cradle, a perpetual motion machine (at least, in a *meta*physically possible world, if not in a *physically* possible world, because of, as widely acknowledged, the violation of the first and second laws of thermodynamics), etc.).

How to account, thus, for the distinction between a natural kind (or entity) and an artefactual one (or a *creat*ure, narrowly taken)? As a whole PhD dissertation may very well be written on this hugely controversial topic, I can only briefly touch the issue here.

The distinction between a natural and an artefactual kind/entity is usually, basically, drawn through the venerable distinction (at a more general level) between, respectively, a *mind-independent* (or *objective*) and a *mind-dependent* (or *subjective*) kind/entity (against natural kinds as mind-independent, cf. Ereshefsky, 2018).

How are we to understand mind-(in)dependence? It is common to hold that a mind-dependent kind/entity is a product of an intentional (e.g. human) action (i.e. an invention, creation, device, fabrication, construction, or even coinage (here for artefactual kind/entity *terms*)), while it is not the case for a mind-independent kind/entity (cf. e.g. Burge, 2010; Hansson Wahlberg, 2014; Searle, 1995; von Wright, 1963; on other plausible understandings of objectivity, Jukola, 2017).

More specifically in the context of kinds (and types), we can argue that an artefactual *type* is a type whose *any* token is *necessarily* brought into existence *only* by a specific (e.g. human) subject through e.g. its perception, desires, preferences, intentions, pleasure, etc.: e.g. a token disease (re)created in a lab, a token living being created (as a whole) as a (token) *clone* (vs. e.g. the creation of a token *robot* or android) or a token artificial lake is as much natural as a token disease, living being or lake not created by a certain intentional action.

Indeed, had a subject not created the token in question, the type would still, nevertheless, (go on to) exist – to the contrary of an *artefactual* type.

If we agree that natural and artefactual kinds are both, or *exist* both as, *specific* (real) kinds, or are out there in the world, in the reality or the *nature* (widely taken) - which absolutely fall under "kind/species"

-, then a *natural* kind may be more strictly *defined* as a mind-independent genus (i.e. a genus not produced by a subject's (e.g. human) action) plus a mind-independent differentia, by opposition with an artefactual kind, albeit definitions of natural and artefactual kinds may be said both two different specific definitions viz., respectively, one given (or found) independently from humans (or other (biological) species), the other given/found dependently from humans (or other (biological) species).

There is, of course, more to say about the difference between natural entities and artifacts than merely differentiating them thanks to the distinction between, respectively, mind-independence and mind-dependence (cf. Preston, 2018); however, for the present purpose, we can stay content with the above minimal account.

Our definition of a natural kind/entity (vs. artifactuality) takes into account the other narrow senses (iii)-(vi) of "naturalness" (cf. Exc. 2.6), in so far as those senses can be easily included in our definition of naturalness, for they can be *reduced* to more fine-grained distinctions neutrally applicable to both natural and artefactual kinds/entities: e.g. sense (iii) is reducible to another more fine-grained distinction between goodness and badness; ditto for the other senses (iv)-(vi).

Indeed, our definition of naturalness, *as such*, remains silent on how to further understand the other narrow senses of "naturalness".

This way of analyzing mind-dependence or, more specifically here, a mind-dependent *kind* does not impinge on the fact that a mind-dependent (or subjective) kind is no less *genuine* (or *irreducible*) than a mind-independent kind; a mind-dependent kind is still arguably a (*real*) kind; it truly exists *as* a (specific) kind viz. a mind-dependent one (Ingthortsson, 2013;Khalidi, 2013; cf. also Lowe, 2014; *pace* Kendig, 2016).

Nevertheless, it is to be noted that Searle (1995) is well-known for defending a *reductive* account (in an absolute intensional sense) of artefactual *tokens* to so-called *brute* (or mind-independent) tokens.

By following roughly the same line (though not along token reductionism), the acknowledgment of artefactual kinds as sui generis kinds to be strictly distinguished from brute ones does not imply that artefactual kinds cannot be said (*indirectly*) constituted by, or (indirectly) absolutely realized by, brute kinds – or vice versa (cf. Sec. 10.2.1).

Furthermore, it is paramount not to equate, as it is unfortunately ubiquitously done (cf. Vinueza, 2002), *subjectivity* (or mind-dependence) with *anti-realism*, for one may want to defend the plausible view that artefactual kinds like paintings or even fictional kinds are *real* (or existent), although *mind-dependent*, i.e. that their existence (or, more precisely, essence) depends on (human) attitudes, or artefactual kinds have no independent existence from humans or other (biological) species (by contradistinction with natural kinds).

While the dichotomy *objectivity* vs. *subjectivity* has arguably to do with the dichotomy *mind-independence vs. –dependence*, the dichotomy *realism* vs. *anti-realism* has arguably to do, by contrast, with the dichotomy (mind-(in)dependent) *existence* (*reality* – which maximally contains both the *actual* world/universe and *possible* ones (or the world *tout court sensu lato*) (on modal realism, cf. Lewis, 1986)

-, or the presence of an essence (or under any *plausible* reading of "essence")) vs. *non-existence* or *unreality*.

Thus, the general debate around scientific *realism* vs. antirealism is to be perhaps better put along the line of, more properly speaking, scientific *objectivism* vs. subjectivism (or anti-objectivism) – albeit, to situate oneself within a certain well-established historical debate, talk about scientific realism can be still plausibly maintained, of course, and a certain scientific antirealist may very well want to be truly antirealist *in lieu of* antiobjectivist (on the vagueness behind the label "scientific (anti)realism", cf. Chakravartty, 2017).

Is not subjectivity itself *objectively* defined, actually? Thus, what we are calling "artefactual kind" runs the risk of being merely a *specific natural* kind itself. The mistake here is to confuse *objectivity* with *realism*; our definition of an artefactual kind just is *realistic*: an artefactual kind is a *specific* (real) kind (*pace* Nagel, 1997).

Applying our account to disease and related kinds, is disease a natural kind? I take *disease* and related kinds (cf. Ch. 3 et sqq.) (or a *value*, in all its generality) as a specific (existing) *natural* kind: e.g. were all human beings (or some other (biological) species) to disappear (or, could we or other species not (re-)create tokens of the type in question any longer), sunsets would still *be* (intuitively and *essentially* here) *beautiful*, a wood thrush's song would still *be melodious*, bees (or ants) would still *be eusocial* (cf. Wilson, 1975), or dogs could still *be diseased*, or there were also diseases (e.g. diseased dinosaurs) before the advent of any intentional agent (cf. Loughlin & Miles, 2015). In that sense, a certain disease is perfectly *natural* (Rolston III, 2001; against normal functions as natural or objective, cf. Amundson, 2000) – that much is no derogation, of course, to the relative extensionality requirement, which is *not* about the *meaning* of terms (cf. Sec. 3.3.1).

Does this mean that e.g. artworks or monuments (as specific *artefactual* entities) cannot *be* e.g. beautiful, or a robot cannot be healthy/diseased (on pain of a category mistake)? A way out here (though not a panacea) is to argue that they, rather, somehow (indirectly) *represent* (imitate, or are inspired by) something naturally beautiful or healthy/diseased. This point should be obviously taken into account in a more complete theory of artifacts (on that, cf. Petroski, 1992; also Pellet, 2019).

Objectivity is to be strictly distinguished, thus, from any value reductionism, value-free or anti-value-laden analyses. Because the philosophical literature on disease (and health) often (wrongly) implicitly assumes a tight connection between values and subjectivity (cf. e.g. Glas, 2019 for the case of mental diseases), any (said) objective theory of disease (or health) is mostly (wrongly) classified as, typically, a value-free theory of disease (or health) (cf. Boorse, 1977; Varga, 2011; against D'Amico, 2007). Note, however, that this mistake is (surprisingly) far less common in the debate about well-being (cf. e.g. Schramme, 2017 for a synoptic perspective).

Three general objections can be raised against the above account of a natural and artefactual kind: (i) several philosophers (cf. Hansson Wahlberg, 2014) hold that some *artefactual* kinds/types (e.g. a cup, a

sheet of A4 paper, a perpetual motion machine, Newton's cradle, etc.) are such that they would still exist, if human beings (or other species) were all to disappear.

Nevertheless, to directly address objection (i), we can maintain (here with Searle, 1995) that, had we (or other (biological) species) not created such kinds/types, then, anyway, they would not (have) exist(ed).

Moreover, we can argue, against orthodoxy (cf. Marcus, 2009), that a *state* is an *enduring* thing in the *minimal* sense that it exists *at time t*, for *instantaneous* states (or *instants*) are still arguably states themselves (for the same point, cf. Stout, 2016): e.g. a cup (as a specific artefactual *state*) can, minimally, *instantaneously* exist at time *t*, when we (or other (biological) species) create it; it does not have to *continuously* or even *pro tem* (temporarily, momentarily or provisionally) *persist* (across time or over a stretch of time) by *enduring* (or, to be *long*-lasting by enduring) (even when human beings (or other (biological) species) do not exist anymore).

Note that the same is true for a process itself: albeit a process unfolds through time (or *perdures*), it can do so *endlessly*; for, if a process is minimally understood as substantial change for a *state* (cf. Box 9.2), then a process does *not* cease to exist, when the state is out there; the presence of the state just is the *end* of the process itself, because the complete fulfillment of the *process*'s essence requires that a state be present – not *until* a state be present (*pace* Kaiser & Krickel, 2017) – albeit a state can be, of course, just *instantly* present.

As a second objection, we could argue that (ii) an artefactual kind/type just is a kind/type whose *origin* is to be found *only* in (human) attitudes, etc., so as to exclude the case where e.g. a token tree which was grown in one's garden (i.e. whose seed has been planted in one's garden) would *not* be as much natural as a token tree which grew up in a forest, or a token IVF- or C-section-procreated baby with respect to a token non-IVF- or non-C-section-procreated baby; etc.

However, against objection (ii), it is completely irrelevant that e.g. a human being be at the origin of, or act for the emergence of, an entity for this entity to be called "artefactual".

Indeed, even if a human being is at the origin of a token, whose type would not exist without this human hand, the type in question is still intuitively natural, and not artefactual; it is counter-intuitive that such types can be claimed mind-dependent: e.g. a token disease not found anywhere else except in a lab, which is at the origin of its existence, is perfectly natural (in the specific narrow sense we are interested here, of course); a gene-edited baby or person, whose genome was edited/engineered by a human hand, is as much natural as a non-gene-edited baby or person; GMOs are also non-artefactual; or a bionic human being (or human cyborg) (e.g. with a dental prosthesis made of synthetic ceramics), however not altogether healthy he can be, is as much natural (vs. artefactual here) as a non-bionic human being.

Note that our reply to objection (ii) is in line with an *anti-reductionism*, where, for *x* to be artefactual, *x*'s (direct) constituents must depend on e.g. human attitudes/actions – and not *indirect* ones.

As a last objection, we can argue that (iii) modifying a natural kind such that the kind would not be found in nature without this modification just is what makes a kind artefactual: e.g. Pegasus as a *winged* white horse; or maybe antibiotics.

However, relatedly to our reply to objection (ii), if Pegasus or an artefactual plant is an artefactual kind, or antibiotics natural ones, actually, that is because, as a *whole*, it is a mind-dependent or –independent entity. On pain of a category mistake and logical inconsistency, *contra* objection (iii), the essence (strictly speaking) of a natural/artefactual kind must be entirely natural/artefactual.

Following our highly realistic metaphilosophy (also leaving room for a *largely* objectivist perspective) (cf. Sec. 2.1), I shall grant here that what I am calling "kind/species" (and related cognates) refers to, *more specifically*, a(n) (existent) *natural* kind, even though our definition of kind/species, *as such*, encompasses the one of an artefactual kind too.

That is why, I shall just use the term "kind/species", where it is, strictly speaking, a *natural* kind. (I will explicitly use the term "natural kind" or "artefactual kind" (or even "kind" *simpliciter*), only when I explicitly distinguish between the two or in contexts where their explicitness is needed).

2.8 Excursus: Values as Facts

Taking diseases as specific natural kinds (cf. Exc. 2.7) means that *evaluative*/*axiological* (or even, more generally, *normative*) properties are, actually, specific *natural* properties i.e. are within the furniture of the world independent from us or other specific (biological) species (cf. Brink, 1989; Railton, 1986).

This does not imply, of course, that normative properties are conflated with, or ((absolutely/relatively) intensionally) *reduce* to, *non-normative* ones (as specific natural properties themselves) – thus, that a(n) *is-ought* (or *naturalistic*) *fallacy* is committed, at least taken here in a certain (weak) Moorean version viz. that a naturalistic fallacy is committed, when normative properties just *reduce* to non-normative ones (on this famous fallacy and its diverse interpretations, cf., amongst others, Ball, 1989; Foot, 1958; Kaiser, 2015; Moore, 1903) -, but only that normative properties are *specific* natural properties themselves; exactly like bodily properties can (indirectly) *constitute* (*without reducing*), or (indirectly) absolutely realize, mental/social ones (cf. Sec. 10.2.1), non-normative properties can also (indirectly) constitute or absolutely realize normative ones (cf. Leary, 2017 on that general idea; *pace* Toppinen, 2018).

The rejection of a distinction between so-called *facts* and *values* or rather here, between, respectively, *natural* properties (narrowly taken) and *evaluative* ones is a common strategy in neo-Aristotelian philosophy, where it has been prominent especially in moral philosophy (on (non-reductive) ethical naturalism (sometimes misleadingly called, rather, "non-naturalism", where "naturalism" is unfortunately considered under its reductionist acceptance only) and virtue ethics, cf. Baertschi, 2014; Foot, 2001; Hursthouse, 1999; Nussbaum, 1993; Thompson, 2008; Zangwill, 2018; Megone, 1998a; 2007 for the case of mental diseases; also Rasmussen, 1999; Wrigley, 2007).

Thoroughly analyzing the relationship between normative (or, more restrictedly, evaluative) properties and non-normative (or, non-evaluative) ones is a complex task.

We can stay content by claiming here that values like disease or health can be arguably said specific *higher-level* natural properties *constituted* by (i.e. *irreducible* to) lower-level/-order natural (as non-normative) properties (on the related analogy between values and higher-order properties like colors (as *secondary qualities*) – but commonly taken in a subjective way -, cf. McDowell, 1985; on the philosophy of color, Maund, 2018).

However, first, a well-known recalcitrant issue with (especially) objectivist views on normative (e.g. axiological – especially *moral*) properties is that, contrary to anti-objectivist views, their link to *motivation* (or, phenomenology, more generally) seems rather obscure or even inexistent (Mackie, 1977; Tappolet, 2000).

With respect to negative values like disease, albeit being a disease is per se a specific *natural* property, if I am right that e.g. negative (or irrational) desires, preferences or emotions (still real and objective) just *are* (indirectly) (in the absolute extension of) diseases themselves (cf. Sec. 4.1.2), and if negative desires, preferences or emotions are, indeed, *motivational* (processes), then this issue does not arise in the context of the value of disease: e.g. a lower-level disease like pain is *necessarily* (cf. Sec. 3.3.1) – though not essentially – relatively realized by a higher-level disease like (indirectly) a negative desire or maybe just a certain bad behavior like crying or screaming.

Ditto for other cases like a feeling of fear (of e.g. a dangerous dog), where this feeling of fear is certainly merely constituted by a certain *attentive* (or *experienced*) access to - under the guise of e.g. a certain perceptual experience of - a dangerous dog (as the intentional object in question), or by having a (mental) *representation* of a dangerous dog (on the closely related thesis of *intentionalism* or, better, *representationalism* about intentional/representational states/processes, cf. e.g. Bain, 2003; Mendelovici, 2013), and where the feeling of fear is very likely (indirectly) relatively realized - but *not defined* - by other complex bodily feelings or an action readiness (Frijda, 1986), just like a belief itself is also certainly relatively realized by a practical reasoning.

Of course, therapy should still be targeted at the precise level, where the disease in question is – not, where its (indirect) realizer(s) are.

Second, the relationship between normativity and *epistemology* is also rarther obscure. One can easily argue, however, that normative properties are, on the one hand, an indirect way of *knowing* (non-normative) things in the world, and, on the other hand, they are also easily *knowable* as being indirect absolute realizers of (non-normative) things.

3

DISEASE VS. DISEASE KINDS

The very concept of disease is far from being clear; indeed, DISEASE is linked to so many different other concepts that it shall be worthwhile for sake of clarity, first of all, to tell some words about what disease is intuitively not.

Such that to better grasp the elusive concept of disease, Ch. 3 purports to clear the path towards a definition of disease by intuitively distinguishing between DISEASE and another related concept viz. DISEASE KIND. This distinction allows us to already take a stance on the debate about what disease is (not): a disease is not a class of disease(s) (kinds). Thence, we reject the widely accepted basic idea behind the famous Bio-Statistical Theory (BST) of health (and disease) viz., in our case, that the notion of disease is statistically established.

Sec. 3.1 begins with a preliminary distinction between our search for what disease is and a search for what a disease kind is. I argue that the notion of disease is ontologically and definitionally prior to the notion of disease kind, in so far as a disease kind is a species falling under the genus disease with a certain differentia.

Sec. 3.2 raises and addresses some objections against our understanding of the genus-species hierarchy.

Sec. 3.3 is an analysis of, more specifically, our definitions of a genus and a differentia; it also raises and directly addresses a specific objection which is an intuitive consequence of our analysis (or understanding) of the genus-species relationship viz. that a disease would be (essentially) a constitutive part of a disease kind.

3.1 Disease and Its Kinds

While Sec. 2.1 and 2.2 were about what it is to *define* disease or to define *disease*, it is now time, in Sec. 3.1, so as to avoid misunderstandings, to tell some more precise words about what our inquiry into the metaphysics of disease really amounts to. Sec. 3.1 aims at making another (more substantive) distinction between a *disease* and a *disease kind*. When one is looking for a definition of the concept DISEASE, of what disease is, or of the word "disease", the most obvious distinction to be drawn, at the outset, is the one between what disease is and what a disease *kind* is as well as what a disease (super/sub-)super/subkind is.

In Sec. 3.1.1, I distinguish between the conceptual clarification enterprise into what DISEASE is and into what DISEASE KIND is. It is important to be clear concerning their relationship. I argue that the former is ontologically and definitionally prior to the latter, if we follow our philosophical methodology (cf. Ch. 2).

Sec. 3.1.2 examines whether and how we can further distinguish between DISEASE KIND and DISEASE (SUPER/SUB-…-)SUPER/SUBKIND. I argue here that two common ways to define DISEASE (SUPER/SUB-…-)SUPER/SUBKIND are to be both rejected. That much does not imply that there is no such concept as DISEASE (SUPER/SUB-…-)SUPER/SUBKIND (indeed, if we argue that a term should have a *relative* extension (cf. Sec. 3.3.1), then "disease super/subkind" is maybe an interesting candidate for the term "disease kind", albeit we shall not pronounce on that point)[28],

[28] Three caveats (with terminological notes) on (a certain understanding of) "_more than_": (i) if "disease kind" necessarily has a relative extension under the guise of a disease super/subkind, then this may be conceived of either "disease superkind" or "disease subkind".
Thus, we can use interchangeably (for our purpose here) "disease subkind" or disease superkind", or, without choosing, simply talk about disease super/subkinds, as long as we are clear about the fact that the dichotomy *super-x* vs. *sub-x* is not the same as the dichotomy between something *more than x* and *less than x* (cf. caveat (ii)).
(ii) If we argue, as we should (cf. Sec. 3.1.2 for rejected definitions), that talk about a disease super/*sub*kind is to be basically understood along talk about something *more than* a disease kind, then, from this, one may be easily *mis*led to the conclusion that talk about a disease *super*kind would be talk about something *more* than a disease kind, while talk about a disease *sub*kind would be talk about something *less* than a disease kind.
However, that much comes from an equivocation on the expression "more than (e.g. a disease kind)", which does not mean here that a certain *intensional* part is *added* to e.g. a disease kind *itself* – which would be against ME -, but that something *extrinsic* to a disease kind (e.g. a specific differentia or genus) is added or *assembled* to it.
Although it may be weird to talk about e.g. a disease subkind as something still *more* (in our terminology) than a disease kind, this becomes less weird, once we distinguish between a disease *sub*(/super)kind and a disease *hypo*(/hyper)-kind (on *super*/*sub*functions vs. *hyper* (or *over*-)/*hypo* (or *under*-)functions, where talk about a disease *hyper*/hypo-kind is here talk about something *less* than a disease kind i.e. a disease kind *losing* (or being *deprived* from) his *intensional* parts, cf. Sec. 9.4.2).
(iii) Finally, we may argue that "_more than *x*" is not to be merely understood as talk about *x* plus a certain differentia (cf. caveat (ii)), but it may be also more trivially understood as talk about a *normal x*: e.g. being more than happy, welcome, problematic, exaggerated, etc., just is being normally or very (very) happy, welcome, problematic, exaggerated, etc.
However, we should avoid using the expression "_more than *x*" as such, for e.g. being more than happy is reducible, along this line, to being more than *defectively* (or *im*perfectly) happy i.e. being *less defectively* happy, or (*perfectly* or *normally*/ordinarily) happy *tout court*.

but only that two common ways to define DISEASE (SUPER/SUB-…-)SUPER/SUBKIND (absolutely intensionally) reduces to either DISEASE KIND or to DISEASE. This also explains why we shall, next, dwell on the definition (or theory)[29] of disease kinds (cf. Ch. 4) – rather than disease (super/sub-…-)super/subkinds.

3.1.1 Disease Kinds: A Primer

Any philosophical investigation into what disease is is to be distinguished from an investigation into the classification[30] of disease *kinds* (nosography) along various studied criteria (nosology) (cf. Ch. 4 for the notion of nosology)[31]. Disease kinds are also usually called "disease entities" (Boorse, 1977; Hucklenbroich, 2014; 2016; 2018; Whitbeck, 1977; but, cf. Jensen, 1984 for a tentative distinction between what he calls "disease kind" and disease entity; on the history of the concept of disease entity, most notably on Virchow (1958)'s pioneering work on this topic, Hucklenbroich, 2018; Whitbeck, 1977).

More precisely, the first distinction to be drawn is between the ontological unit of disease and specific disease kinds like (plausibly if not correctly) infectious (communicable, contagious or microbial) diseases, parasitic diseases, viral diseases, bacterial diseases, or Sexually

[29] Terminological note on theories: although I equate "theory" with "definition", I do not equate "theory" or "account" with "model".
My purpose is not to provide a *model* of *x* (e.g. disease kinds, disease, symptoms, etiopathology, etc.), for we are not interested in this PhD dissertation in the ways *x* is *represented*.
Indeed, we can very well (mis)represent e.g. a disease (kind) as a tin can, but this representation (or model) does not say anything about the real *nature* of disease (kinds), or is not a theory of disease (kinds).

[30] Two caveats on nosology: (i) we should be careful here not to confuse an *intuitive* classification of disease kinds with a strictly *scientific* or scientifically based classification of disease kinds i.e. a description of the taxonomy of disease kinds actually accepted in current scientific practice; the two need not coincide, even if it may happen that they overlap (cf. Ch. 4). Following my metaphilosophy (cf. Ch. 2), my purpose is to argue in Ch. 3 for a certain intuitive way of classifying disease kinds, which is, of course, necessarily realized (in the sciences).
(ii) Why reserving the term "nosology" and cognates to the art of classifying (with respect to the differentia only) what we are calling "disease kinds" (cf. Ch. 4) – and not diseases *tout court*? Albeit the term "nosology" etymologically refers to, indeed, the study of disease (*νόσος*), I am just following here the mainstream usage of the term (under its functionalist acceptance), where nosographic criteria include, generally, negative effects and causes – which correspond to our analysis of disease *kinds* (cf. Sec. 4.2.7), and not disease.

[31] Caveat on the division of labor: from the *mere* distinction between disease and disease kinds we cannot proceed to a division of labor between the philosopher – who would be interested in higher-level taxa – and the scientist – who would be interested in lower-level taxa –, or that scientists are not interested in the notion of disease *simpliciter* (though it is arguably the case: if philosophers are interested in what DISEASE is, (natural) scientists – or, more properly, scientists other than philosophers, since philosophy can be said to also study (higher-level) *natural* phenomena (widely taken) (cf. Exc. 2.1) - are likely interested *only* in *specific* diseases or in the *constituents* of a definition of disease).
As I argue, it only follows from our distinction between disease and disease kinds that disease kinds are based on the notion of disease, and giving a definition of disease is precisely our task, but, again, it does not mean that giving a definition of disease kinds is not also a task for philosophers or is reserved to scientists; it is just a different task from giving a definition of disease *tout court*.

Transmitted Diseases (STDs)[32], exactly like one may want to distinguish between what an animal is and what an animal of a certain kind is (e.g. a human being).

In a nutshell, the distinction between diseases and disease *kinds* aims at capturing the intuitive distinction between talk about diseases *simpliciter* and talk about something (or, another *kind* which is) *more* than diseases viz. disease *kinds* (cf. Sec. 4.1).

The consequences of this mere distinction are three-fold: first, this distinction is highly common in Aristotelian metaphysics and in our intuitive way of defining entities by classifying them within a genus-species hierarchy, or of applying a certain understanding of the genus-species hierarchy to a view about what it is to define entities (cf. Sec. 2.2.1)[33, 34]: e.g. a human being is a certain animal kind/species, and to be a human being is to be itself a kind/species belonging to the taxon *genus* animal (i.e. condition (i)) in a certain way i.e. in *conjunction* with a specific *differentia* (or a differentia specifica) defined independently from the definition of the genus under which the species at issue falls (i.e. condition (ii)) (cf. Box 3.1)[35].

For, if it is true, by following the idea that to define *x* has to do with finding out (the set of) what constitutes a whole (in line with ME), that e.g. a human being is defined as a rational animal (cf. Sec. 2.1), then we observe that our definition of a human being is an application or illustration of both the genus-species hierarchy as understood here i.e., in this case, that the *genus* animal and the *differentia* rationality are the constitutive parts of the *species* human being

[32] Note that the same specific disease (kind) can fall under both e.g. (plausibly) an infectious disease as well as a viral disease, in so far as this specific disease (kind) can be said (in the absolute extension of) (cf. Sec. 3.1.2) *more directly* a viral disease than an infectious disease.

[33] Terminological note on genus: throughout the present dissertation, I shall use the term "specific genus" *simpliciter* to refer to the specific genus *proximum* (except in cases where the distinction does matter) i.e. the closest (or the direct) kind under which the species in question relatively falls (cf. Sec. 3.3.1 on relative extensionality). This is, of course, in line with my requirement of intransitivity on definitions (cf. Sec. 2.1.3).

[34] This classification of entities within (our understanding of) a genus-species hierarchy corresponds to natural kinds (*contra* e.g. Jensen, 1984): the genus-species relationship (as understood here) is used to define them.
Some may want to argue that it is, actually, *non-Aristotelian*, especially for Aristotle's philosophy of *biology* (cf. Walsh, 2006 on this), for a generation of Aristotelian scholars has argued that Aristotelian *biological* essentialism (in especially *PA*) should not be regarded as *typological* (understood in a certain way), but as merely *teleological* (or explanatory) (Balme, 1987; Lennox, 2001; cf. also Richards, 2016; *contra* Linnaeus, 1735).
However, the neo-Aristotelianism I am claiming here directly hangs on Aristotle's *metaphysics* (and logic) – not specifically his philosophy of biology; more precisely, it relies on (a certain interpretation of) Aristotle's Platonist legacy widely known as the "method of (logical or dichotomous) division" or "*diairesis* (*διαίρεσις*)" (Cohen, 1973; Moravcsik, 1973; Smith, 2017).

[35] Two terminological notes (with a caveat) on necessity: (i) for sake of reading clarity, henceforth "necessity" for short refers *restrictedly* (except in some exceptional circumstances) to the different *specific metaphysical* (*de re*) necessities (cf. Sec. 3.3.1), except the one of essence *sensu stricto* (cf. Ch. 2) (where we use, rather, "essential" and cognates) and in the cases where explicit talk about *metaphysical* (*de re*) necessity is necessary (against a (*de re*) modal view of essence, cf. Fine, 1994a; Skiles, 2015; *contra* e.g. Cowling, 2013; Denby, 2014; Kripke, 1980; Wildman, 2013; 2016).
(ii) In addition to metaphysical necessity *tout court* (or broad logical necessity) and the specific ones mentioned in note (i), we can very well take as other genuine specific metaphysical necessities e.g. normative necessity (Mulligan, 2009; *pace* Fine, 2002), natural necessity (narrowly taken), narrow logical necessity, epistemic necessity (vs. Glazier, 2017), or even conceptual necessity (cf. Nimtz, 2012), etc.

considered as a whole (cf. Box 3.1.1-3.1.2) and of the idea that to define *x* is to find out (the set of) its constituents (cf. Exc. 3.1). Box 3.1 is to be read as follows: a species/kind is constituted by a genus (as itself a constitutive part of a species/kind) (cf. Box 3.1.1) in conjunction with a differentia (reducible to a species/kind or a genus) (cf. Box 3.1.2)[36].

Definition of a Species/Kind

x is a species/kind, iff (i) *x* is a genus, and (ii) *x* is a differentia.

Box 3.1. – A definition of a species/kind within a genus-species hierarchy.

Corollary 1: Definition of a Genus

x is a genus, iff *x* is a constitutive part of a species.

Box 3.1.1. – Corollary 1 of Box 3.1. A definition of a genus.

Corollary 2: Definition of a Differentia

x is a differentia, iff *x* is reducible to a species/kind or a genus.

Box 3.1.2. – Corollary 2 of Box 3.1. A definition of a differentia.

Second, the importance of the distinction between disease and disease kinds, and the focus here on disease kinds (cf. Ch. 4), should not be underappreciated, for, although no big *philosophical* debate on the topic of nosology itself has ever really emerged (cf., however, Hucklenbroich, 2014; Jensen, 1984; Reznek, 1987; Whitbeck, 1977) – but, rather, much more on the analysis of the respective nosological criteria (cf. Ch. 5-6) -, confusion around the concepts of disease and of disease kind is proliferating in the medical literature: e.g. in the use of the concept of *disease endotype* (or *subtype*), where a definition of a disease endotype ambiguously oscillates between what we call, properly speaking, "*specific* disease" and what we call, properly speaking, "*specific* disease kind" (cf. especially Sec. 3.1.2 for these notions), in so far as scientists seem to associate a disease endotype both with (i) the idea of a disease variant

[36] I thank Oliver R. Scholz (personal communication, June 2018) for having urged me to be clearer on the relationship between my definition of species/kind and the ones of a genus and of a differentia (cf. Box 3.1; 3.1.1-3.1.2).

characterized by a specific pathophysiology (or pathophysiological mechanism) by opposition with what is sometimes called a "*disease* (or clinical) *phenotype*" i.e. (or, so it seems) the disease characterized in such a way that, in our terminology (cf. Sec. 3.1.2), a disease endotype (indirectly) absolutely falls under it, and with (ii) the idea of a specific disease kind (cf. Lötvall et al., 2011; Scheuermann et al., 2009).

The oscillation between definitions (i) and (ii) is especially noticeable in sentences like "[e]ndotypes are thus a different form of classification from *phenotypes* and describe distinct *disease entities* with a defining *etiology and/or* a consistent pathophysiological mechanism" (Lötvall et al., 2011, p. 356; my emphasis)[37].

This oscillation is very likely explained by the common mistake of using both sometimes a *constitutive* analysis of a certain term like "disease endotype" (i.e. in terms of its parts) (cf. Ch. 2) and sometimes a *functionalist* analysis of the same term "disease endotype" (i.e. in terms of its effects/causes) (Simon, 2011; for the same diagnosis, Stegenga, 2018a)[38].

The explanation behind this oscillation very likely lies in the deep confusion between what a disease is and what it is to *know* what a disease is (for more on all this, cf. Sec. 4.2.7; also Fulford, 1989; 2001 for whom "disease" is used in contexts where the etiology is *known*), as Jonathan Fuller (2018a, p. 8; my emphasis) rightly points out, in other words:

> [n]ot only do scientists *discover* a specific cause of a specific type of disease, they *define* that specific disease as the disease produced by that specific cause.

Third, the very intuitive distinction between disease and disease kinds already excludes any definition of disease focused not on what disease is, but on what a disease kind is, (Hucklenbroich, 2014; 2016) or any *functionalist* approach to disease (in so far as traditional functionalist analyses of disease confound an analysis of disease with an analysis of disease kinds) (cf. Sec. 4.2.7 for that), for, according to the above Aristotelian genus-species relationship, the notion of disease is ontologically and definitionally prior to the notion of disease kind (for the central tenet of the parallel between ontological and definitional (or explanatory) priority, cf. Ch. 2), in so far as a *correct* definition of a disease kind is *non-trivially*

[37] The above authors use, thus, ambiguously the term "disease entity" to refer to what I call "specific disease" or "specific disease kind" (cf. Sec. 3.1.2). Moreover, note that other authors also use the term "disease entity" to refer to what I call "token disease" (cf. Sec. 2.2.2) (cf. e.g. Simon, 2011).

[38] The same hesitation can be also found, especially in philosophy of psychiatry, around the concept of *nosological entity/unit* (cf. e.g. Jablensky, 2012 for a constitution-oriented definition).

(cf. Sec. 4.1.1 on the requirement of non-triviality) based on the notion of disease[39]. More precisely, the definitional and ontological priority of disease over disease kinds is to be understood in the weak sense argued for in Ch. 2 i.e. here that the genus disease is a constitutive part of a disease kind which is a specific whole.

Of course, this does not mean that focusing on a definition of disease kinds cannot bring certain *metaphysical constraints* on a definition of disease (cf. Sec. 3.3.1), to the extent that a definition of disease kinds makes appeal to the notion of disease, but it still remains the case that a disease cannot be defined as e.g. "[…] the whole course of the individual case, from its very beginning or *first cause* to its *outcome* […]" (Hucklenbroich, 2014, p. 613; emphasis original), for this looks like a definition of a disease *kind* (in our terminology) (cf. Box 4.2).

Moreover, a few last terminological notes and caveats about disease kinds have to be highlighted: we have to be cautious in distinguishing between our above intuitive examples of disease kinds and the fact that we are always presupposing that what we are interested in are themselves indirectly kinds/species, genera, classes/sets or even diseases or disease kinds themselves. When we say that e.g. a STD is a specific disease kind, we can venture to say that the kind called "STD" is a certain kind of the genus called "disease"; or, it may also happen that a specific disease (e.g. cardiomyopathy) is plausibly so defined that its constituents are also themselves (what (indirectly) absolutely fall under) specific diseases (e.g. a deleterious mutation in the cardiac desmosomal gene) (cf. Harvey & Leinwand, 2011).

Even if such a presupposition may sound trivial, we should not forget, however, that, if we follow our metaphilosophical framework (cf. Sec. 2.1), then it is not *essential* for e.g. a STD to be (indirectly) (or, in the indirect absolute extension of) a kind or for herpes to be (indirectly) a disease kind (cf. Sec. 3.1.2) – or, to belong to an even more general ontological category[40]. This

[39] Two caveats on (intensional) reductionism and intensionality: (i) the present debate does not imply by itself any (*intensional*) reductionistic stance towards disease kinds and (the elements of) its definiens, for arguing for the priority of the constituents of disease kinds over disease kinds is one thing, and arguing for the reduction of the latter to the former is another thing, even if, of course, the priority of the latter over the former may be a good *argument* for the reduction of the latter to the former, but it still falls short of being a reductionistic thesis itself; indeed, one may accept the *priority* of, say, among other constituents, disease over disease kinds (cf. Box 4.2), but still argue that a disease kind is a sui generis or irreducible entity, or a whole (*numerically*) *distinct* from (the elements of) its essence. Ch. 2 of this PhD thesis has broadly argued for an irreducibility thesis; if we are to follow this irreducibility thesis, the present analysis of the priority of disease over disease kinds (cf. Sec. 3.1.1) is, thus, anti-reductionist.

(ii) Note that the fact that DISEASE is ontologically and definitionally prior to DISEASE KIND can also be *trivially* understood in the sense that DISEASE KIND is (improperly) partly DISEASE, if one allows that the improper parthood relationship be considered a genuine specific relationship of ontological and (here, trivial) definitional priority. However, as being said (cf. Sec. 2.1), albeit it might seem trivial (especially for *complex nouns* – however plausibly or correctly understood), indeed, it remains the case that improper parthood does not yield in itself *correct* definitions (for *kinds*) i.e. *intensional* ones (correctly taken), in so far as it is based on the (strict or loose) identity relationship, but merely *trivial* definitions.

[40] In the highly complex history of the theories of ontological categories (as an entry, cf. Gracia & Newton, 2012), it is important to note here that our attempt at classifying everything or every (existing) entity (with cases apart)

is also true, when we are talking about e.g. a human being as an animal *kind*/*species* – these are all very basic notions that characterize so many different entities that they are not illuminating about what a human being is by opposition with e.g. what an elephant is.

Finally, we may just wonder why what we are referring to with "disease *kind*" should not be properly called, rather, "disease *type*" or "disease *genus*". Is it a mere terminological issue? First, we can perfectly well bestow that, just like there are disease kinds, there are also disease types or even disease genera, for, in addition to kinds, there are also *types* (cf. Sec. 3.2.6) and *genera* (cf. Box 3.1.1).

However, if a disease kind is still (in the indirect absolute extension of) a kind, then a disease type and a disease genus, although they can be said to indirectly fall under a *kind*, are explicitly said by their name to indirect fall under a type and a genus. Since we are interested in this PhD dissertation in defining the (natural) *kind* disease, talking about disease *kinds* in lieu of disease types or genera highlights that "disease kind" absolutely indirectly falls under our own definition of "kind" – instead of "type" (cf. Sec. 3.2.6) or "genus" (cf. Box 3.1.1).

3.1 Excursus: Further Thoughts on the Genus-Species Relationship

If our metaphilosophy must have necessarily an absolute application (or extension) (cf. Ch. 2; Sec. 3.1.2; 3.2), then "kind/species" (defined as a genus plus a differentia) is (certainly) an absolute realizer of it – more intuitively than the other way round. Spelling out the precise relationship between a certain metaphilosophical framework and (our understanding of) the genus-species hierarchy is not an easy matter: one could say that (our understanding of) the genus-species relationship absolutely applies our metaphilosophy, and a species/kind is (one of) the highest *ontological* category(/-ies) under which all the other (more specific) concepts fall i.e. that the definitions of these concepts fall under the definition of the concept of species/kind (or that these concepts are defined within (our understanding of) a genus-species hierarchy).

Does our own way of defining a definition fall within (our understanding of) the genus-species hierarchy (if a species/kind is said (one of) the highest ontological category(/-ies)) (cf. Box 3.1), or, if not, at least within our constitutive framework, *reflexively* speaking?

Our definition of a definition as, roughly, a way of finding out the constituents of a whole (cf. Def_4) may arguably plausibly fall, indeed, if not within (our understanding of) the genus-species hierarchy, at least

(cf. e.g. Sec. 3.3), as far as possible, under a genus-species hierarchy follows, in the broad strokes, the huge neo-Platonistic tradition begun by Porphyry's *Isagoge* through Linnaeus (1735)'s hierarchical system - contemporarily continued by philosophers like Chisholm (1989) and Hoffmann and Rosenkrantz (1994).

within our own metaphilosophical framework itself (e.g. plausibly a definition as a specific whole constituted by a whole with its own constituents), albeit a definition of a definition should not be said an absolute realizer *of itself*. In a nutshell, doing *meta*(-…-)philosophy should still be consistently analyzable as a *reflexive* way of doing (meta-…-)philosophy *tout court*. That is why, we can very well use interchangeably "whole" and "kind", though they are different.

The genus-species relationship is also often understood as a relationship of *membership inclusion* (Fine, 2002). We should not *properly* analyze the genus-species relationship as such, for this inclusion relation, at least in its proper usage in set theory, is a relationship between here a class/set and its members/elements.

Moreover, if e.g. a set (in its relative sense here) (cf. Exc. 3.9) like {lung cancer} *includes* specific lung cancer kinds, in a genus-species hierarchy, actually, the reverse order holds: a lung cancer kind is constituted by lung cancer – lung cancer does not include specific kinds of lung cancer.

More specifically about species/kinds, I follow the dominant view by translating "εἶδος" (Ari. *Met*. Δ.25) interchangeably as "species" or "kind", or, without choosing, talk about *species/kinds* (cf. Koslicki, 2008).

If not otherwise explicitly specified (e.g. as a *plausible* use of the term "kind/species"), "kind/species" refers, of course, throughout this PhD dissertation, to our own definition of the term. In the cases where a misunderstanding may occur, we may, rather, more carefully talk about (explicitly) our own *understanding* of the notion "species/kind". The same caution applies to other related notions like "genus" (cf. Box 3.1.1), etc.

There are two main usages of a term like "whole" or "kind" (when used interchangeably): (i) a usage of "whole", when we talk about e.g. essential properties/constituents of a *whole*, or a *whole* as being numerically distinct from its essence, etc.; (ii) a usage of "whole", when we talk about e.g. cancer as a specific *whole*/kind.

In usage (i), "whole" is referred to as being nothing else than the *thing* in which essential properties/constituents inhere, or as being nothing else than some*thing* numerically distinct from its essence, while, in usage (ii), a whole is seen as being nothing else than some*thing plus*/*with* its (numerically distinct) essence.

With usages (i) or (ii), a *token* whole here is, respectively, a *thin* particular or a *thick* particular (Armstrong, 1989). As long as it is clear whether we are referring to usage (i) or (ii) with "whole" (or "kind"), they can perfectly well be both followed.

However, usage (ii) may be misleading, for it suggests that a whole/kind may be given a reductive *definition*.

We shall avoid throughout the PhD dissertation to use the word "form" for referring to kinds/species, for this is only one of the many interpretations of the controversial (to say the least) notion of *form* in Aristotle (cf., as entries on this topic, Ainsworth, 2020; Cohen, 2020; for contemporary versions of hylomorphism, Koslicki, 2008; 2018; Oderberg, 2007; Sattig, 2015).

Furhermore, although a correct definition of a kind/species within (our understanding of) a genus-species hierarchy is a *conjunction* of a genus and a differentia (cf. Box 3.1) (where the conjuncts are logically independent from each other), that much does not imply that the specific differentia and the specific genus given in the definition are themselves interchangeable: their *place* is interchangeable (for "_and_" is a two-place (binary or dyadic) *symmetric* connective), but, of course, *x* as a specific genus (or as (absolutely) falling under "genus") cannot be changed into a specific differentia.

Note that giving a definition of a kind in terms of a genus plus a differentia constitutively understood follows the WSP (cf. Sec. 2.1), for it says that a *complete* whole (essentially here) has a(n) (intensional here) proper part (i.e. a genus) supplemented by (here in conjunction with) another (intensional here) proper (or non-overlapping) part (i.e. a differentia). In other words, if I am right that a genus *and* a differentia are constitutive *parts* of a species (i.e. a whole), then it respects the WSP, according to which a (*complete*) whole cannot have one single (unsupplemented) part.

However, by contradistinction with a definition of a species/kind as (i) a genus in conjunction with a differentia (cf. Box 3.1), the notion of species/kind may be also understood in a deflationary vein: as (ii) the class/set (or collection/sum) of its members/elements.

Definition (ii) is a so-called *extensional* definition: a species/kind is a class/collection defined with respect to what it applies to. Strictly speaking, for this deflationary reading of the terms "species" and "kind", I shall not employ the words "species" and "kind" themselves, but rather the terms "(natural) class", "collection" or "set".

Thus, according to my terminology e.g. "water" is a (natural) *kind* term, in so far as it minimally refers to a specific genus with a specific differentia such as a certain chemical composition, while $\{H_2O\}$ refers to a natural *class*/*set*, in so far as it refers to a mere collection comprising the members/elements H_2 and O.

We are tempted to say that the distinction between my reading of what a species/kind with respect to its *intension* is (definition (i)) and the deflationary reading of what a species/kind with respect to its *extension* is (definition (ii)) is analogous to the well-known distinction, in set theory, between, respectively, *class inclusion* (where a subclass/subset is included into another superclass/superset) and *class membership*.

However, this analogy should be resisted, for class inclusion, as the name itself indicates, is, properly speaking, about inclusion between *classes* themselves – and not between a *whole* and its *parts*. Class inclusion just is class membership (both in its relative and absolute sense) (cf. Exc. 3.9), where the members of a class are deemed to be themselves specific classes, exactly like when a(n) (im)proper part of a whole is deemed to be itself a specific whole, or when a disease has constitutive parts *x* and *y*, which can be, in a hierarchy of specific diseases, specific diseases themselves viz. *x*, *y* or (*x* & *y*).

3.1.2 How not to Define Disease (Super/Sub-…-)Super/Subkinds

Directly following the distinction between disease and disease kinds (cf. Sec. 3.1.1) is the obvious distinction between disease kinds and disease super/subkinds. Indeed, if a STD e.g. is a specific disease kind, then one may be eager to intuitively say that syphilis, herpes or HIV-AIDS is an STD kind, as well as paranoid schizophrenia or catatonic schizophrenia may be said a schizophrenia kind.

Of course, one may want to go further and further, and, thus, to distinguish between disease kinds and disease super/sub-super/sub-super/sub-…-super/subkinds: e.g. herpes simplex virus-1 or -2 may be plausibly considered a STD super/sub-super/subkind, to the extent that it may be envisaged as a herpes kind. Similarly, if e.g. ehrlichiosis (or tracker dog disease) is a specific disease (negatively) caused by the rickettsial bacterium *Ehrlichia* (disease kind) (cf. Box 4.2), then a certain disease caused by a variant of *Ehrlichia* like *Ehrlichia canis* can be deemed a specific disease *super*/*sub*kind.

What seems to prima facie make a disease a disease *super/subkind* is, thus, the numerous possibilities for a disease kind to be a (widely defined) disease kind, such that a more (and more) fine-grained differentia for a disease super/subkind can be given, by arguing that the definition of the differentia of a certain disease super/subkind is actually dependent on the definition of the disease kind at issue.

In a nutshell, what prima facie makes *x* a disease super/subkind is that *x* is a disease kind with a differentia defined as such that it falls under the definition of the disease kind in question.

If we strictly follow our neo-Aristotelian way to define concepts (cf. Sec. 3.1.1), then nothing can be a disease super/subkind as prima facie defined, for, as said above, the differentia of a disease super/subkind is defined *dependently* upon what a disease kind is[41].

Does it mean that a disease super/subkind is to be defined, above all, as a disease kind *tout court* - thus, that talk about disease super/subkinds appears as redundant with respect to talk about disease kinds?

There seems to be another interesting way to define disease super/subkinds, which may be captured through an illustration: if we say that e.g. lung cancer is cancer of a certain kind, then

[41] Moreover, one can add that, to the extent that we are talking about disease super/sub*kinds*, actually, a definition of a disease super/subkind should obviously fall within our neo-Aristotelian framework of seeing *kinds* as species definable as genera with an *independently* defined differentia (cf. Sec. 3.1.1).

lung cancer is obviously a specific disease super/subkind as prima facie defined, for, if cancer is defined as a specific disease with a specific differentia, then lung cancer is cancer with a differentia whose definition is directly dependent upon what cancer is. However, let us suppose that, actually, lung cancer is *not*, strictly speaking, a *cancer* kind, but a *lung disease* of a certain kind. But, if, contrary to the above prima facie definition of a disease super/subkind, this second definition of disease super/subkinds purports to capture the sense in which something can be defined as a disease kind in conjunction with an *independently* defined differentia, how does it do so?

Let us take again the example of lung cancer. If lung cancer is a certain disease super/subkind, then it could be defined as a lung disease in conjunction with cancer as the differentia. Indeed, in so far as a lung disease is a specific disease kind correctly defined as a disease in conjunction with (by anticipation) such-and-such negative effects and causes (cf. Box 4.2), then the definition of this differentia for lung *cancer* does not *directly* depend upon the definition of a lung disease (the genus) – but upon the definition of cancer. And this is not a problem, since, as a lung disease of a certain kind, a definition of lung cancer does not directly depend on the definition of a lung disease.

However, this second definition suffers from two main problems. First, even if this seems sufficient for independently defining the differentia of a disease kind of a kind, how to explain away the idea that lung cancer, colorectal cancer, or pancreatic cancer all seem to fall under one and the same genus: cancer? Do we have to conclude that, strictly speaking, they do not share this genus, because lung cancer is actually a lung disease kind, colorectal cancer a colon disease kind, and pancreatic cancer a pancreas disease kind?

We can try to answer this question as following: a correct definition of e.g. lung cancer mentions lung disease as the genus *proximum* under which the species lung cancer falls, but that much does not imply that the term “lung cancer” is not in the *indirect extension* of “cancer”, for the definition of the differentia for lung cancer, although not *directly* dependent upon what the genus proximum lung disease is, is, nevertheless, dependent upon the definition of cancer.

The second objection is fatal to our definition of a disease super/subkind: one may wonder why not defining, after all e.g. a lung disease as a disease *tout court*. Indeed, why should we define a lung disease as a disease with such-and-such negative effects and causes (for the definition of disease kinds, cf. Box 4.2), rather than as, more simply, a certain process touching the lung

(under the plausible assumption that a disease would be a specific process)[42]? What reasons are there for considering that a lung disease falls under a disease *kind*?

Moreover, if we agree that the term "disease" should have an *absolute* extension (cf. Exc. 3.2; Sec. 3.3.1), what else than e.g. a lung disease, cancer, or perhaps even lung cancer, etc., could this be? If we follow the idea that a lung disease just is, actually, a specific disease, then we may very well argue that "lung cancer" shall also be defined as a mere specific disease, more precisely as being in the *absolute* extension of the term "cancer" (understood as a disease) or in the indirect absolute extension of "lung disease"[43].

If, however, we notice that lung cancer should be properly defined as a specific disease kind, then we shall say that lung cancer is (plausibly if not correctly) malfunctioning growth (as a specific process) of the cells' lung with such-and-such effects and causes i.e. that it is in the (indirect) absolute extension of the term "cancer" (understood as a specific disease kind).

Of course, all this eventually depends on how we strictly define cancer (on our definition of (lung) cancer, cf. Sec. 9.1.1): if cancer were simply (plausibly) defined as a process of cell growth that is malfunctioning, then the prospect for giving an independently defined differentia seems promising, whilst maintaining that this definition of cancer (indirectly) *absolutely* applies

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[42] Three terminological notes on plausibility vs. correctness: (i) in line with a Maximal Compatibility Principle (MCP), according to which

> (MCP) for a definition to be correct is for it, as an epistemic consequence, to resist to a maximal number of plausible objections against it,

even if it happened that our definition of disease is such that "disease" does *not* strictly absolutely fall under "process", it could still be *plausibly* (or, prima facie i.e. in a non-absurd way) argued that, actually, it does, or that the definition of disease *as such* is *compatible* with this idea.

Thus, if "disease" (plausibly understood as a specific process) necessarily has an absolute extension, then "cancer" may be plausibly argued to be such an absolute realizer – and, as such, it can be *used* as an illustration for (an objection against) a certain thesis, without pronouncing on its *correctness* (i.e. even if it does not strictly or correctly follow our own definition of disease (embedded into our metaphilosophy)) (cf. Ch. 2-3).

More globally speaking, when the different concepts defined throughout this PhD dissertation are *not* used as defined, it is contextually made somehow clear or explicit that they are used under their *plausible* meaning.

(ii) For reasons of textual readability, "correctness" *tout court* reads here as what can be considered, of course, the *most* correct *possible*, except, of course, in cases where explicitly drawing this distinction does matter.

(iii) With the MCP, the point is that a definition of *x* is correct, if it is, as an epistemic consequence, *incontestable* (or *indisputable* or *irrefutable*). However, even if to reach such a level seems practically impossible, a way to increase the correctness of a definition of *x* is to show the *coherence* of the definition of *x* with the definitions of the constituents of *x* or, more generally, a bigger picture.

[43] Caveat on pluralism here: if lung cancer is a disease (plausibly if not correctly) defined as malfunctioning growth of the cells' lung tissues, is it in the indirect absolute extension of "cancer" (understood as a disease here) and/or "lung disease"? In so far as "cancer" and "lung disease" are themselves here in the same absolute extension viz. of "disease" (cf. Exc. 3.2), we can safely say that "lung cancer" is in the absolute extension of *both* "cancer" and "lung disease" – as long as, of course, we do not take this fact as committing us to a *pluralist* position, for "lung cancer" is still here *correctly* defined in a *unique* way. If "lung cancer" *directly* (or, at a same level) absolutely fell under two different realized properties, then it would be, indeed, somehow pluralistically defined.

Is "lung cancer" more directly in the absolute extension of "cancer" or of "lung disease"? We can leave this question unanswered or open for our purpose here, for answering it requires to thoroughly investigate the respective definitions of cancer and of lung disease.

the definition, or rather the intension, more precisely, of a disease *tout court* (i.e. here as a certain dysfunction or abnormal (or aberrating) process viz. a (specific) biological malfunction) – and not of a disease kind; thus, that disease is *not* the specific genus under which cancer (directly or not) falls[44, 45].

But, if cancer is (plausibly) defined as a disease kind itself i.e. as malfunctioning cell growth with such-and-such negative effects and causes (cf. Box 4.2), then one would say that lung cancer is (in the absolute extension of) a disease kind.

Indeed, it is not rare to find, in the medical literature (e.g. Taber's Cyclopedic Medical Dictionary, 2017), *plausible* examples of definitions of cancer *simpliciter* as being essentially related to *malignant* (vs. benign) tumors i.e. with what we may want to take, rather, as an *effect* of cancer.

The same ambiguity can be found in many other cases: e.g. Down syndrome (or trisomy 21), whose name explicitly refers to the (partial) genetic constitution of a certain disease (kind). Indeed, Down syndrome can be intuitively *plausibly* defined as a *specific* disease viz. as a specific process touching human cognition (a certain *mental* disease), which has as an ultimate absolute realizer a certain *genetic* disease, or which has as one of its ultimate constituents a process giving rise to an extra copy of chromosome 21 (as the resulting *state*) (under the assumption that "genetic disease (kind)" is an ultimate realizer of e.g. "mental disease (kind)", or that a genetic disease is an ultimate constituent of e.g. a mental disease (kind)[46]) (cf. Sec. 10.1.1);

[44] If we have to specify the kind of relationship at play here between a disease and cancer, then one might say that the determinable-determinate relationship (understood as a genuine relationship) can perhaps best fulfill this role (cf. Sec. 3.2.3).

[45] From all this, we may wonder, eventually, why not defining e.g. lung cancer as cancer (understood as a disease) with the lung as the differentia – and not as a lung disease with a differentia. In that sense, "lung cancer" seems to be in an indirect absolute extension of "disease kind".
However, as we shall shortly see (cf. Sec. 4.2.8), adding the disease host in the differentia for disease kinds is problematic.
Moreover, one may want to define lung cancer as a lung disease with cancer as the differentia. However, if we say that lung cancer is a specific disease kind, then it is a disease in conjunction with certain effects and causes (cf. Box 4.2). If cancer plays the role of the differentia here, then it is (indirectly absolutely extensionally) reduced to causes and effects. Although we can argue that "cancer" is likely in the very indirect absolute extension of "(negative) cause" or "(negative) effect" (cf. Sec. 4.2.10), lung cancer is intuitively not a specific disease (process) (negatively) *caused* by, and *causing* cancer.

[46] Is e.g. the process giving rise to an extra copy of chromosome 21 (as the resulting *state*) an *ultimate constituent* (or an ultimately relatively realized property) (i.e. a constituent of a constituent of a constituent of etc.) of a certain disease (kind) (e.g. Down syndrome) or is it *an ultimate absolute realizer* (e.g. a specific genetic disease (as a specific bad genetic mutation) (partly constituted by a lower-level bad genetic mutation itself) viz. an extra copy of chromosome 21) of a certain disease (kind) (e.g. Down syndrome)? If genetic (pre)determinism is true (cf. Sec. 10.1.1; for a general defense of *macro-* and *micro-determinism*, Sperry, 1986), then the process giving rise to an extra copy of chromosome 21 can be plausibly deemed as an ultimate constituent as well as an ultimate absolute realizer.

or Down syndrome can be plausibly defined as a specific disease *kind* viz. as a specific process touching human cognition with effects and a cause, and which has as an ultimate absolute realizer a certain genetic disease kind, or whose one of its ultimate constituents is a process giving rise to an extra copy of chromosome 21. "Down syndrome" is in the absolute extension of "mental disease (kind)"[47].

3.2 Excursus: On (Direct or Indirect) Absolute and Relative Extensionality

We can distinguish between (direct or indirect) absolute and relative extensionality. For (direct or indirect) absolute extensionality, e.g. "disease kind" has for *direct absolute* extension (amongst others – but, at least *one*) "infectious disease" (since an infectious disease is (plausibly) a *specific* disease kind), while it has for *indirect absolute* extension (among others, or at least) "STD" (since a STD is itself a specific infectious disease).

For (direct or indirect) relative extensionality, e.g. "disease" has for *direct relative* extension (among others, or at least) "disease kind" (since a disease kind is partly defined as a disease) (cf. Box 4.2), while it has for *indirect relative* extension (among others, or at least) e.g. "disease super/subkind".

I shall use the term "relative/absolute extensionality" *simpliciter* (and cognates) to refer either to *direct* relative/absolute extensionality (or, to the fact that there is (*at least*) *one direct* realizer of *x*), or to *indirect* relative/absolute extensionality, or to both in clear enough contexts. In other less clear-cut contexts, we shall, rather, explicitly talk about *direct*, *indirect* or *(in)direct* relative/absolute extensionality; ditto for terms like "constituent", "effect" and "cause", for the *constitution* relationship as well as the *causal* relationship are both taken as intransitive (cf. Ch. 2; 5-6).

[47] Caveat on the disease host: if precision medicine is the trend making the number of diseases explode (cf. Exc. 3.6; for a highly fine-grained differentiation of the disease host, Sec. 9.3.2), then important terminological issues shall consequently arise: e.g. for which specific disease should a term like "Down syndrome" be used, actually? While some medical terms have been used through time in a more and more *specialized* fashion (cf. e.g. the case of "insanity" in Sec. 7.2.1), other medical terms have been used (and still are) in a *complexification* fashion i.e. as terms describing multiple entities spanning multiple levels, as it seems historically the case for "Down syndrome" which was, first, used to refer to abnormal mental behavior (Down, 1866), then to refer to multiple levels encompassing the genetic one (Lejeune et al., 1959).
If we are right with our definitional scheme that one and the same disease cannot be strictly *defined* as multi-level, then we may want to argue that, far from excluding other necessitation relationships (except the one of, strictly, definition) between multiple levels (on holism, cf. Exc. 3.10), "Down syndrome" should strictly refer either in a more specialized sense (e.g. to a certain single subunit of what is now plausibly called "Down syndrome") or in a generalized sense (e.g. to merely a certain mental disease ultimately constituted/realized by a certain genetic disease), such that to avoid putting more and more multi-level entities under the label "Down syndrome" – "medical precision" (acquired through time) does not have to mean "medical holism" or even "medical reduction". I warmly acknowledge Joachim Kurtz (personal communication, June 2021) for having asked about that point.

When we are talking about *both* absolute and relative extensionality, or one or the other, the explicit use of "*absolute*/*relative* extensionality" (or cognates), or "*absolute* extensionality" or "*relative* extensionality" (or cognates), can be maintained, but it can also be left out in clear enough contexts, where one cannot be easily mistaken about whether it is a talk about mere absolute, mere relative or absolute/relative extensionality.

Talk about, explicitly, *relative/absolute* extension can be also dropped, when the context is clear enough: especially e.g. when we can easily distinguish between what true constituents of *x* are (e.g. human being as a rational animal) and under what these true constituents (indirectly) absolutely fall (e.g. human being as having as constituents specific *kinds* – but, human being is not, *strictly* (*plausibly* yes, of course), *essentially* specific kinds), or a certain disease said constituted by lower-level *diseases* themselves.

The same is true for the related term of art "realization" (or "realizer"), which, when the context is clear enough, can be used per se to refer to different more specific realization relationships (or realizers) like *absolute* or *relative* (*extensional* or *intensiona*l) realizers.

Globally speaking, (in)direct extensionality is to be understood as the (in)direct *application* of the *intension*, strictly or neutrally taken, for extensionality does *not* depend as such on a *specific intensional* framework like (our understanding of) the genus-species hierarchy) of a term to the intension of (*at least*) another one (or, the entity referred to). However, the term "extensionality" *tout court* and cognates shall be used most often throughout the PhD dissertation, for reasons of reading clarity, as referring to, more precisely, a *specific* extensionality viz. the one, whose use is based on the genus-species hierarchy. Other specific uses of "extensionality" and cognates can be easily understood contextually.

More specifically, absolute vs. relative extensionality is to be understood as the application of the *intension* of a term to, respectively, the *whole* vs. *partial* intension of (at least) another term.

However, one should not confuse between absolute/relative extensionality per se and *extensional definitions* (vs., redundantly, intensional definitions or definitions *simpliciter*, properly taken), where, for an extensional definition, the *definition* of a term i.e. a set *is* its (absolute/relative) application to (the extensional definition of) (an)other term(s) i.e. the element(s). As such, the notions of absolute and relative extensionality are orthogonal, strictly speaking, to the notions of an intensional and extensional definition, although the notion of absolute/relative extensionality is, of course, used as the basis for formulating extensional definitions (if, indeed, extensionality is truly and only absolute/relative).

An advantage with absolute extensionality is that it allows us to talk about specific diseases (still at the kind level) (e.g. Cardio-Vascular Diseases (CVDs), etc.), without this implying that we are necessarily talking about disease *kinds* (e.g. bacterial diseases, infectious diseases, etc.) (cf. also Sec. 2.2.2).

There is a first advantage with (indirect) relative extensionality (at least, for *non-singleton* sets): it leaves room for (a well-understood version of) *pleiotrop-y*/*-ism* (*one* gene (process) or genotype-*many* traits or (extended) phenotypes) (also called "genetic polymorphism" at the population level) (on extended phenotypes, cf. Dawkins, 2016).

Talk about (direct) relative extensionality has the following second advantage: it allows us to talk about *emergent* or derivative properties without subscribing to RME: e.g. maybe disease and (human) *attitudes* toward it.

We cannot embark here on such a difficult topic that is emergence (as entries, cf. O'Connor, 2020; Gillett, 2016; for a specific account, Tabaczek, 2019).

However, we can minimally highlight, for the present purpose, that, if, along relative extensionality, *x* and *y* are the parts of a whole *z*, then this whole *z* can be said an emergent (or derivative) whole/property i.e. a whole/property emerging from *x* and *y*; the relationship between, respectively, an emergent property and its emergence basis is, thus, a *constitution* or *intensional* (not a *causal*) relationship.

In the literature, the *semantic* notions of absolute and relative extensionality have been, in parallel, extensively canvassed in the *metaphysics* of concrete objects (or, more specifically, *tokens*) - especially in the puzzles of material constitution - under terms like, respectively, "exact co-location" (Casati & Varzi, 1994) or "superposition" (Simons, 1987) and "entire and sporadic location" (Massin, 2008), albeit they have not been thoroughly analyzed.

Moreover, the relationships of relative and absolute intensionality are sometimes analyzed, especially in the neo-mechanistic philosophy of science, as, respectively, the relationships of *dimensioned* and *flat realization* (cf. Craver, 2007; Gillett, 2016; Polger, 2010).

However, due to the many discrepancies in their respective analyzes and to our own – different – use of the term "realization", it is safer to confine ourselves in using, in this context, the notion of absolute/relative intensionality – albeit the notion of fat/dimensioned realization, *as understood here*, can still be theoretically used, of course, but with much caution.

Absolute extension(ality) can be also described in other ways: first, as a relationship, where (at least) two absolute realizers are a pair of (*sub*)*contraries*: e.g. red vs. blue; non-rest vs. non-motion) belonging to one and the same (coarse- or fine-grained) domain of interest (or of application) i.e. the realized property (e.g. color; etc.);

second, along the famous philosophical term of art "(strong) *supervenience*" (as an entry in the extensive literature about supervenience, cf. Mclaughlin & Bennett, 2018). For, the only uncontroversial feature attributed to the relationship of (strong) supervenience is that a set of properties *A* (strongly) supervenes on a set of properties *B*, only if there cannot be a difference in *A* without a difference in *B*. If a definiens *A* i.e. a set of properties *A* has as an absolute realizer another definiens *B*, and if *A* necessarily has an absolute realizer *B* (on absolute extensionality as a *necessitation relationship*, cf. Sec. 3.3.1), then any change in *A* is accompanied by a change in *B* – but not vice versa – i.e. that *A* (strongly) supervenes on *B*.

Nevertheless, due to the high ambiguity, in the literature, around the notion of (strong) supervenience (mostly used, actually, as an *umbrella term* covering very different (more specific) relationships), and in order to clearly contrast absolute extensionality with relative extensionality, it is preferable not to use the controversial notion of (strong) supervenience.

3.2 A Species as a Genus and a Differentia: Objections and Replies

Sec. 3.2 presents and directly refutes objections (or competing theories) raised against (our understanding of) the genus-species relationship (cf. Box 3.1; 3.1.1-3.1.2)[48].

One could object, by insisting on a different point in the Aristotelian corpus (e.g. *Met*. Δ.25, 1023b12-25), that the genus-species relationship should be differently understood.

Indeed, we have understood so far the genus-species relationship in the following way (cf. Box 3.1; 3.1.1-3.1.2): a species is a genus in conjunction with a differentia i.e. that a species has amidst its constitutive parts a genus and a differentia, or a genus is a constitutive part of a species.

Moreover, as has been said (cf. Sec. 3.1.1), the idea of an ontological and definitional priority of disease over disease kinds should be here understood as following: if ME, according to which a whole has its (im)proper parts essentially, is used within a certain metaphilosophical position (cf. Ch. 2), then one concludes that there is a definitional (and ontological) priority of constitutive parts over wholes; if we conciliate this metaphilosophy with our own understanding of the genus-species relationship (cf. Sec. 3.3), then we can say that there is a definitional and ontological priority of a *genus* (in our case, a disease) over its *species* (in our case, a disease kind), if "disease kind" is correctly defined i.e. in a non-trivial way (cf. Sec. 4.1.2).

Certain neo-Aristotelians will find this picture flawed and will argue against this view, along four different lines: (i) the first line consists in saying that the genus-species relationship should be taken reversely and *conjunctively* (Sec. 3.2.1), while (ii), for the second line, that this relation should be taken reversely and *disjunctively* (Sec. 3.2.2); (iii) the third line is that the genus-

[48] It is important to distinguish here between objections directly raised against our understanding of the genus-species relationship i.e., roughly, what is implied by our use of ME (more specifically here, the genus as essentially a constitutive part of a species, and a species as essentially a genus and a differentia), and objections that could be raised against our account of what a definition is i.e. our idea, roughly speaking, that to define some entity *x* (thus, other than (at least) genus) is to find out the constitutive parts of a whole *x* (cf. Sec. 2.1).
Indeed, since our definition of (specifically) the *genus* as essentially a constitutive part of a species does *not* follow our metaphilosophy (although implied by it) (cf. Sec. 3.3.1), we have to dissociate the issues pertaining to what it is to define *x* and to what it is to define "genus".
As discussions about (natural) kinds (terms) are prolific especially in philosophy of the life science and philosophy of language, I cannot discuss in Sec. 3.2 all the different theories of (natural) kinds that have been proposed in the literature (for a sample of those theories, cf. Bird & Tobin, 2017). I have chosen what I take to be the *widest* theories of kinds/species, under which many other more specific theories of (natural) kinds may be arguably said to (roughly) fall (cf. e.g. Häggqvist, 2005; MacLeod & Reydon, 2013; Reydon, 2009a).

species relationship should be understood as a *determinable-determinate* (or determination) relationship (Sec. 3.2.3); (iv) the fourth line objects that a species is to be understood as an *epistemic* entity (Sec. 3.2.4).
In Sec. 3.2.5-3.2.7, I raise three last related objections (v)-(vii) challenging my understanding of the genus-species relationship: a species as a *cluster kind*, and a species as an *evolving kind*, and the genus-species hierarchy as a *Linnaean hierarchy*.
Objections (i)-(vii) are all consequently and directly disproved. The global conclusion to draw from our replies to objections (i)-(vii) is that an essentialist approach to species/kinds (plausibly if not correctly taken) is ineliminable.

3.2.1 Objection (i): A Genus as a Conjunction of Species (with Differentia)

How are we to understand the claim that a genus has, amongst its constitutive parts (plausibly taken), a species (with a differentia)? For a certain neo-Aristotelian who strictly follows *Met*. Δ.25, 1023b12-25, the genus e.g. animal would have as intensional/constitutive parts, amongst others, the species human being or a rational animal (as a whole), and each specific species would differ from each other with respect to its differentia.
In our case – i.e. with (counter-)examples mostly coming from the philosophy of biology and biomedical sciences (though this is merely an arbitrary restriction due to the present purpose of this PhD dissertation) -, according to objection (i), a specific disease like ischemia is supposedly (and *plausibly*) the conjunction of (amongst others, or at least) specific disease kinds (however understood here) like myocardial infarction *and* mitral valve disease *and* so on and so forth; generally speaking, there is an on-going debate about whether e.g. autism is not to be better considered as an autism spectrum disorder (i.e. here a collection of specific neurodevelopmental disease kinds like (plausibly) Asperger syndrome), or whether dementia should not be envisaged, rather, as a collection of (amidst others, or at least) dozens of disease kinds happening when brain neurons malfunction like Alzheimer's disease (Rao et al., 2014)[49].

[49] Two caveats on objection (i): (i) talk about a conjunction obviously excludes singleton sets here. Note that our own definition of a genus is precisely that {genus} is a certain *singleton* set (understood in its *relative* meaning) (cf. Exc. 3.9) – and not a constitutive part of a *conjunction* or exclusive *disjunction* (cf. Sec. 3.2.2) of a species and/or something else -, although, of course, (indirect) *specific* genera can very well be the constitutive parts of more than one *specific* species.

First, how do we constrain what can(not) enter into the conjunction (or the class/set)? By ordering the conjuncts (or the members/elements of the class/set) according to their resemblances and differences.

Thus, it appears that all the members/elements of the class/set {disease (*x*)} share this very property and differ in some other respect. If that is true, then objection (i) may be arguably said to just collapse into our own analysis of the genus-species relationship (cf. Box 3.1).

Indeed, saying that all the members of a class share the property of being a disease (in a specific way) and differ from each other in some respect amounts to saying that a specific species is falling under a certain genus and differs from the other species of the same genus (or, minimally, from the genus itself) through its differentia - thus, that a genus is a constitutive part of a species.

Secondly, if a specific disease is really the conjunction of (amongst others, or at least) *all* the specific disease kinds, under which they all fall, then for someone to possess the concept of *this* disease is for this person to possess the concepts of (amongst others, or at least) all those specific disease kinds.

In the technical jargon, we say that, if a specific disease is really the conjunction of (among others, or at least) *all* the specific disease kinds, under which they all fall, then, more generally, {disease}, as being, thus, a class or a set, is (here relatively[50]) *extensionally* defined i.e. in terms of its (relative) members or elements (plausibly understood): e.g. the class {disease *x*} with

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The requirement of a *conjunction* or an exclusive *disjunction* (cf. Sec. 3.2.2) is very likely explained by the (mistaken) idea that a genus needs more than one species to exist.

Furthermore, it is important to distinguish objection (i) from another fact about our own account of the genus-species relationship (cf. Box 3.1), for, according to our account, a species is a genus in *conjunction* with a differentia.

As already said (cf. Sec. 3.1.1), even though we are talking about a species as a conjunction of genus and differentia, this fact does *not* imply that we are talking about the class membership relationship, even if it is true that we can always trivially talk about a *set*, once there are at least two elements or even one single element, if we admit the possibility of singleton sets (at least, the element(s) which is/are itself/themselves kinds/species).

The fact that e.g. to be a human being is essentially to be an animal *and* to be rational should not be interpreted along the line that a human being is *essentially* a specific *set*, but that it *is* a whole – (numerically) distinct from its essence('s elements) (cf. Koslicki, 2008) - *intensionally* defined (as we understand intensionality).

(ii) Objection (i) is also based on the strong idea that it may sound prima facie inconsistent to talk about a genus as a *constitutive part* of a species (cf. Box 3.1.1), for we would like to say that a genus is something ontologically *bigger in size* than a species and that a part must be *spatially contained* in a whole. This idea is, of course, reinforced by taking a whole as a set (in its relative sense), along objection (i), where the members of a set are *included* in it, for spatial containment seems related to the relationship of membership inclusion – which is also often taken at the concrete level, because of the fact that the notion of *member* or element seems close to the notion of something (mistakenly) taken as *concrete*.

However, this idea wrongly takes our definition of a genus as a definition of a *concrete* genus. Even if it is true that, at the token level, we have the strong intuition that a specific *token* part must be spatially contained in a *token* whole, this does not imply that a token part is *included* in a token whole – thus, that a concrete genus includes a token species. The mistake is here to interpret the notion of spatial containment along the line of set theory i.e., more precisely, as an inclusion relationship between a set and its elements (against Evnine, 2018).

[50] This fact is to be understood as following: e.g. the set {disease} includes specific disease *kinds* like STDs, viral diseases, bacterial diseases, etc.

(amidst others, or at least) *n* disease kinds (as members) is different from the class {disease *x*} with n_{+1} disease kinds (as members)[51].

Although extensionalist definitions may be said to go against our metaphilosophy (cf. Ch. 2), we have to invalidate objection (i) at the same level at which it is raised – thus, not by merely replying that objection (i) rejects our metaphilosophy -, for objection (i) pronounces here only on a certain conception of *kinds* – and not on a metaphilosophical position.

Objection (i) can be answered as follows: it is obvious that e.g. certain disease kinds (as members of disease *x*) are yet to be conceived of (conceptualized, or discovered) and that some disease kinds deemed correct at time t_0 may very well become incorrect at time t_{+1}. Thus, should we say that nobody truly possesses now the concept of disease *x*? How is it possible for someone to possess, generally speaking, the concept of disease, if disease is an *infinite* conjunction? Is it plausible to say that someone does not possess the concept of e.g. grey or even of color (understood as a specific set in its relative meaning), unless he also possesses the concept of X11 grey or even of a shade of color?

Nevertheless, a proponent of objection (i) may eventually reply that disease *x* is not necessarily an *infinite* conjunction, but a *stable* (enough) conjunction of (amidst others, or at least) e.g. only *some* of the specific disease kinds, under which all these ones fall[52].

However, at a more general level, it is difficult to decide, in this case, in a *non-arbitrary* and *non-vague* way[53], exactly which conjuncts (as elements/members of a set/class) and how many of them amongst all the others should enter into a conjunction, such that we can safely say that

[51] Two terminological notes (with caveats) on (kind) pluralism: (i) pluralism can be understood in two – strong and weak – versions: along the strong version, it is the position, according to which a single sortal *x* is to be analyzed as e.g. *Fx*, *and Gx* (*and* etc.), and that *Fx* and *Gx* cannot be subsumed under a single analysis e.g. *Hx* of *x* (*pace* Brigandt, 2003; Walker & Rogers, 2018) –, where *Fx* and *Gx* would be two specific *x*s or two sort(al)s of *x*: *Fx* and *Gx* are two *irreconcilable* analysis of the sort(al) *x* (cf. Mitchell, 2003).
Along the weak version, pluralism about (supposed) *kinds* – to avoid that weak pluralism applies to trivial entities -, relies on extensional definitions: some kind *x* is weakly pluralistically defined, iff *x* is a (non-singleton) set comprising entities intensionally definable (however understood) or not themselves weakly pluralistically definable (cf. Johansson & Lynøe, 2008): e.g. the concept BIOLOGICAL SPECIES (trivially) defined as a set of different more specific concepts (used and found in different contexts, etc.), which shall be the focus of a philosophical analysis.
(ii) Strong pluralism implies that there is *no* such sortal *x*. In so far as we do not see any longer how *Fx* and *Gx* can be subsumed under a single sortal *x*, then claiming that there is no, actually, such sortal *x*, because the term "sortal *x*" would be empty, is the next logical step (cf. e.g. Machery, 2009 about concepts; Ereshefsky, 1992 about biological species (*pace* Dupré, 1999).

[52] This point is not to be confused with the close idea of a species as a *cluster kind* (cf. Sec. 3.2.5)). I sincerely acknowledge Christian Quast (personal communication, June 2018) for having highlighted this way of making objection (i) stronger and more credible, and to make me avoid attacking a straw man (or, badly rephrasing the arguments of one's antagonist by e.g. radicalizing or weakening them).

[53] It is important not to take the fact that a disease (kind) admits of *degrees* (cf. Sec. 9.4.3 on that) as a reason for sustaining that "disease (kind)" is, thus, a *vague* notion (*pace* Keil et al., 2017; Rogers & Walker, 2017). Indeed, e.g. a work of art can be more famous than another one without making, thence, the notion of fame vague.

they are sufficient – or, that they function as a *threshold* - for the possession of the concept of disease.

3.2.2 Objection (ii): A Genus as an Exclusive Disjunction of Species (with Differentia)

Another way to put into practice the same strategy consisting in reversely understanding the genus-species relationship (cf. Sec. 3.2.1) is to envisage the genus as an *exclusive disjunction*[54] of species (with their differentia).

Objection (ii), thus, amounts to arguing, in our case, that e.g. to have ischemia is supposedly (and plausibly) to have (amidst others, or at least) *either* myocardial infarction *or* mitral valve disease *or* etc., and that each exclusive disjunct is organized along its resemblance and difference with respect to the other exclusive disjuncts.

Objection (ii) has a prima facie advantage over objection (i) (cf. Sec. 3.2.1), because for an exclusive disjunction to be true only one of the disjuncts must be true, to the contrary of a conjunction where for a conjunction to be true all the conjuncts must be true.

Thus, objection (ii) avoids our second criticism of objection (i) (cf. Sec. 3.2.1) that, if a specific disease is a conjunction of (amongst others, or at least) e.g. *some* (if not *all*) of the specific disease kinds, under which all these ones fall, then nobody would ever be able to possess the concept of this disease, for, more generally, to possess the concept of disease would amount to possess the concept of all the conjuncts.

In other words, to correctly possess the concept of disease understood as an exclusive disjunction, we are bound to possess the concept of *one* of the exclusive disjuncts (e.g. disease kinds), under which they fall.

However, this advantage over objection (i) (cf. Sec. 3.2.1) notwithstanding, again, the same issue arises: what are more precisely these resemblances and differences constraining what can(not) enter into the exclusive disjunction? The same answer also applies: all the exclusive disjuncts share the property of being a disease and differ in some other respect. If the idea that

[54] The requirement that it should be an *exclusive* disjunction is justified by the intuition that, if the genus e.g. ischemia is a disjunction, then it must be either e.g. the species myocardial infarction or the species mitral valve disease or etc. – it cannot be both myocardial infarction and mitral valve disease, on pain of collapsing into objection (i) (cf. Sec. 3.2.1).

disease is an exclusive disjunction of e.g., amidst others, or at least, disease kinds makes sense, then the very definiendum should not reappear in (the elements of) the definiens.
However, the ordering of all the exclusive disjuncts in the exclusive disjunction suggests the contrary, since all the exclusive disjuncts share the very property of being a disease. In other words, objection (i) (cf. Sec. 3.2.1) and objection (ii) can be said to put the cart before the horse[55].

3.2.3 Objection (iii): the Genus-Species Relationship as a Determinable-Determinate Relationship

Objection (iii) to our thesis that a genus is ontologically and definitionally prior to a species (cf. Sec. 3.1.1) is that the genus-species relationship should be properly analyzed as a determinable-determinate or determination relationship; even if these two relations are usually taken to be different (cf. Johnson, 1921; Wilson, 2017 on all that), the issue remains whether one is (absolutely intensionally) reducible to the other.
Likewise, the determinable-determinate relationship is also usually contrasted with any *conjunctive* (cf. Sec. 3.2.1) or *disjunctive* (cf. Sec. 3.2.2) analysis of the case study used ad nauseam in the literature viz. the relationship between red and color, but the question remains whether the determination relationship is finally not just a conjunction or a disjunction of a genus.
How to positively understand the determinable-determinate relationship is unsurprisingly a matter of considerable philosophical controversy.
However, the mere following uncontroversial feature of the determination relation is germane to raising an objection against our analysis of the genus-species relationship: a determinate (e.g. red) of a determinable (e.g. color) is taken to be that very determinable *in a specific way*, where the specific way in which e.g. red is said a color cannot be defined *independently* of what the determinable in question is (i.e. that "red" is not in the *relative* extension of "color", but in its *absolute* one): to be red is to be colored in a specific way, and nothing just is colored *tout court* without having one specific shade of color.

[55] If we agree that there is no disjunctive *kind*/*species*, does it mean, nevertheless, that there is no disjunctive *entity* or thing, which would not be a kind? It is not excluded that a disjunctive entity – but, which would not be a non-trivial entity (for, such entities do not have an essence or a definitional structure) or an entity, strictly speaking, taken under a *kind* pluralism (in its weak version) – exists, indeed: e.g. Socrates as being a man or a mountain.

Some commentators of Aristotle's work (Granger, 1984) have argued that this could have been the Stageirite's own position, at least in *Met*. I.3-4, 1054b22-31.

That much is at odds with how we have analyzed so far the genus-species relationship (cf. Box 3.1) i.e. as a relationship where the species can be said (similarly to the determination relation) to be that very genus in the following way: (by opposition here with the determination relation) in conjunction with differentiating characteristics (or differentia) defined independently of what the genus at issue is.

According to objection (iii), the genus-species relationship is (absolutely intensionally) reducible to the determinable-determinate relationship.

The weakness of objection (iii) is how to establish the fact that there is no principled way (or, guideline or rule of thumb) in (or, according to) which e.g. the determinate red can be defined as the determinable color in conjunction with a feature independent of the determinable color i.e. how to figure out more precisely the idea that a species as a determinate is a *specific* genus as a determinable – if not by saying that a species is a genus with an independently defined differentia. That much means that the determination relationship runs the risk of just collapsing into our own analysis of the genus-species relationship.

There are, of course, currently several accounts that try to thoroughly analyze the way in which a determinate can be said a specified determinable: one of the most prominent or insightful accounts states that the precisified way in which a determinate is a determinable is that a determinate (e.g. red) possesses a value (e.g. a specific saturation, hue and brightness) belonging to the value space (or *range*) of the determinable (e.g. saturation, hue and brightness for color) (Funkhouser, 2006).

However, with such an account we still do not understand why it is e.g. the color red which has value *x* belonging to the color value space, rather than the color blue or yellow; indeed, if we want to defend the idea, along objection (iii), that "red" is to be *defined* as being in the absolute extension of "color", then "blue" or "yellow" too is to be defined as such.

But, then, what differentiates them, if not through the fact that either they all share the very same genus (color) but a different differentia, or that they all can still be defined within a genus-species hierarchy as understood here (cf. Sec 4.1.1), while maintaining that "red", "blue" and "yellow" are still in the absolute extension of "color"?

Thus, one would like to say here that one can interpret the strong intuition, that we have to explain away somehow or other, and on which the determination relation is built viz. that e.g. to be colored is to have a specific shade of color, as merely establishing the fact that there is no (specific) genus without a (specific) species under which it falls – a genus is essentially, under

a plausible reading, a constitutive part of a species (or, it essentially has a *relative* extension). As such, the determination relationship is (absolutely intensionally) reduced to our own analysis of the genus-species relationship (cf. Box 3.1.1).

However, we can also read the determination relationship as being about not the definition of a determinate as a non-independent or specific determinable (i.e. a species as being essentially in the absolute extension of a genus), but of a *determinable* as a non-independent or specific determinate (e.g. nothing just is colored without having one specific shade of color) (cf. Jones, 2016b on that point): as such, the intuition that e.g. to be colored is to have a specific shade of color is to be interpreted differently, so as to raise an objection against our analysis of the genus/species relationship (cf. Box 3.1.1): that a *genus* is defined, along objection (iii), *absolutely extensionally*, where its members are species (thus, defined as being absolute realizers of a genus)[56].

[56] Four terminological notes on the determination relationship: (i) here, we are typically using the intuition on which the determination relationship is based to raise an objection against our analysis of the genus/species relationship.
However, if we decide to separate the determination relationship from a definitional relationship (between genus and species) i.e. to take the determination relationship to be sui generis (i.e. absolutely intensionally) irreducible to the genus-species relationship), then it can be said a simple relationship of *absolute extensionality* (but not of relative extensionality, since our analysis of a genus is based on this relationship), and this poses no threat to, or does not jeopardize, our own analysis of the genus-species relationship (cf. Box 3.1; 3.1.1-3.1.2), for both relationships would be, thus, different (cf. Sec. 3.3.1).
(ii) Note, moreover, that the relationship of determination (however understood) has been used for analyzing the famous relationship between *thin* (e.g. goodness vs. badness, rightness vs. wrongness (cf. e.g. Ross, 1930), or maybe even truth vs. falsity, correctness vs. incorrectness, or oughtness vs. non-oughtness) and *thick* (e.g. beauty, health, disease, justice, life, death, etc.) *values* (or *norms*) (Mulligan, 1998; Tappolet, 2004; cf. also von Wright, 1963).
Indeed, the distinction between thin and thick values (or norms) has been explicitly clarified by Scheffler (1987) as a relationship whose relata are values (or norms) and have different degrees of generality or *specificity*; if we are right in taking the determinable-determinate relationship as our absolute extensionality relationship, then e.g. "disease" or "death", as being an indirect *absolute* realizer of "lethal value" (or "fatality") or "badness", shall be said a specific *thicker* value than a lethal value and badness, which are, thus, *thinner* values (on thick-thin values or norms, cf. e.g. Kirchin, 2017; Väyrynen, 2016; in the philosophy of psychiatry, Banicki, 2018).
(iii) Thence, if we are right in analyzing the relationship of thin-thick *values* (or norms) as a *specific* determinable-determinate relationship viz. whose relata are *values* (or norms) – indeed, the notion of thinness and thickness *tout court* may be used, of course, outside the mere realm of *values* (or even norms) (cf. e.g. Mulligan, 1998 for a use of those terms in the debate about internal vs. external relations), albeit, in the literature, talk about thinness and thickness (*tout court*) is often (wrongly) restricted to talk about thin and thick *values* (or norms) - , then thin values (or norms) *simpliciter* are, strictly speaking, always the *thinnest* values (or norms) possible viz. fundamental or general values (or norms) like goodness vs. badness, and rightness vs. wrongness, which may be called "thin values (or norms)" *tout court* (Williams, 1985); indeed, thin and thick values (or norms) are said here to merely differ with respect to their grain or scale of specificity (or maybe (conceptual) richness): e.g. admirability/admiration is (plausibly) a *thinner* value than courage/bravery or generosity, but also at the same time a *thicker* value than goodness.
At the more global level, we might well argue that the *thinnest* entities are precisely those which have no essence (strictly taken) like genera or qua objects.
(iv) Finally, note that, if we are right in taking thin-thick values (or norms) as, respectively, specific determinables-determinates, then a thick value (or norm) cannot be analyzed, thus, as has been often done (cf. Tappolet, 2004), as a thin value (or norm) (i.e. the genus) plus non-normative or -evaluative properties (i.e. the differentia), for e.g. disease is intuitively not something more than bad; it is a *specific* bad thing *simpliciter*.

Objection (iii) is, more precisely, the objection that a genus (e.g. color) should be defined as a conjunction or a disjunction of the species *absolutely* – by contradistinction with objections (i) and (ii) (cf. Sec. 3.2.1-3.2.2) - falling under it (e.g. red, blue, yellow, etc.) (on the related thesis of so-called species (i.e. here biological genera) as sets of organisms (i.e. here specific biological species), cf. Kitcher, 1984).

A well-known illustration of such an approach to defining a kind is the so-called *genotype-first* approach to (some) disease(s) using next-generation sequencing (e.g. whole genome sequencing)[57], where (some) diseases are (plausibly) absolutely extensionally defined with respect to their genotypes; along objection (iii), (some specific) diseases are, thus, classified as either *monogenic* (or *Mendelian*) diseases i.e. with a single mutated genetic process (along objection (iii), e.g. neurofibromatosis, etc.) or *polygenic* (under a deflationary acceptance) diseases i.e. with multiple mutated genetic processes (according to objection (iii), e.g. type 2 diabetes, etc.).

Nevertheless, this charitable reading of objection (iii) is also flawed: first, what constrains which species can(not) absolutely fall under, or be in the absolute extension of, a genus? How to rule out the idea that e.g. the specific genus color be defined, along objection (iii), as being either red or blue or yellow or…a *disease* (cf. Exc. 3.3)?

Precisely by answering that some of these species *truly* absolutely fall under the genus color, while others do not: the definiendum is still present in (the elements of) the definiens[58].

Furthermore, mixing up normativity with non-normativity within one and the same definiens amounts to a category mistake (*pace* Fulford, 1989; 2001).

[57] The genotype-first approach to disease is usually opposed to a *phenotype-first* approach to disease using so-called genome-wide association study, which, in our terminology, aims at determining the (indirect) relative extensionality of "genetic process *x*" (i.e. its (indirect) intensionality).

[58] One could, of course, say that this is not a problem. However, allowing this goes far beyond merely rejecting intensional or extensional definitions (as understood here), but points towards rejecting a definitional relationship, where the definiendum and the definiens are both two (numerically) different (by the PII_1) sui generis entities, by arguing e.g. that the relationship between a definiendum and a definiens is a *numerical identity* relationship between a set or a whole and its singleton or its (single) improper part (if this makes really sense) or a *loose identity* relationship like partial identity, although they still might be, of course, *qualitatively different* (plausibly taken) (by still following the PII_1, but against the PII_2 – thus, against LL) (cf. Forrest, 2010).

If numerical identity seems an implausible candidate for analyzing the relationship between a definiendum and a definiens, for the numerical identity relationship has no *explanatory power* (on essentiality and explanatory power, cf. Gorman, 2005), why not defending, thus, the idea that the definiendum and the definiens may be merely qualitatively different (plausibly taken)?

For, first, even proponents of extensional definitions have to sustain that the definiendum (e.g. a(n) aggregate/conglomeration/sum/total/set/class/fusion/group/range/series/collection/cluster/spectrum) and the definiens (e.g. elements/members) are both two different genuine entities i.e. that talk about *sets* is still an ontological commitment (however shallow, or considered as a free lunch or a logical construction, for we are used to directly refer to the *elements* of a set rather than the set itself) to something different from (vs. *numerically*/*loosely identical* to) its (singleton) element(s) (cf. Koslicki, 2008) – for a set cannot be non-trivially (and on pain of a vicious regress) *numerically* different from the sum/*set* of its elements.

Second, were the definiendum and the definiens merely qualitatively different (plausibly taken), then the *explanatory direction* in a definition would not be unique any more, for loose identity like partial *identity* is arguably a *reflexive* (and *symmetric*) relationship (if ever loose identity proponents want loose identity to be

Second, one may also object here that that much would make the definition of genus *trivial* or empty: the category of cat (plausibly as a genus here) would be what specific cats (as species) are.

A proponent of the idea that a genus should be absolutely extensionally defined with respect to species may still reply that it is implausible to ever find out an *intensional* definition of a specific *quantitative* species/kind like eye (or coat, hair, etc.) color, height, weight, voice (tone) or vocal timbre, etc., such that it includes e.g. for, specifically here, *human* eye color, "green eyes" but excludes "pink eyes" as one of its absolute realizers, if not by considering it as a specific *genus* absolutely extensionally defined with respect to specific species (cf. Exc. 3.3).

As a reply to this last defense of objection (iii), we can, actually, just bite the bullet: for specific (highly widespread) *quantitative* species/kinds (cf. Exc. 3.4), if we do not want to fall into *arbitrariness* (and to avoid *vagueness*), a specific *normal* quantitative kind shall be defined with a *single* deemed *ideal* or *correct simpliciter*, but not Utopian (or (empirically) unachievable) value (in the sense of a quantity here) difficult to reach, of course, though necessarily realized (and indirectly instantiated)[59]: e.g. – here for specific *positive* kinds i.e. specific *perfect* ones -, contemporarily (for, we may guess that such values shall be refined any way with scientific progress), a 20/10 human visual acuity on the Snellen chart (in a bright environment); a human attentional mechanism facilitating learning and survival (in a free environment); a total cholesterol of 5 mmol/L for a human adult (on an empty stomach); a human being with a normal audiogram (in an external noise-free environment) ; a human with a normal volume of breathing and gas exchange (in a non-stressful context); a blood pressure (i.e. the systemic arterial pressure) for an adult human being of 115/75 mmHG i.e. 115 millimeter of mercury systolic (systolic pressure) over 75 millimeter of mercury diastolic (diastolic pressure) (in a non-stressful context); a human blood sugar level of 11.1 mmol/l (or 200 mg/dl) (on an empty stomach); the production, by the thyroid gland, of a certain thyroid hormone (or thyroxin) level due to a thyroid-stimulating hormone of 2.0 μIU/mL (in a non-stressful context); a specific *rate* on the speech naturalness scale (in a non-stressful and external noise-free environment); or a

minimally close to the *logical* concept of identity), while a definitional relationship – be it intensional *or* extensional – is intuitively an *ir*reflexive (and *a*symmetric) relationship (cf. Ch. 2).

[59] Caveat on ideality: the notion of ideality (normalcy, correctness or ordinariness in our vein) is to be differentiated from the notion of *idealization* (on the flourishing literature on idealizations in (theoretical) science, as entries, cf. Frigg, 2020; Weisberg, 2013): e.g. the ideal (normal, correct or ordinary) heart is not the same as the idealized heart.

Indeed, it is very common to hold, on the one hand, that ideal entities cannot exist except as specific entities viz. simplifications, distortions, approximations or idealizations (cf. e.g. Wachbroit, 1994).

But, on the other hand, as for other sciences, biomedicine often confuses *abstraction* (i.e., strictly speaking, an ideal entity (considered in abstraction of parameters judged irrelevant)) with *idealization* i.e. that it may happen that (said) idealized entities are, actually, just ideal entities (considered in abstraction of irrelevant parameters).

specific body temperature of 37.5 °C (for an adult human being in a specific environment) (on those values, cf. e.g. Dorland, 2011); etc.

Any other specific kind with a *lower* or *higher* value (than the one of kind *x*) will be said a(n) (little, much, etc.) *abnormal* (*anomalous*, *incorrect* or unordinary) (on the related issue around enhancement, cf. Exc. 9.2) - more precisely here, *diseased* or *malfunctioning* - kind *x*: e.g. human myopia/hyperopia; human hypo/hypersalience; human hyper/hypocholesteromia (for an adult); human hyper/hypoacusis; human hyper/hypoventilation; human (arterial) hypo/hypertension; human hyper/hypoglycaemia; human hypo/hyperthyroidism; (human) stuttering/cluttering; or human hyper/hypothermia; etc.

Thus, what appeared as an absolute realizer of e.g. human hair or eye color like "red hair" or "brown eyes" (on red hair as "[…] a sort of weakness of hair […]", cf. Ari. *GA*. V.5 (tr. Platt, 1910), because of its too high concentration of pheomelanin and too low concentration of eumelanin), or equine coat color like "chestnut coat" may be possibly judged to be, actually, just a *malfunctioning* human hair or eye color, or equine coat color (cf. also Exc. 3.3-3.4).

An absolute realizer of e.g. "human visual acuity" is, thus, not a realizer with a *different* (lower or higher) value from human visual acuity – which would be defined as a ratio -, but it is a *variant* (a variation, a variety, an exemplar, or even an archetype, etc.), amidst others, or at least, of "human visual acuity" (defined with specific measures) with the *same* value (since it absolutely applies the *same* definition, or rather intension). Thus, a specific quantitative species/kind may still be intensionally defined.

Note that this reply to our final understanding of objection (iii) eliminates (very usual) *definitions* of e.g. "fever" in terms of the many specific diseases of which it is a cause or an effect (for a non-exhaustive list of the specific diseases associated with fever (and rash), cf. Kaye & Kaye, 2015).

Thus, although "fever (of *x*)" or "(exo/endo)toxin release process" can be said in the absolute extension of "effect x_1 of disease x_2" or "cause y_1 of disease y_2" or can have as relative extension "disease kind *z*" (cf. Sec. 3.2.2), "fever (of *x*)" or "(exo/endo)toxin release process" is not to be absolutely (or even relatively) extensionally *defined*; one should not take the absolute *application* of the intension of *x* (i.e. *x*'s absolute extension) for the definition of *x itself* (unless one subscribes, of course, to extensional definitions).

Thus, here, the intuition that there is e.g. no color without one specific shade of color can be interpreted as only requiring that a term should have an absolute extension (cf. Sec. 3.3.1 on this requirement) – but not as saying that a term's intension is its absolute extension (*pace* objection (iii)).

3.3 Excursus: Against Health as a Ratio

Our final reply to objection (iii) (cf. Sec. 3.2.3) seems to rule out the highly common theory of *health* (which goes back to mostly Hippocrates and Galen), according to which being healthy is having a *ratio* (or a proportion, a homeostasis (cf. Virchow, 1958), a regulative mechanism, a harmony, a *golden mean* (or golden middle way) (Ari. *EE*), the scholastic idea that *in medio stat uirtus*, etc.), and, more importantly, having e.g. a normal *biological* function is having *polymorphic* functional traits(/genetic processes) (cf. Boorse, 1977; for an updated contemporary version of such a theory of health, cf. Sholl, 2014, whose theory of health as robust organismic *flexibility* is largely inspired by Canguilhem (1966)'s work): e.g. having a healthy rate of glucose in blood (glycaemia) (on an empty stomach) may be plausibly defined as having a glycaemia between 0.70 and 1.10 g / l; being of a healthy (or normal) *human* blood *group* is (plausibly) being of either blood group *A* or *B* or *AB* or *O*.

Other famous illustrations include, in *quantitative genetics* (under the broad but *plausible* acceptance of what "genetic disease" means) (cf. Sec. 10.1.1), *polygenic* inherited traits - under a certain acceptance of the term; under another deflationary acceptance of the term, polygenetic processes just are multiple conjunct genetic processes all coding for a single trait) – like weight, height, eye or hair color, where we can *plausibly* say that having e.g. a normal *human* eye color here is possessing either green eyes (*indirectly*) due to (or, (partly) having as an (indirect) absolute realizer) a certain single-nucleotide polymorphism in OCA2 gene, or blue eyes (*indirectly*) due to a certain (deleterious) genetic mutation with HERC2 gene, or etc., where "green eyes" and "blue eyes" are plausibly said absolute realizers of "*human* eye color".

Indeed, one would say here that *x* has e.g. a healthy (or normal) glycaemia of e.g. 0.80 g / l, as well as *y* has a healthy glycaemia of e.g. 1.00 g / l; but, if we say that *x* and *y* both have a healthy glycaemia, then what allows us to say this is precisely that *x* and *y* both, actually, have properties falling under a healthy glycaemia. Ditto for other cases: e.g. if having a normal human eye color is having either green eyes, or blue eyes, or etc., then why do not we want to intuitively add to the list of these absolute realizers e.g. "pink eyes"? For these absolute realizers are typically associated with having a *mal*functioning human eye color (e.g. conjunctivitis). We could plausibly argue that, if we can exclude from this list pink eyes, then it is because the other specific eye colors would all apply the definition, or rather the intension of the same specific genus.

If health is not to be absolutely extensionally (and disjunctively here) defined, then disease cannot be defined as a violation of a statistical norm or of a ratio, or as a disproportion (*pace* Canguilhem, 1966). Some remarks are here in order; first, it is important to distinguish between the theory of health as a ratio (cf. Exc. 3.3) and the basic idea behind the famous Bio-Statistical Theory (BST) of health (Boorse, 1977; 2014) i.e. that we should not confuse a norm that is *itself* statistical (or a ratio) with a norm that is

statistically established (as the BST of health (and disease) usually takes it) i.e. through e.g. frequentist or Bayesian inference, for a statistically established norm can very well be itself non-statistical or not a ratio: e.g. if from a sample of 100,000 healthy people we can notice that 80% of them possess a glycaemia with a very precise single value, then we do not have to make a ratio over the whole sample to get a statistical norm, but we can very well conclude that to have a healthy rate is to possess a very precise single value.

As Boorse (1977) himself claims, this very precise single value does not have to be shared by some or even one of the members of the *reference class* over which we average to get this result, but it "[…] is an empirical *ideal* which […] serves as the basis for health judgments in any species where we make such judgments" (p. 557; my emphasis) i.e. that it is a certain *benchmark*.

However, a statistical norm or a ratio, in so far as it necessarily includes *two* extreme values (on a continuum), seems statistically established; even if we proceed by induction from a single healthy *individual*/particular/token, we would, nevertheless, make a ratio over the *number* of the token biological processes of the single individual. Thus, in a nutshell, an *average* over a reference class may be either a *single value* (shared or not by the members of the reference class in question) or a *ratio* (also shared or not by the members of the reference class).

Second, the idea of health as a single perfect value, where any higher or lower (deviating/deviant) value x or y is a disease is not to be confused, of course, with the idea that health is, contrarily, the possession of any value different from x or y, or between x and y. Such a theory of health - which should also be distinguished from the (*positive*) theory of health as a ratio - goes hand-in-hand with (or, encourages to sustain) the general *deflationary* idea of health as the absence of disease – thus, where disease somehow wears the trousers (Boorse, 1977).

3.4 Excursus: Quantitative Kinds

If e.g. there is only one perfect eye color (e.g. *green* eyes – under the assumption that they are here taken as properties *essential* to e.g. attractiveness or light sensitivity), so as to exclude e.g. *red* eyes (i.e. bloodshot eyes) to count as absolute realizers of "eye *color*", we can still, however, *plausibly* defend, of course, that different specific eye colors can be absolute realizers of e.g. "eyes" *tout court* (in a plausible, though obviously incorrect (without pronouncing further on it), sense); or, to the contrary, if e.g. green eyes are said the perfect (or correct) eye color, then it may very well be *plausibly* the case that e.g. *big* or *small* green eyes can be absolute realizers of "green eyes", although big or small eyes are, *strictly* speaking, a *malfunctioning* eye size (on intransitivity in definitions, cf. Sec. 2.1.3).

Note that, if quantitative kinds are here concerned (i.e. kinds defined by *iterations* or duplications of a certain quality like e.g. 25cm taken as 25x1cm), this is not the case for *qualitative* kinds i.e. kinds, *strictly* speaking, constituted by qualities: e.g. scientists do not - at least, currently - describe human *left-*

handedness as a specific disease (or a defect or disorder), but only as a *specificity* or as a specific human handedness (on the genetic factors of human handedness, cf. Annett, 2002; McManus, 2019).
In that sense, we might well argue that left-handed, right-handed, mixed-handed and ambidextrous human beings just are three different *specific* or typical (normal) human beings – not *a*typical ones -, for specific human beings just *cannot* – unlike cases for e.g. their eye color – be otherwise than *either* left-, *or* right-, *or* mixed-handed *or* ambidextrous.
Indeed, we might well argue that human *handedness* is a specific *qualitative* kind (properly speaking) – *not* a quantitative kind, which, if not defined with a *single* deemed normal value, shall be unfortunately defined arbitrarily and vaguely.
Thus, the possible fear that, due to the high *prevalence* of quantitative kinds, there are *only* quantitative kinds (and not also *genuine qualitative* kinds) has no place here. Quantitative kinds are especially necessary for non-arbitrarily and –vaguely ruling out problematic cases, when there is too large a range of (specific) cases for being *qualitatively* definable – without that being, of course, the *raison d'être* of quantitative kinds.
Allowing the existence of qualitative kinds does not preclude, of course, to sustaining to *degrees* of e.g. disease for *qualitative* kinds: e.g. being only *a little bit* left-handed. It is not because *quantitative* kinds are defined with a single deemed perfect value that degrees of e.g. disease apply only to them.
As an objection against the idea of quantitative kinds as being defined with a single value, we may well resort to *contextualist* or perspectivist ideas, according to which the values ascribed to quantitative kinds are different from one context to another one.
However attractive those ideas seem, the question of how to delineate what is normal within *one and the same context* still arises.

3.2.4 Objection (iv): A Species as (Reduced to) an Epistemic Entity

An objection so widespread, that it cannot be passed under silence here, is the one, according to which (natural) species/kinds are (reduced to) *epistemic entities* or *units*[60] i.e., roughly, that species/kinds would not exist as independent from an intentional agent; more precisely, along objection (iv), a species/kind is a specific *artefactual* entity viz. an entity meant to complete

[60] Although our own definition of species/kinds applies, as such, to natural *as well as* artefactual species/*kinds* (cf. Exc. 2.7; Box 3.1), objection (iv) is still relevant here, for it *reduces* (what we take to be) natural kinds to mere artefactual *entities*.

specific epistemic tasks for scientists or researchers or even philosophers themselves (Lemeire, 2018) in different contexts: e.g. a species/kind is anything scientic practice (or philosophy) finds useful, fruitful, interesting, needed, or relevant to postulate, so as to be successful (Ereshefsky & Reydon, 2015; Magnus, 2012); or, species/kinds may be devices merely allowing (scientific) classification (Franklin-Hall, 2015), unification (Schurz, personal communication, April 2019), (inductive) inference or projectibility (Häggqvist, 2005), prediction, or explanation; or, they can be just things having a heuristic value for e.g. (scientific) experimentation (on the *new experimentalism*, cf. Mayo, 1996), modelling (Sterner, 2015) or else (cf. also Kästner, 2017; & Andersen, 2018; Osbeck & Nersessian, 2015 on theories of causation/constitution – or, of individuation practices - based on the researcher).

In other words, according to objection (iv), species/kinds exist (if not fully, at least in the only interesting sense worth to be examined)[61] pragmatically or as *interest-relative* (cf. Craver, 2009; Kaiser, 2018a; Reznek, 1987; also Dasgupta, 2017), or definitions of species/kinds are commonly said to be *operational*(*ized*) (vs. theoretical) (cf. Aucouturier & Demazeux, 2012 for the concept of mental disease; Fuellen et al., 2019; Hacking, 1983; 1991).

One of the main motivations behind objection (iv) is, especially in an era of strongly naturalized metaphysics of science, that philosophers do not merely care about how terms are used by scientists, but they also do claim - (allegedly) in line with the sciences - that what those terms *mean* is intimately related to how they are used, which (epistemic) purpose(s) they serve (mostly *explanatory*), or the scientific framework within which they are embedded (on the related famous thesis of *meaning as use*, cf. Wittgenstein, 1953).

A second motivation is that those philosophers generally think of a primacy of epistemology (of science) over metaphysics (of science) (cf. also the motivations behind objection (v) of Sec. 3.2.5).

