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Samantha Rezap
University of Portland

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Exercise and Cognition in Young Adults

Samantha M. Rezab

University of Portland

Abstract

Exercise had been proven to have a positive effect on physical, mental and cognitive functioning in varying populations. This study looks in to the effect of exercise on episodic memory, perceptual speed and executive function in young adults. Three cognitive tasks and academic performance were correlated with exercise in high school and in college. Results showed a positive correlation between exercise and perceptual speed and a negative correlation between exercise and episodic memory. These results were inconsistent with previous findings, however it is suggested that there is a neurobiological cause underlying the results. Additionally, the assumption that young adults are at the peak of their cognitive ability means that exercise will not cause cognitive abilities to be higher or lower compared to other young adults with more or less exercise.

Literature Review

Exercise has been proven to have beneficial effects on both physical health and mental health. Exercise is associated with improving balance, bone density, strength and endurance, as well as with helping to prevent cardiovascular disease, obesity, and blood pressure. These physical effects of exercise have been studied and seen to have an effect on muscles and organs that in turn modify and regulate the structure and functions of the brain (Dishman et al. 2006). In addition to physical aspects of health, exercise has also been associated with cognitive function. Specifically, it is seen as a viable prevention technique to various neurodegenerative diseases, such as Alzheimer's disease (AD) and seen as a potential way to improve academic achievement, cognitive abilities, and intellectual function in children (Tomporowski et al. 2008).

Cognition is a term that describes higher brain functions such as decision-making, calculating, problem solving, producing and using language, and memory. The purpose of the present study is to examine the effects of exercise on three facets of cognition. Studies looking at exercise and cognition have focused on individuals across the lifespan. However, research in this area has been very controversial with findings that range from exercise positively affecting cognition to findings that say exercise can actually do harm to cognitive function depending on the population and the type of exercise employed.

Some have sought to uncover the specific brain processes and structures that are affected by exercise. The methodology of such studies are still in the early stage of research as technology and the ability to look at people's brains as they function during cognitive tasks are relatively new. The current hypothesis was founded on the basis of neurogenesis, or the making of new neurons and the regulation of their growth by brain derived neurotrophic factor (BDNF) or some other neuronal growth hormone. Some animal studies have focused on studying the

cellular and molecular mechanisms that underlie this phenomenon. In a study done on mice, 14 days of wheel running promoted memory acquisition, memory retention, and reversal learning increased the number of maturing neurons. Mice that had been exposed to the same regime, but sacrificed before the final memory tasks, had an increase in molecules associated with neurogenesis, suggesting that neurogenesis and memory seem to be associated (Van der Borght et al. 2007).

Recently researchers have begun extending studies examining the effect of exercise on brain structures to human participants. The hippocampus is located in the medial temporal lobe of the brain and is involved in the consolidation of information from short-term memory to long-term memory. In rat studies, exercise enhances learning and improves retention, which is accompanied by increased cell proliferation and survival in the hippocampus (van Praag 2005). In a human study, done with 150 older adults, aerobic exercise increased the size of the anterior hippocampus leading to improvements in spatial memory. Increased hippocampal volume is also associated with greater serum levels of BDNF, a mediator of neurogenesis. The results also suggested that physical activity may act as a buffer and protect against hippocampal volume loss. Indeed, aerobic exercise training is effective at reversing hippocampal volume loss in late adulthood, which is accompanied by improved memory function (Erickson et al. 2011).

The effect of not only exercise on memory but also other cognitive functions is an important field to continue to research, as cognitive function is important at every age, from preschool to the elderly. Bunce and Murden (2006), suggest that though there is a correlation between aerobic fitness and cognition, that association weakens with age. They speculate that fitness of the neuroanatomical structures are eventually overshadowed by neurological degeneration that occurs in 70+ years of age. An interesting application of this research is to

Alzheimer's Disease (AD). In a yearlong study, aerobic exercise was observed to stabilize the cognitive functioning in patients with AD. Individuals who walked at least 2h/week saw significant improvement in the Mini-Mental State Examination (MMSE), a questionnaire used to screen for cognitive impairment, whereas those who remained sedentary saw a decline in cognitive ability (Winchester 2013). Scarmeas et al. (2009) also observed that higher physical activity reduced the risk of AD.

