

8-2014

Toward a More Perfect Definition of Learning: Using Biomarkers to Predict and Assess Learning Performance

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TOWARD A MORE PERFECT DEFINITION OF LEARNING: USING
BIOMARKERS TO PREDICT AND ASSESS LEARNING PERFORMANCE

A Dissertation
Presented to
The Faculty of the Educational Leadership Doctoral Program
Western Kentucky University
Bowling Green, KY

In Partial Fulfillment
Of the Requirements for the Degree
Doctor of Education

By
Samuel J. Hunt

August 2014

TOWARD A MORE PERFECT DEFINITION OF LEARNING: USING
BIOMARKERS TO PREDICT AND ASSESS LEARNING PERFORMANCE

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I would like to dedicate this dissertation to all those who have supported me in ways I may not even know while a graduate student at WKU, especially my committee: Barbara Burch, Jie Zhang, and Cheryl Davis; my friends, Joseph Cangemi and James Navalta; and many others.

I also would like to dedicate this to my wife Isis, who, during our first year of marriage, gave constant encouragement to achieve this milestone.

Foremost, I would like to dedicate this to my parents, without whose love and encouragement I would not find myself here today.

ACKNOWLEDGEMENTS

I would like to thank Ellen Lightfoot, who, on two occasions, assisted with the expert advisement and practice during the blood collection of the research. I also would like to thank Graves Gilbert Clinic for their support of the research by providing materials and a phlebotomist, without whose support this research would have been financially prohibitive. I also would like to thank GM Alexandra Kosteniuk, who donated chess training resources from her Chess King software collection.

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August 2014

138 Pages

Directed by: Barbara Burch, Cheryl Davis, Jie Zhang

Educational Leadership Doctoral Program

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This study seeks to establish groundwork for a new definition of learning based on neurogenesis capable of guiding future educational policy and practice. The purpose of the research was to: (1) produce separate increases in neurogenesis and intelligence, (2) measure the changes in neurogenesis using protein biomarkers, and (3) correlate increases in levels of the protein biomarkers with increases in intelligence. The study employed a randomized pretest-posttest, control/comparison group research design. Thirty-eight fourth- and fifth-grade students with diverse academic needs were divided into three experimental groups: chess, exercise, and combined; with an additional control group. Pre-post measures included intelligence (RSPM) and two serum proteins (BDNF) and (VEGF). Multiple one-way ANOVAs between the groups with post-hoc Bonferroni pairwise correction discovered significant differences on post-IQ scores (1) between chess and control; (2) between those groups that received chess treatment and those that did not; and (3) between those groups that received chess and/or exercise treatment versus control. Paired sample t-tests found the exercise group and the combined group significantly increased BDNF pre-post. A Pearson Product Moment correlation revealed that the control group had the only significant post-test correlation between RSPM and BDNF ($p = .049$). Chess and exercise treatment led to increases in intelligence and biomarker levels associated with neurogenesis, as evidenced by increased RSPM and BDNF measures.

The results of this research suggest that a novel process whereby protein biomarkers such as BDNF and VEGF may be useful as a potential measure of neurogenesis in young children. This research successfully produced increases in protein biomarkers in an attempt to correlate neurogenesis to intelligence in human subjects. Exercise treatment initiated increases in protein biomarkers, while chess treatment increased intelligence. Both chess and exercise treatment may be beneficial to increase efficiency of neural networks associated with intelligence in a school-age population.

CHAPTER I: INTRODUCTION

"The issue is not whether to have pullouts or in-class models, but how to develop and test new metaphors about how to stimulate new learning." -- Stanley Pogrow, 1988

The goal of this study is to lay a foundation for the future development of a more accurate definition of learning based on a biological process, neurogenesis, which is capable of guiding future educational policy and practice. To justify this, research will be conducted in an attempt to cause an increase in neurogenesis in an elementary school-age population. This research will measure neurogenesis in humans by testing for two proteins in human blood, both directly and indirectly related to neurogenesis, Brain-derived neurotrophic factor (BDNF), and Vascular endothelial growth factor (VEGF). The treatments chosen for inducing an increase in neurogenesis are chess and exercise. The general hypothesis is that an increase in neurogenesis will cause an increase in cognitive ability (intelligence), which leads to a predictable effect on learning performance, as would be evidenced by improved academic performance.¹ The initial theoretical foundation for the study was proposed by Hunt and Navalta (2012).

This research attempts to provide an experimental basis for the theory that increased neurogenesis affects the outcome of *learning* performance by loosening the biological constraints that inhibit the acquisition of skill, knowledge, or expertise (Hills & Hertwig, 2011). The linear theorized process is:

Physical exercise → Neurogenesis → Intelligence increase → Learning performance

According to the theoretical foundation of this research, inducing the process of neurogenesis should cause the outcome of improved learning performance. Such a cause-

¹ Measuring academic performance, as an effect of learning performance, is beyond the scope of this study.

effect relationship between the combination of mental/physical activity and neurogenesis may lead to a more accurate definition of learning based on neurogenesis that can be assessable by a simple blood test. Such a blood test could be used in conjunction with currently accepted scholastic assessment to measure (1) learning, as newly defined; and (2) content specialization, that should result in a holistic measure of learning performance.

Background and Problem Statement

Since the late 1990s, a growing interest can be seen in attempting to find a more accurate and objective definition of learning. Lachmann (1997) described an accepted definition of learning as being "a relatively permanent change in behavior as a result of practice or experience" (p. 477). However, White (1996) and Christina (1997) noted that an accurate definition of learning in the 1990s remained elusive; and Illeris (2003) wrote that learning remained undefined. According to Daniel and Poole (2009), an accurate, objective definition of learning and the process of achieving it; promoting it; and discovering why, in some cases, it does not occur remained ambiguous and elusive. Joyce and Well (1999) reported the existence of at least 80 different teaching-learning models prior to the new millennium. Daniel and Poole stated that the number of models was increasing as technology advanced and added, "It is clear that the sheer complexity of the environments we are creating for students may pose a serious threat to their motivation to engage in learning" (p. 94).

Illeris (2003) posed four questions surrounding this topic: "(1) What is learning?; (2) How does it come about?; (3) How can it be promoted?; and (4) Why does teaching not always result in learning?" (p. 397). These four questions can be simplified into two

key targets to solve the current crises of the outdated learning paradigm: (a) the development of a more accurate definition of learning, and (b) the description of a process of cognitive enhancement applicable to various populations. In a news report for *Forbes* magazine, Denning (2013) trumpets the age-old line, "A Nation Still at Risk: How We Can Fix Our Schools."

Purpose and Theoretical Base for the Research

The purpose of this study is to define learning more accurately based on the biological process of neurogenesis. The process by which to arrive at that conclusion is to first attempt to induce the process of human neurogenesis and to then accurately assess that it has occurred. First, in order to cause an increase in neurogenesis, the treatments of chess, exercise (Tabata et al., 1997; Tabata et al., 1996), and combined (chess + exercise) will be added to a normal daily scholastic routine. Along with the control group, these treatments represent the independent variables of this investigation. The Raven's Standard Progressive Matrices (RSPM) will be used to measure intelligence, and a blood test will be used to accurately measure levels of two proteins (BDNF and VEGF) associated with neurogenesis. The RSPM and the two blood proteins represent the dependent variables in this investigation. Measures will be taken both pre- and post-treatment to document the degree to which any changes may occur. The treatment period will be nine weeks (45 days) in length. This study will employ a randomized pretest-posttest control/comparison group research design using a population to include a wide spectrum of student abilities (e.g., gifted/talented, normal, at-risk, special needs) from a school in southcentral Kentucky.

The theoretical basis for this study rests on two well-documented methods of

physical and mental enhancement that lead to improved cognition in children: regular bouts of aerobic exercise (Tomporowski, Davis, Miller, & Naglieri, 2008) and playing chess (Frank & D'Hondt, 1979). Additionally, Fabre, Chamari, Mucci, Massé-Biron, and Préfaut (2002) and Gomez-Pinilla, So, and Kesslak (1998) found that subjects in combined treatments (mental + physical) outperformed the mental training only and aerobic training only groups. Exercise and chess offer an experimental basis for using these two treatments in an attempt to cause an increase in neurogenesis, which will be reviewed in Chapter II.

Research Questions and Hypotheses

In order to arrive at the conclusion that increased neurogenesis has been caused by mental (chess) and physical exercise the first research question (Q_1) is, can chess and exercise each produce an increase in measures of intelligence? The following hypotheses guide the answer to this question:

1. H_1 : Exercise intervention will produce an increased effect on cognitive measures of intelligence to a larger degree than the control group.
2. H_2 : Chess intervention will produce an increased effect on cognitive measures of intelligence to a larger degree than the control group.

The second research question (Q_2) relates to the effect of the combined (chess + exercise) treatment: Can a combined treatment produce an added increase in measures of intelligence to a larger degree than chess only and exercise only? The following hypotheses guide the answer to this question:

3. H_3 : The combined group will produce an added effect on cognitive measures to a larger degree than the exercise only group.

4. *H₄*: The combined group will produce an added effect on cognitive measures to a larger degree than the chess only group.

The third research question (Q₃) relates to the effect of treatments on neurogenesis: Can chess and exercise each produce an increase in neurogenesis? This question will be illuminated by determining the effect of chess and exercise on both BDNF and VEGF protein levels separately in the blood stream. The following hypotheses guide the answer to this question:

5. *H₅*: Exercise treatment will produce an increased effect on BDNF levels to a larger degree than the control group.
6. *H₆*: Exercise treatment will produce an increased effect on VEGF levels to a larger degree than the control group.
7. *H₇*: Chess treatment will produce an increased effect on BDNF levels to a larger degree than the control group.
8. *H₈*: Chess intervention will produce an increased effect on VEGF levels to a larger degree than the control group.

The final question attempts to demonstrate a correlation between cognitive measures and BDNF protein levels: Can BDNF levels be associated with an increase in cognitive measures? The following hypothesis guides the answer to this question:

9. *H₉*: Increases in BDNF will be correlated with increases in cognitive measures.

The combination of these hypotheses aid in determining whether intelligence increases are parallel to neurogenesis increases. If these two increases can be shown to have a correlational relationship, then this new knowledge may lay the foundation for a more

accurate definition of learning and its assessment.

Significance of the Study

This study represents a broad interdisciplinary investigation linking research from the fields of education, neuroscience, psychology, exercise science, and educational neuroscience. As such, this study may have significance to each of them in many ways, and in conjunction to one another.

Education. This study may affect social change contributing to curricular revisions by demonstrating a method of daily classroom exercise and chess enrichment that will not disrupt normal academic progress and could lead to improved student learning performance. Also, the findings may aid in the development of a more accurate assessment in the field of education, not only of content specialization, but of actual learning over time, by associating learning to increases in neurogenesis.

Neuroscience. This study also adds to the field of neuroscience, as it is conducted with children in attempt to measure neurogenesis in humans and helps to fill this gap in the neuroscience literature. Three broad reviews on neurogenesis demonstrate the surfeit amount of research that exists on neurogenesis in mammals and the present inference and correlations to assumed process equivalents in humans (Deng, Aimone, & Gage, 2010; Ming & Song, 2011; Zhao, Deng, & Gage, 2008). Pereira et al. (2007) demonstrated groundbreaking research in discovering a mouse-human correlate to measuring neurogenesis in humans based on studies on mice. Those researchers demonstrated the causal links between brain blood flow and neurogenesis, which could be imaged using MRI. The current study adds to this research base by attempting to demonstrate increases in neurogenesis using a far more cost-effective correlate, vascular endothelial growth

factor (VEGF) and brain-derived neurotrophic factor (BDNF). VEGF specifically triggers neovascularization (Carmeliet, 2003) and, according to Jin et al. (2002), is implicated in neurogenesis as well. Cheng, Wang, Cai, Rao, and Mattson (2003) found that BDNF specifically triggers the process of neurogenesis by switching stem cells from proliferation to differentiation into neurons. Both VEGF and BDNF are known to circulate in the bloodstream, which may allow for future testing of neurogenesis *in vivo* by a simple blood draw and analysis of the presence of the two proteins.

Psychology. A number of investigations have been designed to study the perceived association between intelligence and chess. Four of those have shown a positive association between chess and intelligence (Aciego, Garcia, & Betancourt, 2012; Frydman & Lynn, 1992; Hong & Bart, 2007; Horgan & Morgan, 1990). In contrast, Bilalić, McLeod, and Gobet (2007) demonstrated the lack of a relationship between intelligence and highly experienced, expert youth and adult chess players. The current study may illuminate a key to this dichotomy, as this investigation represents the first cause-effect experimental study using chess in an attempt to increase both intelligence and neurogenesis. Chess also has been used as a treatment to improve learning performance, as evidenced by academic outcomes on a population of children with learning disabilities (Scholz et al., 2008). The population chosen for the current study is a mix of students, some with learning disabilities. Thus, it also may illuminate whether chess can provide a therapeutic effect for learners with disabilities and additive effects to increase intelligence in those who do not.

Exercise Science. This study can add to the literature in the field of exercise science related to physical activity and exercise to intelligence and BDNF. In finding a

neurogenesis measurement correlate *in vivo* between mice and human studies, Pereira et al. (2007) used the practice of exercise to induce neurogenesis. Dishman et al. (2006) published an article that established the foundation for a new branch of investigation within the field of exercise science called the “Neurobiology of Exercise.” This brain science branch specifically focuses on the effect of physical activity on the mental health and cognition of humans. In that perspective, they stated that a surprisingly minimal discourse and investigation exists in exercise science on how physical activity and/or inactivity play a role in brain health. A topic focus of research in this discipline is the manner in which physical activity affects the neurotrophic factors of BDNF and VEGF. The current study will add to this new branch within the field of exercise science, as it will describe the neurotrophic effects of exercise on BDNF and VEGF levels in children.

Educational Neuroscience. With the influence of the previously mentioned research fields, the primary location in which the current interdisciplinary investigation would flourish is in the growing field of educational neuroscience. Byrnes and Fox (1998) suggested that delineations between cognitive psychology, educational psychology, and emerging brain sciences were blurred and neither cognitive psychology nor educational psychology would last unless they merged. Fifteen years later, the field of educational neuroscience was born due to technological advances in imaging and neuroscience, along with the sophistication of cognitive science research.

Stern, Grabner, Schumacher, Neuper, and Saalbach (2005) came to the conclusion that foundations should be laid for the collaboration of neuroscience and education, although they renounced that the near-term brain research offered little practicability for education. The current investigation represents the confluence of these previously

divergent fields and converges them into an attempt to amalgamate the traditional definition of learning to a more accurate definition of learning within this interdisciplinary field. This study contributes to the field of educational neuroscience, as it bridges the gap between education, neuroscience, psychology, and exercise science on the commonly, but separately investigated, topic of brain research. It further distills that interdisciplinary topic into a single investigation with practical application in a school setting in an attempt to affect learning performance.

Methodology

This study will use a randomized pretest-posttest control/comparison group research design. Subjects include a class of fourth- and fifth-grade students from a southern Kentucky elementary school. Subjects will be divided into a control group and three treatment groups: chess (chess only), exercise (exercise only), and combined (chess + exercise). Pre-post cognitive measures will be taken using the RSPM and determining percentile ranks. Those scores will be converted into IQ equivalents. Pre-post blood draws will be administered by a certified phlebotomist to collect the amount of blood necessary to analyze for the two proteins, BDNF and VEGF. The treatment period is designed for a period of nine weeks, or 45 school days. Chess treatment will be administered in a group setting by the same teacher each day using online chess lessons and DVD lessons created by chess experts. The chess period is designed to be approximately 40 minutes in length. The exercise protocol will be administered by a second teacher each day for the duration of the study. The exercise period will use online videos in a group setting to lead the students through the correct movements is designed to be for 15 minutes, including a warm-up and cool-down period. Both treatment periods

will be conducted at the same time of day from approximately 1:30-2:30 pm.

Operational Definitions

Several important terms are used in this investigation:

- *Physical exercise*: The increased activity of both the brain and body
- *Neurogenesis* (traditional): The birth, migration, and integration of neurons in the human brain (Aimone, Jessberger, & Gage, 2007)
- *Neurogenesis* (conceptualized for the field of education): The birth, migration, and integration of neurons in the human brain and their resulting plasticity and specialization as effects of potentiation that achieves functional capacity, leading to increases in intelligence and cognitive performance
- *Learning* (traditional): A relatively permanent change in behavior as a result of practice or experience (Lachmann, 1997)
- *Learning* (redefined): The intrinsic six-stage process that moves adult-born neuron(s) from proliferation to summation (neurogenesis), as induced by external factors and leading to increased intelligence and cognitive performance

Assumptions, Limitations, and Delimitations

This study endeavors to demonstrate increases in neurogenesis and intelligence in a young school-age population. However, a few assumptions need to be made in order to test the hypotheses. Additionally, some limitations and delimitations guide this research.

Assumptions. The objective of this study centers around the determination of a more accurate definition of learning based on the process of neurogenesis. The main assumption of the research is that neurogenesis can be increased in humans if BDNF

and/or VEGF are increased in the blood stream due to treatment. This is an assumption based on the literature, but cannot be confirmed in this study without the use of some type of brain imaging. Thus, in this study, neurogenesis is assumed to have occurred if the treatment is able to increase levels of the proteins pre-post. Another assumption is that intelligence can be increased by the treatment of chess in a young population of non-chess players. Many studies in the literature investigate the increase of neurogenesis using exercise and a water maze as treatments for mice and rats. However, studies on neurogenesis in humans remain limited, and studies on children and neurogenesis are almost nonexistent.

Limitations. Deary, Penke, and Johnson (2010) revealed that, based on brain imaging studies, genetics play a role in intelligence throughout the lifespan, of which this investigation has no means to control. Other limitations are present for this research. In nearly all chess studies, a chess expert was the instructor providing chess training. In this investigation, the scholastic teacher is the instructor whose ability is undefined, and online and DVD chess lessons in a group setting are being provided. Although the lessons are created by chess experts, age appropriateness and group setting may become an issue, as individual and expert attention are unavailable to clarify questions and maintain rigor. Another limitation may be the sample size of the groups and the length of the research. The contact time for chess lessons is equivalent; however, most chess research is conducted one hour per week over several months to a year. This study pioneers a daily regimen to examine results, although duration may become a factor.

Delimitations. A delimitation of the study is the decision to use a blood test for BDNF and VEGF proteins as biomarkers for neurogenesis. This method was chosen

because most research in the area of neuroscience investigation used rats and other mammals whose brains can be dissected, sliced, and stained in order to demonstrate increases in neurogenesis. These two biomarkers may be measured non-invasively to predict increases in neurogenesis. Another delimitation is the use of DVD and online chess lessons from experts in a group setting to standardize the chess instruction for all subjects. The exercise instruction will be conducted using videos from fitness leaders in a group setting to standardize the instruction for all subjects.

Another delimitation is the use of the RSPM as the most accurate measure of intelligence (Silverman, 2009). The RSPM is a test of non-verbal intelligence based on the work of Spearman (1927) and directly measures the two main components of Spearman's *g*: eductive ability and reproductive ability; i.e., the cognitive ability to form relationships of, and derive meaning from, complex patterns and/or information and the ability to recall information and reuse it in new and creative ways. Silverman (2009) indicated the RSPM to be the purest form of measuring Spearman's *g*, and wrote, "It also measures cognition of figural relations, spatial ability, and accuracy of discrimination, reasoning by analogy, logical relations, and inference" (p. 948).

Summary

This chapter established the foundation for the investigation of learning, as defined by neurogenesis, by describing the nature and purpose of the investigation, its scope and limitations, as well as operational definitions. The process utilized in this investigation will arrive at a more accurate understanding of human learning to determine whether a cause-effect relationship exists between physical exercise, neurogenesis, and intelligence, and a correlation between neurogenesis and cognitive measures and/or

outcomes. Chapter II will discuss literature from the fields of neuroscience, education, exercise science, and psychology, beginning with an introduction to the field of educational neuroscience.

CHAPTER II: LITERATURE REVIEW

“The goal of bringing the neuroscience of learning to in-service teachers provides a new perspective on instruction, one where teachers come to see themselves as designers of experiences that ultimately change students’ brains.” --- Dubinsky, Roehrig, & Varma, 2013

This chapter presents the theoretical foundation for the investigation of neurogenesis in a school-age population. This review begins with an introduction to the field of educational neuroscience and demonstrates two major themes relevant to this study: (1) the connection between neurogenesis and intelligence in humans; and (2) how chess and exercise relate to increases in BDNF and/or VEGF, intelligence, and learning performance. The chapter also includes a review of the Tabata protocol, the chosen exercise treatment method.

Hunt and Navalta (2012) laid the groundwork for this research, as they linked together the biological cascades and substrates that are directly responsible for initiating neurogenesis. That review specifically centers around the chemical molecule of nitric oxide (NO) and its influence on human physiology and brain morphology. They described how NO interacts with both VEGF and BDNF in their respective functions of neovascularization and neurogenesis. Their review parallels these processes to literature on how exercise, chess, and nutrition each lead to increases in learning and academic ability. The authors theorized that the summation of neural plasticity, as a result of neurogenesis, is the observable process of learning. They represented this process of learning by introducing the “learning curve” (p. 264). This current study is designed to test their theory and to add an experimental foundation, along with the theoretical

foundation, to justify development of a more accurate definition of learning based on the process of neurogenesis.

Introduction to Educational Neuroscience

The debate relative to whether neuroscience in any capacity fits in education, and vice versa, began in the late 1990s (Bruer, 1997) and continues today (Tommerdahl, 2010). Bruer coined the debate as the “education and neuroscience argument.”

Bruer (1997) cautioned researchers about using neuroscience inference in education applications, as the scientific basis for it was extremely limited. The problem was that many education researchers and authors tended to over-simplify neuroscience findings on animal studies and misrepresent them, perpetuating myths that had no research justification to human correlates. According to Bruer, neuroscience findings at that time had little to offer education in the form of instructional practice. However, although he felt education and neuroscience were a “bridge too far,” Bruer offered a more relevant strategy through psychology. He postulated that two relevant bridges already existed to span the gap between brain and education sciences: education → cognitive psychology, and cognitive psychology → cognitive neuroscience. Bruer claimed that cognitive psychology is the basic science of learning, not necessarily concerned with the brain, but with the mind and mental function. Conversely, cognitive neuroscience uses imaging techniques to discover brain activity, which is then related to functions that guide human behavior. Such bridges fit well within the traditional definition of learning (Lachmann, 1997). Over time, as technology and research design have advanced, this bridge too far has become an achievable span.

Geake and Cooper (2003) suggested that a cognitive neuroscience bridge to education is most firmly grounded in the Hebbian Theory of neuronal plasticity and how morphology relates to observed behavioral outcomes. The authors listed the areas of experimental interest within cognitive neuroscience to include: vision, spatial cognition, audition and music, emotions, imitation, memory, motor function, language, and consciousness. They noted that all of these have some implication to learning and memory. However, the brush with which they painted this picture of cognitive neuroscience makes the field so broad in interest to be nearly undefined in approach. Perhaps such dilution is the reason educational policymakers are reluctant to accept such marginalized generalizations to education practice and policy.

Geake (2005) introduced a stark dichotomy: “There exists no mention of schools and classrooms in cognitive neuroscience research, and there exists no mention of brain science in educational policy, curriculum, or assessment” (p. 10). This is a strange division, since no learning takes place without neuronal plasticity, according to Hebbian theory and rules (Hebb, 2005). Geake suggested that the common link between cognitive neuroscience and education is understanding how the brain learns. However, cognitive neuroscience is not the only prospective field from which this bridge may be crossed.

Subsequently, this bridge has evolved into the field of mind, brain, and education, or more specifically, educational neuroscience (Tommerdahl, 2010). The field not only needs to concern itself with how the brain learns, but how pre-service teachers learn how the brain learns (Dubinsky et al., 2013), with a focus on classroom applications (Fischer, Goswami, & Geake, 2010) that result in enhanced learning performance evidenced by increased intelligence and academic outcomes. The perspective that is lacking in the

education and neuroscience argument is that a brain-based model, to date, cannot inform educators *what content* the student learned as a result of neuronal morphology, even if education fully adopts a new definition of learning tied to neurogenesis and/or brain plasticity. Thus, it would seem that, in order to receive full adoption into the field of education, research should work in tandem/parallel with current standardized educational assessments that measure content specialization (Figure 1).

