Physical Activity, Aging and Cognition

A Study of Effects of Physical Exercise on Cognitive Functions of Older Adults

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Presented by

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To
My Parents,
My Sisters and
My Iran

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Abbreviations

ADP	Adenosinediphosphate
AEG	Aerobic group
ANCOVA	Analysis of covariance
ANEG	Anaerobic group
ATP-PC	Adenosinetriphosphate and creatine phosphate
BA	Brodmann area
BDNF	Brain Derived Neurotrophic Factor
BrdU	Bromodeoxyuridine
Ca^{2+}	calcium ion
CG	Control group
CNS	central nervous system
CO_2	carbon dioxide Molecule
E	Energy
e^-	Electron
e.g.	for example
EAM	episodic-autobiographical memory
EG	exercise groups
EM	episodic memory
ETC	electron transport chain
etc.	etcetera
Fig.	Figure
GPx	glutathione peroxidase
H^+	hydrogen ion
H_2O	water molecule
i.e.	that is
IGF-1	Insulin-like growth factor 1

kg	kilogram
Km/h	kilometer per hour is a unit of speed
LDH	Lactate dehydrogenase
LPS-4	Leistungsprüfsystem
LTM	Long-term memory
M	Mean
MAAS	Maastricht Aging Study
max	maximum
MD	mean difference
METs	metabolic equivalent unit, at sitting at rest
min.	minute
ml/kg/min	milliliters of oxygen per kg of body mass per minute
MMT	mammillothalamic tract
mRNA	messenger ribonucleic acid
mtDNA	mitochondrial DNA
MTL	medial temporal lobe
MTLs	left and right medial temporal lobes
MWT-B	Mehrfachwahl-Wortschatz-Test
NGF	nerve growth factor
O_2	Oxygen Molecule
p	p-value or probability
PFC	prefrontal cortex
PM	procedural memory
PPC	posterior parietal cortex
rCBF	regional cerebral blood flow
ROCF	Rey-Osterrieth Complex Figure Test
ROS	Reactive Oxygen Species
s	second

SD	standard deviation
SE	standard error
SM	semantic memory
SPSS	Statistical Package for the Social Sciences
STM	short-term memory
TMT	Trail Making Test
VLMT	Verbal Learning and Memory Test
$VO_{2\ max}$	Maximal oxygen consumption and is derived from V - volume, O_2 – Oxygen and max - maximum
VSTM	visual short-term memory
WM	working memory

1 Introduction

The brain is permanently active during lifetime, whether conscious or non-conscious and is analysing the information by the brain and nervous system. The cognition is one of the most important functions of nervous system in human. Aspects of human physiological such as physical activity and human life style may interrelate with the brain function. Thus, this work tries to show how various kinds of exercises affect several brain functions of healthy older adults.

The current study is an interdisciplinary investigation and confluence of the physiological psychology and human exercise physiology. Thus, before beginning, the theoretical fundamentals related to the study should be explained briefly.

In chapter 2 the theoretical backgrounds of physical education and exercise sciences as well as physiological psychology about the normal aging process and cognitive functions are represented.
The first section (2.1) introduces the preface of lifestyle and aging process.
Given a theoretical framework of exercise physiological principles, the sections (2.2 and 2.3) provide a brief theoretical background of exercise, physical activity and metabolism.
The section (2.4) introduces the theoretical background to memory including the introduction of theories and classifications of memories.
The section (2.5) introduces a few important facets of learning theories related to procedural memory.
The part (2.6) provides a brief framework of gerontology studies related to cognitive functions and exercise.
Finally, the section (2.7) shows a summary of literature on this domain.

Chapter 3 presents questions and hypotheses. A number of questions were formulated from the survey of the theoretical background. For example, whether there is any relationship between various types of physical exercise and older people's cognitive functions? Or if physical activity can improve cognitive functions, whether aerobic or anaerobic conditions would be a better choice to preserve it?
These questions lead to developing four hypotheses that form the basis of this study.

The first hypothesis expounds on systematic and regular light to moderate physical aerobic activities leading to a measurable improvement in cognitive functions of sedentary older adults.

The second hypothesis states that systematic and regular light physical anaerobic activity leads to a robust and beneficial influence on cognitive functions of sedentary elderly.

The third hypothesis states that learning a new motor skill positively influences semantic memory performance in sedentary older adults.

The fourth hypothesis, regarding the effects of motor learning, supposes that developing a new procedural skill is accompanied by a significant improvement in cognitive functions of the sedentary aged.

In chapter 4 the applied methods, the subjects, and the procedures are introduced. This chapter explains the present study (4.1), sampling method (4.2), objectives (4.3), participants and groups (4.4), variables of study (4.5), exercise protocols of groups (4.6) and neurocognitive tests (4.7).

In chapter 5 the samples and population (5.1), procedure of study (5.2), and the statistical analyses (5.3) including descriptive data and data analysis are

considered. The results of current study, which are given in chapter 5, are obtained regarding the data of the neurocognitive tests. The dates of the study are separately presented for each three groups in sections (5.4) till (5.10).

In chapter 6 the discussions of the devised hypotheses are given separately by considering terms such as the aerobic (6.1) and anaerobic conditions (6.2) as well as new motor skill learning (6.3) (or procedural memory).

Ultimately, in chapter 7 the conclusions of this work are presented. Moreover, possible future directions for this study are discussed.

The appendices show raw data of the all participants (Appendix A), a map, which shows the Brodmann areas of the human brain (Appendix B), the results of one-sample Kolmogorov-Smirnov tests (Appendix C), the questionnaire (Appendix D), declaration of consent (Appendix E).

2 Theoretical background

Brain aging probably starts already from adulthood onwards (Blalock et al., 2003; Jiang, Tsien, Schultz, & Hu, 2001; C.-K. Lee, Weindruch, & Prolla, 2000). Along with increasing age some deliberate changes arise in brain structure and function, such as decreases of neurogenesis and granule neurons (Altman & Das, 1965; Cameron & McKay, 1999; Fabel & Kempermann, 2008). Though, the determinants of the changes in age-related cognitive decline are not fully understood (Deary et al., 2009), anyhow some age-related decline in older adults' cognitive functions occur and the normal aging process does not degrade neural areas and cognitive processes similarly (Burke & Barnes, 2006; K. I. Erickson et al., 2009; Kramer et al., 1999; Mather & Carstensen, 2005; Small, 2001).
The normal aging is defined as usual and non-pathological processes of human life (Rowe & Kahn, 1997),

Some studies on executive function and brain regions showed large and disproportionate changes with age, but sometimes many older people did not present cortical atrophy; this, however, didn't indicate that such changes are certain consequence of advancing age (Buckner, 2004; Coffey et al., 1992; Persson et al., 2006; Sullivan, Marsh, Mathalon, Lim, & Pfefferbaum, 1995). Even though there are some interesting findings on aging and brain volume that suggest strong decline in tissue densities as a function of age (Stanley J Colcombe et al., 2003), available evidence is still limited and scarce. The following is probable for a non-demented aging process, executive function and memory performance decline may contribute to structural and functional changes of different brain regions including the medial temporal lobe (MTL) and fronto-striatal circuits (Buckner, 2004).

According to Hedden and Gabrieli, this may contribute to the prefrontal, MTL and white matter tracts (Hedden & Gabrieli, 2004); in other literatures, the frontal and prefrontal is named as a contributor (Kramer et al., 1999); also, the

prefrontal cortex and function of dopamine is mentioned in this context (Braver & Barch, 2002); hippocampal volume and the prefrontal cortex (Persson et al., 2006), even the amygdala is named as a contributor (Grieve, Clark, Williams, Peduto, & Gordon, 2005). Many researchers of human cognition and the aging process have suggested that typically older adults perform more poorly than young adults in terms of memory function (Churchill et al., 2002; Kramer, Larish, & Strayer, 1995; Salthouse, 1996; Small, 2001).

The memory system is one of the most vulnerable ones (Head et al., 2004; Mather & Carstensen, 2005). Prevention of memory-system damage or attempts to decrease the accelerating decline of human memory is plausibly more beneficial and easier than treatment. Considering vulnerability and sensitivity of cognitive functions and the memory system against factors such as the environment, the social, physiological and psychological setting (Teasdale, 1988), and considering the importance of independent life for elderly, researchers are designing and discovering strategies to continue healthy and independent life and also to delay the progression of physical and psychological illnesses.

It may be disputed that engaging in physical activity does not play a protective role on cognition and cognitive disorders, but there are many findings reporting regular physical exercise as an important element in health promotion (Larson & Wang, 2004) and as an effective strategy to delay the onset of dementia (Pate et al., 1995). For quite some time now effects of athletic exercise on cognitive function have received major attention (Blumenthal & Madden, 1988; Botwinick & Thompson, 1968; Green & Bavelier, 2008; Moul, Goldman, & Warren, 1995). By necessity, in this study, we will see a brief description of differences in the physiological kind of such exercises. Previous studies have shown that a program of aerobic training can improve mood (Berger & Owen, 1983; PILC, 2010), self-esteem (Hanson & Nedde, 1974a), cognitive function, and lessen cognitive decline among older adults (Kirk-Sanchez & McGough, 2014; Weuve et al., 2004).

It has also been reported that improvements in aerobic fitness may be related to improvements in performing of executive control processes. 124 subjects, 60 to 75 years old, previously sedentary, were examined over a period of six months. They were randomly assigned to perform either aerobic exercise – for example running and walking – or anaerobic exercises such as stretching and toning (Kramer et al., 1999). In other researches, strength and flexibility exercises were compared with an aerobic exercise program. The aerobically trained subjects demonstrated significant improvement in cerebral metabolic activity and achieved better results in the neuropsychological test battery than the control group (Dustman et al., 1984).

In general, there are studies that have failed to observe the benefits of physical exercise in preserving cognitive function (Broe et al., 1998; Madden, Blumenthal, Allen, & Emery, 1989). In future, we need to better understand effects of exercise on cognition. At the moment results are promising and suggest that physical activity, as a preventive strategy and neuroprotective function, may reduce declines in cognitive performance associated with the normal aging process (Grodstein, 2007; Kirk-Sanchez & McGough, 2014; Kramer, Colcombe, McAuley, Scalf, & Erickson, 2005; Pate et al., 1995). Thus, public policy should focus on ways of increasing self-initiated, lifestyle activity in older people, as well as on increasing the availability and accessibility of senior and community-center programs promoting physical activity (DiPietro, 2001).

2.1 Lifestyle and aging: Delay or acceleration of the aging process

Lifestyle, work, leisure activities and behaviors of individuals and factors associated with the environment may influence directly or indirectly the health and quality of the elderly life (Elavsky et al., 2005; McAuley et al., 2000; McAuley et al., 2006). Participation in physical exercise programs has been associated with the reduction of a number of physical and mental disorders (for

example, cardiovascular disease, obesity and several cancers, as well as depression and anxiety) across the adult lifespan (Hillman, Erickson, & Kramer, 2008). Unfortunately, the sedentary lifestyle (Booth, Chakravarthy, Gordon, & Spangenburg, 2002) and some dementia illnesses (Ewbank, 1999; Larson et al., 2004) are major causes for death among an increasingly aging society in the United States. Increased anti-fatigue ability, muscle weakness, decreased energy expenditure at rest and during exercise, increased body fat, decreased endurance capacity, and muscle wasting are commonly presented in the aging process, and this altogether may lead to decreased physical activity, and then eventually, through other pathways, to further diseases (Hunter, McCarthy, & Bamman, 2004; Stewart, 2005).

Human fitness training studies conducted over the past several decades have produced a varied pattern of results. This demonstrates that the loss of the brain volume in late adulthood is not inevitable and can be reversed with moderate-intensity exercise (K. I. Erickson et al., 2011). Some studies reported a positive relationship between fitness training and cognition (Davranche & McMorris, 2009; Dustman et al., 1984; Kirk-Sanchez & McGough, 2014; Kramer et al., 1999; Weuve et al., 2004) while other studies fail to observe such a relationship but encourage future research to focus on physiological and psychological variables that may serve to mediate the relationship between physical activity and cognitive performance (Etnier, Nowell, Landers, & Sibley, 2006). A study reviewed the effects of age and activity on the need for environmental support in samples of old adults over 75 years old. They found that self-reports of active lifestyle and exercise were positively correlated with memory performance (Hill, Wahlin, Winblad, & Bäckman, 1995). Also, it has been shown that the calorie intake (either the reduced calorie intake in dietary, or reduced activity-induce) can increase the resistance of neurons in the brain to dysfunction in the age-related mental diseases including Alzheimer, Parkinson and Huntington’s disease and stroke (Mattson, 2000).

Physical inactivity accelerates the aging process in many people, whereas increased physical activity slows it down in others (Abbott et al., 2004; Kramer, Erickson, & Colcombe, 2006; Laurin, Verreault, Lindsay, MacPherson, & Rockwood, 2001; Podewils et al., 2005). However, the rate of change is not equal among individuals. What is clear is that there are several modifiable mediating factors on the aging curve. Among modifiable key factors are physical activities, nutrition, body fat, muscle mass, and smoking, each of which can either delay or accelerate the aging process (Stewart, 2005).

In comparison with non-active adults, it is reported that active older adults succeed in cognitive performance as well in counteracting age-related neural decline through a plasticity reformation of neurocognitive networks (Cabeza, 2002; Cabeza, Anderson, Locantore, & McIntosh, 2002). Also, reviews of brain bilateral activity have shown that these activities could improve brain function and cognitive performance. Elderly who displayed a bilateral pattern of prefrontal cortex activity were faster in the verbal working memory (WM) task than those who did not display this pattern (Reuter-Lorenz et al., 2000).

The overactivation as an additional activity serves a beneficial, compensatory function. Determining whether levels of overactivation is beneficial, detrimental or inconsequential in cognitive function on older adults is still the crux of much cognitive and aging research (Reuter-Lorenz & Cappell, 2008; Reuter-Lorenz & Lustig, 2005). There is some evidence on the relationship between low and high fitness, and on cognition performance. A review found that high-fitness individuals were able to perform more quickly and accurately on a wide variety of perceptual, cognitive and motor tasks compared to low-fitness individuals (Etnier et al., 1997).

Some studies have examined the relationship between the amount of physical activity and cognition function. One review studied a large sample of women older than 65 years who were recruited at four clinical centers and were without baseline cognitive impairment or physical limitations. They reported

women with higher levels of baseline physical activity were less likely to experience cognitive decline during the six to eight years of follow-up, indeed, cognitive decline induces more increase when participants were less active (Yaffe, Barnes, Nevitt, Lui, & Covinsky, 2001).

In some perusals physical fitness has been studied, and in others physical activity; so it is important to keep these differences in mind when evaluating findings to understand the distinction between the terms “physical fitness” and “physical activity” and also the relation between “fitness” to “health” and “performance” (Stewart, 2005).

2.2 Exercise and metabolism

Exercise is inherently associated with energy metabolism. The human body obtains the energy needed for exercise from the breakdown of carbohydrate and fatty acids through various biochemical pathways to produce energy in the form of adenosine triphosphate (ATP) (R. K. Murray, 1996; Shahbazi & Maleknia, 2004). The relative amount of energy used by the muscles depends on the type, duration and intensity of the exercise and also on the fitness level of the individual. Since human muscles can’t store too much ATP, human must constantly resynthesize ATP through three metabolic pathways that consist of many enzyme-catalyzed chemical reactions including two anaerobic systems, the phosphagen and anaerobic glycolysis and also the aerobic system (Karp, 2009; Mathews, Fox, & Close, 1976; Morton, 2006; Shahbazi & Maleknia, 1999).

2.2.1 Phosphagen system

In the phosphagen system, throughout short activities – that is, five to six seconds – and intense activities, a vast amount of power needs to be produced by the muscles creating a high demand for ATP, which, in turn, creates a demand for ATP. The system also called the ATP-PC system and this is the most rapid way

to regenerate ATP (Glaister, 2005; McCartney et al., 1986). Creatine phosphate (CP), which is stored in skeletal muscles, donates a phosphate to adenosine diphosphate (ADP) to produce ATP[1]. No foodstuffs e.g. fat or carbohydrate is used in this process; the renewal of ATP comes solely from stored CP. Since this process does not need oxygen to resynthesize ATP and is not oxygen-independent, it is called anaerobic. As the fastest way to regenerate ATP, the phosphagen system is the predominant energy system used for maximum exercise lasting up to about ten to 30 seconds. Because there is a limited amount of stored ATP-PC in skeletal muscles, fatigue occurs in a few seconds (Baker, McCormick, & Robergs, 2010; Enoka & Stuart, 1992). Indeed, this is the primary system after very short, powerful movements like a powerlifting, powerful jumping, and a 100 meters sprint (Mathews et al., 1976).

2.2.2 Anaerobic glycolysis

Anaerobic glycolysis transforms glucose to lactate (lactic acid or $C_3H_6O_3$) if enough amounts of oxygen are not available (R. K. Murray, 1996). Glycolysis[2] is the main energy system used for an exercise lasting from 30 seconds to about a few minutes – however less than four minutes that is the second-fastest way to resynthesize ATP (Fox, 1984). During glycolysis, carbohydrate, either in the form of blood glucose ($C_6H_{12}O_6$) or muscle glycogen[3] is broken down into two

[1] ATP- CP is an abbreviation for adenosine triphosphate and Creatine phosphate. During this breakdown of ATP, which is a water-requiring process, a proton, energy and heat are produced.

$$ATP + H_2O \xrightarrow{ATPase} ADP + Pi + H^+ + E \textit{ for muscle contraction}$$
$$E + ADP + Pi \xrightarrow{ATP\ synthase} ATP$$
$$PCr \xrightarrow{Creatine\ Phosphokinase} Cr + Pi + E$$
$$E + Cr + Pi \xrightarrow{creatin\ kinase} PCr$$
$$PCr + ADP + H^+ \xrightarrow{Creatine\ kinase} ATP + Cr$$

[2] Glycolysis: to break glucose down into two pyruvates, glycolysis produces 4 ATP's and 2 NADH, but uses 2 ATP's in the process for a net of 2 ATP and 2 NADH. Also each NADH produces energy worth of 2 ATP.

[3] Glucose of muscle, first broken down into glucose-6-P

different form of Lactate[4] or pyruvate[5] through a succession of biochemical reactions. When broken down to pyruvate through glycolysis, two/three ATP molecules are produced – that is, for each glucose molecule – as well as two molecules of nicotinamide adenine dinucleotide (NADH). Mainly glycolysis produces four ATP but one or two are used to fuel NADH the process. Depending on kind of fuel the cell gains two or three ATP[6] (Baker et al., 2010; Glaister, 2005; McCartney et al., 1986; R. K. Murray, 1996; Shahbazi & Maleknia, 1999, 2004).

Thus, very little energy is produced through this way, but we get the energy nearly fast. Once pyruvate is formed, it has two destinies; in default of oxygen (O_2) is converted to lactate by Lactate dehydrogenase (LDH)[7] and in the presence of O_2, pyruvate via PDC[8] is converted to a metabolic intermediary molecule called acetyl coenzyme A (acetyl-CoA) (Bowker-Kinley, Davis, Wu, HARRIS, & POPOV, 1998; Linn, Pettit, & Reed, 1969), which then enters the mitochondria to produce more ATP (Baker et al., 2010; R. K. Murray, 1996; Robergs & Roberts, 1997). Is the oxygen supply lower than the demand, then

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[4] Lactic acid ($C_3H_6O_3$) is a chemical compound that doesn't produce from complete broken down of glucose in anaerobic activities and causes an increasing of (+) (lowering of PH) and can be decreased (prevention of Ca^{++} entrance) to ability in muscle contraction.

[5] $C_3H_4O_3$ or Pyruvic acid is an organic acid can be produce from glucose during glycolysis; it is a key intersection in several metabolic pathways. Pyruvate via LDH is converted to lactate ($C_3H_6O_3$): ($C_3H_4O_3 + NADH + H^+ \xrightarrow{LDH} C_3H_6O_3 + NAD^+$).

$$C_3H_4O_3 + NADH + H^+ \xrightarrow{LDH} C_3H_6O_3 + NAD^+$$

[6] There are a variety of beginning points – glucose or glycogen – for glycolysis and produce glucose-6-phosphate. At the steps one and three, in total two ATP are required:The first step: Glucose + ATP $\xrightarrow{hexokinase}$ ADP + Glucose 6-phosphate. The first step is only for glucose and not for glycogen. Thus, glucose needs two ATP as fuel (4-2=2ATP) but glycogen only one, because glycogen starts after the first step.The third step: Fructose 6-phosphate + ATP $\xrightarrow{phosphofructokinase}$ ADP + Fructose 1, 6-bisphosphate

[7] LDH is an enzyme that transfers a hydride from one molecule to another. LDH catalyzes the conversion of pyruvate to lactate and back, as it converts NADH to NAD^+ and back. ($C_3H_4O_3 + NADH + H^+ \rightarrow C_3H_6O_3 + NAD^+$)

[8] Pyruvate dehydrogenase complex (PDC) is a complex of three enzymes included pyruvate dehydrogenase, oxidizes hydroxyethyl and dihydrolipoyl dehydrogenase, which provide alternative fuels.

lactate conversion takes place (i.e., during the 800 and 1,500-metre run). In anaerobic glycolysis, when O_2 is not supplied fast enough to meet the muscles' needs, there is an increase in hydrogen ions, which leads to concomitant decline in the concentration of Ca^{++} which is very essential muscle contraction process (Kowalchuk, Heigenhauser, Lindinger, Sutton, & Jones, 1988; Stackhouse, Reisman, & Binder-Macleod, 2001).

2.2.3 Aerobic system

Since human is adapted for aerobic activities, it's not surprising that human body is dependent on O_2 and aerobic system is the most complex of the three energy systems (Hochachka, Gunga, & Kirsch, 1998; Kulkarni, Kuppusamy, & Parinandi, 2007).

The system through aerobic respiration – the process by which a cell uses O_2 to burn molecules – release energy; in the aerobic respiration, the more carbon atoms would be in the molecule, they can release more energy but need more O_2. The chemical composition for carbohydrates e.g. glucose differs from fats such as palmitic acid. Those fats contain considerably fewer O_2 atoms in proportion to atoms of carbon and hydrogen[9].

Although, aerobic system produces the most of the cellular energy[10], aerobic metabolism is the slowest way to resynthesize ATP (Fox et al., 1975; Mathews et al., 1976; Romick, Fleming, & McFeeters, 1996). In the presence of oxygen, and as another destiny, pyruvate is converted to acetyl-CoA, which enters the mitochondria for oxidation and the production of more ATP. Indeed, when there is enough O_2 available to meet the needs of the muscles (i.e., during

[9] Comparison carbohydrates e.g. glucose and lipids such as palmitic acid ($C_{16}H_{32}O_2$) in the aerobic respiration:

$$\text{Glucose: } C_6H_{12}O_6 + 6\ O_2 \rightarrow 6\ CO_2 + 6\ H_2O$$
$$\text{Palmitic acid: } C_{16}H_{32}O_2 + 23\ O_2 \rightarrow 16\ CO_2 + 16\ H_2O$$

[10] Glucose or $C_6H_{12}O_6 + 6\ O_2 + 38\ ADP + 38\ Pi \rightarrow 6\ CO_2 + 6\ H_2O + 38\ ATP$, this is only for plants, which don't spend an ATP to transport NADH into the mitochondria.

the 10,000-meter run and marathon), pyruvate moves through the aerobic metabolism via acetyl-CoA. The aerobic system includes glycolysis, the Krebs cycle and Oxidative Phosphorylation (Mathews et al., 1976).

2.2.3.1 Glycolysis

In the presence of oxygen, the first stage is known as the aerobic glycolysis. Glycolysis occurs in the sarcoplasm of muscle cells and the normal body cells' cytoplasm, and to move onto to the next stage of metabolism, two pyruvate molecules, two reduced molecules of NADH and two ATP molecules need to be produced. (Shahbazi & Maleknia, 1999). In sum, glycolysis produces four ATP and two NADH, but uses two ATP in the process for a net of two ATP and two NADH (Baker et al., 2010; R. K. Murray, 1996; Shahbazi & Maleknia, 2004).

2.2.3.2 Krebs cycle[11]

In the second stage which is called the Krebs cycle, there are two steps, includes the conversion of Pyruvate to Acetyl CoA and the complete oxidation of acetyl-CoA into two molecules of carbon dioxide (CO_2). In this cycle, two ATP's, two $FADH_2$'s, eight NADH's[12], and six CO_2's per glucose molecule are also produced plus the conversion of pyruvate (R. Murray, Granner, & Rodwell, 2000; Shahbazi & Maleknia, 2004). This stage takes place in the matrix of the cells' mitochondria.

2.2.3.3 Electron Transport or Respiratory Chain

[11] It called also citric acid cycle as well as tricarboxylic acid cycle (TCA cycle), also in this cycle produces one ATP per each CO_2 molecule: in total, two ATP.

[12] The molecules of Nicotinamide adenine dinucleotide (NAD) and Flavin adenine dinucleotide (FAD) are coenzymes found in living cells and involved in redox reactions, carrying electrons from one reaction to another.

Electron Transport chain (ETC) or oxidative phosphorylation is the last stage of the aerobic system, that leads to breaking down of NADH and $FADH_2$ as well as pumping H^+ into the external section of the mitochondria. The majority of the energy generated during aerobic reactions occurs near the end of the metabolic series of reaction (Baker et al., 2010; M. Brand, 2005; Glaister, 2005; Karp, 2009). The hydrogen ions are released during glycolysis and also Krebs cycle should be removed to prevent the acidification of the cell. In this reaction, the ETS creates a gradient which is used to produce ATP (R. K. Murray, 1996; Shahbazi & Maleknia, 1999).

In ETC, couple of reactions occur between NADH and $FADH_2$ as an electron (e^-) donor and an O_2 as electron acceptor to transfer H^+ ions across a membrane, through a set of mediating biochemical reactions that leads to oxidation and form a water molecule (Kregel & Zhang, 2007; Kulkarni et al., 2007; Shahbazi & Maleknia, 2004). The role of oxygen as a final electron receptor in cellular respiration is substantial. The system, through transportation of electrons, meets up with the oxygen of respiration at the end of the chain which is responsible for removing electrons from the system[13]. Without oxygen, the proton pump could not be created, and ATP could not be produced. These H^+ are used to produce ATP. The ATP generated as H^+ moves down its concentration gradient through a special enzyme called ATP synthesis[14]. These redox reactions release energy, which is used to form ATP (M. Brand, 2000, 2005; Stowe & Camara, 2009).

In the chloroplasts of plants, light causes the conversion of water to O_2. The transfer of H^+ in the metabolism of the mitochondria is caused by the

[13] $2\ H^+ + 2\ e^- + {}^1/_2\ O_2 \rightarrow\ H_2O + E$ Notice: 2 hydrogen ions, 2 electrons, and half a mole of oxygen react to form as a product water with energy released in an exothermic reaction ($2\ H_2O \rightarrow 4\ H^+ + 4\ e^- + O_2$)

[14] ATP synthase is also readily reversible (ADP and Phosphate (Pi) are joined together by ATP synthase). Indeed, this is bilateral reaction (see biochemistry references).
$ATP \xrightarrow{ATPase} ADP + Pi + E \rightleftarrows ADP + Pi \xrightarrow{ATPsynthase} ATP$

conversion of O_2 to H_2O and NADH to NAD^+ (R. K. Murray, 1996; Shahbazi & Maleknia, 1999). Hence, the stage is called the electron transport or oxidative phosphorylation, whereas oxygen is the final acceptor of the electrons and hydrogen ions disappear in the stage of aerobic respiration and also because ADP gets phosphorylated to form ATP (Baker et al., 2010; Romick et al., 1996; Shahbazi & Maleknia, 2004). Till now, the first two rounds of aerobic system (aerobic glycolysis and Krebs cycle) have produced only four ATPs and a number of coenzymes.

Complete oxidation of carbohydrates, e.g. a glucose molecule can produce 34 molecules of ATP. In lieu of transportation of each NADH molecule in glycolysis, the conversion of pyruvate to acetyl CoA and Krebs Cycle produce three ATP – but in glycolysis, it costs one ATP to transport the NADH into the mitochondria – and instead, each $FADH_2$ transportation provide the energy needed to resynthesize worth of two ATPs[15] (Baker et al., 2010; M. Brand, 2005; Shahbazi & Maleknia, 1999).

Oxidative phosphorylation is a key part of the aerobic metabolism, also, it facilitates to the propagation of free radicals, leads to the damaging of cells and contributes to diseases and, in cases, encourages the aging process (Blalock et al., 2004; Kregel & Zhang, 2007; Kulkarni et al., 2007; Loerch et al., 2008; Lu et al., 2004).

[15] Since it is against the concentration gradient, using a form of active transport, two ATP molecules are used to force the two pyruvate molecules into the mitochondrion. Complete oxidation of carbohydrates in ETC can yield 32 ATP: Glycolysis yield energy worth of 2 ATP and the Krebs cycle produce also energy worth 2 ATP (2 + 2 = 4). Eight NADH are produced energy worth of 24 ATP – Each NADH produced in the conversion of pyruvate to acetyl CoA and Krebs Cycle is worth 3 ATP ($8 \times 3 = 24$) – and 2 $FADH_2$ produce energy worth of 4 ATP – Each $FADH_2$ is worth 2 ATP – ($2 \times 2 = 4$), thus, theoretically, glycolysis yield in total 32 APT (4 + 24 + 4= 32).

2.3 Exercise and physical activity

In this section, the notion of physical activity and related concepts mentioned in this study such as physical fitness, intensity and type of exercise are elaborated.

2.3.1 Physical fitness

Physiological processes in human body are affected by conditions of lifestyle (Booth et al., 2002; Elavsky et al., 2005; McAuley et al., 2000; McAuley et al., 2006). Exercise can improve levels of athletic performance via adaptation to new conditions (Burgomaster et al., 2008; Holloszy & Coyle, 1984; Mathews et al., 1976).

In sports context, there are some terms that have a variety of possible meanings and are often used interchangeably. Indeed, a human body's motion produced by skeletal muscles in connection with energy expenditure can be defined as physical activity, while physical exercise is every planned and purposeful physical activity that enhances or maintains physical fitness components to create coordination with new conditions (Caspersen, Powell, & Christenson, 1985; Corbin, Pangrazi, & Franks, 2000; Shephard & Balady, 1999). Physical fitness is defined as a general state of health. Among its main merits ranks the ability to perform physical activities such as sports. It is correct nutrition, training, hygiene, et cetera, leading to this state. Physical fitness components can be divided into health-related and skill-related (Caspersen et al., 1985).

The health-related physical fitness components are defined as cardiovascular endurance, muscle strength, muscle endurance, flexibility and body composition. Also, there are some other components of physical fitness that are more skill-related and include agility, balance, coordination, power, reaction time, and speed. These components can be measured and improved using definite

training techniques (Caspersen et al., 1985; Corbin et al., 2000). It is important to know that all components of physical fitness cannot be storable but can be changed, regardless of the stage of life (Caspersen et al., 1985; Fox, 1984; Mathews et al., 1976).

Physiological changes of exercise on muscles and bodily functions e.g. strengthening muscles, muscle development – through exercise programs that often occurs in conjunction with the reduction of body fat – improving cardiovascular system, VO_{2max}, blood pressure, improving motor skills, body composition – by two mechanisms of exercise. First, through a reduction in the percentage of body fat, and second through the increase of lean muscle mass developed –, ideal and healthy weight, physical activity affects on some other aspects of human life such as affective and behavioral responses (Berger & Owen, 1983; Hanson & Nedde, 1974b; McAuley, Jerome, Marquez, Elavsky, & Blissmer, 2003; PILC, 2010; Sebire, Standage, & Vansteenkiste, 2009; Tomporowski & Ellis, 1984), mental health (Folkins & Sime, 1981), and cognitive function (Dustman et al., 1984; Heyn, Abreu, & Ottenbacher, 2004; Kramer et al., 2006; Lowe, Hall, Vincent, & Luu, 2014; Tomporowski, 2003; Weuve et al., 2004).

It should be noted that terms such as sport, physical education as well as recreational and leisure-time activities are quite different (Kirk & Macdonald, 1998; Messner & Sabo, 1990; Winnick, 2011) but they are often confused with other one. A physical activity can range from a walking in park as an activity at leisure-time to running as a competitor in the running match. However, the nature and purpose of physical education, sport and leisure-time activities are not the same. The purpose of physical education is the educational goals related to individual development, while the sport goal is the achievement of motor behavior performance as high as required to win over a rival or sometimes for self-competition. The learning content in physical education is relatively

adaptable to persons' ability level, while in the content of sports training there are targets to be met.

Learning oriented physical education is centered on persons' will, while in sport; the person is responsible for achieving favorable results in match. This means that, in physical education, if the person may not reach the destination in the given time, unlimited times to try over again is available, while in sport there would be no second chance. Also, leisure-time activities are activities that people usually engage in during their free time merely to enjoy, and it encompasses various reasons such as relaxation, competition, or growth and may include playing for pleasure and participating in sports. Those are not objective oriented and don't involve ordinary life e.g. sleeping or cleaning (Caspersen et al., 1985; Chen & Ennis, 2004; Corbin et al., 2000; Haskell et al., 2007; Haskell, Montoye, & Orenstein, 1985; Mathews et al., 1976).

2.3.2 Exercise intensity

Although, decline in cognition and memory function are associated with the reduction of adult hippocampal neurogenesis, physical activity promotes neurogenesis which improves memory function and learning (Cotman & Berchtold, 2002; Kempermann et al., 2010). It has been found that variety of physical activities intensities creates different effects on adaptation to exercise; even excessive exercise can be harmful (Fox, 1984; Mathews et al., 1976; Radak, Chung, & Goto, 2008). In human studies on relationships between the effects of physical exercise and cognitive abilities, from the viewpoint of sport physiology, we should consider several possibilities including: intensity and duration of exercises and the distinction between involved energy systems (Fox, 1984; Mathews et al., 1976; Tomporowski & Ellis, 1986).

It has been reported that cognitive function was affected differently by exercise activity. This means that complex relationships exist between exercise

and cognition. Lambourne and Tomporowski (2010) found that the effect of exercise on cognitive function can be different depending on time of measurement, type of cognitive performance, and type of exercise (Lambourne & Tomporowski, 2010). When describing the amount of physical exercise, there is a notable relationship between the total dose of activity and the intensity of the performed activity (Howley, 2001; Shephard & Balady, 1999).

The terms "dose" or "volume" designate the amount of energy used in exercises which require repetitive muscular activity (Mathews et al., 1976; Shephard & Balady, 1999). Activity intensity refers to the amount of energy expended when exercising. This amount differs depending on whether it's basal energy that is consumed or if it is an exhausting exercise (Fox, 1984).

Exercise intensity is divided into two forms: relative and absolute. The term "relative intensity" is used in the event of a certain percentage of maximal oxygen consumption (%$VO_{2\ max}$) or during maximal heart rate (%HR_{max}), while "absolute intensity" is used when a particular amount of oxygen is consumed per minute (Fox, 1984; Shephard & Balady, 1999). For example, jogging at 4.8 km/h has an absolute intensity of approximately 4 METs[16]. In relative terms, this intensity may be considered light for a healthy young student, though, it must be considered hard for an elderly pensioner[17] (Arriaza Jones et al., 1998; Howley, 2001; Jette, Sidney, & Blümchen, 1990; Shephard & Balady, 1999). Determining whether level of exercise intensity is beneficial, detrimental or inconsequential in cognitive function on older adults is still the crux of much cognitive and aging research (Reuter-Lorenz & Cappell, 2008).

[16] One metabolic equivalent (MET) is defined as the quantity of oxygen consumed while sitting at rest and is equal to 3.5 ml oxygen per kg of body weight per min (3.5 ml O_2 • kg^{-1}•min^{-1}) or (3.5 ml O_2/kg/min). One MET equals the expenditure of 14.6 kJ • kg^{-1} • min^{-1}; 3.5 kcal • kg^{-1} • min^{-1} can be achieved during exercise tests that are graded.

[17] Moderate-intensity activity was defined as activity performed at an intensity of three to six METs.

Since the physical activity intensity may be very different and perceived intensity varies for each individual, hence for a better understanding, it is categorized into low, moderate, and vigorous intensity levels. Either physical inactivity or strenuous exercise leads to increased incidence of a variety of diseases, while regular-moderate physical activity provides a wide range of beneficial effects including, improved physiological function, decreased incidence of disease and a higher quality of life (Radak, Chung, & Goto, 2008; Radak, Chung, Koltai, Taylor, & Goto, 2008).

Studies examining the intensity of exercise required to optimize neurotrophins[18] suggest that cognitive function scores improved after all exercise, but the amount of increase depends on exercise intensity (Ferris, Williams, & Shen, 2007) and it has been reported that continuous increases in neurotrophins levels occur with prolonged low to moderate intensity exercise (Ploughman, 2008).

Young et al., (1999) reported that light and moderate aerobic exercises may have similar effects on cardiovascular factors e.g. blood pressure in previously sedentary older adults. Also, their results revealed that maximal aerobic capacity tended to increase in aerobic exercise (D. R. Young, Appel, Jee, & Miller 3rd, 1999). Lybrand et al. (1954) measured the effects of an exhausting endurance march with about 18 kg packs on the perceptual organization ability of college students. They found that perceptual scores on tasks were higher after mild physical activity than under non-exercise and sleep-deprivation conditions (Lybrand, Andrews, & Ross, 1954).

Tomporowski et al., (1985) have assessed the effects of a strenuous aerobic run to exhaustion on memory. 24 college students classified as of average cardiovascular fitness (mean $VO_{2\ max}$ = 44.7 ml/min/kg) running to exhaustion at 80 percent of their $VO_{2\ max}$. The average time of subjects spent on the treadmill

[18] Neurotrophins are proteins that induce the survival, development, and function of neurons.

was 27.93 minutes immediately after their activity; subjects performed a series of twelve free-recall tests of memory over a 60-minutes period. Post-exercise test scores were compared with the performance of a non-exercised as control group. A slight facilitation in the performance of the exercised subjects was noted during the first 30 min. of the test period; however, the change was not statistically significant (Tomporowski & Ellis, 1986).