A third motivation coming specifically from the domain of the philosophy of medicine is that, because of the constant risk of over-diagnosis and other practical reasons, a definition of disease (kinds) considered as a specific species/kind shall be necessarily vague and stipulative (Rogers & Walker, 2018), or must serve other epistemic purposes (Walker & Rogers, 2018), or just has to be helpful to the medical practictioners (Doust et al., 2017).

A fourth important motivation behind objection (iv) is that it gives a straightforward and limpid answer to the question of why species/kinds exist: they exist, because they are indispensable to the success of science (and philosophy) (Lemeire, 2018).

[61] The notion of existence can be also made interest-relative, of course.

First, beyond the classical problems associated with (strong) anti-objectivist views on kinds/species (cf. Esfeld, 2009), the main issue with objection (iv) is, unsurprisingly, its *reductionism* i.e. the reduction of species/kinds to artefactual ones here – albeit such epistemic entities can be conceived of as having an essence (plausibly speaking, or in compatibility with our own account not restricted here to natural kinds).

Do species/kinds really exist, because they are able to solve such-and-such problems? Without denying that there exist *some* specific things in the world, which are, indeed, artefactual viz. epistemic here, it is obvious that there are - at least, also some - things in the world, which do not exist *for us*, or to serve us (or any other intentional agent), and those things are (natural) kinds (without this meaning that kinds/species are context-insensitive) (on my understanding of context sensitivity, cf. Sec. 3.2.6); they are not there for us, or for giving us knowledge of some good/bad, or for being crucial to our explanatory practices, etc.: e.g. albeit water (as H_2O) is undeniably part of the explanation of many different explananda phenomena (or even, water as H_2O has an *explanatory power* on its own (ontologically taken here i.e., without requesting the presence of any intentional agent asking why- or how-questions, that water is simply constituted by, or is essentially, H_2O)), water, nevertheless, does not exist to allow us to yield those different scientific explanations, or it is in no meaningful sense helpful to us (for whatever epistemic ends/purposes) to define water as H_2O – it is just what water *is* essentially (rather than e.g. XYZ) (and how we try to grasp it).

That much does not mean, of course, that water (as H_2O) is of no *practical* worth (at all) (e.g. for the unificatory or classificatory purposes of science, etc.) – or even, that the act of *defining* or explaining water as H_2O is not a (human) ratiocinative activity -, it only means that water as H_2O, though being (necessarily) part of our epistemic practices, is not (essentially) those practices, or its meaning is not conferred by our epistemic practices.

Second, not every entity is intuitively artefactual or, more specifically, epistemic, for there need to be (at least) some entities able to play the role of fundamental entities, so as not to generate any vicious ontological regress.

Third, as an argument not much discussed in the literature, we could argue that objection (iv) leads to *instrumentalization* – even for an epistemic purpose -, which leads, in turn, to serious (bio)ethical issues: e.g. the *mere* fact of considering an organism - even modified in a lab for an experimental design - as (essentially) a tool, a craftwork, or something which is what I make from it viz. here, more precisely, an epistemic entity (even if many other ethical principles are respected: e.g. that it is a species capable of suffering, etc.) is deeply problematic by itself.

A way out here for objection (iv) to avoid the above absurd consequences is to try to demarcate somehow a theory of (natural) kinds/species from artefactual kinds by arguing that, if what species/kinds are is their epistemic utility, then this epistemic utility is measured, amidst others, with respect to the fact that species/kinds still capture something correct about the world (Magnus, 2012).

However, this way out is already too much of a concession: it goes back to putting primacy of metaphysics over the epistemological practice(s); species/kinds would no longer be (at least, completely) *made* by our epistemic practices. Can they be both somehow or a little mind-dependent and –independent, without being inconsistent? They cannot have it both ways.

Another way out for objection (iv) would be to subscribe to a certain *weak* instrumentalism i.e. the position that only *unobservable* or non-manipulatable entities are purely epistemic entities (Esfeld, 2009; Hacking, 1983).

However, the famous criterion of observability or manipulability for postulating, here, the mind-*independent* existence of entities is *not neutral*, in the sense that it is itself an epistemic criterion, which can, thus, varies from time to time depending on a certain state of knowledge.

If we are right with all this, then how to explain otherwise what was precisely the strength of objection (iv) i.e. an easy understanding of the relationship between (the metaphysics of) (natural) kinds and its epistemology?

Since this is a work in metaphysics of science, more precisely of disease (kinds) and related properties, and due to the high complexity of this question, we can only briefly consider it. If the reason why there are (natural) kinds/species cannot lie in the answer e.g. "Because we need them to make sense of our (scientific) explanatory or unificatory practices" i.e. in some *practical* reason – because of the fact that (natural) species/kinds are not there for us or any other designer -, then the reason why there are species/kinds lies in the answer "Because they have essences (or constituents)" (cf. Dasgupta, 2016 for the same point).

Why are there essences (or constituents)? What is clear is that *we need not* essences (or constituents), for they are not meant to be useful for, or to (epistemically) contribute to, whatever; albeit they are (certainly, very indirectly necessarily) part of our (explanatory) practices (albeit they can be also part of something else, of course), they *are not* (directly) (part of) those (explanatory) practices.

I follow, in the broad strokes, Dasgupta's metaphysical rationalism here, where for him such constituents exist, because they are *autonomous facts* i.e., translated in our own jargon, they are fundamental entities merely relatively realized i.e. that they somehow carry the world on their shoulders (for this point about "genus", cf. Sec. 3.3.1), albeit their credentials are, so to say,

purely *ontological* or only very indirectly epistemological[62]. Ditto for e.g. the subscription to a highly permissive ontology, or even the focus on the concept of disease (and related ones) in a PhD dissertation;
indeed, if we focus on the concept of disease (and related ones), that is simply because it is a *complex* and elusive concept – not because it is a general and widely used concept, whose analysis shall be (essentially) helpful to e.g. scientists or lay people -, whilst not ruling out, of course, that the topic of disease is (indirectly) e.g. relevant to other philosophical and scientific topics (e.g. health, life, death, maladaptation, genes, etc.) or explanatory practices, etc.

3.2.5 Objection (v): A Species as a Cluster Kind

A fifth popular objection that we could raise against our understanding of the genus-species relationship is based on a highly common strategy used in contemporary philosophy (e.g. of science) consisting in envisaging a species/kind as a *cluster kind* i.e. as a specific kind defined as a cluster whose (some, or *most*, of its) members share similar (or common) but non-essential properties (cf. e.g. Lewens, 2012; Ramsey, 2013); the strategy for positing the existence of kinds as cluster kinds is (epistemically) based on the conviction that (i) the history of the meaning of our words (or concepts) shows that we need flexible and revisable definitions of words (or concepts) (Machery, 2009; Walker & Rogers, 2018) (at least, of general words or concepts like SPECIES) – this can be roughly called "pessimistic meta-induction about concept analyses".
Furthermore, it is usually added that (ii) vagueness is inherent to our concept ascriptions (cf. e.g. Keil et al., 2017, for the very concept of mental disease; Bolton, 2008; Sadegh-Zadeh, 2000; 2008 for the concept of disease). This strategy goes back to (at least) Wittgenstein (1953)'s famous idea of *family resemblance* (cf. also Hull, 1965a-b; Pigliucci, 2003), to the widespread theory of concepts as *prototypes* (Rosch, 1978; Rosch & Mervis, 1975) or as *polythetic* (Needham, 1975; cf. Aragona, 2009 about mental diseases; e.g. on the (supposedly) 636, 120

[62] Caveat on the lack of explanatory value of essentialism (about disease): we may fear that, as stated here, essentialism (about disease) or, more generally, natural kind essentialism does not have much explanatory value, if it is only very indirectly related to explanatory practices; thus, why should a scientist subscribe to essentialism (about disease) (cf. Box 9.3 about essentialism about disease)?
Though we have to concede that point, we can still argue, nevertheless, that, generally speaking, as a metaphysical theory, natural kind essentialism ultimately constrains *epistemological* issues: e.g. it is intuitive that to explain the coronavirus is to provide evidence of knowledge of this disease, and this evidence spells out what the coronavirus is, or what its nature is (on that point, cf. Ch. 10).
I acknowledge Ulrich Krohs for having asked about that issue (personal communication, April-June 2021).

ways of having Post-Traumatic Stress Disorder (PTSD), Galatzer-Levy & Bryant, 2013) or as *polyeidic*, according to which, roughly,

> […] a lexical concept *C* doesn't have definitional structure but has probabilistic structure in that something falls under *C* just in case it satisfies a sufficient number of properties encoded by *C*'s constituents (Margolis & Laurence, 2019),

i.e. that the members of *C* all share some similar but non-essential properties, and to the *theory-theory* (of concepts) (Carey, 1985; Keil, 1989), which puts in analogy concepts with (a certain understanding of) scientific theorizing.

On basis of the further incitation that (iii), with the advent of the (neo-)Darwinian theory of evolution and the so-called modern synthesis, we are unable to find out (the set of the) essential properties characterizing a specific biological species (cf. Sec. 3.2.6 for the reply to motivations (i)-(iii)), many philosophers of biology (especially in the debate surrounding the specific concept of *biological* species itself in evolutionary biology (for the debate, as entries, cf. Ereshefsky, 2017; Wilkins, 2018)[63] - but also in e.g. philosophy of psychiatry (Varga, 2011; on, more generally, psychiatric kinds as looping or unstable, cf. Hacking, 1995), of cognitive science (Buckner, 2015), of chemistry (Häggqvist & Wikforss, 2018; *pace* Hoefer & Martí, 2019), or in metaphysics - have recently also followed the same trend (cf. Casetta & Vecchi, 2019; Ferreira Ruiz & Umerez, 2018; Griffiths, 1999; Hacking, 1991; Wilson et al., 2007) by typically relying further on Boyd (1999a-b)'s Homeostatic Property Cluster (HPC) theory of (natural) kinds, according to which, roughly, these similar but non-essential properties shared by the members of a cluster kind are clustered together through homeostatic mechanisms (on problems associated with the general reliance on homeostatic mechanisms, especially their insufficiency in theories of biological functions, cf. Boorse, 1977; Craver, 2009; Dussault & Gagné-Julien, 2015; Ereshefsky & Reydon, 2015; Krohs, 2009; 2011; Reydon, 2009a-b; Slater, 2015).

Thus, a supporter of the view of a *biological* species as a cluster kind shall typically argue, along Boyd's HPC theory of (natural) kinds – or, e.g. along another theory based on causal

[63] Two caveats (with terminological notes) on biological species/kinds and organisms: (i) some (cf. Ereshefsky, 2017) use the term "species" *tout court* to refer to what is for others, strictly speaking, *biological* species, where for the latter ones "species" *tout court* refers, in turn, to (natural) *kind* (cf. Koslicki, 2008). It is clear that I situate myself in this latter tradition.
(ii) The notion of biological species should not be confused with the narrower notion of *organism*, for, although an organism is arguably one *specific* biological species, not every specific biological species is an organism, for e.g. (indirect) (constitutive) *parts* of an organism are arguably specific biological species: e.g. blood can be said a specific biological species without being a specific organism (on my understanding of "organism", cf. Sec. 9.3.2).

networks (Borsboom, 2017) -, that homeostatic mechanisms (or, e.g. causal networks) are responsible for (or, underlie) the similar properties found among the members of the same *biological* species, where, for this supporter, the notion of *essence* has become in philosophy of science (especially in philosophy of evolutionary biology), in light of motivation (iii), out of flavor, old-fashioned, outdated or worth being eliminated, albeit it cannot be said *obsolete* (unlike superseded pseudo-scientific terms like "phlogiston", "miasm", "phrenology", etc.), as long as it is currently (and coherently) (as shown here) – though marginally – used and scrutinized in serious (or non-pseudoscientific) philosophy (of science) or, more specifically here, of evolutionary biology (on pseudoscience, cf. Exc. 9.4; On the indispensability of the notion of essence to solve a debate in philosophy of physics around spacetime substantivalism and determinism, Bartels, 1996).

Indeed, from the Latin translation of the Aristotelian "*οὐσία*" into "*essentia*" to the latest (neo-)Finean (1994a-b) theories of essence, one cannot sweep away more than 2000 years of the notion of essence (and as much controversy around it) by claiming it "obsolete" (without denying that, of course, in philosophy *of science*, the onus is on the essentialist – which explains the presence of Ch. 3).

If we apply now this strategy so as to raise a general objection against our definition of the genus-species relationship, then objection (v) amounts to the argued thesis that a species *tout court* is a cluster whose members share similar but non-essential properties under which the species falls, where the possession of some property *F* (e.g. *having a homeostatic mechanism*) makes true that the members share *these* similar but non-essential properties (or, constrains which properties are relevant for belonging to the same species); of course, as such, the basic idea of SPECIES as a cluster concept remains silent on whether this property *F* is to be conceived of as a genus (with a differentia) under which the species falls or as some absolute realizers of the species; we can stay content here by saying that property *F can* be conceived of as a genus, but it does not have to[64]: e.g. the *biological* species {human being} would be said here a cluster kind whose members share similar but non-essential properties like *being bipedal*, *having an articulated language*, etc., clustered together through the possession of a certain homeostatic mechanistic property (e.g. *being a rational animal*) and found out through e.g. *phenetic* analyses; the *biological* species {*Canis* (*lupus*) *familiaris*} or {dog} would be a cluster kind

[64] If objection (v) is cashed out as such, then many more specific theories of (natural) kinds, roughly, fall under it: e.g. Quine (1969)'s liberal conception of kinds as sets, Dupré (1993; 1999)'s and Hacking (1991)'s promiscuous realistic theory of (natural) kinds, or Ereshefsky and Reydon (2015)'s theory of (natural) kinds.

whose members share specific properties like *having four legs*, *having two eyes*, etc.[65], also clustered together through the possession of a certain homeostatic mechanistic property (e.g. *having a common genetic material*; *being exposed to the same environmental pressures*) – albeit a cluster kind does not *have to* be reductive, of course; the kind {STD} would be a cluster kind defined with respect to the common properties shared by its members clustered together through (or, underlid by) the possession of some property *F* (e.g. *being a disease spread by sex*).

To directly address objection (v), we can stay content with showing that a species is not a cluster kind whose members share similar but non-essential properties, because it is (absolutely intensionally) *reducible* to *plausible* views (among which ours) still appealing, actually, to the notion of essence, without this implying that we are necessarily talking, of course, about a genus in conjunction with a differentia i.e. without turning our reply to objection (v) into a positive argument for our own conception of the genus-species relationship (cf. Box 3.1; also Hommen, 2019 for a similar point).

Indeed, to reply to objection (v), it is sufficient to demonstrate that it is *not* necessary for a species to be a cluster kind (e.g. by showing that {species} can be plausibly conceived of as having an essence), rather than to precisely show that a species is to be strictly conceived of as a genus and a differentia.

A first shortcoming with objection (v) is that, at the reflexive level, we can argue that a theory of kinds as cluster kinds is self-contradictory. Indeed, objection (v) is based on the conviction that specific species/kinds like *Canis* (*lupus*) *familiaris* can be only *vaguely* (i.e. here non-essentially) analyzed, and that a general theory of species/kinds should take into account this fact.

[65] Caveat on taxonomies: some philosophers have argued that *commonsensical* (sometimes called "folk" or "lay") taxonomies (or taxonomies based on the manifest image) do not always correspond, of course, to *scientific* taxonomies (or taxonomies based on the scientific image) – though they may (e.g. *Boa constrictor*): e.g. "lily" does not have the same reference as the one of the biological kind called "*Lilium*" (cf. Dupré, 1993 for this famous example; also Strevens, 2019); or, "mold" has an everyday more inclusive acceptation than the restricted biological notion of mold.

By equating e.g. the biological species *Canis* (*lupus*) *familiaris* with what laymen are used to (commonsensically) call "dog", and by following our own metaphilosophy (cf. Ch. 2-3), I assume here that there is a *single correct* (general) taxonomy (or taxonomical system) viz. a common sense (or intuitive) one - within which some *specific* (typically lower-level) kinds or *constituents* of (higher-levels) kinds are studied and themselves classified, of course, by the (natural) *sciences* other than philosophy (e.g. cancer (kinds)), while others by *philosophy* (e.g. disease (kinds)) -, albeit a scientific taxonomy (typically when it goes outside its proper province) *may* correspond, indeed, to an intuitive one in some cases (cf. e.g. Sec. 4.1 for an intuitive taxonomy of disease kinds broadly corresponding to a scientific one).

Thus, talk about, in a certain *illustration*, e.g. the biological species *Canis* (*lupus*) *familiaris* – or, the use of the strict locution "*Canis* (*lupus*) *familiaris*" in an *example* - should not lead the reader think that we are *necessarily* following a *scientific* taxonomy (by opposition with a commonsense-based one), or that we are *necessarily* talking about something else than a *dog*, although, in the context of an example, we can *plausibly* do so, of course.

However, this general *theory* of species/kinds as clusters is itself not vague, but it can, rather, be interpreted as stating that what is *essential* to a kind/species is that its members share similar but *non-essential* properties.

But, secondly and more importantly, the main shortcoming with objection (v) is that, following our previous answers to objections (i) and (ii) of, respectively, Sec. 3.2.1 and 3.2.2, objection (v) is (absolutely intensionally) reducible to a (minimal) conception of species as having one and the same essence, which variously deals, as far as it can, with the above motivations (i)-(iii) at the basis of objection (v); from this, one can still consistently claim that there are, indeed, *specific* species (*pace* Brigandt, 2003 – which admits the point, but still tries to argue for a certain pluralism with respect to the biological species concept).

Indeed, saying that e.g. what makes true the fact that the members of {*Canis* (*lupus*) *familiaris*} share similar properties is their sharing a homeostatic mechanism viz. a common genetic material or the exposition to the same environmental pressures just amounts to saying that all the members of {*Canis* (*lupus*) *familiaris*} share the same *essence* (cf. Slater, 2015 for a similar point).

That much does not imply, of course, that this specific essence is necessarily the *correct* one for the species *Canis* (*lupus*) *familiaris*, but it still remains true that we can *plausibly* conceive of it as an essence as e.g. Walsh (2006)'s evolutionary essentialism (i.e., roughly, an essence as some goal-directed capacity (e.g. a homeostatic mechanism) for a species/kind) or or even as our own conception of essence does (cf. Ch. 2; Sec. 3.1.1) (cf. also the so-called "relational essentialism" of Austin, 2018; Griffiths, 1999; LaPorte, 2004; Okasha, 2002, or the "intrinsic essentialism" of Devitt, 2008; *pace* Barker, 2010; Lewens, 2012; also Hommen, 2019), or the "origin essentialism" of Kripke, 1980; Forbes, 1986; Salmon 1981)[66]. We see no reason why the word "essence" could not be used, indeed, (with a non-empty meaning) in those circumstances.

3.2.6 Objection (vi): A Species as an Evolving Kind

A sixth and last objection – closely related to objection (v) (cf. Sec. 3.2.5) – comes as follows: a species cannot be essentially a genus plus a differentia, but is, actually, an *evolving kind*

[66] Terminological note on (biological) natural kind essentialism: for us, talk about (biological) natural kind essentialism is either redundant, for the notion of essence only applies, indeed, to kinds/species, or uselessly restrictive, for the notion of essence also applies to *artefactual* kinds, actually.

understood in a certain way i.e. (for the sake of objection (vi)) a kind absolutely extensionally defined with respect to its members changing through time (or, minimally taken, as a *lineage* i.e. descent from a common ancestor)[67]. Along objection (vi), a species/kind *tout court* is an evolving (or changing) kind through time defined with respect to its (diachronic) absolute realizers i.e. that a species is, more precisely, a *collection* (or a class) of specific evolving kinds (at least one) – and not defined as having an essence in the (plausible) form of e.g. a genus and a differentia, or maybe even a cluster kind (cf. objection (v) in Sec. 3.2.5), or as having multiple essences.

As for objection (v) (cf. Sec. 3.2.5), the idea at the basis of objection (vi) is peculiarly widespread in the debate surrounding the concept of *biological* species (Ereshefsky, 2017; cf. e.g. Kronfeldner, 2018 for a focus on human nature), where it is argued in our post-Darwinian era that a biological species cannot be defined by sorting out its specific essence like, in our case, a specific genus and a specific differentia, for a biological species is a specific evolving kind (as understood along objection (vi)) (on, more generally, so-called *Darwinian metaphysics* or, more generally, *evolutionary naturalism*, cf. e.g. Faye, 2016; von Sydow, 2012); David Hull (1965a-b; 1978) e.g. has argued that biological speciation is a gradual process, and that, as such, there is no way to draw a precise boundary between one biological species and another one[68].

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[67] Four caveats on objection (vi): (i) as such, objection (vi) follows in a specific way a process ontology (more generally, on the so-called doctrine or tenet of temporal parts (or *four*-dimensionalism or *perdurantism*), cf. e.g. Hawley, 2020; Lewis, 1986).

(ii) One can say that objection (v) (cf. Sec. 3.2.5) is about how to *synchronically* (i.e. in a *three*-dimensionalist framework or *endurantism*) define a species by allowing sufficient (synchronic) variation within it (i.e. by extensionally defining "species" in terms of some of its (synchronic) members) (cf. Koslicki, 2003 on three-dimensionalism), while objection (vi) is about how to *diachronically* define (i.e. within a process ontology) a species by allowing sufficient (diachronic) variation within it (i.e. by absolutely extensionally defining "species" in terms of its (diachronic) absolute realizers).

In that sense, objection (v) (cf. Sec. 3.2.5) (when specifically about biological species, of course) is more related to *phenetics*, while objection (vi) to *cladistics* or, metaphorically, evolutionary trees (in systematics, narrowly taken).

(iii) It is paramount not to assimilate our constitutive ontology (cf. Ch. 2) with *three-dimensionalism* i.e. with an ontology of *states* or, at a more general level, (commonly but misleadingly said (Stout, 2016) – due to the prima facie exclusion of *instant* states) of *continuants* (on states, cf. e.g. Marcus, 2009; on continuants, Wiggins, 2016). As such, our constitutive ontology is compatible both with a *state* ontology and a *process* ontology (on process philosophy and (causal) processes, cf., amongst others, Rescher, 1996; Seibt, 2017). Indeed, a temporally extended entity viz., more specifically, a *process* can *plausibly* be constitutively defined i.e. within (our understanding of) a genus-species hierarchy (cf. Box 3.1).

(iv) Thus, for objection (vi) to be a proper objection against our definition of species/kinds (cf. Box 3.1) it is not sufficient to draw on a dynamical/evolutionary picture of kinds (since, as such, an evolving kind may be plausibly essentially defined), but on an *extensionalist* dynamical picture of kinds.

Indeed, it is not because a process may be minimally taken as a temporal succession of *states* x, y and z to get a certain state a (cf. Box 9.2) that we are *extensionally* defining a process as a temporal succession of varied specific *kinds*/*species*. Thus, addressing objection (vi) does *not* amount to showing that a species is not to be conceived of as an evolving kind *simpliciter*, but to showing that a species is not to be conceived of as an evolving kind *absolutely extensionally defined* i.e. as a *collection* of specific evolving kinds.

[68] This historical line of thought, actually, goes back to (at least) the famous sorites paradoxes (as an entry, cf. Hyde & Raffman, 2018; on vagueness, cf. Sec. 3.2.5; Sorensen, 2018).

An evolutionary approach to the kind/species *disease* (*kind*) itself - and not specifically to *biological* species - is also very widespread. This approach is sometimes called "evolutionary medicine" (cf. Ananth, 2016; 2017; Nesse, 2001; Nesse & Williams, 1994; Sarto-Jackson, 2018; for the more specific distinction between *evolutionary* and *Darwinian* medicine, where, roughly, Darwinian medicine is the narrow field relying on *natural selection* only, Méthot, 2015; on, more specifically, evolutionary psychiatry, Adriaens & De Block, 2011; for criticisms on evolutionary medicine, Cournoyea, 2017): it is claimed that a disease (kind) should be *evolutionarily* defined (understood in a certain sense, of course)[69].

In a nutshell, evolutionary medicine (in its strong version) applies evolutionary thinking (in a certain way) about disease (kinds) like evolutionary biology about biological species: e.g. instead of defining a specific disease like atherosclerosis (or arteriosclerotic vascular disease) as being constituted by a long generation of atheromatous plaques or a kind of heart attack as being constituted by a heart attack (disease) plus specific negative causes and effects (cf. Box 4.2) (i.e. a *proximate constitutive explanation*), evolutionary medical practitioners are looking for the reasons why people continue *now* to be diseased – or, why natural selection does not fully eliminate diseases - through understanding how past species have developed specific diseases; they typically answer that diseases can be *ultimately* evolutionarily explained through a *temporal mismatch* between inheritance of genetic processes - acknowledged as ultimate or fundamental units - from the Pleistocene era and (rapid) environmental changes through time (i.e. an *ultimate evolutionary explanation* – especially here in the narrow field of Darwinian medicine) (on the distinction between *proximate* and *ultimate* explanations, cf. Mayr, 1963; Tinbergen, 1963; also Kitcher, 1984; Nesse, 2013; on issues pertaining to this famous distinction, Exc. 3.7).

[69] Caveat on evolutionary medicine and fallacies: (i) to remain fully consistent with objection (vi), according to which species are to be *uniquely* defined as evolving kinds (as understood along objection (vi)), I rely here on a *strong* version of evolutionary medicine, according to which diseases are to be *uniquely* defined along an evolutionary line.
Note, however, that most (if not all) evolutionary medical practitioners (and philosophers) hold a pluralist (however understood) or *compatibilist* view between what they take as evolutionary explanations and other specific non-evolutionary explanations: e.g. proximate causal explanations, proximate constitutive explanations, etc. (cf. Méthot, 2015).
Albeit not common, holding a *strong* view on evolutionary medicine does not make our point here a straw man argument, for a straw man argument is a fallacious attack on one's opponent view by intentionally mis(re)formulating the opponent's position or misattributing to the opponent a certain position – which is clearly not the case here with a simple distinction between two (plausible) positions.
Furthermore, a(n) undefended or not much defended view might well be very much plausible or just overlooked; thus, attacking a(n) undefended or not much defended position does not make the argument(s) necessarily *weak*.
I acknowledge Nina Kranke (personal communication, November 2018) for having pointed this to me.

Thus, for evolutionary medicine (in a strong version), a disease is to be defined not e.g. constitutively, but (diachronically) absolutely extensionally through understanding how past species have developed specific diseases.

Another compelling (and pervasive) empirical argument in favor of objection (vi) has been put forward, according to which, if a biological species is necessarily absolutely realized (or necessarily has an absolute extension) (cf. Sec. 3.3.1), then positing (immutable) *essences* (like a genus and a differentia) makes it unlikely to ever find absolute realizers, mostly due to (diachronic) variation (or *variability*) in traits (or genetic processes) within the absolute realizers of one and the same biological species (Sober, 1980; cf. also Walsh, 2006).

A third powerful (and cogent) empirical argument is that the view of biological species as having an essence rests on a picture of kinds as time-less (or *eternal*), and this picture is untenable in light of e.g. the history of species extinction/birth (i.e. speciation) (Richards, 2016).

A first quick and bad reply to objection (vi) is to argue that objection (vi), by defining a species as an evolving kind (as understood along objection (vi)), absolutely extensionally defines a species with respect to its realizers. As such, objection (vi) would be, thus, an objection against intensional definitions (cf. Ch. 2) – and not directly against our definition of the genus-species relationship (cf. Box 3.1).

Nevertheless, even if this is true, it remains the case that objection (vi) does not pronounce beyond a certain (plausible) definition of a species; objection (vi)'s scope is here limited to our analysis of a species, to be precise (cf. Box 3.1). Thus, objection (vi) is to be addressed at that same level.

The second reply to objection (vi) replies, more precisely, to Hull's vagueness argument for (biological) speciation: it is argued that, along objection (vi), it is not clear, in evolutionary theory, at what precise point in time we can safely say that what we get are two different biological species (with different absolute realizers) rather than two different absolute realizers of one and the same biological species.

However, we can still ask: until what *drastic* extent can we really allow (trait or genetic process) variations to obtain among the absolute realizers, which are *themselves* specific (here biological) species or *organisms* (since, along objection (vi), a species is absolutely extensionally defined), of the same (biological) species to still talk about absolute realizers falling under *one and the same* (biological) species (on the phenomenon known as *phenotypic plasticity* or *novelty*, cf. Peterson & Müller, 2015; on e.g. *neuro*plasticity as a certain phenotypic plasticity, Mandolesi et al., 2017; on holobionts and phenotypic variation, Haag, 2018; on the history of variation, Winther, 2000)? Which exact range of (allegedly) *normal* (trait or genetic

process) variations (or, trait/genetic process variations within a same standard or norm) can we allow to obtain? Or, when does (biological) speciation really occur?

E.g., in the course of biological speciation, when can a (biological) variety or specimen (more precisely, an absolute realizer here) of a biological species called "Darwin's finches" be still said a variety (or, an illustration or example) of *this* species or, to the contrary, a variety of another biological species (on Darwin's finches, cf. Sachse, 2011)? Following *modern synthesis* (Dobzhansky, 1937; Fisher, 1930; Mayr, 1942; cf. also Mayr & Provine, 1980), one can bring the answer that, due to different environments of evolutionary adaptedness in the Galápagos, different advantageous genetic mutations are fixed in a variety of Darwin's finches such that this variety cannot interbreed any more with the other varieties, where, at this precise moment, this variety belongs, thus, to another biological species than Darwin's finches.

However *arbitrary* the non-interbreeding condition seems to be – for, the mere possibility to produce such offspring through interbreeding is not excluded (e.g. zebroids or zebra hybrids i.e. the offspring of a zebra and an equine, or ligers/tigons i.e. the offspring of a male lion/tiger and a female tiger/lion, etc.), although they are rarely healthy species (but they are still (diseased) biological species) -, this condition for delineating which varieties can belong to one and the same biological species (absolutely extensionally defined) can also be seen precisely as a(n) (fixed or permanent) *essential* feature for *x* and *y* to belong to one and the same biological species (cf. also Dasgupta, 2016 for a similar point about why-questions). As such, a biological species (even understood as an evolving kind) can be still *plausibly* defined with an essence (cf. Sec. 3.1.1-3.1.2).

Ditto for other (weakly) pluralist attempts claiming that (biological) species e.g. "[…] are segments of metapopulation lineages whose constituents are Mendelian populations characterized by their own gene-phene pool" (Casetta & Vecchi, 2019, p. 3) or that "[…] virtually all contemporary definitions of the species category are based on a common general concept of species: the concept of species as (segments of) metapopulation lineages" (de Queiroz, 2005, p. 6602; cf. also 2007) i.e. an homogeneous population with common ancestry, or as a *clade* – which, like for our reply to objection (v) (cf. Sec. 3.2.5), can be assimilated, indeed, to an *essential* feature (compatible with our own account) for something to be, actually, a (biological) species -, or even definitions of species based on inclusive fitness or reproduction

(Foot, 2001; Thompson, 2008; also Kronfeldner, 2018) or homeostasis, without rejecting, of course, the existence of *different* more specific concepts of a (biological) species[70].

Note that the same is also true for evolutionary medicine (in a strong version). Indeed, although evolutionary medicine can be said to absolutely extensionally define diseases as specific diseases changing through time, these specific diseases seem to be themselves defined not absolutely extensionally – rather, as genetic processes environmentally *maladapted*.

To sum up, a definition of kinds as evolving kinds (as understood along objection (vi)) cannot *replace*, or fails to get rid of, an essentialist (under a plausible if not correct reading) approach to kinds. As for our reply to objection (v) (cf. Sec. 3.2.5), we can argue that objection (vi) can be reduced to a (plausible if not correct) essentialism about species/kinds.

To more directly and generally tackle the vagueness argument, we can use the famous ship of Theseus puzzle (at least, in one of its (most ancient) interpretations): at which moment or is there a moment in time, where the original ship of Theseus constituted by planks, which are gradually replaced in time, ceases to be the original ship of Theseus to become another replaced ship?

A proponent of kinds as evolving kinds (as understood along objection (vi)) shall answer that to have vague boundaries (or, that there is no clear-cut answer to the ship of Theseus paradox) is, actually, a virtue for a theory of kinds (cf. Barnes, 2010; Barnes & Williams, 2011).

But, the question of how to characterize the permissible changes (or *modifications*) for a specific species to remain the same species, and the ones that are not permissible, still remains to be answered.

Along a (neo-)Aristotelian line of thought about change (over time), change comes only in two varieties (on that, cf. Brower, 2010; Cohen, 2012; Gallois, 2017; Mortensen, 2020): (i) *accidental* (or extrinsic) change (also called "*alteration*" in the Aristotelian idiom (*Phys*. A.7) - at least, for qualities) (plausibly taken); (ii) *substantial* change (*GC*. I.4, 319b25-319b31) (or *transformation*, in an Aristotelian vein).

Thus, along this Aristotelian line (*GC*. II.1-5; cf. *Phys*. Z; also Charles, 2017), either a specific kind/species (e.g. a state or a process) does *not*, actually, change (or, becomes different), but only its accidental (or extrinsic, in our jargon) properties change (i.e. *accidental or extrinsic change*), or (a) specific kind(s)/species(s) (typically *states* or *processes* themselves) *do*(*es*) change i.e. that (a) same-level specific kind(s)/species(s) is/are generated or destroyed through

[70] Caveat on *homology*: note that homologuous features are unlikely to count as essential features, for a feature of *x* shared in ancestry with another one does not imply that this feature is (plausibly if not correctly) essential to *x*; indeed, this feature can very well be accidental or extrinsic (though shared in ancestry).

a *process* (i.e. *substantial change* or change *tout court*, when sufficiently clear): e.g., in our vein, from *non*-two daughter cells divided and grouped to two daughter cells divided and grouped through the process of *cell division* (on the distinction between varieties of change (i) and (ii) as a way to solve the so-called *paradox of change*, according to which, if something changes, then it cannot remain one and the same, but if it does not remain somehow one and the same, then it just cannot undergo any change, cf. Gallois, 2017; Hofweber, 2009).

But, if an evolving kind (as understood along objection (vi)) falls under substantial change (of states or of processes) (as understood here), then there are no permissible changes for an evolving kind, for it *is* a change of states or of processes themselves (cf. Box 9.2 for our definition of a process).

However, if so, this evolving kind does not have to be *necessarily* defined, along objection (vi), absolutely extensionally with respect to specific (diachronic) *kinds* – thus, also themselves evolving kinds (defined in the same way) -, for we might argue that there is no more reason to use substantial change as being how a process is (being) generated/destroyed through its intensional parts understood here as (lower-level) processes themselves than to use substantial change as being about how, from/to a contradictory *state* of (¬)*y*, a certain *state* (¬)*y* is (being) generated/destroyed by generating/destroying (¬)*y*'s intensional parts understood *here* as specific *states*. Thus, there is no (positive) reason to *absolutely extensionally* define an evolving kind: it can be plausibly defined along an essentialist line.

The third reply to objection (vi) addresses, more precisely, Sober's argument for the impossibility to find out absolute realizers: a species/kind is not a temporal succession (or change) of miscellaneous (at least, one) specific species/kinds themselves (also defined as evolving kinds – along objection (vi)).

As a first but weak answer to this challenge, it is not definitional to a species/kind to be a temporal succession of *several* specific species/kinds.

Indeed, it may very well happen that e.g. a biological species had just one single variant throughout the history of life on earth (or in other planetary systems or even in possible worlds): e.g. some bacteria are fossilized in the same environmental conditions for billion years; or, so-called "living fossils" (e.g. goblin sharks, platypuses, coelacanths, tadpole shrimps, etc.) have constantly remained through time (almost) alike, while their environmental conditions may have changed.

One could object here that a species/kind is still capable of evolution, or a species/kind is *evolvable* (cf. Bedau, 2008). Beyond the fact that a theory of kinds as evolvable just is different from a theory of kinds as evolv*ing*, the theory of kinds as evolvable also suffers from its own

problems like the issue concerning the fact that, if a specific kind were never to manifest its disposition to evolve, then we would certainly be reluctant in claiming that this kind had, actually, the capacity to evolve, but never used it; we would certainly be more sympathetic to the idea that this kind, rather, just never had the disposition in question.

Nevertheless, all this does not undermine the fact that one can stay content with having necessarily for a species/kind (at least) *one* single variant (or absolute realizer) (cf. Sec. 3.3.1). Second, in order to find out *plausible* absolute realizers of a specific kind/species, it is not necessary to define a kind as an evolving kind (as understood along objection (vi)), but it is sufficient to demonstrate that every (even teeny tiny) (trait or genetic process) *variation* (or absolute realizer of an evolving kind) can be, actually, plausibly defined as a specific (biological) *kind* along an essentialist line (although not necessarily, of course, within a genus-species hierarchy (as understood here)).

There is no (phenotypic or genetic) variation to be allowed (as *defining* features) within one and the same specific (biological) kind/species: e.g. *one and the same* plant *Hieracium umbellatum* does not have, definitionally speaking, phenotypic variations depending on a change of environment, or does not *adapt* to (or, accommodate with) environmental change *through change* (e.g. as a plant with specifically shaped leaves and inflorescences in a rocky environment; or as a plant with differently shaped leaves and inflorescences in a desert environment), but these plants (which may be situated in different environments) can very well be counted just as *two* different or *varied Hieracia umbellata* in their own right (as long as, of course, a *specific* shape of leaves and inflorescences is *not* an essential *quantitative* property of *Hieracium umbellatum*) (on quantitative kinds/species, cf. Exc. 3.4; on our permissive neo-Aristotelian ontology, Exc. 3.5)[71].

A consequence of this idea is that we should acknowledge the existence of many more (highly specific) kinds/species than the ones whose existence is taken prima facie for granted today in quotidian discourse and in the sciences. In that sense, an austere Quinean-style ontology of seeing species as evolving kinds (as understood along objection (vi)) would be replaced by a permissive neo-Aristotelian ontology leaving room for far many more (highly) specific species than are widely acknowledged right now (on precision medicine, cf. Exc. 3.6).

[71] Two caveats here on environmental change: (i) that there is no genetic or phenotypic variation to be allowed should not be taken as implying the rough idea that health is based, to the contrary, strictly speaking, on the *resistance* to e.g. environmental pressures or forces (cf. Sec. 9.3.3).
(ii) Nor should our point be taken as implying a *state* ontology, albeit this is unfortunately often done in the literature (cf. e.g. DiFrisco, 2018).

A major advantage with this permissive neo-Aristotelian ontology is that we can very well acknowledge that (specific) kinds like disease(s) (kinds) can be, in a certain minimal sense, *context-sensitive* or *–dependent* without resorting to, nevertheless, *contextualist* (or *perspectivist*) solutions[72], or making our (monistic) ontology a shallow essentialism *à la* L. A. Paul (2006; cf. also Forbes, 1986), for the multiplication of existing kinds/species makes it unlikely that there are (at least) two different (correct) irreconcilable perspectives on *one and the same* specific kind/species, rather than, actually, two different highly specific kinds/species themselves (with a *single* correct perspective on each one), which *can* very well appear, of course, in two different contexts, settings or environments (of evolutionary adaptedness) i.e. that, even disparate, such entities could still be said specific existent kinds/species themselves perhaps falling under a more general existent species/kind too (however minimally such a general kind is to be defined).

A second advantage is that it allows us to give a simple account – or rather, a dissolution - of the (related) problem of *vestigiality* i.e. the issue over the continuous presence of some species/kinds (e.g. genetic processes/traits) supposed to have lost what they were originally meant for, their original essence or function.

Indeed, if we are right that species/kinds should not be defined as evolving (or as being somehow related to past variants), then there is absolutely no reason to hold that e.g. the human appendix (as contemporarily known) is a vestige, for the human appendix (with what it was meant for) *in the past* is *not* the same as the current human appendix, which has its own essence or function (still much debated) – it was only another (past) *variant* of the human appendix with its own essence or function: e.g. maybe digesting the cellulose of plants (like for other primates).

Or, another way to dissolve the vestigiality problem is to argue that, to the contrary, the current human appendix (as being still different from a past one) is just the host of a certain biological *mal*function.

[72] Caveat on quantitative kinds: with our permissive neo-Aristotelian ontology, there also exist varied normal quantitative kinds (all with different correct – but still *single* - values), which just are different depending on the context or environment where they are to be found: e.g. having a 20/10 visual acuity on the Snellen chart is certainly having for a human being a normal sight *within a bright environment* (necessarily absolutely realized, of course).

However, so as not to fall with the same problem of arbitrariness and vagueness (cf. Sec. 3.2.3), one cannot say that there would exist, thus, one single overall quantitative kind, under which all the other more specific quantitative kinds (with different values) would fall. Does it mean that there is no species e.g. human being or *Canis* (*lupus*) *familiaris* existing out of a certain context?

That there is no, indeed, overall quantitative kind does not mean that there is no overall *qualitative* kind: e.g. the species/kind human being or *Canis* (*lupus*) *familiaris* (*tout court*) would exist as a qualitative kind.

I acknowledge Ulrich Krohs (personal communication, April-June 2021) for having asked about this issue.

Drawing on the acknowledgement of the existence of highly specific kinds/species (for a plea that the Diagnostic and Statistical Manual of Mental Disorders (DSM) should pursue into this direction, cf. Tsou, 2019), it is not necessary to define, thus, a kind as a temporal succession (or change) of varied specific kinds; Sober's argument is not a motivation for subscribing to a view of kinds as evolving kinds (as understood along objection (vi)), if we subscribe to a maximal neo-Aristotelian ontology[73].

A fourth reply to objection (vi)'s last argument is that it is not necessary to argue for kinds/species as evolving (as understood along objection (vi)) to account for species extinction/birth (i.e. speciation).

Indeed, one of the arguments in favor of objection (vi) is that a conception of kinds as having an (immutable) essence (e.g. a genus plus a differentia) presupposes a picture of kinds as *time-less* (or eternal), and that such a picture should be abandoned in favor of a picture of kinds as evolving kinds (as understood along objection (vi)).

However, this presupposition from objection (vi) is false: one can perfectly well sustain an essentialist (under a plausible reading) view of species as well as account for species extinction/birth i.e. being both in favor of seeing species as plausibly having an essence and against seeing them as *time-less*: e.g. the (here natural) kind/species *Tyrannosaurus rex* can very well be conceived of as a specific biological species plausibly having an essence, whose *type* was instantiated at time t_{-1} (or from a certain stretch of time to another one, if *T. rex* is deemed a process), but is no longer instantiated at time t_0 i.e. that no token at time t_0 instantiates it[74].

[73] Caveat on higher/lower-level diseases: exactly like a specific disease may have as constituents (what absolutely fall under) specific lower-level diseases themselves, but *is* not (to be strictly defined as) a set of these lower-level diseases, a certain (yet to be fully generated/born) human being may be (plausibly) said to have as constituents (what absolutely fall under) other specific lower-level human beings themselves (e.g. *Homo sapiens*, *Homo sapiens sapiens*, etc.), but *is* not (to be strictly defined as) a set of those lower-level human beings.

[74] I assume here that *token representations* at time t_0 (e.g. in silico, on a movie screen, in a comics, in a book, etc.) of *T. rex* are here not truly tokens themselves of the (natural) biological species *T. rex* – albeit *T. rex* (as a natural biological kind) may very well reappear at a later time t_{+1}, of course.

Indeed, such representations may be said *model descriptions* (or *implementations*) viz. of a model (or a representation) of *T. rex* (cf. Weisberg, 2013 for the so-called indirect view of models).

Or, if *T. rex* is really to be said a token *tout court* in this context at time t_0, then it is maybe a token of the *artefactual* (or, more precisely, fictional) type *T. rex*.

However, with this distinction in hand, we may worry that a maximal neo-Aristotelian ontology confuses tokens with token *representations*, for we could argue that e.g. a token Pegasus does not exist as such, but only token representations of it truly exist i.e. that only (at least) one token model (or model description) of Pegasus truly exists – not one token Pegasus itself.

Moreover, this confusion can be also easily motivated by holding a representationalist account of tokens, where, roughly, tokens are said *representations* of a type (Szabó, 1999).

However, first, a token model *of Pegasus* still targets Pegasus, albeit, at a more global level, artefactual objects can certainly be considered themselves as being somehow representations (or imitations) of *natural* objects: can there consistently *be* targets which *are non-existent* (on this famous problem, cf. Reicher, 2019)?

And if it happens that a token of the type is present at time t_{+1}, then we shall *not* say that the type *T. rex* was present between the time interval t_0 and t_{+1} (when there is no instance of it), but that the *very same* type just comes into existence (once again) at the same time t_{+1}, when there is (at least) one token of it (on the doctrine of so-called *uniuersalia in rebus* or immanent universals, cf. Armstrong, 1978; Wollheim, 1968)[75].

In other words, following our illustration, the (very same) type *T. rex* (because it is a universal) may be said to have an *intermittent* (or occasional) *existence* (cf. Koslicki, 2008) or, more simply put, to *re*appear, depending on the presence and absence of its tokens.

Ditto, of course, for specific *resurrected* diseases (kinds) themselves (which have been wrongly assessed (or estimated) in the past as completely eradicated *forever*)[76]: e.g. sweating disease

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Second, even though we maintain a strict distinction between a token model and its target (system), *abstract artifacts* (or, more specifically, an artefactual type) like Pegasus may truly have tokens, which are not to be interpreted as token *models* of Pegasus: we can intentionally construct or build e.g. a token computer, without this implying that this construction is a token *model* of a computer, or e.g. the (alleged) *replica* of the Trojan horse situated in Canakkale in Turkey is maybe (here) *one* token of the Trojan horse – rather than a token *model* of the Trojan horse - i.e. that we have to distinguish between a *token* of an artefactual type itself like a computer and a *token representation* of it (e.g. a token drawing/painting/etc. of a computer), albeit the former can also very well be used, of course, as (a vehicle for) a certain representation (e.g. a computer as a representation of a human brain). That much also applies, of course, to *concepts*.

[75] Three caveats with terminological notes on tropes and immanent universals: (i) we may fear that with a maximal or permissive neo-Aristotelian ontology we eventually do not get universals but *tropes* (understood as abstract particulars (plausibly taken)) (on tropes, cf. Campbell, 1990; Maurin, 2013; Mulligan et al., 1984; Smith & Mulligan, 1983), for, if there are so many different specific universals (alongside specific kinds/species), then a trope theorist might argue that there is always, actually, one and exactly only one, here concrete, particular of it. Thus, there are no universals but tropes (on the well-known related idea of so-called *species as individuals* (i.e. here tropes), cf. Ghiselin, 1974; Hull, 1978; Kitcher, 1984).

However, although it is true that with a maximal neo-Aristotelian ontology it may happen in plenty of cases that there is *one* token (or absolute/relative realizer) of a type (or kind) (in so doing, we may also fear here that acknowledging the existence of a type/kind *and* its token/absolute (or relative) realizer is *redundant*), a definition of a type/universal (or of relative/absolute extension) – to the contrary of a definition of a trope (which is arguably also a redundant entity, at least for nominalists) – does not require that one and exactly one token must be present, but leaves open the possibility for more than one token (or absolute/relative realizer) to be present, or *minimally* requires that *one* token (or absolute/relative realizer) be *at least* present.

(ii) We may also fear that rejecting tropes implies rejecting the basic idea behind precision medicine (but, cf. Exc. 3.6). The rejection of tropes only implies the rejection of a *strong* understanding of precision medicine, according to which only a *particular* therapeutic treatment is truly efficacious, for there would be only (concrete) *particular* diseases with their *tropes*.

(iii) Note finally that, in the context of the doctrine of immanent universals, E. J. Lowe (2006) usefully distinguishes further between a *strong* claim about immanent universals and a *weak* one. According to the strong claim (attributed to Armstrong (1978)), universals are themselves concrete (i.e. here spatio-temporal) multi-located entities, while the weak claim denies this.

Whilst the strong claim has been rightly criticized as being incoherent (Keskinen et al., 2015; Lowe, 1998; 2006), we are in no way committed to it. For, we take universals as specific *abstract* entities.

[76] Can a specific disease (kind) really be said resurrected? Some may want to argue, rather, in the case of a specific disease (kind) manifested once again at time t_{+1}, that this specific disease (kind) had not completely disappeared from time t_0 to t_{+1}, but that it was, actually, *dormant* or *latent* (under a certain understanding of it) (cf. Sec. 10.1.5) (e.g. the specific viral process responsible for the Spanish flu only latently present at time t_0 is said to have been *revived* by the effort of scientists to *recreate* (or *reconstruct*) the viral process in question with the disease process i.e. the etiopathological agent) (cf. e.g. Kobasa et al., 2007 for the experiment on macaques), meaning by this that the disease (kind) in question was *somehow* (i.e. as a disposition) still present out there in the world from time t_0 to t_{+1}, but that it had no *manifestation* from time t_0 to t_{+1}.

(also known as English sweating disease or English sweate) is a specific contagious disease said to have spread throughout the European continent especially in England from 1485 to 1551, and to completely vanish after 1551 (Heyman et al., 2014). Sweating disease is a specific higher-level disease kind whose exact specific (higher-level) negative causes and effects are unknown (cf. Box 4.2 on disease kinds), but the disease process in question has been roughly characterized mostly by the physicians John Caius and Thomas Le Forestier as a three-step process from a "cold step" (mostly, sense of apprehension and shivers) through a "hot step" (mostly, sweating and delirium) to the last "fatal step" (mostly, collapse)[77].

Although this is much disputed (by e.g. Bridson, 2001), (a constituent of) an *indirect* absolute realizer, or an indirect constituent itself, of "sweating disease" is said to be a variant of a hantavirus process (Taviner et al., 1998)[78].

However, after its 1551 eradication, sweating disease, understood here as a higher-level contagious disease, *reappears* (under the new name of "Pickardy sweat") in France between 1718 and 1918. If we assume that sweating disease and Pickardy sweat are one and the same specific disease kind (cf. Tidy, 1945), then, as a specific higher-level disease kind, although we can defend that an indirect constituent, or (an intensional part of) an indirect absolute realizer, of "sweating disease" viz. a hantavirus process was still very likely present between 1551 and 1718, sweat disease may be said a specific resurrected disease kind.

In the case of the Spanish flu, I agree that the infection is still somehow present at time t_0, but not *essentially* as a disposition: the infection in question is *partly* present – and not completely eradicated -, for an infectious agent (or the viral process) (taken here as a specific cause) (cf. Box 4.2 for the definition of disease kinds) in question is still present on dead bodies which were infected, and it *necessarily* has a *direct* relative extension (cf. Sec. 3.3 on that) i.e. that this viral process is necessarily a constitutive part of something (viz. here the Spanish flu e.g.); note that this is also the case for many now frozen (or preserved in old permafrost layers) ancient bacteria and viruses (as *states*) (cf. Legendre et al., 2015).

However, against a certain understanding of RME, it remains true that the Spanish flu is not essentially a disposition.

One may eventually worry how a specific disease (kind) can be ever *completely* eradicated (or extinct) - at time *t*, at least.

Indeed, in many cases of what prima facie seems disease (kind) eradication, one can easily argue that a constitutive part of the specific (supposedly completely eradicated) disease (kind) is still, actually, present, or that many famous cases of disease (kind) like scurvy (e.g. still present in France in 2015), tuberculosis, scabies, cholera or even plague (e.g. still present recently in Madagascar) are very unlikely to be ever completely extinct.

However, in specific higher-level disease(s) (kinds) (i.e. *highly emergent* or derivative disease(s) (kinds) like mental disease(s) (kinds)), it is plausible that *only* (constituents of) their *indirect* absolute realizer(s) or their *indirect* constituents are truly present – thus, that the specific disease (kind) be possibly completely eradicated.

[77] For sake of illustration, I assume here that there exists a *plausible* reading of sweating disease, where my three-step characterization captures the disease process in question – rather than what may belong to its causes or effects, for these three steps are also sometimes referred to as the causes and the effects of sweating disease (cf. also Heyman et al., 2014). This latter reading of sweating disease can be, of course, used as an illustration for an objection against what a disease kind is (cf. Sec. 4.2), but this is out of context here.

[78] Following Padula et al. (1998), where it is shown that hantaviral human-to-human transmission can happen, I assume here that a hantavirus process is a plausible candidate as an indirect constituent of sweating disease, or as (a constituent of) an indirect absolute realizer of "sweating disease".

However, in so far as we decide to strictly distinguish between kinds and types, we may worry that our answer confuses *kinds* with *types*, or that, along our picture, although types are instantiated universals, kinds could still be conceived of as Platonic objects i.e. here as eternal beings, for objection (vi) is based on the idea of *kinds* as time-less, and our above answer consists in saying that *types* (or universals) do not exist without (at least) one token (or particular).

Thus, if we want to maintain a strict distinction between kinds and types, there must be some ontological dependency relationship between kinds and types/universals.

Such a relationship has already been the focus from philosophers attempting to define a type simply as a kind (Wolterstorff, 1970) or as a model of all the tokens of the kind (Bromberger, 1992) (on all this, cf. Wetzel, 2006). We can stay content by saying here that "type" is very likely in the absolute extension of "kind" i.e. that, if a kind must be absolutely realized (on this requirement, cf. Sec. 3.3.1), then a type certainly does the job: a type may be, thus, roughly defined as a *specific* kind viz. as a genus plus a differentia with (at least) a token: e.g. if the *kind* human being is essentially an animal (specific genus) which is rational (specific differentia), then we can claim that the *type* human being is a rational animal with (at least) one specific instantiation[79, 80].

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[79] Two caveats on types: (i) we may raise the concern here that the distinction between kinds and types seems, after all, superfluous. Why not defining a kind as an essence having (at least) one instance? But, along this picture, what would be, then, types? Would they (absolutely intensionally) reduce to kinds? Following our metaphilosophical anti-reductionistic stance (cf. Ch. 2), it is a virtue for a theory of types that we are able to define them without (absolutely intensionally) reducing them.

(ii) Are there *super*/*sub*types? If it is arguable that there are, indeed, *disease* super/subtypes (just like there *may* be disease super/subkinds) (cf. Sec. 3.1.2), along the line there are no super/subkinds *tout court* for such a fundamental or general term that is "kind" (cf. Sec. 4.2.5), we can argue along the same line for the term "type".

[80] Two terminological notes (with caveats) on tokens and substrata: (i) types (or universals) and tokens (or particulars/individuals) are, respectively, *specific* abstract and concrete entities (on contemporary (token) substance or Cartesian dualism, cf. Lowe, 2006).

From our definition of a type, how are we to understand what a token is? In the specific *biological* domain, this question is at the basis of the so-called, when properly understood, *problem of biological individuality* (cf., in this extensive literature, Lidgard & Nyhart, 2017; Wilson & Barker, 2013).

If a token is a *particular* i.e. a *concrete* non-repeatable *entity* still constitutively definable, of course (without this making here a token a *specific* abstract entity like a type/universal), to leave room for e.g. tokens of negative types (e.g. absences, holes, etc.) to possibly exist (cf. Casati & Varzi, 2019), "concreteness" (or "spatio-temporal location") cannot be correctly (though still plausibly, of course) equated with "materiality" or "fulfillment/occupancy of a space-time region", for a token of a negative type (e.g. a token (black) hole) is intuitively something which does *not* fill in a space-time region. Can concreteness be (plausibly if not correctly) defined as what is ((in)directly) accessible, or what is *possibly* accessed (otherwise, this makes a token existentially dependent upon the presence of humans), by one's (five) *senses*?

Indeed, we can claim, roughly, that what is ((in)directly) accessible by one's (five) *senses* are *particulars*: nobody has ever seen e.g. the *type* red apple, a (type) suffering or a (type) danger, a (type) hole, etc., but only *token* red apples, the suffering of a particular person, the dangerousness of a particular dog, the hole in a particular doughnout, etc.

While tokens are *a posteriorily* (or empirically) accessed, types are *a priorily* known, where "*S* knows a priori that *p* iff *S* knows that *p* and *S*'s entitlement to believe that *p* is not grounded in experience [i.e., here, in a sensory access] [vs. intuitions]" (Kompa et al., 2009, p. 11).

In that sense, albeit a kind is not the same as a type/universal i.e. that a kind has no instance[81], that much does not make a kind, nevertheless, eternal, for a kind must be absolutely realized by a type (on, contrarily, kinds as specific universals, cf. Hommen, 2019).

We reject here the widespread Platonist view of *types* - as well as kinds/species, consequently - as eternal beings (wrongly associated with the general view of abstract objects (e.g. numbers, sets, etc.) as eternal), which underlies the rejection of the view of species as being essentially definable.

Thus, our fifth reply to objection (vi) is that objection (vi) misinterprets what *essentially* defining a species really implies. Globally speaking, our replies to the arguments on which objection (vi) is based show that they do not give credentials to a view of evolving kinds as absolutely extensionally defined, but that a weak enough *plausible* conception of essence – though not that weak to be implausible, or to make "essence" a catch-all term[82] - (*compatible* here with ours) is still tenable. As Momme von Sydow (2012, p. 1; italics original) brilliantly says, "[e]mphasising change even of evolutionary mechanisms does not preclude that these changes may refer to or take part in – perhaps even eternal – Platonic forms (*methexis*)" i.e. that

(ii) This constitutive definition of a token (cf. note (i)), more precisely a material one, is also tantamount to a certain understanding of Aristotle's hylomorphism.

Indeed, roughly following the basic idea that *one* token material object *is* a *compound* or a bundle of matter and (token) form, and a token material object *is* (nothing else than) a token substratum (or medium) numerically distinct from the bundle of its material and (token) formal constituents (understood in a certain minimal way), a *token* substance (or, a *sensed object* i.e. a(n) (im-/non-)material object, or a *thick* particular in Armstrong (1989)'s sense), at the more general level, is (just, or nothing but) *that* (as a *thin* (or *bare*) particular, a token (essential) *property bearer*/host, a token *subject* of a predicate, or a token *substratum*) (however understood here), in/of which token essential parts (e.g. as a specific token genus plus a token differentia) inhere (under a certain reading)/*are* (for a view sympathetic to those ideas, cf. Keinänen & Tahko, 2019; on bare particularism, in this extensive literature, Connolly, 2015; Garcia, 2014; on the exchangeability between *bare* particulars and *thin* ones, Brower, 2010).

Bare (or thin) *particulars* or, at the level of abstract objects, *substrata* – often accused of being mysterious, enigmatic or extravagant things (Denkel, 1996) - are here to be simply *deflationarily* understood (Chisholm, 1969) i.e. as mere (token) (essential) property bearers/hosts, (token) subjects of (intensional) predication or (token) definienda (however understood) irreducible to (token) (sets/*bundles* of) (essential) properties.

In that sense, a substratum is not, strictly speaking, defined along an essentialist line (on the famous related problem known as the *antinomy of bare particulars*, cf. Armstrong, 1989).

It is clear that a substratum as a mere (essential) property bearer/host (however understood) has *no independent existence* from the (essential) properties it bears/hosts; likewise, the essential parts of a whole are *necessarily* essential parts of a whole (along the relative extensionality requirement) (cf. Sec. 3.3.1).

[81] Terminological note on Platonic objects: under another plausible interpretation of Platonic objects i.e. as (abstract) *uninstantiated* beings, kinds could be said here Platonic objects, albeit our use of the notion of Platonic object (especially the one used in the philosophy of mathematics), throughout this PhD dissertation, is the one of a mere (abstract) *eternal* being.

[82] Caveat on anti- and weak essentialism: the cleavage between *anti*-essentialists and *weak* essentialists is peculiarly difficult to maintain in those circumstances, one may object. However, it remains true that, more specified, the cleavage makes completely sense: e.g. anti-essentialism with a certain understanding *x* of an essence can still be different from essentialism with a *weak* understanding of an essence. Nevertheless, without making objection (vi) a straw man, it is still true that, as found in the literature, objection (vi) remains under-specified – thus, our point here (cf. also objection (v) of Sec. 3.2.5 for the same problem).

there is no incompatibility between a certain essentialism and an appropriate notion of change (cf. Exc. 3.7 for my take on evolution).

Of course, essences of (or, true analyses – i.e. given within a genus-species hierarchy as understood here (cf. Box 3.1; 3.1.1-3.1.2) - of) (specific) kinds/species may be, or are de facto, hard to find out (or, to give) - especially the ones of (specific) coarse-grained kinds like the essence of a man or a woman -, but this is no reason for not *trying* to find them out (or, to give a true analysis of them), or for thinking that there are none (or, no true analysis of (specific) kinds), or that we shall not find them out sometime (or, be able to give such analyzes one day or another), along a pessimistic meta-induction about concept analyzes (cf. Sec. 3.2.5).

In response to the argument of the pessimistic meta-induction (about species/kinds), we can answer that this argument merely shows that finding out the essence of a (specific) kind, as understood here (cf. Ch. 2-3), is not an *easy* task – indeed, have we ever found out any (correct) essence of anything? -, or that a true analysis of a (specific) kind as a (specific) genus plus a differentia, as taken here cannot be *easily* given (cf. Box 3.1; 3.1.1-3.1.2) – but not at all that it is a *useless* or *worthless* task to pursue, on grounds that our concept analyses shall be sooner or later falsified (as Machery (2009; cf. also 2017; Strevens, 2019) suggests for what he calls, somewhat pejoratively, "*folk concepts*" like GOODNESS, KNOWLEDGE, JUSTICE, LIFE, or, very likely, DISEASE itself, or maybe even CANCER, etc.; on philosophical progress, Exc. 3.8)[83].

[83] Caveat on conceptual change: note that, if I am right that species/kinds, generally speaking, do *not* change i.e. here, following objection (vi), that they do *not* have (essentially) variants (through time), then *concepts* (as specific kinds themselves) as well as our (plausible) *analyzes* of them, thus, do not change (on the huge topic of conceptual change (in the sciences), cf. e.g. Amundson, 2005; Brigandt, 2006).

Indeed, even (slight or huge) revisions of (our analyzes of) concepts can be arguably said just *novel* or *innovative* (analyzes of) concepts – however slightly or hugely novel they can be, it remains true that they (fully) exist at time *t* as (numerically different) entities on their own - albeit not necessarily with the ontological status of specific *kinds*/species themselves, but of e.g. *parts* of a kind/species (on the related problem of the pessimistic meta-induction (about kinds), cf. Sec. 3.2.5).

Note that, based on ME (and the WCP), Chisholm (1969) has brought forward the same basic idea with respect to the ship of Theseus puzzle (in its contemporary version):

> Original1 consists of a collection of planks organized in a ship-like manner. […] Original1 gives way to a different ship we may call Original2, constituted from a slightly different collection of planks. In its turn, Original2 gives way to Original3, and so on, all the way to Original, which is constituted from planks entirely different from those constituting Original1. (Gallois, 2017, p. 91)

Maybe Chisholm's point i.e. here the multiplication of the ships of Theseus applies to philosophical problems as well: e.g. the main difference between Descartes's solution to the mind-body problem and contemporary ones is that the former has been *progressively* replaced by the latter ones, which are getting closer to the truth (*pace* Stoljar, 2017 here, for whom problems of the past just are not the same as the current ones, by (slight) contrast with the idea defended here that either the problems are, indeed, different, or only solutions or *analyzes* of these problems are, actually, different) (cf. Exc. 3.8).

Of course, all this does not imply that (analyzes of) concepts are to be necessarily conceived of as eternal Platonic forms: e.g. the type COMPUTER appeared only when there was the first instance of it, or of the type *computer* (on my correspondence principle between concepts, words and (specific) entities, cf. Sec. 2.2.1).

3.5 Excursus: A Permissive Neo-Aristotelian Ontology

Schaffer (2009; cf. also Eklund, 2006) usefully distinguishes between (i) a minimal-style, austere, sparse or parsimonious ontology (basically in Quinean spirit), whose main task is to sort out what there is through ontological commitments from our *best* current (*scientific*) theories (as sets of declarative sentences here) formalized/translated into first-order predicate logic, and (ii) a maximal (or abundant)-style or permissive (basically neo-Aristotelian) ontology, which is mostly interested in fundamentality relationships (e.g. generality, grounding, essence, etc.) holding between *many* – at least, more than the Quinean would allow - different specific entities assumed to exist.

As presented here, a maximal ontology is, thus, such that its ontological commitment criterion just is, actually, (even) more relaxed (or *weaker*) than the one of a minimal Quinean ontology.

Our reply to objection (vi) (cf. Sec. 3.2.6) as well as our metaphilosophy (cf. Box 3.1; 3.1.1; 3.1.2) and the other necessitation relationships investigated (cf. Exc. 3.2; 3.9) make us follow ontology (ii) in a specific way, for we assume the existence of many entities, as long as these are *essentially definable* in a certain way (i.e. within (our understanding of) a genus-species hierarchy) and fulfill other metaphysical constraints (cf. Sec. 3.3.1) (e.g. meta-organisms like perhaps microbial communities, biofilms, ant colonies, or another specific ecological entity like (maybe) a holobiont, an ecological niche, etc.), or they are so under any plausible reading of "essence" (e.g. cf. Sec. 3.3 on genus and differentia).

(Note, however, that some philosophers (e.g. Korman, 2015; cf. also Sider, 2011; Thomasson, 2015) take a permissive or maximal (vs. (said) *conservative*) ontology to be, actually, a(n) (extremely) *deflationary* (or neo-Carnapian (1947)) position, where existence questions are *trivial* – thus, where there is no restriction on what exists. Within the (meta)ontological dispute around ontological realism vs. anti-realism, Chalmers (2009) usefully distinguishes further between *lightweight (anti-)realism* and *heavyweight (anti-)realism*, where ontology (i) or (ii) would fall under heavyweight realism, while this latter (deflationary) interpretation of a maximal ontology would fall under lightweight realism.)

Four objections can be easily raised here: (i) we may fear that with ontology (ii) we give rise to an uncontrolled proliferation of specific entities (in our case, more precisely, specific *kinds/species* or other entities like *genera*) to exist (or goes against Ockham's razor (at least, in an ontological version) for such entities) – where kinds have, according to us, necessarily an absolute/relative extension (cf. Sec. 3.3.1). Thus, with objection (i), the problem of finding absolute realizers becomes pressing.

However, first, that much does not mean that the definition of a specific species should match a scientific description of an absolute realizer of it, but only that there is necessarily a certain *plausible* (i.e. a priorily argued for or non-absurd) description of an absolute realizer of the specific species, which may be followed or not by some scientists.

Second, the proliferation of specific *kinds* as well as of other entities like specific *genera* (cf. Sec. 3.3.1) is still controlled (however shallowly especially for the latter entities), to the extent that sets are relatively/absolutely extensionally defined.

Thus, a threat of *over*-medicalization (or -pathologization) is not to be feared – as long as the diseases in question are correctly defined and not *over*-defined, of course (cf. Walker & Rogers, 2017) -, for our highly permissive (but *not* profligate or exorbitant) or shallowly constrained neo-Aristotelian ontology does not imply, indeed, an *all-or-nothing* or *everything-goes* situation, or is not a slippery slope towards granting the existence of *any*thing, or such an ontology does not preclude, of course, to raise objections against a certain conception of e.g. species/kinds (cf. Sec. 3.2), etc., by using parts of the ontology itself (e.g. reducing kinds/species to sets, etc.).

Some will further object, however, that (ii) a controlled proliferation of specific diseases is still highly *arbitrary*, for "[i]t is not hard to coin new maladies for the purposes of justifying the use of enhancements interventions" (Juengst & Moseley, 2019) i.e. that such diseases would be human inventions.

As shown in the quote, objection (ii) is raised especially in the debate about (*human*) *enhancement*, but it can be extended to the general argument that a proliferation of specific diseases is, actually, not objective or just does not take place, but serves e.g. a commercial, discriminatory or any other pragmatic purpose (e.g. to more easily claim that someone is diseased) (on so-called "disease mongering", cf. Stegenga, 2018a; also Horwitz, 2002), for there are no such (*natural*) kinds as specific essences with their corresponding diseases (or biological malfunctions).

Nevertheless, objection (ii) commits the mistake of relying on scientific anti-*realism* (strictly taken) or anti-*objectivism*, against which, thus, common arguments in favor of scientific realism or objectivism (or our own metaphilosophical framework) can be put forward (for a sample of those arguments, cf. Ch. 2; Chakravartty, 2017; Esfeld, 2009; on pessimistic meta-induction about concept analyzes, Sec. 3.2.5).

With objection (iii) (closely related to objection (i)), philosophers generally argue that an expanded definition of disease leading to a proliferation of specific diseases (directly) contributes to *overdiagnosis* (on that, cf. Biddle, 2016; Hoffman & Cooper, 2012), and over-diagnosis surely is a bad thing.

However, with objection (iii), albeit we agree that *over*-diagnosis is a bad thing, unless we hold a mistaken conception of what a definition of disease is, we fail to see, again, in what sense a definition (of disease), as being a mere description of what there (here objectively) *exists* (cf. Ch. 2), can lead *on its own* to this specific epistemic issue which is overdiagnosis (on overdiagnosis as disease misclassification (and maldetection), but where overdiagnosis is mistakenly related to harm, cf. Rogers & Mintzker, 2016).

Finally, we can fear (iv) that a subscription to a permissive neo-Aristotelian ontology is here an *ad hoc* (unsupported) hypothesis or maneuver for saving a certain (neo-Aristotelian) meta-philosophy or framework (cf. Ch. 2; 3.1.1).

However, objection (iv) misses the mark, for a permissive neo-Aristotelian ontology is, actually, more *general* than the (neo-Aristotelian) meta-philosophy or framework followed throughout this PhD dissertation - which can be said, thus, a *specific* permissive neo-Aristotelian ontology.

The neo-Aristotelian ontology we advocate here has as a consequence that it allows us to easily proceed to a *division of labor* between the proper province of philosophy and of the other sciences: as L. A. Paul (2012, p. 6; emphasis original; cf. also Andersen & Becker Arenhart, 2016; Morganti & Tahko, 2017 for discussion) nicely puts it, "[…] scientific theorizing usually *uncritically assumes* the very organizing principles and deep general truths that metaphysics is concerned to […] develop and understand". C. D. Broad (1924, p. 82; italics in the text and mine) had already (a bit more neutrally) (cf. Sec. 3.3.1 for reservations) expressed that, "[…] whilst all the special sciences *use* these concepts, none of them is *about* these concepts as such. I regard *Critical Philosophy* as the *science* which has this for its most fundamental task", as well as Amie Thomasson (2007, p. 137; emphasis original) "that *using* straightforward empirical descriptive vocabulary "already presupposes grasp of the kinds of properties and relations made explicit by modal vocabulary" (Brandom 2008, 97)".

This way of seeing the division of labor i.e. as a demarcation with respect to different *subject matters* (rather than methods) should not be conflated with the widespread way, especially in the debate about the more fine-grained division of labor between analytic metaphysics and philosophy of physics (French & McKenzie, 2012; *pace* Ladyman & Ross, 2007), of seeing the division of labor as rooted in the heuristic value provided by e.g. philosophy (for the (mistaken) related idea that the division is rooted in the instrument, the tools and the researcher, cf. e.g. Osbeck & Nersessian, 2015).

As a (*weak*) *liberal naturalist* (cf. Exc. 2.1) (or, with a minimal practice-oriented or descriptive philosophy (of medicine), I can claim, thanks to a maximal ontology, that non-philosophical sciences and philosophy may share the same methodology (broadly understood), but different subject matters: e.g. what has philosophy to do with analyzing *correctly* what e.g. a *gene(tic) (process)* (*pace* Stotz et al., 2004) or what *inclusive fitness* is – albeit philosophy can still use, of course, these analyzes or *plausible* versions of them? Or, what have non-philosophical sciences to do with analyzing what *disease* or *life* is?, where e.g. non-philosophical scientists are interested in *specific* disease(s) (kinds) like cancer (kind) or in the *constituents* of a certain definition of disease (on the different ways to reconcile the famous dichotomy of the *manifest* vs. the *scientific image*, cf. Christias, 2019; Cumpa, 2018; Sellars, 1963).

Although we argue for the *irreducibility* of higher-level kinds, they are not, however, ((in)directly) *autonomous* from (more and more) low(er)-level kinds, to the contrary of what has been defended by *strong* liberal naturalists like Hornsby (1997) or McDowell (1994); "(weak (and strong) liberal) naturalism" has as an absolute realizer what we can call, more precisely, "biological or physical naturalism" or "*physicalism*" (albeit most often misleadingly referred to as "naturalism" *simpliciter*) i.e. the thesis that (only) the most fundamental entities (plausibly speaking) discovered or postulated (in an

objectivist/subjectivist sense) by the biological or *physical* sciences (through a certain methodology viz. rational inquiry) exist.

This idea is also pretty close to Chris Peacocke (1992)'s seminal book on *concepts* and their relationship to psychology (reductively understood here), where "[p]hilosophers should specify a priori the possession conditions of specific concepts, such as BELIEF, SQUARE, or RED. Psychologists […] then describe the nature of the states and processes required for someone to meet the possession conditions […] established by philosophers" (Machery, 2009, p. 38; cf. also Engel, 1996) – albeit we also defend that, especially with the requirement of relative extensionality, sciences do have constraints from philosophy (cf. Sec. 3.3.1).

Indeed, if scientists mean to produce *correct* analyses (of kinds/species), then, with respect to the minimal directness requirement of relative extensionality, these shall be (indirectly) *coherent* with our own meta-philosophy (cf. Ch. 2); if not, either there is a problem somewhere, or scientists just have not produced the correct analyses yet (against Kovaka, 2015).

Note that that much also applies, in turn, of course, to the analysis of different *philosophical* concepts themselves (e.g. DISEASE and DISEASE KIND) (cf. Ch. 4), which are to be minimally directly absolutely realized. Thus, with such (mutual) constraints, there is not much of a danger of imposing (as a *norm*) to the sciences an ill-suited philosophical approach (*pace* Kaiser, 2015).

The division of labor cannot be drawn, thus, between the *normative* discourse and the non-normative (or natural) one. For, we can very well argue that the *Naturwissenschaften* (restrictedly taken) also study normative entities (strictly taken): e.g. lung cancer is studied by the *Naturwissenschaften*, but, as being a certain disease (which is a value), it still contains a normative element (on the different senses of "nature", cf. Exc. 2.6), or claiming that the cells in the lung's tissue are cancerous is a value judgment (properly speaking).

Furthermore, our weak liberal naturalism is to be distinguished from the related (ambiguous) position that philosophy would be about, not merely the different concepts *used* by scientists, but also about making explicit the tacit assumptions or the prior a priori understanding from scientists about those concepts – as if science were also investigating the nature of those concepts (Glock, 2017; for oscillations between our own position and this one, cf. Lowe, 2008; 2011).

Indeed, such a position amounts to saying that philosophers and scientists would study, actually, the very same concepts. That science also (a priorily) studies concepts or types is almost trivial, but, with this position, philosophy runs the risk of being a mere chapter of science.

3.6 Excursus: On Precision and Personalized Medicine

Two general remarks are to be made about precision and personalized medicine (on the ambivalence of the term "precision medicine", cf. Darrason, 2017b; Giroux, 2017; on precision medicine, generally speaking, Green & Vogt, 2016; Wiesing, 2018).

First, to differentiate *precision* medicine from *personalized* medicine, the US National Research Council explains, against contemporary orthodoxy (cf. Lister Hill National Center for Biomedical Communications, 2020), that

> […] "precision medicine" refers to the tailoring of medical treatment to the individual characteristics of each patient. It does not literally mean the creation of drugs or medical devices that are unique to a patient, but rather the ability to classify individuals into subpopulations that differ in their susceptibility to a particular disease, in the biology and/or prognosis of those diseases they may develop, or in their response to a specific treatment. Preventive or therapeutic interventions can then be concentrated on those who will benefit, sparing expense and side effects for those who will not. Although the term "personalized medicine" is also used to convey this meaning, that term is sometimes misinterpreted as implying that unique treatments can be designed for each individual. (Committee on a Framework for Developing a New Taxonomy of Disease, 2011, p. 125)

In other words, the US National Research Council prefers using, in this context, "precision medicine", for there exists a wider sense of "personalized medicine" – which does not correspond to what the Council intends to mean in the 2011 report -, where "personalized medicine" refers for them to the idea that each patient could have his own therapeutic treatment adapted to him alone.

Strictly speaking, personalized medicine applies only in the context where what is treated is a *person*, while this is not necessarily the case for precision medicine.

As such, "precision medicine" refers, actually, not to the idea that scientists ought to create drugs necessarily *particular* to a token patient (for there would be only particular diseases) – an ideal which seems to me hardly *practically* (but still *theoretically*, if one subscribes to e.g. tropes) possible to reach, for, if a medical drug is efficacious only to a single individual, then e.g. no clinical population-based Randomized Controlled Trial (RCT)('s result) would be ever useful -, but that they ought to create more and more specialized (or *unique*) drugs, with the hope to cure in a more and more targeted way the (more and more specifically) diseased persons (or other living beings).

If I am right that precision medicine just is the trend which explodes, thus, the number of specific diseases into many more, then this trend did not wait until the recent creation of the label "precision medicine" to already historically happen in medicine (cf. Walker et al., 2019 for a related point).

Indeed, it may be plausibly argued that the whole history of medicine has (discontinuously) followed, actually, such a trend; we may also further argue here that, actually, as understood, precision medicine is in line with a maximal neo-Aristotelian ontology (cf. Exc. 3.5).

Second, *precision* medicine is also sometimes mistakenly associated with the program of *genetic* (or *molecular*) *reductionism* using personalized gene therapy based on e.g. whole genome sequencing or genome-wide association study (cf. e.g. Green et al., 2019). This mistake can be easily explained by the widely held general idea that the more fine-grained (or *specific* or *lower-level*) kind you can get, the more *precise* definition of the specific kind you shall get.
However, "the more *specific*" does not mean "the more *precise*", for a *correct* definition of a *coarse*-grained kind shall be necessarily *precise*. It is, thus, important *not* to take precision medicine as a *reductionist* approach to disease, according to which the proliferation of specific diseases is only about *genetic* diseases.

3.7 Excursus: What is Evolution, or an Evolutionary Explanation?

Does the rejection of a definition of species as evolving kinds (as understood along objection (vi) in Sec. 3.2.6) imply the rejection of evolutionary explanations?
No, it does *not* imply the rejection of a *proper* (*reductive*) understanding of evolutionary (or historical) explanations, but only a rejection of a certain understanding of evolutionary explanations, on which objection (vi) is based.
Indeed, we can very well take evolutionary explanations just as specific *constitutive* explanations viz. *process* explanations, since our neo-Aristotelian framework is compatible with a process ontology. Thence, talk about *evolutionary processes* is redundant, if "evolution" just is to be equated with "process" like in talk about e.g. an evolving situation (on the *generalized* theory of evolution, cf. Feldbacher-Escamilla & Baraghith, 2020; Schurz, 2001).
Formally, the argument for the bold conclusion that evolutionary (or historical) explanations are mere process explanations runs as follows:

(1) the evolution *of* e.g. a human being is not about how a human being changes over time, for a human being is arguably a certain *process* – whose nature is to *be* a substantial *change* (of two states);
(2) indeed, a process is *substantial change* from a *state non-x* to *x* (or from *x* to *non-x*);
(3) an evolution just is a *process*;
∴ therefore, an evolutionary *explanation* just is a *process* (or *mechanistic*) *explanation* interested in what makes a process a process.

Thus, the question of what e.g. the *evolution of* a human being or a disease is just is the question of what a human being or a disease (as *being* a humane or disease *process*, evolution or development) is, or e.g. "How does/did a heart process evolve (or *develop*)?" (i.e. "What is the heart process, or How does a heart process work?") "By, amongst others, pumping blood".

> "[e]volution may be defined as any net directional *change* or any cumulative change in the characteristics of organisms or populations over many generations—in other words, descent with modification. […] It explicitly includes the origin as well as the spread of alleles, variants, trait values, or character states" (Endler, 1986, p. 5; my emphasis);
>
> "[biological evolution] is *change* in the properties of groups of organisms over the course of generations;[…] it embraces everything from slight changes in the proportions of different forms of a gene within a population to the alterations that led from the earliest organism to dinosaurs, bees, oaks, and humans". (Futuyma, 2005, p. 2; my emphasis)

Those classical definitions of *biological* evolution are typically theory-laden (i.e. based on the idea that biological evolution e.g. occurs only at a certain level ((groups of) organisms, populations, etc.), or involves heritable traits/genetic processes); classical definitions of biological evolution are definitions of only, amidst others, *one specific* biological evolution meant to capture only something *that* specific or specified viz. a definition based on natural selection and reproduction (narrowly taken). What is commonly opposed to such classical definitions is also (most of the time), actually, another definition of a specific biological evolution.

The consequences of the above definition of evolution (and evolutionary explanations) are the following: firstly, in opposition to what is commonly accepted in the field of *evolutionary* developmental biology (or evo-devo) (Baedke & Gilbert, 2020; on (said) historical and *a*historical functions, Wakefield, 1992; Walsh, 1996), we reject the dichotomy between evolution and development, where the notion of development is unfairly restricted in evo-devo to specific processes (mistakenly called "ontogenies") viz. the ones of, or underlying, highly specific biological species (or organisms) (e.g. a process starting from an embryonic state), as it is shown by the following definition (for a survey of definitions of development in developmental biology (*tout court*), cf. Pradeu et al., 2016):

> [evolutionary] developmental biology aims to explain [development as the] *organic form* and its origin in the embryo (Amundson, 1994, p. 563; emphasis original; cf. also Craig, 2015).

Secondly, our reductive comprehension of evolution/*development* (or evolutionary explanations) implies the *rejection* of the famous distinction between *ontogeny* (or ontogenesis) and *phylogeny* (or phylogenesis) (Gould, 1977), for, if an evolutionary (genealogical or historical) explanation is a mere *process* explanation (or evolution/development is a mere process), then "*onto*geny" and "*phylo*geny", properly taken, also refer (etymologically) both to a mere process, a development or an evolution of e.g. an organism, a biological species/kind, etc.

Thirdly, however, this reductive understanding of evolutionary (or historical) explanations (*tout court* i.e. not theory-loaded here) does not imply a rejection of the proximate/ultimate distinction; albeit the

distinction between proximate and ultimate explanations is normally used in the context of *causal biological* explanations (Sober, 1984), it can also very well be used in the wider context of *causal*, *constitutive*, *teleological*, *evolutionary*, etc., explanations (however we understand all of these specific explanations).

Proximate and ultimate explanations are orthogonal to causal, constitutive or teleological explanations: e.g. a proximate/ultimate *causal* explanation is obviously an explanation citing proximate (or *direct*)/ultimate (or *indirect*) causes; a proximate/ultimate *constitutive* explanation is an explanation citing proximate/ultimate constituents; a proximate/ultimate *teleological* explanation is an explanation citing proximate/ultimate purposes (cf. Foot, 2001; Thompson, 2008).

Thus, the common association between *ultimate* and *evolutionary* explanations cannot be maintained, as usual in the literature (cf. e.g. Nesse, 2013), since ultimate explanations have nothing to do specifically with evolutionary explanations (however plausibly or correctly understood): e.g. a certain evolutionary explanation can very well be *proximate*, in the sense that e.g. swiftness of a deer may be evolutionarily explained *not* through the natural selection of a specific genetic process on a deer (ultimate evolutionary explanation by natural selection), but through the natural selection of what makes more directly a deer swift (e.g. thin legs) (i.e. a proximate evolutionary explanation by natural selection).

Fourthly, the notion of *adaptation* is to be understood, strictly speaking, *deflationarily* i.e. as the mere fulfillment of the essence of a *process* (understood as substantial *change* of a state): e.g. a(n) (perfectly) adaptive eye or human being just is an eye or human being (as a specific (long-lasting) process) whose essence is being (fully) realized.

The main difference between adaptation and evolution is, thus, that, if evolution is truly a (negative or positive) process (e.g. (a part of) an organism), then adaptation just is the essence of a (negative or positive) process i.e. substantial change of a state: e.g. a bacterium resistant to antibiotics adapts to its environment (on the relationship between adaptation and (biological) *functions*, cf. Huxley, 1974).

Of course, the notion of adaptation can still be *plausibly* understood – and used, indeed, throughout this PhD thesis - in other various ways: e.g. as being only about *positive* genetic processes/traits.

Moreover, if the notion of adaptation is understood along this line, then there exists no such thing as, *properly* understood, *exaptation* i.e. a shift in the adaptiveness of a genetic process/trait, for a genetic process/trait's essence *cannot* change.

However, the notion of exaptation can still be *plausibly* understood (cf. Gould & Vrba, 1973; Lloyd & Gould, 2017) along different lines like the one that a genetic process/trait is *multiply* relatively realized (at different times): e.g. bird feathers with respect to heat regulation and/or bird flight.

3.8 Excursus: On Philosophical Progress

In the general context of a defense of *scientific* realism (or rather, objectivism) - also applicable, actually, to the entities investigated by philosophy (or to philosophical theories), to the extent that philosophy is to be correctly treated as a certain science in its own right (cf. Exc. 2.1) -, the conviction that finding out truth is still a worthy task has been concretized along the line that we are to acknowledge the existence, in a historical or diachronic setting, of, at the *epistemic* level here, (probabilistic) degrees of belief toward a certain scientific theory (at time *t*), or that the notion of *quasi-truth* (approaching truth, or tracking truth) makes sense (on that, cf. da Costa & French, 2003; Psillos, 1999; on *progress* in philosophy, generally, Stoljar, 2017).

With respect to the pessimistic meta-induction about (specific) kinds, we are providing, hopefully, the reader, in this PhD dissertation, with enough evidence for fully embracing our analysis of DISEASE, or, for thinking of our theory of disease, if not as correct or true *simpliciter* (because of having a non-irrefutable theory of disease), at least as *tracking* truth (taken in a minimal or *deflationary* sense) (Engel, 2002; Stoljar & Damnjanovic, 2010; cf. also Künne, 2003), or as the *most* correct/true contribution *possible* (at time *t*) for achieving *correctness*/*truth simpliciter* (as an *ideal*) – or, metaphorically, as a not fully *healthy* contribution – and not as being a theory, if not made just false in the future, made any way *less* (*and less*) probable in the future.

The same also applies to other more mundane concepts/entities like HUMAN BEING/human being or HEART/heart, for the (*completely*) *perfect* (or *ideal*) human being or heart is yet to be (fully) discovered (or just to (fully) appear) as a specific kind here or, more specifically, as a *type* with its token(s).

That is why, when we are talking about e.g. specific ideal values to be reached (realized and instantiated) in specific cases, we are, of course, talking about, more precisely, either (i) ideal values *as known* at time *t* i.e. contemporarily, which are realized and instantiated (in the *actual* world/universe): e.g. a 20/10 human visual acuity on the Snellen chart;

or (ii) *theoretical* ideal values (still possibly achievable, realizable and instantiable) (on the notion of a *regulative ideal* in science, cf. Schaffner, 1993); ways (i) and (ii) are easily contextually recognized throughout the PhD dissertation.

3.2.7 Objection (vii): The Genus-Species Hierarchy as a Linnaean Hierarchy

As a last objection against our understanding of the genus-species hierarchy (cf. Box 3.1; 3.1.1-3.1.2) – closely related to objections (v) and (vi) (cf. Sec. 3.2.5-3.2.6) -, we could argue that a genus-species hierarchy is to be understood along a *Linnaean hierarchy*.

If the notion of a Linnaean hierarchy were *weakly* understood as a mere set of rules for classifying hierarchically organisms with the use of a *binomial nomenclature*, then we would fail to see in what sense objection (vii) could constitute a genuine and novel objection against our own account of the genus-species hierarchy (restricted to the biological domain), for objection (vii) would be reducible to either our own understanding of the genus-species hierarchy (as restricted to the biological domain), or other objections like objection (iii) (cf. Sec. 3.2.3) (e.g. with *Homo sapiens*) or even objection (vi) (cf. Sec. 3.2.6) (also restricted, of course, to the biological domain).

However, the notion of a Linnaean hierarchy (made applicable here to the general notion of species/kind) can be understood more *strongly* as a very specific – irreducible - way to tell what a species/kind is by making it, so to say, more Linnaean in spirit (or closer to Linnaeus's original hierarchy system) than *neo*-Linnaean, but without making objection (vii), of course, gratuitous or just that easy to address.

Indeed, along objection (vii), a species/kind would be just any entity capable of falling within the taxonomic ranks established by Linnaeus (1735), but extended to non-biological kinds/species. In that sense, e.g. a biological species/kind would *not* be – *contra* Linnaeus here -, a certain genus with a differentia, on pain of a collapse with our own understanding of the genus-species hierarchy (cf. Box 3.1; 3.1.1-3.1.2) or with other objections (cf. especially Sec. 3.2.3), but an entity classifiable within a hierarchy of five ranks (for each kingdom) viz. classes, orders, genera, species, and varieties (cf. Ereshefsky, 2001); the species *Rhododendron sikkimense* would be a specific kind, thus, because it is classifiable within, or belongs to, a hierarchy of five ranks.

However, as long as one does not specify how to understand further the five ranks in question, then anything could trivially belong to such a hierarchy; thus, anything would be a kind/species. Linnaeus's proposal to understand these ranks, where

> […] [t]he method of logical division is […] used […] to distinguish genera in an order, and orders in a class [, and where] [e]ach order and each class has its own definition, and the subordinate taxa within them are marked by their distinctive differentiae (Ereshefsky, 2001, p. 202),

does not help solve the problem, unfortunately, for, in other words, the above quote would make objection (vii) reducible to our own conception of a genus-species hierarchy (cf. Box 3.1; 3.1.1-3.1.2) or to another objection viz. objection (iii) of Sec. 3.2.3.

Therefore, with objection (vii), there is a high risk of making this very objection either trivial or non-genuine.

3.3 On Genus and Differentia

Strictly speaking, objections (i)-(vii), as raised (cf. Sec. 3.2.1-3.2.7) here, are not meant to pronounce except on a certain analysis of the genus-species relationship (cf. Box 3.1; 3.1.1-3.1.2); we have used in this context the concept of disease and disease kinds as mere (absolute) *illustrations* (or realizers).

However, cannot we use our own analysis of the genus-species relationship or, more precisely, our analysis of a genus (as essentially a constitutive part of a species) as itself an objection against our constituent ontology (cf. Ch. 2)? Moreover, is it not also the case of our definition of a differentia (cf. Box 3.1.2)?

While Sec. 3.2 defends our understanding (in its generality) of the genus-species relationship, Sec. 3.3 defends, more specifically, our definitions of, respectively, a genus (in Sec. 3.3.1) and a differentia (in Sec. 3.3.2).

3.3.1 A Genus as a Constitutive Part of a Species

An important upshot of our own analysis of the genus-species relationship is that a genus (or *γένος*) is essentially (here along a certain understanding of RME) a constitutive part of a species[84].

Indeed, our definition of a genus (cf. Box 3.1.1) follows an extensionalist line; more precisely, along our definition of a genus, a genus is a specific *set* (in its relative sense) (cf. Exc. 3.9) i.e. that the intension (or rather, the *definition* or even the essence), by using its plausible sense, of "genus" is its relative extension. If a genus is essentially a set (in its relative sense), then there consequently and necessarily exists a species/kind of which a genus is a constitutive part: e.g.

[84] Terminological note on genera: if a genus is essentially (plausibly taken) a constitutive part of a species, then we could argue (typically along a constitutive ontology) that a genus just is, actually, an *essential property* or *intensional predicate*, for essential properties/intensional predicates may be defined as what is constitutive of a species (as a substratum), or what is intensionally predicated of a subject.

the set/genus {animal} includes (or *subsumes*) here, amongst others, or at least, a human being, a rational animal (taken as a specific kind), etc.

In the Aristotelian tradition, especially along its famous Thomistic interpretation (cf. also Ainsworth, 2020), the highly controversial notion of *prime matter* i.e., roughly, a (token) building block can certainly play the role as a specific instantiation of a genus (considered, plausibly, as a type) (e.g. token genes) - exactly like other similar notions such as Plato (*Ti*. 49-52)'s *receptacle* (on the complex relationship between the Aristotelian tradition and atomism (or *minima naturalia*), cf. Berryman, 2016).

A major advantage with our definition of genus (cf. Box 3.1.1) is that it allows us to get a well-founded mereology (strictly taken) (Cotnoir, 2013; & Bacon, 2012), where *Mereological Well-Foundedness* (MWF) is the idea that

(MWF) necessarily, there is no infinite regress or descent of ontological priority i.e. that there exists something – an atom -, which is such that it has itself no (constitutive) parts i.e. a genus.

However, some have argued that MWF is to be rejected (Morganti, 2009 Raven, 2015), on grounds that, instead, *Mereological Infinitism* (MI) is *possible* i.e. that

(MI) there is a (said) possible (atomless) *gunky* world, where everything has a(n intensional or extensional) proper part, or nothing is an atom (however understood).

Although we can argue that there can exist *some* things like certain sets (e.g. qua objects) which may be considered as gunks (understood in a certain way) (cf. Exc. 3.9 on qua objects), not everything can be a gunk, at least in a maximally populated world, along our own definition of e.g., more specifically, *constitutive* or intensional (proper) parthood (cf. Ch. 2); we do reject the possibility of MI, for any non-trivial entity i.e. a species/kind, or an entity with, strictly taken, an essence, a definitional structure or an intension (like a disease, for a disease is obviously *not* a trivial entity) (vs. a set like a genus or a qua object) (cf. Ch. 2), must intuitively inherit somehow its reality from solid foundations (Schaffer, 2016; cf. also Cameron, 2008; *pace* Trogdon, 2018).

If we say that disease is ontologically and definitionally prior to disease kinds (cf. Sec. 3.1.1), then disease just seems to be the genus of the species disease kind. If so, then a disease seems

to be defined (along a certain understanding of RME), like for the case of a genus, as a constitutive part of a certain species.

If we agree that this fundamental term that is "genus" is outside our metaphilosophy (cf. Ch. 2), then it seems that the same point should be also granted to a term like "disease". As such, this objection goes against our definitional framework that to (*correctly*) define some entity *x* (except e.g. genus) is to find out (the set of) its constitutive parts (cf. Exc. 3.10 against holism about kinds)[85].

Moreover, one can add here that we still have to explain away the recalcitrant intuition that there is no (specific) genus without a (specific) species, which is, of course, also applicable to the idea that there is no disease without e.g. (or, at least) a disease kind – by contradistinction with the idea that there is no disease kind without *essentially* a disease (cf. Box 4.2) -, or no color without at least one specific shade of color (cf. Sec. 3.2.3).

We can explain it away in the following two-fold way: that (i) a term like "color" or "disease" must have a *relative* extension[86] (e.g. "red" for "color", "disease kind" for "disease" or even "species" for "genus") (cf. Sec. 3.1.2); and that (ii) a term like "color" or "disease" must have an *absolute* extension (e.g. perhaps "warm color" or even "red" for "color" – we can eventually leave open the idea that red is either a color (i.e. the genus) plus a differentia (as we have used this example until now by following the mainstream view) or that it is rather in the absolute extension of "color" (indeed, it is prima facie odd to claim that red is something *more* than a color) - or "genetic disease" for "disease") i.e. that there is no disease without a *specific* disease (which *absolutely* applies the correct intension (i.e. within (our understanding of) a genus-species hierarchy) of "disease")[87].

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[85] Why not saying, to the contrary, that our definition of kinds/species (cf. Box 3.1) is apart from a certain metaphilosophy, while our definition of e.g. a genus (cf. Box 3.1.1) would be the norm (for definitions) or a *correct* definition?

We can answer that this is because, were it the case, then it would contradict our intuitions about what a (correct) definition is, or our definition of genus would be based on a wrong metaphilosophy (cf. Ch. 2-3 on that).

[86] The requirement of relative (and absolute) extensionality (for kinds) is a minimal *directness* requirement. This minimal directness requirement for relative (and absolute) extensionality obviously emanates from the definition of relative (and absolute) extensionality itself: (at least) one realizer of truly *x* – and not *y* - is a direct one: e.g. if "disease kind" has for absolute extension "bacterial disease", then we would like to say that specific bacterial diseases are only *indirectly* (in the absolute extension of) disease kinds, for they are *directly* in the absolute extension of "bacterial disease".

Moreover, this minimal directness requirement avoids facing the regress that, if a disease is necessarily e.g. a disease kind, and if a disease kind is necessarily e.g. a disease super/subkind, then a disease is necessarily a disease super/subkind, and, if a disease super/subkind is necessarily e.g. a disease supersuper/subsubkind, etc. Requiring that a term like "disease" has at least another term for its *direct* (relative/absolute) avoids this regress.

Note that the chain of extensionality can be, of course, *cumulated* (without allowing, however, transitivity); when there are de facto more than mere direct specific metaphysical constraints, it remains the case these are still indirect specific *constraints*, which should be taken into account.

[87] Let us suppose that there are no more animal kinds except human beings. Does this imply that a human being is no longer an animal *kind*, but would be an animal *tout court*? The philosopher who thinks that we need at least *two*

To summarize, objection (iii) of Sec. 3.2.3 is based on the strong intuition that e.g. to be colored is to have a specific shade of color, or that to be diseased is to have a specific disease (or to have a disease kind).

According to interpretation (i), this intuition does *not* show that e.g. a specific disease is to be *defined* as a conjunction (cf. Sec. 3.2.1) or an exclusive disjunction (cf. Sec. 3.2.2) of the specific disease kinds under which they fall, but it merely shows that a term like "color" or "disease" *necessarily* has a *relative* extension – but not *essentially*, or "disease" is not to be relatively extensionally *defined* (otherwise, objections (i) or (ii) of, respectively, Sec. 3.2.1 and 3.2.2 may be easily raised).

In a nutshell, the idea that the intension of a fundamental term like "genus" is its relative extension is not to be extended to other non- (or less) fundamental (or general) terms like "disease", where relative extensionality is here interpreted as a specific metaphysical requirement.

According to interpretation (ii), this intuition shows that a term like "color" or "disease" must have an *absolute* extension. The philosopher arguing that the determination relationship should be understood along interpretation (ii) could claim that e.g. {disease} refers to the set of all specific diseases – or, that the definition of "disease" is its absolute extension - i.e. that the determination relationship would be a *definitional* relationship.

However, this philosopher – to stay immune to our reply to objection (iii) (cf. Sec. 3.2.3) - can also claim that the determination relationship understood as absolute extensionality is not a definitional relationship. If we want to consistently maintain both our own way to understand the genus-species relationship (cf. Box 3.1; 3.1.1-3.1.2) and the idea that the determination relationship is a *bona fide* relation, then the intuition that there is no disease without one *specific* disease only means that absolute extensionality is *necessary* for a term like "disease" – but not *essential* to it, or "disease" is not to be absolutely extensionally *defined* (otherwise, objection (iii) of Sec. 3.2.3 may be easily raised) -, where the necessity at issue is a specific metaphysical necessity flowing somehow from a kind's essence (on the (related) notion of *consequential essence*, cf. Fine, 1994b; also 2002)[88].

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animal kinds to make sense of our talk about something being an animal kind precisely commits the mistake of thinking that the determinable (or genus) animal - and if he also rejects the possibility of singleton sets/classes/groups - is defined (at least, plausibly taken) (cf. Sec. 2.1) by its determinate*s* (or species/kind*s*); on the notion of *extensional definition*, cf. Sec. 3.1.2).

Moreover, one is not saying that it is part of the correct *intension* of a term like "disease" to have a direct relative (and absolute) *extension* (or, that *specific* genera are essentially, plausibly taken, parts of species); otherwise, one is committed to RME (cf. Sec. 2.1).

I acknowledge Niko Strobach (personal communication, March 2017) for having pointed this to me.

[88] Note that some metaphysicians strictly distinguish between the relationship of *essence* and of (metaphysical) *grounding* (restrictedly taken) (on (metaphysical) grounding, as entries, cf. Bliss & Trogdon, 2014; Correia &

But, if absolute extensionality is to be granted the status of a genuine necessitation relationship, should we not also say that there must exist, correlatively, a certain maximal or top-level entity (however understood), for both relative extensionality and the relationship of ontological priority are granted the status of genuine relationships (cf. Sec. 3.1.1)? In other words, is a (said) *junky* world i.e. a world where everything is a(n extensional or intensional) proper part (however understood) *possible* (for optimists, cf. Bohn, 2009; Morganti, 2009; Trogdon, 2017; *pace* Cotnoir, 2014; Schaffer, 2010)?

Like for the case of gunky worlds, even though we may argue that there possibly exist some things like certain sets (e.g. qua objects) which may be considered as junks (understood in a certain way), not everything can be a junk, at least in a maximally populated world, along our own definition of e.g., more specifically, intensional (proper) parthood (cf. Ch. 2).

Indeed, if one acknowledges the existence of species/kinds, then they can be considered the highest ontological category – at least, as non-trivially or essentially defined entities -, albeit kinds/species do not exist, *because* we judge that there must be a top level; if kinds exist, that is because, in their case, they have an essence; and there is nothing else essentially defined, which is more fundamental (or general) than species/kinds.

Other illustrations of the absolute extensionality requirement (for kinds) include our own (higher-level) metaphilosophy (on ME, cf. Ch. 2), for, if a (higher-level) metaphilosophy necessarily has an absolute application (or extension), then we could say that the genus-species relationship (cf. Box 3.1) is precisely a basic absolute application of it, where the genus-species relationship may be considered itself a (lower-level) metaphilosophy (or, a certain neo-Aristotelian framework) also necessarily having an absolute application (or extension): e.g. disease kinds and disease (cf. Sec. 3.1.1).

Along interpretations (i) and (ii), the requisite of, respectively, relative and absolute extensionality means that relative and absolute extensionality are *specific metaphysical necessities*, where our notion of essence is one of the other specific metaphysical necessities (cf. Ch. 2); metaphysical necessity *tout court* is usually said to include statements of, supposedly, strict or loose (synchronic or diachronic) identity like e.g. "Water is H_2O" or "Gold is the element with atomic number 79" (Kripke, 1980; Putnam, 1975).

These specific metaphysical necessities are intuitively justified by the fact that, when one gives the intension of a term, one has to check (or verify) what *can* be plausibly said to satisfy this intension.

Schnieder, 2012) – though the notion of ground can be still otherwise used viz. non-restrictedly or plausibly, of course (as a reason or a (preceding) cause e.g.).

We can finally notice through the distinction between the genus-species relationship and the determinable-determinate relationship that we often conflate talk about determinates (or realizers) of determinables and talk about species of genera.

For, it is tempting to think that, when we talk about *specific* diseases, we are essentially talking about disease *kinds*, but a specific disease may be in the *absolute* extension of "disease"[89].

It is also important to highlight that, if we say that the determination relationship is just a relation of absolute extensionality – and not a definitional relationship -, then it implies the rejection of the widely accepted basic idea behind the BST of health (and disease) (Boorse, 1977; 1997; 2014; cf. also Giroux, 2016) viz. the idea in our case that the notion of disease is *statistically established* (or defined).

Indeed, for the BST of health and disease, in the case of disease as well as of health, a disease would be established from the members of a reference *class*; a disease held by, for the BST of disease, a reference class is somehow (*mutatis mutandis*) *read off* from the specific diseases held by the members of this class.

Thus, according to the BST of disease, to get what e.g. lung cancer within a reference class is, we generalize from (or average over) (specific) lung cancers hosted by the members of a reference class.

However, if I am right, "disease" is not to be absolutely extensionally defined: we do not generalize from members of a reference class to get what lung cancer is; lung cancer (taking a certain host) is a *general* disease necessarily having for absolute extension (but not for intension) *specific* lung cancers taking specific hosts: e.g. small cell lung cancer; non-small cell lung cancer.

What we eventually get from our reply to objection (iii) of Sec. 3.2.3 is, first, that, although the general level is metaphysically (but *not essentially*) dependent upon, or constrained by, the specific one (but not vice versa), from e.g. specific diseases one cannot somehow *induce* a certain (general) theory of disease (even one with a limited or wide scope of application); if one

[89] Two caveats here: (i) the same mistaken impression is true with commonplace or everyday examples: when we are used to say e.g. "this kind of people is…", we are obviously not talking about something *more* than people, but about *specific* people – thus, we should strictly say "such people are…" –, albeit such a talk can be still maintained as plausible, of course.
This point also applies to so-called "definitions by restriction" (or categorizations through absolute extensionality), where we would like to (wrongly) claim that, once we give a definition of *x* where *x* is a specified (or restricted) *y*, *x* is a *kind of y*, while we should say in those cases, *properly* speaking, that *x* (as a kind) is a specific *y*.
(ii) This case (cf. caveat (i)) is also similar but different from the other case, where, when we talk about e.g. human*kind* or man*kind*, we are obviously not talking about *kinds of* human being or men (strictly taken), or even about *specific* humans or men, but just about the *kind* human or the *kind* man. These examples may accentuate the mistaken idea that diseases and disease *kinds* are, actually, just one and the same thing, but linguistic usage is here at fault.

usually thinks so, it is because one already bears in mind or *assumes*, actually, a certain theory of disease – which can be false, of course[90];

in other words, to somehow induce a certain theory of disease from specific illustrations of disease is *theory-laden* (*pace* Wilkins & Ebach, 2014), or takes the absolute extensionality relationship in the wrong order (on the relationship between philosophy and science, cf. Exc. 3.5; on a related point about functionalism, Sec. 4.2.7; on the related thesis of *modal normativism*, Thomasson, 2007; 2012; on so-called "bottom-up" (vs. "top-down") approaches to philosophy of science, Kaiser, 2015; Weber, 2005).

Thus, looking for specific illustrations of disease (e.g. in the medical literature), so as to critically build – or rather, actively reconstruct, make explicit, uncover or explicate - a (general) theory of disease on one's own or by relying on (or even, oneself conducting or analyzing) experiments, surveys, interviews with scientists, qualitative/quantitative data, etc., is mistaken (on experimental/empirical philosophy of science, cf. Griffiths & Stotz, 2008; Wagenknecht et al., 2015; on experimental philosophy in all its generality, Knobe & Nichols, 2007); this mistake comes from holding a (very) strong version of naturalism.

Rather, there is a certain general theory of disease ((externally) justified) (Janich, 2005), which is still, however, somehow realized, applied or *used* in scientific practice – but, the theory in question is not (essentially) its use (on a related point, cf. Sec. 3.2.4); the rejection of an over-confidence toward the scientific practice does not imply, thus, the subscription to an extreme version of scientific skepticism[91].

A second - related - reason is that e.g., intuitively, Mary could know everything there is to know about what redness is, but still does not know that red is, actually, a specific *color*, for the definition of "redness" *absolutely* applies (for the sake of the argument here) the definition of "color"; or, in the ontological idiom, the existence of e.g. a hand does not entail that there are – at least, one – material objects – the other way round, yes.