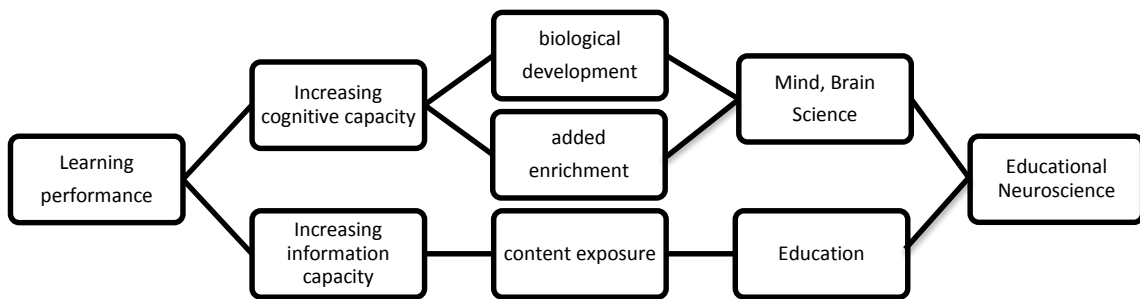


Figure 1. The bridge too far spanned by common goal of learning performance.

Learning involves two factors: increasing both cognitive and informational capacity. Increasing informational capacity through adequate content exposure is the role of the field of education; increasing cognitive capacity by normal biological development and added enrichment is the role of mind/brain science research. To create behavioral change, neither process can act in isolation of the other. Educational neuroscience is the bridge between these two roles that can give full illumination to answering Illeris’s (2003) postulates.

Therefore, it is introduced within the education and neuroscience argument that a linear theory for the investigation of this bridge between biology and learning outcomes can be generalized as:

Physical activity → neurogenesis → intelligence → learning performance

No matter the educational topic under investigation - whether the reading brain, the linguistic brain, the math brain, etc. - the physical exercise of the mind and/or body can be predicted to increase neuronal morphology (i.e., neurogenesis, synaptogenesis, long-term potentiation, etc.). As a result of Hebbian kinetics, it can be predicted to increase intelligence, which can be predicted to increase learning performance in the classroom. Such a theory can be investigated in pieces or as a whole and allows for direct classroom investigations of neuroscience topics that can inform future educational policy.

Ansari (2008) and Tommerdahl (2010) suggested that direct applications are unlikely from neuroscience into the classroom. Ansari attempted to substantiate this by stating that no such direct applications exist from basic research in any other fields. However, the design of the current study has overturned such conjectures by initiating a line of research in the field of educational neuroscience that offers a wide gate for future research with direct applications into classroom practices.

The Connection Between Neurogenesis and Increased Intelligence in Humans

It is now well accepted in the neurosciences that neurogenesis occurs throughout the lifespan (Eriksson et al., 1998; Curtis, Kam, & Faull, 2011; Ming & Song, 2005; Spalding et al., 2013). It appears that the only significant area of neurogenesis known to occur in humans to date is in the hippocampal region of the postnatal brain, an area whose function is commonly associated with learning and memory (Sierra, Encinas, & Maletic-Savatic, 2011). The logical conjecture can be made that the great correlation to learning in non-human mammal studies in association with neurogenesis also would benefit humans in the same way (Deng et al., 2010). However, according to the review by Sierra, Encinas, and Maletic-Savatic (2011), the literature on human neurogenesis is

limited, and much of the discussions on learning and neurogenesis are still extrapolated from studies on mice and rats.

With their knowledge of cognitive psychology, Neissar et al. (1996) explained that, as intelligence increases, learning (and memory) ability increases as well. Thus, it would seem logical that, in order to bridge the distance between brain science and education, one would begin by describing a neural basis for intelligence and once described, attempting to manipulate it and measure its changes from treatment. In order to bridge the span between neurogenesis and intelligence, the underlying factor is functionality. This section reviews literature related to the importance of functional neurogenesis as being important to intelligence and also attempts to describe from the literature the relevant timeline from proliferation of stem cells to functional and mature neurons that could relate to increases in intelligence and learning performance in humans.

Functional neurogenesis and its relation to intelligence. Of importance is the fact that a significant amount of neurogenesis occurs throughout the lifespan only in the hippocampus. If any other neurogenesis occurs in the brain post-perinatal, it is limited to beyond currently detectable methods (Bhardwaj et al., 2006). Some of the functional significance of neurogenesis in the hippocampus includes: memory functions (Lupien et al., 1998; Squire et al., 1992); pattern separation (Bakker, Kirwan, Miller, & Stark, 2008); spatial recognition (Darnaudéry, Koehl, Piazza, Le Moal, & Mayo, 2000); and new learning (Kuhl, Shah, DuBrow, & Wagner, 2010). Although neurogenesis has been directly linked to improved cognition (i.e., intelligence); learning; and memory within the hippocampus in rats (Cao et al., 2004), the relation of neurogenesis to intelligence in humans remains to be discovered. Ironically, although the hippocampus is the focal

region of adult neurogenesis research, and it is certainly part of the network of interrelated regions of the brain associated with intelligence, neuroscience investigation indicates it is not the focal region for intelligence.

Haier et al. (1988) began the search for a neural basis of intelligence using positron emission tomography (PET scan) to image areas of the brain with the greatest uptake of radiated glucose to show which clusters of neurons had the greatest activity during cognitive tasks. Their research used the Raven's Advanced Progressive Matrices (RAPM) as a test of intelligence for adult subjects. Their research compared two groups, the RAPM group and a control group, using a degraded cognitive task consisting of numerical recognition. The interesting objective was to attempt to image the regions of the brain with the highest activation during the intelligence test. In both groups, diffuse activation of various neural clusters occurs throughout parts of the brain. The results show an inverse correlation between region activation (as demonstrated by highest glucose uptake) and performance on the RAPM. According to the study, the more active regions that are found, the greater the association with difficulty of correctly completing the RAPM, i.e., the more difficulty of the task for young adults, the greater the diverse recruitment of neuron focal regions necessary to perform the task. The authors suggested that this indicates that those with low performance, who have the highest levels of neural activity, demonstrate greater inefficiency across the brain and, according to the scores on the RAPM, are less intelligent. This is counterintuitive, as one would expect those with greater activity to demonstrate greater "processing power" and greater intelligence. Surprisingly, this study demonstrates a negative correlation between brain activity and intelligence.

Haier, Siegel, Tang, Abel, and Buchsbaum (1992) followed this research with another PET study testing activation and RAPM, following a learning task of a newly launched video game called Tetris. The authors attempted to determine whether the high ability subjects would show the greatest decrease in activation. They also investigated whether learning affects the inverse relationship between brain activation and intelligence. This investigation used a pre-post control/comparison group research design. Results showed that those who practiced (vs. naïve) during the treatment period had the greatest reduction in activation and the highest intelligence test scores. Surprisingly, the strongest correlation between intelligence level and brain activation was in the naïve (unpracticed) group, indicating the higher the intelligence, the lower the correlation to clusters of neural activation. The authors concluded that general intelligence (Spearman's *g*) relates more to new learning than to task expertise. The researchers, both concluded that studies support the brain "efficiency hypothesis" of intelligence and that Spearman's *g*-factor is not located in any one focal brain region, but, rather, across the brain using interneural loops/templates from distinct brain regions.

Nichelli et al. (1994) conducted a separate investigation using the PET scanning technique to understand the neural networks underlying problem solving using a chess game task. Their results confirmed the studies by Haier et al. (1988) and Haier et al. (1992) and found that problem solving recruited functionally distinct regions of the brain during problem-solving activities.

Research by Duncan et al. (2000) confirmed this trend, also through PET scanning to attempt to discover a neural basis for Spearman's '*g*' using several high-*g* and low-*g* tasks. High-*g* tasks exhibited a problem-solving component, while low-*g* tasks

subtracted the component, although using similar materials. The high-g tasks recruited activation bilaterally from the prefrontal cortex. Additionally, low-g tasks were found to show considerable variation in recruitment of brain areas. The authors concluded that 'g' is more highly associated with the lateral frontal lobe region of the human brain than any other region, although other regions also may, to a lesser extent, be involved.

Amidzic, Riehl, Fehr, Wienbruch, and Elbert (2001) confirmed these studies another way using magnetic imaging of brains of grandmaster chess players versus amateur chess players. They found that recruitment of regions of increased activity are diffuse throughout the brain. However, the more experienced players, the grandmasters, exhibited more activation in the frontal/parietal cortices. Subsequently, in the areas in which the amateur players had the greatest activity, the grandmaster players did not, again confirming the efficiency hypothesis of Haier's investigations. Additionally, a strong negative correlation was found between the activity of these brain regions and level of chess expertise.

An important study by Shaw et al. (2006) tested these results using a population of subjects that were stratified by intelligence level, as measured by the Wechsler intelligence scales. The subjects ranged in age from child to adult. The premise of their study was that, if intelligence (IQ) was related to the frontal cortex, then those with the highest IQ should have the thickest cortical areas. Once divided by age group, a "learning curve" correlation was found that ranged from a strong negative correlation between IQ and frontal cortex thickness in childhood, to a positive correlation in late childhood, which tapered off in adolescent and adult groups. This study demonstrated confirmation of the efficiency hypothesis in the following way: the superior intelligence group has the

greatest increase in cortical thickness of prefrontal cortex (peaking at ~11 years old), but also has the most rapid thinning time of the groups (superior intelligence, high intelligence, average intelligence). For the superior intelligence group, rapid thinning begins in early adolescence; thinning begins in late childhood for the high intelligence group, and even earlier in the average intelligence group. The prediction can be made that groups of lower intelligence may experience cortical expansion or thinning to a minimal degree, remaining chronically underdeveloped/specialized throughout life. This is particularly important, as the frontal lobe is associated with higher order thinking and executive functions. These processes help to clearly define the neural efficiency hypotheses in relation to intelligence.

In relation to research by Bhardwaj et al. (2006), who suggested that no neocortical neurogenesis was detectable in adult humans, the results of the study by Shaw et al. (2006) appear to exhibit frontal lobe cortical plasticity to a “marked” degree in living subjects during the development period from child to adult. When the subjects reached adulthood, primarily static intelligence, such cortical thickness changes also became static. Of importance is the major limitation of Bhardwaj et al. in that the collected samples of brain tissue were from seven cases admitted for autopsy, deceased human adult specimens. Spalding et al. (2013) demonstrated that the adult human generates 1,400 new neurons per day in the hippocampal region of the brain. However, unless those neurons are functionally integrated, they die off. However, Spalding et al. posited that the die off of neurons is relative to age. Additionally, the greatest neurogenesis activity clearly is in the natal-perinatal development of children. One could predict this to be the greatest onset of intelligence change in the human lifespan.

Therefore, the question now becomes: Is neocortical, frontal lobe neurogenesis in humans tied to intelligence increases? Could it be that the static learning curve of the adult intelligence is not sufficient to increase neurogenesis into detectable ranges? Gould, Reeves, Graziano, and Gross (1999) found that in primates neurons are generated in the same regions as humans (the subventricular zone of the hippocampus) and that these new neurons migrated through the white matter into the neocortex areas of the frontal lobe. Could it be that the intelligence level of these primates under human care, in captivity, were developing similarly to a perinatal human child? The research by Shaw et al. (2006) appeared to show that prefrontal cortical plasticity to a great degree is dependent upon age and intelligence levels. They concluded that the level of intelligence is related to prefrontal cortical plasticity, and the group labeled superior intelligence experience the most rapid changes, leading to presumably the greatest neural efficiency at the youngest age.

Subsequent to the research by Haier et al. (1988), more recent studies have reached the same conclusion about the efficiency hypothesis and the potential links between functional neurogenesis and intelligence (Amat et al., 2008; Cole, Yarkoni, Repovš, Anticevic, & Braver, 2012; Lee, Wu, Yu, Wu, & Chen, 2012; van den Heuvel, Neubauer & Fink, 2009; Stam, Kahn, & Pol, 2009). Cole et al. (2012) showed the central mechanism for intelligence as being focalized in the left prefrontal cortex but using brain-wide connectivity. A clear understanding now exists of the neural basis of intelligence in humans; from the first PET study Haier et al. (1988) describing intelligence in the right/left hemispheres, to narrowing this understanding of the neural basis of intelligence to the bilateral frontal cortex by Cole et al. nearly 25 years later. Herein lies a foundation

to begin the engineering of neurogenesis in a way that can increase intelligence and learning performance at the prime age of development.

The brain efficiency hypothesis, relating functional neurogenesis and intelligence, has been observed independently using fMRI imaging of brains of master-level chess players versus novice-level chess players by Campitelli, Gobet, and Parker (2005). They showed both novice and master chess players contrasts between chess position/scenes to non-chess/random position/scenes. Their results revealed that the novices had the greatest activity in the bilateral frontal areas (related to intelligence) as well as posterior areas (visual processing) and the cerebellum (motor processing). No activation was shown during the contrasts in the master-level chess players, confirming the efficiency hypothesis.

These findings are particularly important to the field of education, as strategies can be developed to enrich student learning and increase intelligence. In sum, this section establishes that subjects with the greatest neural efficiency demonstrate the least neural activity, but achieve the highest scores on intelligence tests. These articles also verify that general intelligence testing is more related to new learning and dissociative at some level between naïveté and expertise. Further, a negative correlation exists between neural activity across the brain (in relation to the search for intelligence) and expertise.

The timeline for achieving functional neurogenesis. Sayyah (2009) established that correlations between BDNF plasma levels and IQ scores link neurogenesis to learning performance. The question becomes: How long does it take for functional neurogenesis to result in measurable increases in IQ and improve learning performance

outcomes? This section will highlight the work of Ming and Song (2005) and Deng et al. (2010), and attempt to expand their timeline to learning performance outcomes.

Two in-depth, complete reviews exist outlining the timeline for moving from differentiation to functional integration of a new neuron in the hippocampus (Deng et al., 2010; Ming & Song, 2005). BDNF is the catalyst that initiates the process of neurogenesis (Cheng et al., 2003). Eriksson et al. (1998) demonstrated that the division of progenitor cells and the differentiation of the surviving cells become neurons. Differentiation is the beginning of neurogenesis, wherein BDNF acts as the switching mechanism to move from proliferation of progenitor cells to differentiation of those cells. Marcucci, Paoletti, Jackowski, and Banchio (2010) described:

The sprouting of neurites, the growth of an axon, and the extension of neurite² trees are key morphological features characterizing neuronal differentiation. Neurite outgrowth is important for neuronal plasticity as well as for neuronal regeneration after injuries or neuropathological conditions. (p. 25382)

Ming and Song (2005) clearly outlined the five-stage timeline for neurogenesis from proliferation, as triggered by BDNF, to synaptic integration with mature neurons (Figure 2). Stage one to stage five is approximately 2-4 weeks in length. Deng et al. (2010) reiterated this process with newer information (Figure 3). According to their timeline, a fully mature, indistinguishable adult born neuron fully integrates with surrounding cells in about 2-4 months. Additionally, the suggestion is made here that three more timeline events exist before an outcome on a cognitive test may be realized: modeling of working memory (Del Giudice, Fusi, & Mattia, 2003); positive transfer

² A neurite is an immature neuron.

(Atherton, 2007); and summation³ of functional capacity and general intelligence (Conway, Kane, & Engle, 2003; Kempermann, Wiskott, & Gage, 2004). The Kempermann, Wiskott, and Gage model has three levels of neuronal function and integration with inference to a fourth: (1) cellular, (2) network, (3) system, and (4) individual. The researchers caution others to understand the functional significance of neurogenesis:

“Neuronal development is a lengthy process, a fact that must be considered when judging causes and consequences in experiments that address function and function-dependent regulation of adult neurogenesis.” (p. 186).

In sum, it is suggested here that the complete time course for developing neurogenesis from proliferation to summation is suggested to be:

1. Proliferation to differentiation: sprouting of neurites (~ 4 days)
2. Migration to synaptic integration (~2-4 weeks)
3. Maturation (~ 2-4 months)
4. Modelling of working memory (unknown time span)
5. Positive Transfer (unknown time span)
6. Summation: functional capacity and general intelligence (unknown time span)

³ A term created by this researcher to define complete adult-born neural morphology and integration at all levels, as described by Kempermann, Wiskott, and Gage (2004).

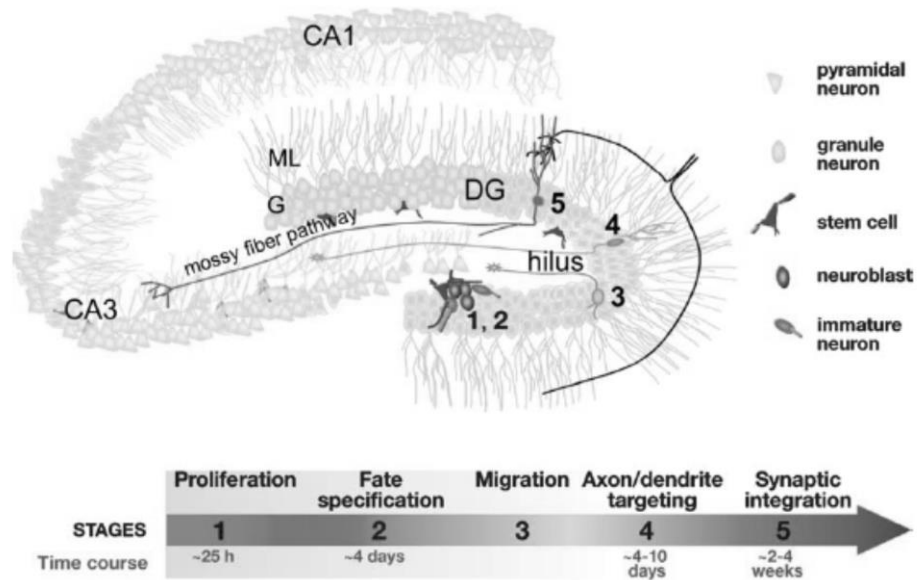


Figure 2. Five stage timeline for neurogenesis in the subventricular zone of the dentate gyrus (DG) in the hippocampus. Stage 1: Proliferation of stem cells in subgranular zone (G). Stage 2: Fate specification of cells into immature neurons. Stage 3: Migration of immature neuron into granule cell layer (G). Stage 4: Extension of axons into mossy fiber pathways of pyramidal cell layer of CA3 (a division of the hippocampus) and dendrites into the molecular layer (ML) of DG. Stage 5: Input is received (black) by new neurons and transferred to regions of hippocampus. Reprinted from “Adult neurogenesis in the mammalian central nervous system,” G. L. Ming and H. Song, 2005, *Annual Review of Neuroscience*, 28, p. 233.

The inference is that, upon completion of this time course, increases in neurogenesis will demonstrate improvement in intelligence in a way that is measurable on the Raven’s Standard Progressive Matrices test of intelligence. Thus, improved academic performance, as a result of enhanced learning ability, could be achieved.

Additionally, findings from research by Jin et al. (2002) implicated that VEGF is not only functional to angiogenesis but in neurogenesis as well in adult rats. Separately, research on the adult songbird brain conducted by Louissaint, Rao, Leventhal, and Goldman (2002), added that BDNF is produced by the endothelial cells that are being differentiated as a result of increases in VEGF. Although not conducted in humans, this research showed a delayed onset of upregulation of VEGF by two weeks and three weeks

by BDNF after increases in testosterone. Alternately, two research studies attempting to measure human plasma VEGF (Brunelli et al., 2012; Kraus, Stallings, Yeager, & Gavin, 2004) and a review by Jelkman (2001) caution researchers on the pitfalls of measuring VEGF, although it is clearly important to neurogenesis, learning, and memory (Cao et al., 2004; Fabel et al., 2003).

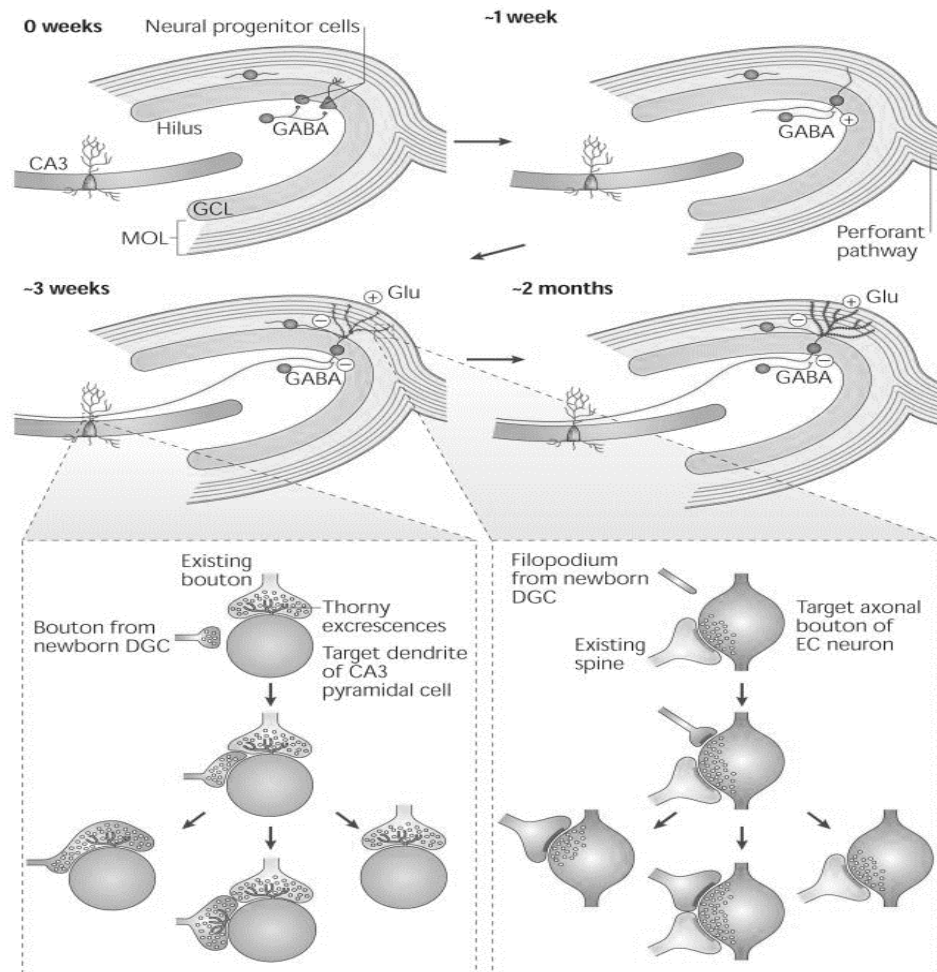


Figure 3. Time scale of adult neurogenesis. Reprinted from “New neurons and new memories: How does adult hippocampal neurogenesis affect learning and memory?”; W. Deng, J. B. Aimone, and F. H. Gage, 2010, *Nature Reviews Neuroscience*, 11(5), p 23.

It is now accepted that interval training increases testosterone (Hackney, Hosick, Myer, Rubin, & Battaglini, 2012), but chess competition increases testosterone levels as well (Mazur, Booth, & Dabbs, 1992). Therefore, these research studies indicate that, not

only should exercise induce increases in BDNF and VEGF, but chess play also should. The process should be initiated by an increase in testosterone initiating uptake of VEGF, stimulating increases in BDNF, which then initiates the process of neurogenesis differentiation through summation. At the very least, this process should take a minimum of seven weeks. While the maximum time course is unknown, in order to observe performance outcomes on cognitive tests and learning, anecdotal research on chess and exercise states that it should take no more than a year.

How Chess and Exercise Relate to Increases in BDNF/VEGF, Intelligence, and Learning Performance

Most research involving chess and exercise in relation to intelligence and learning performance are cross-sectional or correlational in design. They demonstrate that a relationship may exist, but very few, if any, true experimental studies determine cause and effect. This section reviews many of these studies, along with experimental studies relating exercise to changes in BDNF levels in human plasma. The goal is to confirm and expand upon these relationships as a basis to use chess and exercise as treatments to produce increases in neurogenesis and intelligence in young children and to compare the results.

Chess, Intelligence, and Learning Performance. Bilalić et al. (2007) cited six chess studies from 1927-2006 that demonstrate no association of chess skill to higher scores on cognitive tests. Interestingly, all of those studies sampled above average experience to grandmaster-level chess players. Of those six cases, Gruber, Renkl, and Schneider (1994) found a negative correlation between chess skill and intelligence, which appears to contradict the results from Haier et al. (1988), Haier et al. (1992), and Amidzic

et al. (2001). These results, among a host of other chess research, establish that intelligence should increase as chess skill increases. The research by Gruber et al. and Bilalić et al., in conjunction with studies by Haier et al. (1988), Haier et al. (1992) and Amdizic et al. (2001), suggest an interesting postulate that, as chess skill increases, it will increase intelligence. However, as chess skill continues to increase, expertise may continue to increase intelligence in a way that can no longer be measured by current cognitive tests.

