

WHAT HAVE GENES GOT TO DO WITH IT?
**How Social and Genetic Influences Contribute to Differences in Educational
Success within the Family**

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A

Theoretical and methodological framework of the dissertation

What Have Genes got to do with it?

How Social and Genetic Influences Contribute to Differences in Educational Success within the Family

This section provides the theoretical and methodological framework for the following four articles:

Article 1: Diewald, Martin, Tina Baier, Wiebke Schulz, and Reinhard Schunck. 2015. "Status Attainment and Social Mobility: How Can Genetics Contribute to an Understanding of Their Causes?" *Kölner Zeitschrift für Soziologie und Sozialpsychologie* 67(S1):371–95. doi: 10.1007/s11577-015-0317-6.

Article 2: Baier, Tina. 2019. "Does Sibling and Twin Similarity in Cognitive Ability Differ by Parents' Education?" *Journal of Family Research* 31(1):58–82. doi: 10.3224/zff.v31i1.04.

Article 3: Baier, Tina, and Volker Lang. 2019. "The Social Stratification of Environmental and Genetic Influences on Education: New Evidence Using a Register-Based Twin Sample." *Sociological Science* 6:143–71. doi: 10.15195/v6.a6.

Article 4: Baier, Tina, Volker Lang, Michael Grätz, Kieron J. Barclay, Dalton Conley, Thomas Laidley, and Torkild H. Lyngstad. 2019. "Genetic Effects on Educational Success in Cross-National Perspective." (Unpublished manuscript).

1.1 Introduction

Families fundamentally shape individuals' biographies. It is within the family where socialization starts, values are formed, and skills are acquired. Although the family context is important for a variety of reasons, the way in which it directly affects an individual's development, and therefore a person's life chances, is paramount. In stratification research, one of the core questions is how family background affects a child's education, and the dominant approach has been to compare children from different families (a "between-family perspective") (e.g., Blau and Duncan 1967; Breen 2010; Breen and Goldthorpe 1997; Breen and Jonsson 2005; Erikson and Jonsson 1996). Though not explicitly stated, the assumption in such studies is that family background – often indicated on the basis of parents' education, occupation, or income – has a uniform impact on children's stratification outcomes (e.g., Conley 2008; Diewald et al. 2015). However, results of studies that compare children from the same family (a "within-family perspective") clearly challenge this assumption. In terms of educational attainment the correlation between siblings is about 0.5 (e.g., Benin and Johnson 1984; Conley 2008; Hauser and Mossel 1985; Hauser and Wong 1989; Sieben, Huinink, and de Graaf 2001). Thus, stratification mechanisms run not only between families but also within families. Despite being exposed to fairly similar family circumstances, siblings are not equally affected by them and end up attaining different levels of education. Since differences between siblings represent an equally important aspect of a society's inequality structure as between family differences, it is important to understand why siblings develop differently (e.g., Diewald et al. 2015; Grätz 2018). Nevertheless, the differences within families (i.e., within-family stratification) have received much less attention in stratification research than have those between families. The following dissertation investigates the processes that lead to within-family stratification in terms of educational success.

Within-family perspectives are commonly applied in the field of behavioral genetics. Unlike stratification scholars, behavioral geneticists explicitly consider that parents transmit not only their social resources and experiences but also their genes. Genetically sensitive studies based on sibling and/or twin data report that nearly all psychological characteristics –including those that are highly predictive of educational attainment – are heritable (e.g., Ayorech et al. 2017; Plomin et al. 2016; Polderman et al. 2015; Turkheimer 2000). But more distal outcomes such as achievement scores, school grades,

and educational attainment itself are also considerably affected by genes (e.g., Ayorech et al. 2017; Bartels et al. 2002; Branigan, McCallum, and Freese 2013; Gutman et al. 2003; Johnson, McGue, and Iacono 2005, 2006; de Zeeuw, de Geus, and Boomsma 2015). Thus, genes are an important driver of individual differences and need to be considered in order to understand why siblings from the same family end up attaining different levels of education.

Genes and environments do not act independently of one another. According to the bioecological model, human development involves constant exchanges with the environment (Bronfenbrenner and Ceci 1994). Thus, whether and how genes are realized depends on social conditions. One prominent hypothesis for gene–environment interactions is the Scarr–Rowe hypothesis, which claims that the relative importance of genetic influences relevant to one’s cognitive ability is higher in socioeconomically advantaged families, whereas shared environmental influences are more important in socioeconomically disadvantaged families (Rowe, Jacobson, and van den Oord 1999; Scarr-Salapatek 1971). The underlying assumption is that advantaged parents provide environmental conditions under which genetic influences can be realized (Rowe, Jacobson, and van den Oord 1999; Scarr-Salapatek 1971).

Up to now the mechanisms driving the Scarr–Rowe hypothesis have been barely understood. In addition, previous research has predominantly scrutinized IQ (e.g., Bates, Lewis, and Weiss 2013; Guo and Stearns 2002; Turkheimer et al. 2003). In contrast, educational outcomes –which are not solely determined by an individual’s IQ– are under-researched. Furthermore, studies of the Scarr–Rowe hypothesis have focused narrowly on parents’ socioeconomic status and tend to neglect that the broader institutional environment can also shape genetic effects on education (Diewald 2016b; Selita and Kovas 2019; for an exception, see Tucker-Drob and Bates 2016). For instance, more generous welfare states protect against major life risks and provide comparatively high levels of social security. On average, such contexts grant higher living standards and more equal access to relevant resources. In contrast, in less developed welfare states, access to relevant resources is more restricted, which can hinder the realization of genetic potential. Likewise, comprehensive schooling systems provide more homogenous learning environments than stratified schooling systems. Stratified schooling systems limit access to enriched learning environments, which can lower children’s chances for genetic expression. Comparative studies that take systematically interdependencies between family- and macro-level influences into account are largely missing from this literature.

In this dissertation I aim to reconcile research from sociology and behavioral genetics. Sociologists have strong expertise in environmental influences and processes that lead to educational inequality. However, they often apply a between-family perspective and tend to overlook the role of genetic heterogeneity (e.g., Blau and Duncan 1967; Breen 2010; Breen and Goldthorpe 1997; Breen and Jonsson 2005; Erikson and Jonsson 1996). Behavioral geneticists apply a within-family perspective that is sensitive to genetic heterogeneity, but they often overlook the complexity and diversity of environmental conditions. This dissertation contributes to the literature by applying an integrative approach that combines sociological theories on educational inequality with approaches and analytical tools rooted in behavioral genetics.

To gain a better understanding of the processes that lead to within-family stratification in terms of educational success, this dissertation addresses four research questions that are located at the nexus of social stratification research and behavioral genetics. First, I ask whether and how within-family perspectives and genetically informed research contribute to our understanding of the processes that lead to social stratification (see Article 1). Second, I examine within-family stratification in terms of cognitive ability and ask whether the degree of within-family stratification varies according to parents' educational background (see Article 2). Third, I focus on educational attainment and the gene–environment interplay and ask whether genetic and social influences vary according to parents' social background in Germany (see Article 3). Fourth, I put these findings in a comparative, cross-country perspective. This study includes three advanced industrialized societies –Germany, Sweden, and the United States– which differ considerably with regard to their educational systems and represent three different prototypes of welfare regimes. I ask whether genetic influences relevant to educational success –indicated by school grades and by years of education– differ across these countries and whether there are cross-country differences in the social stratification of genetic effects (see Article 4).

1.2 Theoretical Background

The link between family background and children's educational success is well established in the literature (e.g., Breen and Jonsson 2005; DiPrete and Hout 2006). To explain the impact of family background stratification, scholars often refer to the framework of primary and secondary effects of social background (e.g., Boudon 1974; Breen and Goldthorpe 1997; Breen et al. 2014; Erikson and Jonsson 1996; Jackson et al. 2007). Primary effects refer to parents' efforts to improve their children's educational achievement. To actively foster the development of cognitive and noncognitive skills, parents may provide a more stimulating home environment and relevant learning materials and/or private tutoring. In addition, they transmit cultural resources and interests, all of which further school-related skills (e.g., Cunha and Heckman 2008; Lareau 2011; Lareau and Weininger 2003). Secondary effects, in contrast, refer to stratified schooling choices over and above a child's academic achievement. Parents' educational decisions are determined by the anticipated costs, benefits, and likelihood of success and, importantly, by the intention to avoid downward mobility (Breen and Goldthorpe 1997). Consequently, parents having a higher socioeconomic status opt for higher educational tracks for their children more often than do parents with a lower socioeconomic status who maintain their status by opting for lower educational tracks. Thus, despite equal educational achievement, children from disadvantaged families end up with lower levels of education compared with children from advantaged families.

This framework has been used to explain how educational differences emerge among children from different families. However, studies that investigate children from the same family show that they realize different levels of educational attainment even though they share the same family background (e.g., Benin and Johnson 1984; Conley 2008; Hauser and Mossel 1985; Hauser and Wong 1989; Sieben, Huinink, and de Graaf 2001). Hence, the theory of primary and secondary effects falls short in explaining why siblings realize different levels of education.

Differences within Families and Parents' Investments

Current explanations for differences between siblings are mainly rooted in economic approaches that focus on parents' resource allocation decisions (Becker and Tomes 1976; Behrman, Pollak, and Taubman 1982). Becker and Tomes (1976) argue that parents aim to maximize the total returns of the household and invest rationally in children's human

capital formation. According to the “efficiency paradigm,” parents invest in the child for whom they anticipate the highest returns of education. This reinforces differences among their children. Behrman and colleagues (1982), however, argue that parents invest in compensatory fashion, since future returns of education are uncertain. In both perspectives, parents seek to create equal living standards for their children (Becker and Tomes 1976; Behrman, Pollak, and Taubman 1982). How they reach this goal differs; either parents invest selectively in children’s education and redistribute later in the life course or they compensate for differences which reduces the need for redistribution. Conley (2004, 2008) adds a stratification aspect to parents’ investment decisions and argues that equality among siblings is dependent on the amount of resources that are available in the household: Parents with fewer resources allocate their resources efficiently and invest in the most promising child, thus increasing differences among siblings. In contrast, advantaged parents compensate for differences among their children, leading to similarity.

I propose that parents might also make equal investments and are willing to accept differences among their children. To explain how equal investments can lead to systematic differences in the similarity of siblings, I draw on the literature that demonstrates that parents engage differently in childrearing and in their children’s skill-formation processes (Bodovski and Farkas 2008; Cheadle 2008; Cheadle and Amato 2011; Kalil, Ryan, and Corey 2012; Lareau 2011; Lareau and Weininger 2003). Originally, these studies apply a between-family perspective, but I argue that stratified parenting can also affect the extent to which children from the same family resemble each other at least with regard to their cognitive and noncognitive skills. Lareau (2011) differentiates between two logics of parenting that affect children’s development and skill formation processes. Advantaged parents purposely foster skills and behaviors typically found among higher-class families. This parenting concept is called “concerted cultivation” (Lareau 2011). What is important for my expectation regarding within-family differences is that advantaged parents actively manage their children’s learning environment and development by enrolling their children in enrichment activities and lessons, and by providing various types of inputs that stimulate children’s skill development and that foster their individual talents (e.g., through books or additional learning materials). In contrast, disadvantaged parents are engaged in a parenting concept referred to as “natural growth,” according to which parents intervene only little in children’s developmental processes (Lareau 2011). Owing to financial and time

constraints, disadvantaged parents tend to provide uniform investments and inputs that will meet their children's basic needs. Consequently, disadvantaged parents less often structure their children's leisure time in line with their children's talents and provide less individualized investments that enhance children's individual skills.

Differences in parenting can result in stratified sibling similarity in cognitive and noncognitive skills: Investments by advantaged parents address children's specific needs and further their development more individually, and I hypothesize that this more active and strategic parenting tends to accentuate differences among siblings. Investments are not distributed unevenly on purpose but are more in line with children's individual talents and needs. Stratified differences within families are, therefore, not the result of economically driven investment decisions but instead are an unintended consequence of stratified parenting.

In Article 2, I investigate whether sibling similarity with regard to cognitive ability is socially stratified and put these two different expectations under test.

Differences within Families and the Role of Genes

What has mainly been neglected in sociological or economic explanations on sibling similarity is the notion that similarity among siblings might not be driven solely by parents' investments decisions and resources but might also be due to their genes. Behavioral genetic approaches commonly differentiate among shared environmental influences (i.e., those that lead to sibling similarity), nonshared environmental influences (i.e., those that lead to differences among siblings), and genetic influences (for more details, see the next section, Methodological Approaches and Terminology). Related studies based on sibling and/or twin data have consistently shown that predictors of educational success (such as IQ, noncognitive skills, and educational achievement) as well as educational attainment itself are significantly influenced by genes (e.g., Ayorech et al. 2017; Bartels et al. 2002; Branigan, McCallum, and Freese 2013; Gutman et al. 2003; Johnson, McGue, and Iacono 2005, 2006; de Zeeuw, de Geus, and Boomsma 2015). In addition, these studies show that the role of genes clearly exceeds the relative importance of being raised in one family for these outcomes; however, this pattern differs with regard to educational attainment, in that shared environmental influences matter about as much as genes do (i.e., shared environmental influences account for about 36% of the total variation in education and genes account for about 40%) (Branigan, McCallum, and Freese 2013).

These findings have two important implications for how family background affects differences among siblings in educational success: First, not only parents' investments and resources influence the degree of sibling similarity but also their genetic endowments. And second, the role of genes and family-wide characteristics (i.e., shared environmental influences such as parents' education, education or income) vary for different indicators of educational success.

The finding that shared environmental influences play a stronger role for educational attainment could be explained in light of the secondary effects of social background (Boudon 1974). Parents' educational decisions are socially stratified and are driven not only by educational achievement but also by the intention to maintain social status (Breen and Goldthorpe 1997). Consequently, if children from the same family differ in terms of educational achievement, they still end up being more alike with regard to their educational attainment. That implies that parents' schooling choices are to a certain extent independent of children's genetic potential for educational achievement. Shared environmental influences should, therefore, have a larger impact on educational attainment compared to educational achievement. Relatedly, genes should be more important for educational achievement, since educational achievements are less influenced by stratified schooling choices and more directly linked to cognitive ability compared to educational attainment.

However, if we are to accept the role of genes, we also have to take into account that their impact can vary depending on environmental conditions. In the following, I focus on the proximate family environment (i.e., the social position of the family) and, in a next step, also on the broader institutional environment (i.e., the welfare state and the educational system). To elaborate how parents' social position affects the impact of shared environmental and genetic influences on educational success, I combine the literature on parenting with behavioral genetics approaches. As discussed above, the family environment and the kind of inputs children receive vary across the social strata (Bodovski and Farkas 2008; Cheadle 2008; Cheadle and Amato 2011; Kalil, Ryan, and Corey 2012; Lareau 2011; Lareau and Weininger 2003). Differences in the rearing environments are important because individual development involves an ongoing exchange with the environment (e.g., conditions set by families, peers, or institutions; Bronfenbrenner and Ceci 1994). In other words, whether or how genetic dispositions are expressed is dependent on environmental conditions.

The Scarr–Rowe hypothesis, a prominent hypothesis rooted in behavioral genetics, proposes a positive association between the social position of the family and the importance of genetic influences relevant for IQ. Specifically, it is argued that genetic influences on IQ are more important in families with a higher social status, whereas shared environmental influences are more important in disadvantaged families. The notion of stratified family environments can explain the mechanisms that underlie the Scarr–Rowe hypothesis from a sociological perspective: Advanced parents provide more individually adapted environments that match children’s genetic disposition, which enhances genetic expression. Disadvantaged parents provide rearing environments that are less well adapted to their children’s individual abilities. Such environments provide fewer developmental opportunities, which leads to a suppression of genetic potential. Relatedly, less individualized environments explain why shared environmental influences –those that lead to the similarity of siblings– are more important in disadvantaged families.

Article 3 investigates whether shared environmental and genetic influences for educational attainment are socially stratified in Germany, and in this way examines whether an interaction in line with the Scarr–Rowe hypothesis holds for education.

However, not only the proximate family environment but also the broader institutional environment, such as the welfare state and the particular educational system, can shape genetic influences on educational success (Diewald 2016b; Selita and Kovas 2019; Tucker-Drob and Bates 2016). For example, welfare states differ in terms of social benefits provided and in the degree to which they protect their citizens against life risks. In welfare states that provide higher levels of social security with universal access, relevant resources are provided to all citizens and on average living standards are higher. Such conditions provide better opportunities for the realization of an individual’s genetic potential (Selita and Kovas 2019). In contexts where state intervention is minimal and social security systems are weak, individuals are more likely to lack the resources relevant to genetic expression (Selita and Kovas 2019).

The schooling system can also shape genetic expression. In comprehensive school systems, students are taught in the same learning environment, whereas stratified school systems provide different learning environments, with children being grouped according to their abilities. Thus, the quality of the learning environment varies more in stratified school systems, which can lead to a suppression of genetic influences (Selita and Kovas 2019).

Article 4 takes interdependencies between family and macro-level influences into account and investigates whether genetic effects on educational success differ among three advanced industrialized societies –Germany, Sweden, and the United States.

These countries have different educational systems and represent three different types of welfare regimes that are often used in internationally comparative social inequality research (Esping-Andersen 1990). Genetic effects on educational success should be larger in Sweden because of that country’s egalitarian educational system and its more generous welfare regime. The social stratification of genetic effects, by contrast, should be more pronounced in Germany (because of that country’s stratified schooling system, which is characterized by early tracking) and in the United States (because of the meager role of the welfare state).

1.3 Methodological Approaches and Terminology

To study the processes that lead to differences between siblings in educational success, I adopt a within-family perspective and use analytical tools that are applied in stratification research and behavioral genetics.¹ In this section, I first elaborate how twins, as opposed to siblings, can improve estimations on the processes that account for within-family stratification. On the same subject, I describe variance decomposition models that I use in Article 2 to investigate whether within-family stratification in terms of cognitive ability is stratified. Second, I take genetic influences into account and introduce the behavioral genetic perspective on within family differences. I then explain genetically sensitive variance decomposition methods, so-called ACE models, which are used in Articles 3

¹ Behavioral genetics (or quantitative) approaches measure genetic influences indirectly by comparing individuals with different degrees of kinship and a common upbringing. In molecular approaches, in contrast, genetic influences are measured directly. Complex traits, such as educational outcomes, are influenced by many genetic and environmental influences, each of which has fairly small effects. Although the field of molecular genetics is rapidly evolving and has made considerable progress in identifying genetic influences on individuals’ outcomes, quantitative methods are still better suited for modeling whole genome effects and their variation across social conditions. Moreover, quantitative genetics –in contrast to molecular genetics– allows us to study the impact of shared environmental influences (the net of genes), which is of fundamental interest to stratification researchers.

and 4 to study the role of genetic effects on educational success and their variation according to social conditions.

Sibling and Twin Similarity

The similarity of siblings represents a broad measure for the overall impact of family background influences (e.g., Sieben, Huinink, and de Graaf 2001; for a detailed discussion, see Article 2). The idea is intuitive: Because siblings grew up in the same family, everything that makes them alike can be treated as the result of shared family background influences; conversely, differences among siblings are the result of influences that are not shared by siblings and thus are specific to the child.

Sibling designs can be based on different types of siblings, such as (full) siblings or twins. (Full) siblings differ in age and may grow up in very different family environments (Björklund and Jäntti 2012). For example, parents can switch jobs, relocate, repartner, and/or change their parenting behavior from one child to the next. In addition, siblings share on average only 50% of their DNA. Since siblings share family background influences to only a certain extent, estimations of the overall impact of family background influences tend to represent lower bound estimates (Björklund and Jäntti 2012). In addition, differences between siblings can be affected by influences rooted in different family conditions, developmental differences, differences in genetic makeup, and/or a combination of these factors.

Twins, in contrast, are raised simultaneously, grow up under most similar family circumstances, and therefore share much more of the family background influences than do (full) siblings. The twin design provides a unique opportunity to comprehensively control for the impact of the family environment. However, studying twins does not allow us fully to capture the impact of genetic influences, since dizygotic (DZ) twins (i.e., fraternal twins) share on average about 50% of their genes. Thus, differences between twins may still be confounded by genetic differences. Monozygotic (MZ) twins (i.e., identical twins) share 100% of their DNA. Thus, studying MZ twins allows us to control for both, the impact of the family environment and the impact of genes. Any difference between MZ twins is the result of child-specific influences -net of genes (see Table 1.1).

To study whether the similarity of siblings with regard to cognitive ability varies by parents' education, I analyze all three types of siblings (Article 2). Since twins share much more of the family background influences than siblings do, they provide a better unit of analysis to test whether sibling similarity is associated with parents' social background

and related differences in investment behaviors. Results based on DZ and MZ twins show to what extent the similarity in cognitive ability changes when children are raised under the most similar family conditions (DZ twins) and when genetic heterogeneity is also controlled for (MZ twins). Since MZ twins allow to control for both environmental and genetic heterogeneity, the study of MZ twins makes it possible to test more rigorously whether a change in the similarity is associated with parents' educational background.

Table 1.1 Sibling and Twin Designs

| | Full siblings | DZ twins | MZ twins |
|--------------------------|----------------------------------|--------------------------------|----------------------|
| Family environment | Differences in family background | Shared | Shared |
| Genetic overlap | ~ 50% | ~ 50% | ~ 100% |
| Sources of dissimilarity | Nonshared influences and genes | Nonshared influences and genes | Nonshared influences |

Source: Adapted from Baier (2019).

However, sibling and twin designs have limitations. First, results based on siblings and twins could be driven by sibling effects (i.e., influences that siblings have on one another). For instance, siblings can serve as role models and guide each other's decisions and behaviors (e.g., Benin and Johnson 1984). Siblings may also behave in completely different ways to set themselves apart in order to maintain their niche within the family system (Feinberg and Hetherington 2000). Mutual interdependencies among siblings can lead to differences as well as to similarity and can therefore confound estimates regarding the role of shared family and child-specific influences. To rule out the possibility that my findings on siblings' and twins' similarity are affected by sibling effects, I control for the closeness of siblings and twins in Article 2.

Second, and related to twin studies specifically, there is the question of generalizability. Twins are high-risk births. They have on average lower birth weights, are often born prematurely, and are at higher risk for congenital malformations, and all these factors can affect twins' (cognitive) development (Boardman et al. 2002; Liu and Blair 2002; Lytton and Gallagher 2002). In addition, twins –particularly MZ twins– may be different owing to their common upbringing and genetic similarity. However, previous research has found no differences in means and variances between twins and non-twins

with regard to cognitive ability, personality, and, more recently, antisocial behavior (Posthuma et al. 2000; Johnson et al. 2002; Christensen et al. 2006).

Variance Decomposition Methods

The degree of within-family stratification can be estimated by means of variance decomposition models (also known as multilevel models) in which children (level 1) are nested in families (level 2) (e.g., Raudenbush and Bryk 2002; see Article 2). These models decompose the total variation of an outcome in a component associated with shared family influences (“between-family variance”) and a component associated with child-specific influences (“within-family variance”). Based on this multilevel regression setup, the similarity of siblings can be estimated as follows:

$$ICC = \frac{\sigma_b^2}{\sigma_w^2 + \sigma_b^2} \quad (1)$$

The intraclass correlation coefficient (ICC) represents the similarity among siblings. The ICC equals the ratio of the variance associated with shared family influences (σ_b^2) relative to the total variance, which is the sum of between-family variance (σ_b^2) and within-family variance (σ_w^2). A low value for the ICC indicates high within-family stratification, meaning that child-specific influences are more important than shared family influences. For example, if educational differences between families are smaller than educational differences within families, then the ICC is low. Thus, despite growing up in similar family environments, siblings end up with different educational levels. Conversely, if educational differences between families are greater than educational differences within families, then the ICC is high. Shared family influences are more important than child-specific influences, therefore within-family stratification is low.

ACE Variance Decomposition Methods

To acknowledge that differences among siblings are also a consequence of differences in genetic make-up, Article 3 and 4 use genetically sensitive variance decomposition methods based on the classical twin design (CTD).

The CTD is one of most common designs in behavioral genetics (e.g., Plomin et al. 2008). Since DZ twins and MZ twins are raised simultaneously but differ in their genetic

overlap, it is possible to decompose the total variation of an outcome (phenotype) into a component associated with additive genetic influences (A), a component associated with shared environmental influences (C), and a component associated with unique (child-specific) environmental influences that also includes the error term of the variance decomposition (E). The total variance of an outcome (σ_P^2) is therefore conceptualized as the sum of the variance components of σ_A^2 , σ_C^2 , and σ_E^2 :

$$\sigma_P^2 = \sigma_A^2 + \sigma_C^2 + \sigma_E^2 \quad (2)$$

The A, C, and E variance components are usually estimated by means of structural equation modeling or multilevel methods (for more details, see Article 4, Appendix 4.C). *Additive genetic influences* (A) capture the relative importance of genetic influences known as “heritability estimates.” They indicate how much of the total variance of an outcome is associated with genetic as opposed to environmental influences. On an interpretive level it is important to note that heritability estimates are population parameters. For instance, previous research has shown that the heritability of IQ is between 0.6 and 0.8 in adulthood (Tucker-Drob, Briley, and Harden 2013). This does not mean that 60% to 80% of an individual’s IQ can be explained by their genes. Instead, it means that genetic influences account for about 60% to 80% of the total variation in IQ (Plomin et al. 2008). Furthermore, heritability estimates should be treated as any other descriptive statistic (Plomin et al. 2016), that is, they are dependent on the measurement of the outcome, the specifications and assumptions of the identification strategy, and, importantly, on the characteristics of the population studied. Heritability estimates can tell us to what extent genes matter, but not how and under what circumstances.

Common or shared environmental influences (C) represent all nongenetic influences that are shared among siblings and that lead to similarity among siblings. *Unique environmental influences* (E) represent all nongenetic influences that are specific to one child and that lead to differences among siblings. The definitions of shared and nonshared environmental influences are based on their impact (i.e., whether they lead to similarity or dissimilarity among twins or siblings). To give an example, parental divorce is a family event that is experienced by all children and is therefore shared. However, each child can react very differently to the parents’ divorce (Turkheimer and Waldron 2000). Hence, the same conditions cannot automatically be treated as shared environmental influences, because they can lead to differences between siblings. Vice versa, different conditions

can lead to sibling similarity and can therefore not automatically be treated as nonshared environmental influences. On the same subject, behavioral genetic scientists differentiate between “objective” and “effective” environments, with the latter acknowledging that similar circumstances can lead to different individual reactions (Turkheimer and Waldron 2000). The ACE components and their meanings are summarized in Table 1.2.

Table 1.2 Variance Decomposition Based on the Classical Twin Design

| Variance component | Definition | Differences within twin pairs | |
|--------------------|---|-------------------------------|-----|
| | | MZ | DZ |
| A | Additive genetic influences | No | Yes |
| C | Common (shared) environmental influences that make twins alike –net of genes (e.g., shared effects of parents’ education or financial resources) | No | No |
| E | Unique (nonshared or child-specific) environmental influences that lead to differences between twins –net of genes (e.g., selective parenting, selective peer influences, and also measurement error) | Yes | Yes |

Source: Baier and Lang (2019).

The identification of the relative importance of genes and shared environmental influences relies on further assumptions (e.g., Plomin et al. 2008) (see Articles 3 and 4 for a more detailed discussion):

First, the ACE model identifies additive genetic effects –that is, it is assumed that genetic variants have independent effects and do not interact with each other (epistasis).

Second, it is assumed that there are no correlations or interactions between genes and the environment in the population for the outcome under study. Because this clearly contradicts the Scarr–Rowe hypothesis, ACE models for educational success are estimated separately for socially defined groups in Article 3 and 4. This analytical strategy is known as nonparametric gene–environment interaction analysis (Guo and Wang 2002) and addresses this assumption by allowing genetic and environmental influences to vary between socially defined groups.

A third assumption, the equal environment assumption (EEA) (Scarr and Carter-Saltzman 1979), states that the outcome under study should not be affected by differential treatment between MZ and DZ twins. If the EEA is violated, the relative importance of

genetic influences will be inflated, because a higher similarity of MZ twins is driven by a more similar treatment by their surroundings (e.g., parents, friends, peers) and is not the result of genetic influences. Yet, it is likely that MZ twins are treated more similarly than are DZ twins (which is also partly the result of their genetic resemblance). To date, several studies have tested the validity of the EEA for several –mostly psychological– traits. Although there is no study that focuses on educational outcomes, studies on IQ report that more equal environments experienced by MZ twins do not inflate heritability estimates with respect to IQ (Derks, Dolan, and Boomsma 2006).

Fourth, the CTD assumes random mating of spouses. Random mating justifies the assumption that DZ twins share on average 50% of their DNA. However, if parents are similar in characteristics that affect the outcome under study, the genetic similarity of DZ twins increases. This in turn leads to an overestimation of shared environmental influences, because the similarity of DZ twins is higher than would be assumed. Since educational homogamy is a well-established finding across Western societies (e.g., Blossfeld 2009), the analyses in Article 3 and 4 adjust for assortative mating, as suggested by Loehlin and collaborators (2009).

1.4 Data Sources

The empirical articles of this dissertation are based on diverse data sets (Articles 2 to 4). These articles all use novel, large-scale observational twin data from the German Twin Family Panel (TwinLife) (Diewald et al. 2018). Article 4, which applies a comparative perspective, additionally draws on large-scale observational twin data for the United States (Add Health) as well as register data for Sweden.

TwinLife is a longitudinal study that collects information on more than 4000 MZ and same-sex DZ pairs of twins and their families residing in Germany. The TwinLife study surveys twins, twins' biological and social parents, and, if available, one sibling as well as partners of the older twins (“extended twin family design” [ETFD]). In addition, TwinLife applies a cohort sequential design that includes four birth cohorts of twins (2009–2010, 2003–2004, 1997–1998, and 1990–1993). Data collection started in 2014 with face-to-face interviews. Twins were sampled based on administrative data from communal registration offices. Because a probability-based register sampling strategy

was applied (Lang and Kottwitz 2017), the TwinLife study overcomes one of the major weaknesses of many observational twin studies, that is, they are often based on small or convenience samples. Therefore, TwinLife provides a unique opportunity to conduct genetically sensitive analyses covering a broad range of the social spectrum (Lang and Kottwitz 2017).

Zygoty was determined with the use of physical similarity questionnaires (e.g., on eye color, body size, hair structure). Depending on the age of the twins, these questions were either assessed through self-reports or provided by the main caregiver. Similarity questionnaires are often used to assess the zygoty of twins because they are less costly and highly accurate (about 95%) compared with assessments based on twins' DNA (Heath et al. 2003). TwinLife cross-validated the algorithm used to determine twins' zygoty based on DNA samples from more than 300 twin pairs (Lenau and Hahn 2017). The results showed an accuracy of 92% to 97%, depending on twins' ages (Lenau and Hahn 2017).

For Sweden, we use register data (Statistics Sweden 2011). Each individual in Sweden has a unique personal identification number (PIN) by which individual records can be linked across the various administrative registries. This multi-generation register contains information on the PIN of each individual, as well as on the PINs of their parents. This allows us to identify the biological mother and father of each individual and in turn identify any other biological relations. Unfortunately, the information on twins' zygoty was not included in our data access. To approximate twins' zygoty, we use the information on sex, birth year, and birth month: Siblings born on the same date are twins; opposite-sex twin pairs are dizygotic. Same-sex twins, however, can be either monozygotic or dizygotic. We classify all same-sex twins as monozygotic which leads to an overestimation of MZ twins. Following previous research, we correct for the overclassification of MZ twins based on the assumption that same-sex and opposite-sex DZ twin births are equally likely (Figlio et al. 2017) (for more details, see Article 4, Appendix 4.C).

For the United States, we use data from the National Longitudinal Study of Adolescent to Adult Health (Add Health) (Harris et al. 2013). Add Health collects information about individuals' social and economic situation, as well as their psychological and physiological well-being. The data consist of a nationally representative sample of adolescents who were in grades 7 to 12 during the 1994/95 school year. Follow-up waves were collected in 1996, 2001–2002, and 2008 through in-home interviews. In addition to

the core sample, an oversample of about 3000 siblings (including twins) was drawn, which we use for the analyses. Information about respondents' siblings (i.e., twins, half-siblings, or non-related siblings that live in the same household) was retrieved from school rosters. Twins' zygosity was determined by means of similarity questionnaires in wave I. In wave III, the zygosity information was cross-validated with DNA samples, yielding an accuracy of about 91% (Harris et al. 2006).

1.5 Research Agenda

The four articles to be presented aim to provide a coherent research agenda in the area of within-family stratification in terms of educational success. I adopt an interdisciplinary perspective and take into account social and genetic influences, as well as their interplay.

Specifically, this dissertation asks a) whether and how the consideration of genetic variation can improve our understanding of the processes leading to social stratification, b) whether sibling and twin's similarity in cognitive ability differs according to parents' education, c) whether social and genetic influences on educational attainment differ according to parents' social background in Germany, and lastly d) whether genetic effects on educational success differ by country and parents' social position.

*Article 1: Status Attainment and Social Mobility: How Can Genetics Contribute to an Understanding of Their Causes?*²

Martin Diewald, Tina Baier, Wiebke Schulz, and Reinhard Schunck

This chapter provides the relevant theoretical background and discusses empirical findings that motivate the following empirical chapters. We explain why genes can enhance social inquiries related to stratification, introduce the basic concepts and different approaches of genetically sensitive research, and open up routes for future research integrating genetics in stratification research.

² This section summarizes the article by Diewald et al. (2015).

One of the core questions in stratification research is how family background shapes children's life chances. Stratification scholars commonly focus on the role of parents' resources, as indicated in terms of parents' education, occupation, or income (e.g., Blau and Duncan 1967; Breen 2010; Breen and Goldthorpe 1997; Breen and Jonsson 2005; Erikson and Jonsson 1996). However, parents transmit not only social resources but also their genes, which also contribute to differences in stratification outcomes (e.g., Freese 2008; Polderman et al. 2015; Turkheimer 2000). Hence, to identify the impact of social transmission mechanisms, genetic heterogeneity needs to be considered.

But the recognition of genes as a possible confounder represents only a first step, because genes are realized under environmental conditions. To understand how genetic influences contribute to the reproduction of social inequality, it is important to take into account that genes and environments depend on one another. Gene–environment interactions describe processes in which social environments shape genetic expressions, and vice versa. Shanahan and Hofer (2005) distinguish between the following four processes: triggering, compensation, social control, and enhancement. These processes can lead to inequality between groups to the extent that they share genetic variants but differ in their environments.

As an example, the Scarr–Rowe hypothesis proposes that genes are more relevant in advantaged than in disadvantaged families (Rowe, Jacobson, and van den Oord 1999; Scarr-Salapatek 1971). The mechanism that brings about such a gene–environment interaction could be rooted in the benefits of enhancement –that is, advantaged parents may provide rearing environments that match children's genetic endowments and hence facilitate genetic expression. Since children from disadvantaged families do not experience such environments, the chances that they will realize their genetic potential are lowered.

Thus, the consideration of genetic variation is methodologically relevant but also for theoretical reasons. The integration of genetic influences helps to understand how social resources shape children's stratification outcomes –net of genetic influences. In that sense, genetically sensitive research provides estimations on “pure” social effects. Moreover, research that investigates the gene–environment interplay improves our understanding of how social inequality is reproduced across generations: stratification scholars tend to assume that family background influences have a uniform impact on children's attainments. However, as the example of the Scarr–Rowe hypothesis has shown, an important mechanism in the reproduction of social inequality might be rooted

in the provision of child-specific investments that are in line with children's genetic dispositions. Studies that focus on gene–environment interactions can help us to better understand what kind of environments parents must provide for their children to realize their genetic potential for IQ and other characteristics relevant for stratification.

*Article 2: Does Sibling and Twin Similarity in Cognitive Ability Differ by Parents' Education?*³

Tina Baier

Stratification scholars predominantly investigate how differences between children from different families emerge, whereas differences between children from the same family have received much less attention in the literature. In addition, stratification scholars tend to overlook the role of genes. I study within-family stratification in terms of cognitive ability and ask whether sibling and twin similarity varies according to parents' education. I extend the established sibling correlation approach to DZ twins and MZ twins, thus acknowledging that both social and genetic influences affect cognitive ability (e.g., Nisbett et al. 2012; Tucker-Drob, Briley, and Harden 2013). In addition, I shift the theoretical focus from economic perspectives to stratified parenting.

Economic perspectives and their extensions propose that disadvantaged parents reinforce differences, whereas advantaged parents compensate for differences (Becker and Tomes 1976; Behrman, Pollak, and Taubman 1982; Conley 2004, 2008). I propose that parents may also make equal investments and thus accept differences among their children. I extend the literature on stratified parenting that demonstrates that parents are differently engaged in childrearing and their children's skill-formation processes (Bodovski and Farkas 2008; Cheadle 2008; Cheadle and Amato 2011; Kalil, Ryan, and Corey 2012; Lareau 2011; Lareau and Weininger 2003). Owing to financial and time constraints, disadvantaged parents provide more uniform investments and inputs that will meet their children's basic needs. In contrast, advantaged parents are more actively engaged in their children's developmental processes and tend to foster children's individual talents. I argue that stratified parenting leads not only to differences between families but to differences within families. Investments from advantaged parents address

³ This section summarizes the article by Baier (2019).

children's specific needs and further their individual development; however, such investments are not distributed unequally on purpose. Since more active and strategic parenting tends to accentuate the differences among siblings, I hypothesize that siblings from advantaged families are less similar in terms of cognitive ability compared with siblings from disadvantaged families.

Previous research on the stratification of sibling similarity in cognitive skills is based on (full) siblings and provides conflicting evidence (Conley, Pfeiffer, and Velez 2007; Grätz 2018). However, findings based on (full) siblings can be misleading, since the (dis-)similarity of siblings can be influenced by developmental differences, genetic differences, and/or a combination of the two and is not necessarily the direct consequence of varying parental resources. To address this shortcoming, I compare siblings, DZ twins, and MZ twins, which allows me to a) more comprehensively model influences of family background leading to sibling similarity and b) more rigorously test whether the similarity in cognitive ability is directly linked to parents' education and associated investments. To test my hypothesis, I use novel data from the German Twin Family Panel, TwinLife (Diewald et al. 2018), and estimate variance decomposition models.

My results show, first, that within-family stratification in terms of cognitive ability is about the same for young adult siblings and DZ twins. Thus, even most similar family conditions did not lead to greater similarity in cognitive ability among DZ twins. Second, I find that siblings, DZ twins, and MZ twins from highly educated families are less alike in their cognitive ability when compared with their counterparts from less educated families. Thus, the more resources the parents have, the more important are child-specific influences –net of genes. This finding supports my hypothesis concerning equal investments and stratified parenting. Findings on the mean level of cognitive ability provide additional support: siblings' and twins' cognitive ability scores are on average higher among more educated families in which child-specific influences are more important. In contrast, shared family influences –those that make siblings alike– are more important in less educated families. As the mean scores indicate, they are rather detrimental when it comes to the realization of the child's cognitive ability. Since I studied siblings and twins during their young adulthood, my findings indicate that parenting has a lasting impact on children's cognitive ability.

Article 3: The Social Stratification of Environmental and Genetic Influences on Educational Attainment: New Evidence Using a Register-Based Twin Sample⁴

Tina Baier and Volker Lang

Both social and genetic influences matter for education (Branigan, McCallum, and Freese 2013; Nielsen and Roos 2015; Nielsen 2016). We ask whether the relative importance of these influences differs according to parents' social position. Originally, the Scarr–Rowe hypothesis claimed that genetic influences for cognitive ability are stronger in advantaged than in disadvantaged families. We extend this line of research by focusing on education. In addition, we provide a theoretical account for the social stratification of genetic influences from a sociological perspective.

To investigate the social stratification of genetic influences, we combine behavioral genetic approaches with established theories about educational inequality. Specifically, we extend the sociological literature that emphasizes that the rearing environment and parenting differ according to parents' social background (Bodovski and Farkas 2008; Cheadle 2008; Cheadle and Amato 2011; Kalil, Ryan, and Corey 2012; Lareau 2011; Lareau and Weininger 2003). We argue that advantaged parents provide more child-centered learning environments that are adapted to children's potentials and needs. The individual adaptation of the children's learning environment leads to more favorable conditions for gene expression. We hypothesize that the relative importance of genetic influences is stronger in advantaged families, whereas that shared environmental influences are more important in disadvantaged families. In as much learning environments are socially stratified, different opportunities for genetic expression widen over children's educational biography (Dannefer 2003; DiPrete and Eirich 2006). Thus, mechanisms of cumulative advantage and disadvantage can explain how stratified parenting shapes children's education lastingly.