Researchers have sought to understand the mechanisms through which exercise may improve the symptoms of AD. The two primary diagnostic characteristics of AD include two different protein deposits in the brain, "tangles" and Amyloid β ($A\beta$) plaques. Both of these occur naturally with age; however AD patients develop far more of both types of proteins and also follow a particular pattern of development, beginning in important regions of the brain and spreading from there to other areas. Both protein structures are associated with the severity of AD the patient has; if there are more of these protein deposits, the progression of AD is more severe. "Tangles", also known as neurofibrillary tangles, are an abnormal form of the microtubule-associated protein tau (Perl 2010). Tau on its own is a stabilizer for the microtubules in the neuron. The forms of tau that make up the tangles are hyperphosphorylated at specific sites. The pattern of their distribution starts in the deeper layers of the brain in the neocortex, entorhinal cortex, regions of the hippocampus and the amygdala and spread outward from there (Perl 2010).

Amyloid β ($A\beta$) protein is derived from the larger amyloid precursor protein (APP). Normal cell functioning requires APP, which is normally used in Notch processing, a highly conserved cell signaling pathway found in most multicellular organisms that is involved in neurogenesis and neuron differentiation. However, when APP is processed $A\beta$ peptides of

varying lengths are produced between 37-49 amino acids. In a normal individual, the majority of A β produced are 40 amino acids in length (A β -40), only about 5-15% of the A β peptides are A β -42. Both A β -40 and A β -42 are produced by a single γ -secretase enzyme, a type of protease that cleaves APP (Feindeis 2007). Normally the clearance rate for these A β peptides is high and prevents a buildup. In an AD patient, the two main A β peptides found in the plaques in the brain are those of primarily A β -42 and smaller amounts of A β -40 (2007). The predominant and initial A β peptide deposited in the brain parenchyma is A β -42. These initial deposits are then able to seed the formation of both long (A β -42) and short (A β -40) forms of A β . In transgenic animal studies, deposits are associated with high levels of A β -42 and then later on A β -40 as seen in humans (2007). The aggregation of the insoluble A β peptides leads to neuronal toxicity and eventual cell death. Before the death of the neuron however, the A β peptides interrupt the neuronal cell function, which is what is seen in mild forms of AD. In particular long-term potentiation (LTP) is the first brain function to be interrupted. LTP is important in the formation of new memories and learning, explaining why plaque formation may be important in causing the early symptoms of AD (2007).

From previous studies conducted, it is clear that exercise is beneficial to AD patients; however none of these studies examined the mechanism in the brain that drives the observed improvements or lack thereof. Recent evidence has been collected that determined A β plaques to be part of that mechanism. Adlard et al. (2005) used a mouse model of AD to test the hypothesis that long term voluntary exercise would impact the normal progression of AD-like neuropathology. They found that after 5 months of voluntary exercise levels A β -40 and A β -42 was significantly decreased in the cortex of the mice that were given running wheels (exercise) when compared to mice that did not get a running wheel (sedentary). It was suggested that A β

degradation pathways were activated through exercise that may involve neuronal pathways known to regulate APP processing and be reflected in a decreased production of A β . In another study, they found that exercise training reduces A β -42 deposition in mice while their sedentary counterparts had a higher A β -42 deposition. From these findings, they concluded that exercise training could be used to prevent accumulation of the APP because it induces increased degradation and clearance of A β .

On the other end of the age spectrum from those experiencing AD are children. The motivation for studying the effect of exercise on cognition in children is primarily to develop interventions that can be used in the classroom. A child who cannot effectively plan, inhibit impulsive behavior, update working memory, or shift tasks is going to be hard pressed to perform in a classroom setting where those mental processes are required to excel socially and academically (St Clair-Thompson 2006). Several studies have suggested that participation in physical activity has a positive relation or no relation to academic performance. However, it is indicated in these studies that an increase in the amount of time dedicated towards physical activities is not accompanied by a decline in academic performance (Ahamed et al. 1997). In studies focusing on measuring the cognitive nature of exercise, results have varied. In some studies there was a significant increase in creativity measures but not in perceptual-motor skill or visual motor coordination (Tuckman & Hinkle 1986; Hinkle et al. 1993). Positive associations were found between fitness and better cognition and academic achievement in 7-11 year olds (Davis & Cooper 2011). However, later studies have found that exercise does in fact positively change children's cognitive abilities such as planning, executive function and academic achievement.

Academic achievement is an area that many believe is an indicator of other mental capabilities, including cognition but also cooperation, sharing, and learning to follow rules. Similarly, it is thought that types of physical activity that promote those skills often transfer over to the classroom (Tomporowski et al. 2008). In another study that intervened in the lives of overweight children by adding in exercise, it was found that those who partook in the exercise regimen performed better on cognitive tasks than those who did not (Davis et al. 2007).