Results from the research by Campitelli et al. (2005) appear to confirm this limitation. When comparing the brain activity of novice versus master-level chess players, novice chess players have the greatest activity in the brain regions associated with intelligence: the bilateral frontal lobes. However, the master-level chess players, in accordance with the efficiency hypothesis, display *no activation* in these areas and, rather, exhibit activation in other areas of the brain. The efficiency hypothesis states that inverse relationships exist between brain activity and chess skill (Amidzic et al., 2001) and between brain activity and intelligence (Haier et al., 1988; Haier et al., 1992). This indicates that novice players with activity in the frontal lobes should have lower intelligence than masters, who show no activity in those regions but show activity in other regions in the interneural loops of intelligence. In addition, novice chess players should have increased intelligence scores above their non-chess playing peers. It is important to note that the more experienced master completed the tasks at a rate greater than 90%, while the less experienced master completed the tasks at a rate between 75-90%, and even less for novices. Therefore, this would suggest that, as chess players advance in skill, the areas of the brain associated with intelligence become more efficient,

and the brains of the most experienced masters begin recruiting newer areas of the brain as thinking goes beyond that of lower level expertise while increasing efficiency of the neural network associated with intelligence. Lee et al. (2012) stated, “Our commonality analyses support connectivity in the brain as a good indicator of the *g* factor...indicating that the stronger the connectivity strengths, the higher the intelligence” (p. 38).

Research by Duan et al. (2014) appears to substantiate this suggestion. They investigated the functional capacity of neural network differences between chess masters and novices and stated:

We found that, relative to novices, functional connectivity was increased in GM/Ms between basal ganglia, thalamus, hippocampus, and several parietal and temporal areas, suggesting the influence of cognitive expertise on intrinsic connectivity networks associated with learning and memory. (p. 33)

The studies by Lee et al. (2012) and Duan et al. (2014) have added to the literature that, in this case, highly experienced chess players at the master level and above may begin to further increase intelligence by strengthening connectivity in additional focal regions of the “intelligence template,” increasing efficiency in underdeveloped areas as first described by Haier et al. (1988) and Haier et al. (1992). According to the efficiency hypothesis, and in conjunction with the literature review of Bilalić et al. (2007), these findings suggest the existence of a point where current intelligence testing is no longer the correct means of assessing intelligence in chess experts. Perhaps this illuminates the potential divergence of experienced chess players and intelligence test scores in the literature. Studies on chess + young players demonstrate increases in IQ, while studies on chess + expert players demonstrate no IQ effect. Perhaps these results are not due to the

absence of an increase in intelligence, but are simply due to the limitations of intelligence testing on those who have hypertrophic efficiency of multiple focal regions of the brain currently known to be associated with intelligence and cognitive test scores. This raises many questions that cannot be answered by this current study with respect to transfer, intelligence, expertise, memory, and brain activation.

Table 1

Studies of Chess and Intelligence on Young Children with Positive Outcomes.

Authors	Year	Population	Mean grade (age)	Instrument	IV	DV	Design
Horgan & Morgan	1990	15 elite players*	Elem - 4.25 Jr.H - 8.3	RSPM**	chess experience	cognitive scores	cohort
Frydman & Lynn	1992	33 chess players	(11 yrs)	WISC	rating range	cognitive scores	cohort
Hong & Bart	2007	38 non-chess players	(9.7 yrs)	RSPM TONI-3	treatment: chess, control	cognitive scores	experimental
Aciego et al.	2012	chess-170 sport-60	(6-16 yrs)	WISC-R**	treatment: chess, sport	cognitive scores	quasi-experimental

*15 elite players represents a group of top chess players as a subset of 113 active chess players. WISC/WISC-R is the Wechsler Intelligence Scale for Children. TONI-3 is the Test of Non-Verbal Intelligence 3. **There is a strong highly significant relationship between the RSPM and the WISC-R (.56, $p < .001$).

Several chess studies, using chess as an independent variable and cognitive scores as the dependent variable, demonstrate that intelligence increases with the increase of chess skill (Table 1). Horgan and Morgan (1990) split their elite chess player subsample into two groups: elementary and junior high and they demonstrated that the students who played the most games over the year had the highest skill level. Subsequent to the posttest, the elementary sample scored a mean of 37.7 on the RSPM, and the junior high

sample scored a mean of 53.3. The authors noted that the elementary score was nearly equivalent to the 75th percentile norm for fifth-grade children, and the junior high score was nearly at the 90th percentile for 20-year olds, both demonstrating above average scores on RSPM. Of importance is the increases from elementary mean grade and post RSPM score (4.2/37.7) and the junior high cohort (8.3/53.3). Horgan and Morgan were the first researchers to show a unique trend between chess ability and intelligence.

For the first time, Horgan and Morgan (1998) demonstrated that, as chess playing skill level increases, in this case between grades 4 and 8, an associated intelligence curve can be demonstrated based on cognitive practice and time. Horgan & Morgan's study lacked a comparison of the chess treatment to a control group. However, the findings clearly note that a "*learning curve*"⁴ becomes visible as a result of increasing contact time with the learning and practice of chess.

Previous research conducted by Frydman and Lynn (1992) further validated this *learning curve* by dividing the sample by chess rating range (Table 2). Those ranges are: Group 3 (1000-1350; class D player); Group 2 (1350-1550; class C player); and Group 1 (1550+; class C and above). Parallel to Frydman and Lynn, Horgan and Morgan (1990) demonstrated that subjects who had played more games showed significantly greater skill. Chess rating is a measure of chess playing ability or skill level. In the study by Frydman and Lynn, IQ increases as chess playing skill increases.

Figure 4 combines intelligence scores of beginning chess players from data provided by Frydman and Lynn (1992), with an elite subsample of active players whose average rating is 1603 from data provided by Bilalić et al. (2007). The subsample

⁴ This "learning curve" in relation to neurogenesis was first described by Hunt and Navalta (2012) pg. 264.

demonstrated higher IQ scores (WISC-III), experience, and more time playing chess being regularly active in clubs and tournaments.

Table 2

Mean IQ's of Young Belgian Chess Players.

Group	N	Full-scale IQ	Verbal IQ	Performance IQ
1	11	122	110	131
2	11	123	110	132
3	11	117	107	124
total	33	121	109	129

Reprinted from "The general intelligence and spatial abilities of gifted young Belgian chess players," M. Frydman and R. Lynn, 1992, *British Journal of Psychology*, 83, p. 235.

Hong and Bart (2007) continued to demonstrate this trend toward the *learning curve* and further validated the proposal of a new definition of learning based on neurogenesis. They found that, in the chess treatment group, TONI-3 posttest scores are significantly correlated to chess skill rating. Again, this demonstrates that, as chess skill improves, intelligence also improves.

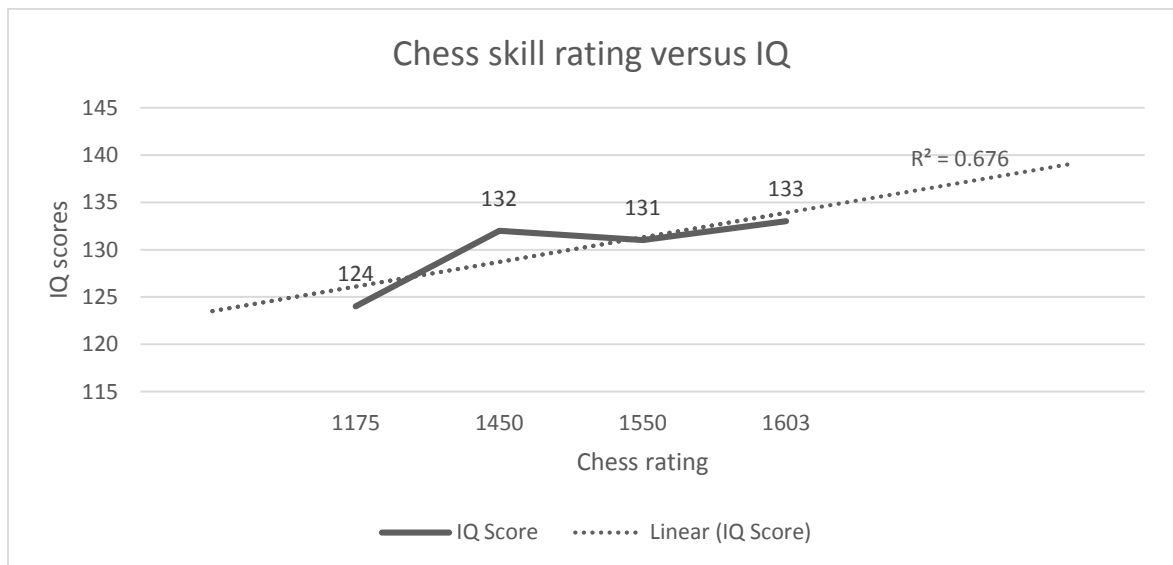


Figure 4. Relation of Chess rating to IQ scores.

Last, Aciego et al. (2012) also demonstrated that the chess group had a significant improvement on within-group and between-group differences on more subsets of the

WISC-R intelligence test than the sport group (basketball/soccer). Their research also found that the socioaffective competence measures demonstrated that the chess group was rated higher in both personal and academic spheres.

The previous paragraphs have clearly demonstrated that chess experience and intelligence are strongly associated with one another. However, as part of the theory to redefine learning based on neurogenesis, a predictable improvement should be found, not only in intelligence, but in learning performance as well. Peer-reviewed journal-published research on chess and academics is surprisingly sparse. Five seminal studies stand out, whose topics focused on improvement of mathematics ability as an effect of chess treatment (Table 3).

Smith and Cage (2000) studied the effects of chess instruction on the mathematics achievement of rural African-American students in grades 9-12. All were enrolled in normal scholastic math courses from algebra to calculus. The study consisted of a pre-post randomized control-comparison group research design. Both groups received 120 hours of instruction in chess or other course electives (including additional math courses). No differences were noted on the pretest scores in either the treatment or control group. Post-test results demonstrate that the treatment group scored higher on mathematics ability, spatial visualization, and nonverbal reasoning skills.

Table 3

Studies of Chess and Academic Outcomes on Young Children

Authors	Year	Population	Mean grade	Duration	Instrument	IV	DV	Design
Smith & Cage	2000	40 male-female non-chess players	Junior-Senior	120 hours chess instruct for 5 months	CAT & NNAT	chess instruction	test scores	pre-post, control-comparison
Scholz et al.	2008	70 learning disabilities	3rd/4th grade	school year-1 hr/week	research based tests	chess instruction	calculation & concentration abilities	ex-post-facto
Barrett & Fish	2011	31 special education	6th-8th grade	30 weeks	TAKS	chess instruction	TAKS scores	ex-post-facto
Kazemi, Yektayar, & Abad	2012	180 male Iranian non-chess players	5th, 8th, 9th grade	6 months	Meta-cog Q; Math exam	chess instruction	Meta-cog & TIMSS scores	pre-post, control-comparison
Gliga & Flesner	2014	20 novice chess & 18 control Romanian	3rd/4th grade	10 weeks-1 session/week	School Perf tests, Kraepelin test, Rey test	chess instruction	test scores	pre-post, control-comparison

Scholz et al. (2008) also studied the effects of chess instruction on mathematics performance but, rather, used a population of students in grades 3 and 4 with learning disabilities. Their study used a pre-post randomized control-comparison group research design. The treatment group received math integrated chess lessons one hour per week for a year consisting of chess basics, notation, chess puzzles, and game play. The control group received regular math curriculum for the same period. No differences were noted on pretests between groups. Mathematics ability increased more significantly in the treatment group versus the control group.

Barrett and Fish (2011) observed that the chess treatment group had better outcomes on end-of-year course grades and overall TAKS math scale scores versus the control group. Kazemi, Yektayar, and Abed (2012) found that the chess treatment group demonstrated increased meta-cognitive ability and math problem-solving ability over non-chess playing students. Another interesting discovery was the strong correlation between meta-cognitive ability and math problem-solving ability in all students. Gliga and Flesner (2014) also demonstrated similar results in the chess training group on their School Performance Test. Although cognitive skills increased in all subjects due to treatment, the chess group increased significantly more than the non-chess playing group on the School Performance Test. Interestingly, the research by Gliga and Flesner is the only study to use blended learning of chess skills in the school curriculum.

This section demonstrates that chess not only increases intelligence but perhaps increases intelligence beyond measureable levels of intelligence tests. Chess also impacts learning performance. A theoretical basis is given from which to predict that chess should

increase intelligence scores and, although beyond the scope of this study, also should increase learning performance.

Exercise, BDNF/VEGF, Intelligence, and Learning Performance. A review of 850 research articles reveals that a child needs approximately 60 minutes of moderate to vigorous age-appropriate daily physical activity while in school (Strong et al., 2005) in order to maintain proper development. However, school systems do not afford that amount of daily physical activity in the US. McCullick et al. (2012) surveyed and analyzed physical education policies in the 50 states. Their interest was to discover which states mandate physical activity, which states follow the NASPE Guidelines for Quality Physical Education, and how many statutes are written in a clear manner to be interpreted by school boards and other governing educational bodies. Although a high percentage of states (> 74%) have mandates for physical education in at least one of three levels (elementary, middle, high), they found that only six adhere to guidelines at the elementary school level, only two at the middle school level, and zero at the high school level. McCullick et al. added that the statutes in all states are written in an ambiguous manner that are merely suggestions and non-explicit, which leaves governing bodies open to the interpretation of the statute as they see fit. According to the Centers for Disease Control and Prevention (2014), only 27% of female and 35% of male high school students participate in any daily physical activity for 60 minutes that increases heart rate and causes heavy breathing. Such over-simplified surveys fuel the fire for ambiguity in regard to necessary physical activity (PA) requirements essential to proper biological development. Dwyer, Coonan, Leitch, Hetzel, and Baghurst (1983) demonstrated that appropriating 60 minutes of school time daily to physical education (and not formal

teaching) does not cause any loss on mathematics and reading scores of students. Another study by Sallis et al. (1999) found that not only did twice the allotted physical activity not hinder academic quality but results verified that the increased amount of physical activity improves academic performance.

Existing correlational evidence on the effect of PA/exercise (PAE) on academic outcomes can be interpreted in light of the framework guiding this current study to determine the causal factors between increases in physical activity that predict improved learning performance. Four groups of researchers, together or individually, dominate the literature on exercise and cognition: (1) Colcombe and Kramer, whose primary research focus was on the effects of exercise on the aging brain in older adults; (2) Scarmeas, whose research focused on diet, exercise, and Alzheimer's disease; 3) Castelli and Hillman (Table 4), whose efforts focus on the fitness, cognition, and academic performance in preadolescent children; and (4) Davis and Tompowroski (Table 5), whose research focused on childhood obesity and its effects on cognition and academic performance. The dichotomy of these research topics demonstrates that certain control factors mitigate quality research on exercise and cognition in order to obtain the best effect sizes - the psychological task/test used, the mode/duration/intensity of the exercise, and the age of the population. These findings were first recognized in an important meta-analysis by Etnier et al. (1997), which established the underlying premises for rigorous research in this area. With respect to the current investigation, they primarily determined that,

To truly establish a cause-and-effect relationship for exercise and cognition, one must use a chronic exercise program in which sedentary participants are randomly

assigned to treatment conditions. To examine this relationship, those studies that used true-experimental designs in randomized trials were examined separately. The results showed that the overall effect size was small ($ES = 0.18$) but still positive and significantly different from zero. This would suggest then that implementing a chronic exercise program in sedentary individuals can cause increased cognitive function. (p. 267)

Taken together, a few studies show a connection between hippocampal neurogenesis, exercise, BDNF, and learning in humans: Pereira et al. (2007); Winter et al. (2007); Griffin et al. (2011); and Cooper, Bandelow, Nute, Morris, and Nevill (2013). However, no studies were found that (a) investigated neurogenesis in young children, (b) measured BDNF/VEGF in young children after exercise treatment, and (c) attempted to correlate BDNF increases to increases in intelligence.

This section attempts to show relevant research that improves the picture and suggests how children's intelligence may be affected after exercise treatment with respect to levels of BDNF/VEGF in plasma. Tables 4 - 6 demonstrate a sample of the research relative to the impact of exercise on academic performance.

Exercise, BDNF, and Cognition. The research by Winter et al. (2007) illuminated the picture by demonstrating that BDNF appears to serve as a mediator in the process in which physical activity improves learning. Their study included 27 healthy college-age subjects in three conditions: 15 minutes of sedentary behavior, 40 minutes of low-impact running, and an intense condition consisting of two intermittent sprints of increasing speed for a period of three minutes each. Results indicate that the intense condition improved learning by approximately 20% above the low-impact and sedentary

treatments. The ANOVA analysis demonstrates significant interactions based on time and condition in relation to plasma BDNF levels; i.e., the longer the BDNF levels are sustained in the intense training condition, the greater the immediate learning success. School systems should be encouraged by such results, as this indicates that, after an acute intermittent burst of exercise lasting no more than 10 minutes, learning ability improves. Such bursts of exercise may be strategically utilized in the classroom to enhance test performance and new learning/skill acquisition.

Similarly, Griffin et al. (2011) verified that acute exercise modulated increased learning performance through increases in BDNF. They also found that, after three weeks of training, the immediate post-exercise BDNF effect on learning is lost; after five weeks, the spike in BDNF levels do not appear until 30 minutes post-exercise. However, five weeks of training resulted in increased fitness and long-term improved learning performance, suggesting that long-term training may modulate the individual's BDNF physiology.

Cooper et al. (2013) added to this timeline for cognitive effects of acute exercise on human subjects by administering cognitive tests 30-minutes pre-treatment and 10- and 60-minutes post intermittent exercise treatment. They found that, at 60-minutes post treatment, no difference existed between treatment and control groups on cognitive tests. The only cognitive effect differences between treatment and control existed at the 10-minute posttest. Although chronic exercise of five weeks reveals long-term benefits on learning performance, the results demonstrated that an additional acute burst of high intensity intermittent exercise could offer an additional short-term boost to testing performances, in agreement with Winter et al. (2007).

Table 4

Sample of Studies on Exercise and Cognition from Hillman and Castelli

Authors	Year	Population	Duration	Methods	Measures	IV	DV	Results
Hillman, Castelli, & Buck	2005	High/Low-fit children & adults	N/A	cross-sectional	Fitnessgram, EEG Recordings	Fitness level	behavioral measures, ERP analysis	High-fit children/adults had faster neurocognitive processing
Castelli, Hillman, Buck, & Erwin	2007	3rd/5th grade students	N/A	cross-sectional	Fitnessgram, ISAT Achievement Test	Fitness level	ISAT score	Fitness is positively related to academic performance
Buck, Hillman, & Castelli	2008	children (7-12 yrs)	N/A	cross-sectional	Fitnessgram, Stroop Task, KBIT Intelligence Test	Fitness level	Stroop score	Higher fitness and IQ was associated with better executive control
Hillman et al.	2009	children (age 9.6)	Acute exercise bout	within-subjects, crossover	EEG, WRAT3 achievement test, fitness test	Exercise & rest conditions	ERP & test performance	Acute exercise improved reading performance and cognitive control
Kamijo et al.	2011	children (7-9 yrs)	9 months	randomized, pre/post, control/comparison	Fitness, EEG, Sternberg Task	Phys Activity	ERP & cognitive performance	Physical activity intervention improved working memory

Table 5

Sample of Studies on Exercise and Cognition from Davis and Tomporowski

Authors	Year	Population	Duration	Methods	Measures	IV	DV	Results
Davis et al.	2007	overweight children (age 9.2)	15 weeks	randomized, pre/post, control/comparison	Cognitive Assessment System (CAS)	Exercise	Cognitive performance	High-dose exercise significantly improves cognitive ability
Wittberg, Cottrell, Davis, & Northrup	2010	5th grade students	2 years	cross-sectional	WESTEST Academic Test, Fitnessgram	Fitness level	Academic test scores	Highly significant correlations exist between fitness and academic test scores
Davis et al.	2011	children (7-11 yrs)	15 weeks	randomized, pre/post, control/comparison	CAS, Woodcock-Johnson Tests of Achievement III, fMRI	Exercise treatment	Cognitive test scores	High-dose exercise improves cognitive ability and math achievement; also increased prefrontal cortex activity
Fischer et al.	2011	children (age 6.2)	10 weeks	randomized control trial	CAS, CANTAB, Attention Network Test	Exercise treatment	Cognitive test scores	No meaningful differences were found
Krafft et al.	2014	children (8-11 yrs)	8 months	randomized, pre/post, control/comparison	fMRI, antisaccade task, flanker task	Exercise treatment	Brain activation, cognitive control	Exercise group showed significantly decreased activation during cognitive tasks from pre-post

Table 6

Sample of Studies on Exercise and Cognition.

Authors	Year	Population	Duration	Methods	Measures	IV	DV	Results
Coe, Pivarnik, Womack, Reeves, & Malina	2006	6th-grade students	School year	longitudinal	3-d physical activity recall, school grades, terra Nova standardized test	Physical activity level	grades	Students who met Healthy People 2010 guidelines had better academic scores
Verret, Guay, Berthiaume, Gardiner, & Béliveau	2012	ADHD children (age 9.1)	10 weeks	randomized, pre/post, control/comparison	Test of Everyday Attention for Children; Child Behavior Checklist; Test of Gross Motor Development	Physical activity training	test scores	Experimental group showed improved information processing ability; improved behavior; improved motor performance
Everhart, Dimon, Stone, Desmond, & Casilio	2012	Special Education children	30 days	pre/post, group comparison	Reading/Math assessment	Exercise treatment	assessment scores	Academic performance increased in intermediate grades immediately after exercise bout
Syväoja et al.	2013	Finnish school children (age 12.2)	10 weeks	cross-sectional	Self-reported physical activity and screen time	Physical activity; sedentary behavior	grade point averages	Physical activity had inverse-U association with GPA; a negative linear association existed for screen time

Tang, Chu, Hui, Helmeste, and Law (2008) also confirmed study results from Winter et al. (2007) and Cooper et al. (2013). Their study demonstrated that increases in BDNF are significant at 25-minutes after exercise initiation, but not significant after 50-minutes. Additionally, research by Brunelli et al. (2012) supported these findings, as exercise levels of BDNF spiked immediately post exercise, but were reduced to pre-exercise levels at 30-minutes post. Brunelli et al. also demonstrated that BDNF levels were regulated “in a manner related to physiological stress exerted” (p. 1871).

The previously mentioned line of research demonstrates a timeline at which BDNF increases and affects learning performance, but tapers off toward baseline levels within one hour. This information suggests that, as BDNF is the switching mechanism for neurogenesis, a post-initiation of exercise window of opportunity captures a short-term enhancement of academic performance. In the long term, exercise will improve BDNF function, as a catalyst for neurogenesis, and boost learning and memory performance. Bekinschtein, Oomen, Saksida, and Bussey (2011) these findings as follows:

1. Voluntary exercise is associated with learning and memory.
2. Voluntary exercise increases both neurogenesis and BDNF.
3. Both BDNF and neurogenesis are required for pattern separation.
4. BDNF might be a third variable responsible for learning and memory enhancement.

Therefore, according to the current framework guiding this investigation, vigorous exercise treatment should produce increases in plasma BDNF, thus producing increases in neurogenesis, which should increase intelligence and which enhances learning performance in humans.

Rasmussen et al. (2009) also supported this trend by discovering that the brain contributes 70-80% of circulating BDNF, which increases two- to three-fold above base level during exercise and which returns to base level 60 minutes post exercise. Extending this line of inquiry, an important study by Lee et al. (2014) became the first report on the interaction of exercise, neurotrophic factors BDNF/VEGF, and performance on neuropsychological assessment. Their study included 45 regular, sport-training adolescents and 46 matched sedentary controls. Subjects were matched based on intelligence, as measured by the RSPM, right-handedness, and psychomotor speed. A battery of neuropsychological tests was used to determine changes in the frontal and medial-temporal lobes of participants' brains. A unique demographic finding revealed that the exercisers had significantly lower levels of both BDNF and VEGF prior to the start of the testing session. This study involved no exercise treatment and was a between-group design that compared exercisers with controls on neurotrophic factors and cognitive functions. Results demonstrated significantly better performance in the exercise group with respect to assessments targeting the frontal and medial-temporal lobes of the brain. Interestingly, the researchers found a negative correlation between BDNF/VEGF and the neuropsychological tests administered to the exercise group.

Aligned with the previous research, an earlier study investigated brain activation and cognitive performance after exercise treatment (Kubitz & Pothakos, 1997). The study involved a randomized pre-post control-comparison group research design with the hypothesis that brain activity would be reduced in the exercise group, as measured by EEG at four time points: baseline, recovery, 5 minutes, and 15 minutes post. Cognitive functioning also was measured based on a vigilance task that is simply a test of

concentration at 5-minutes and 15-minutes. The same trend was demonstrated as spikes in BDNF, in that brain activation was decreased in the exercise group at recovery and 5-minutes, but not at 15-minutes. Inconsistent with literature on exercise and cognition, Kubitz and Pathakos (1997) found that the lower levels of activation were detrimental to cognitive function in the exercise group, although they admitted in their research that the cognitive test scores were “small and quite variable” (p. 299). Whether a decrease in activation causes a detrimental effect on cognition appears to be inconsistent with the aforementioned studies as the type of test chosen was unreliable or invalid with the research. Most studies used the Stroop test to measure cognitive function in relation to exercise.