3.9 Excursus: On Sets (and Qua Objects)

[90] Caveat on inductive inference: inductive inference (however limited) also has, of course, other well-known problems on its own, amongst which e.g. the issue that one just cannot read off some *normative* claim (however taken) from mere commonalities, regularities or statistics found out (or whose tokens are observed) in a certain case study (cf. Hume, 1748 [1910]), or that (ab)normality (or any normative claim) has simply nothing to do with its frequency.

[91] Note, moreover, that, on *both sides*, biases of providing *ad hoc* theories are to be avoided – philosophy should not *make* scientific theories match its own theories, and ditto for science (with the minimal requirement of direct relative extensionality).

Where does the requirement of absolute and relative extensionality come from? If there is no disease without one specific disease, and no disease without a disease kind, then the necessity for "disease" to have an absolute extension is somehow conferred from the idea that *there is* fundamentally something absolutely extensionally defined, exactly like the necessity for "disease" to have a relative extension is somehow conferred from the idea that "disease" can be said in the fundamental or general absolute extension of "genus" (which is an entity relatively extensionally defined).

The notion of *set* (in all its complexity) can certainly play the role of the entities, whose existence is required here.

Indeed, intuitively, the notion of set/class can be understood in a two-fold way: (i) as being *absolutely* extensionally defined (however plausibly understood) (i.e. a set in an *absolute sense*): e.g. the specific set {disease kind} includes specific disease kinds like (amongst others, or at least) STDs (cf. Sec. 3.2.3; 4.1.1);

or, (ii) as being *relatively* extensionally defined (however plausibly understood) (i.e. a set in a *relative sense*): e.g. the specific set {animal} includes (amongst others, or at least) a human being (cf. Sec. 3.2.1-3.2.2), or {genus} includes a species.

Under its absolute sense (i), to follow here a certain (neo-Aristotelian) literature (cf. e.g. Fine, 1982), a set is better called a "*qua object*" – though talk about a set in its absolute sense can be maintained, of course.

Qua objects go back to the Aristotelian (*Met*. Δ.6, 1015b17; Δ.9, 1017b31; *Phys*. A.7, 190a19-21) notion of so-called "kooky objects" (Matthews, 1982) or "accidental unities (or beings)" (Cohen, 2008; 2013; Koslicki, 2008): classical examples include the musical Coriscus, the seated Socrates, Socrates as being fond of dogs, or maybe the statue of clay/bronze/gold (cf. Gallois, 2017), etc. – where, for Aristotle (*Met*. Δ.6, 1015b17; Δ.9, 1017b31) e.g. Socrates and the seated Socrates *accidentally coincide* (along the interpretation of Cohen, 2008); some understand this relationship as the *grounding* relationship (restrictedly taken) (cf. Correia & Skiles, 2019).

Applied to our case study, a disease may be considered itself, thus, *fundamentally* (but not indirectly *extensionally* reductively) a specific qua object i.e. a disease qua e.g. lung cancer.

According to this understanding of qua objects, the intension (or rather, definition), plausibly speaking, of a qua object may be said its absolute extension: a qua object is essentially, plausibly taken, an object which has an absolute extension, or *at least* one absolute realizer; or, the nature of a qua object is that an object (e.g. disease) exists only under a *specific* form (i.e. qua…): e.g. disease as merely a specific disease like lung cancer; or Socrates exists only under specified forms like seated (and etc.) (or, Socrates is the totality of his specified forms).

From this, it is useful, for the present purpose, to distinguish between *accidental* properties (i.e. here those properties that an object possesses (weakly taken) and which *coincide* with the object's essential properties) (e.g. the seated Socrates) and *extrinsic* properties (i.e. here those properties that an object

possesses (weakly taken) and which do *not coincide* with the object's essential properties, and where this object and its extrinsic property are both two essential parts of a whole): e.g. a disease negatively caused by/causing something as a disease kind (cf. Box 4.2).
Thus, if extrinsic properties (e.g. instrumental/consecutive value properties) are taken here as the properties which do not coincide with an object's essential properties, then *essential* (e.g. intrinsic *final* value properties) and *accidental* properties (e.g. intrinsic accidental value properties) (as taken here) may be said, contrariwise, *intrinsic* properties (on intrinsic *values*, cf. Rabinowicz & Rønnow-Rasmussen, 2000; *pace* Moore, 1903 here). Along our definition of extrinsicality, *intrinsic* properties may very well be *relational* properties (on extrinsic (often wrongly taken as relational) vs. intrinsic properties, cf., amongst others, Kagan, 1998; Plate, 2018; Rønnow-Rasmussen, 2015).

3.10 Excursus: Holism (About Kinds)

Why not, actually, admitting that a kind should be defined both *constitutively* (along a certain reading of ME) and *teleologically* (along a certain reading of RME)? A heart process e.g. would be, thus, essentially both the pumping of blood (constituents) and a part of a circulatory system (telos)? Although this answer would certainly please biologists and highly naturalistic-minded philosophers understanding *holistically* biological systems, organismic complexity and interconnection between mereological levels, it is to be rejected on the firm grounds that RME is, strictly taken, false (cf. Sec. 2.1).
Therefore, any use of RME should be taken in situations, where the term "essence" is read under a *plausible* meaning.
The conjunction of (restricted or *strong*) ME and RME very likely falls under so-called *holistic* approaches to (physical, biological, biomedical, social, linguistic, moral, etc.) *kinds*, although the notion of holism (in all its generality) is far from being clear in the literature (on holism in the (special) sciences, cf. Weber & Esfeld, 2003).
The main issue with holism (about kinds here) (or the conjunction of (strong) ME and RME) is that it takes different ontological dependency relationships to be all just *definitional* relationships – thus, where "definition" would be a much encompassing notion.
In a nutshell, holism (about kinds or in all its generality) takes definitions of phenomena as *multi-faceted* (not necessarily taken in a pluralistic (e.g. perspectivist) sense), *complex*, *multi-level* (especially in the neo-mechanistic philosophy of science (Craver, 2007)), or as having a lot of *ramifications* (on e.g. cancer as a disease of a cell('s part) *and* of the whole organism, cf. Laplane, 2016).
However, first, in the special case of holism about *kinds*, we can very well admit the existence of different ontological dependency relationships (e.g. specific *necessitation* relationships - other than the one of essence - between e.g. a man and his ecological system), while maintaining that a definitional

relationship is only a certain one i.e., so to say, without (unlike holism) painting everything with one and the same brush.
Second, we can very well argue that a permissive neo-Aristotelian ontology, by multiplying the number of specific existing *kinds* (or, more generally, of other specific entities) here, precisely allows us to give *non*-multi-faceted definitions of phenomena.

3.3.2 A Reductive Account of a Differentia

A specific kind/species is defined as a conjunction of a specific genus plus a *certain* differentia (or a differentia specifica) defined independently from the definition of the genus in question (cf. Box 3.1), but where a differentia *tout court* (or *διαφορά*) is reducible to a kind/species or a genus i.e. that *x* is a differentia, iff this very *x* is a kind/species or genus itself (cf. Box 3.1.2); in that sense, a differentia is not a genuine entity, and is, thus, outside our meta-philosophy - a differentia *tout court* is not itself defined dependently upon the definition of a genus, because of its being a non-genuine entity falling outside our meta-philosophy.
First of all, the requirement that a *specific* differentia be defined independently of the definition of the genus in question is intuitively justified by the fact that, if we argue that a species is a genus in *conjunction* with a (same-level) differentia, then, were the definition of a differentia dependent upon what a genus is, a genus would cease to fulfill its role as being (a constitutive) (*proper*) *part* of a species; the two conjuncts would not be logically independent any more.
This understanding of the differentia mainly follows its Scotist and Thomist interpretation (cf. also Ari. *Met*. B.3, 998b23-998b26 – but against *Met*. Z; H; *Top*. VI (cf. Deslauriers, 2007 on that controversial question)), where for Thomas Aquinas e.g. "[b]eing […] is not divided as a genus (e.g. animal) is into species (e.g. human) by means of differences (*differentiae e.g.* rational), because such differences need to be *outside* the genus's essence (e.g. rationality is not included in an animal), and nothing lies outside being" (Gracia & Newton, 2012; italics original and personal) i.e. that, indeed, a differentia is defined independently from the definition of a genus[92].

[92] Of course, we may eventually wonder which reasons there are for giving a definition as a conjunction. The main reason is that, if the neo-Aristotelian metaphilosophical framework consisting in giving a definition within (our understanding of) a genus-species hierarchy is a *specific* metaphilosophical framework consisting in giving *metaphysically* necessary (in the sense of Fine, 2002; cf. also Kment, 2014) and sufficient conditions for the definiendum (cf. Ch. 2), then, were a differentia – as a specific (direct) *constitutive part* (with the genus as the other (same-level) constitutive part) of a species/kind - definitionally dependent on what a genus is, a genus would not be essentially a component of a species but of a differentia.

However, along the Scholastic tradition of the predicables, one may want to counter-argue here that the differentia does not differentiate a specific genus from its species, but one species of the genus from the others.

Again (cf. Sec. 3.3.1), one should be careful here in not thinking that one needs at least two peculiar species of a genus to make meaningful one's talk about *genera*. If one thinks so, one would be committed either to RME (under a certain reading) (cf. Sec. 2.1) or to objections (i)-(iii) (cf. Sec. 3.2.1-3.2.3), for one would think that the definition of a genus is dependent upon what its species *are* – and if one also excludes singleton sets/classes/groups.

But, we need not two species, in this case, in so far as the definition of a genus does not depend upon what its species/kinds are (cf. Sec. 3.3.1), though we argue, however, that a genus is a constitutive part of a species (cf. Box 3.1.1).

Second of all, the requirement that a differentia *tout court* is simply not a genuine entity, but is reducible to a kind/species or a genus, is motivated by the fact that, given our *minimal* definition of a genus as a constitutive part of a species as well as the one of a species/kind (cf. Box 3.1; 3.1.1), we fail to see in what sense a differentia, such that it would be something different from a genus or a species, can really be something at all, if not, actually, (reduced to) a genus or a species/kind: e.g. rationality has, at the general level, intuitively no other special ontological status than a *species/kind* itself or, even more generally, a specific *genus* itself - and if one also tries to avoid category mistakes (e.g. a differentia could not be just a certain (differentiating) property); the ontological status of a differentia is inexistent.

From this, it does not change that a whole still has a differentia as a constitutive part per se (cf. Sec. 2.1; Box 3.1) – but, which is itself a kind/species or a genus; a differentia *specifica* has to be, of course, a *different* kind/species or genus from (trivially) the defined kind/species and (especially) the genus in question.

However, first, does this not mean that we fundamentally need at least two species (or genera) to make sense of a differentia, since a differentia is reducible to a species or a genus?

In parallel with the case of the genus, which does not require that two species be (at least) present (e.g. were a human being the only surviving species, a human being would still be intuitively an *animal* kind), albeit (the concept of) a differentia is reducible to (the concept of) a kind or a genus, only a differentia *specifica* (e.g. rationality), in line with the WSP, shall exist as a different specific kind or genus itself (from e.g. humanity or animality).

Second, although, in a definiens, both conjuncts' order can be swapped or *commutated* (thanks to their truth-functionality) (e.g. a rational animal (Gy & Fx) is obviously also an animal which is rational (Fx & Gy)) – i.e. that e.g. a human being is something more than a mere animal *or*

mere rationality -, we may eventually ask: why has a human being, then, for its genus animality instead of rationality?

In other words, if *what* stands for a specific genus and *what* stands for a specific differentia cannot be swapped (albeit their *order* is commutative), how to justify that e.g. animality - rather than rationality - is the specific genus of a human being (or humanity)? With our minimal definition of a genus as well as a reductive definition of a differentia (cf. Box 3.1.1-3.1.2), what would this change for a human being (or humanity) to have rationality rather than, as commonly accepted, animality as its genus?

We cannot but answer here that, in parallel with the case of lung cancer, which can very well be said more directly (in the absolute extension of) e.g. a cancer than a lung disease - or maybe the contrary -, a human being has for genus animality – and not rationality -, because we deem that a human being has somehow more animality in him than rationality, in the sense that a human being has a higher number of indirect constitutive parts having as (indirect) relative realizer animality rather than rationality; in one word, a human being has more indirect parts in him that are animal rather than rational. The same point applies, of course, to other cases like disease kinds (cf. Box 4.2).

A first consequence of our reductive account of a differentia (cf. Box 3.1.2) is that we can make sense of the difference between a specific genus and a differentia specifica in a precise way (without that being part of the definition of a differentia *tout court*): it is something more peculiar (or determining) than a genus for a certain species, precisely because it is somehow *rarer* in the species in question – not because it would be something *unique* to the species, which would differentiate it from other species with the same genus.

A second related consequence is that, contrary to what schoolmen generally say of a differentia (specifica), a differentia specifica does not have to be unique to a specific kind/species, albeit it can happen that this is the case, of course: e.g. rationality can very well constitute another kind than a human being, or negative causes-effects can very well be the differentia of another species/kind than disease kinds (cf. Box 4.2) – and, thanks to our relaxed definition of a differentia (cf. Box 3.1.2), that much is of no problem, for the kind in question just is distinguished with respect to its entire specific essence viz. a genus *and* a differentia (cf. Box 3.1).

II

CONCEPTS RELATED TO DISEASE

4

NOSOLOGY

Of course, saying that there is a distinction to be made between diseases and disease kinds is not defining what a disease kind is or what disease kinds are, to the contrary of defining what disease is. Ch. 4 is about how to intuitively classify disease kinds. I shall give two different criteria (negative effects and causes) i.e. the differentia (cf. Ch. 3), which allow us to distinguish between disease kinds and disease.

Part II of the present PhD thesis is about the different concepts related (pretty directly) to the one of disease.

Sec. 4.1 provides a classification of disease kinds along various criteria (i.e. a nosology) viz. negative effects and negative causes. This nosology will allow us to approach more directly the notion of disease by dissociating DISEASE from other concepts (cf. Ch. 5-6).

Sec. 4.2 addresses objections, which can be raised against our definition of disease kinds, more precisely against the definition of a disease kind as a disease (i.e. the genus) in conjunction with negative causes and effects (i.e. the differentia).

Ch. 5 and Ch. 6 will more thoroughly analyze the symptoms (i.e. the negative effects of a disease) and etiopathology (i.e. the negative causes of a disease).

4.1 A Definition of Disease Kinds

Drawing a distinction between disease and a disease kind along the genus-species relationship i.e. merely saying that a disease kind falls under disease with a differentia (cf. Sec. 3.1.1), is not saying much about what a disease kind is more precisely. To avoid, at the outset, conceptual confusions between DISEASE and DISEASE KIND, it is important to devote Sec. 4.1 to the clarification of *what a disease kind is* i.e. that it is important to review the differentia (or the criteria) allowing us to tell what distinguishes disease from a disease kind.

Sec. 4.1 is organized as following: Sec. 4.1.1 is about how the distinction between disease and disease kinds can tell us about what disease is intuitively *not*.

Sec. 4.1.2 aims at reviewing the different criteria that allow us to distinguish between, on the one hand, disease and, on the other hand, disease kinds viz. negative effects and causes related to a living being.

Sec. 4.1.3 examines the prospect of defining a disease kind as a syndrome.

4.1.1 Defining Disease Kinds vs. Defining Disease

The purpose of the present PhD dissertation is to give a definition of disease and not of disease kinds. Nevertheless, focusing, first of all, on a nosology (i.e., as taken throughout the PhD dissertation, the study of the differentia for disease *kinds*) is justified by the fact that from an intuitive classification of disease kinds along diverse criteria it shall be easier to directly approach DISEASE and its related concepts (cf. Ch. 5 et sqq.).

Indeed, from the trivial idea that for *x* to have a disease kind is for *x* to be diseased in conjunction with a differentia (or criteria here) (cf. Sec. 3.1.1), it follows that the focus on this differentia by which a disease kind is made a disease kind by contradistinction with a disease highlights what distinguishes a disease kind from a disease *tout court*.

In a nutshell, to the extent that these differentiating characteristics are properly parts of what disease *kinds* are, it is useful to review them, for they tell us something about what disease is intuitively *not*.

More formally put, if to have a disease kind is to have a disease in conjunction with (a) specific characteristic(s) i.e. if to have a disease kind is to fall under the genus disease with a differentia, then it follows from our analysis of the genus-species relationship (cf. Box 3.1) that this

differentia is to be defined *independently* of the definition of the genus in question i.e. disease; otherwise, the genus-species relationship collapses into a determinable-determinate relationship (cf. Sec. 3.2.3).

If a definition of disease kinds should really follow a genus-species hierarchy, then this definition is to be contrasted here with another common way to define disease kinds: {disease kind} as a class/set of members/elements. There are two plausible ways to understand in what sense {disease kind} may be said a class/set:

(i) in listing the whole absolute extension of the term "disease kind" by saying that {disease kind} is a class/set whose members/elements are specific disease kinds: e.g. a bacterial disease is a specific disease kind, an infectious disease is a specific disease kind, a STD is a specific disease kind[93], etc.;

(ii) in trivially defining a disease kind as the genus disease of a certain kind or species. According to point (ii), {disease kind} is a class/set comprising the members/elements called "_of a certain kind" and "disease". Since one of our requirement on what it is to provide a *correct* definition is precisely to avoid triviality or reflexivity (cf. Sec. 2.1), it follows that we cannot stay content with a definition of a disease kind as merely a disease of a certain kind, but that a substantial definition is required; saying that a disease kind is a disease kind is hardly explanatory on the nature of disease kinds – or only for a pseudo- or "dormitive virtue" explanation.

Cannot we stay content, however, by simply stating that to have a disease kind is to have a disease (i.e. condition (i)) with a specific differentia (i.e. condition (ii)), without saying what this differentia is (cf. Box 4.1)? Such a prima facie definition would amount to saying that to have a disease kind is to be diseased with anything that differentiates a disease kind from a disease.

As such, this prima facie definition does not avoid reflexivity, to the extent that the term "disease kind" also appears in (the elements of) the definiens.

Furthermore, one can argue that, if there are such kinds as disease *types* or disease *genera* to be distinguished from disease *kinds*, then a *correct* definition of disease kinds has to be precise enough – thus, not prima facie as here (cf. Box 4.1) - for avoiding being confounded with disease types or disease genera.

[93] Thence, it does not follow that e.g. a bacterial disease is to be *defined* as a disease kind in conjunction with a differentia. Were this true, then a bacterial disease could be indirectly said a disease kind of a certain kind or species. It seems better to define a bacterial disease, strictly speaking, as a disease with a differentia. *Being a disease kind* can be, thus, considered an indirect property of a bacterial disease, or we can say that "bacterial disease" is in the indirect *absolute* extension of "disease kind" (cf. Sec. 3.1.2 on that), for listing the indirect extension of a term does not imply that this term is a direct property of what the (definition of the) term applies to.

However, if a disease kind is to be counted as a specific kind/species itself (as we try to show it (cf. Box 4.2)), the following prima facie definition of disease kinds (as given here) can serve as a *preliminary* step towards a *correct* definition of disease kind (cf. Box 4.2):

Prima Facie Definition of a Disease Kind
x is a disease kind, iff (i) *x* is a disease (i.e. the genus), and (ii) *x* is *F* and etc.

Box 4.1. – A prima facie definition of disease kind within (our understanding of) a genus-species hierarchy.

What is, thus, this differentia that allows us to distinguish between a disease kind and disease?

4.1.2 Negative Effects and Causes as the Differentia between Disease Kinds and Disease

One differentiating characteristic between disease and disease kinds which intuitively comes, first, to mind is the *effect*/sign of a disease; more precisely, in the present context, when we talk about the effect *of a disease*, we usually talk about a *symptom*.
Indeed, specific infectious diseases like influenza or tuberculosis may be commonly defined by symptoms; for influenza: fever, runny nose, sore throat, muscle pains, headache, feeling tired and coughing; for tuberculosis: fever, chills, night sweats, loss of appetite, fatigue, weight loss and nail clubbing[94].
A second differentiating characteristic intuitively coming to mind is the *cause*/etiology of a disease; again, in the context where we talk about the negative cause *of a disease*, we are used to employ the term "*etiopathology*"/"*etiopathological agent*" (cf. Ch. 6).

[94] Caveat on the illustrations of symptoms: I rely here on a plausible pre-conception of what a symptom is. Even if it happens that our illustrations here of a symptom do not strictly (or *correctly*) follow our own definition of a symptom (embedded into a certain metaphilosophy) (cf. Box 5.2), in so far we may argue that these *can* be *plausibly* argued to be specific symptoms (e.g. as being *functionally* defined: e.g. fever *simpliciter* – rather than, strictly speaking, fever *of* a disease *x* – as a sign of a disease *x*), or that they are *compatible* with our own definition of symptoms (cf. Sec. 5.1.1), we can still *use* them, for our purpose *here*, as illustrations of symptoms.
The same is true for illustrations of etiopathological agents as well as of disease kinds themselves (or even diseases), as long as these illustrations, depending on a certain use, of course, fall under a *plausible* interpretation (or reading) of a certain definition of etiopathology or of disease kinds (or even of disease).
Indeed, what it is for a definition (i.e. the definiens) of disease kinds to be *correct* is, along the MCP, for it to be weak enough, such that the definition at issue is *compatible* with as many *plausible* conceptions (or understandings or interpretations) of the definiendum in question as possible.

Indeed, specific disease kinds like bacterial or viral diseases or STDs are typically defined by their etiopathology; a specific viral disease like ordinary-type smallpox is usually said a specific disease (negatively) caused by the presence of the virus variant *Variola maior* (as a starting *state*). Similarly, a specific STD such as syphilis or *Chlamydia* infection is commonly said a specific disease caused by the bacterium *T. pallidum* or the bacterium *Chlamydia* (cf. Box 4.2), or Lyme disease (or borreliosis) is a specific disease caused by the bacterium *Borrelia* (as starting states).

The advantage with such a two-fold way to define the differentia between disease and disease kinds is that, if having a disease kind is having a disease in conjunction with specific effects *and* causes viz. negative ones[95], then the nosology seems sufficient (but, cf. especially Sec. 4.2.8 for concerns) for dealing with cases, where the same symptoms or etiopathological agents are found within what are plausibly *different* specific disease kinds: e.g. cough, headache, runny nose and sore throat are said symptoms constitutive of e.g. common cold and pharyngitis – and they might well (*plausibly*) constitute many more specific disease kinds. Similarly, Human Papillomavirus (HPV) disease (or infection) is a specific infectious disease which can be said constituted by HPV as an etiopathological agent (plausibly as a starting *state*), but HPV is also known for being the etiopathology constitutive of other specific disease kinds like cancer (kind)

[95] Four terminological notes (with caveats) about negative causes/effects (of disease): (i) to get a precise enough definition of disease kinds, the causes and effects being the specific differentia for disease kinds (cf. Box 4.2) must be *specific* causes and effects viz. *negative* ones. Indeed, even though the specific differentia is still defined independently from the genus in question i.e. a definition of a *specific* causation, on pain of a category mistake, these causes and effects must be specific, for they must be defined at a same *compatible* level as the one of a disease, and, if a disease is intuitively a certain *negative* entity, then, to be *coherent* both with our metaphilosophy (cf. Ch. 2) and our own definition of disease (cf. Box 9.3), and precise, then, in a *correct* definition of disease kinds, the differentia shall be *negative* causes and effects themselves too.
(ii) As being among the fundamental or general categories, if a *negative cause*/*effect* is still *itself* a specific *cause*/*effect tout court* viz. a negative one, then, if a disease kind is a disease plus a negative cause-effect, then it is also at the same time (more indirectly) a disease plus a cause-effect, although a disease kind is *more directly* a disease plus a *negative* cause-effect than a disease plus a cause-effect *simpliciter*. Thus, in the context of disease kinds, the notions of negative cause-effect (*tout court*) or cause-effect *tout court* can be both *used*.
(iii) However, even though both notions can be used, the former notion is to be preferred over the latter one, for talk about cause/effect *tout court* easily leads to the misleading impression that one is necessarily talking about a *positive* cause/effect, but it is not so, for the (highly) fundamental or general ontological categories are arguably *value-neutral*;
moreover, a *strict definition* of a disease kind (cf. Box 4.2), to be coherent with a certain definition of disease, is phrased such that it is about, more precisely, *negative* causes-effects (exactly like disease is a specific *negative* kind before being a specific kind *tout court*, for, if a disease were, above all, a *specific* kind *simpliciter*, then it could be either positive or negative or value-free, but a disease is intuitively *negative*) (cf. Sec. 4.2.2).
(iv) Note that notes (ii)-(iii) apply to the other (highly) fundamental ontological categories like kind/species, genus, whole, (constitutive) part, constitution, process, state, essence, etc. On grounds of textual readability and simplicity, I shall, thus, use e.g. the term "kind", "species", "genus", etc., *simpliciter* to refer to, more precisely, either *positive* or *negative* kinds, species, genera, etc., - for these notions are meant to apply here beyond the mere area of negativity; they are related to a *universal* metaphilosophical framework (but still restricted to e.g. kinds, of course) (cf. Ch. 2-3) -, except in the cases where, of course, the distinction between positivity and negativity does matter or needs to be made explicit on pain of easy misunderstandings.

(Muñoz et al., 2006) and perhaps even Cardio-Vascular Disease (CVD) kinds (Kuo & Fujise, 2011)[96].

However, it is important to note here that, properly speaking, the differentia between disease kinds and disease is the presence of a negative *effect*/sign and a *cause*/source *tout court* – and not of a symptom and an etiopathological agent.

Indeed, it is intuitive that a symptom (in its literal meaning) is essentially a negative effect *of a disease* (cf. Ch. 5), while an etiopathological agent (in its literal meaning) a negative cause *of a disease* (cf. Ch. 6). Thus, were the differentia for disease kinds a symptom and etiopathology, then this differentia would be defined dependently on what a disease is, since a symptom is an effect of a disease and etiopathology the etiology of a disease.

Hence, if we strictly and coherently follow our definitional structure corresponding to a genus-species hierarchy (cf. Sec. 3.1.1), then a disease kind is to be defined as a disease (i.e. condition (i)) in conjunction with a specific effect and a cause (of an entity) viz. negative ones (i.e. condition (ii)). As such, a definition of the specific differentia (causes and effects) can be easily given independently of the definition of the specific genus (disease)[97].

Moreover, to leave room for as many *plausible* interpretations of our definition of disease kinds as possible (but still compatible with our own theory of disease) (cf. Box 9.3), but still to be more precise with our definition of disease kinds (with an independently defined differentia), those negative causes and effects, in this context, have to be obviously related somehow to *living beings* (even when we plausibly ascribe diseases to artefactual beings, or take them as states, or take symptoms as highly indirect): e.g. a *strict* interpretation of "related to a living

[96] Of course, one may still answer here that such symptoms or etiopathological agents are far from being sufficient for defining the disease kind in question, but cf. Sec. 4.2.1 for this objection.

[97] Caveat on the MF: it is important here of not being guilty of the so-called "mereological fallacy" (Bennett et al., 2007), where

(MF) a predicate of a whole is attributed to one of its (*im*)proper parts.

In the present context, it means that we cannot say that *x* is a(n) etiopathological agent/symptom *of* a disease *kind*. Indeed, one should more properly say that a disease kind is constituted by a(n) etiopathological agent/symptom. However, so as to maintain (or highlight) our definitional structure corresponding to a genus-species hierarchy (cf. Box 3.1), it is better to talk about a disease kind constituted by a disease (i.e. the genus) in conjunction with negative causes and effects (i.e. the differentia).

We would like to intuitively say that that much amounts to saying that a disease kind just is a symptom with a cause or an etiopathological agent with an effect – but not a symptom *and* etiopathology, for it would be redundant, since a symptom is undeniably (synonymously) the negative effect *of a disease* (cf. Box 5.1) and an etiopathological agent the negative cause *of a disease* (cf. Box 6.1).

However, were this true, then the differentia would be defined *dependently* upon the definition of the genus in question. This is, thus, not a *correct* definition (within (our understanding of) a genus-species hierarchy) of disease kinds.

To define more properly a disease kind by following our definitional scheme (cf. Box 3.1), we have to *tripartitely* divide the definition of a disease kind, where a disease kind is *strictly* (or *correctly*) defined as (i) a disease and (ii) negative effects and (iii) negative causes (cf. Box 4.2).

being" shall take this expression to refer to a processual part of a living being situated at the same level as the one of the genus disease; a plausible interpretation of our definition of disease kinds shall take e.g. smoking as a negative cause related to a living being in the sense of being related to a person (who is smoking) (cf. also Box 5.2; 6.2 for the same condition)[98].
In doing so, we get the following correct definition of a disease kind (cf. Box 4.2):

Definition of a Disease Kind

x is a disease kind, iff (i) *x* is a disease (i.e. the genus), and (ii) *x* is a (specific) negative cause related to a living being, and *x* is a (specific) negative effect related to a living being (i.e. the differentia).

Box 4.2. – A definition of a disease kind within (our understanding of) a genus-species hierarchy.

Of course, since in the present context we are specifically talking about the negative effect and cause *of a disease* – and not of something else -, we shall obviously focus (cf. Ch. 5-6) more specifically on the *symptoms* and *etiopathology*, but we should not forget that focusing on symptoms and etiopathology is focusing on something *else* than on the mere differentia between disease kinds and disease, which is a negative cause (of a negative effect *simpliciter*) and a negative effect (of a negative cause *simpliciter*).
Moreover, this two-fold way to define this specific differentia is far from being arbitrary or unjustified. Indeed, in a now common *functionalist* (or, redundantly, causal-role functionalist) vein most notably in philosophy of mind (cf. Levin, 2018 as an entry), one may be tempted to define what diseases are by focusing on what their functions are (or what diseases *do*) in the sense here of what their causal role is; thus, one can dare to define diseases according to what they cause – what their negative effects are -, and also what causes them – what their negative causes are. Hence, we account here for our intuition that a disease should be functionally defined by saying that focusing on the function (as understood here) of a disease is required for defining what, actually, a disease *kind* is.
Another argument in favor of our definition of the differentia for disease kinds is that it allows us to deal with other (said) typical cases of specific disease kinds like communicable vs. non-communicable disease, iatrogenic disease, or idiopathic disease.

[98] Terminological note on "related to a living being": whilst being essential for a better understanding of our definition of a disease kind (cf. Box 4.2), the specification "related to a living being" can be easily left aside in the other contexts, where it is clear and explicit enough that we are talking about causes-effects related somehow to a living being.

Indeed, if they are all really specific disease kinds, then they must be defined as diseases with such-and-such effects and causes: communicable (infectious, or transmissible) vs. non-communicable diseases are diseases whose cause is an infectious agent vs. everything but an infectious agent; iatrogenic diseases are diseases whose causes are indirectly provoked by medical intervention (e.g. maybe post-treatment Lyme disease syndrome); idiopathic diseases are diseases whose etiology is absent[99].

4.1.3 Disease Kinds as Syndromes?

One might eventually think that the best way to define a disease kind is as a *syndrome*. Indeed, if, among specific disease kinds, one finds e.g. Down *syndrome* (under one of its plausible acceptances), then one may argue that a disease kind is a syndrome. But, if a disease were defined as a syndrome, a syndrome would be consequently defined as a disease in conjunction with effects and causes. Thus, a disease kind would be only *indirectly* a disease with effects and causes.

There are two main reasons for avoiding defining disease kinds as syndromes. The first reason is that talking about a disease kind *directly* as a disease in conjunction with effects and causes allows us to clearly notice the embodiment of this definition within our neo-Aristotelian definitional framework (cf. Sec. 3.1.1).

Indeed, if we define a disease kind as a syndrome, then we do not see any more in what sense a disease kind falls under a certain neo-Aristotelian definitional framework.

One could answer, of course, that it merely means that "disease kind" and "syndrome" are not to be defined in terms of one another, but that they are co-intensional; however, there is a second reason for preferring using the word "disease kind" rather than "syndrome" to refer to one and the same thing.

The second reason is that the term "syndrome" is ambiguous, and is *used* ambiguously in the medical literature: it seems to refer to (i) negative effects plus a disease (e.g. Parkinson-plus syndrome) (cf. e.g. Marcovitch, 2010); the term "syndrome" is very likely used as understood under point (i) in the DSM-5 (American Psychiatric Association (APA), 2013, p. 20); indeed,

[99] Caveat on this list of (plausible) disease kinds: iatrogenic diseases are not artefactual kinds, for an intentional agent is never directly (causally) responsible for a certain disease, though an intentional agent can be at the *origin* of the emergence of a certain disease or a certain effect (as a disease); this is not, strictly speaking, the intervention itself, which causes a certain disease – it does so only indirectly. At a more general level, no intentional agent ever really creates a *disease* (*kind*) (properly taken).

> [a] mental disorder is a syndrome characterized by clinically significant disturbance in an individual's cognition, emotion regulation, or behavior [i.e. here specific signs] that reflects a dysfunction in the psychological […] processes […].

In other words, a syndrome is a certain disease (viz. here a psychological malfunctioning process) and negative effects (viz. here psychological disturbances).
"Syndrome" also refers to (ii) negative effects *and* causes plus a disease (e.g. Down syndrome, etc.) – probably the most common use in medicine (cf. e.g. Martin, 2010);
it also refers to (iii) a mere disease (process) (e.g. AIDS vs. HIV-AIDS i.e. HIV infection as a cause of AIDS taken as a specific disease (process) here; or maybe even Stockholm syndrome), or even, *extensionally* defined, to (iv) a set of signs (and/or causes) of any clinical condition (except a disease) (e.g., plausibly, a slightly malfunctioning condition not considered as pathological: e.g. maybe a metabolic syndrome, or, plausibly taken in a literal sense here, Proust syndrome i.e. anything that is likely to trigger a certain memory).
A nice illustration of the ambiguity behind a term like "syndrome" is lactose *intolerance* (or *mal*absorption), which is commonly said to be a specific *syndrome*, but which is described, in the medical literature, as a specific disease kind (National Institute of Diabetes and Digestive and Kidney Diseases, 2020), or sometimes as a specific disease *tout court* – along with other food intolerances generally classified according to their (patho-)mechanism only (Gluckman et al., 2016; cf. also Kurtz & Hemming, 2014).
Only under sense (ii) "disease kind" and "syndrome" refer to one and the same thing.
Thus, the use of the notion of syndrome is to be avoided throughout the dissertation – except in the cases where, so as not to depart too much from the medical literature, the term is maintained (in its plausible usage under senses (i)-(iv) above) in the common denominations of some diseases (e.g. Down syndrome).

4.2 Disease Kinds as Disease with Causes and Effects: Objections and Replies

While Sec. 3.2 addresses objections against the priority of a genus over a species i.e. against our definition of species/kinds, Sec. 4.2 raises and consequently tackles objections against,

more precisely, our definition of disease kinds (cf. Box 4.2), which follows our definition of species/kinds (cf. Box 3.1); these objections are, thus, against the idea that a disease is a constitutive part of a disease kind or that, when embedded into our definitional framework (cf. Sec. 2.1), a disease is an element of the definiens of a disease kind.

Moreover, Sec. 4.2 raises and consequently addresses objections against the second part of the definition of disease kinds viz. the differentia between disease kinds and disease: negative effects and causes related to a living being (cf. Box 4.2).

Sec. 4.2 begins with the objections that can be raised against the necessity of our definition of disease kinds (Sec. 4.2.1-4.2.6), then follows with the objections against the sufficiency condition (Sec. 4.2.7-4.2.11)[100].

4.2.1 Objection (i): Effects or Causes as Unnecessary

[100] Three caveats on the subsequent objections: (i) the objections are free to *plausibly* interpret or understand in a certain way the definition of disease kinds given in Box 4.2: they do not have to subscribe to our neo-Aristotelian framework to be true and *direct objections* against our definition of disease kinds, or even, more generally, against a specific definiendum (e.g. symptoms (cf. Box 5.2), etiopathology (cf. Box 6.2), etc.) defined within our metaphilosophical framework (cf. Ch. 2-3).

(ii) Although our definition of disease kinds is, of course, phrased such that it *correctly* (and *coherently*) follows our own metaphilosophy, the definition of disease kinds *as such* (cf. Box 4.2) can very well fall merely under a *plausible* interpretation (or reading) of the specific metaphilosophical framework into which it is embedded (or, it can be read even *outside*, or *inconsistently* with, our own metaphilosophy), or is *compatible* with many different plausible conceptions of the definiendum in question.

In so far as we strictly distinguish between a *correct* and a *plausible* interpretation of a definition, then the latter can very well be *incoherent* with the correct definitions of other related concepts, while the former shall be *coherent* with these definitions.

With the former, we are proposing what C. D. Broad (1924)) calls "*speculative philosophy*" i.e. the embodiment of specific definitions into a more overarching picture (of reality as a whole) – but without this being a holistic position (cf. Exc. 3.10 on holism).

Due to its very diverse interpretations, we avoid calling this speculative philosophy the "(wide) *reflective equilibrium method*" (taken also outside practical philosophy, of course) (Goodman, 1983; Rawls, 1999). Indeed, the (wide) reflective equilibrium method oscillates between a Rawlsian *coherentist* version (Daniels, 2016), and a Goodmanian *empirical* one (Stich, 1990).

(iii) If *plausible* objections can and are, indeed, raised against our definition of disease kinds (cf. Box 4.2), then this means, along the MCP, that our definition of disease kinds is not completely *irrefutable* (or compatible with *all* plausible conceptions of disease kinds) and must, therefore, address these objections – where "irrefutability" does not mean, of course, "triviality" ("platitude" or "banality") i.e. e.g. "circularity" or "reflexivity", but is closer to the notion of so-called *analytic truths* - to be contrasted with a minimal understanding of (unrevisable or defeasible) apriori(ci)ty (Spohn, 2009) - like "all bodies are extended" (cf. e.g. Boghossian, 1996; Nimtz, 2003; 2009).

Albeit we may rightly feel that correctness is almost *practically* impossible to reach under the present understanding of the notion, this, by no means, is a reason for not *striving* to achieve correctness by e.g. reducing at a minimum the plausibility of different objections against a certain theory through addressing them.

A first objection coming to mind against the necessary conditions of our definition of disease kinds (cf. Box 4.2) is that symptoms or etiopathological agents are not necessary for defining disease kinds.

Indeed, along objection (i), one may argue that a fine-grained differentiation of the causes (or, to the contrary, the effects) of the disease is sufficient for differentiating the disease kind at issue – which would make, thus, a definition of disease kinds either as symptoms *and etiopathology* as unnecessary or as etiopathology *and symptoms* as unnecessary.

Moreover, after all, as we shall shortly see (cf. Ch. 5-6), symptoms and etiopathology are also specific disease *kinds*, in the minimal sense that they are both kinds and something more than a disease (cf. Box 5.2; 6.2). So, why not calling them disease *kinds*? Why reserving the term "disease kind" for as we do (cf. Box 4.2)?

One may add here that either effects or causes are necessary for differentiating disease kinds, for one may argue that specific disease kinds also differ with respect to their disease process, in so far as a disease kind would be a *disease* in conjunction with a differentia. Thus, one may say that it is sufficient to appeal to either symptoms or etiopathology for a definition of disease kinds.

However, it is not difficult to find cases where we would judge that we have *two different* specific disease kinds, while both have the same disease process with the same effects: e.g. hepatitis A and E. If we limited our definition of disease kinds to (here) symptoms (cf. Box 4.2), we would fail to notice that we have actually two different specific disease kinds constituted by two different (plausibly argued) *causes*: hepatitis A virus and hepatitis E virus (as starting *states*).

The prospect for differentiating disease kinds with only etiology and the disease process prima facie seems more promising, in so far as it is able to account for the case of Hepatitis A and E. However, it is also possible to find cases where we would intuitively judge that we have two different specific disease kinds, while both can be described as having the same disease process with the same causes: it is common to describe e.g. Kearns-Sayre syndrome and chronic progressive external ophthalmoplegia as being both constituted by the same etiopathology viz. a cellular energy deficit (which has as an absolute realizer a genetic cause constituted by mtDNA deletions) (Hutchinson, 1879; Kearns, 1965; Kearns & Sayre, 1958), and as having the same disease process viz. a weakening (or attenuation) of extra-ocular muscles; were our definition of disease kinds limited to etiopathology, then we would fail to notice that Kearns-Sayre syndrome and chronic progressive external ophthalmoplegia are usually considered as two different specific disease kinds differing from each other through their symptoms: Kearns-

Sayre syndrome is said to be constituted by the same symptoms as chronic progressive external ophthalmoplegia like ptosis, as well as by extra ones like pigmentary retinopathy and cardiac conduction malfunctionalities (DiMauro & Hirano, 2005; Harvey & Barnett, 1992)[101].

These examples show that a simple analysis of disease kinds as symptoms *or* etiopathology is doomed to failure, but an analysis as symptoms *and* etiopathology is more likely to succeed in differentiating in a sufficient fashion what a disease kind is; having a differentiation of disease kinds as symptoms *and* etiopathology also allows us to distinguish between so-called *variants* (being in the absolute extension) of one and the same disease kind[102].

However, we can further object that, once *all* the effects or causes of the disease discovered and recognized i.e. uncovered or disclosed, we will have differentiated the disease kind in question in a sufficient fashion for excluding other possible disease kinds constituted by (some of) the same symptoms or etiopathological agents (cf. Kendell & Jablensky, 2003 for the case of mental diseases).

In that sense, knowing and recognizing all the precise effects (or causes) of a disease would be enough for differentiating the disease kind at issue; we need not resort to the causes *and* effects of a disease, once all are known and recognized: e.g. a disease kind would be defined in a sufficiently fine-grained manner as a (complex) set of etiopathological agents *or* of symptoms.

But, is it enough? Can we really differentiate the causes (or the effects) of a disease in such a fine-grained way that it differentiates a (highly) specific disease kind, by opposition with another one? That much sounds unlikely, for the more fine-grained the differentiation of the causes (or the effects) of the disease is, the more specific (or fine-grained) the disease kind will also turn out to be: to a specific negative cause and effect of a disease, also a specific disease. Indeed, there is a fair chance that, once we merely differentiate a specific disease kind with

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[101] With Kearns-Sayre syndrome and chronic progressive external ophthalmoplegia, do we really have the same disease process with different effects, or rather different disease processes? Where does the disease process really end? The issue is about how to know that *x* properly belongs to the (end of the) disease process *itself* rather than to the *effect(s) of* the disease process; this well-known problem has already been pointed out by Whitbeck (1977). We may argue that the onus (or burden of proof) is on the medical scientists to *correctly* define the disease kind in question and to tell whether there is some disease kind *x* (partly) constituted by disease *y* and effect *z*, or whether *x* is actually (partly) constituted by disease *y*-*z* and effect *a*.

It is sufficient to show here (in line with our MCP) that Kearns-Sayre syndrome and chronic progressive external ophthalmoplegia can be *plausibly* - without pronouncing on their *correctness* - said to differ here with respect to their symptoms. Note that the same issue arises for the causes of a disease.

[102] Terminological note on "variant" and cognates: as I use the term "variant", "variation" or "variety" (or even "example", "illustration", "exemplar", "archetype" or "specimen") throughout the PhD dissertation i.e. as an absolute/relative realizer: e.g. the notion "specimen" - whose name is explicitly (and etymologically) reminiscent of the notion of (especially biological) *species* - is also used in contexts where we refer to a constitutive *part* of a whole (typically a biological species) i.e. that it is here a relative realizer.

However, the use of "*sample*" (or "portion") as a synonym for "variety" is to be avoided, for, first, it seems to *merely* refer to a relative realizer, and, second, it also seems, actually, extensionally-loaded i.e. that "sample" seems to refer to an *extensional* part of a whole (*plausibly* taken here as a set), albeit the term "sample" is also often interchangeably used with "*example*" (e.g. a writing *sample*).

such-and-such etiopathological agents (or symptoms), the etiopathological agents (or the symptoms) shall be so much fine-grained that they do not capture anymore the targeted disease kind but, rather, an even more specific one.

As an answer to the last point of objection (i), according to which symptoms and etiopathology should be also considered as disease kinds, we, actually, just bite the bullet: symptoms, etiopathology and what we are calling "disease kinds" are all *specific* disease kinds, indeed, in the minimal sense that they are more than a disease and they are kinds. The specific ontological entity we refer to with "disease kind" is to be taken, strictly speaking, as a *specific* disease kind, but, since the other specific disease kinds have their own very distinct name, the specific disease kind we are talking about here i.e a disease plus negative causes *and* effects (cf. Box 4.2) can be simply referred to as "disease kind" *tout court* for sake of textual readability (but on cost of some misleadingness, if there had been no explanation for this) – maybe other (more complete) linguistic systems have a distinct name for this complex entity; be that as it is, the absence of a distinct or proper way to name the entity in question *in a certain language* does not mean, of course, that the entity in question is not a species/kind; it only shows the limitations of the expressive power of a certain language[103].

4.2.2 Objection (ii): Disease Kinds as Diseases/Positive Biological Processes plus Positive/Negative Causes-Effects

A second objection against the sine qua non condition of our definition of disease kinds (cf. Box 4.2) is that it is not necessary to strictly define a disease kind as a disease plus a negative cause-effect. Objection (ii) may be understood in a two-fold way: either (i) a disease kind is to be defined as a *disease* plus a *positive* cause-effect (where *positive* or *negative* causes-effects are *direct* absolute realizers of "cause" and "effect")[104]; or (ii) a disease kind is to be defined as

[103] We could, of course, take a neologism for referring to this specific disease kind. However, finding out a neologism is never an easy task, and there is always the risk of being accused to *invent* (and control) a reality through the invention of a word (cf. e.g. George Orwell (1949)'s famous "Newspeak"), though the primary goal is, to the contrary, to correctly *describe* a certain reality through a word missing in a certain linguistic system i.e. through a neologism.

[104] Note that, in the case where a disease is caused by, and causing, something *positive*, then a functionalist shall take the opportunity to argue here that diseases could be themselves, thus, *positive* – and not specific *negative* kinds (at least, in this case). To the extent that this point is *not* about how to understand interpretation (i) of objection (ii) – indeed, this would be inconsistent with (our reply to) objection (ii), for we assume here that "disease

a *positive* biological process – as a *direct* absolute realizer of "kind" - plus *negative* causes-effects.

Along interpretation (i) of objection (ii), one of the most well-known specific disease kinds used in this context is, likely, sickle-cell anemia as having (or, so it seems) (among others, or at least) a certain *positive* effect viz. - at least, for heterozygotes - a resistance to malaria (Luzzato, 2012). Other examples include a specific disease kind defined here as (primary) polycythemia whose effect (amongst others, or at least) is *positive* viz. an increase in red blood cells (i.e. in endurance capacities, generally speaking) (cf. Dorland, 2011). It is also not rare to find specific disease kinds such that a *cause* (among others) of a disease is plausibly deemed as something *positive*: e.g. acute coronary disease may be typically caused by (among others, or at least) vigorous exertion (Corrado et al., 2006).

Examples falling under interpretation (i) can be easily multiplied, where they are common especially in evolutionary medicine:

> Diseases are not [positive] adaptations shaped by selection. There is nothing useful about pneumonia, schizophrenia, epilepsy, or cancer. Trying to understand diseases as if they are [positive] adaptations is a mistake, one that is unfortunately as common as it is serious. However, many *symptoms* of disease, such as pain, fever, vomiting, cough, and fatigue, are *adaptations*. The systems that regulate such defenses are, for good evolutionary reasons, prone to failures that cause chronic pain, anxiety disorders, and many other diseases. (Nesse, 2016; my emphasis)

In other words, whilst, for Darwinian medicine, disease are not positive, symptoms are positive in the sense that they are adaptations or evolutionarily selected processes.

Indeed, at a more general level, most of evolutionary medicine relies (at least, for (proper) functions) on an (empirical and methodological) weak *adaptationist* approach (cf. Godfrey-Smith, 2001; Lewens, 2004; Méthot, 2015; *pace* Gould & Lewontin, 1979; Lloyd, 2015) i.e., roughly, an approach to evolutionary thinking privileging (both empirically and methodologically) the basic idea that evolution (understood as a positive process here) leads to (positive) *adaptation* through natural selection *and*/*or* other evolutionary forces like higher-level processes (besides the *genetic* level especially) (e.g. on cross-species hybridization, cf. Lewontin & Birch, 1966) or (non-random) mutations (on the so-called *extended evolutionary*

kind" is strictly defined in terms of "disease" plus "negative causes-effects" (cf. Box 4.2) -, it can still be *independently* used, however, as an argument against ascribing necessarily a *negative* value to disease.

synthesis, cf. Laland et al., 2015; Pigliucci & Müller, 2010), but still acknowledging the existence of biological *mal*adaptations[105].

According to this *weak* adaptationist view, diseases are seen, *to the contrary*, as (evolutionary) (biological) *mal*adaptations, but which may be followed by (or lead to) *positive effects* i.e. here effects which have been evolutionarily selected (e.g. through natural selection, or etc.) (on the general idea of (positive) biological functions as (*naturally*) selected effects, cf. Neander, 1991; Wright, 1973)[106]: e.g. senescence is said to be followed by a positive (selected) effect viz. ensuring the steady turnover of the realizers of a biological species, such that to let this species evolve (Nesse, 2005b); or, menopause is said to be followed by the positive (selected) effect - although this is currently an adaptive hypothesis – which is to increase the life (and reproductive) success of the already existing tokens (and of the host) (Williams, 1957).

Along interpretation (ii) of objection (ii), we find plausible illustrations like pre-menstrual syndrome, for it may be plausibly defined as a specific disease kind constituted by specific *negative* causes (e.g. changes in hormone level) and effects (e.g. cramps, bloating, etc.), but

[105] Two caveats on weak adaptationism: (i) I do not define, thus, as is unfortunately commonly done (cf. e.g. Orzack & Sober, 2001), *weak adaptationism* as a theory privileging the *principle of natural selection* over other (acknowledged, however) (positive) evolutionary forces, for weak adaptationism must leave room, according to me, to the idea that *other* evolutionary forces than natural selection (narrowly taken) like higher-level processes may very well also lead, indeed, to (positive) adaptation. In other words, I do not just equate, strictly speaking, *Darwinian medicine* and weak (or strong) *adaptationism* (*pace* e.g. Méthot, 2015).
(ii) Although the notion of adaptation *tout court* is very often (*plausibly*, of course) related to the principle of natural selection (narrowly taken as a positive mechanism) – thus, to the idea that adaptations can be only *positive* -, *mal*adaptations are to be, *strictly* speaking, conceived of, actually, as specific adaptations themselves viz. *negative* ones (on my highly *deflationary* view on adaptation, cf. Exc. 3.7).

[106] Two caveats on strong adaptationism: (i) if diseases are widely acknowledged as (specific) biological *dys*functions i.e. here as maladaptations, along the famous etiological theory of (biological) functions (Neander, 1991; Wright, 1973), cannot they be a *positive* bearer, nevertheless, of a biological function? Indeed, if diseases may be said followed by *positive* selected effects, then it seems that they can truly be positive bearers of biological functions.
However, were this true, then it would lead to *strong* adaptationism: every trait/genetic process (diseases included) would be the positive bearer of a biological function.
But, beyond classical problems with strong adaptationism - among others, the issue of how to explain, thus, the presence of *bad* entities) (on the related *problem of evil* or theodicy, cf. Tooley, 2015) -, the main reason there is for *not* envisaging diseases as positive bearers of biological functions is that the etiological theory of (biological) functions is, actually, not so much about what (biological) functions are than about how to explain, correlatively, the presence of positive *bearers* of (biological) functions themselves; Karen Neander (1991, p. 181) once said: "tumors simply don't have proper functions [i.e. here that they are not positive bearers of biological functions]" – albeit, by mistakenly relying on an evolutionary theory of biological functions and a theory of evolution as a *positive* process only, one generally means by this that tumors do not have functions *at all*.
Talk about diseases as being followed by, or leading to, *positive* (selected) effects may easily mislead us think that diseases are (essentially) positive bearers of biological functions, while they only, actually, (*extrinsically* here) *have* positive (selected) effects – they are not positive bearers of biological functions (along the etiological theory of biological functions, of course).
(ii) One of the main pitfalls to avoid in evolutionary medicine is precisely *strong* adaptationism. It is not rare to find occurrences, where evolutionary (or, more specifically, Darwinian) medical practitioners inconsistently state that diseases are maladaptations *and* are still positive bearers of biological functions (cf. Nesse, 2005a-b; Nesse & Stein, 2012 for a striking illustration (e.g. emotional diseases)).

with here a specific *positive* (or normal) biological process (i.e. indirectly a *positive kind*) viz. a specific - still controversial - phase of the menstrual cycle (Dickerson et al., 2003).

Nonetheless, the main issue with objection (ii) in interpretations (i) as well as (ii) is that it amounts to *category mistakes* (within the same definiens): we claim, with interpretation (i), e.g. that vigorous exertion is here a specific positive cause of acute coronary *disease*; but, we would like to argue that, actually, vigorous exertion is, rather, *properly*, a specific positive cause of a *non-disease* like a reduction of the risk of sudden cardiac arrest (cf. Corrado et al., 2006) i.e. a *positive* cause of (indirectly) a *positive* kind (or, so it seems); ditto for the other examples: senescence as a *negative* kind followed by a *negative* effect like dementia; or, menopause as a *negative* kind followed by a *negative* effect, during the post-menopause, like heart palpitations or urinary incontinence.

With interpretation (ii), we claim that a *positive* (or normal) biological process is here surrounded by *negative* causes-effects, but we would like to argue that, rather, a biological process having a negative cause is to be itself, *properly*, *negative* i.e. a negative biological process (or indirectly a negative kind) with a *negative* cause-effect.

In other words, we can argue that objection (ii) leads to *contrariety* within *one and the same definiens* of a certain definiendum viz. here a disease kind: e.g., along interpretation (i), if disease is (indirectly) a specific *negative* kind, and if a cause-effect may be here also (at the same indirectness level) a specific *positive* kind, then they are two (indirect) contraries (of a kind).

Ditto for interpretation (ii): if disease is (indirectly) a specific *positive* kind, and if a cause-effect may be here also (at the same indirectness level) a specific *negative* kind, then they are both (indirectly) contraries (of a kind) (cf. Sec. 5.3.2 for a related point).

However, as a last defense of objection (ii), we may want to argue that a way out of these contrarieties, or at least a way to dwindle them, would be to admit degrees of badness and goodness to a certain disease kind: e.g. having *a little bit* some stress – but not too much – has some positive effect viz. that it motivates one to act or increase concentration (*pace* Engel, 2019).

Nevertheless, first, however weakened, the contrarieties remain (a bit) present. Secondly, we can answer that what *essentially* motivates one to act or increase one's concentration is not e.g. some minimal stress – which, even made minimal, remains (a little bit) *bad* -, but, to the contrary, some, however soft or strong, *positive* (mental) state/process.

To summarize, the same specific kind (e.g. vigorous exertion) cannot (essentially) have (however plausibly understood) e.g. a *positive final* value, whilst also at the same time a

negative instrumental (or, more generally, extrinsic) value – and vice versa -, for this leads to contrariety within one and the same definiens (cf. Exc. 3.9 for this axiological terminology).

Second, one can argue that objection (ii) (weak adaptationism, especially) is, actually, more *psychologically* than, strictly speaking, *philosophically* motivated, or is grounded on a certain psychology (cf. Pinker, 2018) – instead of a philosophy[107]: *optimistic* people often endeavor to *unilaterally* look for the positive behind evil/bad act(ion)s (on the Panglossian paradigm, cf. Leibniz, 1710)[108], or they think that their misery or pain can eventually lead to a certain advantage or superiority (or, *teaches* them): e.g. patience, resignation, humility, etc. (*pace* Ogien, 2017); or that stress helps boost physical productivity and mental concentration; or, more generally, negative emotions help cope (better) with, adapt to, a certain situation or the environment; or even those people are - more psychologically than philosophically - convinced that (knowledge of) badness leads to (knowledge of) what goodness is[109].

This psychology is often summarized by the popular Nietzschean (1889, Sprüche und Pfeile) motto: "what does not kill me makes me stronger" ["Was mich nicht umbringt, macht mich starker"].

However, this psychology is, in turn, certainly (*philosophically*) highly indirectly if not fundamentally grounded on a certain (mis)understanding of the famous but misnamed *principle*

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[107] Note that our essentialism can be also accused of being "[…] mandated by *psychological* dispositions […]" (Wilkins, 2013, p. 414; my emphasis) i.e. that essentialism would be psychologically necessary for e.g. neatly categorizing entities.
Nevertheless, even if this may be true, it remains the case that our defense of essentialism is (also), first and foremost, *philosophical* (cf. Ch. 2-3).

[108] From this, it is not hard to get to the point that *any* negative thing can be seen under the Panglossian lens; at a more general level, the Panglossian trend is so much widespread and psychologically-well entrenched that, in addition to its interpretation within *evolutionary* biology, it pervades many other areas of (especially) *philosophy* like the philosophy of (negative) emotions (or moods) or, more generally, practical philosophy (or, the biological sciences, more generally): e.g. shame as having a good/positive *instrumental value* viz. by promoting one's reflection on one's own values and showing one's attachment to them (Deonna et al., 2011); or, anxiety by aiding one's social interactions and improving one's own deliberation and decision process (Kurth, 2018).

[109] Indeed, it is not rare to find epistemological arguments in favor of the idea that e.g. a *dys*functional process (or, a dysfunction *tout court*) can lead to *knowledge* of a functional process, especially in the contemporary debate about causation, where causation is sometimes analyzed through the rough idea of the absence of a disruption between a cause and an effect, or of a difference in the effect (where this disruption or difference is generally provoked or made by (human) *interventions* on the cause) (on, relatedly, so-called manipulationist accounts of causation, cf., as entries, Hagmayer et al., 2007; Woodward, 2016; also Ch. 6 about causation).
If e.g. health is the absence of disease (Boorse, 1977) and can be plausibly deemed an effect of this definiens, then one easily notices that, indeed, (knowledge of) badness (generally speaking) leads to (knowledge of) goodness, for badness would be an element of the definiens of goodness. I acknowledge Kevin Mulligan (personal communication, August 2017; cf. also Engel, 2019) for having pinpointed this to me.
Of course, that much seems to rest on an (unfortunately mistaken) analysis of e.g. functions, causation, health or goodness as, respectively, (*plausibly* speaking) absence of dysfunctions, disruption, disease or badness, while we would like to (more) intuitively sustain that, reversely, negative entities like *dis*ease essentially depend (even as sui generis entities) on positive ones like health – unless one holds a stricter or even complete separation between ontological and epistemological issues.
Indeed, one would not intuitively say that e.g. *bad* people are essentially parts of what *good* people are, that suffering is essential to what it is *not* to suffer, or even that sadness is essential to empathy.

of sufficient reason, according to which, roughly, things are out there for a (good) reason (on the compatibility between the principle and essentialism(s), cf. Raven, 2020; on a constitutivist approach to the principle, Dasgupta, 2016) – which should be probably called, more properly, "principle of sufficient *explanation*"; along this (especially Spinozist) (mis)understanding of the principle of sufficient reason,

> [s]ince existing is something positive, we cannot say that it has nothing as its cause […]. Therefore, we must assign some *positive* cause, or reason, why [a thing] exists—either an external one, i.e., one outside the thing itself, or an internal one, one comprehended in the nature and definition of the existing thing itself. (Spinoza, I/158/4–9; my emphasis; tr. Curley, 1985-2016)

In other words, to look for the good behind bad existent things is, along the above (mis)understanding of the principle of sufficient reason, normal, for existence is something positive.

However, if negativity cannot be mixed up with positivity (at least, *within one and the same definiens* of a certain definiendum viz. a *process*, for a *process* itself is a substantial *change* from positive/negative/value-free *states* to contradictory negative/positive/value-free *states* (cf. Box 9.2)), how to account for the above intuitive examples?

We can offer the following alternative solution based on the principle that a process *simultaneously implies* – which may be *plausibly* (though not correctly) interpreted as a cause-effect relationship - the generation/destruction of a (*sub*)*contrary* state of (the contradictory state) (¬)x (= (¬)y) until its own contradictory state (¬)y by generating/destroying (more and more) the essence of state (¬)y (on the neo-Aristotelian WCP, cf. Exc. 2.3): e.g. if sickle-cell anemia or pre-menstrual syndrome is a specific disease kind, then this *negative* process (i.e. from x to ¬x) concurrently *implies* a positive process (and *vice versa* – against weak adaptationism) consisting in the generation of a contrary state of x viz. y (i.e. from ¬y to y), where e.g. resistance to malaria or a certain phase of the menstrual cycle may be said a constitutive part of this *positive* process – and not of the negative process in question.

More generally speaking, with the WCP, we are used to claim (somewhat misleadingly) that e.g. – almost proverbially - a *half full* glass (of water) is also, at the same time, *half empty* (but taken as a *contrary* here). Mixing up negativity with positivity within *one and the same* definiens of a certain definiendum (viz. a disease kind), as objection (ii) does, is, thus, confusing what a process *is* with what a process (as defined) *implies*.

4.2.3 Objection (iii): Asymptomatic and Idiopathic Disease Kinds

Objection (iii) against the necessity condition of the definition of disease kinds (cf. Box 4.2) directed against symptoms and etiopathology is that there are specific disease kinds like (or, so it seems) hypertension or diabetes that can be defined as *asymptomatic* (or, as not being constituted by (a set of) symptoms) (Reaven, 1976), and specific disease kinds like (or, so it seems) ankylosing spondylitis that are deemed *idiopathic* i.e. whose etiopathology is absent (Kuperus et al., 2018; for the distinction between the (negative) causes-effects and the constituents of a disease, Sec. 4.2.10).

There are two ways to address objection (iii): the first way is, in light of the objection that such disease kinds are asymptomatic or idiopathic, to simply reassess them as diseases *simpliciter* – and no longer as disease kinds: an asymptomatic and/or idiopathic disease just is a disease; and this is not a problem, since we have said that, in our essentialist jargon, there is e.g. no disease *kind* without a disease, but not (essentially) vice versa (cf. Sec. 3.1.1).

Nevertheless, it still remains true that, as a specific metaphysical constraint, there is no disease without e.g. a disease kind (cf. Sec. 3.3.1) or, minimally, a symptom or etiopathology (cf. Ch. 5-6). That is why, a second way to address objection (ii) is needed.

The second way begins with the following question: are there really specific disease kinds that show no symptoms at all, or that have no etiopathology? Of course, the answer to this question depends on how we precisely define what symptoms (cf. Ch. 5) and etiopathology (cf. Ch. 6) are.

But, however precisely a symptom or etiopathology is defined, if we hold the idea that the *recognition* of the (negative) effects and causes of a disease is commonly part of an attempt at recognizing a disease (cf. Sec. 4.2.7), then, in this context, “asymptomatic” and “idiopathic” do not refer to the absence of effects and causes of a disease, but to the incapacity to recognize the effects and causes of a disease, thus, to an *epistemic* failure (or fault) of recognizing a disease kind.

Nevertheless, how can we *know* that a disease has still effects and causes, if we are unable (at time *t*) to *recognize* them as *effects* and *causes* of a disease? This question is asked in the name of a(n) (strong) *internalist* conception of knowledge, according to which for *S* to know that *p*

(*Kp*) is for *S* to know that it knows that *p* (*KKp*), or what *S* does not *reflexively* know is what is unbeknownst to *S* (on the so-called *KK*-principle, cf. Ramachandran, 2012).

A solution here is to adopt an *externalist* conception of knowledge (Williamson, 2000) or a *weak* internalist one (Engel, 2007), according to which, in our case, it is not necessary to (reflexively) *recognize x* as a symptom or as an etiopathological agent for knowing that *x* is a symptom or an etiopathological agent, or that it is merely possible to recognize *x*.

Thus, one may argue that, on an externalist or weak internalist reading, "asymptomatic" and "idiopathic" mean that there is, actually, a failure of *internalizing* the knowledge that *x* is a symptom or an etiopathological agent.

A very recent illustration of externalism or weak internalism about knowledge in the context of disease kinds is likely the so-called "disease (kind) X", which has been claimed by the WHO as the next *potential* epidemics (on the same line as e.g. Ebola or Zika) (WHO, 2018b). Talk about disease (kind) X can be interpreted as about the fact that we *know* that disease (kind) X exists (or, shall exist sooner or later at time t_{+1}), but we are unable to *recognize* it (cf. also Sec. 10.1.7)[110].

4.2.4 Objection (iv): Disease Kinds as Diseases plus Simultaneous Processes

A fourth objection that we can easily raise against the necessity condition of our definition of disease kinds (cf. Box 4.2) is a very common and intuitive one: objection (iv) consists in saying that a disease kind is not a disease plus negative causes-effects, but a disease plus another simultaneous process (if a disease is a specific process), where these simultaneous processes "[…] are not properly understood as effects of the disease process but states of affairs that occur within it" (Whitbeck, 1977, p. 634) i.e. as, indeed, processes concomitant with the disease process.

[110] Caveat on disease (kind) X: some may want to argue that, contrarily, disease (kind) X just is a specific scientific thought experiment i.e. – at least, for right now at time *t* – a merely *imaginary* disease (cf. Sec. 10.1.6 on that). However, again (cf. Sec. 3.2.6), a weak (or light) conception of what it is for a disease (kind) to *exist* can be put forward: talk about disease (kind) X is not to be interpreted, thus, as talk about how to model an *imaginary* (*fictional*, or *mind-dependently existent*) target, but only a *non-recognized* (or non-differentiated) target (assessed at time *t*) (for a typical confusion between *imaginary* and *non-recognized* diseases, cf. e.g. Autret, 2016).

However intuitive it may be to think that a disease kind is a disease giving rise at the same time to another *exactly concurrent* (or *concomitant*) process (as Whitbeck (1977) seems to take it), it is hard to see what this really means[111].

Indeed, on the one hand, if the simultaneous process is understood as a specific kind whose "disease" is a specific absolute realizer – indeed, if a certain process *exactly* co-occurs with a disease, and we claim that there is a relationship between them, then this relationship is likely absolute extensionality -, then then how can such an account deal with the case of a certain *highest*-level disease (e.g. irrationality), where no simultaneous process (as understood here) is guaranteed (or warranted), so that they form a specific disease kind? Indeed, the absolute extensionality requirement (for kinds) does not say that from the presence of an absolute realizer there is necessarily an absolute realized property, but the other way round (cf. Sec. 3.3.1).

On the other hand, if the simultaneous process in question is understood, to the contrary, as a specific absolute realizer of "disease", then objection (iv) amounts to the thesis that a disease kind is a disease plus one of its absolute realizers, for a disease *necessarily* has an absolute extension.

We may argue that one of the problems with this interesting proposal is that, with objection (iv), a disease kind could not be defined within our neo-Aristotelian framework (cf. Sec. 3.1.1) any more, for, with absolute extensionality, a disease kind would be defined as a disease (i.e. the genus) plus a kind which absolutely applies the definition, or rather the intension of a disease (i.e. the differentia).

However, along objection (iv), we can add here that this last reply to objection (iv) is not a *direct* reply.

To directly address objection (iv), we can stay content by arguing that, if "simultaneous process" is an *absolute* realizer of "disease", then there is no definitional relationship between a disease kind and a simultaneous process.

4.2.5 Objection (v): Disease Kinds as Kinds (with a Differentia)

[111] Note, however, that Whitbeck (1977; cf. also Fuller, 2018a) understands these simultaneous processes as, more precisely, (clinical) *manifestations* of the disease. However, this is to concede too much, for who talks about (clinical) manifestations of a disease talks about diseases as *dispositions*. The vaguer notion of a simultaneous process here leaves open the question of whether a disease is really to be considered a disposition (for the association between these simultaneous processes (understood as manifestations) and symptoms, cf. Sec. 5.3.3).

A fifth objection against the necessity condition is the following one: if we say that a species is constituted by a genus in conjunction with a differentia (cf. Box 3.1), how can we know among the constituents which one is the genus and which one is the differentia? In other words, why not defining, after all, a disease kind as a *kind* (i.e. the genus) in conjunction with a certain differentia – rather than as a disease (i.e. the genus) in conjunction with a certain differentia (cf. Sec. 3.1.1)? Likewise, as already seen in the context of absolute extensionality (cf. Sec. 3.1.2), is e.g. lung cancer a cancer or a lung disease?

So as to not trivially define a disease kind, it cannot be defined as a kind in conjunction with a disease (cf. Sec. 4.1.1), but with another differentia; and nothing precludes to give the same differentia (causes and effects) as for our own definition of disease kinds (cf. Box 4.2); as such, objection (v) does not go against our differentia.

So, to strengthen objection (v) we need to be more precise about the differentia for disease kinds: let us define, thus, a disease kind as a kind in conjunction with a certain biological dysfunction or an abnormal (anomalous or incorrect) process.

The reply to objection (v) is that we fail to see in what sense a disease kind can be really (defined as) some*thing more* than a kind viz. a kind *plus* a (certain) biological dysfunction or abnormal process[112].

[112] Two caveats here on objection (v): (i) why not defining, after all, a disease kind as a negative cause-effect relationship (i.e. the genus) plus a disease (i.e. the differentia)?

The main problem with this interesting proposal is that it does not do justice to the intuitive idea of what it is for a disease kind to be a *kind of disease*: e.g. a human being is intuitively said a specific kind *of* animal, where the emphasis is clearly put here on the specific genus (as a constitutive part) of a human being viz. animality, albeit rationality is also, of course, a constitutive part of a human being.

In other words, the *kind* human being is constituted by (or made *of*) an animal as its genus. By reasoning by analogy, a disease kind, as a kind *of* disease by its very name, is intuitively a *kind* constituted by (or made *of*) disease as its genus, or, if a disease kind is a specific kind/species *tout court*, the very word itself indicates that there is a *constitutive relationship* between a disease kind (i.e. the species) and a disease, where disease intuitively is the genus in this case

Moreover, we can argue that e.g. "seated Socrates" or "white horse" intuitively refers to the sitting position *of Socrates* or the whiteness *of a horse*, thus here to a *specific Socrates* (rather than – at least, more *directly* than - a specific sitting position viz. the one of Socrates) or to a specific *horse* (rather than to a specific whiteness viz. the one of a horse).

Indeed, we are truly talking about – at least, more directly - here a seated *Socrates* or a white *horse* – and not (or, less directly) about a "Socratized sitting position" or a "horsed whiteness". Along the same line, "kind *of disease*" intuitively refers – not here to a specific disease -, but to a *disease* plus something else (a differentia) (for a further reply to this objection, cf. Sec. 3.3.2).

This answer also excludes, therefore, any strict definition of disease kinds, where the elements of the definiens in question are both the *definition* of disease (i.e. the genus) - and not disease itself - and the *definition* of kinds (i.e. the differentia); moreover, such an attempt also commits the mistake of taking a *single* thing i.e. a whole for two independent entities on basis of the fact that two terms viz. "disease" and "kind" are used to name this whole.

(ii) From this last attempt, we may wonder: why not defining, then, a disease kind as a disease (i.e. the genus) plus the definition of a kind (i.e. the differentia)? Again, the same answer applies: e.g. a rational animal is not to be *correctly* defined as an animal (i.e. the genus) plus the *correct* (i.e. within (our understanding of) a genus-species hierarchy) definition of rationality (i.e. the differentia), for this would amount to just taking rationality as an

But, should not "kind" have a (at least direct) relative extension itself like for "disease"? There is (or exists) nothing more – and else (at the same level) – than a kind i.e., in our vein, no super/sub(natural) kind.

Indeed, the term "kind" should be considered fundamental or general (like for "type"), and, thus, an exception to the requirement that any term necessarily has a relative extensionality (cf. Sec. 3.3.1), on pain of inconsistency, for the notion of relative extensionality precisely makes sense *within* a certain intensional framework like (our understanding of) a genus-*species* hierarchy; had "kind" a (direct) relative extension, then there would exist a *kind strictly* defined as a kind *plus* a differentia, whilst absolutely being a *kind* itself – which is meaningless.

Of course, that much does not mean that the constitutive parts of a disease kind – as being specific wholes themselves – do not absolutely fall under "kind". If a disease kind is a disease (negatively) caused by and causing something (cf. Box 4.2), then it can be truly said something more than a *disease* or a negative *cause-effect*, but, if "disease" indirectly absolutely falls under "kind" itself, does it mean that a disease kind is *indirectly* something more than a kind? No, for this would amount to mix up levels of relative extension with levels of absolute extension.

Nevertheless, objection (v) can eventually reply that there plausibly exists, actually, something more than a kind, for this *thing* need not fall, after all, under a genus-species hierarchy (as understood here). Of course, a disease kind can be said more than a *kind* (e.g. an entity or thing), in the same way as a disease kind is said something more than a *disease* (or a negative *cause-effect*.

The main issue with this last defense of objection (v) is that, even if we grant that there plausibly exists something more than a *kind* (however understood), it still remains true that a definition of, strictly, a disease *kind*, although it is something more than a disease (or, a negative cause-effect) (cf. Box 4.2), is still a specific kind, exactly like a disease *type* is not something more than a type.

But, if we truly think that a disease kind can be something more than a kind, then we would not call it properly "disease kind", but e.g. "disease *entity*". In other words, we can argue that, by sustaining that a disease kind is something more than a kind, objection (v) can be accused of just shifting the definiendum in question (i.e. disease *kind*) for, actually, another one.

independent whole itself – which can be done, of course -, but, in the present context, it is still not an animal *and* rationality which are to be defined, but a *rational animal* (taken as a *single* whole itself). Ditto for a disease kind.

4.2.6 Objection (vi): A Disease Kind as an Internal Relationship between Disease with Causes-Effects and Disease

A sixth and last objection against the necessity condition of our definition of disease kinds (cf. Box 4.2) is that a disease kind should not be considered as a thing in its own right, but *being a kind of* (*y*) is a certain *relationship* obtaining between two items *x* and *y*: e.g. a disease with a cause and an effect enters into a relationship *being a kind of* with a disease, but there is no such weird thing as a disease kind – only a disease with its causes-effects and a disease.
Furthermore, along objection (vi), the relationship *being a kind of* (*y*) is, actually, *internal* (vs. external), where an internal relation is, as commonly interpreted, a relation implied by the nature of the relata: e.g. the relationship of being greater than (*y*) is implied by the presence of two entities viz. e.g. the number 1 and 2, or other (said) formal/material ontological relations like the relationship of essential dependence or maybe absolute extensionality (Hakkarainen et al., 2018; Keinänen & Tahko, 2019) – whilst this is not the case for *external* relations, as commonly understood: e.g. (*x*) *causing*, or *being caused by*, a disease (on the debate about internal vs. external relations, especially about the eliminability of one of the two relations or about eliminativism about internal relations, cf. Johansson, 2011; Moore, 1919; Mulligan, 1998; Russell, 1912; von Wachter, 1998).

> It is [also] a shared, distinguishing feature of internal relations [but not uncontroversial] that they are not entities in addition to their existing relata. They are internal to their relata in the sense of being relatednesses of entities without there being anything that relates these entities. (Hakkarainen et al., 2018, p. 97)

In other words, internal relations are not sui generis entities. In that sense, along objection (vi), the relation *being a kind of* (*y*) is, actually, not a genuine relationship, for it is arguably implied by the presence of the relata viz., in our case, a disease with its causes-effects and a disease.
Objection (vi), thus, puts the emphasis on the "*mode*" or "attitude" (so to say) obtaining between a disease with causes and effects and a disease (e.g. a human being *is a kind of* animal), while our own theory puts the emphasis on the "*content*" (so to say) by allowing that there is such a thing as a disease kind (cf. Sec. 4.1.1) (e.g. a *human being* is a *kind of animal*).

But, if our theory makes the relationship between a disease kind and a disease easier to capture through extending our ontology to disease kinds, objection (vi) asserts that, more widely speaking, there are no such things as e.g. an animal kind, but only e.g. an animal.

A first reply to objection (vi) is that, even if we grant that there are no disease kinds, it remains true that the relationship *being a kind of* (*y*) still itself arguably *exists* (as a relationship) out there in the world – even taken as an internal relation.

Indeed, it is not at all clear that a (said) internal relationship like *being greater than* (*y*), where the relata are e.g. the numbers 2 and 3 cannot be just taken as e.g. a genuine *intrinsic* (accidental or essential) property of the relatum number 2 (on the rejection of the association between extrinsicality and relatedness, cf. Exc. 3.9), or maybe even as, minimally, *ontological free lunches* i.e. (derivative) existent entities, which do not add anything to being (however understood) (Armstrong, 1997).

Following this line of thought, an *internal* relation, properly taken, could be perhaps a specific *intrinsic* property, while an *external* relation could be a certain *extrinsic* property; *being a kind of* (*y*) e.g. animal can very well be deemed, thus, a certain property of e.g. a human being.

To sum up, for objection (vi) not to allow disease kinds in an ontology, it implies, nevertheless, the admission of the relationship *being a kind of* (*y*); and we do not necessarily win at the exchange of the former ontology in favor of the latter one.

A second reply to objection (vi) – directly related to the first reply – is that, granted that *being a kind of* (*y*) is a(n internal) relationship, it obtains between e.g. a symptom and a disease (cf. Ch. 5).

However, this may mean that one's ontology incorporates a *single* (*relational*) entity called "being(-)a(-)kind(-)of(-*y*)", but such an entity could seem as strange to incorporate in one's ontology as the one rejected in such an ontology viz. a disease kind. Thus, an ontology or *Weltanschauung* willing of getting rid of strange entities like disease kinds can be accused of having to incorporate, eventually, other as much bizarre ones.

A third - also related - reply to objection (vi) is that, by saying that e.g. a human being enters into the relationship *being a kind of* with an animal, we are still saying (in a literal and genuine way) that a human being *is* a kind of animal, and the predicate "_is_" is usually said to convey existential (though not necessarily *essential*) import (or, to be *ontologically committing*) (on this notion, cf. Bricker, 2014).

A fourth reply to objection (vi) is that, if *being a kind of* (*y*) were a(n internal) relationship, how to further analyze this relationship between e.g. a human being and an animal: would a human being be *essentially* a kind of animal (which is rational), but not an animal *tout court* (which is

rational)? By allowing animal kinds to enter into an ontology, we solve this issue: a human being is (in the absolute extension of) an animal kind, whilst a human being is (essentially) a rational animal *simpliciter*.

A fifth and last reply to objection (vi) is to argue that, generally speaking, *relations* are, actually, (intensionally) reducible to specific *complex kinds* called with - at least - two terms viz. a joining term (e.g. "kind (of)") and (a) relatum/relata term(s) (e.g. "disease"). Thus, along this line, "disease kind" (or "*kind of* disease") can very well refer to, actually, a certain relation (but, against the reduction of relations to relational *properties*, cf. Mulligan, 1998; 2008; on the complex history behind the relationship between relations and properties, Brower, 2018; also Marmodoro & Yates, 2016).

4.2.7 Objection (vii): Disease as Negative Effects and Causes

A seventh objection that can be raised this time against the sufficiency (or completeness) of our definition of disease kinds (cf. Box 4.2) is, along a functionalist line, that negative effects and causes of a disease are worthwhile for analyzing what DISEASE is – not what DISEASE KIND is, for it is common to define e.g. "pain" *tout court* – and not "pain kind" - in terms of the mental state (or process) (negatively) caused or triggered by a bodily injury, and itself causing other mental states (or processes) like the desire of not being in this very mental state (or process) any more[113].

Actually, this functionalist approach is omnipresent in recent and classical literature on disease. Indeed, some philosophers of medicine propose theories of disease based on the idea that

[113] Two caveats about objection vii: (i) as such, objection (vii) is against the idea that our definiens for disease kinds (cf. Box 4.2) is sufficient for defining what disease kinds are, by arguing that it seems to define, contrariwise, what disease *tout court* is.
Thus, objection (vii) does *not* say that an analysis of disease should be done through an analysis of disease kinds; were this true, it would just collapse into the objections raised against our analysis of the genus-species relationship (cf. Sec. 3.2).
In a nutshell, objection (vii) merely says that the proposed definiens works for the definition of disease *tout court* – and not for a definition of disease kinds (cf. Box 4.2).
(ii) Moreover, it is important not to confound between the use of functionalism as an objection against my constituent ontology (cf. Ch. 2) and the use of functionalism as an objection against our definition of disease kinds (cf. Box 4.2) (as it is the case with objection (vii)).
In other words, the scope of the functionalist approach is here limited to the very case of disease (for the sake of objection (vii)); it is not meant to be a general approach to conceptual analysis (vs. a constitutive one). Thus, objection (vii) is also to be answered at the same level – thus, not by replying that objection (vii) just goes against our neo-Aristotelian framework.

analyzing what disease is is nothing else than analyzing what its causes and effects are (cf. e.g. Darrason, 2014 for a focus on what she calls the genetic causes of disease; Fuller, 2017; Smart, 2016; also, relatedly, Vosgerau & Soom, 2018 for a causal power or dispositional account of disease; Thagard, 1998 for a causal network account; on a general account, Danner Clouser et al., 1981).

This tendency to differentiate a disease - not through its (internal) *constitution* (i.e. by *black-boxing* it, or making it *irrelevant* for defining (or, alien to define) disease) -, but *functionally* i.e. here through its negative causes (and effects) is historically well entrenched especially in the (intensionally, here) reductionist discourse (cf. Thagard, 1999), where diseases have been said (in the 1960s) reducible to so-called *molecular* diseases (e.g. sickle cell anemia historically described as a disease *caused by* a malfunctioning hemoglobin molecule) (Pauling et al., 1949), or (more recently) to so-called *genetic* diseases (e.g. sickle cell anemia historically described as a disease *caused by* a bad genetic mutation situated on chromosome 11) (cf. Strasser, 1999; Strasser & Fantini, 1998; on emotions as functional states, Adolphs & Andler, 2018).

Another illustration of the idea that disease is to be functionally defined originates from contemporary epistemology of medicine, where medical knowledge is commonly said to be a specific *causal* knowledge viz. knowledge of disease (and health) through its causes (Sadegh-Zadeh, 2015; Solomon, 2015; cf. also Campaner, 2012; Cartwright, 2002; Lemoine, 2017).

Following this claim, one of the most well-known and discussed naturalized (or applied) theories of (the nature of) *causation* (in medicine) is the so-called Epistemic Theory of Causality (ETC) (Russo & Williamson, 2007; 2011; Williamson, 2013; also Broadbent, 2011), which is that

> (ETC) causation is the justified belief that *z* is a cause of an entity,

where, in the more specific context of medical causation,

> (RWT) the belief that *z* is a negative cause of disease is justified by (i) a statistical correlation between a disease and its cause, and (ii) a mechanism linking the cause to the disease;

points (i) and (ii) constitute the Russo-Williamson Thesis (RWT) (for discussions, cf. Illari, 2011; Wilde & Parkkinen, 2019; Williamson, 2019a; for criticisms, Canali, 2019), which can be taken as a specification of the ETC.

Thoroughly analyzing point (i) is the focus of a medical methodology widely known as Evidence-Based Medicine (EBM) (as entries, cf. Ashcroft, 2004; Howick, 2011; Parkkinen et al., 2018; for applications to pharmacology, Landes et al., 2018; for criticisms on EBM, Cartwright & Hardie, 2012; on medical nihilism (or rather, skepticism), Stegenga, 2018b).

For EBM, statistical correlations between a cause and its effect (i.e. a disease here) are established (mostly) through interventions done in RCTs[114], where one maskedly (or blindly) tests the efficacy of a therapeutic treatment on diseased individuals by preventing (with a therapeutic treatment) the disease at stake to occur through preventing its cause to occur (e.g. removing the headache of individuals through aspirin), and by averaging over the results gathered through the tested individuals to get population-based results (Reiss & Ankeny, 2016; on the famous internal and external validity problems in the epistemology of clinical RCTs, cf. Pellet, mss.b)[115].

The importance of point (ii) has been emphasized especially by the RWT (Clarke et al., 2013), where a mechanism joining a cause to a disease is discovered through clinical studies (e.g. Randomized Controlled Trials (RCTs), or maybe even observational studies) (Williamson, 2019a).

This detour toward contemporary epistemology of medicine shows us that, especially with point (ii), the RWT is based on a definition of disease as the *effect* of a mechanism (cf. Fuller, 2018b): e.g. the evidence to believe that lung cancer is malfunctioning growth of the cells in the lung's tissues lies in the causes (e.g. smoke inhalation, etc.) further analyzed (partly) mechanistically by the RWT.

Objection (vii) may be addressed as follows: first, one may suspect that what many functionalists have in mind when they talk about causes and effects is, actually, exactly what we are talking about with our notion of constitution (cf. Ch. 2). This is due, of course, to the high ambiguity behind a term like "causation" (and "constitution" too). In that sense, such (tentatively speaking) *vertical functionalists* are, actually, just proponents of constitutive explanations.

[114] We find in the literature two interpretations of what the relata of the causal relationship established by a clinical RCT are: (i) the relata are a *disease* and its *cause* (Williamson, 2019a); (ii) the relata are a *medical treatment* and its *outcome* (Thompson, 2011). If an interventionist theory of causation is held in this case here (Woodward, 2003), then interpretation (ii) just is a way to establish the truth of interpretation (i) (cf. Boniolo & Campaner, 2019).

[115] Terminological note on the notion of population: in its *strict* usage, I follow here the mainstream view about the notion of population by distinguishing it (like e.g. in population genetics or sociobiology) from, especially, the notion of (biological) species (or organism). I take "population" to refer, at the general level, when properly used, to a certain set, group or total viz. a (concrete) set of concrete elements viz. (indirectly) living beings located in a geographically restricted area: e.g. Geneva population, a population of birds, a citizen population, etc.
Of course, the notion of population can still be used under its *plausible* meaning throughout the PhD dissertation.

Second, to be more charitable with functionalism, one can argue that a functionalist theory of *disease* exclusively focuses, actually, on the *epistemology* of disease (for the link between disease kinds and epistemology, cf. Nordenfelt, 2013a; for the link between causation and disease kinds, Whitbeck, 1977) i.e. more precisely here on the differentiation (or recognition) of a disease through the differentiation (or recognition) of the causes and effects of a disease, or mixes up *ontological* theories of disease with its *epistemology*, for functionalists often mean to provide, rather, theories of (the *nature* of) certain processes or states.

Indeed, one can argue that the most common way for medical scientists to *recognize* or *differentiate* a disease consists not in the direct differentiation (or recognition) of the disease (process), but in *inferring* the disease (process) through what medical scientists are directly able to differentiate (or recognize) i.e. most often the causes and effects of the disease[116].

In other words, one can argue that recognizing (or differentiating) a disease mostly goes through recognizing (or differentiating) a disease *kind*, since a disease kind is a disease with negative effects and causes related to a living being (cf. Box 4.2); from the partial differentiation (or recognition) of a specific disease kind i.e. from the differentiation (or recognition) of such-and-

[116] Three caveats here on the epistemology of disease: (i) of course, it does not happen like that in every clinical circumstance: e.g. in the (clinical) circumstance where the medical scientist has tried to differentiate (or recognize) specific symptoms (e.g. renal colic), but is finally able to directly differentiate (or recognize) the specific disease (process) (e.g. kidney stone disease differentiated (or recognized) through fMRI), the preliminary (clinical) attempt at differentiating (or recognizing) the effects of the disease in question shall appear, thus, as pointless, once the disease (process) at issue has been directly differentiated (or recognized).
Directly being able to recognize (or differentiate) – nicely called "*direct localization*" by Bechtel and Richardson (2010) - a disease without passing through the differentiation (or recognition) of a disease kind (i.e. through the effects and causes of the disease) can be, thus, seen as an ideal in clinical medicine
(ii) One could well reply here that the differentiation (or recognition) of, especially, the *causes* of a disease would be useful for preventing the disease to occur by e.g. eliminating the causes in question (typically in a clinical RCT), thus, that the recognition of the causes of a disease is not idle (cf. e.g. Whitbeck, 1977).
However, one should separate the question of how to best *treat* a disease that is ipso facto present from the question of how to best *prevent* a disease to occur. Likewise, crime scene investigation is far different from crime prevention. Even if one maintains that it is always better to be able to prevent the occurrence of the disease than to treat the disease (process) itself, exactly like one is used to colloquially tell "Prevention is the best medicine", these two relations should be kept separate.
Moreover, (iii) one has to be careful in not confusing between (i) prevention of a disease to exist through prevention of its negative *causes* to occur and (ii) prevention of a disease to exist through prevention of its *constituents* to occur (cf. Sec. 2.1).
Strictly speaking, with point (i), preventing the negative cause of a disease to exist does *not* prevent *specifically* a disease to occur or to exist, but *any* specific effect followed by the negative cause, since one has argued that the intuitive idea that there is no disease without e.g. a disease kind is not about the definition (or, about the *essence*) of a disease as a disease kind (cf. Sec. 3.3.1), and that the differentia for disease kinds is defined independently from what the genus in question is (cf. Box 4.2), and that a disease is only *indirectly* (in the absolute extension of) a(n) (negative) effect/cause (cf. Sec. 4.2.10).
In other words, preventing a negative cause/effect of a disease (or, preventing a negative cause or effect whose effect or cause is specifically a disease) to occur is preventing *any* negative effect/cause of *this* cause/effect.
Rather, when one speaks about preventing a disease to occur, one seems to refer to point (ii): preventing a disease to exist through preventing its constituents to exist. Along my constituent ontology (cf. Sec. 2.1) and our definition of disease kinds (cf. Box 4.2), one can *plausibly* argue that preventing the negative cause(s) of a disease to occur is actually preventing a *constituent* of a disease *kind*, viz. the negative cause(s), to occur (cf. also Sec. 4.2.10 on that).

such negative causes and effects, scientists are, thus, able to recognize (or differentiate) by inference the disease at stake[117], by being able to somehow *circumscribe* this specific disease through its causes and effects, or by knowing, first, so to say, what is *around* this specific disease i.e. by investigating other *necessity relationships* (e.g. relative extensionality, or even absolute extensionality) between this disease and other kinds, so as to differentiate, then, the true essence of this specific disease (on the idea of a negative sign of a disease as a *clue* (a hint or an intimation) cf. Sec. 5.1.1)[118].

Hence, one may answer that objection (vi) says nothing about the nature of disease itself, but only about how to (epistemically) proceed for *indirectly* differentiating the nature of disease, or mixes up ontological with epistemological issues about disease by wrongly deducing a certain theory of disease from a certain epistemology (cf. also Sec. 4.2.3)[119, 120].

Third, one may want to argue that a functionalist analysis of disease is actually nothing else than, strictly speaking, an analysis of what disease *kinds* are.

Indeed, in the case of e.g. pain, one can argue that a functionalist analysis of pain really amounts to an analysis of what pain *kinds* are, in the sense that, as functionalists like insisting upon it (Levin, 2018), if this mental state (or process) that is pain is multiply realized (on *multiple realization*, as an entry, cf. Bickle, 2020; for criticisms on the multiple realization thesis, Polger

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[117] Two caveats on medical epistemology: (i) from this above we can usefully distinguish between two specific medical skills: (i) *medical discovery* or detection, which is about how scientists come to *establish* (or know) that *x* counts as a specific disease; (ii) *diagnosis*, which is about how clinicians come to *recognize x* as a specific disease (mostly by inference through recognition of the corresponding specific disease *kind* in a differential diagnosis) (on (Bayesian) diagnostic reasoning, cf. Chiffi & Zanotti, 2017a; Gebharter, 2017; Poellinger, 2012 on formal Bayesian reasoning).

Medical skills (i) and (ii) are, of course, widely acknowledged in today epistemology of medicine (Reiss & Ankeny, 2016), but they have to be strictly distinguished from purely *metaphysical* distinctions.

(ii) A strict distinction between the metaphysics of medicine and its epistemology (without ruling out, however, complex interactions between them) allows us, indeed, to maintain the intuitive idea that e.g. leprosy found in skeletons dating from 2000 BCE and in 2020 is one and the same disease, while our understanding or *knowledge* of it has, of course, evolved or has just not been the same over time.

[118] If we argue that it is common to recognize (or differentiate) a disease by means of something (ontologically) *higher* viz. a disease *kind*, then this will be also true for the case of disease kinds, where they would be themselves recognized through something (ontologically) higher, and so on.

Since the ideal situation is arguably the one where we are directly able to differentiate (or recognize) a disease without passing through the differentiation (or recognition) of a disease.

[119] I acknowledge Guillaume Schlaepfer (personal communication, May 2017) for having pressed me to be clearer on the link between the metaphysics and the epistemology of disease.

[120] Note that this mistake is highly common in *agentive* theories of causation (and constitution) (Craver, 2007; Kästner, 2017; Kästner & Andersen, 2018; Woodward, 2016), where, from a certain epistemology of causation (and constitution) (or causal/constitutive *relevance*, strictly taken) based on ideal interventions/manipulations, one wrongly deduce a certain – though anti-objectivist - theory of the *nature* of causation (and constitution) based, precisely, on the possibility for a free agent to ideally (directly or indirectly) intervene on the relationship in question.

However, it is obvious that there already *existed* (in a mind-independent way) causal relationships on earth before the advent of any free agent capable of (in)directly intervening on them – where nothing was known, of course, thus, about them i.e. here where there was no possibility to (in)directly intervene on them (cf. also Sec. 3.2.4).

An anti-objectivist perspective on the history of science is still possible, but very counter-intuitive here.

& Shapiro, 2016) viz. as having here at least one relative extension (cf. Sec. 3.3.1), then human pain may be said relatively realized by a human pain of a certain kind viz. a pain causing, and caused by, other specific mental states (or processes).

Thus, it seems that a functionalist analysis of pain, by focusing on the causes and effects of pain, intuitively captures something *more* than the mere meaning of "pain" viz. the meaning of "pain *kind*".

In that sense, the thesis that a *functionalist* analysis of (or, so it seems) PAIN is actually nothing else than, or can be easily *replaced* by, a(n) (constitutive) analysis of PAIN KIND is compatible with the thesis that (complex) concepts should be *constitutively* defined (cf. Sec. 2.1), since we are saying that, when we think that PAIN is functionally defined, this is actually another *single* (more coarse-grained) (complex) concept, which is (still constitutively) defined viz. PAIN KIND (however *plausibly* understood)[121].

Along a constitutive line, the same can be said of other mundane notions: we can distinguish between e.g. "smoke" as being *not essentially* the effect of e.g. fire and "smoke(-)of(-)fire" as being essentially (or *constitutively*) the specific effect of a cause viz. fire.

Therefore, our reply to objection (vi) does not imply the rejection of a (properly understood) *semiotics* i.e., in the case of disease, that symptoms (and etiopathological agents) as specific effects and causes may very well be said to have an *epistemological* role to play in the differentiation (or recognition) of diseases, albeit they are not (essentially) parts of, strictly speaking, the *meaning* of "disease", even though they are still considered as specific *metaphysical* constraints on a definition of disease.

Indeed, a kind would intuitively still have the *essence* it has even without ever being caused by, or itself causing, something. Albeit we can argue that it is part of the *existence* of a kind that it is caused by, or itself causing, something, e.g. a billiard ball causing another billiard ball to

[121]Caveat on functionalism: note that both proponents of a constitutive (or constituent) ontology (Loux, 2006; van Inwagen, 2011; Wolterstorff, 1970) and of a functionalist one (Levin, 2018) usually both claim to hold a specific neo-Aristotelian ontology; the former bases his claim mostly on *Met*. Z, while the latter on *DA*. II.
However, the latter holds that functionalism goes hand-in-hand with RME, for he typically holds that what makes something the very entity it is depends on the causal role the entity plays (essentially) *in a system* of which the entity is a part.
Functionalism can be taken here in the *weaker* (more *plausible*, and also *original*) version called "*machine-state functionalism*" (Putnam, 1960) or tentatively "*horizontal functionalism*": "[i]f the machine is in state S_i, and receives input I_j, it will go into state S_k and produce output O_l (for a finite number of states, inputs and outputs)" (Levin, 2018).
In other words, in this weak version of functionalism – still understood in *causal* terms -, there is no reference to a *higher-level* system of which an entity would be (essentially) a part of, or, *plausibly*, a cause of, but only a cause-effect or input-output relationship.
However, functionalism can also be taken in other *stronger* (but more implausible) versions; indeed, if functionalism is the (stronger) thesis that an entity x should be defined with respect to its causes and effects, and if the causal relationship is taken as e.g. *inter-level*, then this version of functionalism is also arguably *reducible* to any (plausible) constitutive analysis of a more coarse-grained entity having x as one of its constituents.

move does not cause *what* a billiard ball's moving *is*, but only the existence of a billiard ball's moving.

4.2.8 Objection (viii): The Disease Host as a Differentia for Disease Kinds

The *intuitive* nosology argued for in Sec. 4.1.2 has followed until now the two most salient nosological criteria (negative effects and causes) highlighted by the most famous (and inclusive) *scientific* taxonomical system of disease kinds: the so-called International Classification of Diseases (ICD) system updated and released by the WHO, if we agree, of course, that the ICD is to be taken as, actually, an analysis of what we are calling, strictly speaking, "disease *kinds*" - currently released in its eleventh edition (for the ICD-11, cf. WHO, 2018a) (on the highly common confusion between a metaphysics and an epistemology of disease, cf. Sec. 4.2.7)[122].

An eighth objection comes as following: however, one may wonder, along a different line, whether our definition is still sufficient for differentiating disease kinds (cf. Box 4.2). In the ICD-11 e.g. one also finds a third nosological criterion: the *disease host* i.e. what is diseased (cf. Sec. 9.3.1)[123].

Indeed, it is common to think of e.g. a CVD as a disease of a certain kind, in the sense that it is a disease which *has* a specific disease host viz. the cardio-vascular system; or, more widely, to think of a *somatic* disease as a disease of a certain kind viz. as a disease whose host is the body.

[122] Of course, there exist other classificatory systems like the Systematized Nomenclature of Medicine (SNOMED) (2020), the Research Domain Criteria (RDoC) (National Institute of Mental Health, 2020), or the DSM now released in its fifth edition (for the DSM-5, cf. APA, 2013; on the history of the DSM, Tsou, 2016).
However, the ICD is deemed to be the reference for medical scientists, for it is intended to be the (most) exhaustive taxonomy of diseases (e.g. the SNOMED merely focuses on specific disease hosts and the DSM-5 only on a certain disease host viz. human cognition), while the DSM-5 may be used as the reference when specifically talking about mental diseases (e.g. the more and more famous RDoC unfortunately relies on a certain reductionism, where mental diseases are brain diseases, somehow coupled with a holistic approach) (as a general critical entry on the RDoC, cf. Demazeux & Pidoux, 2015; for criticisms against the DSM-5, Bueter, 2019; Cooper, 2005; Poland & Tekin, 2017). That is why, I choose to focus *here* on the ICD.

[123] Caveat on disease hosts: it is important not to confuse between the disease host i.e. entities/kinds that are *diseased* or that *host* a disease, and the extension of the term "disease" i.e. entities/kinds/kinds that *are* (directly or indirectly) (more than) diseases, or e.g. between entities/kinds *having*/hosting an aesthetic value (e.g. a person) and entities *being* (in the extension of) aesthetic values (e.g. the value *of* sublimity, of ugliness, etc.).
Indeed, some philosophers (e.g. Récanati, 2008) understand, generally speaking, the notion of the extension of a predicate as being about which *subjects* have/host the predicate at issue (e.g. the extension of "_is beautiful" comprises "John", "Mary", etc.). We distinguish here between the issue of what the extension of a predicate is and of what has/hosts the predicate in question.

The main problem at stake with objection (viii) is that allowing the disease host to be, strictly speaking, a differentia for disease kinds is precisely that it must be the host (e.g. the cardio-vascular system) *belonging to a certain kind* (viz. a disease)[124] i.e. that it makes the definition of the differentia dependent upon the definition of a disease (*tout court*). As such, it goes against our requirement that the differentia between disease and disease kinds be independently defined from the definition of disease (cf. Sec. 3.1.1).

But, how, then, to define a CVD, if not as a disease (i.e. the genus) in conjunction with a specific disease host (i.e. the differentia)?

There are two possible answers to this question. The first shall be rejected, whilst the second shall be maintained. The first answer is as follows: like red is a specific shade of color, we would like to maintain that a CVD is a specific disease kind, but that it cannot be defined as a disease with a specific disease host. How, then, to define a CVD?

The ICD-11 itself seems to provide us with an answer: a CVD may be defined with our differentia (negative effects and causes), for it may be said a disease in conjunction with specific negative effects and causes (e.g. pain or discomfort in the center of the chest, in the arms, in the left shoulder, in the elbows, in the jaw or the back).

But, how to account, with such a solution, for our talk about, precisely, a *CV*D? Do we have to conclude, thence, that, actually, a CVD, as being a disease kind, has nothing to do with the cardio-vascular system, but only with specific symptoms and etiopathological agents which are contingently hosted by one and the same system viz. the cardio-vascular system?

Two arguments could be put forward in favor of this thesis: the first consideration is that a disease kind term like "bacterial disease" or "HIV infection/disease" seems to *plausibly* refer – not to, only when properly taken, a mere bacterial or viral process/invasion/infection -, but to a bacterial or viral infection as a *cause* of a certain disease (or as an etiopathological agent), but, if we agree that a bacterial disease or HIV infection/disease is a certain disease kind and that the differentia is effects *and* causes (of a disease), then, as such, the term does not seem to refer to the effects (of the disease) at all – and, *strictly* speaking, not even to the causes of the disease. Ditto for other specific disease kind terms like (or, so it prima facie seems) "CVD": even if it seems that such disease kind terms refer to a specific disease host, one may argue that, as for the case of a bacterial disease or HIV infection/disease, the name itself is misleading.

[124] It cannot be here a host *tout court*, for a host is essentially either a *property* bearer or the entity *of* which there is a negation (cf. Sec. 9.3.1 on that), alongside other negative entities like an absence *of*, a lack *of*, a hole/perforation *in*/*of*, a destruction/privation *of*, a disturbance *of* (cf. Karmo, 1977), a deviation *from*, a certain *positive* entity.

But, why, then mentioning in the name of such disease kinds either a specific process or a certain disease host? By contrast with other specific disease (kinds) like viral diseases, which seem to have different specific hosts (to the extent that a viral process can *plausibly* infect different specific cells of an organism) like vital cells of the human immune system with HIV infection, or respiratory epithelial cells for rhinovirus infection, a CVD seems so defined (i.e. as a disease with such-and-such effects and causes) that it happens that only a malfunctioning cardio-vascular system can absolutely *realize* it (cf. Sec. 3.3.1).

Thus, as such, we could argue that the name "CVD" strictly refers to a specific disease kind (a disease with such-and-such effects and causes), but, since the disease kind in question is uniquely realized, the name ambiguously seems to directly refer to the absolute realizer (a malfunctioning cardio-vascular system) of the disease kind.

However, this first answer is to be rejected, on grounds that, if a malfunctioning cardio-vascular system is an absolute realizer of a CVD, then it makes a malfunctioning cardio-vascular system a specific disease super/subkind. And there is no such thing as a disease super/subkind as tentatively defined in Sec. 3.1.2.

Moreover, thinking that a viral disease can have many different disease (kind) hosts is a category mistake. A viral disease is had by a single category of hosts: anything that can have a viral disease viz., *plausibly*, a cell, where e.g. HIV infection/disease would be a *specific* disease (kind) whose host is the vital cells of the immune system.

From this, one already sees the second answer that we can propose: as has already been argued for (cf. Sec. 4.2.2), one can say that, actually, a CVD is not a specific disease kind, but a specific disease *simpliciter*; there is no a priori reason to define a CVD as a disease kind.

Once we distinguish between *relative* and *absolute* extension (cf. Sec. 4.2.2), one can easily argue that "CVD" is in the absolute extension of "disease", while "viral disease" is plausibly in the absolute extension of "disease kind" (or, in the relative extension of "disease").

The same objection can be raised for the cases of mental (e.g. psychological, emotional, mood, etc.) diseases. These diseases seem to be properly called "disease kinds", if one thinks that they are essentially felt, and if we define a symptom as a feeling of a disease (but, cf. Ch. 5), and they explicitly mention a specific disease host in their name.

However, there is no reason to think that such diseases should not be treated along the same line as for the case of CVDs or even physical (or somatic, bodily or physiological), neural, etc., diseases.

This means that a definition of an *emotional* (or *affective*) disease (an *affect*, or affection (pejoratively taken)) is kept separate from a definition of a (bad) feeling or *felt* disease (cf. Sec.

5.1.2), if we link symptoms to feelings (but, cf. Box 5.2 for a definition of a symptom). This may sound a surprising result, but it is a virtue for a definition of an emotional disease to dissociate (bad) emotions from (bad) feelings (cf. Sec. 5.1.2), in the sense that *correct* (bad) emotions are neither felt nor unfelt (on theories separating emotions from feelings, cf. Adolphs & Andler, 2018).

4.2.9 Objection (ix): A Process as the Differentia for Disease Kinds

A ninth objection is that one is used to prima facie define disease kinds by describing a specific process plus a differentia i.e. a process of a certain kind. When one talks about e.g. cancer, one talks about malfunctioning cell growth, or "myocardial infarction" refers to a specific process (an infarction) plus a specific host (the myocardia).
However, if so, then (against objection (ix)) the differentia for a disease kind would not be defined independently from what disease and the disease host are, under the vague (or general) plausible assumption that a disease is here a certain process.
Along the same line as our previous reply to objection (viii) (cf. Sec. 4.2.8), one could answer that the focus on the processual aspect of cancer means that, given a definition of a cancer kind as a disease with such-and-such effects and causes, only a cell growth process (with such-and-such effects and causes) would absolutely realize this specific disease kind.
However, the same concern as the one for considering a CVD as a specific disease kind applies (cf. Sec. 4.2.8): there is no a priori reason for considering e.g. cancer as a specific disease kind, in the sense here of a process (a disease) having for its differentia the fact that it is a process *of a certain kind* – and not as a specific disease *tout court*, if we argue that "disease" necessarily has (at least) an *absolute* extension (cf. Sec. 3.3.1).

4.2.10 Objection (x): Against the Independency of the Definition of the Differentia

We can raise the following objection (x): the differentia between disease kinds and disease is, actually, not defined independently of what a disease is. Indeed, if we argue that there is no disease without e.g. a disease kind (cf. Sec. 3.3.1), and if we take the differentia between disease and disease kinds as a negative cause and an effect (cf. Box 4.2), then we reach the conclusion (along objection (x)) that there is no disease without a negative cause and an effect. In that sense, objection (x) validates a functionalist approach to disease (cf. Sec. 4.2.7): negative causes and effects - not positive ones, on pain of a category mistake - would be essential to disease.

In a nutshell, if we say that a disease kind is a disease with negative cause and an effect, then, according to objection (x), does not that much also make a disease necessarily a negative effect, once a negative cause is present (e.g. fever (alone) or a(n) (exo/endo)toxin release process can be described as a disease itself, but is also often considered (especially when coupled as fever *of x* or a(n) (exo/endo)toxin release process *from*/*to x*) as a negative effect of many different specific diseases), or itself a negative cause, once a negative effect is present?