The biological mechanisms behind exercise and the development of the brain is a budding area of research. It is suggested that exercise has a role in the development of various brain structures and neuron development. For example, between infancy and early childhood, there is a dramatic increase in gray matter and later, from age 7 to early adulthood there is a gradual decrease and an increase in the myelination and connectivity in the frontal cortex (Sowell et al. 2004). This is a critical period for refinement of neuronal pathways, strengthening of synapses. Physical activity leads to the production of neurotrophins that regulate the survival, growth and differentiation of neurons during development. Neurotrophins also play a part in synaptogenesis, myelination and angiogenesis, which are all important in the developing brain (Tomporowski et al. 2008).

Research focused on the relationship between chronic exercise and cognition in young adults is extremely limited and has yielded mixed results. Most studies that focus on young adults have studied the immediate effects of acute bouts of exercise on cognition. For example, Coles and Tomporowski (2008) evaluated the effects of a brief bout of exercise on executive function, short term memory, and long term memory tests using eighteen young adult men. It was found that exercise-induced arousal does not influence working memory, however it may facilitate consolidation of information into long term memory. While studying the population of

young adults, this study primarily focused on acute bouts of exercise rather than studying the cumulative effect of exercise on cognition in young adults, Overall, research on the effect acute bouts of exercise has been very inconclusive. Some studies suggest that an acute bout of exercise increases the speed of mental performance and does so without significantly affecting accuracy (McGlynn et al. 1977; Hogervorst et al. 1996; Tomporowski & Ganio 2011; Blumenthal & Madden 1988). Furthermore some studies suggest that visuospatial memory and long term memory seem to be positively associated with an acute bout of exercise (Coles & Tomporowski 2008), while other studies suggests the opposite, that acute bouts of exercise do not enhance memory processes (Tomporowski and Ganio 2011). It is clear from the review of this literature that the ways exercise helps our brain function is still in a preliminary phase and requires additional research to tease out claims that are not supported.

While there is considerable research on the effect of chronic exercise on cognition in elderly and juvenile populations, there is a missing body of research on the effect of chronic exercise on cognition in a population consisting of young adults, and of the research conducted there are discrepancies. The intent of this study is to look at precisely that. Since in both populations, children and elderly, high aerobic and anaerobic exercise benefits cognitive ability, it is hypothesized that there will be a positive correlation between vigorous physical activity in young adult years (high school through college) and cognitive abilities measured in episodic memory, perceptual speed and executive function.

Method

Participants

Participants were undergraduate students from the University of Portland between 18 and 21 years of age. Participants were recruited predominantly by Psychology 101 courses. As part of their course credit they are required to participate in studies. In addition, announcements were made to various other psychology classes and programs on campus to encourage participation.

Participant ($N=27$), were predominantly female: 21 females and 6 males. The average age of the participants was 18.89 ($SD = 1.05$) years old. Participants were assigned a number upon entry so that all participants remained anonymous. An envelope was designated to each participant to collect all results for later analysis. None of the participants received any type of compensation for their participation. All study materials and procedures were approved by the Institutional review Board at the University of Portland and all participants gave written informed consent.

Procedures and Materials

Vigorous Exercise History Questionnaire. Participants were taken into the psychology lab with the experimenter and asked to fill out the “Vigorous Exercise History Questionnaire”, a self-report measure to assess physical activity, level of fitness and academic performance in high school and college. This questionnaire inquired about the number of times per week they partook in vigorous physical activity, the average duration of these sessions, the number of athletic teams they were a member of and their level of fitness on a scale from 1-10. Academic performance was measured in terms of SAT, ACT, High School GPA, and College GPA.

Verbal Auditory Learning Test. A variant of the Verbal Auditory Learning Test was used to assess episodic memory. All lists had been previously tested for equivalent memorability (Potter and Keeling, 2005). An auditory recording of 15 words (e.g. drum, bell, coffee, school etc.) was played. Immediately after the presentation, participants were asked to write down as

many of the words they could remember in any order in one minute. The list presentation and recall was repeated five times with different lists. After the participant left, the experimenter counted and recorded how many words they remembered correctly and how many words they remembered incorrectly. The average between all six trials was calculated for each participant.

Trail Making Test A and B. The Trail Making Test A and B was used to assess perceptual speed. This test was confirmed to be a “relatively pure indicator of perceptual speed” (Sanchez-Cubillo et al. 2009) and inter-rater reliability is high, suggesting very little variance of scorers (Fals-Stewart 1992). Participants were shown a sample part A. The experimenter demonstrated the process of connecting the numbers in numerical order from 1-8. The participants were given sample part A and asked to complete it knowing that it is not timed. If any errors were made the experimenter stopped the participant, addressed the error and explained why it was wrong. Once they completed sample A correctly, participants were given part A and asked to complete as fast and accurately as possible connecting the numbers. If any mistakes were made here, the experimenter stopped the participant and asked them to go back to the last correct circle and continue, while still timing. The experimenter recorded the time.