Lee et al. (2014) and Kubitz and Pothakos (1997) provide intriguing context with respect to the brain efficiency hypothesis of Haier et al. (1988), Haier et al. (1992), who discovered a negative correlation between brain activation and intelligence, and a negative correlation between expertise and activation. Lee et al. found that basal BDNF/VEGF were lowest in those who were chronic exercisers, and a negative correlation was discovered between BDNF/VEGF and neuropsychological outcomes. Kubitz and Pothakos found a negative correlation between exercise and brain activation. Additionally, based on the literature review, the focal location of intelligence in the brain is in the frontal cortices. After acute exercise, a short-term increase in BDNF is correlated to an increase in cognitive test outcomes. Lee et al. found that the exercisers performed much better on neuropsychological assessment than the controls.

This section suggests that increased efficiency of the frontal and medial-temporal lobes of the brain can be predicted as an effect of increased PAE. As this is the focal

region of intelligence in the brain, PAE activity level should create a positive correlation to intelligence, a positive correlation to brain efficiency, and a negative correlation to brain activity in the frontal and medial-temporal lobes. Additionally, long term PAE, which will increase brain efficiency in the long-term, can be predicted to affect the basal levels of BDNF/VEGF in the plasma of human subjects, as PAE expertise increases the physiological system's efficiency of the body.

A caveat can be noted in all of this: although research on acute exercise treatment demonstrates a clear trend of short-term increases in cognitive test outcomes, this is not in conflict with the time-course of neurogenesis from proliferation to summation, as described earlier in this review. According to the literature, while acute exercise increases BDNF levels and, therefore, neurogenesis, this does not contribute to the short-term outcomes of cognitive test performance in those studies. Rather, Hunt and Navalta (2012) posit that those increases are due to the following processes:

1. Increases in exercise lead to increases in nitric oxide (NO) intake across the nasal sinus and into the lungs.
2. Increased amounts of NO transfers across the mucus membrane and expands microvessels in the brain.
3. Expansion/contraction of microvessels and increases in heart rate increase brain blood flow, which increases electrical impulses of glial connectors between microvessels and neurons.

Thus, as an outcome of acute exercise, the brain increases NO uptake, electrical stimulation, and increased blood flow; increases short-term efficiency; and enhances acute learning and memory performance. Only one study in the literature review

investigated the effect of physical exercise on intelligence based on RSPM outcomes. The researchers found no between-group differences, and no changes on RSPM pre-post could be demonstrated (Sparrow & Wright, 1993).

The Tabata Protocol: High Intensity Intermittent Exercise. High intensity intermittent training (HIIT) has become increasingly popular due to its perceived benefits on metabolism and its shortened time frame to complete the routine. The Tabata and Gibala protocols are both peer-reviewed HIIT subcategories. The Gibala protocol (Little, Safdar, Wilkin, Tarnopolsky, & Gibala, 2010) utilizes 8-12 intervals of 60 seconds of peak exercise, with 75 seconds of rest between each bout. In that study, considered a low-volume HIIT workout, only three exercise periods over two weeks were used with successful significant increases in multiple physiological processes associated with known athletic endurance training.

Tabata et al. (1996) compared HIIT with endurance training in a single study. The first experiment consisted of endurance training of moderate intensity five days per week for six weeks, each session lasting 60 minutes. The HIIT training consisted of training four days per week for six weeks, one additional day consisting of a 30-minute low-intensity, non-exhaustive workout. Each HIIT training bout consisted of cycling at a minimum of 85 RPM for 20 seconds, with 10 seconds of rest for seven to eight sets. Both modes of training, endurance and HIIT, were conducted on a stationary cycle ergometer. Measures included both maximal aerobic capacity (VO_2Max) and anaerobic capacity, the two standards by which fitness is measured. Results indicated that both training methods increased VO_2Max by 5 ml/kg^{-1} in endurance training and by 7 ml/kg^{-1} in HIIT training. However, anaerobic capacity does not increase significantly in endurance training, while

the HIIT training increases anaerobic capacity by 28%. This protocol became known as IE1 and was repeated in a second study by Tabata et al. (1997), which added a second HIIT protocol labeled IE2. The IE1 protocol also was found to improve both aerobic and anaerobic physiology in the second study.

This type of exercise has been researched and shown to meet American College of Sports Medicine (ACSM) guidelines for improving cardiorespiratory endurance (Emberts, Porcari, Doberstein, Steffen, & Foster, 2013). This protocol has been shown to cause significant weight loss and increase in muscle tone (fat-free mass), as well as aerobic power in young males (Heydari, Freund, & Boutcher, 2012) and similar outcomes in young females (Trapp, Chisholm, Freund, & Boutcher, 2008). Additionally, HIIT of differing protocols are shown to be at least as effective as endurance training in both fit (Sperlich et al., 2011) and unfit children (de Araujo et al., 2012). de Araujo et al. (2012) may have only found equal results between endurance and HIIT modes of training because they did not follow either the Tabata or Gibala methods of HIIT. However, they were equal. The important factor in the de Araujo et al. and Sperlich et al. (2011) studies is that the HIIT protocol is a condensed version of training that provides the same benefit as endurance training, making it a perfect fit to use in the classroom during a normal school day. In fact, Hazell, Olver, Hamilton, and Lemon (2012) found that VO_2 outcomes were similar to 30 minutes of endurance training, in only two minutes of sprint interval training.

With the inclusion of the results of the studies by Winter et al. (2007) and Cooper et al. (2013) showing that intermittent interval training increases both BDNF and intelligence, a clear link becomes apparent between exercise and neurogenesis, and

neurogenesis and intelligence, as well as intelligence to learning performance as a result of adequate levels of exercise.

Conclusion

This literature review has endeavored to establish the theoretical justification for conducting a non-invasive study on neurogenesis research in young children in the elementary classroom. From this, an experimental basis for the development of a newer, more holistic definition of learning, based on neurogenesis, should result. The literature has described the links between neurogenesis and intelligence in humans, as well as the timeline from differentiation to summation by which that process may occur. This review has established the relation of BDNF/VEGF to intelligence and learning performance, as described in studies on chess and exercise, and also chronicles a multidisciplinary axis from which to span the previously described “bridge to far” by demonstrating methods to increase neurogenesis. Increases in neurogenesis will lead to predictable increases in both intelligence and learning performance that can be hypothesized, investigated, and measured. Last, this review offers a practical, inexpensive, highly-adoptable prescription to improve physical and cognitive fitness in young scholastic students through the adoption of chess and exercise in the classroom:

1. Add 10 minutes of in-classroom Tabata protocol exercise at the beginning of each of six periods (totaling 60 minutes/day).
2. Add 60 minutes of chess study/play weekly (additional after-school club may be necessary as interest increases in the competitive side of the sport).

The inclusion of such activities does not hinder academic progress; and each method, individually and combined, has been shown to improve physical and cognitive fitness.

Such fitness provides acute and aggregate benefits on testing scores and learning ability. Exercise is shown to increase BDNF, which is the switching mechanism initiating the differentiation to summation process. However, if the neurons do not become functionally integrated, they die off. The addition of chess (along with regular classroom instruction) assists in the functional integration of neurons in the hippocampus, which leads to the belief that outcomes in children should be similar to those of Fabre et al. (2002), in which the combined mental training + exercise group outperformed both exercise only and mental training only.

A “one-size-fits-all” test for accurately assessing learning outcomes has been the goal of education institutions for some time (Douglass, Thomas, & Zhao, 2012). However, the process by which professionals in the field have attempted to arrive at such an assessment has been fraught with controversy (Lederman, 2012). As has been discussed previously, this is due to the incorrect definition and misunderstanding of learning and the means to properly define learning. It becomes clear, based on the literature review, that the traditional definition of learning, based on Lachman (1997), is inadequate and stands in need of revision. Chapter III will focus on the methods and practices by which learning can be assessed based on the biological process of neurogenesis.

CHAPTER III: METHODOLOGY

The literature review provided clear evidence of an association between neurogenesis, intelligence, and learning performance. Cross-sectional, correlational, and experimental studies demonstrate that this appears to be caused by increases in vigorous physical and mental exercise. It appears that no studies exist to fill the following gaps in the literature:

- Measurement of neurogenesis in young children
- Cause/effect studies between neurogenesis and intelligence in humans
- Comparison of effects of chess and exercise on intelligence and protein biomarkers
- Bridging the gap between neuroscience investigation and educational policy with direct application/recommendations to practices in the classroom

The goal of the current study is to lay a foundation, within the neuroscience of education, by which a new definition of learning, based on neurogenesis, may be established to guide future educational policy and practice. To this end, this research is designed to lay the foundation for a replicable method by which neurogenesis may be measured non-invasively, cost-effectively in young children. The methodology accomplishes this task by measuring the levels of two protein biomarkers of neurogenesis, brain-derived neurotrophic factor (BDNF), and vascular endothelial growth factor (VEGF) in plasma before and after treatment.

Parallel to this, measures of intelligence pre- and post-treatment also have been included in order to determine whether increases in neurogenesis produce increases in intelligence. The well-researched treatments chosen in an attempt to elicit increases in

neurogenesis and intelligence include chess only (chess), exercise only (exercise), and chess + exercise (combined).

This chapter defines an objective process by which learning (as newly defined) can be measured accurately over time. In conjunction with current academic progress assessment practices that measure content specialization, this will provide a holistic and accurate quantification and valuation of learning. Such data may empower schools with the ability to advance intrinsic learning performance and academic outcomes through enhanced biological development of the mind, brain, and behavior of students.

Subjects

Participants ($N = 40$, mean age 10 years) were recruited from a rural elementary school in southern, Kentucky, in grades 4 and 5 and consisted of 21 male and 17 female students. The school represents a population of which 27% live in poverty.

Approximately 70% of students receive free or reduced lunches. Subjects were randomly assigned into four groups: a chess only group ($N = 10$, 6 male, 4 female), an exercise only group ($N = 9$, 4 male, 5 female), a combined group ($N = 10$, 6 male, 4 female), and a control group ($N = 9$, 5 male, 4 female). Groups were matched based on grade level and gender (Table 7). The participants are a mixture of gifted/talented, developmentally challenged, at-risk, and special education students. Application was made to the WKU HSRB, and the study passed a full board review prior to beginning the research. With permission of the superintendent and principal of the school, informed consent forms were sent to parents and children prior to the start of the research. Both parents and children were made aware of the nature of the treatments and the blood tests pre- and post-intervention (Appendix A1).

Table 7

Subjects, Their Groupings, and Their Classifications

Combined				Chess only				Exercise only				Control			
#	Grade	Gender	Class	#	Grade	Gender	Class	#	Grade	Gender	Class	#	Grade	Gender	Class
18	4th	F	N	37	4th	M	G/T	11	5th	F	N	5	5th	F	N
25	5th	M	N	4	4th	M	G/T	28	4th	M	IEP	14	5th	F	D/C
20	4th	F	N	34	4th	M	N	1	4th	F	N	13	5th	M	R
15	4th	F	N	3	5th	F	G/T	10	4th	M	D/C	32	5th	F	N
6	4th	F	G/T	23	5th	M	D/C	24	5th	F	N	29	4th	M	N
9	5th	M	N	22	5th	F	G/T	35	4th	F	N	33	5th	M	N
12	5th	M	N	8	5th	F	D/C	19	5th	M	N	36	4th	M	IEP
16	5th	M	N	31	5th	F	N	21	5th	F	N	30	5th	F	N
2	4th	M	N	26	4th	M	D/C	17	4th	M	D/C	27	5th	M	N
7	5th	M	N	38	5th	M	N								

Note: Subjects 17 and 21 dropped out of the study. *Abbrev:* gifted/talented (G/T), normal (N), developmentally challenged (D/C), at-risk (R), special education (IEP).

A physical activity readiness questionnaire (Thomas, Reading, & Shephard, 1992) also was required of children prior to participation (Appendix A2). The involved teachers were instructed on the schedule and training program for both chess and exercise regimens. The primary investigator was scheduled to be on site, at a minimum, at the beginning of each week to answer chess related questions and begin the next set of exercises. Both regimens were accessible in online chess lessons and online exercise videos. During the treatment period, students at no time fell behind in the curricular standards for the school system.

Procedures

The training protocols were designed to run daily for a period of nine weeks or 45 consecutive school days. The optimal time period was from September 15 through November 22, 2013, prior to the school’s Thanksgiving break. However, a full week for fall break in this school system fell between the fourth and fifth weeks of training. Additionally, three of the days were missed by the teachers; therefore the students were

not involved with training on those days. Students received 42 days of treatment, for the equivalent of 28 hours of chess and 10.5 hours of exercise. One classroom teacher supervised the chess group, and one supervised the exercise group throughout the term of the study. Both received an orientation on the chess and exercise protocols, but neither were experts. All treatments were conducted at the same time of day in the following manner: 1:30-1:50 p.m. exercise and combined groups; 1:50-2:30 p.m. chess and combined groups. Prior to beginning of the treatment period, and after the conclusion of the treatment period, each participant was required to take the Raven's Standard Progressive Matrices (RSPM) test, 2000 edition (Raven, Raven, & Court, 2003) and submit to a blood draw.

RSPM and blood draw. The purpose for using the RSPM test was not only for its measure of non-verbal intelligence, but also the aesthetically pleasing nature of the test for children. The RSPM uses a format similar to a puzzle with a missing piece (Figure 5).

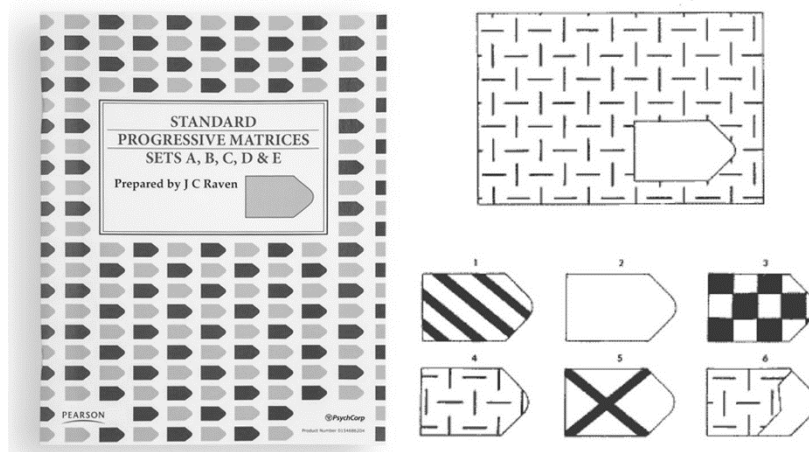


Figure 5. An image of the RSPM test booklet used and sample question.

The simple instructions direct the individual to find the picture piece below the puzzle that best fits the blank in the puzzle. Participants must match the pattern and shape, then mark the score sheet. The test includes 60 items divided evenly into five

groups, A-E (Figure 6). Questions become harder as one progresses across groupings from question 1-60; questions 55-60 being the most difficult.

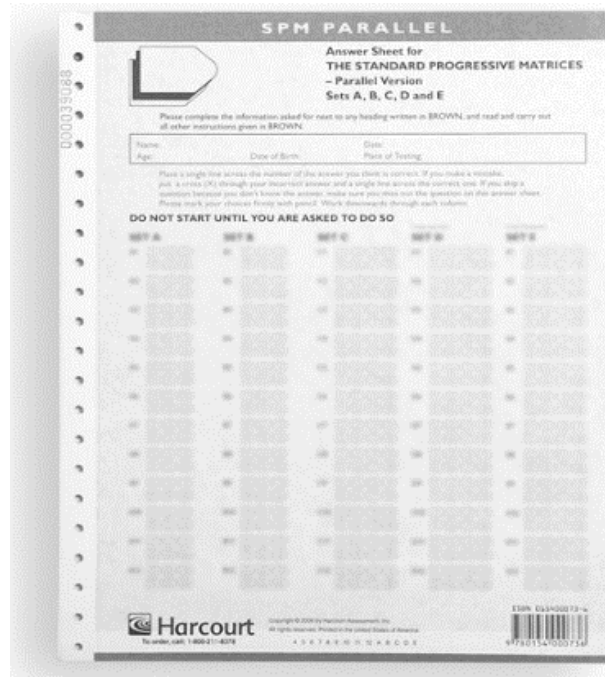


Figure 6. An image of the RSPM score sheet layout.

Tests were administered in two groups of 20 students, with a teacher, the researcher, and a teaching assistant present in each group. No time limit is imposed which reduces test-taking anxiety. Sixty minutes was allotted for students to complete the test prior to lunch. If any student needed more time to complete the test, they were allowed to move to the second group in order to finish. Only one student on the pretest, who had extreme ADHD, required such an accommodation. No students required more than 60 minutes on the posttest and no one took longer than 45 minutes to complete the assessment.

The test is well validated and central to studies using psychometric tests across many cultures and populations (Raven, 2000). The benefits of using this test are: (a)

reliability and validity of measuring cognitive ability, (b) ease of use and appeal to young children, and (c) ease of administration in group settings. Additionally, Sparrow and Wright (1993) previously used the RSPM to measure cognitive ability after only a 6-minute, acute regimen of exercise. Smith and Cage (2000) and Hong and Bart (2007) used tests of non-verbal intelligence to measure cognitive ability after chess treatments.

On the same day, and immediately after completion of the RSPM, students were required to submit to a blood draw to obtain 4 ml of human blood, according to volumes and procedures of New London Hospital (Appendix B). A certified phlebotomist employed at Graves Gilbert Clinic (Bowling Green, KY) was recruited to perform the blood draw on both pre and post samples. After the first blood draw, two students, both from the exercise group, decided not to take the post blood draw. They were allowed to remain as part of the treatment group, but results were dropped from analysis. All blood samples were collected in green top heparin tubes in the school nurse's office with her direct supervision of each child. Blood samples were placed on ice at 2-4° Celsius, remaining on ice for approximately two hours while the blood was collected in preparation for transport to the Western Kentucky University Biotech Center. Samples were immediately spun in a Fisher accuSpin™ 1/1R Benchtop Centrifuge (Thermo Fischer Scientific, Inc., Waltham, MA) at 2000xg for 15 minutes. Samples were immediately placed back on ice and transferred to a level two biosafety hood, where 38 .5 ml aliquot samples were prepared in triplicate. Samples were de-identified, numbered and placed in cold storage at -20° Celsius until analysis could be conducted.

BDNF and VEGF levels present in plasma samples were measured using an enzyme-linked immunoabsorbant assay (ELISA) following the RayBio® Human BDNF

and VEGF ELISA Kit Protocols (RayBiotech, Inc., Norcross, GA) for each (Appendix C). The dilution factors chosen for the study were two-fold for VEGF and ten-fold for BDNF. At completion of the assay procedure, samples were read immediately at 450nm using a Synergy H1m Monochromator-Based Multi-Mode Microplate Reader and Gen5™ Data Analysis Software (BioTek® Instruments, Inc., Winooski, VT).

Chess instruction. Chess has enjoyed a long, diverse history in psychological studies on improving cognitive ability, spanning from the earliest known reference to chess and the mind in a peer-reviewed journal (Verdon, 1877) to the present study. Sixteen online chess lessons were created as a distance learning course for all ages of beginner chess players who had none, or perhaps minimal, knowledge of the rules of the game. These were created to standardize basic instruction for group lessons and as a way to alleviate the classroom teacher's responsibility of teaching chess to students. The format of the daily chess regimen (Appendix D) was created for the teacher who supervised the 40-minute chess period. The regimen consisted of the 16 online lessons and selections of daily activities from the Chess King Training DVD course authored by former women's world champion and chess grandmaster Alexandra Kosteniuk (ChessQueen, Inc., Key Biscayne, FL). Prior to beginning the protocol, and after conclusion of the treatment period, each student in both the chess and combined groups was given a chess assessment. They were instructed to answer six chess puzzles from page 119 in the Chess Tactics Workbook (Woolum, 2000) by circling the piece to be moved first in order to reach checkmate (Appendix D). However, at approximately the fifth week, it became apparent to the teacher that the lesson level was increasingly beyond age-appropriateness of the students and the level of teaching ability of the

instructor, if questions were asked. At that point, the lesson plan was abandoned and the students were allowed, with teacher supervision, to free play and self-learn chess for 40 minutes per day for the remaining four weeks.

Exercise protocol. The daily exercise protocol consisted of a 5-minute warmup wherein the teacher demonstrated the exercise and then the students were asked to practice the movement prior to beginning the 4-minute exercise. A 5-minute cool down period commenced at the completion of the exercise. The total exercise period lasted no longer than 15 minutes due to two reasons: (1) the movements were easy for children to learn and follow; and (2) the duration of exercise, in addition to the chess period for the combined group, would adapt to the daily routine without disruption to the regular curriculum. The exercises were contained in a small area, allowing all subjects to remain in the classroom for exercise, and did not require any additional rooms in the school.

The exercises were based on the Tabata regimen, or IE1 protocol (Tabata et al., 1996; Tabata et al., 1997), which is a subcategory of high intensity interval (HIIT) training. The IE1 protocol for this study began with an intense burst of exercise for 20 seconds, with a 10-second rest period. This was repeated four times with two sets of exercises, for a total of four minutes of HIIT training. Students were encouraged to perform the movements as fast as possible in 20 seconds with good form. For example, the exercise routine may have included the following order, or other similar movements, to comprise the four-minute routine:

- squat-thrusts x 20 seconds
- rest period x 10 seconds
- mountain climbers x 20 seconds

- rest period x 10 seconds
- jumping jacks x 20 seconds
- rest period x 10 seconds
- high knees x 20 seconds
- rest period 10 seconds
- repeat

For this research, the participants needed to complete the routine only twice. However, the IE1 protocol can be repeated up to 10 times in a single exercise session. The format of the daily exercise regimen (Appendix D) was created for the teacher who supervised the 15-minute exercise period. The routine was changed on a weekly basis in order to create novel motor skill movements. Thus, during anaerobic HIIT training, the body and brain are depleted of needed O₂, and breathing rate increases to replenish supply.

In an ideal experiment, the exercise routine would have been conducted in an exercise science lab under proper supervision using metabolic equipment to measure physiological changes in fitness. Measurement of student exercise performance was accomplished using five exercises adopted from the Insanity® workout routine Fit Test (Beachbody, LLC, Santa Monica, CA). The participants were instructed to perform the exercise movement while watching the video for 60 seconds, with a 20-second rest period between each of the five movements. The five movements included: (1) switch kicks, (2) power jacks, (3) power knees, (4) globe jumps, and (5) push-up jacks. They were required to count the number of repetitions they were able to perform in the time period and score them on the sheet provided. Pre-post scores were accumulated by adding the total repetitions across all five movements and dividing by five. The researcher observed

dramatic changes in the confidence, coordination, and skill of executing the movements from pre-post for all participants in the exercise and combined groups.

The combined group received both the chess and exercise treatment, while the control group received no treatment other than their normal daily curriculum schedule. Black, Isaacs, Anderson, Alcantara, and Greenough (1990) demonstrated that exercise, in the form of motor learning, produces distinct physiological brain changes, as compared to mental training that creates its own brain enhancing changes. Therefore, the hypothesis for this study is that a combined group, with both the potential increase in neovascularization and the potential increase in neurogenesis as caused by respective treatments, should receive the highest scores on the RSPM. The antithesis of this, the control group, hypothetically, should show flat results on both the VEGF/BDNF analysis and maintain the lowest outcomes on the RSPM due to lack of treatment.

Data Procedures and Analysis

The independent variables included the chess, exercise, combined, and control treatments (Table 8). The dependent variables included the protein biomarkers (BDNF and VEGF) and the cognitive test scores on the RSPM. The scores on the RSPM are converted into their intelligence quotient equivalent (IQ), which is accomplished in the following steps: (1) convert RSPM percentile rank using the scoring matrix RSPM table, (2) convert percentile number to a Z-score using a table for converting percentiles to Z's, and (3) convert z-score to matching IQ equivalent using a score conversion table for commonly used psychometric tests.

Table 8

Variables and Their Relationships

Control Variables	Independent Variables	Dependent Variables
Grade level	Chess intervention	Protein Biomarkers
Gender	Exercise intervention	Cognitive test scores
Chess experience	Combined intervention	
Exercise experience		
Special needs classification		

Statistically, once the RSPM score is converted into a z-score value, the score is multiplied by the standard deviation of the curve (s.d. = 15) and added to the mean of the curve (mean = 100) using a normal distribution curve for IQ. Additional data analysis is conducted using SPSS version 21 (SPSS IBM, New York, USA).

Summary

These processes allowed for the objective collection and analysis of protein biomarkers and intelligence test scores from young students pre and post treatment. These methods were intended to demonstrate the process by which learning, based on neurogenesis, can be assessed more holistically and accurately to allow for a better valuation of learning performance in the classroom. Chapter IV will describe analysis of the data and findings of the research.

