

# Descriptive Study of Role Played by Exercise and Diet on Brain Plasticity

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**Abstract**—In today's world, everyone has become so busy in their to-do tasks and daily routine that they tend to ignore some of the basal components of our life, including exercise and diet. This comparative study analyzes the pathways of the relationship between exercise and brain plasticity and also includes another variable diet to study the effects of diet on learning by answering questions including which diet is known to be the best learning supporter and what are the recommended quantities of the same. Further, this study looks into inter-relation between diet and exercise, and also some other approach of the relation between diet and exercise on learning apart from through Brain Derived Neurotrophic Factor (BDNF).

**Keywords**—Basolateral amygdala, brain derived neurotrophic factor, brain plasticity, diet, exercise, mediterranean diet.

## I. INTRODUCTION

EXERCISE is shown to have brought various benefits to humanity. Some of the known direct advantages of exercise are the prevention of chronic health diseases, improvement in insulin sensitivity, enhancement in skin health, and also it is shown to boost our energy, and hence leading to weight loss. Studies have shown that exercise not only facilitates in boosting our energy but also helps in the process of learning and memory consolidation [3]. However, failure to exercise has consequences of its own. Some of them include osteoporosis in which bones become brittle, poor posture, increased risk to coronary heart disease, a condition caused by the building of layers of fats and lipids in our heat arteries leading to blockage. Nearly 1.4 billion (27.5%) of the people do not do enough exercise on a week to week basis leading to a sedentary life. What is more devastating is that these numbers have not changed much since 2001. A person is classified as inactive if he/she is unable to meet to do at least 150 minutes of moderate exercise or 75 minutes of vigorous exercise weekly as guided by the World Health Organization. One thing also must be noted that this physical inactivity does not seem to be guided by a lack of resources. A survey was conducted which found out that in high-income countries, 36.8% of the adult population does not get enough sufficient exercise, followed by 26% in middle-income countries and 16.2% in the low-income nations [4].

Everyone in this world follows a particular diet, be it healthy or junk. It is to be noted that good health does not just

come with physical activity; preferably, a combination of diet and exercise produces the best results [5]. Unhealthy dietary habits are developing various problems like hypertension, type 2 diabetes at young ages [6]. Certain substances in the diet lead to those conditions, which include alcohol, saturated fats, trans fats, salt, sugar. According to WHO, total fat intake should be below 30% of the total energy intake. Out of that, saturated fats should be less than 10%, and trans fat less than 1%. Also, sugar is advised to not exceed 10% and no more than 5 g of salt per day [7]. Despite this, one in 5 deaths globally is because of poor diet [8].

Brain plasticity is referred to as the ability of the brain to modify its existing connections to accommodate gained knowledge. With the increase in brain plasticity, there is also an increment in the learning process since it becomes easier to form new neural connections [2]. In this research, we check the effect of exercise and diet on brain plasticity in 2 regions of the brain: the basolateral amygdala (BLA) and hippocampus. BLA is the region associated with the formation of fear and emotional memory [9], and the hippocampus is considered to play a crucial role in the long term and short term memory consolidation [10]. It is the central connecting point between the relation of exercise and diet, and brain plasticity is a protein called BDNF [1]. Recent studies have shown the paradoxical nature of BDNF in the two different regions of the brain: BLA and hippocampus [11]. Of course, there are other factors as well that play a significant role in brain plasticity like oxidative stress and inflammation, which are caused due to poor diet and increased stress levels, which will be discussed later in this paper [12]. It has also been found that hippocampus is associated with conditional conditioning while the amygdala was associated with cued conditioning [13].

Depression is also taken into account as a mediator between the chosen variables. This is because studies have shown that depression tends to down-regulate neurogenesis and hence also negatively impacts learning [14]. So, it becomes imperative not to ignore this central linker.