The experimenter then demonstrated part B, which asks the participant to connect the numbers and letters in numerical and alphabetical order (e.g. 1-A-2-B-3-C-4-D etc.). The participant was given sample part B and asked to complete it, knowing that it is not timed. If any errors were made the experimenter stopped the participant, addressed the error and point explained why it was wrong. Once participants completed the sample accurately, they were given part B and asked to complete it as fast and accurately as possible connecting the numbers and letters. If any mistakes were made here, the experimenter stopped the participant and asked

them to go back to the last correct circle and continue, while still timing. The experimenter recorded the time.

Stroop Test. A variant of the Stroop test was used to assess executive functioning (Stroop1935). The Stroop test has demonstrated high validity and reliability (Jenson and Rohwer 1966). First participants were shown a piece of paper with 44 congruent color words (e.g. “RED” was in red ink) and asked to read as fast as possible. The experimenter recorded the time. Next, the participants were presented with 44 incongruent color words (e.g. “RED” was in blue ink) and asked to read as fast as possible. If there were errors made in either the congruent or incongruent color words, the experimenter stopped the participant and asked the participant to start from the last correct word. The experimenter recorded the time. These two tasks were repeated two additional times. The average was calculated for the time in both tasks. The difference between both average times was calculated and averaged for use in statistical analyses.

Data Analysis

Spearman’s correlation was used to analyze the relationship between exercise in high school and level of fitness, exercise in college and level of fitness, GPA, exercise in both college and high school level of fitness with the significance set at $p < .05$. The only measure of academic performance used was high school GPA, as all other measures of academics (SAT, ACT and College GPA), were not complete. Many participants didn’t remember or declined to provide that information.

Pearson’s correlation was used to analyze many relationships: high school GPA v. exercise in both, exercise in both v. correct words remembered, exercise in both v. trail A time,

exercise in both v. trails B time, and exercise in both v. the difference in stroop test times. Correlations were calculated and analyzed with the significance threshold set at $p < .05$.

Results

Descriptive Statistics

For all participants, in high school the average number of hours of exercise per week ranged from 1 to 16.41 hours, with an average of 8.33 (SD = 4.44) hours per week, the average number of athletic teams ranged from 0 to 4.75 teams with an average of 1.61 (SD = 1.06) teams, and the level of perceived fitness ranged from 2-10 with an average of (SD = 1.61). For college, the average number of hours of exercise per week ranged from 0-19 hours, with an average of 5.33 (SD = 4.07) hours per week, the average number of athletic teams ranged from 0 to 1.25 teams with an average of 0.44 (SD = 0.056) teams, and the level of perceived fitness ranged from 2 to 9 with an average of 6.67 (SD = 1.44).

Examining data from each participant, between high school and college the average number hours of exercise per week decreased for 23 of the participants while 4 increased. The average change from high school to college was decreased by 3 hours per week. The perceived level of fitness decreased for 21 of the participants while 6 had an increase in perceived level of fitness. The number of teams decreased for all of the participants.

Correlations

Significant positive correlations were found in exercise in both v. trail A time ($r = .418$, $p < .05$). Positive correlations were also found between exercise in exercise in both v. trails B time ($r = .143$, $p > .05$) and exercise in both v. the difference in stroop test times ($r = .205$, $p > .05$), however the relationships are not significant.

The only significant negative correlation found was between exercise in both v. correct words remembered ($r = -.389, p < .05$). High school GPA v. exercise in both high school and college showed an insignificant negative correlation ($r = -.287, p > .05$).

Discussion

From this study, it is clear that there is a significant negative correlation between episodic memory and a positive correlation between exercise and perceptual speed in one part of the trails test that measured cognitive processing speed and is considered the easier part of the test. However, the results do not point to a clear correlation between exercise and executive function or cognitive flexibility, such as task switching. A majority of previous research conducted with young adults has been done on a comparative level to older populations to look at cognitive decline and deficits as the population ages. What few studies there are, focus on the effect of acute bouts and various cognitive facets affected by the exercise such as verbal memory, visuospatial memory, psychomotor ability, perceptual speed and more. A possible reason for this lack of research is that young adulthood is believed to be a time where we see a peak in cognitive ability so there isn't a reason to see an increase in cognitive abilities (Salthouse & Hasker 2006). The lack of correlation between exercise and executive function or cognitive flexibility, could be explained by the fact that, young adults are supposedly at the top end of their cognitive ability, therefore there isn't much to improve as they have tapped out the cognitive ability, resembling a ceiling effect.