CHAPTER IV: RESULTS

This section presents the findings of the research conducted. Four main findings were revealed among other interesting results: (1) the chess group increased cognitive test scores significantly greater than the control group, (2) the exercise group increased BDNF protein levels significantly greater than the control group, (3) the control group had a highly significant correlation between BDNF and RSPM scores, and (4) the combined group did not perform significantly higher on any analysis when compared to chess only or exercise only treatments.. This section will present an overview of the statistical analysis process, results from analysis of BDNF, VEGF, and RSPM measures in all four groups, and will conclude with a summary of discoveries.

Overview

The goal of the research was to provide an experimental basis for developing a more accurate definition of learning and its assessment capable of guiding future educational policy and practice. The theory underlying the investigation is based on the following linear projection:

Physical exercise → Neurogenesis → Intelligence increase → Learning performance

Physical exercise causes neurogenesis, which results in an increase in intelligence leading to improved learning performance. The measurement of this new definition of learning, based on the process of neurogenesis, was accomplished by measuring two proteins from human plasma, BDNF and VEGF, which are known switching mechanisms that move neural stem cells from proliferation to differentiation. Four research questions (RQ) were considered when designing this study:

1. Can chess and exercise each produce an increase in measures of intelligence?

2. Can a combined treatment produce an added increase in measures of intelligence more than chess only and exercise only?
3. Can chess and exercise each produce an increase in neurogenesis?
4. Can BDNF levels (as a biomarker of neurogenesis) be associated with increases in cognitive measures?

The methods conducted to answer these questions divided a young school-age population ($n = 38$) into four treatment groups: chess only (chess), exercise only (exercise), chess + exercise (combined), and no chess + no exercise (control). The groups included a fourth/fifth grade mixed cohort of gifted, normal, developmental, and special education students, both male and female. This study employed a randomized pretest-posttest control/comparison group experimental research design. Pre-post chess and exercise scores were collected, along with pre-post scores on a non-verbal intelligence test (RSPM) and two levels of proteins in the blood associated directly and indirectly with increases in neurogenesis (BDNF and VEGF).

Data collection and hypotheses. The data collection instruments utilized in this study were the Ravens Standard Progressive Matrices (RSPM) and RayBio® human ELISA kits for vascular endothelial growth factor (VEGF) and brain-derived neurotrophic factor (BDNF). The data collection instrument for cognitive outcomes consisted of the RSPM, which is a test of non-verbal intelligence that has a test-retest reliability range of .69 to .85 and a factorial validity range from .73 to .89 (Abdel-Khalik, 2005). Scores on the RSPM were converted to Wechsler Intelligence Scale for Children (WISC) IQ Equivalents, which converts the value from a norm standard of the RSPM to the normal distribution for IQ.

The data collection method for VEGF and BDNF utilized a commercially available ELISA kit protocol to analyze levels of the two proteins in human plasma. Results showed a test-retest reliability on the RSPM ($r(33) = .82, p = .000$), and BDNF ($r(33) = .56, p = .000$). The recovery rate of BDNF and VEGF in both pre and posttest were above 95%.

The treatment protocols for the various groups involved exercise, chess, chess + exercise (combined), and no chess-no exercise (control). A paired sample t-test was conducted for the subjects in each of the experimental groups who received treatment - chess, exercise, and combined groups - to determine the existence of a significant gain pre-post due to treatment. The chess group $t(1, 9) = -2.59, p = .029$, and the combined group $t(1, 9) = -3.77, p = .004$, performed significantly higher ($\alpha = .05$) on posttest chess scores than on pretest chess scores. The exercise group $t(1, 6) = -2.149, p = .076$, and the combined group $t(1, 9) = -2.16, p = .059$, performed only marginally higher on posttest exercise scores than on pretest scores.

Data from the RSPM, BDNF, and VEGF were analyzed using a mixed factorial design of within-subjects and between-subject's variables (Table 9). Within-subjects variables included the RSPM, BDNF, and VEGF test with two levels pre-post, and the between-subjects variables of the treatment condition with four levels of chess, exercise, combined, and control.

Table 9

Mixed Factorial Design of Within-subjects Factors and Between-subjects Factors

		Within-subjects Factors					
		BDNF (ng/ml)		VEGF (pg/ml)		RSPM score	
Between-subjects factors	Chess	Chess pre	Chess post	Chess pre	Chess post	Chess pre	Chess post
	Exercise	Exercise pre	Exercise post	Exercise pre	Exercise post	Exercise pre	Exercise post
	Combined	Combined pre	Combined post	Combined pre	Combined post	Combined pre	Combined post
	Control	Control pre	Control post	Control pre	Control post	Control pre	Control post

Although within-subjects design limits the potential for rejecting a false null hypothesis (type II error, β), a limitation of this study may have been the small size of the groups after attrition, which increases the chance of finding no effect, even if one actually existed. An additional inherent limitation of this design may have existed in a potential fatigue effect on the RSPM posttest.

The following hypotheses guided this investigation:

1. H_1 : Exercise intervention will produce an increased effect on cognitive measures of intelligence more than on the control group.
2. H_2 : Chess intervention will produce an increased effect on cognitive measures of intelligence more than the on control group.
3. H_3 : The combined group will produce an added effect on cognitive measures more than on the exercise only group.
4. H_4 : The combined group will produce an added effect on cognitive measures more than on the chess only group.
5. H_5 : Exercise treatment will produce an increased effect on BDNF levels more

than on the control group.

6. H_6 : Exercise treatment will produce an increased effect on VEGF levels more than on the control group.
7. H_7 : Chess treatment will produce an increased effect on BDNF levels more than on the control group.
8. H_8 : Chess intervention will produce an increased effect on VEGF levels more than on the control group.
9. H_9 : Increases in BDNF will be correlated with increases in cognitive measures.

Results of Analysis of BDNF, VEGF, and RSPM

The statistical analyses were conducted using IBM SPSS 21 statistical package. A two-way repeated measures ANOVA was conducted to compare the mean difference between the groups pre-post (Figure 7). Pre-post means and standard deviations are presented in Table 10. The observed power analysis was very strong for both VEGF (.996) and BDNF (.936), but not for RSPM (.075). Because of the small sizes of the groups, both significant and marginally significant ($p = 0.05 - 0.07$) will be reported.

Table 10

Pre-post Means and Standard Deviations

treatment	BDNF ng/ml				VEGF pg/ml				RSPM score			
	pre mean	SD	post mean	SD	pre mean	SD	post mean	SD	pre mean	SD	post mean	SD
chess	.57	.49	1.20	1.44	.00	.00	.011	.01	43.40	4.88	44.70	5.23
exercise	1.33	1.06	3.36	2.82	.00	.00	.026	.02	37.71	5.82	36.71	6.08
combined	.75	.48	1.99	1.73	.00	.00	.027	.04	40.50	8.07	41.70	5.96
control	.52	.55	.81	1.07	.00	.00	.016	.02	34.13	12.43	33.63	9.81

Note: chess ($n = 10$), exercise ($n = 7$), combined ($n = 10$), control ($n = 8$).

In all analyses, a post hoc pairwise Bonferroni correction was conducted for BDNF, VEGF, and RSPM with respect to all group comparisons. The Bonferroni correction is used in simultaneous tests of multiple hypotheses to guard against a false value of significance. As the number of hypotheses testing increases, the probability of getting a test to reach significance increases as well. The Bonferroni correction sets α/n , to reduce spurious positives.

RQ1: Can chess and exercise each produce an increase in measures of intelligence? A two-way repeated ANOVA (DV: IQ score; IVs: chess, exercise, control) showed no significant differences on the pretest IQ scores between the chess, exercise, and control groups. Figures 8 and 9 present the changes pre-post in both the exercise and chess groups versus the control group.

The same analysis revealed significant between-subjects effects, $F(3,31) = 4.421$, $p = 0.01$, of the groups. Pairwise comparison revealed a significant interaction between the chess group and the control group ($p = 0.01$). No significant between-subjects interactions were found for exercise vs. control or chess vs. exercise.

RQ2: Can a combined treatment produce an added increase in measures of intelligence more than chess only and exercise only? Multiple one-way ANOVAs (DV: IQ score; IVs: combined, chess, exercise) showed no significant differences on the pretest IQ scores between the combined and chess and exercise groups. The same analysis revealed no significant interaction between either the exercise group, ($p = 1.0$; Figure 10) or chess group ($p = 1.0$) (Figure 11) with the combined group. A post-hoc Bonferonni pairwise analysis revealed that the combined group did marginally greater than the control group ($p = 0.07$).

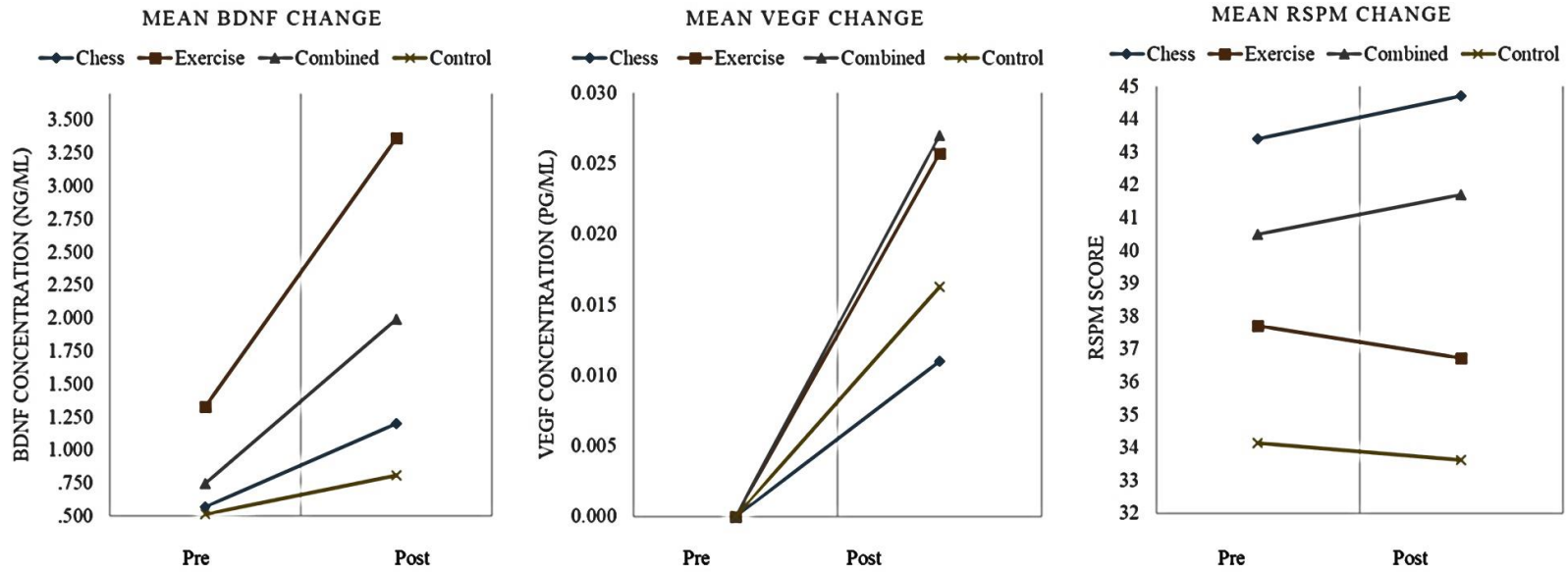


Figure 7. Mean changes of protein biomarkers, BDNF and VEGF, and RSPM within-subjects measures pre-post.

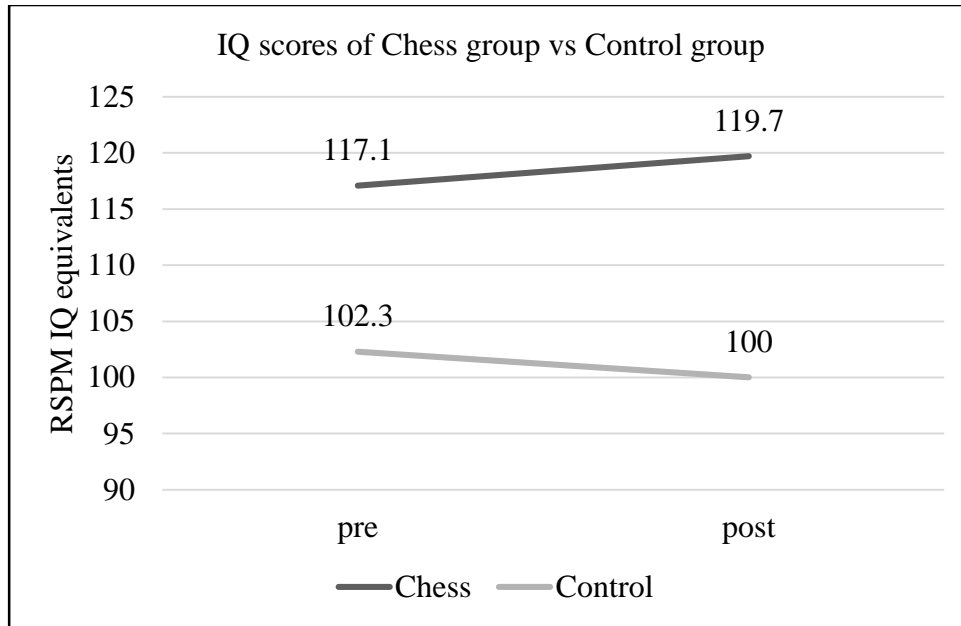


Figure 8. Chess and Control group pre-post score comparisons.

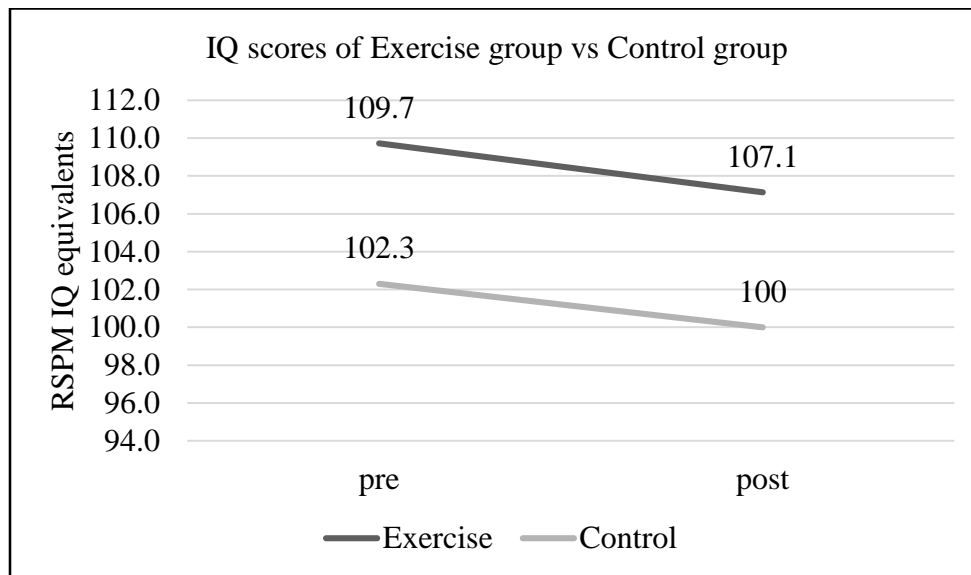


Figure 9. Exercise and Control group pre-post score comparisons.

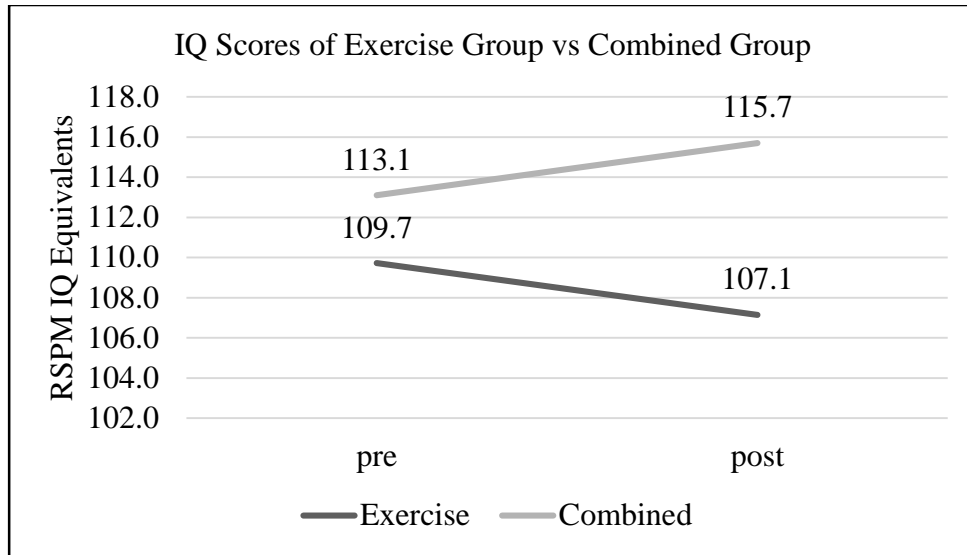


Figure 10. Exercise and Combined group pre-post IQ score comparisons.

Additional analysis of groups and IQ score effects. A paired sample t-test was used to compare the mean post treatment IQ scores of the groups who received chess treatment (chess, combined) against the IQ scores of the groups who did not receive chess treatment (exercise, control). Scores showed a highly significant difference ($p = .002$) (Figure 12) on post treatment IQ scores.

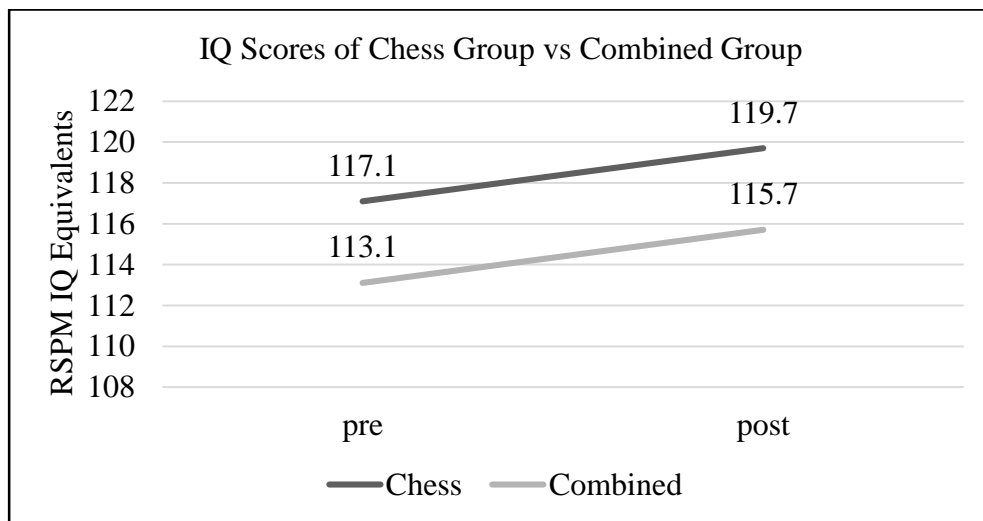


Figure 11. Chess and combined group pre-post IQ score comparisons.

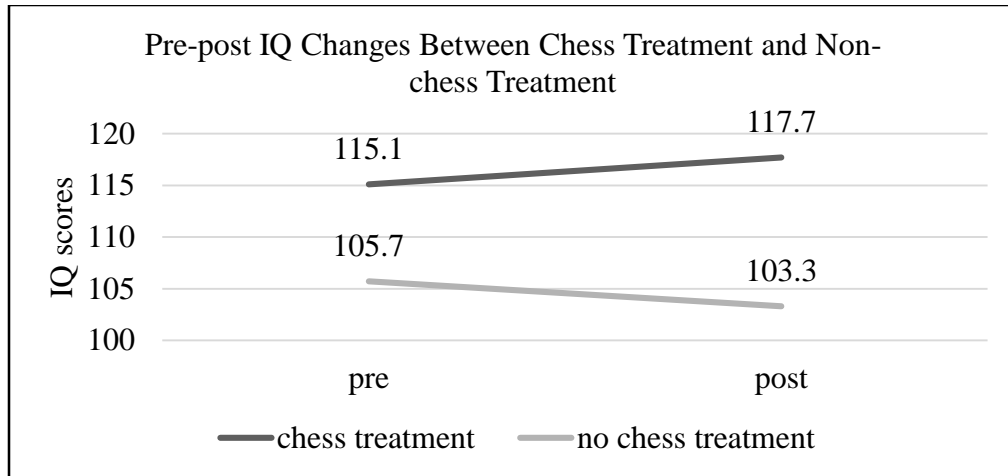


Figure 12. Pre-post IQ changes in the chess + combined groups versus the exercise + control groups.

The same analysis was conducted for those groups that received chess and exercise treatment (chess, exercise, combined) against the group that received no treatment (control). A highly significant difference was noted on post treatment IQ scores for those groups that received a treatment versus the control group that received normal curricular instruction ($p = .003$) (Figure 13).

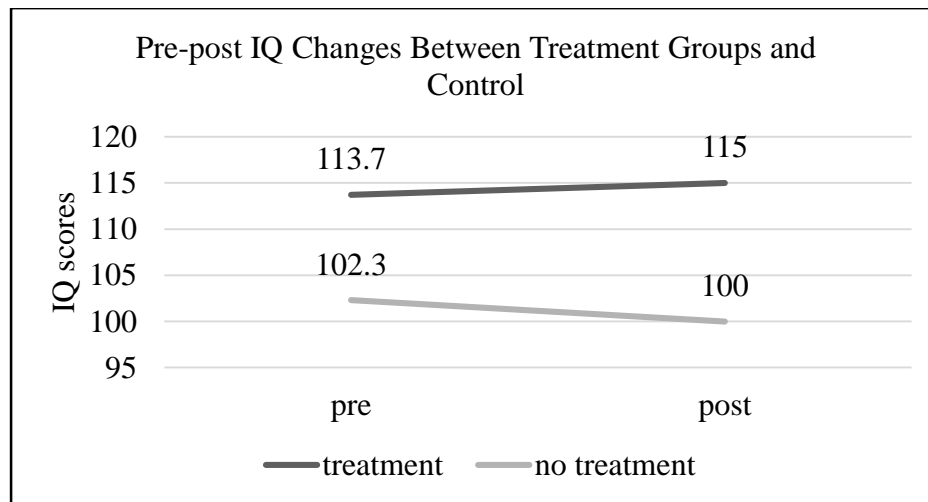


Figure 13. IQ score changes for treatment groups compared to no treatment group.

RQ3: Can chess and exercise each produce an increase in neurogenesis?

Multiple one-way ANOVAs (DVs: VEGF, BDNF; IVs: chess, exercise, control) showed

no significant differences on the pretreatment VEGF and BDNF plasma levels between the chess, exercise, and control groups. Between chess and exercise groups, only the exercise group significantly increased plasma levels of BDNF $t(1, 6) = -2.730, p = .034$ as a result of treatment. Neither the chess group $t(1, 9) = -1.247, p = .244$, nor the control group $t(1, 7) = -.615, p = .558$, showed significant changes on BDNF levels.

Due to the very small amounts of VEGF in the blood stream, all participants began with a score equivalent to 0.00 pg/ml. All groups - chess ($t(1, 9) = -2.905, p = .02$); exercise ($t(1, 6) = -2.789, p = .03$); and control ($t(1, 9) = -2.303, p = .06$) – significantly increased VEGF plasma levels as a result of treatment.

Figure 14 shows the differences between the BDNF plasma levels pre-post for the chess and control groups. Multiple one-way ANOVAs (DV: BDNF level; IVs: chess, exercise, control) revealed significant between-subjects effect, $F(1, 3) = 2.940, p = 0.05$, on posttest BDNF levels. A Bonferonni adjusted pairwise comparison demonstrated that the significant interaction did not exist in the chess/control comparison ($p = 1.0$), but existed in the exercise/control comparison ($p = 0.06$) (Figure 15).

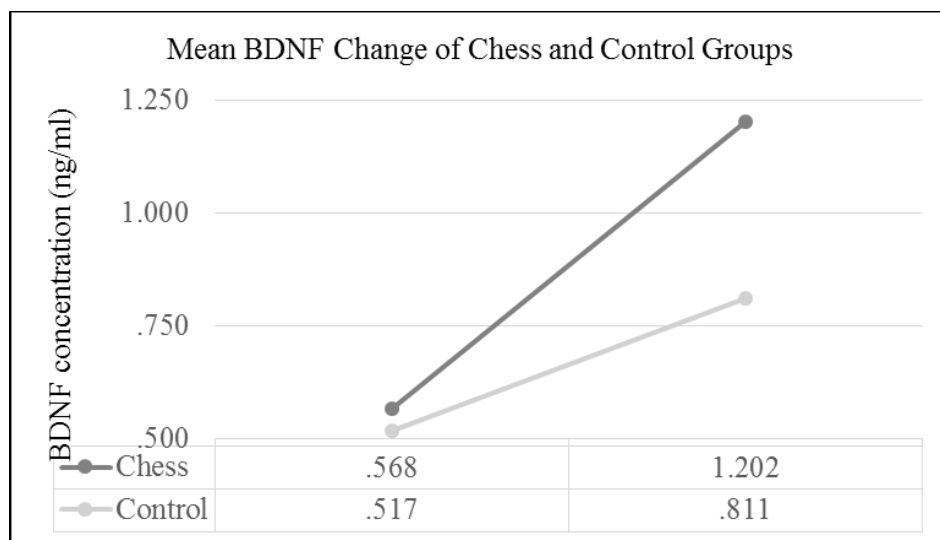


Figure 14. BDNF Plasma level changes for chess and control groups pre-post treatment.