We test our hypothesis for Germany, which represents an interesting case because of its highly stratified schooling system, which is characterized by early tracking. Furthermore, part-time schools are more common in Germany than in other contexts, which gives parents more freedom to form their children's skill development. We

⁴ This section summarizes the article by Baier and Lang (2019).

therefore expect that the social stratification of genetic and shared environmental influences on education will be comparatively strong in Germany. We estimate ACE models for years of education and analyze the German Twin Family Panel study (Diewald et al. 2018).

Our results provide support for the social stratification of shared environmental and genetic effects on educational attainment in Germany: genetic influences on educational attainment are more important in highly educated families, whereas shared environmental influences matter more in less educated families. Moreover, the mean level of educational attainment increases with the level of the parents' education. Thus, shared environmental influences in less educated families are rather detrimental for educational attainment.

In sum, our findings support the expectation of stratified parenting and systematic differences in the quality of the family environment that shape the realization of genetic dispositions and thus contribute to social disparities in educational attainment.

Article 4: Genetic Effects on Educational Success in Cross-National Perspective⁵

Tina Baier, Volker Lang, Michael Grätz, Kieron J. Barclay, Dalton Conley, Thomas Laidley, and Torkild H. Lyngstad

The final study extends previous research on the gene–environment interplay in terms of education by applying a comparative framework. We ask whether genetic influences on educational success vary among Germany, Sweden, and the United States and whether there are differences in the social stratification of genetic influences across these three countries.

Our expectations about cross-country differences in genetic influences on educational success and social stratification are rooted in different types of educational systems and welfare regimes (Esping-Andersen 1990). With regard to the educational system, we focus on differences in tracking. The German educational system places children at an exceptionally young age (10 to 12) on one of the three hierarchically structured secondary-school tracks. Sweden and the United States, in contrast, have a longer period of comprehensive schooling and less strict tracking (Bol et al. 2014). Different tracks

⁵ This section summarizes the manuscript by Baier et al. (2019).

represent distinct learning environments based on children's ability. We expect that early tracking, as compared with more flexible or later forms of tracking, restricts children's chances to realize their genetic potential (Selita and Kovas 2019).

In addition, the structure of the welfare state may affect the quality of children's rearing environments. Liberal welfare states such as the United States provide only minimal social security (DiPrete 2002; DiPrete and McManus 2000; Esping-Andersen 1990). Disadvantaged parents may face more severe economic hardship and are exposed to higher levels of stress compared with the more generous social security regimes in Germany and Sweden (Diewald 2016a). Both resource restrictions and stress may lower parents' capacity to provide enhanced rearing environments and inputs tailored to their children's genetic endowment, thus restricting these children's chances to develop their genetic potential (Selita and Kovas 2019).

We hypothesize that genetic influences on educational success are overall less important in Germany and the United States than in Sweden. In keeping with the Scarr-Rowe hypothesis (Rowe, Jacobson, and van den Oord 1999; Scarr-Salapatek 1971), we expect that the social stratification of genetic influences is stronger in both Germany, owing to the early tracking system, and the United States, owing to the meager role of the welfare state, when compared with Sweden.

To test these expectations, we use large-scale observational twin data for Germany (German Twin Family Panel [TwinLife]) (Diewald et al. 2018) and for the United States (National Longitudinal Study of Adolescent Health [Add Health]) (Harris et al. 2013), as well as register data on twins from Sweden (Statistics Sweden 2011). We study genetic influences on educational achievement (school grades) and educational attainment (years of education). The birth cohorts of the twins in the different samples range from 1975 through 1993.

Results based on the ACE models show that, independent of country, genetic influences are more important for educational achievement than for educational attainment. With regard to cross-country variation, we find that genetic influences on educational success are least important in Germany, and matter most in Sweden. With regard to the social stratification of genetic influences, we do not find robust evidence. However, we find indications of gene-environment interactions in line with the Scarr-Rowe hypothesis for educational success in Germany and the United States. Our findings therefore point to the positive effects of more egalitarian educational systems on the development of genetic potentials for educational success.

1.6 Conclusion

In this framework paper I aimed to show how within-family approaches –particularly when combined with genetically sensitive approaches– can significantly contribute to our understanding of stratification processes. I provided the theoretical and empirical background; introduced the terminology, the methods, and data; and embedded my four articles in the emerging literature that integrates genetics in stratification research.

In my research I have reconciled expertise from the fields of both sociology and behavioral genetics. Specifically, I took social and genetic influences as well as their interplay into account, adopted designs rooted in behavioral genetics, and used established theories concerning educational inequality. The three empirical articles of this dissertation were devoted to processes that drive within-family stratification in terms of educational successes and their variation according to social conditions. I focused on educational success as one of the most relevant indicators of social stratification and hence later life chances. Specifically, I analyzed cognitive ability as one of the major single input factors for educational success, school grades as an indicator of educational achievement, and years of education as an indicator of educational attainment.

In following I synthesize the results of this dissertation with a focus on those findings that challenge common understandings of how family influences operate:

First of all, the findings provided evidence against the implicit assumption of between-family perspectives that parents' resources have a uniform impact on their children's educational success. Despite being raised in the same family, siblings and twins realized different cognitive ability scores, school grades, and education levels.

For cognitive ability my results showed that the similarity of siblings, DZ twins, and MZ twins is socially stratified. Contrary to the expectation that advantaged parents invest in compensatory fashion, siblings, DZ twins, and MZ twins in more educated families were less alike compared with those in less educated families. This finding contradicts the expectation based on economic perspectives that advantaged parents invest in compensatory fashion (Conley 2004, 2008) and instead supports my hypothesis based on equal investments and stratified parenting (e.g., Lareau 2011; Lareau and Weininger 2003). Advantaged parents are more likely to make child-specific investments that tend to accentuate differences. In addition, cognitive ability scores for siblings, DZ twins, and MZ twins were on average higher among highly educated families. This finding shifts the

focus from the role of shared family influences to child-specific influences –net of genetic influences– that further the development of cognitive ability.

Second, the results supported the increasing evidence that not only parents' investments but also children's genes need to be considered to understand the emergence of differences among siblings in educational success (e.g., Ayorech et al. 2017; Bartels et al. 2002; Branigan, Mccallum, and Freese 2013; de Zeeuw, de Geus, and Boomsma 2015). In the studies on educational achievement and educational attainment, I differentiated between shared environmental, nonshared environmental (i.e., child-specific), and genetic influences. In line with previous genetically sensitive research the results provided evidence of genetic influences for both educational achievement and educational attainment (e.g., Ayorech et al. 2017; Bartels et al. 2002; Branigan, Mccallum, and Freese 2013; Gutman et al. 2003; Johnson, McGue, and Iacono 2005, 2006; de Zeeuw, de Geus, and Boomsma 2015). What is more, in all countries under investigation, that is, Germany, Sweden, and the United States, genetic effects were stronger for educational achievement than for educational attainment. This supports the expectation based on stratified schooling decisions (Boudon 1974; Breen and Goldthorpe 1997; Erikson and Johnsson 1996). Genetic effects are stronger for educational achievement than for educational attainment because educational decisions operate over and above children's genetic potential for educational achievement: even if children differ in genetic potential for educational achievement, they end up with the same educational level.

Third, the results of this dissertation emphasized the role of environmental conditions in the realization of genetic potential. Specifically, the results provided evidence for differences in genetic effects across socially defined groups and countries. For educational attainment, the results provided evidence for a gene–environment interaction in line with the Scarr–Rowe hypothesis for Germany: genetic influences on education were more important in highly educated families, whereas shared environmental influences mattered more in less educated families. In addition, shared environmental influences were associated with lower levels of education.

This has important implications for stratification scholars, because these findings divert from our common understanding of how family influences affect children's educational success. Sociologists often perceive family influences as a global family effect of parents' social resources, which has a positive and uniform impact on their children's education (see also Diewald 2016b). However, results for Germany showed

that shared environmental influences were more important for twins from less educated parents and associated with lower levels of education. Shared environmental influences are nongenetic influences that lead to the similarity of siblings (such as parents' education, income or occupation). Thus, family influences tend to make siblings in regards to educational attainment more alike in disadvantaged families and lower children's chances for educational success. In addition, genetic influences were less important in disadvantaged families which indicates that shared family influences constrain the realization of children's genetic potential for educational success. This is an important aspect, and more genetically sensitive research is needed to understand what kind of family influences lead to the enhancement of genetic potential and which ones affect children in a similar fashion and suppress genetic expression.

This dissertation provided a theoretical account for the Scarr–Rowe hypothesis rooted in the quality of the family environment and parenting (e.g., Lareau 2011; Lareau and Weiniger 2003). Advantaged parents provide learning environments that are more child-centered and are more adapted to their children's potential and needs. This individual adaptation of children's learning environment matters, since it leads to better conditions for gene expression. Future research based on genetically sensitive designs is needed to test for the proposed mechanisms using direct measures of parenting behaviors.

Not only the proximate family environment but also the broader institutional environment mattered for the realization of genetic effects. The internationally comparative study including Germany, Sweden, and the United States, revealed substantial cross-country differences in educational inequality: Genetic influences on educational success were least important in Germany, and most important in Sweden. Within this comparative framework, evidence for the social stratification of genetic influences on educational success was weak. Nonetheless, the results provided indications of a social stratification of genetic effects in line with the Scarr–Rowe hypothesis for educational success in Germany and the United States, but not in Sweden. The results indicated that more egalitarian educational systems have a positive impact on the development of genetic potentials for educational success, and that early tracking might be an important factor in the suppression of related genetic effects. Yet, further research is needed to scrutinize the role of tracking. For example, after the 1960s, all Nordic countries changed from tracked to comprehensive schooling systems (for an overview of the educational reforms in these countries, see Gustafsson 2018). One could systematically study whether the relative importance of genetic influences increased after

the detracking period. Comparisons over socio-historic time based on fairly culturally homogenous groups increase the generalizability of the results. In addition, future studies using longitudinal twin data should study whether tracking is indeed associated with lower chances of genetic expression, and whether twins that attend different school tracks have different chances of genetic expression. “Most similar case designs” (Lijphart 1971) in addition to studies within single countries can help to better understand to what extent and why differences in tracking suppress genetic effects on children’s educational success.

At its core, this dissertation has shown that the consideration of genetic variation helps to better understand how differences in educational success among siblings emerge. While genes have been widely ignored in stratification research, the results showed that genes contributed significantly to the realization of educational success. However, to what extent children could realize their genetic potential for educational success differed by the environments they encountered. The findings showed that parents’ social position and stratified family environments mattered for genetic expression as well as macro-structural influences, particularly the strictness of the tracking system seemed to reinforce social disparities in genetic expression.

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B

Articles

Note: The format of Articles 1 through 3 differs slightly from that of the original published version.

1. Status Attainment and Social Mobility – How Can Genetics Contribute to an Understanding of Their Causes?¹

Martin Diewald, Tina Baier, Wiebke Schulz, and Reinhard Schunck

Abstract

This paper discusses why and how the consideration of individual genetic variation can enhance the explanatory power of sociological inquiries of status attainment and social stratification. We argue that accounting for genetic variation may help to address longstanding and in some cases overlooked causality problems in explaining the emergence of social inequalities –problems which may interfere with both implicit and explicit interpretations of a society as “open” or “closed,” as meritocratic or non-meritocratic. We discuss the basic methodological tenets of genetically informative research (section 1.2) and provide empirical examples and theoretical conceptualizations on how genetic variation contributes to status attainment (section 1.3). This is followed by a discussion of gene–environment interplay in relation to more abstract ideas about social mechanisms that generate inequality, touching on normative implications of these ideas as well as considerations from a social justice perspective (section 1.4). Finally, we briefly review the potential benefits as well as pitfalls of incorporating genetic influences into sociological explanations of status attainment. As we will argue, understanding how social influences impinge on the individual and how genes influence our lives requires sophisticated research designs based on sound sociological theory and methodology (section 1.5).

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1.1 Introduction

A central goal of sociological research is to explore how society shapes the individual life course and structures individual opportunities. But how should “the individual” exposed to societal influences be conceptualized? Social stratification and inequality research has addressed this question by focusing on social origins, which are generally defined in terms of parental social class, status, resources, and family structure. In this contribution, we demonstrate why and how the consideration of individual genetic variation over and above social origin can enhance the explanatory power of sociological and particularly social mobility research. Furthermore, we discuss how this endeavor might help to address longstanding and in some cases overlooked causality problems in explaining the emergence of social inequalities –problems which may interfere with both implicit and explicit interpretations of a society as “open” or “closed,” as meritocratic or non-meritocratic. Moreover, this discussion provides an example of how genetically informative research can contribute more generally to established sociological theories and research.

Sociology has developed several approaches to investigate the relationship between social origin and destination and the pathways that mediate between them. Social mobility research tends to proceed by studying associations between social origins and social outcomes, be they in social class (Erikson and Goldthorpe 1992), socioeconomic status, or material resources. The basic status attainment model developed by Blau and Duncan (1967) enlarged the connection between social origin and destination to include two additional pathways: One between social origin and education and another between education and destination. The idea of this model was to test whether status attainment based on social origins was being replaced by meritocracies based on education, which channel social mobility through educational and vocational degrees. The weaker the direct path from origin to destination and from origin to education and the greater the influence of education on destination, the more open in terms of the equality of opportunity provided to its citizens is a society assumed to be (Breen and Jonsson 2005). However, this interpretation is often dismissed as invalid since a strong family influence may also entail meritocratic processes, such as skill formation or motivation (Saunders 2002).

Over the years, this basic status attainment model has been extended in a number of ways, foremost through the Wisconsin model, which integrated interpersonal influences

and aspirations as mediating mechanisms and later cognitive and noncognitive skills (i.e., Haller and Portes 1973; Hauser et al. 2000; Heckman 2006). Life course research has added ever more detailed pathways from social origin to destination in different phases of life that are affected by a wide range of life experiences and social contexts, which are beyond the scope of this article to discuss in detail.

Nevertheless, even with relatively comprehensive measurements of social origins and skills, the overall impact of social origins and individual characteristics on educational and status attainment is still not fully understood, and the relative contributions of both may be biased by unmeasured characteristics (Jencks and Tach 2006; Smeeding, Erikson, and Jäntti 2011). Educational certificates are not simply an indicator of achievement and meritocratic selection, but may reflect social closure as well (Collins 1979). Conversely, residual impacts of the family of origin in status attainment models, not to speak of social mobility tables, may reflect not only ascription but also ability and effort. In other words, the research on individual characteristics and social influences that link social origin and destination is still ongoing and far from complete.

So far most of the studies on this subject have focused on unequal chances between members of different families. Status attainment models assume that children from the same family are influenced in the same ways and to the same degree by family processes and resources. Much less attention has been paid to possible inequalities created within families. Sibling research shows that the assumption of equality between siblings may need to be reconsidered, with attainment correlations between siblings of only about 0.5 (e.g., Benin and Johnson 1984; Conley 2008; Hauser and Mossel 1985; Hauser and Wong 1989; Sieben, Huinink, and de Graaf 2001). Thus, within-family differences in attainment may indeed constitute an important part of a society's inequality structure –yet one that has gone largely ignored so far in the research. The obvious differences between children from the same family point to the complex familial dynamics structuring unequal life opportunities far beyond those usually captured in status attainment research.

Moreover, parents not only pass on resources and experiences to their children, but also their genetic predispositions. Because of this, inequalities exist between individuals from birth on, not only in their social origins but also in their genetic endowments, negating the assumption underlying much of the standard social research that human beings are a “blank slate” at birth. As some sociologists have already suggested, acknowledging the role of genetics and incorporating it into sociological research designs may help to overcome the incompleteness and ambiguity of model parameters as

measures of achievement versus ascription (Adkins and Vaisey 2009; Freese 2008; Nielsen 2006).

In this paper, we try to develop the arguments underlying this suggestion a bit further. We start with a general discussion of what the heritability of social outcomes implies, including a brief introduction to the methodological tenets of genetically informative research, to address the question of how genetic variations shape social forces, and conversely, how social forces shape genetic influences (section 1.2). Section 1.3 explores the implications of this discussion for status attainment research and life chances at large: The genetic dimension contributes to a more complete and useful definition of the family of origin than purely social conceptualizations and allows addressing the interplay between genes and social environments. In section 1.4, we discuss processes of gene–environment interplay in relation to more abstract ideas about social mechanisms that generate inequality. This discussion also touches on normative implications of these ideas as well as considerations from a social justice perspective. Finally, we briefly review the potential benefits as well as pitfalls of incorporating genetic influences into sociological explanations of status attainment. As we will argue, understanding how social influences impinge on the individual and how genes influence our lives requires sophisticated research designs based on sound sociological theory and methodology (section 1.5). As we will demonstrate, considering both social and genetic factors jointly in such a way is also valuable for demographic research and the explanation of fertility (Kohler and Rodgers 2003; Kohler, Rodgers, and Christensen 1999; Tropf and Mills 2015) and mortality (Carey and Vaupel 2005; Vaupel 2004).

1.2 Genetic Influences and Social Science Research

1.2.1 Genetically Informative Research Designs: Methodological Tenets

There is increasing evidence that genetic variation plays an important role in explaining differences in individual outcomes (e.g., Freese 2008, Turkheimer 2000). However, integrating genetic influences empirically is a challenging endeavor. This section gives a broad overview of the methodologies and research designs rooted in the field of behavioral genetics that allow for a genetically sensitive investigation of social scientific research questions (for a more detailed discussion, see Kim 2009; Plomin et al. 2013).

There are two basic strategies for investigating how the interplay between nature and nurture influences life outcomes: quantitative and molecular genetic approaches. The first is to use behavioral genetic designs in which family members with different degrees of genetic and/or environmental similarity are compared, and the second is to include molecular genetic information. Quantitative approaches can be used to quantify the extent to which variation in a phenotype –any observable trait or characteristic of an organism– is related to genetic variation as a whole without knowing about which specific genetic variants are at work. Molecular genetic approaches offer techniques that can be used to analyze how and to which degree specific genetic variants directly affect phenotypes, which might be a smaller or bigger part of the overall genetic influence. These two approaches are not mutually exclusive but rather complementary strategies (e.g., Kendler 2001; Weinstein, Vauper, and Wachter 2008). We will discuss both approaches, but with a stronger emphasis on quantitative genetic approaches as they seem currently better suited to provide a more comprehensive picture of genetic influences on mobility outcomes. Additionally, we point to the importance of the gene and environment interplay that needs to be considered within these two approaches.

Quantitative Genetics

Quantitative genetics offers a means of indirectly assessing the relative contributions of genetic and nongenetic (i.e., environmental) factors in observable phenotypic variation by looking at phenotypic similarity in relatives with known (and different) *average* degrees of genetic relatedness.² The underlying idea is straightforward (Plomin and Daniels 2011): If a certain characteristic is influenced by genetic factors, relatives who are genetically more similar will be more similar in the characteristics of interest. This approach is best illustrated with the classical twin design, the “workhorse” of behavioral genetics (Plomin and Kosslyn 2001:1154).³ Monozygotic twins are genetically identical; dizygotic twins, in contrast, share –like full siblings– on average only half of their DNA.

² It is important to note that these designs rely on average known degrees of relatedness. For instance, dizygotic twins share 50% of their genes on average. A particular dizygotic twin pair may also share more, or fewer, genes.

³ There are also other types of genetically informative designs (i.e., the adoption design). All of them follow the same idea and use information on known degrees of genetic and/or environmental similarity (for an overview see i.e., Plomin et al. (2013)).

But both mono- and dizygotic twins grow up under same, shared familial conditions, so that influences of the shared environment can be assumed to be the same.

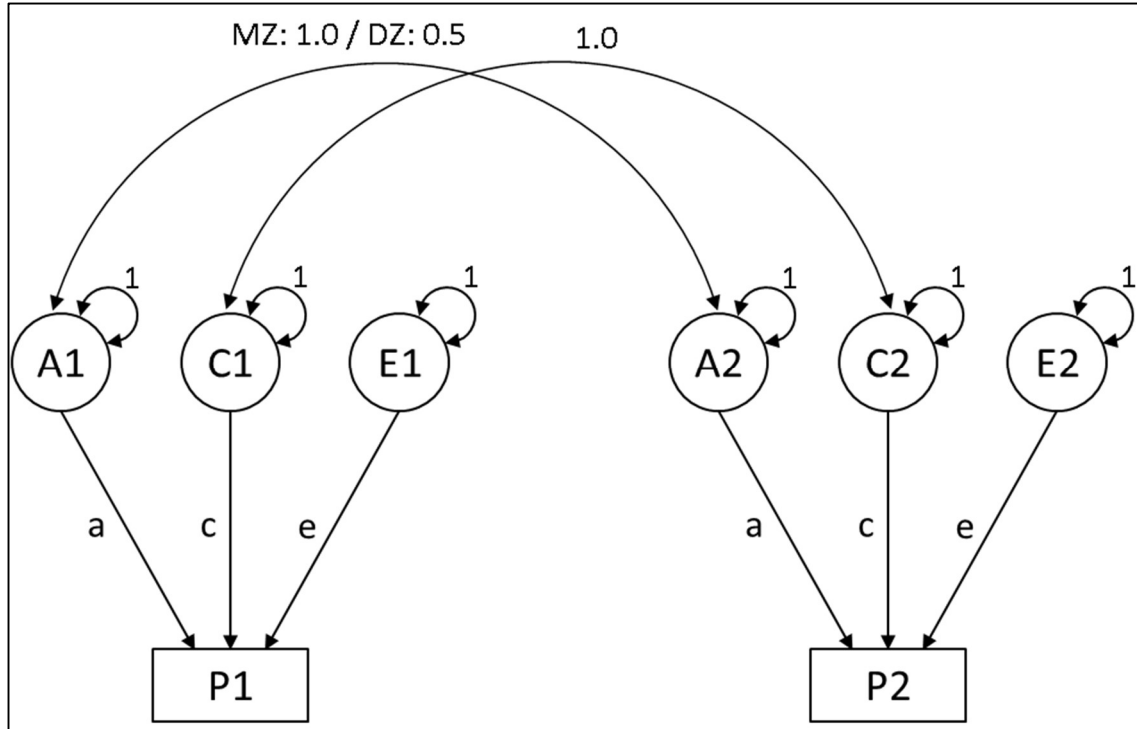
This information can be used to differentiate between the relative importance of genetic and environmental influences. The ACE model assumes that the trait under study (P, the phenotype) is produced through *additive* influences of alleles (A, the genotype), shared environmental factors (C), and nonshared environmental factors (E). With this model, we can estimate how much phenotypic variance is due to genetic variance and how much is due to environmental variance –that is, we can estimate *heritability*.⁴ Total phenotypical variance (σ_P^2) is therefore assumed to being the sum of the variance components of A, C, and E

$$\sigma_P^2 = \sigma_A^2 + \sigma_C^2 + \sigma_E^2 \quad (1)$$

These variance components can be estimated, for instance, via structural equation modeling, as displayed in Figure 1.1.

⁴ This is called narrow-sense heritability, because it only estimates the proportion of variance due to additive genetic effects (Purcell 2013:381).

Figure 1.1 ACE Path Diagram



Note: ACE Path diagram includes expected correlations among MZ and DZ twins.

Heritability in the narrow sense (h^2) is defined as the share of the total variance attributable to additive variance of additive genetic effects⁵

$$h^2 = \frac{\sigma_A^2}{\sigma_A^2 + \sigma_C^2 + \sigma_E^2} \quad (2)$$

For instance, a heritability estimate for IQ of about 50 to 60% (Bouchard and McGue 1981; Deary et al. 2009; Plomin et al. 2013), shows that 50 to 60% of the total observable variance in IQ is based on variance in additive genetic factors. The model is simplistic and relies on rather strict assumptions. It ignores non-additive effects, that is that alleles can interact with each other (I, epistasis) or suppress other alleles (D, dominance deviations) or that genes and environment may correlate or interact (see below) (e.g., Plomin et al. 2013). Additionally, it is assumed that there is no assortative mating of parents, and that MZ and DZ twins grow up under similar conditions (the so-called “equal environment assumption”) and are treated equally by their social environment (Derks,

⁵ Heritability can be also be estimated through mixed effects (multilevel) models and DeFries-Fulker models.

Dolan, and Boomsma 2006; Scarr and Carter Saltzman 1979). If there are non-additive genetic effects or, more generally, if any of these assumptions are violated, estimates of heritability will be biased (Visscher, Hill, and Wray 2008). The main reason for imposing these strict assumptions lies in data limitations. More complex models require data on more than just twins.

Besides estimating the relative influence of genetic and environmental factors on individual traits, multivariate models can also be used to assess the extent to which variance in different phenotypes is due to the same genetic or environmental factors (Posthuma 2009; Purcell 2013:393).

Taking the aforementioned assumptions and limitations into account, it becomes clear that only by properly accounting for social influences genetic factors can be estimated accurately –and vice versa. The “extended twin family design” (ETFD) is a promising research strategy as it includes not only mono- and dizygotic twins but also various other types of family members (e.g., Keller et al. 2009). These differences in kinship can be exploited to provide more rigorous estimates of genetic influences (Coventry and Keller 2005; Posthuma and Boomsma 2000). Adopting the ETFD makes it possible to relax assumptions and thereby capture the different influences more accurately. In particular, the ETFD can help to distinguish the effects of shared and nonshared environments and thus to identify the different causes of a given outcome.

Interpreting Heritability

Although estimating heritability has been a major focus of behavioral quantitative genetics in recent decades, this line of research is relatively new in other social sciences and may be misunderstood. Before we come to a substantial interpretation of heritability estimates we first discuss the underlying concept of heritability estimates and their limitations (Plomin et al. 2013; Shanahan, Hofer, and Shanahan 2003, Turkheimer 1998, Visscher, Hill, and Wray 2008). First, it is important to note that heritability estimates are population- and time-specific (Plomin et al. 2013:92). A high heritability estimate of approximately 80% in height (Carmichael and McGue 1995), for instance, does not indicate that the environment is unimportant. Height has increased substantially in Western societies over the twentieth century due to environmental factors including nutrition (Shanahan, Hofer, and Shanahan 2003:608). Heritability estimates refer to a specific social system, point in time and population (or sample). They can therefore be “expected to vary across societies, historical periods and social contexts” (Nielsen

2006:208). The fact that heritability estimates vary according to environmental influences is an important clue towards the interplay of environment and genes. The consistently higher heritability in educational achievements for men as compared to women is one finding that suggests that context influences work differently for the realization of the genetic dispositions of men and women (Branigan, McCallum, and Freese 2013). Second, high heritability therefore does not imply that environmental factors cannot mitigate or even override genetic effects, as the height example indicates. Third, heritability estimates cannot be treated as “fixed” properties of a given trait: Any increase in environmental differences in a sample automatically decreases the extent to which genetic factors contribute to the variation, and vice versa, as equation (2) indicates. Fourth, heritability estimates are population parameters, and cannot be used to explain genotype-phenotype links at the individual level (Shanahan, Hofer, and Shanahan 2003:607). A heritability estimate of 0.8 for height means that on average 80% of observed differences in height in a population can be attributed to genetic and 20% to environmental differences. It does not mean that 80% of individual height is determined by an individual’s genes. Heritability by no means implies genetic determinism (Plomin et al. 2013:93–94), as it does not say anything about the specific genes and causal mechanisms that produce a specific phenotypic expression (Conley, Strully, and Bennett 2003; Johnson et al. 2009; Turkheimer 1998). Fifth, some phenotypic traits that are under strong genetic control – for instance, bipedalism – will show no heritability in standard behavioral genetic designs because there is no (or too little) variation (Shanahan, Hofer, and Shanahan 2003:608) as evident in (2), although they are obviously inherited.

Taken together, heritability estimates do not tell us anything about the causal mechanisms that eventually lead to an observable outcome (Turkheimer 1998). Nonetheless, heritability has important implications for sociological explananda. If we accept that all traits are heritable to some degree, a correlation between parents and children cannot be simply seen as “prima facie evidence for sociocultural causal mechanisms” (Turkheimer 2000:162). Conversely, however, heritability cannot be seen a prima facie evidence of causal genetic mechanisms.

Causal Environmental Influences

Estimating heritability is just one possible way of exploiting the genetically sensitive twin design. One interesting implication of the idea that genetics affect all life outcomes –now general consensus in behavioral genetics (Johnson et al. 2009; Smith and Hatemi 2013;

Turkheimer 2000)— is that twin designs are capable of estimating causal *environmental* influences (Johnson et al. 2009). Standard empirical research in the social sciences, which does not control for genetic endowments, implicitly assumes that the observed correlations are not linked by genetic factors (Smith and Hatemi 2013). If social mechanisms are confounded by genetic factors, however, neglecting genetic influences will give us incorrect answers. For instance, if there is heritability in ability and schooling (as evidence shows, see below), then any assessment of how social origin impacts education and of how education impacts social outcomes will be severely biased due to unobserved genetic heterogeneity. However, by focusing on discordance in twin pairs, we open up the possibility of adjusting for (unobserved) genetic and shared environmental confounders (Johnson et al. 2009; Kohler, Behrman, and Schnittker 2011). If we focus on discordance, that is, differences within twin pairs, we can estimate twin fixed effects models controlling for genetic confounding (Conley and Rauscher 2013; Fujiwara and Kawachi 2009; Kohler, Behrman, and Schnittker 2011). Suppose we are interested in estimating the effect of education (x) on occupational status (y). Using information on monozygotic twins and displaying this as a regression model leads to

$$y_{ij} = \beta_{MZ}x_{ij} + A_i + D_i + I_i + C_i + E_{ij} \quad (3)$$

P is substituted by y_{ij} with the subscript i denoting family (or twin pair) and j the respective twin. As monozygotic twins are genetically identical, A_i, D_i, I_i are the same for every twin pair—as are the shared environmental influences C_i . However, this model will be biased if there is any unobserved heterogeneity in genetic or environmental influences. Focusing on discordance, an MZ twin fixed effects model as in

$$(y_{i1} - y_{i2}) = \beta_{MZ}(x_{i1} - x_{i2}) + (E_{i1} - E_{i2}) \quad (4)$$

is much less restrictive, since all genetic (A_i, D_i, I_i) and shared environmental components (C_i) drop from the equation. Thus, no assumptions on possible correlations with the independent variables are necessary, and we can estimate the effect of x on y controlling for *all* genetic and shared environmental endowments.

Molecular Genetics

Molecular genetic techniques examine genetic influences directly. Thus they are able to provide analysis of specific genetic influences that go beyond heritability estimates. This is supported by an increasing number of large-scale studies that have begun to provide molecular genetic data (e.g., the National Longitudinal Study of Adolescent Health [Add Health], the Panel Study of Income Dynamics [PSID], or the Framingham Heart Study [FHS]; Beauchamp et al. 2011). Molecular genetic studies seek to identify specific genetically determined biological processes affecting behavior and provide a variety of techniques to examine the relationship between genetic variation and individual differences (for an introduction, see Purcell 2013). Genetic variation between individuals is detected through genotyping. Genotyping procedures scan the entire human DNA and determine the individual's exact genotype (Purcell 2013). Two approaches that can detect these effects are being used to an increasing degree in the social sciences (see Beauchamp et al. 2011; Hatemi et al. 2011): The candidate gene association approach and the genome-wide association approach. Broadly speaking, association studies seek to pinpoint to associations between differences in individual human DNA and the trait of interest. Whereas the genome-wide association approach focuses on finding associations (quantity), the candidate gene approach is more interested in understanding the associations (quality).

As promising as it sounds to directly pinpoint the genetic variation that leads to phenotypic variations, we are far from being able to infer causal relationships. The difficulties inherent in this method result from social scientists' interest in complex traits (determined by genetic and environmental factors) rather than monogenetic traits (determined by a single gene) (Adkins and Guo 2008). To date, these approaches suffer from our limited knowledge about the effects of specific candidate genes on behavioral outcomes (Conley 2009). Here, it is likely that other mechanisms are causing spurious relationships (e.g., Beauchamp et al. 2011; Hatemi et al. 2011; Purcell 2013) and that results are confounded by interaction effects (between different genes or between genes and environment) that cannot be accounted for without deeper knowledge of how DNA operates. So far, results of association studies have seldom been replicated (i.e., Beauchamp et al. 2011). As Beauchamp et al. (2011) remark in light of the difficulties entailed in measuring genotypes and phenotypes, it is important to include environmental factors. Further research has to integrate both factors, as one cannot be estimated without the other. Molecular genetics and quantitative genetics can play a complementary role in

this approach, thereby producing more sensitive estimations (Kendler 2001; Weinstein, Vauper, and Wachter 2008).

1.2.2 Genotype-Environment Interference

The most interesting and promising pathway for integrating genetically sensitive research designs into the research on social stratification and inequality is to investigate how genes and social environment produce phenotypic outcomes in the form of gene–environment interactions (GxE) and gene–environment correlations (rGE).⁶

A gene–environment interaction refers to processes by which genes alter an individual's actions towards specific features of the environment and vice versa (Shanahan and Hofer 2005). Put differently, genetic effects can vary across social groups, situations, and societies (i.e., Johnson and Krueger 2005). The social context can operate in various ways, and so far four ideal types of GxE interactions have been differentiated (Shanahan and Hofer 2005).

The first type, triggering, means that a person has a genetic vulnerability that is expressed only in specific social situations. For example, individuals with a genetic predisposition for depression are more likely to suffer from depression when having experienced a stressful life event earlier in their lives (Silberg et al. 2001). Here the social context works detrimental and triggers the occurrence of a genetic risk.

The second type, compensation, refers to the opposite: Here, the social context is enriched and positively impacts individual functioning by hindering the expression of a genetic risk. Aggressive behavior can be prevented when growing up in intact families with warm relationships for instance (Kendler et al. 1995). Compensation and triggering do not necessarily represent an absolute dichotomy, they can rather be seen as two ends of a continuum.

In the third type, the environment serves as a mode of social control, which sounds similar to the latter but refers to (institutionalized) belief systems (i.e., norms) that are embedded in the social context. Here, individual behavior is restricted by the inherent

⁶ This section describes patterns of the interplay of environmental and genetic factors. Genetic expression can be triggered by many mechanisms which are not discussed in this article. However, the newly evolving field of epigenetics provides promising insights on how environmental factors affect genes and therefore alter genetic expression without being inherited (for a discussion on epigenetic mechanisms, see Shanahan and Hofer (2011)).

rules of the system. The difference to compensation (i.e., avoidance of low levels of functioning) lies in the substantial mechanisms. The social control mechanism describes the limitations to individual's behavior which prevent the realization of a genetic predisposition.

The fourth type, enhancement, describes a social context that increases the genetic predisposition towards socially valued or accepted characteristics or behaviors. The difference to the first type is that enhancement refers to processes and interactions which increase positive functioning. The effect of genetic predispositions is accentuated via training or good parenting for example.

Other processes in which genes and environment affect each other are referred to as gene–environment correlations. Despite their name, gene–environment correlations describe a causal relation between context and behavior. A gene–environment correlation occurs when individual exposure to an environmental context depends on the genotype and vice versa (Jaffee and Price 2007). Three types of gene–environment correlations have been identified (Plomin, DeFries, and Loehlin 1977): Passive, evocative, and reactive.

A *passive gene environment correlation* occurs when social environments appear according to inherited characteristics. Take the example of musical parents and their children. Musical parents raise their children in an environment that motivates their children to become musician themselves (i.e., instruments at home, listening to music). Being musical might also be genetically transmitted. These children passively receive a social context that fits to their genetic predisposition. An *evocative correlation* describes a situation in which genetically transmitted characteristics provoke specific reactions from the environment. For example highly talented children might receive special attention from teachers which reinforces their talents. Lastly, an *active correlation* can be understood as a self-selection process in which individuals actively seek contexts or niches that matches their genetically transmitted interests.

Considering both processes –gene–environment interactions as well as gene–environment correlation– will provide a more profound understanding of how the interplay of social and genetic force jointly shapes life outcomes. Gene–environment interactions reveal how genes take effect through the environment and vice versa. Gene–environment correlation comes into play when the individual genetic make-up affects environmental influences –either directly, through individual behavior, or indirectly, through selection. The existing literature clearly indicates that genes and environmental

factors do not affect life outcomes independently from each other. In situations in which genetic predispositions only unfold in certain social environments, heritability estimates tend to overestimate the impact of genetic factors as they can only tell us that genes matter but not how and under which circumstances. Heritability estimations appear in this sense to be a good starting point as they indicate that social outcomes are genetically confounded. But without further investigations heritability estimation should not be over-interpreted as we do not know whether social conditions mediate these effects.

Neglecting these processes may lead to mistaken conclusions about social influences if one interprets behavior as driven solely by social causation. Acknowledging unobserved individual genetic heterogeneity therefore substantially improves our understanding of how social inequality outcomes are shaped. Sensitive estimations have to take into account the mutual dependency between genes and environment. However, disentangling these complex patterns of genome-environment interrelationships requires interdisciplinary expertise and sophisticated research designs. Applying genetically informative designs makes it possible to go beyond a mere statistical association between genome and outcome and derive explanations based on a chain of interlinking causal factors.

1.3 The Relevance of Genes for Status Attainment: The Interaction of Genetic Variation and Social Mechanisms

Up to now, there have been surprisingly few genetically sensitive analyses of occupational status, one of the most frequently employed operationalizations of inequality in sociological research. The few studies that have examined the heritability of occupational status indicate that genetic factors play a substantial role in explaining individual differences in occupational status. Fulker and Eyseneck (1979) find that MZ twins are more similar in occupational status than DZ twins which indicates a heritable component. Tambs et al. (1989) replicate the heritability of occupational status across cohorts born in the first half of the twentieth century. However, both of these studies base their analyses on rather crude measurements of occupational status. Further investigations are needed to gain a precise assessment of the association between genetic factors and status attainment.

There are, however, an abundance of heritability estimates concerning psychological and physical antecedents of attainment. Most studies have focused on the heritability of cognitive skills such as IQ, with an average variation in IQ of around 50% to 60% due to genetic influences (Bouchard and McGue 1981; Dearly et al. 2009; Plomin et al. 2013). Noncognitive abilities have been studied in the form of economic preferences (Cesarini et al. 2009; Zyphur et al. 2009) and personality traits (for a review of genetic influences on the Big Five personality traits, see Johnson, Vernon, and Feiler 2008). Considerably fewer studies focus on classical elements of the status attainment model such as educational attainment or income. A growing number of studies in this domain assesses the heritability of years of schooling (Behrman and Taubman 1989; Behrman et al. 1980; Rowe, Vesterdal, and Rodgers 1998), examination performance in school achievement tests (Bartels et al. 2002; Nielsen 2006; Plomin et al. 2013), and broader measures of school achievement such as grades (Johnson, McGue, and Iacono 2005, 2006, 2007). However, there is considerable variability of genetic influences on educational attainment across different contexts (Branigan, McCallum, and Freese 2013), indicating a complex interplay between genes and environments. A relatively large number of studies assess the genetic components of income, on average, earning correlations in the incomes of MZ twins are around 0.6 (Bowles and Gintis 2002; Rowe, Vesterdal, and Rodgers 1998). Most recently, Benjamin and collaborators (2012) calculated the heritability of income: For men, 58% of 20-year income can be explained by genetic factors, compared to 46% for women.

Taken together, the current research unequivocally demonstrates that excluding the genetic component of intergenerational transmission omits an integral part of the story (Freese 2008). Nevertheless, for social inequality research to fully benefit from information on genetic variation, it is necessary to understand precisely how this information can enrich the existing theory and research. We discuss this in two steps. First, we explore the consequences of considering genetic variation either in addition to or instead of social origin in the study of status attainment. Second, we apply the formal interaction and covariance patterns presented in section 1.2 to processes and social mechanisms discussed in the sociological status attainment research.

1.3.1 Social Mechanisms as Generative Processes: The Family of Origin as a Social and Genetic Point of Departure

In the research on social inequalities, parental social class, status, resources, and more recently family type are treated as the key features to assess the impact of the family of origin for later life chances. However, this convention raises theoretical as well as methodological concerns, especially for a mechanism-based explanation of status attainment. A fundamental theoretical concern is that if we want to explore how individuals maneuver themselves through the opportunity structures of a society, we need a conceptualization of individuals prior to being subjected to these socially shaped opportunities. Genetic variation offers a potential starting point. Namely, social background and other familial circumstances are already part of this opportunity structure and do not predate them (Diewald 2010). A commonly held ontological understanding of social mechanisms, as substantive mechanisms (Diewald and Faist 2012; Gross 2009), requires that a clearly defined point of departure, or cause, be distinguished from an effect and the generative processes that actively produce this effect (e.g., Machamer 2004:34). Taking social origin as the starting point thus confounds cause and generative processes. This statement does not completely preclude taking social origin as a point of departure for the study of status attainment. The argument put forward here is that social origin is a poor concept for “origin” in a strict sense.

As we have illustrated in section 1.2 whole genome effects as well as the effect of shared environmental influences are “black boxes” as they capture (quantify) both types of influences without having them specified. The effect of the shared environment includes family characteristics usually measured in attainment research but also those usually not measured for example infrastructural and cultural environments such as neighborhoods. Especially for young children, the shared environment estimate should closely approximate a total family effect. Thus, the systems of stratification in different societies could be described by quantifying the influence of genetic forces on the attainment process compared to shared environment or social origin (Nielsen 2006).