In this study, the conception of physical exercise is a low to moderate physical activity without any competition and also risk-free for the study participants. Indeed, there was no competitional physical training or activity as leisure.

2.3.3 Kinds of exercises

Although, there are several physical activity types such as endurance, strength, balance, and flexibility, but since there are only two sources to the energy supply system for human physical activity, thus, exercises are divided into two general categories including aerobic and anaerobic (Morton, 2006).
Aerobic exercise is any physical activity that uses large muscle groups for long time (e.g. more than ten minutes), and causes body to use more oxygen than it would do while resting. The primary goals of aerobic exercises are improvement and maintenance of health, lowering the risk of disease, and managing fitness and weight (Fox et al., 1975; Mathews et al., 1976).

Aerobic exercises induce faster breathing and take in more oxygen, – aerobic conditioning is a key factor in losing or managing weight by lipolysis and Beta-oxidation[19] – improves cardiovascular fitness, up-regulates the immune system, reduces cognitive decline possibly by enhancing concentration of

[19] Lipolysis and Beta-oxidation are the breakdown of lipids and fatty acid in the mitochondria to generate acetyl-coA and NADH and $FADH_2$, which are used by ETC, to produce ATP.

neurotrophins as well as creating greater emotional equanimity as benefits of aerobic conditioning (Haskell et al., 2007; Haskell et al., 1985).
Examples of aerobic training include cycling, swimming walking, long slow runs, rowing, require a great deal of oxygen to generate the energy needed for prolonged exercise (Fox, 1984; Mathews et al., 1976).

Anaerobic exercise is also called Strength or Resistance training. There are two types of anaerobic energy systems: ATP-PC and Anaerobic glycolysis. Anaerobic exercises also include weight training, functional training, eccentric training, Interval training, sprinting. High-intensity interval training increases short-term muscle strength (Fox et al., 1975; Mathews et al., 1976). In this study the term “anaerobic” will be used in the same way as “non-aerobic”.

2.4 Memory

In psychology, memory is defined as a process in which information is registered and encoded, consolidated and stored, and ultimately recalled and retrieved (Jensen & Lisman, 2005; Markowitsch, 2003; Sara, 2000; Tulving & Markowitsch, 1997). Memory is not a single entity but it is a complex system of separate entities which depend on different brain systems (Squire & Zola, 1996; Tulving & Markowitsch, 1998). Functional imaging studies of the human brain indicated multiple memory systems with different functions and separate anatomical organizations (Andrews-Hanna et al., 2007; Bishop, Lu, & Yankner, 2010); there is an understanding that cognitive decline in normal aging arises from functional disruption in the coordination of brain systems that support cognitive function (Andrews-Hanna et al., 2007).

Memory process starts with perception of stimuli through the senses (Shiffrin & Atkinson, 1969). The perceived information may be conscious or unconscious (Markowitsch, 2013). Then, registered information is transferred through a specific code into human memory processing. The role of frontal cortex and specifically the dorsal extent of the inferior frontal gyrus (BA 6/44) have been explained in this stage (McDermott, Buckner, Petersen, Kelley, & Sanders, 1999). A selection of registered information allows continuing the memory process via attention. Encoding is maybe the first voluntary stage of a new memory. Indeed, encoding is the process of putting information into memory for storage. Encoding process can occur deliberately or accidental (Kapur et al., 1994). Figure 1 shows the main processes from registration until recall and retrieval of information.

Some types of encoding encompass acoustic, visual and semantic encodings. The acoustic type is encoding of sounds, words or other auditory information for storage and retrieval (Baddeley, 2003). In this kind of encoding, the phonological loop allows input within echoic memory to be rehearsed in

order to facilitate remembering (A. Baddeley, 2000; Baddeley, Gathercole, & Papagno, 1998).

Visual encoding is the process of encoding images and visual sensory input. Visual items are temporarily stored within the iconic memory before being encoded into long-term storage. The amygdala plays an important role in visual encoding (Sperling, 1960). Semantic encoding is a specific process of encoding in which the meaning of particular information, for example a word, phrase, picture or event. Words with semantic and deep meaning can recall the information better than both easy and hard of non-semantic (Craik & Tulving, 1975).

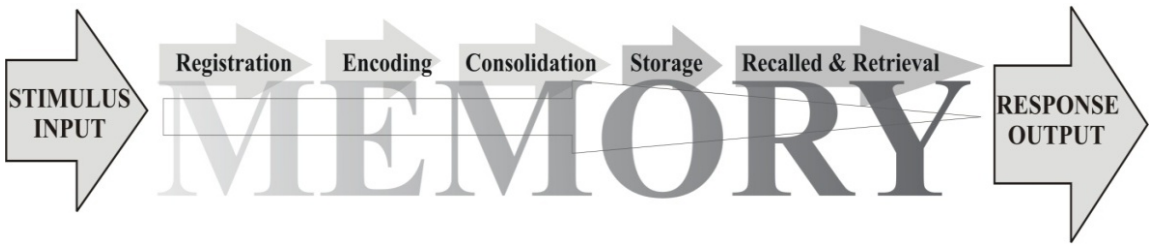

Figure 1: Illustration of the main storage process (modified from Markowitsch, 2003)

Learning process in procedural memory is ongoing and gradual (Ullman, 2004) and the rules of material apply automatically (Squire & Zola, 1996). So, it is acceptable that encoding of motor skill occurs on a constant basis during multiple performances of stimuli and responses.

Consolidation process expresses the neural processes of transpiring newly encoded information that contribute to the permanent storage of memory (Alberini, 2005; Nadel & Moscovitch, 1997). The processes of consolidation may last minutes to hours, months and even years (McGaugh, 2000). This stage of memory is commonly considered in two specific parts. One of them occurs in all memory systems within the first minutes to hours after learning and encoding

process and is called synaptic consolidation. Another one is called system consolidation and it lasts much longer than synaptic consolidation. Many types of memories depend upon hippocampal processing during the first few weeks and may become hippocampal-independent (Dudai, 2004). Hippocampal processing plays a time-limited role – first few weeks – in the consolidation of some types of memory (Anagnostaras, Maren, & Fanselow, 1999; Knowlton & Fanselow, 1998; Zola-Morgan & Squire, 1990).

In consolidation stage, the stability of the new memory formation is enhanced (McGaugh, 2000). During consolidation process, protein synthesis is required to transform newly learned information into stable changes (Alberini, 2005; McGaugh, 2000). Insights into the neurology of the process of new memory consolidation utilizes a phenomenon called long-term potentiation (LTP) provides an important key to understanding the mechanisms which induces memories which are formed and stored (Teyler & DiScenna, 1987). Indeed, LTP makes it possible for synapses to grow in strength as advanced numbers of signals are passed between the two neurons.

Since, sleep can play an important role in the plastic cerebral changes that underlie learning and memory (Maquet, 2001). It has been shown that sleep might enhance the consolidation of declarative memories, but do not improve procedural memory consolidation of hippocampus-independent (Rasch, Büchel, Gais, & Born, 2007). However, there is a misconception that procedural memory do not depend on hippocampal function, studies using fMRI have indicated that hippocampal activation during declarative memory and motor skill learning at least at initial skill acquisition engages hippocampal function (Schendan, Searl, Melrose, & Stern, 2003). Thus, there is currently evidence that sleep promotes the consolidation of declarative memory and motor skill learning (Diekelmann, Wilhelm, & Born, 2009; Marshall & Born, 2007).

Connection between the changes in synaptic function and both declarative memory and motor skill learning in the neural system supports the view that modifications in synaptic strength may be a symbol of a general mechanism of memory storing (Mayford, Siegelbaum, & Kandel, 2012). Memory storage is a extensive continuous biological process, which requires new proteins synthesis after learning (Kandel, 2001). In addition, altered protein synthesis, growth of new synaptic connections play a role in memory storing stage (Bailey, Bartsch, & Kandel, 1996). Via engrams, which are defined as the storage of new memory traces in the neuronal network (Markowitsch, Emmans, Irle, Streicher, & Preilowski, 1985; Penfield, 1968); it appears that, the new learned materials will embed through the neuromorphological changes and protein synthesis in the brain and other neural tissues (Markowitsch, 2013).

Certainly, memories are not separately stored in the brain like books on library shelves. Also, memories can be a combination of content-different information. Since each kinds of memories are stored in different areas of the brain. Interestingly, sometimes, retrieval of stored information requires new nerve pathways in the brain and not the pathways formed during encoding process (P. C. Fletcher et al., 1995).

Retrieval is final presentation of memory process. Because, after memories are encoded, consolidated and stored, without recalling the stored information, the memory is a meaningless process. The recovery of memory which is also called ecphory (Markowitsch, 2013) is the last stage in memory process that reactivates memory traces and may come to pass through one specific trigger. The retrieval may occur without external help and cueing, like a process in which a person is given a list of items to remember and then is asked to recall them (Tulving, 1967; Tulving & Colotla, 1970). It is called free retrieval. While, in cued recall, to retrieve the items presented previously, the person receives a cue as external stimuli. For example when we hear the first

letter of a word then we recall the whole complete word that we searched (Markowitsch, 2013).

Free retrieval displays evidence of primacy and recency effects. When the person can remember items stored at the beginning of the list earlier and more often, it indicates that they were already transferred via primacy effect into long-term memory. while, recency effect occurs when last items of a long list is remembered better (Markowitsch, 2013).

Also, contiguity effect shows that persons who more effectively form and retrieve relations between items that happen nearby in time encoding perform better in episodic retrieval tasks (Sederberg, Miller, Howard, & Kahana, 2010). Also, in expressing the effects of environmental context change on memory process, it has been stated that memory retrieval of integrated-imagery items is better when the environments are similar in both the encoding and recall phases (Eich, 1985). Indeed, suggesting that similarities of context cues during recall process are important.

In procedural system, unlike declarative memory, it appears that those information retrieval are encapsulated (Squire & Zola, 1996). Therefore, recalling of motor skill is triggered by the stimulus without conscious control.

2.4.1 Memory taxonomies

Memory is commonly classified into a number of forms. In next section, two of them have been explained based on subdividing the memory according to the duration or content.

2.4.1.1 Duration and time dependent memory

Regarding the duration and relation between memory and time, memory is generally classified to short-term (STM) and long-term memory (LTM), although, to this classification one may add the sensory store – ultra-short-term memory or sensory memory – as well as working memory (WM) which is connected with STM. The thought of the division of memory into short- and long-term dates back to the last century. However, this division is disputed. Clear boundaries between STM and LTM are difficult to pinpoint; researchers proposed that, in fact, clear-cut distinctions have not been made (Tarnow, 2008, 2009). Markowitsch (1999) believed that some information can last for just few minutes, while some of them last forever (Markowitsch, 1999).

Broadbent (1984) proposed a model consisting of four different stores including a sensory store, a short-term store, a long-term store and a motor output store (Cowan, 1997). Sensory memory is the shortest-term part of memory. It acts for stimuli received through the human senses e.g. vision; although, information are retained accurately, they are very brief (Sperling, 1960). In general, the brain has developed to process information which may be needed later. Thus, sensory memory is an ultra-short-term memory and decays quickly, typically, in about 200-300 milliseconds; indeed, less than a second past perception (Loftus, Duncan, & Gehrig, 1992). Because of this short-term nature it is often understood as part of the perception process itself. Also, it is an important stage for STM storage (Atkinson & Shiffrin, 1968). An example is the remembrance and recognition of something after only a split-second of observation. When a stimulus is noticed it can either be deliberately ignored or enter sensory memory. Sensory memory does not require any conscious attention and is often considered to be outside of conscious control (Schmidt, 1990). Unlike other types of memory, the sensory memory cannot be extended by rehearsal (Cowan, 1997).

Atkinson and shiffrin (1968) explained a multi-store model, which assumed that all memories pass from a short-term to a long-term store after a short time. Figure 2 shows the multi-store model of memory processes. This model assumes that information flows through three different stages before it gets stored in memory. The information first enters the sensory store, which can hold large amounts of data for one or two seconds. Information that gets selected for further processing moves on to the STM. The final destination is LTM, which can hold apparently unlimited amounts of information for an unlimited amount of time (Atkinson & Shiffrin, 1968; Shiffrin & Atkinson, 1969). Information in STM can become LTM through the process of consolidation, involving rehearsal and meaningful association (Baddeley, 1992).

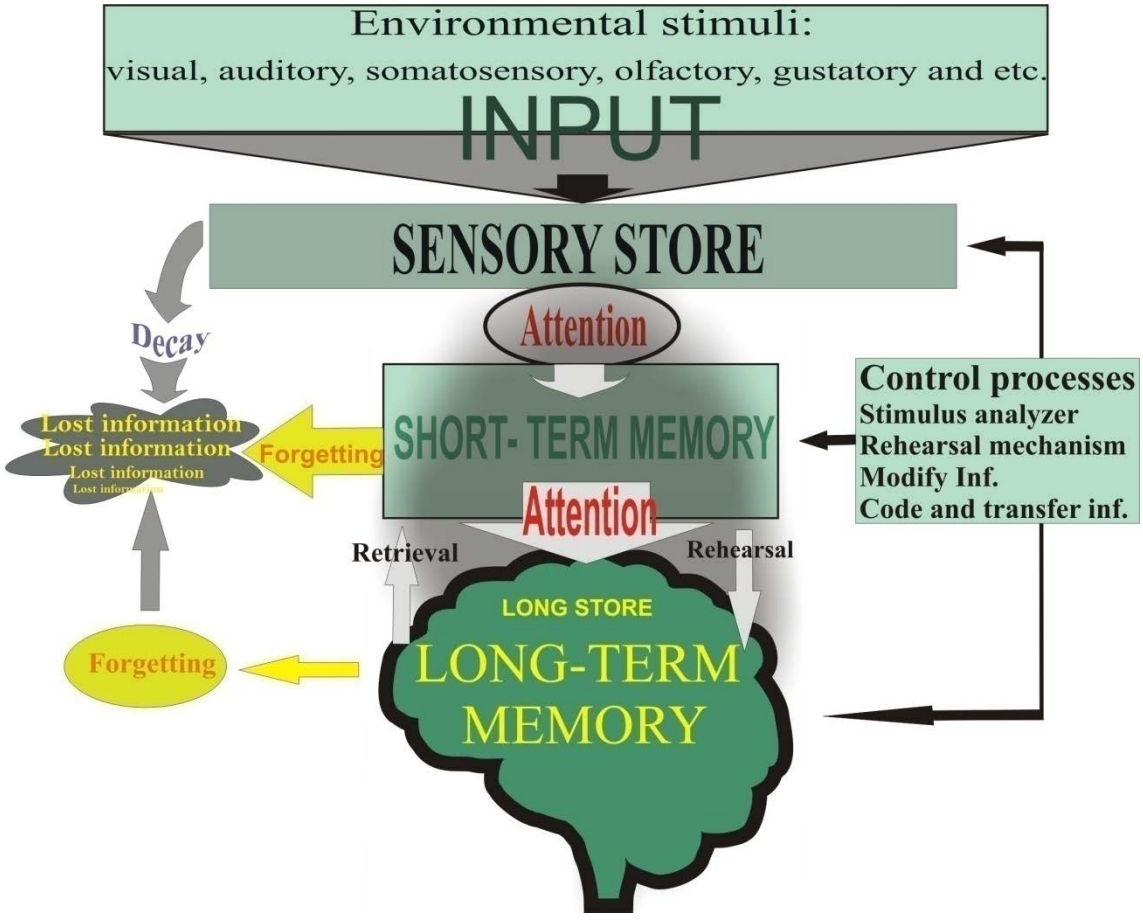

Figure 2: Multi-store model connecting the serial information processing along the time (modified from Atkinson & Shiffrin, 1968)

STM is the capacity for holding small amounts of information in one's mind in an actively available state for a brief span of time. Indeed, STM temporarily acts as a recall ability concerning information that is processed at any point in time. The duration of short-term memory is believed to be within the range of up to 30 seconds and a commonly capacity is estimated about seven, plus or minus two elements (G. A. Miller, 1956); if more items are added here, previous items are lost.

In Markowitsch opinion (2013) STM lasts seconds to a few minutes, which can store digit span of five bits of information. He believes that the first and the last perceived terms are remembered better than those in the middle (Markowitsch, 2013).

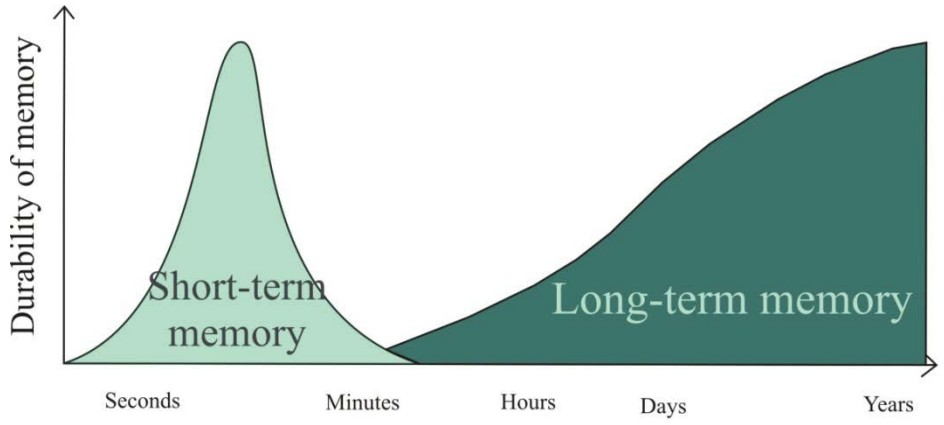

Figure 3: Schema of the difference between short-term and long-term memory, as well as relations between memory durability and duration (modified from Markowitsch, 2013)

It has also reported that there is a strict capacity limit in the number of objects that human can store in visual short-term memory (VSTM). The storage capacity VSTM is interpreted about three or four items (Todd & Marois, 2004, 2005). According to recency effect, it has been assumed that the words perceived first have already been transmitted for LTM, while that information perceived last are still in STM storage due to primacy effect (Markowitsch, 2013).

As mentioned earlier, the memory is a complex system with different functions and separate anatomical organizations. The prefrontal cortex isn't the only STM-engaged component of the brain, yet, injury to the prefrontal cortex in primates brought about STM deficits (Jacobsen, Wolfe, & Jackson, 1935). It takes only a very short amount of time until STM information vanishes irreversibly unless a conscious effort is made to remain it. STM is vital for reaching the next stage of LTM's retention (Schmidt, 1990). However, it is possible that STM may be extended by repetition, attention and rehearsal.

In Atkinson & Schifrin's multi-store model, LTM is the final stage of the memory, which may hold a huge volume of information for a long time, even lifetime. LTM is obviously suitable to store information over a long period of time. It may be that LTM decays very little over time. Information is transferred from the STM to the LTM within just a few seconds (Atkinson & Shiffrin, 1968; Shiffrin & Atkinson, 1969). Physical changes in the structure of neurons in the brain are involved in the creation of LTM. Circuits of such nerve cells are altered whenever something is learned. These neural circuits are a composition of neurons that use synaptic junctions in order to communicate with each other (Bailey & Kandel, 1993).

Now, it is generally accepted that learning requires synaptic changes, these changes in synaptic strength occur as a consequence of certain forms of learning (Martin & Morris, 2002) ; indeed, through an electrochemical transfer of transmitters in junctions between neurons and through the formation of new proteins in the brain. A stronger communication of certain neural circuits in the brain is the result. Long-term memory storage is a wide cell-biological process, which requires transcription of synaptic changes. Hence, the process needs new protein synthesis immediately after training (Kandel, 2001). With repeated use and training, the efficiency of these synapse connections increases.

2.4.1.2 Content dependent memory

Over the years, several classifications of memory as content have been distinguished. Endel Tulving (1972) first proposed the idea for a distinction between kinds of declarative material of memory such as semantic and episodic information. Tulving's "episodic memory" is the active, conscious recall of experience, while his "semantic memory" refers to the memorizing of general knowledge (Tulving, 1972). Another important division of memory made by Tulving is the one of explicit and implicit memory. Tulving proposed that these different memory phenomena reflected different brain systems (Tulving, 1985). Unlike Squire's model, it did not define two declarative and non-declarative memories. Tulving subdivided human LTM into five organized memory systems that involves episodic, semantic, perceptual, priming and procedural memory (Tulving, 1972, 1995; Tulving & Markowitsch, 1998).

Episodic-autobiographical memory (EAM), referring to memories of specific events, may be a subset of episodic memory or episodic memory itself. Like semantic memory, episodic memory is a declarative memory system, and also, there is an overlap between the semantic and episodic memory, which has been introduced by Tulving in 1994. He pointed the hemispheric encoding/retrieval asymmetry model (HERA). He showed that during episodic encoding and semantic retrieval the left prefrontal region was activated, while the right prefrontal cortex was more different engaged in episodic memory retrieval than the left prefrontal region (Nyberg, Cabeza, & Tulving, 1996). For further description about the overlap see Endel Tulving 1994 and Tulving and Markowitsch 1998.

The episodic memory (event) system is probably unique to humans (Tulving, 2002) and is used to retrieve past experiences and also may include all information of personal autobiography, for example what happened on one's last birthday. Though, episodic memory is engaged in more personal memories, such as the emotions, sensations, and personal associations of a specific memory, it is

not limited to a specific place or time. The semantic memory is concerning context-free facts (e.g. words, such as knowing who the first astronaut was, or what swim styles there are). Semantic memory allows the encoding of abstract knowledge about the world, such as the capital of a country. (Tulving, 1989; Tulving & Markowitsch, 1998; Tulving & Thomson, 1973; Vargha-Khadem et al., 1997).

The perceptual memory is expressed in developed identification of object as a physical-perceptual system and refers to the process of learning developed skills of perception because of the similarity of the stimuli. Although, perceptual memory is conscious action, it's still a presemantic level. This means that, the identification of the object requires less stimulus information or occurs more quickly. This ability can be a simple sensory identification such as distinction between two colors or two musical tones (Schacter & Tulving, 1994; Tulving, 1995).

Priming is a non-conscious form of human memory and describes the ability to recognize a stimulus faster because exposure to one stimulus influences a response to another stimulus (Tulving & Schacter, 1990); for example, "Student" is recognized more quickly following "School" than following "Car".

The simplest memory system is the procedural memory system (Thöne-Otto & Markowitsch, 2004). Perhaps, the procedural memory has simply been measured because it recalls unconsciously of past memory (Kandel, 2001). The procedural memory is often acquired by trial and error method (Mochizuki-Kawai, 2008). Indeed, this memory system includes the acquisition, storage, and retrieval of knowledge expressed through changes of experience-induced in performance. Schacter (1994) has been highlighted the procedural memory as the first major memory system (Schacter & Tulving, 1994). Procedural memory is a type of long-term memory that in a taxonomy is divided into three types: motor skills, perception and cognitive skills (Mochizuki-Kawai, 2008).

In procedural memory learning, knowledge and performance are generally unconscious; hence, it is also called implicit and non-declarative memory (Budson & Price, 2005). Unlike declarative memory, the knowledge applies quickly, triggering a response not by conscious control, but by stimulus (Squire & Zola, 1996). This memory system is an ability to learn behavioral and cognitive skills at an unconscious level (such as learning a sport skill), which are separate from declarative memory and it is composed of a network of brain structures (Ullman, 2004). It has also shown anatomical regions and their structures involved in declarative and procedural memories in the brain's system are distinct (see section 2.4.2 and Table 1, for more information). The five long-term memory systems have been shown in Figure 4.

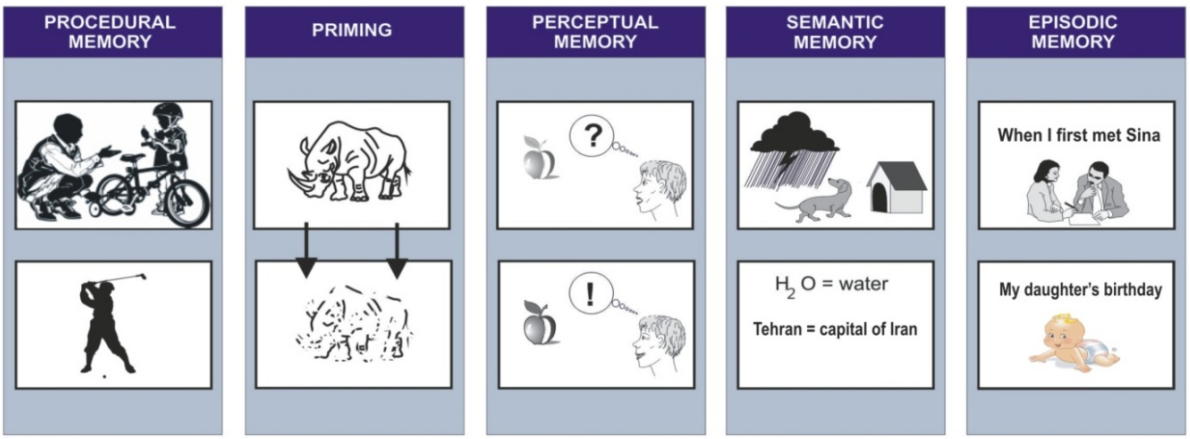

Figure 4: The five memory systems (modified from Markowitsch, 2013)

The other classification was stated by Larry R. Squire (1987). He has focused on the different levels of consciousness in human memory systems. Hence, Squire illuminated distinction between declarative and non-declarative memory. Thus, Squire stated two main memory systems (Fig. 5) included the non-declarative (or implicit) and the declarative (or explicit) memory system (Squire, 1987). In this model, declarative memory is one of two types of LTM, and refers to memories, which may be consciously recalled including facts and general knowledge (semantic memory) as well as storage of the specific and personal experiences (episodic memory) (Squire & Zola-Morgan, 1991; Ullman,

2004). In Squire's opinion declarative memory differs from procedural memory, which encompasses skills and motor learning such as the use of objects or movements of the body that are performed in level of unconscious (Squire, 2004).

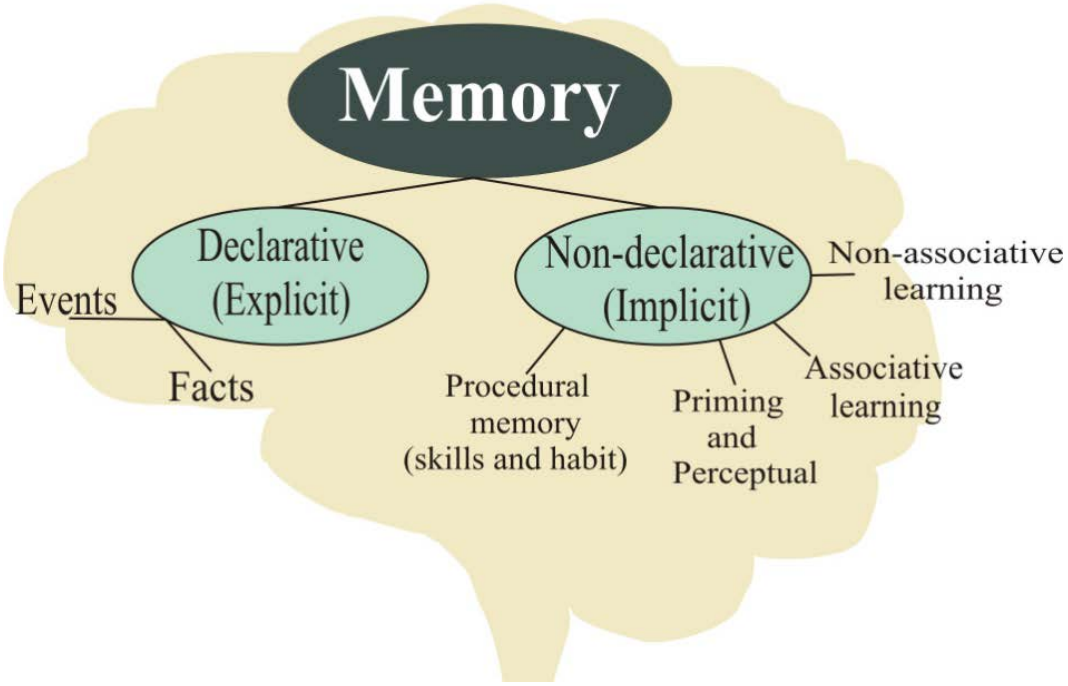

Figure 5: Squires taxonomy of long-term memory (modified from Squire, 1992)

In other word, Squire's model highlights a distinction between declarative memory, which can be accessed consciously and non-declarative memory, which is mainly unconsciously processed memory systems (L. Squire, 1992; Squire & Zola, 1996). Non-declarative memory is subdivided into unconsciously memory systems, which involves procedural learning (e.g. motor skill learning such as e.g. learning to ride a bicycle), associative learning (e.g. classical conditioning), non-associative learning (e.g. habituation and sensitization), as well as priming and perceptual learning (e.g. understanding an incomplete sketch) (L. Squire, 1992).

2.4.2 Neural correlates

At the moment, the knowledge of memory and the corresponding brain structures declared that along with increasing age some changes arise in brain structure and function (Altman & Das, 1965; Cameron & McKay, 1999; Fabel & Kempermann, 2008).

Some studies on executive function and brain regions showed the normal aging process does not degrade neural areas and cognitive processes similarly (Burke & Barnes, 2006; K. I. Erickson et al., 2009; Kramer et al., 1999; Mather & Carstensen, 2005; Small, 2001). Synapses as the functional units of the brain are morphologically and molecularly diverse (Mayford et al., 2012). This variety may be served to different functions of synapses in learning and memory. Repeatedly, engagement of prefrontal cortex during memory-associated tasks has been observed (P. Fletcher & Henson, 2001; Markowitsch, 1995).

Most research regarding short-term memory showed that STM tasks including verbal, visual and spatial are supported by transient patterns of neuronal communication in the different regions (Jacobsen et al., 1935); while LTM is maintained by more permanent and consistent changes in neural networks throughout the brain. Indeed, forgetting occurs more in the LTM when the formerly fortified connections among the neurons in their network lessen in strength (Martin & Morris, 2002), while decay happens more in sensory store and STM (A. D. Baddeley, 2000).

To recognise better of the neural areas and cognitive processes, in the next section, a brief outline is given of the involvement of brain regions in short- and long-term memory processes and also associated areas in the brain.

2.4.2.1 Neural regions and short-term memory

As already noted, different STM tasks are supported by neuronal communication in the different regions. One of the old works has shown that some STM tasks are activated regions of the frontal (BA 4/6/8-11/24/25/32/33/44-47), prefrontal (BA 9-12/46/47) and parietal lobes (BA 1-3/5/7/39-43) of the brain (Jacobsen et al., 1935). It seems that the central executive task cannot be localised only in one area but activates a larger network involving the frontal regions (Baddeley, 2003).

In 1971, a research on monkeys has demonstrated that nerve cells in the prefrontal cortex (BA 9-12/46/47) and part of the thalamus, associated with the attentive process are involved in short-term memory (Fuster & Alexander, 1971).

Courtney et al., (1996) study the neural correlated on object and spatial visual of working memory through the control of increasing cerebral blood flow (rCBF). They demonstrated that the neural systems involved in working memory for faces and spatial location are functionally segregated. Face working memory, is associated on superior and inferior parietal cortex (BA 40), while spatial information working memory, is related on parahippocampal, fusiform, inferior frontal, and a part of anterior cortices, and in right thalamus and midline cerebellum (Courtney, Ungerleider, Keil, & Haxby, 1996).

Nyberg et al., (2002) showed that working memory tasks that is associated to STM activated the premotor (BA 6) and parietal of cortex brain regions (Nyberg, Forkstam, Petersson, Cabeza, & Ingvar, 2002). It has been reported that posterior parietal cortex (PPC) (BA 7) is related with visual short-term memory tasks and is strongly correlated with the limitation amount of scene information that is stored in visual short-term memory (VSTM) (Todd & Marois, 2004). They also found a correlation between VSTM capacity with activity in the posterior parietal and the visual occipital (BA17-19) cortex (Todd & Marois, 2005). A

number of investigator like Beeson et al., (1993), Butters et al., (1970) and Cermak and Tarlon (1978) reported that left inferior frontal cortex related with Broca's aphasia area (BA 44) and this area can limit auditory and verbal of STM and some researchers for example Awh et al., (1996), Cohen et al., (1994), Grasby et al., (1993), Rypma et al., (1999), Smith et al., (1996) have confirmed that this area is activated by verbal STM and may be modulated by the constraints of STM demand (Cooke et al., 2002). Salmon et al., (1996) have also confirmed that the left inferior supramarginal gyrus and the premotor (BA 6) are the main regions in verbal STM processes, and the superior occipital gyrus (BA 19) is the key regions of the visual STM processes (Salmon et al., 1996).

Briefly, different STM tasks are supported by distinct areas of brain. For instance the frontal and prefrontal regions related to the central executive task. Plus a part of prefrontal cortex, part of the thalamus associated with attentive process is involved in STM. Working memory is associated on the premotor (BA 6) and parietal cortex (BA 1-3/5/7/39-43). The posterior parietal cortex (BA 7) was found activated for visual STM tasks, while the left temporal-parietal and left inferior frontal cortex are related to auditory and verbal of STM tasks.

2.4.2.2 Neural regions and long-term memory

Although, the hippocampal region is dedicated to memory performance (Scoville & Milner, 1957) and this region of brain has been identified as fundamental capacity of declarative memory (Eichenbaum, 2000). The encoding and consolidating of new information of declarative memory including events and factual knowledge requires two brain circuits, which are located within the limbic lobe and are called the Papez and the basolateral limbic circuits (Markowitsch, 1997).

James Papez (1937) introduced this circuit that is proposed to be predominantly involved in the transfer of new information into the long-term

declarative memory system. The circuit of Papez is one of the main pathways responsible for processing of emotion and memory. With length about 350 millimeters in this circuit, the information processing begins in the hippocampus and continues into the fornix to reach the mamillary body, which are connected via the mammillothalamic tract (MMT) (or bundle of Vicq d'Azyr) after that continues to the anterior nucleus of the thalamus that is connected by means of the thalamo-cortical pedunculi with the cingulate gyrus. The cingulum courses around the corpus callosum to end in the entorhinal cortex, which then projects to the hippocampus and circuit is completed (Shah, Jhawar, & Goel, 2012). Although, it has also been illumined that there are connections between the hippocampal formation, hypothalamus and septal area via fornix and MMT (Rajmohan & Mohandas, 2007). Figure 6 shows the papez circuit.

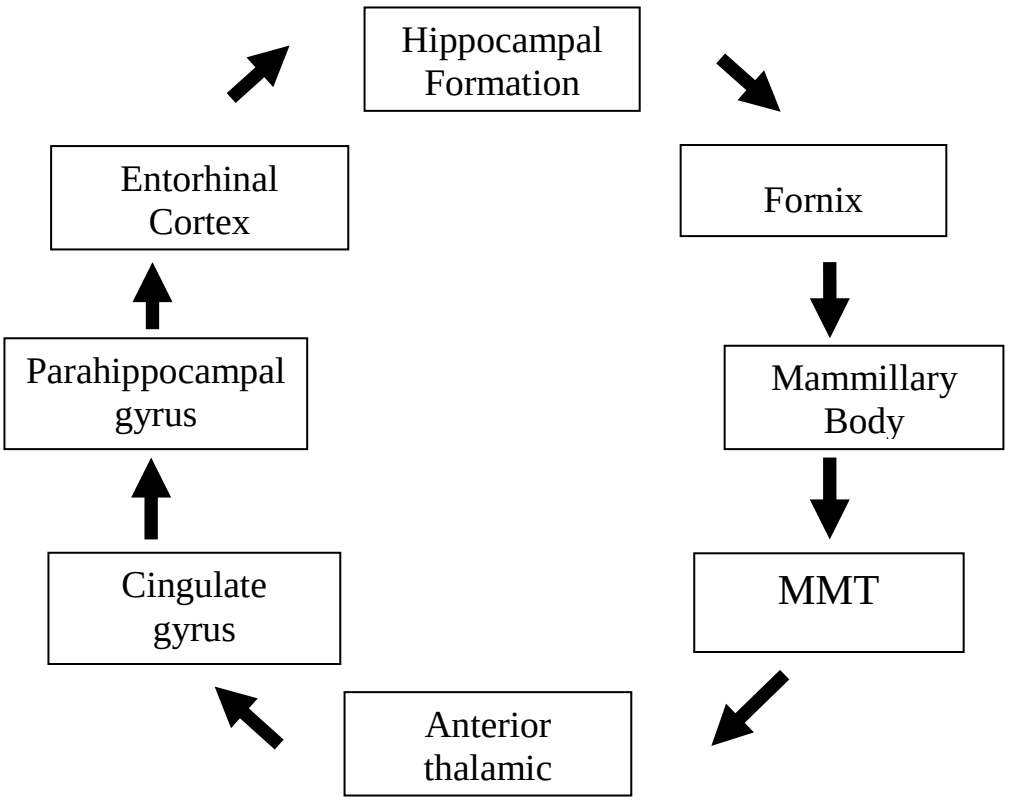

Figure 6: Illustration of the Papez circuit (modified from Rajmohan & Mohandas, 2007)

In other limbic circuit, the information processing is relayed via the basolateral complex of the amygdala (BLA). Since the amygdala is an important area of brain for emotional arousal (McGaugh, 2000) this circuit is known to be responsible for the processing of emotion and affective functions (Markowitsch, 2000). Amygdala uses two major pathways in limbic circuit (Rajmohan & Mohandas, 2007) and contain:

- Dorsal route or amygdalo-septal pathway
- Ventral route or ventral amygdalofugal pathway

The basolateral pathway consists in parts of the orbitofrontal, prefrontal and temporal cortices, amygdala, medial dorsal thalamus, hypothalamus and septal area, anterior temporal cortex. Indeed, basolateral circuits serve to process information and emotional reactivity between association cortices including the prefrontal and temporal, and the hypothalamus (Markowitsch & Staniloiu, 2011a; Rajmohan & Mohandas, 2007). In addition to effects of the amygdala in affective and emotion information, the amygdala plays a special role in encoding, consolidation and retrieval processing of declarative (events) memory (Markowitsch & Staniloiu, 2011a). Also, the findings of animal and human studies have shown the role of the amygdala in modulating the consolidation of LTM (McGaugh, 2002, 2004).