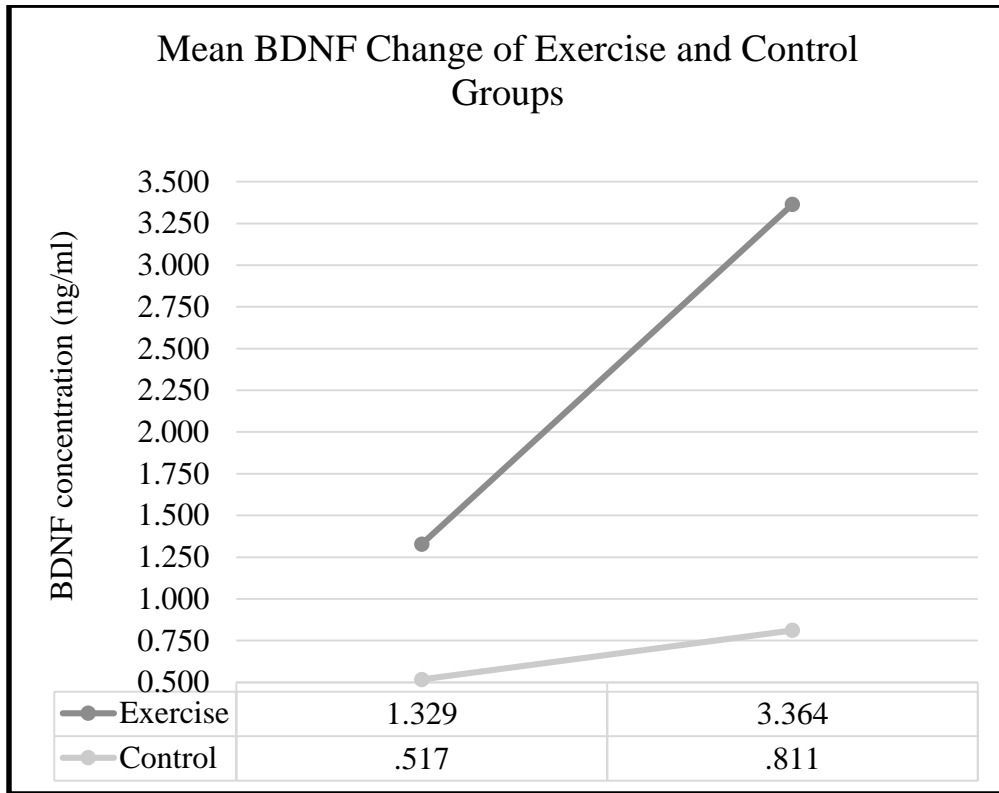


Figure 15. BDNF Plasma level changes for exercise and control groups pre-post treatment.

Figures 16 and 17 show the differences between the VEGF plasma levels pre-post for the chess and control group comparison and the exercise and control group comparison. The control group (0.016 pg/ml) demonstrated a greater increase in VEGF levels after treatment than the chess group (0.011 pg/ml). A one-way ANOVA (DV: VEGF; IV: chess, exercise, control) revealed no between-subjects effect, $F(1, 3) = .898$, $p = 0.453$, on posttest VEGF plasma levels in either the chess/control comparison or the exercise/control comparison.

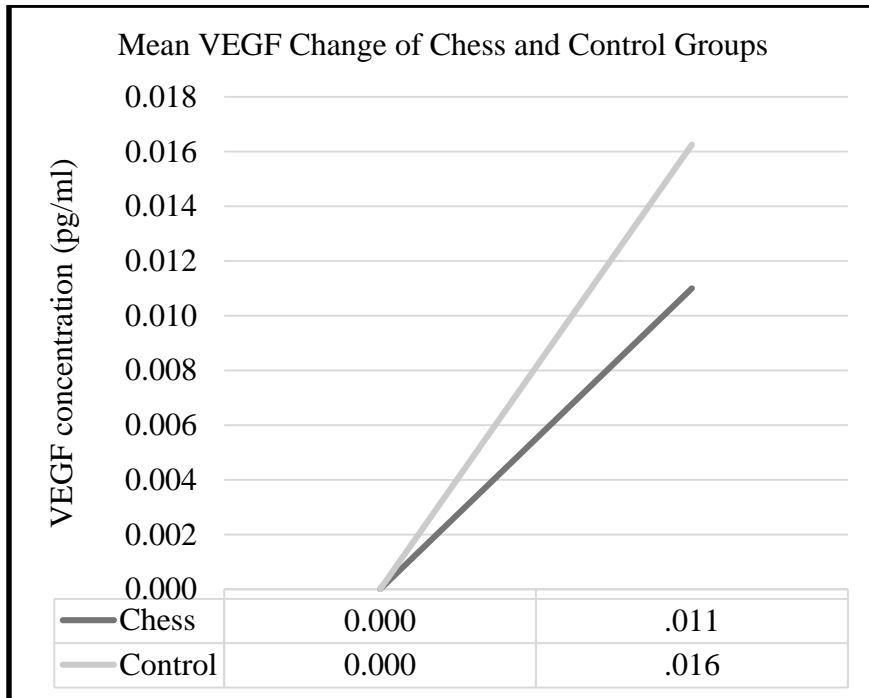


Figure 16. VEGF Plasma level changes for chess and control groups as a result of treatment.

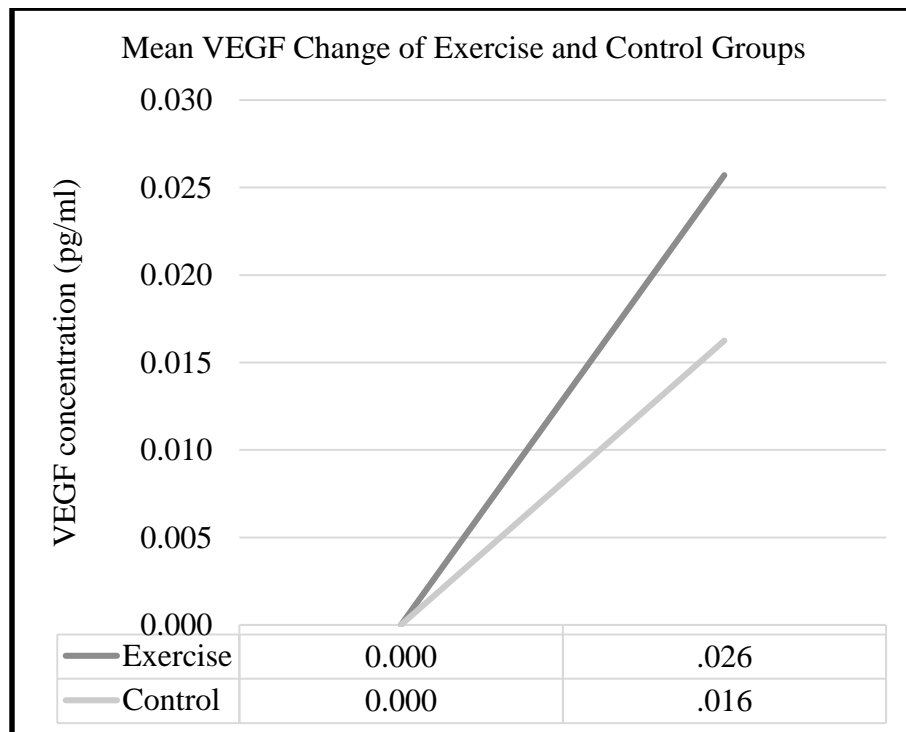


Figure 17. VEGF Plasma level changes for exercise and control groups as a result of treatment.

Additional analysis of the combined group on increasing neurogenesis.

Additional analysis of the combined group, as compared with the chess, exercise, and control groups with respect to BDNF levels, was conducted to determine effects of pre-post treatment (Figure 18). Multiple one-way ANOVAs (DV: BDNF; IVs: combined, chess, exercise, control) showed no significant differences on the pretreatment BDNF plasma levels between the four groups. As a result of treatment, the combined group demonstrated a significant increase in BDNF levels from pre to post measurement ($t(1,7) = -2.527, p = .03$). A repeated measure ANOVA with Bonferroni adjustment for multiple pairwise comparisons demonstrated no significant difference of any group, compared with the combined group on pre-post BDNF level changes.

RQ4: Can BDNF levels (as a biomarker of neurogenesis) be associated with increases in cognitive measures? A Pearson Product Moment analysis was used to determine the existence of a positive correlation between intelligence and neurogenesis. To perform this test, data from only a subsample of subjects was chosen from those who had both a positive increase in BDNF and a positive increase on RSPM scores (Table 11).

Table 11

Means and Standard Deviations of Population Subsample used for Correlation Analysis

	RSPM	SD	BDNF	SD
Chess	5	5.66	1.92	0.16
Exercise	2	1	2.26	2.24
Combined	1	0	1.66	1.05
Control	5.5	0.71	0.34	0.4

Note: chess ($n = 2$), exercise ($n = 3$), combined ($n = 3$), control ($n = 2$).

Results revealed no correlation between increases in RSPM and increases in BDNF measures ($r(8) = -0.29, p = .17$). A separate correlation was conducted using *change* in

RSPM and BDNF measures pre-post among all subjects ($n = 38$). Analysis showed a strong negative correlation between RSPM change and BDNF change pre-post for the combined group, $r(8) = -0.68$, $p = .03$. Total correlation of all subjects scores between RSPM and BDNF change pre-post was significantly negatively correlated, $r(33) = -0.44$, $p = .008$. Of all groups, only the chess group had a very small positive correlation trend $r(8) = .138$, although not significant. The same analysis was conducted on IQ scores. Surprisingly, the only significant correlation was found in the control group whose results showed a very high positive correlation $r(6) = 0.71$, $p = .049$ between BDNF and IQ post treatment.

Summary of Findings

This chapter presented the empirical results of the analyses related to the four research questions and nine hypotheses that guide this investigation. The four main results include: (a) the chess group increased IQ significantly more than the control group as an effect of treatment; (b) the exercise group increased BDNF protein levels significantly more than the control group as an effect of treatment; (c) the control group had a highly significant correlation between BDNF and IQ scores; and (d) the combined group did not demonstrate, in any comparison, significantly better improvements when compared to chess only or exercise only treatments. Another significant finding included: (e) for the two groups who received chess as a treatment (chess, combined), a highly significant pre-post IQ increase was noted compared to the groups who received no treatment (exercise, control).

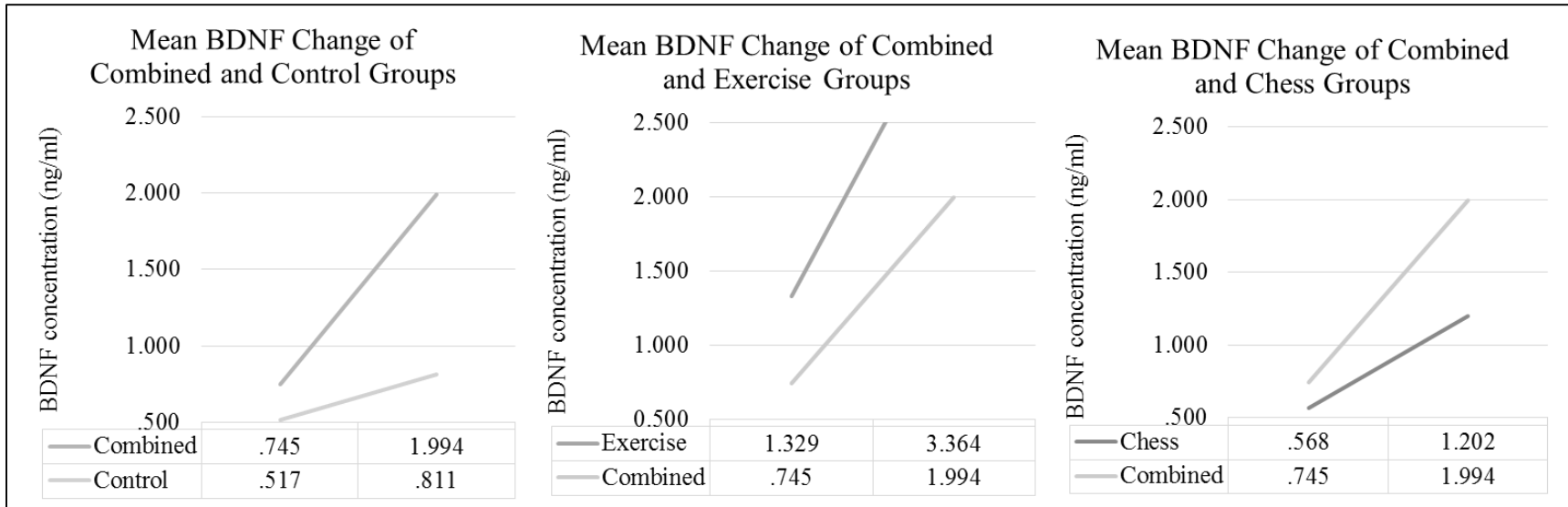


Figure 18. BDNF Plasma level changes for combined group as compared to control, exercise, and chess groups as a result of treatment.

The data analysis also revealed a few interesting, although statistically non-significant, results: (a) the control group had higher post treatment VEGF plasma levels than the chess group; and (b) the chess group had the only positive correlation between RSPM score and BDNF score, although it was very small and non-significant.

These findings warrant acceptance and rejection of certain hypotheses related to this study (Table 12). The four research questions that were considered during this analysis were:

1. Can chess and exercise each produce an increase in measures of intelligence?
2. Can a combined treatment produce an added increase in measures of intelligence more than chess only and exercise only?
3. Can chess and exercise each produce an increase in neurogenesis?
4. Can BDNF levels (as a biomarker of neurogenesis) be associated with increases in cognitive measures?

With respect to Research Question One, chess increased IQ significantly more than the control group post treatment, while exercise did not. Therefore, the first hypothesis is rejected and the second is accepted. With respect to Question Two, while the combined group significantly increased BDNF levels from pre-post and showed marginally significant improvement on IQ scores, no analysis revealed that the combined group did significantly better on measures than either the chess or exercise only groups. Therefore, in this study, hypotheses three and four warrant rejection. With respect to Question Three, exercise had a profound effect on increasing BDNF levels, while chess had no effect. Additionally, although all groups demonstrated significant increases from pre-post on levels of VEGF, no group experienced any significant increase above the

control group. Therefore, these results warrant acceptance of hypothesis five and the rejection of hypotheses six, seven, and eight.

Table 12

The Acceptance or Rejection of the Hypotheses and Their Relationship to the Four Questions

RQ#	H#	Alternate Hypothesis	Verdict
RQ1	1	Exercise intervention will produce an increased effect on cognitive measures of intelligence more than on the control group	<i>Rejected</i>
	2	Chess intervention will produce an increased effect on cognitive measures of intelligence more than on the control group	<i>Accepted</i>
RQ2	3	The combined group will produce an added effect on cognitive measures more than on the exercise only group	<i>Rejected</i>
	4	The combined group will produce an added effect on cognitive measures more than on the chess only group	<i>Rejected</i>
RQ3	5	Exercise treatment will produce an increased effect on BDNF levels more than on the control group	<i>Accepted</i>
	6	Exercise treatment will produce an increased effect on VEGF levels more than on the control group	<i>Rejected</i>
	7	Chess treatment will produce an increased effect on BDNF levels more than on the control group	<i>Rejected</i>
	8	Chess treatment will produce an increased effect on VEGF levels more than on the control group	<i>Rejected</i>
RQ4	9	Increases in BDNF will be correlated with increases in cognitive measures	<i>Rejected</i>

Last, Question Nine attempted to understand whether a relationship existed between increases in BDNF levels and increases in cognitive measures. No such relationship was demonstrated based on increases in scores. Therefore, hypothesis nine warrants rejection based on these results. Chapter V will discuss the importance of these results in light of existing literature.

CHAPTER V: DISCUSSION

The goal of this study was to contribute to a foundation for the future development of a more accurate definition of learning based on a biological process, neurogenesis, which is capable of guiding future educational policy and practice. An experiment was conducted in an attempt to cause an increase in neurogenesis to occur in an elementary school-age population. Neurogenesis was measured indirectly by testing for two proteins in human blood, BDNF and VEGF. Two treatments were chosen to induce increases in neurogenesis: chess and exercise. The general theory was described in a linear process stating that increases in neurogenesis would produce increases in intelligence, which would produce improvement in learning performance:

Physical exercise → Neurogenesis → Intelligence increase → Learning performance

The purpose of this investigation was to more accurately define learning based on the biological process of neurogenesis. The method attempted to (1) cause an increase in neurogenesis and intelligence, (2) measure those changes, and (3) correlate neurogenesis to intelligence. The research questions were: Can chess and exercise each produce an increase in measures of intelligence? (Q₁); Can a combined treatment produce an added increase in measures of intelligence more than chess only and exercise only? (Q₂); Can chess and exercise each produce an increase in neurogenesis? (Q₃); and Can BDNF levels be associated with an increase in cognitive measures? (Q₄).

When presenting the finding and implications of this research, it is important to recognize the main inherent and emergent limitations:

- There is no means of control for potential genetic variances in basal or production levels of BDNF/VEGF.

- No chess expert provided chess instruction, limiting the learning opportunity of the groups who had contact with chess
- Sample size of the groups being less than 10 limits findings.
- There was only a single measurement of proteins pre-post.

This research found four main effects: (1) the chess treatment demonstrated a more immediate impact on cognitive performance by significantly increasing scores on tests of intelligence, (2) the exercise treatment demonstrated a more immediate impact on the process of neurogenesis by significantly increasing levels of both VEGF and BDNF, (3) the control group was found to have a highly significant correlation between levels of neurogenesis and intelligence, and (4) the combined treatment did not perform significantly better on any analysis when compared to chess only or exercise only treatments.

This chapter presents the summary and interpretation of findings, along with their context and implications, in the order of the research questions that guided the study. In addition, this section discusses recommendations for future research in this field.

Q1: Can Chess and Exercise each Produce an Increase in Measures of Intelligence?

While the findings show no effect of exercise on intelligence, both groups that received chess intervention, chess only and combined, demonstrated significant increases in intelligence versus the groups that did not receive chess treatment. This demonstrates that adequate amounts of chess play/learning, in accordance with the extensive amount of literature, cause an increase in intelligence.

Chess and intelligence. The findings of this research demonstrate a significant cause-effect relationship between chess and intelligence for both groups that received

chess treatment: chess and combined. These findings support previous chess studies on chess and intelligence of young players. Horgan and Morgan (1990) provided interesting context in light of current results. The mean grade and post RSPM results in the current study (4.6/42.5) compare nicely with their elementary mean grade and post RSPM score (4.2/37.7) and mean grade and post RSPM score of their junior high cohort (8.3/53.3). This trend substantiates the causal mechanism of chess to increase intelligence, simultaneously with expertise, over time (Figure 19). The results likewise help to establish the *learning curve*, as described by Hunt and Navalta (2012). Additionally, in agreement with the results from Frydman and Lynn (1992) and Bilalić et al. (2007), the findings in the current study continue to establish the learning curve from novice to expert, in accordance with intelligence increases, as an effect of chess play/learning (Figure 20). These figures clarify the previous suggestion by Hunt and Navalta (2012) that a learning curve, based on the operational definition provided herein, exists for cognitive development (figure 21). These findings indicate that chess can be used in schools to accelerate and rehabilitate intelligence and disabilities of the students.

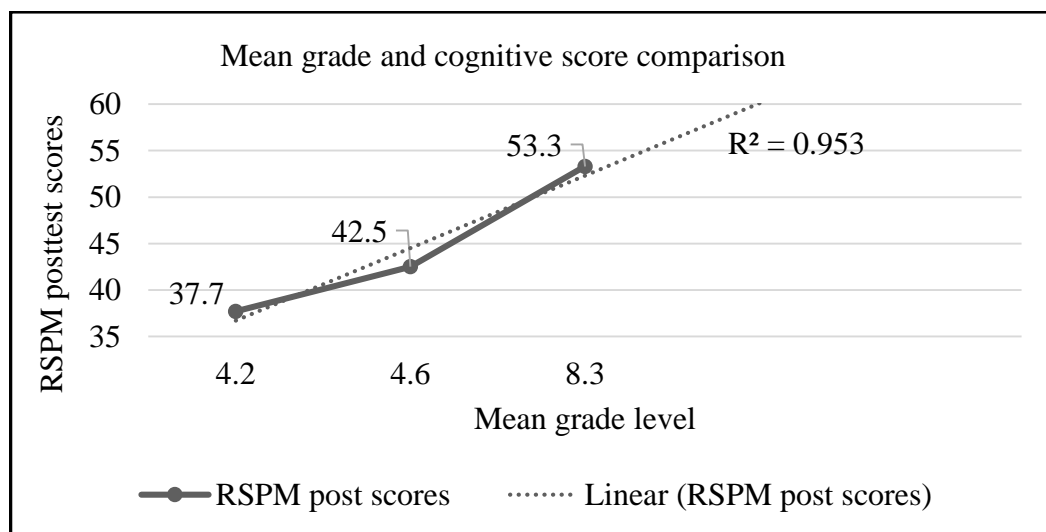


Figure 19. Current posttest RSPM scores after chess treatment, as compared to Horgan and Morgan (1990).

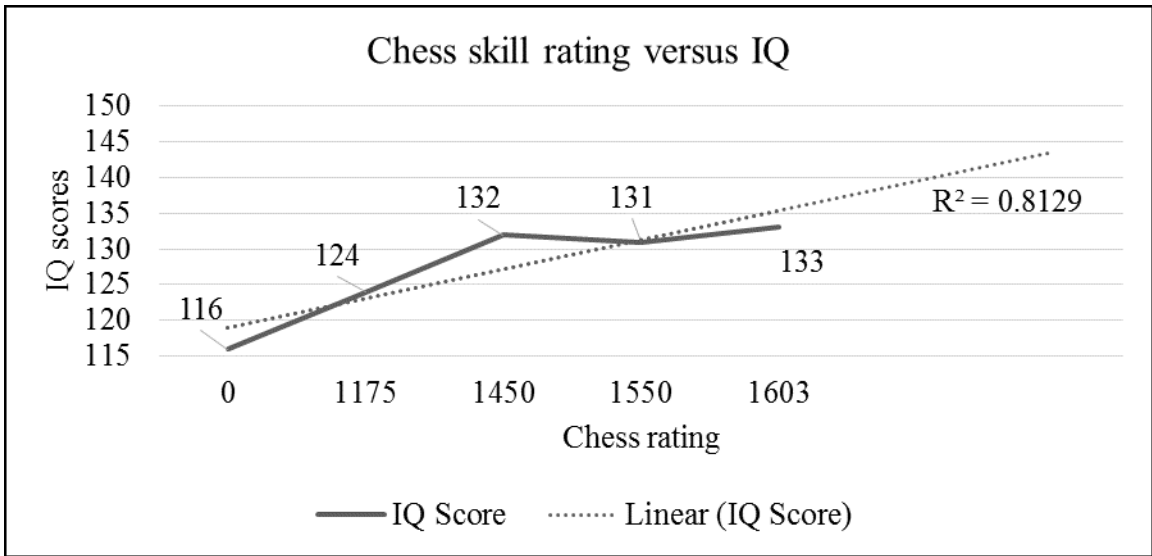


Figure 20. Current findings in relation to chess rating to IQ scores as compared to Frydman and Lynn (1992) and Bilalić, et al. (2007).

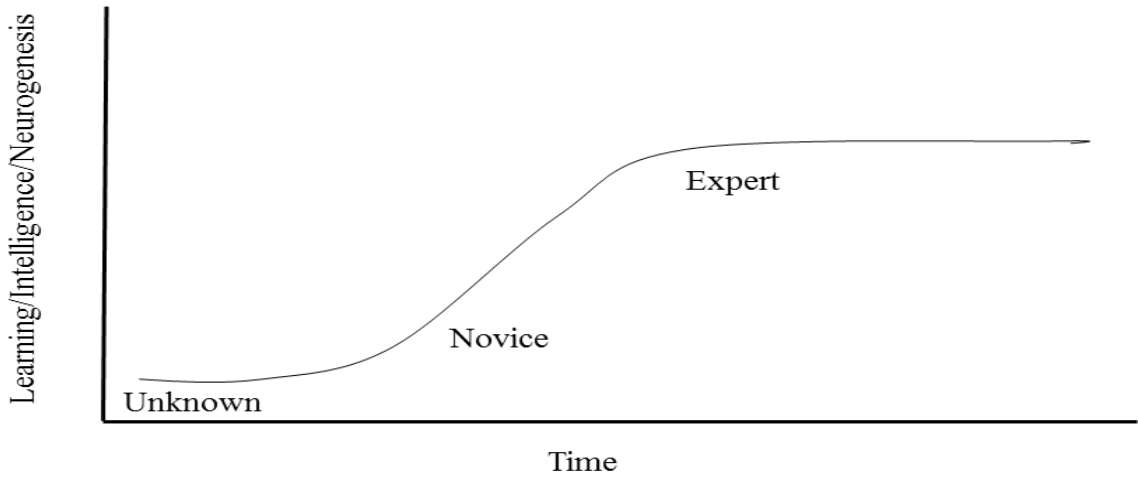


Figure 21. The Learning Curve.