Heritability of attainment can be compared across subgroups (e.g., men versus women, native versus immigrants (Branigan, McCallum, and Freese 2013)), over historical time (e.g., during an economic crisis) or between national contexts (e.g., stable societies and societies in transition). Such comparisons can provide valuable information about the variability of genetic expression with respect to a specific outcome. One example is the study of the heritability of educational attainment in relation to historical

changes in educational policies. According to Heath and collaborators (1985), parental education and genetic factors are each responsible for around 40% of the variation in educational attainment in cohorts born early in the twentieth century. Later in the twentieth century, among men, the relative importance of genetic differences increased and that of family background decreased. In women, over the same period, the heritability of educational attainment changed little. The authors attribute the increase in genetic influences to changes in educational policies that increased access to education (see also Branigan, McCallum, and Freese (2013) for a meta-analysis of educational attainment).

Such comparisons of heritability across subgroups can be understood as relational inequality (Tilly 1998). Here, the social distribution of opportunities for attainment or for social mobility is examined by comparing the levels at which different social groups are achieving their genetic potential for success. Thus, looking at genetic variation as a cause of differential attainment fits into the broader sociological frameworks of social mechanisms that transform heterogeneities into inequalities (Diewald and Faist 2012). Higher heritability implies that genetic endowments can realize and lead to socially unhindered opportunities for attainment. Lower heritability estimates indicate that social factors limit the realization of genetic potential. A number of recent studies illustrate the variability of the genetic components of IQ depending on the socioeconomic status (SES) of the family. In low-SES families, most variation in IQ is attributable to shared environment and very little to genetic influences. In more affluent families, this relation is reversed: Most variation in IQ is due to genetic influences and very little to the shared environment (Turkheimer et al. 2003; Nisbett et al. 2012).

Recent research has also shown how parental SES and the quality of parent-child relationships interact with genes. Social and genetic influences are interwoven from the very beginning (Chen et al. 2011; for a summary, see Shanahan 2013). In consequence, it is difficult to interpret what role social origin and other social influences play in a particular outcome such as educational attainment, because measured social origin effects may partly reflect genetic predispositions for effort and ability as well. And ability and effort, even when measured at early ages, might not only reflect innate talent but influences of social origin. Therefore to interpret trends in attainment and mobility, several authors (Björklund, Jäntti, and Solon 2005; Jencks and Tach 2006) have emphasized the importance of studying patterns of genetic variation, arguing that if family environment is not separated from genetic relatedness, this can mask differential or even contradictory developments in gene expression in the family's social characteristics (see

Branigan, McCallum, and Freese 2013). What the “shadow of the family of origin” actually means may change over time, even if the total family effect remains the same. And if it changes, this could be due to variability in the influence of either genetic relatedness or the family’s social characteristics, or both.

1.3.2 Patterns of Gene–Environment Interference Determining Socioeconomic Attainment

Genes matter for a person’s position in society, though there is no gene for income, socioeconomic status, or social class. The only characteristics directly influenced by genes are those that lie “underneath the skin.” In other words, genetically based similarities in attainment between parents and children must be explained by physical or psychological characteristics that are relevant to reach status relevant outcomes. Genetically transmitted characteristics influence individual behaviors and evoke different reactions in the environment, resulting, for example, in different labor market outcomes and recruitment to different jobs.

A common extension to better assess the effect of social origin on status attainment is to examine the impact of cognitive and noncognitive skills, which are considered important for success in education, training, and employment (Bihagen, Neramo, and Stern 2013; Jackson 2006; Kanfer, Wanberg, and Kantrowitz 2001). These traits, which are considered to be productivity-enhancing (Bowles, Gintis, and Osborne 2001), also have a heritable component, as described above. Other possible important characteristics which are included less often in the analyses of status attainment are physical and mental health, physical attractiveness, height, and weight. Their status as productivity-enhancing attributes is more doubtful, although they might function as such in some areas and not in others (Jackson 2006). Even less positively valued traits such as aggression may contribute to successful attainment as well. Others, such as skin color, definitely do play a role, while having no relation at all to ability or effort. This still incomplete set of very heterogeneous characteristics reveals that whole-genome effects are difficult to interpret in a substantial way.

However, the role of such personality characteristics and skills to mediate the influence of genes on socioeconomic attainment may be overestimated. As Jencks and Tach (2006:38) state, “... genes are not generating intergenerational economic resemblance primarily by influencing IQ.” The moderate effect of skills on the link between genes and

attainment may also be due to the fact that concepts like IQ, risk aversion, time preferences, conscientiousness, and health are less proximal to genes.

An alternative strategy for studying how physical and psychological characteristics affect the interplay between genes and socioeconomic attainment is to investigate endophenotypes, which refer to more general patterns of the organism's reaction to environmental influences that are also more proximal to genetic influences (i.e., Chen et al. 2011). Moreover, they refer to mechanisms of transcription regulation that are relevant for a broader range of developments, some of which –like behavioral problems and deviant behavior– are often unobserved, despite being relevant for attainment. Shanahan (2013) provides a number of examples of a “durable programming of the stress response system”, distinguishing between “fight or flight” responses to stressors. Such patterns of transcription regulation may play a crucial role in the link between social origins and socioeconomic outcomes, because on the one hand they begin to operate very early in the life course during the sensitive period around birth, with parental SES and parent-child relationships exerting a major impact on the activation or repression of genetic activity that regulates stress (Shanahan 2013). On the other hand, stress regulation appears to play a crucial role in brain development, which in turn is important for later educational and socioeconomic attainment, as reflected, for example, in a higher IQ (Nisbett et al. 2012:152).

We refer to both characteristics and endophenotypes as well as to the four types of gene–environment interactions mentioned in section 1.2: Triggering, compensation, social control, and enhancement (Shanahan and Hofer 2005). However, we differentiate consistently between characteristics and behaviors as distinct levels at which development can occur. Characteristics and behaviors can be favorable for or detrimental to attainment. Because of this, these behavioral genetic concepts can be integrated into a more general framework of risk, risk accumulation, and risk compensation (Diewald 2011).

In life course research, risk exposure is commonly defined by the presence of risky events or episodes such as divorce, unemployment, or poverty in the life course. However, from a behavioral genetic perspective, the definition of risks starts with heterogeneity in the genetic propensity to exhibit certain “embodied” characteristics that play a role in socioeconomic attainment. These characteristics may result in either risk-averse or risk-prone behaviors or serve as criteria for institutional and organizational selection into more or less risky locations and positions. Contrary to the conventional view, this

understanding of “risk” should not be confined to the emergence of negative characteristics and behaviors (e.g., aggression, anxiety). Risk also comprises a low or no propensity to exhibit favorable characteristics or the failure to realize existing genetic potential in areas such as cognitive skills or self-control. Social risks or risk compensation emerge in three steps from genetic propensities to exhibit different characteristics and behaviors:

- a) as the development of favorable or detrimental physical or psychic characteristics;
- b) as the manifestation of such embodied characteristics (i.e., aggression) in favorable and detrimental observed behaviors;
- c) as unequal attainment resulting from these characteristics and behaviors.

In short, the blocking of detrimental characteristics and behaviors and the activation of favorable ones is good for socioeconomic attainment. Step c is then the traditional realm of sociological life course and attainment research.

To give an example of the second step: In line with the sociological adage “a gene for aggression lands you in prison if you’re from the ghetto, but in the boardroom if you’re to the manor born,” sociologists often question whether general, genetically based traits and skills ultimately constitute important factors determining life opportunities (Conley 2009:238). There are at least two possible reasons why the same genetic propensity could express itself in such divergent ways: A disposition toward aggression in upper-class children is either transformed into situation-specific, culturally accepted “know-how” – skills that make a positive difference in the sense of “power” or assertiveness– or this disposition is effectively eliminated. The traditional thinking on the gene–environment interaction tends towards the latter interpretation, which sees this as a social control mechanism by which upper-class parents attempt to socialize their children and discourage overtly offensive behavior. But the latter interpretation may be valid as well.

The bulk of gene–environment interaction studies deal with such proximate contexts as family environment, measured as socioeconomic status (Turkheimer et al. 2003) and extensions which include family form and ethnicity (Guo and Stearns 2002). However, contexts shaping gene expression are located also at more distal levels: In neighborhoods, educational and work contexts, and societal institutions. Up to now, these multilevel interdependencies have been researched little with respect to genetic influences (for notable exceptions, see Boardman, Daw, and Freese 2013; Branigan, McCallum, and Freese 2013). The proximate and distal levels do not work independently of one another but may constitute chains of risk generation and risk compensation over the life course.

For example, the family context may trigger or exacerbate a genetic predisposition toward deviant behavior that threatens educational success. Although this threat may be counteracted by mentoring programs in schools, such programs may fail to produce the desired long-term effects because the schools are situated in disadvantaged neighborhoods. Thus, in sum, the extent to which genetic predispositions toward specific traits that may affect socioeconomic attainment are expressed and actually affect the life course is shaped by the multilevel contexts in which individuals live, both simultaneously and successively. Nevertheless, recent interdisciplinary life course research suggests that experiences in the sensitive, very early years of life are especially important in the long run, though not in a deterministic way (Shanahan et al. 2014). Insofar as they trigger or block genetic predispositions to traits that affect attainment repeatedly, and that are exacerbated by active as well as evocative gene–environment covariance, these experiences are decisive in cumulative advantage or disadvantage over the life course (DiPrete and Erich 2006). Genetic differences also affect the ways members of a society treat one another and how they choose their environments. Thus, there are hardly any environmental effects that are not confounded with genetic differences (gene–environment covariance; see Manuck and McCaffery 2014:62).

1.4 Genetics and Attainment: Normative Implications

In the discussion above, we underscored that genetic information can extend our knowledge of intergenerational transmission and can help to more precisely identify social causes of attainment. In the following, we discuss how genetic information can be treated in the framework of abstract-theoretical social mechanisms prominent in sociological inequality research (Diewald and Faist 2012) and what normative implications this has for interpreting the genetic causes of attainment.

While the interpretation of social origin is a subject of widespread discussion in sociology, the impact of genetic variation and its interpretation in the light of equal opportunity concepts is far less discussed. Nevertheless, sometimes implicitly, sometimes explicitly, sociologists tend to interpret the whole-genome effect as “opportunity for achievement” (Nielsen 2006:193), or openness of the opportunity structure: “Favorable environments, permitting fuller expression of potential, are characterized by high

heritability. Unfavorable environments, inhibiting expression of native talent, are characterized by low heritability” (Nielsen 2006:198). The underlying assumption is that the whole-genome effect on attainment is due to meritocratically legitimate differences in genetic endowments, and that the higher the proportion of socioeconomic attainment explained by genes, the more this genetic potential can develop without social barriers. In other words, the development and effect of talent is not restricted by social closure in access to favorable educational tracks and jobs, and not restricted by exploitation in cooperative relationships. To put it in a nutshell: In a world without social barriers, the heritability of status attainment would be 100%. Moreover, if we appreciate a society with a less restricted unfolding of genetic predispositions for socioeconomic attainment as “open”, we implicitly agree that individuals must accept their good or bad luck in the gene lottery in the sense of self-ownership which means that “a person has a right to benefit from his personal genetic constitution, [because] [...] it is an important part of what constitutes him as a person” (Roemer 2012:484).

However, this view can be challenged in at least two respects. First, as discussed in section 1.3, inherited traits comprise not only meritocratically legitimate talents but also skin color, height, and other ascriptive characteristics, which can by no means be seen as achievement-related and legitimate sources of inequality. If ascriptive characteristics play a decisive role, heritability does not necessarily represent openness but to some unknown extent social closure as well. From this discussion, it is evident that one should not speak of heredity as a measure of openness or “opportunity for achievement”, based on meritocratically legitimate means but in a more neutral way as “opportunity for socioeconomic attainment” based on whatever inherited characteristics. Without further information, opportunities for attainment could be defined by the unrestricted realization of innate talent or by the use of stereotypes for opportunity-hoarding, or both. Only if ascriptive characteristics are removed from the whole-genome effect by comparing the heritability of attainment in related subgroups, such as men and women, blacks and whites, migrants and non-migrants, can we approach a substantive interpretation.

Second, it can be argued against the self-ownership argument that individuals should be compensated for “bad luck” in the gene lottery since it is a fate for which they cannot be held responsible. Why should social origin, but not genetic origin, be interpreted as social closure? Is the opportunity structure more open if life chances are largely inscribed in the gene instead of being shaped by parental resources? In other words, the ultimate outcomes of the gene lottery can hardly be interpreted as pure individual achievements.

From one point of view it could be argued that the gene lottery represents social closure. On the other hand individual endowments may be able to compensate for social closure by increasing openness. To be clear: The difference between this and the former, more common interpretation is not a difference in content but in the underlying (philosophical) justice considerations (for a more detailed discussion, see Nussbaum 2000).

These normative issues are easier to discuss when using clearly defined and conjointly judged characteristics and behaviors that lead to specific socioeconomic outcomes. If genetic endowments with talent can unfold more freely in the “richer” environments of high-SES families but to a much more limited degree in low-SES families (Guo and Stearns 2002), this would indicate a need for social policy interventions. And if children’s genetic propensities for stress resistance are blocked in low-SES families, producing detrimental long-term effects, a society should address this problem by compensating for unwanted social closure or by preventing it more effectively from the outset.

Finally, genetic variation alone can lead to social closure and even to social exclusion or exploitation if genetic traits are used by a society and its institutions as selection criteria. Young (1958), who coined the term meritocracy in his satirical science fiction novel “The Rise of the Meritocracy”, described how genetic tests are introduced in the Great Britain of the future to screen for achievement potential. Here, contrary to any notion of openness, and justified by efficiency arguments, a favorable screening result provides subjects with an exclusive ticket to higher educational opportunities and better jobs. Yet such symbolic mechanisms are not completely unknown today. While the German school system does not make direct reference to genetic endowments, its tripartite structure is founded on the idea of providing different tracks for the different types of innate talents present in the population.

1.5 Conclusion

Can genetic variation make a significant contribution to sociological inquiry? Could the inclusion of genetic information challenge the purely social explanation of attainment and social mobility, alter the size and significance of social origin effects, or even lead to a new understanding of the social mechanisms linking social origin and destination? Our answer to these questions based on the current research is affirmative. Including genetic

factors to complement and enrich the conventional way to assess social origin influences opens up new perspective in social scientific research.

The recent literature indicates that genetic and social origins play varying roles in the overall family-of-origin effect depending on historic and other contextual conditions. Nevertheless, genetically sensitive approaches stress the vital importance of different parental influences, both early as well as later in life. Sibling studies and genetically informed studies have demonstrated repeatedly that the common approach of looking at standard indicators of between-family variation in social origin captures only part of what decisively affects a child's educational and socioeconomic attainment. What is more, these measures are confounded with genetic variability, calling into question the validity and the relevance of the results (Arrow, Bowles, and Durlauf 2000; Bowles, Gintis, and Osborne 2001).

As there is still a widespread lack of rich data encompassing various social contexts as well as genetic factors, we do not yet know how much we will profit from behavioral genetics when analyzing social mobility and status attainment processes. Nevertheless, our discussion points in several potentially fruitful directions for future social mobility research. To address the complex interplay between environmental and genetic influences (section 1.2 and 1.3), research should focus on gathering data that comprise a number of differently related individuals from heterogeneous social environments. This is achieved on the one hand by collecting representative data that include families and respondents from the whole range of social strata. On the other hand, the data need to be longitudinal to allow an individual to be followed across the life course through encounters with broader social contexts including neighborhoods, schools, and work environments, all of which relate to the individual's social and genetic origins and earlier experiences. The extended twin family design (ETFD), combined with molecular genetic information, offers the most promising approach to assess the interplay between social and genetic influences and how this interplay unfolds over the life course.

The benefits of disentangling the genetic and social components of the total family of origin effect are by no means restricted to social inequality research. This is not least demonstrated in demographic research and especially research on fertility behavior. Several investigations have shown that fertility is partly in our genes and that genetic and social effects depend on one another (Kohler and Rodgers 2003; Kohler, Rodgers, and Christensen 1999; Mills and Tropf 2015). As such this statement is not astonishing, since genetic variation may be related to genetically influenced variation in fecundity.

However, genes related to fecundity can neither explain the development of fertility (and the varying contribution of genes to it) over historical time nor the changing role of educational attainment as determinant of fertility (Kohler, Rodgers, and Christensen 1999). Cohort comparisons show that the difference between no parenthood and at least one child and the age at first attempt to have a child seem to be more influenced by genetic variation than the completed fertility as the number of children one gets over the life course. As Kohler, Rodgers, and Christensen (1999) suggest, genetic variation contributes to fertility more over variation in preferences for parenthood than over –biological and/or material– resources to take over the responsibility for (many) children. These results are also relevant for social inequality research: If we conceive of realized fertility in the sense of unequal chances to realize preferred life goals, then we have to take into account that genetic influences on inequality may not only be due to genetic sources of resources and skills but by genetic propensities for specific preferences as well. For education and fertility there are presumably different genes at work: “overlapping sources of genetic influences are relatively small” (Kohler and Rogers 2003:82). In other words, genetic variation obviously contributes to the variation in inequalities across different inequality dimensions. It can be assumed that this holds also true for different dimensions of status attainment and social mobility, namely class, status, prestige, and income.

Finally, our discussion of the empirical and normative implications of genetic variability in social stratification points to some fundamentally important issues. It is important to understand that genetic influences are far from deterministic. A high heritability estimate of an outcome does not imply that environmental factors are unimportant. There are numerous examples that illustrate this issue: Heritability in intelligence is contingent on parental socioeconomic position (Nisbett et al. 2012), heritability of fertility depends on social context (Kohler and Rodgers 2003; Kohler, Rodgers, and Christensen 1999), social control may effectively prevent genetic dispositions to aggression or drug use from unfolding (Shanahan and Hofer 2005), to cite but a few. This is vital to realize because it illustrates how environmental variability may enhance, remedy, or counter genetic influences, but it also –falsely– implies a static idea of environment. It is misleading to think of people as genetically advantaged or disadvantaged in general. The effect of genes is always contingent on the environment – an advantage under certain conditions may be a disadvantage under others.

Moreover, the discussion of the normative issues involved in the interpretation of genetically sensitive research may enrich the long-standing discussion on the legitimacy

of openness and social closure. The challenge then lies in understanding how environment and genes interact, which will bring about a refined and better understanding on how the individual is exposed to societal influences and how this affects mobility outcomes, which may ultimately contribute to the development of policies directed at increasing equality of opportunity.

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2. Does Sibling and Twin Similarity in Cognitive Ability Differ by Parents' Education?¹

Tina Baier

Abstract

Stratification scholars predominantly investigate how differences among children from different families emerge and tend to neglect differences among children from the same family. I study sibling similarity in cognitive ability and examine whether their similarity varies by parents' education. Although economic approaches and their extensions argue that disadvantaged parents reinforce differences while advantaged parents compensate for differences, I argue that parents may also make equal investments and thus accept differences among their children. I refer to the literature on stratified parenting, which demonstrates that parents are engaged differently in childrearing and their children's skill formation processes. Because advantaged parents foster children's talents more individually compared with disadvantaged parents, I propose that sibling similarity is lower in advantaged than in disadvantaged families. Previous studies based on sibling correlations provide conflicting evidence. To account for observable and unobservable differences among siblings, I extend the established sibling correlation approach and study dizygotic and monozygotic twins in addition to full siblings. The analyses draw on novel data from a population register-based study of twin families. I find that young adult siblings and twins are less alike in cognitive ability in highly educated families than in less educated families. Hence, my results support the hypothesis concerning equal investments and indicate that stratified parenting has a long-lasting influence on children's cognitive ability.

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2.1 Introduction

The link between family background and children's education is well established in the literature (e.g., Breen 2010; Breen and Jonsson 2005; Torche 2015). Most of what we know about the impact of family background influences derives from studies that examine children from different families. Yet, a smaller body of literature studies differences that emerge among children from the same family. These studies highlight that shared family background influences, such as parents' education, occupation or income, do not affect siblings equally. Indeed, for most stratification outcomes, including education, siblings correlate at about 0.5 (e.g., Benin and Johnson 1984; Conley 2008; Hauser and Mossel 1985; Hauser and Wong 1989; Sieben, Huinink, and de Graaf 2001). Thus, stratification mechanisms run not only between families but also within the family itself: despite being exposed to fairly similar family conditions, siblings end up with different levels of education. This challenges the common –though mostly not explicitly stated– assumption that shared family influences affect children in similar fashion (e.g., Conley 2008; Diewald et al. 2015).

An emerging scholarship investigates whether the similarity of siblings varies depending on parents' social background (e.g., Anger and Schnitzlein 2017; Conley 2008; Conley and Glauber 2008; Conley, Pfeiffer, and Velez 2007; Grätz 2018). Despite excellent research in this field, studies do not explicitly take into account the fact that differences among siblings are not only the result of parents' social background and associated resources but are also driven by differences in genetic make-up. Behavioral geneticists provide consistent evidence that genes are an important source of individual differences and that they can shape reactions to and from the social environment (e.g., Freese 2008; Polderman et al. 2015). To understand why differences among siblings emerge, it is therefore important to consider genetic heterogeneity as well. I build on previous studies on a possible stratification of sibling similarity and study sibling and twin similarity in cognitive ability, which is a major predictor of educational success and is strongly influenced by genes (e.g., Strenze 2007; Polderman et al. 2015).

Current explanations for within-family differences are mainly rooted in economic perspectives that model parents' investment decisions within the household (Becker and Tomes 1976; Behrman, Pollak, and Taubman 1982). Adding a stratification aspect, Conley (2004, 2008) proposes that advantaged parents are more likely to invest in a way that

compensates for differences among their children, whereas disadvantaged parents reinforce differences due to efficiency considerations. I argue, however, that parents might also invest equally in their children and thus accept differences among them. I draw on the literature on stratified parenting (e.g., Cheadle and Amato 2011; Kalil, Ryan and Corey 2012; Lareau 2011; Lareau and Weininger 2003), which originally emphasized the role of parenting in the emergence of differences between families and propose that differences in parenting also influence the extent to which siblings resemble one another. Lareau differentiates between two logics of parenting (2011). Disadvantaged parents are engaged in a parenting concept referred to as “natural growth” and intervene little in their children’s skill formation processes. Because resources are limited, parents more often invest primarily to meet the basic needs of their children. Advantaged parents, by contrast, have more resources and can afford investments in addition to those needed fundamentally. They engage in a parenting concept referred to as “concerted cultivation” and intend to further skills and behaviors typically found in higher class families. Importantly, parents embrace an active parenting strategy that shapes developmental processes of their children. Over and above “concerted cultivation” in accordance with higher class habits such active investments can also address children’s individual potentials and needs. Such investments are more child-specific. Because children develop depending on their unique interests, talents, and related specific inputs, I expect them to end up being less alike in their cognitive ability than siblings from disadvantaged backgrounds. Hence, I propose a competing hypothesis –namely, that siblings are less similar in terms of cognitive ability in advantaged families than in disadvantaged families.

Previous research on sibling similarity (i.e., sibling correlations) in cognitive skills is limited and provides conflicting evidence (Anger and Schnitzlein 2017; Conley, Pfeiffer, and Velez 2007; Grätz 2018). Yet, findings on sibling correlations have recently been criticized (e.g., Björklund and Jäntti 2012): First, (full) siblings differ in age and, because family contexts are not necessarily stable over time, might grow up in different family environments. Second, (full) siblings differ in their genetic make-up. Consequently, findings concerning the link between parents’ social background and the similarity of siblings might be influenced by developmental differences, genetic differences, and/or a combination of the two –and are not necessarily the direct consequence of varying parental resources.

To address this shortcoming, I study the similarity of (full) siblings, dizygotic (DZ), and monozygotic (MZ) twins. DZ twins are born at the same time and thus share much more of the family influences than (full) siblings do. However, DZ twins differ in their genetic make-up, which also affects the degree of similarity. MZ twins, by contrast, are genetically alike. The similarity between MZ twins therefore captures family influences most comprehensively. MZ twins allow one a) to accurately differentiate between shared family and child-specific influences and b) to rigorously test whether the similarity changes if parents' education increases.

Sibling and twin similarity is estimated with multilevel models. I draw on the newly collected dataset from the TwinLife study. TwinLife is a population register-based sample of more than 4000 twins and their families residing in Germany (Diewald et al. 2018). Unlike many observational twin studies TwinLife has applied a probability-based sampling strategy. These data make it possible to investigate, for the first time for Germany, sibling and twin similarity in cognitive ability and a possible stratification covering a broad range of the social spectrum (Lang and Kottwitz 2017).

I contribute to the literature by acknowledging that family influences comprise both social resources and genetic transmission. In addition, I control for the relationship of siblings and twins, which addresses a major limitation of studies analyzing within-family stratification. This enables me to model family influences more comprehensively and to analyze systematic differences in the similarity of siblings that are not influenced by differences in the rearing environment, genetic influences or even the sibling relationship. Finally, I extend current theoretical explanations based on economic approaches and emphasize the role of stratified parenting instead.

2.2 Theoretical Background

How can we explain differences in cognitive ability among children from the same family? And do differences vary according to parents' social background? To address these questions, I apply a within-family perspective and link parents' investments and parenting to sibling similarity. I then refer to the sibling correlation framework, which is widely applied to test

the proposed mechanisms indirectly. Incorporating findings from behavioral genetics, I argue that twins as opposed to siblings provide a more suitable unit of analysis to test whether a change in similarity is associated with parents' social background.

Sibling Similarity and Parents' Investments

To explain how differences among children from the same family emerge, scholars predominantly refer to economic perspectives that model parents' resource allocation decisions within the household. Becker and Tomes (1976) propose a general model according to which parents rationally invest various types of resources in children's human capital formation and, thus, in later-life outcomes. Following the investment paradigm, parents aim to maximize the total returns of the household. Accordingly, their investment decisions are driven by efficiency considerations, and resources are directed to the child from whom they anticipate the highest returns. Later in the life course, parents seek to create equality among children by monetary transfers. According to the "efficiency paradigm", parents purposely reinforce differences in human capital, which increases differences among their children.

Behrman and colleagues (1982) counter this perspective and add a different motivational aspect of parents' investments decisions. Because future returns on investment are uncertain, parents seek to compensate differences among children and tend to create equal outcomes in children's human capital. Thus, parental investments actively reduce differences among siblings, leading to higher sibling similarity with respect to education and, presumably, later income. This ultimately reduces the need to make monetary transfers in order to create equal living standards for their children. In this sense, parents invest in exactly the opposite way from that predicted by Becker and Tomes (1976) (see also Conley 2008).

Thus, in both perspectives, parents allocate their resources unequally among their children: If their decision is guided by efficiency considerations, parents increase differences among their children by favoring the most promising child (lower similarity). If, on the other hand, parents intend to create equal outcomes, they compensate for differences and favor the less talented child (higher similarity).

Parents' Investments and Social Background

It is also important to take into account the fact that the quality and quantity of parents' investments might differ depending on their social background. According to the family investment model (FIM), which extends the investment paradigm, advantaged parents have more resources that are conducive to cognitive and noncognitive skill development than do disadvantaged parents (e.g., Conger, Conger, and Martin 2010). Resources include not only various goods and services, such as better housing and healthy food, but also skill-enhancing activities and a stimulating home environment (e.g., Cunha and Heckman 2007; Cunha et al. 2006). The family stress model (FSM) focuses on the influence of intra-family dynamics and marital conflicts triggered by economic hardship; due to increased levels of psychological stress, disadvantaged parents become less involved in their children's affairs, are less capable of meeting their children's emotional needs, and often respond with harsh parenting (e.g., Conger and Elder 1994; Conger, Conger, and Martin 2010). The related nonmaterial consequences of financial strain are the relevant pathways through which parents' social position influence children's skills and well-being. Both the family investment model and the family stress model have made major contributions to our understanding of how parents' social background leads to systematic differences between children from advantaged and disadvantaged families. Nonetheless, whether and how parents' social background leads to differences or similarity among children from the same family remains unclear.

Conley (2004, 2008) adopts a within-family perspective and links parents' social background to their resource allocation decisions. He argues that parents' investment rationale is contingent on their social position: Depending on the resources available, parents invest either in a compensatory fashion or in line with the efficiency paradigm. Accordingly, parents with fewer resources minimize the risk of failure by directing resources to the most promising children, whereas advantaged parents can afford both –investments in the most promising child and compensatory investments in the less gifted one. In this perspective, equality among siblings is a goal that can be attained once enough resources are available (higher sibling similarity); otherwise, parents will have to pick one of their children and direct their resources selectively (lower sibling similarity) (Conley 2004).

However, parents might also make equal investments and accept that their children develop differently. To elaborate how equal investments might accentuate differences between children from the same family, I draw on the literature on stratified parenting.

Broadly speaking, parenting refers to parent-child interactions that affect children's development. Hereby, we can distinguish between parenting goals, parenting styles, and parenting practices (Darling and Steinberg 1993). Parenting goals, or socialization goals, refer to the outcomes that parents seek for their children. Parenting styles denote the emotional climate in which parent-child interactions are embedded, and parenting practices refer to parental actions and activities that parents provide for their children in order to achieve their goals. The study of parenting styles has a long research tradition among developmental psychologists pioneered by Baumrind (1971), whilst recent sociological studies focus on parents activities, that is parenting practices, as expression of distinct cultural taste (e.g., Cheadle and Amato 2011; Kalil, Ryan, and Corey 2012; Lareau 2011; Lareau and Weininger 2003).

In her qualitative study, Lareau (2011) identifies two different logics of parenting that describe systematic differences in childrearing and involvement in children's skill formation processes. These logics are rooted in the parents' distinct cultural practices and habits and influence children's skills, educational attainment, and hence their subsequent life chances.

Advantaged parents adopt a parenting concept referred to as "concerted cultivation" (Lareau 2011) Parents seek to promote –that is, cultivate– their children's unique talents and to give them the ability to speak up for themselves in order to increase their chances of later-life success. To achieve this, parents invest various types of resources that support the skill development of their children. Importantly, parents actively shape the development of their children and plan interactions and activities with their children. Parents are very controlling paired with responsiveness. Parents provide clear guidance and are strongly involved in structuring their children's daily lives. Consequently, children grow up in a home environment in which the parents structure their leisure time and actively further their children's interests. To be effective (i.e., to foster the children's talents), such parents provide child-specific inputs: they customize their children's daily activities in line with the children's interests; they monitor the children's educational processes individually; and they provide support if needed. Hence, stimulating activities and resources are provided for every child, but what kind of investment each child receives will depend on their specific needs.

Disadvantaged parents adopt a parenting concept referred to as "natural growth" (Lareau 2011). Here, the children's development is perceived more as something that naturally evolves over time. Parents intervene less in the developmental processes of their children and

provide those inputs that are fundamentally needed for development. Parents are less involved and more authoritarian, set strict rules which are not questioned. Due to limitations of time and money, parents often lack the capacity to discover their children's individual talents and/or to provide stimulating activities or resources to further those interests. Consequently, such parents adopt a less active role in their children's development and skill formation. Disadvantaged parents also provide their resources for all of their children; investments are, however, rather uniform and thus less child-centered.

Quantitative studies provide support for different parenting concepts in line with Lareau's notion on different logics of parenting (e.g., Cheadle 2008; Cheadle and Amato 2011) and their association with children's academic performance (Bodovski and Farkas 2008; Roksa and Potter 2011) and facets of personality (Kaiser 2017; Kaiser and Diewald 2014). It is important to note that Lareau does not adopt a within-family perspective; rather, she shows how culturalized habits lead to systematic differences between advantaged and disadvantaged children. Furthermore, the notion of stratified logics of parenting is not mutually exclusive from either the family investment perspective or the family stress perspective. In fact, Lareau's notion of different logics of parenting is supported by the finding that more advantaged parents provide more skill-enhancing inputs compared with disadvantaged parents (e.g., Conger, Conger, and Martin 2010). Related to that, psychological stress as proposed by the family stress model (e.g., Conger and Elder 1994; Conger, Conger, and Martin 2010) might provide a mechanism that explains why disadvantaged children receive less attention from their parents. Nonetheless, I argue that an important mechanism behind the emergence of within-family differences is rooted in active and strategic parenting behaviors found in advantaged families: Parents that seek to cultivate distinct skills and behaviors are also more actively involved in shaping the development of their children. Such investments can address children's potentials and needs more individually which promote differences in cognitive ability among siblings to a greater extent (lower sibling similarity) than investments from disadvantaged parents. Disadvantaged parents often lack the capacity and/or resources to make those skill-enhancing investments and provide fairly uniform inputs, which leads to higher similarity. That siblings in advantaged families end up being more different than siblings in disadvantaged families is not intentional –it is a side effect of parents' distinct parenting behavior.

Taken together, the literature provides competing hypotheses for a stratification of sibling similarity. Conley (2004, 2008) argues that parents allocate their resources selectively: If resources are limited, parents will allocate their resources efficiently; if resources are not restricted, parents tend to compensate. If Conley's argument holds, I expect *siblings to be less similar in disadvantaged families compared with siblings from advantaged families (H1)*. If, however, parents make equal investments and adopt different parenting concepts I expect the opposite pattern –that is, I expect *siblings to be more similar in disadvantaged families compared with siblings from advantaged families (H2)*.

Previous Findings

The link between social background and sibling similarity has been studied for socioeconomic outcomes such as education, income, and earnings (Conley 2008; Conley and Glauber 2008), as well as for cognitive and noncognitive skills (Anger and Schnitzlein 2017; Conley, Pfeiffer, and Velez 2007; Grätz 2018). Most studies refer to the United States, although more recent studies have been conducted for Germany. Given that educational decisions are different from investments that further the development of cognitive ability (Boudon 1974; Breen and Goldthorpe 1997; Erikson and Jonsson 1996), I focus in the following on studies that analyze sibling similarity in cognitive and noncognitive skills. Conley, Pfeiffer and Velez (2007) analyzed sibling similarity in cognitive skills and behavioral outcomes during early childhood (between ages 6 and 12) based on the Panel Study of Income Dynamics (PSID) for the United States. These authors used literacy, numeracy, reading comprehension, and problem-solving skills as indicators of cognitive skills, and the Behavior Problem Index (BPI) for behavioral outcomes; social background was approximated using mothers' education. The results offer some support for a systematic variation according to social background: Sibling similarity in literacy and the BPI was significantly higher for siblings whose mothers were less educated. Anger and Schnitzlein (2017) examined sibling similarity in cognitive ability, noncognitive skills (i.e., the Big Five), and locus of control for adult siblings (aged between 20 and 54) in Germany using the Socio-Economic Panel study (SOEP). Because they had only small sample sizes, they examined the link with social background only for noncognitive skills. The results show that sibling similarity for most indicators of noncognitive skills was higher for siblings whose mothers are more educated. Grätz (2018) examined sibling similarity in cognitive ability for

young adult siblings (aged between 17 and 28) based on the SOEP as well. He used more recent waves and examined systematic differences in the similarity of cognitive skills according to social background, as indicated by parents' education, occupation, and social class (based on the Erikson-Goldthorpe-Portocarero social class scheme [EGP]). Regardless of the indicator of social background, sibling similarity did not change systematically according to social background.

In sum, the empirical literature provides conflicting evidence for the country and the outcome under study. In the United States, sibling similarity in the BPI and in literacy skills tend to be higher in disadvantaged families. For Germany, however, sibling similarity in noncognitive skills is higher in advantaged families. Sibling similarity in cognitive skills, by contrast, did not systematically differ.

There are two factors that might explain why these findings diverge between the United States and Germany. First, institutional differences might play a role. Germany and the United States vary greatly in the institutional set-up of the welfare state. Social inequality is much more polarized in the United States context, and the welfare state there is less invasive and provides only a weak insurance structure. The German welfare state, by contrast, provides more generous social benefits and a safety net. At least regarding cognitive skills, evidence for the United States shows that poverty is strongly linked to children's cognitive outcomes, which is less so in Germany (Biedinger 2011). Thus, the fact that sibling similarity in literacy skills in the United States is associated with social background might be explained by differences in marginalization that are experienced in these two countries (see also Schulz et al. 2017). Yet, evidence for a systematic variation in cognitive outcomes is weak, because it was found for only one indicator of cognitive skills during early childhood. In addition, it is striking that the pattern identified for noncognitive skills tends in the opposite direction.

The second important factor that might explain the divergent findings is the age range of the siblings, which is closely linked to the development of cognitive and noncognitive skills (e.g., Cunha and Heckman 2007; Haworth et al. 2010). The two studies for Germany analyzed young adult siblings, whereas the study for the United States analyzed siblings during childhood. Given that children are more sensitive to environmental influences (i.e., family inputs) during childhood (e.g., Cunha and Heckman 2007), divergent findings might indicate that the influence of parents' social background varies over the children's life

courses. However, this remains an empirical question and will require more research that also takes systematically children's developmental stage into consideration.

A major limitation of previous studies besides possible life course variation is that they have not sufficiently accounted for genetic influences. Genetically sensitive studies provide consistent evidence that cognitive and noncognitive skills, as well as more distal outcomes such as achievement scores, grades, and educational attainment, are significantly influenced by genetics (e.g., Ayorech et al. 2017; Branigan, McCallum, and Freese 2013; Johnson, McGue, and Iacono 2005; de Zeeuw, de Geus, and Boomsma 2015). IQ research in particular has a long tradition in behavioral genetics, and previous studies show that heritability of adults' cognitive skills (i.e., IQ) is between 0.6 and 0.8 (Tucker-Drob, Briley, and Harden 2013). Thus, genetic influences account for about 60 to 80% of total variation in IQ. This does not mean that environmental (i.e., social) influences are unimportant, because genetic tendencies are realized under social conditions provided by the proximate environment (Bronfenbrenner and Ceci 1994).

Moreover, environments that humans encounter are not random but are a function of an individual's genotype, referred to as "gene-environment correlations" (Plomin, DeFries, and Loehlin 1977; Rutter 2007). Passive gene-environment correlations describe situations in which individuals are selected into environments that match their talents. For example, parents who favor classical music not only transmit such preferences, they also expose their children more often to this type of music. Thus, children inherit genetic dispositions but are also exposed to environmental influences in line with these dispositions. Evocative gene-environment correlations describe individuals' reactions to the genetic endowments of others; for example, gifted children might receive special treatment from teachers. Recent evidence shows that children's genetic make-up also influences how parents treat their children; for example, extrovert children might be treated differently from introvert children. Children's genetic make-up can therefore influence how parents react to them (Avinun and Knafo 2014; Klahr and Burt 2014). Finally, individuals actively search for environments that match their innate talent (niche picking), which is referred to as active gene-environment correlation.

If we do not take genetic heterogeneity into account, findings concerning the link between similarity and parents' social background (i.e., social transmission mechanisms) remain misleading. Genes affect cognitive ability directly, but they also operate indirectly in that

genes influence how parents react to their children and/or how children react to their parents' investments. Thus, any similarity or dissimilarity of siblings might be driven by differences in genetic make-up.

Sibling, DZ Twin, and MZ Twin Similarity

As noted, sibling similarity (i.e., sibling correlation) serves as an indirect test for parents' investment strategies. Sibling similarity can be understood as a summative measure for all measured and unmeasured influences of family background ("total family effect") (e.g., Sieben, Huinink, and de Graaf 2001). The idea is straightforward: Because siblings are born and raised in the same family, everything that makes them alike can be attributed to shared family influences. The more alike siblings are, the stronger the impact of shared family influences. Conversely, differences among siblings emerge as a result of influences that are not shared by siblings and thus are specific to the child.

On an interpretive level, it is important to note that a low sibling correlation does not necessarily imply that family background is not important, because differences among siblings may be rooted in parents' actions (e.g., Björklund and Jäntti 2012; Conley 2008). In line with the theoretical assumptions outlined above, parents' efforts may lead to either sibling similarity or sibling dissimilarity. If parents compensate for differences, sibling similarity increases and shared family influences increase (H1). If, however, for efficiency reasons, only child benefits from the parents' resources, differences increase but shared family influences decrease. And even if parents allocate their resources equally (H2), shared family influences decrease to the extent that initial differences are reinforced. Thus, differences among siblings –lower sibling correlations– may be triggered by parents' efforts. Given that nonshared or child-specific influences may be the result of parents' selective resource allocation, the interpretation of the similarity of siblings as the "total family impact" can be misleading (Conley 2008). Nevertheless, sibling correlations, as a descriptive measure, reveal whether stratification mechanisms on the societal level are associated with intra-familial dynamics that lead to differences among siblings (Conley 2008).

On a conceptual level, it is important to note that sibling similarity summarizes not only the influence of parents' characteristics and associated resources, but also the impact of influences associated with the broader family context (i.e., neighborhood influences), genetic endowments, and effects that siblings have on one another (e.g., Conley and Glauber 2008).