Those circuits are not only interconnected together, but also connected with other regions through some of the structures (Rajmohan & Mohandas, 2007). In addition, the components of the circuits are known to have different tasks. As already noted, the amygdala plays a special role in encoding of emotional information (Markowitsch, 2000; Markowitsch & Staniloiu, 2011a), the amygdala with the thalamus and the basal forebrain can allow a better encoding of affective memories (Piefke, Weiss, Zilles, Markowitsch, & Fink, 2003). There is evidence that the amygdala through interconnections with other

brain systems plays a special role in modulation of memory as well as regulating attention (Gallagher & Chiba, 1996).

Much evidences have supported that the amygdala plays a considerable role in storage processes during emotional learning conditions (Kilpatrick & Cahill, 2003; McGaugh, Cahill, & Roozendaal, 1996). Anyhow, pleasant or aversive events are memorized better than neutral (or non-emotional) events. Despite of fact that the visual information is stored in the occipital lobe (BA 17-21) (in the visual area of the brain cortex) the amygdala feedback during emotional situations can influence the processing of visual information (Amaral, Behniea, & Kelly, 2003). If the amygdala is activated by affective arousal, it modulates memory storage processes in other brain regions such as parahippocampal gyrus and parts of prefrontal cortex (Kilpatrick & Cahill, 2003).
Even through the amygdala activation via emotionally arousing can improve episodic memory through modulation of hippocampal activity (Hamann, Ely, Grafton, & Kilts, 1999). Although, the storage process depends on the type of memory (Tulving, 1972); it has been suggested that parts of the brain such as amygdala may have the role of substantial modulator control (Amaral et al., 2003). Also, the amygdala contributes not only to the conscious processing with a higher degree of cognitive-emotional functions for instance in episodic memory, but also to non-conscious forms of memory such as procedural memory and priming (Markowitsch & Staniloiu, 2011a).

Some processes of declarative memory depends on the temporofrontal junction area, but, in view of, lateral hemisphere predominance, the factual knowledge or semantic memory is more correlated with the left cerebral cortex and episodic information or personal experiences with the right part (Markowitsch, 1997). Also, about hippocampus in human, studies has shown that the right hippocampus is associated with encoding processes of spatial memory and left hippocampus is related to processing verbal information and declarative

memory (Burgess, Maguire, & O'Keefe, 2002). Aside from the specific role of hippocampus in autobiographical memory, but not for the acquisition of factual knowledge (Tulving & Markowitsch, 1998; Vargha-Khadem et al., 1997), the hippocampal formation that are connected to the frontal lobe can create the basis for developing spatial-temporal episodic memories (Burgess et al., 2002).

Milner in 1965 emphasized the importance of the medial temporal lobe (MTL) for encoding processes of the declarative memory (Milner, 1965a). In 1953, the famous patient H.M. who suffered from intractable epilepsy was referred to neurosurgeon William Scoville. Scoville diagnosed the patient's epilepsy to both MTLs (left and right) and ordered their surgical resection. The hippocampal formation was included in the patient's MTL; most of the entorhinal cortex and the amygdaloid complex needed to be removed. Also, it appeared that his hippocampal tissue was entirely nonfunctional; even parts of the anterolateral temporal cortex were destroyed.
After surgery and after a successful bilateral medial temporal lobectomy, he suffered from severe anterograde amnesia; the patient's procedural and working memory remained intact. He was able to learn new skills such as mirror drawing; although, he was unaware of doing it. Also he could not apply new events to his declarative memory (Scoville & Milner, 1957). The structure of MTL including the hippocampus, entorhinal, perirhinal, and parahippocampal cortices, may be involved in storing of new information (Squire & Zola-Morgan, 1991).

Memory storage for declarative human learning is notably dependent upon structures within the temporal lobe, for instance the hippocampus (L. R. Squire, 1992). Also, the prefrontal and anterolateral temporal cortex which provides a connecting link to posterior cortical centers of integration as the major storage places (Markowitsch et al., 1985). Hippocampal region plays a key role in the storing of new memories, but over time the storage of non-declarative memories becomes independent of this area (Graham & Hodges, 1997). Briefly, declarative memory depends on the hippocampal region, a set of greatly interrelated medial

temporal lobe structures, parts of cortex such as entorhinal and perirhinal, as well as parahippocampal cortical regions (Squire & Zola, 1996; Suzuki & Eichenbaum, 2000; Tulving & Markowitsch, 1998; Vargha-Khadem et al., 1997).

Semantic memory depends primarily on the cerebral cortex (Markowitsch & Staniloiu, 2012; Vargha-Khadem et al., 1997) and also the inferolateral temporal lobe (Budson & Price, 2005; Markowitsch & Staniloiu, 2011b). The episodic memory system is dependent mainly on the hippocampal component system (Tulving & Markowitsch, 1998; Vargha-Khadem et al., 1997) as well as on the limbic regions, and frontotemporal and cerebral cortex (Markowitsch & Staniloiu, 2012).

Retrieval of LTM is strongly related to the parts of the right lateral temporo-frontal junction area (Kroll, Markowitsch, Knight, & von Cramon, 1997). Though, it has been observed that the prefrontal cortex (BA 9/10/11/12/46/47) acts as a kind of control center for beginning of recall (Jetter, Poser, Freeman Jr, & Markowitsch, 1986). Retrieval of declarative memory containing semantic and episodic information is triggered by the synchronous action of the temporo-frontal junction area (Markowitsch, 1997).

Brain lateral hemispheres have features in the activation and recovery of stored explicit material. The right hemisphere supports episodic information retrieval, while left brain hemisphere may support retrieval of stored semantic information or factual knowledge (Markowitsch, 1995).

As already noted, Tulving (1994) showed that during semantic retrieval, the left prefrontal region was activated (Nyberg et al., 1996). Like the HERA of Tulving has shown, there is an overlapping of brain regions during episodic encoding and semantic retrieval, and also in the left prefrontal region, as Cabeza et al. (2000) explained, by the engagement of encoding of new autobiographical information during the retrieval of old semantic memory (Cabeza & Nyberg, 2000). Also, the encoding of autobiographical information has been related to the

activation of retrosplenial (BA 29/30) regions in the cingulate cortex and in the left prefrontal cortex (BA 9-12/46/47), whereas recall of autobiographical information is furthermore depended on activation of the prefrontal cortex (BA 9-12/46/47) – this time, though, on the right hemisphere –, and also on the precuneus bilaterally (BA 7) (P. C. Fletcher et al., 1995).
It has been found that retrieval of episodic memories is associated with parts of the prefrontal cortex (BA 10/11), the temporo-parietal-occipital junction (BA 19/37/39), and the medial frontal cortex (BA 9). Indeed, the parts of anterior frontal (BA 8), orbitofrontal (BA 11), and dorsolateral frontal cortex (BA 46/47) of the right are involved in the retrieval of episodic-autobiographical memories (EAM) (P. Fletcher & Henson, 2001). The clear functional roles of the fronto-polar region (BA10) are not well described in human (Strange, Henson, Friston, & Dolan, 2001) but, the retrieval process is mainly linked to frontal regions of the brain (Wagner, 2002).

In general, the retrieval of episodic memory activates fronto-polar prefrontal cortices (BA 10) (Strange et al., 2001). In page 51, Table 1 presents these five memory systems and their relevant regions of the brain.

2.4.2.3 Neural regions and procedural memory

Penfield (1968) believed that the procedural memory is developed in both hemispheres of the brain (Penfield, 1968). Also, the basal ganglia collects information from almost the cerebral hemispheres and in accompany with the frontal cortex constitutes an integrated, distributed neuronal structure (Wise, Murray, & Gerfen, 1996). Anatomy of the basal ganglia has been shown in Figure 7. The basal ganglia are a set of sub-cortical structures, included of the striatum, pallidus, subthalamic nucleus (STN), as well as the substantia nigra

(SN), which is within brainstem (Kurita, Sasaki, Suzuki, & Kirino, 1998; Miall, 2013; Parent & Hazrati, 1995; Wise et al., 1996).

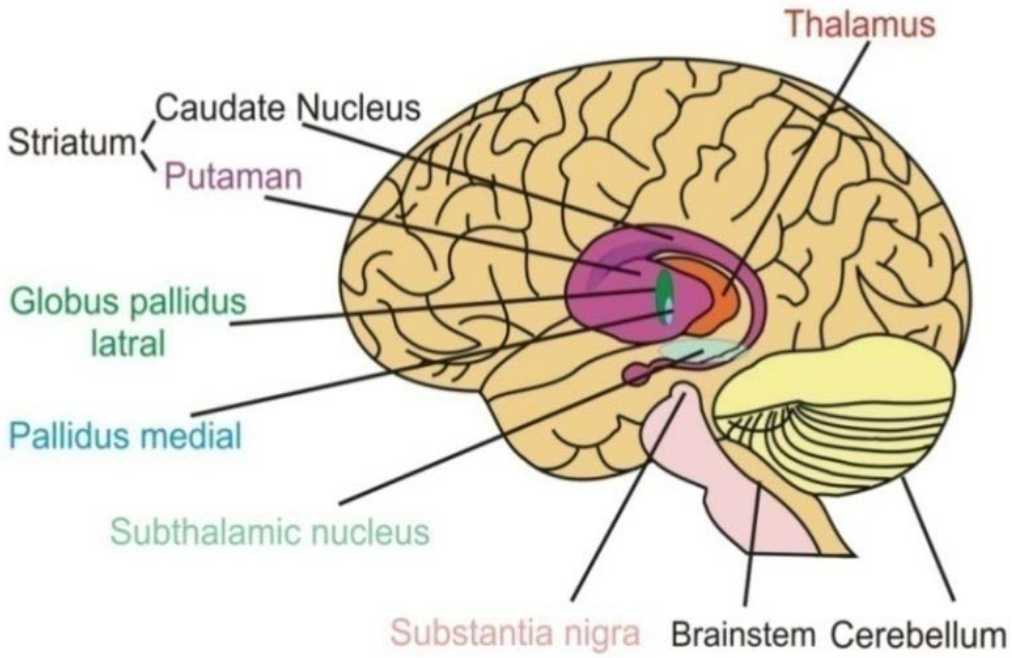

Figure 7: The components and structure of the basal ganglia in sagittal view (modified from Alexander et al., 1986)

In primates, the striatum (or neostriatum) consists of two segments (Kurita et al., 1998; Miall, 2013); one of them is putamen, which plays an important role in motor learning. The other one is caudate nucleus that engages in aspects of cognition (Alexander, DeLong, & Strick, 1986). It has also been demonstrated that both the putman and caudate of the striatum indicate a role in learning and memory (Packard & Knowlton, 2002). Although, the striatum is required for the formation of long-term procedural memory, the striatum also effectively supports a motor skill consolidation in procedural memory (Mochizuki-Kawai et al., 2004).

Alexander (1986) has suggested a theory that is called segregated circuits hypothesis of basal ganglia. This implies that the basal ganglia contains parallel and mainly functionally segregated circuits (Alexander, Crutcher, & DeLong,

1989; Alexander et al., 1986). Hence, in the basal ganglia via the circuits might be performed similar neuronal functions (Ullman, 2004). Some of the circuits may mediate in many dysfunctional aspects of human behavior containing executive function deficits, imbalances disinhibition, movement disorders, depression and obsessive disorders (Cummings, 1993). Figure 8 shows the attitude of the basal ganglia-thalamocortical circuit and also the elements of circuits include separate, essentially non-overlapping parts of the striatum, globus pallidus, substantia nigra, thalamus, and cortex.

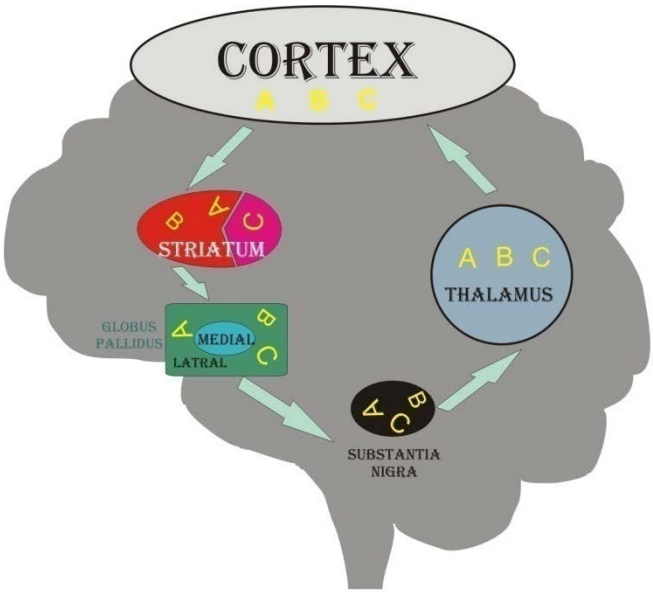

Figure 8: The attitude of the basal ganglia-thalamocortical circuit and also the elements of circuits (modified from Alexander et al., 1986)

It has repeatedly been stated that the basal ganglia circuits' damage appears cognitive and motor indications, such as deficits during new learning and differences in motor tasks (Alexander et al., 1986; Cummings, 1993; Jenkins, Brooks, Nixon, Frackowiak, & Passingham, 1994; Middleton & Strick, 2000). Although, a wide range of basal ganglia functions is still unknown, they are implicated in the release (or inhibit) and control (or hibit) of generated movements (Miall, 2013). However, this is consistently emphasized that

preservation of sensorimotor skill depends upon the basal ganglia (Gabrieli, 1998; Gabrieli, Corkin, Mickel, & Growdon, 1993; Gabrieli, Stebbins, Singh, Willingham, & Goetz, 1997; Milner, 1962, 1965b).

In sum, the basal ganglia can play an important role in a number of learning functions, including in stimulus-response learning (Packard & Knowlton, 2002), as a strengthening factor in consistent relationship between stimuli and responses (White, 1997), for the gradual learning of habit learning via caudate nucleus and putamen (Knowlton, Mangels, & Squire, 1996), real-time motor-skill learning (Wise et al., 1996), the switching or selection among multiple motor-skill performances (Haaland, Harrington, O'Brien, & Hermanowicz, 1997), and in general, in procedural learning (Markowitsch, 1997; Mishkin, Malamut, & Bachevalier, 1984; Mochizuki-Kawai et al., 2004; Schacter & Tulving, 1994; Squire & Knowlton, 2000). Indeed, it has unanimously pointed out that the procedural learning system depends on a network of the brain regions that involves parts of prefrontal and basal ganglia parietal and cerebellar structures (Mochizuki-Kawai, 2008; Nicolson & Fawcett, 2007).

The neostriatum and other components of the basal ganglia are associated with the MTL and the frontal cortex to obtain input projections from many cortical regions and in this way are created several circuits such as motor circuit (Alexander & Crutcher, 1990). The basal ganglia structures themselves are really interlinked. For example, there are two pathways on outputs of the basal ganglia to the frontal lobe via the thalamus. The direct and indirect pathways are two of them paths, which have contrasting effects. The inhibitory projections is inhibited by the direct pathway from the basal ganglia to the thalamus, while the inhibitory projections are disinhibited by the indirect pathway on the same path between the basal ganglia and the thalamus (Ullman, 2004). In fact, frontal cortical regions activity is released by the direct pathway, and reduced by the indirect pathway.

Dysfunctions of the frontal cortical regions that depend on the basal ganglia can be related to imbalances between these pathways (A. B. Young & Penney, 1993). For case, Parkinson's disease is often associated with pathological changes in sensorimotor portions of the striatal region (Kish, Shannak, & Hornykiewicz, 1988; Middleton & Strick, 2000). Also, some of the frontal regions are vital for motor learning and procedural memory (Ullman, 2004). Each of these regions can play key roles in the motor skill learning.

The encoding, consolidation and retrieval processing of procedural memory are related to the basal ganglia (Alexander et al., 1986; Markowitsch, 1997; Markowitsch & Staniloiu, 2012), portions of the cerebellum (Markowitsch, 1997; SANES, DIMITROV, & HALLETT, 1990; Willingham, Koroshetz, & Peterson, 1996) and the supplementary motor area (BA 6) (Budson & Price, 2005).
The premotor regions (BA 6) are related to motor skills learning (Jenkins et al., 1994), and particularly encoding of procedural memory (Schubotz & von Cramon, 2001).

Broca's area (BA 44) is another important component of the motor skill learning. It has been reported that Broca's area can be associated with rhythm of physical performance (Schubotz & von Cramon, 2001; Ullman, 2004). Also, evidence suggests that this area of the brain may be involved during mental imagery of human movement (Binkofski et al., 2000).

Some areas of parietal cortex (BA 1-3/5/7/39-43) also play a critical role in the procedural memory. In human, a lobule of inferior parietal (BA 40) has been implicated in several of brain functions, including attention shifts (Perry & Zeki, 2000), and the execution and retrieval of motor skills that were previously learned (Heilman, Watson, & Rothi, 1997).

The cerebellum has traditionally been associated with motor control (Ivry & Fiez, 2000; Thach, 1998; Wolf, Rapoport, & Schweizer, 2009), but much evidence have shown the cerebellum involvement in non-motor and cognitive functions (Cutting, 1977; Doya, 2000; Fiez, Petersen, Cheney, & Raichle, 1992; Ramnani, 2006; Schmahmann & Pandyat, 1997; Thach, 1998; Wolf et al., 2009). The role of the cerebellum in motor control as well as both cognitive and affective functions is not surprising; because, the cerebellum is strongly interconnected with non-motor cortical and sub cortical areas associated with emotional processing, including the limbic system (Markowitsch, 1997; Schmahmann & Pandyat, 1997; Wolf et al., 2009).

The cerebellum also connects with cortical areas of the prefrontal cortex (Ramnani, 2006). It has also been shown that the cerebellum and the cerebral cortex are specialized for different types of learning and also reinforcement learning (Doya, 2000; Thach, 1998). Also, results of an investigation about separating the effects of changes in performance from motor skill learning suggests that the cerebellum may be engaged primarily in the modification of motor performance (Seidler et al., 2002). However, the cerebellum plays vital role in the coordination and balance of motor skill as well as in procedural motor-skill learning (Ivry & Fiez, 2000; Nicolson et al., 1999).

According to Brindley theory (1964) motor skill learning begins as a conscious act mostly under the control of the cerebral cortex, without help from the cerebellum (Brindley, 1964; Thach, 1998). When, a new motor learning is going to be skilled, there are too many things that should be refined, as well as muscles are not yet related jointly in the correct combination, or level of activation. The cerebellum would acquire control of the task by recognizing the contexts in which each piece of consciously initiated movement occurs (Thach, 1998). This relationship between motor and sensory cortices has long been documented (Ramnani, 2006). During practice and repeat of movement, the

cerebellum would relate that context to the movement generators, so that reappearance of the context would automatically trigger the movement.
Finally, the cerebellum would largely take over the process, as a background subconscious mental subroutine, with minimal help from the cerebral cortex (Thach, 1998). Studies with using functional magnetic resonance imaging (fMRI) have shown that memory systems such as declarative and procedural may compete with each other. It has been reported that despite the similarity of the learned material and the level of performance interaction between the medial temporal lobe and basal ganglia occur (Poldrack et al., 2001).

Human and animal studies have clearly shown that the declarative and procedural memory systems are largely independent from each other, but they interact in several pathways (Mishkin et al., 1984; Schacter & Tulving, 1994; Squire & Knowlton, 2000). Interestingly, indeed, there are anatomical and functional confluence between the ganglia basal and cerebellum in motor and non-motor function; thus, it may interact in a number of functions of the brain. In motor skill learning process, the interaction of procedural and declarative memory systems has been seen (Willingham, 1998). Also, evidence has suggested that the interaction between hippocampus and the part of basal ganglia can optimize learning in human (Poldrack & Packard, 2003). Though, when both implicit and explicit systems are intact they can help one another, for example the brain structures which underlie non-declarative memory can also influence some components of declarative memories via brain structures that play roles in both systems (Ullman, 2004).
A good example can be patient H.M (see section 2.4.2.2.), when he could learn new motor skills, while cannot remember, how has learned them (Scoville & Milner, 1957). However, despite this apparent independence, extensive evidence indicates that multiple memory systems in the human brain may interact with each other.
Mochizuki-Kawai et al., (2008) explained that the three types of procedural memory (motor skills, perception and cognition) are intertwined; they depend on

different brain regions – one of which may be the motor-type procedural memory – and are supported by other multiple brain regions including the frontal and parietal regions as well as the basal ganglia and cerebellum (Mochizuki-Kawai, 2008). In Mochizuki-Kawai's opinion both the perceptual and cognitive types of the procedural memory are maintained by multiple brain regions, indeed, the related cerebral areas depend on type of memory which is either perceptual or cognitive. It has been suggested that the fusiform area (BA 37) may support the perceptual type's acquisition of procedural memory, whereas the frontal and parietal cerebellar regions as well as the basal ganglia may maintain acquisition of cognitive procedural memory (Mochizuki-Kawai, 2008). Table 1 shows the five memory systems and their brain region.

Table 1: Memory systems and the brain regions (modified from Markowitsch,2011)

	Encoding and consolidation	Storage	Retrieval
PROCEDURAL MEMORY	Basal ganglia, cerebellum, premotor areas	Basal ganglia, cerebellum, premotor areas	Basal ganglia, cerebellum, premotor areas
PRIMING	Primary association cortex	Primary association cortex	Primary association cortex
PERCEPTUAL MEMORY	Posterior sensory cortex	Posterior sensory cortex	Posterior sensory cortex
SEMANTIC MEMORY	Cerebral and preforotal cortex, limbic circuits	Cerebral cortex and limbic circuits	Frontotemporal cortex, left
EPISODIC-AUTOBIOGRAPHICAL MEMORY	Preforotal cortex and limbic circuits	limbic circuits and cerebral cortex	limbic circuits and the right of frontotemporal cortex

2.5 Learning theories related on procedural memory

Several models are used to describe these learning stages. The most popular are the Gentile two-stage model and the Fitts and Posner three-stage model. Unlike the Fitts and Posner model, the Gentile model draws on the account of the learning environment and distinction between open and closed skills.

2.5.1 Fitts and Posner's model

Fitts & Posner (1967) proposed one model which is also called Fitts and Posner's three stage models for learning motor skill acquisition. They believed that the learning process is continuous and steady. This model suggests the idea that the new learning is possible through the completion of three stages including cognitive phase, associative phase, and autonomous phase (Fitts & Posner, 1967).

Cognitive stage (Verbal-motor stage)

This stage is important for the acquisition of skills. In the present stage, a learner acquaints components of the skill, because the learner does not have any corrective schema. The learner then forms a mental picture of the skill. Learners gather information and performance is still inconsistent. Indeed, this stage concerns learning what to do. In this stage, the learner divides the desired motor-skill into smaller parts for a better understanding of how these parts operate together as a unique skill.

Associative stage (Motor stage)

Hitherto learners have had large gains, but they will have small gains in this stage. This stage involves connecting the components into a unit performance through repetition and practice and using feedback to the motor skill pattern. In simple words, this phase is refining the movement pattern. In this stage an

individual's sensory system acquires the accurate symbolic and spatial data which required completing the desired skill. The decreased gross errors by developing appropriate error correction strategies and increased attention are specifications of this stage.

Autonomous stage (Automatic stage)

In this final stage, the skill of learner is developing so as to prefect skill acquisition. However, any learner may not be able to reach this stage. In the autonomous stage less thought process is required and in this phase also the decrease in attention occurs, while performing the skill almost remains in good level. The learned skill seems unconscious and learner needs time and practice. Gradually, performance becomes automatic – involves little or no conscious thought or attention whilst performing the skill in this stage create the ability to differentiate important from unimportant stimuli. Thus, a skilful athlete can easily ignore the negative stimuli, at highest level of proficiency, because the skill has become automated. A skilled person makes few errors and can generally detect and correct those errors. Figure 9 shows the Fitts and Posner's three stage models of motor learning.

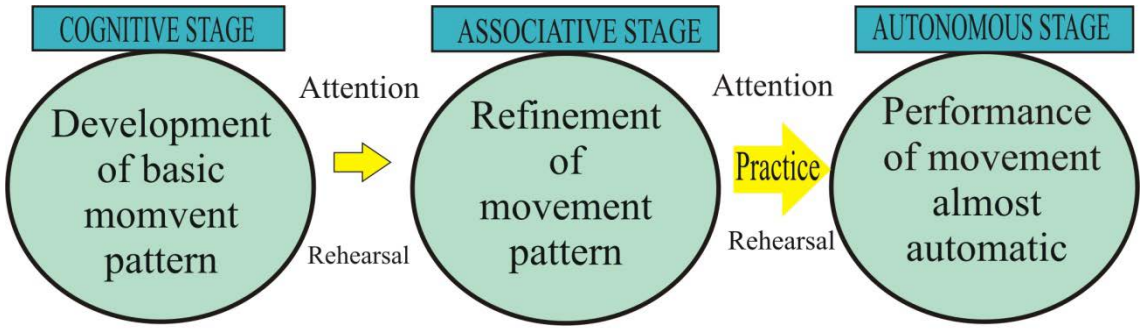

Figure 9: Three stage of motor learning (modified from Fitts and Posner's 1967)

2.5.2 Gentile's model

Gentile's two-stage model of learning is almost similar to Fitts and Posner's model in a way that the initial stage linked cognitive processes.

In early (or initial) stage, the learner is trying to understand the concept of movement. Within this first stage Gentile refers to learners beginning to gain an understanding of different factors that can affect the performance of the skill (Equivalent to Fitts and Posner cognitive stage). The second stage (or the late stage) is about a skill being fixed and the learner to be able to modify the skill, like the Associative and Autonomous stages of Fitts and Posner's. In second stage the skill gradually becomes fixated. She believed that for static and dynamic performance learning is different; she used the terms open and closed skills. Open skills have to do with a changing environment. Learners adapt to such changing environments – for example during a game of water polo where players need to counter and react to each other's moves – in order to succeed.

Concerning close skills, which applies for example during swimming, environmental fixations is necessary to achieve success (Gentile, 1972).

2.5.3 Adams' closed-loop theory

Adams' closed-loop theory is based on motor learning that focused on slow and graded tasks; detection of errors following corrections allows achievement of optimal performance. Adams' views processing of afferent information as a central player in human motor control. He believed, to learn a skill, a motor program consisting of two states of memory require to involves the memory trace and the perceptual trace (Adams, 1971). The memory trace is a brief motor program – less than one second – and occurs before the perceptual trace. The memory trace chooses and starts the response preceding feedback and is independent of the perceptual trace. A reference mechanism is proposed by Adams that is called the perceptual trace. The perceptual trace involves in guidance of the limb to the correct position along a presentation and creates the

next response different from the last one, with less error (Adams, 1987). According to Adams, two stages in motor learning process includes the verbal-motor stages which is without perceptual trace and the motor stage that the subject has a good perceptual trace (Adams, 1971).

2.5.4 Schmidt's schema theory

Schmidt in 1975, in opposition to closed-loop theory, proposed the open-loop theory which is called the schema theory for motor control. In Schmidt's opinion, the motor response schema need four kinds of information that store in memory after a motor learning program:

1. The initial conditions of the motor learning skill
2. The response specifications for the motor skill
3. The sensory consequences or information about the performance
4. The knowledge of results as a response outcome of the learned information (or feedback)

This information is stored into two forms of the motor response schema including:

1. The recall schema that related to intended outcome.
2. The recognition schema that compares the intended outcome with the actual outcome.

The schema theory introduced motor learning as a continuous process. It appears that continuous comparing of the recall schema with the recognition schema is performed for each motor skill.

2.6 Aging, cognition and exercise

Research on risk factors for reduced cognitive function in aging adults is of a necessary public health importance.

Beneficial effects of physical education may improve quality of life, prolong independency and life expectancy, it may also reduce economic cost (Elavsky et al., 2005; Larson & Wang, 2004) and is an effective strategy to delay the onset of dementia (Larson et al., 2006; Pate et al., 1995). Functional changes of age that are increasing in the hippocampus induce cognitive shortage that is associated with an excessive decrease in neurogenesis in the hippocampal dentate gyrus (K. I. Erickson et al., 2011; Kim et al., 2010).

Studies on animals have shown benefits of physical activity, that is, aerobic exercise can increase the factors which helps learning and brain performance in adult animals (Churchill et al., 2002; Neeper, Gómez-Pinilla, Choi, & Cotman, 1996; Van Praag, Christie, Sejnowski, & Gage, 1999). For example, on the adult mouse, voluntary physical activity such as swimming, walking and toning yielded an increase in cell proliferation, cell survival, neurogenesis and improved learning, and it also enhanced long-term potentiating and synaptic plasticity (Van Praag, Christie, et al., 1999; van Praag, Kempermann, & Gage, 1999; Van Praag, Shubert, Zhao, & Gage, 2005).

In 1997, the Maastricht Aging Study (MAAS) indicated that aerobic fitness is a factor of relative importance in the cognitive aging process. It was stressed that all evidence pointing towards a factor capable of preventing/postponing age-related decline should be most intensely researched in order to uncover any underlying mechanisms (Van Boxtel et al., 1997). Often the relation between activity and memory has been reviewed under the hypothesis that participation in regular physical activities may help preserve cognitive activity and decrease dementia risk, which, in turn should exercise and maintain memory (K. I. Erickson et al., 2011; Hess, 2005; Heyn et al., 2004; Podewils et al., 2005). Collectively exercise can improve cognitive performance (Intlekofer et

al., 2013) and delay the onset of decline brain diseases (Cotman & Engesser-Cesar, 2002).

The findings derived from the study of fitness training on brain function and structure in animal models (Van Praag, Christie, et al., 1999; van Praag, Kempermann, et al., 1999; Van Praag et al., 2005) provide a conceptual framework for better understanding of the cognitive function-cardiovascular fitness relationship in humans and factors that may moderate this relationship (K. Erickson & Kramer, 2009; Kramer et al., 1999; McAuley, Kramer, & Colcombe, 2004).

Researchers who have studied the effects of anaerobic exercise on cognitive functions have consistently failed to detect a clear relation between the exercise and cognitive processes, thus, there is limited data on the effects of resistance training and anaerobic exercise in the cognitive function of older adults. For example, an eight weeks investigation of resistance training reported some effects in cognitive function, though not significantly; in a follow-up one year later, researchers observed a long-term effect on memory performance (Peig-Chiello, Perrig, Ehrsam, Staehelin, & Krings, 1998).
It was reported that resistance exercise programs between moderate and high intensity had beneficial effects on cognitive function of older adults (Cassilhas et al., 2007). It also was suggested that aerobic training interventions that also included a strength training protocol may provide greater overall benefits on cognitive performance than those that only have an aerobic training component (S. Colcombe & Kramer, 2003; K. I. Erickson et al., 2009; Heyn et al., 2004).

A study on the dose-response correlation between resistance exercise intensity and cognition has found a complicated relation between the intensity of exercise and cognitive performance for example the high-intensity exercise benefits speed of processing but moderate intensity exercise is most beneficial for executive function (Chang & Etnier, 2009).

Evidently, converging results about the efficiency of exercise and physical fitness for the maintenance of cognitive function of elderly is a necessity. There are reports on the importance of aerobic activity having specific benefits on such things as maintenance or improvement of cognitive skills (Kramer et al., 1999), cell growth in the hippocampus and dopamine receptor density in the brain (S. Colcombe & Kramer, 2003), and also there are reports on affected cognition through aerobic exercise (Blumenthal & Madden, 1988), stretching exercise (K. I. Erickson et al., 2011), toning (Kramer et al., 1999), and on positive effects of strength training on neuronal growth (Borst, Vincent, Lowenthal, & Braith, 2002) and cognitive performance (S. Colcombe & Kramer, 2003). It has also explained that engagement in stimulating activities, either mentally or socially may decrease the risk of developing dementia via a protective effect due to social interaction and intellectual stimulation (Hertzog, Kramer, Wilson, & Lindenberger, 2008; Wang, Karp, Winblad, & Fratiglioni, 2002).

In sum, human research suggests that exercise as a simple means could have similar benefits for brain health as seen in aging animals; and this may, in fact, extend to aging humans for cognitive function, and may improve learning (Stanley J Colcombe et al., 2003). It is now clear that voluntary exercise is an important factor in improving neuronal growth, creating more stimulation of neurogenesis, improving mental performance and increasing resistance to brain disorder in the adult brain (Cotman & Berchtold, 2002; K. I. Erickson et al., 2011; Mattson, Maudsley, & Martin, 2004; PILC, 2010; Rhyu et al., 2010; Webster, Weickert, Herman, & Kleinman, 2002).

It has also been shown that aerobic exercise could amount to enhancing the Brain Derived Neurotrophic Factor (BDNF) and messenger ribonucleic acid (mRNA) in the hippocampus and several brain areas (Gómez-Pinilla, Dao, & So, 1997; Neeper et al., 1996). In behaviors concerned with activity and metabolism such as aerobic exercise, BDNF may have played a critical role relation between metabolism and cognitive function (Vaynman & Gomez-Pinilla, 2006). In fact, a

close association must exist between metabolism and proper neuronal function. Despite the important role of BDNF protein expression in age-related hippocampal atrophy and despite the effects of exercise on hippocampal volume and function, it is suggested that long-term and also higher amounts of physical activity could be advantageous for brain volume and cognitive performance in older adults (K. I. Erickson, Miller, & Roecklein, 2012).

In a study 1993, effects of aerobic and anaerobic training executive and non-executive control processes were examined. The main focus rested on the question why performance in tasks with components of executive control processes improved in the aerobic program, but not in the anaerobic one; it was puzzling that non-executive control processes experienced equal trends in both programs (R. D. Rogers & Monsell, 1995). Also , it was confirmed that physical activity intervention is important for maintenance or even improvement of cognitive health and function during a lifetime (Etnier et al., 1997).

It is probable that, alongside the general effects of physical education, executive and non-executive cognitive processes benefit from various kinds of exercises, physical trainings could intervene specifically on some cognitive processes and brain regions (Hillman et al., 2008). A recent study has reviewed only a single session of aerobic exercise on executive function of 34 students from 18 to 27 years old. They were randomly assigned to one of three aerobic exercises which conditions included: mild, moderate and vigorous intensity exercise. After the exercise, the participants completed three standardized tasks that demanded executive function, including Stroop, Go-No Go and Stop Signal measures. The duration of the exercise amounted to 35 minutes and all three executive function tasks lasted almost 30 minutes. Thus, it was suggested that effects of exercise are not uniform across all measures of cognitive function, although they confirmed beneficial effects of moderate aerobic training on some measures of cognition (Lowe et al., 2014).

Therefore, although exercise is known to create a flow of biological processes that support brain plasticity (Cotman & Berchtold, 2002; Knaepen, Goekint, Heyman, & Meeusen, 2010), it is possible that different kinds of physical exercises establish dissimilar response (Caspersen et al., 1985; Dishman, Sallis, & Orenstein, 1985; O'Sullivan, Phyty, Twomey, & Allison, 1997); for this reason, to explore our hypothesis, we sought two protocols for this human study.

2.6.1 Exercise and brain

Although, research on humans has demonstrated improved cognitive performance as a result of physical activity in older adults; however, there are clearly limitations on the human brain studies. Non-human animal research can directly examine the cellular and molecular changes that are occurred by exercise, which in humans can only be indirectly studied.
In current study, the literature of the human and non-human have been separately produced.

2.6.1.1 Non-human, animal researches

Aging is a biological process described by a progressive decline in physiological functions that leads to mortality. These changes in the hippocampus may lead to cognitive decline (Van Praag et al., 2005) and can degrade memory performance in adult individuals (Buckner, 2004; Hedden & Gabrieli, 2004; Kim et al., 2010).
Findings on lesions in non-human primates and studies on functional neuroimaging with healthy human subjects have led to a de-emphasis of the hippocampus' role in learning and memory, and have pointed to hippocampus regions and beyond that in the MTL as well as the neocortex, as to be equally important components of memory circuits in the brain (Small, 2001; Tulving & Markowitsch, 1997). It has also been observed that some regions of the cortex – such as the frontal, prefrontal and parietal cortices – show the greatest age-related

declines in humans and it is interesting that these regions are identified to engage cognition and executive functions (Stan J Colcombe, Kramer, McAuley, Erickson, & Scalf, 2004; Kramer et al., 1999).

Research on animals has demonstrated that aerobic exercise is associated with increased neurogenesis (Kim et al., 2010; Kramer et al., 1999; Kronenberg et al., 2006) and angiogenesis or, in other words, the increased number of capillaries in the brain (Fordyce & Farrar, 1991; Swain et al., 2003). It is even reported that exercise previous to brain injury in animals may lower the extensity of such injury (Cotman & Engesser-Cesar, 2002).

It is also reported that physical activities such as voluntary running, increases neurogenesis of granule cells in the dentate gyrus of adult mice. It is stated that an increase in cell proliferation, cell survival in the dentate gyrus and also in the production of granule cells of the hippocampus are the most observed exercise effects when adult mice are exposed to voluntary running (Brown et al., 2003; Van Praag, Christie, et al., 1999; van Praag, Kempermann, et al., 1999). Those results have shown that neurogenesis has only increased in mice with unlimited access to running activity.

Moreover, both voluntary physical activity and enrichment induced almost double the total number of surviving new-born cells in the dentate gyrus. The production of new cells in the brain requires an increased nutrient (Fordyce & Farrar, 1991; Van Praag et al., 2005). There is confirmation that consumption of dietary supplements in concurrence with exercise can enhance neurogenesis, cell survival, synaptic plasticity and vascular function (van Praag, 2009; Van Praag, Christie, et al., 1999; van Praag, Kempermann, et al., 1999). A significant difference has formed during Bromodeoxyuridine[20] (BrdU-positive) between cell amounts of young and old mice in the dentate gyrus. It has been confirmed that

[20] Bromodeoxyuridine is a synthetic nucleoside that is an analog of thymidine, and BrdU is used in the detection of differentiation, survival, proliferating and cycle cells in vivo and vitro.

cell genesis declines in aging mice, and that they have fewer new cells than in young mice; it is concluded that voluntary exercise ameliorates some of the deleterious morphological and behavioral consequences of aging (Van Praag et al., 2005).