There appears to be a divergence of chess expertise and the linear IQ line (Figure 20). This may be a predictable relationship, which, as noted in the literature review and by Bilalić et al. (2007) that the difference of IQ between expert-master chess players and highly intelligent non-chess players ceased to exist as chess levels continue higher. This appears to happen in this graph at an approximate rating level of 1600, which serves to

underscore the suggestion that IQ testing may have a limitation in regard to assessing such relationships.

Exercise and intelligence. Of all literature reviewed, only Sparrow and Wright (1993) used the RSPM as a test of intelligence pre-post exercise. The results of the current study match their findings. In both studies, no between-group differences and no pre-post differences were found between physical exercise and intelligence, as measured by the RSPM. Sparrow and Wright used a different duration of exercise (an acute regimen lasting 6 minutes), a different mode of exercise (a step-up task), and a different population (50 men, mean age = 24.8). They concluded that acute, short duration exercise had no immediate effect on intelligence. The current research added to the literature by demonstrating that chronic short-duration exercise in young children also showed no pre-post improvement in intelligence when measured by the RSPM.

The literature review determined that a timeline exists that demonstrated a short-term increase in cognitive performance post exercise (Cooper et al., 2013). However, this was not seen when using the RSPM to measure increases in cognitive performance, but was demonstrated using other forms of cognitive and academic measures after acute exercise regimens (Brunelli et al., 2012; Tang et al., 2008; Winter et al., 2007; Tables 4-6).

A potential explanation may be due to limitations of the validity of the RSPM test to measure intelligence variances associated with exercise. No studies were found that compared various cognitive test outcomes as an effect of PAE, and only one other study was found to use the RSPM to measure intelligence pre-post PAE. This suggests that there may be a difference between intelligence, as caused by exercise, and intelligence

caused by other factors, i.e., mental exercise. Yet, according to the literature, although not found in the current study, an improvement in learning performance due to intelligence increases caused by PAE has been demonstrated.

Q2: Can a Combined Treatment produce an Added Increase in Measures of Intelligence More Than Chess Only and Exercise Only?

Although the combined group performed marginally better than the control group on measures of intelligence, no between-group differences were noted when compared to chess only and exercise only groups. This result is contradictory to the literature (Fabre et al., 2002). Depending upon the level of chess expertise of the subjects, a positive relationship exists between chess and intelligence. Chess, in the current study, demonstrates a significant effect on intelligence over controls. Likewise, a positive relationship exists between increases in PAE and increases in BDNF and cognitive performance, particularly with the regions of the brain associated with intelligence (Lee et al., 2014).

This study was the first to attempt to use a daily chess routine to determine the effects on intelligence. Additionally, this is the first study to determine the effect of the Tabata protocol on increases in intelligence. The methods used were developed with the consideration of using chess and exercise daily in the classroom, without disruption of normal curriculum.

The duration of the research was only nine weeks, which did not allow the benefit of a combined treatment effect on test subjects. However, when comparing the two groups having chess instruction (chess & combined) with the groups that did not receive any chess treatment (exercise & control), a significant difference was demonstrated in

intelligence levels of those groups receiving chess treatment versus the groups that did not. This finding suggests that the duration of the research was long enough to provide a chess effect on intelligence, but no exercise effect on intelligence.

Previous literature recapitulates that a timeline exists for functional integration of new neurons. The time frame from proliferation to integration may take from 2-4 weeks up to four months and to reach maturation. Additional amounts of time may be required to reach a positive outcome on cognitive assessment. The total amount of contact time for both chess and exercise in the current study is equivalent with the literature. However, the duration of the study in regards to chess (Aciego et al., 2012; Barrett & Fish, 2011; Hong & Bart, 2007; Kazemi et al., 2012; Scholz et al., 2008; Smith & Cage, 2000) and the mode (Tabata Protocol) relative to exercise differed from the previous literature.

However, results of the chess and combined groups may indicate that chess, at lower levels of initiation, causes a more immediate morphological effect on existing neural networks related to intelligence. Additionally, combined treatment may be predicted to cause an added effect in longer duration studies.

Q3: Can Chess and Exercise Each Cause an Increase in Neurogenesis?

The purpose of this question was to target the underlying causes of increased cognitive ability as an effect of exercise and chess in previous studies. The hypothesis stated that both chess and exercise would increase neurogenesis. The findings demonstrate that exercise treatment had a significant effect on plasma levels of BDNF, while chess did not.

Exercise and neurogenesis. Hunt and Navalta (2012) traced the causal mechanism behind the pluripotent, paracrine messenger, nitric oxide (NO), to be oxygen

(O₂) intake across the nasal sinus. This has a profound effect on physiology relating to O₂ uptake of the vasculature, musculature, and even how NO influences electrical stimulation and morphology in the brain. In a cascading mechanism, increases in PAE will increase intake of O₂ across the sinus, which increases NO effects on physiology. One of those effects is on the production of VEGF. Increases in NO, as a switching mechanism, lead to increases in VEGF. As traced in the literature review, research demonstrates that VEGF is a mediator between NO and BDNF (Cao et al., 2004; Fabel et al., 2003; Jin et al., 2002; Louissaint et al., 2002). BDNF is the known switching mechanism that initiates neurogenesis (Cheng et al., 2003). The causal mechanism of the cascade between increases in VEGF, producing increases in BDNF, and producing increases in neurogenesis is increases in NO as a result of inhalation across the nasal sinus stimulated by increases in PAE. Therefore, the findings of this research suggest that increases in PAE may also lead to increases in neurogenesis. This discovery was made through the measurement for increases in VEGF and BDNF as a result of PAE. Although this cascade, with emphasis on the role of BDNF/VEGF initiation of neurogenesis, was nearly immediate in physiology, the time course for deriving an outcome of intelligence is much more deliberate in practicality. In fact, Louissaint et al. (2002) showed that a delayed onset of upregulation of VEGF (two weeks) and BDNF (three weeks) may exist in relation to the hormone testosterone which increased as a result of exercise in humans (Hackney et al., 2012). This matched the time scale from proliferation to maturation as described by Ming and Song (2005) and Deng et al. (2010).

Understandings of these cascades and time frames help to illuminate the implications for reasons that a positive significant result was not found in the exercise

and combined groups in relation to intelligence scores. A lack of time allocated to this research to reach summation of neurogenesis at a system level prohibited observance of increases in intelligence on the RSPM. In any case, the results of this study show a significant effect of exercise treatment, specifically the Tabata protocol, to increase levels of BDNF and VEGF in young children. A host of studies have demonstrated that increases in BDNF levels are associated with increases in cognitive performance as a result of both acute and chronic PAE (Brunelli et al., 2011; Griffin et al., 2011; Lee et al., 2014; Rasmussen et al., 2009; Tang et al., 2008; Winter et al., 2007).

Chess and neurogenesis. No studies were found in the literature that measured BDNF levels or VEGF levels as a result of playing chess. Additionally, no studies implicated chess as a correlate for increases in neurogenesis. Several studies led to an inference that chess might increase BDNF/VEGF and produce an increase in neurogenesis. Primarily, chess was shown to increase cognitive performance and should follow the same biological cascade as demonstrated for exercise to achieve neurogenesis, which was hypothesized in this study as the causal mechanism for increase in intelligence. Additionally, research showed that chess play likewise increases testosterone, which had effects on BDNF/VEGF modulation.

The results in this study show no between-group differences of chess on BDNF or VEGF. In fact, even the control group had higher levels of VEGF over time than the chess group. The main difference between chess and exercise treatment, in achieving similar results on BDNF/VEGF to initiate increases in neurogenesis, rests in the fact that chess play does not increase the intake of O₂ across the nasal sinus.

A lack of cause was apparent for chess to initiate the increased activity of the

physiological cascade that leads to increases in BDNF/VEGF and neurogenesis.

Therefore, the mechanism by which chess leads to increases in intelligence appears to differ significantly from the mechanism by which exercise may lead to increases in intelligence. Also revealed is the finding that the time courses that lead to increases in intelligence are different for both chess and exercise treatments. One may surmise that chess has a more immediate impact on existing, functioning neural networks associated with intelligence, whereas exercise has a more immediate impact on the production of new neural morphology that builds stronger neural networks associated with increases in intelligence. This substantiates the prediction that research using the same methods, but longer duration, may demonstrate a robust combined effect of chess plus exercise treatment on increasing intelligence.

Q4: Can BDNF levels be associated with increases in cognitive measures?

The purpose of this question was to determine whether neurogenesis can be correlated to increases in intelligence. The hypothesis stated that increases in BDNF will be correlated to increases in cognitive measures. The finding demonstrates two significant relationships: (a) a highly significant negative correlation exists between all subjects' changes in values pre-post between BDNF and RSPM, and (b) a very high significant correlation is demonstrated in the control group between IQ and BDNF values post treatment.

Based on the literature, it was not surprising to discover a strong positive correlation in the control group on values of BDNF and RSPM. First, in all measures, the control group saw no significant changes, and therefore, remained the most stable. Second, Haier et al. (1992) found that the highest brain activation was in the naïve,

unpracticed group that demonstrated the lowest cognitive test scores. The current study's results appear to match their results, in that the control group had the lowest IQ scores and the least amount of change in BDNF.

Haier et al. (1992) concluded that general intelligence related more to new learning, which would suggest that those children who received no treatment did not increase learning. As new learning was initiated, the treatment groups saw increases pre-post on measures of intelligence and neurogenesis. This gave context to the other finding that a negative correlation exists between change in BDNF and change in RSPM scores of all subjects. According to the brain efficiency hypothesis, multiple negative relationships exist between brain activation and intelligence (Haier et al., 1988; Haier et al., 1992); brain activity and expertise (Amidzic et al., 2001); BDNF/VEGF levels and cognitive performance (Lee et al., 2014), and exercise and brain activation (Kubitz & Pothakos, 1997). The findings from this study appear to corroborate those in support of the brain efficiency hypothesis. Also, as no correlation was found in the chess group, or the exercise or combined group, between BDNF and RSPM scores, other factors are suggested: (a) the chess group, which improved RSPM, saw no improvement in BDNF; (b) the exercise group, which saw an increase in BDNF, saw no increase in RSPM; and (c) the duration of the study was not long enough to realize a combined effect. These factors suggest that neurogenesis may be a disruptor of achieving improved cognitive outcomes until reaching a level of functional integration. Once functional integration is achieved, mental exercises such as chess continue to impact the morphology of neural networks from functional integration to summation in order to accomplish increases in intelligence.

Recommendations for Future Research

Based on the results, two lines of future study are implicated. The first is relative to educational neuroscience; the other is relative to brain/learning disability rehabilitation. Simply, future research could reproduce this study with larger populations and a longer duration. The research could be conducted with single types of populations that exhibit learning disabilities/impairments: ADHD, Autism spectrum disorders, etc. Another means of manipulating the subject groups would be to stratify based on age in those with deficits due to Alzheimer's or brain insult. A recommended method to manipulate the length of the study would be to measure changes longitudinally at different time points, which would help to develop a more accurate learning curve.

Another means of expanding knowledge in this area would be to add manipulators to the treatments to compare individual and combined condition. Several well-researched nutritional supplements exist, including EPA/DHA fatty acids, ginkgo biloba, and other nutritive and herbal medicine substances known to increase neural stimulation. Replication of the current study could validate the use of blood protein correlates of neurogenesis with the inclusion of imaging technologies such as fMRI, fNIRS to measure cortical changes, and PET/EEG to measure regional activation. Such increased investigations would become useful tools for the fields of education and brain rehabilitation for treatment and remediation of brain related and mental health disabilities.

Conclusion

The significance of the research rests on the observation that an increase in the brain efficiency of subjects was demonstrated through increases in intelligence for the

chess group, and increases in BDNF as a marker for neurogenesis for the exercise group. This demonstrates that the underlying biological constraints inhibiting the acquisition of skill, knowledge, and expertise can be loosened and enhanced in a way that leads to improved learning performance. This is illustrated uniquely by findings that showed exercise to increase neurogenesis, and to capitalize on the effect, chess was shown to assist the functional integration of new neurons to the point of exhibiting increases in intelligence.

This study is not only important for educational policy-makers, as well as a host of populations that experience brain disorders and/or learning impairments. The findings can help to inform several fields of science and may impact millions of students' and patients' cognitive rehabilitation and learning performance. Perhaps even more significant is the redefinition of learning and its valid and objective assessment. This research demonstrates that BDNF and VEGF, as potential markers of human neurogenesis, may be measured in young children *in vivo* and associated with levels of intelligence. Therefore, this simple blood test exists as a measure of a holistic, modern definition of learning over time. That operational definition is:

Learning: The intrinsic six-stage process that moves adult-born neuron(s) from proliferation to summation (neurogenesis), as induced by external factors, and leading to increased intelligence and cognitive performance.

The bridge too far, described by Bruer (1997) as being between neuroscience and education, has been spanned by the current investigation offering improved definitions and understandings of “learning,” “neurogenesis,” and even education. That bridge allows both neuroscience and education research to meet at the precipice of advancing

human understanding of intelligence and learning performance. Last, this study helps to illuminate the totality of chess studies since 1877, revealing the cause of why chess appears as a panacea to improve so many behavioral outcomes of psychological studies. Chess improves the efficiency of the neural networks related to intelligence; intelligence being a transferrable skill to many domains requiring higher cognitive ability. Educational institutions seeking to improve learning performance outcomes should adopt chess and exercise as in classroom activities on a daily and weekly basis.

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APPENDIX A1: Informed Parent and Child Consent Forms

Informed Consent Form for Parents Whose Children Can Participate in WKU Educational Leadership Research Study at Clarkson Elementary

This informed consent form is for adults whose children we are inviting to participate in research that will determine if a child's involvement in exercise and chess could improve their learning ability and enhance their natural brain development.

Principle Investigator: Samuel J Hunt

Name of Organization: Western Kentucky University

Department: Educational Leadership Doctoral Program

Faculty Sponsor: Dr. Barbara Burch

Project Title: *A Novel Use of Biomarkers: Predicting and Assessing Academic Ability*

This Informed Assent Form has two parts:

Information Sheet (gives you information about the study)

Certificate of Assent (this is where you sign if you agree to allow your child to participate)

PART I: Information Sheet

Purpose: Why are we doing this research?

We want to find better ways to help children perform better in school and improve their brain development. We have chosen two activities that are fun, smart, and healthy for students. In order to find out if students doing exercises and playing chess every day could help them develop their brain faster and become smarter and healthier; we have created this research study.

We would like for your child to be a part of this research study, which would ask them to participate in exercise and chess routines, take a simple assessment (not for a grade), and participate in a simple blood test that will draw some blood from their arm to test for 2 specific proteins only. We are conducting this research to understand more about how to improve children's academic ability and how that relates to their natural brain development. Everything is completely confidential and your child cannot be identified in any way with their test scores or their blood sample.

Participation is voluntary: Does my child have to do this?

We are testing chess and exercise on children at this time due to the benefits that children might receive developmentally from such a program. Clarkson Elementary offers a large and diverse population of children to choose from. It is entirely up to you whether you agree to have your child participate in this research. They/ You may also choose to change your mind later and have your child stop participating, even if you agreed earlier, there are no penalties for withdrawing. Your child's education will not be affected by your decision. You may contact me or my research advisor, your school's principal or the superintendent. **Procedures: What is going to happen to your child, what are your responsibilities?**

If you allow your child to help out with our research, we will:

- Place them in groups that may follow chess, exercise, or both routines while receiving their normal education
- Have them complete a quick test before and after their participation in the research
- Have a certified phlebotomist quickly take about 5 tablespoons of blood from a

vein or hand before and after their participation in the research (lasts approximately 10-20 seconds)

Your child may be encouraged to perform the daily activities for the study and to complete all assessments. The exercises we have chosen are normal callisthenic-style exercise. The routine will consist of:

- A short warm-up period (5 min)
- 8 intervals of 20 sec. high intensity exercise followed by 10 sec. of rest (4 min) doing squat-thrusts, mountain climbers, jumping jacks and high knees, or similar exercise
- A cool-down period (2 min)

Discomforts & Risks: Will it hurt?

The risks involved with participation in this study are as follows. If they do not exercise often they may feel like they are out of breath during the first couple of sessions. In addition, after the first couple of exercise sessions there may be some muscle soreness. This also is completely normal and will pass after a few sessions once they get used to doing the routine. The investigator is certified in Heartsaver First Aid by the American Heart Association and is capable of handling any unexpected incidence that may arise from the exercise protocol. There will be a very slight prick when the phlebotomist is taking the blood sample. This will pass after a few seconds and there is typically no pain at all during such a procedure. If your child has a fear of pin pricks, then we will do everything to accommodate them and ease their fears of the short procedure. The risks of taking blood include a quick prick, and in some cases may cause bruising at the point where the blood is taken, redness and swelling of the vein, and a rare risk of fainting or

infection. If you have problems with blood clotting or are on a medication that might decrease clotting, please alert the phlebotomist prior to blood being drawn. The investigator should be alerted to any medical problems so that appropriate precautions can be taken to limit your child's involvement.

Benefits

While we cannot guarantee that your child will receive any benefit above and beyond their education; if your child participates in this research, your child may receive the following benefits:

- They may become healthier, stronger, and faster while following the exercise routine
- They may begin to perform better in school and on tests
- They may have fun playing chess and participating in exercise

All children who participate, and complete all the requirements for nine weeks will receive *free tickets to a Bowling Green Hot Rods game* after all data collection is complete. Groups will compete against each other based on attendance and effort in a competition to earn \$1 of play money each daily. The team will accumulate this total daily for the nine weeks and the individuals on the winning team will each receive a very nice "Star Performer" trophy. All participants and their parents will be invited to a pizza party on Friday afternoon November 21st.

Confidentiality

The information that we collect from this research project will be kept confidential. Identifiable data will be coded to protect any records. Only the researchers will know what that number is and we will lock that information up so no one else will have access

to it. Afterwards, we will publish the confidential results in order that other interested people may learn from our research.

Right to Refuse or Withdraw

Refusal to participate in this study will have no effect on any future services you may be entitled to from the University. Anyone who agrees to participate in this study is free to withdraw from the study at any time with no penalty. Those who refuse to participate or withdraw will continue to participate in normal school activities. There will only be two periods with disruptions: the enrichment period and the gym period. During those times, those who refuse to participate or withdraw from research will not have any data collected regarding their activities.

You understand also that it is not possible to identify all potential risks in an experimental procedure, and you believe that reasonable safeguards have been taken to minimize both the known and potential but unknown risks.

Who to Contact

This proposal has been reviewed and approved by the Western Kentucky University Human Subjects Review Board, which is a committee whose task it is to make sure that research participants are protected from harm. If you wish to find out more about the WKU IRB, contact:

Office of Research Integrity

Address: Western Kentucky University College Heights Blvd. #11026 Bowling Green, KY 42101-1026

Phone: 270.745.2129

Fax: 270.745.4221

Email:Paul.Mooney@wku.edu

<http://www.wku.edu/compliance/>

PART II: Certificate of Consent

Certificate of Consent

I have been invited to have my child participate in research of a method for improving learning and brain development in Jr. High and High School children. I have read the foregoing information. I have had the opportunity to ask questions about it and any questions that I have asked have been answered to my satisfaction.

I consent voluntarily for my child to participate as a participant in this study.

Print Name of Participant _____

Print Name of Parent/Guardian _____

Signature of Parent or Guardian _____

Date _____ Day/month/year

Informed Assent Form for Children Who Can Participate in WKU Educational Leadership Research Study at Clarkson Elementary

This informed assent form is for children in fourth/fifth grade who attend Clarkson Elementary who we are invited to participate in research that will determine if a child's involvement in exercise and chess could improve their learning ability and enhance their natural brain development.

Principle Investigator: Samuel J Hunt

Name of Organization: Western Kentucky University

Department: Educational Leadership Doctoral Program

Faculty Sponsor: Dr. Barbara Burch

Project Title: *A Novel Use of Biomarkers: Predicting and Assessing Academic Ability*

I, _____, understand that my parents (mom, dad, or guardians) have given permission (said it's okay) for me to take part in a project about playing the game of chess and doing gym exercises daily under the direction of Samuel J Hunt. The exercise will consist of normal callisthenic-style exercise. The routine will consist of:

- A short warm-up period (5 min)
- 8 intervals of 20 sec. high intensity exercise followed by 10 sec. of rest (4 min) doing squat-thrusts, mountain climbers, jumping jacks and high knees, or similar exercise
- A cool-down period (2 min)

I also understand that I will need to take a written test (not for a grade) and a blood test for proteins at the beginning and the end of the project.

The risks of taking blood include a quick prick, and in some cases may cause bruising at the point where the blood is taken, redness and swelling of the vein, and a rare risk of fainting or infection. If you have problems with blood clotting or are on a medication that might decrease clotting, please alert the phlebotomist prior to blood being drawn.

I am taking part because I want to. I have been told that I can stop at any time I want to and nothing will happen to me if I want to stop.

Signature _____ Date _____

Signature of Witness _____ Date _____

APPENDIX A2: Physical Activity Readiness Questionnaire (PARQ)

Physical Activity Readiness
Questionnaire - PAR-Q
(revised 2002)

PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO	
<input type="checkbox"/>	<input type="checkbox"/>	1. Has your doctor ever said that you have a heart condition <u>and</u> that you should only do physical activity recommended by a doctor?
<input type="checkbox"/>	<input type="checkbox"/>	2. Do you feel pain in your chest when you do physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	3. In the past month, have you had chest pain when you were not doing physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	4. Do you lose your balance because of dizziness or do you ever lose consciousness?
<input type="checkbox"/>	<input type="checkbox"/>	5. Do you have a bone or joint problem (for example, back, knee or hip) that could be made worse by a change in your physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?
<input type="checkbox"/>	<input type="checkbox"/>	7. Do you know of <u>any other reason</u> why you should not do physical activity?

If
you
answered

YES to one or more questions

Talk with your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want — as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

NO to all questions

If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can:

- start becoming much more physically active — begin slowly and build up gradually. This is the safest and easiest way to go.
- take part in a fitness appraisal — this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively. It is also highly recommended that you have your blood pressure evaluated. If your reading is over 144/94, talk with your doctor before you start becoming much more physically active.

DELAY BECOMING MUCH MORE ACTIVE:

- if you are not feeling well because of a temporary illness such as a cold or a fever — wait until you feel better; or
- if you are or may be pregnant — talk to your doctor before you start becoming more active.

PLEASE NOTE: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

Informed Use of the PAR-Q: The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

No changes permitted. You are encouraged to photocopy the PAR-Q but only if you use the entire form.

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

"I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction."

NAME _____

SIGNATURE _____

DATE _____

SIGNATURE OF PARENT _____
or GUARDIAN (for participants under the age of majority)

WITNESS _____

Note: This physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if your condition changes so that you would answer YES to any of the seven questions.



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APPENDIX B: New London Blood Draw Standards and Procedures

BAKER MEMORIAL LABORATORY

VENIPUNCTURE (INCLUDING ORDER OF DRAW and LABELING)

PURPOSE

To standardize the process of drawing and labeling blood samples throughout the New London Hospital Association.

SAFETY

1. Venipuncture will not be performed in the following situations:
 - Non-emergency in-patients and ER patients not properly identified with a NLH wristband.
 - Patient who refuses to have blood drawn
 - From a limb with an IV that is open.
 - The laboratory staff do not draw from any catheter, cannula or IV line or from arteries.
 - From any leg or foot veins
2. Do NOT attempt a venipuncture more than twice. A second phlebotomist may evaluate veins and attempt a third venipuncture ONLY if they are confident in the vein selection. Microcollection methods will be used if acceptable for the required tests. Notify Charge RN if sample cannot be obtained on an Inpatient or Clough resident and notify the provider's office if an outpatient.
3. Needles are never recapped, removed, broken, or bent after phlebotomy procedures.

4. Contaminated surfaces must be cleaned with hospital approved disinfectant.
5. In the case of an accidental needlestick, immediately wash the area with a hospital approved hand cleaner and follow current guidelines for needlesticks.