In the following, I explain why twins provide a better unit of analysis to capture shared family influences and how twins enable us to test more rigorously whether a change in the similarity is associated with varying resources of the parents.

(Full) siblings are born and raised at different points in time and share about half of their DNA. Twins, by contrast, are born and raised at the same time, while MZ twins are, at conception, genetically alike (see Table 2.1).

Table 2.1 Similarity and Dissimilarity of Siblings and Twins

| | (Full) Siblings | DZ Twins | MZ Twins |
|---|--------------------------------|--------------------------------|----------------------|
| Exposure to <i>same</i> family conditions | No | Yes | Yes |
| Genetic overlap | ~ 50% | ~ 50% | ~ 100% |
| Sources of dissimilarity | Nonshared influences and genes | Nonshared influences and genes | Nonshared influences |

It is common in stratification research to study the similarity of (full) siblings. Siblings may grow up under different family conditions (i.e., families might relocate, parents might switch jobs and/or repartner) and differ in their genes. Thus, their similarity might result from either of these influences or from a combination of the two. Consequently, a change in the similarity might not be a direct consequence of varying parental resources and the associated investments. DZ twins are raised simultaneously, and hence they grow up under almost the same family conditions. For example, when twins grow up, their parents have the same occupational and educational status, and the twins live in the same neighborhood and probably attend the same school (or at least a school that is nearby). It is less likely that differences in the strength of the similarity between DZ twins can be attributed to the broader family context (because most of the contextual influences are shared). DZ twins are raised under most similar family conditions, while the rearing environment of (full) siblings can be very different. Thus, a change in the strength of similarity of DZ twins is more likely to be associated with systematic differences in parents' resources. Nonetheless, differences between DZ twins might still be due to their genetic differences. MZ twins are genetically identical and thus provide the most comprehensive measure for shared family influences

because of their common upbringing and shared genes. Any difference among MZ twins must be the result of nonshared influences –net of genetic factors. Studying MZ twins, therefore, makes it possible to test more rigorously whether the similarity is associated with parents' social position and related investments.

Given the above, I argue that studying MZ twins provides the most rigorous test for the two hypotheses proposed earlier. The findings concerning the link between sibling similarity and parents' social background based on (full) siblings serve as baseline findings, because this is the general approach in stratification research. Results for DZ twins and MZ twins show to what extent the similarity changes when children are raised under the most similar family conditions (DZ twins) and if genetic heterogeneity is also controlled for (MZ twins).

Nonetheless, similarity between twins may also be the result of sibling effects –that is, of the influence siblings have on one another. This is a general concern when studying siblings and possibly even more so when studying twins. Previous studies have found that siblings have an effect on cognitive development (Azmitia and Hesser 1993; Brody 2004; Dunn 1983). Siblings may serve as teachers, which is beneficial to both the one being taught and the one teaching, because the teaching sibling has to reconsider a given subject, reduce the level of complexity, and find appropriate and/or easier explanations. Siblings are even more effective teachers than are peers, which may possibly be explained by their greater familiarity and knowledge of their siblings' unique talents and weaknesses (Azmitia and Hesser 1993). Studies in this field analyze (full) siblings. And it might be argued that interactions are not directly transferable to twins, who might have more similar knowledge than siblings who differ in age. However, as Dunn (1983) noted, sibling relationships are characterized by both reciprocity and complementarity, with the latter being positively associated with sociocognitive development. Reciprocal interactions, however, are very likely among twins, who share even more time with each other and know each other probably even better than (full) siblings know one another. I therefore argue that such learning processes are also prevalent in twin dyads.

Importantly, mutual influences among siblings might differ depending on how parents allocate their resources. When resources are scarce, sibling rivalry might be increased, which in turn lowers mutual influences among siblings as they struggle for scarce resources. As competitors, it is unlikely that they will teach each other. However, siblings might also interact less with each other if there are plenty of resources, because they seek to set

themselves apart to maintain their niche in the family system (Feinberg and Hetherington 2000). Given that there is no empirical research on a possible stratification on siblings' relationships and their influence on cognitive outcomes, both scenarios are equally plausible. To rule out the possibility that sibling effects are not the main driver of sibling and twin similarity, I provide a sensitivity analysis that controls for the closeness of the twin and siblings relationships.

2.3 Data and Methods

The analyses draw on newly collected data from the TwinLife panel study, a population register-based study of twins and their families residing in Germany (Diewald et al. 2018). These data make it possible –for the first time for Germany– to examine sibling and twin similarity in cognitive ability. Data collection started in 2014. TwinLife applies an extended twin family design in which the twins, their biological and social parents, and one sibling (if available) are surveyed. The information on zygosity (i.e., whether a twin is mono- or dizygotic) was obtained by means of physical similarity reports (self-reports or parents' reports) (see Lenau and Hahn 2017).² Due to the probability-based sampling strategy, TwinLife provides a unique opportunity to examine correlations in cognitive ability on a broad range of the social spectrum (Lang and Kottwitz 2017).

The analyses are based on young adult siblings and twins from the two oldest birth cohorts, aged between 17 and 25 years (birth cohorts 1997/98 and 1990–93, respectively). I excluded younger birth cohorts from the analysis (twins aged between 5 and 12), because age is a strong moderator of cognitive development (e.g., Cunha and Heckman 2007; Haworth et al. 2010), and the question how parents' social background affects cognitive ability at different stages of children's life course represents a study in its own right. Studying

² The algorithm to determine the zygosity of twins was additionally cross-validated through genotyping procedures with a subsample of about 300 twin pairs (Lenau and Hahn 2017).

young adults is particularly interesting, because I can examine whether social background and associated allocation decisions have a lasting influence on cognitive ability.

To investigate sibling and twin similarity, I generated three samples: a sibling sample, a DZ twin sample, and an MZ twin sample. TwinLife samples twin families only (with or without additional non-twin siblings). Thus, siblings in the sibling sample are (full) siblings of twins who are randomly paired with one twin from the mono- or dizygotic twin pair (i.e., non-twin sibling-twin dyads). I restrict the minimum age of the siblings to the minimum age of twins (17 years) and the age difference to up to 8 years (i.e., two standard deviations from the age difference) in order to avoid the level of cognitive ability being affected by developmental differences within the sibling-twin dyad. Given the design of the TwinLife study, the sibling sample includes families with at least three children (i.e., the twin pair and one non-twin sibling), which is not necessarily the case for the twin samples considering that twins do not necessarily have a non-twin sibling. To ensure the results will not be influenced by fewer resources among families who have more than two children, I restrict the analyses to families with at least 3 and no more than 8 children (the maximum number of children in all three samples). The sibling sample comprises 726 siblings, the DZ sample 1148 twins, and the MZ sample 1232 twins.

Variables

The outcome of interest is that for cognitive ability. Cognitive ability is measured using the Culture Fair Intelligence Test (CFT 20-R), which is a standard psychometric test to measure nonverbal (fluid) intelligence (Weiß 2006). Individuals' cognitive ability scores are estimated using structural equation modeling. As recommend by the TwinLife group, I used age-corrected CFT scores (Gottschling 2017). I deleted observations with missing values for the cognitive testing (14% of the sample). Information on cognitive ability was missing more often among low-educated families ($p < 0.05$). Because lower-educated families are to some extent underrepresented, the findings concerning sibling and twin similarity tend to represent lower-bound estimates.

I use parents' education as an indicator of social background –that is, the highest level of education achieved by the parents (dominance principle).³ I chose parents' education because it covers not only transmission mechanisms that run through economic resources but also resources that can be summarized as cultural capital. For instance more educated parents provide a stimulating home environment, additional learning material, and cultural goods, all of which foster children's cognitive ability (e.g., Cunha and Heckman 2007; Duncan et al. 1998; Lareau and Weininger 2003). Based on the CASMIN classification scheme (see Appendix Table A2.1), I distinguish low-educated (CASMIN 1a-c, 2b), medium-educated (CASMIN 2a, c), and highly educated parents (CASMIN 3a, b). CASMIN 2b refers to individuals with intermediate levels of general education but without vocational training. They are included in the lowest educational category for two reasons. First, the German labor market is highly credentialized and it is very uncommon to enter the labor market without any vocational training (e.g., Allmendinger 1989; Solga 2005). Second, due to educational expansion, the proportion of individuals with low levels of secondary education is decreasing, while the proportion of individuals with intermediate levels of secondary education is increasing (Solga 2005).⁴

CASMIN information was missing for 7.8% of the mothers and 32% of the fathers. I used multiple imputation with chained equations with 20 data sets for each observation to impute the missing information on education (van Buuren et al. 2006). The main variables for the imputation model are at the family level (i.e., they come from the twins' parents).

In sensitivity analyses, I investigate the role of mutual influences among siblings and twins. To indicate the closeness of a relationship, three questions were asked: (1) *How often do you talk about important things with (name of the other sibling)?* (2) *How often do you attempt to cheer up (name of the other sibling)?* (3) *How close do you feel to (name of the other sibling)?*⁵ The response categories were never, rarely, occasionally, often, and very often. I used confirmatory factor analysis based on structural equation modeling to create an

³ The results do not change substantially if mother's education is used to indicate educational background instead (see Appendix Figure A2.1)

⁴ The results remain stable if individuals with CASMIN 2b are in the group with medium education.

⁵ Twins were asked the same questions.

index of closeness (the coefficient of determination is 0.8). Table 2.2 shows the descriptive statistics for the sibling, DZ, and MZ samples.

The distribution of the main variable is fairly similar across all three samples. However, with regard to the closeness of the sibling and twin relationship, there are substantial differences: MZ twins are closest, followed by siblings and then by DZ twins. Differences between DZ twins and MZ twins have previously been found in the literature and might be explained by their closer resemblance in terms of both the rearing environment and their genetic make-up (Fortuna, Goldner, and Knafo 2010). The fact that siblings are closer to one another than DZ twins are to each other is contrary to previous findings (which, however, were reported in studies based on small samples) and therefore requires more empirical investigation (Fortuna, Goldner, and Knafo 2010).

Table 2.2 Descriptive Statistics

| | Siblings | | | | DZ | | | | MZ | | | |
|---|------------------|-------|------|-----|------------------|-------|------|------|------------------|-------|------|------|
| | Mean/ SD | Min | Max | N | Mean/ SD | Min | Max | N | Mean/ SD | Min | Max | N |
| <i>Individual (twin) Level Variables:</i> | | | | | | | | | | | | |
| Cognitive ability | 98.96 (16.46) | 56 | 143 | 726 | 98.21 (16.49) | 55 | 143 | 1148 | 99.30 (15.83) | 55 | 146 | 1232 |
| Age | 20.73 (3.20) | 17 | 31 | 726 | 19.82 (3.00) | 17 | 25 | 1148 | 20.13 (3.02) | 17 | 25 | 1232 |
| Male | 0.45 (0.50) | 0 | 1 | 726 | 0.41 (0.49) | 0 | 1 | 1148 | 0.42 (0.49) | 0 | 1 | 1232 |
| Closeness ^a | 0.00 (1.00) | -2.70 | 1.69 | 726 | -0.23 (1.02) | -3.71 | 1.03 | 1148 | 0.22 (0.93) | -3.71 | 1.03 | 1232 |
| Family size | 3.56 (0.90) | 3 | 8 | 726 | 3.54 (0.91) | 3 | 8 | 1148 | 3.59 (0.90) | 3 | 8 | 1232 |
| <i>Family-Level Variables:</i> | | | | | | | | | | | | |
| Parents' CASMIN (imputed) | | | | | | | | | | | | |
| Low | 0.18 (0.39) | 0 | 1 | 726 | 0.18 (0.38) | 0 | 1 | 1148 | 0.19 (0.39) | 0 | 1 | 1232 |
| Medium | 0.47 (0.50) | 0 | 1 | 726 | 0.47 (0.50) | 0 | 1 | 1148 | 0.46 (0.50) | 0 | 1 | 1232 |
| High | 0.35 (0.48) | 0 | 1 | 726 | 0.35 (0.48) | 0 | 1 | 1148 | 0.35 (0.48) | 0 | 1 | 1232 |
| Parents' CASMIN (unimputed) | | | | | | | | | | | | |
| Low | 0.20 (0.40) | 0 | 1 | 712 | 0.18 (0.39) | 0 | 1 | 1120 | 0.22 (0.41) | 0 | 1 | 1198 |
| Medium | 0.46 (0.50) | 0 | 1 | 712 | 0.47 (0.50) | 0 | 1 | 1120 | 0.44 (0.50) | 0 | 1 | 1198 |
| High | 0.34 (0.48) | 0 | 1 | 712 | 0.35 (0.48) | 0 | 1 | 1120 | 0.34 (0.47) | 0 | 1 | 1198 |

Source: TwinLife wave 1; own calculations. Standard errors in parentheses. ^a) Closeness is mean-centered.

Analytical Strategy

To examine the similarity among siblings and twins, I use multilevel modeling in which siblings (level 1) are nested in families (level 2) (e.g., Raudenbush and Bryk 2002). Multilevel models (also known as variance decomposition models) are well suited for the question under study because they make it possible to separate out the different sources of variation in children's cognitive ability that is, shared family and nonshared, child-specific, influences. Given that the variance components are of particular interest, I separately specify

empty models for each sibling sample. Based on this regression set-up, the intra-class correlation coefficient ICC can be calculated as follows:

$$ICC = \frac{\sigma_b^2}{\sigma_w^2 + \sigma_b^2}$$

The ICC is defined as the ratio of the variance due to between-family differences (shared family influences) (σ_b^2) relative to the total variance (i.e., variation that can be attributed to the family [σ_b^2] plus variation that can be attributed to the child [σ_w^2]). A low ICC indicates high within-family stratification: despite shared family influences, siblings' outcomes differ from each other. Vice versa, a high ICC indicates a greater importance of shared family influences.

I first estimate variance decompositions for each sample (siblings, DZ twins, MZ twins) separately. I then estimate these models for each sample, differentiated by parents' education. The test for a systematic variation according to parents' education is based on the z-value of the differences in the ICCs (Conley and Glauber 2008; Conley, Pfeiffer, and Velez 2007; Kenny, Kashy, and Cook 2006). It is common in the sibling correlation literature to consider only the ICC, which is a standardized measure of the importance of the between-family (random effect) variance, at the expense of the variance components in absolute terms. However, the ratio stays the same if both variance components change simultaneously. Thus, the relative importance of shared family influences may change even if the ICC does not. To better understand the ongoing processes, I also provide information about the variance components in absolute terms (see also Erola 2012).

I estimate two-level random intercepts models with the mixed command in Stata 14.2 using the restricted maximum-likelihood option.

2.4 Results

Table 2.3 shows the results for sibling and twin similarity in the unrestricted sample (column 1) and their variation according to parents' education (columns 2 to 4). I report three estimates of empty multilevel models: 1) the variance components in absolute terms as an indication of the underlying structure of the variation (Var.: Family and Var.: Child); 2) the intra-class correlation (ICC), which specifies the relative importance of shared family influences; and 3) the mean level of cognitive ability (constant), which provides information about the direction of shared family influences. Figure .1 visualizes the findings for the ICCs and variance components.

Table 2.3 Sibling and Twin Similarity in Cognitive Ability by Parents' Education

| | Parents' Education | | | | | | | |
|---------------------------|--------------------|---------|-----------------|---------|-----------------|---------|------------------|---------|
| | Overall | | Low | | Medium | | High | |
| | b/ var | z-value | b/ var | z-value | b/ var | z-value | b/ var | z-value |
| Siblings | | | | | | | | |
| Constant | 98.95 (0.71) | 139.09 | 90.77 (1.96) | 46.36 | 98.63 (1.09) | 90.22 | 102.91 (1.07) | 95.88 |
| Var.: Family | 96.47 | | 147.38 | | 73.09 | | 58.62 | |
| Var.: Child | 174.55 | | 125.03 | | 186.97 | | 181.65 | |
| ICC | 0.36 (0.05) | 7.76 | 0.54 (0.10) | 5.60 | 0.28 (0.08) | 3.72 | 0.24 (0.08) | 2.87 |
| N | 726 | | 122 | | 324 | | 280 | |
| <i>Differences in ICC</i> | | z-value | | | | | | |
| Medium vs. high | | 2.03 | | | | | | |
| High vs. low | | 0.35 | | | | | | |
| Low vs. medium | | 2.34 | | | | | | |
| DZ Twins | | | | | | | | |
| Constant | 98.21 (0.58) | 170.7 | 90.39 (1.63) | 55.37 | 96.22 (0.90) | 106.96 | 103.36 (0.90) | 115.06 |
| Var.: Family | 108.13 | | 112.15 | | 91.54 | | 71.79 | |
| Var.: Child | 163.72 | | 181.09 | | 159.57 | | 161.73 | |
| ICC | 0.40 (0.04) | 11.32 | 0.38 (0.09) | 4.02 | 0.36 (0.06) | 6.11 | 0.31 (0.07) | 4.69 |
| N | 1148 | | 176 | | 510 | | 462 | |
| <i>Differences in ICC</i> | | z-value | | | | | | |
| Medium vs. high | | 0.19 | | | | | | |
| High vs. low | | 0.54 | | | | | | |
| Low vs. medium | | 0.61 | | | | | | |
| MZ Twins | | | | | | | | |
| Constant | 99.30 (0.58) | 169.81 | 92.77 (1.51) | 61.50 | 99.63 (0.94) | 106.11 | 101.83 (0.92) | 110.42 |
| Var.: Family | 170.72 | | 175.53 | | 169.17 | | 146.35 | |
| Var.: Child | 79.87 | | 66.67 | | 80.83 | | 84.65 | |
| ICC | 0.68 (0.02) | 31.54 | 0.72 (0.05) | 14.44 | 0.68 (0.04) | 19.23 | 0.63 (0.04) | 15.41 |
| N | 1232 | | 212 | | 536 | | 484 | |
| <i>Differences in ICC</i> | | z-value | | | | | | |
| Medium vs. high | | 0.63 | | | | | | |
| High vs. low | | 0.88 | | | | | | |
| Low vs. medium | | 1.41 | | | | | | |

Source: TwinLife wave 1; own calculations. Standard errors in parentheses

I start with the results for the unrestricted sample (Table 2.3, column 1). These are baseline results for the degree of within-family stratification by sibling type. The similarity of (full) siblings is 0.36 (see ICC). Thus, more than a third of the total variation in cognitive ability can be attributed to shared family influences; child-specific influences account for about two-thirds of the total variation. On average, siblings share about 50% of their DNA. Thus, genetic

influences are included in the shared family component (if they lead to sibling similarity) and also in child-specific influences (if they lead to differences) (see Table 2.1). Since (full) siblings differ in age and genetic make-up, their similarity is comparatively low. However, the similarity of DZ twins is only slightly higher (40%). As noted earlier, DZ twins are born and raised at the same time. The rearing environment for DZ twins is therefore much more similar compared with the rearing environment for siblings. Nonetheless, the correlation – and hence the degree of within-family stratification– in cognitive ability for siblings and DZ twins is about the same (0.40 for DZ twins and 0.36 for siblings). Thus, even under the most similar family conditions DZ twins are differently affected by them. The similarity of MZ twins is considerably higher (0.68), which can be explained by their common upbringing and genetic similarity. The fact that the similarity is considerably higher for MZ twins reflects the importance of genetic influences for cognitive ability (e.g., Nisbett et al. 2012; Tucker-Drob, Briley, and Harden 2013). Any difference between MZ twins results from nongenetic nonshared influences (see Table 2.1). About 30% of the total variation in cognitive ability is associated with child-specific influences –net of genes.

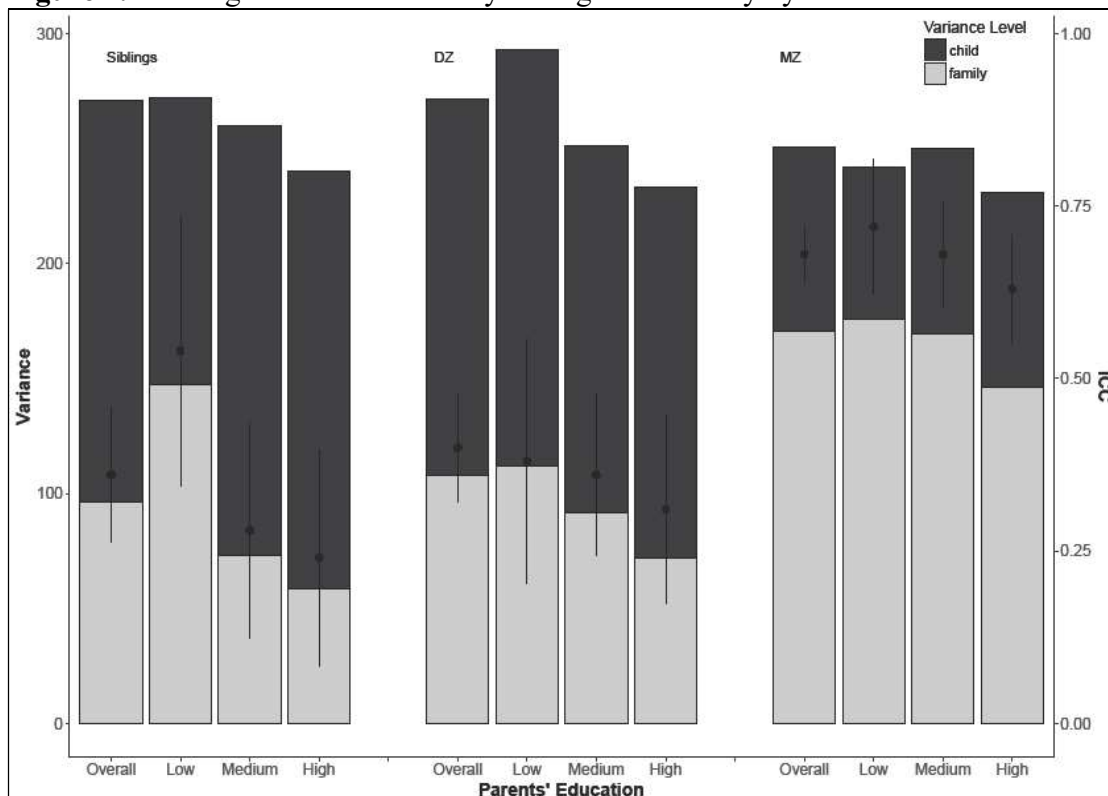
Next I evaluate how the overall similarity changes according to parents' education Table 2.3, columns 2-4). The results show that the degree of similarity decreases in all three samples from less to highly educated parents. The decrease in the similarity is most pronounced in the sibling sample. In less educated families, sibling similarity is about 0.54. Thus, half of the total variation in cognitive ability can be attributed to shared family influences. In highly educated families, by contrast, siblings correlate at about 0.24. Thus, child-specific influences are more important in highly educated families. As indicated by the z-values, differences in the similarity between medium- and highly educated families are statistically significant (z -value=2.03), as are differences between less and medium-educated families (z -value=2.34). Also in absolute terms, shared family background influences decrease sharply, whereas child-specific influences increase in families from low to medium-educated families. Given that the cognitive ability scores are more different in more highly educated families than in less educated families, the results provide preliminary support for hypothesis H2.

The similarity of DZ twins also decreases from less to highly educated families. In less educated families, the similarity of DZ twins is 0.38; in highly educated families it is 0.31. Although the decrease in the degree of similarity is not statistically significant, the results

tend in the same direction. Given that DZ twins and siblings differ only in the extent to which they are simultaneously exposed to the same family conditions, the significant decrease among siblings must be rooted in different family environments in which (full) siblings grow up.

Results for MZ twins reveal the same pattern. The similarity decreases from 0.72 in less educated families to 0.63 in highly educated families. The results for the variance components in absolute terms show the same trend: shared family influences decrease steadily from less to highly educated families, whereas child-specific influences –net of genes– become more important in the MZ sample. This provides further indication that parents use their resources selectively once additional resources are available. Thus, even for MZ twins, who are overall more similar than siblings and DZ twins because of their shared genetic make-up and common upbringing, differences are more pronounced the more educated parents are.

Figure 2.1 Sibling and Twin Similarity in Cognitive Ability by Parents' Education



Source: TwinLife wave 1; own calculations.

Finally, I report the findings on the mean of cognitive ability (Table 2.3, Constant). For siblings, DZ twins, and MZ twins the mean level of cognitive ability increases with parents' education. The more resources parents have, the higher the mean value of cognitive ability. Since parents transmit 50% of their genes to their children the increase in the mean value of cognitive ability is also driven by direct genetic effects. To parcel out genetic transmission, I would need the information on the correlation of children's and parents' genotypes, which I consider to be a study in its own right. However, parents' genes that are not transmitted also affect children's outcomes, since parents select environments based on their genetic make-up (indirect genetic effects) (Belsky et al. 2018). Previous research shows that environmental conditions created by more educated parents enhance genetic expression for cognitive skills such as IQ (i.e., they provide a rearing environment in which children can actualize their genetic potential (e.g., Guo and Stearns 2002; Turkheimer et al. 2003)). Thus, parents pass down genetic influences that affect children's cognitive ability; however, whether children realize their genes and innate talent depends on the rearing environment their parents provide.

Interpreting the results for the mean values of cognitive ability along with the findings concerning the variance components, I find lower means for disadvantaged siblings and twins but a greater importance of shared family influences. This supports my expectation concerning stratified parenting: Disadvantaged parents often lack the resources to make stimulating investments, which explains why disadvantaged children have, on average, lower levels of cognitive ability scores than do advantaged children. Given that investments of disadvantaged parents are more uniform and are intended to meet basic needs, siblings are also more alike in terms of cognitive ability (shared family influences are more important). Advantaged parents, by contrast, provide more child-specific inputs and address their children's need individually, which accentuates differences in cognitive ability among siblings more strongly (shared family influences are less important). In all three samples, the relative importance of shared family influences is most pronounced in less educated families, which leads me to conclude that the same family influences that account for the similarity of siblings and twins in less educated families are rather detrimental to the realization of cognitive ability.

As discussed earlier, effects that siblings have on one another might lead to misleading results, particularly if sibling effects systematically differ according to parents' education.

Sensitivity analyses have shown that the pattern identified exists over and above siblings' and twins' closeness (Appendix Figure A2.2). The change in the similarity of siblings and twins cannot be attributed to systematic differences in the closeness of the sibling and twin relationship.

Taken together, the results show that in all three samples, shared family influences are more important in less educated families. The more education parents have, the less alike the cognitive ability scores of siblings, DZ twins, and MZ twins. This contradicts the expectation that highly educated parents compensate for differences, whereas less educated parents reinforce differences for efficiency reasons (H1). Instead, the results support the expectation that parents make equal investments and but adopt different parenting concepts that accentuate differences among advantaged siblings (H2). Given that the analyses are based on a sample of young adults, the results show that shared family influences have a lasting impact on cognitive ability, which is stronger for less educated families. As the findings concerning the mean value of cognitive ability have shown, these influences are not necessarily conducive to the realization of cognitive ability –in fact, quite the opposite.

2.5 Conclusion and Discussion

I studied sibling similarity in cognitive ability and asked whether the degree of similarity varies with parents' education. In contrast to previous research, I extended the established sibling correlation approach to DZ twins and MZ twins. This acknowledges that genetic influences account for differences in cognitive ability (e.g., Nisbett et al. 2012; Tucker-Drob, Briley, and Harden 2013) and allows us to capture shared family influences more comprehensively, and thus to test more rigorously the link between sibling similarity and parents' education.

To explain a varying degree of similarity, I first referred to economic approaches that model parents' investment decisions within the household (Becker and Tomes 1976; Behrman, Pollak, and Taubman 1982). Against this backdrop, I tested the hypothesis that sibling similarity in disadvantaged families is lower for efficiency reasons, whereas highly educated families compensate for, and thus equalize, differences among siblings (Conley

2004, 2008). I then introduced the idea that parents might also invest equally in and accept differences among their children. I drew on the literature on stratified parenting (e.g., Cheadle and Amato 2011; Kalil, Ryan, and Corey 2012; Lareau 2011; Lareau and Weininger 2003) and put it in a within-family perspective. Because advantaged parents adopt an active role in shaping the developmental processes of their children and tend to provide more skill-enhancing and specific inputs in line with children's potentials and needs, I hypothesized alternatively that siblings from advantaged families are less similar in terms of cognitive ability compared with siblings from disadvantaged families.

My analyses yielded two findings. First, young adult siblings, DZ twins, and MZ twins in highly educated families are less alike in terms of cognitive ability compared with young adult siblings, DZ twins, and MZ twins in less educated families. This contradicts the hypothesis concerning stratified investments rationales, according to which sibling similarity increases with parents' social background (H1), and supports the hypothesis concerning equal investments and stratified parenting (H2).

Systematic differences in the degree of similarity in cognitive ability are significant in the sibling sample. This is in line with findings for the United States for literacy skills (Conley, Pfeiffer, and Velez 2007) but differs from the finding for Germany (Grätz 2018). One explanation of the divergent findings could be that the families I studied have more children (twins and at least one sibling) than the families in the study by Grätz (2018). Unfortunately, this study does not provide information about the variance components in absolute terms. The ICC is a standardized measure that does not change if the variances of shared family and child-specific influences in absolute terms change at the same time. Thus, there might be some variation in the relative importance of shared family influences that did not show up in the ICC. To evaluate to what extent results differ substantially, we would also need information on the family level variation in absolute terms.

For both DZ twins and MZ twins the results reveal the same pattern. The similarity decreases according to parents' education, though it is not statistically significant. Nonetheless, both the results for the variance components in absolute terms and for the ICC confirm that shared family influences decrease the more educated parents are. Thus, the more resources parents have, the more important are processes within the family that accentuate differences within the family.

In addition, I found that the mean level of cognitive ability increases with parents' education, whereas the relative importance of shared family influences decreases. These divergent trends show that the same shared family influences that make siblings and twins more alike are also associated with lower levels of cognitive ability. This is a very important aspect, and more research is needed to understand what kind of influences affect siblings equally and hamper the realization of cognitive ability in less educated families. In advantaged families, by contrast, parents often provide additional inputs that foster children's talents. These influences are more child-specific, which leads to higher levels of cognitive ability and promotes differences in cognitive ability among their children. Given that differences between siblings and twins from advantaged and disadvantaged backgrounds remain even as the children grow older, my results indicate a long-lasting impact of parenting on cognitive ability.

Second, my results show that the association between parents' educational background and sibling and twin similarity is not affected by the closeness of the sibling and twin relationship. I thereby address a major limitation of studies on sibling similarity. In a similar vein, my results reveal a very similar trend for siblings, DZ twins, and MZ twins, which shows that there is no "twinning effect" –that is, that twins behave profoundly differently from (full) siblings.

However, it is important to note that I used an indicator that was measured at the same time as cognitive ability. Since the quality of the sibling and twin relationship might change over the life course, it is important to back up my results –ideally, with longitudinal data. To the extent that there are no profound changes in the sibling and twin relationship until early adulthood, my results are reliable.

This study is the first to adopt a genetically sensitive approach to study the stratification of sibling similarity in cognitive ability. The results provide strong indications for parent's investment decisions that are not in line with economic theories, rather parents invest equally in their children but in distinct ways that differ according to parents' educational background. My findings challenge the implicit assumption that shared family influences such as parents' education influence children in similar fashion. Moreover, if children are raised in advantaged families, shared family influences –those that differ between families– are less important. Genetically sensitive research can help us to better understand what kinds of parental investment –net of genetic influences– result in within-family stratification, and to

formulate informative policy suggestions to enhance the skill development of children from less educated families.

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doi:10.1177/0002716214547476.

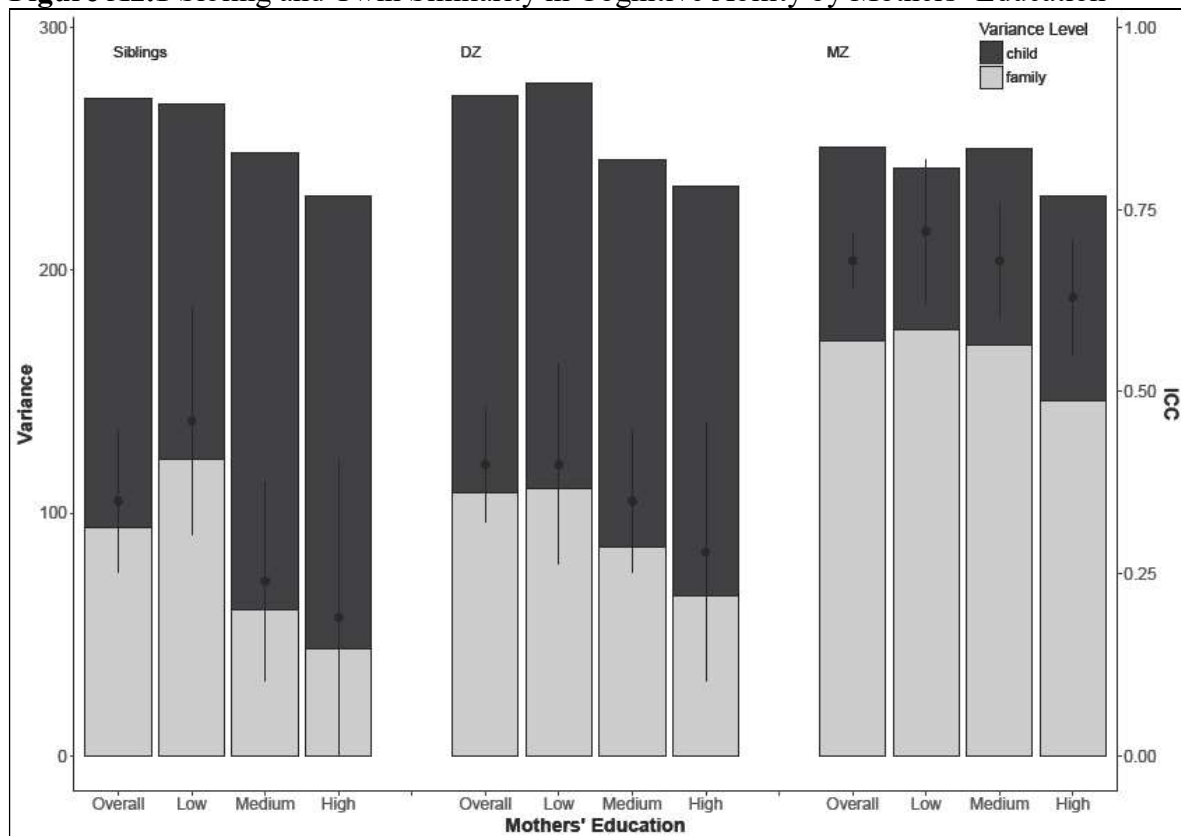
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Appendix

Table A2.1 CASMIN Educational Classification

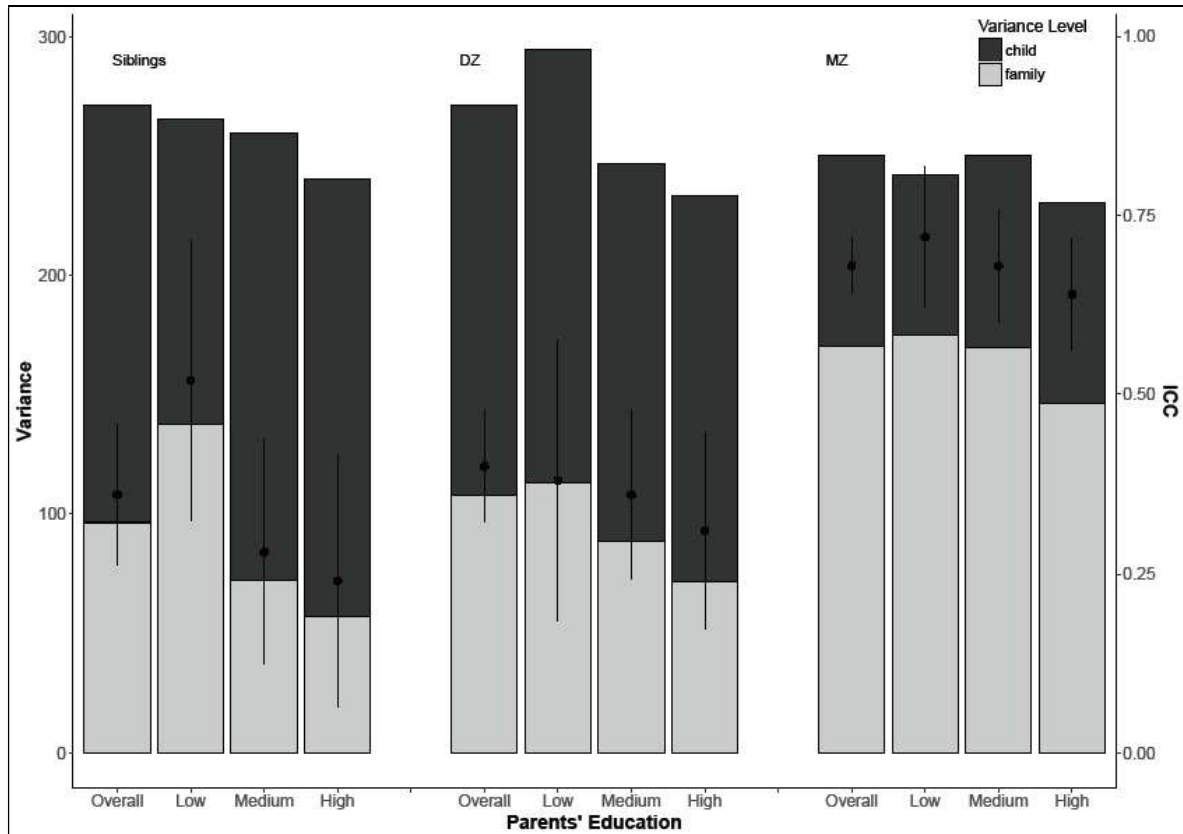
| | |
|--------|---------------------------------------|
| 1a | Inadequately completed |
| 1b | General elementary education |
| 1c | Basic vocational qualification |
| 2a | Intermediate vocational qualification |
| 2b | Intermediate general qualification |
| 2c_gen | General maturity certificate |
| 2c_voc | Vocational maturity certificate |
| 3a | Lower tertiary education |
| 3b | Higher tertiary education |

Figure A2.1 Sibling and Twin Similarity in Cognitive Ability by Mothers' Education



Source: TwinLife wave 1; own calculations.

Figure A2.2 Sibling and Twin Similarity in Cognitive Ability by Parents' Education – Controlled for Closeness



Source: TwinLife wave 1; own calculations.

3. The Social Stratification of Environmental and Genetic Influences on Education: New Evidence Using a Register-Based Twin Sample¹

Tina Baier and Volker Lang

Abstract

The relative importance of genes and shared environmental influences on stratification outcomes has recently received much attention in the literature. We focus on education and the gene–environment interplay. Specifically, we investigate whether –as proposed by the Scarr–Rowe hypothesis– genetic influences are more important in advantaged families. We argue that the social stratification of family environments affects children’s chances to actualize their genetic potential. We hypothesize that advantaged families provide more child-specific inputs, which enhance genetic expression, whereas the rearing environments of children in disadvantaged families are less adapted to children’s individual abilities, leading to a suppression of genetic potential. We test this relationship in Germany, which represents an interesting case due to its highly selective schooling system characterized by early tracking and the broad coverage of part-time schools. We use novel data from the TwinLife panel, a population-register-based sample of twins and their families. Results of ACE variance decompositions support the Scarr–Rowe hypothesis: Shared environmental influences on education matter only in disadvantaged families, whereas genetic influences are more important in advantaged families. Our findings support the growing literature on the importance of the gene–environment interplay and emphasize the role of the family environment as a trigger of differential genetic expression.

¹ Original published version: Baier, Tina, and Volker Lang. 2019. “The Social Stratification of Environmental and Genetic Influences on Education: New Evidence Using a Register-Based Twin Sample.” *Sociological Science* 6:143–71.

3.1 Introduction

The influence of family of origin on children's education is a recurring topic in stratification research. What has largely been neglected in this literature is that not only social resources but also genetic influences lead to differences in education. Behavioral genetic scientists, by contrast, commonly differentiate between genetic and environmental sources of individual variation. Recent findings based on an international meta-analysis show that shared environmental influences account for an average of about one-third of the total variation in educational attainment, whereas the relative importance of genes is only slightly higher (i.e., about 40%: Branigan, McCallum, and Freese 2013). Thus, both shared environmental and genetic influences are important to understanding individual variation in education. However, what is missing are studies addressing whether and how genetic influences on education depend on social conditions.