Nevertheless, again (cf. Sec. 3.3.1), first, the intuition that there is no disease without e.g. (amidst others, or at least) a disease kind is not to be taken as indicating (plausibly taken) that a *definition* of disease is based on a disease kind.

Indeed, the claim that a disease is not essentially a disease kind does not contradict the intuition that there is no disease without e.g. (amongst others, or at least) a disease kind (i.e. a disease negatively caused by, and causing, something), thus that a disease kind is *necessary* to a disease, once we are clear that we are talking about the necessity for the term "disease" to have a *relative* extension viz. e.g. "disease kind".

Second, objection (x) mistakenly associates the intuition that there is no disease without e.g. a disease kind with the idea that there is no disease without a negative cause and an effect. The requirement of relative extensionality (cf. Sec. 3.3.1) does not imply this latter idea[125].

[125] But, if we say that a disease is necessarily a disease kind, then *being a disease kind* is a necessary but insufficient property for *being a disease*. Thus, a disease could be said *defined* as a disease kind plus (a non-necessarily independently defined) *x*, where both conditions are necessary and sufficient for a disease.
Such an objection aims at blurring our distinction between absolute and relative extensionality (cf. Sec. 3.3.1). To address it, two points are to be raised: (i) the general idea of giving strictly necessary and sufficient conditions (for a definition) is different from our neo-Aristotelian framework of giving a definition within (our understanding of) a genus-species hierarchy, although our neo-Aristotelian framework can be said to be a *specific* way of giving strictly necessary and sufficient conditions (cf. Ch. 2; Sec. 3.1.1);
(ii) it may be argued that, if *x* has for its absolute extensionality *y*, then there is no way in which *y* can be (within (our understanding of) a genus-species hierarchy) *x plus* an *independently defined* differentia: e.g. if "human being" has for intension "rational *animal*" – and not "rational *mammal*" -, then we may argue that, actually, a human being is a *specific* mammal (or, "human being" is in the *absolute* extension of "mammal") – and not a mammal of a certain kind.

However, is not "disease (of *x*)" (e.g. cancer (of *x*)) *also* intuitively in the indirect *absolute* extension of "(specific) negative cause of *x*" or "(specific) negative effect of *x*" [126, 127]?

For, it is true that, if *x* is a negative cause of a disease, then a disease is *indirectly* a negative effect of *x* (but this does not also make – at least *directly* - a disease a symptom itself, since a disease would be here indirectly the negative effect of a mere cause *x*); likewise, if *y* is a negative effect of a disease, then a disease is *indirectly* a negative cause of *y* (but this does not also make – at least, directly – a disease an etiopathological agent, since a disease would be here indirectly the negative cause of a mere negative effect *y*).

Let us say that e.g. cancer is a specific disease (plausibly) defined as a specific complex *causal* process (malfunctioning cell growth) of (minimally) (i) dysplasia (viz. here hyperplasia) and (ii) the generation of a (benign) tumor (or neoplasia) – where steps (i) and (ii) are deemed specific *diseases* themselves.

If so roughly defined, we can argue that step (ii) is (indirectly) a specific negative *effect* of step (i) and consequently step (i) a specific negative *cause* of step (ii), or that step (i) is (indirectly) a specific *disease* (negatively) causing step (ii), *or* that step (ii) is (indirectly) a specific disease (negatively) caused by step (i)? If this is the case, then steps (i) and (ii) can be judged to be both (in the indirect absolute extension of) a disease as well as a mere negative cause or effect (of a disease).

4.2.11 Objection (xi): A Specific Causal Relationship as the Differentia for Disease Kinds

[126] Another objection is that, if we agree that there is no cause without an effect and vice versa (thus, that a cause or an effect is a *necessary* (but not essential) condition for an effect or a cause), and if we argue that "disease" is intuitively in the indirect absolute extension of "cause" or "effect", then "disease" prima facie seems also in the indirect *relative* extension of "effect", when it is in the absolute extension of "cause" and vice versa.
Again (cf. Sec. 4.2.5), this is to misuse here the relation of (indirect) relative extensionality. *Indirect* relative extensionality means that the relation of relative extensionality is cumulated through a genus-species hierarchy – not that the relationships of absolute and relative extensionality are mixed up.

[127] Terminological note on secondary diseases: diseases considered as mere effects (of, especially, other specific diseases themselves) are commonly (and intuitively) called "*secondary diseases*" – which should not be confused with *symptoms*, for symptoms are not diseases whose signs are necessarily (though it is most often the case) *diseases* themselves (e.g. an effect of Diogenes syndrome can be (plausibly) waste accumulation) (cf. Box 5.2): e.g. influenza resulting in fever, or a microbial process resulting in a(n) (exo/endo) toxin release process, where fever or a(n) (exo/endo) toxin release process shall be considered itself (indirectly) a specific disease.
Note that primary and secondary diseases may very well both form a *single* (whole) disease itself, only if they can be considered as a specific whole or species, of course (cf. Box 3.1).

An eleventh and last objection against the sufficiency of our definition of disease kinds (cf. Box 4.2) – closely related to objection (x) (cf. Sec. 4.2.10) – is the following one: if we say that a disease kind is a disease in conjunction with a negative cause and an effect, do we necessarily talk about the negative cause *of a disease* and the negative effect *of a disease*? Why could not we understand this definition (cf. Box 4.2) differently? E.g. a disease kind is a disease followed (or preceded) by a negative cause-effect/causal relationship – thus, where the negative cause and the effect are *not* of a disease.

A first quick reply to objection (xi) relies on the intuitive idea that there is no cause without an effect and no effect without a cause (cf. Sec. 4.2.10) i.e. that the existence of a cause entails the existence of the resulting effect and vice versa. If this is the case, then, if we explicitly mention in the differentia for disease kinds negative causes *and* effects (cf. Box 4.2), then, were the negative causes and effects not about a disease, mentioning both of them in the differentia for disease kinds would be redundant (along objection (xi)).

Indeed, if we agree that there is no cause without an effect and vice versa, and if we think that negative causes and effects are *not* here of a disease (along objection (xi)), then mentioning in the differentia either the negative cause or the effect is sufficient.

So far, so good. But, then, objection (xi) goes on to say that, even if we agree that the fact that the differentia for disease kinds consists in negative causes *and* effects means that (at least) a third term *x* is involved, it still does not mean that, when we say that a disease kind is a disease plus a negative cause and an effect (cf. Box 4.2), that this cause and this effect are specifically *of the disease*: e.g. we could define a disease kind as a disease followed by (or preceded by) a negative cause and an effect of *x* (or a *specific* cause and effect), where *x* is not, strictly speaking, a disease; or, we could maybe even define (along objection (xi)) a disease kind as a disease plus a negative cause of *x* and a negative effect of *y*.

Objection (xi) can also add here that the absence of a third term *x* other than "disease" in a definition of a disease kind as a disease plus negative causes and effects (cf. Box 4.2) does not indicate that the negative causes and the effects are of a disease, for the definition of the differentia should remain independent from the definition of the genus: a disease kind is a disease plus a negative effect and cause, where the specific effect and the specific cause are defined independently from the definition of a disease; it merely indicates that these negative causes and effects can just be *interpreted* as being of a disease. Indeed, if the differentia is to be taken here as negative causes and effects of *x*, then *x* can be taken as the formalization of "disease".

However, we can eventually reply that, if this is how objection (xi) is to be taken, then it is *not* an objection against our definition of disease kinds.

Indeed, *strictly* speaking, a disease kind is not a disease plus negative causes and effects *of it*. Were this true, then the differentia would be defined *dependently* upon what the genus in question is viz. a disease.

Although talk about a disease kind can be *plausibly understood* (or interpreted) as, or is compatible with, talk about symptoms and etiopathology i.e. the negative effects and causes of disease – for, we can argue that we can pass from the idea that a disease kind is a disease plus a negative cause of a specific *process* and a negative effect of a specific *process* to the idea that the process in question is a disease -, a strict or *correct* (interpretation of the) definition of disease kinds (i.e. embedded into our metaphilosophical framework) (cf. Ch. 2-3) cannot state, indeed, that the differentia is the negative effects and causes *of disease*.

Thus, we can eventually reply to objection (xi) by saying that it is, actually, not an objection against our definition of disease kinds, but just a plausible re-statement of our own definition of disease kinds (cf. Box 5.2).

5

SYMPTOMS

Ch. 5-6 of Part II are devoted to analyze – not the differentia for disease kinds i.e. negative causes and effects *simpliciter* -, but, more specifically symptoms (Ch. 5) and etiopathology (Ch. 6).

In order to avoid further confusions between DISEASE and related concepts, it is important to thoroughly analyze SYMPTOM and ETIOPATHOLOGY.

Ch. 5 is devoted to the analysis of the first concept viz. SYMPTOM. Ch. 5 is organized as following:

Sec. 5.1 prima facie defines a symptom as a negative sign of a disease, and argues that felt and unfelt symptoms are specific symptoms.

In Sec. 5.2, I detail the sense in which a symptom is a negative sign of a disease by giving a correct definition of a symptom within (our understanding of) a genus-species hierarchy as a disease (i.e. the genus) and a directly temporally succeeding entity related to a living being (i.e. the differentia).

In Sec. 5.3, I raise and consequently address objections against our (prima facie) definition of a symptom.

5.1 A Prima Facie Definition of a Symptom

Sec. 5.1 aims at giving a prima facie definition of a symptom, such that it can be used as a *preliminary* step into giving a correct definition of a symptom (cf. Box 5.2). I am giving here a prima facie definition of a symptom as a negative sign (indicating the past presence, or left by the past presence) of a disease. Not much of the philosophy of medicine has ever focused on the concept of symptom (with one notable exception: Whitbeck, 1977).

Although there is a burgeoning literature on *causation* in medicine (cf. Russo & Williamson, 2007 as a starter), philosophers of medicine have been, unsurprisingly, focusing until now, more precisely, on the notion of (negative) *cause* in medicine or etiopathology (cf. Ch. 6), but not on the precise notion of (negative) *effect* in medicine or symptoms, as if analyzing the notion of (negative) medical cause itself were sufficient for analyzing medical causation *tout court*.

I show in Sec. 5.1 that the notion of symptom itself is ambiguous and deserves, thus, that some philosophical attention is directed towards it.

Sec. 5.1 is divided into two further sections: In Sec. 5.1.1, I argue that, to remove any ambiguity behind the meaning of "symptom", a symptom is to be prima facie defined as a negative sign of a disease.

In Sec. 5.1.2, I argue that felt and unfelt symptoms are both *specific* symptoms.

5.1.1 A Symptom as a Negative Sign of a Disease

What are we talking about, when we are saying that such-and-such disease kind is constituted by (or defined with) such-and-such *symptoms*? There are three intuitive ideas (or insights) at the basis of what symptoms are:

(i) a symptom is a *feeling* or an awareness (or *consciousness*) indicative of a disease (on consciousness or feelings, cf. Sec. 7.1.2)[128];

(ii) a symptom is a negative *clinical* (or *medical*) *unfelt sign*(*al*) (trace, mark, *effect* or result) (indicating the past presence) of a disease – or, simply put, a negative unfelt sign(al) of a disease;

[128] Caveat on felt signs of a disease: it is important not to confuse, at the outset, between a feeling *of* a disease (or *illness* or *bad feeling*) and a feeling *indicative* (or, of a sign) of a disease, or between a feeling of a disease and a negative felt *sign* of a disease.

(iii) a symptom is both (i) a feeling indicative of a disease *and* (ii) a negative unfelt sign(al) of a disease[129].

In the medical literature, the use of the term "symptom" clearly oscillates between points (i), (ii) or (iii). Point (i) clearly describes the most common usage of the word "symptom" that we find in any standard medical textbook like "Harrison's Principle of Internal Medicine" (Kasper et al., 2015, p. 2; my emphasis):

> For example, abnormalities of hepatic function may provide the clue to […] symptoms such as generalized *weakness* and increased *fatigability*, suggesting a diagnosis of chronic liver disease […] [, thus *p*]*ointing to* a particular disease […].

[129] Five terminological notes on symptoms here: (i) point (ii) above excludes, thus, talking about *prognostic* signs *tout court* viz. signs indicating that e.g. a disease (or death) shall occur *in the future* i.e., in daily speech, early (warning, precursor, forerunning, etc.) signs of a disease (or death): e.g. the famous Hippocratic face said a sign of death. Indeed, this is to take signs of disease for *causes*/*sources* of disease here (cf. Ch. 6).
However, only signs of disease strictly called "anamnestic" in the medical literature (cf. King, 1982) and "pathognomonic" i.e. signs indicating the *past* presence/existence of a (*peculiar*) disease are *truly* negative signs of a (peculiar) disease *simpliciter*.
(ii) The terms "prodrome" and "postdrome" are sometimes used to refer to, respectively, early and late signs occurring (temporally) after a disease (e.g. migraine). Are they not just symptoms, actually?
We can very well consider prodromes and postdromes as specific symptoms themselves viz., respectively, early and late effects of a disease (cf. Sec. 6.3.2 on indirect etiopathologies/symptoms).
However, the notion of prodrome is also very widely used to refer to, not early *effects* of a disease, but premonitory *causes* of a disease: e.g. an aura as a prodrome of a migraine.
Therefore, due to the ambiguity behind "prodrome", it is preferable to stick ourselves in using the term "symptom" or "etiopathology".
(iii) Are *complications* of disease symptoms? The main issue with disease complications is that it is unclear, in the medical usage, whether complications of a disease are really *signs of* a disease or parts of a disease itself. Indeed, "disease complication" commonly refers to either the *negative consequence* or effect of a disease (e.g. thrombosis as a complication of phlebitis) or the *severity evolution* of the very disease (process) (e.g. generalized septicemia as a complication of an infected wound). That is why, it is preferable or more recommended to avoid using "disease complication".
(iv) Symptoms or (more often) signs of disease are also commonly called "*stigmata*" e.g. in "Miller-Keane Encyclopedia and Dictionary of Medicine, Nursing and Allied Health" (2003) or "The Concise Dictionary of Modern Medicine" (Segen, 2010).
However, talk about a stigma should be avoided here because of the high ambiguity and the many discrepancies surrounding it. For, "stigma" is also said to more narrowly (or exclusively) refer to what are, according to us, some *specific* symptoms or signs of disease, especially *mental* or *bodily* ones (e.g. petechia, stains/spots, scars, etc.) (cf. e.g. Dorland, 2011), or to a specific vice viz. "[…] an attribute that links a person to an undesirable stereotype, leading other people to *reduce* the bearer from a whole and usual person to a tainted, discounted one" (Goffman, 1963, p. 11; my italics) i.e. the reduction of a complex whole viz. a person here to some of its parts (cf. also Sec. 10.2.1).
(v) The term "bio*marker*" (or "biochemical *sign*ature") is also often used to refer to, generally, specific signs of a disease or any other healthy process viz. lower-level signs like physiological (or somatic) ones or, even more specifically, e.g. signs of the motor system, signs at the cellular level, etc. (e.g. antibodies as biomarkers of an infection); the Biomarkers Definitions Working Group (2001, p. 91; my emphasis) defines a biomarker as "[…] a characteristic that is objectively measured and evaluated as an *indicator* of normal biological processes, pathogenic processes, or pharmacologic responses to a therapeutic intervention" i.e. as a mere biological sign (cf. Nesse & Stein, 2012; Strimbu & Tavel, 2010; *pace* Baker 3rd & Sprott, 1988; Fuellen et al., 2019, for whom biomarkers, in the context of aging, are predictors).
With this definition of a biomarker in hand, symptoms (as taken under point (ii) above) can be considered, thus, as specific biomarkers.

In the same medical textbook, it is also said, later on, that "[…] "fatigue" is best used to describe a *feeling* of low physical or mental energy but without a tendency to actually sleep" (Czeisler et al., 2015, p. 188; my emphasis).

In other words, fatigue or weakeness is a symptom, in the sense that it indicates a specific disease, and is, at once, a certain feeling.

That much fits nicely with the characterization of symptoms as feelings indicative of (or, like here, "pointing to") a disease (cf. point (i)).

Moreover, it is also highly common to talk about patients *experiencing* symptoms (cf. e.g. Armstrong, 2003).

As such, symptoms understood as feelings indicative of disease are often clearly *opposed* to how point (ii) describes what a symptom is viz. as a negative *unfelt* sign of a disease. However, it is not rare to find usages of the word "symptom" complying with point (ii).

Indeed, we can use our very same above quote from "Harrison's Principle of Internal Medicine" to argue that, if *weakness* is truly taken as a symptom here, there is no reason to treat it as a feeling indicative of a disease. For the idea of weakness is clearly separated from the idea that "[…] [m]any disorders can cause *feelings of* weakness […]" (Czeisler et al., 2015, p. 189; my emphasis. Moreover, to add more credentials to the idea that "symptom" is also used to refer to negative unfelt signs of a disease, there is no a priori reason why we could not also *widely* use the term "symptom" for analyzing disease kinds whose hosts can only have unfelt or unconscious diseases i.e. *unconscious* organisms (mostly for reasons of low organic complexity and evolution) (cf. Sec. 5.1.2).

Although it is usual to oppose symptoms to mere negative unfelt signs of a disease, it is also usual to find usages of the word "symptom" complying with point (iii) i.e. with both points (i) and (ii): e.g. there are numerous cases where fever and rash (of *x*) are both said *symptoms* (constitutive of the same specific disease kind(s) like measles), in the sense that fever is taken as a *feeling* indicative of some disease *x*, while rash is taken as an *unfelt* sign of the same disease *x* (for a non-exhaustive list of the diseases in question, cf. Kaye & Kaye, 2015)[130].

It is obvious that a correct – at least, prima facie - definition of symptoms should, thus, coherently take into account both points (i) and (ii) i.e. that it should somehow comply with (or, be based on) point (iii). We can argue that a way to do so is to propose a wide enough definition

[130] Note that, if I am right that to each specific disease kind its own symptoms or etiopathology with the same *grain* of specificity, then, although it can be plausibly defended that a specific disease kind can be constituted by both negative *unfelt* and *felt* signs of the same disease, it is, nevertheless, incorrect.
Of course, this should not be taken as a motivation for providing a definition of symptoms which does *not* take into account *both* points (i) and (ii).

of symptoms, where both "felt symptom" (cf. point (i)) and "unfelt symptom" (cf. point (ii)) can be said in the absolute extension of "symptom"; among others, or at least (e.g. "mental symptom" or "somatic symptom"), "felt symptom" and "unfelt symptom" are absolute realizers of "symptom".

In light of this requirement, the proposal for a prima facie definition of symptoms, as a preliminary step before giving a correct definition of symptoms (cf. Box 5.2), intuitively comes as follows: a symptom is a negative sign of a disease; or, for *x* to be a symptom is for *x* to be a negative sign (i.e. condition (ii)) of the past presence of a disease (i.e. condition (i))[131]:

Prima Facie Definition of a Symptom

x is a symptom, iff (i) *x* is a disease, and (ii) *x* is a negative sign of it.

[131] Five caveats here on our prima facie definition of symptoms (cf. Box 5.1): (i) we may worry that prima facie defining a symptom as a mere negative sign of a disease just amounts to above point (ii): a symptom as a negative *unfelt* sign of a disease.
However, the specification "unfelt" is here essential: it explicitly specifies that we are talking about something else than a *felt* symptom (cf. Sec. 7.1.3 on unfeelings).
(ii) Does this prima facie definition (cf. Box 5.1) commit the fallacy of affirming the consequent: from the presence of a symptom can we deduce that there is a disease? Indeed, if smoke is a sign for fire, it does not imply that from the mere presence of smoke we can deduce that there was fire, for smoke can also be the sign for something else like a smoke grenade.
However, can a symptom be something else, properly speaking, than a negative sign for *disease* (cf. Sec. 4.2.2; 5.3.4)? We are allowed to prima facie define a symptom as a negative sign for disease, to the extent that, in its proper meaning, "*symptom*" cannot (arguably) refer to a sign of something else than a disease (*pace* First & Wakefield, 2013); if it appeared that there is no disease as the source of the sign, then we would just refrain ourselves in talking about symptoms here.
(iii) Talking about a symptom *of a disease* is pleonastic, for a symptom is undeniably (a *synonym* for) a negative sign *of a disease* (cf. Box 5.1). Indeed, it attributes a predicate of what we can *plausibly* take as an element of the definiens to a definiendum, in so far as a symptom is prima facie defined as a negative sign of a disease (cf. Box 5.1).
In the context of the (*im*)proper part-whole relationship (for its link with definitions, cf. Sec. 2.1), we can talk here about, more precisely, a *reverse* MF, in so far as a MF is defined as the misattribution of the predicate of a whole to one of its (im)proper parts (cf. Bennett et al., 2007).
Common pleonastic talk about a symptom of a disease is to be put along the same line as talk about (*plausibly*) e.g. *p* as a definition *of what q is* (or of the *nature* of *q*), but a (linguistic) definition can be itself prima facie defined as a proposition telling us what *q* is or the nature of *q* (cf. Sec. 2.1).
Thus, strictly speaking, we should say that *x* is a symptom (*tout court*) or (synonymously) a negative *sign of a disease*, and that *p* is a (linguistic) definition of *q* or a *proposition stating what q is* (or *the nature of q*).
As long as we are clear that, when we talk about a symptom *of* a disease, we are committing a reverse MF, it is allowed to follow this very widespread pleonastic talk, but it should still be avoided, when possible, for sake of clarity. The same remark also applies for common talk about the etiopathology *of a disease*, if an etiopathological agent is prima facie defined as a negative cause *of a disease* (cf. Ch. 6).
(iv) Why not prima facie defining a symptom as a negative sign plus a disease, or, more generally, a negative sign as a negative sign plus *x* – rather than as a disease plus a negative sign (cf. Box 5.1)? The choice for this latter solution rests on the willingness to respect more here (even though with a prima facie definition) the basic idea that "*y* (e.g. a (negative) sign) of *x*" can be taken as a constitutive relationship between "*y of x*" (as a whole) and "*x*".
(v) Why could not we *correctly* define a symptom as a disease (i.e. the genus) plus a specific sign (i.e. the differentia)? For, this specific sign, so as to be a specific differentia defined independently from what the genus in question is, cannot be, thus, of a disease, but a symptom cannot but be a negative sign *of a disease*.

Box 5.1. – Prima facie definition of a symptom.

5.1.2 Felt and Unfelt Symptoms as Specific Symptoms

In so far as we argue that, actually, a symptom is to be prima facie defined as a negative sign of a disease (cf. Box 5.1), thus, retrospectively, point (i) is envisaged as the definition of a *specific* symptom viz. a *feeling of a symptom* defined as a negative felt (or feeling of a) sign of a disease, while point (ii) is considered as the definition of another specific symptom viz. an *unfelt symptom* defined as a negative unfelt sign of a disease.

However, are feelings of a symptom really specific symptoms themselves? We can argue that a feeling of a negative sign of a disease is itself to be considered as a (high(er)-level) negative sign of a (high(er)-level) disease (cf. Exc. 5.1 on levels).

The reason for this is the following: there are certain disease kinds that are (plausibly) (partly) defined as symptoms that are *necessarily felt*; even though we may argue that the negative effects of higher-level diseases like mental (e.g. emotional) diseases are not necessarily felt, one may argue that, actually, other higher-level properties of e.g. a human being are necessarily felt or experienced, to the extent that a human being would be a *sentient* (or conscious) organism, and that an effect of a disease situated at this higher (or maybe even highest) level would be also necessarily felt (e.g. a *feeling* of a headache or of a migraine as the effect of a *feeling* of unwellness).

We draw here on a general distinction between *bad* (or *negative* or, more specifically, *diseased*) *emotions* (or, more generally, bad mental states, or bad bodily states/processes) (*kinds*) (or emotional, etc., disease(s) (kinds)) and *bad* (or *negative* or, more specifically, *diseased*) *feelings* (*kinds*) (or felt disease(s) (kinds))[132]; we would intuitively say, indeed, that e.g. anxiety or

[132] Two caveats on phenomenology: (i) this fine-grained distinction already allows us to take a clear stance on the debate about the necessity (or not) to rely on a so-called *phenomenology* (in my terminology, strictly speaking, on feelings or awareness) to account for the effects of mental or, more specifically, emotional diseases (as an entry, cf. Radden, 2019).

Indeed, by separating bad emotions (and, more generally, mental states or processes as well as bodily states or processes) from bad feelings, we can consistently both reject the claim that a theory of mental disease kinds must be phenomenologically based (or, strictly speaking, based on bad feelings) and keep the idea that a phenomenology is, nevertheless, useful, but for theories of other more specific disease kinds than mental and emotional disease kinds.

(ii) The separation of bad emotion kinds from bad feeling kinds seems good news for the *reductionist* who tries to account for the effects of mental diseases purely in unfeeling-based terms (Forest, 2016; Guze, 1992).

However, any reductionist aspiration actually evaporates, for, even if we strictly distinguish between the effects of bad feelings and those of bad emotions, that much does not imply that bad emotion kinds are themselves

anguish/angst ((indirectly) measured through e.g. (arterial) hypertension) can be continuously present, even when there is no necessarily *felt* anxiety – we would *not* judge, furthermore, that a subject ought to feel anxious.

This distinction also has the advantage of being able to take into account some famous cases of disease like prosopagnosia (or visual agnosia, more generally), where we could say that it is a disease, whose host is specifically a certain *feeling* (or awareness) associated with (face) perception; or blindsight could be (plausibly if not correctly) described in such a way that a certain (at least, partial) feeling of perception (or visual *experience*) still exists, where there is, actually, no unfelt perception i.e. in a blind visual field.

Put in a broader perspective, we may wonder: is a feeling of a disease (thus, a bad feeling) *itself* a specific (higher-level) disease? There is no a priori reason to not envisage bad feelings as specific (higher-level) diseases themselves (cf. also Sec. 7.1.1) – thus, to not also consider a negative *effect* of a bad *feeling* (or of a feeling of a disease) as being itself necessarily *felt*.

If this is the case, then the same diagnosis (so to say) can also be made, more generally, about (un)feelings *tout court*: if we assume that, exactly like a felt disease is itself a specific (higher-level) disease as well as a negative felt sign of a disease is itself a specific (higher-level) negative sign of a disease, then a negative *felt* sign of a *bodily* disease is to be strictly analyzed, actually, as a specific negative *bodily sign* (viz. a negative *felt bodily* sign) of a specific bodily disease viz. a *felt* bodily disease (e.g. felt lung cancer), as well as a negative *unfelt* sign of a *mental* disease is to be itself analyzed as a specific negative *mental sign* (viz. a negative *unfelt mental* sign) of a specific mental disease viz. an *unfelt* mental disease (e.g. unfelt depression, or unfelt anxiety, etc.).

One may want to argue that, if one allows diseases to be not only physical (or somatic) but also mental (e.g. psychological or emotional), then a *felt* symptom would be essentially the sign (indicating the past presence) of e.g. an *emotional* disease, as well as an *unfelt* symptom would the sign of a *physical* or *somatic* (neural, etc.) disease.

But, as we have seen (cf. Sec. 4.2.8), as being a specific disease, a definition of an emotional disease *tout court* is to be kept separate from a definition of a (bad) feeling. Thus, a certain effect of an *emotional* disease is a specific sign viz. an *emotional* sign, whilst a certain effect of

(absolutely extensionally) reducible to specific *unfelt* disease kinds: felt emotional disease kinds and unfelt emotional disease kinds are two different specific emotional disease kinds.

Moreover, we may suspect that what many have in mind when they talk about mental and, more specifically, emotional disease kinds just is what I am calling, actually, "bad feeling kinds".

an *unfelt* emotional disease is a specific *unfelt* emotional sign, and a certain effect of a *felt* emotional disease (i.e. a certain bad feeling) is a specific *felt* emotional sign[133, 134].

In a nutshell, we have to be careful in not thinking that it is because we talk about higher-level properties like affective ones that these properties are to be defined as feelings (cf. Sec. 2.1), even if we grant that a human being is a sentient organism; we may want to argue that higher-level properties of a human being are *conscious* properties (or feelings), but that much does not imply that other higher-level properties such as affective, linguistic (as specific mental properties), or, more generally, mental or even social ones are themselves conscious too (cf. e.g. Sec. 7.2).

To summarize, we can argue here that the fact that an effect of a bad (or negative) feeling is necessarily felt is due to the disease in question (i.e. a bad feeling), which the symptom at issue is a sign of. The felt character of a symptom is determined by (or, follows) the disease kind at issue one tries to define: e.g. a *felt mental* disease kind is essentially a *felt mental* symptom,

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[133] Caveat on granularity: that much above may sound a convoluted thesis, but the basic idea behind it is rather simple to grasp and almost trivial: to each specific thing (e.g. a disease, a felt disease, an unfelt disease, etc.), the *same* degree (or grain) of specificity applies (e.g. a sign, a felt sign, an unfelt sign, etc.) (cf. Exc. 5.1 on levels).
Hence, talk about a felt sign of a mental disease or an unfelt sign of a bodily disease is a category mistake: a sign of a *mental* disease is necessarily a *mental* sign, and a sign of a *bodily* disease is necessarily a *bodily* sign. The *felt*/*unfelt* dichotomy does not correspond to the *mind*/*body* dichotomy (cf. Sec. 7.1.2).
Allowing both coarse-grainedly differentiated (negative signs of) diseases as well as highly fine-grainedly differentiated (negative signs of) diseases – without allowing, however, any (absolute extensional) reduction between them -, has the benefit of avoiding disjunctions: e.g. if fever (of *x*) is strictly defined as a specific symptom, then it is the specific sign of a single disease, while e.g. a *felt* fever or an *unfelt* fever (of *x*) shall be a specific sign of another specific (single) disease (with the same grain of differentiation): to every (coarse- or fine-grained) specific disease its *own* (coarse- or fine-grained) specific effect/sign. Subscribing to a permissive neo-Aristotelian ontology (cf. Exc. 3.5) allows to both avoid disjunctions and to somehow take into account the intuition that e.g. fever (of *x*) as a specific symptom is associated with many specific diseases.
However, with such a view, the temptation to *reduce* (or *dispense* with) *coarse*-grained (or *higher*) levels to (maintain only) the most *fine*-grained (or the *lowest*) levels is high: this is a(n) ((in)direct absolute extensional) *reductionist* stance, according to which things as e.g. horses are reducible to e.g. Arabian horses, Andalusian horses, etc., or even Rabicano Arabian horses, Carthusian Andalusian horses, etc. In line with our *anti*-reductionist neo-Aristotelian framework, such a(n) ((in)direct absolute extensional) reductionistic temptation should be, however, resisted.

[134] Caveat on unfelt symptoms and coma: from this thesis it implies that e.g. a (deeply) comatose (or a permanent/persistent vegetative), maybe catatonic or just sleeping person (or under general anesthesia), because this person is unaware, *cannot* by definition have certain (high(est)-level) disease kinds viz. disease kinds (e.g. measles) constituted by negative *felt* effects. (Note that the point extends to diseases *tout court*, of course.)
With a very permissive ontology of disease(s) (kinds), we can deal with such cases as following: e.g. it may happen that a (deeply) comatose or sleeping person can very well develop a variant of measles viz. *unfelt* measles;
indeed, if fever and rash (of *x*) are truly negative effects constitutive of measles, then we may argue that there is a sense in which a (deeply) comatose or sleeping person can develop *specific* measles viz. *unfelt* measles constituted by (among others) *unfelt* fever and *unfelt* rash (of *x*), where "fever" and "rash" (of *x*) could be roughly defined as, respectively, "malfunctioning increase of bodily temperature of *x*" and "appearance of skin lesions of *x*".
As such, the difference between a comatose person having measles and a non-comatose person having measles is that the former has specific measles viz. *unfelt* measles, while the latter has (most often) another *variant* of measles viz. *felt* measles (for our understanding of unfeelings, cf. Sec. 7.1.3).
I acknowledge Ulrich Krohs and Niko Strobach (personal communication, July 2017) for having reported to me this illustration.

while a *mental* disease kind *tout court* is essentially a *mental* symptom *tout court*, for it does not pronounce on the felt character of its mental symptom.

Even if we have here high(est)-level possible diseases viz. e.g. negative *feelings* themselves or bad preferences i.e., roughly, a poor weighting between two specific options (or other negative (propositional) attitudes), while preferences are adjusted with respect to, or are differently weighed depending on, the specific host in question, it does not mean that these diseases and their signs(/causes) are themselves *subjective* (or mind-dependent), or – quite misleadingly said – inner, in the eye of the sufferer, or accessible to a *first-person* or internal perspective (e.g. through patients' own experience reports to physicians) (*contra* Carel, 2016; Kay Toombs, 1993) i.e. *defined* (strictly speaking) through other (negative propositional) attitudes themselves, but their (e.g. linguistic) signs(/causes) can equally well be treated as mere specific negative signs(/causes) of a disease defined mind-independently or – quite misleadingly said – through a view from nowhere (Nagel, 1986) or a *third-person* or external perspective (*pace* Canguilhem (1966), where for him a condition will count as malfunctioning (to the extent that a disease kind is linked to malfunctions), only if it is subjective) (cf. Exc. 7.2; on Canguilhem's aftermath, Gaille, 2014; Giroux, 2010; on social (or mental) practice as a *second nature*, McDowell, 1998; Testa, 2015): e.g. in a (complete or total) locked-in state or quadriplegia, or during an episode of sleep paralysis, albeit there is a loss of an *indirect coordination* between awareness and communicability, the host is commonly said to be perfectly aware, but unable to communicate by any means whatsoever (e.g. verbally, etc.).

Thus, we can treat cases where e.g. mottling is said an unfelt sign of mosaic dasheen virus infection, which is just a specific infection of leaves, on a par with cases where e.g. a feeling of tiredness is said a felt sign of a feeling of depression[135].

[135] Caveat on depression: it is important to distinguish between (i) unfelt depression and (ii) a feeling of depression, although we can argue that both are specific diseases viz. (iii) depression *tout court*.
However, if we have to explicitly distinguish between cases of *unfelt* depression and of *felt* depression, and if they are two very different specific diseases (e.g. felt depression would be constituted by emotional exhaustion i.e. specific sensations of mental clouding, etc.), where the former is commonly considered as a specific *unfelt* affective or mood disorder, and the latter a specific bad feeling (or a specific *felt* mood disorder), then depression *tout court* is neither a specific unfelt nor felt mood disorder (on depression in all its generality, cf. Forest, 2016; Horwitz & Wakefield, 2007; Ratcliffe & Stephan, 2014). What is it, thus? We can stay content here that it is simply a specific mood disorder *tout court*.
The main reasons for distinguishing between points (i), (ii) and (iii) are that, when depression is unfelt, it does *not* mean that the host in question is delusional, distracted, or that the host just does *not* pay attention to it, while it *ought* to, but only that the host is just *un*aware of it (with specific mechanisms different from the ones constituting a felt depression) – which is different from cases of *care*lessness; and, as such, there is no reason to hold that the host should pay attention to his (unfelt) depression instead of something else or nothing at all.
Moreover, when depression is felt, there is no reason to hold that, actually, depression *tout court* is *essentially* unfelt.

5.1 Excursus: Levels of (Causes/Effects of) Disease

How do we measure that some (effects/causes of) diseases can be said higher/upper, or at a higher/upper *level*, than other ones (or, more coarse-grained than other ones)? The measure in question is intuitively done through the use of *levels of* (*partial*/*absolute/relative*) *intension* and *absolute extension* (at a token or type level): a token/type (effect/cause of) disease is at a lower level than another *one*, iff a token/type (effect/cause of) disease can be deemed as (i) a(n) (indirect) constituent of a token/type (effect/cause of) disease (as a whole): e.g. dysplasia can be plausibly considered a specific lower-level disease with respect to cancer;

or as (ii) an (indirect) absolute intension of a token/type (effect/cause of) disease (as a whole): e.g. dysplasia (viz. hyperplasia here) with benign neoplasm (as a specific whole disease itself) with respect to cancer;

or as (iii) a(n) (indirect) relative intension of a token/type (effect/cause of) disease (as a whole): e.g. plausibly neoplasm with metastases i.e. a malignant neoplasm (as a certain disease kind itself) with respect to a cancer kind);

or as (iv) a less direct/more indirect absolute realizer of another one: e.g. a *genetic disease* with respect to a *mental disease*, where "mental disease" is a (more) direct absolute realizer of "disease" than "genetic disease").

The different measures (i)-(iv), along which we are to understand levels of (causes/effects of) diseases, are made, throughout this PhD dissertation, contextually clear or explicit, only when a confusion between one or the other may easily occur.

Two remarks are in order. First, if we are agree that measures (i)-(iv) are measures for levels, then the comparison between the levels of specific token/types (effects/causes of) disease is not merely *mereological* (i.e. here made within (our understanding of) a genus-species hierarchy of specific (effects/causes of) diseases.

Second, is a higher-level token (effect/cause of) disease than another one necessarily a *spatially bigger token* (effect/cause of) disease than this other one? Are not there spatially bigger token (effects/causes of) diseases than other ones, but which we would judge as being at a lower level than these ones? Indeed, we would like to intuitively say that e.g., although a token (effect/cause of) disease of the digestive (or gut) system of earthworms – which spreads over the whole earthworm -, is bigger in size than a token (effect/cause of) disease of the central nervous system of earthworms, the latter seems at a higher level than the former. But, is it? If the relationship between the latter and the former is along measure (i), (ii) or (iii), then the former cannot be a higher-level token (effect/cause of) disease than the latter one; however, if their relationship is along measure (iv), then a higher-level token (effect/cause of) disease is not necessarily a spatially bigger one, for there is no intensional relationship between them.

5.2 A Definition of a Symptom

If we want to give a correct definition of a symptom (i.e. within (our understanding of) a genus-species hierarchy), as being a mere *synonym* of a negative sign of a disease, a symptom cannot be *correctly* defined along the line of our prima facie definition of a symptom (cf. Box 5.1), but a definition of a symptom shall absolutely apply the definition, or rather the intension of a negative sign (result or effect) *tout court*, for a symptom is intuitively not something *more* than a negative sign: it is a *specific* negative sign viz. a negative sign of a *disease*.

By using our prima facie definition of a symptom (cf. Box 5.1), in Sec. 5.2, I detail the sense in which we say that a symptom is a negative sign of a disease. So as to avoid mistakes in the definition of a disease, it is important to devote Sec. 5.2 to a more thorough analysis of what a symptom as a negative sign of a disease really is.

I argue here that a negative sign/effect/result of a disease (or, a symptom) is to be properly defined as a disease in conjunction with a negative entity directly temporally following (another entity) and related to a living being. More generally, I argue that a (negative) sign is a (negative) entity plus another directly temporally succeeding (negative) entity.

As being a synonym of a negative sign of a disease, a symptom cannot, thus, be strictly defined as (or constituted by) a disease in conjunction with a negative sign/effect *of it* (cf. Box 5.1), for this would make, thus, the differentia in question (viz. a negative sign of a disease) defined dependently upon what the genus at issue (viz. a disease) is, though the prima facie definition of symptoms can be easily used as a basis for providing a correct definition of symptoms, and objections against our definition of symptoms can be based, of course, on this (plausible) understanding of our definition of symptoms (cf. Sec. 5.3; for the same point, Sec. 4.1.2).

I would like to argue in Sec. 5.2 that a symptom is to be properly defined within (our understanding of) a genus-species hierarchy as a disease (i.e. the *genus*) and a directly temporally succeeding negative entity related to a living being negative (i.e. the *differentia*) (cf. Box 5.2). At a more general level, if “symptom” truly absolutely falls under “(negative) sign”, then a (negative) sign is to be consequently defined as a (negative) entity (i.e. condition (i)) (i.e. the *genus*) plus another directly temporally following (negative) entity (i.e. condition (ii)) (i.e. the *differentia*).

To summarize, we get the following correct definition of a symptom[136]:

Definition of a Symptom

x is a symptom, iff (i) *x* is a disease (i.e. the genus), and (ii) *x* is a directly temporally succeeding negative entity related to a living being (i.e. the differentia).

Box 5.2. – Definition of a symptom within (our understanding of) a genus-species hierarchy.

A nice (plausible if not correct) illustration of our definition of symptoms (cf. Box 5.2) is very likely *Parkinson-plus syndrome*. Indeed, Parkinson-plus syndrome is commonly acknowledged, in any well-reputed student medical textbook like the "Goldman-Cecil Medicine" (Goldman & Schafer, 2019), the "Oxford Textbook of Medicine" (Firth et al., 2020) or "Mosby's Medical Dictionary (2016), as a specific (single) entity, which may be (plausibly, if not correctly) understood, in our jargon (cf. Ch. 2; Box 3.1), as a specific (single) complex negative *whole* or kind viz., more precisely, as a specific (single) *symptom* (cf. Box 5.2).

For, Parkinson-plus syndrome may be defined, roughly, as Parkinson's disease (cf. condition (i)) plus, directly temporally following it, Pick's disease (i.e. an over-accumulation of tau proteins in the brain) (cf. condition (ii)).

Other plausible illustrations of our definition of symptoms include e.g. "fever of disease *x*"[137]; indeed, we could roughly define "fever of disease *x*" here as a single whole constituted by disease *x* (cf. condition (i) of Box 5.2) temporally followed by an increased bodily temperature set point through heat generation (cf. condition (ii)), while "fever of disease *x*" also indirectly

[136] Caveat on negative signs here: it does not make this definition of symptoms (cf. Box 5.2), of course, a disease *kind* itself, in so far as a disease kind is a disease plus a negative *cause* and an effect – and not of a disease - (cf. Box 4.2), but only something *more* than a disease, and merely being something more than a disease is not the same as being a disease kind along, of course, our own definition of it (but, cf. also Sec. 4.2.1). Ditto for etiopathology (cf. Box 6.2).

[137] Caveat about the absolute realizer(s) of symptoms: (i) while a specific disease like fever (of disease *x*) is only (highly) *indirectly* a (negative) cause (of disease *y*) and/or an effect (of disease *z*) (cf. Sec. 4.2.10), is there anything which is *directly* a cause or an effect of a disease? In so far as "symptom" must have a direct absolute extensionality, there must exist something which directly absolutely falls under "symptom" as defined (cf. Box 5.2).

However, within our neo-Aristotelian framework or constituent ontology (cf. Ch. 2-3), there does not seem anything directly absolutely falling under "symptom", for it is very likely that any specific symptom is arguably *more directly* a specific disease itself (but, cf. Sec. 5.3.2).

Nonetheless, we do not have to pronounce on which direct absolute realizers of "symptom" there are, but it is sufficient to show that there are *plausible* specific symptoms: e.g. even if fever (of *x*) may be plausibly argued to be itself a specific (secondary, e.g. along a *functionalist* line,) disease – for, strictly speaking, fever *tout court* as well as fever *of* disease *x* may be *both* considered specific (indirect) diseases per se, only if, of course, "fever of *x*" absolutely falls under "disease" -, we can also *plausibly* argue that, actually, "fever (of *x*)" is a *direct* absolute realizer of "symptom".

refers to a certain disease itself (which happens most of the time with symptoms) (but, cf. Sec. 4.2.10).

Condition (i) of our definition of symptoms (cf. Box 5.2) can be easily explained by the idea that, if a symptom is synonymously a negative sign *of* disease (cf. Box 5.1), and if a sign/effect is an *extrinsic property* of a disease (cf. Exc. 3.9), then a symptom is to be intuitively defined within (our understanding of) a genus-species hierarchy as something more than a disease, where a disease is here the *genus* in question – and not the differentia.

For the sake of providing an independently defined differentia, condition (ii) of our definition of symptoms (cf. Box 5.2) is required. Indeed, if a symptom is truly something *more* than a disease (i.e. the genus) (i.e. condition (i)) viz. a negative *sign* of a disease, and if it is to be correctly defined within (our understanding of) a genus-species hierarchy, then it should have an independently defined differentia as given in condition (ii).

The first part of condition (ii) mentions the notion of *directness*; indeed, one should strictly distinguish between direct signs of disease and indirect ones (cf. Sec. 6.3.2 on indirect etiopathologies). The second part of condition (ii) is about *temporal succession*; indeed, it is almost trivial that an effect of *x* is temporally following *x* (on the notion of "related to a living being", cf. Sec. 4.1.2). With our definition of symptoms (cf. Box 5.2) – compatible with the idea that an effect is plausibly a (negative) *state* (and not necessarily a process); ditto for etiopathologies (cf. Box 6.2) -, the requirement of directness (and of *intensionality*, if the definition of a symptom (taken as a single *whole*) is strictly interpreted within our meta-philosophical framework) allows us to avoid the first strong objection that mere (temporal) *correlation* is not causation.

5.3 Objections against the Definition of a Symptom (with Replies)

Sec. 5.3 is divided into further sections (Sec. 5.3.1-5.3.11) raising and consequently addressing objections against the necessity (Sec. 5.3.1-5.3.5) and sufficient conditions (Sec. 5.3.6-5.3.11) of our definition of symptoms as negative signs of disease (cf. Box 5.2).

5.3.1 Objection (i): Symptoms as Meanings of "Disease"

In a growing body of philosophical literature on the notion of a sign *simpliciter*[138], most notably in philosophy of language, with its long tradition associating "sign" with notions like "reference" or "meaning" (e.g. Grice, 1957; Lepore & Stone, 2015; Peirce, 1977), theories of signs have been proposed on the basis that (natural or conventional) signs as understood here - i.e. taken synonymously with *effects* (cf. Sec. 4.1.2) - are parts of a theory of (natural or conventional) meaning *à la* Grice (1957): e.g. "[t]hose spots [i.e. the signs here] *mean* (meant) measles" (Grice, 1957, p. 377; my emphasis).

Along objection (i), which can be raised against the necessity condition of our definition of symptoms (cf. Box 5.2), a symptom or a negative sign of a disease would be, thus, part of what "disease" means.

In other words, for "disease" to have a meaning there must be a negative sign of a disease, which confers to "disease" its meaning.

Although objection (i) is obviously based on a *functionalist* theory of disease – which has been already rejected (cf. Sec. 4.2.7) -, again, objection (i) is to be addressed at the same level at which it is raised i.e. against our definition of *symptoms* (cf. Box 5.2) – not of disease or kind/species itself.

An issue that we can raise against objection (i) is that, if the meaning of a term like "lung cancer" is (partly) conferred by the existence of an effect or sign of it, then the term "lung cancer" would be meaningless, if there were e.g. only lung cancers in a plausible world.

Indeed, we would like to intuitively say that there is a plausible world in which *only* lung cancers (and nothing else or just effects of it) exist (and have a meaning), but, according to objection (i), this is an *implausible* world, for "lung cancer" can only have a meaning, if there is *also* a sign/effect of it; or lung cancer exists (and has a meaning) in a plausible world, if there exists in this world, in addition to it, an effect or a sign of lung cancer conferring to it its meaning. Exactly like there can exist fire without smoke (e.g. through complete combustion), we can argue that there *can* plausibly exist (with a meaning) (specific) diseases without anything else in addition to them or just (specific) negative signs of disease.

[138] Terminological note on signs: by "sign" I mean, of course, in line with our theory of kinds restricted to *natural* kinds (cf. Exc. 2.7), "*natural* sign" by contradistinction with "artefactual (or, more specifically, conventional) sign" (cf. Grice, 1957).

Thus, "lung cancer" may arguably be present (and have a meaning), without that a negative sign of it (or anything else) be also present[139].

However, a proponent of objection (i) could still reply that, if there were, indeed, nothing in addition to "lung cancer" conferring to it its meaning, then its meaning would be *trivial* – and a trivial meaning is not a true meaning. So, there must exist something in addition to "lung cancer" conferring to it its meaning.

As a reply to this last defense of objection (i), we may argue that that much still does *not* imply that the meaning of "lung cancer" is conferred to it by its *effects* – but only that there would be, indeed, something additionally existent – but hardly said an effect - conferring to "lung cancer" its meaning: e.g. constituents (however, on the possibility of *downward causation*, cf. Sec. 5.3.5).

5.3.2 Objection (ii): "Symptoms" as Absolute Realizers of "Disease"

A second objection, which we can raise against the necessity condition of our definition of symptoms (cf. Box 5.2), is that "symptom" is an absolute realizer of "disease", or a symptom is a typical illustration of a disease: under the assumption that e.g. syphilis is here a specific disease, then one of the commonly said symptoms of syphilis viz. *chancre* may be arguably

[139] We could argue that this point also seems to go, actually, against our own constituent ontology (or even against absolute/relative extensionality as a specific *necessitation* relationship), in so far as we have defended that a whole is (numerically, thus qualitatively (plausibly taken), by the PII_1) *distinct* from the sum of its constitutive parts (cf. Ch. 2).
Thus, there is no possible world in which *only* lung cancers (as having a meaning) exist. Indeed, for "lung cancer" to have a meaning its constituents must also exist in the same possible world.
However, although a *correct* definition of lung cancer is to be put within (our understanding of) a genus-species hierarchy, the definition *itself* can be plausibly interpreted (or, is compatible with conceptions of it situated) outside this metaphilosophical framework.
Moreover, to address objection (i), for the sake of the argument, it is sufficient to *plausibly* show that there exists a world in which only lung cancers exist (or lung cancers without effects of it), where "lung cancer" is meaningful – thus, that there exists a plausible conception of lung cancer *incompatible* with objection (i): e.g. the defensible idea that the meaning of "lung cancer" is conferred, through the PII_1, by something to which it is qualitatively distinct (plausibly taken), although numerically (or loosely like part(ial)ly) identical; or, that the meaning of "lung cancer" just is trivial, for there is nothing in addition to lung cancer, which can confer to it its meaning.
This is the case, even though a certain plausible conception of e.g. lung cancer *could* still be also used as another objection (or counter-example) itself against our own conception of lung cancer.
But, in the present context, our reply to objection (i) is still *compatible* with a *plausible* reading of our own definition of disease (cf. Box 9.3) viz. here the reading that a disease would be (numerically) identical to (the elements of) its definiens.

said to be an absolute *realizer* of, or a way to absolutely realize, the disease process – and not a negative sign/effect of it (cf. Box 5.1).

Objection (ii) has the prima facie advantage to understand in an intuitive way the *metaphorical* or analogical usage of the word "symptom", or the use of "symptom" as a mere *façon de parler* (on "disease" with its cognates and metaphors, cf. Boyd, 2000).

Indeed, it seems to be the case that "symptom" is metaphorically used to refer to a *typical* (or characteristic) *illustration* of something deemed bad (or only metaphorically taken as a disease) (however, cf. Sec. 5.3.8 for another interpretation of the metaphorical usage of "symptom").

Moreover, objection (ii) can be said to follow somehow the Greek etymology of symptom viz. "*σύμπτωμα*" translated as e.g. "that which concurrently occurs with something else", for an absolute realizer is precisely something happening *simultaneously* with the realized property.

Nevertheless, in spite of this, and beyond the problems with absolute extensional definitions (cf. Sec. 3.2.3), objection (ii) does not go without its own problems.

Indeed, if "symptom" is an absolute realizer of "disease", then a symptom just is, actually, a specific disease, among which we would like to intuitively also count e.g. mental diseases, or bad feelings, etc.

But, it is obvious that a symptom cannot be put on a par with, or on the *same* (absolute extensional) *level* as, e.g. mental diseases. Were this true, then, exactly like a(n) (in)direct absolute realizer of "mental disease" cannot fall (as (*in*)*directly* as with "mental disease") under "physical disease", a(n) (in)direct absolute realizer of e.g. "mental disease" could not also (as (*in*)*directly* as with "mental disease") absolutely fall under "symptom": along objection (ii), it should (indirectly) absolutely fall under *either* "mental disease" *or* "symptom".

However, we would intuitively judge that one and the same absolute realizer (e.g. tic disease) can fall under *both* e.g. "mental disease" and "symptom" (e.g. tics i.e. abnormal repetitive motor or phonic movements as signs of an obsessive-compulsive disorder). But, for that to be true, "symptom" and e.g. "mental disease" cannot be put on the same (absolute extensional) level: e.g. "tic disease" may be plausibly said to absolutely fall more directly under "mental disease" than under "symptom". Therefore, "symptom" cannot be an absolute realizer of "disease".

5.3.3 Objection (iii): Symptoms as Manifestations of Disease

A third objection that we can raise against the *necessity condition* of our definition of symptoms (cf. Box 5.2) is the following one: a symptom is the manifestation (or actualization) of a disease. Thus, when the disease is actualized, then the symptom concurrently occurs (or is manifested). Like for objection (ii) (cf. Sec. 5.3.2), objection (iii) can also be said to rely on the Greek etymology of the notion of symptom i.e. "*σύμπτωμα*" translated as "coincidence" or "that which concurrently occurs with something else". This position about symptoms has been common in the 1980s especially about mental disease kinds (cf. Boorse, 2011 on that):

> [a] mental disorder [kind] is a medical disorder whose *manifestations* are primarily *signs* or *symptoms* of a psychological (behavioral) nature, or if physical, can be understood only using psychological concepts. (Spitzer & Endicott, 1978, p. 18; my emphasis)

In other words, and to generalize this position outside the mental domain, symptoms are, indeed, those manifested signs of a disease.

The main issue with objection (iii) is that, beyond the problems related to the adoption of a dispositionalist theory of disease i.e. its reliance on RME (cf. Sec. 5.3.10), it is *ambiguous*: if a symptom (exactly) concurrently occurs with a disease, then either "symptom" can be plausibly argued to be an absolute realizer of "disease" (cf. Sec. 5.3.2), or "disease" can be plausibly argued to be an absolute realizer of "symptom".

Indeed, if a symptom exactly concurrently occurs with a disease, then a symptom cannot be said (in our terminology) constituted by a disease, or a disease is not a constitutive *part* of a symptom. We can, thus, reply to objection (iii) by saying that there is no definitional relationship between a disease and a symptom (for the same problems about the related idea of a disease kind as a disease plus a simultaneous process, cf. Sec. 4.2.4).

5.3.4 Objection (iv): Symptoms as Negative Signs of Clinical Conditions

A fourth objection that we can raise against the necessity condition of our definition of symptoms (cf. Box 5.2) is as follows: symptoms are not negative signs of disease (cf. Box 5.2), but they are negative signs of *any* clinical condition, which may be *good* or *bad* (cf. Sec. 4.2.2 for a related objection, but about *disease kinds*). It is not rare to use the word "symptom" to refer to the negative signs (e.g. gestational diabetes, gestational hypertension, tiredness, etc.,

for pregnant (*wo*)*men*) of e.g. (fe)male pregnancy (where pregnancy is not considered as a specific disease).

In this case, the term "syndrome" is also sometimes used to refer to a set of symptoms that are signs of any clinical condition (but except a disease) (cf. Sec. 4.1.3).

Objection (iv), thus, denies that symptoms are properly negative signs of *disease* (cf. Box 5.2), but claims that symptoms are negative signs of whatever (good or bad) clinical condition – thus, where objection (iv) should not be taken as an objection against the sufficiency condition of our definition of symptoms (cf. Box 5.2).

However, can we really properly use the word "symptom" in this sense? A first weak reply to objection (iv) consists in saying that objection (iv) confuses a *literal* sense of "symptom" with, plausibly speaking, a *metaphorical* one i.e. here a symptom as a negative sign of a clinical condition (cf. Sec. 5.3.2; 5.3.8 for other metaphorical usages of "symptom").

Objection (iv) may reply here: but, why should the *literal* sense of "symptom" be reserved to talk about a negative sign of *disease*? Where does literacly really end, and metaphoricality begin, in our case here (about the general idea that literalcy ends when there is no possible transfer of justification from one case to another one, cf. Feldbacher-Escamilla & Baraghith, 2020)?

Nevertheless, it is hardly defensible that the literal sense of "symptom" is about any clinical condition. Indeed, one may be against expanding the meaning of "symptom" to any clinical condition, for this would result in a non-fine-grained enough definition of symptoms, which still has to neatly and non-confusedly distinguish "symptom" from other related but more general and non-overlapping cognates like "biological effect/sign" or etc.

A second reply to objection (iv) consists in arguing that, if e.g. pregnancy is deemed *positive* (or a non-disease), then its effects cannot be *negative*, on pain of a category mistake.

However, if we maintain that a symptom is a negative sign of *disease*, how to explain away the intuition that a non-disease like pregnancy may have *negative* signs? Do we have to bite the bullet by arguing that pregnant (fe)males are, actually, diseased (on basis of their physiological changes), and on the grounds that we are used to talk about negative signs of pregnancy? Or, is not a pregnant male/female just a *specific* (fe)male? More precisely, is not *being pregnant* (or *in gestation*) an *accidental property* of a certain *part* of the (fe)male body (e.g. a specific uterus of a (fe)male)? Moreover, are they not specific diseases or deadly processes of the pregnant (fe)male (e.g. miscarriage)?

Negative signs associated to pregnancy are not specific symptoms, for pregnancy is not a certain disease, but they are negative signs of specific *diseases implied by* the gestational process

(understood as a normal or positive process) (cf. Sec. 4.2.2 for a related reply; about the neo-Aristotelian WCP, Exc. 2.3).
Note, finally, that most of the diseases implied by gestation or pregnancy (e.g. diseases having as negative signs (gestational) diabetes, (gestational) hypertension, or tiredness) can very well be implied by *another* normal or positive process (which is not (fe)male pregnancy), for a contrary may have *several* contraries.

5.3.5 Objection (v): Symptoms as Constituents of Disease

A fifth (and last) objection that we can raise against the necessity condition of our definition of symptoms (cf. Box 5.2) is the following one: Jonathan Fuller (2018a) has elaborated an interesting so-called "constitutive disease model", according to which, roughly, symptoms are constituents of disease – rather than signs following a disease. According to Fuller, this theory of disease is not new; it goes back to the early 19th century, where specific diseases were defined with respect to their symptoms taken as components (cf. Porter, 2002). Indeed,

> [b]efore Koch isolated the tubercle bacillus, tuberculosis was understood symptomatically and pathologically in terms of 'tubers' in the lungs. […] HIV disease is roughly defined as an infection with HIV […] [i.e. that "tubers" would be, indeed, signs of tuberculosis understood as constituents of it, just like HIV would be a symptom-as-a-constituent of HIV disease]. (Fuller, 2018a, pp. 10-11; 13)

If symptoms are truly to be taken as constituents of disease, and if symptoms are intuitively related to *signs* or effects of disease, then objection (v) just relies on *downward causation* i.e. on the idea of an emergent property *z* causing a lower-level property *x*. Thus, objection (v) can be rejected by ruling out the possibility of downward causation.
A problem with downward causation is that it seems to go against the idea of *temporal asymmetry*, which is one of the desiderata for causation. Indeed, if e.g. HIV disease (as a specific disease here – not disease *kind*) were really to be defined as requiring, *amongst others*, the presence of HIV as a constituent, then there does not seem to be there a temporal asymmetry, but, rather, a temporal synchonicity between a whole and its constituents.

However, temporal asymmetry can be maintained in the case where e.g. HIV disease just is the disease having only one specific symptom. This is a pretty implausible move, for defining a disease with only one constituent would amount to either a clearly insufficient definition or a trivial one (cf. also Borsboom, 2017).

Thence, the main issue with objection (v) is that it commits a *category mistake* by relating to each other two very different notions viz. the one of constitution and of symptom.

As a last defense of objection (v), we could follow Borsboom (2017) by claiming that symptoms are truly *constituents* of a disease, but they are *causally* connected to each other (as nodes) in a certain network – and not, or so it seems, to a certain disease. In other words, Borsboom tries to make sense differently of the complex relationship between symptoms, constitution and causation by not conflating constitution with causation.

As ingenious as this solution may sound, it does not go without difficulty. Indeed, along Borsboom's theory, how to make sense, thus, of the idea that symptoms are related to signs/effects of *disease*? This intuitive idea is completely evacuated, because of the claim that symptoms are causally connected to *each other*.

5.3.6 Objection (vi): Symptoms as Negative Kinds of Sign

Sixth, one can raise an objection against the *sufficiency* condition of our definition of symptoms as negative signs of a disease (cf. Box 5.2) by arguing that a symptom is to be understood as a negative sign *of a certain kind* viz. of a disease – thus, as something *more* than a sign. Along the same line, with objection (vi), one could add that a feeling of a symptom is also to be understood as a *feeling of a certain kind* viz. of a symptom; a feeling is a mode (or attitude) directed toward an object (or, having a content), and a feeling of a symptom would be, thus, according to objection (vi), a mode directed toward an object of a certain kind (or, having a content of a certain kind).

But, is a symptom really a sign of a certain kind? It seems prima facie so: a symptom is a negative sign of the past presence of a disease. If not, what is it? Is a symptom a disease whose past presence is indicated by a negative sign (cf. Box 5.2)? If so, then a symptom looks like (partly) a disease kind (cf. Box 4.2).

Objection (vi) can be overcome as following: a symptom is not a sign of a certain kind, but "symptom" is obviously in the *absolute* extension of "sign of a process" or "sign" *tout court*. The same answer applies to the case of the feeling of a symptom: "feeling of a symptom" is in the indirect absolute extension of "feeling of an entity" or "feeling" *tout court*, where, in turn, it indirectly absolutely falls under "entity" *tout court*.

5.3.7 Objection (vii): Symptoms as Negative Unfelt Signs of Disease

A seventh objection that can be raised against the sufficiency condition is the following one: it is not sufficient to define a symptom as a mere negative (clinical) sign of a disease (cf. Box 5.2), for a symptom is to be defined, rather (along objection (vii)), as a negative (clinical) *unfelt* sign of a disease. In that sense, a symptom would be said a negative (clinical) sign of a disease of a certain kind.

Indeed, along objection (vii), if the definition of symptoms as negative signs of disease is not sufficient (cf. Box 5.2), that is because it is too much wide: feelings of a symptom would count as symptoms, but a feeling of a symptom should be defined, along objection (vii), as something more than a specific symptom viz. as a *symptom of a certain kind*: a negative (clinical) unfelt sign of a disease, which is precisely felt by a host.

According to objection (vii) e.g. a(n) (unfelt) bloody nose (of disease x) at time t_0 is a specific symptom, while, when felt by the host at time t_{+1}, it becomes a symptom of a certain kind.

But, if precisely a bloody nose (of x) becomes felt at time t_{+1}, then it cannot be consistently at the same time *more* than a specific negative *unfelt* sign of a disease viz. a specific negative *unfelt* sign of a disease (i.e. the genus) which is precisely *felt* (i.e. the differentia).

Moreover, if a disease kind y is constituted by (partly) a specific *symptom* (cf. Sec. 4.1.2) – and not a specific symptom *kind* -, the bloody nose (of x), *because* it is felt at time t_{+1}, would cease to be at time t_{+1} (strictly speaking) a specific symptom *tout court* (of a specific bad feeling) to become a symptom of a certain kind. Thus, a feeling of a symptom can never (partly) constitutes a disease *kind*.

However, if true, then objection (vii) has to reject the claim that a bad feeling may be a specific disease kind (cf. Sec. 4.1.2).

5.3.8 Objection (viii): Symptoms as Negative Felt Signs of Disease

An eighth objection which naturally comes after objection (vii) (cf. Sec. 5.3.7) is, contrariwise, to claim that it is not sufficient to define a symptom as a negative sign of a disease (cf. Box 5.2): a symptom is, along objection (viii), a negative *felt* sign of a disease (cf. Sec. 7.1.1). Again (cf. Sec. 5.3.6), a symptom would be here a (clinical) negative sign of a disease of a certain kind.

Indeed, should we not say that a symptom is essentially a feeling indicative of a disease? Could not we draw a neat distinction between, on the one hand, symptoms and, on the other hand, negative (clinical) *unfelt* signs of a disease (cf. e.g. Whitbeck, 1977) i.e. precisely those signs of a disease that are unexperienced or unfelt by the host, or those negative signs of a disease, which the host is unconscious (or unaware) of?

But, if so, then what is a negative unfelt sign of a disease? Objection (viii) shall answer here, by contradistinction with objection (vii) (cf. Sec. 5.3.7), that a negative unfelt sign of a disease is a *symptom of a certain kind*.

However, first, if we want to reject the definition of symptoms as negative signs of disease (*pace* Box 5.2), by arguing, rather (along objection (viii)), that symptoms are negative (clinical) signs of a disease of a certain kind, then we should argue against the possibility to understand the *metaphorical* usage of "*x* (as a negative kind) is symptomatic of (¬)*y* (as a negative kind)" as "*x* is a(n) (mere) indication of (¬)*y*": e.g. "dilapidated buildings are symptomatic of a city's decay", where what is metaphoric is here the understanding of the term "symptom" as a mere *negative* (non-clinical) sign (i.e. *x*) of something deemed *negative* (i.e. (¬)*y*) (which can be itself (*plausibly*) understood metaphorically as a disease: e.g. a city's decay[140].

[140] Two caveats on literal vs. metaphorical uses of "sign (of disease)" or "death": (i) it may seem difficult to distinguish, strictly speaking, between *literal* and *metaphorical* cases of, amidst others, disease(s) (signs), death, etc.; indeed, with a case like "serious labor shortage is symptomatic of population decline (or *de*population), is depopulation truly a specific (higher-level) disease (or death)?
With a fine-grained differentiation of disease(s) (signs), and if we hold a minimal view on what can host a disease (sign) (or death here) (cf. Sec. 9.3), then a strict distinction between literalcy and metaphoricality about e.g. disease (signs) (or death) can be maintained: e.g. a population can be plausibly said a certain meta-organism, while this is hardly the case for the above example of a city.
(ii) Furthermore, it seems that the notion of symptom (or even etiopathology) – however plausibly or correctly understood - cannot be used literally or even metaphorically in value-*neutral* contexts.
Indeed, if a symptom just is a sign of disease, and if disease (*simpliciter*) is deemed something *bad* (or negative), then, to avoid a category mistake, "symptom" – be the term used *literally* or *metaphorically* - belongs to the same

That much implies that the metaphorical usage of "symptom" is taken to refer here to a mere (non-clinical) sign of (¬)*y* – and not to something more than a (non-clinical) sign of (¬)*y* viz. a (non-clinical) sign of (¬)*y of a certain kind*; however, other endeavors at understanding (or interpreting) the metaphorical usage of "symptom" are, of course, still possible (cf. Sec. 5.3.2 for such an attempt).

Second, negative (clinical) *unfelt* signs of a disease are clearly not considered as symptoms of a certain kind in the context of *plant* diseases, where it is controversial whether (vegetal) hosts can truly have feelings: e.g. there is maybe a plausible sense in which we can say that the young sunflower *feels* that it is growing, when turned towards some source of light.

However, let us grant here without argument that a basic (vegetal) host *cannot* have feelings (in our sense). It is said that a usual effect of a viral plant disease called "dasheen mosaic virus infection" is for the leaf to exhibit off (darker or lighter) colors referred to as mottling (or mottle) or just discoloration. In this context, it is obvious that negative (clinical) *unfelt* signs of a disease are considered specific symptoms – and not symptoms of a certain kind.

5.3.9 Objection (ix): Symptoms as Negative either Felt or Unfelt Signs of Disease

A ninth objection can be raised against the sufficiency of our definition of a symptom as a (mere) negative sign of a disease (cf. Box 5.2): indeed, we may wonder whether we should not define, rather, a symptom *simpliciter* as a negative *either felt or unfelt* sign (indicating the past presence) of a disease, instead of taking felt and unfelt symptoms as two different specific symptoms (cf. Sec. 5.1.2).

A strong argument in favor of objection (ix) can be put forward as follows: (i) something that is usually judged as a negative (clinical) unfelt sign of a disease typically becomes at some point experienced or felt by a specific host: e.g. an athlete who is racing with a muscle contracture - usually said a negative (clinical) unfelt sign of spasticity (cf. Sec. 5.1.1) –, nevertheless, feels it after his race.

Likewise, (ii) something that is deemed a feeling of a sign of a disease may very well become unfelt in a situation where the host is just unaware of it: e.g. an athlete racing with a bleeding

(fundamental or general) ontological category. I acknowledge Niko Strobach (personal communication, July 2018) for having pressed me on this point.

nose (of *x*) – defined here as a specific feeling of a sign of a disease -, which is unfelt during his race.

In situations (i) and (ii), would we say, respectively, that the host in question ceases to have a certain symptom (as a negative unfelt sign of a disease) and gets *another* specific one at a different level (as a negative *felt* sign of a disease), or that, *because* a certain host is unaware of it, he does not have the same specific symptom as the one when he *feels* it? No, because, in situation (i), we would like to say that the *same specific* symptom is *still* present even when it becomes felt; and, in situation (ii), we would like to intuitively say that the same specific symptom is present even when it is unfelt. Situations (i) and (ii) seem different from the situations where a specific symptom is suppressed (*cured* or *treated*), and a new specific one arises.

We can address objection (viii) as following: first, for situations (i) and (ii), we have to bite the bullet: we have to distinguish between the cases where there is a sign *x* of e.g. a bad emotion or, more generally, a mental disease (not necessarily felt), and a sign *y* of a bad feeling.

Thus, as we have seen (cf. Sec. 5.1.2), that we are dealing with a symptom as a negative *felt* sign of a disease or as a negative *unfelt* sign of a disease is determined by the nature (and the level) of the specific disease (e.g. as a bad feeling, a bad emotion, a bodily injury, etc.), which the symptom is a specific sign of.

In a nutshell, we have to distinguish between an unfelt sign of a(n) (unfelt) disease *y* and a felt sign of a (felt) disease *z*: e.g. my feeling *a headache* (where the headache is understood as a (mere) negative sign of a disease *x*) is itself (in the indirect absolute extension of) a headache, while my *feeling* a headache is also itself a specific (felt) sign of e.g. my *feeling* unwell (or a disease *z* understood as a bad feeling), and my *unfelt* headache is (in the indirect absolute extension of) a headache and is itself a specific (unfelt) sign of e.g. my *unfelt* unwellness (or disease *y* understood as an unfelt emotion (or, more generally, mental) disease)[141].

Second, with our differentiation of diseases and their signs, we can consistently argue that e.g. a felt bloody nose and an unfelt bloody nose (of *x*) are two different specific *felt* and *unfelt* symptoms, while a bloody nose (of *x*) *tout court* is a specific symptom *tout court*.

[141] Caveat on emotions and feelings: if we say that bad feelings are to be counted as specific high(er)-level diseases themselves as well as bad emotions (cf. Sec. 5.1.2), and if we say that there is a neat or clear-cut distinction between bad emotions and bad feelings (cf. Sec. 4.2.8), then we may want to object that the distinction between bad emotions and bad feelings becomes, after all, futile.
Indeed, for some philosophers what I call "bad feelings" just are "bad emotions", to the extent that emotions and feelings go hand-in-hand for them (Deonna & Teroni, 2012). The main reason for keeping this distinction neat is that it allows us to distinguish between cases of mental or, more specifically, emotional disease and cases of illness (cf. Sec. 7.1).

The same answer applies to another familiar situation: e.g. a person gets a (token) headache as a result of a laborious, tiring and painful workday, relieves it for a while by taking some aspirin or drinking a lot of water, but eventually gets *again*/*still* a (token) headache.

There are two intuitive ways to interpret this short story: (i) the very *same* token headache eventually *came back*, when it is *felt* again; the same token headache was there since the very beginning; it is just that it was merely *unfelt* for a while.

(ii) The person eventually got *another* token headache, precisely when the person *felt* it once more; the token headache *was gone*, when it was unfelt; it is just that a new token headache appeared, when the person felt it again.

However paradoxical as it sounds, interpretations (i) and (ii) can be coherently rephrased with our idea that felt and unfelt symptoms are *specific* symptoms (cf. Sec. 5.1.2): in interpretations (i) and (ii), the same token headache (*tout court*) *x* is present since the very beginning; however, headache can be said to have two different shapes (or two different absolute realizers) with three different tokens: first, a token *felt* headache *y*; second, a token *unfelt* headache *z*; finally, once more, another token *felt* headache *a* (for a token is a non-repeatable entity or a particular); during this time, the same token headache (*tout court*) *x* was somehow always present.

The same solution also applies to other (even more controversial) general cases (e.g. specific diseases): e.g. when we say that a token pain was gone, but that *it* finally came back, we seem to say (prima facie inconsistently) both that a token pain was gone and another one came into existence later on, and that the very same token pain was somehow always present.

There is a sense in which we are talking, indeed, about the same token pain *tout court*, but where "pain" has here two different shapes (or absolute realizers) viz. "felt pain" and "unfelt pain"; there is another sense in which we are talking about a different *specific* token pain, which came into existence only when felt i.e. a *felt* pain.

Lastly, against objection (ix), a definition of symptoms as negative either felt or unfelt signs of disease commits exactly the same mistake as when we try to define a genus as a disjunction of its species (cf. Sec. 3.2.2). Indeed, what constrains the elements which can(not) enter into the disjunction is precisely their sharing the very property of being a symptom.

5.3.10 Objection (x): Symptoms as Negative Possibly Felt Signs of Disease

Tenth, one may object that a symptom *tout court* is merely a negative possibly felt sign of a disease, or that it is a *disposition* to feel a disease. Indeed, why not defining, after all, a symptom *tout court* as a negative sign (of the past presence of a disease) of a certain kind viz. possibly felt?

Objection (x) has a prima facie plausibility: a muscle contracture e.g. would be judged as a negative sign of a disease possibly felt (or, a negative sign of a disease that one is disposed to feel) under certain circumstances (cf. also Sec. 5.3.9).

However, are there not some negative (clinical) signs of a disease that we would judge as symptoms, but which are not possibly felt? Of course, it is not an easy matter of how to determine the *impossibility* for a symptom to be felt.

There are two main paths for negatively answering this question: the first, strong, path is Spinozist (*Ethics* 2p12): I *am*, actually, now aware - even at a very low or confusing level - of every symptom happening in my body.

The second weak - and more plausible - path consists in weakening the Spinozist thesis: there is always the possibility for a host to be aware - even at a very low or confusing level - of every symptom that happens in its body, in the sense that this host would be extra-lucid.

The main problem with this second path is that, when we have a symptom, it is not *essential* for it to be possibly felt (although, of course, "symptom" *necessarily* has an absolute extension like "felt symptom" or "unfelt symptom") (cf. Sec. 5.1.2). From the presence of a symptom as a negative sign of a disease (cf. Box 5.2) one cannot infer that it is essential for it to be possibly felt. It is not part of what a symptom is that it is possible to feel it, but a feeling of a symptom is a specific symptom.

In other words, one can argue that it is not part of the essence of a symptom that it can be felt, but that it is necessarily absolutely realized e.g. through a felt symptom (i.e. an attention mechanism making one's aware, generally speaking, of what is happening in one's body (and mind) (cf. Sec. 7.1.3), where one can be simultaneously aware, of course, of *several* somatic (or mental) processes)[142].

[142] Two caveats (with a terminological note) on (un)felt symptoms here: (i) if *x* is considered as having a specific symptom (or, generally speaking, as being specifically diseased), then the specific symptom (e.g. bodily symptom; mental symptom; etc.) is necessarily absolutely realized through e.g. *x*'s (un)feeling the symptom (or, the disease): e.g. if *x* as a person may be overweighted (understood as a specific bodily symptom), then, *necessarily* (but *not essentially*), *x* feels this symptom or *x* has a unfelt symptom, though a(n) (un)felt bodily symptom like (un)felt overweight is only *one* specific absolute realizer of overweight (among others, or at least one); or, generally speaking, *x* as a person cannot be said specifically diseased without necessarily e.g. (un)feeling like a diseased person.
(ii) It should be noted, at the outset, however, that *un*feeling like a *diseased person* and (positively) feeling like a *healthy person* are two very different things.

Saying that a negative sign of a disease is disposed to become under certain circumstances a *feeling* of a sign of a disease is, along a certain understanding of RME, making of a negative sign of a disease a part that essentially belongs to its whole i.e. a part becoming under certain circumstances (e.g. when combined with other parts) a whole (cf. Exc. 5.2).

A second problem for objection (x) has to do with the fact that there are many specific lower-level disease kinds happening in our body (e.g. deleterious genetic mutation kinds) that it is not even sure that it is possible to feel their symptoms in a sufficiently (conceptually) clear manner. Thus, here, we are rejecting the weak Spinozist idea that it is possible to feel, even at a very low or confusing level, every symptom happening in our body - the absolute realizers for such lower-level symptoms are perhaps precisely those that are unfelt. Indeed, if it is always possible to *confusingly* feel every symptom, how can we ascertain (certify or confirm) that an effect of e.g. some bad *genetic mutation* is ever felt - rather than being actually an effect of depression? Moreover, not every lower-level disease is necessarily relatively realized by e.g. a higher-level *feeling* of a disease – it can be very well relatively realized by a higher-level *unfelt* disease.

5.2 Excursus: Against (Pre)Dispositions

Along dispositional theories of disease (and health) (Boorse, 1977; 2014; Whitbeck, 1977), if e.g. to be myopic is to be disposed to have a blurred sight of an object, and if the disposition is actualized or manifested under the circumstance that this object is distant, then the property of being myopic can be said, along a certain understanding of RME, to belong essentially to a certain whole (under certain circumstances viz. when the object is distant); ditto for the case, where to have sight is to have the disposition to see, which is manifested when e.g. the bearer is awake – without being necessarily aware (of seeing or of being awake), of course.

If I am right that RME is not to be followed (at least for kinds), then myopia cannot be defined as a part that essentially becomes, in addition to given circumstances, a whole understood as a process here (viz. a blurred sight of an object).

More generally, there are no such things as (genetic, etc.) *(pre)dispositions* to develop such-and-such disease (kind) (e.g. human beings whose fair skin is exposed to sunlight are more risky to develop skin cancer than human beings whose darker skin is exposed to sunlight (Green & Trichopoulos, 2002)), in so far as a disease (kind), of course, is seen as being *essentially* a (pre)disposition, but there are only specific disease(s) (kinds) having *x* and *y* as (direct or indirect) *constituents*.

Globally speaking, we can argue that, even in a permissive neo-Aristotelian ontology, we can, actually, dispense with dispositions or dispositional properties (vs. e.g. Dumsday, 2016; 2019); thus, all properties

(even trivial ones) would be *categorical* (or, they can be simply put as properties *tout court* throughout this PhD dissertation) (on *categoricalism*, cf. Armstrong, 1997; on the difficulties in defining (mal/dys)*functions* as specific dispositions, Jansen, 2018; Kroes, 2001 (*pace* Lange, 2007; Müller-Strahl, 2014; Spear et al., 2016)): e.g. a disease belongs *necessarily* to e.g. a disease kind, a symptom or an etiopathological agent, but a disease is not disposed to become a disease kind, a symptom or an etiopathological agent, when all the other parts shall be also present – the whole (as a disease kind, a symptom or an etiopathological agent) just is *partly* there until all the other parts are also present (on the requirement of relative extensionality, cf. Sec. 3.3.1).
However, note that our specific use of Aristotelian metaphysics (cf. Sec. 3.1.1) is here at odds with the current revival of Aristotle's theories of potentiality/potency (*δύναμις*) (vs. theories of possibilities) and actuality (*ενέργεια*/*έντελέχεια*) in the metaphysics of science, which is dominantly noticeable in the subscription of, precisely, theories of essential properties as dispositional properties (on *dispositional essentialism*, cf. e.g. Austin, 2018; Bird, 2007) as well as theories of essential properties as *causal* (or functional, understood in a certain way) properties (cf. Sec. 4.2.7; on *causal essentialism*, e.g. Hawthorne, 2001).

5.3.11 Objection (xi): Symptoms as Negative Potential Signs of a Disease

An eleventh and last objection – closely akin to objection (x) (cf. Sec. 5.3.10) - against the sufficiency condition of our definition of symptoms (cf. Box 5.2) is that a symptom is a negative *potential* sign of a disease. In the now classical 1982 book "Medical thinking: A historical preface", Lester S. King seems to have argued for such a view:

> The belief that a symptom is a subjective report of the patient, while a sign is something that the physician elicits, is a 20th-century product that contravenes the usage of two thousand years of medicine. In practice, now as always, the physician makes his judgments from the information that he gathers. […] If the data, however derived, lead to some inferences and go beyond themselves, those data are *signs*. If, however, the data remain as mere observations *without interpretation*, they are *symptoms*, regardless of their source. Symptoms *become* signs when they lead to an interpretation. (King, 1982, p. 89; my emphasis)

In other words, by understanding in a specific way the above quote, objection (xi) is, more precisely, the objection that symptoms are signs-of-a-disease-yet-to-become, and symptoms become negative signs of a disease, when they got interpreted.

The main drawback with objection (xi) is that, beyond the problems akin to adopting a dispositionalist stance on symptoms (cf. Exc. 5.2), it mixes up ontology with epistemology. Indeed, by *defining* symptoms as negative potential signs of a disease, where this potential is actualized (or realized) when there is an *interpretation*, objection (xi) states that the existence of this specific entity that is a symptom is dependent upon a scientist's competence in being able to interpret this symptom (by making it a sign) (and, thus, to access, first, to this symptom taken as a raw datum), but it may be plausibly defended that a symptom is obviously out there in the world, without being necessarily the case that a scientist *accesses* to - and potentially interprets - this symptom (cf. Sec. 4.2.2).

6

ETIOPATHOLOGY

While Ch. 5 of Part II is devoted to analyze symptoms, Ch. 6 is about etiopathological agents or etiopathology. Ch. 6 is divided into three sections: Sec. 6.1 provides a prima facie definition of etiopathology as a (preceding) cause of disease.

Sec. 6.2 argues that an etiopathological agent is, within (our understanding of) a genus-species hierarchy, a disease (i.e. the genus) in conjunction with a directly temporally preceding negative entity related to a living being (i.e. the differentia).

Sec. 6.3 is about the issue of the possibility of multiple and indirect causes of diseases and, by extension, also effects of disease; it is argued here that there is a minimal sense of multiple causation i.e. where only the indirect constituents of an etiopathology (or a symptom) are multiple. Furthermore, I argue that indirect causes of disease *x* are just those etiopathologies themselves causing the cause of disease *x*.

Ch. 6 does not contain its own section of objections against our definition of etiopathology, for the reason that the objections raised (and consequently addressed) in Sec. 5.3 of Ch. 5 about our definition of symptoms (cf. Box 5.2), and many if not most of what is said about symptoms in Sec. 5.1 and 5.2, apply, actually, to our own definition of etiopathology as well.

Indeed, those objections as well as their replies (and the remarks made *passim*) can be very easily rephrased in the context of etiopathology. That is why, devoting a section to objections against our definition of etiopathology would be redundant. I take for granted that all this is extensible to etiopathological agents.

6.1 A Prima Facie Definition of Etiopathology

To the contrary of the case of symptoms (cf. Sec. 5.1), to motivate that we have to devote (at least) a whole chapter of a PhD thesis on etiopathology or causes of disease (by contrast with symptoms taken as effects/signs of disease) is not really necessary[143]. For, a whole PhD dissertation can – and has been, actually (Clarke, 2011) – devoted to the topic of causes in medicine, but it is not our present purpose.

Indeed, the topic of causation or, more precisely, the topic of what a cause is being a very widespread subject of interest to philosophers since the dawn of time, it is, thus, unsurprising that philosophers of medicine have become very quickly interested in causation in medicineor, more precisely, medical causes since the birth of the discipline "philosophy of medicine" (cf. e.g., as entries, Campaner, 2012; Whitbeck, 1977)[144].

However, once we have given an account of what a symptom is (cf. Box 5.2), do we need to spend some time on etiopathology, for it seems that to give an account of symptoms is already to give somehow an account of etiopathology (or vice versa), if there is, indeed, no effect without a cause (and vice versa)?

This legitimate worry echoes objection (xi) of Sec. 4.2.11. Although it goes without saying that an account of etiopathology shall mirror the one of symptoms (cf. Box 5.2), there can very well exist symptoms or effects of *disease*, without that causes of *disease* or etiopathological agents exist as well.

A second reason for spending some time on etiopathology is more *pragmatic*: because of their parallelism, focusing not only on symptoms, but also on etiopathological agents, can help get a

[143] Terminological note on negative causes of disease: talk about negative causes of disease is often replaced, in the current medical usage, by talk about *pathogenesis* (or pathogenicity) or *pathogens* (cf. e.g. Dorland, 2011). However, the term "etiopathology" or "etiopathological agent" is much more suitable for talking about a cause of disease here, for, as such, "patho*genesis*" (or "patho*genicity*") does not refer to the (preceding) *causes* of disease, but to the beginning and development of a pathology (or disease) as a *process* i.e. from the start/origin to the end/completion of a disease (process): if e.g. "leukemia" or "cancer" refers to a certain disease process, then the term "leukemo*genesis*" or "carcino*genesis*" (or "carcino*genicity*") just insists upon the fact that we are talking about a certain whole disease process from start to end – unless, of course, these processes are, as it is also highly common, understood in a functionalist setting (cf. e.g. Williamson, 2019b).
To the contrary of "pathogenesis" or "pathogen", the term "*etio*pathology" (or "*etio*pathological agent" or "*etio*pathogenesis") explicitly refers, etymologically speaking, to the *causes* of disease – albeit the term is also not devoid of defects (cf. Sec. 7.3.1). This latter term is, thus, to be preferred over "pathogenesis" or "pathogen", although "pathogenesis" or "pathogen" can be still *plausibly* used, of course, as referring to the causes of disease. If so, this plausible use of "pathogenesis" or "pathogen" shall be made explicit throughout this PhD dissertation.

[144] Terminological note on causation: note that "causation" or "causal relationship", albeit the word itself insists upon the notion of a *cause* – and not to an effect, though the former implies the presence of the latter (and vice versa) -, is to be taken as a general term having as more *specific* and *different* relational predicates "_is a cause of_" and "_is an effect of_".

better understanding of one notion thanks to the other one (and vice versa), or can give the reader a better overview of medical causation in all its *generality*, by having chosen to give not a single detailed account of medical causation, but two separate accounts of two specific causal relationships viz. *being an effect of disease* (cf. Ch. 5) and *being a cause of disease*, which also parallel our analysis of the differentia for disease kinds viz. negative effects and causes (cf. Box 4.2).

What is an etiopathological agent (etiopathology)? If we are to list some common causes of disease, then one can find e.g. parasites (e.g. ticks, mosquitoes, etc.), bacteria, or viruses – all causing specific diseases (and plausibly as starting *states*). To this list, one should also include other etiopathologies like a hostile environment causing a certain disease (e.g. pollutants like some solvants or metals causing cancer) (on that point, cf. Rappaport et al., 2014)[145].

More concretely, a parasitic disease (i.e. a disease caused by a parasite) like toxoplasmosis involving *T. gondii* is very likely to count as an etiopathology or etiopathological agent. Although such a whole may sound strange or quite unintuitive (prima facie) to be called an "etiopathological *agent*", *T. gondii causing* a certain disease (where toxoplasmosis is the whole) is the etiopathological agent in question – not *T. gondii tout court* (against functionalist interpretations, cf. Sec. 4.2.7); that much is in line with our comprehension of a symptom (cf. Box 5.1-5.2), where a symptom is the effect *of a disease* – not a specific negative effect *tout court*, for there is no such thing, or there are only effects/causes *of*; this weirdness evaporates, once we realize that quoting e.g. *T. gondii* as an etiopathological agent should be taken, in the present case, as a shortcut for *T. gondii* causing disease *x*, or being a cause (temporally) preceding (the presence of) disease *x*.

However, it remains true that *T. gondii* (*tout court*) just is (*essentially*) a certain parasite (invading/infecting a certain host) – indeed, the parasite, as being a negative entity here, is nothing without its host[146] -, or only indirectly (*necessarily*) e.g. a negative cause (temporally)

[145] Caveat on internal vs. external causes (and effects): it is arbitrary to try to delineate *internal* causes (or effects) of disease (e.g. bacteria) from *external* ones (e.g. pollutants); indeed, first, the respect to which we shall talk about internalness vs. externalness in this situation shall likely apply to *tokens* only (as having a spatio-temporal location);
second, it is obvious that some tokens disease (because of precisely what they are) can only but have (direct) tokens internal cause or effect (in whatever sense) (e.g. a token HIV infection), while others can only but have (direct) tokens external causes or effects (e.g. a physical injury).

[146] Caveat on negative processes and their constituents: the term "parasite" (as for many other cognates like "bacterium" or "virus"), indeed, often refers to only a certain *constituent* of the negative *causal* process in question: e.g. typically the starting or resulting *state* of the process in question (e.g. a certain cell) – *not* to a whole negative causal process. That much goes hand-in-hand with, probably (though not necessarily), their very common functionalist acceptance (cf. Sec. 4.2.7).

preceding (the presence of) another (negative) entity (for the same point about e.g. fever (of *x*), cf. Sec. 5.2).

Thence, a prima facie definition of e.g. toxoplasmosis (as a certain etiopathology) shall be as a certain disease (i.e. condition (i)) with *T. gondii* as a negative cause temporally preceding it (i.e. condition (ii) (cf. Box 6.1) – though this definition is given with a dependently defined differentia.

Prima Facie Definition of Etiopathology
x is an etiopathology, iff (i) *x* is a disease, and (ii) *x* is a negative cause of it.

Box 6.1. – Prima facie definition of etiopathology.

6.2 A Definition of Etiopathology

Sec. 6.2 is divided into two further sections: in Sec. 6.2.1, I provide a definition of etiopathology, according to which an etiopathological agent is a disease (i.e. the genus) plus a directly temporally preceding negative entity related to a living being (i.e. the differentia).

In Sec. 6.2.2, I compare my account of etiopathology to Glennan (1996)'s mechanistic account of causation.

6.2.1 Etiopathology as a Disease with a Temporally Preceding Entity

Conditions (i) and (ii) of our prima facie definition of etiopathology (cf. Box 6.1) do not clearly enunciate a *correct* or strict definition of etiopathology. Indeed, this prima facie definition merely provides a synonym for "etiopathology".

Nevertheless, as for the case of symptoms (cf. Box 5.2), this prima facie definition of an etiopathological agent can be used as a basis for providing a correct definition of etiopathology: e.g. HIV-AIDS is a nice (plausible if not correct) illustration of a certain (complex) etiopathology.

Indeed, if we hold that HIV infection causing AIDS (or stage 3 HIV infection) is an etiopathological agent, then this seems true simply in virtue of the fact that AIDS is a certain disease (i.e. the genus) (cf. condition (i) of Box 6.2) directly temporally – to avoid subscribing to counterintuitive (token) causes and effects e.g. too much remote from each other (cf. also Sec. 5.2) - preceded by another negative entity viz. HIV infection (i.e. the differentia) (cf. condition (ii) of Box 6.2), which is defined independently from its genus, in so far as HIV infection can very well temporally precede other (negative) entities than, necessarily, AIDS; HIV-AIDS is also indirectly, of course, a certain disease (which also happens most of the time with etiopathologies) (but, cf. Sec. 4.2.10).

From this illustration, in parallel to our own definition of symptoms (cf. Box 5.2), we can argue, thus, that an etiopathology is constituted by a disease (as a genus) in conjunction with a negative entity directly temporally preceding (another entity) and related to a living being (as the differentia) (cf. Box 6.2). As such, this definition of etiopathology is compatible with the *plausible* (though incorrect, strictly speaking) idea that an etiopathological agent is a (negative) *state* (cf. also Sec. 5.2).

Definition of Etiopathology
x is an etiopathology, iff (i) *x* is a disease (i.e. the genus), and (ii) *x* is a directly temporally preceding negative entity related to a living being (i.e. the differentia).

Box 6.2. – Definition of etiopathology within (our understanding of) a genus-species hierarchy.

In other words, if HIV-AIDS (S's ϕ-ing)'s causing T's ψ-ing) is a certain etiopathology, that is (partly) because, following condition (ii) of Box 6.2 (cf. also Fig. 6.1 below), albeit HIV infection is *essentially* a certain viral process invading/infecting the vital cells of the human immune system (S's ϕ-ing), HIV infection just directly temporally precedes AIDS (just like it also precedes other different diseases like (plausibly) some specific flu[147].

Along condition (i) of Box 6.2, if HIV infection causes precisely AIDS, that is *not* because AIDS is this disease temporally following HIV infection (on pain of getting a differentia defined dependently upon the genus in question) – albeit the definition can still be *plausibly* interpreted

[147] Caveat on infectious diseases: the literature here, in line with a certain functionalism, often confuses what e.g. HIV infection *is* with what HIV infection *causes*: e.g. HIV infection is, strictly speaking, of course, an infection or invasion; it does not cause it. This mistake is also certainly related to, or coupled with, the idea that HIV infection is not the whole viral (causal) process, but only some *part* of it *causally* preceding another one.

as such, of course (cf. Box 4.2 for the same point) -, but that is just (partly) because HIV-AIDS (as a complex kind itself) is AIDS (as a genus) (T's ψ-ing) plus condition (ii) of Box 6.2.

6.2.2 Towards a Mechanistic Account of Etiopathology

To better grasp our definition of etiopathology (cf. Box 6.2) – and also, *en passant*, our definition of symptoms, of course (cf. Box 5.2) -, it is necessary to put it within the more general debate around the metaphysics (and epistemology) of cause (and effect).

David Hume (1748 [1910]) is famous for, amidst others, having once asked: what is the necessary connection between cause and effect?

In other words, how do we get the impression of *necessitation*, because all we can observe are regularities, and not the secret or hidden connection or power binding together two events? Roughly, his famous skeptical solution is that a constant conjunction and a spatio-temporal contiguity between two events (rather, *processes* here or even maybe *states*) is all there is to say about causality: there is no (i.e. a *metaphysical thesis*) or unknowable (i.e. an *epistemological thesis*) such thing as a causal connection, a bond, a cement, or a causal nexus (i.e. *causal anti-realism*).

Although it merely follows the Humean idea of temporal contiguity between two entities, our definition of etiopathology as a disease plus a directly temporally preceding negative entity related to a living being (cf. Box 6.2) – on pain of a category mistake – is basically *anti-Humean*, for it *realistically* grounds the idea that a cause makes its effect necessary (and vice versa) on the idea, for the more specific case of (negative) causes (of disease) here (cf. Ch. 5 for symptoms), that, if x is the (negative) cause of y (e.g. a disease), then there exists *necessarily* a (negative) whole.

In other words, in the case of an etiopathological agent, the secret connection is analyzed through the idea that, from two causal relata, there exists necessarily a negative emerging whole.

This abstract analysis of a negative cause of a disease (cf. Box 6.2) is close, at a more general level (cf. also Ch. 5), to (a certain understanding of) the well-known *mechanistic*/*mechanical theory of causation* elaborated by Stuart Glennan (1996; 2002; cf. also Glennan, 2017)[148], according to which, roughly, an event c situated at the non-fundamental level causes an event

[148] Note that I am not the first philosopher of medicine, of course, to compare (or even base) his theory of medical causation (even partially) on Glennan (1996)'s mechanical theory of causation (cf. e.g. Sec. 4.2.7 for the RWT).

e situated at the non-fundamental level, iff there is (i) a *mechanism m*, which (ii) *connects c* to *e*[149].

Indeed, if desideratum (ii) of Glennan's mechanistic theory of causation is understood in a certain way viz. that there exists an *underlying* (or *constitutive*) mechanism connecting *c* to *e* - thus, not a mechanism situated at the same level between *c* and e, or *producing e* (from *c*) - (cf. also Gebharter, 2019 for this classical interpretation of the mechanical account of causation), then Glennan's mechanistic theory of causation is, at the more general level of (non-fundamental) causation *simpliciter*, obviously close to, though not identical with, our own definition of etiopathology (understood in a *processual* way, of course) as being about a disease (the relatum *e*) directly temporally preceded by a negative entity related to a living being (the relatum *c*) (cf. Box 6.2), but with the important difference that a negative cause of disease is taken here as a specific (single) kind/whole itself, and that the relata *c* and *e* are merely conjunctively related[150].

Finally, one major advantage of our definition of a cause of disease (or, at a more general level, of causes – or even effects (cf. Box 5.2) - is that, as such, it is compatible with a permissive neo-Aristotelian ontology acknowledging the causal efficacy of higher-level phenomena like mental ones causing, or being caused by, other phenomena situated at that *same* level/order[151]. Indeed, our definition of etiopathology (as well of symptoms) is applicable to (highly) emergent (or derivative) phenomena as well (cf. Box 5.2; 6.2) – without making them, thus, *epiphenomenal* i.e., minimally, just as having no causal efficacy (*pace* Walter, 2010; on my non-reductive physicalism, cf. Exc. 7.3), or *causally overdetermined* i.e. here with redundant different-level causes (for, to every cause/effect its own same-level effect/cause).

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[149] What a mechanism *according to* Glennan really is is a rather complicated matter. Indeed, for him,

> "[m]echanism" is used to describe two distinct but related sorts of structures. First, mechanisms are *systems* consisting of a collection of parts that interact with each other in order to produce some behavior. […] Second, mechanisms are *temporally extended processes* in which sequences of activities produce some outcome of the mechanism's operation". (Glennan 2008, 376; my emphasis)

In other words, along this line of thought, a mechanism can be understood (i) *vertically* as the structured underlying components giving rise to a certain phenomenon, or (ii) *horizontally* as a process leading to an effect *e* (cf. Kincaid, 2012 for this terminology).

However, for the present purpose, we can stay content with a more deflationary meaning of "mechanism" as a causal *process* (along a plausible understanding) (cf. also Kistler, 2004; Railton, 1978; Salmon, 1984; on the burgeoning literature on what mechanisms are, e.g. Craver & Darden, 2013; Kaiser, 2018a; Krickel, 2018; Machamer et al., 2000).

[150] About this important difference, note that Glennan (1996; 2002)'s mechanistic theory of causation has been subject to extensive criticisms, especially on how to correctly (or, in a non-circular way) grasp the notion of *connection* between an event *c* and *e*, which Glennan makes appeal to (for a sample of those criticisms, cf., amongst others, Casini, 2016; Craver, 2007).

[151] Note that this point follows one of the famous Bradford Hill (1965) criteria in epidemiology viz. the one of getting between the cause and the effect the same grain of specificity.

6.3 Complex Etiopathologies

Sec. 6.3 is devoted to the question of, given our account of etiopathology (cf. Box 6.2) and, by extension, of symptoms (cf. Box 5.2), how to make sense of the intuitive idea to have *multiple* (direct) causes of disease and *indirect* ones.

Sec. 6.3 is divided into two further sections: in Sec. 6.3.1, I defend that our definition of an etiopathological agent does not preclude us to talk about multiple causes of disease (as well as symptoms) taken in a certain minimal sense i.e. where only the indirect constituents of an etiopathological agent are multiple – at least, more than two.

In Sec. 6.3.2, to take into account all the complexity in the discussions around causes of disease, we distinguish between direct and indirect etiopathologies; I argue that many etiopathological agents quoted as such in the literature are, actually, only indirect ones; I provide an account of indirect etiopathologies as etiopathologies themselves causing the cause of another disease.

All those points also apply, of course, to symptoms as well as to causation in all its generality.

6.3.1 Multiple Etiopathologies

Why to dwell on the issue of whether there is always only *one* (specific) cause of disease or *multiple* ones? First, beyond being of a metaphysical worth, answering this question also has some *practical* worth; it can have, indeed, huge implications in several bioethical debates: e.g., in the debate about the end of life or euthanasia, for reasons, partly, of responsibility, we seem to rely on a very fine-grained differentiation of the specific cause of disease or death: e.g. in euthanasia, is the cause of death the intention to actively intervene on a patient, or to passively let the patient die by an absence of treatment?

Second, unlikely as it may sound, there is, indeed, a certain lively debate in contemporary philosophy of medicine about whether we should allow multiple causes of disease or not (on the history of the so-called "multifactorial disease model (plausibly taken)" and the late 19th century "monocausal disease model", cf. Fuller, 2018a; Ross, 2018; forth.), albeit some have tried to minimize the current debate by claiming that

> […] notions of multiple causation and multivariate analysis are so commonplace and so embedded in modern epidemiologic reasoning that they hardly merit discussion as a model or as an approach to understanding disease. (Krieger, 1994, p. 891)

However, that the notion of multiple causation seems so much practically entrenched, as the above quote takes it, is not a reason for not debating it, for it is not clear what "multiple causation" really means; the current debate has certainly emanated from our more and more precise knowledge of causes of disease[152].

Thus, it is worth asking about whether etiopathological agents can be multiple or not. In what sense can etiopathological agents be, indeed, multiple? Can there be a web of causation? Our definition of an etiopathological agent (cf. Box 6.2), in line with our permissive neo-Aristotelian ontology (cf. Exc. 3.5), seems to imply that there is always one single specific cause of a specific disease, for there is one kind or *whole* viz. an etiopathological agent: e.g. HIV-AIDS is a single etiopathology, or it is *one* specific cause of a specific disease. That there is, in this sense here, only one specific cause for a specific disease is due to the fact that etiopathologies are *intensionally* defined (cf. Ch. 3).

However, that there is one specific cause for a disease does not prevent that this specific cause of disease (as a whole) has *multiple indirect constituents* – at least, more than two -, which are themselves (indirectly) causes (of disease): e.g. HIV-AIDS (S's ϕ-ing's causing T's ψ-ing), as a single complex whole/kind, is one specific etiopathology, but the constituents of HIV infection (S_1's ϕ_1-ing & S_2's ϕ_1-ing), as being multiple (i.e. through the whole replication cycle), can very well play the role of multiple causes, a chain or path of (intermediate) causes (Ross, 2018), or a web of causation (Krieger, 1994) (cf. Fig. 6.1).

First, note that those are not multiple causes of e.g. AIDS, for AIDS has truly a *single correct* cause); rather, they are a chain or a path of causes between *each other*, or an underlying *causal* mechanism or process (cf. Sec. 6.2.2). Referring to them as multiple causes *of AIDS* is committing a MF (cf. Sec. 4.1.2). In that minimal sense, but still with the commitment to a fallacy, we can talk about, indeed, multiple causation (in all its generality).

Second, note that our minimal sense of multiple causation or, more specifically here, multiple causes, excludes that multiple causation or, more precisely, causes can be understood as, so to say, different *parallel* (and independent) paths or chains towards/from the same effect/cause

[152] Note that this debate also occurs in the philosophy of causality (in all its generality): e.g., on basis of Hume (1748 [1910])'s legacy (and Mackie (1974)'s subsequent work), Baumgartner (2008) has argued for the case of multiple causes and effects.

(*pace* Baumgartner, 2008). As Lauren Ross (2018; my emphasis) nicely expresses it (but, in an interventionist vein):

> [w]hat [the idea of multiple causes along a single path] does deny is that there are other factors–*off this path*–that also have interventionist control over the disease. What about factors such as oxygen, the immune system, and genes? Don't these factors play a causal role in all diseases and, thus, figure in the multicausal etiology of any disease? Notice that we do not typically cite these factors as causing infectious diseases such as tuberculosis, anthrax, and cholera. The reason for this is that […] [w]hen these factors are manipulated they can control a variety of outcomes (including whether an organism lives or dies, as in the case of oxygen) but they lack control over the effect of interest, viz. the presence and absence of disease in question.

In other words, rephrased within our own framework, multiple causes *of disease* are, actually, those multiple indirect constituents belonging all and only to *a single* etiopathology like HIV-AIDS; there are no other (off-path) causal factors (for the disease at issue) (e.g. the absence of oxygen), on grounds of irrelevance for the disease in question.
Fig. 6.1 summarizes all this as follows (cf. Sec. 6.2.1 for an illustration):

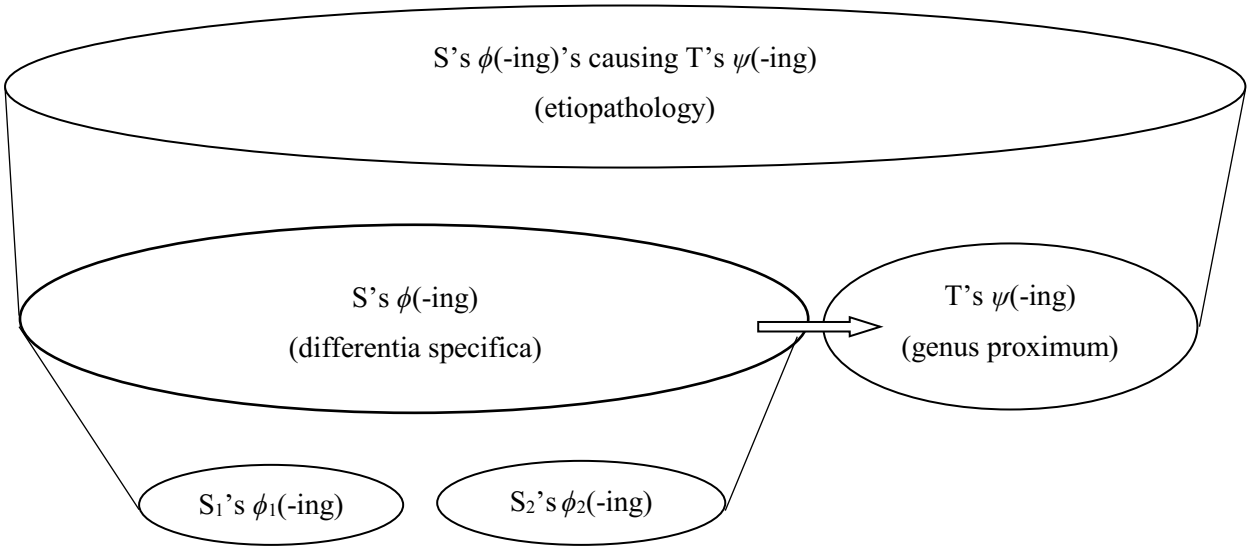

Fig. 6.1. – Depiction of our definition of etiopathology (cf. Box 6.2).
Legend: "S's ϕ-ing's causing T's ψ-ing" stands for e.g. "HIV-AIDS"; the oblique bars represent a constitutive relationship; the arrow a (lower-level) relationship of direct temporal asymmetry.

6.3.2 Indirect Etiopathologies

With our minimal account of multiple etiopathologies (cf. Sec. 6.3.1), are we able to deal with the whole complexity of the (said) causes of disease in the literature? How can we be sure that a path, chain or cascade of *true* causes (of disease) could be multiple constituents of a single correct cause of a disease? E.g., pemphigus is a rare autoimmune disease creating blisters on the skin, whose causes seem to include both the development of specific autoantibodies and acantholysis i.e. the loss of connection between the basal cells – without that it seems possible to count them as multiple constituents of a *single* cause of pemphigus.

A way out here is, in addition to admitting a minimal form of multiple causation, to distinguish further between direct and *indirect* causes of disease (and effects, by extension).