PATIENTS RECEIVING IV FLUIDS

- Blood should be obtained from the arm opposite the one receiving IV solution. If this is not possible, a distal or proximal sample can be collected.

DISTAL (below IV site)

1. Ask the caregiver to turn off the IV infusion for 2 minutes to ensure flow is completely discontinued.
2. Apply tourniquet between IV and intended venipuncture site.
3. Proceed with venipuncture.

PROXIMAL: (above IV site)

NOTE: not recommended – only use when all other alternatives (including capillary) have been exhausted

1. Ask the caregiver to turn off the IV infusion for 2 minutes. Care should be taken to ensure flow has been completely discontinued.
 2. Apply tourniquet 3 to 4 inches above the antecubital fossa.
 3. Proceed with venipuncture.
- When blood is obtained from the arm receiving IV fluids, a note should be added to the report to inform the provider that the sample was obtained in the same arm as an IV and the relevance to the IV (above or below after IV shut off 2 minutes.)

MATERIALS

- Gloves

- Tourniquet
- Alcohol Prep
- Gauze
- Needle holder/needle or butterfly needle
- Vacutainer Tubes
- Syringes (for butterfly collection)
- Tape
- Sharps container

VENIPUNCTURE PROCEDURE

1. Identify the patient. Outpatients are called into the phlebotomy area and asked their first and last name and their date of birth. This information must match the requisition. Inpatients or emergency department patients are identified by their wrist-band and by asking their name and date of birth. This information must match the collection label. Resolve any discrepancy before the patient's blood is drawn. If the wristband has been removed, a nurse must attach a new one before the patient can be drawn (unless an emergent draw).
2. Assess patient's ability to understand the process (e.g. language for non-English speaking persons, children, etc.). Adjust processes as needed based on age. Ask caregiver or persons accompanying patient for assistance. Contact admissions or social worker for assistance with language barriers. **DO NOT** proceed with venipuncture if the patient refuses.
3. **BLOOD BANK COLLECTION ONLY:** Gather correct blood collection tubes to fill for lab tests ordered. If a crossmatch or other pre-infusion testing has been

ordered (or is anticipated), the following protocol must be initiated at the time of collection of the blood sample.

- a. Complete the label on the blood bank wristband and attach it to the patient at the time the blood is obtained. Press hard with ballpoint pen.
 - i. PT: is the patient's name.
 - ii. MR#: is the patient's medical record number or DOB if medical record number not available.
 - iii. DATE: is the date of collection.
 - iv. PB: is the initials of the person that drew the patient's blood
 - b. Remove the white label with patient information from the wristband and place onto the sample of blood.
 - c. Detach tail with 14 small pre-numbered labels at perforation after last hole and send to lab with tube.
 - d. Wrap the band around the patient's wrist (or ankle). Place 2 fingers under the band before closing the snap to allow some slack in the band. For increased length, attach white extension band to third hole of blood band. Size band and close snap.
4. Assemble the necessary equipment appropriate for this venipuncture.
- Needle holder and needle are disposable and come prepackaged.
 - Butterfly Needle
 - a. Remove the needle from the sterile package.
 - b. Twist the rubber sheathed puncture end onto a holder or remove it and twist the hub onto a sterile syringe.

5. Wash hands and put on gloves.
6. Position the patient with the arm extended to form a straight-line from shoulder to wrist.
7. Apply a tourniquet 3-4 inches above the collection site and select the best vein.
Never leave the tourniquet on for more than one minute. If a tourniquet is used for preliminary vein selection, release it and reapply just prior to the venipuncture.

Vein Selection:

- Avoid scarring or healed burn areas
 - Do NOT use veins on underside of wrist.
 - Do NOT collect sample on same side as mastectomy without consultation with physician.
 - Avoid areas of hematomas
 - Do not obtain from an arm having a cannula, fistula, or vascular graft.
 - Allow 30 minutes after a completed blood transfusion prior to collecting a blood sample.
8. Swab the site with an alcohol prep pad. Air dry thoroughly before proceeding.
 9. *If using a butterfly needle / syringe set up, skip to section Venipuncture Using a Syringe below.*
 10. Remove plastic cap over needle and hold bevel up.
 11. "Fix" the vein in position and with the needle at an acute angle, quickly penetrate the skin and vein in one smooth motion.

**** BUTTERFLY USAGE NOTE:** If a light blue sodium citrate tube (used for coagulation studies) or a Vital Diagnostics ESR tube is to be drawn, the tubing must be cleared of air before filling the tube. This can be done by drawing or filling a no additive waste tube (red with clear plastic top) first as a discard or by drawing an appropriate tube for other testing, following order of draw requirements.

12. Holding the hub securely, insert the first vacutainer tube following proper order of draw. Puncture the tube stopper by pushing the tube forward. This initiates the vacuum suction and blood should flow into the tube.

13. After the blood starts to flow, release the tourniquet.

14. When the blood flow into the tube stops, remove the tube by holding the hub securely and pulling the tube off the needle. Tubes should be inverted while other tubes continue to be filled. Invert tubes as indicated in the chart below. Do NOT shake or mix vigorously.

Order to draw	Tubes	Invert (# of times)
1	Blood Culture Bottles	8-10
2	Light Blue (citrate)	4
3	Vital Diag. ESR tube	4
4	Red or Gold	5
5	Green	8-10
6	Lavender	8-10
7	Pink (EDTA)	8-10
8	White	8-10
9	Gray	8-10

15. Place a dry gauze pad over the venipuncture site and withdraw the needle carefully. Remove the entire assembly from the arm when completed. Engage the needle safety device and discard the entire needle assembly. If using a butterfly –

push the black button prior to removing needle from arm to safely retract the needle. Do NOT use cotton balls.

16. Immediately, apply slight pressure. Ask the patient to apply pressure while you label the tubes. If the patient applies pressure, continue to observe for adequate pressure.
17. When the bleeding stops, apply a fresh Band-Aid or gauze and tape. Instruct the patient to leave the bandage on for at least 15 minutes.
18. Dispose of vacutainer needle and holder into biohazard sharps container as one unit.
19. Label all tubes with:
 - Patient's first and last name
 - DOB (or medical record number)
 - date of collection
 - initials of collector
 - label from blood bank wristband (if applicable)
20. Non-lab personnel: Complete the requisition form. (See separate procedure) and place specimen tubes in a plastic biohazard specimen bag with the requisition form in the outside pocket of the bag.

VENIPUNCTURE USING A BUTTERFLY / SYRINGE

1. Remove plastic cap over needle and hold bevel up.
2. "Fix" the vein in position and with the needle in line with the vein, quickly penetrate the skin and vein in one smooth motion.
3. Draw the desired amount of blood by pulling back slowly on the syringe stopper.

4. Release the tourniquet within one minute.
5. Place a gauze pad over the puncture site and quickly remove the needle.
Immediately apply pressure. Ask the patient to apply pressure. When the bleeding stops, apply a fresh bandage, gauze and tape.
6. Transfer blood drawn into the appropriate tubes as soon as possible using a needleless transfer device. Use chart for order to fill tubes. Invert tubes as indicated in the chart below. Do NOT shake or mix vigorously.

Order to fill	Tubes	Invert (# of times)
1	Blood Culture Bottles	8-10
2	Light Blue (citrate)	4
3	Vital Diag. ESR tube	4
4	Red or Gold	5
5	Green	8-10
6	Lavender	8-10
7	Pink (EDTA)	8-10
8	White	8-10
9	Gray	8-10

7. Dispose of syringe and needle as one unit into appropriate sharps container.

TROUBLESHOOTING HINTS FOR BLOOD COLLECTION

- If blood is not flowing into the vacutainer tube:
 - Reposition the needle: Pull back slightly or push in slightly. Do not probe.
 - Ensure that the collection tube is completely pushed onto the back of the needle in the hub.
 - Loosen the tourniquet.
- Hematoma:
 - If a hematoma forms, withdraw the needle immediately and elevate the arm. Apply pressure. Do not bend the patient's arm.

- Repeat venipuncture in a different site if needed.
- Collapsed Vein:
 - Tighten the tourniquet by grasping the ends with one hand and twisting them together. If the blood does not resume, remove the tube from the needle, wait a few seconds for the blood flow to reestablish and insert a smaller volume tube.
 - Remove the needle.
- Patient experiences sharp or shooting pain:
 - This could be a sign of contact with a nerve.
 - Remove tourniquet and withdraw needle immediately.

Reference:

H3-A6 Procedures for the Collection of Diagnostic Blood Specimens by Venipuncture;
Approved Standard

<http://www.drgreene.org/body.cfm?id=21&action=detail&ref=1616>

BD Vacutainer Order of Draw for Multiple Tube Collections 01-2010

APPENDIX C: Lab Procedure for Blood Analysis

Samples were prepared ahead of time by spinning in a Fisher accuSpin™ 1/1R Benchtop Centrifuge (Thermo Fischer Scientific, Inc., Waltham, MA) at 2000xg for 15 minutes. Samples were immediately placed back on ice and transferred to a level two biosafety hood, where 38 .5 ml aliquot samples were prepared in triplicate. Samples were de-identified, numbered and placed in cold storage at -20° Celsius until analysis could be conducted. Lab procedure was conducted in the following manner:

1. Pull kit and samples from freezer and bring to room temperature.
2. Label and set-up microfuge tubes according to plate layout design.
3. Pipet 100 µL of assay diluent A from RayBio® kit into each labeled microfuge tube.
4. Prepare standards according to RayBio® kit directions.
5. Add 100 µL of sample to each microfuge tube in the following process:
 - a. Vortex sample three sec @ 3000rpm using VWR® Signature Digital Vortex Mixer (VWR International, LLC, Radnor, PA).
 - b. Pipet 100 µL of sample into each microfuge tube as labeled.
 - c. Mix sample by pipetting up and down 10x.
 - d. Close tube and discard tip after each transfer and mix.
6. Add 100 µL of prepared sample and standard in duplicate into appropriate wells. Discard tip after each transfer.
7. Label and incubate overnight at 4° C with gentle mixing on Orbitron II Rotator (Boekel Scientific, Feasterville, PA).
8. Discard solution wash 4x and blot dry per RayBio® kit directions.

9. Add 100 μ L of biotinylated antibody to each well (dilution factor .1 ml).
10. Cover and incubate on rotator with gentle mixing for 60 minutes.
11. Discard solution and repeat wash procedure.
12. Prepare HRP-streptavidin concentrate (dilution factor .05: 14.45 ml) and add to each well.
13. Incubate for 45 minutes on rotator.
14. Discard solution and repeat wash procedure.
15. Add 100 μ L of TMB substrate reagent to each well. Cover and wrap in aluminum foil. Incubate 30 minutes in dark on rotator.
16. Set-up software and prepare plate reader.
17. Remove foil and apply 50 μ L stop solution to each well and read immediately on 450nm.

APPENDIX D1: Daily Exercise and Chess Regimen

Daily Chess Class Regime						
Date	Chess Lessons		Chess Practice		Pass out	Approx. Time
Friday 9/13	Online Lesson 1	L 1 Supp video 1	Chess Pre Quiz		Lesson 1 Handouts	39 min.
Week 1						
Monday 9/16	L1 Supp video 2	Online lesson 2	L 2 Supp video 1 & 2		Lesson 2 handouts	39 min.
Tuesday 9/17	Lesson #2 group quiz	L 2 Supp video 3	Online lesson 3		Lesson 3 handouts	35 min.
Wednesday 9/18	L3 supp videos 1-3	Lesson 3 group quiz	Online lesson 4	L4 supp video	Lesson 4 handouts	32 min.
Thursday 9/19	Lesson 4 group quiz	Online Lesson 5	L5 Supp video	Play chess (20 min.)	Lesson 5 handouts	40 min.
Friday 9/20	Lesson 5 group quiz	Online Lesson 6	L6 Supp video	Play chess (20 min.)	Lesson 6 handouts	40 min.
Week 2						
Monday 9/23	Lesson 6 group quiz	Online Lesson 7	L7 Supp video	Play chess (15 min)	Lesson 7 handouts	40 min.
Tuesday 9/24	Lesson 7 group quiz	Online Lesson 8	L8 Supp video 1 & 2	Play chess (15 min)	Lesson 8 handouts	40 min.
Wednesday 9/25	Lesson 8 group quiz	Online Lesson 9	L9 Supp video	Hands-on Practice link	Lesson 9 handouts	40 min.
Thursday 9/26	Lesson 9 group quiz	Online lesson 10	L10 Supp video 1	Hands-on Practice link 1	Lesson 10 handouts	40 min.
Friday 9/27	L10 Supp video 2	Hands-on Practice link 2	Lesson 10 group quiz	Play chess (15 min)		40. min
Week 3						
Monday 9/30	Online Lesson 11	L11 supp videos 1 & 2		Play Chess (10 min)	Lesson 11 handouts	40 min.
Tuesday 10/1	Lesson 11 group quiz	Online lesson 12		Play chess (20 min)	Lesson 12 handouts	40 min.
Wednesday 10/2	L12 Supp videos 1-4	Lesson 12 group quiz		Play chess (10 min)		40 min.
Thursday 10/3	L12 Supp videos 5			Play chess (20 min)		40 min.
Friday 10/4	L12 supp video 6 (x2)			Play Chess (10 min)		40 min.

Daily Chess Class Regime cont'd						
Week 4						
Monday 10/7	Online Lesson 13	L13 Supp video		Play chess (10 min)	Lesson 13 Handouts	40 min.
Tuesday 10/8	Lesson 13 group Quiz	Online Lesson 14	L14 Supp video 2 (both)	Play chess (15 min)	no handouts	40 min.
Wednesday 10/9	L14 Supp video 1 (both)	Lesson 14 group quiz				35 min.
Thursday 10/10	Online Lesson 15			Play chess (20 min)	Lesson 15 handouts	40 min.
Friday 10/11	L15 Supp. Link 1 (watch 1-3 in list)			Play chess (10 min)		40 min.
Saturday 10/12	Chess Tournament at McNeill Elementary!!!					
Week 5						
Monday 10/14	L15 Supp. Link 1 (watch 4-6 in list)			Play chess (10 min)		40 min.
Tuesday 10/15	L15 Supp. Link 1 (watch 7-8 in list)			Play chess (15 min)		40 min.
Wednesday 10/16	L15 Supp. Link 1 (watch 9-10 in list)			Play chess (15 min)		40 min.
Thursday 10/17	L15 Supp. Link 1 (watch 11-13 in list)					35 min.
Friday 10/18	L15 Supplement videos 2 & 3	Lesson 15 group quiz		Play chess (10 min)		37 min.
Week 6 Chess King DVD – Chess Course Titles (gray set)*						
* NOTE: IN addition to the Chess DVD course you will need to install the Tactics Level 1 (orange), Strategy (purple), and Endgames (brown) Practice DVDs and store them on your computer.						
Monday 10/21	Opening Principles DVD ch. 1-7			Play Chess (15 min)		40 min.
Tuesday 10/22	Tactics/Strategy DVD ch. 1-6					35 min.
Wednesday 10/23	Endgames DVD ch. 1-7			Play Chess (15 min)		40 min.
Thursday 10/24	Opening Principles DVD ch. 8-9	<i>Tactics Practice: Mate in 1, Few figured position, solve 5 from ea. Category</i>				40 min.
Friday 10/25	Tactics/Strategy DVD ch. 7-13			Play chess (10 min)		40 min.

Daily Chess Class Regime, cont'd						
Week 7	Chess King DVD – Chess	Course Titles (gray set)				
Monday 10/28	Endgames DVD ch. 8-12	<i>Endgame Practice:</i> Opposition - 8 exercises				40 min.
Tuesday 10/29	Opening Principles DVD ch. 10-12	<i>Strategy practice:</i> Pawn structure & Weak pawns				40 min.
Wednesday 10/30	Tactics/Strategy DVD ch. 14-16	<i>Tactics Practice:</i> Mate in 1, many figured position, Rook checkmates, solve 10				35 min.
Thursday 10/31	Endgames DVD ch. 13-16	<i>Endgame practice:</i> Triangulation, Rule of the Square, Outside Passed Pawn				40 min.
Friday 11/1	Opening Principles DVD ch. 13-15			Play chess (15 min)		35 min.
Week 8	Chess King DVD – Chess	Course Titles (gray set)				
Monday 11/4	Tactics/Strategy DVD ch. 17-20			Play chess (15 min)		38 min.
Tuesday 11/5	Endgames DVD ch. 17-22	<i>Strategy practice:</i> Pawn majority all				38 min.
Wednesday 11/6	Opening Principles DVD ch. 16-20			Play chess (15 min)		40 min.
Thursday 11/7	Tactics/Strategy DVD ch. 21-23	<i>Tactics Practice:</i> Mate in 1, many figured position, Queen checkmates, solve 10				40 min.
Friday 11/8	Tactics/Strategy DVD ch. 24-27			Play Chess (10 min)		40 min.
Week 9	Chess King DVD – Opening Ideas (Blue set)					
Monday 11/18	Open Games DVD ch. 1-6					35 min.
Tuesday 11/19	Open Games DVD ch. 7-9	<i>Endgame Practice:</i> Playing for Stalemate - 4 exercises				35 min.
Wednesday 11/20	Closed Games DVD ch. 1-3	<i>Tactics Practice:</i> Mate in 1, many figured position, Bishop checkmates, solve 10				35 min.
Thursday 11/21	Closed Games DVD ch. 4-6	Take Post Chess Quiz		Play chess		40 min
Fri day 11/22	CONGRATULATIONS!!! POST-TEST DAY!!!					

Exercise video list: http://www.youtube.com/playlist?list=PLVBHOy3ZBi1T9uwgDNSr2swAIS5hhOzAa							
Week one exercise routine:		Repeat all x1			Total 4 minutes		
Video #1							
Duration	20 sec.	10 sec.	20 sec.	10 sec.	20 sec.	10 sec.	20 sec.
movement	Squat thrusts	rest	mountain climbers	rest	high knees	rest	jumping jacks
Week two exercise routine:		Repeat all x1			Total 4 minutes		
Video #2							
Duration	20 sec.	10 sec.	20 sec.	10 sec.	20 sec.	10 sec.	20 sec.
movement	Jump Lunges	rest	Jump squats	rest	Squat Thrusts	rest	Push-ups
Week three exercise routine		Repeat all x1					
Video #3							
Duration	20 sec.	10 sec.	20 sec.	10 sec.	20 sec.	10 sec.	20 sec.
movement	High knees	rest	Squat Jumps	rest	Knee-up hops	rest	Knee grab abs
Week four exercise routine:		Repeat all x1			Total 4 minutes		
Video #4							
Duration	20 sec.	10 sec.	20 sec.	10 sec.	20 sec.	10 sec.	20 sec.
movement	Spiderman crawl	rest	half burpee	rest	Push-up	rest	forward lunges
Week five exercise routine:		Repeat all x1			Total 4 minutes		
Video #5							
Duration	20 sec.	10 sec.	20 sec.	10 sec.	20 sec.	10 sec.	20 sec.
movement	Sumo Squats	rest	Knee drives Right Leg	rest	Knee drives Left Leg	rest	squat thrusts

APPENDIX D2: Pre-Post Chess and Exercise Test Forms

Diagram 1

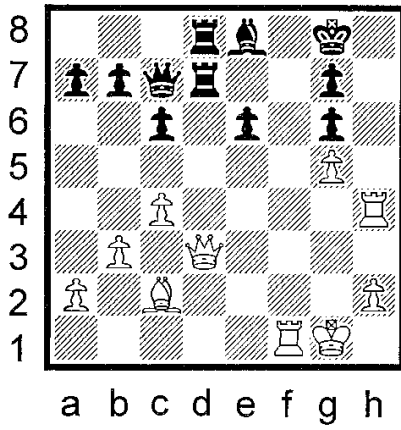


Diagram 2

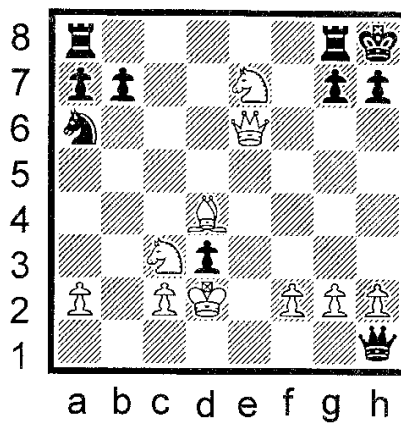


Diagram 3

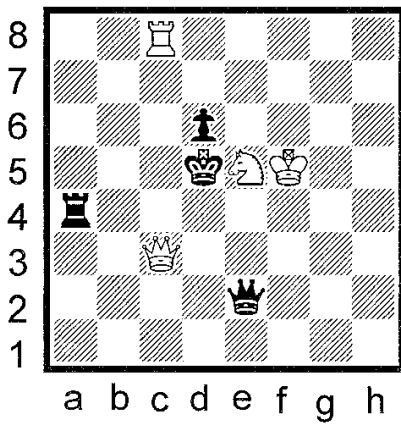


Diagram 4

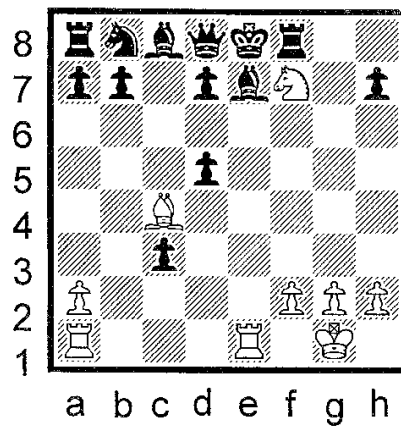


Diagram 5

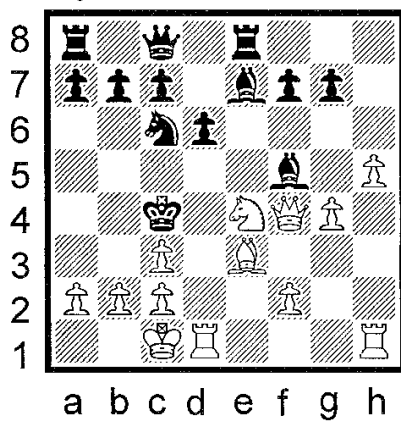
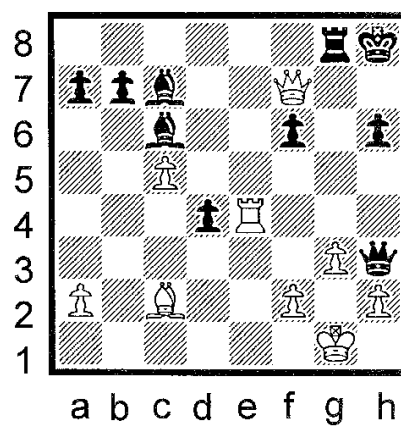



Diagram 6




INSANITY[®]


FIT TEST


1. SWITCH KICKS
2. POWER JACKS
3. POWER KNEES
4. POWER JUMPS



2 KICKS = 1 REP





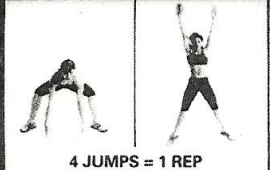


MOVE	FIT TEST 1 (DAY 1)	FIT TEST 2 (DAY 15)	FIT TEST 3 (DAY 36)	FIT TEST 4 (DAY 50)	FIT TEST 5 (DAY 63)
1. SWITCH KICKS					
2. POWER JACKS					
3. POWER KNEES					
4. POWER JUMPS					

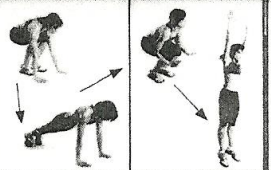
You can also find this Fit Test on the DIG DEEPER DVD. Perform the moves listed above. Do as many reps as you can in 1 minute, then record your results after every exercise. Repeat the entire fit test and test when needed.

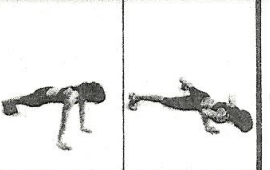
FIT TEST, CONT.

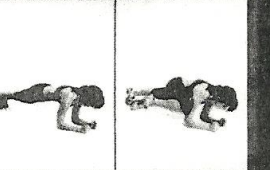
5. GLOBE JUMPS
6. SUICIDE JUMPS
7. PUSH-UP JACKS
8. LOW PLANK OBLIQUE



4 JUMPS = 1 REP







MOVE	FIT TEST 1 (DAY 1)	FIT TEST 2 (DAY 15)	FIT TEST 3 (DAY 36)	FIT TEST 4 (DAY 50)	FIT TEST 5 (DAY 63)
5. GLOBE JUMPS					
6. SUICIDE JUMPS					
7. PUSH-UP JACKS					
8. LOW PLANK OBLIQUE					

INSANITY is an extreme and physically demanding workout. This is NOT for beginners or individuals with any pre-existing condition that may be aggravated by extreme cardio and strength exercise. Consult your physician for membership eligibility and other medical advice regarding this program.

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