The Scarr–Rowe hypothesis claims that the relative importance of genes is higher in advantaged families, whereas shared environmental influences are more important in disadvantaged families (Rowe, Jacobson, and van den Oord 1999; Scarr-Salapatek 1971). The underlying assumption is that environmental conditions provided by advantaged families fit better with children's genetic dispositions, which enhances genetic expression (Bates, Lewis, and Weiss 2013; Guo and Stearns 2002; Tucker-Drob and Bates 2016; Turkheimer et al. 2003). The Scarr–Rowe hypothesis emerged in the context of studies of intelligence (IQ). Certainly, IQ is strongly correlated with outcomes that are relevant to social stratification. Nonetheless, mechanisms that determine an individual's social position are more complex and are not only determined by an individual's IQ (e.g., Strenze 2007). Thus, for stratification research, it is important to extend studies on the Scarr–Rowe hypothesis beyond IQ. This article focuses on education as one of the most relevant indicators of social stratification.

To explain the relative importance of social compared to genetic influences on education, we combine sociological theories with behavioral genetic approaches. We use the conceptual differentiation between primary and secondary effects of social background to explain the substantial impact of shared environmental influences on educational attainment (Boudon 1974). To elaborate on how social background shapes the relative importance of genetic influences on educational attainment, we draw on the literature on stratified logics of parenting (e.g., Bodovski and Farkas 2008; Cheadle and Amato 2011; Kalil, Ryan, and Corey 2012; Lareau 2011; Lareau and Weininger 2003).

Advantaged parents typically engage in so-called “strategic” or “planned” types of parenting; disadvantaged parents are more focused on addressing children’s basic needs, leading to so-called “natural growth” (Lareau 2011). Strategic parenting often entails a cultivation of skills and behaviors that are distinctive to higher social classes, so-called “concerted cultivation” (Lareau 2011). We argue that strategic types of parenting tend to be accompanied by the provision of rearing environments that are more adapted to children’s potential. In other words, such parenting behavior not only promotes skills that are rewarded in contemporary societies but also involves parenting strategies that are more child centered and hence individually adapted.

The specificity of rearing environments is important because the development of humans’ genetic potential is dependent on environmental conditions (Bronfenbrenner and Ceci 1994). Hence, if children from advantaged parents grow up in an environment that matches their individual abilities, this enhances the expression of genetic potentials, whereas the less-adapted environments more often provided by disadvantaged families lead to a suppression of genetic potential. This theoretical mechanism can explain why shared environmental influences on IQ –and potentially on education as well– play a stronger role in disadvantaged than in advantaged families. In addition, different opportunities for gene expression can accumulate over the life course (e.g., Dannefer 2003; DiPrete and Eirich 2006), as children are selected in distinct learning environments related to their social background.

We test the Scarr–Rowe hypothesis for educational attainment in Germany, which is an especially interesting case for two reasons: First, Germany has a highly stratified schooling system characterized by early tracking, leading to comparably strong secondary effects on educational attainment (e.g., Blossfeld and Shavit 1993; Hillmert and Jacob 2010; Neugebauer 2010). And second, schooling is often part time, leaving more room for parents to shape the developmental environment of their children through investments in parenting and other resources. Given these institutional features, we expect the social stratification of the relative importance of shared environmental and genetic influences to be stronger compared to countries with a comprehensive schooling system.

Our analyses are based on the TwinLife panel (Diewald et al. 2018). Since 2014, TwinLife has collected a wealth of information from monozygotic and same-sex dizygotic twins and their families residing in Germany. These data allow, for the first time, genetically sensitive analyses of educational attainment in Germany. Importantly, TwinLife overcomes one of the major weaknesses of many observational twin studies, as

a probability-based register-sampling strategy was applied (Lang and Kottwitz 2017). We estimate genetically sensitive variance decomposition models (ACE-models) of completed years of education based on twins, which enables us to distinguish between environmental and genetic influences (Guo and Wang 2002; Plomin et al. 2008; Rabe-Hesketh, Skrondal, and Gjessing 2008).

Our article makes two main contributions. First, we extend research on the Scarr–Rowe hypothesis on IQ to education and provide evidence for a social stratification of environmental and genetic influences on educational attainment in Germany. Second, we provide a theoretical account for the strong impact of shared environmental influences on education and their variation according to social background. Our explanation emphasizes the role of parental behavior in shaping children’s chances of their genetic potential being expressed.

3.2 Theoretical Background

Shared Environmental Influences on Education

The impact of shared environmental and genetic influences on education is well established (Branigan, McCallum, and Freese 2013; Heath et al. 1985; Nielsen 2016; Silventoinen et al. 2004; Taubman 1976). According to an early study of the United States based on the Veteran Twin Registry, shared environmental influences explain about one-third of the total variation in education, and genes explain about 40% (Taubman 1976). In Norway (Norwegian Twin Panel) and Finland (Finnish Twin Cohort Study), genetic influences on education are about the same, but at 40%, the relative importance of shared environmental influences is somewhat higher (Heath et al. 1985; Silventoinen et al. 2004). The findings of a recent international meta-analysis (including Australia, Denmark, Finland, Germany, Italy, Norway, Spain, Sweden, the United Kingdom, and the United States) are in line with findings based on single countries (Branigan, McCallum, and Freese 2013): Shared environmental influences account for an average of about 36% and genes account for about 40% of the variation in education. In almost every country, shared environmental influences account for more than 20% of the variation in education (Branigan, McCallum, and Freese 2013). Most of the samples

are not population based (only the samples for Finland, Italy, and parts of the United States are), and it is therefore important to replicate those findings.

Nonetheless, current findings for shared environmental influences on education diverge strongly from the pattern identified for most other adult characteristics, including those relevant to education, such as cognitive and noncognitive skills. Here, the relative importance of genes far exceeds the relevance of shared environmental influences (e.g., Freese and Jao 2017; Nielsen 2016; Polderman et al. 2015; Turkheimer 2000).

Before we discuss current explanations for the comparatively strong impact of shared environmental influences on education, we briefly explain their meaning. Shared environmental influences represent all nongenetic influences that lead to similarities among siblings. They cannot simply be equated with indicators of shared family background because such factors can also make children from the same family less alike. For example, siblings may share the experience of their parents' divorce but can have very different reactions to it (Turkheimer and Waldron 2000). This in turn is a nonshared environmental influence, as it leads to differences among siblings (Turkheimer and Waldron 2000). Thus, only factors that affect children from the same family in similar fashion are shared environmental influences. It is standard fare in the behavioral genetics literature that the definition of shared environmental influences is based on their effects (i.e., whether children are equally affected or not). Shared environmental influences on education, therefore, represent transmission mechanisms that affect siblings in a similar way – net of genetic factors (Freese and Jao 2017; Nielsen and Roos 2015).

To date, the literature provides two sets of explanations for shared environmental influences on education. The first set discusses the impact of assortative mating, sibling effects, and the measurement of education (Freese and Jao 2017; Nielsen and Roos 2015).

The standard approach to estimating environmental and genetic influences (see section Data and Methods) assumes that spouses mate randomly with regard to the characteristic under study. This justifies the assumption that siblings share on average about 50% of their genes. However, previous research shows that spouses are more similar in their education than expected given random mating across Western societies (e.g., Blossfeld 2009). It is plausible to assume that spouses are also more similar in genetic endowments relevant to education, which in turn increases (on average) the similarity of their children with respect to genes. Assortative mating, therefore, leads to an underestimation of genetic influences and an overestimation of shared environmental influences because the genetic similarity of siblings is higher than

assumed by standard models building on the random-mating assumption (e.g., Plomin et al. 2008).

Sibling effects may also account for comparably strong shared environmental influences on education. Such sibling effects can occur if the educational decisions of one sibling guide those of other siblings (Freese and Jao 2017; Nielsen and Roos 2015). For example, older siblings can serve as a role model for younger ones (Benin and Johnson 1984). In that case, shared environmental influences are the consequence of the mutual influences of siblings rather than being the direct consequence of parents' actions.

Finally, the measurement of educational attainment can affect estimations of shared environmental influences. In general, fine-grained measures are better suited to detect differences between individuals. If coarse measures are used (e.g., tertiary vs. nontertiary education), the estimates for shared environments tend to be larger compared to more nuanced measures (e.g., years of education) (Freese and Jao 2017; Nielsen and Roos 2015).

The second set of explanations discusses family resources, parents' educational expectations, and the broader family context (Freese and Jao 2017; Nielsen and Roos 2015). In light of the large body of sociologically inspired work that links parents' material and nonmaterial resources with children's educational attainment (e.g., Blau and Duncan 1967; Breen and Jonsson 2005; Haller and Portes 1973; Torche 2015), we focus on this set of explanations. We address this first set of explanations, which we label "alternative explanations," in our analysis to rule out the possibility that our findings (with respect to shared environmental influences on education) are driven by any of these aspects.

To integrate current explanations of family resources, we draw on the theory of primary and secondary effects of social background on educational attainment (Boudon 1974). This theory is well established in the stratification literature to conceptualize how parents' social background and related resources affect children's education. Primary effects describe how parents influence children's academic performance, and the most important input factors are cognitive and noncognitive skills. Advantaged parents provide more stimulating home environments, provide relevant learning materials, and often transmit cultural resources and interests, all of which further school-related skills (e.g., Chedale and Amato 2011; Cunha and Heckman 2008; Kalil, Ryan, and Corey 2012; Lareau 2011; Lareau and Weininger 2003). Secondary effects, by contrast, describe

systematic differences in educational decisions by social background –net of academic achievement. Parents evaluate the costs, anticipated benefits, and likelihood of success differently according to their own social position and educational experience (Breen and Goldthorpe 1997; Erikson and Jonsson 1996). An important driver of stratified educational choices is the intention to avoid downward mobility. In order to maintain social status, advantaged parents tend to have higher educational aspirations for their children compared to disadvantaged parents, who can reach this goal with lower levels of education (Breen and Goldthorpe 1997). Thus, even if the academic performances of advantaged and disadvantaged children do not differ, children from higher social backgrounds are still more likely to choose higher levels of education than their counterparts from lower social backgrounds (Boudon 1974; Breen and Goldthorpe 1997; Erikson and Jonsson 1996).

Primary and secondary effects do not work independently of each other. Parents with high educational aspirations for their children are also more likely to prompt children's academic skills accordingly (for a discussion on anticipatory effects, see Erikson et al. 2005). Such anticipatory behavior leads to an underestimation of secondary effects because such efforts affect children's academic performance and are hence inadequately attributed to primary effects (Erikson et al. 2005). Nonetheless, this conceptual differentiation acknowledges that differences in education emerge not only because of differences in academic skills but also because of stratified cost-benefit calculations.

In light of the secondary effects, shared environmental influences on educational attainment are comparatively strong because parents choose educational routes based not only on their children's academic performance. Parents tend to have similar, social class-specific educational aspirations and educational choices for their children in order to maintain social status. As a consequence, even if their children differ in school-related skills, they still end up being more alike with regard to their education. Because similar educational choices within families affect the development of cognitive and noncognitive skills less directly than they affect educational attainment, we expect shared environmental influences to be stronger for educational attainment than for cognitive and noncognitive skills, as is reported in the literature (e.g., Polderman et al. 2015; Turkheimer 2000). Based on this framework, we formulate our first hypothesis:

Hypothesis 1 (H1): Secondary effects of social background explain a substantial part of the shared environmental influences on educational attainment.

The Social Stratification of Environmental Influences and the Gene–Environment Interplay

The environment in which children develop their skills can also vary within socially defined groups (Bodovski and Farkas 2008; Cheadle 2008; Cheadle and Amato 2011; Kalil, Ryan, and Corey 2012; Lareau 2011; Lareau and Weininger 2003). Specifically, how differently parents treat their children and the variety of inputs they provide can differ between advantaged and disadvantaged families. Such differences in rearing environments are important because human development is embedded in proximal processes (i.e., specific environmental influences that help individuals realize their genetic potential [e.g., conditions set by families, peers, or institutions]; Bronfenbrenner and Ceci 1994). To be effective, such environmental influences have to be encountered regularly (Bronfenbrenner and Ceci 1994). One prominent hypothesis concerning such a gene–environment interaction is the Scarr–Rowe hypothesis, which claims that proximal processes leading to the actualization of genetic potential are more prevalent in enriched environmental settings (Rowe, Jacobson, and van den Oord 1999; Scarr-Salapatek 1971).

Against the backdrop of stratified family environments, we argue that this type of gene–environment interplay is important to understanding how genetic and environmental influences affect educational attainment. In her U.S. study, Lareau (2011) scrutinizes how distinct cultural habits consolidate daily parenting behavior. Different logics of parenting affect children’s development and skill-formation processes. Advantaged parents follow a parenting concept labeled concerted cultivation, which describes parenting practices that foster behaviors and skills that are distinctive to higher social classes (Lareau 2011). More importantly for this study is that parents from higher social backgrounds adopt an active role in their children’s development (Lareau 2011). Parents frequently plan activities with their children and provide educationally relevant inputs (e.g., books or other learning materials) to raise their children’s interests and motivation to learn. Parents more often structure children’s time outside of school and engage them in extracurricular activities (e.g., music lessons or sports clubs (Covay and Carbonaro 2010; Dumais 2006)). As a consequence, children from higher social backgrounds grow up in stimulating home environments. We argue that a more active role in parenting also implies that home environments are more adapted to children’s individual potential and needs because parents focus on planned interactions and stimulating activities with their children. In addition, children learn to speak up for themselves and communicate with institutions, especially instructors and educators.

Moreover, parents extend their efforts to the schooling context, as they interact frequently with teachers and intervene on behalf of their children if needed (Bodovski and Farkas 2008; Cheadle 2008; Cheadle and Amato 2011). The acquisition of such skills and parents' efforts can lead to further individual adaptation of learning environments outside of the family context.

In contrast, disadvantaged parents tend to perceive their children's development as a naturally evolving process (Lareau 2011) and follow a parenting concept labeled natural growth. Parenting is focused more on fixed interaction routines. Given their limited resources, disadvantaged parents are usually occupied with meeting the basic needs of children and are less, if at all, involved in children's learning and out-of-school activities. This also implies that parents less often customize children's leisure time or provide tailored inputs to foster children's skills. As a consequence, a fit between environments and potential (or the specific abilities of children) is more often coincidental and less often planned than it is in the case of advantaged families. Furthermore, there are fewer discussions between parents and their children, and children follow their parent's instructions instead of questioning them. And because disadvantaged parents more firmly believe that children's education is the responsibility of teachers, parents question teachers' behavior toward their children less often (Lareau 2011).² Previous research shows that differences in parenting behavior are significantly associated with scholastic performance (Bodovski and Farkas 2008; Roksa and Potter 2011).

Originally, the concept of different logics of parenting was applied to understand how class-related differences between families emerge (i.e., why advantaged children tend to outperform their counterparts from disadvantaged families). However, we argue that the notion of different logics of parenting also allows for conclusions about differences between children from the same family: Advantaged parents plan and can afford investments that promote specific talents and interests of their children. Such individualized investments can be expected to be more effective. By contrast,

² Neither of the logics of parenting is better or worse, per se. Different practices provoke different types of skills (e.g., disadvantaged children can become more autonomous as they decide by themselves what they want to do in their leisure time; they are also better in learning by experience). The concept of concerted cultivation, however, fosters skills that are rewarded more by contemporary educational institutions.

disadvantaged parents have lower levels of resources and might not have the time to discover their children's specific talents, or they lack the resources to further them individually.

The social stratification of parenting behavior, therefore, provides an understanding of the theoretical mechanisms underlying the Scarr–Rowe hypothesis from a sociological perspective. Stratified differences in parenting trigger different conditions for genetic expression. Disadvantaged parents provide environmental conditions that are less adapted to children's individual abilities and hence less often match children's genetic dispositions. This increases the relative importance of shared environmental influences on educational attainment. By contrast, advantaged parents provide inputs to actively foster children's specific talents. Environmental conditions are more often in line with children's genetic make-up, which enhances genetic expression.

In addition to environmental conditions provided by the family, children are also exposed to different school environments and related peer groups (Freese and Jao 2017); these are the consequence of stratified educational choices. For example, in the United States, which has a within-school tracking system, it is mostly disadvantaged students who attend lower tracks with less-qualified teachers (Heubert and Hauser 1999; Lucas 1999). Due the persistent stratification of learning environments, systematic differences in the realization of developmental potential can be exacerbated over the life course (Dannefer 2003; DiPrete and Eirich 2006). Given this literature, we formulate our second hypothesis:

Hypothesis 2 (H2): Shared environmental influences on educational attainment are more important for children from disadvantaged families, whereas genetic influences matter more for children from advantaged families.

To date, the Scarr–Rowe hypothesis has not been tested for educational attainment based on sibling or twin data. Nonetheless, studies report that the relative importance of shared environmental influences is sensitive to macrostructural differences (Branigan, McCallum, and Freese 2013; Heath et al. 1985; Nielsen and Roos 2015). In most countries, shared environmental influences on educational attainment declined over the twentieth century, although they have increased in the United States (Branigan, McCallum, and Freese 2013; Heath et al. 1985; Nielsen and Roos 2015).

In addition to twin studies, in which genetic influences are indirectly assessed based on the information on their genetic relatedness and common upbringing (for an overview, see Plomin et al. 2008), researchers also use direct measures of genes based on DNA samples (for an overview, see Conley 2016) to study the impact of genes. Studies in this research area have used polygenic scores (PGS) for education –a measure constructed based on DNA samples– and examined whether the association between these PGS and education changed by social background (Conley et al. 2015; Domingue et al. 2015). Conley et al. (2015) report that the effect of PGS on education did not systematically vary by social background. The study by Domingue et al. (2015), by contrast, shows that the association between PGS and education decreased with social background for younger birth cohorts. Thus, previous studies based on PGS do not support the Scarr–Rowe hypothesis. PGS are conceptually much closer to the pathways of genetic transmission than twin studies. But until recently, PGS studies related to social stratification outcomes (such as educational attainment) had limited predictive power (Okbay et al. 2016). For example, the PGS used in these studies explained only about 2 to 3% of the total variation in educational attainment (Conley et al. 2015). However, genomic analyses are rapidly evolving, and advancements in genotyping procedures and larger sample sizes have already led to significant improvements in the predictive power of PGS for educational attainment (Okbay et al. 2016). According to a recent study, current PGS account for about one-fifth of the variation in educational attainment (Liu 2018). It is likely that these improvements also affect findings on the moderating effect of social background on genetic influences (see also Conley et al. 2015). Thus, the findings of previous studies are preliminary and need to be replicated before we can draw conclusions about the moderating effect of social background (Domingue et al. 2015).

A comparatively large body of literature has tested the Scarr–Rowe hypothesis for IQ based on twin or sibling designs (e.g., Bates, Lewis, and Weiss 2013; Figlio et al. 2017; Guo and Stearns 2002; Tucker-Drob and Bates 2016; Tucker-Drob, Briley, and Harden 2013). Here, the evidence is also mixed. Most research refers to the United States and supports the Scarr–Rowe hypothesis. However, these findings have recently been challenged by studies that include countries other than the United States (Tucker-Drob and Bates 2016), and the most recent study for the United States also finds no support for the Scarr–Rowe hypothesis regarding IQ (Figlio et al. 2017).

The German Context

Germany represents an especially interesting case for our study, as the link between social background and children's education is exceptionally strong (e.g., Blossfeld and Shavit 1993; Hillmert and Jacob 2010; Neugebauer 2010). The educational system plays a pivotal role: Even though tracking is a common feature of European educational systems, children in Germany are tracked as early as the age of 10 to 12 years into one of the three hierarchically structured secondary-school types (Hauptschule, Realschule, and Gymnasium). Secondary schooling tracks differ strongly in their curricula and length and are linked to postsecondary alternatives. Only a diploma from the highest secondary level (Gymnasium) entitles a student to study in tertiary-level institutions. Only recently have reforms been implemented to loosen the link between secondary education and postsecondary alternatives (Betthäuser 2017).

It is possible to switch between secondary-schooling tracks. However, upward moves are rare and linked to social background (Henz 1997; Jacob and Tieben 2010). Secondary-school-type decisions are –with variations in regulations between federal states– made jointly by teachers and parents. Teacher recommendations are provided for all students, though they are not binding in every federal state. Recommendations are based on children's academic performance and expectations about future development. However, research shows that children from lower social background are (*ceteris paribus*) less likely to receive a recommendation for the highest academic track than are children from higher social backgrounds (Baumert, Trautwein, and Artelt 2003). Furthermore, the quality of the learning environment differs between the tracks due to compositional effects (i.e., students are more similar in terms of social background and achievement) and institutional effects (i.e., curricula and teaching personnel; Maaz et al. 2008).

Another distinct feature of the German educational system is that primary and secondary schools are frequently part-time. Full-time schools, by contrast, end about late afternoon and provide food, extracurricular activities, and usually support with homework. Initiatives toward an expansion of full-time schooling took place from 2002 to 2008 (Sekretariat der Kultusministerkonferenz 2008, 2010).³ Despite these efforts,

³ During 2002 and 2008, the share of full-time schooling increased substantially, although different school tracks were expanded unevenly: Full-time schooling in primary schools and intermediate secondary-schools (Realschule) tripled and quadrupled in the lowest secondary school track

part-time schools are overall still widespread in Germany. However, there is large variation in part-time schooling, especially, between eastern and western Federal States. Previous research finds that extracurricular activities for children can lower social disparities in performance (Covay and Carbonaro 2010; Dumais 2006). Thus, part-time schools leave much more room for parents' resources and parenting practices to influence children and can (depending on their quality) reinforce social disparities in academic performance, whereas full-time schools can serve as an equalizer (Fischer and Theis 2014; Kuhn and Fischer 2011). Taken together, the stratified schooling system and the broad coverage of part-time schools make children's educational attainment particularly sensitive to social background. Therefore, we argue that the proposed mechanisms driving the social stratification of shared environmental influences on education are comparatively strong in Germany.

3.3 Data and Methods

Sample

We use data from the first wave of the German twin panel TwinLife (Diewald et al. 2018). TwinLife collects extended twin family information (i.e., on twins, their parents, siblings, and partners) for monozygotic (MZ) and same-sex dizygotic (DZ) twins residing in Germany. Due to the social and regional stratified probability-based sampling strategy, TwinLife provides a unique opportunity to analyze a broad range of the social spectrum with behavioral genetic methods (Lang and Kottwitz 2017). We examine twin pairs from the oldest birth cohort (1990–1993), who were between 22 and 25 years old at the time of the survey. We study twin pairs in which both twins provided valid information on their education (for 3% of the sample, this information is missing or incomplete). The analysis sample comprises 1930 twins (47% DZ; 53% MZ).

(Hauptschule), whereas full-time schools for the highest schooling track (Gymnasium) doubled (Sekretariat der Kultusministerkonferenz 2008, 2010).

Variables

Educational attainment is measured in years of education. We transformed the categorical information on respondents' educational level (general education and professional training) into corresponding years of education using established coding schemes for Germany (Socio-Economic Panel Group 2017; see Appendix Table A3.1). The coding scheme is based on educational levels and not on the actual time spent in educational institutions. The continuous measure has the advantage that linear genetically sensitive variance decompositions can be estimated (see section Analytical Strategy). We centered educational attainment to 13 years of education in all our analyses.

Due to the age range of twins, about 59% were still enrolled in professional training at the time of the interview. For these twins, we do not know whether they finish their education or not. To address this uncertainty, we ran two sets of analyses. The first set assumes that all twins currently enrolled finish their professional training and that they do not attend additional professional training leading to a higher educational degree at some later point. In this scenario, twins get assigned the associated additional years of education of the degree they are currently pursuing (so-called "upper-bound scenario" with respect to final years of education). The second set of analyses relies on the assumption that all twins currently enrolled drop out and again that they do not attend additional professional training leading to a higher educational degree at some later point. In this case, twins do not get assigned the additional years of education of the degree they are currently pursuing (so-called "lower-bound scenario" with respect to final years of education).

To evaluate the findings, it is important to note that in Germany, the share of young adults (aged between 20 and 30 years) without vocational training or tertiary education fluctuated between 14.6% and 12.9% from 1996 to 2015 (Bundesinstitut für Berufsbildung 2017). Thus, most young adults who do not finish their current professional training will switch to another professional training and will not entirely drop out. Moreover, given the strong linkage between secondary education and postsecondary alternatives in Germany, a switch within the current type of profession (horizontal change) is more likely than a vertical switch between vocational training and tertiary education. The majority of twins currently in professional training will, therefore, receive a certificate either from vocational training or tertiary education. Thus, the final distribution of educational attainment –which is observable in a few years– is most likely closer to the upper-bound than to the lower-bound scenario.

We indicated social background with parents' education measured in years.⁴ We chose education because of its significant role in shaping an offspring's chances of educational attainment (e.g., Bukodi and Goldthorpe 2013; Hout and DiPrete 2006). Parents' education covers not only transmission mechanisms that run through economic resources but also transmission mechanisms driven by socially stratified educational choices and specific parental habits and practices, all of which affect children's skill formation (Breen and Goldthorpe 1997; Erikson and Jonsson 1996). We used the same coding scheme that we used for the twins (see Appendix Table A3.1).⁵ We took the information on the parent with the highest educational level (dominance principle) and centered parents' years of education on 13 years of education.

For the subgroup analyses, we distinguished between the following overlapping educational subgroups: 7 to 11, 9 to 12, 10 to 13, 11 to 14, 12 to 15, 13 to 16, 14 to 17, 15 to 18, 16 to 19, and 17 to 20 years of parental education. If we had created mutually exclusive subgroups based instead on several years of education, we would run the risk of our findings relating to a change in the variance components being driven by the cutoff points we have chosen. At the same time, however, analyses that compare variance components for every single year of parents' education are not feasible due to small sample sizes. Overlapping groups have the advantage that our conclusions are not dependent on cutoff points and offer a more nuanced understanding of the changes in the variance components over the entire range of parents' education.

To account for sibling effects, we included a measure of the closeness of twins in our analyses based on three items with a five-point rating scale. We used the following three items: (1) *How often do you talk about important things with (name of the other twin)?* (2) *How often do you attempt to cheer up (name of the other twin)?* (3) *How close do you feel to (name of the other twin)?* We used confirmatory factor analysis based on a structural equation model to construct a single indicator of these items. The coefficient of determination for this indicator was 0.90.

As information on parents' education is sometimes missing (4.6% for mothers; 37.9% for fathers), we imputed missing information by means of multiple imputation with

⁴ Results remain robust when we used parents' occupational status (as indicated by the International Socio-Economic Index of Occupational Status [ISEI]) instead of parents' education.

⁵ In addition to the codes used for the young adult twins, we included a doctoral category for the parents (see Appendix Table A3.1).

chained equations and created 20 imputations for each observation (van Buuren et al. 2006). All of the predictors used for the imputation were at the family level (i.e., the imputation model was based on information about the parents). Thus, the imputation model is not suitable to predict missing information for twins individually. Our results are robust with and without the imputed information on parents' education (see Appendix Table A3.2 and Table A3.3).

Table 3.1 presents the distributions of the sample characteristics. The main variables are distributed fairly similarly among MZ and DZ twins. The closeness indicator is exceptional, as MZ twins are significantly closer to one another than DZ twins ($p < 0.001$). Differences in closeness between MZ and DZ have been found previously (Fortuna, Goldner, and Knafo 2010) and might be rationalized in terms of the stronger similarity of MZ twins.⁶

⁶ Because MZ twins are closer to each other than DZ twins, we run an additional analysis in which we accounted for differences in closeness by zygosity. In addition, we examined whether the closeness of MZ and DZ twins varies by parental education. The results remained stable for both the upper- and lower-bound scenarios.

Table 3.1 Descriptive Statistics

| | DZ twins | | | | MZ twins | | | |
|--|-----------------|-------|------|-----|-----------------|-------|------|------|
| | Mean/ SD | Min | Max | N | Mean/ SD | Min | Max | N |
| <i>Individual (twin) level variables:</i> | | | | | | | | |
| Twins' years of education: | | | | | | | | |
| Lower-bound scenario | 12.80 (2.35) | 7 | 18 | 900 | 13.00 (2.23) | 7 | 18 | 1030 |
| Upper-bound scenario | 14.20 (3.04) | 7 | 18 | 900 | 14.50 (2.90) | 7 | 18 | 1030 |
| Closeness | -0.23 (1.03) | -4.06 | 1.03 | 900 | 0.21 (0.93) | -4.06 | 1.03 | 1030 |
| <i>Family-level (twin-pair-level) variables:</i> | | | | | | | | |
| Twin pair's age | 23.03 (0.81) | 21 | 25 | 450 | 23.05 (0.83) | 21 | 25 | 515 |
| Twin pair's sex (1 = female) | 0.57 (0.50) | 0 | 1 | 450 | 0.59 (0.49) | 0 | 1 | 515 |
| Parents' years of education: | | | | | | | | |
| Highest in family | 13.37 (2.98) | 7 | 20 | 449 | 13.15 (3.09) | 7 | 20 | 513 |
| Highest in family (imputed) | 13.86 (2.76) | 7 | 20 | 450 | 13.66 (2.80) | 7 | 20 | 515 |
| Mother | 12.72 (2.89) | 7 | 20 | 425 | 12.46 (2.68) | 7 | 20 | 482 |
| Mother (imputed) | 12.71 (2.84) | 7 | 20 | 442 | 12.43 (2.62) | 7 | 20 | 509 |
| Father | 13.29 (3.09) | 7 | 20 | 257 | 13.31 (3.17) | 7 | 20 | 276 |
| Father (imputed) | 13.20 (2.62) | 7 | 20 | 406 | 13.09 (2.70) | 7 | 20 | 451 |

Source: TwinLife wave 1; own calculations.

Analytical Strategy

Our analyses are based on a classical twin design (CTD) (e.g., Plomin et al. 2008). The CTD is a method in behavioral genetics that is widely used to estimate the relative importance of environmental and genetic influences (Plomin et al. 2008). Twins are born and raised at the same time, and MZ twins are additionally genetically alike; DZ twins share on average 50% of the 1% of all genes in which humans tend to vary. A CTD uses this knowledge to decompose the variance of an outcome into a component associated

with additive genetic influences (A), a component associated with shared environmental influences (C), and a component associated with unique environmental influences, including the error term of the decomposition (E) (Table 3.2). This type of analysis is called ACE variance decomposition.

Table 3.2 Variance Decomposition Based on the CTD

| Variance component | Definition | Differences within twin pairs | |
|--------------------|--|-------------------------------|-----|
| | | MZ | DZ |
| A | Additive genetic influences | No | Yes |
| C | Shared environmental influences (that make twins alike –net of genes; e.g., shared effects of parents’ education or financial resources) | No | No |
| E | Nonshared environmental influences (leading to differences between twins –net of genes; e.g., selective parenting, selective peer influences, and measurement error) | Yes | Yes |

To identify the A and C components, the CTD relies on additional assumptions. First, it is assumed that genetic effects are additive, which means that the effects of different genes are independent. This implies that there is no epistasis (i.e., there are no interactions between genetic influences that affect the outcome under study). Second, the CTD assumes that the genetic and environmental components are additive (i.e., that there are no correlations or interactions between genes and their environment). This assumption is challenged by the Scarr–Rowe hypothesis and therefore is tested in the second part of our analysis focusing on H2. The third assumption is the equal environments assumption (EEA), which states that the trait under study is not affected by the fact that MZ twins are differently treated by their environment than DZ twins (Scarr and Carter-Saltzman 1979). The EEA has been critically evaluated for several, mostly psychological, traits. We are not aware of any study that investigates the EEA with regard to education, though studies report that more similar environments of MZ twins do not lead to an overestimation of genetic influences on IQ (Derks, Dolan, and Boomsma 2006). The fourth assumption states that spouses mate randomly. Given random mating, the genetic similarity of siblings is on average about 0.5. As stated above, assortative mating

increases the genetic similarity of siblings. If information on parents' education is available, it is possible to estimate an average genetic correlation for DZ twins that corrects for assortative mating (Loehlin, Harden, and Turkheimer 2009). The correction is given by $0.5 + 0.5 * h_0^2 * r_p$, where h_0^2 denotes the share of genetic influences (A) estimated without correction for assortative mating and r_p denotes the correlation of parents with respect to the trait under study (Loehlin, Harden, and Turkheimer 2009). In our sample, r_p is 0.46, whereas h_0^2 is 0.43 for the lower-bound scenario and 0.46 for the upper-bound scenario. These inputs imply an assumed average genetic correlation for DZ twins of 0.60 for both scenarios.

We test our hypotheses as follows: We first estimated ACE variance decompositions for the lower- and upper-bound scenarios using the linear multilevel mixed-effects parameterization developed by Rabe-Hesketh and collaborators (2008) without controlling for parental educational background (base model). This parameterization can be extended (like the standard regression approach) by including explanatory variables on which the mean of the outcome (in our case, twins' years of education) is regressed. We then controlled for parents' education in model 1 to test H1. Next, we stepwise controlled for parents' assortative mating (model 2) and sibling effects (model 3). To test H2, we estimated ACE variance decomposition models separated by parents' years of education. This research strategy is also known as nonparametric gene-environment interaction analysis (Guo and Wang 2002). All analyses were computed with the statistical software Stata (14.2) using `acelong.ado` (Lang 2017).

3.4 Results

Table 3.3 and Figure 3.1 present the results for the ACE variance decompositions for twins' years of education. We start with the base model. In the lower-bound scenario, the mean value for years of education was about 12.9 years (constant +13) and 14.4 years for the upper-bound scenario. The total variances were 5.3 and 8.8 years of education, respectively. Genetic influences (A) accounted for about 44% of the total variation in educational attainment in the lower-bound scenario and about 41% in the upper-bound scenario; shared environmental influences (C) accounted for 27% in the lower-bound scenario and about 40% in the upper-bound scenario. Accordingly, in the lower-bound

scenario, 30% can be attributed to nonshared environmental influences and measurement error (E), and 20% can be attributed as such in the upper-bound scenario. In line with theoretical expectations and previous findings (i.e., 36% C and 40% A; see Branigan, McCallum, and Freese 2013), we find substantial shared environmental influences on educational attainment in Germany.

Table 3.3 ACE Variance Decompositions for Twins' Years of Education

| | Lower-bound scenario b/var | Upper-bound scenario b/var |
|---|-------------------------------|-------------------------------|
| Base model: | | |
| Assumed genetic DZ correlation | 0.50 | 0.50 |
| Constant | -0.10 (0.07) | 1.38* (0.09) |
| Total variance | 5.26* (0.23) | 8.84* (0.23) |
| A in % | 43.57* (7.97) | 41.00* (6.52) |
| C in % | 26.59* (7.14) | 39.73* (6.21) |
| E in % | 30.32* (2.88) | 19.72* 1.78 |
| Model 1 (parents' education): | | |
| Assumed genetic DZ correlation | 0.50 | 0.50 |
| Parents' years of education | 0.25* (0.02) | 0.38* (0.03) |
| Constant | -0.29* (0.06) | 1.08* (0.09) |
| Total variance | 5.26* (0.23) | 8.84* (0.23) |
| A in % | 43.36* (7.83) | 41.50* (6.70) |
| C in % | 16.33 ^b (7.03) | 24.46* (6.31) |
| E in % | 30.34 (2.63) | 19.67* (1.77) |
| Explained variance (R ²) in % | 9.97 | 14.37 |
| Model 2 (assortative mating): | | |
| Assumed genetic DZ correlation | 0.60 | 0.60 |
| Parents' years of education | 0.25* (0.02) | 0.38* (0.03) |
| Constant | -0.29* (0.06) | 1.08* (0.09) |
| Total variance | 5.26* (0.23) | 8.84* (0.23) |
| A in % | 54.28* (9.77) | 51.40* (8.30) |
| C in % | 5.41 (8.76) | 14.56 (7.76) |
| E in % | 30.34* (2.63) | 19.67* (1.77) |
| Explained variance (R ²) in % | 9.97 | 14.37 |

Table continued on next page

| Model 3 (sibling effects): | | |
|---|--------|--------|
| Assumed genetic DZ correlation | 0.60 | 0.60 |
| Parents' years of education | 0.25* | 0.38* |
| | (0.02) | (0.03) |
| Closeness of twins | 0.24* | 0.28* |
| | (0.05) | (0.07) |
| Constant | -0.29* | 1.09* |
| | (0.06) | (0.09) |
| Total variance | 5.26* | 8.84* |
| | (0.23) | (0.23) |
| A in % | 55.24* | 53.60* |
| | (9.71) | (8.38) |
| C in % | 2.28 | 10.89 |
| | (8.58) | (7.84) |
| E in % | 30.58* | 19.56* |
| | (2.64) | (1.78) |
| Explained variance (R ²) in % | 11.90 | 15.95 |

Note: All models are based on 965 twin pairs, and clustered standard errors are calculated at the twin-pair level. Clustered standard errors are in parentheses. ^b P ($Z > |z|$) < 0.05. * P ($Z > |z|$) < 0.01 (two-tailed tests). Source: TwinLife wave 1; own calculations.

In model 1, we examine the role of secondary effects and controlled for education. First of all, we found that parental education has a strong effect on twins' education in both scenarios. In the lower-bound scenario, twins' educational attainment increases by 0.25 years for each additional year of parental education. About 10% of the total variation in twins' years of education is explained by parents' education (see explained variance [R²] in %). In the upper-bound scenario, the effect of parents' education is about 0.38 years, and 14% of the total variance is explained. In both scenarios, the relative importance of genetic influences remained stable. By contrast, the relative importance of shared environmental influences was smaller. Shared environmental influences explain about 16% of the total variation in the lower-bound scenario and 25% in the upper-bound scenario. Hence, the results for both scenarios show that the relative importance of genes hardly changed once parents' education was controlled for. By contrast, the relative importance of shared environmental influences was about 11 to 15% lower if parents' education is controlled for. These results indicate that parents' education mostly accounts for shared environmental influences and to a much less extent for genetic influences. As a consequence, the results offer support for H1 (i.e., that socially stratified educational choices [secondary effects of social background] explain a substantial part of shared environmental influences on education). However, a substantial impact of shared environmental influences on education remains unexplained after controlling for parents' education.

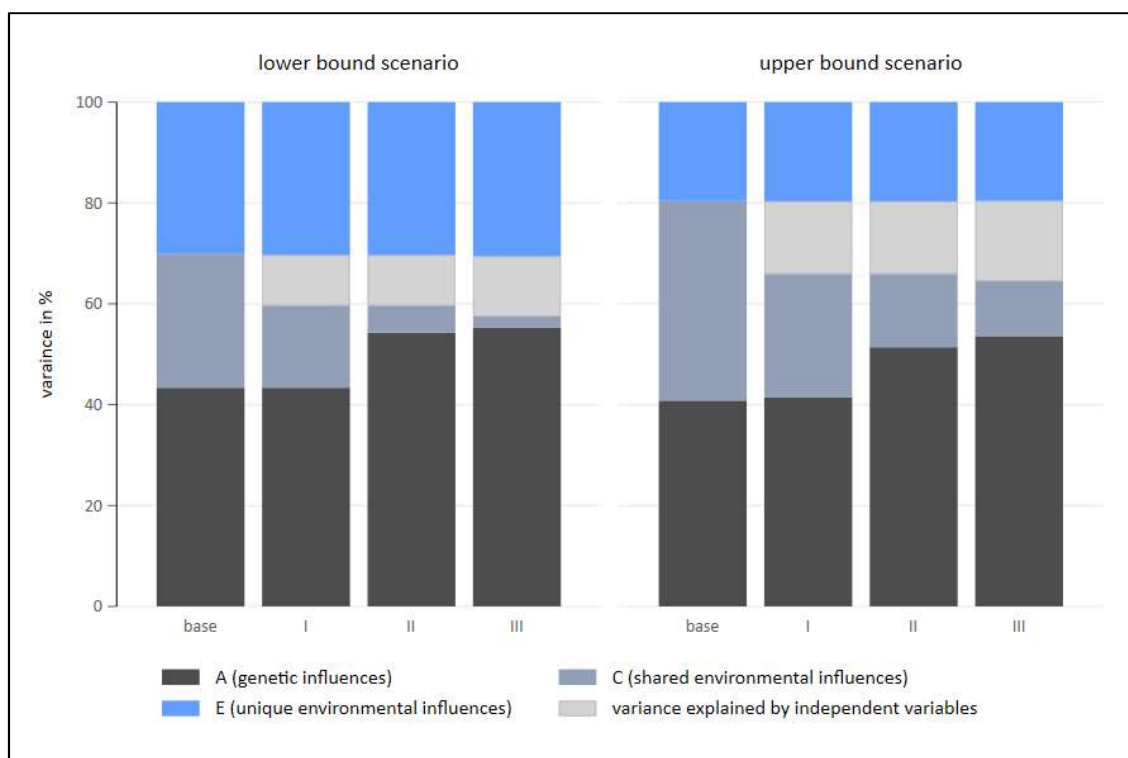
In model 2 we analyzed the role of alternative explanations. We assess their effects in conjunction with the findings for H1. In our sample, assortative mating increased the genetic similarity to 0.6 (see assumed genetic DZ correlation).⁷ The relative importance of genetic influences was about 54% in the lower-bound scenario and about 51% in the upper-bound scenario, whereas the relative importance of shared environmental influences was only about 5% in the lower-bound scenario and 15% in the upper-bound scenario. Thus, in both scenarios, shared environmental influences are about 10% lower compared to the findings in which random mating is assumed. Furthermore, the impact of the shared environmental component is no longer significant. Thus, both parents' education and assortative mating account for shared environmental influences on education in Germany.