In 1992, a study compared the morphology of the cerebellar cortex in adult rats that were exposed to repetitive exercise with motor learning; it found the distance from blood vessels to be of a shorter diffusion under aerobic conditions compared to motor-skill learning tasks. Here, the rat's motor skills increased notably. The volume of the molecular layer per Purkinje[21] neuron increased, as well as the number of blood vessels, which were capable of maintaining the diffusion distance. (Isaacs, Anderson, Alcantara, Black, & Greenough, 1992). Additionally, it has been suggested that motor skills training induce remarkably changes that could create beneficial impact in the aged brain (Kleim, Jones, & Schallert, 2003).

Effects of aerobic exercise on the Brain Derived Neurotrophic Factor[22] (BDNF), messenger ribonucleic acid (mRNA) in the hippocampal and in amygdala sub regions were also investigated. It had already been established that exercise treatments can develop BDNF level (Cotman & Berchtold, 2002). An aerobic running program can increase BDNF (Cotman & Engesser-Cesar, 2002), it may also increase mRNA in the dentate gyrus and the basolateral amygdala (Greenwood, Strong, Foley, & Fleshner, 2009), cell proliferation, cell survival and neurogenesis in the hippocampus; indeed, spatial learning is dependent on an improved hippocampus (Van Praag, Christie, et al., 1999; van Praag, Kempermann, et al., 1999). Research has shown that physical activity can change

[21] Purkinje cells are found inside the Purkinje layer in the cerebellum and degeneration of Purkinje cells leads to severe motor disorders such as incoordination and disturbances of fine motor skills. It has been reported that loss of cerebellar Purkinje cells, along with generalized atrophy of the brain (Vonsattel & DiFiglia 1998).

[22] The BDNF protein is known to regulate neurogenesis, synaptic plasticity and survival actions in various parts of the central nervous system.

in the hippocampus' gene expression[23] which could improve the memory system (Cotman & Engesser-Cesar, 2002), as well as enhance the gene expression of BDNF and other growth factors that promote neurogenesis, angiogenesis and synaptic plasticity (Intlekofer & Cotman, 2013).

2.6.1.2 Human researches

Recently, the relation between exercise and cognition on seniors has been explained. In human studies on the relationship between physical training and cognition, some possibilities – that merit further attention – include: cognitive processes that were examined, intensity and duration of exercises, the distinction between involved energy systems, gender, age range, health and diseases as well as the education of participants (Barnes, Yaffe, Satariano, & Tager, 2003; S. Colcombe & Kramer, 2003; Hillman et al., 2008; Kramer & Erickson, 2007). Effects of aerobic-exercise condition on the cognitive function of older individuals have been examined and it has been reported that executive-control processes were more affected by the exercise intervention than other cognitive processes (Kramer et al., 1999; Kramer et al., 2001).

Another study reported that aerobic programs generally improve cognitive function and spatial executive control more than other cognitive functions (S. Colcombe & Kramer, 2003). In a six-month perusal, 59 aged sedentary, however healthy women, participated in the anaerobic (toning and stretching) and aerobic groups. Also 20 young adults served as controls for the magnetic resonance imaging. Increases were observed in the brain volume due to the fitness training of only the subjects who participated in the aerobic exercise but not for the participants of the anaerobic and younger control group. The study's results have suggested that aerobic fitness without sparing any brain tissue has a strong

[23] Gene expression, in short, is the review of information on DNA, genomic sequences and genetic codes.

biological role in maintaining and enhancing cognitive function and the central nervous system's health in the human aging process (Stanley J Colcombe et al., 2006). Results of another study with 1324 subjects have demonstrated that any rate of moderate exercise performed in midlife or late life is associated with reduced chances of having mild cognitive impairment. In that review, researchers have compared the frequency of physical exercise of 198 subjects suffering from mild cognitive impairment (MCI) with that of 1126 subjects with normal cognition (Geda et al., 2010).

In other research, aerobic and stretching exercises with 120 older adults did show that exercise intervention is effective at increasing the size of the hippocampus. In this study, both aerobic and stretching groups showed improvements in the hippocampal volume and in memory performance (K. I. Erickson et al., 2011). It is known that strength training increase neurogenesis (Vukovic, Colditz, Blackmore, Ruitenberg, & Bartlett, 2012), levels of Insulin-like Growth Factor 1 (IGF-1) (Borst et al., 2002), which in turn is known to have positive effects on neuronal growth, survival, differentiation and performance, and perhaps on function of BDNF and mRNA transcription (Mackay et al., 2003). It has been suggested that at the aging process, BDNF signaling decreases in the brain and amounts of BDNF decrease in hippocampal regions, and that dentate granule cells decrease during aging in animals (Neeper et al., 1996).

Another study revealed that acute aerobic training increases basal peripheral BDNF concentrations which is opposed by findings that don't support those effects for strength exercise (Knaepen et al., 2010). Aerobic physical activity can increase levels of nerve growth factors, such as IGF-1 and BDNF which serves to enhance synaptic efficiency by supporting the survival and growth of a number of neuronal subtypes. As an ability of the brain to activity-dependent remodeling (Bruel-Jungerman, Davis, & Laroche, 2007), these effects may help to accentuate the effects of aerobic training, to support plasticity by enhancing the learning and memory system (McAuley et al., 2004). Furthermore,

it has been stated about several physiological mechanisms of exercise – like decreasing blood pressure, lipid levels and the inhibition of platelet accumulation – (R. L. Rogers, Meyer, & Mortel, 1990), enhanced aerobic capacity and cerebral metabolic demands (Dustman et al., 1984), that they could serve as prevention factors on older adults' cognitive decline (Laurin et al., 2001).
Physical exercise can improve adult hippocampal neurogenesis via the ability to increase neural precursor cell endogenous microglia (Vukovic et al., 2012). Such exercise may improve memory performance and hippocampal-dependent learning by induction of BDNF into the hippocampus (Intlekofer & Cotman, 2013). Thus, transmission of foodstuffs accompanied by the blood during exercise can enhance neurogenesis, cell survival, synaptic plasticity and vascular function and affects as a useful tool the health promotion and cognitive decline prevention in normal aging (Cutuli et al., 2014).

Briefly, non-human examinations clearly suggest an affirmative answer to the question if there are positive effects on cognitive performance due to aerobic exercise. Although, results of human studies nearly confirm beneficial effects of aerobic training on the human brain system, the literature on human non-aerobic training appears to be more equivocal; indeed, there is no consensus.

2.6.2 Brain and metabolism

The cardiovascular system encompasses the heart as an efficient pump, arteries and veins. This system provides blood, transports of all necessary nutrients – such as glucose and O_2 – to every cell in the human body (Waldstein, Snow, Muldoon, & Katzel, 2001). Naturally, the brain depends on the cardiovascular system. Although the human brain uses 20 percent of the oxygen consumed by the body (Dustman et al., 1984), the brain is comprised only two percent of the body's weight (Clarke & Sokoloff, 1999; Dringen, Gutterer, & Hirrlinger, 2000). Looking at the total amount the cardiac output in blood, approximately one liter

is supplied to the brain per minute. This is one fifth of the heart's total output (Waldstein et al., 2001).

Regarding that the level of oxygen is very low inside brain tissue, the brain is one of the neediest oxygen consumers in the body (Zhang, Zhu, & Fan, 2011), consequently, any pause of this blood supply for even a few minutes can be detrimental to the brain. Indeed, this amount of oxygen consumption is one of the potential major causes of age-related destruction of brain tissue (Reiter, 1995), because compared with the kidney or the liver, the brain contains only little antioxidants, such as catalase, glutathione peroxidase[24] (GPx) activity, et cetera – these combine to be an important antioxidant defense in nearly all cells (Cooper & Meister, 1997; Dringen et al., 2000; Ji, 1999). Moreover, the brain is rich in lipids with unsaturated fatty acids (Cassarino & Bennett Jr, 1999; Heales et al., 1999).

Also, in comparison with other organs, the brain appears to be especially endangered in regard to the generation and detoxification of Reactive Oxygen Species (ROS) or Reactive Nitrogen Species (RNS); the mitochondrion, the cell's energy powerhouse, and also low and insufficient in its mitochondrial functions, could be considerable and important on this matter (Cassarino & Bennett Jr, 1999; Cooper & Meister, 1997; Heales et al., 1999; Paradies, Petrosillo, Paradies, & Ruggiero, 2010).

By adding a single electron to the oxygen molecule, ROS is generated (Fisher-Wellman & Bloomer, 2009). The electron transport chain (ETC) transfers electrons from electron donors such as NADH to molecule acceptors. Indeed, the ETC transfers electrons across cell membranes by means of redox reactions and by pairing the electrons' transfer with the protons' transfer (for example H^+ ions), cause to transfer the electrons across a cell membrane (R. K. Murray,

[24] Glutathione (GSH) is a tri-peptide which makes of the amino acids cysteine, glutamic and glycine. To see more information, review biochemistry references.

1996; Shahbazi & Maleknia, 1999). ETC causes an electrochemical proton gradient that works using the energy of ATP or by the generation of chemical energy in the form of phosphocreatine[25]. Since the oxygen molecule is a key electron acceptor, the production of free radicals such as ROS is inevitable.

To complete the whole series minor percentages of electrons do not suffice. Such small amounts only leak to O_2, followed by formations of ROS which may contribute to oxidative stress; implications to aging and a number of diseases can also be made.

ROS is required for many normal physiologic functions; most of production of ROS coupled with their insufficient scavenging by endogenous antioxidants will lead to detrimental oxidative stress. ROS, the most important free radicals in the body, are generated continuously as a natural byproduct of oxidative phosphorylation in all tissues including muscle fibers and, especially, in the mitochondrial respiratory chain – by estimation, 90 percent –, and have important roles in cell signaling and homeostasis (Balaban, Nemoto, & Finkel, 2005; Devasagayam et al., 2004; Kregel & Zhang, 2007; Kulkarni et al., 2007; Schulz, Lindenau, Seyfried, & Dichgans, 2000); nevertheless, all this happens as a mitochondrial dysfunction. In this context, mitochondria act like biosensors and they enable cells to endure changes in aging and age-related diseases (Stowe & Camara, 2009; Wei & Lee, 2002).

Probably such derangement of the brain indicates that a loss of neurons in adult brains cannot be compensated by the generation of new neurons (Dringen et al., 2000). However, the brain is able to function throughout, which indicates the presence of an effective antioxidant system in different brain regions (Cassarino & Bennett Jr, 1999; Heales et al., 1999; Schulz et al., 2000).

[25] Phosphocreatine or creatine phosphate (or PCr) is a substance that, in its chemical partnership with adenosine triphosphate:

$$PCr \xrightarrow{\text{Creatine Phosphokinase}} Cr + Pi + E$$

$$Cr + Pi \xrightarrow{\text{creatin kinase}} \text{Phosphocreatine}$$

Recently, gerontological studies from a biological perspective have revealed different molecular pathways involved in the aging process and pointed out mitochondria as vital regulators of longevity (Bratic & Trifunovic, 2010). The roles of mitochondria are known to continuously produce energy (Dringen et al., 2000) and regulate the cellular metabolism through the aerobic/oxygen system (Miquel, Economos, Fleming, & Johnson Jr, 1980; R. K. Murray, 1996; Voet, Voet, & Pratt, 2007). The function of the mitochondrion and the aging process are considered to be intimately intertwined, and this happens by means of the respiratory chain dysfunction and the formation of ROS, which may lead to damage of the mitochondrial constituents including mitochondrial proteins, lipids and DNA. This DNA damage can change neuronal survival (Miquel et al., 1980; Pak et al., 2003).

It isn't unexpected that very low levels in the generation of ROS occur during mitochondrial respiration under normal physiological conditions, but progressive oxidative damage to the aged mitochondrial DNA (mtDNA) may lead to DNA strand breaking down and to the occurrence of somatic mtDNA mutations (Paradies et al., 2010; Richter, 1995; Wei, 1992). Accumulation of these mtDNA mutations may lead finally to the progressive decline in cellular and tissue function (Judge & Leeuwenburgh, 2007; Linnane, Ozawa, Marzuki, & Tanaka, 1989; Wei, 1992), this also points to an association between mtDNA mutations and muscle fiber atrophy age-related loss (Pak et al., 2003). There are significant examples in animal research which show that reduction of mitochondrial function would be expected to impair health and shorten lifespan (Rea, Ventura, & Johnson, 2007) and may speed up the brain's aging (Bishop et al., 2010; Sánchez-Blanco, Fridell, & Helfand, 2006).

It has also been demonstrated that older mitochondria alter functionally, indeed, they work economically beneficial, producing less ATP and more oxidants (Shigenaga, Hagen, & Ames, 1994). In general, it is accepted that age-associated decline of respiratory function can result in enhanced production of

ROS in mitochondria (Wei & Lee, 2002). Conversely, augmentation of mitochondrial function has been shown to extend life (Schriner et al., 2005). Thus, mitochondrial function seems to have an important modulating influence on the aging process with either positive or negative effects on a lifespan (Blalock et al., 2004; Loerch et al., 2008; Lu et al., 2004).

DNA damage may change the expression of genes involved in learning, memory and neuronal survival (Lu et al., 2004). Even gene expression studies provide evidence of an association between mitochondrial function during aging and specific changes in gene expression (Blalock et al., 2003; Loerch et al., 2008; Yankner, Lu, & Loerch, 2008; Zahn et al., 2007), they also provide evidence of reduced expression of genes involved in the mitochondrial metabolism. The latter may become more clear in humans with cognitive decrease (Andrews-Hanna et al., 2007; J. A. Miller, Oldham, & Geschwind, 2008; Wei & Lee, 2002; Yankner, 2000).

In some conditions, surprisingly, it has been observed that reduced mitochondrial function could cause a lifespan to increase (Bishop et al., 2010; Branicky, Bénard, & Hekimi, 2000). Indeed, only a modest reduction of function in some genes affecting the electron transport chain can increase a lifespan as a remedy function (Dillin et al., 2002; S. S. Lee et al., 2003; Rea et al., 2007).

The term “uncoupling to survive” is a hypothesis pointing to an inefficiency in the mitochondrial ATP generation which may be necessary to reduce the production of reactive oxygen species (ROS), which in turn could be important in helping to reduce oxidative DNA damage and in slowing aging (M. Brand, 2000). There are studies demonstrating that animals with higher metabolic intensities and oxygen consumption live longer than animals with lower metabolic intensities.

Indeed, increases of metabolic uncoupling suggest that these animals may decrease ROS generation even in the setting of increased oxygen consumption by reducing the mitochondrial membrane potential (Balaban et al., 2005; Speakman et al., 2004). This theory provides a possible explanation of how ROS in a moderately increased concentration may act as a signal to activate a survival pathway and enhance longevity (Bishop et al., 2010). Thus, uncoupling has been proposed as an important mechanism to reduce ROS levels (M. D. Brand et al., 2004; Casteilla, Rigoulet, & Pénicaud, 2001).

2.6.3 Metabolism and health

It appears that physical activity may have the potential to increase ROS production and subsequent oxidative stress. Oxidative stress is defined as an imbalance between the systemic manifestation of reactive oxygen species and a biological system's ability to readily detoxify (Fisher-Wellman & Bloomer, 2009). In other words, physiological-respiratory modifications and increased ATP need during exercise cause the production of ROS (Radak, Chung, & Goto, 2008) which is depended on the oxygen amount consumption (Ramel, Wagner, & Elmadfa, 2004), exercise intensity (Goto et al., 2003) and duration (Bloomer, Davis, Consitt, & Wideman, 2007).

Although the initial view as a common assumption was that increased mitochondrial oxygen consumption leads to a higher production of ROS which may raise the risk of cell damaging (Balaban et al., 2005). Attention to the question of optimal levels of physical exercise is important; such an optimal level may end where an ROS-related disease risk begins to increase (Knez, Coombes, & Jenkins, 2006). Thus, optimal health is dependent on optimal levels of ROS generation (Fisher-Wellman & Bloomer, 2009; Ji, GOMEZ-CABRERA, & Vina, 2006).

It seems optimal levels of exercise inducing ROS are able to stimulate cytokine production from skeletal muscles, and that cytokine has an important role in the regulation of cell signaling (Fisher-Wellman & Bloomer, 2009; Scheele, Nielsen, & Pedersen, 2009). Briefly, it appears that exercise-induced ROS can serve as signal to activate a mechanism of adaptive responses to the ROS-leveling process at rest, during as well as subsequent to exercise (Ji et al., 2006; Knez, Jenkins, & Coombes, 2007).

There are some effects resembling the phenomenon of hormesis[26] when ROS is generated during exercise (Calabrese & Baldwin, 2002; Ji et al., 2006). The similarity lies in the adaptive response to exercise in skeletal muscles, the liver and the brain including increased antioxidant enzyme activity as well as increased resistance to oxidative stress; beneficial effects of exercise can decrease the vulnerability of the body to oxidative stress and to several diseases significantly (Gomez-Cabrera, Domenech, & Viña, 2008; Radak, Chung, & Goto, 2008). What has been reported is contradicting since the relationship between oxidative stress and exercise training is not simple and depends on several factors including mode, duration and intensity of exercise, individual differences, cardiovascular disease, diabetes, hypercholesterolemia, obesity and chronic obstructive pulmonary disease as well as smoking (Fisher-Wellman & Bloomer, 2009). Discrepancies in literature are likely related to the diversity and sometimes incomparability of those factors mentioned above.

The important roles of oxygen on the brain are not only showed in the development, but also illustrated in the brain dysfunctions. Preserving the correct ranges of tissue pressure of the Oxygen (po_2) can be beneficial to normal brain function. Higher or lower levels of tissue oxygen pressure (pO_2) may affect

[26] Hormesis: A repeated low dose stimulation followed by high dose inhibition (Calabrese & Baldwin, 2002 & Ji et al., 2006).

normal chemical production, possibly leading to pathological disorders and brain cell damage (Zhang et al., 2011).

It would appear that acute exercise can aggravate the state of oxidative stress (Sanchez-Quesada et al., 1995), a lower antioxidant status (Ramel et al., 2004) and disease risk (Knez et al., 2006). On the other hand, there are reports supporting that health benefits of physical exercise are connected to the optimal level of exercise-induced ROS including positive effects of regular exercise on cognitive diseases such as Alzheimer and Parkinson's (Scheele et al., 2009).

Furthermore, it has been demonstrated that there is a capacity for an increase in muscle resistance against fatigue (Vollaard, Shearman, & Cooper, 2005). It has also been shown that submaximal resistance exercise increases plasma antioxidants which could enhance antioxidant defenses in response to the oxidative stress of physical exercise (Ramel et al., 2004). There is research on the vulnerability of the body to oxidative stress and diseases being significantly enhanced in sedentary individuals (Radak, Chung, & Goto, 2008).

Benefits of mild and light but not vigorous exercise have been reported, and it is recommended that individuals should dedicate at least 30 minutes of moderate-intensity physical activity each day in order to improve and maintain their health (Armstrong, 2006; Fisher-Wellman & Bloomer, 2009; I.-M. Lee, Hsieh, & Paffenbarger, 1995).

2.7 Summary of literature

To review the study's literature, a number of previous works are listed:

Table 2: Summary of exercise and cognition studies

Date	Author	Results and Conclusion	Domain
1964	Altman, Das	An increase in the weight and the volume of the cortex in the enriched animals	Animal
1988	Blumenthal	Aerobic exercise can improve some aspects of memory-search performance	Human
1996	Neeper	Exercise changes Brain Derived Neurotrophic Factor (BDNF)	Animal
1998	Oliff H.S	Increase in the brain's resistance to damage with exercise and BDNF mRNA expression	Animal
1998	Peig-Chiello, Perrig	No significant effects on cognitive function occurred with the training	Human
1999	Van Praag	Positive effect of running on learning level and neurogenesis	Animal
1999	Kramer	Aerobic exercise improves executive control tasks more than anaerobic training	Human
2001	Yaffe	Women with more physical exercise are less likely to develop cognitive decline	Human
2001	Laurin	Regular exercise could be a potent protective factor for cognitive decline and dementia in elderly	Human
2002	Cotman	Exercise can have effects on gene expression, BDNF, neurogenesis	Animal
2003	Colcombe, Erickson	Benefits of aerobic exercise on the brain health	Human
2004	Colcombe, Kramer	Aerobic exercise can activate several cortical regions	Human

2004	Heyn, Abreu	Exercise increases cognitive function and can have other positive effects in/on people with dementia	Human
2005	Van Prrag	In young animals, exercise increases hippocampal neurogenesis and improves learning	Animal
2006	Kronenberg	Physical exercise induces adult hippocampal neurogenesis	Animal
2006	Colcombe	Aerobic exercise can increase brain volume, nervous system health, improve cognition	Human
2006	Larson	Sufficient and regular exercises decrease probability of dementia	Human
2007	Etnier	Association of aerobic fitness and Apo-lipoprotein E with memory performance	Human
2009	Chang, Etnier	Intensity of resistance exercise can create different response in cognition	Human
2010	Sung Kim	Exercise improves short-term memory, enhancing neurogenesis in the hippocampus	Animal
2010	Geda Yanes	Rate of moderate exercise can reduce odds of having mild cognitive impairment	Human
2010	Creer	Running can increase born newly neurons and neurons of dentate gyrus	Animal
2011	Erickson	Aerobic exercise improves hippocampal volume, BDNF and memory function	Human
2012	Erickson	Long-term and also a higher amount of physical activity could better brain volume and cognitive performance in elderly	Human
2013	Intlekofer	Physical activity can improve hippocampal function through enhanced neuroplasticity	Human
2014	Kirk-Sanches	Exercise can modify metabolic, structural and functional dimensions of the brain that preserve cognitive performance in older adults	Human

2014	Snigdha	Acute and chronic exercise can improve cognitive function even with progressing age	Animal
2014	Lowe	Effects of exercise are not uniform across all measures of cognitive function	Human

As noticed in introduction of the study (section 1), the current study is an interdisciplinary investigation.

In this section, the terms of physiology and psychology related to aging, cognitive function and physical activity are introduced. These terms include physiological principles (for example and metabolism of exercise activity, kinds of exercises and intensity of physical activity) and some main concepts in psychology (for example memory, divisions of memory, neural correlates and several learning theories).

In the next section (3), questions and hypotheses concerning the previously introduced information about aging, several cognitive performances and physical activity are provided.

3 Questions and hypotheses

Age-related cognitive decline or normal and non-pathological cognitive aging is an essential human experience that differs in level between population. Though, the determinants general bodily aging also influence in older adults' cognitive performances, but this factors are not fully known. Progress in the field is taking place across many areas of health-related sciences.

The purpose of this investigation is to study different kinds of physical exercises to preserve cognitive functions for people of advanced age and to provide a new comfortable and practical template for these exercises. Although, physical activity can improve brain health and cognitive performance, there are still some ambiguities in literature about the kind of exercise on cognition. Advanced age can create musculoskeletal disorders and may cause limitations in activities such as running, jogging and walking. Thus, there is still this question: What kinds of physical exercises are more favorable?

3.1 Questions

- At the beginning, we ask whether there is any relationship between various kinds of physical activity and older people's cognitive functions.
- How aerobic and anaerobic exercises may affect cognitive functions in older adults?
- Can decline of cognitive functions during normal aging process be reversed with exercises of light to moderate intensity?
- Can aerobic and anaerobic exercises affect intellectual functions in older adults?
- Can improvement of motor learning develop procedural memory?
- Can development of motor learning improve declarative memory?
- Can development of procedural memory improve declarative memory?
- Which exercise is more effective on memory performance of older adults?
- Can different kinds of exercises preserve short-term and long-term memories similarly in older people?
- Can different kinds of exercise preserve declarative and non-declarative memories similarly in older people?
- Does new motor learning of non-aerobic is more effective on memory performance or repeated aerobic programs such as running.
- If physical exercise can improve cognitive functions, may either aerobic or anaerobic conditions be better to preserve it?

3.2 Hypotheses

In the following, the hypotheses that arise from these questions are introduced.

3.2.1 Hypothesis I

Systematic and regular light to moderate physical aerobic activity leads to a measurable improvement in cognitive functions of sedentary older adults.

A robust relationship exists between the circulatory system and the brain. If any disturbance occurs by vascular disease, it may affect normal brain function. Human's cognitive functions are of the most important mental abilities for independent life. Considering the importance of independent life for elderly, researchers are designing and finding strategies to delay the progression of physical and psychological illnesses and also to continue healthy and independent life.
Considering vulnerability and sensitivity of cognitive functions against factors such as the environment, social, physiological and psychological setting, the fact that prevention or attempts to decrease the accelerating decline of cognition is plausibly more beneficial and easier than treatment (Teasdale, 1988).

In normal aging process arise some changes in brain structure and function that suggest strong decline in tissues density of the brain as a function of aging (Altman & Das, 1965; K. I. Erickson et al., 2009). Physical inactivity accelerates the aging process in many people, whereas increased physical activity slows it down in others (Kramer et al., 2006; Laurin et al., 2001; Podewils et al., 2005).

At present, it appears that all forms of exercise, both aerobic and anaerobic, possess the potential to result in decreased cognitive function loss in healthy aged individuals. Although, there are studies that have failed to observe

the benefits of physical exercise in preserving cognitive function (Broe et al., 1998; Madden et al., 1989); it may be disputed that engaging in physical activity does not play a protective role on cognition and cognitive disorders, but there are many findings reporting regular physical exercise as an important element in health promotion. At the moment, results are promising and suggest that physical aerobic activity, as a preventive strategy and neuroprotective function, may reduce declines in cognitive performance among older adults (Kirk-Sanchez & McGough, 2014; Kramer et al., 1999; Pate et al., 1995). However, the rate of change is not equal among individuals.

In human physiology, the role of physical activity to create exercise-induced adaptations is elucidated and is based on the extensive body of literature. The aerobic exercise may improve aerobic capacity and cerebral nutrient supply as well as increase oxygen saturation and angiogenesis in brain areas crucial for task cognitive performance (Dustman et al., 1984; Fordyce & Farrar, 1991; Kleim, Cooper, & VandenBerg, 2002).

What is clear is that there are several modifiable mediating factors on the aging curve. Among modifiable key factors are physical activities, nutrition, body fat, and muscle mass, each of which can either delay or accelerate the aging process (Stewart, 2005). Furthermore, several other factors appear to play a significant role in the exercise-induced effects including duration and intensity of exercise. Intense exercise appears to have harmful effects on different aspects of human health especially in aging process (Radak, Chung, Koltai, et al., 2008). Determining whether or not the level of exercise intensity is beneficial in seniors' cognition has not solved the problem of much cognitive and aging research yet (Reuter-Lorenz & Cappell, 2008).

3.2.2 Hypothesis II

Systematic and regular light physical anaerobic activity leads to a robust and beneficial influence in cognitive functions of sedentary elderly.

Physical exercise has the potential to result in decreased cognitive functions loss in healthy aged population. It is now clear that increased voluntary physical activity is an important factor to improve neuronal growth, creates more stimulate neurogenesis and improves mental performance in the adult brain (Cotman & Berchtold, 2002; Knaepen et al., 2010; Mattson et al., 2004; O'Sullivan et al., 1997; PILC, 2010).

The ability of physical exercise to impact systems that promote cell survival, neurogenesis and plasticity may be applicable for combating the deleterious effects of aging on brain health and cognitive function. Though, several mechanisms may underlie the potentially protective effects of physical activity on cognitive function. It has been shown that exercise, probably sustain the brain's vascular health by lowering blood pressure, promoting endothelial nitric oxide production, and improving lipoprotein profiles (Taddei et al., 2000). It also increases non-neural components of brain, for instance vasculature, maintains the generating of new neurons in response to exercise and by learning selectively increases synaptogenesis in later life (Churchill et al., 2002). Also, physical activity likely upregulates neurotrophins such as Nerve growth factor (NGF), BDNF and also IGF-1 that support cell proliferation, cell survival, neurogenesis and dendritic branching in the adult brain (Cotman & Engesser-Cesar, 2002; McAuley et al., 2004).

Since that effects of physical exercise enhance older adults' cognitive abilities (Dustman et al., 1984; K. I. Erickson et al., 2011; Kirk-Sanchez & McGough, 2014; Kramer et al., 2006; Kramer et al., 1999; Weuve et al., 2004),

and considering essential differences of exercise types such as aerobic and anaerobic, dynamic and static, concentric and eccentric training, strength and endurance, power and speed, balance, coordination, stretch and flexibility (Fox et al., 1975; Mathews et al., 1976); as well as different physiological responses in the human body to different exercises (Mathews et al., 1976); functions and responses different of brain against different exercise types can create also a wide range of changes such as increasing the blood and oxygen flow to the brain, increasing levels of nerve growth factors that help neurogenesis, support the survival and growth of a number of neuronal cells, and enhancement of synaptic plasticity (Arida et al., 2007; Cotman & Berchtold, 2002; Kempermann et al., 2010; Knaepen et al., 2010; Kramer et al., 2005).

Furthermore, physical exercises improve cognitive function via improvement of learning process, synaptic plasticity and neurogenesis (Intlekofer & Cotman, 2013; McAuley et al., 2004; Van Praag et al., 2005). Thus, physical activity, which has been emphasized as a strategy to slow or reverse cognitive decline is not limited only to aerobic exercises.

Even, regarding the view that oxidative stress is a causal factor in brain senescence (Forster et al., 1996) maybe non aerobic exercises can better reduce the risk of developing dementia in older adults.

3.2.3 Hypothesis III

Learning a new motor skill positively influences semantic memory performance in sedentary older adults.

Although, it has repeatedly been pointed that physical exercise has considerable effects on brain morphology and function (e.g. Dustman et al., 1984; Hill et al., 1995; Kramer et al., 1999; Laurin et al., 2001; Abbott et al., 2004; Weuve et al., 2004; Podewils et al., 2005; Kramer et al., 2006; Davranche & McMorris, 2009; K. I. Erickson et al., 2011; Kirk-Sanchez & McGough, 2014), yet current knowledge is not completed about the connection of motor skill and semantic memory system.

Anyway, it has been found that the motor skills which previously learned, were intact in patients with declarative memory disorders due to amnesia or Alzheimer's disease (Gabrieli et al., 1993; Milner, 1962). Durability and protection of motor learning skills in amnesia diseases indicates that motor skill learning is not dependent on declarative memory areas-related in the brain (Gabrieli, 1998; Milner, 1965b). Although, basal ganglia and cerebellum dysfunction can cause deficits in procedural memory (Mochizuki-Kawai, 2008), but these diseases don't have homogeneous effects on motor skill learning (e.g. Gabrieli et al., 1997; Sanes et al., 1990; Nicolson & Fawcett, 2007). It has been reported that defects in dyslexia (developmental reading disorder) are attributed to an impaired procedural learning system, while there was the intact declarative learning system (Nicolson & Fawcett, 2007).

Also, patients with Huntington's disease (HD) that have mildly impaired mirror-reading skill – which is a procedural ability – despite the pretty well declarative memory for words and the reading experiences (Martone, Butters, Payne, Becker, & Sax, 1984).

Gabrieli et al., (1997) showed that basal ganglia dysfunctions can usually impair motor skill learning and procedural memory system (Gabrieli et al., 1997).

The work of Sanes (1990) showed that Cerebellar injuries can impair procedural memory system. These results indicate that the cerebellum and its associated input pathways are involved in motor skill learning (SANES et al., 1990).

In order to acquire motor sequences, components of basal ganglia are assumed to play a critical role. In contrast, to learn mappings between motor responses and visual cues, it is the cerebellum that seems to be of importance (Willingham et al., 1996). Also, to learn closed-loop skills[27] – and, therefore, engaging in movement's ongoing visual, external feedback – it is the cerebellum who's involvement is needed. In comparison, to acquire any open-loop skills – including delayed feedback and planning of movements – the basal ganglia is crucial (Gabrieli et al., 1997). Studies have also shown that interactive, dynamic neural networks are involved in procedural memory (Gabrieli, 1998).

In case of a dysfunction, other regions may support the defective functioning; for instance, it has been shown that parts of the forebrain such as the striatum which is known as the primary input of the basal ganglia system, is required in some processes of motor learning and procedural memory for example consolidation; developments in procedural memory are not preserved unless the striatum is normal. However when the striatum is defective, other regions of the brain may support optimal and necessary function (Mochizuki-Kawai et al., 2004).

There are also examples where memory on a declarative task is correlated to memory on a related non-declarative (such as procedural memory) task (Bowers & Schacter, 1990). Aspects of cognitive learning are known to depend on the basal ganglia and diencephalon structures that support declarative memory

[27] See Adams' closed-loop theory in section (2.5.3)

(Knowlton, Mangels, et al., 1996; Knowlton, Squire, Paulsen, Swerdlow, & Swenson, 1996).

Also, it has been stated that the basal ganglia is vital for developing perceptual and cognitive skills in addition to procedural learning (Alexander et al., 1986). Thus, it appears that procedural memory system may contribute through the functions of basal ganglia and cerebellum in processes of declarative memory function.

Also, regarding animal literature, it appears that motor skill training induce remarkable changes in the brain that may improve learning and cognitive function of elderly (Cotman & Berchtold, 2002; Isaacs et al., 1992; Kleim et al., 2003; Van Praag et al., 2005).

3.2.4 Hypothesis IV

Developing a new procedural skill is accompanied by a significant improvement of cognitive functions of sedentary aged.

Given a theoretical framework of this work that emphasizes the undeniable presence of age-related decline in cognitive functions (Altman & Das, 1965; Blalock et al., 2003; Burke & Barnes, 2006; Cameron & McKay, 1999; K. I. Erickson et al., 2009; Fabel & Kempermann, 2008; Jiang et al., 2001; Kramer et al., 1999; C.-K. Lee et al., 2000; Mather & Carstensen, 2005; Small, 2001); remembering that since 1962 the neurogenesis in the adult brain has repeatedly been evidenced (Altman, 1962; Altman & Das, 1965).

Also, much evidence of a relation between an active lifestyle and improved cognitive performances (Blumenthal & Madden, 1988; Botwinick &

Thompson, 1968; Dustman et al., 1984; Green & Bavelier, 2008; Kirk-Sanchez & McGough, 2014; Kramer et al., 1999; Larson & Wang, 2004; Pate et al., 1995; Weuve et al., 2004). Unlike the literature on inactive lifestyle, researches are suggesting that physical exercise improves cognitive performances in adult population.

Much evidences show that engagement in intellectually stimulating, mental and social activities through training can create better cognitive functioning and may protect against dementia in future (Hertzog et al., 2008; Wang et al., 2002). Accordingly, the changes in structure of a neuron consequent motor skill learning has been reported (Kolb & Whishaw, 1998) and bearing in mind the role of synaptic strengthening in learning and memory (Bruel-Jungerman et al., 2007), it can be expected that the motor skill learning is able to compensate for inactive lifestyle or disabilities of the elderly such as walking and running. It has repeatedly been stated that the brain regions which are related to procedural memory such as the basal ganglia and cerebellum (Gabrieli, 1998; Gabrieli et al., 1997; Milner, 1962, 1965b; Mochizuki-Kawai, 2008; Mochizuki-Kawai et al., 2004; SANES et al., 1990; Willingham et al., 1996) according to result of some researches, it has been shown that basal ganglia can support cognitive performance (Knowlton, Mangels, et al., 1996; Knowlton, Squire, et al., 1996). Also, studies suggested that aside from physical exercises, the mental activities during learning and specifically during motor skill learning can similarly increase neurotrophic factor production and neurogenesis (Isaacs et al., 1992; Kleim et al., 2003; Mattson, 2000). Thus, there is a considerable preserved potential in older adults' cognitive capacity that may be reached through mental and physical training.

4 Method

The current study is accomplished with the experimental method through the pretest and the post-test. There are three groups which encompasses aerobic, non-aerobic and control conditions. For present study normal, sedentary and healthy subjects (aged 65 to 75 years) were tested. In the following section a description of the conducted procedure in current study is introduced. Furthermore, a brief overview of groups, exercise protocols and neurocognitive tests are included.

4.1 Present study

It has been reported that both aerobic and anaerobic physical exercise can improve cognitive functions and memory performance. In an effort to test our hypotheses about effects of various types of physical activity on cognition and memory function we organized and supervised aerobic or anaerobic programs for sedentary older adults aged 65 to 75, none of them suffering from dementia. All exercise protocols were led by an experienced exercise leader. The exercise programs were conducted three times a week for three months. It should be noted that subjects in the control group didn’t have physical tasks and they only took part in pre- and post-test.

At the beginning (Pre-test) and at the end (Post-test) of the program every subject was tested on two types of memory tests, the Verbal Learning and Memory Test (VLMT) (Mueller, Hasse-Sander, Horn, Helmstaedter, & Elger, 1997) and the Rey-Osterrieth Complex Figure Test (ROCF) (Fastenau, Denburg, & Hufford, 1999).

Also, several other cognitive functions were tested with a neurocognitive test battery including Mirror Reading Task, Trail Making Test, Mehrfachwahl-Wortschatz-Test (MWT-B), Leistungsprüfsystem (LPS-4).

4.2 Sampling method

In the study a voluntary sampling method is used, which is one of the non-probability sampling methods. In fact, we have found only 89 subjects, which have volunteered to participate in our three month experiment. Hence, they were assigned non-randomly into three groups of current study. Often, these participants have a strong interest to participate in their suitable exercise of the survey groups.

4.3 Objectives

Physical activity may improve brain health, cognitive performance and lower the risk of cognitive decline. There are some ambiguities in literature about effects of exercise on cognition; however, there is no unanimous viewpoint. Increased age can create musculoskeletal disorders and may cause limitations in activities such as running, jogging and walking. The purpose of this investigation was to study different kinds of physical exercise to protect cognitive function for people of advanced age and to provide a new comfortable and practical template for these exercises.