We hope, with this distinction, to be able to maintain our idea that there is always one single correct cause of disease (cf. Sec. 6.3.1), and to take into account the complexity in the discussions around the notion of etiopathological agents. Indeed, if we are right that there are direct as well as indirect causes of a certain disease, then it shall turn out that many (said) causes of disease are, actually, only (very) indirect ones (on the impossibility to transitivize here, cf. Sec. 2.1.3).

What are indirect causes of disease? If our definition of causes of disease *simpliciter* (cf. Box 6.2) stands for, actually, only *direct* ones, then (very) indirect causes of disease *x* are just those etiopathologies themselves causing the (indirect) cause of disease *x*; ditto for the case of symptoms, of course.

For the case of pemphigus, we may well argue that, plausibly, acantholysis is the *direct* cause of pemphigus, for there is a certain kind called “acantholytic pemphigus” which fulfills conditions (i) and (ii) of Box 6.2; indeed, there is the genus “pemphigus”, and acantholysis directly temporally precedes different diseases, amongst which pemphigus (i.e. the differentia). Furthermore, if the presence of specific autoantibodies (i.e. antibodies produced by the immune system) is a(n) (very) *indirect* cause of pemphigus, that is because those autoantibodies can be deemed as a (direct or maybe even indirect) cause of acantholysis. For, autoantibodies (as destroying desmoglein) temporally precedes, in turn, different diseases (like acantholysis).

7

ILLNESS, SICKNESS, DISABILITY, HARM, DISORDER, ETC.

Ch. 7 of the present PhD dissertation is devoted to the analysis of our different concepts related to the one of disease, with which they are sometimes confounded or very vaguely used with almost no difference with the one of disease.

To the contrary of the concepts of disease kinds and related ones, providing a correct definition or thorough analysis for each of the concepts of illness and related ones is useless for our present purpose, for, as we shall shorly see, they are, actually, mere specific diseases i.e. illustrations of the category of disease, or just synoyms.

That is why, we choose, with prima facie definitions of those concepts, to merely show, in Ch. 7, how close (or far) such concepts are intuitively from DISEASE; in that sense, we remain neutral here about whether such concepts, though certainly not trivial, are (intensionally) *reducible* or not to other ones. In line with the goal of Part II, this task aims at clarifying what disease is *not*, such that to more directly and less confusedly approach the concept of disease (cf. Ch. 8).

Other plausible meanings of all these terms can, of course, still be used throughout the PhD dissertation; if so, the reader shall easily recognize whether the meaning of the word in question follows the one given in Ch. 7 or not.

In Sec. 7.1, I defend a minimal view of illness as the mere feeling of a disease (taken as a specific disease itself).

In Sec. 7.2, we review other complex concepts (like SICKNESS, DISABILITY and HARM) directly related to the one of disease which, as I argue, are cases of higher-level diseases.

In Sec. 7.3, we review several notions that are very often used just exchangeably with the one of disease.

7.1 A Prima Facie Definition of Illness

Sec. 7.1 is about the concept of illness. In Sec. 7.1.1, I defend the minimal view that illness is a mere feeling of a disease, which is itself a certain disease.
In Sec. 7.1.2, I focus, more specifically, on the illness *host* i.e. on the notion of a *feeling* of disease by arguing that, more generally, a feeling is an attention mechanism.
In Sec. 7.1.3, I argue more thoroughly that an attention mechanism is, generally speaking, based on a mechanism of reaction to an entity's own neural stimuli.

7.1.1 Illness as a Specific Disease

There is already an extensive body of literature on the notion of illness (cf. e.g. Boorse, 1975; 1997; Carel, 2016; Hofmann, 2002; 2017; Kay Toombs, 1993; Radden, 2019; Schramme, 2016). At the basis of any account of illness, there is intuitively the notion of a *bad feeling* or the feeling of a disease. However, how to further understand what a bad feeling is is subject to many interpretations.
First, the notion of a bad feeling has been associated to the one of a *mental disorder* (Radden, 2019). However, it is clear that the notion of a bad feeling is not essential to the one of a mental (or even affective) disease, as has been already clarified (cf. Sec. 4.2.8)[153]. In that sense, the dichotomy feelings/unfeelings does not correspond, thus, to the dichotomy mind/body, respectively.
This theory of illness goes along with the general view, according to which for *x* to count as a disease (kind) *tout court* (in our terminology) (cf. Sec. 2.2.2) is for *x* to be essentially *felt* (however plausibly understood) as a disease (kind) by a specific host, or that *x* constitutes some *harm* (*plausibly* understood here as a bad *feeling*) to the host (Cooper, 2002; Fulford, 1989): e.g. one can reject the idea that people having hearing loss are diseased people (from the auditory system) on the firm grounds that these people may *not* feel like diseased people.
However, we would hardly deny that e.g. Ted Bundy or Charles Manson is a specific diseased human being on the firm grounds that he is (realistically) not feeling like a specific diseased

[153] This point goes, thus, against the influential view of *mental* diseases put forward, most notably, by Wakefield (1992), according to which a mental disease is a *harmful* dysfunction (plausibly taken), where "harm" seems closely related for him to what I call "bad feeling" (cf. Sec. 5.1.2) or "illness" (cf. Box 7.1), albeit Wakefield's view is not at all clear on that precise crucial point.

human being; the same point more widely applies: e.g. are we really ready to accept that human beings torturing animals are not to be judged diseased human beings, because we could say that they do not (realistically) *feel* like diseased human beings – indeed, it could be further argued that they are feeling, to the contrary, just as being different (healthy) human beings (Barnes 2009; 2014), for torturing animals is *for them* part of what they are (or their culture)?

Thus, it is not because *x* is no *harm* (as taken here) to a host that *x* should not be considered a disease: bad feelings are not the *only* existing specific diseases. A disease host need not *feel* (i.e. e.g. acknowledge, notice, or be aware) that it is diseased to be deemed diseased (on *externalism* about knowledge, cf. Sec. 4.2.3).

Moreover, although a disease (or e.g. a symptom) is *necessarily* – but not essentially (or definitionally) - absolutely realized (cf. Sec. 3.3.1), it is not necessarily so by a *felt* symptom (or disease) i.e. taken as a specific disease, for it can be absolutely realized as well by e.g. an *un*felt symptom (or disease).

However, if a disease is necessarily absolutely realized by e.g. a *felt* disease or illness (as a specific disease), it is still not essential to a disease that it be felt, or a disease is not (strictly) *definitionally* dependent upon a (propositional) attitude like a feeling. Thus, a negative value like disease is still *objective* – not subjective (cf. Exc. 7.2).

Of course, all this does not imply that feeling oneself well is not important, crucial, essential for, or just directly related to, having some healthy process *x*, while still hosting another disease *y* having nothing to do with *x*: e.g., albeit a person with an abnormal audiogram cannot feel to have a healthy audiogram, the person, nonetheless, can correctly feel her/himself well in her/his skin in many other ways viz. e.g. in having a healthy Body Mass Index (BMI), etc[154].

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[154] Three caveats here on felt and affective diseases, and unfeelings: (i) can "*feeling* of disease" have as (indirect) constituents or as (indirect) absolute realizers "*un*felt disease", or vice versa? Indeed, it is very widespread, especially in the philosophy of emotions, to claim either that it is *correct* to *feel* e.g. frightened/fearful from *x*, when there is, actually, some (*unfelt*) frightfulness/fear from *x*, or (*unfelt*) frightfulness/fear from *x* is part of the *correctness* conditions (under a plausible meaning) of a *feeling* of frightfulness/fear from *x* (cf. Deonna & Teroni, 2012), or that it is exactly in the reverse order (cf. Tappolet, 2011).

First, we can reply that a *feeling* of disease (e.g. the feeling of frightfulness/fear) is a mere *absolute realizer* (amongst others, or at least) of a disease (e.g. frightfulness/fear).

Even if it is true that a disease *necessarily* has an absolute realizer like a *felt* disease, it still does not imply from this that a *felt* disease is, strictly speaking, among the correctness conditions of a disease (*or vice versa*), to the extent that we associate, of course, correctness conditions with the notion of *essence*: a disease is not made correct by the presence of a felt disease (or vice versa), although a disease is necessarily absolutely realized.

Second, following our largely *objectivist* (and highly *realistic*) metaphilosophy (cf. Ch. 2), we may argue here that a *feeling* of disease can be, indeed, (indirectly) constituted by, or can have as an (indirect) absolute realizer, an *unfelt* disease (on the sophisticated distinction between (un)felt *x* and the mind-body relationship, cf. Sec. 5.1.2).

(ii) Felt or e.g. affective diseases remain *bad* or negative, even if all their *correctness* conditions are met: e.g. being frightened by a dog, who is truly frightening, or even frightening someone who deserves to be frightened (i.e. where all the correctness conditions for e.g. frightening someone are reunited), remains bad or negative.

That much is not to be interpreted as meaning that there are (negative) *values* (e.g. *x* is frightening), which are part of the correctness conditions of (negative) *emotions* (on the huge literature about the complex relationship(s)

Second, the notion of bad feeling has been associated to the subjective and/or (said) evaluative aspect of disease (Boorse, 1975; Carel, 2016; Kay Toombs, 1993).

Albeit it is true – but not to the contrary of disease – that illness has an evaluative dimension, in the minimal sense that illness (as a *bad* feeling) is trivially something *bad* (like for disease), a bad feeling (like for disease, thus) is not subjective, but (completely) objective, as we have also already seen (cf. Sec. 5.1; 5.2; Exc. 7.2; also Schramme, 2016 for this possibility).

From the decoupling between disease and subjectivity, we can easily argue, thus, that *illness* just is a specific higher-level – maybe the highest-level - disease itself (as a more encompassing category, and taken also as a *value* (fully) objectively defined) viz. a *bad* feeling or the feeling of a disease (cf. Exc. 7.1).

Thus, we get the following prima facie definition of illness:

Prima Facie Definition of Illness
x is an illness, iff *x* is a feeling of a disease.

Box 7.1. – Prima facie definition of illness.

Note that our point here excludes other specific subjectivist theories like preference-, desire-, enjoy- or pleasure-based theories of health (or welfare) (Sen, 1979), for it seems that one can perfectly well judge that somebody fares well without that any (even core or basic) preference, desire or pleasure be made or taken.

Another general consequence of the dismissal of any *subjectivity* in disease (kind) attributions – by following here the DSM-5 (APA, 2013; cf. also Lalumera, 2016) for the case of mental diseases - is the rejection of a whole body of philosophical literature emphasizing the importance to rely in medical diagnoses e.g. "[…] on the individual's [own] experience as a complex and multidimensional person" (Stegenga et al., 2017, p. 356) i.e. on the patient's

between emotions and values, cf. e.g. Mulligan, 1998); for, negative *emotions* (as conceived of here i.e. decoupled from negative *feelings*) are specific diseases (e.g. *x* is fearful/frightening) – which *are* themselves (in the indirect absolute extension of) negative values or badness (e.g. *x* is negatively evaluated) -, albeit there are connections, of course, between e.g. frightfulness, *felt* frightfulness and *unfelt* frightfulness (cf. caveat (i)).

(iii) Cannot someone really feel to have a healthy *x*, while hosting a disease *x*? Indeed, if from the feeling of *x* we cannot infer that there is necessarily some (natural) *x*, then nothing should impediment that someone is *feeling* to have a healthy BMI, while still having, actually, a diseased BMI, for the diseased BMI might well be absolutely realized by an *unfelt* one.

The main problem with this interesting issue is that it is sufficient that there is (at least) a *single* absolute realizer. If so, then this would amount to a contradiction: if, for the sake of the argument, only feelings were the absolute realizers here, then, in our case, someone would contradictorily feel to have both a healthy BMI and a diseased one.

feelings or, even more, his story (on so-called "narrative (or humanistic) medicine", cf. e.g. Marcum, 2008; Solomon, 2015; Tekin & Mosko, 2015 (for mental diseases); for criticisms, Ferry-Danini, 2018; 2019).

But, as being a specific disease itself, what is really a feeling of disease?

7.1 Excursus: The (Bio)Medical Model

Following our own account of illness as a specific genuine (highest-level) disease (cf. Box. 7.1), it is not clear whether we have to *fully* embrace the so-called (bio)*medical model* (of health and disease) (so labeled by Laing, 1969) (also called "disease-centered model" especially in psychopharmacology) (cf. Moncrieff, 2008), for the (bio)medical model seems to take a ((relative/absolute) intensional) *reductionist* stance (at least) on *higher-level* diseases like illness (along our understanding of it, or as a mere mental disease), or those that are not, strictly speaking, biological (restrictedly taken) or physical, such that to certainly avoid referring to values taken (mostly) as subjective (cf. Groopman, 2007; Guze, 1992; Lemoine, 2017; Murphy, 2006; Sarto-Jackson, 2018):

> [i]n my opinion, there can only be one sound foundation for psychiatry, that based on the medical model, and only one legitimate domain of expertise, that pertaining to mental illness. […] What distinguishes the medical model from nonmedical models […] is not so much its reliance on scientific method but rather [the] philosophical orientation […] that sufficient deviation from normal represents disease, […] and that elimination of [disease] will result in cure or improvement in individual patients. […] [P]sychiatric disease or mental illness would be defined as any debilitating, cognitive-affective behavior disorder due primarily to known, suggestive, or presumed biological *brain* dysfunctions, either biochemical or neurophysiological in nature. (Ludwig, 1975, p. 603; my emphasis)

In other words, despite that our (anti-reductionist – even at the token level) treatment of higher-level diseases like illness (or even mental or social diseases, as well as all the other lower-level diseases) still follows, actually, one of the core tenets of the (bio)medical model (on antireductionist psychological realism, cf. Broome & Bortolotti, 2009a; but, also 2009b) - according to which any objective deviation from the (biological) normal is a disease, or "[…] [if a biological part] is not in the right natural condition, that [is because] it has been [objectively] damaged by diseases […]" (Bolton, 2008, p. xv) -, it departs from the (bio)medical model in that the model tends to be reductionistic for higher-level diseases (as a follower of this idea, cf. Fuller, 2017; *pace* Petrolini, 2015).

7.2 Excursus: Subjectivism about Disease

How to understand the idea of subjectivity about disease? Subjectivity (or *mind-dependence*) about disease (kinds) is just a way of taking disease (kinds) (even negative (propositional) *attitudes* themselves like mental states or feelings) as being *definitionally dependent* (strictly understood) upon (other negative (propositional) or *con*-) *attitudes* i.e. as specific artefactual kinds (for further thoughts on our taking of artefactual kinds, cf. Exc. 2.7; on how to understand subjectivity at different levels, Schramme, 2008; Stroop, 2013): e.g. (negative) desires (on irrational desires, cf. Culver & Gert, 1982); (negative) feelings themselves; etc.

A few remarks are in order. First, it is important not to confound subjectivism about disease i.e. the thesis that all diseases are subjective with the issue about whether for *x* to count as a disease is for *x* to be essentially *felt*.

Indeed, distinguishing between disease (negative feelings included) and subjectivity (in all its generality) is one thing, and between disease and, *specifically*, *feelings* (or awareness) is another one.

Second, can even the highest-level diseases like illnesses (or higher-level diseases like affective diseases) be really objectively defined? All this shall depend, of course, on how those diseases are defined (e.g. on emotions as (mere) perceptions of values, cf. Prinz, 2004); but, for them to be said objective is merely for them to have a definiens not referring to con-attitudes themselves; and this condition is fulfilled for a case like illness (cf. Sec. 7.1.2-7.1.3).

Third, subjectivism about disease should not be confounded with the thesis of *anti-realism* about disease i.e. the thesis that there are no real truth conditions for disease.

Indeed, one can be consistently *subjectivist about disease* and *anti*-antirealist (about disease) by e.g. realistically (or even objectively) understanding further the notion of *harm* (as taken here) (cf. Fuellen et al., 2019 for such a strategy), or by insisting e.g. that even the highest-level diseases (subjectively defined here) are real, for they are accessible by other people by the high(est)-level means like mind reading or mental simulation of others' mind; the ideal physician need not his patients' own reports to diagnose a specific disease (kind) – whatever the specific disease (kind) it is i.e. even a highest-level one (although, of course, not *reports*, as such, but patients' own words or speech (acts) may very well be deemed as e.g. a certain negative sign of a higher-level disease).

However, subjectivism and antirealism about disease usually go hand-in-hand in the literature, for subjectivity is often understood as having no possible (real) normative conditions, as (partially) for Wakefield (1992)'s famous definition of disease as *harmful* dysfunction (plausibly taken) (cf. Sec. 7.2.3; also Amoretti & Lalumera, 2019a-b; Cooper, 2015).

7.1.2 Illness as the Feeling of a Disease

By “feeling”, generally speaking, I do not restrict myself to so-called “bodily feelings (or, bodily *sensations*) or kinesthesia” (James, 1884). For, if I can have e.g. a feeling of pleasure, then, if a feeling just is a *bodily* feeling (or, a feeling of a body), then what appear as specific *mental* states or processes (like pleasure) just are specific *physical* (bodily or somatic) states or processes (cf. Exc. 7.3).

To avoid using too much a technical jargon, I just employ the term “feeling” *simpliciter* throughout the PhD dissertation to refer to one’s (positive or negative) attention toward one’s (positive) mental *or* somatic states or processes (or, reflexively, feelings themselves).

However, this might be wrongly interpreted as the claim that one’s (*positive*) mental *or* somatic states or processes (or, reflexively, feelings themselves) are the *intentional objects* of a feeling (e.g. a feeling *of* pleasure), while the attention mechanism would be the *subject* of a feeling.

Although this is the case for e.g. mental or, more specifically, affective or even mood diseases (vs. Bordini, 2017), where the disease host (as a higher-level part of a living being) is (plausibly) *about* or directed toward (in an intentional sense) an intentional object (e.g. the fear *of* a dog), this is not true for feelings, since a feeling is here equated with *awareness*: e.g. a feeling of a diseased foot is a diseased felt *foot*, where, thus, a foot (as the host) is the (positive) *subject* in question - not a specific intentional object.

This conception of feelings goes well with our intuition that e.g. (a token) pain (understood as a specific negative *feeling* here) is not intentionally directed toward an object, but is located in, loosely taken, this very object (e.g. a painful foot) (cf. Aydede, 2009; *pace* Borg et al., 2020).

Of course, in so far as I link throughout the work “feeling” with “consciousness (of one’s (positive) mental *or* somatic states or processes (or, reflexively, feelings themselves)” (or “awareness”), then I am using “consciousness (of one’s (positive) mental *or* somatic states or processes (or, feelings themselves)” *simpliciter* to refer for the philosopher of consciousness to states (or processes), which one is simply aware of, or which one’s attention is directed toward (in a non-intentional sense for the case of feelings) (cf. Sec. 7.1.3; Rosenthal, 1986).

This way of understanding consciousness is different from what is often called “*phenomenal* consciousness” (i.e. what-it-is-like states/processes) as well as to what is also often called “*access* consciousness” (i.e. those states/processes interacting with other mental states/processes and guiding an organism’s action) (Block, 1995). Thus, feeling *x* is interpreted here as being aware of *x*, or as having one’s attention (non-intentionally) directed toward *x*.

Some philosophers of emotion will find this picture of what a feeling is far too much minimal (cf. e.g. Deonna & Teroni, 2012); according to them, there is more to say about what a feeling

is than appealing to an attention mechanism; such philosophers typically rely on a picture of feelings as closely related to *phenomenal* conscious – or *what-it-is-like* (Nagel, 1974) - states or processes (or *qualia*) (but, on the vagueness behind the famous notion of qualia, cf. Tye, 2017), as well as on feelings (or even emotions) as related to action tendencies (e.g. the fear of a dangerous dog tends a subject to make him leave).

I cannot thoroughly embark here on such difficult and controversial topics. I can only highlight for the present purpose that, with a weak enough definition of (bad) emotions, a rejection of a first-person or internal perspective in this context (*pace* Bueter, 2019; Tekin, 2016), and a subscription to the WCP (cf. Exc. 2.3), all what is left to what a feeling really is is likely a mere attention mechanism (cf. Sec. 5.3.10), and action tendencies are maybe just implied by feelings (or emotions) (cf. Exc. 2.8).

Although reflexive/introspective consciousness, meta-representations or *self*-awareness of a disease is a specific disease itself properly understood along our line that a feeling is a mere attention mechanism, *reflexive* consciousness (or consciousness) should not be interpreted, thus, as *access* consciousness: e.g. people (plausibly) said to be (self-)*aware* (or, to (self-)acknowledge) that they are diseased are most of the time, actually, not (merely) *feeling* (that they are feeling) diseased, but (also) *believe*, *know*, *are convinced*, etc., that they are diseased, and e.g. knowing that one is diseased is, of course, not itself a specific disease, for it is *not* being (self-)conscious of one's disease (in our minimal sense), but, rather, merely having a (possibly unconscious) *positive* mental state intentionally directed toward a disease[155].

It is important to also distinguish between "awareness" (or "feeling") and often related (but very different, actually) cognates like "arousal" and "awakening" (or "wakefulness").

Indeed, first, one can very well be intuitively aware of *x* without being aroused by *x* (e.g. having one's attention directed toward *x* without being excited/stimulated by *x*), or one can be aroused by *x* without being aware of *x* (e.g. sexual arousal during sleep).

Second, albeit one cannot be aware of *x* without being awakened, one can well be, nevertheless, awakened without being aware, when one's attention just is not directed toward anything precisely (or, one is *un*aware of what one is doing) like in e.g. daily repetitive tasks or movements.

[155] Caveat here on those complex (positive) mental states: being convinced that *p* or knowing that *p* is, of course, not the same as e.g. *boasting* that *p* (e.g. for a man to boast of being a manipulator, of being violent, of being clever, etc.), which is something *negative* (cf. Sec. 9.4.2).
In other words, the important distinction drawn here is the one between e.g. for *x* to be offensive and for *x* to boast of being offensive; being de facto offensive is one (negative) thing, to boast of it is another - certainly worse – one.

Furthermore, what about judgments of (dis)taste (e.g. "I dislike being diseased"; "I like being healthy"; etc.)? In opposition here to the Kantian (and Humean) tradition, which takes judgments of (dis)taste as being essentially subjective i.e. that they are judgments about things in terms of (dis)pleasure, I take judgments of (dis)taste as purely objective: they are not to be taken as grounding things (however understood) like beauty or ugliness on (dis)pleasure, but as mere expressions of certain (positive or negative) feelings – which are themselves objective (cf. Exc. 7.2).

Thus, *inappropriate* judgments of taste or – also possibly inappropriate - judgments of *dis*taste (widely taken) (based on e.g. (dis)liking being hurt; over-(dis)liking things like (dis)liking being hurt; or, under-(dis)liking things like (dis)liking being healthy) can be treated as specific higher-level diseases themselves for the case of inappropriate judgements of taste, or healthy processes. Of course, all this does not imply that there are not diverse *specific* (correct) judgements of (dis)taste.

Finally, it is important not to relate, generally speaking, feelings of disease with, necessarily, *unwellness*, as it is unfortunately often done (Schramme, 2016), for one might very well *be* unwell without having any illness viz. any bad feeling, or one can also very well have an illness without being unwell – though for one to *feel* unwell is a different story (cf. Sec. 7.4.1).

7.3 Excursus: The Mind-Body Relationship

Taking a full-fledged stance on the so-called mind-body problem i.e. the issue over what the mind-body relationship is is far beyond the reach of the present PhD dissertation (as an entry, cf. Armstrong, 1999; also Jaworski, 2016).

However, we can assume a certain minimal (Cartesian) *mind-body dualism,* in so far as the position takes both the mind and the body to be genuine (or ontologically *irreducible*) kinds - thus, with their own (genuine) same-level diseases - i.e. that "mind" (widely understood, where e.g. *affective* states/processes belong to the mind) and "body" can be safely considered to be both in the absolute extension of "(natural) kind" – a thesis forcefully rejected by antipsychiatrists (or reductionists) like Kendell (1993), Murphy (2006) or Szasz (1974; cf. Megone, 2000; Schramme, 2013 for discussions) in the case of mental diseases (considered as genuine (natural) kinds), for whom it does not make any sense to separate the mental domain from the physical one.

In other words, more precisely, mind and body are both two (indirect) intensional parts of an organism. If mind-body dualism is truly to be adopted here, then can a *mental* (sign/cause of) disease still be genuine and have as (indirect) constituents, or be indirectly absolutely realized by, *bodily* (signs/causes

of) diseases i.e., more generally, can we sustain to a certain *non-reductive* (type/token) *physicalism* (for concerns about whether a *physicalist* must be a *reductionist* (e.g. a physicalist may claim that only the most *fundamental* entities are biological or physical (restrictedly or technically taken)), cf. Baker, 2009; Pereboom, 2002)?

Our minimal understanding of mind-body dualism does not prevent us to claim that there are, of course, necessary *interactions* (or dependencies) between the mind and the body, but how are we to understand these interactions along a certain non-reductive physicalist line?

I can only briefly touch the question here: exactly like states or processes can be said to be constituted by specific (lower-level) states or processes themselves or to have as absolute realizers specific (lower-level) states or processes, a specific mental (sign of) disease can be said to be (indirectly) constituted by – to avoid a category mistake -, or to have as (*indirect*) absolute realizers, specific (lower-level) mental (signs of) diseases themselves, where these specific (lower-level) *mental* (signs of) diseases can very well be arguably (more *direct*) specific *somatic* (signs of) diseases.

In other words, so as not to reject a whole body of medical literature, especially in *psychiatry* taken seriously here along a permissive neo-Aristotelian ontology (cf. Exc. 3.5), which is now the dominant attitude in philosophy of medicine (Boorse, 1976; 1997; Papineau, 1994; Wakefield, 2006; cf. also Demazeux, 2016), mental (e.g. affective) (signs/causes of) diseases are (indirectly) based on specific *physiological* (or somatic) processes constitutive of/absolutely realizing, generally speaking, any higher-level (sign/cause of) disease: e.g. nervousness or anxiety (as a specific mental (sign of) disease) can have (indirect) constituents absolutely falling under e.g. "stomach ache", "eczema", "hair loss", "(arterial) hypertension", etc., or it can have as an indirect absolute realizer a specific *somatic* (sign of) disease; an attention deficit hyperactivity disease (as a specific *mental* disease) or an addiction can have as (indirect) constituents/absolute realizers functional impairments of neurotransmitter systems like the dopaminergic pathways (Pickard, 2012); or even obsessive-compulsive disorder with (indirect) constituents/absolute realizers like a *damaged* hippocampus, or apraxia with a *lesion* in the dominant brain hemisphere as a(n) (indirect) constituent/absolute realizer (on the difficulties in finding out such (indirect) constituents or realizers, cf. Lemoine, 2015; Poland & Tekin, 2017; Ross, forth.; Uher & Zwicker, 2017; on the current scientific immaturity in psychiatry, Hucklenbroich, 2017).

Two remarks are following from this: first, finding out those indirect constituents/absolute realizers is not an easy task at all, and should not be confused with mere (e.g. neural) *correlates*, which are contingent.

Second, our minimal mind-body dualism also means that, on pain of a category mistake, *one and the same* (very) specific disease cannot be, thus, *psycho-somatic* (viz. e.g. psycho-dermatic), or even (*bio-*)*psycho-social* (or social-ecological), or have mixed mechanisms (Ghiara & Russo, 2018) – although that much does not prevent that there may be, of course, different specific diseases at different (higher or lower) levels with ontological dependency relationships between them (on the famous biopsychosocial model of health (and disease), cf. e.g. Bolton & Gillett, 2019; Farre & Rapley, 2017).

7.1.3 Illness as a Certain Attention Mechanism

How to more precisely analyze what a feeling, generally speaking, as an attention mechanism really is is unsurprisingly a very difficult issue: an attention mechanism can be plausibly (or *indirectly*) defined as a mechanism of reaction to an entity's own neural stimuli.

A bad feeling (the feeling of a disease, or an illness) would be just a *certain* attention mechanism viz. a bad one i.e. that, when ill, a person still has – rather, hosts – feelings in a certain way viz. bad ones. More precisely, an illness (as a specific disease) is arguably the destruction of a certain *positive* entity, which is its host (cf. Sec. 7.1.2 on that): e.g. the (good) feeling of a (healthy) hand or, likely, the *distinct* experiencing of a hand – which can be taken as a *specific* mechanism of reaction to a specific entity's own neural stimuli.

Something (positively or negatively) *un*felt would refer, thus, along this general picture, to any specific mechanism with other properties than (indirectly) a reaction to neural stimuli i.e. that e.g. an unfelt or unconscious disease is *not* a disease, which the host ought to be conscious of; instead, an illness or felt disease is the privation of a certain positive attentional mechanism (turned to itself) an host has[156].

This picture fits well with typical consciousness diseases or disruptions of the (positive) attentional mechanism like the clouding of (good or positive) consciousness, where the host in question is experienced as foggy – taken as a certain *hypo*attention mechanism; or, with e.g. a somatization disorder minimally defined as a certain *hyper*attention mechanism i.e. a mechanism of overreaction to a certain positive mental or bodily (indirect) part's own neural stimuli.

Indeed, when e.g. the *felt* hand i.e. a specific hand – to be distinguished from an *unfelt* hand – is becoming more and more diseased, then the specific (positive) attention mechanism at issue here is also being at the same time diseased; so, as long as I can (positively, or more or less

[156] Caveat (with a terminological note) on unfeelings: a noteworthy concern arising, of course, from decoupling (un)feelings from the mind-body relationship (cf. Sec. 7.1.1) is that it is difficult to define what an *unfelt* (sign/cause of a) disease is, for we would like to typically define "unfeeling" with terms referring to the body.
However, this temptation should be resisted, but note that it does not imply that "unfeeling" is to be understood as a *non*-(natural) kind term: with a broad understanding of "body" (e.g. as the specific part of an organism whose tokens are material or fill in a space-time region), unfeelings are such that they are just incompatible with (or *contraries* to) feelings – not that they are *non*-feelings (or, a *lack* of awareness, while one ought to have it) in the sense of being *contradictories* to feelings, or that "unfeeling" is a general category (e.g. unfelt depression as depression *tout court*), under which "feeling" can absolutely fall.
Indeed, "*un*feeling" or "*un*awareness" does not have to refer only to *negative* states or processes: e.g. the *un*conscious belief that 2 + 2 = 4.

distinctly) feel my hand, the essence of the *felt* hand is not *fully* destroyed – even if there is no e.g. unfelt hand.

However, when I say that *I* - which refers to my person - am *feeling* diseased *from x*, what is diseased is obviously the *felt x* (e.g. a felt hand, etc.), but not – at least, directly – *me* (taken as a genuine entity). If it seems that the host of an illness is the *person* rather than a specific positive mental or bodily part, that is because illnesses are certainly the highest-level diseases easily misidentifiable, thus, with a person (taken as a very higher-level entity too) (cf. Sec. 7.1.1-7.1.2).

Nevertheless, it remains true that, by *x*'s hosting a certain illness, a *person* (as a genuine higher-level entity) is *indirectly* diseased (on the relative extension requirement, cf. Sec. 3.3.1).

7.2 Other Cognates of "Disease"

As a very important concept on its own (with its own growing body of scholarly literature), a separate treatment of the concept of illness is perfectly understandable (cf. Sec. 7.1). To the contrary, Sec. 7.2 reunites diverse – nevertheless important – notions directly related to the one of disease.

As said in the title of Sec. 7.2, the main idea here is to argue that such complex notions related to "disease" can be taken, actually, as specific higher-level diseases themselves.

Sec. 7.2.1 is devoted to the concept of *sickness*, where it is argued that sickness is a suffering or pain;

Sec. 7.2.2 is about the concept of *disability*, which is, as I argue, the feeling of suffering (or pain).

Sec. 7.2.3 is devoted to the concept of *harm*, which is prima facie defined as a strong suffering or pain.

7.2.1 A Prima Facie Definition of Sickness

Sec. 7.2.1 is about the concept of sickness. In Sec. 7.2.1, I defend the prima facie definition of sickness that sickness, like illness (cf. Sec. 7.1.1), just is a specific disease viz. a suffering.

Albeit illness and sickness have been already recognized a long time ago as the two most important pillars (or just examples) of disease or malady (Twaddle, 1968; on malady, cf. Sec. 7.3.2) – which explains, thus, our time spent on them in this doctoral dissertation -, it is quite surprising that there is, to my knowledge, no book-length study of sickness, to the contrary of illness (cf. Sec. 7.1.1), until now (but, cf. Hofmann, 2002; 2017), though the concept of sickness is far from being uncontroversial to define.

Indeed, first, the term "sickness" is, actually, used by medical scientists with no really clear-cut difference from their use of "disease" (cf. e.g. Taber's Cyclopedic Medical Dictionary, 2017) or even "illness" (cf. e.g. Dictionary of Medical Terms, 2004).

However, albeit the etymology of "sickness" is complicated and unclear (however weak as an argument it is to rely on etymology), there is a clear sense of "sickness" originally (at least, from the 16th century) referring to the *twisted mind* – which, as a mental disease, is, thus, something much more *specific* than a mere disease -, and it is hardly believable that the contemporary meaning of "sickness" has changed in such a drastic way that it is just reduced to the one of "disease"[157]; the etymology of "sickness" certainly explains also its common synonymicity with "illness" (cf. Sec. 7.1.1).

Nevertheless, the idea to reduce here sickness to illness is not much appealing neither, for it is intuitive that there are lots of specific sicknesses, which we seem reluctant to also call "illnesses": e.g. motion sickness, homesickness, etc.

Secondly and more importantly, "sickness" is widely taken to be related – but, with no clear reason - to soci(et)al norms (Hofmann, 2002; 2017). More precisely, "sickness" would refer to any pathological condition, for which the authorities, society, and so on, judge that the sick person is not capable of e.g. doing correctly his job, participating to society, etc.: e.g. hypertension would not count as a sickness on this account, but home*sickness* would.

However, on this account, sickness can be considered a certain disease viz. one highly incapacitating. From this, beyond the issue of *arbitrariness* with respect to what should count as *highly* incapacitating, we fail to see – if not by *petitio principii* – why it is up to (public) authorities or society to decide what counts as a sickness or not, or why sickness has to be (inter)subjective; sickness could be just a certain disease, such that, as a mere *consequence* of

[157] Note, however, that the opposite tendency does – and did, indeed – happen, actually: e.g. "in*sanity*" was originally a genuine synonym for "dis*ease*", but the term became very early (and exclusively) used in a more *specialized* sense viz. to refer to *mental* disease (cf. e.g. the classical studies of insanity by Foucault, 1972; also Cooper, 2005 on madness).
The tendency toward the specialization of words – rather than the opposite – can certainly be explained by the historical increased presence of novel (and often borrowed) words into a given vocabulary.

it, the sick person cannot e.g. do correctly or optimally his job, participate to soci(et)al activities, etc.

From our replies to these two attempts at defining (or just reducing) sickness, we can retain that, like illness (cf. Sec. 7.1.1), sickness is a specific disease. But, by contradistinction here with illness, what is this specific disease that sickness is?

What is clear from our replies from the above two attempts is that sickness is a genuine *higher-level* disease viz. one which is both a mental disease and one incapacitating the sick person such that, as a consequence, to prevent her from being part of, globally, society. Finding a coherent middle way between those two ideas at the basis of sickness, while maintaining that sickness is intuitively a specific disease, is not an easy task at all; however I would like to suggest that we can prima facie define sickness as, in all its generality, *suffering* or pain (cf. Box 7.2).

Prima Facie Definition of Sickness
x is a sickness, iff x is a suffering.

Box 7.2. – Prima facie definition of sickness.

From the idea that, consequently, a sick person is not part of, globally speaking, society any longer, we can safely conclude that the sick person is, actually, someone *suffering* or having some pain from something bad (e.g. an unfair situation), a disease (e.g. the suffering of brain cancer would not allow one to be optimally part of society), or even an illness, where, thus, suffering or pain is not to be confused with the *feeling* of disease (for, one can theoretically merely *feel* to have e.g. muscle stiffness, without this being muscle soreness i.e. the suffering or, more precisely, the *bodily* pain associated with it) (cf. also Sec. 5.3.9).

Furthermore, prima facie defining sickness as suffering or pain (cf. Box 7.2) can also easily take into account the idea that sickness is related to the twisted mind. Indeed, a twisted mind is typically someone suffering viz. by having some *mental* pain.

7.2.2 A Prima Facie Definition of Disability

Another important concept related to the one of disease is, of course, the concept of *disability* (handicap or incapacity) (cf. Edwards, 2017; Thompson & Upshur, 2018).

With this case that is disability, we find, in the literature, generally two different accounts of disability (cf., as an entry, Wasserman et al., 2016a-b): (i) according to the "mere-difference" view on disability (Barnes, 2016), for *x* to be disabled just is for *x* to be (positively) different or specific;

(ii) according to the "bad-difference" view on disability, for *x* to be disabled is, to the contrary of theory (i), for *x* to have some bad difference i.e. some impaired, debilitating or damaged function.

Theory (ii) is still today the dominant view in analytic philosophy (Parfit, 1984) and also in medicine, actually (cf. e.g. the Americans with Disabilities Acts (ADA) amendments acts of 2008).

The main argument behind theory (i) of disability is, of course, that it captures how many people say to *experience* disability i.e. not as something bad (vs. theory (ii)), but as something they can very well enjoy.

However, independently of the fact that disability is, obviously, closely related to *disease* (*kinds*) and that it has already been argued that a disease (kind) is a natural kind (cf. Ch. 4), theory (i) of disability can be easily struck with pellets on its own grounds.

First of all, it is strange to still call someone "disabled", if "disability" is understood along theory (i), for the term itself seems to have a pejorative connotation, unless one pleads for a reductive account of disability.

Second, let us assume that overweight is a certain disability; obesophobia - as here a discriminative behavior related to overweight, not as, strictly speaking, a (complete) *fear* of overweight - is also a disease itself viz. a vice (cf. Sec. 10.2.1). Philosophers who argue that overweight is not a disability in the sense of theory (ii) above (but that it is – almost *trivially* (by following PII_2), actually – something just (positively) different, as theory (i) states) want most of the time avoid that these (said) disabled people be discriminated (or offended) (i.e. *ableism*), on basis of their overweight, by typically arguing that what is called a defect is, actually, not one, because it is something natural (as good here) or experienced as good.

For those philosophers, it is as if such a discriminative attitude toward disabled people could be legitimate, or that this attitude could be correct, if such people could be truly said disabled (along (their comprehension of) theory (ii)), but, as the argument goes, they are *not* – at least, not along theory (ii).

For, such philosophers shall prefer arguing that they are not disabled along (their understanding of) theory (ii), or that, if they are truly to be said disabled, then it is along theory (i), by trying to make disability a mere (positive) difference or specificity.

That much can be easily explained by the general (mistaken) idea that disability (like disease) is a certain negative (or maybe positive) value (*subjectively*) defined as a *con-* (or *pro-*) *attitude*. However, we can consistently maintain that overweight is a disability (along theory (ii)) and obesophobia is a specific disease, by rejecting the idea that disability (or disease) is a con-attitude: although it is bad to be overweighted (or e.g. to have a bad sight, etc.), it does not imply that it is right to discriminate overweighted people (or e.g. people carrying glasses because of a bad sight, etc.) on basis of this disability, for such an attitude is also to be judged as a specific disease itself. There is no reason to argue that overweight is not a disability (with theory (ii)), for the notion of disability is not to be grounded on attitudes that we have toward disabled people.

If many people do not want to be called "disabled" (along theory (ii)), it is because they mistakenly make a (direct) *correlation* between disability and negative attitudes that people have towards them: e.g. this idea is at the basis of the anti-psychiatry movement (cf. Papineau, 1994), where psychiatry is seen as a way to discriminate people by pathologizing them with plenty of mental diseases. They prefer, thus, suppressing the problem by arguing that they are *not* disabled (along (their understanding of) theory (ii)), rather than *questioning* the (direct) correlation in question, which should not hold, actually – furthermore, we see no reason why *this* implication holds even indirectly (though a lower-level disease is necessarily relatively realized by another higher-level one belonging to one and the same living being) (cf. Exc. 2.8). Indeed, *x* can be disabled or diseased without giving the right to people to discriminate *x* on basis of the fact that *x* is disabled or diseased. There is *no* (direct) *implication* between *x*'s disability (or disease) and negative attitudes toward *x*[158]; talk about overweight is not talk about the *drifts* related to overweight i.e. obesophobia – that much is not to deny, of course, the reality of those drifts, but only the toxic tendency to count those (natural) concepts as being *theory-driven* or as artifacts (cf. Sec. 3.2.4; for the related controversial concept of race, e.g. Sesardić, 2010 on race objectivism).

Nevertheless, the question remains: if theory (ii) of disability is the one to go along with, what is, more precisely, this *specific* bad difference making someone disabled?

I would like to argue now that, like for the cases of illness (cf. Sec. 7.1) and sickness (cf. Sec. 7.2.1), disability just is, actually, a specific disease. Which one is it?

According to the ADA amendments act (2008, Sec. 3.1),

[158] Other negative attitudes toward disabled people include, to the contrary, e.g. *pity* – especially in the cases, where we judge that the disabled person is *not responsible* for her disability.

> The term 'disability' means, with respect to an individual—
>
> (A) a physical or mental impairment that substantially limits one or more major life activities of such individual;
>
> (B) a record of such an impairment; or
>
> (C) being regarded as having such an impairment […].

According to the WHO's report on disabilities (2020),

> [d]isabilities is an umbrella term, covering impairments, activity limitations, and participation restrictions. An impairment is a problem in body function or structure; an activity limitation is a difficulty encountered by an individual in executing a task or action; while a participation restriction is a problem experienced by an individual in involvement in life situations.

On the one hand, like for sickness (cf. Sec. 7.2.1), a disabling condition also seems a very complex and much encompassing *higher-level* phenomenon, if we are to follow the above attemps from the ADA and the WHO at clarifying what a disability is, according to which a disability is a substantial impairment in one's life (in general) i.e., amongst others, "[…] caring for oneself, performing manual tasks, seeing, hearing, eating, sleeping, walking, standing, lifting, bending, speaking, breathing, learning, reading, concentrating, thinking, communicating, and working" (ADA amendments act, 2008, Sec. 3.2).

However, following this line of thought, and if we would like to maintain the *genuine* character of disability (by not restricting it to a list of major life activities, which shall be inevitably arbitrary), there might exist a collapse with our prima facie definition of sickness as suffering or pain (cf. Box 7.2).

On the other hand, there also exists the possibility to *deflate* the concept of disability by merely stating that having for *x* a *dis*ability just is having for *x* the impossibility to accomplish what he should be *able* to do i.e. any conscious (bodily or mental) activity: e.g. even experiencing the moving of a toe, etc.

However, again, there might also exist here a collapse with our own prima facie definition of illness as the feeling of a disease (cf. Box 7.1).

Nevertheless, a middle ground between those two (extreme) options is still possible i.e., so to say, as a mixture of illness and sickness, but without making it a general concept under which ILLNESS and SICKNESS would fall or an arbitrary concept. Accordingly, a disability might be prima facie defined as a conscious or *felt suffering* or pain (cf. Box 7.3).

Prima Facie Definition of Disability
x is a disability, iff *x* is the feeling of suffering.

Box 7.3. – Prima facie definition of disability.

With Box 7.3, e.g. the incapacity to move one's toe would count as a specific disability, only when it is, in a more specific way, (i) conscious, and (ii) associated with some suffering or pain. Conditions (i) and (ii) are necessary for making disability a *genuine* ontological category – which is intuitively the case -, where a disability would be neither (purely) an illness nor a sickness (cf. Box 7.1-7.2), though many philosophers may want to argue that pain or suffering is essentially conscious or experienced (but, cf. Sec. 5.3.9; 7.2.1 for a rebuttal).
Furthermore, condition (i) allows us to take into account the *experiential* character of disability (cf. theory (i)), while condition (ii) allows us to take into consideration the *higher-level* character of disability, as stated by the ADA amendments act and the WHO[159].

7.2.3 A Prima Facie Definition of Harm

A last important concept, which is worth a section in a PhD dissertation on the concept of disease, is the one of *harm*. Indeed, the concept of harm is at the basis of one of the most influential definitions of a (mental) disease viz. the one proposed by Jérôme Wakefield (1992). Although Wakefield does not give any very precise and clear indication of how to take his notion of harm in his 1992 paper – nor in his subsequent work -, we can suggest, on basis of hints from his definition, where the notion of harm is clearly put alongside what he calls the "*value* criterion" (cf. Sec. 8.2.3 for his definition), that his taking the notion of harm is closely related to the *subjective* evaluation of disease.

[159] Caveat on the relationship between sickness and disability: an obvious consequence of our prima facie definitions of sickness and disability is the following one: if disability is prima facie the feeling of suffering (cf. Box 7.3), and if sickness is prima facie suffering (cf. Box 7.2), then disability is prima facie coextensional with the feeling of sickness. But, are all disabled people really feeling sick? From our definitions of disability and of sickness, we fail to see in what sense this could not be the case, indeed: e.g. a person feeling nauseous and suffering from it can be counted both as a disabled person (i.e. the conscious suffering from the incapacity to correctly digest food in her stomach) and as a person merely feeling sick.
I acknowledge Ulrich Krohs (personal communication, April 2021) for having pointed this consequence to me.

Moreover, along the DSM-5 (APA, 2013, p. 20), the notion of harm is related to "[…] significant distress or disability in social, occupational, or other important activities" i.e. that harm is related to distress or disability, which is mostly taken along theory (i) of disability i.e. subjectively (cf. Sec. 7.2.2).

However, relating harm to subjectivity is a non-starter, for we can very well just *objectively* suffer from a harm or just having a harm (cf. Exc. 7.2).

What is harm? Albeit the – far from being uncontroversial - notion of harm is at the basis of (bio)ethics and political philosophy (on the *primum non nocere* or non-maleficence principle, cf. Hipp. *Epid*. 1. 2, 5), little if anything has been said about how to define the term in all its generality (but, cf. Amoretti & Lalumera, 2019a-b).

As a starting point, in the medical literature (cf. Dictionary of Medical Terms, 2004), "harm" is often related to a term like "(mental or physical) *injury* (or hurt)" or "affliction". If we take them as synonyms, then there is, indeed, a high risk of just confounding harm and disease (in all its generality).

Nevertheless, if we are ready to acknowledge that "harm" is - prima facie, at least - a sui generis notion i.e. here different from disease, then, like for illness, sickness and disability (cf. Sec. 7.1; 7.2.1-7.2.2), harm is (arguably) a *specific* disease. But, what is this specific disease that is harm? Intuitively, we would like to defend that a harm is a peculiarly *strongly* suffered disease (on *sickness*, cf. Sec. 7.2.1). More precisely, a harmful disease could be just this last part of the process of a suffering, which is strong or painful enough to be said, more properly, "harmful". In any case, the boundaries between suffering (or pain) and harm (hurt or injury) (and even agony) look blurry, or they may partially overlap, but our characterization of harm as strong suffering suffices for a prima facie definition (cf. Box 7.4).

Prima Facie Definition of Harm
x is a harm, iff *x* is a strong suffering.

Box 7.4. – Prima facie definition of harm.

Following our prima facie definition of harm as strong suffering (cf. Box 7.4), we can, thus, argue that e.g. a wound would be a specific (bodily) harm or injury viz. the last – strong – part of the suffering process provoked by a damaged skin.

Another typical illustration of harm, as prima facie defined here (cf. Box 7.4), is, of course, the case of detriment, or of *prejudice* i.e. here the strong suffering provoked by a judgment or an action against one's legal rights.

7.3 Why not "Disorder", "Ailment", "Pathology", or etc.?

Why to make DISEASE the overarching concept to study? Is there not a more suitable concept than this one to express what we are talking about? Why not talking about the notion of e.g. disorder, ailment or pathology? Why is what we want to talk about best captured through the notion of *disease*, actually, instead of (an)other existing one(s)?

While we leave open the possibility, of course, to find *true* synonyms for "disease" (cf. Sec. 7.3.2), Sec. 7.3 is about the common notions, which are used exchangeably with the one of disease. Whilst Sec. 7.1. and 7.2 are more about the concepts commonly (and vaguely) related (directly) to the one of disease, Sec. 7.3 is devoted the the different concepts merely used (in the literature) exchangeably with DISEASE.

Sec. 7.3 is divided into two further sections: in Sec. 7.3.1, I highlight mistaken synonyms for "disease";

Sec. 7.3.2 suggests some true synonyms for "disease".

In Sec. 7.3.3, we interrogate ourselves about whether using the term "disease" for referring to what we want is really the best option, after all.

7.3.1 Almost but not Synonyms of "Disease"

To keep things clear for the reader, I pass under review several plausible candidates – the most common ones - supposed to be synonymous with "disease" (largely used as such in the literature, and *passim*) – though they can still be used under their plausible reading, of course, as genuine synonyms for "disease"[160]:

[160] For some of them, it might be even true that we just *have to* use them under a plausible meaning, because of a lack of linguistic resources: e.g. "etio*pathology*" is used to refer to, in all its generality, the negative causes of *disease* – and not of a specific disease.

- *disorder*: as the most commonly used synonym for "disease", the term "disorder" is, actually, highly misleading; to use it as a mere synonym for "disease" – especially, when talking about *mental* diseases (e.g. "autism" has becomed "autism spectrum *disorder*") – should be, *strictly* speaking, avoided.

  Indeed, the term "dis*order*" is merely *normative* – and not (also, or more specifically) axiological (to the contrary of what we are after with "disease"): e.g. it is normal or *ordinary* for a psychopath to lack empathy; thus, for a psychopath, not lacking empathy would be a *dis*order, albeit this is clearly not a disease; or e.g. a sleep disorder does not necessarily mean, as such, that it is something bad, for "sleep disorder" may very well refer to a disorder of some *bad* sleep.

  Moreover, "disorder" does not have also to refer merely to (parts of) organisms, but can very well *literally* apply to other cases.

  Of course, the term "disorder" can still be plausibly used synonymously with "disease" - especially when we follow some common scientific disease denominations (e.g. "somatization disorder", "obsessive-compulsive disorder", "PTSD", etc.), for the sake of situating the example within a certain given (and widely accepted) tradition, or for the sake of simplicity, or to avoid misleading denominations; for the other cases, where one could not be easily misled, using the term "disease", instead, is the best option: e.g. "mental disease" rather than "mental disorder";
- *ailment*: "ailment" is an old term referring to, actually, some *specific* higher-level disease viz. the one including other (more) specific diseases like a pain (cf. Sec. 7.2.1), a trouble, a (di)stress, an affliction (cf. Sec. 7.2.3), a torment, a turmoil, a trauma, etc.

  However, the term "ailment" is still not as much of a generality than the one of disease, for e.g. a lower-level disease like hypertension is generally not referred to as an ailment (cf. Dictionary of Medical Terms, 2004);

  Furthermore, "ailment" can also have a positive dimension in the sense of being an *affection*;
- *pathology*: if there is a term that we would like to unabashedly use as a synonym of "disease", then it is certainly the term "pathology" ("pathosis" or even "pathomechanism"). And yet, etymologically speaking (and also *strictly* speaking, actually), "*patho*logy" goes back to the discipline studying (or the subject matter that is) the *πάθος* - and not *disease* (or, so it seems) - i.e. a (positive) feeling or a *pass*ion (etymologically taken).

  In other words, first, "pathology" seems more restricted than what we want to talk about with "disease", for it does refer to a passion, which can be taken as a mere specific disease

(cf. Sec. 7.1 on *illness*); second, "pathology" does not merely refer to something negative, but also positive (unlike disease): e.g. "a-*pathy*" or "anti-*pathy*" seems to refer to the absence of something *positive*, indeed, viz. some *πάθος* taken as a positive feeling here (though linguistic usage can be easily misleading in related cases, of course);

- *ill health*: the main advantage of this – almost – synonym of "disease" is that it puts emphasis on the priority of health over disease, by explicitly showing us that disease as an ill health is the absence of *health* - and not the contrary (*pace* Boorse, 1977).

  Beyond that very weak point – which can very well be misleading -, we fail to see in what sense an *ill* health is different from, indeed, an *illness*, which is a *specific* disease (cf. Sec. 7.1); we can further notice that, if there exist many different specific diseases like illness, as the expression "ill *health*" shows us, the term "health" does not seem to have its own counterpart for "illness" – even "wellness" cannot play this role, actually;
- *unwellness*: indeed, as another very common term used exchangeably with "disease", *as such*, unwellness cannot be a synonym of "disease". For, along a deflationary or minimal theory of wellness (welfare or well-being) (but, for other theories in this extensive literature, cf., as an entry, Crisp, 2017), the well-being of *x*, generally speaking, can be just taken as *x* being well, where *x* is not necessarily (the part of) an organism: e.g. my washing machine can be or fare well, just like my heart process can be or fare well; thus, to the contrary, my washing machine or my heart process can be unwell;
- *infirmity*: what about "infirmity" (or "invalidity") as a synonym for "disease? As the word "validity" (or "firmity") comes, at the origin, from condensed matter physics, "invalidity" (or "infirmity") would certainly be suitable to somatic diseases. Albeit the word "infirmity" (or "invalidity" has been extended, of course, also to *mental* diseases, we fail to see in what sense this extension can be really litteral – and not merely *metaphorical* through the very widespread use of the metaphor of the mind as a machine (or as a computer).

  Thus, "infirmity" (or "invalidity") is suitable to some diseases only viz. bodily ones;
- (biological) *parafunctions*: the first advantage coming to mind with the use of parafunctions is that, etymologically, the prefix *para-x* also refers to something resembling *x*.

  However, beyond this, we fail to see in what sense the notion of parafunction can be a genuine synoym for "disease, for like for "disorder" the term "parafunction" is only *normative* – and not also axiological.

7.3.2 Synonyms of "Disease"

To the contrary of Sec. 7.3.1, Sec. 7.3.2 passes under review some different *genuine* synonyms of "disease" – which are used *passim* throughout the whole PhD dissertation -, albeit the term "disease" is certainly the one to be preferred for referring to what we want to (cf. Sec. 7.3.3):

- *unhealthiness*: there is no a priori reason not to use the term "unhealthiness" as a true synonym of "disease". Again, as for ill health (cf. Sec. 7.3.1), unhealthiness has the advantage of putting priority of health over disease, although this weak point should be taken with much precaution.

  However, the term "disease" is certainly to be preferred, for reasons having to do, first, with *syntactic simplicity* – for, we shall hardly refer to specific diseases as specific *unhealthinesses*, but rather as specific *unhealthy processes* (like for disease processes); second, with the common *scientific denominations* of specific diseases (or unhealthy processes): e.g. we are used to talk more about bacterial diseases than about bacterial unhealthy processes.

  Nevertheless, beyond this argument from syntactic simplicity for preferring the use of "disease" over "unhealthiness", "unhealthiness" is a genuine synonym of "disease";
- *unwholesomeness*: the term "unwholesomeness", as its etymology suggests, seems prima facie applicable beyond the field of medicine. However, this usage is not attested (any longer). That is why, there is also no reason not to use "unwholesomeness" synonymously with "disease".

  Again, beyond the syntactic simplicity associated with the term "disease" – instead of "unwholesomeness" – (and, thus, the more widespread use of "disease" rather than "unwholesomeness), a further reason for preferring using "disease" is precisely that it is (still) not entirely clear whether "unwholesomeness" cannot be applied not merely to medicine;
- *morbidity*: as a much used term in medicine, I see no reason why "morbidity" (or "morbus") could not be used as a mere synonym of "disease"[161].

[161] Terminological note on co-/multi-morbidity: our anti-reductionistic stance (cf. Exc. 3.5), in general, allows us to save the phenomenon of *co-* (or *multi-*)*morbidity* i.e. that, if a specific disease is *necessarily* (at least, directly) absolutely realized by another specific *lower-level* disease (cf. Sec. 3.3.1), then there are *simultaneously* (or *concomitantly*) (*at least*) two specific *independent* - in the sense that, like for the case of a differentia defined independently from what a genus is, the specific realizing disease is not *essentially* what the specific realized disease is and vice versa - genuine diseases.
The relationship of absolute extensionality between two specific diseases allows us to give a precise or accurate meaning of "co- (or multi-)morbidity", which is (vaguely) said to refer to (at least) two specific simultaneous and

However, again, to the contrary of "morbidity", - however weak as an argument it is -, the term "disease" seems to have the slight advantage of capturing in a greater number specific diseases or morbidities, by following their common scientific denominations: e.g. if "STD" is for "Sexually Transmitted Disease", there does not exist, to my knowledge, the denomination "STM" for "Sexually Transmitted Morbidity";

- *malady*: as a last genuine synonym for "dis*ease*", "malady" (or "mal*aise*") can, thus, be used exchangeably in principle with "disease".
  However, its lack of predicative function makes the term "malady" *less complete* than "disease": e.g. a disease*d* heart process (by contrast with a heart malady/disease), a disease*d* liver (by contrast with a liver malady/disease), etc.

7.3.3 Why "Disease"?

Of course, depending on the specific theory of disease adopted (cf. Ch. 9), many more genuine (and complex) synonyms can be used for "disease" (e.g. "defect of a living being", "destructive process of health", etc.), just like there will also inevitably be other mistaken synonyms.
However, in Sec. 7.3.1-7.3.2, our point was precisely to discuss potential synonyms for "disease, no matter which *specific* theory of disease is argued for.
If we are now clear about which almost but not (cf. Sec. 7.3.1), and genuine, synonyms there are for "disease", even though we can accept the above reasons given for still preferring the term "disease" over its competiting terms (cf. Sec. 7.3.2), we might wonder whether "disease" is really the best - or, at least the least bad – term to use for referring to what we want to refer.
Although all the genuine synonyms for "disease" (or even "diseasedness") have their own defect, maybe this is also the case for "disease" – which would not make it, thus, really preferable to use over its other competitors.
As "ease" etymologically refers to (physical) comfort, pleasure, peace of mind, tranquility, freedom in one's movements, or even freedom from pain or moral constraint, dis*ease* (like for mal*aise* or mal*ady*, actually) seems to be, rather, a *certain* morbidity – and not a general

independent diseases (of different *levels*): e.g. *x* can host both a certain (generally characterized) disease (kind) and indirectly a more specific one;
cases of two (or more) *same-level* diseases are not to be considered, thus, as cases of co- (or multi-)morbidity: e.g. deafblindness is *not nothing* else than two diseases viz. deafness and blindness, but a *single* specific disease constituted by two other specific diseases.
Of course, not everyone agrees on this (traditional) definition of co- (or multi-)morbidity (Valderas et al., 2009).

category encompassing all the specific morbidities, or being at the same level of generality than the category of morbidity or unhealthiness.

Albeit the terms “unhealthiness”, “unwholesomeness” and “morbidity” all have also their own defects (cf. Sec. 7.3.2), the term “disease” does not seem to fare better – worse, it seems to behave more like illness, sickness, disability, or harm, than morbidity or unhealthiness (cf. Box 7.1-7.2-7.3). Is there not, thus, a better term than “disease”?

Actually, the above etymology of “ease” is already the *specialization* of an older – much more general – sense of “ease” *still* attested *literally*, which somehow unites all the above more specialized senses under a single idea, and allows us to maintain the intuition that disease is a much more encompassing category (for, to the contrary of e.g. illness or sickness (cf. Sec. 7.1; 7.2.1), “disease” does not refer intuitively to (a) specific higher-level morbidity(/-ies)).

Indeed, there also exists a general sense of “ease” referring to any specific healthy process happening to an organism(’s parts); to the contrary of the term “disorder” (cf. Sec. 7.3.1), “disease” is, thus, *axiological* – and not merely normative.

Though not perfect – mainly because of the plural meaning of “ease” tending to make “disease” several specific morbidities -, amidst the genuine synonyms of “disease” (cf. Sec. 7.3.2), the term “disease” itself is, eventually, the least bad (available) term to use, or so it seems, for referring to what we want to.

And, indeed, what is it that we want to refer to?

III

THE CONCEPT OF DISEASE

8

THE PHILOSOPHICAL DEBATE AROUND DISEASE

Once that we are clear about what disease is not, and to which terms the notion of disease should not be confounded, as well as about the concepts in which DISEASE is embedded (cf. Part II), we can now, in Part III, begin directly our analysis of what disease is. Part III of the present PhD dissertation is devoted to the concept of disease.

More specifically, Ch. 8 of the present thesis aims at situating, first, our own correct definition of disease (cf. Ch. 9) within a certain tradition or line of thought i.e. three current groups of theories of disease viz. axiologism about disease, malfunctionalism about disease, and hybridism about disease. These three groups of theories of disease are distinguished with respect to the emphasis the theories of disease they include put on one or both of the two (main) intuitions that we have about disease.

In Sec. 8.1, I present the two main intuitions that any complete theory of disease should ideally take into account.

Sec. 8.2 is devoted to present the above three groups of theories of disease viz., respectively, axiologism about disease, malfunctionalism about disease and hybridism about disease, by briefly and critically illustrating them (with the most discussed theories).

8.1 Two Intuitions about Disease

Any reader who is reading this sentence has already been diseased at least once in his/her life (cf. Sec. 9.3.3 for how to understand ascriptions of health/disease to whole living beings). Diseases are ubiquitous in our lives (or other organisms); indeed, the concept of disease intuitively includes cases as diverse as fatigue, fever, cardiomyopathy, AIDS, psychopathy, lung cancer, headache or genetic mutations; but the concept of disease seems to also exclude cases like drapetomania (i.e. the tendency of African-American slaves to flee from their servitude), while other cases are more controversial such as intersexuation (cf. Office of the High Commissioner for Human Rights, 2015) or the pre-menstrual syndrome. Which justified, non-arbitrary and -vague criteria are there for distinguishing between diseases and non- (or pseudo-) diseases? What is disease?

From the Hippocratic theory of disease as disturbance (or imbalance) between the four humors (or temperaments) to the BST of disease (and health) as a biological process of a part of a living being deviating from a norm statistically established (within a reference class) through other various theories of disease (cf. *passim* for a sample of those theories; also Pellet, 2016; 2018), philosophers have been trying (unsuccessfully until now) for a long time already to thoroughly analyze this elusive and complex concept that is DISEASE.

In the contemporary literature about the nature of disease, we can distinguish between three groups of theories of disease, which may be labeled "axiologism about disease", "malfunctionalism about disease" and "hybridism about disease" (cf. Ereshefsky, 2009; Pellet, 2018; 2019); these three groups of theories of disease are distinguished with respect to the emphasis the theories of disease they include put on one or both of the two (main) intuitions that we have about (what) disease (is) (cf. Sec. 8.2)[162]: (1) saying that e.g. cell growth is

[162] Caveat on (philosophical) intuitions: of course, we have in mind here, as a starting point, our own metaphilosophy based on a priori reasoning (cf. Ch. 2-3).
However, not accepting this metaphilosophy does not imply, necessarily, the rejection of this starting point here i.e. with (philosophical) intuitions.
Indeed, as we shall see (cf. e.g. Sec. 8.3), e.g. a theory belonging to the group "malfunctionalism about disease" does not have to follow, strictly speaking, our own metaphilosophy (or even, can reject it), but it remains true that this theory can be *objectively* classified as belonging to this group based on the main intuition about disease the group tries to take into account.
Subscribing to this starting point here does not imply that one has to subscribe to my metaphilosophy (as a whole package, so to say).
Indeed, this starting point says nothing about the nature of intuitions themselves: e.g. one could very well take intuitions as having to do merely somehow with a *discovery* process, but nothing to do with the *justification* of a theory (on this famous dichotomy, cf. Reichenbach, 1938), or one could also take intuitions more seriously as *evidence* for a theory, or as *pre-theoretical judgments* (on my *common sense*-based philosophy, cf. Ch. 2-3), etc.
With a weak enough (plausible) conception of (philosophical) intuitions, it seems rather implausible to reject from the outset our starting point here.

cancerous is making a specific *negative value judgement* toward cell growth, where the value at issue is intuitively a certain *lethal* one (like death), by contradistinction with a vital value like health or life; (2) saying that cell growth is cancerous is saying that cell growth is *biologically malfunctioning* in a specific way[163].

Sec. 8.1.1-8.1.2 are devoted to present into more details intuitions (1) and (2), respectively.

8.1.1 Disease as a Specific Lethal Value

How do we come to intuit that disease is, amongst others, a specific *negative value* viz. a specific *lethal* value? We have the intuition about disease that attributing to a subject *x* (e.g. "liver", "lung's tissues", "cell growth", etc.) the predicate "_is diseased" is negatively evaluating (or, *disvaluing*) *x in a specific way*. In a nutshell, saying that *x* is diseased is a (specific) *negative evaluative judgment*.

However, as they are many different specific (positive or negative) value judgments (e.g. aesthetic judgments, etc.), what is this specific negative evaluative judgment that we intuit about (the nature of) disease?

We have the intuition that the specific way in which disease is a negative value is that disease is a certain *lethal value* (as a thinner value than disease).

Indeed, if it is common, especially in the Anglo-Austrian tradition, to hold that *life* and *health* are specific positive values viz. specific *vital* values (Mulligan, 2009; Scheler, 1921; von Wright, 1963) with the idea in mind that those values have to do with, indeed, what is primordial

[163] Two caveats on intuitions (1) and (2): (i) what about the case where one does not share intuitions (1) and (2) as a starting point for investigating the nature of disease (cf. Box 8.1-8.2)? In addition to being outside the classical analytic literature on the concept of disease, if one does not want to follow intuitions (1) and (2), there is still the possibility to argue, nevertheless, that, if one shares other intuitions about (supposedly) the nature of disease, then those intuitions can certainly be classified as genuine intuitions not so much about the nature of disease than about, rather, the nature of something else *related* to disease: e.g. about human attitudes towards disease, the experience of disease, our knowledge of disease, etc.
I thank Ulrich Krohs for having pointed this to me (personal communication, April 2021).
(ii) How to treat *plausible* cases of diseases, which are not essentially defined as we do (e.g. in different medical specialties) (cf. Box 9.3)? For such cases, we can argue that either these are diseases, which are, indeed, only plausibly non-essentially defined i.e. that, *strictly*, these diseases are to be essentially defined as we do, or these are *not* (true) diseases; these can be considered as, typically, destructive processes of *accidental* or *extrinsic* properties like, plausibly, *having a sound* for a heart process (while a heart sound is in itself a certain (qua) object with its own diseases like a heart murmur), or *stopping being a musician* for a human being (on accidental change, cf. Sec. 3.2.6; Exc. 3.9, for our understanding of accidental and extrinsic properties; for controversial cases of disease, Sec. 10.2-10.3).
I deeply acknowledge Ozan Altinok for this question (personal communication, June 2021).

to a *living* being, then we can say that disease is, amidst others (e.g. *death*), to the contrary, a specific *lethal* value (cf. Box 8.1).

Intuition (1) about Disease
Disease is a specific negative value viz. a specific lethal value.

Box 8.1. – Intuition (1) about disease.

8.1.2 Disease as a Specific Biological Malfunction

How do we come to intuit that disease is a certain *biological malfunction*? In addition to intuition (1), according to which disease is a specific lethal value (cf. Box 8.1), we have another intuition about disease, according to which, when we judge that an entity *x* (e.g. liver, lung's tissues, cell growth, etc.) is diseased, we mean by this that *x badly biologically* functions (or, biologically *mal*functions, or is biologically malfunctioning) (cf. Box 8.2) *in a specific way* (where a living being as a whole can also badly biologically function) – by using widely or narrowly the notion of biology (cf. Sec. 9.3.2 on that)[164].

Intuition (2) about Disease
Disease is a specific biological malfunction.

Box 8.2. – Intuition (2) about disease.

8.2 Groups of Theories of Disease: A Framework

[164] Caveat on intuitions (1) and (2) (cf. Box 8.1-8.2): by saying, with intuition (2), that disease is a specific biological malfunction in the sense that disease is something *badly* biologically functioning in a specific way, we may rightly wonder whether, actually, intuition (2) does not reduce to intuition (1), which takes disease as a certain *disvalue*.
Though we argue for an *intertwinement* between intuitions (1) and (2) (cf. especially Fig. 9.4), they can be meaningfully kept separate here for the sake of providing a neutral framework, for nothing precludes, actually, to sustain to a value-free analysis of intuition (2) e.g. by taking the notion of a bad biological malfunction as a mere biological dysfunction i.e. an *incorrect* biological function (cf. Sec. 8.2.2).

If we are clear that any *complete* theory of disease should be based somehow on intuitions (1) and (2) (cf. Sec. 8.1), what is it to say, more precisely, that disease is a specific negative value viz. a specific lethal value? What is it to say, more precisely, that disease is a specific biological malfunction?

A framework for (thinking) the debate about disease, or for classifying the current theories of disease, can be provided on basis of *which* of these (most basic and vague) intuitions (1) and (2) (cf. Box 8.1-8.2) that we have about disease different theories of disease take into account *the most seriously*, and *how* (*differently*) those theories further analyze or understand intuition(s) (1) and/or (2), however how those theories understand what "analyzing intuition(s) (1) and/or (2)" means: viz. e.g. "taking intuition(s) (1) and/or (2) as defining features of disease"; or "taking intuition(s) (1) and/or (2) as a mere basis for investigating how scientists use the term "disease""; or etc.

Three groups of theories of disease can be distinguished with respect, thus, to the emphasis the theories of disease they respectively include put on intuition(s) (1) and/or (2):

1. the first group, which can be labeled - somewhat arbitrary, but still quite precisely - "*axiologism about disease*", includes theories of disease seriously accounting for and further analyzing intuition (1), and somehow explaining away intuition (2);
2. the second group of theories of disease, which can receive the – quite neutral - label "*malfunctionalism about disease*", includes theories of disease seriously taking into consideration and further understanding intuition (2), and somehow explaining away intuition (1);
3. the third and last group of theories of disease, which can be coined, in line here with the literature, "*hybridism about disease*", includes theories of disease seriously based on and further analyzing intuitions (1) *and* (2)[165].

[165] Three caveats (with terminological notes) on axiologism and malfunctionalism about disease here: (i) in the literature, the first group is most often called "normativism about disease", while the second "naturalism about disease" (cf. e.g. Ereshefsky, 2009; for the case of mental disease, Graham, 2013; Thornton, 2007).
Concerning the first group, though the expression "normativism about disease", as such, does not exclude *objectivist* value analyses, actually, talk about "axiologism about disease" is, first, *more precise* – for, it specifies that disease is a value, and not merely a normative entity -, and, second, due to the fact there is a third hybrid group of theories of disease, talk about axiologism and malfunctionalism about disease seems *less opposing* than talk about normativism and naturalism about disease (cf. caveat (iii)).
Thus, it is preferable not to use the expression "normativism about disease".
(ii) By contradistinction with (said) normativism about disease, where values, generally, are most often taken as *subjective* –, naturalism about disease is especially understood as a *value-free* (understand: objective) analysis of disease (cf. Exc. 2.6; also Boorse, 1977; also Wakefield, 1992).
However, we can claim the superiority of our label "malfunctionalism about disease" here, which, by being situated a little bit outside the common denomination found out in the philosophical literature, is likely to be less prone to theory-laden analyses or other common *doxas* i.e. to be (too much) *exclusive*: e.g. which mis-conceived ideas talk about malfunctionalism about disease is really likely to elicit, by contrast with naturalism about disease? Ditto here for axiologism about disease.

A major motivation for subscribing to axiologism or malfunctionalism about disease, instead of hybridism about disease, is that many philosophers of medicine (wrongly) take disease to be either normative in the sense of subjective (i.e. belonging for them to axiologism about disease) or natural in the sense of objective (i.e. belonging for them to malfunctionalism about disease) (cf. Sec. 8.2.1-8.2.2).

Sec. 8.2 is further divided into three sections corresponding to, respectively, axiologism (Sec. 8.2.1), malfunctionalism (Sec. 8.2.2), and hybridism about disease (Sec. 8.2.3).

Sec. 8.2.1-8.2.3 more thoroughly present those three groups of theories of disease by briefly and critically illustrating them (with, mostly, targeted objections, but also some more general ones) with the most discussed theories of disease.

8.2.1 Axiologism about Disease: Nordenfelt's Subjectivist Theory

Axiologism about disease is a group of theories of disease, which put the emphasis on intuition (1), while somehow explaining away intuition (2).

Intuition (1), according to which disease is a specific negative value viz. a lethal value (cf. Box 8.1), has been variously further analyzed in the philosophical literature (cf. e.g. Megone, 2000; 2007), but most theories of the group "axiologism about disease" subscribe to a *subjectivist* account of (negative) value, generally, according to which, disease is defined as a specific *negative attitude (*or *con-attitude*) (cf. Cooper, 2002; Engelhardt Jr., 1976 ; Sedgwick,

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That much is more than a mere terminological issue; this is about finding out the least bad non-theory-laden label(s) (e.g. by being close to "functionalism", "malfunctionalism" is also not quite perfect, though still less prone to theory-laden analyses than "naturalism"), which shall not exclude, thus, from the outset any theory of disease.

Due to its many diverse and inconsistent interpretations, it is preferable not to use the label "*naturalism* about disease" to refer to our second group of theories of disease, though that much does not mean that talk about naturalism (about disease) should be banned *tout court*, of course; ditto for normativism (about disease).

(iii) A major problem already pointed out with the current dichotomy normativism/naturalism about disease (cf. caveat (i)) is that, if naturalism has really to do with a value-free analysis of disease, while this being contradictory to normativism about disease, then *hybridism* about disease is a contradictory thesis (cf. Sec. 8.2.3): a disease would be both something (partly) normative and (partly) natural; but, note that this point is present, indeed, in some theories of thick values, according to which a thick value like disease contains both a *normative* and a *descriptive* (understand: natural) element (cf. Gibbard, 1992; Hare, 1952; also the *secondary qualities* of McDowell, 1985).

1982; Tappolet, 2011): e.g. a liver disease, lung cancer or headache is an undesirable or unpleasant state (or process).

Intuition (1), more precisely, of disease as a specific *lethal value* has been further understood, most notably by Lennart Nordenfelt (1995; 2000) in his attempt to define disease as an undesirable or unpleasant state preventing a subject to accomplish his *vital goals*, which "[…] are necessary and together sufficient for his minimal happiness [in the long run]" (Nordenfelt, 1995, p. 68; cf. also e.g. Fritzson, 2018; Richman, 2004; Venkatapuram, 2013) i.e. that those vital goals are the ones making the overall organism happy in the sense of having a minimally decent life (Nordenfelt, 2000; cf. also Nussbaum, 2000 for a list of what constitutes a minimally decent life).

This theory is, of course, not immune to objections. First, because of its reliance on subjectivism about (negative) values, we can raise against Nordenfelt's subjectivist theory of disease the same objections commonly raised against subjectivism about (negative) values: e.g. that entities have (negative) values, not because we have (con-)attitudes toward them, but we have (con-)attitudes toward entities, because those entities have (negative) values (for further objections, cf. Massin, 2010; on *objectivism about* (negative) *values*, Foot, 2001; Megone, 2000; Pellet, 2018; Thompson, 2008; also Hamilton, 2010; *pace* Glackin, 2016; Wakefield, 2000).

Secondly, another (weak) objection (against the sufficiency condition) is due to Thomas Schramme (2007): e.g. an athlete becomes unhappy, because of his non-participation to the 2024 Olympics, where *for him* participating to the 2024 Olympics was a vital goal in the sense that it makes his life minimally indecent; but, the non-participation to the 2024 Olympics (as somehow an undesirable state for an athlete) does *not* seem a case of disease (even for an athlete); talk about vital goals (even minimally understood i.e. as what tends a subject towards its minimal happiness in the sense of having a minimally decent life) seems *too broad* a condition (cf. Venkatapuram, 2013 for the same point; as a tentative reply, Nordenfelt, 2013b); furthermore, a list of what constitutes a minimally decent life has a high risk of *arbitrariness* (and human-centerness).

Finally, the way in which Nordenfelt's subjectivist theory of disease can be a *complete* theory of disease belonging truly to the group "axiologism about disease" is not clear at all; indeed, it tells no word about how to explain away in a certain way intuition (2).

8.2.2 Malfunctionalism about Disease: Boorse's Bio-Statistical Theory (BST)

Malfunctionalism about disease is a group of theories of disease, which focus on and further analyze intuition (2) about disease, while explaining away somehow intuition (1) (cf. Box 8.1). Again (cf. Sec. 8.2.1), intuition (2) about disease, according to which disease is a certain biological malfunction (cf. Box 8.2), has been also variously further analyzed in the literature: the most famous theory of the group "malfunctionalism about disease" is certainly the BST of disease (for other theories, cf. e.g. Chin-Yee & Upshur, 2017; Griffiths & Matthewson, 2018), according to which *x*'s biologically malfunctioning is *x*'s deviating from its normal biological functioning i.e. a statistically typical contribution of *x* (within a reference class) to the inclusive fitness of the organism of which *x* is a part (cf. Boorse, 1977; 1997): e.g. saying that a heart process is diseased is saying that a heart process badly functions (or is malfunctioning), in the sense that it deviates from the heart process's "[…] pumping in the appropriate way, [i.e.] when, given statistically normal circumstances, it makes its species-typical contribution to the individual's survival and reproduction" (Nordenfelt, 2018, p. 11) i.e. when the heart process's biological function is statistically determined within a chosen reference class with respect to a heart process's typical contribution to the inclusive fitness of the whole organism of which the heart process is a part.

Moreover, the BST of disease tries to explain away intuition (1) about disease by merely *rejecting* it i.e. by arguing that DISEASE is a *value-free* concept i.e. here a concept not defined as a con-attitude - to the contrary of ILLNESS (cf. Sec. 7.1.1; Boorse, 1975; 1977).

As being perhaps the most well-known theory of disease, the BST of disease has been also variously criticized, most notably on grounds that the choice of the reference class is *arbitrary*, and that the notion of a deviation from a statistically typical contribution to inclusive fitness is *vague* (on those criticisms, cf. Azevedo, 2015; Casini, 2017; Cooper, 2002; Giroux, 2009; Kingma, 2007; 2010;Sarto-Jackson, 2018; *contra* Broadbent, 2019; Dussault & Gagné-Julien, 2015; Hausman, 2014).

Another interesting criticism against the BST of disease (and health) is that the method of conceptual analysis – based on *value-laden* assumptions, mostly because of the arbitrary choice of the reference class (DeVito, 2000; Varga, 2018) - is incompatible with the (said) *naturalism* (i.e. here the value-free analysis) a proponent of the BST of disease (and health) advocates for

the concept of disease (and health) (but, cf. Schwartz, 2014; for further criticisms, Sec. 2.1; 3.3.1).

8.2.3 Hybridism about Disease: Wakefield's Harm-Malfunction Theory

To the contrary of the theories of disease belonging to the groups axiologism and malfunctionalism, which all put the emphasis on *either* intuition (1) *or* (2) (cf. Sec. 8.2.1-8.2.2), hybridism about disease comes with the prima facie advantage of having theories seriously taking into account *both* intuitions (1) and (2) (cf. Box 8.1-8.2); nevertheless, this advantage is counter-balanced by the difficulty for those hybrid theories of disease to find some *coherence* (and logical consistence) between intuitions (1) and (2) – which is not really easier to find than to somehow explain away either intuition (1) or (2), actually.
One of the most famous and discussed hybrid theories of disease is the *harm-malfunction theory* developed, first and foremost, - in the context of mental diseases, but which can be easily generalized -, by Jérôme Wakefield (1992; p. 384; my emphasis; for other hybrid theories, cf. e.g. Matthewson & Griffiths, 2017; Megone, 2000; 2007; Thompson, 1995):

> [a] condition is a disorder [understand: disease] if and only if (a) the condition causes some harm or deprivation of benefit to the person as judged by the standards of the person's culture [or to any other organism as judged by their own standard[166]] (*the value criterion*), and (b) the condition results from the inability of some internal mechanism to perform its natural function, wherein a natural function is an effect that is part of the evolutionary explanation of the existence and structure of the mechanism (*the explanatory criterion*).

Condition (a) i.e. the idea that disease is a lethal value, in the sense that it causes some harm to a person (or any other organism), and condition (b) i.e. the idea that disease is a certain biological malfunction, in the sense that a biological function taken as a naturally selected effect is unable to be performed try to clearly capture the *dual* aspect of the concept of disease i.e. intuitions (1) and (2) in a single coherent way (cf. Box 8.1-8.2).

[166] This addition is necessary, for Wakefield's theory is developed, above all, for the cases of mental disease, which he seems to ascribe only to human beings.

However, the harm-malfunction theory is not exempt from shortcomings. First, the notion of harm, as it seems understood here, i.e. as having to do with a *subjective* aspect behind the concept of disease is *not necessary* for defining certain clear cases of disease (cf. also Sec. 7.2.3): e.g. we would hardly deny that there can be cancerous processes – at least, their starting point -, which are not subjectively evaluated as negative; in that sense, albeit they are clearly diseases, they do no *harm* (as understood here) to the person, for Wakefield, by using the DSM-3-R, clearly associates harm with "[…] present distress (a painful symptom) […]" (APA, 1987, pp. xxii), and relies on the dichotomy between values and facts.

Second, one can also argue that conditions (a) and (b) of Wakefield's harm-malfunction theory are not jointly sufficient for a theory of disease; indeed, one might well argue that there was a certain historical period in a certain cultural environment, where *drapetomania* was, actually, deemed a certain disease i.e. where, to the contrary, slavery was considered no harm to enslaved people, and where for *x* to be enslaved is for *x* to perform its biological function as a naturally selected effect (cf. also Ari. *Pol*. 1, 1254b16-21); but, we would like to answer here that drapetomania was never, is not, and shall never be a certain disease in whatever culture and at any historical period whatsoever; Wakefield's cultural *relativism* is here problematic.

Third, as a fatal flaw for Wakefield's theory, we fail to see in what sense the above definition is not *contradictory* (or incoherent) in its terms, by affirming that disease would have both, under condition (a), a *subjective* aspect related to its being a specific negative value viz. a lethal one and, under condition (b), an *objective* aspect related to its being a certain biological malfunction (for further objections to Wakefield's theory, cf. Jablensky, 2007; Murphy & Woolfolk, 2000a-b; Sadler & Agich, 1995).

9

A DEFINITION OF DISEASE

Ch. 9 of the present PhD dissertation is devoted to propose and defend our own original definition of disease, according to which, in a nutshell, disease is the destruction (or deprivation) of the essence of a processual part of a living being.

In Sec. 9.1, after a plea for hybridism about disease (cf. Sec. 8.2.3), I defend this prima facie hybrid definition of disease, and I show how it takes coherently into account both intuitions (1) and (2) about disease (cf. Box 8.1-8.2).

In Sec. 9.2, I refine our prima facie definition of disease to get a correct definition of disease i.e. one situated within a genus-species hierarchy (cf. Box 3.1), according to which disease is a negative process of a part of a living being (i.e. the genus), and is the destruction of a positive state p until the negative state $\neg p$ (i.e. the differentia).

How to understand further this definition? How does it analyze into more details intuitions (1) and (2) about disease?

Starting from our correct definition of disease, in Sec. 9.3, I analyze further what the disease host as the essence of a processual part of a living being is.

In Sec. 9.4, I analyze into more details what “essence destruction” really means.

9.1 A Prima Facie Hybrid Definition of Disease

Sec. 9.1 is devoted to presenting our prima facie definition of disease. Like for our correct definition of disease (cf. Box 9.3), our prima facie definition of disease is to be intuitively situated within the group *hybridism about disease* (cf. Sec. 8.2.3) i.e. within a group of theories of disease seriously taking into consideration both intuitions (1) and (2) that we have about disease (cf. Box 8.1-8.2).

To the contrary of axiologism and malfunctionalism about disease (cf. Sec. 8.2.1-8.2.2), we see, actually, no a priori reason why either intuition (1) or (2) should be somehow explained away in favor the other one, or why one rather the other should be privileged, if not because of the mistaken idea that intuition (1) and (2) are contradictory, or cannot be both coherently taken into account in a single theory of disease, though, of course, this latter task is not easier than the one of explaining away either intuition (1) or (2).

Sec. 9.1 is divided into three further sections: in Sec. 9.1.1, before even giving our prima facie hybrid theory of disease, I thoroughly analyze two clear cases of disease;

in Sec. 9.1.2, I provide a novel prima facie definition of disease, while, in Sec. 9.1.3, I explain how our prima facie definition of disease coherently takes into account intuitons (1) and (2) about disease.

9.1.1 Two Examples: Phenylketonuria (PKU) and (Lung) Cancer

Rather than to provide *ex abrupto* our prima facie hybrid definition of disease, I begin with a certain intuitive analysis of two cases of disease, which have been chosen because of their limpid, clear and (apparently, at least) simple character, and also because of the fact that our theory of disease must be *empirically realized* (i.e. by being absolutely realized) (cf. Box 9.1) – at least, under a plausible reading of these two cases of disease -, and those two very different examples are perfectly suitable to illustrate (also two different aspects of) our theory of disease (cf. Sec. 9.4.2); the choice of these examples is also justified by their *ubiquity* (and fascination) both in the scientific sphere and in everyday talk.

As a first example, phenylketonuria (PKU) is ususally defined as a specific metabolic disease touching the level of the *enzymatic* (or organic catalytic) action of the phenylalanine hydroxylase (PAH) in a body, whose function (judged as a processual part of a certain living being) is to convert the amino acid phenylalanine into the amino acid tyrosine (i.e. a metabolic pathway) (cf. e.g. the ICD-11 on all that).

If this (part of the) metabolic – or, more precisely, catabolic - pathway cannot happen, or if the function of the process of phenylalanine hydroxylation (PAH-tion) is *absent*, then there is a decrease of tyrosine in the body, as well as, as a consequence, an *over*-accumulation of phenylalanine, which is what is commonly called "(classical) PKU", and which leads to other metabolic errors (like tyrosinemia or alkaptonuria), as illustrated by the chemical pathways of Fig. 9.1:

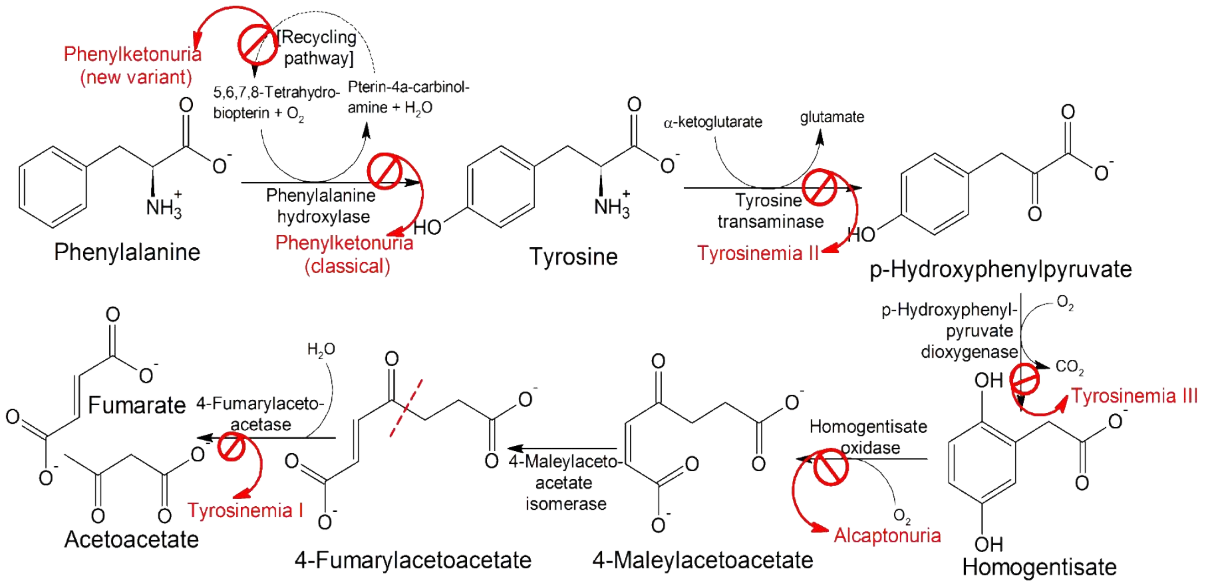

Fig. 9.1. – A scheme of a malfunctioning metabolic pathway because of PKU (as a first step). Reprinted from the Liberal Dictionary, 2019.

What is *fundamentally* responsible for the PAH-tion malfunction, or so it seems, is generally acknowledged to be a deleterious genetic mutation on the PAH *gene* situated on chromosome 12 (MedlinePlus, 2020).

If left untreated, PKU can lead to higher-level diseases like seizures or other mental damages. The treatment for PKU is generally said to be a low diet; this is due, of course, to the over-accumulation of phenylalanine in the body.

However, what about tyrosine diminution? Is not PKU some catabolic disease *preventing* the conversion into tyrosine (from phenylalanine, of course) – rather than to be some disease, which would be the over-accumulation of phenylalanine? Indeed, this over-accumulation seems, instead, just the direct *consequence* of PKU – not what PKU *is* (essentially).

Moreover, one also usually distinguishes between PKU and *hyper*phenylalaninemia i.e. the over-accumulation of the amino acid phenylalanine in the body (Ogier de Baulny et al., 2007).

PKU is a very good and simple example for starting our inquiry into the very concept of disease. Indeed, from our presentation of this example, first, *what* is diseased is clear: the enzymatic action of the PAH or PAH-tion;

second, what *the disease* in question is is also clear: PAH-tion, as a processual part of a certain living being, does not do what it is supposed to do i.e. being a certain catalyst (cf. Fig. 9.1);

third, in line here with our meta-philosophy and, more specifically, our requirement of intransitivity in definitions (cf. Sec. 2.1.3), PKU is essentially distinguished both from its *genetic* level (i.e. that PKU is *not* a genetic disease) and the other *higher-level* diseases it (necessarily) leads to, though there are still, of course, relationships of fundamental (and indirect) constitution and of necessitation between all those levels – though, as being a *kind/species* itself (cf. Box 3.1), defining PKU is not an easy task and is far beyond the reach of the present PhD dissertation. In other words, PKU is not (strictly) defined as a disease encompassing different levels.

As a second example, we can take the case of (*lung*) *cancer*. First, differentiating *what* is really and specifically diseased in the case of (lung) cancer is a much disputed issue; many authors choose to have e.g. a *holistic* perspective on cancer (in all its generality) having to do with the level of tissue formation or even the whole organism (cf. Exc. 3.10; also Bertolaso, 2016; Laplane, 2016; Laplane et al., 2018; Sonnenschein & Soto, 2000).

However, “cancer” generally means “abnormal cell growth (or division)” or “uncontrolled cellular proliferation” (cf. e.g. the ICD-11 on that). Thus, what is diseased in a (lung) cancer is, very precisely, *cell growth* or *division* (in the lung’s tissues), which is a phase of the cell cycle.

One could object that a definition of cancer as malfunctioning cell growth is oversimplistic or implausible. Of course, defining in a *correct* (within our framework i.e. within a genus-species hierarchy) way cancer is not an easy task (cf. Box 3.1); that the topic of cancer is getting more and more attention from philosophers of medicine proves it (cf. e.g. Germain, 2012; Plutynski, 2018; 2019).

For our present purpose, it is enough to relate the *general* notion of cancer with cognates like “dysplasia” and “benign tumor (or neoplasm)” (cf. Karindas, 2017; Sporn, 1991 for

terminological issues around cancer). To be more precise, we could plausibly (if not correctly) define (the generation of) cancer as, roughly, the complex process of *oncogenesis* (or carcinogenesis) i.e. (at least) (i) the start of a malfunctioning cell growth (or a specific *dysplasia* viz. hyperplasia) (as a specific process) – where the start of cell growth, deemed as a certain processual part of a living being, is cell division or reproduction) (cf. Hanahan & Weinberg, 2000; for a defense of the so-called somatic mutation theory of cancer, Vaux, 2011) -, occurring (temporally) *after* proto-oncogenetic processes and tumor suppressor genetic processes have mutated (where the deleterious genetic mutation in question is (a constituent of) an ultimate absolute realizer, or an ultimate constituent, of "cancer kind") (cf. Sec. 3.1.2), and (ii) the generation of a *benign* tumor (or *carcinoma in situ* for specific cancers, or neoplasia) i.e. the mass of the non-controlled grouped cells (very often) *metastasizing*, afterwards (i.e. as a *malignant* tumor), through (or, invading) other bodily sites (as the *effect* of cancer) (cf. Sec. 3.1.2; Fig. 9.2).

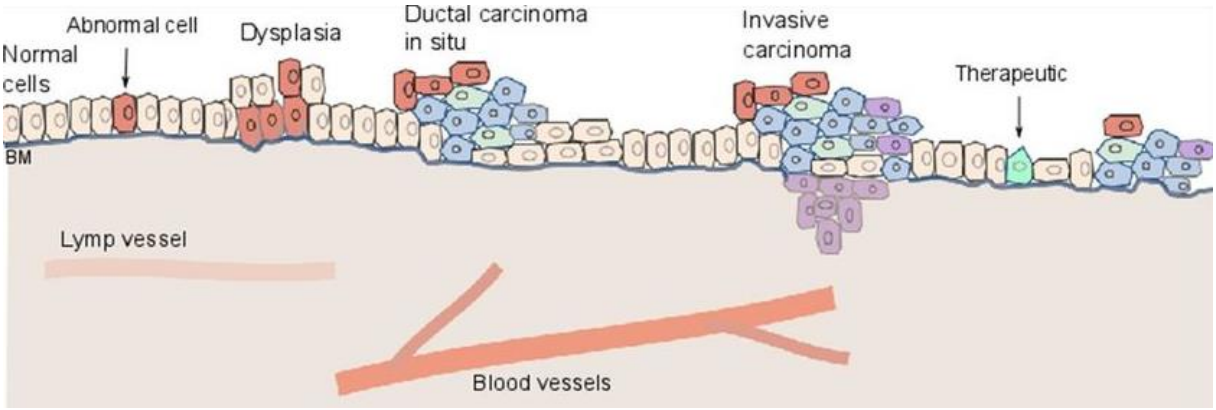

Fig. 9.2. - A rough scheme of cancer (in all its generality) from dysplasia to a malignant tumor. Reprinted from Kumar et al., 2018.

Thus, with points (i) and (ii), we can maintain our usage of the term "cancer" to refer to malfunctioning cell *growth* or division.

Second, following this highly fine-grained differentiation of what is diseased, in the case of (lung) cancer, what *the disease* in question is is, then, pretty clear: with (lung) cancer, cell growth or division (of the lung's tissues), as a processual part of a living being, does not function as it is supposed to do i.e. as a phase of a normal cell cycle (cf. Fig. 9.2), which divides or

reproduces (in a regulatory way) into two daughter cells (cf. point (i)), and groups them (cf. point (ii)).

Third, however complex the case of (lung) cancer is, from a very simple and intuitive characterization of (lung) cancer as abnormal cell growth, one can see that a (correct) definition of cancer does not cross multiple levels (until especially the genetic one), or is not holistic in the sense that, like for PKU, cancer necessarily (but not essentially) leads to higher-level diseases like malformed tissues.

9.1.2 A Prima Facie Definition of Disease

While Sec. 9.1.1 presents in details two clear (and uncontroversial) examples of disease viz. PKU and (lung) cancer, Sec. 9.1.2 is devoted to providing a prima facie hybrid definition of disease on the basis of our analysis of PKU and (lung) cancer.

As already stated, PKU is the disease, according to which the enzymatic process of the PAH does not *function* as it should i.e. by converting the amino acid phenylalanine into the amino acid tyrosine; being this catalyst is intuitively, not merely what PAH-tion just does, it is what PAH-tion (as a process) *is essentially*.

Following this line of thought, PKU is the disease *preventing* this *healthy process* to occur. In other words, PKU is the disease *destroying* PAH-tion (or making PAH-tion deficient, lacking or absent) or, more precisely, destroying what PAH-tion is or the *essence* of PAH-tion i.e. what PAH-tion (as a part of a living being) does.

To be more precise, PKU is a *specific way* of destroying PAH-tion i.e. by making tyrosine *decrease* (or subtracted) (rather than making phenylalanine increase, actually).

The same scheme of analysis can be applied to the case of (lung) cancer. (Lung) Cancer is also a destruction of, or *deprivation* from, this healthy process that is cell growth or, more precisely, what cell growth or division (of the lung's tissues) is or the essence of cell growth (of the lung's tissues) i.e. what cell growth (as a processual part of a living being) does: to divide into two daughter cells, and to group them.

Again, to be more precise, (lung) cancer is a certain way of destroying the essence of cell growth (of the lung's tissues) by making it *hyper*function i.e. through an uncontrolled cell proliferation or division (cf. Sec. 9.4.2).

From this scheme of analysis applied to two clear cases of disease, we can easily generalize to get a prima facie hybrid definition (or theory) of disease: a disease would be a process of

destruction (*loss* or *negation*) of a healthy process, which seems to be the essence of a *part* of a living being (e.g. what the enzymatic action of the PAH or cell growth is, etc.) (cf. Box 9.1). Put in a slogan, to be *diseased* is for a processual part of a living being to be *deceasing* (or to cease to exist).

Prima Facie Definition of Disease
x is a disease, iff *x* is the destructive process of the essence of a processual part of a living being.

Box 9.1. – Prima facie definition of disease.

9.1.3 Disease as a Specific Lethal Value and Biological Malfunction

Can our prima facie definition of disease (cf. Box 9.1) be (already) said *hybrid* i.e. to belong to the group hybridism about disease (cf. Sec. 8.2.3)? If yes, how does it *coherently* take into account both intuitions (1) and (2) about disease (cf. Box 8.1-8.2)?

That our prima facie definition of disease takes into consideration in a certain way intuition (2) about disease is rather clear. Indeed, e.g. PKU and (lung) cancer are typically two destructive processes of the (normal) *biological function* of, respectively, PAH-tion and cell growth or division (of the lung's tissues) (both judged also as parts of a living being). In that sense, a disease is, generally speaking, a biological malfunction, to the extent that it is, plausibly, the mere destruction of a (normal) biological function.

However, independently of any specific theory of biological functions, it seems also true that PKU and (lung) cancer can be said to have their own (normal) biological functions too viz., respectively, to prevent the conversion from phenylalanine to tyrosine, and to have an uncontrolled cellular proliferation (in the lung's tissues) (cf. Sec. 9.1.2), in the minimal sense that this is just what PKU and (lung) cancer, as (biological) processes, (normally or correctly) *do* (cf. also Sec. 9.4.1 about that).

Thus, merely talking about disease as a certain biological malfunction, in the plausible sense of being the mere destruction of a (normal) biological function is not sufficient for analyzing what disease is.

Nevertheless, that much is not what our prima facie definition *fully* states (cf. Box 9.1): as a specific biological malfunction, disease is the destruction of the biological function (or essence) of a processual part of a *living* being i.e. a *healthy* process – not whatever process.

The *value-laden* character of this prima facie definition, as a resort or constraint for completing in a certain way the definition in question – rather than to address the above point in a non-value-laden way, and to somehow explain away intuition (1), which would put our definition in the group malfunctionalism about disease (cf. Sec. 8.2.2) - suggests that it also tries to take into account seriously intuition (1).

How does it do so? Following intuition (1) that disease is a lethal value (cf. Box 8.1), we can argue that our prima facie definition of disease (cf. Box 9.1) takes intuition (1) into account, in so far as a lethal value can be also taken – not as the destructive process of a (normal) biological function -, but just as the destruction of this *vital value* that is health or, more precisely here, a healthy process (like e.g. what PAH-tion or cell growth is).

In other words, to the contrary of Wakefield (1992)'s hybrid definition trying to account for intuitions (1) and (2) in a *conjunctive* – and, thus, independent - way (cf. Sec. 8.2.3) (where an internal coherence is hardly present) our own prima facie hybrid definition of disease unifies those two intuitions, as two sides of the same coin (or two synonyms), under the single expression "destruction of a processual part of a living being"; one cannot talk about disease-as-biological-malfunction without talking at the same time about disease-as-lethal-value – and vice versa (cf. Fig. 9.4); along our analysis, intuitions (1) and (2) are *inseparable*.

9.2 A Correct Hybrid Definition of Disease

While Sec. 9.1 is devoted to providing a prima facie hybrid definition of disease, Sec. 9.2 is about a *correct* definition of disease.

Exactly like we already did with the cases of symptoms and etiopathology (cf. Box 5.1; 6.1), we use our prima facie definition of disease as a template for providing a correct definition of disease.

What can be retained from the above two uncontronversial cases of disease (cf. Sec. 9.1.1) is that disease is, generally, a specific *process*.

To provide a definition of disease situated within a genus-species hierarchy (cf. Box 3.1), it shall be helpful to develop, first, in Sec. 9.2.1, a certain (general) theory of processes, according to which, roughly, a process is substantial change of states.
Second, in Sec. 9.2.2, on the basis of our theory of processes, we give a correct definition or theory of disease – which can be usefully called "*essentialism about disease*" -, according to which disease is a negative process of a part of a living being (i.e. the genus), and is the destruction of a positive state *p* until the negative state ¬*p* (i.e. the differentia) (cf. Box 9.3).

9.2.1 Disease as a Process

As a starting point toward a correct definition of disease, our prima facie hybrid definition of disease relies on the general idea that disease is a certain (negative) *process* (cf. Box 9.1).
For the present purpose, since this is not a work on the concept of process, we can be satisfied, in Sec. 9.2.1, with only a *plausible* or prima facie definition of a process, albeit an ontology of kinds (as here) includes *states* as well as *processes*, as long as both are correctly defined i.e. constitutively, of course (cf. Box 3.1)[167]; moreover, relying on a plausible conception of processes is sufficient for implementing a correct definition of disease.
Sec. 3.2.6 has already touched upon this huge and complex topic that is a process by defending the idea that a process *is* basically (*substantial*) change *of states* (minimally understood as *instants*); more precisely, a (positive or negative) process is minimally analyzed as the *generation*/*destruction* of (the essence of) a (positive or negative or value-free) *state* (¬)*x* at time t_{+1} from its contradictory state (¬)*x* at time t_0; more simply put, a process is the generation of a state (¬)*x* present at time t_{+1}, which is generated from the contradictory state (¬)*x* at time t_0 (cf. Box 9.2): e.g. PAH-tion is a process, for it is the change from the *state* where tyrosine is *not* present to the *state* where tyrosine is present; PAH-tion is the generation of tyrosine, while this is the opposite for PKU by making tyrosine absent; cell growth is a process, because cell growth is the change from the state of non-two daughter cells divided and grouped to the state

[167] Caveat on essence and state ontology: although the notion of *essence* is often related to a *state* ontology (cf. e.g. Boniolo & Carrara, 2004; Guay & Pradeu, 2015), it does not have to be so (cf. also Sec. 3.2.5): albeit processes are to be correctly understood as (essentially) substantial change of *states* i.e. that processes "[…] are not things that change but are the changings themselves" (Charles, 2017, p. 3), processes may still very well be (specific) temporally extended *kinds*/species.
The subscription to an ontology of processes (or, things which are extended through time does *not* imply, as such, indeed, that talk about essences should be jettisoned, or that one should subscribe to (semantic) *vagueness* or (metaphysical) *indeterminacy* (cf. especially DiFrisco, 2018).

of two daughter cells divided and grouped; cell growth is the generation of two daughter cells grouped, while cancer is the opposite by making non-two daughter cells divided and grouped (but rather a multiplication of them)[168].

Prima Facie Definition of a Process
x is a process, iff (i) *x* is the generation of a state (¬)*y* at time t_{+1} from the contradictory state (¬)*y* at time t_0.

Box 9.2. – Prima facie definition of a process.

Further remarks on this prima facie definition of a process are in order: we have to be careful, first, to distinguish between two dimensions in a *process*: (i) a process is basically *substantial change* for a (positive or negative or value-free) *state* i.e. the *generation* (or birth/life for an organism)/*destruction* (or extinction/death/the killing for an organism) of a (positive or negative or value-free) state (¬)*x* at time t_{+1} from its *contradictory* state (¬)*x* at time t_0, where states *minimally* exist at these precise times (cf. Exc. 2.7); it does so, of course, by generating/destroying the *essence* of state (¬)*x* i.e. that it *gathers together* (puts together, assembles, or generates)/destroys (more and more of) the intensional parts of state (¬)*x*; if a process must be said somehow the *passage* (or transition) – however misleading this term can be, for it looks like a mere synonym of "process" - from a state to another one, then it is from a state to another *contradictory* one[169].

Thus, instead of conceiving a process as the passage from e.g. redness to blueness, it should be more properly conceived of as the putting together of e.g. all the pieces of a puzzle from the beginning until the end.

[168] Terminological note on our definition of processes: (i) our definition of a process (cf. Box 9.2) does apply, indeed, to specific processes like *positive* processes (e.g. a healthy process), *negative* processes (e.g. a disease process), or *value-free* processes (e.g. building something).
In the case of a value-free process, the state ¬*y* does not refer, of course, to a negative existing state, but only to the *absence of* a certain value-free state *y*.
However, for sake of textual readability (though somewhat sacrificing accuracy), we maintain the same formulation (with the negation) also for *negative* entities themselves (but, cf. Sec. 9.3.1 for our understanding of negations).

[169] Caveat on processes and contradictions: Parmenides may ask: "isn't *contradictory*, indeed, to claim that a process is substantial change from a state to a contradictory one?" A philosopher from a Heraclitean inspiration shall be happy with this fact, but a way out here to avoid this consequence is to claim that two propositions (as types here) are contradictory, only if they also happen at the same time or during the same time interval (cf. Horn, 1989 for the same point).
If this is true, then, indeed, because there is, with a process, a difference *in time* between a certain state and its contradictory state, then there is no contradiction here, strictly speaking.

Furthermore, (ii) a process also *implies at the same time* the *generation*/*destruction* of a (*sub*)*contrary* state of (the contradictory state) (¬)*x* (= (¬)*y*) until its own contradictory state (¬)*y* by *generating*/*destroying* (more and more) the essence of state (¬)*y*; if a process *implies* the generation/destruction of a (*sub*)*contrary state* of (the contradictory state) (¬)*x* (i.e. another process itself), then these two *processes* can also be deemed (sub)contraries themselves: e.g. a diseased body (*Leib*) vs. the generation of a corpse (*Körper*); the destruction of a caterpillar vs. the generation of a butterfly[170].

By dimensions (i) and (ii), we distinguish, thus, between what a process is and what a process *implies*, respectively.

The confusion arising between dimension (i) i.e. what a process is and dimension (ii) i.e. what a process implies is very widespread in the biomedical sciences: e.g. one usually confuses between what a viral process *is* (e.g. as a certain *negative* entity depriving its host from e.g. some of its cellular constituents by invading/infecting it) and what is *implied* by (what) a viral process (is) i.e. e.g. a certain *positive* entity with its own genetic processes and mode of reproduction viz. the process of virion assembly (on the controversial status of viruses as organisms, cf. Dupré & O'Malley, 2009) - though a *functionalist* interpretation of a viral process as an etiopathological agent is also plausible, of course (but, cf. Sec. 4.2.7 for criticisms).