In model 3, we accounted for sibling effects. The closeness of twins had a positive significant effect on their educational attainment. An increase of one standard deviation in twins' closeness is associated with an increase of about 0.25 years of education and an about 2% increase in explained variance compared to model 2. Hence, sibling effects account for shared environmental influences to some extent, but they were not as strong of an explanatory factor as parental education and parental assortative mating.

In sum, when all theoretical explanations are accounted for, the relative importance of shared environmental influences on educational attainment is no longer significant. In fact, shared environmental influences were only about 2% in the lower-bound scenario and about 11% in the upper-bound scenario.

⁷ The adjustment for assortative mating changes only the estimations for the relative importance of shared environmental and genetic influences.

Figure 3.1 ACE Variance Decompositions for Twins' Years of Education



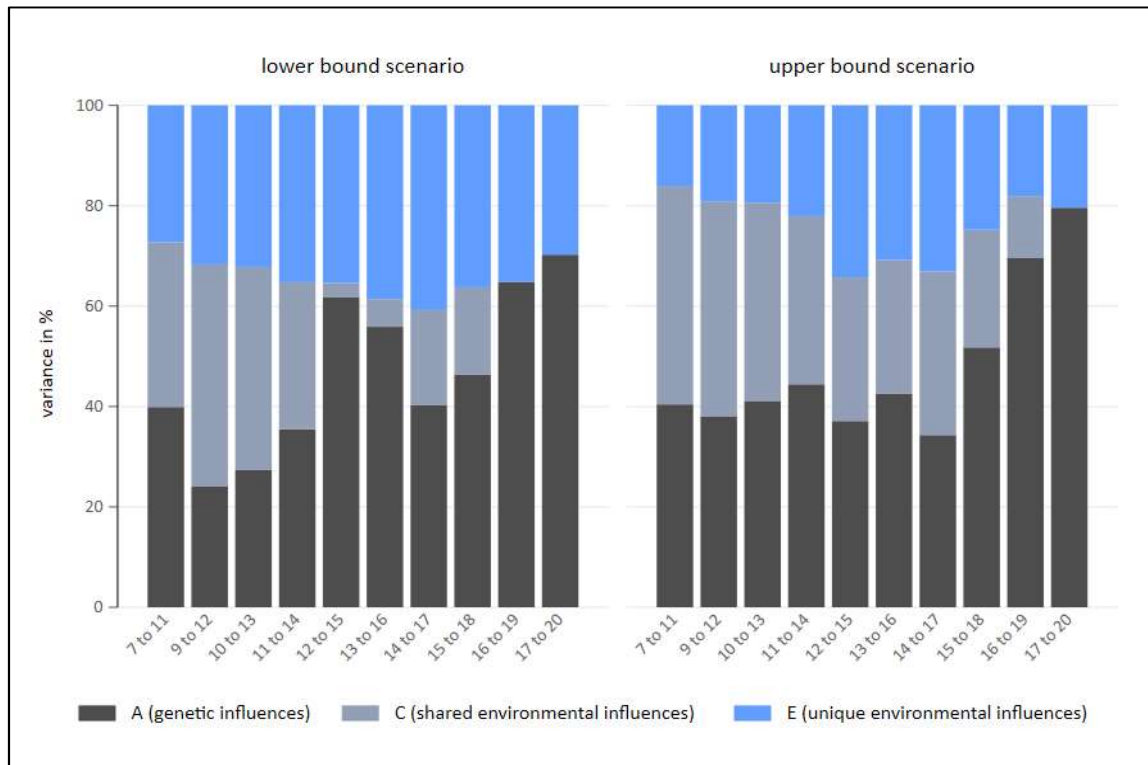
Note: The base refers to the unconditioned model, I refers to model 1 (parents' education is controlled for), II refers to model 2 (parents' education and assortative mating are controlled for), and III refers to model 3 (parents' education, assortative mating, and siblings are controlled for). Source: TwinLife wave 1; own calculations.

Now we turn to the results for the social stratification of shared environmental and genetic influences on education. The findings are visualized in Figure 3.2 (see Appendix Table A3.4 for additional information on the estimation results). The following subgroup-specific ACE decompositions are conditional on parental education. As a consequence, the variance related to the effect of parental education on children's educational attainment is excluded in these variance decompositions.⁸ Our results in models 1 to 3 have shown that this variance is mainly associated with shared environment influences. In line with theoretical expectations and our previous analyses (Table 3.3, model 1), twins' mean level of educational attainment increases continuously with parents' education in both scenarios. In the lower-bound scenario, we found that twins whose parents have the lowest level of education (7 to 11 years of education) have about 11.7 years of education. Twins whose parents have the highest level of education (17 to 20

⁸ Furthermore, we base our analysis for the subgroups by parental education on the assumption of random mating because the majority of assortative mating between parents based on education is already accounted for by conditioning on parental education.

years) have about 13.9 years of education. For the upper-bound scenario, mean values range from 12.7 to about 16.1 years. Overall, the total variances of educational attainment were quite stable across the subgroups.

Figure 3.2 ACE Variance Decompositions for Twins' Years of Education by Parents' Years of Education



Source: TwinLife wave 1; own calculations.

In the lower-bound scenario, shared environmental influences accounted for about one-third of the variation among twins whose parents have the lowest level of education. Shared environmental influences mattered most (about 44%) among twins whose parents have 9 to 12 years of education and were absent among twins whose parents have 16 years of education or more. By contrast, the relative importance of genetic influences was smallest (about 24%) among twins whose parents have 9 to 12 years of education and highest (about 70%) among twins whose parents have the highest level of education. Thus, the relative importance of shared environmental influences tended to be smaller among twins whose parents have more education, whereas the relative importance of genetic influences tended to be larger in these educational subgroups.

This pattern was even more pronounced in the upper-bound scenario: The relative importance of shared environmental influences was most relevant (about 44%) among twins whose parents have the lowest level of education, whereas shared environmental influences are absent for twins whose parents have the highest level of education. The relative importance of genetic influences was about 40% among twins whose parents have the lowest level of education and about 80% among twins whose parents have the highest level of education.

These findings support our second hypothesis: In both scenarios, shared environmental influences were most pronounced in families with lower levels of education, whereas genes were more important in families with higher levels of education. In neither the lower-bound nor the upper-bound scenario did we find shared environmental influences among twins whose parents are highly educated over and above shared environmental influences induced by parents' education. Interpreting these findings in conjunction with the mean levels of education, our results show that shared environmental influences in less-educated families are rather detrimental for educational attainment because, on average, twins attained lower levels of education than twins with highly educated parents where shared environmental influences were absent.

3.5 Conclusion and Discussion

In this article, we investigated how shared environmental and genetic influences affect educational attainment and tested the Scarr–Rowe hypothesis for educational attainment (i.e., we analyzed whether shared environmental and genetic influences are socially stratified).

Our theoretical explanations combine sociological perspectives with behavioral genetic approaches. Firstly, to explain the comparatively strong shared environmental influences on education compared to IQ, we use the framework of primary and secondary effects of social background on educational attainment (Boudon 1974). Because schooling decisions are socially stratified and operate over and above academic performance, we proposed that secondary effects explain a substantial part of the shared environmental influences on education. Secondly, to motivate the social stratification of gene expression proposed by the Scarr–Rowe hypothesis, we extend the sociological

literature on stratified parenting (e.g., Lareau 2011): We argue that advantaged parents provide learning environments that are more child centered and adapted to children's potential and needs. This individual adaptation of children's learning environment matters because it leads to better conditions for gene expression. In consequence, we expected the relative importance of genetic influences to be stronger in advantaged families and shared environmental influences to be more important in disadvantaged families. Furthermore, it is plausible that if learning environments are persistently socially stratified, then the different likelihoods of genetic potentials being realized are amplified as children get older (Dannefer 2003; DiPrete and Eirich 2006). Mechanisms of cumulative (dis-)advantage might therefore explain why differences in parenting can have a long-lasting impact on children's educational biographies.

Our results based on a socially stratified random sample of young adult twins show that shared environmental influences account for an average of about one-third of the total variation in education in Germany. These findings are in line with previous findings (Branigan, McCallum, and Freese 2013). The impact of shared environmental influences was driven partly by parents' education, which supports our first hypothesis on the importance of socially stratified educational choices. However, assortative mating also accounts for shared environmental influences on education in Germany. Thus, both parents' education and assortative mating are main explanatory mechanisms of shared environmental influences of education. Future research should, therefore, systematically account for assortative mating to avoid an overestimation of shared environmental influences on education.

Our findings on the social stratification of shared environmental and genetic influences provide evidence for the Scarr–Rowe hypothesis applied to education. For twins whose parents have low levels of education (up to 12 years), shared environmental influences accounted for about 40% of the total variation in educational attainment. By contrast, for twins whose parents are highly educated (more than 17 years), shared environmental influences were absent, and genetic influences accounted for about 75% of the total variation in educational attainment. These findings provide support for the impact of socially stratified family environments and different logics of parenting on gene expression with respect to educational attainment: More-educated parents provide rearing and learning environments that are more often in line with children's individual abilities and genetic make-up, helping them to realize their developmental potential. In disadvantaged families, environmental influences are less adapted to children's potential

and needs and hence less specific to children's genetic disposition. This can explain why genetic influences are much weaker, and thus why relative shared environmental influences are much stronger, in less-educated families. In addition, lower mean levels of education for young adults whose parents are less educated indicate that those shared environmental influences tend to be detrimental for children's education and constrain the realization of children's innate talents.

Our results relate to Germany, which has a highly stratified schooling system and a broad coverage of part-time schools. Both of these institutional facets imply a higher sensitivity to social background influences compared to educational systems that lack these features. Given these institutional features, the social stratification we found can be expected to be strong in comparison to other national contexts. Future research is needed to systematically assess the effects of cross-country differences in educational institutions on gene expression with regard to education and to examine to what extent the social stratification of environmental and genetic influences depends on factors such as the timing of tracking or the coverage of full-time schools.

It is important to keep in mind that the individuals we studied are predominantly still in education. We addressed the uncertainty by providing lower- and upper-bound estimates of environmental and genetic influences on education. These estimates are based on assumptions about the future educational career of the twins we analyzed, which can also influence our estimates of variance components. It is therefore important to replicate our results once twins finished their education. Nonetheless, results for both scenarios provided a clear pattern that supports the Scarr–Rowe hypothesis.

Furthermore, we did not explicitly analyze factors leading to primary and secondary effects (e.g., educational choices or aspirations, extracurricular activities, and parenting behaviors). Thus, more genetically informed research on these mechanisms is needed (e.g., to assess how parenting behaviors suppress or enhance children's potential to realize their genetic disposition).

Finally, two limitations that come along with the CTD need to be discussed: First, we cannot rule out that our findings are driven by systematic differences in genetic variation across the social strata. Such differences could stem from stratified assortative-mating patterns: If assortative mating is less pronounced among more-educated compared to less-educated parents, our findings on the relative importance of genetic influences on education would be upwardly biased (Conley et al. 2015). Although this argument is reasonable, current evidence based on PGS on education within sibling samples does not

support this assumption (Conley et al. 2015). Sibling analysis provides a powerful tool in this context because siblings are exposed to the same family background influences, whereas differences in their genes are random. The results show that the effect of PGS on education is smaller in models that examine children from different families (“between-family analysis”) compared to models that examine different children from the same family (“within-family analysis”). Thus, controlling for unobserved influences within the family, the effect of genetic endowment measured through PGS on educational attainment is stronger than without these controls. These results contradict the expectation that greater genetic variance among more-educated parents upwardly biases the findings on the relative importance of genes for educational attainment.

Second, we cannot rule out that gene-by-gene instead of gene-environment interactions are at work. In other words, it could be that genetic variants of children interact with genetic variants of parents that are associated with parents’ education. Such mechanisms can only be detected if genotyped data of parents and children are available. Such data are not available in Germany yet. However, additional analysis from the study of Conley et al. (2015) shows that parental genotype has not had an independent effect on children’s education if children’s PGS and parents’ schooling are controlled for.

In light of recent developments in molecular genetics, findings on the Scarr-Rowe hypothesis on education need to be replicated for Germany based on genotyped data. Nonetheless, we acknowledge the added value of the CTD. Molecular genetic studies are interested in scrutinizing the role of specific genetic variants, whereas behavioral genetic studies in general are also interested in the relative importance of overall genetic influences on individual variation. Complex traits, such as educational outcomes, are influenced by many different genetic and environmental factors, with each single factor having a rather small effect. Twin designs, however, facilitate the investigation of whole-genome effects –rather than specific genetic variants– across different social conditions. Importantly, these approaches are not mutually exclusive, and we believe that they fruitfully complement each other, which will significantly enhance our current understanding on how social and genetic influences shape individuals’ life chances.

Overall, our results provide evidence for socially stratified environmental and genetic influences on educational attainment in Germany based on a quantitative genetic design. We provided a theoretical account for the underlying mechanisms that are rooted in differences in the quality of the learning environment and related parenting. The social

stratification of learning environments shapes the realization of genetic predispositions and thus contributes to social disparities in educational attainment.

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Appendix

Table A3.1 Coding Scheme for Years of Education

| Level of Education | Years of Education |
|---|--------------------|
| General Education | |
| No diploma | 7 |
| Lower secondary (<i>Hauptschulabschluss</i>) | 9 |
| Intermediate secondary (<i>Realschulabschluss</i>) | 10 |
| Diploma to enter a professional college (<i>Fachhochschulreife</i>) | 12 |
| Upper secondary (<i>Abitur</i>) | 13 |
| Other general educational | 10 |
| + Occupational Training | |
| Apprenticeship | 1.5 |
| Technical schools (including health-care schools) | 2 |
| Civil servants' apprenticeship | 1.5 |
| Higher technical college | 3 |
| University degree | 5 |
| Ph.D. (coded only for parents of twins) | 7 |

Table A3.2 ACE Variance Decompositions for Twins' Years of Education –Without Imputation

| | Lower-Bound Scenario | | | Upper-Bound Scenario | | |
|---------------------------------------|----------------------|--------|----------|----------------------|--------|----------|
| | b/var | c.s.e. | z-value | b/var | c.s.e. | z-value |
| Base Model: | | | | | | |
| Assumed genetic DZ correlation | 0.50 | | | 0.50 | | |
| Constant | -0.10 | 0.07 | -1.53 | 1.38 | 0.09 | 15.59*** |
| Total variance | 5.26 | 0.23 | 23.00*** | 8.84 | 0.23 | 38.91*** |
| A in % | 43.57 | 7.97 | 5.47*** | 41.00 | 6.52 | 6.29*** |
| C in % | 26.59 | 7.14 | 3.73*** | 39.73 | 6.21 | 6.40*** |
| E in % | 30.32 | 2.64 | 11.47*** | 19.72 | 1.78 | 11.09*** |
| Model 1 (Parents' Education): | | | | | | |
| Assumed genetic DZ correlation | 0.50 | | | 0.50 | | |
| Parents' years of education | 0.26 | 0.02 | 12.86*** | 0.41 | 0.02 | 16.50*** |
| Constant | -0.17 | 0.06 | -2.72** | 1.28 | 0.08 | 15.82*** |
| Total variance | 5.27 | 0.23 | 22.99*** | 8.85 | 0.23 | 38.80*** |
| A in % | 43.06 | 7.74 | 5.19*** | 41.58 | 6.72 | 6.19*** |
| C in % | 15.17 | 6.88 | 2.20* | 21.90 | 6.24 | 3.51*** |
| E in % | 30.42 | 2.63 | 11.55*** | 19.72 | 1.78 | 11.09*** |
| Explained var. (R ²) in % | 11.35 | | | 16.81 | | |
| Model 2 (Assortative Mating): | | | | | | |
| Assumed genetic DZ correlation | 0.62 | | | 0.62 | | |
| Parents' years of education | 0.26 | 0.02 | 12.86*** | 0.41 | 0.02 | 16.50*** |
| Constant | -0.17 | 0.06 | -2.72** | 1.28 | 0.08 | 15.82*** |
| Total variance | 5.27 | 0.23 | 22.99*** | 8.85 | 0.23 | 38.80*** |
| A in % | 56.53 | 10.10 | 5.59*** | 54.59 | 8.81 | 6.20*** |
| C in % | 1.71 | 9.02 | 0.19 | 8.89 | 8.15 | 1.09 |
| E in % | 30.42 | 2.63 | 11.58*** | 19.72 | 1.78 | 11.09*** |
| Explained var. (R ²) in % | 11.35 | | | 16.81 | | |
| Model 3 (Sibling Effects): | | | | | | |
| Assumed genetic DZ correlation | 0.62 | | | 0.62 | | |
| Parents' years of education | 0.26 | 0.02 | 12.96*** | 0.41 | 0.02 | 16.60*** |
| Closeness of twins | 0.25 | 0.05 | 4.93*** | 0.29 | 0.07 | 4.32*** |
| Constant | -0.16 | 0.06 | -2.76** | 1.29 | 0.08 | 16.12*** |
| Total variance | 5.27 | 0.23 | 22.99*** | 8.85 | 0.23 | 38.80*** |
| A in % | 55.82 | 4.04 | 13.83*** | 56.47 | 8.81 | 6.41*** |
| C in % | --- | --- | --- | 5.38 | 8.14 | 0.66 |
| E in % | 30.81 | 2.50 | 12.35*** | 19.61 | 1.78 | 11.02*** |
| Explained var. (R ²) in % | 13.36 | | | 18.54 | | |

Note: All models besides the base model (N = 965) are based on N = 962 twin pairs and clustered standard errors are calculated at the twin pair level. Legend: *: $P(Z > |z|) < .05$; **: $P(Z > |z|) < .01$; ***: $P(Z > |z|) < .001$ (two-tailed tests). Source: TwinLife wave 1; own calculations.

Table A3.3 ACE Variance Decompositions for Twins' Years of Education by Parents' Years of Education –Without Imputation

| | Lower-Bound Scenario | | | Upper-Bound Scenario | | |
|-------------------------------------|----------------------|--------|-----------|----------------------|--------|----------|
| | b/var | c.s.e. | z-value | b/var | c.s.e. | z-value |
| Parents' Years of Education: | | | | | | |
| ≥ 7 to ≤ 11 | | | | | | |
| Constant | -1.26 | 0.12 | -10.45*** | -0.30 | 0.18 | -1.70 |
| Total variance | 3.98 | 0.35 | 11.35*** | 8.07 | 0.56 | 14.45*** |
| A in % | 33.49 | 18.86 | 1.78 | 40.08 | 15.64 | 2.56** |
| C in % | 33.37 | 16.40 | 2.03* | 41.16 | 15.31 | 2.69** |
| E in % | 33.14 | 6.94 | 4.78*** | 18.76 | 3.46 | 5.42*** |
| N _{twin pairs} | 219 | | | 219 | | |
| ≥ 9 to ≤ 12 | | | | | | |
| Constant | -0.61 | 0.09 | -6.61*** | 0.52 | 0.12 | 4.23*** |
| Total variance | 4.93 | 0.32 | 15.25*** | 8.57 | 0.35 | 24.49*** |
| A in % | 23.04 | 12.79 | 1.80 | 38.01 | 9.74 | 3.90*** |
| C in % | 45.35 | 12.08 | 3.76*** | 42.38 | 9.42 | 4.50*** |
| E in % | 31.61 | 4.01 | 7.88*** | 19.60 | 2.52 | 7.79*** |
| N _{twin pairs} | 484 | | | 484 | | |
| ≥ 10 to ≤ 13 | | | | | | |
| Constant | -0.45 | 0.09 | -4.96*** | 0.76 | 0.12 | 6.20*** |
| Total variance | 4.81 | 0.32 | 15.14*** | 8.41 | 0.34 | 25.00*** |
| A in % | 27.39 | 13.57 | 2.02* | 43.32 | 10.24 | 4.23*** |
| C in % | 40.03 | 12.55 | 3.19*** | 36.85 | 9.84 | 3.74*** |
| E in % | 32.59 | 4.25 | 7.67*** | 19.84 | 2.64 | 7.51*** |
| N _{twin pairs} | 474 | | | 474 | | |
| ≥ 11 to ≤ 14 | | | | | | |
| Constant | -0.17 | 0.10 | -1.78 | 1.13 | 0.13 | 8.76*** |
| Total variance | 4.88 | 0.34 | 14.35*** | 8.05 | 0.34 | 23.75*** |
| A in % | 41.34 | 15.38 | 2.69** | 46.63 | 11.66 | 4.00*** |
| C in % | 25.02 | 13.55 | 1.85 | 30.81 | 10.84 | 2.84** |
| E in % | 33.64 | 45.76 | 7.35*** | 22.56 | 3.14 | 7.18*** |
| N _{twin pairs} | 400 | | | 400 | | |
| ≥ 12 to ≤ 15 | | | | | | |
| Constant | 0.19 | 0.10 | 1.89 | 1.68 | 0.13 | 12.95*** |
| Total variance | 4.32 | 0.30 | 14.26*** | 6.94 | 0.29 | 23.92*** |
| A in % | 63.17 | 7.92 | 7.97*** | 37.05 | 16.36 | 2.26* |
| C in % | --- | --- | --- | 25.88 | 14.01 | 1.85 |
| E in % | 36.83 | 4.83 | 7.63*** | 37.08 | 5.14 | 7.21*** |
| N _{twin pairs} | 316 | | | 316 | | |
| ≥ 13 to ≤ 16 | | | | | | |
| Constant | 0.20 | 0.11 | 1.89 | 1.74 | 0.14 | 12.37*** |
| Total variance | 4.40 | 0.33 | 13.26*** | 7.08 | 0.34 | 20.67*** |
| A in % | 57.92 | 8.47 | 6.84*** | 43.94 | 16.81 | 2.61** |
| C in % | --- | --- | --- | 22.36 | 14.34 | 1.56 |
| E in % | 42.08 | 5.68 | 7.41*** | 33.70 | 5.31 | 6.35*** |
| N _{twin pairs} | 275 | | | 275 | | |

Table continued on next page

| | | | | | | |
|-------------------------|-------|-------|----------|-------|-------|----------|
| ≥ 14 to ≤ 17 | | | | | | |
| Constant | 0.27 | 0.12 | 2.22* | 1.88 | 0.16 | 11.74*** |
| Total variance | 4.38 | 0.38 | 11.66*** | 7.11 | 0.38 | 18.71*** |
| A in % | 38.64 | 23.00 | 1.68 | 33.84 | 19.85 | 1.70 |
| C in % | 15.39 | 19.91 | 0.77 | 29.03 | 16.97 | 1.71 |
| E in % | 45.97 | 6.96 | 6.60*** | 37.13 | 62.93 | 5.90*** |
| N _{twin pairs} | 215 | | 215 | | | |
| ≥ 15 to ≤ 18 | | | | | | |
| Constant | 0.67 | 0.11 | 5.84*** | 2.74 | 0.14 | 20.03*** |
| Total variance | 4.92 | 0.36 | 13.65*** | 6.60 | 0.43 | 15.23*** |
| A in % | 44.10 | 19.88 | 2.22* | 58.96 | 17.95 | 3.28*** |
| C in % | 16.46 | 16.91 | 0.97 | 14.46 | 16.34 | 0.89 |
| E in % | 39.44 | 6.10 | 6.47*** | 26.58 | 4.51 | 5.90*** |
| N _{twin pairs} | 279 | | 279 | | | |
| ≥ 16 to ≤ 19 | | | | | | |
| Constant | 0.78 | 0.14 | 5.67*** | 3.05 | 0.16 | 18.80*** |
| Total variance | 5.06 | 0.43 | 11.74*** | 6.39 | 0.58 | 11.02*** |
| A in % | 61.65 | 24.87 | 2.48** | 82.43 | 10.06 | 8.19*** |
| C in % | 0.34 | 20.28 | 0.02 | --- | --- | --- |
| E in % | 38.01 | 7.62 | 4.99*** | 17.57 | 4.10 | 4.29*** |
| N _{twin pairs} | 194 | | 169 | | | |
| ≥ 17 to ≤ 20 | | | | | | |
| Constant | 1.09 | 0.15 | 7.26*** | 3.48 | 0.15 | 22.88*** |
| Total variance | 5.29 | 0.42 | 12.54*** | 5.30 | 0.62 | 8.61*** |
| A in % | 70.43 | 10.42 | 6.76*** | 78.39 | 12.88 | 6.09*** |
| C in % | --- | --- | --- | --- | --- | --- |
| E in % | 29.57 | 6.84 | 4.32*** | 21.61 | 5.39 | 4.01*** |
| N _{twin pairs} | 175 | | 175 | | | |

Note: Clustered standard errors are calculated at the twin pair level. Legend: *: $P(Z > |z|) < .05$; **: $P(Z > |z|) < .01$; ***: $P(Z > |z|) < .001$ (two-tailed tests). Source: TwinLife wave 1; own calculations.

Table A3.4 ACE Variance Decompositions for Twins' Years of Education by Parents' Years of Education

| | Lower-Bound Scenario | | | Upper-Bound Scenario | | |
|------------------------------------|----------------------|--------|----------|----------------------|--------|----------|
| | b/var | c.s.e. | z-value | b/var | c.s.e. | z-value |
| Parents' Years of Education | | | | | | |
| ≥ 7 to ≤ 11 | | | | | | |
| Constant | -1.32 | 0.16 | -8.4*** | -0.35 | 0.23 | -1.51 |
| Total variance | 4.05 | 0.45 | 8.94*** | 8.11 | 0.73 | 11.09*** |
| A in % | 39.90 | 21.87 | 1.82 | 40.42 | 18.23 | 2.22* |
| C in % | 32.85 | 19.74 | 1.66 | 43.52 | 18.01 | 2.42** |
| E in % | 27.25 | 7.44 | 3.66*** | 16.06 | 4.34 | 3.70*** |
| N _{twin pairs} | 160 | | | 160 | | |
| ≥ 9 to ≤ 12 | | | | | | |
| Constant | -0.69 | 0.1 | -6.74*** | 0.44 | 0.14 | 3.18** |
| Total variance | 4.87 | 0.36 | 13.37*** | 8.56 | 0.4 | 21.33*** |
| A in % | 24.15 | 14.35 | 1.68 | 37.94 | 10.66 | 3.56*** |
| C in % | 44.16 | 13.58 | 3.25** | 42.93 | 10.45 | 4.11*** |
| E in % | 31.69 | 4.51 | 7.03*** | 19.13 | 2.72 | 7.03*** |
| N _{twin pairs} | 434 | | | 434 | | |
| ≥ 10 to ≤ 13 | | | | | | |
| Constant | -0.58 | 0.1 | -5.84*** | 0.60 | 0.13 | 4.47*** |
| Total variance | 4.76 | 0.36 | 13.34*** | 8.44 | 0.38 | 21.93*** |
| A in % | 27.34 | 14.67 | 1.86 | 41.12 | 11.03 | 3.73*** |
| C in % | 40.45 | 13.82 | 2.93** | 39.49 | 10.75 | 3.67*** |
| E in % | 32.21 | 4.58 | 7.04*** | 19.39 | 2.83 | 6.86*** |
| N _{twin pairs} | 443 | | | 443 | | |
| ≥ 11 to ≤ 14 | | | | | | |
| Constant | -0.32 | 0.1 | -3.07** | 0.94 | 0.14 | 6.55*** |
| Total variance | 4.75 | 0.36 | 13.06*** | 8.14 | 0.37 | 21.9*** |
| A in % | 35.49 | 15.86 | 2.24* | 44.44 | 12.12 | 3.67*** |
| C in % | 29.28 | 13.89 | 2.11* | 33.47 | 11.54 | 2.90** |
| E in % | 35.23 | 5.35 | 6.58*** | 22.09 | 3.37 | 6.55*** |
| N _{twin pairs} | 399 | | | 399 | | |
| ≥ 12 to ≤ 15 | | | | | | |
| Constant | 0.06 | 0.11 | 0.52 | 1.50 | 0.15 | 10.1*** |
| Total variance | 4.40 | 0.35 | 12.55*** | 7.29 | 0.36 | 20.39*** |
| A in % | 61.83 | 18.96 | 3.26*** | 37.11 | 16.33 | 2.27* |
| C in % | 2.79 | 16.81 | 0.17 | 28.63 | 14.41 | 1.99* |
| E in % | 35.37 | 5.37 | 6.59*** | 34.26 | 4.96 | 6.90*** |
| N _{twin pairs} | 331 | | | 331 | | |

Table continued on next page

| | | | | | | |
|-------------------------|-------|-------|----------|-------|-------|----------|
| ≥ 13 to ≤ 16 | | | | | | |
| Constant | 0.10 | 0.11 | 0.88 | 1.58 | 0.15 | 10.34*** |
| Total variance | 4.47 | 0.38 | 11.9*** | 7.39 | 0.41 | 17.89*** |
| A in % | 55.87 | 19.79 | 2.82** | 42.59 | 16.5 | 2.58** |
| C in % | 5.53 | 16.69 | 0.33 | 26.62 | 14.58 | 1.83 |
| E in % | 38.60 | 6.26 | 6.17*** | 30.79 | 5.14 | 5.99*** |
| N _{twin pairs} | 309 | | | 309 | | |
| ≥ 14 to ≤ 17 | | | | | | |
| Constant | 0.17 | 0.12 | 1.34 | 1.71 | 0.17 | 9.99*** |
| Total variance | 4.48 | 0.42 | 10.63*** | 7.43 | 0.45 | 16.66*** |
| A in % | 40.22 | 23.93 | 1.68 | 34.32 | 19.08 | 1.80 |
| C in % | 18.97 | 21.24 | 0.89 | 32.55 | 16.61 | 1.96* |
| E in % | 40.81 | 6.8 | 6.00*** | 33.13 | 5.99 | 5.53*** |
| N _{twin pairs} | 247 | | | 247 | | |
| ≥ 15 to ≤ 18 | | | | | | |
| Constant | 0.53 | 0.12 | 4.62*** | 2.47 | 0.15 | 16.7*** |
| Total variance | 5.02 | 0.37 | 13.48*** | 7.30 | 0.46 | 15.95*** |
| A in % | 46.36 | 19.16 | 2.42* | 51.77 | 16.73 | 3.09*** |
| C in % | 17.38 | 16.68 | 1.04 | 23.41 | 15.18 | 1.54 |
| E in % | 36.26 | 5.76 | 6.30*** | 24.82 | 4.42 | 5.61*** |
| N _{twin pairs} | 326 | | | 326 | | |
| ≥ 16 to ≤ 19 | | | | | | |
| Constant | 0.63 | 0.14 | 4.5*** | 2.72 | 0.18 | 15.42*** |
| Total variance | 5.16 | 0.44 | 11.83*** | 7.23 | 0.59 | 12.15*** |
| A in % | 64.83 | 9.75 | 6.65*** | 69.55 | 19.45 | 3.58*** |
| C in % | --- | --- | --- | 12.34 | 18.29 | 0.67 |
| E in % | 35.17 | 6.52 | 5.39 | 18.11 | 4.26 | 4.26*** |
| N _{twin pairs} | 231 | | | 231 | | |
| ≥ 17 to ≤ 20 | | | | | | |
| Constant | 0.93 | 0.15 | 6.02*** | 3.14 | 0.18 | 17.59*** |
| Total variance | 5.41 | 0.44 | 12.31*** | 6.24 | 0.67 | 9.31*** |
| A in % | 70.25 | 9.94 | 7.07*** | 79.60 | 10.49 | 7.59*** |
| C in % | --- | --- | --- | --- | --- | --- |
| E in % | 29.75 | 6.61 | 4.50*** | 20.40 | 5.15 | 3.96*** |
| N _{twin pairs} | 209 | | | 209 | | |

Source: TwinLife wave 1; own calculations. Clustered standard errors are calculated at the twin pair level.
Legend: *: $P(Z > |z|) < .05$; **: $P(Z > |z|) < .01$; ***: $P(Z > |z|) < .001$ (two-tailed tests).

4. Genetic Effects on Educational Success in Cross-National Perspective

Tina Baier, Volker Lang, Michael Grätz, Kieron J. Barclay, Dalton Conley, Thomas Laidley, and Torkild H. Lyngstad

Abstract

Both social and genetic influences contribute to differences in education. Yet, we know little about their variation according to social conditions. Previous research has emphasized the role of the proximate family and tends to neglect that the broader institutional environment can also shape genetic effects on education. To account for interdependencies between the family and macro-level influences, we adopt a comparative perspective. Specifically, we ask first, whether genetic effects on educational success differ in Germany, Sweden, and the United States, and second, whether genetic effects vary by parents' social background. We hypothesize that genetic effects on educational success are stronger in more egalitarian educational systems and more generous welfare regimes, while the social stratification of genetic effects should be less pronounced in these countries. We focus on two indicators of educational success: educational attainment, indicated by years of education, and educational achievement, indicated by school grades. We use large-scale observational twin data for Germany and the United States –TwinLife and Add Health– and register data for Sweden. Our results based on genetically sensitive variance decomposition models show that genetic effects on educational success are least pronounced in Germany and most pronounced in Sweden. Evidence for differences in genetic effects according to social background is weak. However, we find indications for a social stratification of genetic effects for educational success in Germany and the United States. Our findings therefore suggest that more egalitarian educational systems have a positive effect on the development of genetic potential for educational success.

4.1 Introduction

That genetic and social influences shape individuals' chances for education is well established in the literature (Branigan, McCallum, and Freese 2013; Freese and Jao 2017; Nielsen 2016; Nielsen and Roos 2015). However, their relative importance can vary according to social conditions (Baier and Lang 2019; Domingue et al. 2015). Social conditions are important for the realization of genetic potential since human development takes place in constant exchange with conditions provided by the proximate environment, such as the family (Bronfenbrenner and Ceci 1994). Yet, not only the proximate environment but also more distal environments, such as educational systems and welfare regimes, can shape genetic effects on education (Diewald 2016b; Selita and Kovas 2019). Generous welfare states protect against major life risks and provide comparatively high levels of social security. Such contexts grant, on average, higher living standards and more equal access to relevant resources. By contrast, in less developed welfare states, access to relevant resources is more restricted, which can lower the realization of genetic potential. Likewise, stratified schooling systems limit access to enriched learning environments, which can lead to untapped genetic potentials for education. Though differences among countries and their implication for genetic effects on education have been discussed in the literature, cross-country differences have not yet been systematically studied (Diewald 2016a; Selita and Kovas 2019, Tucker-Drob and Bates 2016).

To date, research on gene–environment interactions has focused on cognitive skills, such as IQ, and the role of parents' socioeconomic standing. This line of research is motivated by the Scarr–Rowe hypothesis claiming that the relative importance of genes relevant for the development of cognitive ability is positively associated with parents' social background (Rowe, Jacobson, and van den Oord 1999; Scarr-Salapatek 1971). The mechanism supposed to bring about a Scarr–Rowe interaction (SRI) is that socioeconomically advantaged families provide environmental conditions that match children's genetic dispositions, while there are fewer developmental opportunities in socioeconomically disadvantaged families, thus hindering the realization of genetic potentials.

We extend previous research on gene–environment interactions in two crucial ways: First, we acknowledge that both the proximal environment, as well as the broader institutional environment, can shape genetic effects on education. Second, we shift the

initial focus of the Scarr–Rowe hypothesis from cognitive skills to educational success. While cognitive ability is a major predictor for educational attainment, educational attainment is not only affected by cognitive skills but also driven by socially stratified schooling choices (Boudon 1974; Breen and Goldthorpe 1997; Erikson and Jonsson 1996). To understand the reproduction of social inequalities, it is therefore important to extend this line of research to other characteristics related to social stratification. We address this research gap by focusing on school grades and educational attainment that are directly linked to social mobility.

Previous research on SRIs related to cognitive skills reveals differences across countries. Several studies found evidence for larger genetic effects on IQ in socioeconomically advantaged compared with disadvantaged families in the United States (Bates, Lewis, and Weiss 2013; Kirkpatrick, McGue, and Iacono 2015; Rhemtulla and Tucker-Drob 2012; Schwartz 2015; Tucker-Drob et al. 2011; Turkheimer et al. 2003). One study found socioeconomic differences for Sweden (Fischbein 1980) as did one for Germany (Gottschling et al. 2019). However, there is also conflicting evidence for the United States (Figlio et al. 2017; Grant et al. 2010), and also studies for the United Kingdom (Asbury, Wachs, and Plomin 2005; Hanscombe et al. 2012), the Netherlands (van der Sluis et al. 2008), and Australia (Bates et al. 2016) found no evidence for socioeconomic differences in the effects of genes on cognitive skills. An international meta-analysis, however, found support for a SRI in the United States but not in Australia, Germany, the Netherlands, and Sweden (Tucker-Drob and Bates 2016). In sum, research on IQ points to cross-country variations in regards to the SRI, but the results are partly conflicting.

To date, few studies have tested the Scarr–Rowe hypothesis for education (Baier and Lang 2019; Conley et al. 2015; Domingue et al. 2015). A recent study for Germany showed that genetic influences on educational attainment were more important in highly than in less educated families (Baier and Lang 2019). This study, in line with ours and the most research on the Scarr–Rowe hypothesis, applied genetically sensitive variance decompositions, that is, ACE models, to identify the effects of genes on educational attainment. In this approach, genes are not directly measured, but their influences are inferred via knowledge of the degree of relatedness of the individuals under study.

Other studies tested the Scarr–Rowe hypothesis for educational attainment in the United States using direct measures of genes (Conley et al. 2015; Domingue et al. 2015). These studies used polygenic scores and analyzed whether their predictive power for

educational attainment differed by parental education. Polygenic scores are based on genotyped data and estimate the cumulative impact of measured genes on educational outcomes. The findings of these studies remained inconclusive: one study found no evidence for a social stratification of genetic effects (Conley et al. 2015), while the other found that the effect of polygenic scores on education decreased with parents' education (Domingue et al. 2015). Since the predictive power of polygenic scores on educational attainment has further increased over the time since these studies were conducted, future research with larger sample sizes and improved polygenic scores may lead to more conclusive results. We believe that behavioral genetic and molecular approaches are complementary and that both strategies are needed to obtain a better understanding of how genetic influences affect education within and across countries.

Overall, the current state of research on the Scarr–Rowe hypothesis is characterized by a lack of systematic cross-country comparisons and a narrow focus on cognitive skills. We address these gaps by investigating genetic effects on educational success and their social stratification from an international comparative perspective using the same analytic approach and similar definitions of variables for all countries included in our study.

4.2 Current Study

We study genetic effects on educational achievement (school grades) and educational attainment (years of education). In light of the conceptual framework of primary and secondary effects of social background on education (Boudon 1974; Breen and Goldthorpe 1997; Erikson and Jonsson 1996), we expect that the role of genes differs for educational achievement and educational attainment. Primary effects describe parents' efforts to improve children's educational performance. Parents actively foster the development of cognitive and noncognitive skills and provide various goods and services to enhance school-related skills, such as extra learning material and/or private tutoring. Secondary effects, by contrast, refer to stratified schooling choices over and above children's educational performance. Parents' educational decisions are determined by the anticipated costs, benefits, likelihood of success, and importantly, the intention to avoid downward mobility (Breen and Goldthorpe 1997). Consequently, parents from higher social backgrounds opt more often for higher educational tracks than parents from lower

social backgrounds who maintain their status with lower levels of education. Since parents' schooling decisions are to some extent independent of children's genetic potential for educational success, genetic effects should be stronger for educational achievement compared to educational attainment.

Our expectations about cross-country differences in genetic influences on educational success and their social stratification are rooted in different types of educational systems, their welfare states, and the related degrees of social inequality (Esping-Andersen 1990). These macro-level differences have also been used to explain international differences in the heritability of educational outcomes and to motivate comparative studies on the Scarr-Rowe hypothesis for cognitive skills (Selita and Kovas 2019; Tucker-Drob and Bates 2016). Following these criteria, we selected a sample of three advanced, industrialized societies for our cross-national analysis: Germany, Sweden, and the United States.

First, differences in the effects of genes and their stratification may be a consequence of differently structured educational systems. Here, we focus on tracking, which comprises the formal selection of students, based on their academic ability, and placing them in different schools, classes or set of courses. Tracking is a common characteristic of Western educational systems. However, differences exist in regard to the timing. The German educational system assigns children as young as 10 to 12 years of age to one of three hierarchically structured secondary schooling tracks. By contrast, Sweden and the United States have longer periods of comprehensive schooling and less strict tracking (Bol et al. 2014). In the United States, there is, however, a high degree of internal tracking (Lucas 1999). Different secondary school tracks represent different learning environments, since children are grouped by early ability, which is more closely related to social origin than ability at a later age. Since tracking in Germany takes place at an exceptionally young age of the children and is strongly linked to social background (e.g., Breen and Jonsson 2005; Dustmann 2004; Hillmert and Jacob 2010; Müller et al. 1993; Shavit and Blossfeld 1993), we expect that genetic effects on educational success are comparably small in Germany, while the social stratification of genetic effects should be comparably strong.