4.4 Participants and Groups

89 older adults aged 65 to 75 years without dementia took part in our study; 37 men and 52 women. They were assigned non-randomly – due to individually interested subjects – into one control group (CG) and two exercises groups (EG); the CG consisted of 12 men and 16 women with an average age of 67.93 years. The EG were divided into two sub-groups performing either aerobic (for example, walking and jogging) or anaerobic exercises (stretching, toning, coordination, Stroop training during movement training, equilibrium, muscle activity, etc.). The aerobic sub-group included 15 men and 15 women with an average age of 68.52 years. The anaerobic group consisted of ten men and 21

women with an average age of 68.20 years. Participants of both EG groups have been trained three times a week for three months. Both exercise groups trained about 45 minutes each time with an academic supervisor. In contrast, subjects in the CG only participated in the pre-test and the post-tests.

4.5 Variables

In current study, the dependent variables are older adults' cognitive functions that encompasses visual and verbal memory performances, visual search, scanning, Speed of information processing, mental flexibility, executive functions, Mirror reading task and intelligence includes verbal and non-verbal.
The independent variables are two various physical exercise conditions, which are compared to the inactivity condition and also together.

4.6 Groups and exercise protocols

In this medium case and prospective study we showed tested 89 adults aged 65 to 75 years without dementia took part in the study. They were assigned non-randomly – due to *individually interested subjects* – into one control group (CG) and into two exercise groups (EG). The EG divided into two sub-groups doing either aerobic exercises (for example, walking and jogging) or anaerobic[28] exercises (e.g., stretching, toning, coordination, Stroop training during movement training, equilibrium, muscle activity, etc.). The aerobic sub-group (AEG) consisted of 30 participants with an average age of 68.52 years. The anaerobic group (ANEG) consisted of 31 participants with an average age of 68.20 years. The control group (CG) consisted of 28 participants with an average age of 68.03 years. Participants of both EG groups trained three times a week for three

[28] For more information see sections (2.2 and 4.6.3).

months. In contrast, subjects in the CG only took part in the pre-test and the posttest.

4.6.1 Control group (CG)

As mentioned, the control group wasn't given any physical tasks and they only took part in pre- and post-test. They were sedentary, healthy, lived routine lives without dementia or other major diseases.

4.6.2 Aerobic exercise group (AEG)

The aerobic exercise condition was designed to influence physical fitness as typified by cardiorespiratory endurance (Kramer et al., 2001). The exercise program was conducted three times a week for twelve weeks. Basic principles of exercise programming were followed, including adequate warm-up – five min. more slowly than walking in main program – and five min. of cool-down periods, progressive and gradual increments in exercise duration and energy expenditure, and instructions regarding avoidance of exercise-related injuries. The exercises consisted of aerobic activities such as walking, brisk walking, Nordic walking, jogging and running. The duration of exercises remained constant, fixed around 45 minutes and with a light speed almost each kilometer in nine to 13 minutes. Additionally, the aerobic participants were training in each session about 3000 till 4500 meters. It was very important for the aerobic-group subjects to continue the task for 45±5 minutes. In respect to the exercise prescription and subjects' situations, the moderate intensity level began light and gradually increased throughout the program. Exercise sessions were initially conducted at Bielefeld University on the Finnbahn, which is an uneven running track, and involved participants walking outdoors on a premeasured route of almost 500 meters.

4.6.3 Anaerobic exercise group (ANEG)

First it should be noted that when the term “anaerobic” exercise is mentioned, regarding the section exercise and metabolism (2.2), it does not mean that this exercise induces the increasing of lactic acid in bodies’ participants. Because in the study, the intensity of anaerobic protocol is farther down than anaerobic threshold levels and consequently it doesn’t induce the increasing of lactic acid. Thus, it will be used in the same way as “non-aerobic”.

The program of the anaerobic group was conducted three times a week for three months each session 45±5 minutes. The participants trained in a small gymnasium of Bielefeld University. The focus of this program laid on providing an organized program of stretching, toning, coordination, balance, Stroop exercise, and equilibrium, muscle activity, calisthenics, and circuit training. Each session was preceded and followed by ten minutes of warm-up and cool-down exercises. The stretching-exercise battery was constant, controlled and contained about 40 exercises and every stretch was done only once and lasted for nearly ten seconds. This program emphasized stretches for all large muscle groups of both the upper and lower body as well as fine and gross motor skills. Our Stroop exercises (Hillman et al., 2008; Lowe et al., 2014; MacLeod, 1992) were designed for body movement and displacement, combining Stroop and other exercises. For example, for the participants to take a step forward, backward or sideways we used train numbers, names of flowers, colors, animals, etc instead of actually saying the direction.

Fine motor skills are small movements with hands, wrists, fingers, feet, toes, lips and tongue, whereas gross motor skills involve movements of/with arms, legs, feet or the whole body. We often accomplished finger movements as a fine motor skill, for example finger abductions and adduction for both hands and legs.

Toning exercises are physical exercises that are used with the aim of developing a figure with a large emphasis on musculature. Toning exercises are isotonic and isometric (Fox et al., 1975; Mathews et al., 1976).

In an isotonic exercise – exemplary for most exercises – a contracting muscle shortens against an increasing load; the resistance – meaning, the weight lifted – does not remain the same throughout the exercise (Fox, 1984).

Tension is at the highest level when the body part in question eases off and is parallel with the floor, above and below. Following, tension changes with muscle length. Concerning isotonic contractions, muscles length changes the, while weight remains unchanged. Pushing an object at a constant length of muscle is an example for isotonic contractions (Fox et al., 1975; Mathews et al., 1976). Almost fifteen motions of body muscles were achieved by our groups every session. Those exercises were performed with about ten repetitions and moderate resistance, along with very short rest periods.

Concerning isometric exercises, they shall be understood as a type of strength training. Here, muscle length and the joint's angle are constant whilst contracted; they are realized within static positions. In an isometric contraction muscle and joint – while confronted with a resisting object – remain static (Mathews et al., 1976).

In our groups, every such motion has been performed for about ten seconds and with moderate resistance, allowing short resting periods. Approximately thirty particular motions were performed every session.

These were, for example:

1. Chair-leg extension; aiming to strengthen quadriceps and thighs
2. Hand press; aiming to strengthen biceps, triceps and the chest
3. Wall push-off; aiming to strengthen chest, triceps and shoulders

4. Overhead press; aiming to strengthen shoulders
5. Drawing-in Manoeuvre; aiming to strengthen core
6. Side bend; aiming to stretch the back and sides
7. Cross arm; aiming to stretch the upper back
8. Neck stretch; aiming to stretch the neck
9. Isometric squats; aiming to strengthen the front thighs
10. Palm press; aiming at biceps, shoulders and chest

When performing an isometric exercise we don't move or put muscles through any range of motion. We simply hold a pose for as long as we can; for example, when holding a static push-up position or a dumbbell in one hand with a mid-bicep curl, or even pushing against an immovable object, such as a wall.

Coordination is an ability related to fitness and performance (Stewart, 2005) that is required to climb stairs, walk, run, prevent injuries and continue an active lifestyle. With advancing age comes a gradual decline in coordination (Paquette, Paquet, & Fung, 2006), the latter itself being a product of strength, mobility, neuromuscular control and balance. Each of these elements is potentially responsive to appropriate exercise training, thus coordination could improve in response to exercise. Coordination skills include eye-hand and hand-leg coordination, simple and complex one-leg balancing – which may be controlled movements of hands, legs or the head –, bilateral coordination and also smooth, controlled movements of the body and so on, all without falling over.

Equilibrium and balance: poor balance affects people of all ages and can lead to injuries. The elderly are especially susceptible to injuries related to incoordination and poor balance such as dislocated or broken hips. Balance improving exercises can decrease the risk of injury and boost a person's confidence. It may be the first element of a complete workout regime that includes stretching and strengthening exercises.

Neurocognitive tests

Cognitive functions were compared in all three groups with a neuropsychology test battery including Rey-Osterrieth Complex Figure Test (ROCF), Verbal Learning and Memory Test (VLMT), Mirror Reading Task, Trail Making Test Parts A and B (TMT-A&B), Mehrfachwahl-Wortschatz-Test (MWT-B), Leistungsprüfsystem (LPS-4).

4.6.4 Rey-Osterrieth Complex Figure test

The ROCF is a neuropsychological test extensively used in clinical practice to investigate visuospatial constructional functions, visuographic memory and some aspects of planning and executive function (Caffarra, Vezzadini, Dieci, Zonato, & Venneri, 2002).

Copy:

In the Copy condition the participant is given a piece of paper and a pencil and the stimulus Figure is placed in front of participant (test person or examination candidate). The participants reproduce the Figure to the best of their abilities onto the paper. The test is not timed, but the length of time needed to copy the Figure is observed. Once the copy is completed, the stimulus Figure and the participant's copy are removed from view. Each copy is scored for the accurate reproduction and placement of 18 specific design elements.

Immediate recall:

After a three-minute delay, the participant is asked to reproduce the Figure from memory.

Delayed recall:

After a longer delay of almost 30 minutes the participant may again be asked to draw the Figure from memory. Participants are not told beforehand that they will be asked to draw the Figure again. The immediate and delayed recall conditions are therefore tests of incidental memory.

4.6.5 Verbal Learning and Memory Test (VLMT)

During VLMT verbal working memory components' were examined, including recall and recognition. Subjects were asked to remember words from different lists. In trials 1-5, some participants had to read 15 nouns from list A, while others needed to remember and repeat words in a particular order. In trial 6, another list of 15 words was introduced. This interference list is read to the subjects, after which they are required to recall as many items as possible. In trial 7 (while no list is read) participants are asked to name words from the A list; as many as they are able to recall.

After a 30-minute break, trial 8 follows: the examinees are again asked to recall items from list A.

With trial 9, recognition is tested: 50 words are read by the examiner; then, participants are instructed to identify items from list A by simply stating "Yes" or "No" when asked (Mueller et al., 1997).

4.6.6 Mirror reading task

To assess procedural memory we used a new version of the German mirror reading task (Borsutzky, Fujiwara, Brand, & Markowitsch, 2010). A booklet of 15 pages with German mirror writing was given to the subjects; 30 words in total, two words per page, and eight to ten letters per word. The participants were

required to read the words out loud accurately and quickly. Any incorrect reading was pointed out to the subject. After a correct reading of one page, the next one was given. The time a subject needed to successfully read a page was used as a measure. All incorrect responses were counted.

After a delay of 30 minutes an unannounced second trial was administered. Here, 30 words on 15 pages were used; ten words of which were identical with the one's used during trial 1. Another ten were used for interference; they were new, but still similar (e.g. "***Verbrecher***" and "***Verbrechen***" see Table 3). Word sequences were kept random throughout trials (Borsutzky et al., 2010).

Table 3: one example of the mirror reading task items, how to look like the words

	Priming	***Procedural memory***	***Interference***
Trial 1	Ergebnis	Hindernis	Waldbrand
	Vorhaben	Verachtung	Verbrechen
Trial 2	Ergebnis	Detonation	Waldrand
	Vorhaben	Erkenntnis	Verbrecher

The English translation of the words:
Priming: trial 1: "result" and "purposes", in trial 2 similar to trial 1.
Procedural: trial 1: "obstacle" and "contempt" and in trial 2: "detonation" and "insight".
Interference: trial 1: "forest fire" and "crime", and in trial 2: "forest edge" and "criminal".

If the subject's reading speed changed from trial 1 to trial 2, a priming performance was measured. To create an index of skill acquisition (for procedural learning), reading-time improvements of new words were used as a measure. It was this study's assumption that our subjects would be disturbed in their automatic reading processes by the mentioned interference items by virtue of their similarity to trial-1 words; due to priming effects, subjects may have tend

to recognize those similar trail-1 words. Then, during reading, they may notice differences and proceed to initiate re-analysis of said words. This may cause reading times to prolong. Concerning interference words of trial 2, though, the number of reading mistakes produced – as compared to items for the priming-and-procedural memory – may rise when subjects experience shortcomings in suppressing activated memory traces.

4.6.7 Trail Making Test (TMT)

Cognitive Functions were compared in all three groups with a neuropsychology test battery including Trail Making Test Parts A and B (TMT-A&B). It has been shown that increasing age and decreasing levels of education can significantly decrease the visual search, scanning, speed of processing, mental flexibility, and executive functions. (Corrigan & Hinkeldey, 1987; Gaudino, Geisler, & Squires, 1995; Reitan, 1958; Tombaugh, 2004).

Guadino et al., (1995) have indicated that part B is more difficult than part A not only because it is a more difficult cognitive task, but also because of its increased demands in motor speed and visual search (Gaudino et al., 1995).
The attention, visual search, scanning, Speed of information processing were assessed by the TMT-A, and to assess the mental flexibility and executive functions we used the TMT-B. In the study, the levels of education intervene only in statistical analysis of TMT results.

4.6.8 Intellectual functions

To assess verbal intelligence functions we used the German version of the “Mehrfachwahl-Wortschatz-Test” (or MWT-B) (Lehrl, 1977). It was used to estimate non-verbal intelligence functions from subtest ‘Reasoning’ form the “Leistungsprüfsystem” (or LPS-4) (Horn, 1983) in all three groups.

4.7 Physiological test

We didn’t measure any physiological abilities of both exercise groups. Aerobic group participants performed light- to moderate-intensity of walking, Nordic walking, jogging and running. These levels of activities were easy to be done for all of participants, and it was never been vigorous. At first, they have been trained the main program, in almost 35 min., 3000 to 3500 metres, but finally they did more than 4000 metres. Anaerobic group participants often have been performed a same program of stretching, toning, coordination, balance, Stroop exercise, and equilibrium, muscle activity, calisthenics, and circuit training without any physiological measurement.

For this study, both exercise groups have trained synchronous for twelve weeks. Also, the both protocols have been accomplished in four successive times; till we get enough subjects.

Though, more than 120 individuals were participated for this study. A number of them due to individual reasons have cancelled their participation. Ultimately, 89 of participants have continued the program. Consequently, the results of 89 subjects were analysed.

5 Results

In the following chapter, first the study data reported the means and standard deviations of groups and afterwards the results Pairwise comparisons of groups.
Our results are based on a medium sample, using a prospective design, avoiding biases related to retrospective assessment of regular physical activity and other exposures.
All statistical analyses were performed with the version 22 of IBM Statistical Package for the Social Sciences (SPSS) Statistics.

5.1 Samples and population

A group of 89 samples (36 male, 53 female) healthy and sedentary was tested. The samples ranged in age from 65 to 75 years (M = 68.26, SD = 2.97). 48 persons of them had studied less than twelve years and 41 persons more than twelve.
All have lived in Bielefeld, Germany. Thus, statistical universe current study encompasses older adults people aged 65 to 75 years from Bielefeld.

5.2 Procedure of study

Samples were assigned non-randomly – due to *individually interested subjects* – into one control group (CG) and into two exercise groups (EG). The EG is divided into two sub-groups doing either aerobic exercises (for example, walking and jogging) or anaerobic exercises (e.g., stretching, toning, coordination, Stroop training during movement training, equilibrium, muscle activity, etc.). At the beginning, all of participants in three groups (Aerobic, Anaerobic and Control) were measured with a neuropsychological test battery. Participants of both EG groups trained three times a week for three months. In contrast, subjects in the CG only took part in the pre-test and the posttest. After the experiment, subjects

of both EG with the same neuropsychological examination as post test were controlled.

5.3 Statistical analyses

First, to determine whether or not our observations are obtained from a normal distribution, we used one-sample Kolmogorov-Smirnov test. The results showed that data distribution is Normal (see results of Kolmogorov-Smirnov tests in Appendix C).

5.3.1 Descriptive data

Descriptive data of samples involves number of samples, distribution of gender, frequency of age, and level of education in groups as well as accumulation of samples in the age. Table 4 shows Means (M), Standard Deviations (SD) of age in groups.

Also, the descriptive data encompasses the frequency of participants in groups (N), frequency of gender – female (f), male (m) –, as well as education levels (Edu. Lvl.), – less than twelve years (< 12), or more than twelve years (> 12) – of groups are listed in Table 4. Moreover, Figures ten to 16 show this information. To review all the raw data of the study, see Appendix A.

Table 4: Descriptive data of the participants

Groups	Mean	SD	N	f	m	Edu. Lvl. < 12		Edu. Lvl. > 12	
						f	m	f	m
Control	68,0357	2,87366	28	17	11	11	5	6	6
Anaerobic	68,2000	2,99885	30	15	15	8	7	7	8
Aerobic	68,5161	3,11845	31	21	10	13	4	8	6
sum	68,2584	2,97538	89	53	36	32	16	21	20

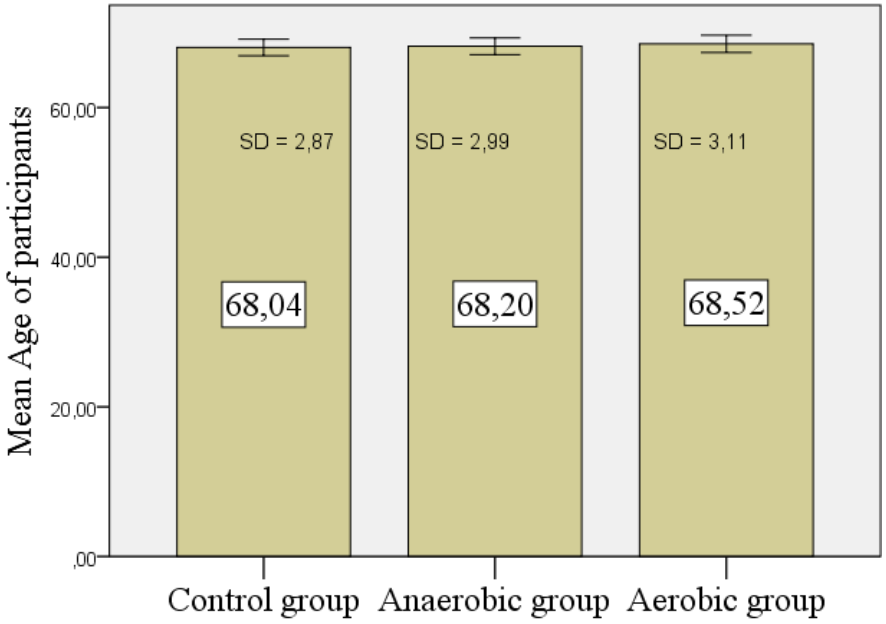

Figure 10: Means and Standard Deviations of groups aged.

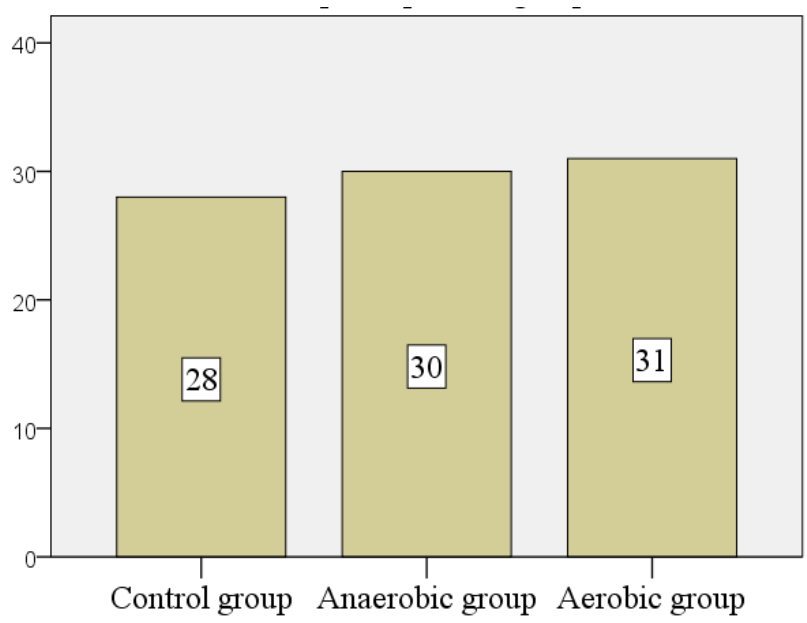

Figure 11: Frequency of participants in groups.

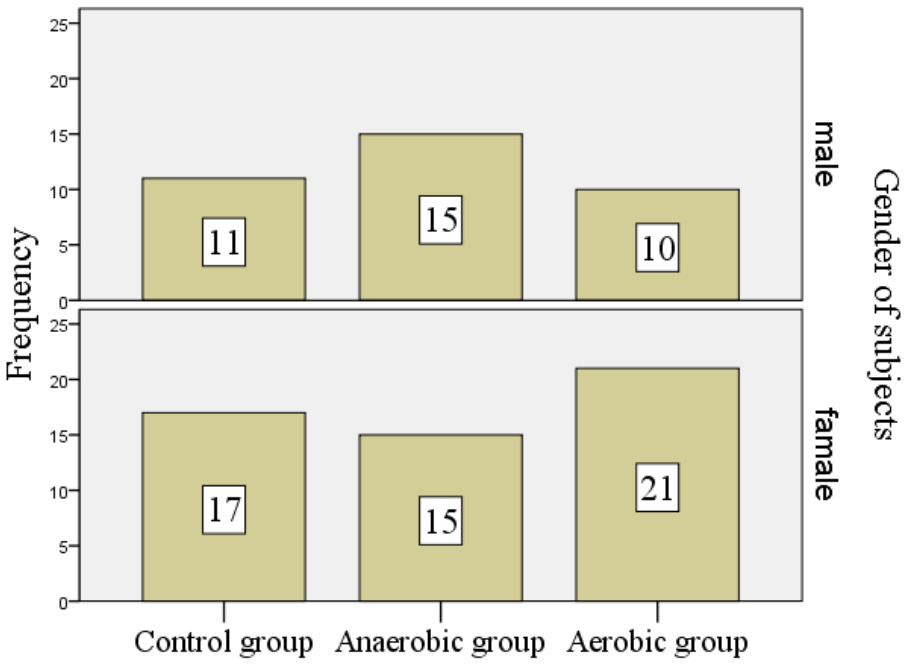

Figure 12: Distribution of gender participants in groups.

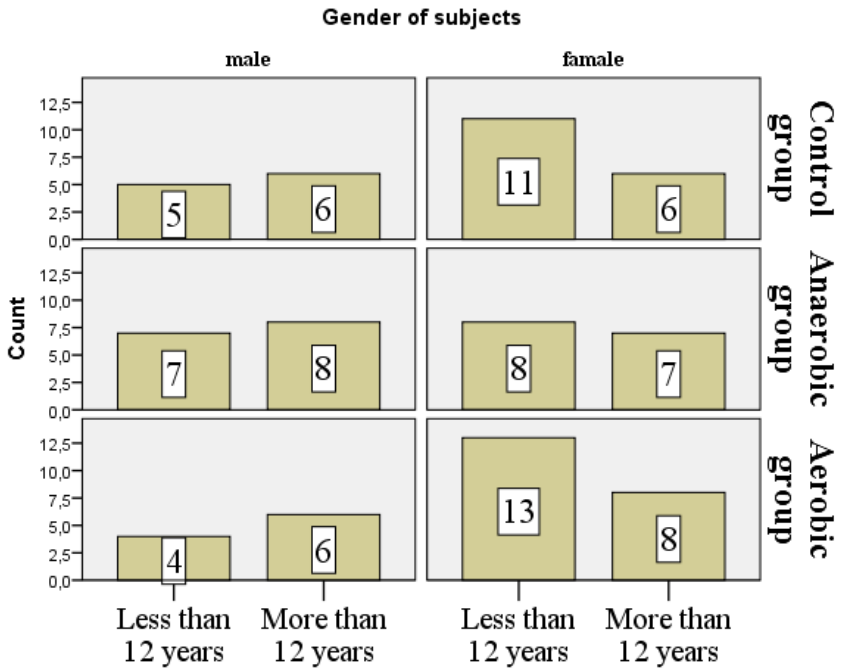

Figure 13: Distribution of education levels of participants in groups.

Also, the following Figures show frequency of participants on the basis of age (Fig. 14) and distribution of age within the groups (Fig. 15 and Fig. 16)

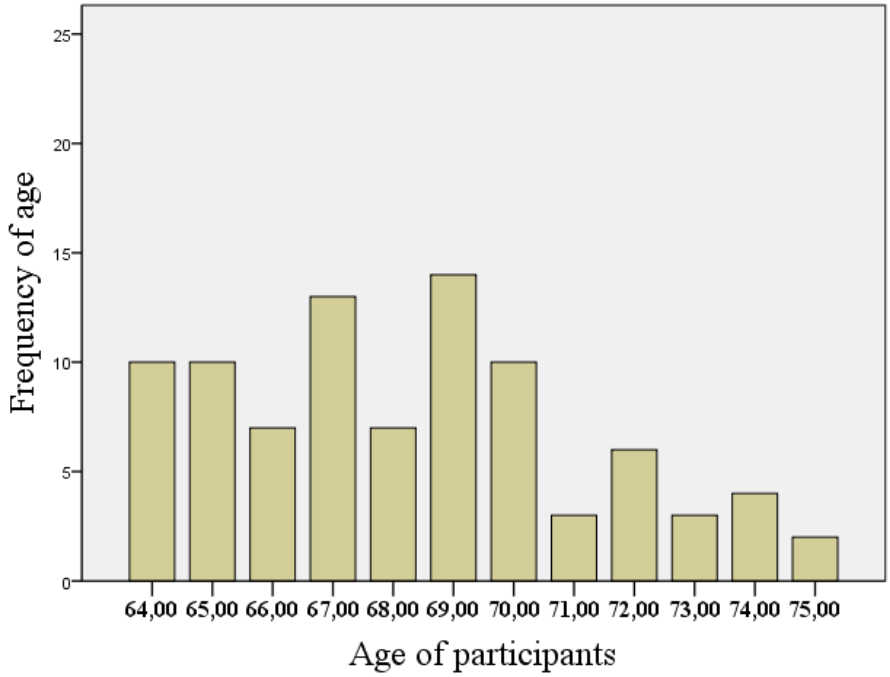

Figure 14: Frequency of participants on the basis of age.

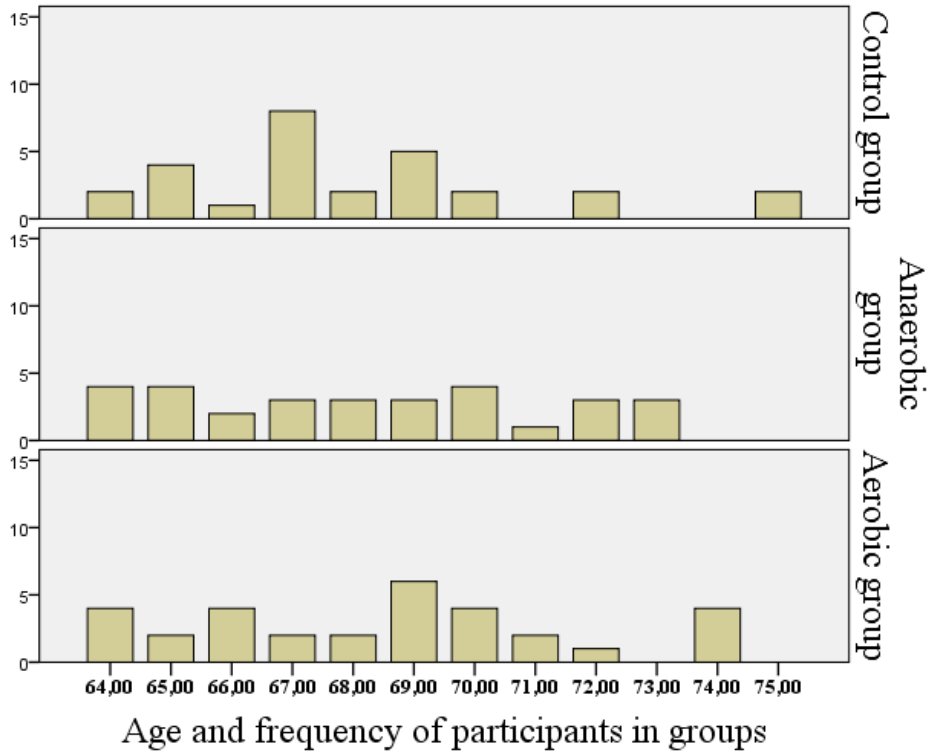

Figure 15: Distribution of age in the groups.

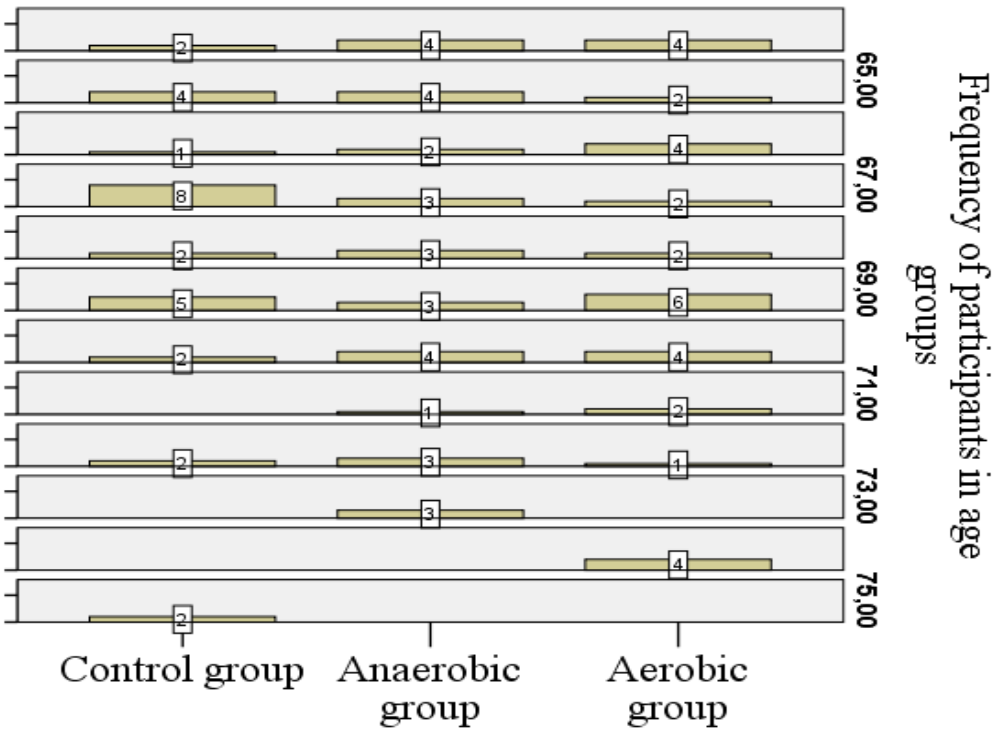

Figure 16: Distribution of age in the groups.

5.3.2 Data analysis

Results of the neurocognitive tests encompass the Means (M) and Standard Deviations (SD) of these three groups as listed in Table 5. Also, the Pairwise comparisons of groups are presented in Table 6. The effects of various kinds of physical activity on cognitive functions were analyzed using a univariate analysis of covariance (ANCOVA) and was performed on the data in which the exercise group was the between-subjects variable. The separate analyses were performed to assess the associations between kinds of physical activities and cognitive protection. The Bonferroni test was used to investigate what changes occurred among groups.

The test Bonferroni was used to investigate changes occurs between which groups.

Table 5: Means (M) and Standard Deviations (SD) of variables

Test Variable		Control group (N = 28)		Aerobic group (N = 31)		Anaerobic group (N = 30)	
		Pre-test	Post-test	Pre-test	Post-test	Pre-test	Post-test
ROCF[a] immediate recall[1]	M	71.71	63.96	63.97	73.52	60.07	76.57
ROCF immediate recall	SD	10.43	11.20	10.60	12.05	12.15	8.90
ROCF delayed recall[1]	M	70.89	64.07	63.13	74.26	60.30	75.97
ROCF delayed recall	SD	11.77	11.61	11.43	10.56	13.49	10.29
VLMT[b] immediate recall[2]	M	59.25	50.20	62.98	65.65	61.83	64.42
VLMT immediate recall	SD	31.33	28.99	26.87	23.25	26.42	27.87
VLMT after learning[2]	M	42.38	35.96	40.45	41.37	33.77	51.22
VLMT after learning	SD	26.21	26.30	22.11	24.69	21.33	26.44
VLMT after interference[2]	M	46.14	33.32	45.89	41.81	36.50	51.03
VLMT after interference	SD	32.55	29.23	26.16	31.27	24.53	31.88
VLMT delayed recall[2]	M	41.38	27.95	39.23	42.61	35.55	46.03
VLMT delayed recall	SD	31.62	24.20	25.83	32.40	26.43	31.60
Mirror reading priming [s]	M	191.85	214.67	207.77	154.45	182.38	103.14
Mirror reading priming	SD	108.65	130.55	211.59	171.60	138.95	53.89
Mirror reading procedural [s]	M	198.04	234.15	242.29	152.87	197.52	115.31
Mirror reading procedural	SD	99.99	126.04	264.30	134.90	109.39	45.98

Mirror reading interference [s]	M	181.59	176.74	188.48	111	158.66	90.41
Mirror reading interference	SD	98.56	94.69	149.68	63.49	93.73	36.04
TMT[c]-A[d]	M	52.50	40.00	49.68	60.65	53.00	67.00
TMT-A	SD	29.01	28.02	25.23	53.89	30.53	26.28
TMT-B[e]	M	46.43	33.21	49.68	62.58	48.67	66.67
TMT-B	SD	28.05	27.49	31.36	28.75	31.81	30.10
MWT-B[f] verbal intelligence	M	117.50	114.29	119.84	122.39	121.80	127.63
MWT-B verbal intelligence	SD	10.99	10.83	12.55	10.66	11.31	10.88
LPS-4[g] non-verbal intelligence	M	112.11	110.39	112.23	115.90	112.57	117.93
LPS-4 non-verbal intelligence	SD	11.28	10.96	10.47	11.60	10.07	11.06

[a] *Rey-Osterrieth Complex Figure Test (ROCF)*

[b] *Verbal Learning and Memory Test (VLMT)*

[c] *Trail Making Test (TMT)*

[d] *Speed of information processing*

[e] *Executive functions*

[f] *Mehrfachwahl-Wortschatz-Test (MWT-B)*

[g] *Leistungsprüfsystem (LPS-4)*

[s] *Second*

[1] *Raw score*

[2] *Percentile*

Mean difference (MD), Standard error (SE) of between groups are listed in Table 6. Also as shown in Table 6, statistical analyses showed a significant interaction indicating beneficial contributions of training on cognitive performances.

Although number, gender and education of subjects in groups are not equal, but one-sample Kolmogorov-Smirnov test showed that data distribution was Normal.

As previously mentioned in section (4.7.4), our study didn't intervene the education levels of subjects in statistical analyses unless in TMT.

Table 6: Pairwise Comparisons of Groups

Comparisons of Groups	Dependent Variable	Mean Difference	Std. Error	Significance
Control & Anaerobic	Visual memory, immediate recall	-19.968	2.454	p < .001
Control & Aerobic	Visual memory, immediate recall	-15.673	2.362	p < .001
Control & Anaerobic	Visual memory, delayed recall	-19.219	2.205	p < .001
Control & Aerobic	Visual memory, delayed recall	-16.182	2.151	p < .001
Control & Anaerobic	Verbal short-term memory	-13.164	6.462	p = .134
Control & Aerobic	Verbal short-term memory	-14.113	6.417	p = .092
Control & Anaerobic	VLMT, recall after learning	-22.584	5.036	p < .001
Control & Aerobic	VLMT, recall after learning	-6.860	4.936	p = .505
Aerobic & Anaerobic	VLMT, recall after learning	-15.725	4.895	p = .006
Control & Anaerobic	VLMT, recall after interference	-27.363	4.693	p < .001
Control & Aerobic	VLMT, recall after interference	-8.223	4.597	p = .232
Aerobic & Anaerobic	VLMT, recall after interference	-19.140	4.851	P < .001
Control & Anaerobic	Verbal long-term memory	-22.556	5.565	p < .001
Control & Aerobic	Verbal long-term memory	-15.813	5.503	p = .015
Control & Anaerobic	Mirror reading, Priming performance	110.97	13.43	p < .001
Control & Aerobic	Mirror reading , Priming performance	73.34	13.21	p < .001

Aerobic & Anaerobic	Mirror reading , Priming performance	37.62	12.99	p = .015
Control & Anaerobic	Mirror reading , Procedural memory	131.24	14.40	p < .001
Control & Aerobic	Mirror reading , Procedural memory	111.51	14.15	p < .001
Aerobic & Anaerobic	Mirror reading , Procedural memory	19.73	13.87	p = .477
Control & Anaerobic	Mirror reading , Interference	77.54	11.75	p < .001
Control & Aerobic	Mirror reading , Interference	66.63	11.48	p < .001
Aerobic & Anaerobic	Mirror reading , Interference	10.91	11.37	p = 1.00
Control & Anaerobic	Speed of Info. processing TMT-A	-26.770	5.420	p < .001
Control & Aerobic	Speed of Info. processing TMT-A	-21.897	5.384	p < .001
Aerobic & Anaerobic	Speed of Info. processing TMT-A	-4.874	5.290	p = 1.00
Control & Anaerobic	Executive functions TMT-B	-31.889	5.453	p < .001
Control & Aerobic	Executive functions TMT-B	-27.263	5.413	p < .001
Aerobic & Anaerobic	Executive functions TMT-B	-4.626	5.311	p = 1.00
Control & Anaerobic	Verbal intelligence	-10.073	1.971	p < .001
Control & Aerobic	Verbal intelligence	-6.062	1.941	p < .007
Aerobic & Anaerobic	Verbal intelligence	-4.012	1.901	p = .113
Control & Anaerobic	Non-verbal intelligence	-7.113	1.560	p < .001
Control & Aerobic	Non-verbal intelligence	-5.411	1.548	p < .002
Aerobic & Anaerobic	Non-verbal intelligence	-1.702	1.521	p = .799

5.4 Results of visual memory (ROCF)

Aerobic and anaerobic exercises can improve visual memory in older adults. This study aims to clarify the effects of physical activity on memory performance in older adult participants. Group sizes were for the control, aerobic and anaerobic n = 28, n = 31 and n = 30.