Second, to fix some potential terminological issues, some philosophers use the notion of (*mere*) *Cambridge change* (cf. Mortensen, 2020; Strobach, 1998; Ujvari, 2004) for what I am calling (following the neo-Aristotelian tradition) "substantial change of a state", but others more widely use this technical notion to refer to what is in stark contrast to *real change* and associated to a certain theory of (substantial and *accidental* or extrinsic) change viz. the cinematographical (or "at-at") theory of change (cf. Cleland, 1990; Geach, 1969; Lowe, 2006; Russell, 1903 for the "at-at" theory); other philosophers also use the term "succession" (Strobach, 1998) to refer to dimensions (i) *and* (ii) i.e. the *passage* (or transition) from one *state* to another – contrary, but not *sub*contrary - state (e.g. from redness to blueness), as long as the notion of succession is not taken as meaning that dimension (ii) comes temporally after dimension (i).

As long as these quite vague labels are made clear or explicit, using them (by contrast to, or interchangeably with, our own terminology) is, of course, fine, even though, due to their high vagueness, it is more preferable or advisable not to use them.

[170] Terminological note on (sub)contrariety: (sub)contrariety is to be minimally understood as *incompatibility* between two (intensional or not) predicates, however plausibly or correctly understood, with a *same predication* restricted to a specific (as fine-grained as possible) *domain of application*: e.g. "_is a caterpillar" vs. "_is a butterfly" as two contrary intensional predicates of "a living being" (i.e. the domain of application).

Third, the gathering together/destruction of (more and more of) (¬)*x*'s intensional parts (as specific states) is not merely the generation/destruction of state (¬)*x* (cf. dimension (i)), but also the generation/destruction of this very process – with the difference, however, that the (gathered together/destroyed) intensional parts of *state* (¬)*x* shall be themselves intuitively (on pain of a category mistake) specific (lower-level) *states* (e.g. (¬)*a*, (¬)*b* and (¬)*c*), while the (gathered together/destroyed) intensional parts of a specific process shall be themselves intuitively specific (lower-level) *processes*: e.g. the *gathering together*/destruction of states (¬)*a* and (¬)*b*; the *gathering together*/destruction of states (¬)*b* and (¬)*c*.

Fourth, although they go hand-in-hand, one should not confuse what it is to be a process (i.e. the generation/destruction of a *state*) with what it is for a process to be generated/destroyed or what generation/destruction (of a state or a process) *simpliciter* is; indeed, it is of a primordial importance not to confuse processes with *mere* substantial change.

One of the biggest mistakes to avoid with our definition of a process (cf. Box 9.2) is to reduce *change* (or generation/destruction *simpliciter*) to generation/destruction of *contrary* relata: e.g. from red to blue. This mistake comes from the intuition that substantial change *tout court* is not really a change, for there is the high temptation of *not* considering *negations* of a positive state/process as specific genuine kinds (or entities) themselves (cf. Sec. 9.3.1 about that point).

Finally, one may doubt that disease is, actually, only a certain negative process. Indeed, it is not rare, after all, to talk about disease is *static* terms, typically when one commonly says that *x* is in a diseased *condition* or state: e.g. experiencing a flu state; ditto for the case of health, of course.

However, independently of the fact that disease is intuitively a specific biological malfunction (cf. Box 8.2) – which is something closely related to dynamicity or processes -, it is not difficult to interpret the above case as a mere attempt at capturing the *end* of the disease process, which tends towards, indeed, a certain negative state, along our conception of processes, or just a part of this process through an *intermediate* state[171, 172].

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[171] Caveat with a terminological note on processes and events: in the literature (cf. e.g. Charles, 2017; Stout, 2016; also Mourelatos, 1978), we often distinguish between the ontological category of (*on-going*, or perhaps even *ever going*) *processes* (e.g. walk*ing*) and the ontological category of (*completed*) *events* (e.g. *a* walk).
According to me, the (supposed) *metaphysical*-cum-semantic rule of distinguishing between an *imperfective* and a *perfective* verb aspect does not correspond, actually, to a *metaphysical* distinction, but merely to a(n) (*epistemic*) way of grasping one and the same *process* (i.e. a specific *temporally extended* kind understood as substantial change of *states*) by the means of a (constitutive) temporal *part* of it: e.g. at the very *beginning*/*end* of a process itself (e.g. *a* walk, *a* race, *a* jump, *a*(*n*) earn/win, being disease*d*, etc.), or in the *course* of it (e.g. walking, racing, jumping, earning/winning, being in the course of a disease, etc.).
Thence, I do not distinguish between "*process*" and "*event*" (or even "activity" (*pace* e.g. Seibt, 2008) or "mechanism"), where these latter terms can be used interchangeably with "process".

[172] Two terminological notes on processes and states: (i) as said above, our ontology of kinds/species includes processes as well as *states*; especially in the biological and biomedical sciences, many terms seem to ambiguously

Another objection against the idea of disease as merely a process may be put as follows: we are used to say that it is *unhealthy that p* (e.g. to smoke, etc.), while smoking is commonly interpreted in those circumstances as a (negative) *fact* or state of affairs (cf. Mulligan, 1998). That much amounts to our taking diseases as processes, actually; indeed, as being abstract entities, those processes can very well be phrased as facts or states of affairs themselves: e.g. smoking, etc.[173].

9.2.2 Essentialism about Disease

How can our rough theory of processes (cf. Box 9.2) help us give a *strict* definition of disease? According to our prima facie hybrid theory of disease (cf. Box 9.1), disease is a certain negative process viz. the destruction of the essence of a processual part of a living being.
It takes time for something to become e.g. a (healthy) PAH-tion or cell growth. PAH-tion or cell growth is a certain *positive* process (as a part of a living being), which is the generation of the (resulting) state *p* (e.g. tyrosine being present, or two daughter cells divided and grouped) from the state ¬*p*, where the intermediary *states x*, *y*, *z*, etc., and the ending state *a* are all the constitutive parts of the (static) entity *p*.

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refer to either processes or states or both (for a focus already on that issue, cf. e.g. Machamer et al., 2000): e.g. does the very term "gene" or "virus" (or etc.) refer to a certain state or process or both? The answer shall obviously depend on the meaning of those words. Intuitively, one would like to say that "gene" as meaning, roughly (and partly), "sequence of nucleotides" refers, thus, to a certain state; ditto for other cases like "virus", which intuitively refers to a certain state, or even "heart", which refers to a certain organ (thus, to a certain state).
Throughout the entire PhD dissertation, I distinguish between, strictly speaking, e.g. genes and genetic processes, or viruses and viral processes, or even heart and heart processes, or PAH and PAH-tion, etc; this distinction is made explicit as above, or in cases where e.g. "genes" or "viruses" are referred to as (resulting or starting) *states* (most often for the sake of a plausible illustration and to follow a mainstream scientific terminology).
(ii) Even though it is clear that the relationship between e.g. a gene and a genetic process has to do with the fact that a gene understood as a state is obviously the *resulting* state of a genetic process (cf. note (i)), or a virus as a state is the *starting* state of a viral process, one should not, however, take a gene or a virus as being essentially a *causal* state (on functionalism, cf. Sec. 4.2.7).
I warmly acknowledge Ulrich Krohs and Niko Strobach (personal communication, April-June 2021) for having pressed me for clarification on that issue.

[173] Caveat on negative facts: sentences like "it is unhealthy to smoke" are arguably incomplete or are shortcuts for more complex sentences like "it is unhealthy for *x* to smoke" or "it is unhealthy that *x* smokes" i.e. that there is something that is for *x* an unhealthy process, and this thing is smoking.
Contra Mulligan (1998) here, those former sentences are *not* to be put in parallel with sentences like "*x* (e.g. a heart process) is unhealthy/diseased, where what we talk about is the disease host – and not the process itself; if doing so, one shall be misled by thinking that e.g. smoking can be a disease *host* (like for e.g. a heart process) (cf. Sec. 9.3), while, actually, smoking is an unhealthy process itself.
A short argument in favor of this is to claim that sentences like "smoking is unhealthy" and "the heart process is unhealthy/diseased" are two *incomplete* sentences, which are ambiguous, and do not focus on one and the same thing: the former insists upon the disease process itself viz. smoking, while the latter upon the disease host viz. the heart process.

The more we advance in the states *x*, *y*, *z*, etc., the more there is intuitively a (*healthy*) PAH-tion or cell growth (cf. Sec. 9.3.3): there is, first, a little bit of PAH-tion or cell growth (*x*), a bit more (*x* & *y*), even more (*x* & *y* & *z*), etc.; PAH-tion or cell growth is the process – not the resulting state *p* we get at the end once all its parts have been gathered together. During this process, states are generated, whose essence is to be *x*, or to be *x* & *y*, or, etc., to be *x* & *y* & *z* & *a* (which is the essence of *p*).

PKU or (lung) cancer, as a negative process, is itself a certain existing process preventing this generation process to correctly happen – which is the essence of PAH-tion or cell growth (in the lung's tissues); thus, first of all, PKU or (lung) cancer is a certain negative process of a part of a living being – the disease host being, in this case, PAH-tion or cell growth (in the lung's tissues) (cf. Sec. 9.3 on the disease host); being a negative process of a - strictly, *processual* - part of a living being is intuitively the *genus* (proximum) under which a disease falls (if correctly interpreted within our understanding of the genus-species hierarchy) (cf. Box 3.1-3.1.1). Indeed, exactly like a sign *of* a disease (or symptom) has a disease for the genus (cf. Box 5.2), a disease of *x* has *x* for the genus.

However, a disease cannot be merely a negative process of a part of a living being, it has to be something *more* (precise) than that – otherwise, "disease" is a far too weak notion (against *being diseased* as an accidental property, cf. Sec. 9.3.1); if PAH-tion or cell growth (in the lung's tissues) is *essentially* the generation of a certain positive state *p* from the negative state ¬*p* (cf. Box 9.1 for our prima facie definition of a process), then PKU or (lung) cancer shall also have to be, at the opposite, the destruction of this positive state *p* until the negative state ¬*p*.

Based on this illustration, we get the following strict definition of a disease or theory of disease, which we can label "essentialism about disease": a disease is essentially a negative process of a part of a living being (i.e. the genus), and is the destruction of a positive state *p* until the negative state ¬*p* (i.e. the differentia) (cf. Box 9.3)[174].

[174] Caveat on essentialism about disease: the following charitable interpretation of our definition of disease (cf. Box 9.3) has been suggested to me: *x* is a disease, iff *x* is a process (i.e. the genus), and there is some *x'*, such that *x'* is a living being; there is some *y'*, such that *y'* is a processual part of *x'*; there is some *z'*, such that *z'* is the essence of *y'*; there is some *p* such that *p* is a positive state and *p* results from *z'*, and *x* is destructive of *z'* by leading from *p* to ¬*p*.
However, first, it is preferable to be already as much specific as possible in the genus (*proximum*) given in our definition of disease by talking about a negative process of a part of a living being; this also has as a consequence a brief differentia.
Second, our own differentia has the advantage of being defined independently from the genus in question.
Third, though called "essentialism about disease", our definition of disease is still compatible with a *plausible* non-essentialist reading of it.
Nevertheless, beyond these differences, essentialism about disease is very close to the charitable interpretation of it offered by Niko Strobach (personal communication, April 2021), whom I deeply acknowledge.

Definition of Disease (or Essentialism about Disease)
x is a disease, iff (i) *x* is a negative process of a part of a living being (i.e. the genus), and *x* (ii) is the destruction of a positive state *p* until the negative state ¬*p* (i.e. the differentia).

Box 9.3. – Definition of disease within (our understanding of) a genus-species hierarchy, or essentialism about disease.

As for our other correct definitions of a disease kind, a symptom and an etiopathological agent (cf. Box 4.2; 5.2; 6.2), and since a (positive or negative or value-free) process is essentially the generation/destruction of a positive or negative or value-free state from its contradictory state (cf. Box 9.2), it is obvious that, when we talk about the destruction of a *positive* state *p* until the *negative* state ¬*p* (cf. condition (ii) of Box 9.3)), we are talking, strictly speaking, about the destruction of the *essence* of a certain positive process, where this positive process is precisely the one of the disease host mentioned in condition (i); however, again, as for the other correct definitions, a differentia defined independently from its genus has to be provided.

Furthermore, Box 9.3 is clearly a refinement of our prima facie definition of disease (cf. Box 9.1), to the extent that it details in which sense disease is a destructive process i.e. as substantial change from a positive state *p* to the negative state ¬*p*.

We can argue that what resorts the most importantly both from our prima facie and correct definition of disease (still made as much compatible as possible with diverse plausible interpretations) (cf. Box 9.1; 9.3) are certainly expressions like "*essence of a processual part of a living being*" and "*destructive process*" – which seem to be, thus, the two most important key points at the basis of what disease is.

Sec. 9.3 and 9.4 are devoted to further analyzing those expressions, instead of detailing in a separate way conditions (i) and (ii) of our definition of disease, by focusing on the notions of the essence of a processual part of a living being and of a destructive process, and to shedding some light, actually, on how to understand into details conditions (i) and (ii) of our original definition of disease i.e how to understand in a coherent (or unified) way, actually, intuitions (1) and (2) at the basis of disease (cf. Box 8.1-8.2; also Sec. 9.1.3).

9.3 The Disease Host

What is diseased? In other words, what is the disease *host*? Both our prima facie definition of disease and the correct one (cf. Box 9.1-9.2) talk about (the essence of) a processual part of a living being. Why does it have to be so? In a nutshell, Sec. 9.3 is devoted to answering this question by unpacking the complex expression "(essence of a) processual part of a living being".

Sec. 9.3 is divided into four further sections: in Sec. 9.3.1, by understanding in a certain way negative entities, I argue that that the relationship between the disease host and a disease process is the one between a certain positive kind viz. a healthy process and the *negation* of it.

In Sec. 9.3.2, I argue that the disease host is to be taken as a (processual) *part* of a living being – and not e.g. as a living being itself.

In Sec. 9.3.3, I provide a prima facie definition of health, according to which health is the essence of a processual part of a living being, and I argue for the idea that health is not the absence of disease.

In Sec. 9.3.4, I extrapolate from Sec. 9.3.3 a prima facie definition of a lethal value, in all its generality.

9.3.1 Disease as a Negative Entity

To unpack the complex expression "(essence of a) processual part of a living being", it is important to spend some time on the relationship between a disease process and what is diseased i.e. the disease host; to do this, we need to dig deeper into the nature of negative entities.

From intuition (1), we know that disease is a specific *negative* value i.e. that disease is, indeed, a specific negative entity, while, from intuition (2), we know that disease is a certain biological *mal*function (cf. Box 8.1-8.2).

There are, generally, two ways of taking negative entities: (i) when we talk about e.g. PAH-tion or cell growth being diseased as a disease *of* PAH-tion or cell growth, we would like to talk about PAH-tion or cell growth as (*plausibly*) a *positive bearer* of an *accidental* property viz. *being diseased*: a diseased PAH-tion or cell growth would be, thus, *itself* also a specific PAH-tion or cell growth.

Indeed, if e.g. PKU or cancer (as a specific disease) is nothing else than, actually, a diseased PAH-tion or cell growth, then it sounds like talk about a specific disease like PKU or cancer is talk about a specific PAH-tion or cell growth *itself* viz. a diseased (or bad or negative) PAH-tion or cell growth.

That much is part of a more general problem: are *bad* or *negative* (e.g. mental, or, more specifically, affective) states/processes specific (mental or affective) states/processes *themselves* (Hommen, 2013; Paolini Paoletti, 2017), or are they *negations of* (positive) (mental, or, more specifically, affective) states/processes (Zangwill, 2011)?

However, (ii) we can also claim that a specific disease like PKU or cancer is not to be analyzed as a specific PAH-tion or a specific cell growth.

Indeed, if we hold the claim that, when e.g. PAH-tion or cell growth is *not* healthy, then it is diseased, then *being diseased* cannot *be* an accidental property of PAH-tion or cell growth, or PAH-tion or cell growth cannot be, plausibly speaking, a *positive bearer* of the property of being diseased; thus, a diseased PAH-tion or cell growth cannot be a specific PAH-tion or cell growth itself.

At a more general level, a *negative* value (or disvalue) would be, thus, the negation of a (*mere*) value (which would be necessarily positive).

In other words, if disease is a *negation of a specific positive property* (alongside holes, absences, privations, etc.), then, on pain of a category mistake, the negation of a specific positive property cannot be borne by a *positive bearer* like PAH-tion or cell growth.

Ditto for the reverse relationship between a *positive* property and a *negation* of a positive bearer: e.g. a healthy psychopath; if psychopathy is a disease i.e. here a negation, then a psychopath cannot *be* healthy, or, exactly like a diseased PAH-tion or cell growth is not a specific PAH-tion or cell growth, a healthy psychopath is not a specific psychopath, although, of course, a *negative bearer* can still possess/bear *negative* properties (e.g. a psychopath can *be*, among others, *without* empathy or *without* a capacity to formulate correct moral judgments) (on the difficulty in defining psychopath(olog)y, cf. Kincaid & Sullivan, 2014; Lepine, 2015; Zachar, 2014).

At a more general level, way (ii) is associated with the idea that talk about negative entities is talk about *non-existent* things (Hommen, 2018; Moore, 2009).

To sum up, ways (i) and (ii) disagree over whether a specific disease like PKU or cancer is, respectively, either a *true* specific PAH-tion or cell growth itself viz. a negative one, or the *negation* of a (*mere*) entity like PAH-tion or cell growth (which would be necessarily something positive).

More generally, are negations of *x* (or *negated* entities) specific kinds (or entities) themselves, or, as more usually put, is a *denial* of a positive predicate (e.g. “Socrates is not wise”), actually,

an *affirmation* itself of a negation (e.g. "Socrates is non-wise" or "It is true/correct that it is false/incorrect that Socrates is wise")?[175] What *are* negations?

Strictly speaking, a negation *of x* cannot be, of course, *x*, but it still can be, or *exist* as, (indirectly) a specific *y* itself viz. a *negative* one, where "*y*" very likely stands for more *fundamental* or general (*axiologically neutral*) items (than *x*) like "process/state", "cause/effect" or "species/kind" – in that sense, *negations* can be judged as being still *inside* our metaphilosophical framework (cf. Ch. 2-3).

Indeed, it is intuitive that e.g. a negative cause is *not* the negation of a (*mere*) *cause* (which would be necessarily positive) i.e. not a cause, but *is* still itself, actually, a specific cause viz. a negative one – which can still be consistently treated, however, as the *negation* of a *positive* cause (taken as a specific cause itself, and not as a *mere* cause) (Barros, 2013; Mebius, 2014; Schaffer, 2004; *pace* Stepanians, 2017; Varzi, 2007): e.g. the *lack of* blood flow to tissues (i.e. ischemia) causes a lack of oxygen at the level of tissues (i.e. (local) hypoxia), where the blood flow to tissues is a positive cause of the supply of oxygen to tissues (on negative causality, generally speaking, cf. e.g. Birnbacher & Hommen, 2012; Hommen, 2014; McGrath, 2005; on the (more) general idea of negative causes as (indirectly) norm violations, Beebee, 2004; Clarke, 2014 for the same point about omissions).

In other words, a middle path between ways (i) and (ii) can be found: PKU or cancer can be treated as a negative entity i.e. as a specific *kind* itself – but not as a *specific* PAH-tion or cell growth (against way (i)) -, for PKU or cancer is truly the negation of PAH-tion or cell growth taken as a *specific positive* entity itself – but not as a *mere* entity, which would be necessarily positive (against way (ii)).

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[175] Three caveats (with terminological notes) on contradictions: (i) the *affirmation* of a negative property (e.g. *being non-red*) does not amount to making it no longer the *negation of* a certain (positive) property (e.g. *being red*) (i.e. a contradictory property) by making it, to the contrary, a *contrary* property of being red (*pace* Massin, 2010; cf. also Horn, 1989).

Indeed, even if *being non-red* is taken as a negative property, it does not imply that, within a given (restricted) domain of application, there exist things that are neither red nor non-red (e.g. numbers are out the domain in question) i.e. that *being non-red*, taken as a negative property, is not a contrary property of being red – but really the contradictory one.

(ii) Talk about non-*x* (e.g. non-red) is often confused with directly talking about a *contrary* of *x* (e.g. blue, etc.) – not the contradictory of *x*: e.g. *non*-reductive physicalism is generally meant to include any *contrary* position to reductive physicalism. For sake of terminology or to situate oneself within a certain well-established debate (or also because of linguistic restrictions), this confusion can, however, be pragmatically maintained.

(iii) Is not the affirmation of a negation a *contradiction* (e.g. it is true that it is false that Socrates is wise) (cf. Beall et al., 2016)? A thorough answer to this difficult question is far beyond the reach of the present PhD dissertation. However, just like for the case of (sub)contrariety (cf. Sec. 9.2.1), by restricting the scope of the truth/falsity connective (or predicate) to a domain of application as fine-grained as possible, we can prima facie non-paradoxically claim that the truth/falsity connectives (or predicates), in this case, do not have the same domain of application: "it is true that_" applies here to a *complex* sentence (viz. "it is false that Socrates is wise), while "it is false that_" applies here to a *simple* sentence (viz. "Socrates is wise").

Diseases are, thus, specific *negations* (or (de)privations) of *specific positive* kinds (cf. Box 9.1-9.2): e.g. bad feelings or bad emotions are negations of *positive* feelings or emotions - not of emotions *tout court* (vs. way (ii)); more precisely, disease is this specific negative property, which is the negation of a *specific positive* property viz. the property of being *healthy* or of being the biological function of something *good* (cf. Box 9.3).

However, if the relationship between a disease and what is diseased cannot be seen, properly speaking (though not implausibly), as a relationship between a property and its (positive) *bearer*, then how to conceive of it?

In the literature on negative entities or, more specifically, on *holes* (Casati & Varzi, 2019), the term "host" is often used instead of "bearer", where, in our case, the notion of a *disease host* is to be interpreted as a specific *negated entity* (or a certain negation *of x*) viz. a disease *of x*[176].

However, we can object here that, generally speaking, negativity cannot be *mere* destruction/negation of positivity. For, otherwise, there would not be anything positive in the world any more. And, if positivity truly grounds somehow negativity – and not the other way round -, then positivity must in one way or another be always present.

If disease is the deprivation of a certain positive entity, then how can it be that there are still positive entities, after all[177]?

First of all, through the WCP, positive entities are necessarily existent; they stand in equilibrium with negative ones.

Second, it may also well happen that a positive kind exists without ever being destroyed; whilst a (positive) host (*tout court*) exists without that any negative entity is necessarily present to destroy *it* (or, a *specific* positive host could also not exist at all), a negative entity (like disease) implies (essentially) the presence of some positive host.

Third, we can argue, more precisely, that a negative *kind* like the kind disease just is the destruction of a positive *kind*[178] – without this meaning that it is the destruction of a positive

[176] Terminological note (with caveat) on (disease) hosts: the term "host" has a wider acceptance in the biological context, where it is used to describe any organism interacting with another one viz. a symbiont (or guest); this interaction can be many-fold: e.g. the host can play the role of a shelter or food reserve for the symbiont.
This latter – much more inclusive (than ours) – comprehension of what a host is is risky (and should be avoided), for this can easily lead to an under-appreciation of what are, indeed, very different specific ontological units.

[177] I acknowledge Ludger Jansen (personal communication, February 2019) for having pinpointed this to me.

[178] Caveat on the destruction of essences: what does it really mean for a negative kind to destroy a positive kind? Since the destruction of a whole intuitively implies the destruction of what this whole is i.e. its essence, the destruction of a whole (thus, with its essence) does imply, of course, that this whole with its essence does not exist any longer, but it does not imply the destruction of a whole understood as a *bare* entity i.e. a mere subject (of predication), a property bearer/host or a substratum i.e. an entity with no essence (cf. Sec. 3.2.6 on bare particularism): e.g. destroyed PAH-tion is like (but for an artefactual kind) the switching (a light), which does not work any longer; one would intuitively say that there still exists some*thing* like e.g., for the case of tokens, the very last remnants of a hardware switch as the basis for a proper switching, but there is, nevertheless, no switching (cf. Sec. 9.2.1 for the relationship between e.g. a switch and a switching).

type, and the *type* disease would be, thus, just the destruction of a positive *kind* instantiated – not the positive type in question, which still has, thus, instances. In this case, we just simply distinguish between the destruction of (the essence of) a kind and the destruction of (the essence of) a type; if a positive kind (and its essence) go on to exist, that is here because somehow it is still necessitated by the presence of a positive type (as an absolute realizer of it)[179].

9.3.2 The Disease Host as a Part of a Living Being

Why should the disease host be a (processual) *part* of a *living being*, rather than something else? First, thanks to our permissive neo-Aristotelian ontology (cf. Exc. 3.5), many fine-grainedly differentiated kinds – but, without that implying that *anything* can play the role of a disease host - can play the role of the disease host: e.g. PAH-tion, cell growth, or other higher-level kinds like positive emotions or feelings; we follow here contemporary medicine, which tends to, indeed, categorize more and more as diseases even the most teeny tiny (biological) – and very common – defects (cf. Exc. 3.6).

Secondly, we can argue that, if the disease host can only be, indeed, a *part* of a living being, that is, because what hosts a living being *tout court*, instead of a part of it, is, actually, *death* – not disease (as entries on the debates around death, cf. Luper, 2019; Bradley et al., 2013). What can be diseased is intuitively, of course, those (indirect) intensional parts of organisms, which are *not* themselves also specific living beings (e.g. cells), for those latter ones cannot be healthy or diseased, for they are alive or dead/dying[180].

I thank Ulrich Krohs and Niko Strobach (personal communication, April-June 2021) for having asked me for clarification on that issue.

[179] Caveat on ceasing to exist: how do positive tokens cease to be, finally? If positive tokens are more fundamental than negative ones, then, since a negative kind has necessarily a negative type (with its own negative tokens, thus), it just *contingently* happens that more and more positive tokens fall under the negative type – until it happens that the positive type is itself consequently destroyed.

[180] Three terminological notes (with caveats) on the notion of a living being and organism: (i) the notion of a *living being* is to be distinguished from other notions: e.g. the term "biology" can be used to refer either, etymologically speaking, (i) *widely* to the study of any living (and dying) being and its (supposed) parts (e.g. if being virtuous, rational or social is part of the human being taken as a living being, then the study of virtue, rationality or sociality is part, actually, of the biological sciences) – as Chris Megone (1998b, p. 223; emphasis in the text) nicely puts it, "[…] the Aristotelian view categorizes humans *biologically* as rational [or even social] animals" -, or (ii) *narrowly* to certain parts of a living (or dying) being viz. those that are (non-)material. "Biology" is used to refer, throughout the PhD dissertation, either under sense (i) or (ii), albeit we have to be aware that sense (ii) – the technical one - is far the most common one, of course.

(ii) The term "organism" has two different large (and vague) senses: it can be taken as (i) a mere synonym for "living (or dying) being" (or "biological entity"), or as (ii), etymologically speaking, what is taken to *make*, the most generally, a living (or dying) being a living (or dying) being or an organism (under sense (i)) i.e. organization (however understood here). The term "organism" is used implicitly or explicitly (when needed) under either sense (i) or (ii).

Indeed, one can argue that what distinguishes disease from death is the specific *host* in question: e.g. a human being, a cell, an ant colony, or maybe even a fetus - however difficult to be defined those living beings are - can be said *dying* or dead, while only *parts* of e.g. a human being, a cell, an ant colony (as a meta-organism or, for sake of simplicity, organism *tout court*), or a fetus can be said diseased or in the course of a disease (on death, cf. Exc. 9.1; on *meta*organisms as specific organisms themselves, O'Malley, 2013).

However, cannot the disease host be a part of something else than a living kind? We fail to see in what sense a disease host could be *literally* part of something else than a living being – a minimal definition of life can certainly take into account most of the intuitive judgments that we have about what counts as a disease host, while non-arbitrarily relegating the other uses as metaphorical (for a starter on a minimal definition of a living being, cf. Sec. 9.3.3) -, while other related terms can better capture to a greater degree of generality what we want to refer to (on e.g. *disorder*, cf. Sec. 7.3.1).

9.1 Excursus: On Death

As being very close to disease, it is important to devote some time in briefly explaining what death is and what its relationship with disease is.

Intuitively, death is not the *permanent* end of life; the possibility to recover from it (or to resurrect) is always theoretically possible (on cryo-preservation/-conservation, cf. Lauria, 2019; also Yourgrau, 2019).

Along our own essentialist theory of disease (cf. Box 9.3), *deprivationism about death* (Deng, 2015; cf. also Nagel, 1970) has been also interestingly defended, according to which, roughly, death (as a process) is bad for the one who is deceasing, for it *deprives* him of some goods the host possessed viz. life.

In other words, the death host is always the entire living being. A consequence of this idea is that values are not merely ascribed to whole living/artefactual beings, but also to their parts (as for health and disease).

However, first, since there is no consensus on what life or a living being is, most of the time, what is referred to by "death" or "causes of death" is *mistakenly* merely a highest-level *disease* (or its causes): e.g. there has been a historical shift in medicine from a view on human death as of a *cardio-vascular*

(iii) "Organism" cannot be taken, thus, as a mere synonym for "*living* being", for we do intuitively count also as organisms what is not, strictly speaking, a *living* thing: e.g. a viral processis intuitively an organism, although it is not a *living* being, strictly speaking – while the term "organism" is sometimes used, in the literature, to refer only to a living being.

This latter use of "organism" is, of course, incorrect, strictly speaking, but still plausible (and can be used so) with a very rough acceptation of what a *living* being is.

death (i.e., actually, the cardio-vascular system as (directly) constitutive of a human being) to the idea of *cerebral* death (i.e. the brain as (directly) constitutive of a human being) (Machado, 2010); or in talk about the (said) causes of death by e.g. strangulation, while strangulation is truly the cause of a certain higher-level *disease* viz. respiratory arrest.
Second, if death is truly the destruction of a living being (as a whole), then is a living being ever really completely dead? It is not rare to find cases, where one would judge that the person is dead, but that some of her highly indirect constituents are still (a little bit) functioning: e.g. cadaveric spasms, or (said) postmortem DNA degradation.
If death is, like disease, also a *process*, then we may well argue that those very indirect constituents functioning barely properly are just at the end of the process of death.
Finally, and relatedly, if death is truly the destruction of a living being (as a *whole*), how to understand the *transplantation* of organs or tissues from a *deceased* donor, for those organs or tissues seem, if not completely healthy, at least partly so (to be intuitively transplanted to another body)? A plausible if not correct – at least, empirically realized - answer here is that, like any other process, (*complete*) death takes time; even if it is true that death occurs at the level of the whole living being, while the whole living being is dying (and that e.g. consciousness is already gone), those lower-level organs or tissues are also (necessarily simultaneously) becoming more and more diseased. During this (short) period of time, those half-diseased organs or tissues are retrieved, put into another body and healt (through surgical intervention or thanks to the novel body's own system).

9.3.3 A Prima Facie Definition of Health

What is, actually, the relationship between health and disease? If disease is intuitively the destruction of a *healthy process* (cf. Sec. 9.1.1), it is also, as we have seen (cf. Box 9.3), the destruction of (the *essence* of) a *processual part of a living being*.
Following this line of thought, one would say, thus, that a healthy process just is the essence of a processual part of a living being (cf. Box 9.4).

Prima Facie Definition of Health
x is a healthy process, iff *x* is the essence of a processual part of a living being.

Box 9.4. – Prima facie definition of health.

To say that health is the essence of a processual part of a living being is not to say much about what health really is (cf. Box 9.4).

Although a whole PhD dissertation can be devoted to analyzing the concept of health (as well as of life or even death), we can touch the topic of health and, relatedly, life here, only by making a few comments on it (and providing only a prima facie definition of health); ditto for the case of a biological malfunction (cf. Box 9.6).

Firstly, the bearer of health cannot be the whole living being (*pace* Nordenfelt, 2018); there is no *overall* health (*pace* Hausman, 2015)[181]; ditto for disease (cf. Exc. 9.1 on death).

Secondly, and most importantly, health, as a positive entity, is more *fundamental* than disease (as a negative entity) (cf. Sec. 9.3.1); health is *not* the absence of disease; following essentialism about disease (cf. Box 9.3), to the contrary, disease is the absence of health[182].

[181] Two caveats on overall health: (i) the above impossibility of overall health is to be distinguished from the impossibility of *complete health*. For, the WCP does not imply that attaining a perfect *x*, or attaining a healthy *x*, is pointless, but only that, if someone does decide to do so, it shall necessarily come with a cost (within one and the same specific entity) i.e. that having a healthy *x* implies that it is impossible not to have some diseased *y* at once.

If it is true, with Boorse (1977) here, that *complete* health, in the plausible sense of having for a certain living kind *all* of its parts healthy, cannot be achieved because of the WCP, it remains true that e.g. higher-level health or other lower-level healthy *x*s *can* still be reached (and are certainly worth to): e.g. being the perfect man is not being e.g. the best sprinter *and* the best boxer *and* the best tennis player *and* etc. (*pace* Boorse, 1977), for this is not, first, what it is to be, strictly, a man, and, second, along our permissive neo-Aristotelian ontology (cf. Exc. 3.5), "sprinter", "boxer" snd "tennis player" are certainly just three different (indirect) absolute realizers of "man".

Within one single (healthy) realizer there should not be in principle *incompatibilities* like the (said) above ones.

Boorse's main argument against health as a positive ideal (beyond the mere absence of disease) or against what he calls "positive health" i.e. as being unattainable misses, thus, its target, if there is no such thing, from the outset, as complete health (cf. Dinkmeyer & Dinkmeyer Jr., 1979).

(ii) If there is no such thing as overall health (and disease too), what are we saying, thus, when we say that *I*/we/(s)he/etc. is healthy/diseased? Likewise, when we say that this *person* is healthy/diseased, is it really this person (as a higher-level part of a living being), who is healthy/diseased, or rather one of its (direct or indirect) *parts*?

There are two possible answers to this question: first, we can answer that, in such cases, we are mistakenly referring, indeed, to, not the *direct* bearer/host itself, but an *indirect* higher-level kind (e.g. the whole living being, or a part of it), which the true bearer/host is necessarily a(n indirect) lower-level part of;

or, second, we can very well answer that those sentences do not *really* ascribe health or disease to the wrong bearer/host (to the contrary of the first answer), but that they are shortcuts for e.g. "*I* am *indirectly* healthy/diseased (*from x*)".

The explanation for all this is certainly *epistemic* (and practical): we are used to identify (and it is also more convenient) some lower-level entity by another higher-level one.

[182] Caveat on the mere existence of values: how to address the issue that all (morally) evaluative (and normative) statements are false (on the famous *error theory* generally restricted to the field of metaethics, cf. Jaquet & Naar, 2019; Mackie, 1977)?

Since error theory goes against common sense thinking, it is generally agreed that the burden of proof is on error theorists; to support the claim that (moral) value (and normative) ascriptions are illusory, error theorists globally rely upon a certain evolutionary thinking, according to which, roughly, (human) evolution does not presuppose that moral (or, more generally, value and normative) beliefs are true, but only *useful* (to e.g. human cooperation).

However, with such an argument, we fail to see in what sense, thus, *non*-moral (or non-evaluative and –normative) beliefs would be really different from moral (or value and normative) beliefs i.e. that e.g. the arithmetic belief that 1 + 1 = 2 would not be true, but only useful (to human survival).

Our claim that health is not the absence of disease does not follow, as I understand it, the WHO (1946, Pref.)'s definition, according to which health is a "[…] state of complete physical, mental and social well-being, and not merely the absence of disease or infirmity", for, first, health is not to be ascribed to the *overall* living being, and, second, the claim that health is not *merely* the absence of disease (but something more) is not to be read here at the ontological level – rather, as a mere expression for "There is more to say about the concept of health than as a mere absence of disease" - ditto for Boorse (1977)'s notion of positive health.

Third, to distinguish our prima facie definition of health from other well-known conceptions, it is noticeable that our prima facie definition of health goes against Boorse (1977)'s famous BST of health, according to which, roughly, health is biological function (as a contribution to inclusive fitness) statistically established (against the BST of disease, cf. Sec. 8.2.2);

another conception of health is the so-called free-energy principle – at least, in the present state of the art (but, for promising prospects, cf. Kirchhoff, 2018) –, where the free-energy principle "[…] tries to explain the ability of biological systems to resist to a *natural* tendency to disorder" (Friston, 2012, p. 2100; my emphasis) i.e. that health would be e.g. a resistance to environmental pressures or forces.

However, a healthy process would intuitively (continue to) be so even outside a context of resistance to environmental pressures or forces, or when there is just nothing which to withstand: a living being shall still be said healthy, even when there exists no disease at all, which health can be an absence or a lack of, e.g. in a context where a living being is put in quarantine.

Fourthly and finally, to the extent that our prima facie definition of health relies on the notion of a living being (cf. Box 9.4), how to understand life? Again, we can only touch upon this huge topic here. As Aristotle says (*DA*. II.4, 415b4), "[…] in the case of living things, their being is to live […]". In other terms, what it is for a living being to *live* is what it is for it to *be* or to exist (as a living being) (cf. also Thompson, 2008 for the same idea) i.e. that life – and, thus, indirectly health (as being a part of a living being) – is a value grounding the *existence* of a certain bearer: e.g. a *living* human just is an *existing* human being, or a *healthy* PAH-tion or cell growth just is a(n) (existing) PAH-tion or cell growth (with all its constituents) (as being *necessarily* a part of an *existent* living being). The (positive) *value* of life (and, indirectly, of health) is based on what makes the thing – that we are willing to ascribe the value of life (and, indirectly, of health) to - the very thing it is, though life and health are not essentially or *strictly* the essence of something, for, otherwise, they would not be kinds/species themselves (cf. Box

3.1)[183]; this "what makes" the thing it is i.e. its *essence* is, of course, what is precisely hugely contentious and disputed in theories of life (and, indirectly, our theory of health) (on the *organicist* movement trying to ground the notion of life mostly through the refreshed use of old notions like homeostasis, self-reproduction, or metabolism, cf. El-Hani & Emmeche, 2000; Nicholson, 2018; also Baedke, 2018; Meincke, 2018).
In other words, talk about the values of life and of health is related to the mere presence of something *specific* and its *specific* essence.

9.3.4 A Prima Facie Definition of a Lethal Value

If we agree that health is a specific *vital* value (cf. Sec. 8.1.1), and if we agree with our prima facie definition of health, according to which health is the essence of a processual part of a living being (cf. Box 9.3), then we can easily give also a prima facie definition of a *lethal* value, in all its generality i.e. where *disease* and *death* are (certainly) its two absolute realizers.
In order to notice more clearly and into greater details the interwinement between intuitions (1) and (2) about disease (cf. Box 8.1-8.2; also Sec. 9.4.1), it shall be useful to briefly provide such a prima facie definition.
As said above (cf. Sec. 9.3.3), the value of life is based on what it is for something to *be* a *living* being i.e. on its essence, and the value of health is also based on what it is for something to *be* that very *healthy* thing it is (as being necessarily a part of a living being).
Following this, we can claim that, plausibly speaking, *p* is vital to *x*, iff *p* is a *specific* essence (or the specific set of essential properties) of *x*, where *x* is a specific kind: e.g. *possessing a metabolism* is (partly) vital, in a specific way, to a living being, for it is (at least, partly) this specific essence we are looking for, when we call something a "living being".
What is a lethal value? Along the line that negative entities are generally deprivations from, or negations of, positive ones (cf. Sec. 9.3.1), if we take vital values to be *positive*, then a lethal value is obviously the *deprivation* of a vital one. We get, thus, the following prima facie definition of a lethal value:

Prima Facie Definition of a Lethal Value

[183] In that sense, a *strict* definition of health shall not have to follow RME i.e. that it does *not* say that health is *essentially* e.g. a part of a living being's inclusive fitness (as an essential feature of it) (*pace* Boorse, 1977), or etc.

x is a lethal value, iff *x* is the deprivation from the essence of a living or healthy being.

Box 9.5. – Prima facie definition of lethal value.

9.4 Disease as Essence Destruction

Sec. 9.4 is devoted to analyzing more thoroughly the notion of an *essence destruction*, which is at the basis of our definition of disease (cf. Box 9.3). While Sec. 9.3 focuses more on intuition (1) viz. that disease is a specific lethal value (cf. Box 8.1), by focusing on the notion of a *healthy process* (as the disease host), Sec. 9.4 is more related to intuition (2) about disease, according to which disease is a specific biological malfunction (cf. Box 8.2).

Sec. 9.4 is divided into three further sections: in Sec. 9.4.1, I argue that, as a specific biological malfunction, disease is the destruction of a healthy process, where talk about *functions*, in all its generality, just is talk about the *essence* of a process.

In Sec. 9.4.2, I argue that there are two different specific ways for a disease to destroy the essence of a processual part of a living being (or a healthy process) viz. by *hyper*-functioning or *hypo*-functioning.

Sec. 9.4.3 deals with disease *comparisons*. Indeed, with our essentialist account of disease, we might wonder how we are to understand how to compare diseases between them; that much also helps in distinguishing, again, the concept of disease from other closely related ones and easily misidentified with.

9.4.1 A Prima Facie Definition of a Biological Malfunction

Again, as for many other concepts analyzed or just touched upon in this PhD dissertation (cf. e.g. Box 9.5), a whole book (or, more likely in the present case, several major books) can be written about the topic of (biological) functions, *dys*functions and *mal*functions.

In Sec. 9.1.3, we defend the idea that, to take coherently into account both intuitions (1) and (2) (cf. Box 8.1-8.2), the notion of a biological malfunction at the basis of disease is to be roughly

analyzed, for the cases of PKU or (lung) cancer, as the *destructive process* of the biological function of PAH-tion or cell growth (in the lung's tissues) (as a processual part of a living being).

What is it to say, more precisely, that disease is a certain biological malfunction, in the sense that it is a destructive process of the *biological function* of PAH-tion or cell growth (as a processual part of a living being)? Following Box 9.3 and Sec. 9.3, according to which, globally, disease is the destruction of (the *essence* of) a processual part of a living being, we come intuitively to the thesis that the biological function of PAH-tion or cell growth just states what PAH-tion or cell growth (as a process) is i.e. its *essence*.

However, in all its generality, talk about (biological) functions is related to talk about the essence of a *positive* or *negative* (biological) process (cf. Pellet, 2019; *pace* Ari. *GA.* II.4, 740b19–34; II.6, 743a16–36; Neander, 1991): e.g. if the function of a washing machine is to clean clothes, then cleaning clothes is intuitively just what the washing machine (as a process) *is*; or, if the function of PAH-tion or cell growth is to convert phenylalanine into tyrosine or to divide into two daughter cells and to group them, then the conversion of phenylalanine into tyrosine or the division into two daughter cells and their grouping is what PAH-tion or cell growth (as a process) *is*; or even, if the function of a psychopath is, amidst others, to lack empathy and to be deficient in formulating correct moral judgements, then lacking empathy and being deficient in formulating correct moral judgements are, amidst others, what psychopathy is – which, as a negative entity, also has, of course, its own correctness conditions (as long as *norms* are strictly distinguished from *values* here, of course).

If we are right here that talk about functions is merely related to the *correctness conditions* (or the essence) of a process, then talk about (biological) *dys*functions (or, strictly speaking, *para*functions) just is talk about the incorrect functioning of a *positive or negative* (biological) process itself: e.g. albeit PAH-tion or cell growth can be said *dys*functional, in the sense that its essence is being destroyed, or that its correctness conditions are no longer present, PKU or cancer (as a negative process) can be also said *dys*functional, in the sense that it does *not* destroy (correctly) the essence of PAH-tion or cell growth i.e. that, contrariwise, PAH-tion or cell growth functions correctly[184].

[184] Three caveats (with terminological notes) on functional properties (or (dys)functions): (i) for *x* to be said to be correctly, normally or ordinarily (dys)functional (*simpliciter*) is always a matter, actually, for *x* to be *ultra* (intensively, extremely or ideally) (in)correctly (dys)functional - but not *excellently* (*perfectly* or *optimally*) (in)correctly (dys)functional, for the notion of an excellent/perfect/optimal function is related to the function of a *good* bearer *only* (also sometimes referred to, throughout this dissertation, as "function" *tout court*, when the context is not confusing);
otherwise, *x* shall be said to be only too (or excessively) little/the least (possible)/lowly/(very) little/a little bit/pretty/(very) much/highly/the most (possible)/too (or excessively) much *(in)*correctly (dys)functional.

A first major consequence of our taking the dichotomy functions vs. dysfunctions as a purely *normative* dichotomy (in a narrow sense i.e. as merely related to norms (vs. Exc. 2.6)) i.e. as the correct/normal functioning of a positive or negative process vs. the *in*correct/*ab*normal function of a positive or negative process is that it is, precisely, not axiological; in that sense, we do not associate so tightly, unlike here Aristotle's famous function argument (*EN*. I.7), according to which, roughly, the human good resides in human function (cf. Gotthelf, 2012; Korsgaard, 2008), talk about functions (*tout court*), *at their basis*, with goodness (Wakefield, 2000) – but with correctness or essence only; moreover, we de not ground goodness on functions (on enhancement, cf. Exc. 9.2).

A second consequence is that *norms* are more fundamental than *values*.

But, if the notion of disease as the destruction of a *healthy* process (or the essence of a processual part of a *living* being) cannot be captured somehow, indeed, through the notion of a biological *dys*function (for, it is a purely normative notion), we can expect, however, that the word "(biological) *mal*function" (taken as a medical natural kind, cf. Lange, 2007; also Sydenham, 1741) – by contradistinction with "(biological) *dys*function" - truly captures, indeed, the notion of a biological function related to disease taken as something *bad*, while health would be associated not to biological functions *simpliciter*, but only the specific ones whose bearer is *good* or positive (cf. Pellet, 2019) (cf. Exc. 9.3).

In other words,

> [h]ealth [is] a *specific* biological function viz. the correct functioning of something (biologically) *good*, while disease [is] also a *specific* biological function viz. a *specific* biological *dys*function, or the incorrect functioning of something (biologically) *good* i.e. a biological *mal*function. (Pellet, 2019, p. 56; italics original)

Fig. 9.3 summarizes the above remarks as follows:

If not otherwise explicitly stated or made somehow contextually clear (i.e. where the notion of (biological) (dys)function is to be loosely (or *plausibly*) understood), this is how the word "(biological) (dys)function" is used, in its *strict* sense, throughout the PhD dissertation.

(ii) When the context is clear enough, talk about functions *tout court* may well be plausibly used, throughout the PhD dissertation, to refer to, more specifically, *good* (*biological*) functions i.e. (biological) functions whose bearer is something positive. But, such a talk is to be avoided in easily misleading cases.

(iii) The notion of *ultra-function* should not be confused with the notion of *over-function* (cf. Sec. 9.4.2 on over-functions).

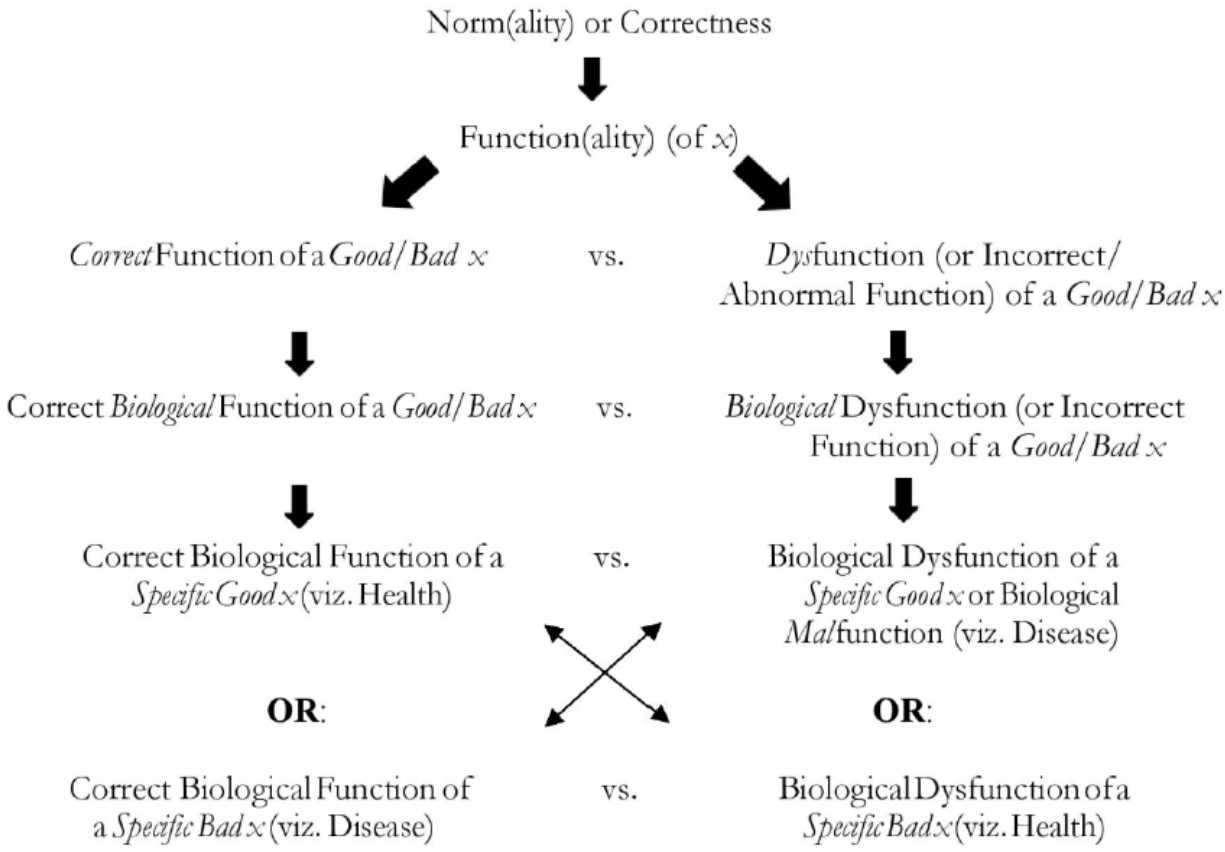

Fig. 9.3. – Depiction of the relationships between (ab)normality and (biological) (dys/mal)functions. Reprinted from Pellet, 2019, p. 57.
Legend: the one-way arrows stand for a relationship of specificity; the double arrows for a relationship of equivalence.

From all this (cf. Fig. 9.3), we can provide a prima facie definition of a biological malfunction, according to which a biological malfunction is the destructive process of a healthy process (like *what* PAH-tion or cell growth *is*) or a life process (e.g. like what a human being is) – thus, destroying PAH-tion or cell growth *themselves* (or a human being) (as a whole) (cf. Box 9.6).

Prima Facie Definition of a Biological Malfunction
x is a biological malfunction, iff *x* is a destructive process of the essence of (a processual part of) a living being.

Box 9.6. – Prima facie definition of a biological malfunction.

We eventually come to see more clearly and into more detail how intuitions (1) and (2) about disease (cf. Box 8.1-8.2) are unified into a single definition of disease. Indeed, our prima facie

definition of both a lethal value (in all its generality) and the one of a biological malfunction are obviously very much alike (cf. Box 9.5-9.6); intuitions (1) and (2) are both analyzed in the same way i.e. as basing the notion of disease on the one of a destructive process of (the essence of) a processual part of a living being (or, a healthy process) (cf. Box 9.3) (cf. Fig. 9.4)[185, 186].

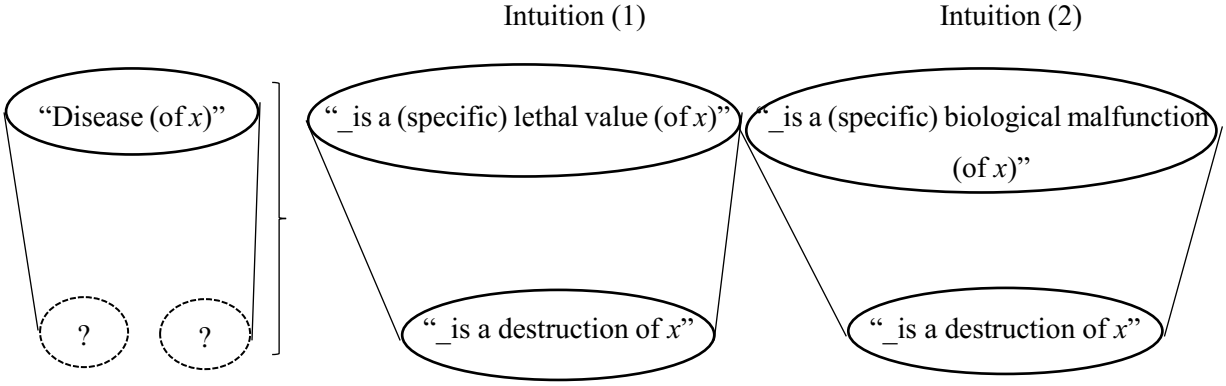

Fig. 9.4. – Depiction of the analysis of intuitions (1) and (2) about disease (cf. Box 8.1-8.2), on basis of our definitions of a lethal value and a biological malfunction (cf. Box 9.5-9.6).

[185] Two caveats on our analysis of intuitions (1) and (2) (cf. Box 8.1-8.2) and our framework for thinking about disease (cf. Sec. 8.1): (i) we may wonder whether, with our stance on intuitions (1) and (2) as two sides of the same coin (cf. also Sec. 9.1.3), we are not, actually, just *explaining away* intuition (1) in terms of the other – or vice versa – i.e. that essentialism about disease would not be a hybrid theory of disease (cf. Box 9.3).
To explain away intuition (1) or (2) is typically e.g. to *reject* intuition (1) or (2) or to *reduce* intuition (1) or (2) to the other. It is clear that essentialism about disease, first, does not reject intuition (1) or (2) – it tries to take both of them into account; second, essentialism about disease does not reduce intuition (1) or (2) to the other, for, analyzed as two genuine (or *irreducible*) synonyms, the notions "(specific) lethal value" and "biological malfunction" are, thus, merely (non-explanatorily) equivalent, while "_reduces to_" is intuitively an asymmetric relationship.
(ii) If we claim that intuitions (1) and (2) are *inseparable*, then can our framework for thinking the debate around disease still be used, for, with its three groups of theories of disease, it is based on the idea that intuitions (1) and (2) are clearly separable? Should we throw away the ladder after having climbed it up? In light of essentialism about disease, we have to eventually reject the presented framework; however, it does not imply that essentialism about disease cannot be meaningfully considered as prima facie a *hybrid* theory of disease, *globally* speaking, for its starting point is still to coherently take into account both intuitions (1) and (2); moreover, as such, the groups axiologism, malfunctionalism and hybridism about disease are all developed (and presented) *independently* from a certain ontology, though the theories within these groups have to be *coherent* with a certain *Weltanschauung* (though not necessarily ours).

[186] Caveat on intuitions (1) and (2) as two sides of the same coin: how are we to understand more precisely the image of our analysis of intuitions (1) and (2) (cf. Box 8.1-8.2) as two sides of the same coin (cf. Fig. 9.4)? By this metaphor, we do *not* mean that a specific lethal value like disease and a specific biological malfunction are just one and the same thing – to the contrary, both are two sui generis entities i.e. that, though synonyms, they are irreducible to each other (like for the infamous cases of Clark Kent and Superman) (cf. caveat (i) above).
While our analysis of the notion of (biological) *mal*function is already normatively *and* axiologically based – which obviously explains its synonymicity with our analysis of intuition (1) -, it is far beyond the reach of the present PhD dissertation to thoroughly explain, at the global level, the complex relationship between values and (biological) functions.
However, if we are right in claiming that functions have to do merely with norms, then values seem to be irreducible higher-level entities, which have emerged from specific biological functions (evolutionarily selected for their relationship, likely, to *affective* states/processes).
I thank Ulrich Krohs and Niko Strobach (April-June 2021) for having asked for clarification on that point.

Legend: the oblique bars represent a constitutive relationship.

9.2 Excursus: On Enhancement

As being close to the notion of health as a positive entity, as understood throughout this PhD dissertation (cf. Box 9.4), how to understand the notion of (biological) *enhancement*? Along a deflationary understanding of (biological) enhancement, enhancement is the mere treatment/remedy for a *good* organism('s part) or a means to merely repair or regenerate a good organism('s part) i.e. (*restitutio ad integrum*/*transformation ad optimum*) for a good organism('s part) (so as) to achieve *perfection* (by having a normal (or ideal) - rather, *perfect* here - *life*, or by being *healthy* (Nordenfelt, 1998).
In other words, talk about e.g. an *enhanced psychopath* makes no sense: albeit Ted Bundy can be a certain (partly) incorrect(ly functioning) psychopath (as a psychopath), we do not obviously (partly) *cure*/*enhance* him by making him a correct(ly functioning) psychopath, although he may well be (partly) *reconstituted* or *regenerated* into a certain correctly functioning psychopath; in other words, because it is (arguably) a specific *bad* (part of an) organism, (partly) incorrectly functioning psychopathy can be (partly) *worsened* (or pejorated) into correctly functional psychopathy - not *bettered* or enhanced (on biological enhancement (with different understandings), in all its generality, cf. e.g. Baertschi & Mauron, 2011; Bayertz & Quante, 2003; Juengst & Moseley, 2019; Harris, 2007; Quante & Stoppenbrink, 2011; Schöne-Seifert et al., 2009).

9.3 Excursus: On the Logical Equivalence between Generation and Destruction

The generation of (state or process) x is logically equivalent to the destruction of $\neg x$, in so far as $\neg\neg x$ is logically equivalent to x; likewise, the generation of $\neg x$ is logically equivalent to the destruction of x: e.g. suffering or fearing less or more than what one ought to given a context is equivalent to something (a bit/very/etc.) *positive*.
From this, first, we can argue that talk about either the (*mere*) generation of ($\neg$)x or the (*mere*) destruction of ($\neg$)x is *value-neutral*.
Thus, talk about e.g. *de*generation (or *de*composition) synonymously with destruction (of a whole) should not mislead us think, therefore, that generation (or composition) is a certain *positive* entity.
More generally, linguistic usage or, more precisely, *morphology* is misleading – at least, here in the search for the correct meaning of a term: e.g. "*dis*abuse", "*dis*encumbrance", or "*anti*-AIDS" does not

imply that “abuse”, “encumbrance”, or “AIDS” is a *positive* term (or, somehow wears the trousers – for (axiologically) neutral terms).

(However, this is not the case for *etymology*, for etymology, when correctly used, does not reveal, actually, something idiosyncratic to a certain language, but something about the *origin* of the conceptual structures underlying language, in all its generality – thus, not necessarily about the *current* meaning of a term (or concept) due to its possible changes through time, of course; that different linguistic systems may share the very same etymology attests to that point.)

Although e.g. disease can be seen as the *destruction*/privation/negation of a certain entity until its negation (cf. Box 9.3), it can also be seen as the *generation* of a certain entity viz. a negation from its (own) negation, while the value of disease remains (intuitively) the same in both cases viz. *negative*.

If, generally speaking, badness is seen as a *privation* of goodness (or *deprived* goodness) (on this classical thesis, cf. Oderberg, 2014; 2020) goodness cannot be seen, thus, as a *mere generation* (or fulfillment, as Oderberg puts it), for badness (under this analysis) is also (logically equivalent to) the *generation* of some*thing*. In other words, talk about *mere* absence vs. presence, or destruction/elimination vs. generation, etc., does not carry *by itself* any ontological priority or definitional relationship between one over the other.

Even if there is a logical equivalence relationship between x and $\neg\neg x$, it does not mean that positivity is (to be defined as) *privation of negativity* (i.e. of $\neg x$). Were this true, then, indeed, positivity could be analyzed as an *absence* of negativity.

Rather, the logical equivalence relationship merely indicates that positivity is privation of privation of positivity i.e. that, trivially, positivity is (privation of privation of) positivity. Positivity can be, indeed, considered as (logically equivalent to) an absence of negativity, *just like* negativity can be considered as (logically equivalent to) an absence of positivity (through the logical equivalence relationship), but it does not change that positivity *ontologically* grounds negativity.

Second, if the destruction of x is also at the same time equivalent to the generation of $\neg x$, the generation of $\neg x$ shall be intuitively interpreted as the generation of a *specific* entity/kind viz. a *negative* one – rather than the *negation of* a (*mere*) entity/kind -, while, even if the destruction of $\neg x$ is also at the same time equivalent to the generation of x (due to the logical equivalence between $\neg\neg x$ and x), the generation of x shall be intuitively interpreted as the generation of a specific kind itself viz. x – rather than the *negation of* $\neg x$.

Thus, the logical equivalency between generation and destruction tends to privilege negative entities as specific kinds themselves, instead of negations of (*mere*) kinds (cf. Sec. 9.3.1).

Third, it goes without saying that, if badness is seen as the destruction of something positive, and if destroying something negative is equivalent to generating something positive, then we do not cure a disease (as something negative) by generating something *negative* too (unless no other strategy is available): e.g. curing oneself through self-deception or false beliefs, through hypnosis (considered as an altered state of consciousness), etc.

Finally, it is important not to confuse the logical equivalency between generation and destruction with the neo-Aristotelian WCP, for the destruction of a *positive* kind *x* is logically equivalent to the generation of a *negative* one, as well as *entails* (by the WCP) the generation of another *positive* kind – which is a contrary to *x* -, and vice versa (cf. Exc. 2.3).

9.4.2 Hypo/Hyper-functions, or How to Destroy an Essence

If a disease is truly the destruction of the essence of a processual part of a living being (e.g. of what PAH-tion or cell growth is) – and, thus, of PAH-tion or cell growth (as wholes) themselves – (cf. Box 9.3), then how does it do so?

There are basically (and intuitively) two different ways for a disease to destroy (or remove) a healthy process or just a processual part of a living being: (i) through *subtracting* or decreasing intensional parts from e.g. PAH-tion (’s essence) until (complete) destruction or annihilation i.e. to get a hypo-PAH-tion, or *hypo-* (or *under-*)*functioning* PAH-tion;

or, (ii) through *adding* or increasing intensional parts to e.g. cell growth(’s essence) until (complete) destruction or di*s*assembly i.e. by getting hyper-cell growth or *hyper-* (or *over-*)*functioning* cell growth[187].

If ways (i) and (ii) are two different specific ways for a *positive or negative* whole to lose its essence or to have its essence removed (or deficient) – for, as for the notion of (biological) function *tout court*, the notions of *hypo-* and *hyper-*functions are not by themselves axiological notions, but purely *normative* ones (cf. Sec. 9.4.1) -, then ways (i) and (ii) are also, through logical equivalency, two different specific ways for a (positive or negative) whole to *generate*/gather together/assemble its essence: e.g. destroying *x*(’s essence) until ¬*x* through

[187] Three caveats on hyper/hypofunctions: (i) hyper/hypofunctions may be interestingly put in parallel with the two different ways to raise objections against a certain definition viz. by claiming that there are, respectively, *unnecessary* or *insufficient* conditions to a certain definition.

(ii) If the elements *Fx*, etc. of a definiens (*Fx* & …) are merely *conjuncts* (cf. Def_4), then there is no structure (taken in a certain way), order, configuration, shape, interdependence, etc., *in* the definiens or *amongs*t the elements of the definiens.

Although there are intuitively different *ways* of destroying (¬)*x*(’s definiens) (e.g. by *subtracting* (¬)*Fx* from (¬)*x*’s definiens; or *adding* (¬)*Hx* to (¬)*x*’s definiens), as well as of generating (¬)*x*(’s definiens) (on generation/destruction as two sides of the same coin, cf. Exc. 9.3), the *order of destruction* of (¬)*x*(’s definiens) (due to the *truth-functionality* of the definiens) does not matter: e.g. whether (¬)*x*(’s definiens) is deprived from, first, (¬)*Fx*, then, from (¬)*Gx* (by, in a more specific way, subtracting them or adding extra elements).

(iii) The notion of hyper/hypofunctions has nothing to do with, specifically, *quantitative* kinds. Indeed, a hyper/hypofunctioning kind may very well be qualitative (just like it can be also quantitative).

subtracting *x*'s essence from *Fy* & …, or through adding to *x*'s essence *Hy* & …, is (logically) equivalent to the generation of *two specific* ¬*x* viz., respectively, *hypo-x* and *hyper-x*.
PKU and (lung) cancer can serve to perfection as two illustrations of, respectively, the destruction of PAH-tion through a *hypo*-function, and the destruction of cell growth through a *hyper*-function;
indeed, as we have seen (cf. Fig. 9.1-9.2), PKU is a *specific way* of destroying PAH-tion i.e. by making tyrosine *decrease* (or subtracted); (lung) cancer is a specific malfunctioning growth, or uncontrolled proliferation (s*ensu lato*), of (specialized) cells (e.g. respiratory epithelial cells), or *hyper*plasia followed by neoplasia (in the lung's tissues).
Other clear and everyday examples include:

- *hypo-x* (e.g. perhaps *cowardice* as hypo-courage/-bravery) vs. *temerity* as hyper-courage/-bravery) as two different ways of being *non*-courageous/brave (cf. Exc. 3.3);
- *hypo*somnia vs. *hyper*somnia as two different ways of having a *para*somnia i.e. some bad sleep here.
- more generally, *under*reaction vs. *over*reaction/sensitivity/tolerance (e.g. of the immune system in the cases of allergies) as two different ways of being *non*-/*un*reactive;
- or even *oligo*dactyly vs. *poly*dactyly as two different ways to destroy some whole i.e. by having too few or too many fingers (or toes) (cf. also Sec. 10.1.3)[188].

In pharmacology, medicines have been traditionally classified by the famous Anatomic Therapeutical Chemical (ATC) classification system (WHO Collaborating Centre for Drug Statistics Methodology, 2020) along (amongst other criteria) their different *mode of action*.
In the ATC system, one can intuitively distinguish between specific medicines or medication (or even, at a more general level, *therapeutic treatments* or *remedies*)[189], which act as (i)

[188] Caveat on amputations: cannot we destroy a whole by *separating* it from its constitutive parts, or by literally *removing* its parts? But, is e.g. a severed hand no longer a constitutive part of the same whole as before? An amputated hand could still be said a *remote* constitutive part (or, a non-attached token part) of a (token) living body from which it was severed (cf. Exc. 2.2 on scattered objects).
However, we would like to intuitively answer that literally *spatially* separating a token part from a token whole is partly destroying this token whole (e.g. scattering or dislocating the parts of a (token) whole), even if is true that the token part still functions correctly (spatially "outside" this token whole) – it also has to be necessarily e.g. *transplanted* to another token whole.
An amputation may be considered (in a certain way) as a specific way to destroy a token whole by removing its token parts or by making such that they are not any more *spatially contained* in this token whole i.e. that an amputation is a destruction of relational properties, where the relata can still very well function correctly.
In other words, at the token level, our two different ways to destroy (or eliminate) the essence of a token whole are very well illustrated: e.g. by putting aside (or tearing out) its essence.
[189] Two caveats on philosophy as a therapeutical science: (i) we may want to argue that *philosophy* (or metaphysics) itself (understood under a prescriptive or revisionary (*sensu* Strawson, 1959) – and not merely normative - guise, of course) can be considered, *plausibly* speaking, a *non-medical* therapeutic science: e.g. the (commonly said) second Wittgenstein (1953) sees philosophy as a specific therapy i.e., roughly, as a way to cure humans from the

inducers: e.g. amino acids (like tyrosine) inducers, insulin injection, protein substitutes/supplements, cholecalciferol absorption (in case of (a certain) vitamin D deficiency or deficit), prostheses, transplants, etc;
or, as (ii) *inhibitors*: e.g. chemotherapy, β-blockers (reducing blood pressure), anti-histamines (against allergies), antidepressants (e.g. to cure hypersomnia), antibiotics, etc.
Medicines (or, more generally, therapeutic treatments or remedies) (i) or (ii) are two different manners of curing, respectively, the above ways (i) or (ii) of being deprived from (the essence of) a *positive* entity (cf. Exc. 9.4).

9.4 Excursus: On Pseudo-Sciences, and Treatments for Higher-Level Diseases

If illness is, indeed, a specific disease (cf. Box 7.1), then anything (claimed to be) able to cure *this* (and not also other diseases) (like placebos, homeopathic drugs, etc.) shall count, actually, as a *true* therapeutic treatment (or remedy).
If (said) alternative or *pseudo- medicines*/*sciences* like (contemporary) homeopathy *claim*(*ed*) to cure *merely* e.g. (some) illnesses by bringing about e.g. a *placebo response* i.e. the relief of a feeling of

troubles/torments arising from philosophical issues by dissolving them (Peterman, 1992; on so-called *quietism*, cf. e.g. Pettit, 2004; Virvidakis, 2008); or e.g. reading (and understanding) this very PhD dissertation can make the reader healthier viz. by curing him from irrational thinking i.e., more precisely, unclear or confused thinking (about what disease is), or how to not to be diseased (or, less diseased), when thinking about what disease is, to the extent that e.g. some specific *irrational thinking* (or belief) can be seen as *the* specific higher-level disease (plausibly) cured.
(ii) However, strictly speaking (unlike with caveat (i)), as we have seen with the level at which our own meta-philosophical framework is situated (cf. Ch. 2-3), our meta-philosophy is, more globally taken, merely *normative* – and not also *prescriptive* or intentional (cf. Sec. 10.2.1); we merely give the reader reasons or *evidence* for which he ought to *believe* in the truth of our theory.
More precisely, we can say that we are still *describing* (as a descriptive metaphysics) norms of thought or epistemic norms (more specifically, the norms of belief) (Strawson, 1959); or even, philosophers might still be interested in describing *oughts* or duties – this is no prescription, exactly like feelings are themselves objectively defined, while they have a role in subjectivity.
In other words, the distinction between philosophy and non-philosophy is here the same as the distinction between, respectively and properly (but at a different level), a biomedical scientist (as a researcher and/or teacher) and the physician or *practitioner* scientist (even in his role as a theorist scientist giving *prescriptions*) – thus, the distinction in question does *not* just correspond to the simple distinction between theory and practice (for a more specific division of labor, cf. Exc. 3.5).
Nevertheless, does not philosophy, as conceptual clarification analysis, correct somehow our knowledge of the world or make us pass from unclarity (or ignorance) to clarity (or knowledge)? Is it not eventually a therapy or a cure? Philosophy's aim is even more modest: just to try to correctly describe (by giving good reasons) what are taken to be high-level (controversial) *complex* concepts or words hard to analyze – but, which are not essentially unclear; *we*, strictly speaking, are unclear with those concepts; we do not aim at curing humans from their unclarity with those concepts (cf. Berges, 2012), or even *improve* or further our conceptual representations (on so-called "conceptual engineering", cf. Cappelen, 2018; Eklund, 2017); as Antoine de Rivarol says (1856, *Pensées inédites*; personal tr.), "philosophers are more anatomists than physicians: they dissect and do not heal".

disease due to the administration of a placebo – whose essence is, arguably, to induce such an effect -, and if illnesses are meant to be cured by *medicine/sciences*, then they are/would *not* (be) pseudo-sciences(/medicines) (Haresnape, 2013; Shang et al., 2005; on bioethical issues about alternative medicines, cf. Anlauf et al., 2015; against the idea of the use of placebos as deceptions, Charlesworth et al., 2017; Chiffi & Zanotti, 2017b; but, against the placebo response as the scientific guarantee (or warrant) of homeopathy, or for envisaging the administration of a placebo as the mere absence of treatment or as a deceptive treatment, Stoessl, 2020).

They are to be deemed pseudo-sciences(/medicines), only if they claim to be able to cure specific diseases *beyond* the ones, which they *can truly* cure with the proposed treatment (National Health and Medical Research Council, 2015; cf. also the critics of the European Academies Science Advisory Council, 2017); or if they sustain to certain theories (e.g. of disease), which they claim to be scientific, despite *stronger evidence* to the contrary or no supporting evidence (at a certain time given a certain total amount of available knowledge) i.e. the intentional attitude of trying to raise to the rank of science what is not (vs. e.g. Arts, properly understood, i.e. disciplines claiming *not* to be scientific): e.g. *currently* sustaining to a miasma(tic) theory of disease, where miasma are said to be some bad air (Hahnemann, 1921); or treating a *para*normal action or process as an unexplain*able* (non-illusional or -hallucinated), mystic or esoteric phenomenon transcending (possible) rationalization;

or, if they have, of course, (self-)*deceptive* intentions (in the proposed treatment): e.g. the over-pathologization i.e. the invention of mental health issues – non-matching, thus, natural kinds -, which would have to be settled (e.g. by using vagueness): e.g. drapetomania (on the famous demarcation problem between science and pseudo-science, cf., as an entry, Hansson, 2017; Pigliucci & Boudry, 2013).

9.4.3 Disease Degrees and Comparisons

How do we *compare* diseases between them? How to judge the *severity* (or seriousness) of a certain disease?

Devoting some time now to these questions (rather unexplored in the literature) is necessary to any analysis on the nature of disease, for they are at the core of our daily ascriptions of disease to a host. How does our essentialism about disease (cf. Box 9.3) leave room, thus, to disease comparisons?

Disease, as a process unfolding through time until complete destruction of a healthy process, comes, indeed, with *degrees* or severities – which does not make "disease" a vague notion (*pace* Keil et al., 2017). While health (or the biological function of something *good*) can be said the

value *polarly* opposed to disease (cf. Sec. 9.3.3), disease is also a *scalar value* i.e. that it has degrees opposed to each other, and inversely equivalent to each other (e.g. to be only a little bit courageous is equivalent to be *e.g.* very much coward): if *x* is not diseased *tout court* (in the sense, more precisely, of being *completely* so) – for, each disease tends towards the complete destruction (or destruction *tout court*) of its (highly fine-grainedly differentiated) host -, *x* can still be, of course, only a *little bit* diseased, *very much* diseased, etc. i.e. that the process of destruction remains *unfinished* – which is, actually, what probably happens (contingently) most of the time for many specific diseases (thanks to e.g. the immune system or drug administration); disease degrees express, thus, the different *temporal parts* of the disease process (as a whole): e.g. to be a bit/very much diseased from PKU or (lung) cancer is for PKU or (lung) cancer to have its destruction process a little bit/almost finished[190]; thus, *at its own level*, every disease (as being a process) is more or less serious.

Note that, if there are disease degrees, there are also degrees of *death* to be conceived along the same line.

Disease comparisons are both intra- and inter-; disease intra- and inter-comparisons are assessed along disease (and death) severity. A disease *intra*-comparison is a measure of the progress (of the destruction process) of (at least) two tokens of one and the *same* type disease: e.g., plausibly taken, John has a more serious PKU or (lung) cancer than Mary i.e. that the (token) destructive process of PKU or (lung) cancer of (the token) John is more advanced than the one of (the token) Mary.

A disease *inter*-comparison, to the contrary, is the measurement of (at least) two *different* diseases belonging indirectly to one and the same living being, based on the idea that, in the higher level (of intension) the disease in question is, the more severe it is (on levels, cf. Exc. 5.1) i.e. that a host is dying more with a higher-level disease than a lower-level one, for it destroys more of its essence (or it is more *direct* than another one): e.g., plausibly taken, it is worse for a human being to have PKU or (lung) cancer than to have a heart murmur, for PKU

[190] Three caveats (with terminological notes) on the degree scheme: (i) it is important not to confuse the above degree scheme with the notions of *hyper-* and *hypo-functions*.
Indeed, the degree scheme (as presented here) is a mere measure for the progress of disease as a (*complete*) destruction process, while hyper- and hypo-functions are two different *specific* ways to incarnate somehow this destruction process (cf. Sec. 9.4.2): e.g. to be very much diseased can be done through something *hyper*functioning very much or *hypo*functioning very much.
(ii) Note that *states* too can have degrees: e.g. my cup of tea can very well be *partly* present (on degrees of being, cf. McDaniel, 2017).
(iii) Talk about degrees of disease may perhaps take into account the notion of an *infra-pathological* process.
Indeed, this rather vague notion seems prima facie hard to categorize, if not as having to do, precisely, with degrees of disease.
I warmly thank here Carole Fry (personal communication, November 2019) for having pinpointed note (iii) to me.

or (lung) cancer destroys more of, or is more direct to, a human being's essence than a heart murmur.

If e.g. for a human being to have heart murmur is a disease, then does this imply that a human being ought to go for treatment? Should he strive to attain such an ideal? Is it worth doing it? As being a mere constituent of a *very indirect* absolute realizer, or an indirect absolute realizer itself, of what a human being is – or even of e.g. happiness (as a highest-level part) -, there is no reason not to *privilege* or *prefer* other higher-level ideals to reach: e.g. having a healthy PAH-tion or cell growth, or just being happy; having a heart murmur should not, thus, make a human being very unhappy – but only a very little bit.

Disease inter-comparisons provide an *objective* measure for ordering such preferences, while still retaining, of course, the idea that, however higher- or lower-level, any disease remains bad *tout court*[191].

More generally, we can argue that the value of life is to be placed higher than the value of health (although they are both specific vital values); the ideal that we think worth reaching shall be always, first, the fulfilling of the essence of the living being (as a whole), while, only after, the fulfilling of the essence of the living being's (indirect) parts.

To summarize, essentialism about disease (cf. Box 9.3), with the rough idea of disease as a *progressive* destruction of the essence of a *part* of a living being (or, a healthy process) (cf. Box 9.1), understands disease comparisons through their basing on the notion of progress and on the idea of diseases as parts of a *dying* being (cf. Exc. 9.1).

However, from all this, the following remarks are in order: first, we usually judge that, e.g., it is worse to have a broken nose than a stuffy nose, if we suppose that in both cases the nose has lost exactly the same amount of essential properties – but not the same ones, of course. Were

[191] Three caveats on *Moorean monism* here (Massin, 2015; Rønnow-Rasmussen, 2011): (i) Moorean monism is the view that only goods/bads *period* exist; there are no *personal* values; goods/bads *for* reduce to *impersonal*, absolute (in the sense of not personal here) goods/bads or goods/bads period/*tout court*. Goods/bads for are not sui generis entities.
Indeed, first, saying that e.g. pumping blood is a *good for* the heart process or that a disease is a *bad for* its host seems like saying that a constituent is *essentially* a part of a *positive/negative* whole (against RME, cf. Sec. 2.1.2). Second, *good/bad for* can be also interpreted as the claim that something is a mere *cause* of something good/bad. Third, good/bad for may be *deflationarily* understood just as quoting the *positive bearer* (for good for) or the *host* (for bad for) in question – and *not* as assuming that what is good for *x* is also bad for *y*: "pumping blood is a good for the heart" means "pumping blood is a *good* with a *positive bearer* viz. the heart process" – it can never be turned into a bad for something else; the value in question is exemplified by something, and does not apply to other things. In that deflationary sense, good/bad for is reducible to good/bad, period.
(ii) If good/bad for is *strongly* understood as implying that what is good for *x* is also necessarily bad for *y*, then we argue against this view through the WCP: e.g. having an injury is always a bad, period; it can never be turned into a *good for y*, although it *implies* something good.
(iii) Note that the necessity of relative extensionality does not mean that a good for *x* is also a bad for *y*, for this would amount to category mistakes: e.g. "disease" has for relative extensionality something which is *negative* itself (on relative extensionality, cf. Exc. 3.2).

the essence of the nose (*Fx*) a mere conjunction of properties (*Gx* & *Hx* & *Ix*), then in cases where the nose is diseased in two different ways with the same amount of remaining essential properties like *Gx* & *Hx* (e.g. the nose is broken), or *Hx* & *Ix* (e.g. the nose is stuffy), no comparison of the severity of disease between the two cases could be done.

However, we can defend here that two different essences are destroyed here – and not the sole essence of the nose in two different ways.

Second, how, with our account of disease intra- and inter-comparisons, can we compare - as we intuitively and commonly do – between (at least) two different diseases of two different species/kinds themselves? It is often said, indeed, that e.g. humans have a worse sight than cats, or a worse smell than dogs with respect to e.g. the number of olfactive receptors, etc.

These cases can be easily treated exactly like with disease *intra*-comparisons: we can postulate the existence of an overall (theoretical) biological species/kind (and its parts), which, also as a type, would have e.g. a human being, a cat and a dog (as well as their parts) as tokens here. In that sense, *with respect to* that overall (theoretical) biological *type*, and by comparison with another token, e.g. the token human sight or smell – at least some peculiar part of it (even as much perfect as it can be) - would be, actually, worse than the one of the token cat or dog[192].

Third, how to understand the notion of *virulence* in accordance with what is said above? Literally speaking, the notion of virulence has to do with, obviously, the notion of *virus* i.e. that the term "virulence" should be *literally* used only in the context where a viral process is somehow involved: e.g. a *virulent disease* just is a specific (complete) destructive process of a healthy process resulting from the presence of a virus (as a starting *state*) as the cause of this process (cf. Box 6.2 on etiopathologies), or just is a *viral process*.

In line here with the dominant view in medicine (cf. e.g. Pirofski & Casadevall, 2012), comparisons made with the notion of virulence refers, thus, to either a certain etiopathological

[192] Caveat on species (intra-)comparisons: if there is no good/bad *for x*, but only goods/bads *period*, then e.g. *blindness* cannot be said a bad for a man but a good for a mole. Blindness is, indeed, a bad *tout court*, period or absolutely (in the sense here of not being a bad *for x*). As Aristotle points it out (Ari. *Met*. Δ.22, 1022b29-30; tr. Ross, 1924; my emphasis):

> […] blindness is a *privation*, but one is not 'blind' at any and every age, but only if one has not sight at the age at which one would naturally have it. Similarly a thing is called blind if it has not sight in the medium in which, and in respect of the organ in respect of which, and with reference to the object with reference to which, and in the circumstances in which, it would naturally have it.

If blindness is, in other words, privation of sight, and if sight is *not* a constituent of a mole (to the contrary of a man, etc.), then a mole cannot be said, *strictly speaking*, blind. There is no way in which a mole ought to have (naturally) sight, but is deprived of it.

If we happen to ascribe blindness to moles, it is only through those *species* (intra-)comparisons: e.g. with respect to an overall (theoretical) biological kind/species, a certain biological species with a (perfect) sight shall be judged better than a species without it (like a mole).

severity (with a virus as the cause) (e.g. HIV-AIDS) or just a viral process severity (as a certain disease process itself) (e.g. HIV infection); of course, that much does not mean that no comparisons, in general, can be ever made with e.g. disease kinds or symptoms themselves (cf. Ch. 4-5).

However, just as the notion of virus itself, in its *metaphorical* meaning, is very widespread, the same is true for the notion of virulence, in its metaphorical meaning; ditto for other well-known cases like malignancy (e.g. a malignant joy).

Fourth, the notion of (e.g. food or drug) *intolerance* (or sensitivity) seems directly related to disease degrees.

Indeed, the idea of intolerance to something is intuitively connected to the idea that one is not, strictly speaking, talking about a disease process (in its entire length), but about something less severe than a disease i.e. some (temporal) *parts* of it.

Lastly, what about the notion of *vulnerability* – at least, the more specific one related to the notion of disease? According to Samia Hurst (2008, p. 195; italics original), vulnerability (with respect to a population or person *x*) is, in all its generality, "[…] *an identifiably increased likelihood of incurring additional or greater wrong* [to *x*]" i.e. that for *x* to be vulnerable is for *x* to have likely *more* wrongs *than y*.

However, we might wonder whether this *comparative* aspect behind the concept of vulnerability is really essential to it; cannot we just say that *x* is vulnerable *tout court*?

Indeed, being vulnerable *simpliciter* is one thing, being *more* vulnerable *than* something else is another one; we fear, thus, that the above definition reduces the notion of vulnerability *tout court* to the relational (and comparative) predicate "_is more vulnerable than_" (for the same mistake about the concept of health, cf. Schroeder, 2013); the latter one is not more fundamental (somehow) than the former.

While disease has essentially *degrees*, that much does not imply by itself that disease *comparisons* are essential to the concept of disease: e.g. *x* can be a *little bit* diseased, while there is absolutely no other *y*, which to make a comparison with.

For *x* to be vulnerable is certainly just for *x* to have a *certain likelihood* in being hurt, harmed or injuried (cf. Box 7.4) – rather than merely diseased, if we strictly follow the etymology of "vulnerability", – which does not imply, as such, that *x* is necessarily *more* likely *than y* in being hurt, harmed or injuried (for, *y* may well be inexistent).

10

CONTROVERSIAL DISEASES AND OTHER DIFFICULTIES

To what extent is our definition of disease applicable? Is not our definition of disease too much exclusive or too much inclusive (cf. Box 9.3)? Is our definition of disease complete (or universal)? Ch. 10 is devoted to answering those questions by investigating the consequences of our definition of disease and by trying to defeat it.

Indeed, at a general level, it is constantly important to tackle the point of how far we can go with a certain definition i.e. how many peculiar philosophical issues it can (help) *solve* (cf. Sec. 3.2.4).

Furthermore, passing those difficult and controversial cases of disease under review also provides us with the advantage of grounding empirically (even more) our own definition of disease, and of avoiding with our definition of disease both the potential *over-restriction* of cases of disease and the potential trivialization or *over-pathologization* of cases of disease.

Ch. 10 is structured as follows: in Sec. 10.1, I review plausible counter-examples to our definition of disease, and show how our definition of disease is able to include them – thus, pointing towards the universalization of essentialism about disease.

In Sec. 10.2, I pass under review several controversial cases of disease directly following from essentialism about disease, and give reasons why essentialism about disease considers them as genuine cases of disease.

In Sec. 10.3, I review other controversial cases of disease, and give reasons why our essentialism about disease does *not* consider them as genuine diseases.

Other very well-known controversial cases have been also already treated or just highlighted in due course of this PhD dissertation: e.g. on pre-menstrual syndrome, cf. Sec. 4.2.2; on drapetomania, Sec. 8.2.

The choice of the present case studies is motivated both by a desire to variously illustrate (at best) our theory of disease, by a willingness to take into account the most intuitive and common complex and controversial cases of disease a scientist or a lay person can think about.

10.1 Difficult Cases of Disease

While Ch. 9 focuses on the cases of PKU and (lung) cancer (cf. Fig. 9.1-9.2) as clear and uncontroversial illustrations of essentialism about disease (cf. Box 9.3), Sec. 10.1 is devoted to analyzing different difficulties that our definition of disease may encounter. Can talk about disease as, roughly, the destruction of a healthy process account, in a necessary and sufficient way, for all what we take to be intuitive cases of disease (cf. Box 9.1-9.2)?

We might rightly doubt that: e.g. what about genetic diseases? acquired vs. inherited diseases? (non-)congenital diseases? etc. If they are not diseases, what are they, and can they be taken into account in the more general picture drawn in this PhD dissertation? It is not at all obvious, indeed, that essentialism about disease (or even the definitions of the cognates of "disease" or our own neo-Aristotelian framework) can prima facie take all of them into account (cf. Box 9.3).

Sec. 10.1 is structured with respect to the different counter-examples one can give against the necessity (Sec. 10.1.1-10.1.5) or sufficient (Sec. 10.1.6-10.1.9) condition of essentialism about disease.

In Sec. 10.1.1, I show how our definition of disease somehow takes into account the famous and difficult case of *genetic diseases* i.e. by making genetic diseases fundamental.

In Sec. 10.1.2, I argue, relatedly, that the dichotomy *inherited vs. acquired* diseases can also be easily taken into account by making inherited diseases genetic diseases, whose mutation has been passed from the ancestors, while acquired diseases are diseases, whose mutation has *not* been passed down from the ancestors; this dichotomy is not the same as the dichotomoy *congenital vs. non-congenital* diseases.

In Sec. 10.1.3, I argue that birth diseases are to be understood in terms of the *higher-level* (disease) process, of which they are a part.

In Sec. 10.1.4, I take into account *cultural* diseases as specific higher-level diseases (like any other one) whose host is a certain belief (or behavior), and whose *origin* can be traced back to a certain homogenous group.

In Sec. 10.1.5, I show that essentialism about disease is able to take into account *latent diseases* by taking them as either diseases partly present or not as diseases, but as specific etiopathologies.

In Sec. 10.1.6, I show that *chronic* diseases are just to be taken as long-lasting disease processes, by contrast with – but, often confounded with - *recurrent* or *common* diseases; as such, they can be perfectly well integrated to essentialism about disease.
In Sec. 10.1.7, I show that we are able to take into account *imagination-related* diseases as specific cognitive diseases, by contradistinction with imaginary diseases.
In Sec. 10.1.8, I focus on *deformities*, and show that a deflationary understanding of deformities as destructions of relations is still coherent with our essentialism about disease (cf. Box 9.3).
In Sec. 10.1.9, I show that there are no *artefactual diseases* (properly understood).

10.1.1 Genetic Diseases

How to coherently take into account genetic diseases with the picture of diseases drawn in this PhD dissertation? Are they not potential *counter-examples* to our definition of disease?
Indeed, as fundamental units, genetic processes do not seem to be essentially definable; in that sense, there seems to be no essence that a genetic disease can really destroy (against the idea that genes can fit into a single world picture or structure, cf. Waters, 2017).
First, we can take (plausibly if not correctly) genetic diseases to be specific ultimate or *fundamental* diseases *simpliciter* (cf. Sec. 3.3.1 on the notion of *genus*) – and not specific disease *kinds*. In a nutshell, a genetic disease can be arguably minimally understood as a specific genetic (non-random) *mutation* viz. a *deleterious* (or bad) one i.e. destroying a genetic process coding for a *trait* - narrowly used here as what is (directly) constituted by a gene(tic) (process) (*pace*, generally, Beurton et al., 2000); this fits with e.g. autosomal/X-linked/Y-linked recessive/dominant diseases as *monogenic* diseases (one genetic process-one trait), as well as *polygenic* diseases (many genetic processes-one trait) (cf. Exc. 3.3): e.g. as of 2011 more than 36 genetic processes have been said to (indirectly) constitute (or, to be an (indirect) absolute realizer of) type 2 diabetes (Herder & Roden, 2011).
If that is true, then, indeed, genetic diseases, as being fundamental diseases, cannot be essentially defined – for, genes do not have, strictly, essences -, but only *teleologically* or relatively extensionally i.e. that, as bad mutations, they, *consequently*, destroy the trait (as a process) a genetic process codes for, for this is what a genetic process is; in that sense, genetic diseases, as being fundamental diseases, are apart from our meta-philosophy, and, thus, to essentialism about disease (cf. Box 9.3).

However, in the literature (cf. e.g. Dekeuwer, 2016; Rheinberger et al., 2015), much more is usually put under the label "genetic disease" including diseases (kinds) like Down syndrome, sickle-cell anemia, etc. It seems that the term "genetic disease" refers (in our terminology), in such cases, to a specific disease (*kind*) *defined* as such that its *ultimate* absolute realizer(s)/constituent(s) is/are (a) bad genetic mutation(s) (with its causes-effects).

We should not use so widely the term "genetic disease", for (i) there is a threat of *trivialization* i.e. here of *geneticization* (e.g. as of September, 2018 6259 genetic diseases (over 3961 genetic processes) are reported in the Online Mendelian Inheritance in Man (2020)), to the extent that all disease (kinds) can be said to have, indeed, as ultimate absolute realizers bad genetic mutations or to have as ultimate constituents bad genetic mutations (for an illuminating analysis of this problem, cf. Darrason, 2014; 2017a);

and (ii) following our metaphilosophy (cf. Ch. 2-3) and our definition of disease (kinds) (cf. Box 4.2; 9.2), a certain disease (kind) is not *correctly* defined such that it can cross different levels of specific disease (kinds) (viz. here levels of absolute extension or of partial intension), where its ultimate absolute realizer(s) is/are a certain genetic disease, or its constituents are ultimate ones (on intransitivity in definitions, cf. Sec. 2.1.3).

Point (i) is said to lead to the so-called *paradox of contemporary medical genetics* (Darrason, 2017a), where "genetic disease" refers to either all specific disease (kinds) or to none. Taking genetic diseases as *certain* diseases *tout court* viz. fundamental or ultimate ones (thus, relatively extensionally defined like for "genus") is a way of dissolving this paradox.

Our taking genetic diseases as fundamental diseases does not imply that there are *only* genetic diseases, but that all specific disease(s) (kinds) can be said (merely) *fundamentally* genetic – thus, that a genetic disease is not any specific disease (kind) *defined* such that its ultimate absolute realizer(s) is/are (a) bad genetic mutation(s) or that its ultimate constituents are the processes giving rise to a bad trait: e.g. we can stick ourselves to the plausible (if not correct) definition of Down syndrome as, roughly, a specific mental disease (kind) having as an ultimate absolute realizer a certain genetic disease (kind) viz., roughly, the destruction of the process giving rise to chromosome 21 (as the resulting *state*) by adding an extra copy of it (and its causes-effects), or having as an ultimate constituent the process giving rise to an extra copy of chromosome 21.

That is why, contrary to what is usually claimed in the literature (cf. e.g. Selikowitz, 2008), Down syndrome is not a specific genetic disease (kind), for it is *not* a certain disease (kind) defined as a bad genetic mutation (with its causes-effects) (along our line), or as a disease whose ultimate realizer is a bad genetic mutation (with its causes-effects) or whose one of its ultimate

constituents is the process giving rise to an extra copy of chromosome 21 (as the resulting *state*) (for, no disease ever crosses multiple levels) (cf. point (ii)).

Moreover, we cannot say that Down syndrome is a specific *mental* disease (kind) viz. a specific *genetic* disease (kind), for we reject ((in)direct (or ultimate) absolute extensional and partial intensional) *genetic reductionism* to the extent that we reject any specific reductionism (cf. Exc. 2.5).

However, even if it remains true that all specific diseases (except genetic diseases themselves, of course) are fundamentally *constrained* by genetic diseases, cure must always intervene at the very specific level in question, never at a higher- or lower-level one: e.g. PKU can be also (plausibly) said to have as one of its *ultimate* constituents a genetic disease touching the PAH *gene* on chromosome 12, but this genetic disease is also commonly said to be (indirectly) an ultimate constituent of other specific disease(s) (kinds) (in our terminology) like hyperphenylalaninemia.

The rejection of genetic reductionism does not imply, of course, the rejection of a well-understood version of *genetic* (*pre*)*determinism* i.e. a *non-reductionistic* one (Carrier & Finzer, 2006; Kitcher, 2001) also called (most often in its reductionistic acceptance) “geno-centrism” (Darrason, 2017a; also Dawkins, 2016; *pace* (but especially with respect to evolutionary theory) Eldredge, 1995; Jablonka & Lamb, 2014), “genetic imperialism” (Juengst, 2000), or even sometimes “(genetic) preform(ation)ism” (weakly taken) (Maienschein, 2005), for we can consistently maintain both the thesis that a disease (or a healthy process) (kind) is *not* ((in)directly (or fundamentaly) absolutely extensionally) *reducible* to its fundamental realizer(s) viz. to a genetic disease (kind) or to its fundamental constituents (e.g. Down syndrome as a specific mental disease (kind)), as well as the thesis that a disease (kind) is – though *not defined* as being - *ultimately* absolutely realized by a genetic disease (kind) or has for ultimate constituents what gives rise to bad traits (e.g. Down syndrome as a specific mental disease (kind) ultimately absolutely realized by a specific genetic disease (with its causes-effects); or Down syndrome as a specific mental disease (kind), whose one of the ultimate constituents is the process giving rise to an extra copy of chromosome 21 (as the resulting *state*)).

10.1.2 Inherited vs. Acquired Diseases, and Congenital vs. Non-Congenital Diseases

In what sense can inherited vs. acquired diseases be a challenge to our definition of disease (cf. Box 9.3)?

First, as being commonly related to genetic diseases, inherited diseases pose the threat of how to strictly distinguish them from genetic diseases *simpliciter*, while genetic diseases are (already) taken as fundamental diseases (cf. Sec. 10.1.1).

Second, it is not at all obvious how essentialism about disease can interpret the notion "acquired disease" by contradistinction with "inherited disease" – for, how to make sense of acquired diseases within a picture sustaining to genetic determinism?

We can argue that an *inherited* (or innate) disease is a *specific genetic* disease viz. a genetic disease (cf. Sec. 10.1.1), whose mutation has been passed down from the parents (or the progenitors), while an *acquired* disease is a disease, whose constituents have *not* been passed down from the ancestors: e.g. half cases of neurofibromatosis are reported to be fundamentally constituted, or absolutely realized, by *acquired* bad genetic mutations i.e. bad genetic mutations, which have not been passed down from the ancestors (National Institute of Neurological Disorders and Stroke, 2020).

However, note that it is common to mistakenly equate inherited diseases with genetic diseases *tout court*, and acquired diseases with diseases, whoses causes are environmental.

A common argument which has been put forward in favor of this latter understanding of the inherited vs. acquired diseases distinction is based on, respectively, the so-called *nature vs. nurture divide* i.e. the debate about the acquisition of traits solely (pre)determined by one's genetic processes vs. the environment (cf. Kronfeldner, 2009; 2018; Maienschein, 2005; Tabery, 2014).

However, the nature vs. nurture divide is largely rejected in contemporary biology on the firm grounds that there are *gene-environment interactions* (however understood) (Oyama, 2000).

Therefore, the above understanding of the distinction between inherited and acquired diseases is to be rejected.

Gene-environment interactions may be deflationarily understood along the line that e.g. a certain genetic disease *kind* may very well be essentially an inherited (or acquired) genetic disease with an *environmental cause* (having fostered, promoted or stimulated the genetic disease) (and an effect); ditto, of course, for interactions at other (non-genetic) levels.

In that sense, a *weak* version of (genetic) preform(ation)ism leaving room for gene-environment interactions is tenable (cf. Morgan, 1901; also Oyama et al., 2001).

Furthermore, acquired vs. inherited diseases should be also distinguished from non-congenital vs. *congenital* (also mistakenly said *birth*) diseases, to which they are often conflated due to the mistaken association between inheritance (or innateness) and birth disease (cf. e.g. Kronfeldner, 2009; Stich, 1975).

Indeed, congenital diseases may be said specific diseases, whose specific *host* is an (indirect) intensional *part of a fetus* (cf. e.g. Behrmann & Avidan, 2005 on the specific host involved in congenital prosopagnosia), while an inherited or innate disease is a specific genetic disease present at the birth of e.g. a teenager, an adult, etc. – not necessarily a baby, of course (cf. Sec. 10.1.3).

10.1.3 Birth Diseases

If congenital diseases are specific diseases viz. diseases whose host is a part of a fetus (cf. Sec. 10.1.2), what are, thus, birth diseases? Are they not inherited diseases?

Birth diseases are diseases already present at the birth of a certain living being. Indeed, birth (or *ontogenesis*) – minimally understood as a certain process (e.g. an organism) (cf. Exc. 3.7) – is arguably, *as such*, not only about babies, but also about children, adults, teenagers, etc., in so far as these latter are all taken as (highly) specific living beings themselves.

Moreover, to the contrary of innate diseases, birth diseases are not to be restricted at the genetic level.

How to understand a living being born with e.g. baldness or without a hand, leg or arm (i.e. *amelia*), or even without a brain (i.e. *anencephaly*)? Can this living being be (indirectly) diseased from e.g. the hand, while this living being has never been (indirectly) diseased (in the sense of a disease process) *from the hand*?

What is the difference, or is there one, between a human being *born with* baldness - whose generation process of hair never took place (while it ought to), but was destroyed *from the start* -, and a human being having lost his hair e.g. during chemotherapy?

In other words, how to understand in a more detailed way birth diseases represents, thus, a big challenge to essentialism about disease (cf. Box 9.3).

A solution comes as follows: if it is true, indeed, that the process in question never took place (for the specific host in question, while the process may have happened for another one viz. e.g. (the part of) a fetus), it is not the case for a *higher-level* process i.e. that for a living being to be born with e.g. baldness just is for this living being to have only *partially* a higher-level healthy

process, where this healthy process is (partly) constituted by hair; or, for a living being to be born without a hand just is for this living being to have only partially this higher-level healthy process (partly) constituted by a hand; e.g. it is intuitive to say that a person born without well-functioning legs is a person (partly) unable, at a higher level, to *walk* (where, thus, well-functioning legs (partly) constitute, of course, what it is to walk) – which is not to be confused with the case, where someone e.g. could experience losing his legs due to a car accident, and then is (completely) unable to walk *any longer*; in this latter case, indeed, if we know all what happened to that person, then we would rather say that this person has just been diseased from his legs.

In that sense, the major difference between e.g. a human being born with baldness and a human being having lost his hair during chemotherapy lies in the fact that the former, to the contrary of the latter, never (indirectly) hosted the disease process leading to the complete loss of hair.

If there is no possible higher-level (started) *disease* process, then the host in question is just, actually, *dying*: e.g. maybe the case of an anencephalic living being, which is intuitively just a dying entity able to survive only a few hours or, at best, a couple of days (if we decide, of course, to treat death as *cerebral* death) (on death, cf. Exc. 9.1).

10.1.4 Cultural Diseases

Can essentialism about disease take into account cultural diseases, for cultural diseases seem prima facie to have, indeed, by being precisely cultural, no (universalizable) essence, or they seem to be diseases subjectively defined? How to coherently understand cultural (or better, *culture-bound*) diseases (as listed in the DSM-5) (APA, 2013; cf. also Hacking, 1998)?

Culture-bound diseases may be treated as specific diseases *tout court*; a minimal - some would say “skeptical” (Stegenga, 2018b) - understanding of culture-bound diseases (compatible with our largely *objectivist* (and highly *realistic*) metaphilosophy) (cf. Ch. 2) is the following one: a culture-bound disease just is a certain (higher-level) disease (like any other one) whose host is a certain behavior (or belief) (with a(n indirect) biological basis, of course), whose *origin* can be uniquely traced back to a certain homogenous (or non-heterogeneous) group (or community) i.e. that a (*type*) culture-bound disease just is a certain (type) disease, which (like any other type disease) can very well be, in principle, *widely* instantiated (or not) i.e. not necessarily by the community in question only – a culture-bound disease remains, thus, (plausibly) essentially definable and universally applicable (and not arbitrarily and vaguely delineated) (cf. Cooper,

2010), at least to the suitable hosts: e.g. (running) amok is a certain frenzy attack against a mass (of people) in a momentum of killing spree (i.e. a drift of a specific (male) honor), whose origin is widely accepted to be Malaysian or Indonesian (Gomez, 2006).
In that sense, even a cultural disease is not *defined* subjectively i.e. here with respect to cultural beliefs (however understood).
To the extent that we attempt at tracing back the origin of the disease (host) to a certain community, then it happens that either many (if not most) (higher-level) diseases, indeed, absolutely fall under culture-bound diseases (as understood here), or only very few (if any) diseases are really culture-bound, for some (said) culture-bound diseases are, actually, very similar (if not identical) to other (said) culture-bound diseases – which make them no longer so *culture*-bound, after all: e.g. (running) amok is said similar to another (said) culture-bound disease called "hwabyeong", whose origin is widely acknowledged to be Korean;
or, we fail to see in what sense couvade disease (sometimes said a cultural disease) i.e. for a soon-to-be-father to reproduce pregnancy can be somehow characteristic, at the origin, of a certain (restricted) community (cf. Stegenga, 2018a).
Along our minimal understanding of culture-bound diseases, the chief difficulty in avoiding either their potential trivialization or their over-restriction lies in the capacity to show that the disease in question has really a single cultural origin (otherwise, it is not cultural any more, of course): highly specific diseases based on religious or spiritual beliefs are certainly good candidates for being said cultural diseases: e.g., if one counts the belief in angels as irrational (and as genuine), then, to the extent that irrationality is considered as a disease, one can easily argue that believing in angels is a culture-bound disease, whose origin can be certainly traced back to the old Testament, and which has spread out of its community through time alongside the religious spreadth.

10.1.5 Latent Diseases

Latent diseases seem prima facie hard to be taken into account by our definition of disease (cf. Box 9.3), for talk about latency, in all its generality, is related to talk about dispositions, while essentialism about disease relies on the notion of a *categorical* essence (cf. Exc. 5.2); talk about latency is also used in situations where we talk about an incubation period i.e. a significant temporal gap between *x* and *y*, while essentialism about disease does not allow that.

Along the first use of the term "latency", a latent disease is like a dormant volcano, which shall be sooner or later active again; this use is peculiarly widespread in talk about *viral* latency, where a viral process is said present in a cell without that any replication activity is happening. Though this case may easily serve as a perfect example for a dispositional theory of disease, we can also very well interpret this example along our own non-dispositionalist theory of disease i.e. as showing that only a *part* of a certain disease is present viz., more specifically here for the case of a (bacterial) viral process, only the lysogenic cycle i.e. the integration of the viral DNA inside the host's DNA - and not also the lytic cycle i.e., roughly, the destruction of the host's DNA. Following that sense of "latency", a latent disease would be, thus, just a disease partly present.

Along another also widespread use of the term "latency", a latent disease is unlike a dormant volcano; rather, "(disease) latency" refers here to a *latent period* – sometimes used synonymously with the notion of an *incubation period* - i.e. the time elapsed between the presence of a (generally infectious) cause of a disease and the onset of a disease process.

How to interpret this? Are we (necessarily) talking about an *indirect* disease with respect to a certain cause (cf. Sec. 6.3.2)? We can argue that "latent disease" refers, in this latter use, to a certain *etiopathology* (cf. Box 6.2), where there is simply some temporal gap between a cause and a (caused) disease.

Essentialism about disease (cf. Box 9.3) is able, thus, to take into account latent diseases by making them either diseases partly present or not specific diseases at all, but specific etiopathologies i.e. by making them interpretable either way within our general framework about "disease" and cognates.

10.1.6 Chronic vs. Recurrent vs. Common Diseases

Is essentialism about disease *sufficient* for taking into account chronic diseases (cf. Box 9.3)? Indeed, if any disease process (with its host) is highly fine-grained with very likely brief-lasting processes, then how are we to understand chronic diseases?

First, *chronic* diseases (vs. *acute* diseases like seizures) are to be strictly distinguished from *recurrent* diseases, where recurrent diseases just are diseases present in a long period of time – not having (necessarily) a *long-lasting* process or course like, intuitively, for chronic diseases; in our terminology (cf. Sec. 3.2.6), a recurrent disease can be simply understood as a *type* disease having *tokens* - not necessarily many - present in a long period of time.

Second, recurrent diseases should be also nuanced from *common* vs. *rare* diseases, which are (plausibly) diseases whose type has *indirectly* many vs. few tokens *living beings* present at time t_0, from time t_0 to t_{+1} or during a longer period of time, such that a token living being has a high/little chance to indirectly fall under the type disease in question, where risk indicators are generally given by the Global Burden of Disease (WHO, 2017) – indeed, a rare type disease (like a rare genetic disease) can have many (to say the least) tokens themselves inside one token living being.