Second, differences in genetic effects on education may be rooted in the welfare state and, particularly, the way social security is institutionalized. Liberal welfare states such as the United States provide only limited social security structures (DiPrete 2002; DiPrete and McManus 2000; Esping-Andersen 1990). Disadvantaged parents in liberal welfare states may face more severe economic hardship and are exposed to higher levels of stress

compared to Germany and Sweden where individuals are protected against major life risks (Diewald 2016b). Both resource restrictions and stress may lower parents' capacity to provide rearing environments and inputs tailored to their children's genetic endowments. This, in turn, decreases children's chances to develop their genetic potential. In consequence, we expect genetic effects on educational success in the United States to be comparably small. Since access to relevant resources is dependent on individuals' socioeconomic standing, we also expect the stratification of genetic influences to be comparably strong in the United States.

Overall, we hypothesize that the genetic effects on educational success are smaller in Germany and the United States than in Sweden. Furthermore, we expect that the impact of parents' socioeconomic status on children's chances to realize genetic potential relevant for education is stronger in Germany and in the United States than in Sweden. In Germany, the social stratification of genetic effects should be more pronounced because of the early tracking system, and in the United States, because of the meager role of the welfare state.

To test these expectations, we use large-scale observational twin data for Germany (German Twin Family Panel [TwinLife]) (Diewald et al. 2018) and for the United States (National Longitudinal Study of Adolescent Health [Add Health]) (Harris et al. 2013), as well as register data on twins for Sweden (Statistics Sweden 2011). The birth cohorts of the twins in the different samples range from years 1975 to 1993. The datasets are described in greater detail in Appendix section 4.A, and Table 4.D.1 provides an overview of the analytical samples.

Our outcomes of interest are measured as follows: As measure for school grades we use grade point averages at age 16 in Sweden (i.e., the end of comprehensive schooling). In Germany and the United States we use final grade point average from secondary schooling. For years of education, we use a harmonized measure across countries (see Appendix Table 4.D.2 for a description). We differentiate in all countries between basic education, upper secondary education (vocational track), upper secondary education (academic track), post-secondary non-tertiary education, and tertiary level, and assign 9, 11, 12, 14, and 15.5 years for the corresponding educational levels. Since less differentiated measures of outcomes tend to lower estimates of genetic influences in behavioral genetic variance decompositions (Freese and Jao 2017), such a harmonization across countries, is necessary for the substantive interpretation of our results. We z-standardize all outcomes used in our analyses. Further details on the variables are reported

in Appendix 4.B. Summary statistics on the variables are provided in the Appendix Tables 4.D.3–4.D.5.

We analyze twin data from the different countries using genetically sensitive variance decomposition models (ACE models) based on the classical twin design (CTD) (Lang 2017; Plomin et al. 2008; Rabe-Hesketh, Skrondal, and Gjessing 2008). Twins are born at the same time; dizygotic (DZ) twins share 50% of the DNA, while monozygotic (MZ) twins are genetically identical. This information can be used to divide the total variance of an outcome into variances attributable to additive genetic influences (A), to shared environmental influences (C), and to unique environmental influences including measurement error (E). For Sweden twins' zygosity (whether a twin is mono- or dizygotic) was unknown. Here we use twins' sex to approximate zygosity. Twin pairs who are of opposite sex are dizygotic. Same-sex twin pairs can be both –monozygotic or dizygotic. For our analysis, we classify all same-sex twins as MZ twins. Due to the over classification of MZ twins in the Swedish sample, we apply an adjustment for using sex as a proxy (see section 4.C).

The standard ACE model assumes that spouses mate randomly in regard to the outcome under study. Given that assortative mating based on education is a well-established phenomenon across Western societies (e.g., Blossfeld 2009), we adjust our estimates for assortative mating (Loehlin, Harden, and Turkheimer 2009) (see Appendix section 4.C). To test for systematic differences in genetic effects, we estimate ACE models separately for different groups by parents' social background.¹ This analytical strategy is known as nonparametric gene–environment interaction analysis (Guo and Wang 2002). ACE models have a long research tradition in studies on SRIs (Asbury et al. 2005; Baier and Lang 2019; Bates et al. 2013; Figlio et al. 2017; Fischbein 1980; Grant et al. 2010; Guo and Stearns 2002; Harden, Turkheimer, and Loehlin 2007; Kirkpatrick et al. 2015; Schwartz 2015; van der Sluis et al. 2008; Tucker-Drob et al. 2011; Turkheimer et al. 2003). Applying these techniques makes our analyses comparable to those in this body of literature. Further details on the methods are reported in the Appendix section 4.C.

¹ The subgroup analyses are based on the assumption of random mating because the stratification accounts already for a large part of assortative mating (see also Baier and Lang (2019)).

4.3 Results

Table 4.1 shows the means and variances for school grades and years of education for each country. The mean value for grades differs only slightly across countries, and is smaller in Germany than in Sweden and the United States. The mean for years of education, however, is higher in Germany, followed by Sweden and the United States.

Table 4.1 ACE Variance Decomposition Results for Twins' School Grades and Years of Education –Adjusted for Assortative Mating

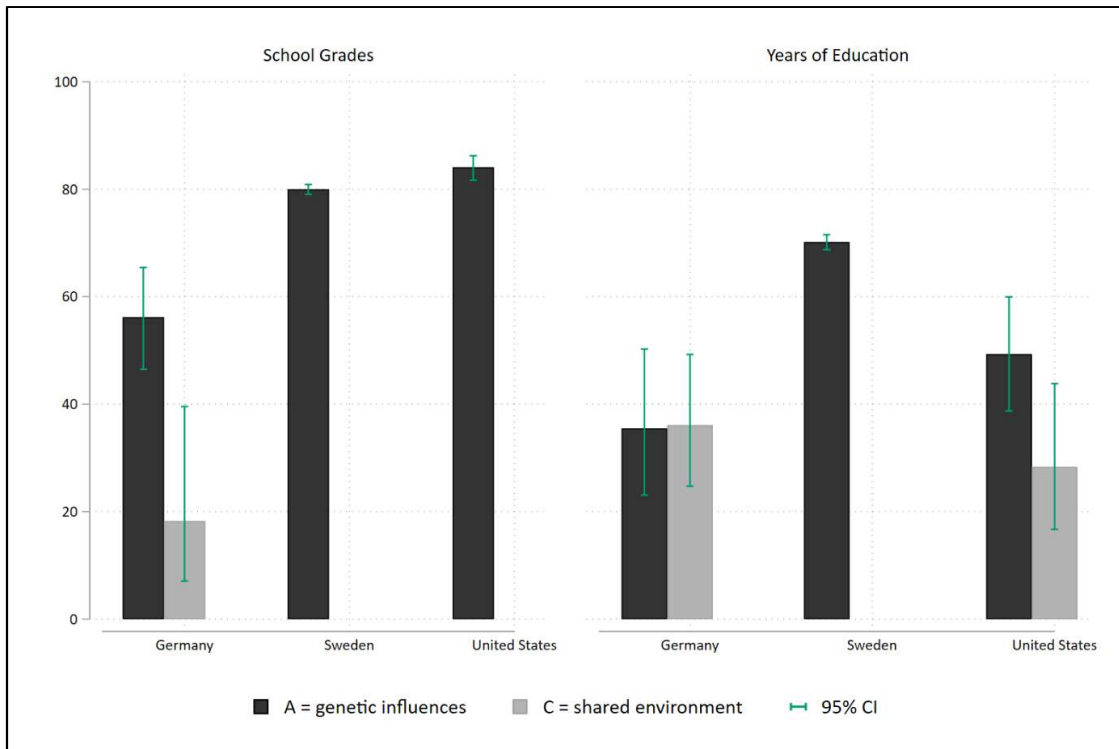
| | Germany | | Sweden | | United States | |
|---------------------------|---------|-------|--------|-------|---------------|-------|
| | b/var | c.s.e | b/var | c.s.e | b/var | c.s.e |
| School Grades | | | | | | |
| Means | 0.00 | 0.03 | 0.06 | 0.01 | 0.04 | 0.05 |
| Total Var. | 1.00 | 0.04 | 1.02 | 0.02 | 1.03 | 0.07 |
| N(Pairs) | 849 | | 6510 | | 364 | |
| Years of Education | | | | | | |
| Means | 0.86 | 0.08 | 0.49 | 0.03 | 0.32 | 0.06 |
| Total Var. | 3.78 | 0.16 | 2.62 | 0.06 | 2.03 | 0.13 |
| N(Pairs) | 956 | | 3873 | | 539 | |

Notes: Clustered standard errors are calculated at the twin pair level. Sources: Add Health, Swedish Registers, and TwinLife.

Figure 4.1 shows how genetic influences (A) and shared environmental influences (C) contribute to differences in school grades and years of education in Germany, Sweden, and the United States.² Detailed information on the results is reported in the Appendix Table 4.D.6, Figure 4.E.1 in the Appendix visualizes the results without adjustment for assortative mating.

² For the sake of clarity, we present the results for the A and C components. If the percentage of the C component was close to zero (smaller than 3%), we estimated AE models instead of ACE models. In these cases, the graphs only display the A component.

Figure 4.1 ACE Variance Decompositions Results for Twins’ School Grades and Years of Education –Adjusted for Assortative Mating



Sources: Add Health, Swedish Registers, and TwinLife; own calculations.

Independent of country, we find that genetic influences contribute substantially to differences in grades and, as expected, to a lesser extent to differences in years of education.

For grades, we find that genes account for more than half of the total variation in Germany, and in Sweden and the United States for about 80%. In comparison, genetic influences on school grades are least pronounced in Germany. In addition, shared environmental influences matter only in Germany and account for about one-fifth of the total variation in grades.

For years of education, genes and shared environmental influences contribute to equal shares (i.e., about 35%) in Germany. In Sweden, genes account for about 70%, while shared environmental influences are again absent. In the United States, genetic influences exceed the relative importance of shared environmental influences (50% vs. 30%). In comparison, genetic influences on years of education are more important in Sweden than in Germany and the United States.

With the exception of Sweden, findings of the substantial impact of shared environmental influences on education are in line with the findings based on an international meta-analysis (Branigan, McCallum, and Freese 2013). Different findings

for Sweden could be explained by the fact that previous research did not adjust for assortative mating, and also by differences in the samples under study (this is outlined in greater detail in the Discussion and Conclusion section).

Next, we test for stratified genetic effects for school grades in the three countries. As an indicator for social background, we used parents' occupation based on the Erikson-Goldthorpe-Portocarero (EGP) class scheme (Ganzeboom and Treiman 1996).³ Higher EGP classes are indicated by lower numbers (see Appendix 4.B). Table 4.2 displays the means and variances of school grades by parents' EGP class for each country. The results show that, in all countries, children from higher EGP classes have, on average, better grades and that the total variances for school grades are smaller in higher EGP classes.

Table 4.2 Means and Total Variances of Twins' School Grades by Parents' EGP Class

| | Germany | | Sweden | | United States | |
|---------------------------|----------------|-------|---------------|-------|-----------------------------|-------|
| | b/var | c.s.e | b/var | c.s.e | b/var | c.s.e |
| EGP V–VII or non-employed | | | | | EGP III–VII or non-employed | |
| Mean | –0.36 | 0.07 | –0.24 | 0.02 | –0.20 | 0.07 |
| Total var. | 1.13 | 0.09 | 1.06 | 0.04 | 1.10 | 0.09 |
| N(Pairs) | 231 | | 2542 | | 181 | |
| EGP III–IV | | | | | | |
| Mean | –0.16 | 0.07 | 0.03 | 0.02 | | |
| Total var. | 1.08 | 0.09 | 0.90 | 0.05 | | |
| N(Pairs) | 238 | | 1256 | | | |
| EGP II | | | | | EGP I–II | |
| Mean | 0.16 | 0.06 | 0.26 | 0.02 | 0.27 | 0.06 |
| Total var. | 0.89 | 0.08 | 0.87 | 0.04 | 0.78 | 0.06 |
| N(Pairs) | 213 | | 1703 | | 183 | |
| EGP I | | | | | | |
| Mean | 0.34 | 0.06 | 0.50 | 0.03 | | |
| Total var. | 0.62 | 0.07 | 0.84 | 0.06 | | |
| N(Pairs) | 167 | | 1009 | | | |

Notes: Clustered standard errors are calculated at the twin pair level. Sources: Add Health, Swedish Registers, and TwinLife; own calculations.

³ We used EGP since this information is available for all countries, and we intend to measure another dimension of social background related to parents' occupation and the economic situation of the family.

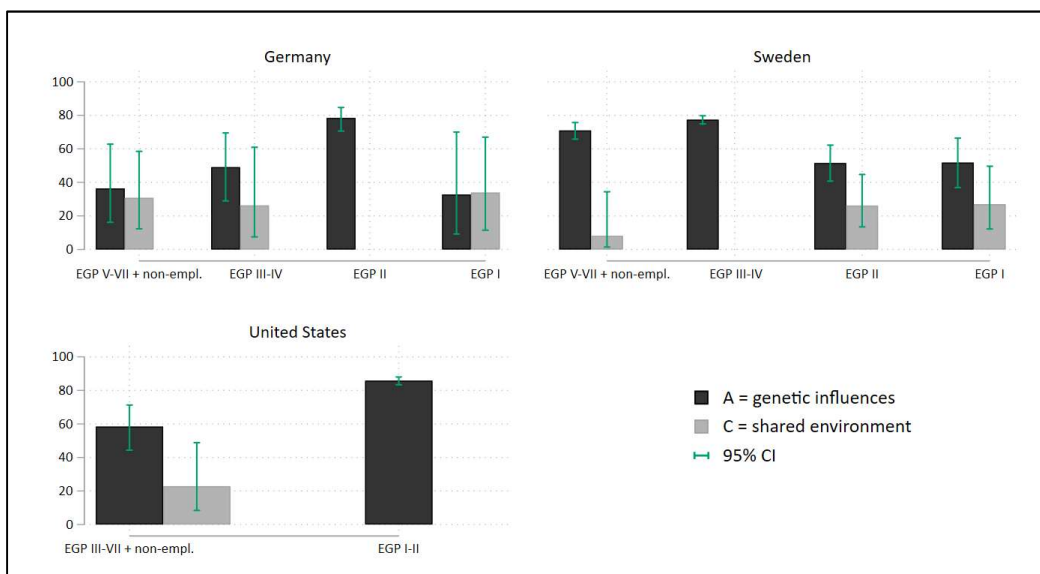
Figure 4.2 visualizes the ACE variance decomposition results for school grades differentiated by parents' EGP class. Detailed results are displayed in Appendix Table 4.D.7.

For Germany the results show that genetic influences on grades increase in the three lower EGP classes, while shared environmental influences decline. In EGP classes V–VII, including the non-employed, genetic influences account for about 35% of the variation, while genes account for about 80% in EGP class II. However, in the highest EGP class genetic effects explain only about 30% of the total variation in grades.

In Sweden genetic influences decline with parents' EGP class. In families belonging to the lower two EGP classes, genetic influences account for up to about 75% of the total variation, while shared environmental influences explain less than 10%. For families belonging to EGP classes I and II, genetic influences account for about half of the variation, and shared environmental influences account for about one-fourth.

For the United States, we can only differentiate between the upper two EGP classes and EGP classes III to VII, including the non-employed, due smaller sample sizes. The results support an interaction in line with the Scarr–Rowe hypothesis: For children from families belonging to the lower EGP classes, genetic influences account for roughly 60%, while for those belong to the higher EGP classes genes account for about 80%. Moreover, shared environmental influences are absent in the upper EGP class.

Figure 4.2 ACE Variance Decompositions Results for Twins' School Grades by Parents' EGP Class



Sources: Add Health, Swedish Registers, and TwinLife; own calculations.

Analyses using parents' education instead of EGP class are presented in Figure 4.E.2 in the Appendix. The results for Sweden and the United States provide no support for a social stratification of genetic effects in line with the Scarr–Rowe hypothesis. For Germany, we find support for the Scarr–Rowe hypothesis: genetic influences are about 40% in less and about 60% in more educated families. However, differences are not statistically significant.

In sum, we find indications for socially stratified genetic effects in line with the Scarr–Rowe hypothesis for school grades by parents' EGP class in the United States and by parents' education in Germany.

Next, we turn to the results that put the Scarr–Rowe hypothesis for educational attainment to the test. To address the accumulation of years of education on the upper tail of the distribution, we estimate Tobit ACE models (see Appendix 4.C). Table 4.3 reports the means and variances of years of education by parents' EGP class for each country.

Table 4.3 Means and Total Variance for Twins' Years of Education by Parents' EGP Class

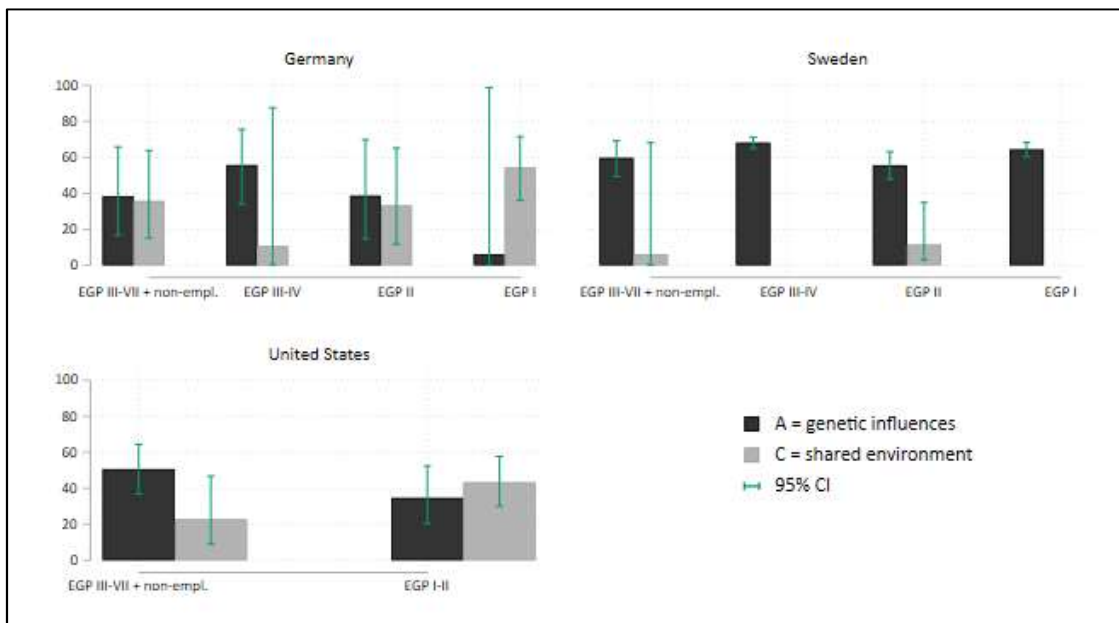
| | Germany | | Sweden | | United States | |
|------------------------------|----------------|-------|---------------|-------|--------------------------------|-------|
| | b/var | c.s.e | b/var | c.s.e | b/var | c.s.e |
| EGP V–VII or non-employed | | | | | EGP III–VII or non-employed | |
| Mean | 0.06 | 0.13 | –0.14 | 0.04 | –0.12 | 0.07 |
| Total var. | 2.88 | 0.27 | 1.72 | 0.07 | 1.71 | 0.13 |
| N(Pairs) | 279 | | 1290 | | 277 | |
| EGP III–IV | | | | | | |
| Mean | 0.56 | 0.14 | 0.29 | 0.05 | | |
| Total var. | 3.23 | 0.28 | 2.13 | 0.11 | | |
| N(Pairs) | 272 | | 865 | | | |
| EGP II | | | | | EGP I–II | |
| Mean | 1.29 | 0.18 | 0.85 | 0.06 | 0.80 | 0.10 |
| Total var. | 3.98 | 0.38 | 3.07 | 0.14 | 2.02 | 0.22 |
| N(Pairs) | 228 | | 1034 | | 262 | |
| EGP I | | | | | | |
| Mean | 1.84 | 0.23 | 1.77 | 0.10 | | |
| Total var. | 4.55 | 0.49 | 4.09 | 0.24 | | |
| N(Pairs) | 177 | | 684 | | | |

Notes: Clustered standard errors are calculated at the twin pair level. Sources: Add Health, Swedish Registers, and TwinLife; own calculations.

In line with previous research (e.g., Breen and Jonsson 2005; Sirin 2005) and similar to the results for school grades, we find that twins' years of education increase with

parents' EGP class in all countries. Moreover, the total variance for years of education is larger for the higher EGP classes in Germany and Sweden, while the total variance is only slightly higher in the United States. Figure 4.3 displays the results from Tobit ACE models for years of education differentiated by parents' EGP class (estimates are provided in Appendix Table 4.D.8).

Figure 4.3 Tobit ACE Variance Decompositions Results for Twins' Years of Education by Parents' EGP Class



Sources: Add Health, Swedish Registers, and TwinLife; own calculations.

Results for Germany do not support the Scarr–Rowe hypothesis. In EGP classes V–VII, including the non-employed, as well as in EGP class II, genetic influences account for about 40%, and in EGP classes III–IV, for about 55% of the total variation in years of education. In EGP class I, genes account for only about 5%. However, the uncertainty of this estimate is very high.

In Sweden, genetic effects on years of education are rather stable across EGP classes (about 55 to 68%), while genetic effects decline from EGP III–IV class to EGP II class. However, the dip in genetic effects in the EGP class II should not be overstated, since genetic influences in the EGP class I are about 65%.

In the United States, we find—similar to Sweden—that the relative importance of genes decreases. Genetic influences on years of education account for about 50% in EGP classes V–VII, including the non-employed, and for around 35% in the upper EGP classes.

However, using parents' education the results do not support a systematic decline, neither in Sweden nor in the United States (see Appendix Figure 4.E.3). In Germany, results tend in the direction of the Scarr–Rowe hypothesis, but again, differences in genetic effects between the single educational groups are not statistically significant. Thus, we only find indication for a stratification for Germany for years of education and only if parents' education is used as an indicator for social background.

4.4 Conclusion and Discussion

This study extended previous research on gene–environment interactions for education in two crucial ways. First, we acknowledged that not only the proximate family but also the broader institutional environment can shape genetic effects on education. Second, we extended previous research that focused originally on IQ to indicators of educational success, namely educational achievement measured in school grades and educational attainment measured in years of education. Specifically, we addressed the following research questions: Do genetic effects on educational success vary across countries, and are there differences in the social stratification of genetic effects on educational success among these countries?

We selected three advanced industrialized societies for our study: Germany, Sweden, and the United States. These countries largely differ in the setup of their educational systems and represent prototypically three different types of welfare regimes, which are often used in internationally comparative social inequality research. We hypothesized that genetic influences on educational success are overall weaker in Germany and the United States than in Sweden. Furthermore, we expected that the association between parents' socioeconomic standing and genetic effects on educational success is stronger in Germany and in the United States than in Sweden. For Germany, our hypothesis was rooted in the early tracking system and for the United States in the less extensive welfare regime.

Our study yielded three important findings: First, we found that genetic effects on years of education are smaller than genetic effects on school grades –independent of country. Hence, genes are more important for educational achievement than for educational attainment. In addition, shared environment environmental influences on

educational attainment were stronger in Germany and the United States. This supports the notion of socially stratified schooling decisions that operate over and above educational achievement (Boudon 1974; Breen and Goldthorpe 1997; Erikson and Jonsson 1996). However, we did not find effects of the shared environmental influences on educational attainment in Sweden, which diverts from previous findings based on an international meta-analysis (Branigan, Mccallum, and Freese 2013). There are three reasons that could account for conflicting results. First, our results are based on more recent birth cohorts (i.e., we studied birth cohorts for 1975–1982, while meta-analysis examined birth cohorts for 1926–1958), and previous research shows that genetic influences on education have increased among birth cohorts born in the second half of the twentieth century (Branigan, Mccallum, and Freese 2013; Heath et al. 1985). Second, the samples used in the meta-analyses were not all population based, including the sample of Sweden where the Swedish Twin Registry was used. Third, the meta-analysis did not account for assortative mating. Without such an adjustment, genetic influences tend to be underestimated, while shared environmental influences are overestimated (Freese and Jao 2017). That shared environmental influences were absent for educational attainment in Sweden indicates that educational choices are more closely related to educational achievement, which could be explained with the less selective comprehensive schooling system.

Second, we identified cross-country differences in genetic effects on educational success. Genetic effects on educational success were least pronounced in Germany, and most pronounced in Sweden. Our hypothesis on cross-country differences was therefore supported for Germany, since genetic effects were comparatively small for both indicators of educational success. For the United States, our hypothesis was only partly supported, since genetic effects on educational attainment were comparatively small, while genetic effects on educational achievement were at least as large as in Sweden. Together, these findings supported our expectation that more egalitarian educational systems have a positive effect on the development of genetic potential for educational success and that early tracking might be an important factor for the suppression of related genetic effects. Future research should build upon our findings and focus in a more detailed manner on the impact of the tracking system. For instance the educational system in the Nordic countries changed from a tracked to a comprehensive schooling system (see for an overview on the educational reforms in Denmark, Finland, Norway, and Sweden (Gustafsson 2018)). If tracking lowers genetic effects on education, genetic effects on

educations should increase after comprehensive schools were introduced. Systematic cross-countries using a culturally homogenous set of countries (“most similar case design (Lijphart 1971)) increase the generalizability of the results.

Third, we found indications for a social stratification of genetic effects in line with the Scarr–Rowe hypothesis for educational success in Germany and the United States. We did not find any evidence for a gene–environment interaction in line with the Scarr–Rowe hypotheses in Sweden. If anything, this underlines the positive impact of more egalitarian educational systems on the development of genetic effects relevant to education. However, differences between countries are too small and not robust enough to clearly support our hypothesis. Yet, the evidence for an interaction in line with the Scarr–Rowe hypothesis for Germany is weaker than previously found using a more fine-grained measure for years of education (Baier and Lang 2019). Thus, differences in the results for Germany between this and the previous study are likely to be driven by the harmonized measure of education which comes at the cost of preciseness. For the international comparison, however, it is crucial to investigate the same measure of education in each country; otherwise, results on genetic and environmental influences can be differently affected by the way educational attainment is measured and, thus, cannot be meaningfully interpreted across countries.

It is important to note that twins’ zygosity was unknown for our sample from Sweden. We adjusted in line with previous research for the missing information based on the assumption that same-sex and opposite-sex dizygotic twin births are equally likely (Figlio et al. 2017). This assumption is fairly reasonable. In addition, there is no reason to believe that the distribution same-sex and opposite-sex dizygotic twin births varies by parents’ social background which would have affected our results in regards to the Scarr–Rowe hypothesis. Nonetheless, future research is needed to gain the precise estimates of genetic influences on educational success. Since some twin pairs tend to be misclassified, our adjustment can lead to an underestimation of genetic differences between monozygotic and dizygotic twins. Therefore, our results represent lower bounds of genetic influence on educational success. Hence, the overall conclusions we draw from our cross-country comparison should not be affected by this adjustment. If anything, we underestimated the role of genes in Sweden.

For the United States, our sample sizes were comparatively small, and analyses for parents’ EGP class were based on broad categorizations (i.e., EGP classes I and II versus EGP III–VII, including the non-employed). However, the Add Health data are currently

the only nationally representative dataset that includes twins. Since the quality of educational institutions varies considerably among federal states, the representativeness across states is crucial for our study purposes. Nonetheless, more research for the United States is needed to test in a more fine-grained way for the social stratification of genetic influences on educational success.

In sum, our study is the first to study cross-country differences in genetic effects on educational success. We found substantial differences in genetic effects on educational success among Germany, Sweden, and the United States. An important factor that causes these cross-country differences may be rooted in the stratification of educational systems, specifically in the strictness and timing of tracking.

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Appendix

4.A Data Sources

Germany: For Germany, we used data from the German Twin Family Panel (TwinLife) (Diewald et al. 2018). TwinLife provides a population register-based sample of four birth cohorts of monozygotic and same-sex dizygotic pairs of twins and their families residing in Germany (Lang and Kottwitz 2017). TwinLife applies an extended twin family design in which the twins, social and biological parents, and one sibling (if available) are surveyed. Twins' zygosity was determined by means of similarity reports (Lenau and Hahn 2017). For our study, we used data of twins from the oldest birth cohort (1991–1993) from waves I and II.

Sweden: We used register data for Sweden (Statistics Sweden 2011). In Sweden, each individual has a unique personal identification number (PIN). This PIN makes it possible to link the records of an individual across the various administrative registers, thus providing information on education, occupation, and grades. This study used the Swedish multi-generational register to link individuals to their siblings (including twins). The multi-generational register contains information on the PIN of each individual, as well as on the PIN of the individual's parents. This allowed us to identify the biological mother and father of each individual and, in turn, to identify any other biological kin relations. The main family members of interest in this study were the mother, father, and siblings. We used information on the biological mother and father to identify siblings. Unfortunately, our access did not include the information on twins' zygosity. We used twins' sex to approximate zygosity and applied an adjustment for this proxy (see section 4.C).

United States: For the United States, we used data from the National Longitudinal Study of Adolescent to Adult Health (Add Health) (Harris et al. 2013). The Add Health data consists of a nationally representative sample of adolescents in Grades 7–12 during the 1994/1995 school year. In addition to the core sample, an oversample of siblings (including twins) was taken which we used for our analyses. Twins' zygosity was also determined by means of similarity questionnaires (Harris et al. 2006). School grades were measured at Wave III (in 2001–2002), and years of education attained in adulthood were measured at Wave IV (in 2008).

4.B Variables

The original measurements of educational success, school grades, and years of education varied among countries. In Germany, we used twins retrospective self-reports on final grade point average for secondary education. Grades were reported to the first decimal place and ranged from 1 (very good) to 4 (sufficient). We coded all students who failed as 5. To account for differences in grades between secondary schooling tracks in Germany, we rescaled grades. Since the highest secondary school track is the most demanding, we subtracted 1 grade if students graduated from the intermediate and 2 grades if students' graduated from the lowest school track. Hence, the adjusted scale for grades in Germany ranges from 1 to 7. Further, we rotated grades to match the scale direction of grades across countries. The sample for the analyses of school grades in Germany included 849 twin pairs aged 21 to 27.

For Sweden, grade point averages were calculated based on results for single subjects recorded in the registers. Since the system for assigning grades in the Swedish high school system has changed several times over the past decades, we limited our analyses to the period 1998–2007 during which the grading system remained constant. During this period, grades in the Swedish compulsory schooling system included pass with special distinction, pass with distinction, pass, and fail. Each of these grades was assigned a numerical value of 20, 15, 10, or 0, respectively. The overall grade point average was calculated by summing up the values for the 16 best grades achieved by any given student, and the overall range was therefore 0 to 320. School grades are observed at the end of the ninth grade (i.e., at the end of comprehensive schooling) when students are around age 16. The Swedish sample for the analyses of school grades comprised 6510 twin pairs.

For the United States, we used retrospective self-reports on grades. Twins reported their total high school grade point average up to the first decimal place using a four-point scale (4 indicated the best grade; 0 indicated the worst or failure). The analytical sample for school grades consisted of 364 twin pairs aged 18 to 25.

To measure years of education, we constructed a harmonized measure based on twins' educational certificates. The harmonization of education across countries is crucial for the analyses since ACE variance decompositions are sensitive to the granularity of the measurement (Freese and Jao 2017). In brief, the fewer categories of educational attainment are distinguished, the larger the estimates for shared environmental influences and the smaller the estimates for genetic influences tend to be. Since school grades are

measured in a very detailed format in all countries, there was no need to harmonize this measure.

For education, we differentiated in all countries among basic education, upper secondary education (vocational track), upper secondary education (academic track), post-secondary non-tertiary education, and tertiary level and assigned 9, 11, 12, 14 and 15.5 years for the corresponding levels, respectively (see Table 4.D.2). The assigned values were based on the coding used in the Swedish register data. Our measure of years of education indicated achieved degrees and did not reflect the actual time spent in educational institutions.

For Germany, years of education were measured from 21 to 27 years of age, and the sample contained 956 twin pairs; for Sweden, years of education were measured at age 30, and the sample consisted of 3873 twin pairs; and for the United States, years of education were measured from 25 to 32 years of age, and the sample comprised 539 twin pairs.

The samples for Germany and the United States included twins that were still in education at the time when years of education were measured. For the United States, about 16% of the students were still enrolled in school for credits. These students were assigned the years of education associated with the track they were enrolled in. We found that dropping the youngest students did not affect our results.

In the sample for Germany, twins were even younger, and about 49% of them were still in vocational training or tertiary education. To address the related uncertainty about the final degree attained, we ran two sets of estimations (Baier and Lang 2019). For the first set, we assumed that all twins that were still in education would finish the track they were currently enrolled in (as in the United States). This is the so-called “upper-bound scenario” which we report in the Results section. For the second set, labeled “lower-bound scenario,” we assumed that all twins that were still enrolled would drop out of their current track, and assigned the years of education associated with their prior educational degree (results for the lower bound estimations are visualized in Figure 4.E.4). For both scenarios, we assumed that twins would not afterwards pick up a track changing their final attainment. These assumptions led to differences in the years of education assigned in the two scenarios for 24.9% of the twins.

In all countries, the distributions for years of education were heaped on the right tail. In the sample for Germany, 54.8% of the twins had 15.5 years of education in the upper bound-scenario, and 33.0% of the twins had 15.5 years of education in the lower-bound

scenario. In Sweden, 41.6% of the twins had 15.5 years of education, and in the United States 33.2%. Since ACE variance decompositions tend to be sensitive to such clustering of responses (Freese and Jao 2017), we estimated Tobit ACE models in addition to standard ACE models (see section 4.C). The estimates based on Tobit ACE models are reported in the Results section, while those using standard ACE models are shown in Table 4.D.9.

To facilitate the comparability of our results for school grades and years of education across countries, we z-standardized both outcomes. In Sweden, where twins' zygosity is unknown, we z-standardized the outcomes separately for women and men to account for nongenetic similarity among same-sex twins induced by having the same gender (Figlio et al. 2017).

We measured socioeconomic background with the highest level of parents' occupational class and education (dominance principle). Parents' occupation was indicated by the EGP class scheme (Ganzeboom and Treiman 1996). We differentiated among the following four groups: 1) higher-grade professionals (EGP I), 2) lower grade professionals (EGP II), 3) routine non-manual employees (EGP III-IV), and 4) workers and farmers (EGP V-VII). In group 4, we included families where both parents were non-employed. Due to a small sample size in the United States, we differentiated between professionals (EGP I-II) and non-professionals (EGP III-VII) including families where both parents were non-employed. For parents' education, we differentiated among the following three groups: 1) basic education and upper secondary education (vocational track), 2) upper secondary education (academic track) and post-secondary non-tertiary education, and 3) tertiary education.

In Germany, parental EGP class and parental education is based on parents' reports and is measured when twins are between 21 to 27 years of age. We found that missing information for mothers was modest: 6.5% for the EGP class and 4.0% for education. The number missing for fathers was higher, 40.9% for the EGP class and 38.5% for education. To account for missingness in the data we used multiple imputation with chained equations and created 20 imputations for each missing observation (van Buuren et al. 2006).

For Sweden, we measured parental education and EGP during childhood. Information on parental social class was derived from the 1990 census, and parental education was defined as the maximum level attained. Since few people pursue additional formal education after childbearing (which was particularly the case for the birth cohorts of the

parents we were studying), differences in the level of parental education following childbirth were found to be minor.

For parents' EGP class and education in the United States, we used resident mother's and father's attainment in the first wave (1994–1995) when the respondents were in Grades 7–12 (about 13–18 years of age). We constructed this measure starting with the parents' reports, and when these were missing (because not all parents of the sampled children had taken part in the surveys), we used children's reports on their parents instead. If children provided discordant information on their parents (e.g., the first twin reports the resident mother as a high school graduate, and the second specifies some college education with no degree), we took the maximum value of the different reports. Missing information on parental social background was modest among mothers, only about 6% for EGP class and 5% for education. A greater proportion of data was missing for resident fathers' EGP class and education (about 30% for each), which was largely due to the prevalence of single-parent households in the United States. For cases where we had a report for one parent, we used their information to code the household. Because only a small proportion of households lack data for either parent and because, in many cases, this missing information was not due to nonresponse but acceptable reasons (e.g., children raised by grandparents), we did not impute values for the sample for the United States. Tables 4.D.3–4.D.5 provide descriptive statistics of the samples for each country.

4.C Methods

We estimated ACE models using a multilevel mixed-effects specification developed by Rabe-Hesketh et al. (2008). This is a two-level random effects model with constrained and weighted random effects. Like other ACE models, this specification builds on the rules of Mendelian inheritance and assumes that the genetic correlation within MZ twin pairs is at 1, while the population average genetic correlation within DZ twin pairs is at 0.5 (e.g., Plomin et al. 2008). These assumptions are used to weight the two random effects that model the additive genetic variance component (A) of a phenotype by zygosity in a multilevel framework:

$$y_i = b_0 + b_1 * A_j(0, A) + b_2 * A_i(0, A) + C_j(0, C) + e_i(0, E)$$

with i indicating the twin level, j indicating the twin pair level, y the outcome, b_0 an intercept, b_1 a weight of 1 for MZ twin pairs, of $\sqrt{0.5}$ for DZ twin pairs, b_2 a weight of 0 for MZ twins pairs, and of $\sqrt{(1 - 0.5 = 0.5)}$ for DZ twin pairs, A_j and A_i two random effects which are constraint to be equal ($A_j=A_i$) to capture the additive genetic variance (A), C_j a random effect modeling the environmental variance shared by a twin pair (C), and the residual e_i , an estimate of the environmental variance specific to each twin (E) which includes measurement error in the phenotype. Figure 4.E.5 displays a related path diagram. We estimated the ACE models using Stata 14 and the `acelong.ado`-package (Lang 2017).

Estimating an ACE model requires a set of further assumptions. First, it is assumed that environments influence MZ and DZ twin pairs in the same way (equal environments assumption [EEA]). Yet, it is likely that MZ twins are treated in a more similar fashion than DZ twins (which is also partly the result of their genetic resemblance). To date, several studies have tested the validity of the EEA for several—mostly psychological—traits, and studies that focus on educational outcomes are missing. For IQ, however, studies report that the more equal environments that MZ twins encounter do not lead to an overestimation of the heritability (Derks, Dolan, and Boomsma 2006).

Second, the CTD assumes that there is no assortative or selective mating of parents in regard to the characteristic under study. Under the assumption of random mating, it can be assumed that DZ twins share, on average, about 50% of their DNA. Assortative mating increases the genetic similarity of spouses and, hence, the average similarity of

DZ twins. Since assortative mating based on education is a well-established phenomenon across Western societies (e.g., Blossfeld 2009), we corrected for assortative mating as follows (Loehlin, Harden, and Turkheimer 2009): $0.5 + 0.5 * h_0^2 * r_p$ while h_0^2 denotes the heritability –or share of genetic influences (A)– estimated without correction for assortative mating, and r_p the correlation of parents with respect to the trait under study (here, education) (Loehlin, Harden, and Turkheimer 2009). In Germany, the correlation of parental education was at 0.39, in Sweden at 0.46, and in the United States at 0.53. This led to a corrected genetic correlation of DZ twins of 0.56 in Germany, 0.64 in Sweden, and 0.61 in the United States.

Third, the CTD assumes that there are no interactions among the influences of different genes, which implies that there are no non-additive or dominant genetic effects on the phenotype (no epistasis). And fourth, it is assumed that there are no correlations or interactions between genes and the environment in the population for the phenotype studied. The fourth assumption –neither gene–environment interactions (GxE) nor gene–environment correlations (rGE)– contradicts the Scarr–Rowe hypothesis. We relaxed this assumption when we tested for stratified genetic effects by estimating ACE models separately for socially-defined groups. This type of modeling is called nonparametric gene–environment interaction analysis (Guo and Wang 2002). It relaxes the assumption of additive genetic and environmental effects insofar as genetic and environmental influences on the phenotype are allowed to vary between socially defined groups. In contrast to parametric gene–environment interaction models (e.g., Turkheimer et al. 2003), this type of analysis does not require that the variation of genetic and environmental influences by social background follows a specific functional form (mostly in linear or quadratic fashion). On the downside, nonparametric gene–environment interaction analyses have less power to statistically identify the variance components and their differences, since they are based on subgroups.