Generally, with the exception of two tests between aerobic and anaerobic conditions, findings did not significantly differ. Yet, when comparing the outcomes of the aerobic and anaerobic groups with the control group's outcome there were some differences. The results of the memory tests of these three groups are listed in Table 6.

The comparing results of the Rey-Osterrieth Complex Figure Test (ROCF) among all groups showed a significant interaction between the anaerobic condition and short-term[29] visual memory (MD = -19.968, $p < .001$) and long-term[30] visual memory (MD = -19.219, $p < .001$). Also, there was a significant difference between the aerobic condition and short-term visual memory (MD = -15.673, $p < .001$) as well as long-term visual memory (MD = -16.182, $p < .001$). As illustrated in Figure 17, we found aerobic and anaerobic exercises to be improving short-term visual memory in older adults.

[29] In this statistical analysis and in the following discussion "short-term memory" shall be synonymous with "immediate recall".

[30] The phrase "long term memory" may be understood synonymously with "delayed recall"

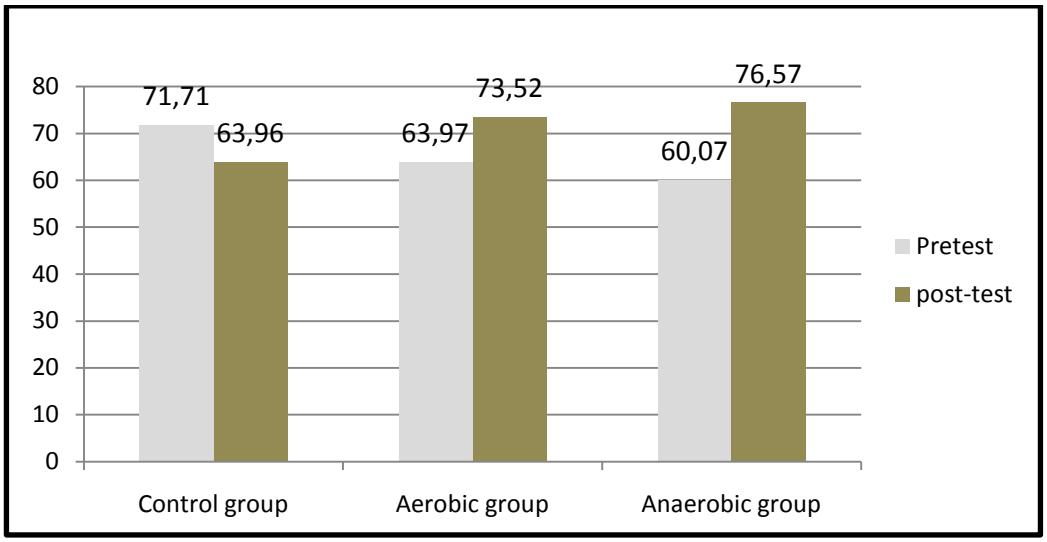

Figure 17: Interventions of physical activity on short-term visual memory. The Figure shows effects of twelve weeks aerobic and anaerobic exercises and also physical inactivity in older adults.

The Figure 18 shows that aerobic and anaerobic conditions enhance long-term visual memory in the elderly, too. As indicated, there is a significant relation between both aerobic and anaerobic conditions with short-term and long-term visual memories in older adult participants.

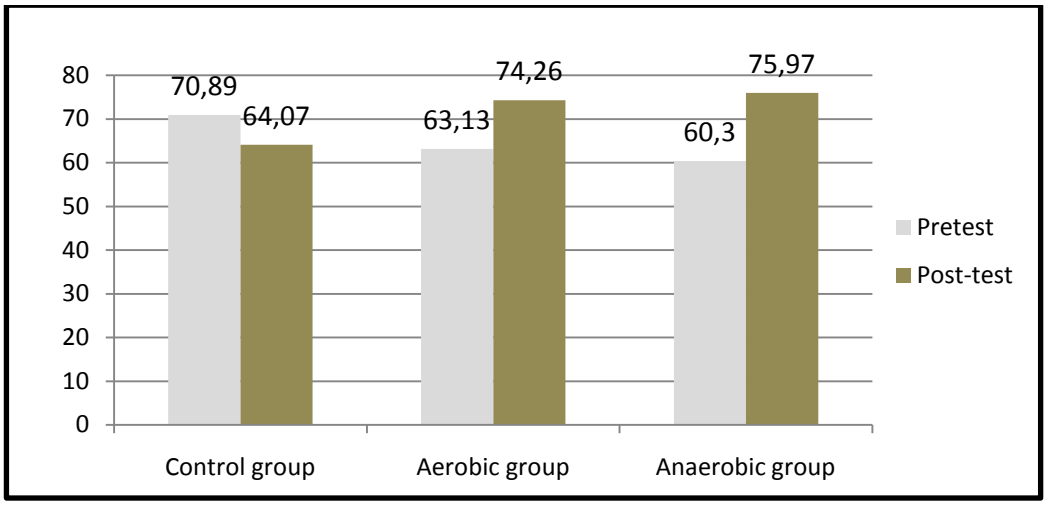

Figure 18: Interventions of physical activity on long-term of visual memory. The Figure shows effects of twelve weeks aerobic and anaerobic exercises and also physical inactivity on older adults. It shows means of long-term memory performance or delayed recall in ROCF.

5.5 Results of Verbal Memory Test (VLMT)

Aerobic and anaerobic exercises are different in their improvement of Verbal Learning and Memory in older adults.

In VLMT, the outcomes of aerobic and anaerobic conditions for immediate recall, recall after learning in the 5th trial, recall after interference and delayed recall differed a lot. As shown in Table 6 there were no significant differences between all three groups in short-term memory (immediate recall). Apart from some increasing appearances in aerobic and anaerobic interaction in short-term memory (see Fig. 19) we didn't find a significant increasing impact of exercise conditions on immediate recall for verbal learning memory.

In all events, the influence of exercise conditions lead to better results compared to the control group, even though, it was non-significant.

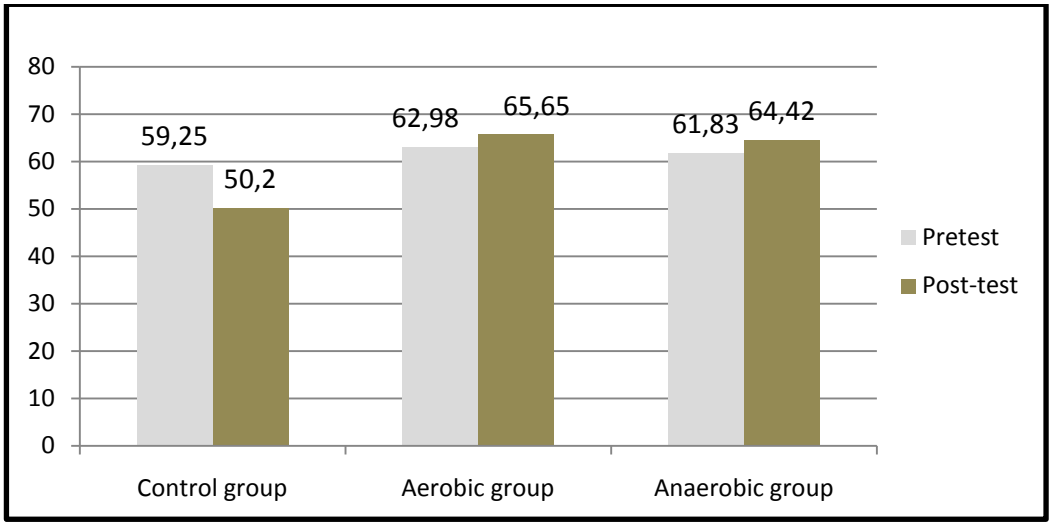

Figure 19: Interventions of physical activity on short-term verbal memory. The Figure shows effects of twelve weeks aerobic and anaerobic exercises and also physical inactivity in 89 adults. It demonstrates means of short-term memory performance or immediate recall in VLMT.

As illustrated in Figure 20, interventions of anaerobic effects indicate a significant difference at recall after learning (5th trial in VLMT), with both the aerobic (MD = -15.725, p = .006) and the control group (MD = -22.584, p < .001), whereas there was no significant difference between these two groups, and the intervention of aerobic exercise conditions created positive effects for recall after learning in the 5th VLMT trial, again with no significant value (MD = 6.860, p = .51).

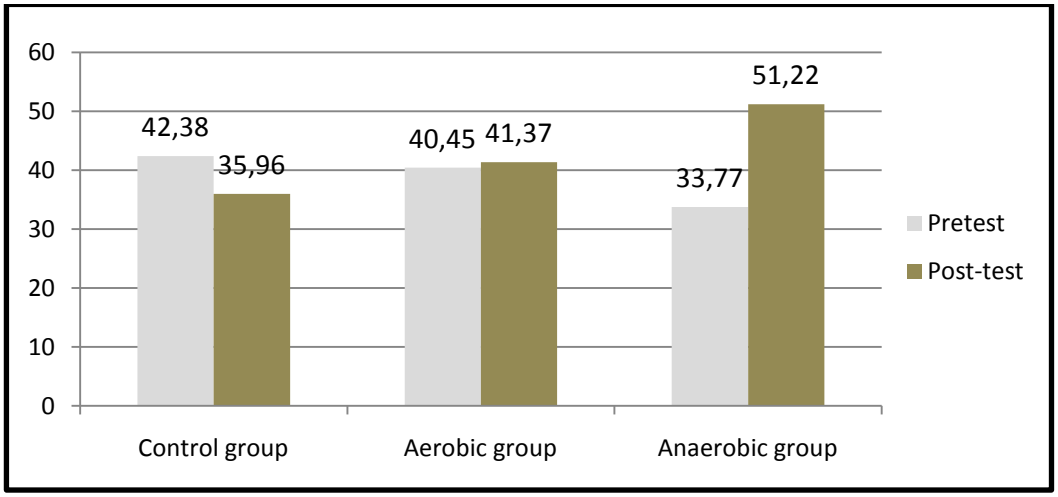

Figure 20: Interventions of physical activity on recall after learning in VLMT. The Figure shows effects of twelve weeks aerobic and anaerobic exercises and also physical inactivity in 89 adults.

In Table 6 and Figure 21 , we can see that the intervening anaerobic condition can produce a significant difference at recall after interference in the VLMT with both the aerobic (MD = -19.14, p < .001), and the control group (MD = -27.363, p < .001), and this more than in the aerobic and control group; the aerobic condition has not only positive effects, but also there is a negative effect of a decrease in outcomes in this phase (Pretest M = 45.89, Post-test M = 41.81, MD = 8.223, p = .23). This is unexpected.

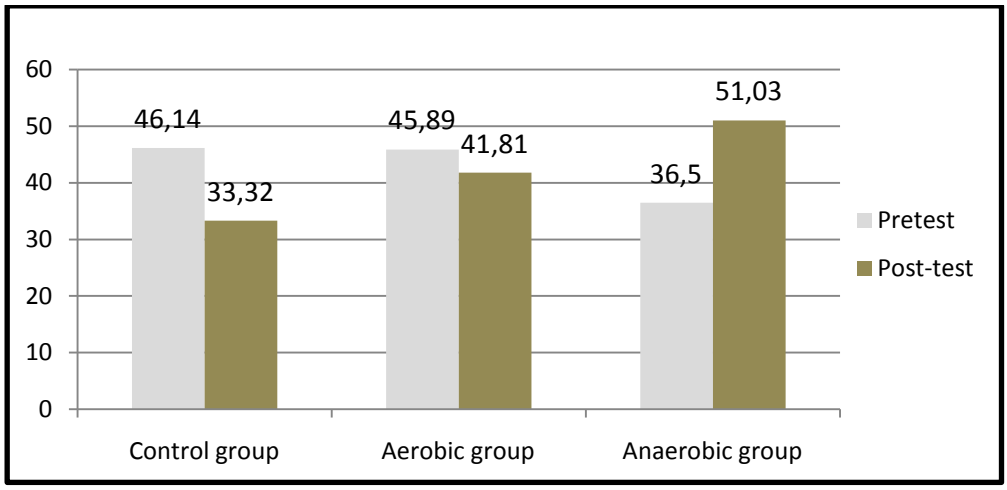

Figure 21: Interventions of physical activity on recall after interference in VLMT.
The Figure shows effects of twelve weeks aerobic and anaerobic exercises and also physical inactivity on adults' participants.

In Table 6 and Figure 22, the compared results of long-term verbal memory at delayed recall in VLMT are significant for the anaerobic group when contrasted to the control group (MD = 22.556, $p < .001$). Also, there are significant results for the aerobic group when contrasted to the control group (MD = 15.813, $p < .015$).

In short, the aerobic and anaerobic condition groups yielded significant differences for the short-term and long-term memory in the ROCF.

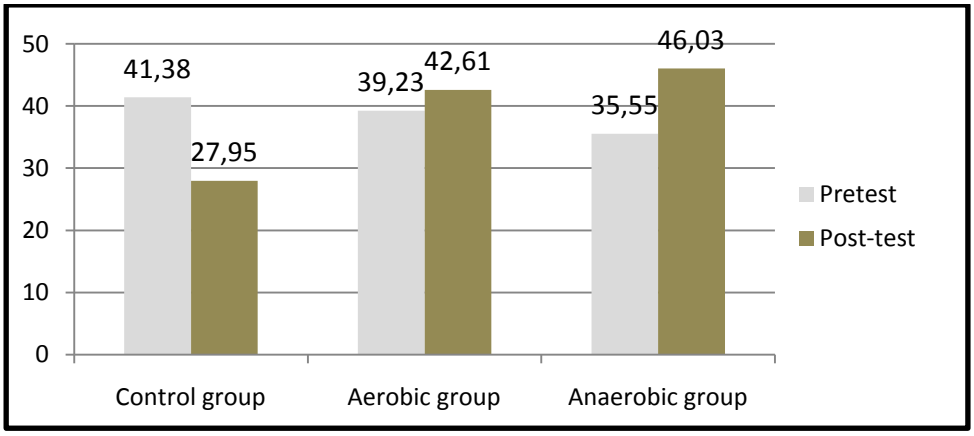

Figure 22: Interventions of physical activity on delayed recall in VLMT.
It shows effects of twelve weeks aerobic and anaerobic exercises and also physical inactivity on older adults.

The VLMT for the anaerobic condition was able to reveal significant differences in recall after learning within the 5th trial, recall after interference and delayed recall for our participants, yet, the aerobic condition could only yield significant differences at delayed recall. In terms of the immediate recall in the VLMT, neither the aerobic nor the anaerobic condition caused significant differences in subjects. Overall, the control group's results were always inferior to the results of the experimental groups.

5.6 Results of mirror reading task

As illustrated in Figure 23, interventions of anaerobic effects indicate a significant difference at priming performance (priming, mirror reading), with both the control group (MD = 110.97, $p < .001$) and the aerobic (MD = 37.62, $p = .015$). Also, we can see that the intervening aerobic condition can produce a significant difference at priming, mirror reading with the control group (MD = 73.34, $p < .001$).

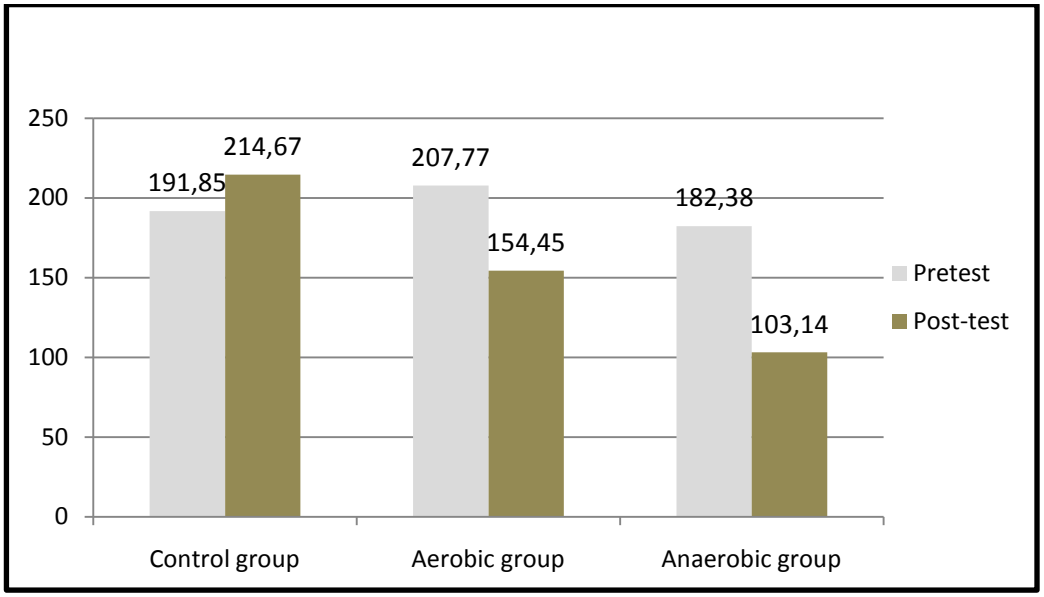

Figure 23: Interventions of physical activity on priming of mirror task.
The Figure shows effects of twelve weeks aerobic and anaerobic exercises and also physical inactivity in older adults.

In Table 6 and Figure 24, we can see that the both EG intervening condition can produce a significant difference for the ANEG (MD = 131.24, p < .001), and the AEG (MD = 111.51, p < .001) on procedural memory performance of mirror reading test in older adult participants, whereas there being no significant difference between these two EG (MD = 19.73, p = .477).

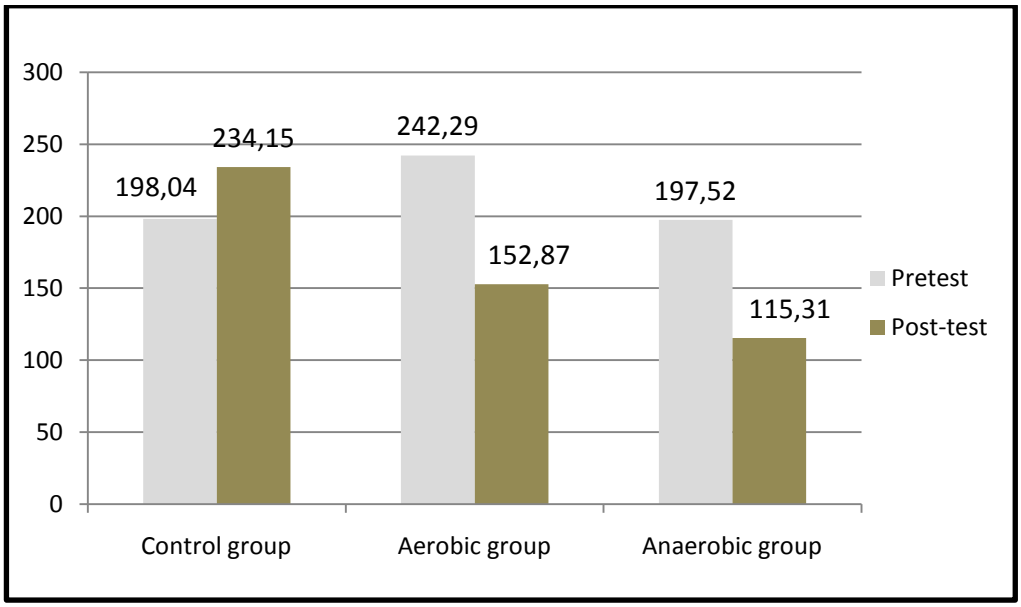

Figure 24: Interventions of physical activity on procedural memory of mirror task.
The Figure shows effects of twelve weeks aerobic and anaerobic exercises and also physical inactivity in 87 adults.

As shown, in Figure 25, we can see that the both EG intervening condition can produce a significant difference for the ANEG (MD = 77.54, p < .001), and the AEG (MD = 66.63, p < .001) on Interference mirror reading in older adult participants, whereas there was no significant difference between these two EG (MD = 10.91, p = 1.00).

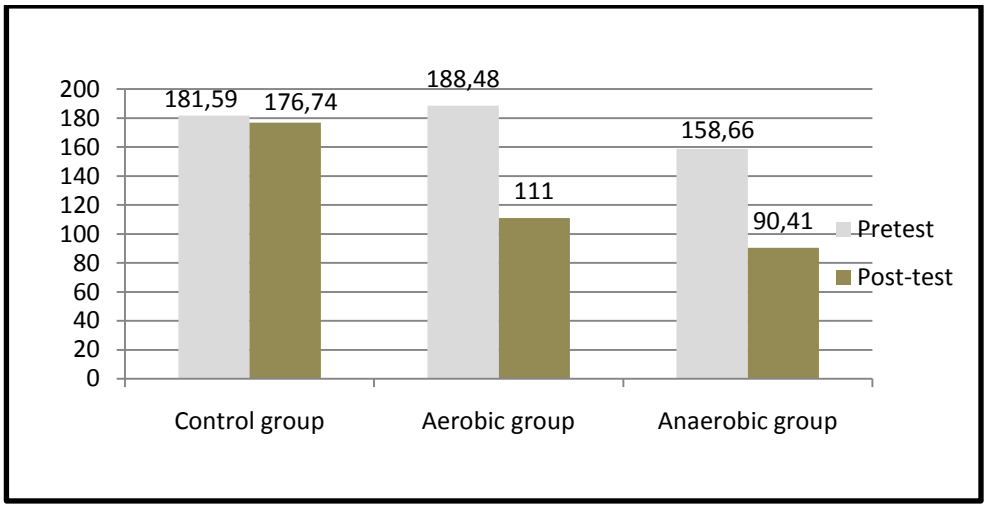

Figure 25: Interventions of physical activity on interference of mirror reading task.
The Figure shows effects of twelve weeks aerobic and anaerobic exercises and also physical inactivity in 89 adults.

5.7 Results of visual search, scanning and speed of information processing

As illustrated in Figure 26, we found aerobic and anaerobic exercises to be improving on speed of information processing (TMT-A) in older adults. As shown in Table 6 and Figure 26, interventions of both exercises effects the ANEG (MD = -26.770, p = .001) and the AEG (MD = -21.897, p < .001) indicate a significant difference on speed of information processing, whereas there being no significant difference between these two EG (MD = -4.874, p = 1.00).

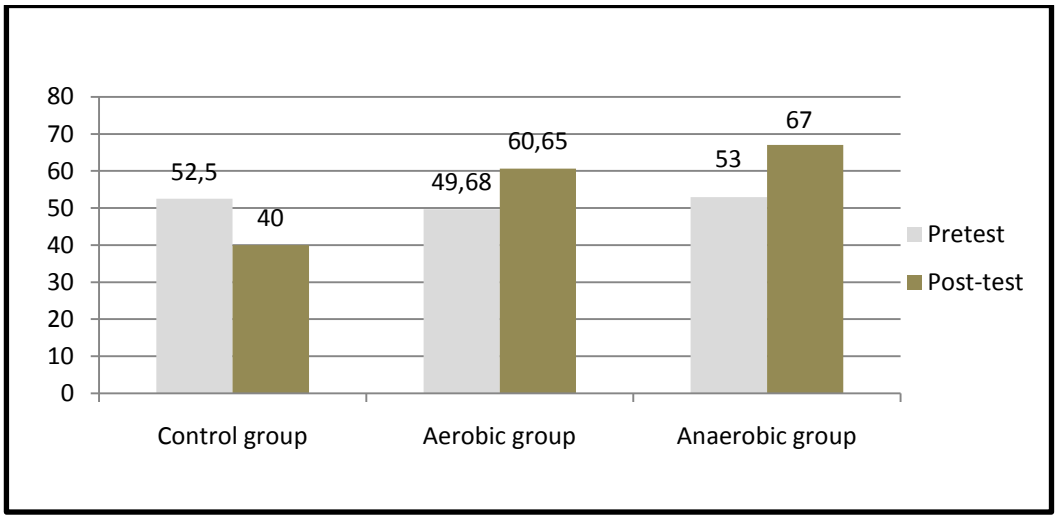

Figure 26: Interventions of physical activity on attention and speed of information processing. The Figure shows effects of twelve weeks aerobic and anaerobic exercises and also physical inactivity in older adults.

5.8 Results of visual search, scanning, mental flexibility and executive functions

As illustrated in Figure 27, interventions of both exercises effects the ANEG (MD = -31.889, p = .001) and the AEG (MD = -27.263, p < .001) indicate a significant difference on mental flexibility (TMT-B), whereas there being no significant difference between these two EG (MD = -4.626, p = 1.00).

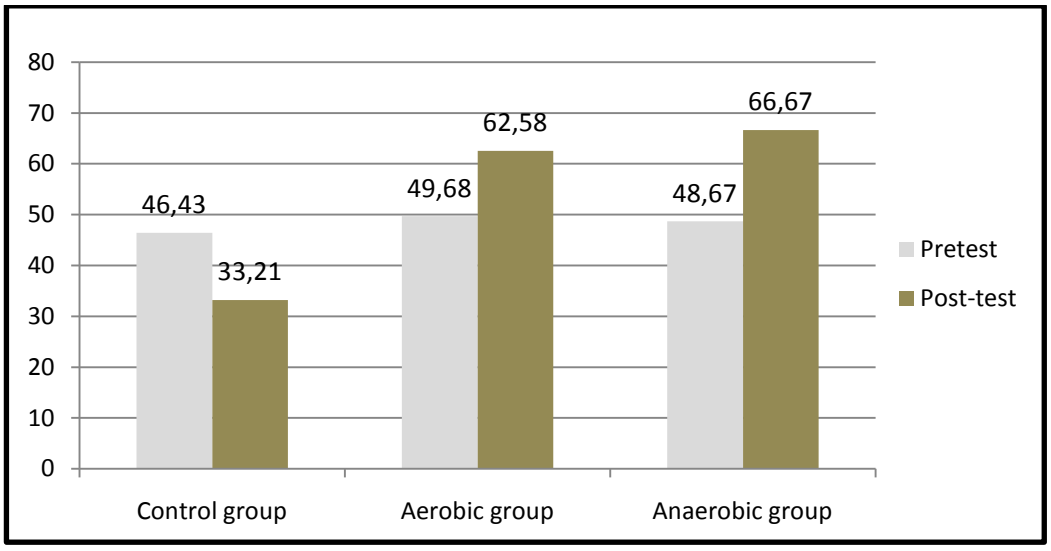

Figure 27: Interventions of physical activity on mental flexibility.
The Figure shows effects of twelve weeks aerobic and anaerobic exercises and also physical inactivity in 89 adults.

5.9 Results of verbal intelligence

As illustrated in Figure 28 and Table 6, interventions of both exercises effects the ANEG (MD = -10.073, p = .001) and the AEG (MD = -6.063, p < .007) indicate a significant difference on verbal intelligence, whereas there being no significant difference between these two EG (MD = -4.012, p = .113).

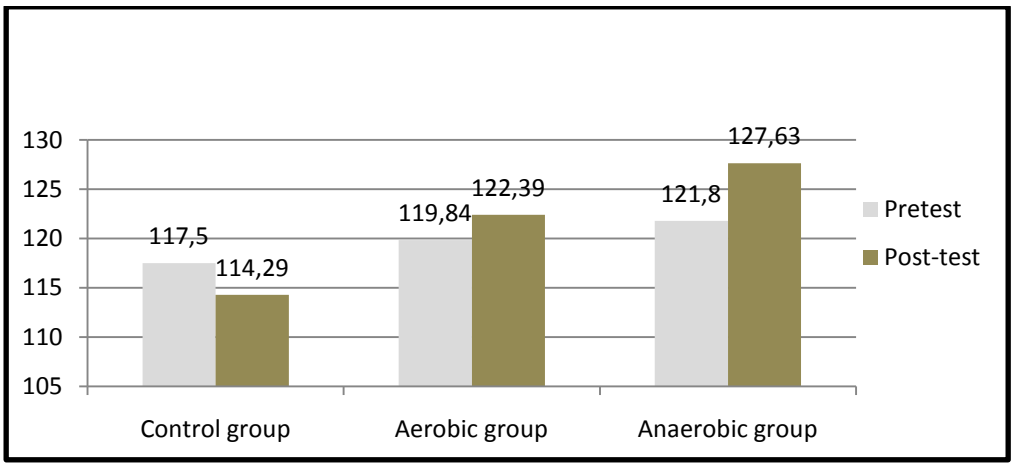

Figure 28: Interventions of physical activity on verbal intelligence.
The Figure shows effects of twelve weeks aerobic and anaerobic exercises and also physical inactivity in 89 adults.

5.10 Results of non-verbal intelligence

As illustrated in Figure 29, we found aerobic and anaerobic exercises to be improving on non-verbal intelligence in older adults. As shown in Table 6 and Figure 28, interventions of both exercises effects the ANEG (MD = -7.113, p = .001) and the AEG (MD = -5.411, p < .002) indicate a significant difference on non-verbal intelligence (LPS-4), whereas there being no significant difference between these two EG (MD = -1.702, p = .799).

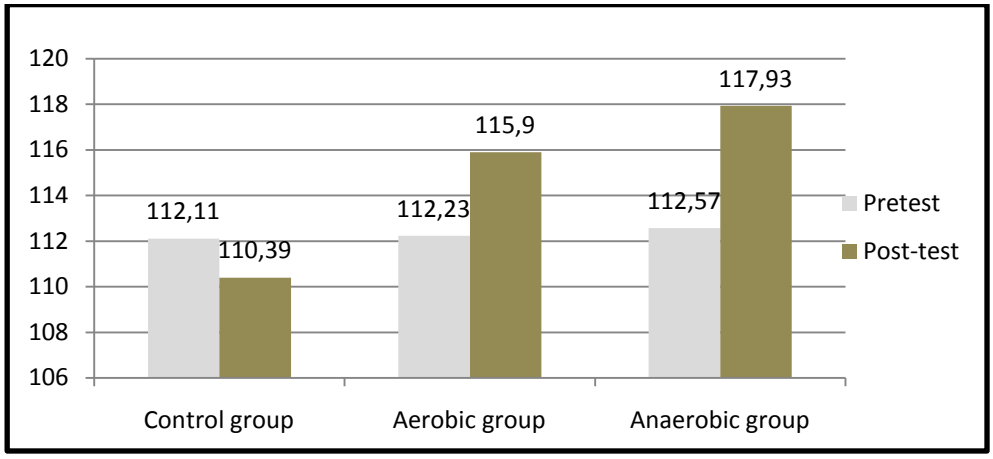

Figure 29: Interventions of physical activity on non-verbal intelligence.
The Figure shows effects of twelve weeks aerobic and anaerobic exercises and also physical inactivity in 89 adults.

6 Discussion

In this chapter, the results of present study are discussed in the context of previous research carried out on aging, exercise and cognitive functions.

There is a consensus about the occurrence of a decline in cognitive functions during the aging process (Altman & Das, 1965; Burke & Barnes, 2006; Cameron & McKay, 1999; K. I. Erickson et al., 2009; Fabel & Kempermann, 2008; Kramer et al., 1999; Mather & Carstensen, 2005; Small, 2001). Animal and human literature have suggested an affirmative effect of exercise on cognitive function and memory performance (Blumenthal & Madden, 1988; K. I. Erickson et al., 2011; Larson et al., 2006; Neeper et al., 1996; Pate et al., 1995; Van Praag, Christie, et al., 1999) .

In sum, our findings demonstrated:

1. Aerobic and anaerobic exercises can create a significant change in older adults' cognitive functions.

2. Although intervention of both aerobic and anaerobic condition could improve cognitive abilities of older adults, but it appears that these condition are not able to improve at a similar rate.

3. Anywhere the aerobic exercise could create a noteworthy change, the anaerobic exercise could also too, but there are some abilities that the anaerobic condition could significantly improve in older adults' cognitive functions that the aerobic condition could not. The first question raised in this study asked whether there is any relationship between various kinds of physical activity and older people's cognitive functions; and afterwards, in regard to different effects of various physical exercises, the aerobic and non-aerobic protocols are arranged. For each of current study parameters like the aerobic, anaerobic as well as developing a new procedural skill which is as a condition that have been intervened in the study, we have made separate discussion, and ours results with previous works are compared and interpreted together.

6.1 The aerobic exercise condition and cognitive functions of sedentary older adults

The physical activity leads in energy expenditure. In fact, the energy expenditure is needy cardiorespiratory activity such as physical fitness, and it also improves through the physical exercise. Up to now, many results of studies have indicated that exercise-induced physiological increases in aerobic exercises have beneficial effects on physical, physiological and psychological outcomes of human population. Indeed, the aerobic training creates an improvement in parts of physical fitness that relates to the ability of circulation and respiration to supply oxygen during sustained physical activity.

The beneficial effects of aerobic exercise may not be merely limited to physiological advantages such as increasing oxygen consumption, blood circulation, the number of capillaries, and vascular function. Bearing in mind that the human brain with about two percent of weight uses almost 20 percent of the oxygen consumed by the body, and also despite the low-oxygen level inside brain tissue, the brain is one of the neediest oxygen consumers in the body.

In addition of oxygen roles on the brain development, it also has been shown in the pathological processes and the brain dysfunctions (Zhang et al., 2011). Accordingly, the increasing of oxygen transportation may be an important factor in improving neuronal growth, cell proliferation, cell survival, and creating more stimulation of neurogenesis. All these consequences provide an ideal condition for improving mental performance and increasing resistance to brain disorder in adults' brain. However, it is expected that the participation in regular aerobic activities should help to preserve the brain cells and their functions through the improvement of blood circulation and cardiorespiratory fitness. Thus, this is surely established that participants in aerobic activity programs receive suitable levels of physical and psychological health.

Many studies on animals (e.g. Churchill et al., 2002; Neeper, Gómez-Pinilla, Choi, & Cotman, 1996; Van Praag et al., 1999, 2005) have shown that aerobic exercises like walking could create a considerable increase in cell survival, neurogenesis and improve synaptic plasticity. Also, human cognitive studies indicate the influence of exercise on some brain regions (Stanley J Colcombe et al., 2006; Cotman & Berchtold, 2002; Hillman et al., 2008; Kramer et al., 1999; Weuve et al., 2004).

Currently there are several theories that can explain how exercise may affect cognitive function. The first, physical activity and especially the aerobic activities improve aerobic capacity. During physical exercise regional cerebral blood flow, in major cerebral arteries and also blood flow in the internal carotid artery enhance, causing an increase in blood flow to a wide regions of the brain (Ide & Secher, 2000). Aerobic exercise increases oxygen saturation (Kramer et al., 1999) angiogenesis and improves vascular function (Fordyce & Farrar, 1991; Kleim et al., 2002) in brain areas crucial for task cognitive performance. These changes are generally associated with aerobic exercise and endurance exercise. Additionally, during exercise the regional cerebral uptake of O_2 increase (Ide & Secher, 2000).

There is confirmation that the transmission of foodstuffs accompanied by the blood during exercise can improve neurogenesis and affects as a useful tool on the cognitive decline prevention in adults' population (Cutuli et al., 2014). Thus, it could be expected that aerobic exercise increases the rate of oxygen consumption in healthy older adults and enhances performance of cognition. Additionally, aerobic exercise can increase level of BDNF, an effective element for supporting the survival of existing neurons in some areas of the brain including hippocampus, medial temporal lobe, amygdala, frontal, prefrontal and parietal cortices. These are vital areas related to learning process, cognitive function, memory performance, and notably the long-term memory.

Results of animal and human research have frequently revealed that aerobic exercise can improve cognitive function and brain performance through neurogenesis (Cotman & Berchtold, 2002; Kramer et al., 1999; van Praag, 2009; Weuve et al., 2004), angiogenesis (Fordyce & Farrar, 1991; Swain et al., 2003), synaptic plasticity (van Praag, Kempermann, et al., 1999; Van Praag et al., 2005), through changes in gene expression and through the increasing if basal BDNF concentrations (Cotman & Engesser-Cesar, 2002; Intlekofer & Cotman, 2013) and other several physiological mechanisms (Dustman et al., 1984; Laurin et al., 2001; R. L. Rogers et al., 1990).

The second theory suggests that physical exercise can directly enhance synaptic plasticity through the change of synaptic structure and strength, thus, supports neurogenesis (Cotman, Berchtold, & Christie, 2007). One of the key mechanisms of exercise and physical activity on the brain is induction of growth factors, which plays an important role in structural and functional changes as well as neuroprotective effects in aging. The aerobic exercise upregulates neurotrophins like BDNF and IGF-1 that support neuronal survival and differentiation in the developing brain and dendritic branching in the adult brain (Cotman et al., 2007; Cotman & Engesser-Cesar, 2002; McAuley et al., 2004). Thus, physical aerobic activity may also influence brain regions engaged with the cognitive abilities and memory system.

As previously mentioned in section (4.5) one of the independent variables was a protocol of an aerobic prolonged and sub-maximal activity like walking, brisk walking, Nordic walking, jogging and running. It was conducted three times a week for twelve weeks. The duration of exercises remained constant, fixed around 45 minutes.

However, our findings support that the participation in regular aerobic activities can help to preserve cognitive activity and to decrease the risk of dementia, and that such exercise should be able to maintain memory performance. The results of our study confirmed that aerobic condition was able

to significantly improve older adults' visual memory in both short (MD = -15.673, $p < .001$) and long-term (MD = -16.182, $p < .001$).

As seen in Table 6, there were no significant differences between all three groups in short-term verbal memory (immediate recall) in VLMT (Fig. 19).
These results of both exercise groups have the weakest impact on cognitive function, which are documented in the study. Indeed, neither of the both exercise conditions can create any significant difference on the immediate recall in VLMT. In this task, participant was provided with the number of components of verbal working memory including recognition and recall of a list – which were only heard once – of ordered stimulus. This task is very near to sensory store. Sensory memory is an ultra-short-term memory and decays quickly.

The findings of the visual memory component revealed that both exercises condition were able to significantly improve older adults' short-term. Probably, the generalizations of results' immediate recall in VLMT to short-term memory cannot indicate the nature of task in meaning short-term. Thus, in the review of sensory store and short-term memory, it appears that measuring of the immediate recall in VLMT cannot assess short-term memory.