However, of the results in studies conducted with young adults as the target population, there seem to be a few conclusions of significance. First, aerobic exercise can improve the updating component of working memory. Stroth et al. 2010, found a decrease in reaction times in

the two-back test, which requires constantly updating the working memory. Second, aerobic fitness is associated with top-down modulation of responses in tasks that rely on selective attention and inhibitory control. This indicates that as humans, we direct our focus to certain stimuli and ignore other stimuli and this process plays an important role in consciously selecting to pay attention to other things and inhibiting other stimuli from perception. In relation to exercise, highly fit individuals are better able to use executive control processes to modulate their responses in a selective attention task (Guiney & Machado 2013). If participants made an error, they were much slower after, reflecting the effortful modulation of responses to avoid making another error. The results from these studies support the claim that executive function and episodic memory are enhanced by exercise. The results obtained in this study point in the opposite direction, finding no significant correlation in the case of executive function and a negative correlation in episodic memory.

While human studies are limited, there are some animal studies that may lend a hand in explaining the results. Smith et al. (2013) provides evidence for the increase in working memory in rats that had a combination of exercise and working memory training. Specifically they saw an improvement more than double of working memory training alone. Conclusions from rat studies can be extended to humans, as rats are one of the main animal models used for studies in neuroscience. The results from these studies can be used to explain the neurobiological causes underlying the results obtained in this study.

The negative correlation between episodic memory and exercise is perhaps the most baffling of the results obtained in this study. A neurobiological line of study looks into neurotrophins, especially brain derived neurotrophic factor (BDNF), which is implicated in the differentiation, extension and survival of neurons in various brain structures that play important

roles in cognition. These include the hippocampus, cortex, striatum, and cerebellum. BDNF also works in another way, by increasing the docking and fusion of neurotransmitter vesicles within synapses. These synapses respond better leading to enhanced long-term potentiation and learning (Lojovich 2010). Injecting BDNF directly into the hippocampus of rats, there was an increase in the number of neurotransmitter containing vesicles docked on dendritic spines in the hippocampus. In turn, this increased the excitatory postsynaptic potentials, increased dendritic growth and spine density, which all increase the number of future synapses (Tyler & Pozzo-Miller 2001; van Praag et al. 1999). In studies looking at the effect of exercise on BDNF, they found that exercise increased the amount of BDNF mRNA in rats that exercised and as the distance ran increased, so did the level of BDNF mRNA (Neeper et al. 1995). Moreover, looking specifically into the relationship with cognition, Komulainen et al. (2008) looked at the amount of serum BDNF in aging women. From this they concluded that women with higher serum levels scored significantly better on various cognitive tasks than their lower serum level counterparts. This research has provided evidence supporting a positive relationship between episodic memory and exercise. However, this study failed to agree with these findings.

The positive correlation between exercise and perceptual speed seen in this study could be accredited to two different factors, the first being the participant being in a state of arousal. In a study that looked at acute bouts of exercise and the effect that bout of exercise had on perceptual speed, they found that perceptual speed increased post-exercise (Hogervorst et al. 1996). The possible mechanism proposed has to do with the increased heart rate of the participant and thus highly aroused participant. This higher state of arousal could be the main driving factor behind the increased speed in perceptual tasks. However, this study and studies similar to this assume that it is post-exercise, while the participants in my study were not aroused

by exercise. The second possible mechanism, may have to do with the increase in brain circuitry due to the increase in neurotransmitter and neurotrophin concentrations and as such synapses occur quicker.

Due to time constraints on this study, there are certain limitations to the study. First, my sample size was relatively small, thus limiting statistical power and the generalizability of the results. Second, the number of male participants in the study was lacking. There were a much greater number of female participants than male participants. If repeated, I would want to determine a sexual difference between males and females in cognition as well as exercise habits and how they have changed. For example, observing if in high school the majority of their exercise was based on team workouts, such as soccer practice, versus individual workouts, such as going to a personal fitness center.

Future research on the relationship between exercise and cognition should consider including other measures. Specifically, examining physiological evidence such as resting heart rate blood pressure and observing how those differ across the spectrum of activity levels would be an interesting line of study. In addition, studying the neurotransmitter and neurotrophin concentrations in the brain of those who have a higher than average number of hours exercised across their lifetime without intervention, to see how they changed and how they are regulated by exercise. As previously mentioned, many studies on mice or rats have looked at BDNF levels in the hippocampus after an intervention of exercise. A similar study would be informative into the activity of how BDNF concentrations change in humans.

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