To account for the accumulation of years of education on the right tail of the distribution in every country, we estimated ACE models using a Tobit link function (Tobin 1958) instead of the identity link function that is used in standard ACE models. Finally, we addressed the uncertainty related to unknown zygosity in Sweden. Under the assumption that same-sex and opposite-sex DZ twin births are equally likely, the number of MZ twin pairs among same-sex (ss) twin pairs is given by the number of same-sex twin pairs minus the number of opposite-sex (os) twins pairs. We used this information to adjust our assumption about the genetic similarity for same-sex twin pairs in our ACE

models for Sweden. We calculated the genetic correlation among same-sex twins as follows (Figlio et al. 2017): $\frac{ss-os}{ss} + 0.5 * \frac{ss}{ss}$. The adjustment led to a corrected genetic correlation of same-sex twin pairs of about 0.79 for school grades and of about 0.82 for years of education.

4.D Tables

Table 4.D.1 Overview of the Sample Sizes

| | Germany | Sweden | United States |
|---------------------------|----------------|---------------|----------------------|
| School Grades | | | |
| Birth cohorts | 1990–1993 | 1982–1991 | 1976–1982 |
| Age | 21–27 | 16 | 18–25 |
| N(Pairs) | 849 | 6510 | 364 |
| Years of Education | | | |
| Birth cohorts | 1990–1993 | 1975–1982 | 1975–1982 |
| Age | 21–27 | 30 | 25–32 |
| N(Pairs) | 956 | 3873 | 539 |

Sources: Add Health, Swedish Registers, and TwinLife; own calculations.

Table 4.D.2 Coding Scheme for Years of Education

| Level of Education | Years | Germany | Sweden | United States |
|---|--------------|---|--|--|
| Basic education | 9 | No diploma, Lower secondary, Intermediate secondary | Basic education | 8th grade or less, some high school |
| Upper secondary school (vocational track) | 11 | No diploma but apprenticeship, lower secondary and apprenticeship, intermediate secondary and apprenticeship | Upper secondary school (vocational track) | High school graduate, some vocational/ technical training (after high school) |
| Upper secondary school (academic track) | 12 | Upper secondary intermediate and technical school, diploma to enter tertiary education | Upper secondary school (academic track) | Completed vocational/ technical training (after high school) |
| Post-secondary non-tertiary | 14 | Diploma to enter a professional college and technical school, diploma to enter a professional college and apprenticeship, upper secondary and technical school | Post-secondary non-tertiary | Some college |
| Tertiary level | 15.5 | Diploma to enter a professional college and higher technical college, upper secondary and higher technical college, diploma to enter a professional college and university, upper secondary and university | B.A., M.A., University college degree, Licentiate and doctorate degree | Completed college (B.A.), some graduate school, some graduate training beyond a master's degree, some post- baccalaureate professional education (e.g., law, medical, and nursing schools) |

Table 4.D.3 Summary Statistics: Germany

| | Samples Used for School Grades | | | | | | | | Samples Used for Education in Years | | | | | | | |
|-----------------------------|---------------------------------------|------|------|------|-----------------|------|------|------|--|------|------|------|-----------------|------|------|------|
| | Monozygotic Twins | | | | Dizygotic Twins | | | | Monozygotic Twins | | | | Dizygotic Twins | | | |
| | mean | SD | Min | Max | Mean | SD | Min | Max | Mean | SD | Min | Max | Mean | SD | Min | Max |
| School grades | 4.95 | 1.22 | 1 | 7 | 4.83 | 1.26 | 1 | 7 | | | | | | | | |
| Years of education | | | | | | | | | 13.76 | 2.17 | 9 | 15.5 | 13.64 | 2.29 | 9 | 15.5 |
| Age | 24.14 | 1.36 | 21 | 27 | 24.14 | 1.24 | 21 | 27 | 24.14 | 1.36 | 21 | 27 | 24.12 | 1.26 | 21 | 27 |
| Birth year | 1991.53 | 1.14 | 1990 | 1993 | 1991.64 | 1.09 | 1990 | 1993 | 1991.54 | 1.13 | 1990 | 1993 | 1991.60 | 1.09 | 1990 | 1993 |
| Male | 40.22% | | | | 43.49% | | | | 40.23% | | | | 43.02% | | | |
| Parents' EGP class | | | | | | | | | | | | | | | | |
| EGP V-VII or non-employed | 28.39% | | | | 25.78% | | | | 29.30% | | | | 29.05% | | | |
| EGP III-IV | 28.82% | | | | 27.08% | | | | 29.88% | | | | 26.80% | | | |
| EGP II | 23.87% | | | | 26.56% | | | | 22.85% | | | | 25.00% | | | |
| EGP I | 18.92% | | | | 20.57% | | | | 17.97% | | | | 19.14% | | | |
| Education of parents | | | | | | | | | | | | | | | | |
| Low | 49.89% | | | | 46.61% | | | | 51.56% | | | | 47.97% | | | |
| Middle | 15.27% | | | | 17.71% | | | | 15.43% | | | | 18.47% | | | |
| High | 34.84% | | | | 35.68% | | | | 33.01% | | | | 33.56% | | | |
| N(Pairs) | 465 | | | | 384 | | | | 512 | | | | 444 | | | |

Source: TwinLife; own calculations.

Table 4.D.4 Summary Statistics: Sweden

| | Samples Used for School Grades | | | | | | | | Samples Used for Education in Years | | | | | | | |
|-----------------------------|--------------------------------|-------|------|------|-----------------|-------|------|------|-------------------------------------|------|------|------|-----------------|------|------|------|
| | Monozygotic Twins | | | | Dizygotic Twins | | | | Monozygotic Twins | | | | Dizygotic Twins | | | |
| | Mean | SD | Min | Max | Mean | SD | Min. | Max | Mean | SD | Min | Max | Mean | SD | Min | Max |
| School grades | 217.73 | 57.82 | 0 | 320 | 217.06 | 58.45 | 0 | 320 | | | | | | | | |
| Years of education | | | | | | | | | 13.52 | 1.97 | 9 | 15.5 | 13.38 | 1.99 | 9 | 15.5 |
| Age | 16 | | | | 16 | | | | 30 | | | | 30 | | | |
| Birth year | 1986.94 | 2.86 | 1982 | 1991 | 1987.17 | 2.809 | 1982 | 1991 | 1978.52 | 2.27 | 1975 | 1982 | 1978.46 | 2.31 | 1975 | 1982 |
| Male | 49.56% | | | | 50.00% | | | | 49.86% | | | | 50.00% | | | |
| Parents' EGP class | | | | | | | | | | | | | | | | |
| EGP V–VII or non-employed | 39.60% | | | | 37.70% | | | | 32.90% | | | | 34.42% | | | |
| EGP III–IV | 19.21% | | | | 19.48% | | | | 22.90% | | | | 22.53% | | | |
| EGP II | 26.48% | | | | 25.38% | | | | 27.42% | | | | 24.74% | | | |
| EGP I | 14.70% | | | | 17.43% | | | | 17.42% | | | | 18.31% | | | |
| Education of parents | | | | | | | | | | | | | | | | |
| Low | 31.40% | | | | 30.75% | | | | 37.17% | | | | 38.54% | | | |
| Middle | 35.57% | | | | 32.02% | | | | 33.04% | | | | 33.75% | | | |
| High | 33.03% | | | | 37.23% | | | | 29.79% | | | | 27.71% | | | |
| N(Pairs) | 4611 | | | | 1899 | | | | 2830 | | | | 1043 | | | |

Source: Swedish Registers; own calculations.

Table 4.D.5 Summary Statistics: United States

| | Samples Used for School Grades | | | | | | | | Samples Used for Education in Years | | | | | | | |
|-----------------------------|--------------------------------|------|------|------|-----------------|------|------|------|-------------------------------------|------|------|------|-----------------|------|------|------|
| | Monozygotic Twins | | | | Dizygotic Twins | | | | Monozygotic Twins | | | | Dizygotic Twins | | | |
| | Mean | SD | Min | Max | Mean | SD | Min | Max | Mean | SD | Min | Max | Mean | SD | Min | Max |
| School grades | 2.72 | 0.77 | 1 | 4 | 2.54 | 0.87 | 1 | 4 | | | | | | | | |
| Years of education | | | | | | | | | 13.71 | 1.86 | 9 | 15.5 | 13.50 | 2.10 | 9 | 15.5 |
| Age | 21.97 | 1.61 | 18 | 25 | 21.70 | 1.68 | 18 | 25 | 29.04 | 1.59 | 25 | 32 | 28.72 | 1.66 | 25 | 32 |
| Birth year | 1978.86 | 1.60 | 1976 | 1982 | 1979.14 | 1.67 | 1976 | 1982 | 1978.82 | 1.60 | 1975 | 1982 | 1979.13 | 1.66 | 1975 | 1982 |
| Male | 49.00% | | | | 50.07% | | | | 46.20% | | | | 50.08% | | | |
| Parents' EGP class | | | | | | | | | | | | | | | | |
| EGP III–VII or non-employed | 43.25% | | | | 54.17% | | | | 47.11% | | | | 54.01% | | | |
| EGP I–II | 56.76% | | | | 45.83% | | | | 52.89% | | | | 45.92% | | | |
| Education of parents | | | | | | | | | | | | | | | | |
| Low | 26.35% | | | | 33.33% | | | | 28.37% | | | | 34.74% | | | |
| Middle | 34.46% | | | | 26.38% | | | | 32.21% | | | | 27.19% | | | |
| High | 39.19% | | | | 40.28% | | | | 39.42% | | | | 38.07% | | | |
| N(Pairs) | 148 | | | | 216 | | | | 208 | | | | 331 | | | |

Source: Add Health; own calculations.

Table 4.D.6 ACE Variance Decomposition Results for Twins' School Grades and Years of Education –Adjusted for Assortative Mating

| | School Grades | | | | | Years of Education | | | | |
|----------------------|---------------|-------|---------|--------|-------|--------------------|-------|---------|--------|-------|
| | b/var | c.s.e | z-value | 95%-CI | | b/var | c.s.e | z-value | 95%-CI | |
| Germany | | | | | | | | | | |
| Mean | 0.00 | 0.03 | 0.10 | -0.06 | 0.06 | 0.86 | 0.08 | 11.04 | 0.71 | 1.01 |
| Total var. | 1.00 | 0.04 | 23.53 | 0.92 | 1.09 | 3.78 | 0.16 | 23.27 | 3.48 | 4.12 |
| A% | 56.20 | 11.16 | 5.04 | 46.50 | 65.44 | 35.51 | 11.00 | 3.23 | 23.08 | 50.25 |
| C% | 18.23 | 10.02 | 1.82 | 7.06 | 39.56 | 36.08 | 9.97 | 3.62 | 24.72 | 49.25 |
| E% | 25.57 | 2.62 | 9.77 | 21.94 | 29.57 | 28.41 | 3.05 | 9.33 | 24.34 | 32.87 |
| N(Pairs) | 849 | | | | | 956 | | | | |
| Sweden | | | | | | | | | | |
| Mean | 0.06 | 0.01 | 5.34 | 0.04 | 0.08 | 0.49 | 0.03 | 17.12 | 0.43 | 0.54 |
| Total var. | 1.02 | 0.02 | 43.77 | 0.97 | 1.07 | 2.62 | 0.06 | 42.31 | 2.50 | 2.74 |
| A% | 79.99 | 2.33 | 34.32 | 79.06 | 80.88 | 70.17 | 2.40 | 29.27 | 68.75 | 71.56 |
| C% | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| E% | 20.01 | 0.86 | 23.26 | 18.70 | 21.40 | 29.83 | 1.27 | 23.48 | 28.11 | 31.60 |
| N(Pairs) | 6510 | | | | | 3873 | | | | |
| United States | | | | | | | | | | |
| Mean | 0.04 | 0.05 | 0.83 | -0.05 | 0.13 | 0.32 | 0.06 | 5.22 | 0.20 | 0.44 |
| Total var. | 1.03 | 0.07 | 15.54 | 0.91 | 1.16 | 2.03 | 0.13 | 15.86 | 1.79 | 2.30 |
| A% | 84.10 | 7.26 | 11.58 | 81.70 | 86.23 | 49.32 | 10.85 | 4.55 | 38.74 | 59.96 |
| C% | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 28.35 | 9.82 | 2.89 | 16.71 | 43.83 |
| E% | 15.90 | 2.86 | 5.56 | 11.73 | 21.19 | 22.33 | 2.99 | 7.46 | 18.11 | 27.22 |
| N(Pairs) | 364 | | | | | 539 | | | | |

Notes: Clustered standard errors are calculated at the twin pair level. The assumed genetic correlation for same-sex twins in Sweden is 0.79 for the school grades sample, and 0.82 for the years of education sample. Results for years of education are estimated with a Tobit ACE model. Sources: Add Health, Swedish Registers and TwinLife; own calculations.

Table 4.D.7 ACE Variance Decomposition Results for Twins' School Grades by Parents' EGP Class

| | Germany | | | | Sweden | | | | United States | | | | | | |
|---------------------------|---------|-------|---------|--------|--------|-------|---------|-----------------------------|---------------|-------|---------|--------|-------|-------|-------|
| | b/var | cse | z-value | 95%-CI | b/var | cse | z-value | 95%-CI | b/var | cse | z-value | 95%-CI | | | |
| EGP V–VII or non-employed | | | | | | | | EGP III–VII or non-employed | | | | | | | |
| Mean | –0.36 | 0.07 | –5.14 | –0.50 | –0.22 | –0.24 | 0.02 | –13.23 | –0.28 | –0.21 | –0.20 | 0.07 | –2.82 | –0.33 | –0.06 |
| Total var. | 1.13 | 0.09 | 12.13 | 0.96 | 1.33 | 1.06 | 0.04 | 28.33 | 0.98 | 1.13 | 1.10 | 0.09 | 12.65 | 0.94 | 1.28 |
| A% | 36.38 | 20.11 | 1.81 | 16.21 | 62.82 | 70.99 | 8.72 | 8.14 | 65.79 | 75.69 | 58.41 | 16.90 | 3.46 | 44.34 | 71.23 |
| C% | 30.80 | 18.13 | 1.70 | 12.32 | 58.52 | 8.08 | 7.36 | 1.10 | 1.45 | 34.38 | 22.86 | 13.64 | 1.68 | 8.43 | 48.84 |
| E% | 32.82 | 5.88 | 5.59 | 25.60 | 40.97 | 20.93 | 1.45 | 14.47 | 18.78 | 23.26 | 18.73 | 5.21 | 3.59 | 11.78 | 28.45 |
| N(Pairs) | 231 | | | | 2542 | | | | 181 | | | | | | |
| EGP III–VI | | | | | | | | | | | | | | | |
| Mean | –0.16 | 0.07 | –2.33 | –0.30 | –0.03 | 0.03 | 0.02 | 1.45 | –0.01 | 0.08 | | | | | |
| Total var. | 1.08 | 0.09 | 12.53 | 0.92 | 1.26 | 0.90 | 0.05 | 18.45 | 0.81 | 1.00 | | | | | |
| A% | 49.08 | 21.56 | 2.28 | 28.95 | 69.51 | 77.39 | 5.50 | 14.07 | 74.86 | 79.74 | | | | | |
| C% | 26.25 | 19.81 | 1.33 | 7.50 | 60.97 | --- | --- | --- | --- | --- | | | | | |
| E% | 24.67 | 4.86 | 5.08 | 18.21 | 32.50 | 22.61 | 1.85 | 12.19 | 19.92 | 25.54 | | | | | |
| N(Pairs) | 238 | | | | 1256 | | | | | | | | | | |
| EGP II | | | | | | | | EGP I–II | | | | | | | |
| Mean | 0.16 | 0.06 | 2.72 | 0.05 | 0.28 | 0.26 | 0.02 | 12.90 | 0.22 | 0.31 | 0.27 | 0.06 | 4.65 | 0.16 | 0.39 |
| Total var. | 0.89 | 0.08 | 10.53 | 0.74 | 1.07 | 0.87 | 0.04 | 20.79 | 0.79 | 0.96 | 0.78 | 0.06 | 12.88 | 0.67 | 0.91 |
| A% | 78.43 | 16.63 | 4.72 | 70.59 | 84.64 | 51.54 | 11.49 | 4.49 | 40.72 | 62.21 | 85.82 | 8.31 | 10.33 | 83.34 | 87.97 |
| C% | -- | -- | -- | -- | -- | 26.20 | 11.02 | 2.38 | 13.46 | 44.75 | --- | --- | --- | --- | --- |
| E% | 21.56 | 5.21 | 3.74 | 12.52 | 28.99 | 22.27 | 2.11 | 10.54 | 19.21 | 25.65 | 14.18 | 2.62 | 5.41 | 10.32 | 19.19 |
| N(Pairs) | 213 | | | | 1703 | | | | 183 | | | | | | |
| EGP I | | | | | | | | | | | | | | | |
| Mean | 0.34 | 0.06 | 6.12 | 0.23 | 0.45 | 0.50 | 0.03 | 19.29 | 0.45 | 0.56 | | | | | |
| Total var. | 0.62 | 0.07 | 8.87 | 0.50 | 0.77 | 0.84 | 0.06 | 14.09 | 0.73 | 0.96 | | | | | |
| A% | 32.74 | 26.19 | 1.25 | 9.21 | 70.01 | 51.81 | 16.08 | 3.22 | 36.91 | 66.40 | | | | | |
| C% | 33.92 | 23.79 | 1.43 | 11.49 | 66.99 | 27.01 | 13.50 | 2.00 | 12.20 | 49.63 | | | | | |
| E% | 33.35 | 8.72 | 3.82 | 23.05 | 45.52 | 21.18 | 3.01 | 7.04 | 16.90 | 26.19 | | | | | |
| N(Pairs) | 167 | | | | 1009 | | | | | | | | | | |

Notes: Clustered standard errors are calculated at the twin pair level. The assumed genetic correlation for same-sex twins in Sweden is 0.79. Sources: Add Health, Swedish Registers and TwinLife; own calculations.

Table 4.D.8 Tobit ACE Variance Decomposition Results for Twins' Years of Education by Parents' EGP Class

| | Germany | | | | Sweden | | | | United States | | | | | |
|---------------------------|---------|-------|---------|-------------|-----------------------------|-------|---------|-------------|---------------|-------|---------|-------------|--|--|
| | b/var | c.s.e | z-value | 95%-CI | b/var | c.s.e | z-value | 95%-CI | b/var | c.s.e | z-value | 95%-CI | | |
| EGP V-VII or non-employed | | | | | EGP III-VII or non-employed | | | | | | | | | |
| Mean | 0.06 | 0.13 | 0.43 | -0.20 0.31 | -0.14 | 0.04 | -3.89 | -0.21 -0.07 | -0.12 | 0.07 | -1.64 | -0.26 0.02 | | |
| Total var. | 2.88 | 0.27 | 10.68 | 2.39 3.46 | 1.72 | 0.07 | 22.98 | 1.58 1.87 | 1.71 | 0.13 | 13.53 | 1.48 1.98 | | |
| A% | 38.34 | 22.06 | 1.74 | 16.76 65.76 | 59.60 | 12.64 | 4.72 | 49.33 69.10 | 50.68 | 14.67 | 3.45 | 36.81 64.44 | | |
| C% | 35.73 | 20.95 | 1.71 | 14.97 63.69 | 6.10 | 10.86 | 0.56 | 0.20 68.09 | 22.71 | 12.68 | 1.79 | 8.96 46.74 | | |
| E% | 25.93 | 5.21 | 4.98 | 19.10 34.17 | 34.30 | 2.45 | 14.02 | 31.22 37.51 | 26.61 | 4.55 | 5.85 | 20.59 33.64 | | |
| % upper limit | 40.14 | | | | 26.01 | | | | 21.48 | | | | | |
| N(Pairs) | 279 | | | | 1290 | | | | 277 | | | | | |
| EGP III-VI | | | | | EGP I-II | | | | | | | | | |
| Mean | 0.56 | 0.14 | 3.97 | 0.29 0.84 | 0.29 | 0.05 | 5.61 | 0.19 0.39 | | | | | | |
| Total var. | 3.23 | 0.28 | 11.36 | 2.72 3.83 | 2.13 | 0.11 | 20.06 | 1.93 2.34 | | | | | | |
| A% | 55.67 | 25.50 | 2.18 | 33.85 75.50 | 68.07 | 4.93 | 13.82 | 64.91 71.07 | | | | | | |
| C% | 10.52 | 21.98 | 0.48 | 0.20 87.58 | | | | | | | | | | |
| E% | 33.81 | 7.15 | 4.73 | 25.23 43.60 | 31.93 | 2.84 | 11.25 | 28.27 35.83 | | | | | | |
| % upper limit | 49.95 | | | | 36.53 | | | | | | | | | |
| N(Pairs) | 272 | | | | 865 | | | | | | | | | |
| EGP II | | | | | EGP I-II | | | | | | | | | |
| Mean | 1.29 | 0.18 | 7.04 | 0.93 1.65 | 0.85 | 0.06 | 13.55 | 0.73 0.97 | 0.80 | 0.10 | 8.39 | 0.61 0.99 | | |
| Total var. | 3.98 | 0.38 | 10.36 | 3.29 4.81 | 3.07 | 0.14 | 22.32 | 2.81 3.35 | 2.02 | 0.22 | 9.13 | 1.63 2.50 | | |
| A% | 38.59 | 25.58 | 1.51 | 14.63 69.73 | 55.57 | 8.81 | 6.31 | 47.83 63.05 | 34.62 | 12.91 | 2.68 | 20.31 52.38 | | |
| C% | 33.12 | 22.40 | 1.48 | 11.62 65.09 | 11.41 | 8.29 | 1.38 | 3.01 34.87 | 43.27 | 12.85 | 3.37 | 29.88 57.72 | | |
| E% | 28.29 | 7.76 | 3.64 | 18.73 40.32 | 33.02 | 2.69 | 12.28 | 29.59 36.64 | 22.11 | 4.84 | 4.57 | 15.60 30.36 | | |
| % upper limit | 65.35 | | | | 49.27 | | | | 45.61 | | | | | |
| N(Pairs) | 228 | | | | 1034 | | | | 262 | | | | | |

Table continued next page

| EGP I | | | | | | | | | | |
|---------------|-------|-------|------|-------|-------|-------|------|-------|-------|-------|
| Mean | 1.84 | 0.23 | 8.08 | 1.40 | 2.29 | 1.77 | 0.10 | 16.97 | 1.56 | 1.97 |
| Total var. | 4.55 | 0.49 | 9.19 | 3.67 | 5.63 | 4.09 | 0.24 | 17.35 | 3.66 | 4.58 |
| A% | 6.08 | 22.10 | 0.28 | 0.01 | 98.77 | 64.34 | 5.75 | 11.19 | 60.23 | 68.25 |
| C% | 54.34 | 20.64 | 2.63 | 36.12 | 71.47 | | | | | |
| E% | 39.58 | 9.71 | 4.07 | 28.82 | 51.45 | 35.66 | 3.86 | 9.24 | 30.96 | 40.66 |
| % upper limit | 72.60 | | | | | 66.01 | | | | |
| N(Pairs) | 177 | | | | | 684 | | | | |

Notes: Clustered standard errors are calculated at the twin pair level. The assumed genetic correlation for same-sex twins in Sweden is 0.82. Sources: Add Health, Swedish Registers, and TwinLife; own calculations.

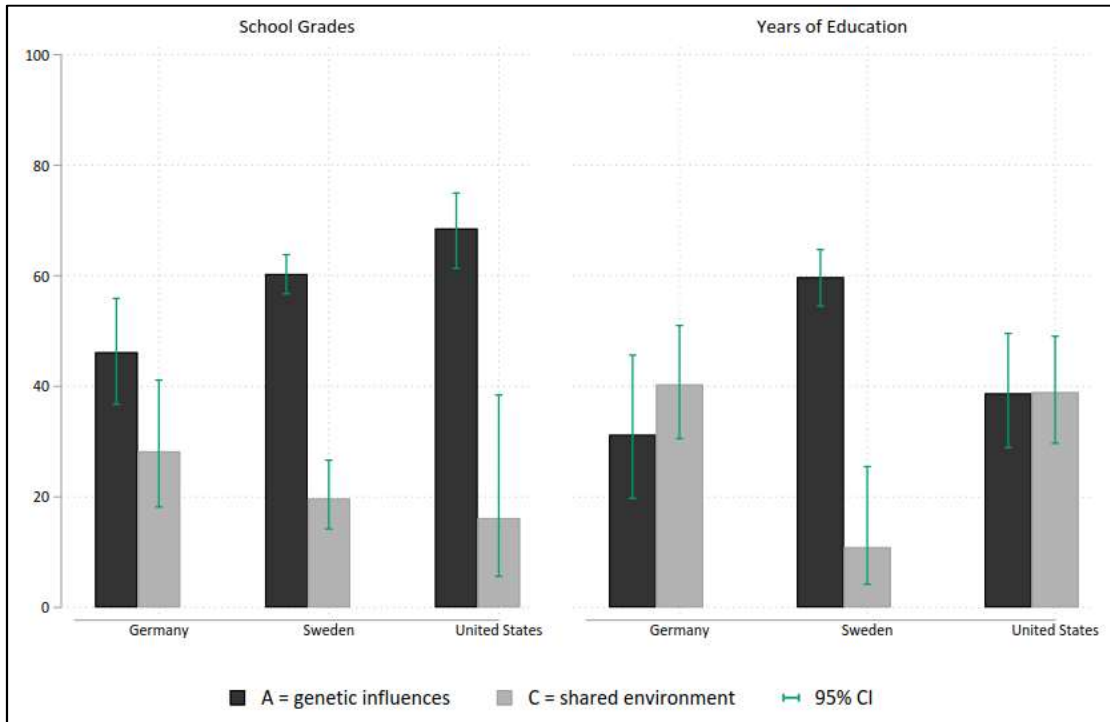
Table 4.D.9 ACE Variance Decomposition Results for Twins' Years of Education by Parents' EGP Class

| | Germany | | | | Sweden | | | | United States | | | | | | |
|---------------------------|---------|-------|---------|--------|-----------------------------|-------|---------|--------|---------------|-------|----------|--------|-------|-------|-------|
| | b/var | c.s.e | z-value | 95%-CI | b/var | c.s.e | z-value | 95%-CI | b/var | c.s.e | z-value | 95%-CI | | | |
| EGP V-VII or non-employed | | | | | EGP III-VII or non-employed | | | | | | | | | | |
| Mean | -0.39 | 0.07 | -5.77 | -0.52 | -0.26 | -0.32 | 0.02 | -13.50 | -0.37 | -0.28 | -0.28 | 0.06 | -5.08 | -0.39 | -0.17 |
| Total var. | 1.20 | 0.05 | 24.26 | 1.10 | 1.30 | 1.00 | 0.03 | 35.47 | 0.94 | 1.06 | 1.13 | 0.06 | 19.59 | 1.02 | 1.25 |
| A% | 39.16 | 20.06 | 1.95 | 19.09 | 63.73 | 51.42 | 12.31 | 4.18 | 39.84 | 62.86 | 51.93 | 14.74 | 3.52 | 38.24 | 65.33 |
| C% | 28.92 | 17.65 | 1.64 | 10.96 | 57.36 | 9.28 | 10.41 | 0.89 | 1.12 | 47.94 | 17.26 | 11.80 | 1.46 | 5.18 | 44.33 |
| E% | 31.91 | 5.57 | 5.73 | 24.97 | 39.76 | 39.30 | 2.51 | 15.68 | 36.36 | 42.31 | 30.81 | 5.50 | 5.60 | 23.89 | 38.73 |
| N(Pairs) | 279 | | | | | 1290 | | | | | 277 | | | | |
| EGP III-VI | | | | | | | | | | | | | | | |
| Mean | -0.10 | 0.06 | -1.66 | 0.22 | 0.02 | -0.04 | 0.03 | -1.42 | -0.10 | 0.02 | | | | | |
| Total var. | 1.03 | 0.05 | 20.87 | 0.94 | 1.13 | 0.96 | 0.03 | 33.95 | 0.91 | 1.02 | | | | | |
| A% | 47.69 | 22.13 | 2.15 | 26.85 | 69.36 | 61.94 | 3.94 | 15.74 | 58.97 | 64.83 | | | | | |
| C% | 10.14 | 18.86 | 0.54 | 0.29 | 81.23 | --- | --- | --- | --- | --- | | | | | |
| E% | 42.18 | 6.90 | 6.12 | 34.61 | 50.12 | 38.06 | 3.01 | 12.63 | 34.47 | 41.77 | | | | | |
| N(Pairs) | 272 | | | | | 865 | | | | | | | | | |
| EGP II | | | | | | | | | | | EGP I-II | | | | |
| Mean | 0.18 | 0.05 | 3.35 | 0.08 | 0.29 | 0.20 | 0.03 | 7.59 | 0.15 | 0.25 | 0.29 | 0.05 | 6.36 | 0.20 | 0.38 |
| Total var. | 0.81 | 0.06 | 12.50 | 0.69 | 0.95 | 0.98 | 0.03 | 31.02 | 0.92 | 1.04 | 0.71 | 0.07 | 9.97 | 0.58 | 0.86 |
| A% | 43.77 | 23.10 | 1.90 | 21.68 | 68.65 | 51.48 | 13.38 | 3.85 | 38.94 | 63.84 | 29.81 | 18.17 | 1.64 | 11.39 | 58.38 |
| C% | 22.18 | 19.59 | 1.13 | 4.80 | 61.68 | 6.67 | 11.16 | 0.60 | 0.27 | 65.44 | 34.90 | 14.84 | 2.35 | 18.90 | 55.23 |
| E% | 34.05 | 7.27 | 4.68 | 25.35 | 43.97 | 41.84 | 3.15 | 13.26 | 38.30 | 45.48 | 35.29 | 7.40 | 4.77 | 26.56 | 45.14 |
| N(pairs) | 228 | | | | | 1,034 | | | | | 262 | | | | |
| EGP I | | | | | | | | | | | | | | | |
| Mean | 0.34 | 0.05 | 6.62 | 0.24 | 0.44 | 0.53 | 0.03 | 19.43 | 0.47 | 0.58 | | | | | |
| Total var. | 0.64 | 0.07 | 9.75 | 0.52 | 0.78 | 0.72 | 0.04 | 19.52 | 0.66 | 0.80 | | | | | |
| A% | 3.68 | 19.97 | 0.18 | 0.00 | 99.94 | 53.64 | 5.61 | 9.56 | 48.52 | 58.69 | | | | | |
| C% | 41.96 | 18.95 | 2.21 | 22.99 | 63.66 | --- | --- | --- | --- | --- | | | | | |
| E% | 54.36 | 9.87 | 5.51 | 45.49 | 62.96 | 46.36 | 4.43 | 10.47 | 41.75 | 51.03 | | | | | |
| N(pairs) | 177 | | | | | 684 | | | | | | | | | |

Notes: Clustered standard errors are calculated at the twin pair level. The assumed genetic correlation for same-sex twins in Sweden is 0.82. Sources: Add Health, Swedish Register, and TwinLife; own calculations.

4.E Figures

Figure 4.E.1 ACE Variance Decompositions Results for Twins' School Grades and Years of Education –Without Adjustment for Assortative Mating



Sources: Add Health, Swedish Registers, and TwinLife; own calculations.

Figure 4.E.2 ACE Variance Decompositions Results for Twins' School Grades by Parents' Education

Sources: Add Health, Swedish Registers, and TwinLife; own calculations.

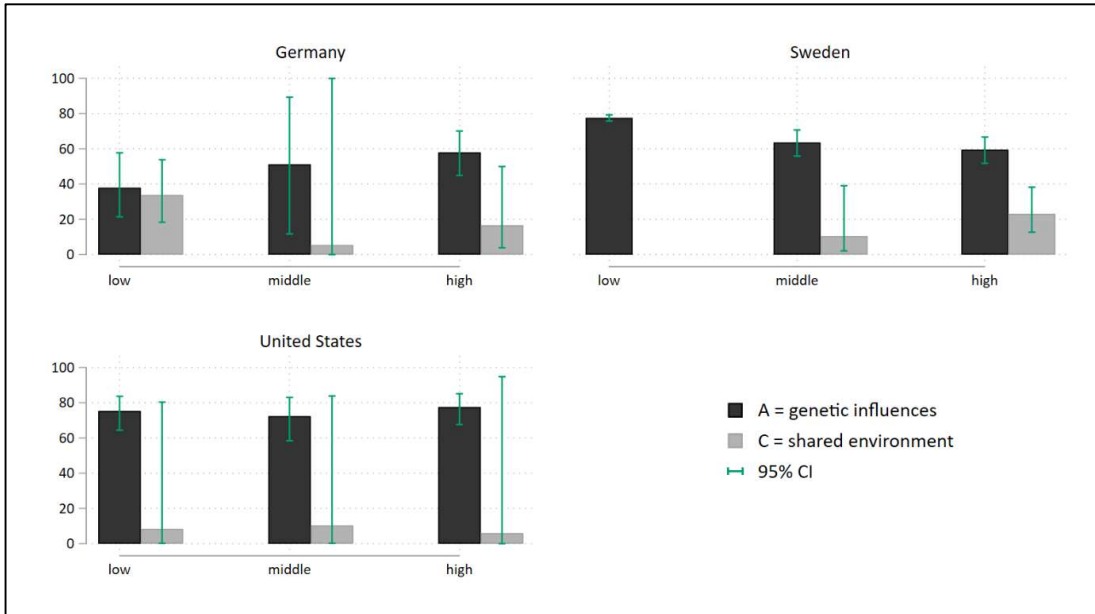
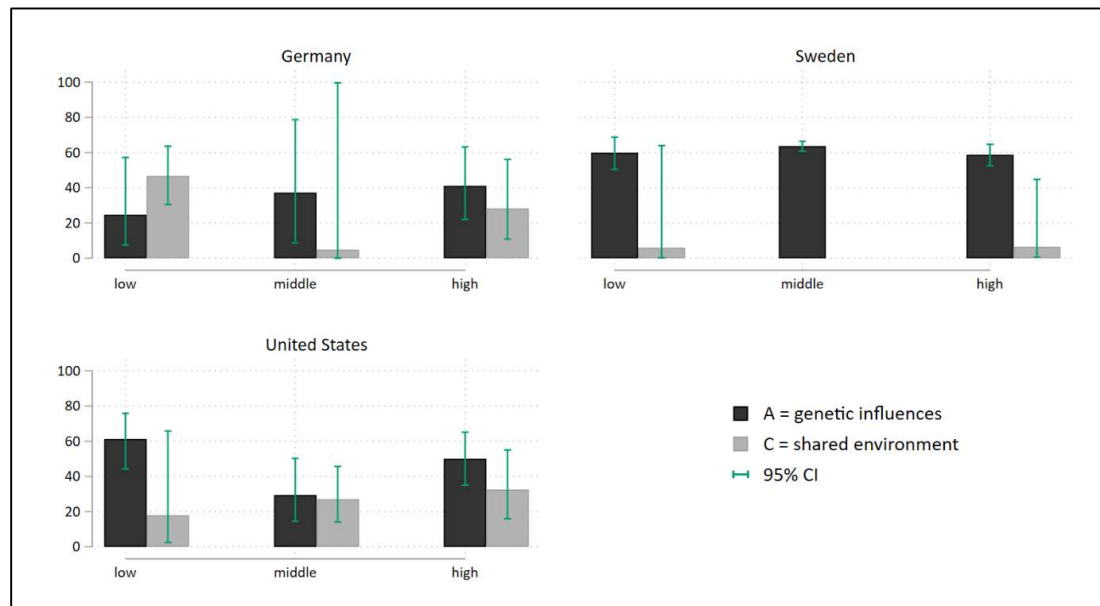
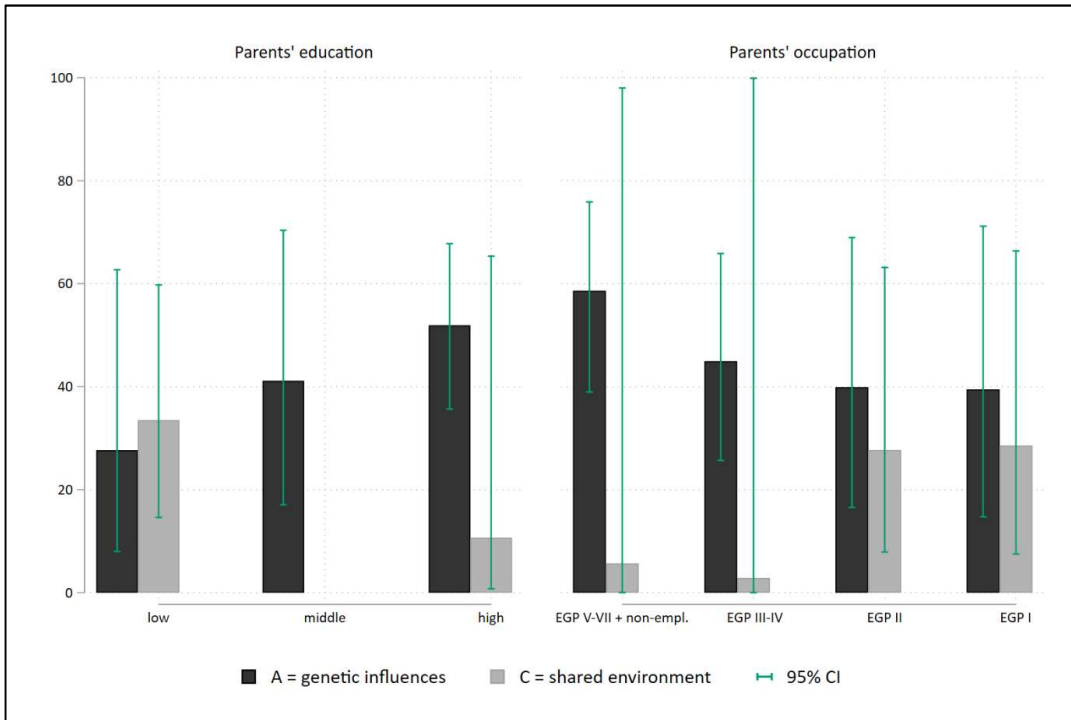


Figure 4.E.3 Tobit ACE Variance Decompositions Results for Twins' Years of Education by Parents' Education



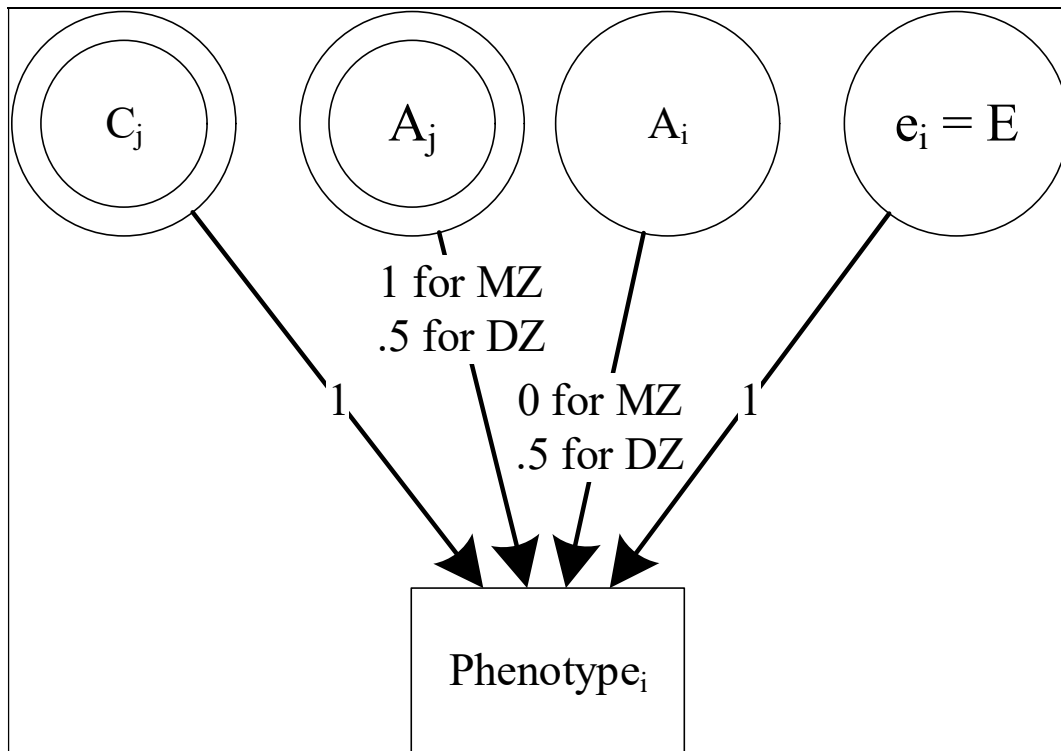
Sources: Add Health, Swedish Registers, and TwinLife; own calculations.

Figure 4.E.4 Tobit ACE Variance Decompositions Results for Twins' Education by Parents' Education and EGP Class for Germany –Lower Bound Estimations



Source: TwinLife; own calculations.

Figure 4.E.5 Path Diagram of Rabe-Hesketh, Skrondal, and Gjessing ACE Model



Source: Rabe-Hesketh, Skrondal, and Gjessing (2008).