Essentialism about disease does not exclude chronic diseases, to the extent that chronic diseases just are diseases, whose destructive process is long-lasting; *higher-level* diseases are most likely good candidates for chronic diseases like CVDs; indeed, as being at a higher level, they take more time to develop, for they have many indirect constituents, and they are closer to the death of the entire living being fighting, thus, the most efficaciously possible against those diseases.

10.1.7 Imagination-Related Diseases

What about imagination-related diseases like counterfeiting to be diseased? Can essentialism about disease take them into account? It is not clear whether essentialism about disease has enough resources to take into consideration their complexity (cf. Box 9.3).

Merely *imagining* (or maybe even fantasizing) being diseased (or even, pretending to be diseased) i.e. having the (mere) belief (or desire, etc.)-*like* (subject to the will) that one is diseased is, of course, not itself a specific *disease*, for imagination (as a belief-like, desire-like, perception-like, or even maybe behavior-like, etc.), as such, is obviously not a specific disease (on imagination, cf. e.g. Currie & Ravenscroft, 2002; Kind, 2016) – albeit a lack of imagination (e.g. aphantasia with respect to mental images) is a certain disease.

First, imagining being diseased is to be strictly nuanced from *imagination-related diseases* like e.g. counterfeiting to be diseased (e.g. malingering), hallucinating (that e.g. one's own amputated limb still exists) (for a survey of the past attempts at explaining the phantom limb phenomenon, cf. Hill, 1999; on the peripheral and central neural mechanisms surrounding the phantom limb phenomenon, Subedi & Grossberg, 2011), or having an illusion viz. e.g. false pregnancy (or *pseudo*cyesis).

How to understand imagination-related diseases? There is no reason not to treat imagination-related diseases as specific higher-level *cognitive diseases* viz., more precisely, specific mental and affective diseases: e.g. illusions and hallucinations are directly related to malfunctioning

perception or *un*intentional perception-like (Strawson, 1970), while counterfeiting to be diseased is directly related to the act of making believe (or, behaving-like) that one is diseased, but *with a deceptive purpose* i.e. the malfunctioning pretense of being diseased (with its good purpose).

Second, (the mere *act* of) imagining being diseased must also be strictly distinguished from, *plausibly* speaking, *imaginary* (or imaginative) diseases themselves, which would be arguably *fictional*/fictive diseases.

But, if disease is a specific *natural* kind, then, on pain of a category mistake, there is no, strictly speaking, fictional disease or, more generally, artefactual disease.

Is it really the case for any specific disease? It seems so, for, albeit a (bad) human action can certainly be (indirectly) the *cause* of a disease, it is not at all clear that a type disease (kind), whose all tokens are *created* in a lab (e.g. a variant of the viral disease H5N1, but transmissible from human to human), shall not eventually have tokens spread outside the lab (without any human intervention).

Thus, those disease(s) (kinds) created in the lab turn out to be not (really) artefactual diseases, but perfectly natural ones (all things considered in a certain predictable period of time – otherwise, we could argue that with in(de)finite time nature shall be able to build on its own almost all what we considered to be (human) artifacts).

For other cases of (said) fictive or artefactual diseases (e.g. a diseased robot, a diseased Sherlock Holmes), which cannot be reduced to (natural) diseases, we shall simply deny that they are specific diseases at all, by arguing that the predicate "_is a disease" is not to be *truly* attributed to such cases (on artefactual kinds, cf. Exc. 2.7).

Imaginary diseases must be subtly distinguished, in turn, from, at a more global level, false, *fake* (or even factitious, artificial, or *non*-) diseases (e.g., historically, drapetomania), which are, indeed, not (specific) diseases at all, but are logically equivalent to healthy processes (by the law of double negation).

10.1.8 Deformities

As a case of disease, how to understand with essentialism about disease or, more largely, our neo-Aristotelian framework, a *de*formity (or *mal*formation) (cf. Box 9.3)? Indeed, as such, the term "deformity" or "malformation" (or even "dysmorphism") seems to refer to the destruction of a certain structure or *form*, and, as we have seen (cf. Box 3.1), a species/kind is the *mere*

conjunction of a genus and a differentia; there is no additional form or structure (on Aristotle's notion of deformities and monstrosities, cf. Connell, 2018).

A way out here still in accordance with essentialism about disease is to argue that deformities can be minimally or deflationarily understood as specific disease processes (with a very specific host) viz. destructions of the *relational properties* responsible for the *shape* of e.g. a certain organ: e.g. a congenital heart process defect is a certain *malformation* of the heart process or great vessels (cf. Sec. 10.1.2), in the sense that it is a disease preventing the heart process or the great vessels (of a fetus) to have the structure they ought to have i.e. that, more specifically for ventricular septal defect, this specific relational property, that is the ventricular septum (as a wall separating the left and right ventricles), is being destroyed.

10.1.9 Artefactual Diseases

Is essentialism about disease sufficient for taking into account diseases, which seem to be *essentially* based on artefacts (cf. Box 9.3)? E.g., how to take into account alcoholism i.e. taken as an addiction essentially based on something artefactual viz. alcohol? Or, diseases (partly) cured only (or, so it seems possible today) through artefacts viz. e.g. orthodontic functional appliances?

However, are there really diseases essentially based on artefacts? We may rightly doubt this: e.g., for alcoholism, the (indirect) constituents of the disease in question may be so refined, that alcoholism (as a higher-level disease) not refer, in any case, essentially to some artefacts, by arguing along the line that alcohol (or ethanol) is a perfectly natural chemical substance (e.g. as a residue of yeast metabolism), which truly triggers (indirectly) an addictive response in the dopaminergic neurons and the opioid brain receptors; ditto for other related cases of addiction like drug addiction.

The loophole does not consist here in trying to provide a *reductive* definition of artefactual diseases, but only to show that highly fine-grained definitions of so-called artefactual diseases are perfectly natural i.e. *not* artefactual, actually: e.g., with respect to the (indirect) constituents of alcoholism as seen above, alcoholism, as a *psychological* disease, shall not be defined any longer as referring to alcohol *as such*, but as the psychological impossibility to stop swallowing those chemical substances (mostly ethanol) (indirectly) constituting alcohol. In that sense, as surprising for a conclusion it is, alcoholism is not an addiction essentially based on this human product that is alcohol.

Other cases of so-called artefactual diseases can be treated along (almost) the same line: e.g., for the case of orthodontic functional appliances and related ones, they either merely reflect the limitation of our knowledge (e.g. an artefactual cardiac pacemaker), or they can be treated as mere *means* to get some healthy process, and, since a disease is not *essentially* related to cure (and its means) (cf. Sec. 10.3.1), then those means can very well be artefactual – indeed, they are very often so.

10.2 Controversial Diseases as Genuine Diseases

In addition to spend some time on the difficulties that our definition of disease may encounter (cf. Sec. 10.1; Box 9.3), it is also important to spend some time on how our analysis of disease or, more largely, our neo-Aristotelian framework may be illuminating to preculiar controversial cases of disease; we are interested here in those controversial cases, which essentialism about disease can especially illuminate, or which directly follow from essentialism about disease.
Sec. 10.2 is devoted to the analysis of some controversial cases of disease, which are treated – arguably - as *genuine* cases of disease.
In Sec. 10.2.1, I show that *vices* are truly to be (objectively) taken as genuine higher-level cases of disease, for which the living being is responsible, where the responsibility in question is analyzed as having to do with a bad intention.
In Sec. 10.2.2, I show that *bad personality/character traits* (or *temperaments*) are, indeed, specific higher-level diseases destroying good personality traits consisting in having continuous positive mental states/processes.

10.2.1 Vices

How to understand *vices* is a big topic, which can in itself constitute a single monograph – or even several ones. The controversiality of vices as diseases very likely lies in that they go far beyond the proper domain of biology; another reason for their controversiality as diseases also lies certainly in their relationship to morality and responsibility i.e. that they prima facie seem more related to bad inner traits of a person than to true diseases very often taken as something that the person is not responsible for.

Essentialism about disease can illuminate the case of vices in the following way: from a highly fine-grained differentiation of the disease host and a permissive neo-Aristotelian ontology (cf. Sec. 9.3.2; Exc. 3.5), it follows that there exist e.g. specific (positive or negative) acts for which the living being is held (directly) *responsible* or not i.e. that this act results (indirectly constitutively) from a (positive or negative) *intention* (as a genuine mental state) (Zhu & Buckareff, 2006), or any other (positive or negative) non-intentional mental (or affective) state (e.g. the mere belief/desire that *p*, or being manipulated in believing/desiring that *p*), respectively.

As a consequence of essentialism about disease, there is no reason why vices are not to be truly counted as diseases i.e. as destructions of a healthy process (cf. Box 9.3). For the case of vices, we can argue that a vice is a specific higher-level disease viz. the destructive process of a *positive intention* (or virtue): e.g. being sexually *addicted* is a vicious behavior resulting from a vice viz. a bad intention, which destroys the positive intention (or the virtue) to have an equilibrated sexuality by making it hyper-function (on the biological basis of sexual addiction, cf. Blum et al., 2012); by contrast, being sexually *dependent* is also a certain disease viz. a deviant behavior resulting – not from a vice (as a bad intention) -, but from an irrational desire (as a certain disease itself), which destroys the desire (over which one has *no control*) – not the (positive) intention - to have an equilibrated sexuality by making it hyper-function (against vices as specific mental diseases, cf. Sadler, 2008; 2013)[193].

By allowing higher-level diseases like vices to count as specific diseases per se, we address Rachel Cooper (2007)'s objection - targeted especially against Chris Megone (2000)'s account of disease (kinds) - that, generally, neo-Aristotelian accounts of disease (kinds) are over-inclusive by simply biting the bullet, actually: vices *are* specific diseases, albeit that much does not make talk about disease (kinds) trivial, as long as we are able to give, of course, at least

[193] Caveat on intentions and disease: is the dropping of a positive intention a disease? First of all, we can argue that, at the general level, an intention is unlike a mere desire, a weak will or a wish; a correct (i.e. realizable) intention to *p* may be understood as implying that an agent has to do anything necessary or in his power to achieve *p*. If someone drops a positive intention, then we can understand this point as meaning that, actually, this person never truly had a positive *intention*, but perhaps a mere positive desire or wish or even a hope – while dropping e.g. a positive wish is unlikely to count as a certain disease, in so far as a wish to *p* (like for other related entities) is intuitively not so tightly connected (to the contrary of an intention) to the ((non-)immediate) obtaining by whatever means *p*; rather, they seem more related to *attempts* (cf. Massin, 2014 on that).
Secondly, if there is a dropping of a *real* positive intention, then, indeed, we shall have to argue that there is something wrong with the positive intentional process going on (even e.g. in the case where someone merely positively intends to open a door) i.e. that it is a vice (e.g. for a person not to have her intention to open a door fulfilled is for her to be *epistemically* vicious).
However, we should not forget that there are certainly as many *specific* positive intentions on earth as there are *specific* human beings (as the bearers of positive intentions here) (cf. Sec. 3.2.6); many paths lead to virtue.
I acknowledge Ulrich Krohs (personal communication, April 2021) for this interesting question.

minimal criteria for distinguishing disease(s) (kinds) from non-disease(s) (kinds) (Charland, 2004; 2006).

However difficult it is for scientists to find out (indirect) constituents/absolute realizers for higher-level diseases – typically *neural bases* (and not mere correlate*s*) -, vices are also (indirectly) constituted or realized by somatic diseases.

Though situated at the level of intentions as genuine mental states, vices (as, generally, mental diseases) are also specific; as illustrated above, some vices are specific *social* diseases – still grounded on, or (indirectly) constituted by, somatic diseases (i.e. brute or non-social kinds), where a social disease may be very well taken as a specific natural kind – not an artefactual one -, as commonly taken (Hansson Wahlberg, 2014; Searle, 1995, for we may perhaps further distinguish between *social* kinds (e.g. carrying clothes) and *institutional* ones, where the latter only would be, indeed, specific artefactual kinds.

Other examples of social vices are the following: an insult (or offense) is arguably a vicious behavior resulting from a certain vice viz. the intention to *mis*attribute (a) specific bad predicate(s), or to (indirectly) (absolutely/relatively) *reduce* (a specific subject) *x* to (one) specific bad or good predicate(s) (or to (partially) *degrade* a specific (positive or negative) whole to (one of) its (indirect) (positive or negative) parts), or to *subjectively* attribute (a) specific bad predicate(s), which destroys the positive intention to objectively and truly attribute (an) (negative or positive) predicate(s)[194];

[194] Three caveats on offense: (i) we may want to argue that e.g. being offended/offensive *without* (*good*) *reasons* (or justifications) is, actually, a specific disease, but not just being offended/offensive.
However, there are never (good) reasons or justifications for e.g. being offended/offensive – for offense is not a *good* thing (or, offense is always *wrongful* or, more specifically, *arational* (as put by Hursthouse, 1991)) and *bad* reasons or justifications are arguably *no* reasons or justifications at all -, unless the notion of (good) reason or justification is, in a deflationary (but still plausible) way, equated with the mere notion of (causal or constitutive) *explanation* – which is quite often the case, especially with a certain (plausible) wide meaning of "rationality";
in other words, a (higher-level) disease like offense is never justified (or even *un*justified) or, more specifically *reason*able (rational, or even less *motivated*), although it is *explained* or grounded in a certain way, or fitting viz. through *negative* causes/constituents: e.g., generally speaking, a mental disease can be (plausibly) explained by the idea that it is a *normal*, appropriate or legitimate reaction to a stressful environment (Horwitz, 2002), or maybe it can be even sometimes excused (or forgiven) or tolerated, or, to the contrary, punished.
We could even go further by arguing, along a neo-Davidsonian (1963) line (broadly understood), that reasons (for an act(ion)) or justifications (of a process) are, indeed, *specific* causes (or constituents) (of an action or a process) viz., in our case, only *positive* ones.
(ii) *Conflating* (willingly) *mere explanations* or grounds (plausibly taken) (i.e. good or bad ones) with *justifications* or *reasons* – which are, actually, only *specific* explanations viz. good ones or of something good - is a powerful psychological tool to make appear to oneself evil/bad act(ion)s (or just processes) as, actually, (morally) good ones.
(iii) Nevertheless, if e.g. offense without any (good) reason is deflationarily understood i.e. that offense is, strictly speaking, *unexplained* (or inappropriate) i.e. where none of its correctness conditions is present, then the predicate "_is offended/offensive" is simply not present.
But, if "offense" is still referred to in such circumstances, then the predicate "_is offended/offensive" is, actually, itself *mis*attributed.

discrimination (as commonly taken) is a vicious act resulting from a certain vice viz. the intention to (unjustifiedly) grant *un*equal rights to *x* (or, more generally, consider *x* less well) compared to *y* (mostly, reductively) grounded on the idea that *x* is (plausibly if not correctly) *ab*normal and *y* normal (taken in a *subjective* way), which destroys the positive intention to grant equal rights to *x* and *y* (or consider *x* as well as *y*), where *x* and *y* are merely distinguished, respectively, as (plausibly if not correctly) *objectively* abnormal and normal (European Commission against Racism and Intolerance, 2020).

Other vices can also be found at the *epistemic* level: e.g. a lie results from a vice viz. the bad intention to violate a sincerity norm by presenting *p* (as true) or asserting that *p* (is true), while believing that ¬*p* (is - more likely - the case) (on lie (and (self-)deception, cf. Engel, 1995; Fallis, 2009; Mahon, 2015; Marsili, 2019)[195].

10.2.2 Bad Personality/Character Traits or Temperaments

It is common to hear someone aggressive often saying that (s)he needs no cure for his/her aggressiveness, because (s)he judges him/herself not diseased, but that aggressivity is just a much anchored trait of his/her *personality* or *character*, or is part of his/her *temperament*[196], however bad it is (and aware of this badness the person can be).

[195] Caveat on pathological lie and other related cases (like morbid jealousy or curiosity): if a lie (or jealousy or curiosity) is already by itself a certain higher-level mental disease viz. a vice, then what is a *pathological* lie (or *morbid* jealousy/curiosity)? Is it like to redundantly talk about diseased psychopathy, where psychopathy is already itself a disease (or about a healthy heart process) i.e. just about a negative entity *hosted by* a negative property? Or, is it a question of *intensity*?

We are used to distinguish, indeed, between a lie and a pathological/compulsive lie (or jealousy and pathological jealousy, or curiosity (vs. *interest* – which is positive) and pathological curiosity), etc.

According to me, this distinction does not reveal that e.g. a lie (or jealousy/curiosity) *tout court* is *not* to be counted as a specific disease – by contrast with a *pathological* lie (or *morbid* jealousy/curiosity) -, but it can be interpreted in a two-fold way: first, talk about a pathological lie (or morbid jealousy/curiosity) rather than lie (or jealousy/curiosity) *simpliciter* in a certain context is just to insist upon the *severity* of a (pathological) lie (or jealousy/curiosity) by (intra-)comparison with a lie (or jealousy/curiosity) *tout court* (cf. Sec. 9.4.3);

second, the distinction between a lie (or jealousy/curiosity) and a pathological lie (or morbid jealousy/curiosity) can also indicate that, indeed, a lie (or jealousy/curiosity) is a *vice*, while a pathological/compulsive lie (or morbid jealousy/curiosity), by being precisely compulsive (or non-intentional), is not a vice, but another (mental) disease.

[196] Terminological note on temperaments: classically, some psychologists (and philosophers) strictly distinguish between personality/character traits and temperaments, by arguing that there is an *innate* (and dispostional) structure with temperaments (taken as psychological tendencies) (Thomas et al., 1968).

However, without entering into the details of this complex debate, we fail to see in what sense the classical studies on babies have shown that, to the contrary of temperaments, personality/character traits are *not* (partly) innate.

The controversiality of bad character traits/temperaments as diseases lies precisely in the *innerness* or anchoreness of a personality/temperament within a living being, which would make it something on which physicians cannot intervene (cf. Sec. 10.3.1).

A direct consequence of essentialism about disease is that there is no formal reason that bad personality traits/temperaments should not also count as specific diseases themselves (cf. Box 9.3), which are also (indirectly) somatically realized/constituted (on neurotransmitter imbalances in neurobehavioral regulatory systems as biological bases for bad character traits/temperaments, cf. Sulis, 2020; Trofimova & Sulis, 2018).

Indeed, there is no reason why good personality/character traits or temperaments cannot play the role of the disease host as a higher-level part of a living being i.e. that a good *personality* (in all its generality) is intuitively a higer-level part of a certain living being (e.g. a part of the human being's essence), while a bad personality is the destruction of what it is to have a good personality.

What constitutes a good personality is widely held, in the philosophical literature (Olson, 2019), to be those positive psychological states/processes (i.e. *traits*) that are continuously present (within a single living being) i.e. *recurrent* positive mental states/processes, which are absolutely realized in different ways: e.g. to be humble is certainly to have a certain good personality trait, while to be ambitious is also certainly to have another specific good personality trait.

If a bad personality trait/temperament is the destruction of a good personality trait, then e.g. aggressiveness, as a bad personality trait, is, indeed, this inner psychological tendency or this recurrent psychological trait, which destroys one's own good character trait consisting in continuously desiring peaceful social interactions.

10.3 Controversial Diseases as Non-Diseases

While Sec. 10.2 is about some controversial diseases (directly following from essentialism about disease) (cf. Box 9.3), which are to be arguably counted as genuine cases of disease, to the contrary, Sec. 10.3 is about some controversial diseases to be counted *not* as genuine cases of disease.

Devoting Sec. 10.3 to that is also essential for avoiding any threat of *trivialization* of essentialism about disease.

Several of non-genuine cases of disease – on which we shall not come back here - have already been (briefly) highlighted in the due course of this PhD dissertation: e.g. drapetomania (cf. Sec. 8.2.3).

In Sec. 10.3, I review other controversial non-genuine cases of disease as follows: in Sec. 10.3.1, I show that *incurable diseases* are not specific diseases, for no specific disease is such that it must be cured.

In Sec. 10.3.2, I argue that *aging*, when minimally understood for a type process of a living being to go through time, is not to be taken as a specific disease.

10.3.1 Incurable Diseases

Are incurable diseases genuine diseases? Above all, an incurable disease is *not* a certain disease *kind* i.e. a disease whose effects and causes cannot be suppressed (cf. Box 4.2; *contra* Boorse, 1977), for nothing in the term "incurable disease" makes directly reference to the negative causes and/or effects of disease, but only to a certain disease process, which cannot be cured, or whose destructive process cannot be stopped by any possible intervention. Is there such a disease?

The controversiality over incurable diseases as genuine diseases or not very likely lies in the fact that we relate the notion of a disease process, which is *independent* from the human hand, with human capability to intervene on it.

However, first, if there were incurable diseases, then this would also suggest that there are *curable diseases*. As a direct consequence of essentialism about disease (cf. Box 9.3), no disease can ever be *essentially* something on which physicians can(not) intervene such that to cure it - or even, just something needed or worth to be cured (but which cannot be so) -, or a disease is not essentially a certain attitude consisting in curing (or healthcare) (but which cannot be so): *x* is not diseased, because there is someone judging that *x* needs to be cured (though it cannot be so).

More generally, negativity is not *essentially* something we want to get rid of or to eliminate, or something which we want to avoid, or which is undesirable.

Second, if there were incurable (and curable) diseases, they would also probably exist some diseases, on which physicians can intervene (such that to cure them), but do not do so: e.g. a benign heart murmur. If so, then where to non-arbitrarily and –vaguely put the boundaries between curable diseases and those latter ones?

Taking incurable diseases as specific diseases is confusing issues about the nature of disease with issues about the attitudinal reactions that we have towards disease, or, more generally, *metaphysical* issues with *practical* ones (cf. Cooper, 2002; Stegenga, 2015); that much does not imply, of course, that studying the *nature* of those attitudes is worthless or *un*philosophical, it only means that such studies shall also turn out to be, actually, somehow metaphysical – and not practical.

However, if no disease is ever incurable in the sense of being essentially something that physicians cannot cure by intervening on it, as we have already seen (cf. Exc. 2.8), there is still an indirect *necessitation* relationship between a disease and the corresponding intention (or just the hope) to cure or other con-attitudes, though not every disease is to be put, of course, on the same level of *healing emergency* (cf. Sec. 9.4.3), and such desires or preferences may also very well be absent: e.g. *x* lacks the (positive) effort to correct *x*'s flaws.

Thus, we are also not saying, at the other extreme, that diseased people should just stay content with their own fate, should be fatalist, should resign themselves, should not complain, or should not even want to get cured.

10.3.2 Aging

As a central concept with its own growing body of literature (cf. e.g. Scarre, 2016), a PhD dissertation on the concept of disease cannot but spend some time on the concept of aging.

The controversiality over aging as a genuine disease or not lies precisely in the unclarity behind the concept of aging: e.g. while one judges, on the one hand, that aging is natural (in a certain narrow sense), one also judges, on the other hand, that aging as mental and physical decline (i.e. senility) is a certain disease (De Winter, 2015).

Along with essentialism about disease (cf. Box 9.3), we fail to see in what sense there is something bad with aging i.e. in what sense aging, as such, can be the destruction of a healthy process.

Indeed, "aging" (or "becoming old") only means "passing from a certain age to another one" i.e., more precisely here in a dynamic framework (cf. Sec. 9.2.1 on processes), for (negative or positive) type processes (of a type living being) to (go on to) exist through a certain stretch of time (*pace* Fuellen et al., 2019), whose existence is constantly renewed by the presence of tokens (for a (type) process cannot *endure* through time) (*pace* Steward, 2013); if minimally understood as such, then aging (or becoming old) is neither positive nor negative.

If it is true that, with respect to a higher-level process called “humanity”, we are used to distinguish between different “ages” viz. (human) new borns, babies, children, teenagers, adults, and elderlies, in the minimal sense here that one and the same process viz. “humanity” just goes through (a certain stretch of) time, then we shall just take e.g. childhood or adulthood as a mere absolute realizer of “humanity”; in other words, children, adults, elderlies, etc., are just all different *specific* (healthy) human beings on their own with, thus, their own diseases too: e.g. a certain dementia can happen to elderlies, but another one can also happen to teenagers, etc.

A consequence of decoupling aging from the idea of being more and more diseased is that there is absolutely no necessity for passing from e.g. childhood to adulthood and so on (cf. Sec. 10.1.3 for the same idea about the fetus); it is *contingent*, thus, that there is a passage from e.g. a fetus to a baby.

However, how to understand the intuitive idea that aging is related to mental and physical decline? “Humanity” has not merely “new borns”, etc., as absolute realizers themselves (with their own diseases), but also any specific human being passing, indeed, from a new born until an elderly through an adult, etc.

Though passing through the ages is not itself a specific disease – for, it is *not* because the longer an organism lives, that, as such, the more likely it shall be, in principle, diseased -, mental and physical decline (associated to the idea of aging as a disease) can be simply understood as a *chronic* disease i.e. a disease with a (very) long-lasting destructive process (cf. Sec. 10.1.6), whose host is this specific human being passing through the ages.

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CONCLUSION

We are all diseased – to a certain degree and at a certain level. What is it to say? Is it to say that some biological part of us is just malfunctioning in a certain way? Or, that we are just in a situation evaluated as bad in a specific way? Any analysis of the concept of disease is *complex*, for we are obviously unclear with what DISEASE is.

How is DISEASE related to the concept of biological malfunction (taken in a specific way)? Or, to the concept of a situation evaluated as bad in a specific way? Any analysis of the concept of disease is *significant*, for the concept of disease intersects with other complex concepts.

The immense literature on the concept of disease starting from the Hippocratic theory of disease as imbalance between the four humors to (until now) our own contribution to this debate testifies to the importance of the concept of disease.

Facing this immensity, the present PhD dissertation follows a very simple structure, which consists mostly in the *positive* line of defense of an original theory of disease. In the image of the specific mereology defended throughout this PhD dissertation (cf. Ch. 2), the synoptic structure is meant to stand as a *single whole*, which is not the sum of its parts, but still with chapters with their own major point(s) understandable (minimally) *independently* from each other or self-contained (cf. Fig. 11.1).

The original theory of disease defended is called "essentialism about disease": x is a disease, iff x is a negative process of a part of a living being (i.e. the genus), and x is the destruction of a positive state p until the negative state $\neg p$ (i.e. the differentia) (cf. Box 9.3).

In other words, a disease is the destructive process of the essence of a processual part of a living being; that to be diseased is to have a malfunctioning biological part, as well as that to be diseased is to be in a situation evaluated as bad in a specific way are both analyzed through the idea of a destructive process of the essence of a processual part of a living being (cf. Box 9.5-9.6).

To reach this conclusion about the nature of disease, we have come a long way. Above all, with this controversial topic with its huge history that is disease, we have to be clear about *how* to make an inquiry into the concept of disease. Part I of the present PhD thesis argues for a certain meta-philosophy.

In Ch. 2, we defend, first, the universal meta-philosophical position (at least, to all the suitable entities) that to define the predicate *D* is to find out those conditions necessarily (modally speaking) equivalent to the sentence "*x* is *D*", where those conditions are the (intransitive) *intensional*, *constitutive* or *essential* parts (independent from each other) of a numerically distinct whole, which is *D* (cf. Def_4); second, we defend the *correspondence principle*, according to which defining the predicate e.g. "_is a disease" is equivalent to defining the concept DISEASE or disease as another specific existent entity i.e. that an inquiry into the nature of disease (as a specific ontological unit) turns out to be at the same time a *conceptual* analysis of DISEASE as well as a *semantic* analysis of "disease".

Through an investigation into other necessitation relationships (than the one of definition) viz. the one that a whole (and a kind) must be necessarily – also indirectly *empirically* - realized in a specific form i.e. through the subscription of a minimally practice-oriented meta-philosophy (or a liberal naturalism) (cf. Exc. 2.1; 3.5), Ch. 3 is already a specification of the meta-philosophy argued for in Ch. 2. Through the preliminary distinction between disease and disease kinds, we argue, first, that disease is ontologically and definitionally prior to disease kinds, in so far as disease is the *genus* under which a disease kind as a (*natural*) *species* falls – where *values* (like disease (kind)), generally, are taken as natural or objective/mind-independent kinds (cf. Exc. 2.7).

More generally, we understand a (natural or artefactual i.e. subjective/mind-dependent) species or *kind* as a whole numerically distinct from its genus (proximum) and differentia, which are its intensional or constitutive parts (cf. Box 3.1); this conception of the genus-(natural) species hierarchy is defended against numerous objections, amongst which the one *reducing* natural kinds to specific artefactual entities viz. epistemic entities (e.g. the meaning of "disease" is its use) (cf. Sec. 3.2.4) – we counter-argue that there are necessarily natural kinds (like disease

(kinds)) -, and that a kind is an *evolving* kind (cf. Sec. 3.2.6) – we counter-argue that evolution (as substantial change) does not rule out that processes (as substantial changes themselves) have essences (like genera and differentiae). Second, although we argue for a very permissive neo-Aristotelian ontology leaving room for many different specific *irreducible* entities highly *fine-grainedly* differentiated like (natural and artefactual) kinds and sets (cf. Exc. 3.5; 3.9), not all of these entities – only kinds/species - are amenable to a proper definition, or are part of a (universal) meta-philosophy, for some of them are *trivial* entities (like sets); we defend that a genus, as a fundamental entity, is a constitutive part of a species – thus, a genus cannot be considered as a kind/species itself (cf. Box 3.1.1); furthermore, as being apart from our neo-Aristotelian ontology, we propose a reductive definition of a differentia, according to which a differentia (or differentiating characteristics) reduces to merely either a kind or a genus (cf. Box 3.1.2), where a specific differentia, along the Thomistic tradition, is *defined independently* from the specific genus with which it is part of a certain species. In that sense, as an implementation of our own meta-philosophy, a species/kind is truly a whole numerically distinct from its essential parts i.e. a genus and a differentia, which are merely two conjuncts (independent from each other).

Once we are clear about how to proceed for studying the concept of disease, Part II of this PhD dissertation begins to *apply* more directly our philosophical methodology of Part I to find out the essence of disease. However, like with any other highly complex concept, it is fruitful to start with the concepts DISEASE is embedded in (or surrounded by), and with what DISEASE is not, as well as those concepts easily confused with DISEASE.

In Ch. 4, we develop, first, a theory of disease *kinds*, according to which a disease kind is constituted by a disease (i.e. the genus) plus a negative cause related to a living being and a negative effect related to a living being (i.e. the differentia) (cf. Box 4.2), however plausibly or correctly those constituents are taken; as the differentia (defined independently from the genus in question), the negative causes and effects are what we take to be *nosological criteria*; many diseases defined in the medical literature are certainly more likely to be called “disease kinds” along our line of thought. Second, this theory of disease kinds is defended against multiple objections, amongst which the (weak) *adaptationist* claim that a disease is followed by positive effects (cf. Sec. 4.2.2) – we counter-argue, along the Weak Conservation Principle (WCP), that a disease (as always a bad *tout court*, and not also a *good for* something else) concurrently implies something good (and vice versa) (cf. Sec. 9.4.3 on Moorean monism) -, and a famous *functionalist* interpretation of disease, where causes and effects are the differentia for disease, not for disease kinds (cf. Sec. 4.2.7) - we reply here that a functionalist interpretation of disease

confuses the metaphysics of disease with its *epistemology*, where finding out the causes and effects of a certain disease are certainly a good way for knowing this disease (by circumscribing it), but they are not *what* this disease is.

Ch. 5 and 6 are devoted to analyzing more precisely, and respectively, what a negative effect of disease and a negative cause of disease are. In Ch. 5, we argue, first, that a negative effect or sign of a disease (or *symptom*) is a disease (i.e. the genus) in conjunction with a directly temporally following negative entity related to a living being (i.e. the differentia) (cf. Box 5.2) i.e., plausibly reinterpreted, that a disease (however plausibly or correctly understood) and what temporally follows it both constitute a single complex whole (as a state or process/mechanism); an interesting case study is here certainly Parkinson-plus syndrome; we defend this conception of symptoms against many objections, amongst which that symptoms are manifestations of a disease (cf. Sec. 5.3.2) – we counter-argue that we can dispense with *dispositions* (as ontological units), for they are encumbrant entities. Second, we defend the idea that felt and unfelt symptoms are *specific* symptoms themselves; at a more general level, we neatly separate bad feelings from bad emotions and mental states/processes i.e. that the dichotomy feelings vs. unfeelings does *not* correspond to the dichotomy mind vs. body.

Along the same line of thought, Ch. 6 is about the causes of disease or the *etiopathology* (or etiopathological agent). An etiopathology is a disease (i.e. the genus) plus a directly temporally preceding negative entity related to a living being (i.e. the differentia) (cf. Box 6.2) (however plausibly or correctly understood); an interesting case study is here HIV-AIDS, where, plausibly reinterpreted, HIV-AIDS is a whole constituted (or underlied) by AIDS and directly temporally preceded by HIV infection. Furthermore, we relate our own theory of medical causes to the general *mechanistic* theory of causation developed by Stuart Glennan (1996; 2002), according to which, roughly, an event *c* situated at the non-fundamental level causes an event *e* situated at the non-fundamental level, iff there is a(n) (underlying) mechanism *m*, which connects *c* to *e*. Finally, we argue that *multiple causes* of disease are to be simply understood as the multiple constituents of an etiopathological agent, while *indirect causes* of disease are just those causes causing a cause of a disease.

In Ch. 7, we continue our analysis of diverse concepts related to the one of disease or sometimes even confounded with the concept of disease. Ch. 7 is devoted, more precisely, to those entities, which we take to be, not things that are *more* than disease (cf. Ch. 5-6), but *specific higher-level* diseases themselves, which, under the assumption that they are genuine, are still (indirectly) absolutely realized by somatic diseases (cf. Exc. 7.2). First, *illness* is analyzed as the feeling of a disease (cf. Box 7.1), where, by following even for higher-level diseases the

core tenet of the (*bio*)*medical model*, according to which any objective deviation from the (biological) normal is a disease (cf. Exc. 7.1), feeling *x* (or being aware of *x*) is further objectively analyzed as an attention mechanism directed towards *x* (cf. Sec. 7.1.2-7.1.3); if illness is a specific disease, then disease is not essentially related to bad feelings. Second, for *x* to have a *sickness* is analyzed as for *x* to have a suffering (cf. Box 7.2). Third, against the idea that *disability* is a certain value subjectively defined as a con-attitude or against the idea of a (direct) correlation between disability and negative attitudes that people have towards disabled persons – e.g. discriminatory attitudes are to be counted as specific higher-level diseases themselves viz. vices (cf. Sec. 10.2.1) -, we argue that disability is to be prima facie defined as the feeling of suffering (cf. Box 7.3). Fourth, as an important concept on its own especially in bioethical debates, the concept of *harm* is analyzed here as a strong suffering (cf. Box 7.4). Finally, we give reasons why some concepts, amongst which UNHEALTHINESS and MORBIDITY, rather than others, amidst which DISORDER (as a mere normative concept) and PATHOLOGY (as a positive concept too), can be interchangeably used with DISEASE.

Part III of the present PhD dissertation is devoted to analyzing directly the concept of disease. Ch. 8 is meant to situate our own definition of disease within a certain debate around the concept of disease. As a framework for thinking the debate around disease, we can distinguish between three groups of theories of disease: *axiologism* about disease, *malfunctionalism* about disease, and *hybridism* about disease; these three groups of theories of disease are distinguished with respect to the emphasis the theories of disease they include put on one or both of the two (main) intuitions that we have about what disease is. The first main intuition (1) that we have about disease is the following: saying that e.g. cell growth is cancerous is making a certain negative value judgement toward cell growth, where the value in question is a specific *lethal value* (like death) (cf. Box 8.1); the second main intuition (2) that we have about disease is that saying that cell growth is cancerous is saying that cell growth is *biologically malfunctioning* in a specific way (cf. Box 8.2); axiologism about disease is a group of theories of disease, which put the emphasis on intuition (1), while somehow explaining away intuition (2); malfunctionalism about disease contains theories of disease seriously accounting for intuition (2), while explaining away intuition (1); hybridism about disease includes theories of disease seriously based on both intuitions (1) and (2). A major motivation for subscribing to axiologism or malfunctionalism about disease, instead of hybridism about disease, is that it is highly common to take disease either (as a lethal value) to be normative in the sense of *subjective* or (as a biological malfunction) to be natural in the sense of *objective*. However, hybridism about disease is not to be taken prima facie as a contradictory thesis; to the contrary, it comes with

the advantage of seriously taking into account intuitions (1) and (2), however difficult this task is. Furthermore, Ch. 8 briefly illustrates and criticizes the most well-known theories of disease belonging to our three groups. As a theory belonging to axiologism about disease, Nordenfelt (1995; 2000)'s subjectivist theory, according to which, disease is the unpleasant state preventing a subject to accomplish his vital goals, which make the overall organism happy in the sense of having a minimally decent life, suffers from several objections, among which the one that this theory is too broad (cf. Sec. 8.2.1). As a famous theory belonging to malfunctionalism about disease, Boorse (1977; 1997)'s Bio-Stasticical Theory (BST) of disease, according to which disease as a value-free entity is *x*'s biologically malfunctioning is *x*'s deviating from its normal biological functioning i.e. a statistically typical contribution of *x* (within a reference class) to the inclusive fitness of the organism of which *x* is a part, can be also objected on grounds, amidst others, that the choice of the reference class is arbitrary (cf. Sec. 8.2.2). Finally, as a hybrid theory of disease, Wakefield (1992)'s harm-malfunction theory that disease is both a harm and a certain biological malfunction (as a naturally selected effect) is also not exempt from objections, amongst which that the notion of harm (taken subjectively) is not essential for a disease to happen (cf. Sec. 8.2.3).

Ch. 9 defends an original *hybrid* theory of disease – at least, prima facie so, for our framework must be eventually rejected on basis of the inseparability between intuitions (1) and (2); we see, indeed, no a priori reason why either intuition (1) or (2) should be explained away in favor of the other one; our theory of disease is called "essentialism about disease", according to which, roughly, disease is the destructive process of the essence of a processual part of a living being (cf. Box 9.1-9.2); put in a slogan, to be *diseased* is for a processual part of a living being to be *deceasing*. To get this definition, we consider into details two case studies: phenylketonuria (PKU) and (lung) cancer (cf. Sec. 9.1.1); PKU is the disease *destroying* in a specific way the *essence* of the process of phenylalanine hydroxylation (PAH-tion) (or its biological function) i.e. what PAH-tion (as a processual part of a living being) does i.e. converting the amino acid phenylalanine into the amino acid tyrosine by making tyrosine decrease; (lung) cancer is a specific way of destroying the essence of cell growth (of the lung's tissues) (as intuitively a *healthy process*) i.e. dividing into two daughter cells and grouping them (as its biological function) by making it *hyper*function i.e. through an uncontrolled cell proliferation. How does essentialism about disease take into account *coherently* both intuitions (1) and (2) about disease? Intutions (1) and (2) are taken as two sides of the same coin (cf. Fig. 9.4): the notion of a lethal value associated to disease is to be objectively understood as the negation or destruction of a certain vital value viz. *health*, which is the essence of a processual part of a

living being (cf. Box 9.4-9.5), where life just is the value grounding the existence of a certain bearer (e.g. a living human being just is an existing human being, exactly like a healthy PAH-tion or cell growth just is an existing PAH-tion or cell growth) through its having a specific *essence* like homeostasis, self-reproduction, or metabolism; from this, disease is the absence of health – not the opposite. The notion of a specific biological malfunction is captured by our definition of disease (cf. Box 9.3) through the idea that disease is the destruction of a certain biological function (cf. Fig. 9.3), which is intuitively the essence of a processual part of a *living* being, or is a healthy process (cf. Box 9.6): e.g. what PAH-tion does or what cell growth does is taken as its biological function i.e., where PAH-tion or cell growth is a processual part of a living being, its essence. The remainder of Ch. 9 focuses more thoroughly, first, on the notion of a *disease host* i.e. what is diseased. Disease is this specific negative property, which is the negation of a *specific positive* property viz. the property of being *healthy* or the property of being a biological function of something *good*; the relationship between disease and what is diseased is to be envisaged as the relationship between a host and a specific negative property – thus, *being diseased* is not an accidental property of a certain positive bearer (like PAH-tion or cell growth) (cf. Sec. 9.3.1). Why is the disease host a *processual* part of a living being? Disease (like its host) is intuitively a certain process, where a process is prima facie analyzed as the *generation*/*destruction* of (the essence of) a (positive or negative or value-free) *state* $(\neg)x$ at time t_{+1} from its contradictory state $(\neg)x$ at time t_0 (cf. Box 9.2): e.g. PAH-tion is a process, for it is the change from the *state* where tyrosine is *not* present to the *state* where tyrosine is present (the contrary for PKU); cell growth is a process, because cell growth is the change from the state of non-two daughter cells divided and grouped to the state of two daughter cells divided and grouped (the contrary for cancer). Why is the disease host a processual *part* of a living being? As being another lethal value, *death* (as a thick value) is intuitively distinguished from disease with respect to its specific host: only a whole living being can be said dead/dying, while only a (highly fine-grainedly diffentiated) part of it can be said diseased (cf. Exc. 9.1). Second, Ch. 9 focuses on the idea of disease as an *essence destruction*. We argue that there are basically two ways for the essence of a process to be destroyed: through *hypo*-functioning or *hyper*-functioning (cf. Sec. 9.4.2): e.g. PKU is the destruction of PAH-tion by making PAH-tion hypo-function through subtracting its intensional parts i.e. making tyrosine absent (until complete destruction); cancer is the destruction of cell growth by making cell growth hyper-function through adding other intensional parts to it i.e. producing an uncontrolled cell proliferation (until complete destruction). Finally, Ch. 9 spends some time on the idea that disease is not only a value *polarly* opposed to health (or the biological function of something good), but it is also

a *scalar* value i.e. a value with degrees (cf. Sec. 9.4.3); the severity of a disease is analyzed along the line that disease degrees express the different *temporal parts* or the progression of the disease process (as a whole): e.g. to be a bit/very much diseased from PKU or (lung) cancer is for PKU or (lung) cancer to have its destruction process a little bit/almost finished; from this idea (and of *death* severity), we distinguish between disease *intra*- and *inter*-comparisons: a disease intra-comparison is a measure of the progress of the destruction process of (at least) two tokens of a type disease, while a disease inter-comparison is the measurement of (at least) two different diseases belonging indirectly the same living being, where the severity of the disease is judged with respect to the level (of intension) at which it is situated.

As a last chapter of the present PhD dissertation, in an effort to variously illustrate and empirically ground our definition of disease, Ch. 10 considers the extent to which essentialism about disease is applicable. First, we study potential counter-examples to essentialism about disease. If our permissive neo-Aristotelian ontology allows for the existence of fundamental entities, which cannot be defined constitutively, then there are also certainly *fundamental diseases*; we argue that *genetic diseases* are those fundamental diseases, which merely destroy genetic processes, where a genetic process is not constitutively defined, but *teleologically* i.e. as the coding for a trait. Following an anti-reductionist line, we reject *genetic reductionism*, but subscribe to a certain form of *genetic determinism* i.e. the idea that all diseases (or healthy processes) are fundamentally but irreducibly realized or constituted by genetic diseases (or genes) (cf. Sec. 10.1.1), where a genetic disease may very well have an environmental cause. As other potential counter-examples to essentialism about disease, we pass under review the following cases: *inherited diseases* are genetic diseases, whose mutation has been passed from the ancestors, while *acquired diseases* are genetic diseases, whose mutation has *not* been passed down from the ancestors; *congenital diseases* are those diseases whose host is a part of a fetus (cf. Sec. 10.1.2); *being born* without e.g. well-functioning legs is, at a higher-level, to be prevented from walking (where, well-functioning legs (partly) constitute what it is to walk) (cf. Sec. 10.1.3); *cultural diseases* are specific higher-level diseases (like any other one) whose host is a certain belief (or behavior), and whose *origin* can be traced back to a certain homogenous group (cf. Sec. 10.1.4); *latent diseases* are either diseases partly present or specific etiopathologies (cf. Sec. 10.1.5); *chronic* diseases are just to be taken as long-lasting disease processes, by contrast with *recurrent* or *common* diseases (cf. Sec. 10.1.6); *imagination-related* diseases are specific cognitive diseases, by contradistinction with imaginary diseases (cf. Sec. 10.1.7); *deformities* are destructions of relations (taken as complex kinds) (cf. Sec. 10.1.8; also 4.2.6); there are no *artefactual diseases* (properly understood) (cf. Sec. 10.1.9).

Second, we show how essentialism about disease sheds new light on controversial cases of disease directly following from our definition of disease: from a highly fine-grained differentiation of the disease host, we see no reason why *vices* – whose controversiality as a disease lies in the notion of *responsibility* associated to it - taken as bad intentions destroying positive intentions (or the virtue) to do something cannot be counted as specific higher-level diseases themselves (cf. Sec. 10.2.1); ditto for the case of bad personality/character traits or temperaments – whose controversiality as disease lies in the *innerness* associated to them -, which can very well be also treated as genuine higher-level diseases destroying good personality traits consisting in having continuous positive mental states/processes (cf. Sec. 10.2.2). Finally, to rule out any threat of trivialization, we reject the controversial case of *incurable diseases* as genuine diseases – whose controversiality lies in the *human dependency*: as a consequence of essentialism about disease, no disease is ever essentially something on which physicians cannot intervene such that to cure it, or something needed to be cured (but which cannot be so); a disease is not essentially a certain attitude consisting in curing (but which cannot be so) (cf. Sec. 10.3.1). Along with an ontology of processes, we fail to see in what sense *aging* - whose controversiality lies in its dual aspect as both something natural and a disease - can be a certain disease, for "aging" merely refers to the passing through the ages i.e. passing through time; the idea of mental and physical decline associated to aging (as a disease) can be simply understood as a chronic disease i.e. a (very) long-lasting disease process (cf. Sec. 10.3.2).

In the image of the part-whole theory defended in Ch. 2-3, where a whole is numerically distinct from its parts, which are independent from each other, we have constantly striven to provide the reader with independent reasons or *evidence* to sustain to, or reject, a certain theory, though the diverse theories argued for are not, of course, *irrefutable* (along the Maximal Compatibility Principle (MCP)), according to which for a theory to be correct is for it to be compatible with a maximal number of plausible objections); although each chapter can be read minimally independently from each other (e.g. our theory of symptoms or etiopathological agents can be read autonomously from our theory of disease; or even, though embedded into our essentialist picture of disease, our theory of vices can also be read somehow in an independent fashion; ditto for our theory of a process; etc.), a way to make those theories as correct as possible is to show their *coherence* within a whole system presented i.e. that each chapter is also meant to make sense within a single coherent picture as a single whole composed of all the chapters of this PhD thesis; in other words, though *independent* from each other, the parts of a whole still "make sense together" to compose, indeed, a *single* whole. Fig. 11.1 summarizes this idea.

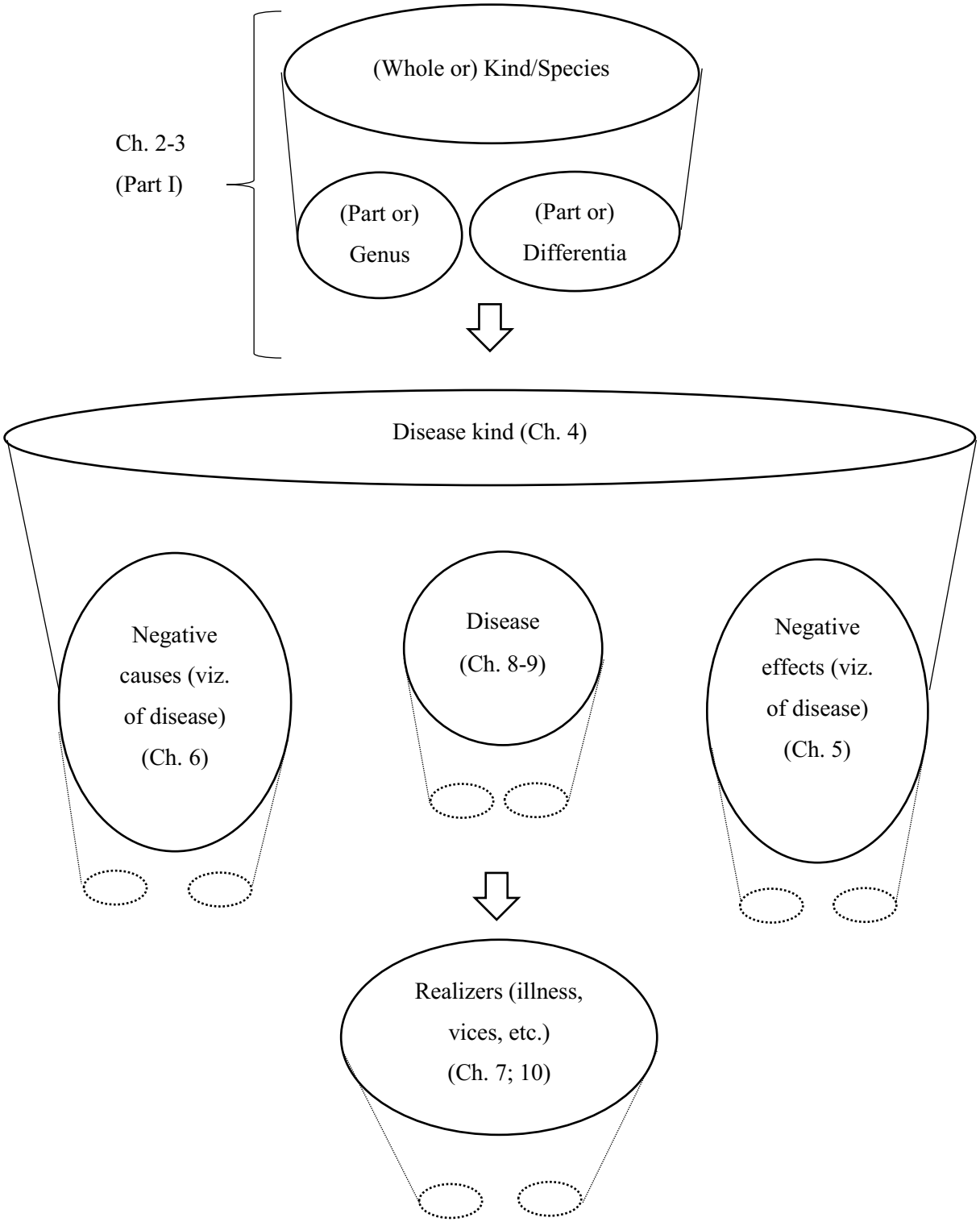

Fig. 11.1. – Depiction of the synoptic structure of the present PhD dissertation following our mereology. Legend: the oblique bars represent a constitutive relationship; the arrow a relationship of absolute extensionality.

Without ruling out other aspects of disease (e.g. its epistemology or ethics), one of the greatest achievements of the present PhD dissertation, by focusing on the metaphysical aspect of disease, is to have shown the constant relevance and fruitfulness of an *analytical* approach to the concept of disease coupled with a minimally practice-oriented metaphilosophy. In that sense, the meta-philosophy advocated in this PhD thesis departs from the current highly naturalistic tradition in philosophy of science and medicine, but is still somehow continuous with the huge tradition of analytic philosophy (cf. Engel, 1997); more precisely, even if the reader disagrees with the proposed methodology, it is hardly deniable that we have shown how fruitful is the (*re*-)*connection* between the current debate around disease and traditional metaphysical issues around e.g. the notion of essence or negative entities – indeed, this has allowed us to get an original and interesting theory of disease as well as of related entities.

What are the consequences of our theory of disease? Albeit the most direct *epistemic* and *ethical* consequences of our metaphysical inquiry into disease have already been studied throughout the whole PhD dissertation (cf. e.g. Sec. 2.1.2; 2.2.1; 3.1.1; 3.2.4; 4.2.7; 6.3.1; 7.2.2; 7.2.3; 10.2.1; Exc. 3.5; 10.1), first, a very interesting extension of the present PhD dissertation for the epistemology of science or, more specifically, the one of medicine lies in how from a certain theory of disease (and health) – i.e. our essentialism about disease -, one passes to a certain theory of medical explanations viz. a *constitutive* one. Indeed, if to provide a medical explanation is to provide evidence of knowledge of disease (and health), then this evidence *spells out* what disease (and health) is(/are) in terms of its(/their) constituents. The implications of this are two-fold: first, it goes against contemporary orthodoxy, which still takes medical explanations as (naturalistically) *causal*(-*statistical*) (Russo & Williamson, 2007), to the contrary of the neo-mechanistic view in philosophy of science, generally speaking (on constitutive mechanistic explanations, cf. Craver, 2007); second, if we are right that the explanations given are nothing else than statements on the nature of health and disease, then, though there is obviously a ratiocinative activity involved in explanations (in all their generality) with a term like "spell out", this points toward a *weak ontic* conception of explanations, according to which explanatory relationships are objective (cf. Illari, 2013).

Second, another interesting (and very general) (bio)ethical consequence of essentialism about disease is the idea that, if we agree that death and disease are both specific *lethal* values, while life and health are two specific highest-level values i.e. *vital* ones, which are the essences of e.g. a human being (as a whole) and his parts, then e.g. a general theory of justice should be built upon the idea that life and health are just those values that a human being and his parts aim at attaining, for they are *what* a human being and his parts are; a theory of justice should

focus, thus, on how, in a fair society, to prevent death and disease i.e. the extinction of life and health.

References

Adolphs, R., & Andler, D. (2018). Investigating emotions as functional states distinct from feelings. *Emotion Review, 10*(3), 191-201. doi: 10.1177/1754073918765662

Adriaens, P. R., & De Block, A. (Eds.). (2011). *Maladapting minds: Philosophy, psychiatry, and evolutionary theory*. New York: Oxford University Press.

Ainsworth, T. (2020). Form vs. matter. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/form-matter/

Allen, J. (2003). G. E. Moore and the principle of organic unity. *Journal of Value Inquiry, 37*(3), 329-339. doi: 10.1023/b:inqu.0000013345.47521.35

Americans with Disabilities Acts (ADA) *Amendments Act*. (2008). URL: https://www.eeoc.gov/statutes/ada-amendments-act-2008

American Psychiatric Association (APA) (Ed.). (1987). *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) (3rd ed. rev.). Washington; London: American Psychiatric Publishing.

American Psychiatric Association (APA) (Ed.). (2013). *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) (5th ed.). Washington; London: American Psychiatric Publishing.

Amoretti, M. C., & Lalumera, E. (2019a). Harm should not be a necessary criterion for mental disorder: Some reflections on the DSM-5 definition of mental disorder. *Theoretical Medicine and Bioethics, 40*(4), 321-337. doi: 10.1007/s11017-019-09499-4

Amoretti, M. C., & Lalumera, E. (2019b). A potential tension in DSM-5: The general definition of mental disorder versus some specific diagnostic criteria. *Journal of Medicine and Philosophy, 44*(1), 85-108. doi: 10.1093/jmp/jhy001

Amundson, R. (1994). Two concepts of constraint: Adaptationism and the challenge from developmental biology. *Philosophy of Science, 61*(4), 556-578. doi: 10.1086/289822

Amundson, R. (2000). Against normal function. *Studies in History and Philosophy of Biological and Biomedical Sciences, 31*(1), 33-53. doi: 10.1016/s1369-8486(99)00033-3

Amundson, R. (2005). *The changing role of the embryo in evolutionary thought: Roots of Evo-Devo*. New York: Cambridge University Press.

Ananth, M. (2016). Human organisms from an evolutionary perspective: Its significance for medicine. In T. Schramme, & S. Edwards (Eds.), *Handbook of the Philosophy of Medicine* (pp. 243-272). Dordrecht: Springer.

Ananth, M. (2017). *In defense of an evolutionary concept of health*. Abingdon: Routledge.

Andersen, F., & Becker Arenhart, J. R. (2016). Metaphysics within science: Against radical naturalism. *Metaphilosophy, 47*(2), 159-180. doi: 10.1111/meta.12175

Anlauf, M., Hein, L., Hense, H.-W., …Schöne-Seifert, B. (2015). Complementary and alternative drug therapy versus science-oriented medicine. *German Medical Science, 13*. doi: 10.3205/000209

Annett, M. (2002). Handedness and brain asymmetry: The right shift theory. *Cognition, Brain, Behavior, 10*, 235-250.

Aragona, M. (2009). The concept of mental disorder and the DSM-V. *Dialogues in Philosophy, Mental and Neuro Sciences, 2*(1), 1-14.

Aristotle. *De anima*. Ed. by W. D. Ross. Oxford: Clarendon Press. 1956.

Aristotle. *De arte poetica liber*. Ed. by R. Kassel. Oxford: Clarendon Press. 1964.

Aristotle. *De la génération et la corruption*. Ed. and tr. by M. Rashed. Paris: Les Belles Lettres. 2005.

Aristotle. *De generatione animalium*. Ed. and tr. by A. Platt. Oxford: Clarendon Press. 1910.

Aristotle. *Ethica Eudemia*. Ed. by R. R. Walzer & J. M. Mingay. Oxford: Clarendon Press. 1991.

Aristotle. *Ethica Nicomachea*. Ed. by I. Bywater. Oxford: Clarendon Press. 1963.

Aristotle. *Metaphysica*. Ed. by W. Jaeger. New York: Oxford University Press. 1957.

Aristotle. *Metaphysics: A revised text with introduction and commentary*. Tr. by W. D. Ross. Oxford: Clarendon Press. 1924.

Aristotle. *Parts of animals, movement of animals, progression of animals*. Ed. and tr. by E. S. Forster. Cambridge, MA; London: Harvard University Press. 1937.

Aristotle. *Physica*. Ed. by Sir D. Ross. Oxford: Clarendon Press. 1963.

Aristotle. *Politica*. Ed. by Sir D. Ross. Oxford: Clarendon Press. 1963.

Aristotle. *Topica et Sophistici Elenchi*. Ed. by. Sir D. Ross. Oxford: Clarendon Press. 1963.

Armstrong, D. M. (1978). *A theory of universals: Universals and scientific realism* (vol. 2). Cambridge, MA: Cambridge University Press.

Armstrong, D. M. (1989). *Universals: An opinionated introduction*. Boulder: Westview Press.

Armstrong, D. M. (1997). *A world of states of affairs*. Cambridge: Cambridge University Press.

Armstrong, D. M. (1999). *The mind-body problem: An opinionated introduction*. New York; London: Routledge.

Armstrong, T. S. (2003). Symptoms experience: A concept analysis. *Oncology Nursing Forum, 30*(4), 601-606. doi: 10.1188/03.ONF.601-606

Arp, R., Smith, B., & Spear, A. D. (2015). *Building ontologies with basic formal ontology*. Cambridge, MA; London: The MIT Press.

Ashcroft, R. E. (2004). Current epistemological problems in evidence based medicine. *Journal of Medical Ethics, 30*(2), 131-135. doi: 10.1136/jme.2003.007039

Aucouturier, V., & Demazeux, S. (2012). The concept of mental disorder. In H. Carel, & R. Cooper (Eds.), *Health, illness, and disease: Philosophical essays* (pp. 77-95). London: Acumen.

Austin, C. J. (2008). The relation of compresence in the bundle theory: Four problems. *Percipi, 2*, 49-65.

Austin, C. J. (2018). *Essence in the age of evolution: A new theory of natural kinds*. London: Routledge.

Autret, A. (2016). *Les maladies dites "imaginaires": Enquête sur les douleurs et symptômes inexpliqués*. Paris: Albin Michel.

Ayala, F. J. (1998). Teleological explanations versus teleology. *History and Philosophy of the Life Sciences, 20*(1), 41-50.

Aydede, M. (2009). Is feeling pain the perception of something?. *Journal of Philosophy, 106*(10), 531-567. doi: 10.5840/jphil20091061033

Azevedo, M. A. (2015). Health as a clinic-epidemiological concept. *Journal of Evaluation in Clinical Practice, 21*(3), 365-373. doi: 10.1111/jep.12279

Baedke, J. (2018). O organism, where art thou? Old and new challenges for organism-centered biology. *Journal of the History of Biology, 52*(2), 293-324. doi: 10.1007/s10739-018-9549-4

Baedke, J., & Gilbert, S. F. (2020). Evolution and development. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/evolution-development/

Baertschi, B. (2005). *Enquête philosophique sur la dignité: Anthropologie et éthique des biotechnologies*. Genève: Labor et Fides.

Baertschi, B. (2014). The argument of ethical naturalism. In A. Reboul (Ed.), *Mind, values, and metaphysics: Philosophical essays in honor of Kevin Mulligan* (vol. 2) (pp. 89-104). Dordrecht: Springer.

Baertschi, B., & Mauron, A. (2011). Qu'est-ce qu'une veritable amelioration?. *Bioethica Forum, 4*(1), 4-11.

Bain, D. (2003). Intentionalism and pain. *The Philosophical Quarterly, 53*(213), 502-523. doi: 10.1111/1467-9213.00328

Baker, L. R. (2009). Nonreductive materialism. In B. McLaughlin, & A. Beckermann (Eds.), *The Oxford handbook for the philosophy of mind* (pp. 109-120). Oxford: Oxford University Press.

Baker 3rd , G. T., & Sprott, R. L. (1988). Biomarkers of aging. *Experimental Gerontology, 23*(4-5), 22-239. doi: 10.1016/0531-5565(88)90025-3

Ball, S. W. (1989). Facts, values, and normative supervenience. *Philosophical Studies, 55*(2), 143-172. doi: 10.1007/BF00354185

Balme, D. (1987). Aristotle's biology was not essentialist. In A. Gotthelf, & J. G. Lennox (Eds.), *Philosophical issues in Aristotle's biology* (pp. 287-312). Cambridge: Cambridge University Press.

Banicki, K. (2018). Personality disorders and thick concepts. *Philosophy, Psychiatry, and Psychology, 25*(3), 209-221. doi: 10.1353/ppp.2018.0030

Barker, M. J. (2010). Specious intrinsicalism. *Philosophy of Science, 77*(1), 73-91. doi: 10.1086/650209

Barnes, E. (2009). Disability, minority, and difference. *Journal of Applied Philosophy, 26*(4), 337-355. doi: 10.1111/j.1468-5930.2009.00443.x

Barnes, E. (2010). Ontic vagueness: A guide for the perplexed. *Noûs, 44*(4), 601-627. doi: 10.1111/j.1468-0068.2010.00762.x

Barnes, E. (2014). Valuing disability, causing disability. *Ethics, 125*(1), 88-113. doi: 10.1086/677021

Barnes, E. (2016). *The minority body: A theory of disability*. Oxford: Oxford University Press.

Barnes, E., & Williams, J. R. G. (2011). A theory of metaphysical indeterminacy. In K. Bennett, & D. W. Zimmerman (Eds.), *Oxford studies in metaphysics* (vol. 6) (pp. 103-148). New York: Oxford University Press.

Barros, D. B. (2013). Negative causation in causal and mechanistic explanation. *Synthese, 190*(3), 449-469. doi: 10.1007/s11229-011-0040-4

Bartels, A. (1996). Modern essentialism and the problem of individuation of spacetime points. *Erkenntnis, 45*(1), 25-43. doi: 10.1007/BF00226369

Bartlett, J. (2017). Evolutionary teleonomy as a unifying principle for the extended evolutionary synthesis. *Bio-Complexity, 2*, 1-7. doi: 10.5048/BIO-C.2017.2.c

Basic formal ontology. (2017). URL: http://basic-formal-ontology.org/

Baumgartner, M. (2008). Regularity theories reassessed. *Philosophia, 36*(3), 327-354. doi: 10.1007/s11406-007-9114-4

Baxter, D. (1988). Identity in the loose and popular sense. *Mind, 97*(388), 575-592.

Bayertz, K., & Quante, M. (2003). Genetic enhancement. In D. N. Cooper (Ed.), *Nature encylopedia of the human genome* (pp. 824-827). London: Nature Publishing Group.

Bealer, G. (1998). Intuition and the autonomy of philosophy. In M. DePaul & W. Ramsey (Eds.), *Rethinking intuition: The psychology of intuition and its role in philosophical inquiry (pp. 201-240)*. Lanham, Maryland: Rowman & Littlefield.

Beall, J., Glanzberg, M., & Ripley, D. (2016). Liar paradox. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/liar-paradox/

Beaney, M. (2014). Analysis. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/analysis/

Bechtel, W., & Richardson, R. C. (2010). *Discovering complexity: Decomposition and localization as strategies in scientific research* (2nd ed.). Cambridge, MA: MIT Press.

Bedau, M. A. (2008). What is life? In S. Sarkar, & A. Plutynski (Eds.), *A companion to the philosophy of biology* (pp. 455-471). Malden, MA: Blackwell.

Beebee, H. (2004). Causing and nothingness. In L. A. Paul, E. J. Hall, & J. Collins (Eds.), *Causation and counterfactuals* (pp. 291-308). Cambridge, MA: MIT Press.

Behrmann, M., & Avidan, G. (2005). Congenital prosopagnosia: face-blind from birth. *Trends in Cognitive Sciences, 9*(4), 180-187. doi: 10.1016/j.tics.2005.02.011

Bennett, M. R., & Hacker, P. M. S. (2003). *Philosophical foundations of neuroscience*. Malden: Blackwell.

Bennett, M. R., Dennett, D. C., Hacker, P. M. S., Searle, J. R., & Robinson, D. N. (2007). *Neuroscience and philosophy: Brain, mind, and language*. New York: Columbia University Press.

Berges, S. (2012). Virtue as mental health: A Platonic defence of the medical model in ethics. *Journal of Ancient Philosophy, 6*(1), 1-19. doi: 10.11606/issn.1981-9471.v6i1p1-19

Berryman, S. (2016). Ancient atomism. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/atomism-ancient/

Bertolaso, M. (2016). *Philosophy of cancer: A dynamic and relational view*. Dordrecht: Springer.

Beurton, P., Falk, R., & Rheinberger, H.-J. (Eds.). (2000). *The concept of the gene in development and evolution: Historical and epistemological perspectives*. Cambridge: Cambridge University Press.

Bickle, J. (2020). Multiple realizability. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/multiple-realizability/

Biddle, J. B. (2016). Inductive risk, epistemic risk, and overdiagnosis of disease. *Perspectives on Science, 24*(2), 192-205. doi: 10.1162/posc_a_00200

Biomarkers Definitions Working Group. (2001). Biomarkers and surrogate endpoints: Preferred definitions and conceptual framework. *Clinical Pharmacology & Therapeutics, 69*(3), 89-95. doi: 10.1067/mcp.2001.113989

Bird, A. (2007). *Nature's metaphysics: Laws and properties*. Oxford: Oxford University Press.

Bird, A., & Tobin, E. (2017). Natural kinds. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/natural-kinds/

Birnbacher, D., & Hommen, D. (2012). *Negative Kausalität: Ideen & Argumente*. Berlin: de Gruyter.

Bishop, R. (2015). Chaos. In E. N. Zalta (Ed.), The Stanford Encyclopedia of Philosophy. URL: https://plato.stanford.edu/entries/chaos/

Bliss, R., & Trogdon, K. (2014). Metaphysical grounding. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/grounding/

Block. N. (1995). On a confusion about a function of consciousness. *Brain and Behavioral Sciences, 18*(2), 227-247. doi: 10.1017/s0140525x00038188

Blum, K., Werner, T., Carnes, S., …Gold, M. (2012). Sex, drugs, and rock 'n' roll: Hypothesizing common mesolimbic activation as a function of reward gene polymorphisms. *Journal of Psychoactive Drugs, 44*(1), 38-55. doi: 10.1080/02791072.2012.662112

Boghossian, P. A. (1996). Analyticity reconsidered. *Noûs, 30*(3), 360-391. doi: 10.2307/2216275

Bohn, E. D. (2009). Must there be a top level?. *Philosophical Quartely, 59*(253), 193-201. doi: 10.1111/j.1467-9213.2008.573.x

Bolton, D. (2008). *What is mental disorder? An essay in philosophy, science, and values*. New York: Oxford University Press.

Bolton, D., & Gillett, G. (2019). *The biopsychosocial model of health and disease: New philosophical and scientific developments*. Cham: Palgrave Pivot.

Bolzano, B. (1837 [1931]). *Wissenschaftslehre: Versuch einer ausführlichen und grösstentheils neuen Darstellung der Logik mit steter Rücksicht auf deren bisherige Bearbeiter (2nd ed.). Leipzig: Felix Meiner.*

Boniolo, G., & Campaner, R. (2019). Causal reasoning and clinical practice: Challenges from molecular biology. *Topoi, 38*(2), 423-435. doi: 10.1007/s11245-017-9518-0

Boniolo, G., & Carrara, M. (2004). On biological identity. *Biology and Philosophy, 19*(3), 443-457. doi: 10.1023/B:BIPH.0000036163.02581.15

Boorse, C. (1975). On the distinction between illness and disease. *Philosophy & Public Affairs, 5*(1), 49-68.

Boorse, C. (1976). What a theory of mental health should be. *Journal for the Theory of Social Behaviour, 6*(1), 61-84. doi: 10.1111/j.1468-5914.1976.tb00359.x

Boorse, C. (1977). Health as a theoretical concept. *Philosophy of Science 44*(4), 542-573. doi: 10.1086/288768

Boorse, C. (1997). A rebuttal on health. In J. M. Humber, & R. F. Almeder (Eds.), *What Is Disease?* (pp. 1-134). Humana Press: Totowa.

Boorse, C. (2011). Concepts of health and disease. In F. Gifford (Ed.), *Philosophy of medicine* (pp. 13-64). Amsterdam: Elsevier.

Boorse, C. (2014). A second rebuttal on health. *Journal of Medicine and Philosophy, 39*(6), 683-724. doi: 10.1093/jmp/jhu035

Bordini, D. (2017). Not in the mood for intentionalism. *Midwest Studies in Philosophy, 41*(1), 60-81. doi: 10.1111/misp.12066

Borg, E., Harrison, R., Stazicker, J., & Salomons, T. (2020). *Mind and Language, 35*(1), 29-47. doi: 10.1111/mila.12227

Borsboom, D. (2017). A network theory of mental disorders. *World Psychiatry, 16*(1), 5-13. doi: 10.1002/wps.2037

Borsboom, D., Cramer, A., & Kalis, A. (2018). Brain disorders? Not really… Why network structures block reductionism in psychopathology research. *Behavioral and Brain Sciences, 42*, 1-54. doi: 10.1017/s0140525x17002266

Boyd, K. M. (2000). Disease, illness, sickness, health, healing and wholeness: Exploring some elusive concepts. *Medical Humanities, 26*(1), 9-17. doi: 10.1136/mh.26.1.9

Boyd, R. (1999a). Homeostasis, species, and higher taxa. In R. A. Wilson (Ed.), *Species: New interdisciplinary essays* (pp. 141-185). Cambridge, MA: MIT Press.

Boyd, R. (1999b). Kinds, complexity and multiple realization: Comment on Millikan's 'Historical kinds and the special sciences'. *Philosophical Studies, 95*(1-2), 67-98. doi: 10.1023/A:1004511407133

Bradford Hill, A. (1965). The environment and disease: Association or causation?. *Proceedings of the Royal Society of Medicine, 58*(5), 295-300.

Bradley, B., Feldman, F., & Johansson, J. (Eds.). (2013). *The Oxford handbook of philosophy of death*. Oxford: Oxford University Press.

Brandom, R. (2008). *Between saying and doing: Towards an analytic pragmatism*. Oxford: Oxford University Press.

Bricker, P. (2014). Ontological commitment. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/ontological-commitment/

Bridson, E. (2001). The English 'sweate' (Sudor Anglicus) and hantavirus pulmonary syndrome. *British Journal of Biomedical Science, 58*(1), 1-6.

Brigandt, I. (2003). Species pluralism does not imply species eliminativism. *Philosophy of Science, 70*(5), 1305-1316. doi: 10.1086/377409

Brigandt, I. (2006). *A theory of conceptual advance: Explaining conceptual change in evolutionary, molecular, and evolutionary developmental biology*. PhD thesis, University of Pittsburgh. URL: http://d-scholarship.pitt.edu/8849/1/Dissertation_Ingo_Brigandt_08-04-2006.pdf

Brink, D. O. (1989). *Moral realism and the foundation of ethics*. New York: Cambridge University Press.

Broad, C. D. (1924). Critical and speculative philosophy. In J. H. Muirhead (Ed.), *Contemporary British philosophy: Personal statements* (vol. 1) (pp. 75-100). London: Allen and Unwin.

Broadbent, A. (2011). Inferring causation in epidemiology: Mechanisms, black boxes, and contrasts. In P. McKay Illari, F. Russo, & J. Williamson (Eds.), *Causality in the sciences* (pp. 45-69). New York: Oxford University Press.

Broadbent, A. (2019). Health as a secondary property. *British Journal for the Philosophy of Science, 70*(2), 609-627. doi: 10.1093/bjps/axx014

Bromberger, S. (1992). *On what we know we don't know*. Chicago: University of Chicago Press.

Broome, M., & Bortolotti, L. (2009a). Mental illness as mental: A defence of psychological realism. *Humana Mente, 3*(11), 25-44.

Broome, M., & Bortolotti, L. (Eds.). (2009b). *Psychiatry as cognitive neuroscience: Philosophical perspectives*. New York: Oxford University Press.

Brower, J. E. (2010). Aristotelian endurantism: A new solution to the problem of temporary intrinsics. *Mind, 119*(476), 883-905. doi: 10.1093/mind/fzq072

Brower, J. E. (2018). Medieval theories of relations. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/relations-medieval/

Buckner, C. (2015). A property cluster theory of cognition. *Philosophical Psychology, 28*(3), 307-336. doi: 10.1080/09515089.2013.843274

Bueter, A. (2019). On illness, disease, and priority: A framework for more fruitful debates. *Medicine, Health Care and Philosophy, 22*(3), 463-474. doi: 10.1007/s11019-019-09886-7

Burge, T. (1993). Concepts, Definitions, and meaning. *Metaphilosophy*, *24*(4), 309–325.

Burge, T. (2010). *Origins of objectivity*. New York: Oxford University Press.

Cameron, R. P. (2008). Turtles all the way down: Regress, priority and fundamentality. *Philosophical Quarterly, 58*(230), 1-14. doi: 10.1111/j.1467-9213.2007.509.x

Campaner, R. (2012). *Philosophy of medicine: Causality, evidence and explanation*. Bologna: Cooperativa Libraria Universitaria Editrice Bologna.

Campbell, K. (1990). *Abstract particulars*. Oxford: Blackwell.

Canali, S. (2019). Evaluationg evidential pluralism in epidemiology: Mechanistic evidence in exposome research. *History and Philosophy of the Life Sciences, 41*(1), 4. doi: 10.1007/s40656-019-0241-6

Canguilhem, G. (1966). *Le normal et le pathologique*. Paris: Presses Universitaires de France.

Cappelen, H. (2012). *Philosophy without intuitions*. Oxford: Oxford University Press.

Cappelen, H. (2018). *Fixing language: An essay on conceptual engineering*. Oxford: Oxford University Press.

Carel, H. (2016). *Phenomenology of illness*. New York: Oxford University Press.

Carey, S. (1985). *Conceptual change in childhood.* Cambridge, MA: MIT Press.

Carrier, M., & Finzer, P. (2006). Explanatory loops and the limits of genetic reductionism. *International Studies in the Philosophy of Science, 20*(3), 267-283. doi*:* 10.1080/02698590600960937

Carlson, E. (2001). Organic unities, non-trade-off, and the additivity of intrinsic value. *Journal of Ethics, 5*(4), 335-360. doi: *10.1023/a:1013962220004*

Carlson, E. (2020). Organic unities and conditionalism about final value. *Journal of Value Inquiry, 54*(2), 175-181. doi: 10.1007/s10790-019-09688-3

Carnap, R. (1947). *Meaning and necessity*. Chicago: University of Chicago Press.

Cartwright, N. (2002). From causation to explanation and back. In J. Reiss (Ed.), *Center for philosophy of natural and social science: Contingency and dissent in science* (pp. 3-21). London. Technical report.

Cartwright, N., & Hardie, J. (2012). *Evidence-based policy: A practical guide to doing it better*. New York: Oxford University Press.

Cartwright, R. (1975). Scattered objects. In K. Lehrer (Ed.), *Analysis and metaphysics: Essays in honor of R. M. Chisholm* (pp. 153-171). Dordrecht: D. Reidel.

Casati, R., & Varzi, A. C. (1999). *Parts and places: The structures of spatial representation*. Cambridge, MA: MIT Press.

Casati, R., & Varzi, A. C. (2019). Holes. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy.* URL: *https://plato.stanford.edu/entries/holes/*

Casetta, E., & Vecchi, D. (2019). Species are, at the same time, kinds and individuals: A causal argument based on an empirical approach to species identity. *Synthese*, 1-19. doi: *10.1007/s11229-019-02199-5*

Casini, L. (2016). Can interventions rescue Glennan's mechanistic account of causality?. *British Journal for the Philosophy of Science, 67*(4), 1155-1183. doi: *10.1093/bjps/axv014*

Casini, L. (2017). Malfunctions and teleology: On the chances of statistical accounts of functions. *European Journal for Philosophy of Science, 7*(2), 319-335. doi: 10.1007/s13194-016-0163-z

Chakravartty, A. (2017). Scientific realism. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/scientific-realism/

Chalmers, D. J. (2009). Ontological anti-realism. In D. J. Chalmers, D. Manley, & R. Wasserman (Eds.), *Metametaphysics: New essays on the foundations of ontology* (pp. 77-129). Oxford: Clarendon Press.

Chalmers, D. J., Manley, D., & Wasserman, R. (Eds.). (2009). *Metametaphysics: New essays on the foundations of ontology*. Oxford: Clarendon Press.

Charland, L. C. (2004). Character: Moral treatment and the personality disorders. In J. Radden (Ed.), *The philosophy of psychiatry: A companion* (pp. 64-77). Oxford: Oxford University Press.

Charland, L. C. (2006). Moral nature of the DSM-IV cluster B personality disorders. *Journal of Personality Disorders, 20*(2), 116-125.

Charles, D. (2017). Aristotle on agency. In G. Williams (Ed.), *Oxford Handbooks Online: Scholarly Research Reviews*. doi: 10.1093/oxfordhb/9780199935314.013.6

Charlesworth, J. E. G., Petkovic, G., Kelley, J. M., …Howick, J. (2017). Effects of placebos without deception compared with no treatment: A systematic review and meta-analysis. *Journal of Evidence-Based Medicine, 10*(2), 97-107. doi: 10.1111/jebm.12251

Chiffi, D., & Zanotti, R. (2017a). Fear of knowledge: Clinical hypotheses in diagnostic and prognostic reasoning. *Journal of Evaluation in Clinical Practice, 23*(5), 928-834. doi: 10.1111/jep.12664

Chiffi, D., & Zanotti, R. (2017b). Knowledge and belief in placebo effect. *Journal of Medicine and Philosophy, 42*(1), 70-85. doi: 10.1093/jmp/jhw033

Chin-Yee, B., & Upshur, R. E. G. (2017). Re-evaluating concepts of biological function in clinical medicine: Towards a new naturalistic theory of disease. *Theoretical Medicine and Bioethics, 38*(4), 245-264. doi: 10.1007/s11017-017-9410-3

Chisholm, R. M. (1969). On the observability of the self. *Philosophy and Phenomenological Research, 30*, 7-21. doi: 10.2307/2105917

Chisholm, R. M. (1975). Mereological essentialism: Some further considerations. *Review of Metaphysics, 28*, 477-484.

Chisholm, R. M. (1989). *On metaphysics*. Minneapolis: University of Minnesota Press.

Christias, D. (2019). Towards a reformed liberal and scientific naturalism. *Dialectica, 72*(4), 507-534. doi: 10.1111/1746-8361.12279

Clarke, B. (2011). *Causality in medicine with particular reference to the viral causation of cancers*. PhD thesis, University College London.

Clarke, B., Gillies, D., Illari, P., Russo, F., & Williamson, J. (2013). The evidence that evidence-based medicine omits. *Preventive Medicine, 57*(6), 745-747. doi: 10.1016/j.ypmed.2012.10.020

Clarke, R. (2014). *Omissions: Agency, metaphysics, and responsibility*. New York: Oxford University Press.

Cleland, C. E. (1990). The difference between real change and mere Cambridge change. *Philosophical Studies, 60*(3), 257-280. doi: 10.1007/BF00367472

Cleland, C. E. (2012). Life without definitions. *Synthese, 185*(1), 124-144. doi: 10.1007/s11229-011-9879-7

Cohen, S. M. (1973). Plato's method of division. In J. M. E. Moravcsik (Ed.), *Patterns in Plato's thought* (pp. 181-191). Dordrecht: Springer.

Cohen, S. M. (2012). Alteration and persistence: Form and matter in the Physics and De Generatione et Corruptione. In C. Shields (Ed.), *The Oxford handbook of Aristotle* (pp. 205-226). New York: Oxford University Press.

Cohen, S. M. (2008). Kooky objects revisited: Aristotle's ontology. *Metaphilosphy, 39*(1), 3-19. doi: 10.1111/j.1467-9973.2008.00521.x

Cohen, S. M. (2013). Accidental beings in Aristotle's ontology. In G. Anagnostopoulos, & Jr. Miller (Eds.), *Reason and analysis in ancient Greek philosophy: Essays in honor of David Keyt* (pp. 231-242). Dordrecht: Springer.

Cohen, S. M. (2020). Aristotle's metaphysics. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/aristotle-metaphysics/

Committee on a Framework for Developing a New Taxonomy of Disease. (2011). *Toward precision medicine: Building a knowledge network for biomedical research and a new taxonomy of disease*. Washington: The National Academies Press.

Connell, S. (2018). Aristotle's explanations of monstrous births and deformities in Generation of Animals 4.4. In A. Falcon, & D. Lefebvre (Eds.), *Aristotle's Generation of Animals: A critical guide* (pp. 207-223). Cambridge: Cambridge University Press.

Connolly, N. (2015). Yes: Bare particulars!. *Philosophical Studies, 172*(5), 1355-1370. doi: 10.1007/s11098-014-0353-5

Cooper, R. (2002). Disease. *Studies in History and Philosophy of Biological and Biomedical Sciences, 33*(2), 263-282. doi: 10.1016/S0039-3681(02)00018-3

Cooper, R. (2005). *Classifying madness: A philosophical examination of the Diagnostic and Statistical Manual of Mental Disorders*. Dordrecht: Springer.

Cooper, R. (2007). Aristotelian accounts of disease – What ae they good for?. *Philosophical Papers, 36*(3), 427-442. doi: 10.1080/05568640709485208

Cooper, R. (2010). Are culture-bound syndromes as real as universally-occurring disorders?. *Studies in History and Philosophy of Biological and Biomedical Sciences, 41*(4), 325-332. doi: 10.1016/j.shpsc.2010.10.003

Cooper, R. (2015). Must disorders cause harm? The changing stance of the DSM. In S. Demazeux, & P. Singy (Eds.), *The DSM-5 in perspective: Philosophical reflections on the psychiatric babel* (pp. 83-96). Dordrecht: Springer.

Corrado, D., Migliore, F., Basso, C., & Thiene, G. (2006). Exercise and the risk of sudden cardiac death. *Herz, 31*(6), 553-558. doi: 10.1007/s00059-006-2885-8

Correia, F. (2005). *Existential dependence and cognate notions*. München: Philosophia.

Correia, F. (2017). Real definitions. *Philosophical Issues, 27*(1), 52-73. doi: 10.1111/phis.12091

Correia, F., & Schnieder, B. (Eds.). (2012). *Metaphysical grounding: Understanding the structure of reality*. Cambridge: Cambridge University Press.

Correia, F., & Skiles, A. (2019). Grounding, essence, and identity. *Philosophy and Phenomenological Research, 98*(3), 642-670. doi: 10.1111/phpr.12468

Cotnoir, A. J. (2013). Composition as general identity. In K. Bennett, & D. W. Zimmerman (Eds.), *Oxford studies in metaphysics* (vol. 8) (pp. 294-322). Oxford: Oxford University Press.

Cotnoir, A. J. (2014). Universalism and junk. *Australasian Journal of Philosophy, 92*(4), 649-664. doi: 10.1080/00048402.2014.924540

Cotnoir, A. J., & Bacon, A. (2012). Non-wellfounded mereology. Review of Symbolic Logic, 5(2), 187-204. doi: 10.1017/s1755020311000293

Cournoyea, M. (2017). Evolutionary medicine. In M. Solomon, J. R. Simon, & H. Kincaid (Eds.), *The Routledge companion to philosophy of medicine* (pp. 475-486). London: Routledge.

Cowling, S. (2013). The modal view of essence. *Canadian Journal of Philosophy, 43*(2), 248-266. doi: 10.1080/00455091.2013.827395

Craig, L. R. (2015). Neo-Darwinism and evo-devo: An argument for theoretical pluralism in evolutionary biology. *Perspectives on Science, 23*(3), 243-279. doi: 10.1162/posc_a_00167

Craver, C. F. (2007). *Explaining the brain: Mechanisms and the mosaic unity of neuroscience*. Oxford: Oxford University Press.

Craver, C. F. (2009). Mechanisms and natural kinds. *Philosophical Psychology, 22*(5), 575-594. doi: 10.1080/09515080903238930

Craver, C. F., & Darden, L. (2013). *In search of mechanisms: Discoveries across the life sciences. Chicago*. University of Chicago Press.

Crisp, R. (2017). Well-being. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/well-being/

Crowther, K. (2018). What is the point of reduction in science? *Erkenntnis*. doi: 10.1007/s10670-018-0085-6

Culver, C. M., & Gert, B. (1982). *Philosophy in medicine*. New York: Oxford University Press.

Cumpa, J. (2018). Factualism and the scientific image. *International Journal of Philosophical Studies, 26*(5), 669-678. doi: 10.1080/09672559.2018.1533713

Currie, G., & Ravenscroft, I. (2002). *Recreative minds: Imagination in philosophy and psychology*. Oxford: Clarendon Press.

Czeisler, C. A., Scammell, T. E., & Saper, C. B. (2015). Sleep disorders. In D. L. Kasper, A. S. Fauci, S. L. Hauser, D. L. Longo, J. L. Jameson, & J. Loscalzo (Eds.), *Harrison's principles of internal medicine* (19th ed.) (pp. 184-194). New York: McGraw-Hill.

da Costa, N. C., & French, S. (2003). *Science and partial truth: A unitary approach to models and scientific reasoning*. New York: Oxford University Press.

D'Amico, R. (2007). Disease and the concept of supervenience. In H. Kincaid, & J. McKitrick (Eds.), *Establishing medical reality: Essays in the metaphysics and epistemology of the biomedical sciences* (pp. 35-45). Dordrecht: Springer.

Danner Clouser, K., Culver, C. M., & Gert, B. (1981). *Malady: A new treatment of disease*. Hastings Center Report, 11(3), 29-37. doi: 10.2307/3561321

Daniels, N. (2016). Reflective equilibrium. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/reflective-equilibrium/

Darrason, M. (2014). *Y a-t-il une théorie génétique de la maladie ?*. PhD thesis, Paris 1.

Darrason, M. (2017a). From the concept of genetic disease to the geneticization of diseases: Analyzing and solving the paradox of contemporary medical genetics. In G. Boniolo, & M. J. Nathan (Eds.), *Philosophy of molecular medicine: Foundational issues in research and practice* (pp. 57-78). New York: Routledge.

Darrason, M. (2017b). Médecine de précision et médecine des systèmes: La médecine personalisée se trompe-t-elle de cible ?. *Lato Sensu, 4*(2), 66-82.

Dasgupta, S. (2016). Metaphysical rationalism. *Noûs, 50*(2), 379-418. doi: 10.1111/nous.12082

Dasgupta, S. (2017). Constitutive explanation. *Philosophical Issues, 27*(1), 74-97. doi: 10.1111/phis.12102

Davidson, D. (1963). Actions, reasons, and causes. *Journal of Philosophy, 60*(23), 685-700. doi: 10.2307/2023177

Dawkins, R. (2016). *The selfish gene* (4th ed.). Oxford: Oxford University Press.

De Caro, M., & Voltolini, A. (2010). Is liberal naturalism possible?. In M. De Caro, & D. Macarthur (Eds.), *Naturalism and normativity* (pp. 69-86). New York: Columbia University Press.

De Haan, D. (2018). Hylomorphism, new mechanisms, and explanations in biology, neuroscience, and psychology. In W. M. R. Simpson, R. C. Koons, & N. J. The (Eds.), *Neo-Aristotelian perspectives on contemporary science* (pp. 293-326). New York: Routledge.

Dekeuwer, C. (2016). Conceptualization of genetic disease. In T. Schramme, & S. Edwards (Eds.), *Handbook of the Philosophy of Medicine* (pp. 325-343). Dordrecht: Springer.

Della Rocca, M. (1996). Essentialism. *Philosophical Books, 37*, 1-20; 81-9.

Demazeux, S. (2016). Philosopher contre la psychiatrie, tout contre. *Revue de Synthèse, 137*(6), 11-34. doi: 10.1007/s11873-016-0290-x

Demazeux, S., & Pidoux, V. (2015). Le projet RDoC : La classification psychiatrique de demain ?. *Médecine/Sciences, 31*(8-9), 792-796. doi: 10.1051/medsci/20153108019

Demoss, D., & Devereux, D. (1988). Essence, existence, and nominal definition in Aristotle's Posterior Analytics II 8-10. *Phronesis, 33*(2), 133-154.

Denby, D. A. (2014). Essence and intrinsicality. In R. Francescotti (Ed.), *Companion to intrinsic properties* (pp. 87-109). Berlin: de Gruyter.

Deng, N. (2015). How A-theoretic deprivationists should respond to Lucretius. *Journal of the American Philosophical Association, 1*(3), 417-432. doi: 10.1017/apa.2015.1

Denkel, A. (1996). *Object and property*. Cambridge: Cambridge University Press.

Deonna, J. A., Teroni, F. (2012). *The emotions: A philosophical introduction*. London; New York: Routledge.

Deonna, J. A., Rodogno, R., & Teroni, F. (2011). *In defense of shame: The faces of an emotion*. New York: Oxford University Press.

de Queiroz, K. (2005). Ernst Mayr and the modern concept of species. *Proceedings of the National Academy of Sciences of the Unites States of America, 102*(1), 6600-6607.

de Queiroz, K. (2007). Species concepts and species delimitation. *Systematic Biology, 56*(6), 879-886. doi: 10.1080/10635150701701083

de Rivarol, A. (1856). *Pensées inédites suivies de deux discours sur la philosophie moderne, et sur la souveraineté du peuple*. Paris: J.-A. Boudon.

Deslauriers, M. (2007). *Aristotle on definition*. Leiden; Boston: Brill.

DeVito, S. (2000). On the value-neutrality of the concepts of health and disease: Unto the breach again. *Journal of Medicine and Philosophy, 25*(5), 539-567. doi: 10.1076/0360-5310(200010)25:5;1-W;FT539

Devitt, M. (2008). Resurrecting biological essentialism. *Philosophy of Science, 75*(3), 344-382. doi: 10.1086/593566

De Winter, G. (2015). Aging as disease. *Medicine, Health Care, and Philosophy, 18*(2), 237-243. doi: 10.1007/s11019-014-9600-y

Dicken, P. (2015). Three degrees of naturalism in the philosophy of science. In C. Daly (Ed.), *The Palgrave handbook of philosophical methods* (pp. 573-590). London: Palgrave Macmillan.

Dickerson, L. M., Mazyck, P. J., & Hunter, M. H. (2003). Premenstrual syndrome. *American Family Physician, 67*(8), 1743-1752.

Dictionary of Medical Terms (4th ed.). London: A & C Black.

DiFrisco, J. (2018). Biological processes: Criteria of identity and persistence. In D. J. Nicholson, & J. Dupré (Eds.), *Everything flows: Towards a processual philosophy of biology* (pp. 76-95). New York: Oxford University Press.

Dilthey, W. (1883 [1989]). Introduction to the human sciences. In R.A. Makkreel, & F. Rodi (Eds.), *Selected works* (pp. 47–242). Princeton: Princeton University Press.

DiMauro, S., & Hirano, M. (2005). Mitochondrial encephalomyopathies: An update. *Neuromuscular Disorders, 15*(4), 276-286. doi: 10.1016/j.nmd.2004.12.008

Dinkmeyer, D., & Dinkmeyer Jr., D. (1979). Holistic approaches to health. *Elementary School Guidance & Counseling, 14*(2), 108-112.

Dobzhansky, T. (1937). *Genetics and the origin of species*. New York: Columbia University Press.

Doust, J., Walker, M. J., & Rogers, W. A. (2017). Current dilemmas in defining the boundaries of disease. *Journal of Medicine and Philosophy, 42*(4), 350-366. doi: 10.1093/jmp/jhx009

Dorland, W. A. N. (2011). *Dorland's illustrated medical dictionary* (33rd ed.). Philadelphia: Elsevier.

Down, J. L. H. (1866). Observations on an ethnic classification of idiots. *London Hospital Reports, 3*, 259-262.

Dumsday, T. (2014). E.J. Lowe on the unity problem. *Studia Philosophica Estonica, 7*(2), 195-218. doi: 10.12697/spe.2014.7.2.10

Dumsday, T. (2016). Natural-kind essentialism, substance ontology, and the unity problem: Two dispositionalist solutions. *Dialectica, 70*(4), 609-626. doi: 10.1111/1746-8361.12168

Dumsday, T. (2019). *Dispositionalism and the metaphysics of science*. Cambridge: Cambridge University Press.

Dupré, J. (1993). *The disorder of things: Metaphysical foundations of the disunity of science*. Cambridge, MA: Harvard University Press.

Dupré, J. (1999). On the impossibility of a monistic account of species. In R. A. Wilson (Ed.), *Species: New interdisciplinary essays* (pp. 3-22). Cambridge, MA: MIT Press.

Dupré, J., & O'Malley, M. A. (2009). Varieties of living things: Life at the intersection of lineage and metabolism. *Philosophy, Theory and Practice in Biology, 1*(3). doi: 10.3998/ptb.6959004.0001.003

Dussault, A. C., & Gagné-Julien, A.-M. (2015). Health, homeostasis, and the situation-specificity of normality. *Theoretical Medicine and Bioethics, 36*(1), 61-81. doi: 10.1007/s11017-015-9320-1

Edwards, S. (2017). Disability as medical and as social category. In In T. Schramme, & S. Edwards (Eds.), *Handbook of the Philosophy of Medicine* (pp. 147-158). Dordrecht: Springer.

Eklund, M. (2006). Metaontology. *Philosophy Compass, 1*(3), 317-334. doi: 10.1111/j.1747-9991.2006.00026.x

Eklund, M. (2017). *Choosing normative concepts*. New York: Oxford University Press.

Eldredge, N. (1995). *Reinventing Darwin: The great debate at the high table of evolutionary theory*. New York: Wiley.

El-Hani, C. N., & Hemmeche, C. (2000). On some theoretical grounds for an organism-centered biology: Property emergence, supervenience, and downward causation. *Theory in Biosciences, 119*(3-4), 234-275. doi: 10.1078/1431-7613-00017

Endler, J. (1986). *Natural selection in the wild*. Princeton: Princeton University Press.

Engel, P. (1995). Croyance, jugement et *self-deception*. *L'inactuel, 3*, 105-122.

Engel, P. (1996). *Philosophie et psychologie*. Paris: Gallimard.

Engel, P. (1997). *La dispute: Une introduction à la philosophie analytique*. Paris: Editions de Minuit.

Engel, P. (2002). *Truth*. London: Routledge.

Engel, P. (2007). *Va savoir! De la connaissance en général*. Paris: Hermann.

Engel, P. (2019). *Les vices du savoir : Essai d'éthique intellectuelle*. Paris: Agone.

Engelhardt Jr., H. T. (1976). Ideology and etiology. *Journal of Medicine and Philosophy, 1*(3), 256-268. doi: 10.1093/jmp/1.3.256

Ereshefsky, M. (1992). Eliminative pluralism. *Philosophy of Science, 59*(4), 671-690. doi: 10.1086/289701

Ereshefsky, M. (2001. *The poverty of the Linnaean hierarchy: A philosophical study of biological taxonomy*. Cambridge: Cambridge University Press.

Ereshefsky, M. (2009). Defining 'health' and 'disease'. *Studies in History and Philosophy of Biological and Biomedical Sciences 40*(2), 221-227. doi: 10.1016/j.shpsc.2009.06.005

Ereshefsky, M. (2017). Species. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/species/

Ereshefsky, M. (2018). Natural kinds, mind independence, and defeasibility. *Philosophy of Science, 85*(5), 845-856. doi: 10.1086/699676

Ereshefsky, M., & Reydon, T. A. C. (2015). Scientific kinds. *Philosophical Studies, 172*(4), 969-986. doi: 10.1007/s11098-014-0301-4

Esfeld, M. (2009). *Philosophie des sciences: Une introduction* (2nd ed.). Lausanne: Presses polytechniques et universitaires romandes.

European Academies Science Advisory Council. (2017). *Homeopathic products and practices*: *Assessing the evidence and ensuring consistency in regulating medical claims in the EU.* URL: https://easac.eu/publications/details/homeopathic-products-and-practices/

European Commission against Racism and Intolerance. (2020). *Glossary*. URL: https://rm.coe.int/ecri-glossary/168093de74

Evnine, S. (2018). The use of sets (and other extensional entities) in the analysis of hylomorphically complex objects. *Metaphysics, 1*(1), 97-109. doi: 10.5334/met.9

Etxeberria, A. (2016). Biological organization and pathology: Three views on the normativity of medicine. In E. Giroux (Ed.), *Naturalism in the philosophy of health: Issues and implications* (pp. 121-142). Cham: Springer.

Fallis, D. (2009). What is lying?. *Journal of Philosophy, 106*(1), 29-56. doi: 10.5840/jphil200910612

Farre, A., & Rapley, T. (2017). The new old (and old new) medical model: Four decades navigating the biomedical and psychosocial understandings of health and illness. *Healthcare, 5*(4), 88. doi: 10.3390/healthcare5040088

Faye, J. (2016). *Experience and beyond: The outline of a Darwinian metaphysics*. Cham: Palgrave Macmillan.

Feldbacher-Escamilla, C. J., & Baraghith, K. (2020). Cultural inheritance in generalized Darwinism. *Philosophy of Science, 87*(2), 237-261. doi: 10.1086/707564

Ferreira Ruiz, M. J., & Umerez, J. (2018). Dealing with the changeable and blurry edges of living things: A modified version of property-cluster kinds. *European Journal for Philosophy of Science, 8*(3), 493-518. doi: 10.1007/s13194-018-0210-z

Ferry-Danini, J. (2018). A new path for humanistic medicine. *Theoretical Medicine and Bioethics, 39*(1), 57-77. doi: 10.1007/s11017-018-9433-4

Ferry-Danini, J. (2019). Should phenomenological approaches to illness be wary of naturalism?. *Studies in History and Philosophy of Biological and Biomedical Sciences, 73*, 10-18. doi: 10.1016/j.shpsc.2018.05.006

Feser, E. (2019). *Aristotle's revenge: The metaphysical foundations of physical and biological science*. Neunkirchen-Seelscheid: editiones scholasticae.

Fine, K. (1982). Acts, events and things. In W. Leinfellner, E. Kraemer, & J. Schank (Eds.), *Proceedings of the 6th international Wittgenstein symposium: Language and ontology* (pp. 97-105). Wien: Hölde-Pichler-Tempsky.

Fine, K. (1994a). Essence and modality. *Philosophical Perspectives, 8*, 1-16.

Fine. K. (1994b). Senses of essence. In W. Sinnott-Armstrong, D. Raffman, & N. Asher (Eds.), *Modality, morality and belief. Essays in honor of Ruth Barcan Marcus (pp. 53-73)*. Cambridge, MA: Cambridge University Press.

Fine, K. (2002). The varieties of necessity. In T. S. Gendler, & J. Hawthorne (Eds.), *Conceivability and possibility* (pp. 253-281). Oxford: Oxford University Press.

Fine, L. (2012). What is metaphysics?. In T. E. Tahko (Ed.), *Contemporary Aristotelian metaphysics* (pp. 8-25). New York: Cambridge University Press.

Firth, J. D., Conlon, C., & Cox, T. M. (Eds.). (2020). *Oxford textbook of medicine* (6th ed.). Oxford: Oxford University Press.

First, M. B., & Wakefield, J. C. (2013). Diagnostic criteria as dysfunction indicators: Bridging the chasm between the definition of mental disorder and diagnostic criteria for specific disorders. *Canadian Journal of Psychiatry, 58*(12), 663-669. doi: 10.1177/070674371305801203

Fisher, R. A. (1930). *The genetical theory of natural selection*. London: Dover.

Fitting, M. (2015). Intensional logic. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/logic-intensional/

Foot, P. (1958). Moral arguments. *Mind, 67*(268), 502-513. doi: 10.1093/mind/LXVII.268.502

Foot, P. (2001). *Natural goodness*. New York: Oxford University Press.

Forbes, G. (1986). In defense of absolute essentialism. *Midwest Studies in Philosophy, 11*(1), 3-31. doi: 10.1111/j.1475-4975.1986.tb00486.x

Forest, D. (2016). Is an anatomy of melancholia possible? Brain processes, depression, and mood regulation. In J. C. Wakefield, & S. Demazeux (Eds.), *Sadness or depression? International perspectives on the depression epidemic and its meaning* (pp. 95-107). Dordrecht: Springer.

Forrest, B. (2000). Methodological naturalism and philosophical naturalism: Clarifying the connection. *Philo, 3*(2), 7-29.

Forrest, P. (2010). The identity of indiscernibles. In. E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/identity-indiscernible/

Foucault, M. (1972). *Histoire de la folie à l'âge classique*. Paris: Gallimard.

Fox Keller, E. (2000). *The century of the gene*. Cambridge, MA: Harvard University Press.

Franklin-Hall, L. (2015). Natural kinds as categorical bottlenecks. *Philosophical Studies, 172*(4), 925-948. doi: 10.1007/s11098-014-0326-8

French, S., & McKenzie, K. (2012). Thinking outside the toolbox: Towards a more productive engagement between metaphysics and philosophy of physics. *European Journal of Analytic Philosophy, 8*(1), 42-59.

Frigg, R. (2020). Models in science. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/models-science/

Frijda, N. H. (1986). *The emotions*. Cambridge: Cambridge University Press.

Friston, K. (2012). A free energy principle for biological systems. *Entropy, 14*(11), 2100-2121. doi: 10.3390/e14112100

Fritzson, F.-A. (2018). Subjectivism and relational good. *Ethical Theory and Moral Practice, 21*(2), 359-370. doi: 10.1007/s10677-018-9886-6

Fuellen, G., Jansen, L., Cohen, A. A., …Moller, S. (2019). Health and aging: Unifying concepts, scores, biomarkers and pathways. *Aging and Disease, 10*(4), 883-900. doi: 10.14336/AD.2018.1030

Fulford, K. W. M. (1989). *Moral theory and medical practice*. Cambridge: Cambridge University Press.

Fulford, K. W. M. (2001). What is (mental) disease?: An open letter to Christopher Boorse. *Journal of Medical Ethics, 27*(2), 80-85. doi: 10.1136/jme.27.2.80

Fuller, J. (2017). The new medical model: A renewed challenge for biomedicine. *Canadian Medical Association Journal, 189*, E640-E641. doi: 10.1503/cmaj.160627

Fuller, J. (2018a). Universal etiology, multifactorial diseases and the constitutive model of disease classification. *Studies in History and Philosophy of Biological and Biomedical Sciences, 67*, 8-15. doi: 10.1016/j.shpsc.2017.11.002

Fuller, J. (2018b). What are chronic diseases?. *Synthese, 195*(7), 3197-3220. doi: 10.1007/s11229-017-1368-1

Funkhouser, E. (2006). The determinable-determinate relation. *Noûs, 40*(3), 548-569. doi: 10.1111/j.1468-0068.2006.00623.x

Futuyma, D. J. (2005). *Evolution*. Sanderland, MA: Sinauer Ass.

Gaille, M. (2014). Introduction: La philosophie de la médecine après G. Canguilhem. *Revue de Métaphysique et de Morale, 2*, 155-165.

Galatzer-Levy, I. R., & Bryant, R. A. (2013). 636,120 ways to have posttraumatic stress disorder. *Perspectives on Psychological Science, 8*(6), 651-662. doi: 10.1177/1745691613504115

Galen. *Selected works*. Tr. by P. N. Singer. Oxford: Oxford University Press, 1997.

Galen. *Œuvres tome 1: Introduction générale, sur l'ordre de ses propres livres, sur ses propres livres, que l'excellent médecin est aussi philosophe.* Ed. and tr. by V. Boudon-Millot. Paris: Les Belles Lettres. 2007.

Gallois, A. (2017). *The metaphysics of identity*. London: Routledge.

Galton, F. (1904). Eugenics: Its definition, scope, and aims. *The American Journal of Sociology, 10*(1), 1-6.

Garcia, R. K. (2014). Bare particulars and constituent ontology. *Acta Analytica, 29*(2), 149-159. doi: 10.1007/s12136-013-0208-2

Geach, P. (1969). *God and the soul*. London: Routledge & Kegan Paul.

Gebharter, A. (2017). *Causal nets, interventionism, and mechanisms: Philosophical foundations and applications*. Cham: Springer.

Gebharter, A. (2019). A causal Bayes net analysis of Glennan's mechanistic account of higher-level causation. *British Journal for the Philosophy of Science, axz034*. doi: 10.1093/bjps/axz034

Genetics Home Reference. (2020). *PAH gene*. URL: https://ghr.nlm.nih.gov/gene/PAH

Germain, P.-L. (2012). Cancer cells and adaptive explanations *Biology and Philosophy, 27*(6), 785-810. doi: 10.1007/s10539-012-9334-2

Ghiara, V., & Russo, F. (2018). Reconstructing the mixed mechanisms of health: The role of bio- and socio-markers. URL: http://philsci-archive.pitt.edu/15549/

Ghiselin, M. T. (1974). A radical solution to the species problem. *Systematic Zoology, 23*, 536-544.

Gibbard, A. (1992). Thick concepts and warrant for feelings. *Proceedings of the Aristotelian Society, S66*, 267-283.

Gillett, C. (2016). *Reduction and emergence in science and philosophy*. Cambridge: Cambridge University Press.

Giroux, E. (2009). Définir objectivement la santé: une évaluation du concept bio statistique de Boorse à partir de l'épidémiologie moderne. *Revue Philosophique de la France et de l'Étranger, 134*(1), 35–58.

Giroux, E. (2010). *Après Canguilhem: Définir la santé et la maladie*. Paris: Presses Universitaires de France.