Thus, it should be concluded that the both exercises condition didn't create significant changes on ultra-short-term memory and sensory store. This is not unexpected, because, as it has been reported, the sensory memory cannot be extended by rehearsal (Cowan, 1997).
In fact, both exercises condition did not create significant changes between EG and CG. It appears that perhaps the verbal short-term memory depends less on physical exercise. Because, no major divergence is witnessed in both exercise groups; although, it is also possible that the used light intensity that we had accomplished cannot create important differences.

As illustrated in Table 6, and Figure 20, intervention of aerobic effects didn't indicate an important difference (MD = -6.860, p = .505) at recall after learning (5th trial in VLMT). Also, taking into the Table 6 and Figure 21, we can see that the intervening aerobic condition cannot produce a noteworthy difference at recall after interference in the VLMT with the control group (MD = -8.223, p = .232).

However, this failure does not appear to be the reason of this disability and can perhaps be intervention of the low intensity of our aerobic condition. As we know, there is confirmation that has shown the beneficial influence of physical activity produced through aerobic exercise on cognitive performances (Dustman et al., 1984; Hillman et al., 2008; Kramer et al., 2006).

Aside from a few results of aerobic exercise condition on memory function – that sometimes were not affective –, the intervention of aerobic condition produces a significant difference on other cognitive function aspects, which are examined in the study. These remarkable differences were exactly similar (p < .001) for the mirror reading tasks encompasses priming performance, procedural memory and interference as well as for the Trail Making Test (TMT A&B) includes measurements of visual search, scanning, speed of processing, mental flexibility, and executive functions.

While, these significantly changes were able to be observe in (MWT-B test) verbal intelligence (MD = -6.063, p < .007) and in (LPS-4 test) non-verbal intelligence (MD = -5.411, p < .002).

Though the results are promising, findings of our aerobic group suggest that aerobic exercise is not always as effective as its anaerobic counterpart in influencing cognitive abilities and particularly in memory system.

Our results of aerobic condition confirm the level of oxygen consumption (Ramel et al., 2004), roles of mitochondria (Blalock et al., 2004; Bratic & Trifunovic, 2010; Dringen et al., 2000; Loerch et al., 2008; Lu et al., 2004;

Miquel et al., 1980; Pak et al., 2003) and the formation of ROS (Fisher-Wellman & Bloomer, 2009; Radak, Chung, & Goto, 2008); exercise may actually make a difference in the aging process using the aerobic condition (Bishop et al., 2010; Rea et al., 2007; Sánchez-Blanco et al., 2006). It appears that the optimal level of aerobic exercise concerning both intensity (Goto et al., 2003) and duration (Bloomer et al., 2007), as well as the optimal level of ROS generation may be of importance in brain function (Knez et al., 2006; Radak, Chung, & Goto, 2008).

If the modification of the oxygen consumption by physical activity induces the neurogenesis in the brain, a novel idea provides to preserve the ability of learning processes and memory function because of the involvement of neurogenesis in the process of cognitive function. Thus, this study proposes the future investigations study effects of aerobic exercises on older adults' cognitive performance with exact measurement of aerobic condition according to $VO_{2\ max}$ levels of physical aerobic program.

6.2 The anaerobic exercise condition and cognitive functions of sedentary older adults

Now, we know that inactivity lifestyle is one of the important risk factors for many age-related diseases including physical, physiological and psychological diseases. Since, neuron is able to survive and serve for a century or more in many persons that age successfully (Mattson, 2000). Understanding of the mechanisms that permit such cell survival and synaptic plasticity may therefore lead to the development of new preventative and therapeutic strategies for age-related neurodegenerative dysfunctions.

Though, researchers who have examined the effects of anaerobic exercise on cognitive processes have consistently failed to detect a clear relation between exhaustive exercise and cognitive abilities (Stanley J Colcombe et al., 2006; Knaepen et al., 2010; Tomporowski, 2003).

Contrary to those failed studies of anaerobic exercise on cognition, perhaps the most important consequence of our analysis is the implication that robust and specific benefits do occur with anaerobic exercise. Results of our study can confirm findings of Peig-Chiello, Perrig et al., 1998; Colcombe and Kramer 2003; Heyn, Abreu et al., 2004; Cassilhas, Viana et al., 2007; Erickson, Prakash et al., 2009; Chang and Etnier 2009.

As previously mentioned in sections (2.2) and (2.3) anaerobic energy system is the transformation of glucose to lactic acid, when enough amounts of oxygen are not available. The anaerobic system produces the energy nearly fast, but very little. It should be described that when it is said the term anaerobic exercise, it is not right, if we think this exercise induces the increasing of lactic acid in bodies' participants, because in the study, the intensity of anaerobic protocol is farther down than anaerobic threshold levels and consequently it

doesn’t induce the increasing of lactic acid. Thus, it will be used in the same way as “non-aerobic”.

Indeed, our anaerobic protocol content includes stretching, toning, coordination, equilibrium and especially the Stroop training. Through the new motor learning, which is required for the synaptic changes and creation of new proteins within the brain cells and neurons, the facilitating and acceleration of electrochemical transfers and neurotransmitters in synaptic junction contributes to reinforcing the communicative capacity of certain circuits of neurons in the brain and can lead to improvement of the cognitive functions in older adults.

It is now clear that voluntary exercise is an important factor to improve neuronal growth, creates more stimulate neurogenesis, improves mental performance and increases resistance to brain disorder in the adult brain (Caspersen et al., 1985; Cotman & Berchtold, 2002; Dishman et al., 1985; K. I. Erickson et al., 2011; Knaepen et al., 2010; Mattson et al., 2004; O'Sullivan et al., 1997; PILC, 2010; Rhyu et al., 2010; van Praag, 2009; Van Praag, Christie, et al., 1999; van Praag, Kempermann, et al., 1999; Van Praag et al., 2005; Webster et al., 2002). Anaerobic group participants have performed exercises such as new motor learning, Stroop, neuromuscular, cross-brain and body equilibrium exercises; this caused an increase in brain activity and a strengthening of underlying systems that support brain plasticity – including metabolism and vascular function, cell proliferation, cell survival, neurogenesis – which may have caused in turn increased cognitive function and memory performance compared to the aerobic group which have repeated only the same aerobic activity.

This investigation suggests that engaging in systematic and regular physical activity, among other health benefits, can delay or prevent the beginning of cognitive impairment and dementia in the older adults. Although, our results is needy confirmation in further intervention studies, these results suggests that

regular practice of physical activity could represent an important and potent protective factor for cognitive dysfunction and dementia in the adult people.
Like aerobic condition, the results of the investigation show that the effects of anaerobic protocol on short-term (MD = -19.968, p < .001) (Fig. 17) and long-term of visual memory (MD = -19.219, p < .001) in elderly are significant (Fig. 18).

As noted in section (6.1) there were no significant differences (it is concluded in section 6.1, to another concept) between all three groups in immediate recall of the VLMT. Unlike the aerobic group, as seen in Table 6 and Figure 20, interventions of anaerobic effects indicate a significant difference at recall after learning (5th trial in VLMT), with the control group (MD = -22.584, p < .001), interestingly, in this parameter, we can see a significant difference between ANEG and AEG too (MD = -15.725, p = .006).
This implies that engaging in the low intensity anaerobic condition even without the need of running can create robust beneficial effects in seniors' cognition, in contrast regular running and other systematic aerobic program.

In VLMT after interference list, in trial 7 when no list is read and subjects are asked to recall as many words as possible from the A list (see Table 6 and Fig. 21) we can see that the intervening anaerobic condition can produce a significant difference at recall after interference in the VLMT with both the aerobic (MD = -19.14, p < .001), and the control group (MD = -27.363, p < .001), and this more than in the aerobic and control group; the aerobic condition has not only positive effects, but also the negative effect of a decrease in outcomes of this phase (Pre-test M = 45.89, Post-test M = 41.81, MD = 8.223, p = .23). This really is unexpected that in comparison to low intensity exercises of both aerobic and anaerobic and regarding our results, it seems that mild anaerobic intervention can create better improvements in cognitive functions and memory performance of elderly than aerobic.

In Table 6, and Figure 22, the compared results of long-term verbal memory at delayed recall in VLMT are significant for the anaerobic group when contrasted to the control group (MD = 22.556, $p < .001$). Also, there are significant results for the aerobic group when contrasted to the control group (MD = 15.813, $p < .015$).

In comparison to the time and duration of memory function, it appears that low and light types of the aerobic and anaerobic intervention can create almost similar improvements in memory performance of elderly.

The intervention of anaerobic condition produces a significant difference on other cognitive function aspects, which are examined in the study. These remarkable differences were exactly similar ($p < .001$) for the mirror reading tasks encompasses priming performance, procedural memory and interference as well as for the Trail Making Test (TMT A&B) including measurements of attention, visual search, scanning, speed of processing, mental flexibility, and executive functions, verbal intelligence (MWT-B) and in non-verbal intelligence (LPS-4).

Our findings diverge from the data of previous studies, some of which have failed to observe the benefits of anaerobic exercise on cognitive processes (Stanley J Colcombe et al., 2006; Knaepen et al., 2010). Also, findings of the current work suggest that the mild to moderate types of anaerobic exercise are more capable in improving these brain regions effectively than aerobic programs. All this points to the decline of memory function in late adulthood are not inevitable and it can be reversed with light intensity type of our anaerobic protocol. It seems that for supporting cognitive function in older adults that have difficulty with performing aerobic exercise such as walking and running, the light intensity of non-aerobic programs may maintain older adults' cognitive abilities.

6.3 The new procedural skill learning and cognitive functions of sedentary older adults

The beneficial effects of exercise and physical activity may not be merely limited to physiological advantages of aerobic and anaerobic activities such as increasing aerobic and anaerobic capacities, oxygen consumption, blood circulation, development of capillaries network, strength, power, stretching, toning and muscle activity.

Though, the increasing of these factors may be an important factor in general human health as well as improving neuronal growth, cell proliferation, cell survival, and creating more stimulation of neurogenesis, but to obtain an ideal condition for improving mental performance in aging process, it is not confined only by these factors.

Regarding the problems and disabilities of the elderly such as walking and running, which are related to cardiovascular and skeletal muscle disorders due to aging; it seems that for supporting cognitive function in older adults that have difficulty with performing moderate aerobic exercise, an exercise planning based on new motor skill learning can be a useful and preventive strategy for helping mental performance, brain cells activity and neuroprotective function.

Our results demonstrate that the loss of cognitive function and memory performance in late adulthood is not inevitable. Training protocols such as our non-aerobic program could serve as prevention of on older adults' cognitive deterioration and memory decline. In addition, the creation of more activity in brain regions related to procedural memory could support brain cells better and keep the cognitive function as well as memory performance at higher level, by comparison with increased oxygen consumption, blood circulation, and angiogenesis brought by repetitious aerobic exercises without new motor skill learning.

Since moderate to high intensity exercises cannot be endured by some of the sedentary elderly, and also regarding the important role of the exercise intensity in the creation of physiological changes in human, our results suggest that the low intensity non-aerobic exercise – without the need of running – e.g. stretching, toning, coordination and the Stroop training (see section 4.6.3) could support the cognition and memory performance in aged participants better than low intensity aerobic exercise. In other words, these non-aerobic low intensity exercises may create more synaptic changes, new proteins in brain cells, and facilitate electrochemical transfer of neurotransmitters in synaptic junction through the new motor skill learning and intellectual action.

Stimulation of the expression of neurotrophic factors such as the Brain Derived Neurotrophic Factor (BDNF), Nerve growth factor (NGF) as well as messenger ribonucleic acid (mRNA) is one of the basic mechanisms of beneficial effects of physical exercise (Gómez-Pinilla et al., 1997; Neeper et al., 1996).

The increasing of neurotrophic factors induced by physical activity may protect neurogenesis through several biochemical processes (Cotman & Berchtold, 2002; Mattson, 2000; Van Praag, Christie, et al., 1999). Despite the important role of BDNF protein expression in age-related hippocampal atrophy and despite effects of exercise on hippocampal volume and function, it is suggested that physical activity could be advantageous for brain volume and cognitive performance in older adults (K. I. Erickson et al., 2012).

It is repeatedly shown that exercise increases hippocampal neurogenesis (Cotman & Berchtold, 2002; van Praag, Kempermann, et al., 1999; Van Praag et al., 2005) and it may improves learning and cognitive performances of older adults. Inasmuch that has been suggested the motor learning may increase the brain’s plasticity and the capacity for self-repair (Mattson, 2000), our converging results confirm that new motor skill learning can reverse the cognitive performance decline in adults population. Interestingly, like the exercise

intervention, motor skill learning condition associated with neurogenesis, may increase numbers of newly generated neural cells in the adults' brain (Mattson, 2000; Mattson et al., 2004). Hence, it appears, in addition to physical exercise activity; new motor skill learning may ameliorate some of the deleterious morphological consequences of aging. These results have been considerably practical in current study.

It also has been shown that a motor learning condition considerably increased the volume of the molecular layer per Purkinje neuron and increased blood vessel number (Isaacs et al., 1992). Thus, the result of current study supports previous investigations that have suggested that physical exercise and intellectual activity can similarly increase neurotrophic factor production and neurogenesis.
Consequently, physical and intellectual activities in turn improve seniors' cognitive performances.

Since, components of the brain, which engage in multiple memory systems can interact together and help each other; there was a hope that procedural learning contributes to cognition abilities of sedentary seniors in non-pathological and normal aging process. Indeed, the brain regions such as the ganglia basal and cerebellum as well as areas of MTL that engage in non-declarative memories are activated via the motor skill learning of our anaerobic protocol. We observed that motor skills learning can reverse the cognitive function decline of older adults' population.

Our results propose that the elderly can use the similar anaerobic protocol (see section 4.6.3) as an effective strategy to delay brain atrophy, reverse the brain decay, and maintain the neuroprotective function and cognitive abilities. As a result, it slows down the cognitive function decline and the onset of dementia.

Generally, our results confirmed that like physical training programs, participation in intellectual or mental activities has also been shown to predict reduced cognitive decline. Thus, there is a hope that participation in mental activities through new motor skill learning is able to compensate the inactivity of lifestyle or disabilities of the elderly to accomplish activities such as walking and running.

The anaerobic protocol of the study is encompassed various training, which anyone can create various impact. Hence, to find exact effects of these various exercises on older adults' cognition, the components of this protocol should be examined separately. Now, we can only suggest that this combination of non-aerobic protocol reverse the cognitive function decline of adults' population.

7 Conclusion

According to literature of the study the successful aging is defined multidimensional (Rowe & Kahn, 1997), hence, involves success in different aspects of life. Also, exercise is a proven method to improve general health. In this prospective and medium case study has been investigated impact of various physical exercises on cognitive functions of healthy and sedentary older adults.

A short-term (three months) of regular physical activity intervention includes aerobic and anaerobic programs that were conducted three times a week for three months each session 45±5 minutes.

The first condition was a program encompasses an aerobic activity like walking, jogging and running. Walking the equivalent of at least 40 min. and three times per week at a 9 to 13 min/km pace was often associated with better cognitive performance than control group. Other condition organized program of stretching, toning, coordination, balance, Stroop exercise, and equilibrium, muscle activity, calisthenics, and circuit training. This non-aerobic protocol was associated with higher levels of cognitive function and less cognitive decline.

Second, our findings could reflect “speciality of exercises” such that various exercise induce different impact on cognition. Though, any kind of physical exercise is preferred to inactivity for having better levels of cognitive function and less cognition decline

The beneficial effects of exercise is related to physiological advantages of aerobic exercise such as increasing oxygen consumption, blood circulation, the number of capillaries, and vascular function; such that the aerobic activities creates an improvement in aerobic capacity. Aerobic exercise increases oxygen saturation and improves vascular function in brain areas. Hence, during aerobic

exercises regional cerebral blood flow improved, causing an increase in blood flow to wide regions of the brain.

Consequently, the transmission of foodstuffs accompanied by the blood during exercise can improve neurogenesis. Also, aerobic exercise can increase level of BDNF that is associated with oxygen metabolism, as an effective element for supporting the survival of existing neurons. Thus, it could be expected that aerobic activity increases the rate of oxygen consumption in healthy older adults and enhances performance of cognition. In addition to oxygen roles on the brain development, it has also shown in the pathological processes and the brain dysfunctions. Accordingly, the increasing of oxygen transportation may be an important factor in improving neuronal growth, cell proliferation, cell survival, and creating more stimulation of neurogenesis in normal aging process in protecting of cognitive abilities.

Aside from, oxygen consumption and beneficial impact of aerobic metabolism, muscle activity can directly enhance synaptic plasticity through the change of synaptic structure and strength, and lead to supporting neurogenesis.

One of the key mechanisms of muscle activity on neural cells is induction of growth factors and upregulates neurotrophins like BDNF that support neuronal survival and differentiation in the developing brain and dendritic branching in the adult brain.

Hence, even mild intensity of voluntary exercise is an important factor to improve neuronal growth, create more stimulate neurogenesis, improve mental performance and increase resistance to brain disorder in the adult brain.

Since, a creation of activity in neuromuscular junction is not dependent on intensity of physical exercise. There is a hope that low intensity physical exercise via movement games and training games can improve older adults' cognitive function.

This kind of activity could be a potent protective factor for cognitive dysfunction and dementia in the adult people. In fact, disabilities movement of the elderly in aerobic activity such as walking and running should not lead to more inactivity. Hence, our findings suggested that practising motor skills can help to develop older adults' cognitive abilities. Engaging in new motor skill learning leads synaptic changes and creation of new proteins in neurons of the brain. When synaptic junction engages in muscle activity, the facilitating and acceleration of electrochemical transfers and neurotransmitters occur. It appears that increasing of synaptic activity can develop capacity of certain circuits of neurons in the brain and can lead to improvement of the cognitive functions in older adults.

Our protocol (like those ones with light intensity and without running regular) includes new fine learning motor skill, stretching, toning, coordination (for example hand-eye coordination games), equilibrium and also sensory play and simple manipulative games that could represent an important and potent protective factor for cognitive abilities in the elderly population.

We conclude that the participation in aerobic and anaerobic activity programs receive suitable levels of physical and psychological health.
Our study suggests that engaging in light intensity regular type of physical exercise activities that involves the aerobic, non-aerobic and motor skill learning among other health benefits, may delay or prevent the onset of cognitive impairment and dementia in non-pathological and normative aging. Though, our findings need confirmation in further intervention studies.

Limitations

Several limitations to our study should be considered. In this study, results may be confounded by unmeasured factors. Also, the present study has other limitations; our results might possibly be argued and be criticized by some exercise physiological principles, because in current study:

1. Our inhomogeneous population is one of the limitations.
2. At baseline and finish, we did not measure physiological factors in precision – such as the aerobic and anaerobic capacity of our subjects. Physiological measures used to assess changes in aerobic fitness were maximal work load, submaximal heart rate at a standard work load, predicted maximum oxygen uptake, and resting heart rate.
3. Participants were assigned non-randomly into control and exercise groups.
4. A number of exercise group subjects had previously been more inactive than control group members.
5. There can be no certainty about our probands' motivation and if the concentration has remained even-leveled throughout each exercise.
6. Although, it is supposed that major parts of positive effects in anaerobic condition depended on engagement to new motor learning, but the anaerobic protocol of the current study is a set of various exercises that encompasses new motor skills learning, Stroop, neuromuscular, cross-brain and body equilibrium exercises, thus to find the answer that, what exercise has the ability to create better effects in cognitive functions of sedentary older population, in future, these conditions should assay alone and separation.
7. We suggest that to find exact effects of the components of our non-aerobic protocol, anyone of stretching, toning, Stroop training during movement training, equilibrium, coordination and muscle activity should be examined separately.

Summary

In this medium case and prospective study we showed how different kinds of exercise affect cognitive function of healthy older adults. 89 adults aged 65 to 75 years without dementia took part in the study; 37 men and 52 women. They were assigned voluntary and non-randomly – due to individually interested subjects – into one control group (CG) and into two exercise groups (EG); the CG consisted of 28 participants with an average age of 67.93 years. The EG was divided into two sub-groups doing either aerobic exercises (for example, walking and jogging) or anaerobic exercises (e.g., stretching, toning, coordination, Stroop training during movement training, equilibrium, muscle activity, etc.). The aerobic sub-group (AEG) consisted of 30 participants with an average age of 68.52 years. The anaerobic group (ANEG) consisted of 31 participants with an average age of 68.20 years. Participants of both EG groups trained three times a week for three months. In contrast, subjects in the CG only took part in the pre-test and the posttest. The SPSS-Analysis Covariance (ANCOVA) showed a significant interaction that indicated beneficial contributions of anaerobic training on memory function.

The beneficial effects of aerobic exercise may not be merely limited to physiological advantages such as increasing oxygen consumption, blood circulation, the number of capillaries, and vascular function. Though, bearing in mind that human brain is one of the neediest oxygen consumers in the body. In addition of oxygen roles in the brain development, it has also shown in the pathological processes and the brain dysfunctions. Accordingly, the increasing of oxygen transportation may be an important factor in improving neuronal growth, cell proliferation, cell survival, and creating more stimulation of neurogenesis. Thus, this is surely established that participants in aerobic activity programs receive suitable levels of physical and psychological health. During physical exercise regional cerebral blood flow, in major cerebral arteries and also blood flow in the internal carotid artery enhances, causing an increase in blood flow to a wide regions of the brain. Aerobic exercise increases oxygen saturation angiogenesis and improves vascular function in brain areas crucial for task cognitive performance.

These changes are generally associated with aerobic exercise and endurance exercise. This confirms that the transmission of foodstuffs accompanied by the blood during exercise can improve neurogenesis and act as a useful tool for preventing the cognitive decline in adults' population. Thus, it could be expected that aerobic exercise increases the rate of oxygen consumption in healthy older adults and enhances performance of cognition. Additionally, aerobic exercise can increase level of BDNF, an effective element for supporting the survival of existing neurons in some areas of the brain including hippocampus, medial temporal lobe, amygdala, frontal, prefrontal and parietal cortices. These are vital areas related to learning process, cognitive function, memory performance, and notably the long-term memory. Also, physical exercise can directly enhance synaptic plasticity by changing the synaptic structure and strength, thus, supports neurogenesis. One of the key mechanisms of exercise and physical activity on the brain is induction of growth factors, which plays an important role in structural and functional changes as well as neuroprotective effects in aging. However, our findings support that the participation in regular aerobic activities can help to preserve cognitive activity and decrease the risk of dementia in older adults' population.

The results of our study confirmed that both aerobic and anaerobic conditions were able to significantly improve older adults' visual memory in both short and long-term. In verbal memory (VLMT), the outcomes of aerobic and anaerobic conditions for immediate recall (short-term memory or STM), recall after learning in the 5th trial, recall after interference and delayed recall differed a lot. As shown in the text and results, there were no significant differences between all three groups in immediate recall. It appears that perhaps the verbal short-term memory depends less on physical activity and exercise. In fact, both exercises condition did not create significant changes between both exercise groups and control group (CG). We didn't find a significant increasing impact of exercise conditions on immediate recall for verbal learning memory.

In all events, the influence of both exercise conditions lead to better results compared to the control group, even though, it was non-significant. Unlike the aerobic group, interventions of anaerobic effects indicate a significant difference at

recall after learning (5th trial in VLMT), with the CG, interestingly, in this parameter, we can see a significant difference between ANEG and AEG too. This implies that engaging in the low intensity anaerobic condition even without the need of running can create robust beneficial effects in seniors' cognition, in contrast to regular running and other systematic aerobic program. Also, taking into the results, we can see that the intervening aerobic condition cannot produce a noteworthy difference at recall after interference in the VLMT with the control group. In VLMT after interference list, in trial 7 when no list is read and subjects are asked to recall as many words as possible from the A list, we can see that the intervening anaerobic condition can produce a significant difference at recall after interference in the VLMT with both the aerobic and control group, and this is more when comparing with the aerobic and control group; the aerobic condition has not only positive effects, but also the negative effect of decreasing the outcomes in this phase (Pre-test M = 45.89, Post-test M = 41.81). This is really unexpected in comparison of low intensity exercises of both aerobic and anaerobic and regarding our results. It seems that mild anaerobic intervention can create better improvements in cognitive functions and memory performance of elderly than aerobic. Though the results are promising, findings of our aerobic group suggest that aerobic exercise is not always as effective as its anaerobic counterpart in influencing cognitive abilities and particularly in memory system.

The results of long-term verbal memory at delayed recall in VLMT are significant for both the aerobic and anaerobic groups when contrasted to the CG. Our results of aerobic condition confirm the results of studies about relationship between older adults' cognition and level of oxygen consumption (Ramel, Wagner, & Elmadfa, 2004), roles of mitochondria (Blalock et al., 2004; Bratic & Trifunovic, 2010; Dringen, Gutterer, & Hirrlinger, 2000; Loerch et al., 2008; Lu et al., 2004; Miquel, Economos, Fleming, & Johnson Jr, 1980; Pak et al., 2003) and the formation of ROS (Fisher-Wellman & Bloomer, 2009; Radak, Chung, & Goto, 2008); exercise may actually make a difference in the aging process using the aerobic condition (Bishop et al., 2010; Rea, Ventura, & Johnson, 2007; Sánchez-Blanco, Fridell, & Helfand, 2006). It appears that the optimal level of aerobic

exercise concerning both intensity and duration, as well as the optimal level of ROS generation may be of importance in brain function (Knez, Coombes, & Jenkins, 2006; Radak et al., 2008). If the modification of the oxygen consumption by physical activity induces the neurogenesis in the brain, a novel idea provides preserving the ability of learning processes and memory function because of the involvement of neurogenesis in the process of cognitive function. Thus, this study proposes the future investigations study effects of aerobic exercises on older adults' cognitive performance with exact measurement of aerobic condition according to the $VO_{2\ max}$ levels of physical aerobic program.

In comparison of the time and duration of memory function, it appears that low and light type of the aerobic and anaerobic intervention can create almost similar improvements in memory performance of elderly. Also, findings of the current work suggest the mild to moderate form of anaerobic exercise is more capable than aerobic programs in improving these brain regions effectively. Anaerobic group participants have performed exercises such as new motor learning, Stroop, neuromuscular, cross-brain and body equilibrium exercises; this caused an increase in brain activity and strengthening of the underlying systems that support brain plasticity – including metabolism and vascular function, cell proliferation, cell survival, neurogenesis – which may have caused in turn the increased cognitive function and memory performance compared to the aerobic group which have repeated only the same aerobic activity.

The intervention of anaerobic condition produces a significant difference on other cognitive function aspects, which are examined in the study. We saw a remarkable differences for both EG in the mirror reading tasks, the Trail Making Test (TMT A&B), verbal intelligence (or MWT-B) and in non-verbal intelligence (or LPS-4). Though, it was more robust at significant level for anaerobic groups. Our findings diverge from the data of previous studies, some of which have failed to observe the benefits of anaerobic exercise on cognitive processes. Although, our results need confirmation in further intervention studies, these results suggest that regular non-aerobic could represent an important and potent protective factor for cognitive dysfunction and dementia in the adult people.

All these points to the decline of memory function in late adulthood are not inevitable, that it can be reversed with light intensity of our anaerobic protocol. It seems that for supporting cognitive function in older adults that have difficulty with performing aerobic exercise such as walking and running, the light intensity form of non-aerobic programs may maintain older adults' cognitive abilities. Hence, even mild intensity form of voluntary exercise is an important factor to improve neuronal growth, create more stimulate neurogenesis, improve mental performance and increase resistance to brain disorder in the adult brain.

Since, a creation of activity in neuromuscular junction is not dependent on intensity of physical exercise. It is hoped that low intensity physical exercise via movement games and training games can improve older adults' cognitive function. This kind of activity could be a potent protective factor for cognitive dysfunction and dementia in the adult people. In fact, disability in movement of the elderly in aerobic activities such as walking and running should not be lead to more inactivity. Hence, our findings suggested that practicing motor skills can help to develop older adults' cognitive abilities. Engaging in new motor skill learning leads synaptic changes and creating new proteins in neurons of the brain. When synaptic junction engages in muscle activity, the facilitating and acceleration of electrochemical transfers and neurotransmitters occur. It appears that increasing of synaptic activity can develop capacity of certain circuits of neurons in the brain and can lead to improvement of the cognitive functions in older adults.

We conclude that the participation in aerobic and anaerobic activity programs receive suitable levels of physical and psychological health. Our study suggests that engaging in light intensity form of regular physical exercise activity involves the aerobic, non-aerobic and motor skill learning among other health benefits, may delay or prevent the onset of cognitive impairment and dementia in non-pathological and normative aging. Though, our findings need confirmation in further intervention and brain morphological studies.

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Appendix A

Raw data of the study

	VP Code	VP Name	Groups	Gender	Education Level	Age
1	1	ZM	1,00	2	1	65,00
2	2	FG	3,00	2	1	68,00
3	4	DW	2,00	1	1	71,00
4	5	MR	1,00	1	1	69,00
5	11	FM	2,00	1	1	64,00
6	12	GH	2,00	1	1	70,00
7	17	KL	3,00	2	2	66,00
8	18	KC	1,00	2	2	65,00
9	19	KH	1,00	1	1	67,00
10	20	DL	2,00	2	2	65,00
11	25	KV	1,00	1	2	67,00
12	26	KD	1,00	2	1	66,00
13	27	RR	2,00	1	1	67,00
14	28	KM	1,00	1	1	67,00
15	29	SM	1,00	2	1	75,00
16	30	MR	2,00	1	2	70,00
17	33	HG	2,00	2	1	64,00
18	34	MD	2,00	1	2	67,00
19	35	WJ	2,00	2	2	72,00
20	36	MM	3,00	2	1	74,00
21	37	SH	3,00	2	1	67,00
22	38	BM	3,00	2	1	70,00
23	39	WA	3,00	2	1	71,00
24	40	DA	2,00	2	1	69,00
25	41	LM	1,00	2	2	67,00

Control group 1, Anaerobic 2, Aerobic 3.
Education level: 1 < 12 years and 2 > 12 years.
Gender: 1 = male and 2 = female.

	ROCF Immediate Pre-test	ROCF Delayed Pre-test	ROCF Immediate Post-test	ROCF Delayed Post-test	MWT-B Pre-test	MWT-B Post-test	LPS-4 Pre-test
1	51,00	85,00	79,00	79,00	124,00	118,00	120,00
2	57,00	60,00	80,00	85,00	112,00	118,00	122,00
3	80,00	51,00	70,00	62,00	112,00	118,00	110,00
4	71,00	76,00	74,00	70,00	124,00	118,00	110,00
5	77,00	60,00	75,00	71,00	136,00	136,00	115,00
6	60,00	51,00	60,00	63,00	124,00	136,00	118,00
7	85,00	63,00	85,00	85,00	143,00	130,00	134,00
8	40,00	78,00	70,00	68,00	104,00	104,00	107,00
9	75,00	72,00	62,00	66,00	92,00	92,00	94,00
10	85,00	71,00	85,00	85,00	124,00	124,00	130,00
11	63,00	40,00	49,00	56,00	112,00	112,00	125,00
12	51,00	76,00	70,00	71,00	118,00	118,00	122,00
13	75,00	46,00	80,00	80,00	130,00	130,00	130,00
14	76,00	66,00	55,00	55,00	130,00	130,00	100,00
15	69,00	85,00	85,00	85,00	124,00	118,00	110,00
16	52,00	61,00	71,00	70,00	130,00	130,00	122,00
17	53,00	51,00	85,00	80,00	112,00	143,00	120,00
18	51,00	71,00	75,00	78,00	130,00	136,00	115,00
19	61,00	80,00	85,00	85,00	136,00	136,00	120,00
20	70,00	60,00	64,00	61,00	97,00	107,00	94,00
21	53,00	54,00	54,00	55,00	101,00	101,00	125,00
22	56,00	51,00	70,00	66,00	118,00	130,00	115,00
23	42,00	51,00	85,00	85,00	124,00	124,00	115,00
24	63,00	75,00	85,00	85,00	124,00	124,00	102,00
25	58,00	69,00	60,00	60,00	130,00	130,00	107,00

	LPS-4 Post-test	TMT-A Pre-test	TMT-A Post-Test	TMT-B Pre-test	TMT-B Post-Test	Mirror reading Priming Pre-test
1	120,00	30,00	30,00	30,00	30,00	110,00
2	134,00	70,00	80,00	90,00	90,00	76,00
3	110,00	80,00	90,00	90,00	90,00	247,00
4	105,00	80,00	70,00	60,00	60,00	248,00
5	134,00	90,00	90,00	90,00	90,00	56,00
6	120,00	80,00	70,00	60,00	90,00	92,00
7	127,00	90,00	90,00	70,00	40,00	62,00
8	107,00	90,00	30,00	20,00	10,00	357,00
9	90,00	10,00	10,00	10,00	10,00	436,00
10	134,00	90,00	90,00	90,00	90,00	101,00
11	125,00	90,00	70,00	90,00	10,00	180,00
12	122,00	90,00	80,00	90,00	80,00	79,00
13	134,00	90,00	80,00	50,00	80,00	108,00
14	98,00	10,00	10,00	40,00	40,00	101,00
15	110,00	60,00	60,00	90,00	90,00	147,00
16	125,00	80,00	90,00	70,00	90,00	118,00
17	120,00	20,00	60,00	90,00	90,00	84,00
18	120,00	10,00	10,00	10,00	10,00	165,00
19	122,00	50,00	60,00	80,00	90,00	155,00
20	96,00	80,00	90,00	40,00	60,00	183,00
21	125,00	80,00	80,00	40,00	60,00	118,00
22	118,00	40,00	80,00	80,00	80,00	170,00
23	122,00	10,00	70,00	60,00	90,00	129,00
24	113,00	20,00	80,00	60,00	90,00	75,00
25	105,00	70,00	70,00	80,00	80,00	91,00

	Mirror reading Procedural Pre-test	Mirror reading Interference Pre-test	Mirror reading Priming Post-test	Mirror reading Procedural Post-test	Mirror reading Interference Post-test	VLMT Immediate Pre-test
1	126,00	100,00	109,00	132,00	95,00	95,00
2	83,00	90,00	98,00	70,00	82,00	77,50
3	277,00	145,00	180,00	168,00	100,00	7,50
4	183,00	202,00	303,00	240,00	162,00	57,50
5	60,00	62,00	55,00	68,00	57,00	77,50
6	61,00	71,00	64,00	68,00	77,00	57,50
7	129,00	84,00	77,00	128,00	72,00	37,50
8	242,00	272,00	281,00	344,00	339,00	92,50
9	262,00	283,00	478,00	260,00	164,00	4,00
10	155,00	95,00	89,00	140,00	73,00	77,50
11	181,00	144,00	110,00	331,00	119,00	77,50
12	60,00	57,00	61,00	98,00	69,00	37,50
13	122,00	95,00	89,00	115,00	80,00	20,00
14	189,00	105,00	120,00	150,00	105,00	37,50
15	222,00	318,00	180,00	232,00	159,00	95,00
16	164,00	125,00	219,00	109,00	146,00	77,50
17	152,00	73,00	57,00	100,00	37,00	95,00
18	161,00	124,00	97,00	79,00	109,00	37,50
19	147,00	143,00	94,00	104,00	95,00	95,00
20	292,00	309,00	136,00	131,00	146,00	57,50
21	181,00	212,00	54,00	67,00	59,00	20,00
22	224,00	171,00	95,00	205,00	125,00	77,50
23	149,00	191,00	122,00	118,00	99,00	20,00
24	154,00	72,00	50,00	48,00	50,00	37,50
25	164,00	107,00	84,00	102,00	92,00	77,50

	VLMT Immediate Post-test	VLMT after learning Pre-test	VLMT after learning Post-test	VLMT after interference Pre-test	VLMT after interference Post-test	VLMT delayed recall Pre-test	VLMT delayed recall Post-test
1	92,50	70,00	87,50	77,50	97,50	95,00	55,00
2	57,50	55,00	42,50	67,50	57,50	90,00	55,00
3	20,00	4,00	4,00	4,00	15,00	4,00	4,00
4	57,50	55,00	55,00	77,50	57,50	72,50	55,00
5	97,50	32,50	87,50	77,50	97,50	90,00	95,00
6	77,50	22,50	55,00	22,50	4,00	7,50	4,00
7	77,50	32,50	42,50	47,50	22,50	25,00	35,00
8	57,50	15,00	42,50	7,50	15,00	4,00	17,50
9	4,00	4,00	4,00	4,00	4,00	4,00	3,00
10	37,50	70,00	55,00	77,50	67,50	95,00	42,50
11	92,50	42,50	22,50	35,00	15,00	17,50	5,00
12	92,50	22,50	42,50	22,50	4,00	17,50	25,00
13	20,00	42,50	42,50	47,50	57,50	32,50	35,00
14	37,50	10,00	4,00	5,00	4,00	7,50	3,00
15	77,50	55,00	72,50	77,50	47,50	90,00	47,50
16	97,50	15,00	22,50	7,50	4,00	4,00	4,00
17	97,50	70,00	87,50	67,50	97,50	72,50	97,50
18	20,00	22,50	55,00	35,00	87,50	42,50	55,00
19	92,50	55,00	87,50	67,50	97,50	47,50	97,50
20	57,50	22,50	32,50	22,50	4,00	25,00	5,00
21	20,00	22,50	10,00	35,00	5,00	25,00	22,50
22	77,50	87,50	87,50	77,50	97,50	90,00	95,00
23	77,50	32,50	22,50	4,00	22,50	7,50	5,00
24	77,50	32,50	87,50	57,50	97,50	47,50	97,50
25	77,50	15,00	15,00	35,00	47,50	25,00	47,50