Giroux, E. (Ed.). (2016). *Naturalism in the philosophy of health: Issues and implications*. Cham: Springer.

Giroux, E. (2017). Médecine de precision et Evidence-Based Medicine : quelle articulation ?. *Lato Sensu, 4*(2).

Glackin, S. N. (2016). Three Aristotelian Accounts of Disease and Disability. *Journal of Applied Philosophy, 33*(3), 311-326. doi: 10.1111/japp.12114

Glas, G. (2019). Psychiatry as normative practice. *Philosophy, Psychiatry, and Psychology, 26*(1), 33-48. doi: 10.1353/ppp.2019.0002

Glazier, M. (2017). The difference between epistemic and metaphysical necessity. *Synthese*. doi: 10.1007/s11229-017-1626-2

Glennan, S. S. (1996). Mechanisms and the nature of causation. *Erkenntnis, 44*(1), 49-71. doi: 10.1007/BF00172853

Glennan, S. S. (2002). Rethinking mechanistic explanation. *Philosophy of Science, 69*(S3), 342-353. doi: 10.1086/341857

Glennan, S. S. (2008). Mechanisms. In S. Psillos, & M. Curd (Eds.), *The Routledge companion to the philosophy of science* (pp. 376-384). London; New York: Routledge.

Glennan, S. S. (2017). *The new mechanical philosophy*. Oxford: Oxford University Press.

Glock, H.-J. (2017). Impure conceptual analysis. In G. D'Oro, & S. Overgaard (Eds.), *The Cambridge companion to philosophical methodology* (pp. 77-100). Cambridge: Cambridge University Press.

Gluckman, P. D., Buklijas, T., & Hanson, M. A. (2016). The developmental origins of health and disease (DOHaD) concepts: Past, present, and future. In C. S. Rosenfeld (Ed.), *The epigenome and developmental origins of health and disease* (pp. 1-15). Amsterdam: Elsevier.

Godfrey-Smith, P. (2001). Three kinds of adaptationism. In S. H. Orzack, & E. Sober (Eds.), *Adaptationism and optimality* (pp. 335-357). New York: Cambridge University Press.

Goffman, E. (1963). *Stigma: Notes on the management of spoiled identity*. New York: Simon & Schuster.

Goldman, L., & Schafer, A. I. (Eds.). (2019). *Goldman-Cecil medicine* (26th ed.). Amsterdam: Elsevier.

Gomez, M. (2006). Culture-bound syndromes: Amok. In Y. Jackson (Ed.), *Encyclopedia of Multicultural Psychology* (p. 132). Thousand Oaks, CA: SAGE.

Goodman, N. (1983). *Fact, fiction, and forecast* (4th ed.). Cambridge, MA: Harvard University Press.

Gorman, M. (2005). The essential and the accidental. *Ratio, 18*(3), 276-289.

Gotthelf, A. (2012). *Teleology, first principles, and scientific method in Aristotle's biology*. New York: Oxford University Press.

Gould, S. J. (1977). *Ontogeny and phylogeny*. Cambridge, MA: Belknap Press.

Gould, S. J., & Lewontin, R. C. (1979). The spandrels of San Marco and the Panglossian paradigm: A critique of the adaptationist programme. *Proceedings of the Royal Society of London, 205*(1161), 581-598.

Gould, S. J., & Vrba, E. S. (1973). Exaptation - A missing term in the science of form. In D. L. Hull, & M. Ruse (Eds.), *The philosophy of biology* (pp.52-71). London: Hutchinson University Library.

Gracia, J. J. E. (1999). *Metaphysics and its task: The search for the categorical foundation of knowledge*. Albany: State University of New York Press.

Gracia, J. J. E., & Newton, L. (2012). Medieval theories of the categories. In E.N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/medieval-categories/

Graham, G. (2013). *The disordered mind: An introduction to philosophy of mind and mental illness* (2nd ed.). London; New York: Routledge.

Granger, E. H. (1984). Aristotle on genus and differentia. *Journal of the History of Philosophy, 22*(1), 1-23. doi: 10.1353/hph.1984.0001

Green, A., & Trichopoulos, D. (2002). Skin cancer. In H.-O. Adami, D. Hunter, & D. Trichopoulos (Eds.), *A textbook of cancer epidemiology* (pp. 281-300). New York: Oxford University Press.

Green, S., & Vogt, H. (2016). Personalizing medicine: Disease prevention *in silico* and *in socio*. *Humana Mente, 9*(30), 105-145.

Green, S., Carusi, A., & Hoeyer, K. (2019). Plastic diagnostics: The remaking of disease and evidence in personalized medicine. *Social Science & Medicine, 18*, 112318. doi: 10.1016/j.socscimed.2019.05.023

Grice, H. P. (1957). Meaning. *Philosophical Review, 66*(3), 377-388. doi: 10.2307/2182440

Griffiths, P. E. (1999). Squaring the circle: Natural kinds with historical essences. In R. A. Wilson (Ed.), *Species: New interdisciplinary essays* (pp. 209-228). Cambridge, MA: MIT Press.

Griffiths, P. E., & Matthewson, J. (2018). Evolution, dysfunction, and disease: A reappraisal. *British Journal for the Philosophy of Science, 69*(2), 301-327. doi: 10.1093/bjps/axw021

Griffiths, P. E., & Stotz, K. (2008). Experimental philosophy of science. *Philosophy Compass, 3*(3), 507-521. doi: 10.1111/j.1747-9991.2008.00133.x

Groopman, J. (2007). *How doctors think*. Boston: Houghton Mifflin.

Gross, F. (2011). What systems biology can tell us about disease. *History and Philosophy of the Life Sciences, 33*(4), 477-496.

Guay, A., & Pradeu, T. (2015). To be continued: The genidentity of physical and biological processes. In A. Guay., & T. Pradeu (Eds.), *Individuals across the sciences* (pp. 317-357). New York: Oxford University Press.

Guze, S. B. (1992). *Why psychiatry is a branch of medicine*. New York: Oxford University Press.

Haag, K. L. (2018). Holobionts and their hologenomes: Evolution with mixed modes of inheritance. *Genetics and Molecular Biology, 41*(1), 189-197. doi: 10.1590/1678-4685-GMB-2017-0070

Hacking, I. (1983). *Representing and intervening: Introductory topics in the philosophy of natural science*. New York: Cambridge University Press.

Hacking, I. (1991). A tradition of natural kinds. *Philosophical Studies, 61*(1-2), 109-126. doi: 10.1007/BF00385836

Hacking, I. (1995). The looping effects of human kinds. In D. Sperber, D. Premack, & A. J. Premack (Eds.), *Causal cognition: A multidisciplinary debate* (pp. 351-394). Oxford: Clarendon Press.

Hacking, I. (1998). *Mad travelers: Reflections on the reality of transient mental illnesses*. Charlottesville; London: University Press of Virginia.

Häggqvist, S. (2005). Kinds, projectibility and explanation. *Croatian Journal of Philosophy, 5*(1), 71-87. doi: croatjphil20055141

Häggqvist, S., & Wikforss, Å. (2018). Natural kinds and natural kind terms: Myth and reality. *British Journal for the Philosophy of Science, 69*(4), 911-933. doi: 10.1093/bjps/axw041

Hagmayer, Y., Sloman, S., Lagnado, D., & Waldmann, M. R. (2007). Causal reasoning through intervention. In A. Gopnik, & L. Schulz (Eds.), *Causal learning: Psychology, philosophy, and computation* (pp. 86-100). New York: Oxford University Press.

Hahnemann, C. F. S. (1921). *Organon der Heilkunst* (6th ed.). Heidelberg: Haug.

Hakkarainen, J., Keinänen, M., & Keskinen, A. (2018). Taxonomy or relations: Internal and external. In D. Bertini, & D. Migliorini (Eds.), *Relations: Ontology and philosophy of religion* (pp. 93-121). Verona: Mimesis International.

Hamilton, R. P. (2010). The concept of health: Beyond normativism and naturalism. *Journal of Evaluation in Clinical Practice, 16*(2), 323-329. doi: 10.1111/j.1365-2753.2010.01393.x

Hanahan, D., & Weinberg, R. A. (2000). The hallmarks of cancer. *Cell, 100*(1), 57-70. doi: 10.1016/s0092-8674(00)81683-9

Hansson, S. O. (2017). Science and pseudo-science. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/pseudo-science/

Hansson Wahlberg, T. (2014). Institutional objects, reductionism, and theories of persistence. *Dialectica, 68*(4), 525-562. doi: 10.1111/1746-8361.12083

Hare, R. M. (1952). *The language of morals*. Oxford: Clarendon Press.

Haresnape, C. (2013). An exploration of the relationship between placebo and homeopathy and the implications for clinical trial design. *Journal of the Royal Society of Medicine Short Reports, 4*(9). doi: 10.1177/2042533313490927

Harris, J. (2007). *Enhancing evolution: The ethical case for making better people*. Princeton: Princeton University Press.

Harvey, J. N., & Barnett, D. (1992). Endocrine dysfunction in Kearns-Sayre syndrome. *Clinical Endocrinology, 37*(1), 97-103. doi: 10.1111/j.1365-2265.1992.tb02289.x

Harvey, P. A., & Leinwand, L. A. (2011). Cellular mechanisms of cardiomyopathy. *Journal of Cell Biology, 194*(3), 355-365. doi: 10.1083/jcb.201101100

Hausman, D. M. (2014). Health and functional efficiency. *Journal of Medicine and Philosophy, 39*(6), 634-647. doi: 10.1093/jmp/jhu036

Hausman, D. M. (2015). *Valuing health: Well-being, freedom, and suffering*. New York: Oxford University Press.

Hawley, K. (2020). Temporal parts. In E. N. Zalta (Ed.), The Stanford Encyclopedia of Philosophy. URL: https://plato.stanford.edu/entries/temporal-parts/

Hawthorne, J. (2001). Causal structuralism. *Noûs, 35*(s15), 361-378. doi: 10.1111/0029-4624.35.s15.16

Henry, D. (2008). Organismal natures. *Apeiron, 41*(3), 47-74. doi: 10.1515/APEIRON.2008.41.3.47

Henry, D. (2013). Optimality reasoning in Aristotle's natural teleology. In B. Inwood (Ed.), *Oxford studies in ancient philosophy* (vol. 45) (pp. 225-263). New York: Oxford University Press.

Herder, C., & Roden, M. (2011). Genetics of type 2 diabetes: pathophysiologic and clinical relevance. *European Journal of Clinical Investigation, 41*(6), 672-692. doi: 10.1111/j.1365-2362.2010.02454.x

Heyman, P., Simons, L., & Cochez, C. (2014). Were the English sweating sickness and the Picardy sweat caused by hantaviruses?. *Viruses, 6*(1), 151-171. doi: 10.3390/v6010151

Hill, A. (1999). Phantom limb pain: A review of the literature on attributes and potential mechanisms. *Journal of Pain and Symptom Management, 17*(2), 125-142. doi: 10.1016/S0885-3924(98)00136-5

Hippocrates. *Vol. 1: Ancient medicine; airs waters places; epidemics I and III; the oath; precepts; nutriment*. Ed. and tr. by W. H. S. Jones. Cambridge, MA: Harvard University Press. 1957.

Hippocrates. *Places in man*. Ed. and tr. by E. M. Craik. Oxford: Clarendon Press. 1998.

Hoefer, C., & Martí, G. (2019). Water has a microstructural essence after all. *European Journal for Philosophy of Science, 9*(1). doi: 10.1007/s13194-018-0236-2

Hofmann, B. (2002). On the triad disease, illness and sickness. *Journal of Medicine and Philosophy, 27*(6), 651-673. doi: 10.1076/jmep.27.6.651.13793

Hofmann, B. (2017). Disease, illness, and sickness. In M. Solomon, J. R. Simon, & H. Kincaid (Eds.), *The Routledge companion to philosophy of medicine* (pp. 16-26). New York: Routledge.

Hoffman, J. R., & Cooper, R. J. (2012). Overdiagnosis of disease: A modern epidemic. *Archives of Internal Medicine, 172*(15), 1123-1124. doi: 10.1001/archinternmed.2012.3319

Hoffmann, J., & Rosenkrantz, G. S. (1998). On the unity of compound things: Living and non-living. *Ratio, 11*(3), 289-315. doi: 10.1111/1467-9329.00072

Hofweber, T. (2009). The meta-problem of change. *Noûs, 43*(2), 286-314. doi: 10.1111/j.1468-0068.2009.00707.x

Holm, S. (2014). Disease, dysfunction, and synthetic biology. *Journal of Medicine and Philosophy, 39*(4), 329-345. doi: 10.1093/jmp/jhu025

Hommen, D. (2013). Negative properties, real and irreducible. *Philosophia Naturalis, 50*(2), 383-406. doi: 10.3196/003180213815620415

Hommen, D. (2014). Moore and Schaffer on the ontology of omissions. *Journal for General Philosophy of Science, 45*(1), 71-89. doi: 10.1007/s10838-013-9224-6

Hommen, D. (2018). Negative properties – negative objects?. *Acta Analytica, 33*(4), 396-412. doi: 10.1007/s12136-018-0341-z

Hommen, D. (2019). Kinds as universals: A neo-Aristotelian approach. *Erkenntnis*. doi: 10.1007/s10670-019-00105-6

Horn, L. (1989). *A natural history of negation*. Chicago: University of Chicago Press.

Hornsby, J. (1997). *Simple mindedness: A defense of naive naturalism in the philosophy of mind*, Cambridge, MA; London: Harvard University Press.

Horwitz, A. V. (2002). *Creating mental illness*. Chicago: University of Chicago Press.

Horwitz, A. V. & Wakefield, J. C. (2007). *The loss of sadness: How psychiatry transformed normal sorrow into depressive disorder*. New York: Oxford University Press.

Howick, J. H. (2011). *The Philosophy of evidence-based medicine*. Oxford: Wiley-Blackwell.

Hucklenbroich, P. (2014). "Disease entity" as the key theoretical concept of medicine. *Journal of Medicine and Philosophy, 39*(1), 609-633. doi: 10.1093/jmp/jhu040

Hucklenbroich, P. (2016). Die Normativität des Krankheitsbegriff: Zur Genese und Geltung von Kriterien der Krankhaftigkeit. *Analyse & Kritik, 38*(2), 459-496.

Hucklenbroich, P. (2017). Disease entities and the borderline between health and disease: Where is the place of gradation?. In G. Keil, L. Keuck, & R. Hauswald (Eds.), *Vagueness in psychiatry* (pp. 75-92). Oxford: Oxford University Press.

Hucklenbroich, P. (2018). Disease entities, negative causes, multifactoriality, and the naturalness of disease classifications: Remarks on some philosophical misperceptions of medical pathology. In A. Christian, D. Hommen, N. Retzlaff, & G. Schurz (Eds.), *Philosophy of science: Between the natural sciences, the social sciences, and the humanities* (pp. 137-152). Dordrecht: Springer.

Hull, D. L. (1965a). The effect of essentialism on taxonomy – Two thousand years of stasis (I). *British Journal for the Philosophy of Science, 15*(60), 314-326. doi: 10.1093/bjps/XV.60.314

Hull, D. L. (1965b). The effect of essentialism on taxonomy – Two thousand years of stasis (II). *British Journal for the Philosophy of Science, 16*(61), 1-18. doi: 10.1093/bjps/XVI.61.1

Hull, D. L. (1978). A matter of individuality. *Philosophy of Science, 45*(3), 335-360. doi: 10.1086/288811

Hume, D. (1748 [1910]). *An inquiry concerning human understanding*. London: P. F. Collier & Son.

Hurka, T. (1998). Two kinds of organic unity. *The Journal of Ethics, 2*(4), 299-320. doi: 10.1023/a:1009795120631

Hurst, S. A. (2008). Vulnerability in research and health care; Describing the elephant in the room?. *Bioethics, 22*(4), 191-202. doi: 10.1111/j.1467-8519.2008.00631.x

Hursthouse, R. (1991). Arational actions. *Journal of Philosophy, 88*(2), 57-68. doi: 10.2307/2026906

Hursthouse, R. (1999). *On virtue ethics*. Oxford: Oxford University Press.

Hutchinson, J. (1879). On ophthalmoplegia externa, or symmetrical immobility (partial) of the eyes, with ptosis. *Medico-Chirurgical Transactions, 62*, 307-329. doi: 10.1177/095952877906200120

Huxley, J. (1974). *Evolution: The modern synthesis* (3rd ed.). London: Allen & Unwin.

Hyde, D., & Raffman, D. (2018). Sorites paradox. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/sorites-paradox/

Illari, P. McKay (2011). Mechanistic evidence: Disambiguating the Russo-Williamson thesis. *International Studies in the Philosophy of Science, 25*(2), 139-157. doi: 10.1080/02698595.2011.574856

Illari, P. McKay (2013). Mechanistic explanation: Integrating the ontic and epistemic. *Erkenntnis, 78*(2), 237-255. doi: 10.1007/s10670-013-9511-y

Inan, I. (2008). Rigid general terms and essential predicates. *Philosophical Studies, 140*(2), 213-228. doi: 10.1007/s11098-007-9138-4

Ingthorsson, R. D. (2013). The natural vs. the human sciences: Myth, methodology and ontology. *Discusiones Filosóficas, 14*(22), 25-41.

Inman, R. D. (2017). *Substance and the fundamentality of the familiar: A neo-Aristotelian mereology*. New York: Routledge.

Jablensky, A. (2007). Does psychiatry need an overarching concept of "mental disorder"?. *World Psychiatry, 6*(3), 157-158.

Jablensky, A. (2012). The nosological entity in psychiatry: A historical illusion or a moving target?. In K. S. Kendler, & J. Parnas (Eds.), *Philosophical issues in psychiatry II: Nosology* (pp. 77-94). Oxford: Oxford University Press.

Jablonka, E., & Lamb, M. J. (2014). *Evolution in four dimensions: Genetic, epigenetic, behavioral, and symbolic variation in the history of life*. Cambridge, MA: MIT Press.

James, W. (1884). What is an emotion?. *Mind, 9*, 188-205.

Janich, P. (2005). Wissenschaftsphilosophie als kritische Reflexion auf eine historische Praxis. In B. Gesang (Ed.), *Deskriptive oder normative Wissenschaftstheorie?* (pp. 145-166). Frankfurt: Ontos Verlag.

Jansen, L. (2018). Functions, malfunctioning, and negative causation. In A. Christian, D. Hommen, N. Retzlaff, & G. Schurz (Eds.), *Philosophy of science: Between the natural sciences, the social sciences, and the humanities* (pp. 117-135). Dordrecht: Springer.

Jansen, L., & Smith, B. (Eds.). (2008). *Biomedizinische Ontologie: Wissen strukturieren für den Informatik-Einsatz*. Zürich: Vdf Hochschulverlag.

Jaquet, F., & Naar, H. (2019). *Qui peut sauver la morale? Essai de métaéthique*. Paris: Ithaque.

Jaworski, W. (2016). *Structure and the metaphysics of mind: How hylomorphism solves the mind-body problem*. Oxford: Oxford University Press.

Jensen, U. J. (1984). A critique of essentialism in medicine. In L. Nordenfelt, & B. I. B. Lindahl (Eds.), *Health, disease, and causal explanations in medicine* (pp. 63-73). Dordrecht: D. Reidel.

Johansson, I. (2011). Toutes les relations sont internes – La nouvelle version. *Philosophiques, 38*(1), 219-239. doi: 10.7202/1005724ar

Johansson, I., & Lynøe, N. (2008). *Medicine and philosophy: A twenty-first century introduction*. Frankfurt: Ontos Verlag.

Johnson, W. E. (1921). *Logic* (Part 1). Cambridge: Cambridge University Press.

Jones, N. K. (2016a). A higher-order solution to the problem of the concept *Horse*. *Ergo: An Open Access Journal of Philosophy, 3*(6), 132-166. doi: 10.3998/ergo.12405314.0003.006

Jones, N. K. (2016b). *Object* as a determinable. In M. Jago (Ed.), *Reality making* (pp. 121-151). Oxford: Oxford University Press.

Juengst, E. T. (2000). Concepts of disease after the Human Genome Project. In S. Wear, J. J. Bono, G. Logue, & A. McEvoy (Eds.), *Ethical issues in health care on the frontiers of the twenty-first century* (pp. 127-154). London: Kluwer Academic Publishers.

Juengst, E. T., & Moseley, D. (2019). Human enhancement. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/enhancement/

Jukola, S. (2017). On ideals of objectivity, judgments, and bias in medical research - A comment on Stegenga. *Studies in History and Philosophy of Biological and Biomedical Sciences, 62*, 35-41. doi: 10.1016/j.shpsc.2017.02.001

Kagan, S. (1998). Rethinking intrinsic value. *The Journal of Ethics, 2*(4), 277-297. doi: 10.1023/a:1009782403793

Kästner, L. (2017). *Philosophy of cognitive neuroscience: Causal explanations, mechanisms, and experimental manipulations*. Berlin: de Gruyter.

Kästner, L., & Andersen, L. M. (2018). Intervening into mechanisms: Prospects and challenges. Philosophy Compass, 13(11), e12546. doi: 10.1111/phc3.12546

Kaiser, M. I. (2015). *Reductive explanation in the biological sciences*. Cham: Springer.

Kaiser, M. I. (2018a). The components and boundaries of mechanisms. In S. Glennan, & P. Illari (Eds.), *The Routledge handbook of mechanisms and mechanical philosophy* (pp. 116-130). New York: Routledge.

Kaiser, M. I. (2018b). Individuating part-wholes relations in the biological world. In O. Bueno, R.-L. Chen, & M. B. Fagan (Eds.), Individuation, process, and scientific practices (pp. 63-89). New York: Oxford University Press.

Kaiser, M. I., & Krickel, B. (2017). The metaphysics of constitutive mechanistic phenomena. *British Journal for the Philosophy of Science, 68*(3). doi: 10.1093/bjps/axv058

Karindas, M. M. (2017). The multicellular origin of cancer and the evolution of oncogenesis. *Madridge Journal of Cancer Study & Research*, 1(1), 1-7. doi: 10.18689/mjcsr-1000101

Karmo, T. (1977). Disturbances. *Analysis, 37*(4), 147-148. doi: 10.1093/analys/37.4.147

Kasper, D. L., Fauci, A. S., Hauser, S. L., Longo, D. L., Jameson, J. L., & Loscalzo, J. (Eds.). (2015). *Harrison's principles of internal medicine* (19th ed.). New York: McGraw-Hill.

Kaye, E. T., & Kaye, K. M. (2015). Fever and rash. In D. L. Kasper, A. S. Fauci, S. L. Hauser, D. L. Longo, J. L. Jameson, & J. Loscalzo (Eds.), *Harrison's principles of internal medicine* (19th ed.) (pp. 127-134). New York: McGraw-Hill.

Kay Toombs, S. (1993). *The meaning of illness: A phenomenological account of the different perspectives of physician and patient*. Dordrecht: Kluwer.

Kearns, T. P. (1965). External ophthalmoplegia, pigmentary degeneration of the retina, and cardiomyopathy: A newly recognized syndrome. *Transactions of the American Ophthalmological Society, 63*, 559-625.

Kearns, T. P., & Sayre, G. P. (1958). Retinitis pigmentosa, external ophthalmoplegia, and complete heart block: Unusual syndrome with histologic study in one of two cases. *American Medical Association Archives of Ophthalmoplegia, 60*, 280-289.

Keil, F. (1989). *Concepts, kinds, and cognitive development*. *Cambridge, MA: MIT Press.*

Keil, G., Keuck, L., & Hauswald, R. (Eds.). (2017). *Vagueness in psychiatry*. Oxford: Oxford University Press.

Keinänen, M., & Tahko, T. E. (2019). Bundle theory with kinds. *Philosophical Quarterly, 69*(277), 838-857. doi: 10.1093/pq/pqz021

Kendell, R. E. (1993). The nature of psychiatric disorders. In R. E. Kendell, & A. K. Zeally (Eds.), *Companion to psychiatric studies* (5th ed.) (pp. 1-7). Edinburgh: Churchill Livingston.

Kendell, R. E., & Jablensky, A. (2003). Distinguishing between the validity and utility of psychiatric diagnoses. *American Journal of Psychiatry, 160*(1), 4-12. doi: 10.1176/appi.ajp.160.1.4

Kendig, C. (Ed.). (2016). *Natural kinds and classification in scientific practice*. London: Routledge.

Keskinen, A., Hakkarainen, J., & Keinänen, M. (2015). Concrete universals and spatial relations. *European Journal of Analytic Philosophy, 11*(1), 57-71.

Khalidi, M. A. (2013). *Natural categories and human kinds: Classification in the natural and social sciences*. Cambridge: Cambridge University Press.

Kincaid, H. (2012). Mechanisms, causal modeling, and the limitations of traditional multiple regression. In H. Kincaid (Ed.), *The Oxford handbook of philosophy of social science* (pp. 46-64). Oxford: Oxford University Press.

Kincaid, H., & Sullivan, J. A. (Eds.). (2014). *Classifying psychopathology: Mental kinds and natural kinds*. Cambridge, MA: MIT Press.

Kind, A. (Ed.). (2016). *The Routledge handbook of philosophy of imagination*. London; New York: Routledge.

King, C. D. (1945). The meaning of normal. *Yale Journal of Biology and Medicine 17*(3), 493-501.

King, L. S. (1982). *Medical thinking*. Princeton: Princeton University Press.

Kingma, E. (2007). What is it to be healthy?. *Analysis, 67*(294), 128-133. doi: 10.1093/analys/67.2.128

Kingma, E. (2010). Paracetamol, poison, and polio: Why Boorse's account of function fails to distinguish health and disease. *British Journal for the Philosophy of Science, 61*(2), 241-264. doi: 10.1093/bjps/axp034

Kirchin, S. (2017). *Thick evaluation*. Oxford: Oxford University Press.

Kirchhoff, M. D. (2018). The Markov blankets of life: Autonomy, active inference and the free energy principle. *Journal of the Royal Society Interface, 15*(138).

Kistler, M. (2004). La causalité dans la philosophie contemporaine. *Intellectica, 38*(1), 139-185.

Kitcher, P. (1984). Species. *Philosophy of Science, 51*(2), 308-333. doi: 10.1086/289182

Kitcher, P. (2001). Battling the undead: How (and how not) to resist genetic determinism. In R. Singh, K. Krimbas, D. Paul, & J. Beatty (Eds.), *Thinking about evolution: Historical, philosophical and political perspectives* (pp. 396-414). Cambridge: Cambridge University Press.

Klima, G. (2002). Contemporary essentialism vs. Aristotelian essentialism. In J. Haldane (Ed.), *Mind, metaphysics, and value in the Thomistic and analytic traditions* (pp. 175-194). Notre Dame: University of Notre Dame Press.

Kment, B. (2014). *Modality and explanatory reasoning*. New York: Oxford University Press.

Knobe, J., & Nichols, S. (Eds.). (2008). *Experimental philosophy*. New York: Oxford University Press.

Kobasa, D., Jones, S. M., Shinya, K., …Kawaoka, Y. (2007). Aberrant innate immune response in lethal infection of macaques with the 1918 influenza virus. *Nature, 445*(7125), 319-323. doi: 10.1038/nature05495

Kompa, N., Nimtz, C., & Suhm, C. (Eds.). (2009). *The a priori and its role in philosophy*. Paderborn: Mentis.

Korman, D. Z. (2015). *Objects: Nothing out of the ordinary*. Oxford: Oxford University Press.

Korsgaard, C. M. (2008). *The constitution of agency: Essays on practical reason and moral psychology*. Oxford: Oxford University Press.

Koslicki, K. (2003). The crooked path from vagueness to four-dimensionalism. *Philosophical Studies, 114*(1-2), 107-134. doi: 10.1023/a:1024413302197

Koslicki, K. (2008). *The structure of objects*. Oxford, UK: Oxford University Press.

Koslicki, K. (2018). *Form, matter, substance*. New York: Oxford University Press.

Kovaka, K. (2015). Biological individuality and scientific practice. *Philosophy of Science, 82*(5), 1092-1103. doi: 10.1086/683443

Krickel, B. (2018). *The mechanical world: The metaphysical commitments of the new mechanistic approach*. Dordrecht: Springer.

Krieger, N. (1994). Epidemiology and the web of causation: Has anyone seen the spider?. *Social Science & Medicine, 39*(7), 887-903. doi: 10.1016/0277-9536(94)90202-X

Kripke, S. (1980). *Naming and necessity*. Cambridge, MA: Harvard University Press.

Kroes, P. (2001). Technical functions as dispositions: A critical assessment. *Techné: Research in Philosophy and Technology, 5*(3), 105-115. doi: techne2001531

Krohs, U. (2009). Functions as based on a concept of general design. *Synthese, 166*(1), 69-89. doi: 10.1007/s11229-007-9258-6

Krohs, U. (2011). Functions and fixed types: Biological and other functions in the post-adaptationist era. *Applied Ontology, 6*(2), 125-139. doi: 10.3233/AO-2011-0089

Krohs, U., & Kroes, P. (Eds.). (2009). *Functions in biological and artificial worlds: Comparative philosophical perspectives*. Cambridge, MA: MIT Press.

Kronfeldner, M. (2009). Genetic determinism and the innate-acquired distinction. *Medicine Studies, 1*(2), 167-181. doi: 10.1007/s12376-009-0014-8

Kronfeldner, M. (2018). *What's left of human nature? A post-essentialist, pluralist and interactive account of a contested concept*. Cambridge, MA: MIT Press.

Künne, W. (2003). *Conceptions of truth*. Oxford: Clarendon Press.

Kumar, S., Srinivasan, A., & Nikolajeff, F. (2018). Role of infrared spectroscopy and imaging in cancer diagnosis. *Current Medicinal Chemistry, 25*(9), 1055-1072. doi: 10.2174/0929867324666170523121314

Kuo, H.-K., & Fujise, K. (2011). Human papillomavirus and cardiovascular disease among U.S. women in the National Health and Nutrition Examination Survey, 2003 to 2006. *Journal of the American College of Cardiology, 58*(19), 2001-2006. doi: 10.1016/j.jacc.2011.07.038

Kuperus, J. S., Waalwijk, J. F., Regan, E. A., …Verlan, J.-J. (2018). Simultaneous occurrence of ankylosing spondylitis and diffuse idiopathic skeletal hyperostosis: A systematic review. *Rheumatology*, *57*(12), 2120-2128. doi: 10.1093/rheumatology/key211

Kurth, C. (2018). *The anxious mind: An investigation into the varieties and virtues of anxiety*. Cambridge, MA; London: MIT Press.

Kurtz, J., & Hemming, R. (2014). Krankheit und Evolution. In J. S. Ach, B. Lüttenberg, & M. Quante (Eds.), *Wissen.leben.ethik* (pp. 193-201). Münster: Mentis.

Ladyman, J., & Ross, D. (2007). *Everything must go: Metaphysics naturalized*. New York: Oxford University Press.

Laing, R. D. (1969). *The politics of the family and other essays*. London: Routledge.

Laland, K. N., Uller, T., Feldman, M. W., Sterelny, K., Müller, G. B., Moczek, A., Jablonka, E., & Odling-Smee, J. (2015). The extended evolutionary synthesis: Its structure, assumptions and predictions. *Proceedings of the Royal Society B: Biological Sciences, 282*(1813). doi: 10.1098/rspb.2015.1019

Lalumera, E. (2010). Concepts are a functional kind. *Behavioral and Brain Sciences, 33*(2-3), 217-218. doi: 10.1017/S0140525X10000403

Lalumera, E. (2016). Saving the DSM-5? Descriptive conceptions and theoretical concepts of mental disorders. *Medicina & Storia, 9-10*, 109-129.

Landes, J., Osimani, B., & Poellinger, R. (2018). Epistemology of causal inference in pharmacology: Towards a framework for the assessment of harms. *European Journal for Philosophy of Science, 8*(1), 3-49. doi: 10.1007/s13194-017-0169-1

Lange, M. (2007). The end of diseases. *Philosophical Topics, 35*(1-2), 265-292. doi: 10.5840/philtopics2007351/212

Laplane, L. (2016). *Cancer stem cells: Philosophy and therapies*. Cambridge, MA: Harvard University Press.

Laplane, L., Duluc, D., Larmonier, N., Pradeu, T., & Bikfalvi, A. (2018). The multiple layers of the tumor environment. *Trends in Cancer, 4*(12). doi: 10.1016/j.trecan.2018.10.002

LaPorte, J. (2004). *Natural kinds and conceptual change*. Cambridge: Cambridge University Press.

LaPorte, J. (2013). *Rigid designation and theoretical identities*. Oxford: Oxford University Press.

LaPorte, J. (2016). Rigid designators. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/rigid-designators/

Lauria, F. (2019). La mort (grand public). In M. Kristanek (Ed.), L'Encyclopédie Philosophique. URL: http://encyclo-philo.fr/mort-gp/

Leary, S. (2017). Non-naturalism and normative necessities. In R. Shaffer-Landau (Ed.), *Oxford studies in metaethics* (vol. 12) (pp. 76-105). Oxford: Oxford University Press.

Legendre, M., Lartigue, A., Bertaux, L., …Claverie, J.-M. (2015). In-depth study of Mollivirus sibericum, a new 30,000-y-old giant virus infecting Acanthamoeba. *Proceedings of the National Academy of Sciences of the Unites States of America, 112*(38), E5327-E5335. doi: 10.1073/pnas.1510795112

Leibniz, G. W. (1710). *Essais de théodicée sur la bonté de Dieu, la liberté de l'homme et l'origine du mal*. Amsterdam: I. Troyel.

Lejeune, J., Gautier, M., & Turpin, R. (1959). Etude des chromosomes somatiques de neuf enfants mongoliens. *Comptes Rendus Hebdomadaires des Séances de l'Académie des Sciences, 248*(11), 1721-1722.

Lemeire, O. (2018). No purely epistemic theory can account for the naturalness of kinds. *Synthese*, 1-19. doi: 10.1007/s11229-018-1806-8

Lemoine, M. (2013). Defining disease beyond conceptual analysis: an analysis of conceptual analysis in philosophy of medicine. *Theoretical Medicine and Bioethics, 34*(4), 309-25. doi: 10.1007/s11017-013-9261-5

Lemoine, M. (2015). The naturalization of the concept of disease. In P. Huneman, G. Lambert, & M. Silberstein (Eds.), *Classification, disease and evidence* (pp. 19-42). Dordrecht: Springer.

Lemoine, M. (2017). Explanation in medicine. In M. Solomon, J. R. Simon, & H. Kincaid (Eds.), *The Routledge companion to philosophy of medicine* (pp. 296-309). New York: Routledge.

Lennox, J. G. (1993). Darwin was a teleologist. *Biology and Philosophy, 8*(4), 409-421. doi: 10.1007/BF00857687

Lennox, J. G. (2001). *Aristotle's philosophy of biology: Studies in the origins of life sciences*. New York: Cambridge University Press.

Lepine, S. (2015). Le rationalisme moral face à la psychopathie. *Implications Philosophiques, 2015*.

Lepore, E., & Stone, M. (2015). *Imagination and convention*. Oxford: Oxford University Press.

Leunissen, M. (2010). *Explanation and teleology in Aristotle's science of nature*. Cambridge: Cambridge University Press.

Levin, J. (2018). Functionalism. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/functionalism/

Lewens, T. (2004). *Organisms and artifacts: Design in nature and elsewhere*. Cambridge, MA: MIT Press.

Lewens, T. (2012). Species, essence and explanation. *Studies in History and Philosophy of Biological and Biomedical Sciences, 43*(4), 171-757. doi: 10.1016/j.shpsc.2012.09.013

Lewis, D. K. (1986). *On the plurality of worlds*. Oxford: Blackwell.

Lewontin, R. C., & Birch. L. C. (1966). Hybridization as a source of variation for adaptation to new environments. *Evolution, 20*(3), 315-336. doi: 10.1111/j.1558-5646.1966.tb03369.x

Liberal dictionary. (2009). *Homogentisic acid*. URL: https://www.liberaldictionary.com/homogentisic-acid/

Lidgard, S., & Nyhart, L. K. (Eds.). (2017). *Biological individuality: Integrating scientific, philosophical, and historical perspectives*. Chicago: University of Chicago Press.

Linnaeus, C. (1735). *Systema naturae, siue regna tria naturae systematice proposita per classes, ordines, genera & species*. Leiden: Theodore Haak.

Lister Hill National Center for Biomedical Communications. (2020). Bethesda: National Library of Medicine.

Lloyd , E. A. (2015). Adaptationism and the Logic of Research Questions: How to Think Clearly About Evolutionary Causes. *Biological Theory* (2015) 10: 343.

Lloyd, E. A., & Gould, S. J. (2017). Exaptation revisited: Changes imposed by evolutionary psychologists and behavioral biologists. Biological Theory, 12(1), 50-65. doi: 10.1007/s13752-016-0258-y

Lötvall, J., Akdis, C. A., Bacharier, L. B., …Greenberger, P. A. (2011). Asthma endotypes: A new approach to classification of disease entities within the asthma syndrome. *Journal of Allergy and Clinical Immunology, 127*(2), 355-360. doi: 10.1016/j.jaci.2010.11.037

Loughlin, M., & Miles, A. (2015). Psychiatry, objectivity, and realism about value. In P. Zachar, D. St. Stoyanov, M. Aragona, & A. Jablensky (Eds.), *Alternative perspectives on psychiatric validation: DSM, IDC, RDoC, and beyond* (pp. 146-163). New York: Oxford University Press.

Loux, M. J. (2006). Aristotle's constituent ontology. In D. W. Zimmerman (Ed.), *Oxford studies in Metaphysics* (vol. 2) (pp. 207-250). New York: Oxford University Press.

Lowe, E. J. (1998). The possibility of metaphysics: Substance, identity, and time. Oxford: Clarendon Press.

Lowe, E. J. (2006). *The four-category ontology: A metaphysical foundation for natural science*. New York: Oxford University Press.

Lowe, E. J. (2008). Two notions of being: Entity and essence. *Royal Institute of Philosophy Supplement, 83*(62), 23-48. doi: 10.1017/s1358246108000568

Lowe, E. J. (2011). The rationality of metaphysics. *Synthese, 178*(1), 99-109. doi: 10.1007/s11229-009-9514-z

Lowe, E. J. (2014). How real are artefacts and artefact kinds?. In M. Franssen, P. Kroes, T. Reydon, & P. E. Vermaas (Eds.), *Artefact kinds: Ontology and the human-made world* (pp. 17-26). Cham: Springer.

Ludwig, A. M. (1975). The psychiatrist as physician. *JAMA, 234*(6), 603-604.

Ludwig, K. (2010). Intuitions and relativity. *Philosophical Psychology, 23*(4), 427-445. doi: 10.1080/09515089.2010.505877

Luper, S. (2019). Death. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/death/

Luzzato, L. (2012). Sicle cell anaemia and malaria. *Mediterranean Journal of Hematology and Infectious Diseases, 4*(1), e2012065. doi: 10.4084/MJHID.2012.065

McDaniel, K. (2017). *The fragmentation of being*. Oxford: Oxford University Press.

McDowell, J. (1985). Values and secondary qualities. In T. Honderich (Ed.), *Morality and objectivity* (pp. 110-129). London: Routledge.

McDowell, J. (1994). *Mind and world: With a new introduction*. Cambridge, MA; London: Harvard University Press.

McDowell, J. (1998). *Mind, value, and reality*. Cambridge, MA; London: Harvard University Press.

McGrath, S. (2005). Causation by omission: A dilemma. *Philosophical Studies, 123*(1-2), 125-148. doi: 10.1007/s11098-004-5216-z

McLaughlin, B., & Bennett, K. (2018). Supervenience. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/supervenience/

McLaughlin, P. (2001). *What functions explain: Functional explanation and self-reproducing systems*. New York: Cambridge University Press.

McManus, C. (2019). Half a century of handedness research: Myths, truths; fictions, facts; backwards, but mostly forwards. *Brain and Neuroscience Advances, 3*, 1-10. doi: 10.1177/2398212818820513

Macarthur, D. (2017). Liberal naturalism, ordinary things and second-personal space. In A. Garre & W. Hudson (Eds.), *For a new naturalism* (pp. 50-65). Candor, NY: Telos Press.

Macarthur, D. (2019). Liberal naturalism and the scientific image of the world. *Inquiry: An Interdisciplinary Journal of Philosophy, 62*(5), 565-585. doi: 10.1080/0020174X.2018.1484006

Machado, C. (2010). Diagnosis of brain death. *Neurology International, 2*(1), e2. doi: 10.4081/ni.2010.e2

Machamer, P., Darden, L., & Craver, C. F. (2000). Thinking about mechanisms. *Philosophy of Science, 67*(1), 1-25. doi: 10.1086/392759

Machery, E. (2009*). Doing without concepts*. New York: Oxford University Press.

Machery, E. (2017). *Philosophy within its proper bounds*. New York: Oxford University Press.

Mackie, J. L. (1974). The cement of the universe: A study of causation. Oxford: Clarendon Press.

Mackie, J. L. (1977). *Ethics: Inventing right and wrong*. London: Pelican Books.

Macleod, M., & Reydon, T. A. C. (2013). Natural kinds in philosophy and in the life sciences: Scholastic twilight or new dawn?. *Biological Theory, 7*(2), 89-99. doi: 10.1007/s13752-012-0080-0

Magnus, P. D. (2012). *Scientific enquiry and natural kinds: From planets to mallards*. Basingstoke: Palgrave Macmillan.

Mahon, J. E. (2015). The definition of lying and deception. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/lying-definition/

Maienschein, J. (2005). Epigenesis and preformationism. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/epigenesis/

Mandolesi, L., Gelfo, F., Serra, L., …Sorrentino, G. (2017). Environmental factors promoting neural plasticity: Insights from animal and human studies. *Neural Plasticity, 2017*, 7219461. doi: 10.1155/2017/7219461

Marcovitch, H. (Ed). (2010). *Black's medical dictionary* (42nd ed.). London: Bloomsbury.

Marcum, J. A. (2008). *An introductory philosophy of medicine: Humanizing modern medicine*. Dordrecht: Springer.

Marcus, E. (2009). Why there are no token states. *Journal of Philosophical Research, 34*, 215-241. doi: 10.5840/jpr_2009_17

Margolis, E., & Laurence, S. (2019). Concepts. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/concepts/

Marmodoro, A., & Yates, D. (Eds.). (2016). *The metaphysics of relations*. Oxford: Oxford University Press.

Marsili, N. (2019). Immoral lies and partial beliefs. *Inquiry*. doi: 10.1080/0020174x.2019.1667865

Martin, E. A. (2010). *Concise medical dictionary* (8th ed.). Oxford: Oxford University Press.

Massin, O. (2008). Impenetrability. *Swiss Philosophical Preprints Series, 13*.

Massin, O. (2010). *Introduction à la philosophie des valeurs*.

Massin, O. (2014). Quand vouloir, c'est faire. In R. Clot-Goudard (Dir.), *L'explication de l'action : Analyses contemporaines* (pp. 79-114). Paris: Vrin.

Massin, O. (2015). Book Reviews: Toni Rønnow-Rasmussen, *Personal Value*, Oxford, Oxford University Press, 2011, 185 pp., US$ 75 (Hardback), ISBN 9780199603787. *Dialectica, 69*(2), 221-231. doi: 10.1111/1746-8361.12099

Matthews, G. B. (1982). Accidental unities. In M. Schofield, & M. C. Nussbaum (Eds.), *Language and logos* (pp. 223-240). Cambridge: Cambridge University Press.

Matthewson, J., & Griffiths, P. E. (2017). Biological criteria of disease: Four ways of going wrong. *Journal of Medicine and Philosophy, 42*(4), 447-466. doi: 10.1093/jmp/jhx004

Maund, B. (2018). Color. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/color/

Maurin, A.-S. (2018). Tropes. In E. N. Zalta (Ed.), The Stanford Encyclopedia of Philosophy. URL: https://plato.stanford.edu/entries/tropes/

Mayo, D. G. (1996). *Error and the growth of experimental knowledge*. Chicago: University of Chicago Press.

Mayr, E. (1942*). Systematics and the origin of species from the viewpoint of a sociologist*. New York: Columbia University Press.

Mayr, E. (1963). *Animal species and evolution*. Cambridge, MA: Harvard University Press.

Mayr, E. (1988). *Toward a new philosophy of biology: Observations of an evolutionist*. Cambridge, MA; London: Harvard University Press.

Mayr, E., & Provine, W. (Eds.). (1980). *The evolutionary synthesis: Perspectives on the unification of biology*. Cambridge, MA: Harvard University Press.

Mebius, A. (2014). A weakened mechanism is still a mechanism: On the causal role of absences in mechanistic explanation. *Studies in History and Philosophy of Biological and Biomedical Sciences, 45*, 43-48. doi: 10.1016/j.shpsc.2013.11.001

MedlinePlus. (2020). *PAH gene*. URL: https://medlineplus.gov/genetics/gene/pah/

Megone, C. (1998a). Aristotle's function argument and the concept of mental illness. *Philosophy, Psychiatry, and Psychology, 5*(3), 187-201.

Megone, C. (1998b). Response to the commentaries. *Philosophy, Psychiatry, and Psychology, 5*(3), 221-224.

Megone, C. (2000). Mental illness, human function, and values. *Philosophy, Psychiatry, & Psychology, 7*(1), 45-65.

Megone, C. (2007). Mental illness, metaphysics, facts and values. *Philosophical Papers, 36*(3), 399-426. doi: 10.1080/05568640709485207

Meincke, A. (2019). Autopoiesis, biological autonomy and the process view of life. *European Journal for Philosophy of Science, 9*. doi: 10.1007/s13194-018-0228-2

Meinong, A. (1904). *Untersuchungen zur Gegenstandstheorie und Psychologie*. Leipzig: Barth.

Meixner, U. (2006). *The theory of ontic modalities*. Heusenstamm: Ontos Verlag.

Mendelovici, A. (2013). Pure intentionalism about moods and emotions. In U. Kriegel (Ed.), *Current controversies in philosophy of* mind (pp. 135-157). London: Routledge.

Merricks, T. (1999). Composition as identity, mereological essentialism, and counterpart theory. *Australasian Journal of Philosophy, 77*(2), 192-195. doi: 10.1080/00048409912348931

Méthot, P.-O. (2015). Darwin, evolution, and medicine: Historical and contemporary perspectives. In T. Heams, P. Huneman, G. Lecointre, & M. Silberstein (Eds.), *Handbook of evolutionary thinking in the sciences* (pp. 587-617). Dordrecht: Springer.

Miller-Keane Encyclopedia and Dictionary of Medicine, Nursing, and Allied Health (7th ed.). (2003). Philadelphia: Saunders.

Mitchell, S. D. (2003). *Biological complexity and integrative pluralism*. New York: Cambridge University Press.

Modrak, D. (2010). Nominal definition in Aristotle, his successors and his predecessors. In D. Charles (Ed.), *Definition in Greek philosophy* (pp. 252-285). New York: Oxford University Press.

Moghaddam-Taaheri, S. (2011). Understanding pathology in the context of physiological mechanisms: The practicality of a broken-normal view. *Biology and Philosophy, 26*(4), 603-611. doi: 10.1007/s10539-011-9258-2

Moncrieff, J. (2008). *The myth of the chemical cure: A critique of psychiatric drug treatment*. New York: Palgrave Macmillan.

Montévil, M., & Mossio, M. 2015). Biological organisation as closure of constraints. *Journal of Theoretical Biology, 372*, 179-191. doi: 10.1016/j.jtbi.2015.02.029

Moore, D. (2015). Mereological essentialism and mereological inessentialism. *Croatian Journal of Philosophy, 15*(43), 67-85.

Moore, G. E. (1903). *Principia ethica*. Cambridge: Cambridge University Press.

Moore, G. E. (1919). External and internal relations. *Proceedings of the Aristotelian Society, 20*, 40-62.

Moore, M. S. (2009). *Causation and responsibility: An essay in law, morals, and metaphysics*. Oxford: Oxford University Press.

Moravcsik, J. M. E. (1973). Plato's method of division. In J. M. E. Moravcsik (Ed.), *Patterns in Plato's thought* (pp. 158-180). Dordrecht: Springer.

Moreno, A., & Mossio, M. (2015). *Biological autonomy: A philosophical and theoretical enquiry*. Dordrecht: Springer.

Morgan, T. H. (1901). Regeneration in the egg, embryo, and adult. *American Naturalist, 35*(420), 949-973.

Morganti, M. (2009). Ontological priority, fundamentality and monism. *Dialectica, 63*(3), 271-288. doi: 10.1111/j.1746-8361.2009.01197.x

Mortensen, C. (2020). Change and inconsistency. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/change/

Mosby's medical dictionary (10th ed.) (2016). Amsterdam: Elsevier.

Mossio, M., & Moreno, A. (2010). Organisational closure in biological organisms. *History and Philosophy of the Life Sciences, 32*(2/3), 269-288.

Mossio, M., Saborido, C., & Moreno, A. (2009). An organizational account of biological functions. *British Journal for the Philosophy of Science, 60*(4), 813-841. doi: 10.1093/bjps/axp036

Mourelatos, A. P. D. (1978). Events, processes, and states. *Linguistics and Philosophy, 2*(3), 415-434. doi: 10.1007/BF00149015

Müller-Strahl, G. (2014). Mechanisms, patho-mechanisms, and the explanation of disease in scientifically based clinical medicine. In M. I. Kaiser, O. Scholz, D. Plenge, & A. Hüttemann

(Eds.), *Explanation in the special science: The case of biology and history* (pp. 99-130). Dordrecht: Springer.

Mulligan, K. (1998). Relations through thick and thin. *Erkenntnis, 48*(2-3), 325-353. doi: 10.1023/a:1005454805376

Mulligan, K. (2008). Propriétés, processus et priorités. In C. Erismann, & A. Schniewind (Eds.), *Compléments de substance: Etudes sur les propriétés accidentelles offertes à Alain de Libera* (pp. 231-247). Paris: Vrin.

Mulligan, K. (2009). Values. In R. Le Poidevin, P. Simons, A. McGonigal, & R. P. Cameron (Eds.), *The Routledge Companion to Metaphysics* (pp. 401-412). Londres ; New York : Routledge.

Mulligan, K., Simons, P., & Smith, B. (1984). Truth-makers. *Philosophy and Phenomenological Research, 44*(3), 287-321. doi: 10.2307/2107686

Munn, K., & Smith, B. (Eds.). (2008). *Applied ontology: An introduction*. Heusenstamm: Ontos Verlag.

Muñoz, N., Castellsagué, X., Berrington de González, A., & Gissmann, L. (2006). Chapter 1: HPV in the etiology of human cancer. *Vaccine, 24*(S3), S1-S10. doi: 10.1016/j.vaccine.2006.05.115

Murphy, D. (2006). *Psychiatry in the scientific image*. Cambridge, MA: MIT Press.

Murphy, D., & Woolfolk, R. L. (2000a). Conceptual analysis versus scientific understanding: An assessment of Wakefield's folk psychiatry. *Philosophy, Psychiatry, and Psychology, 7*(4), 271-293.

Murphy, D., & Woolfolk, R. L. (2000b). The harmful dysfunction analysis of mental disorder. *Philosophy, Psychiatry, and Psychology, 7*(4), 241-252.

Nagel, T. (1970). Death. *Noûs, 4*(1), 73-80. doi: 10.2307/2214297

Nagel, T. (1974). What is it like to be a bat?. *Philosophical Review, 83*, 435-450. doi: 10.2307/2183914

Nagel, T. (1986). *The view from nowhere*. New York; Oxford: Oxford University Press.

Nagel, T. (1997). *The last word*. New York; Oxford: Oxford University Press.

National Center for Biomedical Ontology. (2020). URL: https://www.bioontology.org/

National Health and Medical Research Council. (2015). *Statement on homeopathy*. URL: https://www.nhmrc.gov.au/about-us/resources/homeopathy#

National Institute of Diabetes and Digestive and Kidney Diseases. (2020). *Lactose intolerance*. URL: https://www.niddk.nih.gov/health-information/digestive-diseases/lactose-intolerance

National Institute of Mental Health. (2020). *Research Domain Criteria* (RDoC). URL: https://www.nimh.nih.gov/research/research-funded-by-nimh/rdoc/index.shtml

National Institute of Neurological Disorders and Stroke. (2020). *Neurofibromatosis fact sheet*. URL: https://www.ninds.nih.gov/Disorders/Patient-Caregiver-Education/Fact-Sheets/Neurofibromatosis-Fact-Sheet

Neander, K. (1991). Functions as selected effects: The conceptual analyst's defense. *Philosophy of Science, 58*(2), 168-184. doi: 10.1086/289610

Needham, R. (1975). Polythetic classification: Convergence and consequences. *New Series, 10*(3), 349-369. doi: 10.2307/2799807

Nesse, R. M. (2001). On the difficulty of defining disease: A Darwinian perspective. *Medicine, Health Care and Philosophy, 4*(1), 37-46. doi: 10.1023/A:1009938513897

Nesse, R. M. (2005a). Evolutionary psychology and mental health. In D. M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 903-927). London: John Wiley & Sons.

Nesse, R. M. (2005b). Maladaptation and natural selection. *Quarterly Review of Biology, 80*(1), 62-71. doi: 10.1086/431026

Nesse, R. M. (2013). Tinbergen´s four questions, organized: a response to Bateson and Laland. *Trends in Ecology and Evolution, 28*(12), 681-682.

Nesse, R. M. (2016). What is evolutionary medicine? …and 9 other questions answered. In R. M. Nesse (Ed.), *Evolutionary Medicine Blog*. URL: https://www.randolphnesse.com/essays/whatisevolutionarymedicine

Nesse, R. M., & Stein, D. J. (2012). Towards a genuinely medical model for psychiatric nosology. *BMC Medicine, 10*(5). doi: 10.1186/1741-7015-10-5

Nesse, R. M., & Williams, G. C. (1994). *Why we get sick: The new science of Darwinian medicine*. New York: Random House.

Nicholson, D. J. (2018). Reconceptualizing the organism: From complex machine to flowing stream. In D. J. Nicholson, & J. Dupré (Eds.), *Everything flows: Towards a processual philosophy of biology* (pp. 139-166). New York: Oxford University Press.

Nietzsche, F. (1878). *Menschliches, Allzumenschliches*. Chemnitz: Ernst Schmeitzner.

Nietzsche, F. (1889). *Götzen-Dämmerung oder Wie man mit dem Hammer philosophirt*. Leipzig: C. G. Neumann.

Nietzsche, F. *Human, All too human* (2nd ed.). Tr. by R. J. Hollingdale. Cambridge: Cambridge University Press. 1996.

Nimtz, C. (2003). Analytic truths – still harmless after all these years?. *Grazer Philosophische Studien, 66*(1), 91-118. doi: 10.1163/18756735-90000813

Nimtz, C. (2009). Conceptual truth defended. In N. Kompa, C. Nimtz, & C. Suhm (Eds.), *The a priori and its role in philosophy* (pp. 137-155). Paderborn: Mentis.

Nimtz, C. (2010). Saving the doxastic account of intuitions. *Philosophical Psychology, 23*(3), 357-375. doi: 10.1080/09515089.2010.490942

Nimtz, C. (2012). Conceptual truths, strong possibilities and our knowledge of metaphysical necessities. *Philosophia Scientiae, 16*(2), 39-58. doi: 10.4000/philosophiascientiae.735

Nordenfelt, L. Y. (1995). *On the nature of health: An action-theoretic approach* (2nd ed.). Dordrecht: Springer.

Nordenfelt, L. Y. (1998). On medicine and health enhancement – Towards a conceptual framework. *Medicine, Health Care and Philosophy, 1*(1), 5-12. doi: 10.1023/A:1009949517421

Nordenfelt, L. Y. (2000). *Action, ability and health: Essays in the philosophy of action and welfare.* Dordrecht: Springer.

Nordenfelt, L. Y. (2013a). Identification and classification of diseases: Fundamental problems in medical ontology and epistemology. *Studia Philosophica Estonica, 6*(2), 6-21. doi: 10.12697/spe.2013.6.2.02

Nordenfelt, L. Y. (2013b). Standard circumstances and vital goals: Comments on Venkatapuram's critique. *Bioethics, 27*(5), 280-284. doi: 10.1111/j.1467-8519.2011.01958.x

Nordenfelt, L. Y. (2018): Function and health: Towards a praxis-oriented concept of health. Biological Theory, 13(1), 10-16. doi: 10.1007/s13752-017-0270-x

Nussbaum, M. C. (1993). Non-relative virtues: An Aristotelian approach. In M. C. Nussbaum & A. Sen (Eds.), *The quality of life* (pp. 242-270). Oxford: Oxford University Press.

Nussbaum, M. C. (2000). *Women and human development: The capabilities approach*. New York: Cambridge University Press.

O'Connor, T. (2020). Emergent properties. In E. N. Zalta (Ed.), The Stanford Encyclopedia of Philosophy. URL: https://plato.stanford.edu/archives/sum2020/entries/properties-emergent/

Oderberg, D. S. (2007). *Real essentialism*. London: Routledge.

Oderberg, D. S. (2014). The metaphysics of privation. In R. Huntelmann, & J. Hattler (Eds), *New Scholasticism meets analytic philosophy* (pp. 63-68). Heusenstamm: Editiones Scholasticae.

Oderberg, D. S. (2020). *The metaphysics of good and evil*. London: Routledge.

Office of the High Commissioner for Human Rights. (2015). *Human rights and intersex people*.

Ogien, R. (2017). *Mes mille et une nuits: La maladie comme drame et comme comédie*. Paris : Albin Michel.

Ogier de Baulny, H., Abadie, V., Feillet, F., & de Parscau L. (2007). Management of phenylketonuria and hyperphenylalaninemia. *Journal of Nutrition, 137*(6), 1561S-1563S. doi: 10.1093/jn/137.6.1561S

Okasha, S. (2002). Darwinian metaphysics: Species and the question of essentialism. *Synthese, 131*(2), 191-213. doi: 10.1023/A:1015731831011

Olson, E. T. (2006). The paradox of increase. *The Monist, 89*(3), 390-417. doi: 10.5840/monist200689318

Olson, E. T. (2019). Personal identity. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/identity-personal/

O'Malley, M. A. (2013). Metaorganism. In W. Dubitzky, O. Wolkenhauer, H. Yokota, & K.-H. Cho (Eds.), *Encyclopedia of systems biology*. Dordrecht: Springer.

Online Mendelian inheritance in man. (2020). Baltimore: Johns Hopkins University.

Orwell, G. (1949). *Nineteen eighty-four: A novel*. London: Secker & Warburg.

Orzack, S. H., & Sober, E. (Eds.). (2001*). Adaptationism and optimality*. New York: Cambridge University Press.

Osbeck, L. M., & Nersessian N. J. (2015). Prolegomena to an empirical philosophy of science. In S. Wagenknecht, N. J. Nersessian, & H. Andersen (Eds.), *Empirical philosophy of science: Introducing qualitative methods into philosophy of science* (pp. 13-35). Cham: Springer.

Oyama, S. (2000). *Evolution's eye: A systems view of the biology-culture divide*. Durham, NC: Duke University Press.

Oyama, S., Griffiths, P. E., & Gray, R. D. (Eds.). (2001). *Cycles of contingency: Developmental systems and evolution*. Cambridge, MA; London: MIT Press.

Padula, P. J., Edelstein, A., Miguel, S. D., …Rabinovich, R. D. (1998). Hantavirus pulmonary syndrome outbreak in Argentina: molecular evidence for person-to-person transmission of Andes virus. *Virology, 241*(2), 323-330. doi: 10.1006/viro.1997.8976

Paley, W. (1809). *Natural theology, or Evidences of the existence and attributes of the Deity* (12th ed.). London: J. Faulder.

Paolini Paoletti, M. (2017). From falsemakers to negative properties. *Theoria, 83*(1), 53-77. doi: 10.1111/theo.12113

Paolini Paoletti, M. (2018). Mechanisms and relations. *Erkenntnis*. doi: 10.1007/s10670-018-0095-4

Papineau, D. (1994), Mental disorder, illness and biological disfunction. *Philosophy, 37*, 73-82. doi: 10.1017/S135824610000998X

Parfit, D. (1984). *Reasons and persons*. Oxford: Clarendon Press.

Parkkinen, V.-P., Wallmann, C., Wilde, M., Clarke, B., Illari, P., Kelly, M., Norrell, C., Russo, F., & Williamson, J. (2018). *Evaluating evidence of mechanisms in medicine: Principles and procedure*. Dordrecht: Springer.

Paul, L. A. (2006). In defense of essentialism. *Philosophical Perspectives, 20*(1), 333-372. doi: 10.1111/j.1520-8583.2006.00110.x

Paul, L. A. (2010). The puzzles of material constitution. *Philosophy Compass, 5*(7), 579-590. doi: 10.1111/j.1747-9991.2010.00302.x

Paul, L. A. (2012). Metaphysics as modeling: The handmaiden's tale. *Philosophical Studies, 160*(1), 1-29. doi: 10.1007/s11098-012-9906-7

Pauling, L., Itano, H. A., Singer, S. J., & Wells, I. C. (1949). Sickle cell anemia, a molecular disease. *Science, 110*(2865), 543-548.

Peacocke, C. (1992). *A study of concepts*. Cambridge, MA: MIT Press.

Peirce, C. S. (1977). *Semiotic and significs: The correspondence between Charles S. Peirce and Victoria Lady Welby*. Bloomington: Indiana University Press.

Pellet, F. (2016). Qu'est-ce que la maladie ?. *Philosophie und Gesundheit/Philosophie et santé (Philosophie.ch: Swiss Portal for Philosophy)*. URL: https://www.philosophie.ch/fr/philosophie-fr/philosophie-et-sante/qu-est-ce-que-la-maladie

Pellet, F. (2018). La maladie. In M. Kristanek (Ed.), *L'Encyclopédie Philosophique*. URL: http://encyclo-philo.fr/maladie-gp/

Pellet, F. (2019). Nature, natural vs. artefactual kinds, normality vs. abnormality, functions vs. dys/malfunctions, and values vs. disvalues: Some general clarifications related to health and disease. *Spazio Filosofico, 9*(23), 49-60.

Pellet, F. (mss.a). *Computer simulations: A definition*. Master thesis, University of Geneva.

Pellet, F. (mss.b). Dissolving the internal and external validity problems in clinical randomized controlled trials.

Pereboom, D. (2002). Robust nonreductive materialism. *Journal of Philosophy, 99*(10), 499-531. doi: 10.2307/3655563

Peterman, J. (1992). *Philosophy as therapy: An interpretation and defense of Wittgenstein's later philosophical project*. New York: State University of New York Press.

Petersen, S. (2014). A normative yet coherent naturalism. *Philo, 17*(1), 77-91. doi: 10.5840/Philo20141715

Peterson, T., & Müller, G. B. (2015). Phenotypic novelty in EvoDevo: The distinction between continuous and discontinuous variation and its importance in evolutionary theory. *Evolutionary Biology, 43*, 314-335. doi: 10.1007/s11692-016-9372-9

Petrolini, V. (2015). Are mental disorders illnesses? The boundary between psychiatry and general medicine. In J. Pathemore, & B. Whitby (Eds.), *Proceedings of the AISB symposium From mental 'illness' to disorder and diversity* (pp. 15-21).

Petroski, H. (1992). *The evolution of useful things: How everyday artifacts – from forks and pins to paper clips and zippers – came to be as they are*. New York: Vintage Books.

Pettit, P. (2004). Existentialism, quietism, and the role of philosophy. In B. Leiter (Ed.), *The future for philosophy* (pp. 304-327). Oxford: Clarendon Press.

Pickard, H. (2012). The purpose in chronic addiction. *American Journal of Bioethics Neuroscience, 3*(2), 40-49. doi: 10.1080/21507740.2012.663058

Pigliucci, M. (2003). Species as family resemblance concepts: the (dis-)solution of the species problem?. *Bioessays, 25*(6), 595-602. doi: 10.1002/bies.10284

Pigliucci, M., & Boudry, M. (Eds). (2013). *Philosophy of pseudoscience: Reconsidering the demarcation problem*. Chicago: Chicago University Press.

Pigliucci, M., & Müller, G. B. (Eds.). (2010). *Evolution: The extended synthesis*. Cambridge, MA: MIT Press.

Pinker, S. (2018). *Enlightenment now: The case for reason, science, humanism and progress*. New York: Viking.

Pirofski, L.-A., & Casadevall, A. (2012). Q&A: What is a pathogen? A question that begs the point. *BMC Biology, 10*(6). doi: 10.1186/1741-7007-10-6

Plate, J. (2018). Intrinsic properties and relations. *Inquiry: An Interdisciplinary Journal of Philosophy, 61*(8), 783-853. doi: 10.1080/0020174x.2018.1446046

Plato. *Euthyphro, Apology, Crito, Phaedo, Phaedrus*. Ed. and tr. by H. N. Fowler. New York: Harvard University Press. 1914.

Plato. *Platonis Opera* (vol. 1): *Tetralogiae I–II: Εὐθύφρων* (399 – 390 BC). Ed. by E. A. Duke, W. F. Hicken, W. S. M. Nicoll, D. B. Robinson, & J. C. G. Strachan. Oxford: Oxford University Press. 1995.

Plato. *Platonis Opera* (vol. 4): *Clitopho, Respublica, Timaeus, Critias*. Ed. by J. Burnet. Oxford: Clarendon Press. 1963.

Plutynski, A. (2018). *Explaining cancer: Finding order in disorder*. New York: Oxford University Press.

Plutynski, A. (2019). Cancer. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/cancer/

Poellinger, R. (2012). *Concrete causation: About the structures of causal knowledge*. PhD thesis, LMU Munich.

Poland, J., & Tekin, Ş. (Eds.). (2017). *Extraordinary science and psychiatry: Responses to the crisis in mental health research*. Cambridge, MA; London: MIT Press.

Polger, T. W. (2010). Mechanisms and explanatory realization relations. *Synthese, 177*(2), 193-212. doi: 10.1007/s11229-010-9841-0

Polger, T. W., & Shapiro, L. A. (2016). *The multiple realization book*. New York: Oxford University Press.

Porphyry. *Isagoge*. Ed. by A. Busse. Berlin: G. Reimer. 1887.

Porter, R. (2002). *Blood and guts: A short history of medicine*. London: Allen Lane.

Pradeu, T., Laplane, L., Prévot, K., …Vervoort, M. (2016). Defining "Development". *Current Topics in Developmental Biology, 117*, 171-183. doi: 10.1016/bs.ctdb.2015.10.012

Preston, B. (2018). Artifact. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/artifact/

Prinz, J. J. (2004). *Gut reactions: A perceptual theory of emotion*. New York: Oxford University Press.

Psillos, S. (1999). *Scientific realism: How science tracks truth*. London: Routledge.

Putnam, H. (1960). Minds and machines. In S. Hook (Ed.), *Dimensions of minds* (pp. 138-164). New York: New York University Press.

Putnam, H. (1975). The meaning of 'meaning'. *Minnesota Studies in the Philosophy of Science, 7*, 131-193.

Quante, M., & Stoppenbrink, K. (2011). Enhancement und kosmetische Chirurgie. In R. Stoecker, C. Neuhäuser, & M. L. Raters (Eds.), *Handbuch angewandte Ethik* (pp. 475-478). Stuttgart: J. B. Metzler.

Quine, W. v. (1969). *Ontological relativity & other essays*. New York: Columbia University Press.

Rabinowicz, W., & Rønnow-Rasmussen, T. (2000). A distinction in value: Intrinsic and for its own sake. *Proceedings of the Aristotelian Society, 100*(1), 33-51. doi: 10.1111/j.0066-7372.2003.00002.x

Radden, J. (2019). Mental disorder (illness). In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/mental-disorder/

Railton, P. (1978). A deductive-nomological model of probabilistic explanation. *Philosophy of Science, 45*(2), 206-226. doi: 10.1086/288797

Railton, P. (1986). Moral realism. *Philosophical Review, 95*(2), 163-207. doi: 10.2307/2185589

Ramachandran, M. (2012). The KK-principle, margins for error, and safety. *Erkenntnis, 76*(1), 121-136. doi: 10.1007/s10670-011-9311-1

Ramsey, G. (2013). Human nature in a post-essentialist world. *Philosophy of Science, 80*(5), 983-993. doi: 10.1086/673902

Rao, B. S., Gupta, K. K., Karanam, P., & Peruri, A. (2014). Alhzeimer disease: An interactome of many diseases. Annals of Indian Academy of Neurology, 17(1), 48-54. doi: 10.4103/0972-2327.128551

Rappaport, S. M., Barupal, D. K., Wishart, D., Vineis, P., & Scalbert, A. (2014). The blood exposome and its role in discovering causes of disease. *Environmental Health Perspectives, 122*(8), 769-774. doi: 10.1289/ehp.1308015

Rasmussen, D. B. (1999). Human flourishing and the appeal to human nature. *Social Philosophy and Policy, 16*(1), 1-43. doi: 10.1017/S0265052500002235

Ratcliffe, M., & Stephan, A. (Eds). (2014). *Depression, emotion and the self: Philosophical and interdisciplinary perspectives*. Exeter: Imprint Academic.

Raven, M. J. (2015). Ground. *Philosophy Compass, 10*(5), 322-333. doi: 10.1111/phc3.12220

Raven, M. J. (2020). Explaining essences. *Philosophical Studies*. doi: 10.1007/s11098-020-01483-2

Rawls, J. (1999). *A theory of justice* (2nd ed.). Cambridge, MA: Harvard University Press.

Rea, M. C. (1995). The problem of material constitution. *Philosophical Review, 104*(4), 525-552. doi: 10.2307/2185816

Reaven, G. M. (1976). Treatment of asymptomatic diabetes mellitus. *Comprehensive Therapy, 2*(11), 22-28.

Récanati, F. (2008). *Philosophie du langage (et de l'esprit)*. Paris: Gallimard.

Reichenbach, H. (1938). *Experience and prediction: An analysis of the foundations and the structure of knowledge*. Chicago: The University of Chicago Press.

Reicher, M. (2019). Nonexistent objects. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/nonexistent-objects/

Reiss, J., & Ankeny, R. A. (2016). Philosophy of medicine. In Zalta, E. N. (ed.). *Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/medicine/

Rescher, N. (1996). *Process metaphysics: An introduction to process philosophy*. New York: State University of New York Press.

Reydon, T. A. C. (2009a). Do the life sciences need natural kinds?. *Croatian Journal of Philosophy, 9*(2), 167-190. doi: croatjphil20099221

Reydon, T. A. C. (2009b). How to fix kind membership: A problem for HPC theory and a solution. *Philosophy of Science, 76*(5), 724-736. doi: 10.1086/605814

Reznek, L. (1987). *The nature of disease*. London: Routlegde & Kegan Paul.

Rheinberger, H.-J., Müller-Wille, S., & Meunier, R. (2015). Gene. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/gene/

Richards, R. A. (2016). *Biological classification: A philosophical introduction*. Cambridge: Cambridge University Press.

Richman, K. A. (2004). *Ethics and the metaphysics of medicine: Reflections on health and beneficence*. Cambridge, MA: MIT Press.

Rosch, E. (1978). Principles of categorization. In E. Rosch, & B. Llyod (Eds.), *Cognition and Categorization* (pp. 27-48). Hillsdale: Lawrence Erlbaum Associates.

Rosch, E., & Mervis, C. (1975). Family resemblances: Studies in the internal structure of categories. *Cognitive Psychology, 7*(4), 573-605. doi: 10.1016/0010-0285(75)90024-9

Rogers, W. A., & Mintzker, Y. (2016). Getting clearer on overdiagnosis. *Journal of Evaluation in Clinical Practice, 22*(4), 580-587. doi: 10.1111/jep.12556

Rogers, W. A., & Walker, M. J. (2017). The line-drawing problem in disease definition. *Journal of Medicine and Philosophy, 42*(4), 405-423. doi: 10.1093/jmp/jhx010

Rogers, W. A., & Walker, M. J. (2018). Précising definitions as a way to combat overdiagnosis. *Journal of Evaluation in Clinical Practice, 24*(5), 1019-1025. doi: 10.1111/jep.12909

Rolston III, H. (1992). Disvalues in nature. *The Monist, 75*(2), 250-278.

Rolston III, H. (2001). Naturalizing values: Organisms and species. In L. P. Pojman (Ed.), *Environmental ethics: Readings in theory and application* (3rd ed.) (pp. 76-86). Belmont: Wadsworth Publishing Co.

Rønnow-Rasmussen,T. (2011). *Personal value*. Oxford: Oxford University Press.

Rønnow-Rasmussen, T. (2015). Intrinsic and extrinsic value. In I. Hirose, & J. Olson (Eds.), *The Oxford handbook of value theory* (pp. 29-42). New York: Oxford University Press.

Rosen, G. (2015). Real definitions. *Analytic philosophy, 56*(3), 189-209. doi: 10.1111/phib.12067

Rosen, G. (2017). Abstract objects. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/abstract-objects/

Rosenberg, A. (2006). *Darwinian reductionism, or How to stop worrying and love molecular biology*. Chicago; London: The University of Chicago Press.

Rosenthal, D. M. (1986). Two concepts of consciousness. *Philosophical Studies, 49*, 329-359. doi: 10.1007/bf00355521

Ross, L. N. (2018). The doctrine of specific etiology. *Biology and Philosophy, 33*(5-6), 37. doi: 10.1007/s10539-018-9647-x

Ross, L. N. (forth.). Explanation in contexts of causal complexity: Lessons from psychiatric genetics. URL: http://philsci-archive.pitt.edu/15728/

Ross, W. D. (1930). *The right and the good: Some problems in ethics*. New York: Oxford University Press.

Russell, B. (1903). *The principles of mathematics*. Cambridge: Cambridge University Press.

Russell, B. (1912). *The problems of universals*. London: Williams & Norgate.

Russo, F., & Williamson, J. (2007). Interpreting causality in the health sciences. *International Studies in the Philosophy of Science, 21*(2), 157-170. doi: 10.1080/02698590701498084

Russo, F., & Williamson, J. (2011). Epistemic causality and evidence-based medicine. *History and Philosophy of the Life Sciences, 33*(4), 563-581.

Saborido, C., & Moreno, A. (2015). Biological pathology from an organizational perspective. *Theoretical Medicine and Bioethics, 36*(1), 83-95. doi: 10.1007/s11017-015-9318-8

Sachse, C. (2011). *Philosophie de la biologie: Enjeux et perspectives*. Lausanne: Presses polytechiques et universitaires romandes.

Sade, D. A. F. (de) (1795). *La philosophie dans le boudoir*. London.

Sadegh-Zadeh, K. (2000). Fuzzy health, illness, and disease. *Journal of Medicine and Philosophy, 25*(5), 605-638. doi: 10.1076/0360-5310(200010)25:5;1-W;FT605

Sadegh-Zadeh, K. (2008). The prototype resemblance theory of disease. *Journal of Medicine and Philosophy, 33*(2), 106-139. doi: 10.1093/jmp/jhn004

Sadegh-Zadeh, K. (2015). *Handbook of analytic philosophy of medicine* (2nd ed.). Dordrecht: Springer.

Sadler, J. Z. (2008). Vice and the diagnostic classification of mental disorders: A philosophical case conference. *Philosophy, Psychiatry, and Psychology, 15*(1), 1-17. doi: 10.1353/ppp.0.0152

Sadler, J. Z. (2013). Vice and mental disorders. In K. W. M. Fulford (Ed.), *The Oxford handbook of philosophy and psychiatry* (pp. 451-479). Oxford: Oxford University Press.

Sadler, J. Z., & Agich, G. J. (1995). Diseases, functions, values, and psychiatric classification. *Philosophy, Psychiatry, and Psychology, 2*(3), 219-231.

Salmon, N. (1981). *Reference and essence*. Princeton: Princeton University Press.

Salmon, W. C. (1984). *Scientific explanation and the causal structure of the world*. Princeton: Princeton University Press.

Sarkar, S. (1998). Genetics and reductionism. Cambridge, MA: Cambridge University Press.

Sarto-Jackson, I. (2018). Time for a change: Topical amendments to the medical model of disease. *Biological Theory, 13*(1), 29-38. doi: 10.1007/s13752-017-0289-z

Sattig, T. (2015). *The double lives of objects: An essay in the metaphysics of the ordinary world*. New York: Oxford University Press.

Scarre, G. (Ed.). (2016). *The Palgrave handbook of the philosophy of aging*. London: Palgrave Macmillan.

Schaffer, J. (2004). Causes need not be physically connected to their effects: The case for negative causation. In C. R. Hitchock (Ed.), *Contemporary debates in philosophy of science* (pp. 197-216). London: Blackwell.

Schaffer, J. (2009). On what grounds what. In D. J. Chalmers, D. Manley, & R. Wasserman (Eds.), *Metametaphysics: New essays on the foundations of ontology* (pp. 347-383). Oxford: Clarendon Press.

Schaffer, J. (2016). Grounding in the image of causation. *Philosophical Studies, 173*(1), 49-100. doi: 10.1007/s11098-014-0438-1

Schaffer, J. (2017). Monism: The priority of the whole. *Philosophical Review, 119*(1), 31-76. doi: 10.1215/00318108-2009-025

Schaffner, K. F. (1993). *Discovery and explanation in biology and medicine*. Chicago: University of Chicago Press.

Scheffler, S. (1987). Morality through thick and thin: A critical notice of ethics and the limits of philosophy. *Philosophical Review, 96*(3), 411-434. doi: 10.2307/2185227

Scheler, M. (1921). *Der Formalismus in der Ethik und die materiale Wertethik: Neuer Versuch der Grundlegung eines ethischen Personalismus*. Halle: Max Niemeyer

Scheuermann, R. H., Ceusters, W., & Smith, B. (2009). Towards an ontological treatment of disease and diagnosis. *Summit on Translational Bioinformatics, 2009*, 116-120.

Schöne-Seifert, B., Talbot, D., Opolka, U., & Ach, J. S. (Eds). (2009). *Neuro-enhancement – Ethik vor neuen Herausforderungen*. Paderborn: Mentis.

Schramme, T. (2007). A qualified defence of a naturalist theory of health. *Medical Health Care and Philosophy 10*(1), 11-17. doi: 10.1007/s11019-006-9020-8

Schramme, T. (2008). Classic concepts of disease. *International Encyclopedia of Public Health, 1*, 726-733.

Schramme, T. (2013). On the autonomy of the concept of disease in psychiatry. *Frontiers in Psychology, 4*, 457. doi: 10.3389/fpsyg.2013.00457

Schramme, T. (2016). What a naturalist theory of illness should be. In E. Giroux (Ed.), *Naturalism in the philosophy of health: Issues and implications* (pp. 63-79). Cham: Springer.

Schramme, T. (2017). Subjective and objective accounts of well-being and quality of life. In T. Schramme, & S. Edwards (Eds.), *Handbook of the Philosophy of Medicine* (pp. 159-168). Dordrecht: Springer.

Schroeder, S. A. (2013). Rethinking health: healthy or healthier than?. *British Journal for the Philosophy of Science, 64*(1), 131-159. doi: 10.1093/bjps/axs006

Schurz, G. (2001). What is 'normal'? An evolution-theoretic foundation for normic laws and their relation to statistical normality. *Philosophy of science*, 68(4), 476-497. doi: 10.1086/392938

Schwartz, P. H. (2014). Reframing the disease debate and defending the biostatistical theory. *Journal of Medicine and Philosophy, 39*(6), 572-589. doi: 10.1093/jmp/jhu039

Searle, J. R. (1995). *The construction of social reality*. New York: Free Press.

Sedgwick, P. (1982). *Psychopolitics*. London: Pluto Press.

Segen, J. C. (2006). *Concise dictionary of modern medicine*. New York: McGraw-Hill.

Seibt, J. (2008). Beyond endurance and perdurance: Recurrent dynamics. In C. Kanzian (Ed.), *Persistence* (pp.133-164). Frankfrut: Ontos Verlag.

Seibt, J. (2017). Process philosophy. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/process-philosophy/

Selikowitz, M. (2008). *Down syndrome* (3rd ed.). New York: Oxford University Press.

Sellars, W. (1963). *Science, perception and reality*. New York: Humanities Press.

Sen, A. (1979). Utilitarianism and welfarism. *Journal of Philosophy, 76*(9), 463-489. doi: 10.2307/2025934

Sesardić, N. (2010): Race: A social destruction of a biological concept. *Biology and Philosophy, 25*(2), 143-162. doi: 10.1007/s10539-009-9193-7

Shang, A., Huwiler-Müntener, K., Nartey, L., …Egger, M. (2005). Are the clinical effects of homeopathy placebo effects? Comparative study of placebo-controlled trials of homeopathy and allopathy. *Lancet, 366*(9487), 726-732. doi: 10.1016/S0140-6736(05)67177-2

Sholl, J. (2014). *Evolution and normativity*. PhD thesis, KU Leuven.

Sider, T. (2011). *Writing the book of the world*. New York: Oxford University Press.

Simon, J. R. (2011). Medical ontology. In F. Gifford (Ed.), *Philosophy of medicine* (pp. 65-114). Amsterdam: Elsevier.

Simons, P. (1987). *Parts: A study in ontology*. Oxford : Clarendon Press.

Simons, P. (2004). Pourquoi presque tout – mais non pas exactement toute chose – est une entité. In J.-M. Monnoyer (Ed.), *La structure du monde : objets, propriétés, états de choses: Renouveau de la métaphysique dans l'école australienne de philosophie* (pp. 265-276). Paris: Vrin.

Skiles, A. (2015). Essence in abundance. *Canadian Journal of Philosophy, 45*(1), 100-112. doi: 10.1080/00455091.2015.1013173

Slater, M. (2015). Natural kindness. *British Journal for the Philosophy of Science, 66*(2), 375-411. doi: 10.1093/bjps/axt033

Smart, B. T. H. (2016). Concepts and causes in the philosophy of disease. Basingstoke: Palgrave Macmillan. URL: http://philsci-archive.pitt.edu/11757/

Smith, B., & Mulligan, K. (1983). Framework for formal ontology. *Topoi, 2*(1), 73-85.

Smith, B., Grenon, P., & Goldberg, L. (2004). Biodynamic ontology: Applying BFO in the biomedical domain. *Studies in Health and Technology Informatics, 102*, 20-38.

Smith, R. (2017). Aristotle's logic. In. E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/aristotle-logic/

Sober, E. (1980). Evolution, population thinking, and essentialism. *Philosophy of Science, 47*(3), 350-383. doi: 10.1086/288942

Sober, E. (1984). *The nature of selection: Evolutionary theory in philosophical focus*. Chicago: University of Chicago Press.

Solomon, M. (2015). *Making medical knowledge*. Oxford: Oxford University Press.

Sonnenschein, C., & Soto, A. M. (2000). Somatic mutation theory of cancer: why it should be dropped and replaced. *Molecular Carcinogenesis, 29*(4), 205-11. doi: 10.1002/1098-2744(200012)29:4<205::aid-mc1002>3.0.co;2-w

Sorensen, R. (2018). Vagueness. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/vagueness/

Spear, A. D., Ceusters, W. & Smith, B. (2016). Functions in Basic Formal Ontology. *Applied ontology, 11*(2), 103-128. doi: 10.3233/AO-160164

Spencer, H. (1898). *The Principles of biology* (vol. 1-2). New York: D. Appleton & Co.

Sperry, R. W. (1986). Macro- versus micro-determinism. *Philosophy of Science, 53*(2), 265-270. doi: 10.1086/289311

Spinoza, B. *Opera*. Ed. by C. Gebhardt. Heidelberg: Carl Winter. 1925-1987.

Spinoza, B. The collected works (2 vol.). Ed. and tr. by E. Curley. Princeton: Princeton University Press. 1985-2016.

Spitzer, R. L., & Endicott, J. (1978). Medical and mental disorder: Proposed definition and criteria. In R. L. Spitzer, & D. F. Klein (Eds.), *Critical issues in psychiatric diagnosis* (pp. 15-39). New York: Raven Press.

Spohn, W. (2009). A priori reasons: Two difficult notions and an even more difficult connection. In N. Kompa, C. Nimtz, & C. Suhm (Eds.), *The a priori and its role in philosophy* (pp. 25-38). Paderborn: Mentis.

Sporn, M. B. (1991). Carcinogenesis and cancer: Different perspectives on the same disease. *Cancer Research, 51*, 6215-6218.

Stegenga, J. (2015). Effectiveness of medical interventions. *Studies in History and Philosophy of Biological and Biomedical Sciences, 54*, 34-44. doi: 10.1016/j.shpsc.2015.06.005

Stegenga, J. (2018a). *Care & cure: An introduction to philosophy of medicine*. Chicago; London: The University of Chicago Press.

Stegenga, J. (2018b). *Medical nihilism*. New York: Oxford University Press.

Stegenga, J., Graham Kennedy, A., Tekin, Ş, Jukola, S., & Bluhm, R. (2017). New directions in philosophy of medicine. In J. A. Marcum (Ed.), *The Bloomsbury companion to contemporary philosophy of medicine* (pp. 343-368). London; New York: Bloomsbury.

Stepanians, M. (2017). Why Schaffer's solution to the problem of absence causation fails.

Sterner, B. (2015). Pathways to pluralism about biological individuality. *Biology and Philosophy, 30*(5), 609-628. doi: 10.1007/s10539-015-9494-y

Steward, H. (2013). Processes, continuants, and individuals. *Mind, 122*(487), 781-812. doi: 10.1093/mind/fzt080

Stich, S. P. (Ed.). (1975). *Innate ideas*. Berkeley; Los Angeles: University of California Press.

Stich, S. P. (1990). *The fragmentation of reason*. Cambridge, MA: MIT Press.

Stoessl, A. J. (2020). Deception and the ethics of placebo. *International Review of Neurobiology, 153*, 147-163. doi: 10.1016/bs.irn.2020.03.030

Stoljar, D. (2017). *Philosophical progress: In defense of a reasonable optimism*. Oxford: Oxford University Press.

Stoljar, D., & Damnjanovic, N. (2010). The deflationary theory of truth. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/truth-deflationary/

Stotz, K., Griffiths, P. E., & Knight, R. (2004). How biologists conceptualize genes: An empirical study. *Studies in History and Philosophy of Biological and Biomedical Sciences, 35*(4), 647-673. doi: 10.1016/j.shpsc.2004.09.005

Stout, R. (2016). The category of occurrent continuants. *Mind, 125*(497), 41-62. doi: 10.1093/mind/fzv138

Strasser, B. J. (1999). Sickle cell anemia, a molecular disease. *Perspective, 286*(5444), 1488-1490. doi: 10.1126/science.286.5444.1488

Strasser, B. J., & Fantini, B. (1998). Molecular diseases and diseased molecules: Ontological and epistemological dimensions. *History and Philosophy of the Life Sciences, 20*(2), 189-214.

Strawson, P. F. (1952). *Introduction to logical theory*. London: Methuen.

Strawson, P. F. (1959). *Individuals: An essay in descriptive metaphysics*. London: Routledge.

Strawson, P. F. (1970). Imagination and perception. In L. Foster, & J. W. Swanson (Eds.), *Experience and theory* (pp. 32-54). Amherst: University of Massachusetts Press.

Strevens, M. (2019). *Thinking off your feet: How empirical psychology vindicates armchair philosophy*. Cambridge, MA; London: Belknap Press.

Strimbu, K., & Tavel, J. A. (2010). What are biomarkers?. *Current Opinion in HIV and AIDS, 5*(6), 463-466. doi: 10.1097/COH.0b013e32833ed177

Strobach, N. (1998). *The moment of change: A systematic history in the philosophy of space and time*. Dordrecht: Springer.

Stroop, B. (2013). *Rethinking well-being in biomedical ethics*. PhD thesis, University of Muenster.

Stroud, B. (1996). The charm of naturalism. *Proceedings and Addresses of the American Philosophical Association, 70*(2), 43-55.

Subedi, B., & Grossberg, G. T. (2011). Phantom limb pain: Mechanisms and treatment approaches. *Pain Research and Treatment, 2011*, 864605. doi: 10.1155/2011/864605

Sulis, W. (2020). The continuum from temperament to mental illness: Dynamical perspectives. *Neuropsychobiology*, 1-13. doi: 10.1159/000509572

Sullivan, A. (2005). Rigid designation, direct reference, and modal metaphysics. *Pacific Philosophical Quarterly, 86*(4), 577-599. doi: 10.1111/j.1468-0114.2005.00243.x

Sydenham, T. (1741). *Opera universa medica*. London: Sydenham Society.

Systematized Nomenclature of Medicine (SNOMED) (2020). London: SNOMED.

Szabó (1999). Expressions and their representations. *Philosophical Quarterly, 49*(195), 145-163. doi: 10.1111/1467-9213.00134

Szasz, T. S. (1974). *The myth of mental illness: Foundations of a theory of personal conduct* (2nd ed.). New York: Harper & Row.

Tabaczek, M. (2019). *Emergence: Towards a new metaphysics and philosophy of science*. Notre Dame: University of Notre Dame Press.

Taber's Cyclopedic Medical Dictionary (23rd ed.) (2017). Philadelphia: F. A. Davis Company.

Tabery, J. (2014). *Beyond versus: The struggle to understand the interaction of nature and nurture*. Cambridge, MA: MIT Press.

Tahko, T. E. (2015). *An introduction to metametaphysics*. Cambridge, MA: Cambridge University Press.

Tappolet, C. (2000). *Emotions et valeurs*. Paris: Presses Universitaires de France.

Tappolet, C. (2004). Through thick and thin: Good and its determinates. *Dialectica, 58*(2), 207-221. doi: 10.1111/dltc.2004.58.issue-2

Tappolet, C. (2011). Values and emotions: Neo-sentimentalism's prospects. In C. Bagnoli (Ed.), *Morality and the emotions* (pp. 117-134). New York: Oxford University Press.

Taviner, M., Thwaites, G., & Gant, V. (1998). The English sweating sickness, 1485-1551: A viral pulmonary disease?. *Medical History, 42*(1), 96-98. doi: 10.1017/s0025727300063365

Tekin, Ş. (2016). Are mental disorders natural kinds? A plea for a new approach to intervention in psychiatry. *Philosophy, Psychiatry, and Psychology, 23*(2), 147-163. doi: 10.1353/ppp.2016.0013

Tekin, Ş., & Mosko, M. (2015). Hyponarrativity and context-specific limitations of the DMS-5. *Public Affairs Quarterly, 29*(1), 109-134.

Testa, I. (2015). Some consequences of Michael Thompson's Life and Action for social philosophy. *Philosophy and Public Issues, S2015*, 69-84.

Thagard, P. (1998). Explaining disease: Correlations, causes, and mechanisms. *Minds and Machines, 8*(1), 61-78. doi: 10.1023/A:1008286314688

Thagard, P. (1999). *How scientists explain disease*. Princeton: Princeton University Press.

Thomas, A., Chess, S., & Birch, H. G. (1968). *Temperament and behavior disorders in children*. New York: New York University Press.

Thomasson, A. L. (2007). Modal normativism and the methods of metaphysics. *Philosophical Topics, 35*(1/2), 135-160. doi: 10.5840/philtopics2007351/27

Thomasson, A. L. (2012). Experimental philosophy and the methods of ontology. *The Monist, 95*(2), 175-199. doi: 10.5840/monist201295211

Thomasson, A. L. (2015). *Ontology made easy*. New York: Oxford University Press.

Thompson, M. (1995). The representation of life. In R. Hursthouse, G. Lawrence, & W. Quinn (Eds.), *Virtues and reasons: Philippa Foot and moral theory* (pp. 247-296). Oxford: Clarendon Press.

Thompson, M. (2008). Life and action: Elementary structures of practice and practical thought. Cambridge, MA: Harvard University Press.

Thompson, R. P. (2011). Causality, theories and medicine. In P. McKay Illari, F. Russo, & J. Williamson (Eds.), *Causality in the sciences* (pp. 25-44). New York: Oxford University Press.

Thompson, R. P., & Upshur, R. E. G. (2018). *Philosophy of medicine: An introduction*. London: Routledge.

Thomson, J. J. (1983). Parthood and identity across time. *Journal of Philosophy, 80*(4), 201-220.

Thornton, T. (2007). *Essential philosophy of psychiatry*. Oxford: Oxford University Press.

Tidy, H. (1945). Sweating sickness and Picardy sweat. *British Medical Journal, 2*(4410), 63-64.

Tinbergen, N. (1963). On aims and methods of ethology. *Zeitschrift für Tierpsychologie, 20*, 410-433.

Tooley, M. (2015). The problem of evil. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/evil/

Toppinen, T. (2018). Non-naturalism gone quasi:Explaining the necessary connections between the natural and the normative. In R. Shaffer-Landau (Ed.), *Oxford studies in metaethics* (vol. 12) (pp. 25-47). Oxford: Oxford University Press.

Trofimova, I., & Sulis, W. (2018). There is more to mental illness than negative affect: Comprehensive temperament profiles in depression and generalized anxiety. *BMC Psychiatry, 18*, 125. doi: 10.1186/s12888-018-1695-x

Trogdon, K. (2017). Priority monism. *Philosophy Compass, 12*(11), 1-10. doi: 10.1111/phc3.12458

Trogdon, K. (2018). Inheritance arguments for fundamentality. In R. L. Bliss, & G. Priest (Eds.), *Reality and its structure* (pp. 182-198). New York: Oxford University Press.

Tsou, J. Y. (2016). Natural kinds, psychiatric classification and the history of the DSM. *History of Psychiatry, 27*(4), 406-424.

Tsou, J. Y. (2019). Philosophy of science, psychiatric classification, and the DSM. In Ş. Tekin, & R. Bluhm (Eds.), *The Bloomsbury companion to philosophy of psychiatry* (pp. 177-196). London: Bloomsbury.

Twaddle, A. (1968). *Influence and illness: Definitions and definers of illness behavior among older males in Providence, Rhode Island*. PhD thesis, Brown University.

Tye, M. (2017). Qualia. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/qualia/

Ujvari, M. (2004). Cambridge change and sortal essentialism. *Metaphysica, 5*(2), 25-34.

Uher, R., & Zwicker, A. (2017). Etiology in psychiatry: Embracing the reality of poly-gene-environmental causation of mental illness. *World Psychiatry, 16*(2), 121-129. doi: 10.1002/wps.20436

Valderas, J. M., Starfield, B., Sibbald, B., Salisbury, C., & Roland, M. (2009). Defining comorbidity: Implications for understanding health and health services. *Annals of Family Medicine, 7*(4), 357-363. doi: 10.1370/afm.983

van Inwagen, P. (1981). The doctrine of arbitrary undetached parts. *Pacific Philosophical Quarterly, 62*(2), 123-137.

van Inwagen, P. (2011). Relational vs. constituent ontologies. *Philosophical Perspectives, 25*(1), 389-405. doi: 10.1111/j.1520-8583.2011.00221.x

van Inwagen, P. (2014). *Metaphysics* (4th ed.). Boulder: Westview Press.

Varga, S. (2011): Defining mental disorder. Exploring the 'natural function' approach. *Philosophy, Ethics, and Humanities in Medicine, 6*(1), 1-10. doi: 10.1186/1747-5341-6-1

Varga, S. (2018). Embodied concepts and mental health. *Journal of Medicine and Philosophy, 43*(2), 241-260. doi: 10.1093/jmp/jhx040

Varzi, A. C. (2007). Omissions and causal explanations. In F. Castellani, & J. Quitterer (Eds.), *Agency and causation in the human sciences* (pp. 155-167). Paderborn: Mentis.

Varzi, A. C. (2016). Mereology. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/mereology/

Vaux, D. L. (2011). In defense of the somatic mutation theory of cancer. *BioEssays, 33*(5), 341-343. doi: 10.1002/bies.201100022

Väyrynen, P. (2016). Thick ethical concepts. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/archives/sum2019/entries/thick-ethical-concepts/

Venkatapuram, S. (2013). Health, vital goals, and central human capabilities. *Bioethics, 27*(5), 271-279. doi: 10.1111/j.1467-8519.2011.01953.x

Vinueza, A. (2002). Realism and mind-independence. *Pacific Philosophical Quarterly, 82*(1), 51-70. doi: 10.1111/1468-0114.00118

Virchow, R. L. K. (1958). *Disease, life, and man: Selected essays*. Ed. and tr. by L. J. Rather. Palo Alto: Stanford University Press.

Virvidakis, S. (2008). Varieties of quietism. *Philosophical Inquiry, 30*(1-2), 157-175. doi: 10.5840/philinquiry2008301/227

von Sydow, M. (2012). *From Darwinian metaphysics towards understanding the evolution of evolutinary mechanisms*. Göttingen: Universitätsverlag Göttingen.

von Wachter, D. (1998). On doing without relations. *Erkenntnis, 48*(2-3), 355-358. doi: 10.1023/A:1005469206285

von Wright, G. H. (1963). *The varieties of goodness*. New York: Humanities Press.

Vosgerau, G., & Soom, P. (2018). Reduction without elimination: Mental disorders as causally efficacious properties. *Minds and Machines, 28*(2), 311-330. doi: 10.1007/s11023-017-9434-5

Wachbroit, R. (1994). Normality as a biological concept. *Philosophy of Science, 61*(4), 579-591. doi: 10.1086/289823

Wagenknecht, S., Nersessian, N. J., & Andersen, H. (Eds.). (2015). *Empirical philosophy of science: Introducing qualitative methods into philosophy of science*. Cham: Springer.

Wakefield, J. C. (1992). The concept of mental disorder: On the boundary between biological facts and social values. *American Psychologist, 47*(3), 373-388. doi: 10.1037/0003-066X.47.3.373

Wakefield, J. C. (2006). What makes a mental disorder mental?. *Philosophy, Psychiatry, and Psychology, 13*(2), 123-131. doi: 10.1353/ppp.2007.0010

Wakefield, J. C. (2010). Aristotle as sociobiologist: The "function of a human being" argument, black box essentialism, and the concept of mental disorder. *Philosophy, Psychiatry, and Psychology, 7*(1), 17-44.

Walker, M. J., & Rogers, W. A. (2017). Defining disease in the context of overdiagnosis. *Medicine, Health Care and Philosophy, 20*(2), 269-280. doi: 10.1007/s11019-016-9748-8

Walker, M. J., & Rogers, W. A. (2018). A new approach to defining disease. *Journal of Medicine and Philosophy, 43*(4), 402-420. doi: 10.1093/jmp/jhy014

Walker, M. J., Bourke, J., & Hutchison, K. (2019). Evidence for personalised medicine: Mechanisms, correlation, and new kinds of black box. *Theoretical Medicine and Bioethics, 40*(2), 103-121. doi: 10.1007/s11017-019-09482-z

Wallace, M. (2014). Composition as identity, modal parts, and mereological essentialism. In A. J. Cotnoir, & D. L. M. Baxter (Eds.), *Composition as identity* (pp. 111-129). New York: Oxford University Press.

Wallace, M. (2019). The polysemy of 'part'. *Synthese, SI:Mereology and Identity*. doi: 10.1007/s11229-019-02088-x

Walsh, D. M. (1996). Fitness and function. *British Journal for the Philosophy of Science, 47*(4), 553-574. doi: 10.1093/bjps/47.4.553

Walsh, D. M. (2006). Evolutionary essentialism. *British Journal for the Philosophy of Science, 57*(2), 425-448. doi: 10.1093/bjps/axl001

Walter, S. (2002). Need multiple realizability deter the identity-theorist?. *Grazer Philosophische Studien, 65*(1), 51-75. doi: 10.1163/18756735-90000793

Walter, S. (2010). Taking realization seriously: No cure for epiphobia. *Philosophical Studies, 151*(2), 207-226. doi: 10.1007/s11098-009-9425-3

Wasserman, D., Asch, A., Blustein, J., & Putnam, D. (2016a). Disability: Definitions, models, experience. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/disability/

Wasserman, D., Asch, A., Blustein, J., & Putnam, D. (2016b). Disability: Health, well-being, and personal relationships. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/disability-health/

Wasserman, R. (2017). Material constitution. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/material-constitution/

Waters, C. K. (2017). No general structure. In M. H. Slater, & Z. Yudell (Eds.), *Metaphysics and the philosophy of science: New essays* (pp. 81-108). New York: Oxford University Press.

Weber, M. (2005). *Philosophy of experimental biology*. New York: Cambridge University Press.

Weber, M., & Esfeld, M. (2003). Holism in the sciences. In G. Hirsch Hadorn (Ed.), *Encyclopedia of Social Sciences and Humanities*. Oxford: EOLSS Publishers.

Wedgwood, R. (1999). The price of non-reductive moral realism. *Ethical Theory and Moral Practice, 2*(3), 199-215. doi: 10.1023/A:1009994827047

Weisberg, M. (2013). *Simulation and similarity: Using models to understand the world*. New York: Oxford University Press.

Wetzel, L. (2006). Types and tokens. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/types-tokens/

Whitbeck, C. (1977). Causation in medicine: The disease entity model. *Philosophy of Science 44*(4), 619-637. doi: 10.1086/288771

WHO. (1946). *Constitution of the World Health Organization*. New York. URL: https://www.who.int/governance/eb/who_constitution_en.pdf

WHO. (2017). *The Global Burden of Diseases, injuries, and risk factors study*. URL: http://ghdx.healthdata.org/gbd-2017

WHO. (2018a). *International Statistical Classification of Diseases and Related Health Problems* (ICD) (11th ed.). URL: https://icd.who.int/

WHO. (2018b). *List of blueprints priority diseases*. URL: https://www.who.int/activities/prioritizing-diseases-for-research-and-development-in-emergency-contexts

WHO. (2020). *Disabilities*. URL: http://origin.searo.who.int/topics/disabilities/en/

WHO Collaborating Centre for Drug Statistics Methodology (2020). Oslo: WHO.

Wiesing, U. (2018). From art to science: A new epistemological status for medicine? On expectations regarding personalized medicine. *Medicine, Health Care and Philosophy, 21*(4), 457-466. doi: 10.1007/s11019-017-9820-z

Wiggins, D. (2016). *Continuants: Their activity, their being, and their identity: Twelve essays*. New York: Oxford University Press.

Wildman, N. (2013). Modality, sparsity, and essence. *Philosophical Quarterly, 63*(252), 760-782. doi: 10.1111/1467-9213.12059

Wildman, N. (2016). How to be a modalist about essence. In M. Jago (Ed.), *Reality making* (pp. 177-196). New York: Oxford University Press.

Wilde, M., & Parkkinen, V.-P. (2019). Extrapolation and the Russo-Williamson thesis. *Synthese, 196*(8), 3251-3262. doi: 10.1007/s11229-017-1573-y

Wilkins, J. S. (2013). Essentialism in biology. In K. Kampouris (Ed.), *The philosophy of biology: A companion for educators* (pp. 395-420). Dordrecht: Springer.

Wilkins, J. S. (2018). *Species: The evolution of the idea*. Boca Raton: CRC Press.

Wilkins, J. S., & Ebach, M. C. (2014). *The nature of classification: Relationships and kinds in the natural sciences*. New York: Palgrave Macmillan.

Williams, B. (1985). *Ethics and the limits of philosophy*. Cambridge, MA: Harvard University Press.

Williams, D. C. (1936). Tokens, types, words, and terms. *Journal of Philosophy, 33*(26), 701-707. doi: 10.2307/2015665

Williams, G. C. (1957). Pleiotropy, natural selection, and the evolution of senescence. *Evolution, 11*(4), 398-411. doi: 10.1111/j.1558-5646.1957.tb02911.x

Williamson, J. (2013). How can causal explanations explain. *Erkenntnis, 78*(S2), 257-275. doi: 10.1007/s10670-013-9512-x

Williamson, J. (2019a). Establishing causal claims in medicine. *International Studies in the Philosophy of Science, 32*(1), 33-61. doi: 10.1080/02698595.2019.1630927

Williamson, J. (2019b). Evidential proximity, independence, and the evaluation of carcinogenicity. *Journal of Evaluation in Clinical Practice, 25*(6), 955-961. doi: 10.1111/jep.13226

Williamson, T. (2000). *Knowledge and its limits*. New York: Oxford University Press.

Williamson, T. (2018). *Doing philosophy: From common curiosity to logical reasoning*. New York: Oxford University Press.

Wilson, E. O. (1975). *Sociobiology*. Cambridge, MA: Belknap Press.

Wilson, J. (2017). Determinables and determinates. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/determinate-determinables/

Wilson, R. A., & Barker, M. J. (2019). Biological individuals. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/biology-individual/

Wilson, R. A., Barker, M. J., & Brigandt, I. (2007). When traditional essentialism fails: Biological natural kinds. *Philosophical Topics, 35*(1/2), 189-215. doi: 10.5840/philtopics2007351/29

Wimsatt, W. C. (2007). *Re-engineering philosophy for limited beings: Piecewise approximations to reality*. Cambridge, MA; London: Harvard University Press.

Winther, R. G. (2000). Darwin on variation and heredity. *Journal of the History of Biology, 33*(3), 425-455. doi: 10.1023/A:1004834008068

Wittgenstein, L. (1953). *Philosophical Investigations*. Oxford: Blackwell.

Wollheim, R. (1968). *Art and its objects*. New York: Harper & Row.

Woodford, P. (2016). Neo-Darwinists and neo-Aristotelians: How to talk about natural purpose. *History and Philosophy of the Life Sciences, 38*(4). doi: 10.1007/s40656-016-0123-0

Woodward, J. (2003). *Making things happen: A theory of causal explanation*. Oxford: Oxford University Press.

Woodward, J. (2016). Causation and manipulability. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*. URL: https://plato.stanford.edu/entries/causation-mani/

Wolterstorff, N. (1970). *On universals: An essay in ontology*. Chicago: University of Chicago Press.

Wright, L. (1973). Functions. *Philosophical Review, 82*(2), 139-168. doi: 10.2307/2183766

Wrigley, A. (2007). Realism and anti-realism about mental illness. *Philosophical Papers, 36*(3), 371-397. doi: 10.1080/05568641.2007.11733499

Yourgrau, P. (2019). *Death and nonexistence*. New York: Oxford University Press.

Zachar, P. (2014). *A metaphysics of psychopathology*. Cambridge, MA; London: MIT Press.

Zangwill, N. (2011). Negative properties. *Noûs, 45*(3), 528-556. doi: 10.1111/j.1468-0068.2010.00776.x

Zangwill, N. (2018). Brutalist non-naturalism and Hume's principle. *Dialectica, 72*(3), 365-383. doi: 10.1111/1746-8361.12235

Zhu, J., & Buckareff, A. A. (2006). Intention are mental states. *Philosophical Explorations, 9*(2), 235-242. doi: 10.1080/13869790600641954

Zimmerman, M. J. (1999). Virtual intrinsic value and the principle of organic unities. *Philosophy and Phenomenological Research, 59*(3), 653-666. doi: 10.2307/2653786