	VP Code	VP Name	Groups	Gender	Education Level	Age
26	42	KM	3,00	2	1	69,00
27	43	AW	2,00	1	2	70,00
28	44	PM	3,00	2	1	68,00
29	45	EH	2,00	1	2	68,00
30	46	HA	2,00	2	2	70,00
31	47	JU	2,00	1	2	69,00
32	48	WA	1,00	2	2	69,00
33	49	BH	2,00	1	1	64,00
34	50	BG	1,00	2	2	67,00
35	51	JU	2,00	2	2	66,00
36	52	WW	1,00	1	2	69,00
37	53	UG	1,00	2	2	75,00
38	55	WK	3,00	1	2	66,00
39	56	PS	2,00	2	1	67,00
40	57	ZB	1,00	2	1	69,00
41	58	FW	1,00	1	1	65,00
42	59	BH	3,00	1	1	69,00
43	60	AU	2,00	2	1	65,00
44	61	PH	3,00	2	2	66,00
45	62	LD	2,00	1	2	73,00
46	63	MR	1,00	1	1	67,00
47	64	WJ	1,00	2	1	69,00
48	65	SU	1,00	2	1	72,00
49	66	GH	2,00	1	2	64,00
50	69	FJ	3,00	1	2	72,00
51	70	HG	1,00	2	1	65,00
52	71	FJ	3,00	1	1	69,00
53	73	HP	2,00	2	1	65,00
54	74	HW	2,00	1	1	68,00
55	76	GM	3,00	2	2	69,00
56	77	UJ	3,00	2	1	70,00
57	78	SR	3,00	2	2	64,00

Control group 1, Anaerobic 2, Aerobic 3.
Education level: 1 < 12 years and 2 > 12 years.
Gender: 1 = male and 2 = female

	ROCF Immediate Pre-test	ROCF Delayed Pre-test	ROCF Immediate Post-test	ROCF Delayed Post-test	MWT-B Pre-test	MWT-B Post-test	LPS-4 Pre-test
26	55,00	39,00	68,00	71,00	104,00	107,00	110,00
27	72,00	57,00	85,00	77,00	136,00	136,00	113,00
28	63,00	44,00	43,00	53,00	136,00	104,00	83,00
29	85,00	64,00	85,00	85,00	124,00	136,00	117,00
30	66,00	53,00	76,00	80,00	136,00	136,00	118,00
31	64,00	48,00	80,00	85,00	118,00	136,00	122,00
32	64,00	73,00	49,00	55,00	130,00	118,00	134,00
33	55,00	66,00	85,00	85,00	101,00	136,00	120,00
34	51,00	85,00	85,00	85,00	112,00	118,00	134,00
35	57,00	78,00	85,00	85,00	130,00	136,00	130,00
36	80,00	68,00	61,00	68,00	101,00	101,00	110,00
37	71,00	65,00	58,00	58,00	112,00	104,00	108,00
38	55,00	60,00	72,00	69,00	130,00	130,00	125,00
39	59,00	55,00	63,00	66,00	124,00	130,00	113,00
40	63,00	59,00	53,00	44,00	107,00	101,00	104,00
41	80,00	76,00	63,00	66,00	124,00	118,00	107,00
42	85,00	85,00	85,00	85,00	130,00	118,00	120,00
43	57,00	62,00	72,00	69,00	107,00	107,00	110,00
44	60,00	57,00	68,00	69,00	112,00	118,00	110,00
45	85,00	85,00	85,00	85,00	143,00	145,00	113,00
46	59,00	60,00	57,00	55,00	112,00	107,00	87,00
47	63,00	78,00	72,00	69,00	112,00	112,00	110,00
48	76,00	73,00	51,00	56,00	124,00	118,00	98,00
49	85,00	85,00	85,00	85,00	118,00	118,00	117,00
50	62,00	65,00	69,00	70,00	124,00	130,00	122,00
51	85,00	85,00	70,00	71,00	118,00	112,00	104,00
52	70,00	73,00	77,00	85,00	118,00	130,00	122,00
53	55,00	53,00	85,00	85,00	118,00	118,00	102,00
54	53,00	57,00	60,00	62,00	124,00	124,00	105,00
55	70,00	62,00	66,00	64,00	143,00	136,00	113,00
56	62,00	68,00	71,00	75,00	124,00	124,00	120,00
57	80,00	75,00	85,00	85,00	130,00	143,00	110,00

	LPS-4 Post-test	TMT-A Pre-test	TMT-A Post-Test	TMT-B Pre-test	TMT-B Post-Test	Mirror reading Priming Pre-test
26	113,00	80,00	80,00	90,00	90,00	255,00
27	115,00	70,00	60,00	50,00	90,00	222,00
28	87,00	30,00	30,00	10,00	10,00	1267,00
29	120,00	90,00	90,00	90,00	90,00	144,00
30	120,00	90,00	90,00	90,00	90,00	87,00
31	134,00	90,00	90,00	80,00	90,00	74,00
32	130,00	10,00	10,00	30,00	10,00	136,00
33	127,00	90,00	90,00	10,00	20,00	212,00
34	130,00	80,00	80,00	70,00	60,00	53,00
35	134,00	20,00	30,00	60,00	90,00	59,00
36	110,00	90,00	30,00	30,00	10,00	303,00
37	100,00	60,00	60,00	30,00	30,00	205,00
38	122,00	30,00	80,00	70,00	90,00	120,00
39	125,00	30,00	50,00	10,00	80,00	163,00
40	102,00	60,00	50,00	60,00	50,00	249,00
41	107,00	80,00	80,00	40,00	30,00	416,00
42	127,00	10,00	10,00	90,00	80,00	90,00
43	115,00	80,00	90,00	80,00	80,00	113,00
44	115,00	40,00	90,00	70,00	70,00	203,00
45	130,00	40,00	80,00	10,00	20,00	123,00
46	87,00	30,00	10,00	40,00	40,00	-
47	110,00	50,00	10,00	40,00	10,00	230,00
48	98,00	40,00	30,00	10,00	10,00	155,00
49	120,00	50,00	80,00	50,00	50,00	131,00
50	118,00	40,00	40,00	10,00	50,00	107,00
51	120,00	80,00	80,00	70,00	70,00	87,00
52	122,00	30,00	30,00	60,00	70,00	149,00
53	110,00	30,00	90,00	40,00	90,00	270,00
54	115,00	20,00	50,00	50,00	60,00	311,00
55	134,00	30,00	90,00	80,00	90,00	116,00
56	127,00	30,00	20,00	60,00	20,00	321,00
57	113,00	40,00	40,00	20,00	30,00	79,00

	Mirror reading Procedural Pre-test	Mirror reading Interference Pre-test	Mirror reading Priming Post-test	Mirror reading Procedural Post-test	Mirror reading Interference Post-test	VLMT Immediate Pre-test
26	289,00	215,00	124,00	171,00	128,00	77,50
27	235,00	239,00	95,00	110,00	111,00	57,50
28	1578,00	901,00	1032,00	826,00	393,00	20,00
29	328,00	142,00	102,00	152,00	128,00	92,50
30	114,00	98,00	87,00	99,00	59,00	77,50
31	240,00	87,00	71,00	87,00	71,00	37,50
32	105,00	74,00	137,00	121,00	103,00	92,50
33	191,00	318,00	165,00	158,00	162,00	77,50
34	57,00	43,00	62,00	57,00	59,00	95,00
35	69,00	83,00	42,00	37,00	51,00	92,50
36	344,00	174,00	218,00	392,00	250,00	92,50
37	331,00	188,00	215,00	472,00	303,00	57,50
38	105,00	153,00	165,00	142,00	158,00	37,50
39	219,00	215,00	75,00	111,00	68,00	37,50
40	263,00	385,00	298,00	337,00	241,00	20,00
41	426,00	355,00	443,00	450,00	453,00	77,50
42	123,00	101,00	90,00	95,00	88,00	57,50
43	196,00	190,00	92,00	199,00	78,00	57,50
44	182,00	269,00	116,00	99,00	82,00	92,50
45	132,00	106,00	69,00	100,00	69,00	37,50
46	-	-	-	-	-	37,50
47	300,00	232,00	396,00	400,00	195,00	92,50
48	144,00	206,00	247,00	220,00	205,00	37,50
49	103,00	82,00	51,00	99,00	55,00	77,50
50	379,00	121,00	96,00	92,00	101,00	57,50
51	85,00	85,00	93,00	91,00	99,00	20,00
52	254,00	125,00	110,00	145,00	87,00	37,50
53	371,00	202,00	116,00	97,00	107,00	57,50
54	135,00	283,00	151,00	133,00	105,00	57,50
55	106,00	12,00	91,00	99,00	107,00	95,00
56	152,00	164,00	260,00	146,00	129,00	92,50
57	85,00	85,00	58,00	78,00	60,00	92,50

	VLMT Immediate Post-test	VLMT after learning Pre-test	VLMT after learning Post-test	VLMT after interference Pre-test	VLMT after interference Post-test	VLMT delayed recall Pre-test	VLMT delayed recall Post-test
26	77,50	42,50	55,00	67,50	57,50	42,50	42,50
27	37,50	10,00	32,50	7,50	15,00	4,00	5,00
28	57,50	4,00	15,00	4,00	4,00	4,00	4,00
29	20,00	70,00	87,50	77,50	87,50	47,50	95,00
30	97,50	70,00	87,50	57,50	77,50	55,00	95,00
31	97,50	42,50	32,50	35,00	67,50	42,50	55,00
32	97,50	70,00	87,50	90,00	87,50	72,50	97,50
33	37,50	42,50	55,00	47,50	57,50	47,50	47,50
34	37,50	87,50	42,50	90,00	77,50	90,00	55,00
35	92,50	32,50	42,50	22,50	35,00	32,50	35,00
36	57,50	32,50	32,50	22,50	5,00	17,50	4,00
37	20,00	22,50	10,00	47,50	15,00	42,50	4,00
38	37,50	42,50	22,50	57,50	22,50	42,50	25,00
39	20,00	42,50	57,50	35,00	67,50	42,50	47,50
40	20,00	15,00	5,00	15,00	4,00	25,00	7,50
41	77,50	22,50	22,50	77,50	77,50	42,50	42,50
42	77,50	10,00	15,00	4,00	22,50	4,00	42,50
43	92,50	42,50	70,00	35,00	47,50	25,00	32,50
44	92,50	42,50	70,00	57,50	67,50	47,50	72,50
45	57,50	4,00	10,00	4,00	22,50	4,00	32,50
46	37,50	32,50	32,50	7,50	7,50	4,00	4,00
47	4,00	55,00	42,50	77,50	57,50	90,00	42,50
48	5,00	32,50	10,00	7,50	15,00	7,50	7,50
49	77,50	22,50	42,50	35,00	47,50	42,50	32,50
50	77,50	32,50	55,00	57,50	22,50	55,00	42,50
51	20,00	70,00	32,50	77,50	57,50	55,00	47,50
52	37,50	15,00	15,00	4,00	4,00	4,00	4,00
53	57,50	32,50	55,00	22,50	67,50	25,00	42,50
54	57,50	10,00	15,00	35,00	35,00	25,00	25,00
55	97,50	55,00	55,00	77,50	77,50	90,00	90,00
56	77,50	55,00	42,50	77,50	77,50	47,50	90,00
57	92,50	42,50	55,00	57,50	77,50	42,50	90,00

	VP Code	VP Name	Groups	Gender	Education Level	Age
58	79	SE	2,00	2	1	66,00
59	80	PB	3,00	2	1	69,00
60	81	NH	2,00	2	1	72,00
61	82	SH	1,00	1	2	68,00
62	83	VF	1,00	2	1	64,00
63	85	SA	3,00	2	2	64,00
64	86	AH	2,00	2	2	65,00
65	87	LK	2,00	2	2	73,00
66	88	MH	3,00	2	2	70,00
67	89	PL	1,00	1	2	70,00
68	90	MU	3,00	2	2	74,00
69	91	RH	3,00	2	1	71,00
70	93	BB	3,00	1	2	65,00
71	94	BD	3,00	2	1	67,00
72	95	MF	2,00	2	2	68,00
73	96	MI	2,00	1	1	69,00
74	97	MU	2,00	2	1	72,00
75	98	HT	1,00	2	1	67,00
76	99	LH	1,00	1	2	64,00
77	100	MH	2,00	1	2	73,00
78	101	AU	1,00	2	1	68,00
79	102	KW	1,00	1	2	67,00
80	105	OK	3,00	1	1	74,00
81	106	HK	3,00	1	2	74,00
82	107	WB	3,00	2	1	65,00
83	108	SM	1,00	2	2	72,00
84	109	WG	3,00	1	1	64,00
85	111	BS	3,00	2	2	64,00
86	112	SU	1,00	2	1	70,00
87	113	BM	3,00	1	2	66,00
88	114	BH	3,00	2	1	69,00
89	115	BH	3,00	1	2	70,00

Control group 1, Anaerobic 2, Aerobic 3.
Education level: 1 < 12 years and 2 > 12 years.
Gender: 1 = male and 2 = female

	ROCF Immediate Pre-test	ROCF Delayed Pre-test	ROCF Immediate Post-test	ROCF Delayed Post-test	MWT-B Pre-test	MWT-B Post-test	LPS-4 Pre-test
58	63,00	62,00	77,00	85,00	124,00	124,00	110,00
59	70,00	78,00	85,00	85,00	112,00	124,00	107,00
60	54,00	54,00	74,00	73,00	112,00	118,00	110,00
61	57,00	57,00	55,00	53,00	104,00	104,00	120,00
62	68,00	75,00	63,00	62,00	107,00	104,00	110,00
63	51,00	53,00	63,00	60,00	118,00	124,00	102,00
64	53,00	48,00	66,00	62,00	107,00	107,00	92,00
65	49,00	39,00	71,00	70,00	118,00	136,00	92,00
66	62,00	70,00	79,00	80,00	107,00	118,00	106,00
67	85,00	85,00	79,00	75,00	107,00	107,00	115,00
68	69,00	77,00	85,00	70,00	136,00	136,00	122,00
69	74,00	70,00	85,00	80,00	112,00	118,00	98,00
70	59,00	57,00	61,00	62,00	107,00	112,00	117,00
71	75,00	66,00	75,00	78,00	130,00	130,00	110,00
72	28,00	26,00	57,00	48,00	104,00	107,00	104,00
73	55,00	57,00	70,00	63,00	112,00	112,00	98,00
74	71,00	70,00	80,00	85,00	136,00	136,00	106,00
75	66,00	69,00	66,00	64,00	124,00	104,00	122,00
76	85,00	85,00	77,00	85,00	118,00	118,00	130,00
77	69,00	73,00	85,00	85,00	104,00	118,00	103,00
78	49,00	43,00	47,00	41,00	124,00	124,00	120,00
79	63,00	62,00	47,00	47,00	136,00	136,00	115,00
80	76,00	85,00	85,00	85,00	107,00	118,00	120,00
81	73,00	70,00	85,00	85,00	112,00	130,00	108,00
82	80,00	80,00	85,00	85,00	107,00	107,00	115,00
83	67,00	70,00	67,00	63,00	136,00	136,00	110,00
84	63,00	58,00	65,00	71,00	107,00	107,00	110,00
85	61,00	60,00	85,00	85,00	124,00	130,00	104,00
86	69,00	70,00	67,00	67,00	124,00	118,00	106,00
87	77,00	62,00	85,00	85,00	136,00	130,00	105,00
88	49,00	48,00	46,00	60,00	124,00	130,00	115,00
89	64,00	56,00	68,00	68,00	130,00	130,00	100,00

	LPS-4 Post-test	TMT-A Pre-test	TMT-A Post-Test	TMT-B Pre-test	TMT-B Post-Test	Mirror reading Priming Pre-test
58	110,00	50,00	60,00	10,00	20,00	120,00
59	115,00	20,00	30,00	40,00	80,00	300,00
60	113,00	20,00	70,00	20,00	80,00	462,00
61	110,00	10,00	10,00	10,00	10,00	153,00
62	113,00	50,00	50,00	80,00	70,00	301,00
63	104,00	80,00	80,00	10,00	90,00	323,00
64	94,00	10,00	10,00	10,00	10,00	330,00
65	94,00	40,00	40,00	60,00	60,00	177,00
66	106,00	80,00	30,00	40,00	30,00	214,00
67	115,00	50,00	40,00	10,00	10,00	271,00
68	127,00	40,00	50,00	10,00	50,00	242,00
69	103,00	70,00	70,00	10,00	80,00	248,00
70	134,00	10,00	80,00	40,00	70,00	246,00
71	127,00	80,00	90,00	90,00	90,00	119,00
72	107,00	10,00	10,00	10,00	10,00	-
73	104,00	20,00	40,00	20,00	70,00	327,00
74	113,00	80,00	80,00	10,00	30,00	70,00
75	122,00	80,00	80,00	70,00	60,00	77,00
76	122,00	50,00	10,00	60,00	10,00	84,00
77	106,00	50,00	90,00	10,00	60,00	693,00
78	102,00	70,00	30,00	80,00	10,00	129,00
79	115,00	10,00	10,00	40,00	10,00	102,00
80	115,00	70,00	40,00	90,00	90,00	195,00
81	108,00	50,00	80,00	10,00	10,00	285,00
82	120,00	60,00	10,00	70,00	70,00	233,00
83	110,00	10,00	10,00	10,00	10,00	322,00
84	102,00	80,00	70,00	10,00	10,00	163,00
85	104,00	50,00	60,00	90,00	90,00	182,00
86	106,00	30,00	10,00	10,00	10,00	158,00
87	110,00	10,00	70,00	10,00	60,00	75,00
88	105,00	60,00	40,00	10,00	10,00	79,00
89	113,00	50,00	80,00	70,00	90,00	92,00

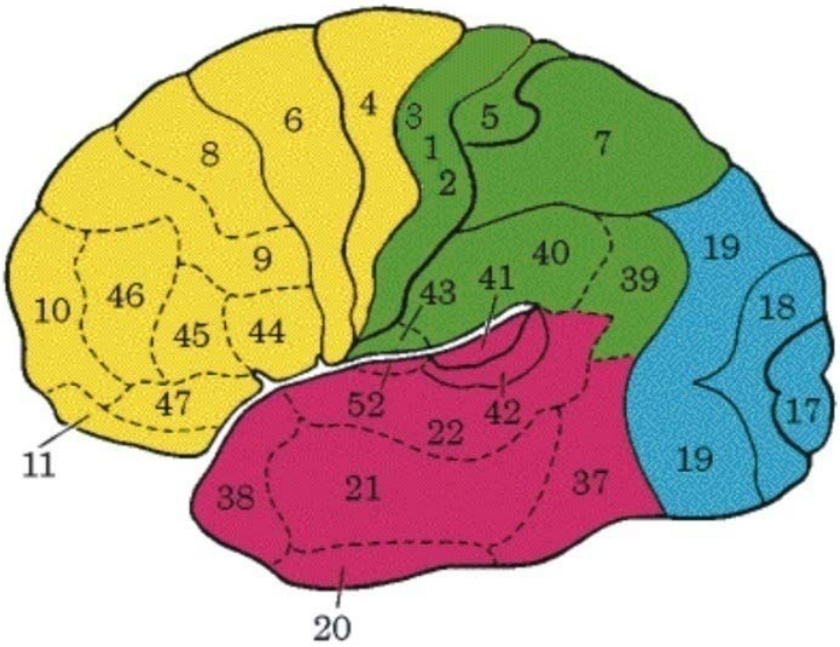
	Mirror reading Procedural Pre-test	Mirror reading Interference Pre-test	Mirror reading Priming Post-test	Mirror reading Procedural Post-test	Mirror reading Interference Post-test	VLMT Immediate Pre-test
58	130,00	90,00	57,00	52,00	65,00	77,50
59	151,00	191,00	108,00	116,00	94,00	37,50
60	385,00	179,00	105,00	160,00	88,00	57,50
61	134,00	144,00	193,00	187,00	206,00	5,00
62	344,00	285,00	477,00	261,00	192,00	57,50
63	384,00	166,00	235,00	218,00	132,00	95,00
64	491,00	295,00	175,00	161,00	167,00	57,50
65	221,00	176,00	91,00	132,00	106,00	92,50
66	350,00	242,00	205,00	126,00	114,00	77,50
67	316,00	222,00	319,00	473,00	313,00	77,50
68	169,00	123,00	57,00	101,00	48,00	57,50
69	243,00	154,00	99,00	147,00	98,00	37,50
70	414,00	248,00	180,00	156,00	126,00	92,50
71	114,00	175,00	142,00	92,00	62,00	92,50
72	-	-	-	-	-	92,50
73	400,00	338,00	194,00	211,00	139,00	5,00
74	78,00	66,00	31,00	57,00	36,00	37,50
75	97,00	69,00	101,00	111,00	82,00	57,50
76	134,00	139,00	111,00	159,00	99,00	92,50
77	237,00	407,00	228,00	190,00	133,00	92,50
78	108,00	96,00	113,00	176,00	135,00	95,00
79	82,00	117,00	166,00	142,00	159,00	37,50
80	159,00	122,00	123,00	127,00	99,00	95,00
81	264,00	309,00	243,00	215,00	220,00	57,50
82	185,00	234,00	180,00	295,00	135,00	92,50
83	202,00	340,00	370,00	263,00	246,00	20,00
84	175,00	167,00	121,00	174,00	80,00	20,00
85	219,00	222,00	129,00	112,00	118,00	95,00
86	246,00	161,00	111,00	121,00	128,00	20,00
87	120,00	89,00	96,00	72,00	69,00	57,50
88	130,00	88,00	66,00	90,00	74,00	37,50
89	123,00	110,00	80,00	86,00	56,00	57,50

	VLMT Immediate Post-test	VLMT after learning Pre-test	VLMT after learning Post-test	VLMT after interference Pre-test	VLMT after interference Post-test	VLMT delayed recall Pre-test	VLMT delayed recall Post-test
58	77,50	22,50	55,00	57,50	57,50	32,50	47,50
59	77,50	42,50	55,00	67,50	67,50	42,50	25,00
60	57,50	5,00	10,00	4,00	4,00	4,00	4,00
61	20,00	42,50	4,00	4,00	4,00	17,50	4,00
62	37,50	87,50	55,00	77,50	47,50	47,50	42,50
63	77,50	70,00	32,50	57,50	67,50	72,50	25,00
64	57,50	32,50	32,50	35,00	57,50	32,50	47,50
65	57,50	70,00	87,50	67,50	77,50	90,00	90,00
66	37,50	22,50	32,50	67,50	22,50	42,50	7,50
67	57,50	42,50	32,50	47,50	35,00	32,50	32,50
68	77,50	22,50	70,00	35,00	67,50	22,50	72,50
69	97,50	22,50	22,50	57,50	47,50	47,50	47,50
70	57,50	32,50	42,50	35,00	7,50	32,50	25,00
71	77,50	70,00	22,50	77,50	67,50	47,50	55,00
72	92,50	32,50	32,50	4,00	4,00	17,50	17,50
73	57,50	10,00	22,50	4,00	7,50	4,00	25,00
74	57,50	10,00	55,00	22,50	22,50	7,50	25,00
75	37,50	70,00	22,50	77,50	35,00	55,00	17,50
76	57,50	70,00	70,00	77,50	57,50	90,00	42,50
77	92,50	42,50	70,00	22,50	47,50	42,50	47,50
78	37,50	87,50	87,50	77,50	35,00	72,50	47,50
79	77,50	10,00	15,00	4,00	4,00	17,50	4,00
80	20,00	70,00	32,50	57,50	22,50	55,00	25,00
81	37,50	32,50	22,50	22,50	22,50	7,50	25,00
82	95,00	87,50	87,50	77,50	90,00	72,50	97,50
83	37,50	32,50	42,50	57,50	15,00	42,50	17,50
84	57,50	10,00	5,00	7,50	4,00	4,00	4,00
85	97,50	70,00	87,50	67,50	97,50	47,50	97,50
86	77,50	15,00	15,00	15,00	4,00	4,00	4,00
87	57,50	22,50	22,50	22,50	15,00	25,00	17,50
88	37,50	42,50	22,50	4,00	7,50	7,50	4,00
89	37,50	42,50	87,50	47,50	47,50	47,50	72,50

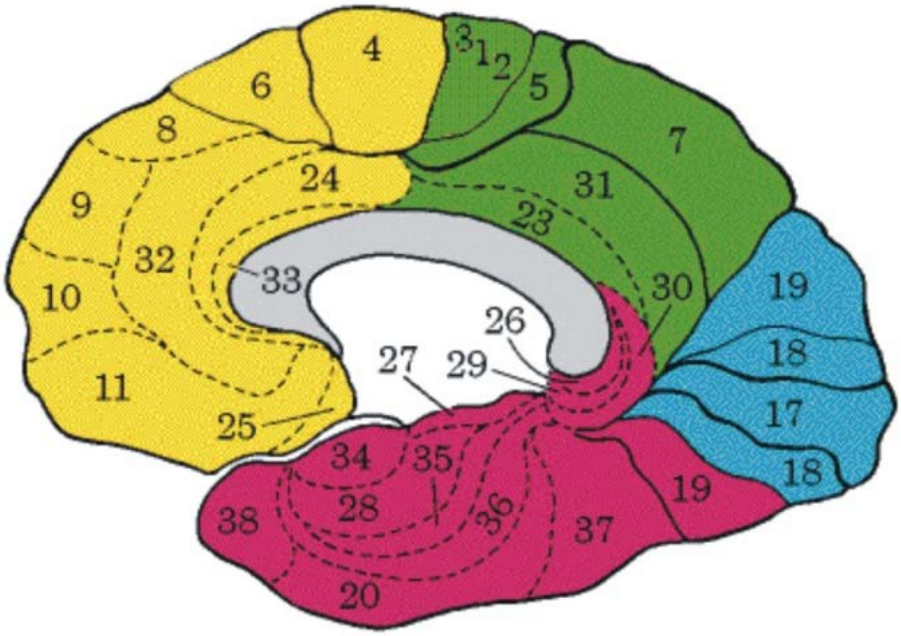
Appendix B

Brodmann Areas

Sagittal lateral view



Sagittal midial view



Appendix C

One-sample Kolmogorov-Smirnov tests

Groups		
N		89
Normal Parameters[a,b]	Mean	2,0337
	Std. Deviation	,81811
Most Extreme Differences	Absolute	,230
	Positive	,211
	Negative	-,230
Test Statistic		,230
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. b. Calculated from data Lilliefors Significance Correction.

Age of participants		
N		89
Normal Parameters[a,b]	Mean	68,2584
	Std. Deviation	2,97538
Most Extreme Differences	Absolute	,113
	Positive	,113
	Negative	-,076
Test Statistic		,113
Asymp. Sig. (2-tailed)		,007[c]

a. Test distribution is Normal. b. Lilliefors Significance Correction.

Pretest of Rey-Osterrieth Figure immediate recall		
N		89
Normal Parameters[a,b]	Mean	65,0899
	Std. Deviation	11,95222
Most Extreme Differences	Absolute	,087
	Positive	,087
	Negative	-,064
Test Statistic		,087
Asymp. Sig. (2-tailed)		,094[c]

a. Test distribution is Normal. b. Lilliefors Significance Correction.

Pretest of Rey-Osterrieth Figure delayed recall		
N		89
Normal Parameters[a,b]	Mean	64,6180
	Std. Deviation	12,91186
Most Extreme Differences	Absolute	,066
	Positive	,057
	Negative	-,066
Test Statistic		,066
Asymp. Sig. (2-tailed)		,200[c,d]

a. Test distribution is Normal..Lilliefors Significance correction.

Posttest of Rey-Osterrieth Figure immediate recall		
N		89
Normal Parameters[a,b]	Mean	71,5393
	Std. Deviation	11,93358
Most Extreme Differences	Absolute	,162
	Positive	,130
	Negative	-,162
Test Statistic		,162
Asymp. Sig. (2-tailed)		,000[c]

a.Test distribution is Normal. b.. Lilliefors Significance Correction.

Post-test of Rey-Osterrieth Figure delayed recall		
N		89
Normal Parameters[a,b]	Mean	71,6292
	Std. Deviation	11,88905
Most Extreme Differences	Absolute	,184
	Positive	,130
	Negative	-,184
Test Statistic		,184
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. Lilliefors Significance Correction.

Pre-test MWT-B verbal intelligence		
N		89
Normal Parameters[a,b]	Mean	119,7640
	Std. Deviation	11,65998
Most Extreme Differences	Absolute	,147
	Positive	,129
	Negative	-,147
Test Statistic		,147
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b.. Lilliefors Significance Correction.

Post-test MWT-B verbal intelligence		
N		89
Normal Parameters[a,b]	Mean	121,6067
	Std. Deviation	11,97594
Most Extreme Differences	Absolute	,152
	Positive	,124
	Negative	-,152
Test Statistic		,152
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

Pre-test LPS-4 Non-verbal intelligence		
N		89
Normal Parameters[a,b]	Mean	112,3034
	Std. Deviation	10,48311
Most Extreme Differences	Absolute	,093
	Positive	,093
	Negative	-,076
Test Statistic		,093
Asymp. Sig. (2-tailed)		,057[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

Post-test LPS-4 Non-verbal intelligence		
N		89
Normal Parameters[a,b]	Mean	114,8539
	Std. Deviation	11,53113
Most Extreme Differences	Absolute	,088
	Positive	,057
	Negative	-,088
Test Statistic		,088
Asymp. Sig. (2-tailed)		,085[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

Pre-test TMT-A		
N		89
Normal Parameters[a,b]	Mean	51,6854
	Std. Deviation	28,01120
Most Extreme Differences	Absolute	,170
	Positive	,118
	Negative	-,170
Test Statistic		,170
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

Post-test TMT-A		
N		89
Normal Parameters[a,b]	Mean	56,2921
	Std. Deviation	28,81690
Most Extreme Differences	Absolute	,188
	Positive	,121
	Negative	-,188
Test Statistic		,188
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

Pre-test TMT-B		
N		89
Normal Parameters[a,b]	Mean	48,3146
	Std. Deviation	30,19768
Most Extreme Differences	Absolute	,167
	Positive	,167
	Negative	-,123
Test Statistic		,167
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

Post-test TMT-B		
N		89
Normal Parameters[a,b]	Mean	54,7191
	Std. Deviation	32,08971
Most Extreme Differences	Absolute	,178
	Positive	,154
	Negative	-,178
Test Statistic		,178
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction

Mirror reading priming pre-test		
N		87
Normal Parameters[a,b]	Mean	194,3678
	Std. Deviation	159,96279
Most Extreme Differences	Absolute	,188
	Positive	,149
	Negative	-,188
Test Statistic		,188
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction

Mirror reading procedural pre-test		
N		87
Normal Parameters[a,b]	Mean	213,6322
	Std. Deviation	178,17571
Most Extreme Differences	Absolute	,190
	Positive	,170
	Negative	-,190
Test Statistic		,190
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

Mirror reading interference pre-test		
N		87
Normal Parameters[a,b]	Mean	176,4023
	Std. Deviation	117,38799
Most Extreme Differences	Absolute	,132
	Positive	,117
	Negative	-,132
Test Statistic		,132
Asymp. Sig. (2-tailed)		,001[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

Mirror reading priming post-test		
N		87
Normal Parameters[a,b]	Mean	156,0345
	Std. Deviation	135,62563
Most Extreme Differences	Absolute	,203
	Positive	,203
	Negative	-,194
Test Statistic		,203
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

Mirror reading procedural post-test		
N		87
Normal Parameters[a,b]	Mean	165,5747
	Std. Deviation	119,27198
Most Extreme Differences	Absolute	,205
	Positive	,205
	Negative	-,151
Test Statistic		,205
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

Mirror reading interference post-test		
N		87
Normal Parameters[a,b]	Mean	124,5402
	Std. Deviation	76,51004
Most Extreme Differences	Absolute	,181
	Positive	,181
	Negative	-,136
Test Statistic		,181
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

VLMT pretest short-term (Immediate recall)		
N		89
Normal Parameters[a,b]	Mean	61,4213
	Std. Deviation	27,92542
Most Extreme Differences	Absolute	,178
	Positive	,141
	Negative	-,178
Test Statistic		,178
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

VLMT post-test short-term (Immediate recall)		
N		89
Normal Parameters[a,b]	Mean	60,3708
	Std. Deviation	27,31805
Most Extreme Differences	Absolute	,184
	Positive	,136
	Negative	-,184
Test Statistic		,184
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

VLMT pre-test after learning		
N		89
Normal Parameters[a,b]	Mean	38,8034
	Std. Deviation	23,26058
Most Extreme Differences	Absolute	,145
	Positive	,145
	Negative	-,124
Test Statistic		,145
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

VLMT post-test after learning		
N		89
Normal Parameters[a,b]	Mean	42,9888
	Std. Deviation	26,27196
Most Extreme Differences	Absolute	,127
	Positive	,127
	Negative	-,112
Test Statistic		,127
Asymp. Sig. (2-tailed)		,001[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

b.

VLMT pretest after interference		
N		89
Normal Parameters[a,b]	Mean	42,8034
	Std. Deviation	27,87804
Most Extreme Differences	Absolute	,139
	Positive	,126
	Negative	-,139
Test Statistic		,139
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

VLMT post-test after interference		
N		89
Normal Parameters[a,b]	Mean	42,2472
	Std. Deviation	31,33907
Most Extreme Differences	Absolute	,174
	Positive	,174
	Negative	-,111
Test Statistic		,174
Asymp. Sig. (2-tailed)		,000[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

VLMT pretest delayed recall		
N		89
Normal Parameters[a,b]	Mean	38,6629
	Std. Deviation	27,75653
Most Extreme Differences	Absolute	,128
	Positive	,128
	Negative	-,106
Test Statistic		,128
Asymp. Sig. (2-tailed)		,001[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

VLMT post-test delayed recall		
N		89
Normal Parameters[a,b]	Mean	39,1517
	Std. Deviation	30,44634
Most Extreme Differences	Absolute	,122
	Positive	,122
	Negative	-,118
Test Statistic		,122
Asymp. Sig. (2-tailed)		,002[c]

a. Test distribution is Normal. b. Calculated from data. c. Lilliefors Significance Correction.

Appendix D

Questionnaire

Fragebogen

VP- CODE: - - - - - - - - -

A) Name:
1. Geschlecht: Weiblich: O Männlich: O
2. Händigkeit: Rechts: O Links: O

3. Geburtsdatum:

4. Familienstand: ledig: O verheiratet/ mit festen Partner: O
Geschieden: O verwitwet: O

5. Gewicht: kg 6. Körpergröße: cm

BMI:

B) Schulbildung:
Hauptschule: O Realschule: O Gymnasium: O Andere Schulart :
Schulabschluss:

Berufsausbildung:

Berufsschule: Lehre: Studium:

Derzeitige Berufstätigkeit: frühere Berufstätigkeit:

C)
Rauchen: Ja: O Nein: O wie viele Zigaretten am Tag:

seit wann:

Alkohol: nie: O selten: O regelmäßig: O

seit wann:

D) Aktivitäten:

Machen Sie regelmäßig Sport: Ja: O Nein: O

wie oft in der Woche: . . . .

1. Weniger als 1 Stunde leicht: O mittel: O schwer: O
2. 1-3 Stunden leicht: O mittel: O schwer: O
3. 3-5 Stunden leicht: O mittel: O schwer: O
4. Mehr als 5 Stunden leicht: O mittel: O schwer: O

Betreiben Sie einen Individualsport: O (wie Schwimmen, Laufen, . . .)

Betreiben Sie einen Zwei-Personensport: O (wie Badminton, Tennis, . . .)

Betreiben Sie einen Kampfsport: O (wie Ringen, Karate, . . .)

Betreiben Sie einen Mannschaftssport: O (wie Fußball, Volleyball, usw.)

-Haben Sie Sportarten selbstständig (autodidaktisch) gelernt oder haben Sie bei einem Lehrer/ Trainer gelernt?

selbst : O bei Trainer: O

-Wenn autodidaktisch, schätzen Sie sich selber als:

schlecht: O mäßig: O gut: O sehr gut: O

In dieser Sport ein?

Möchten Sie neue Sportarten lernen? Ja: O Nein: O

Möchten Sie geistige Fertigkeiten (z. B Schach) und/oder Feinmotorische lernen (z.B. das Kugellabyrint und der Münzentrick)? Ja: O Nein: O

Appendix E

Declaration of consent

Prof. Dr. Hans J. Markowitsch
Gholam Reza Tazkari M.A.

Bielefeld, September 2011

EINVERSTÄNDNISERKLÄRUNG

zur Teilnahme an der Studie

Körperliche Aktivierung und Gedächtnis

Hiermit erkläre ich, Frau/Herr . . . . . , mich bereit, an den neuropsychologischen Untersuchungen sowie einem Training von Hirnfunktionen in der Virtuellen Realität im Rahmen der Studie

„Körperliche Aktivierung und Gedächtnis“

an der Fakultät für Psychologie und Sportwissenschaft der Universität Bielefeld teilzunehmen. Ich bin mündlich und schriftlich umfassend über Inhalt, Zweck und Umfang der Untersuchung informiert worden und habe keine weiteren Fragen zu den Untersuchungen. Die Teilnahme an der Studie ist **freiwillig,** und ich kann die Untersuchungen **jederzeit** ohne Angabe von Gründen abbrechen, ohne dass mir dadurch irgendwelche Nachteile entstehen.

Die Studienleiter und Projektmitarbeiter verpflichten sich, die eingesehenen und erhobenen Daten **anonym** und **streng vertraulich** zu behandeln und unterliegen diesbezüglich der Schweigepflicht. Ich bin einverstanden, dass die Ergebnisse meiner Untersuchung von der Fakultät für Psychologie und Sportwissenschaft der Universität Bielefeld sowie von den an der Studie beteiligten Kliniken in **anonymisierter** Form für wissenschaftliche Zwecke verwendet werden.

_____	_____
Ort, Datum	Unterschrift (Studienteilnehmer/in)
_____	_____
Unterschrift zuständiger Projektmitarbeiter	Unterschrift Studienleiter

Academic Thesis:

Declaration of Authorship

Hereby, I declare that the dissertation presented in this thesis entitled

Physical Activity, Aging and Cognition

A Study of Effects of Physical Exercise on Cognitive Functions of Older Adults

is my own work. None but the cited methods and materials were used. This work has not been submitted in this or another form at any other university or faculty.

Signature: *Gholam Reza Tazkari*

Date: *Germany, Bielefeld, February 2015*