## II. EXERCISE AND THE BRAIN PLASTICITY

Studies have shown that exercise aids in the process of learning. To trace back the relation between these two, a study measured levels of BDNF since it leads to neuron proliferation. Exercise leads to increased BDNF in both the amygdala and hippocampus [15]. To analyze the effects of continuous vs. intermittent exercise, one study included mice who ran on a spinning wheel either continuously or on alternative days, and the BDNF levels in the hippocampus

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were measured. There was an increase of 174% in regular exercising group and a 160% increase in the intermittent exercising group as compared to the control group, which did not exercise at all. Some of the experiments are also conducted to strengthen further the claim that the expression of BDNF in the hippocampus leads to enhancement in learning. This enhancement is also dependent on various other factors, one of which is the intensity. Rats were either underwent low intensity or high-intensity workout, and BDNF was injected in both groups. It was seen that there was an enhancement in memory persistence in those who performed low-intensity exercises, but such a difference was not seen in the high-intensity performers. This also proves that up to a specific limit of intensity of exercise, the injected BDNF can play a role in proliferation, or else, the exercise itself produces sufficient BDNF and does not any external source [1].

TABLE I  
 EFFECTS OF DIFFERENT EXERCISES ON DIFFERENT TYPES OF LEARNING

TYPES OF MEMORY	Aerobic	Anaerobic	Yoga
Visuospatial memory	YES	N/A	N/A
Verbal Memory	NO	YES	N/A
Concentration	NO	N/A	N/A
Positive thoughts	YES	N/A	YES
Spatial memory	N/A	YES	N/A
Executive Functions	YES	YES	N/A
Prevention from cognitive decline	YES	YES	YES

### III. DIFFERENT TYPES OF EXERCISES AND THEIR EFFECT ON THE TYPE OF MEMORY

#### A. BDNF and the Hippocampus

To mention the specific forms of exercise that show the improvement in learning via the hippocampus, one research conducted found that endurance training resulted in an increase in visuospatial memory but did not show significant differences in verbal memory and the concentration as compared to the control group which did not exercise at all [71]. The study also found that there was an increase in positive thoughts through endurance training. It might be a misconception among many that only the aerobic exercises provide the benefits of enhanced learning. This is provided by research, which showed that aerobic exercise improved planning scores in previously overweight individuals by aerobic exercise training intervention, which depicts to enhance the functions necessary for cognitive development [65]. However, anaerobic exercises also do not fall behind in supplying the benefits. A study assessed divided healthy individuals into three groups: the first performing high impact anaerobic sprints, the second performing low-intensity aerobic exercises, and the third at rest. The study found that the sprinters had 20% faster vocabulary learning as compared to the other two groups [66]. This claim is further established by another study that compared the effects of resistance training to moderate training in individuals aged 65-75. Resistance training resulted in better verbal and spatial memory [67]. Another study conducted consisted of women aged 65-75, who either performed resistance training with eight reps for 40

minutes, and the other performed mixed balanced exercises for 60 minutes. The resistance trainers were further categorized as one who exercised either once a week or twice a week. There was an improvement in executive functions like planning, decision making, and multi-tasking for both once a week and twice a week resistance trainees [67].

A study inferred from the meta-analysis that aerobic exercise did not seem to increase the hippocampal volume of the brain. However, when the study included participants and made them undergo aerobic training, the results showed an increase in the volume, more specifically the left hippocampal volume. This increase in the volume is essential as the volume of the neural mass in this region decreases with aging leading to cognitive decline. The exercise slows the attenuation of this neural mass rather than increasing its mass. It was also found that there was no increase in the brain volume in people suffering from depression or mild cognitive impairment [68]. One study has linked increased hippocampal volume with better fitness, more specifically cardiorespiratory fitness [69]. It must also be noted that individuals who have a history of physical or sexual abuse in the past or childhood have decreased left hippocampus size. In several other cases as well, we have found that the left hemisphere of the hippocampus is vulnerable to size changes. BDNF levels in the sprinters were also found higher relative to the other two groups resulting in the more short term learning success [70].

Another exciting relation BDNF shows is its ability to cause a reduction in weight. This means, more the expression of BDNF in the hippocampus, higher the weight reduction [1].

#### B. BDNF and Amygdala

Amygdala is also one of the forebrain structures composed of several nuclei, one of which is BLA [16]. The impact of exercise on BLA is not that straight. An experiment was conducted in which mice ran either on a treadmill or a spinning wheel, and the two parts of the brain: the amygdala and hippocampus were observed. The results showed that both exercises up-regulated the synaptic proteins (TrkB and SNAP-25) in the hippocampus leading to increased plasticity, but only the spinning wheel increased spine density and dendritic field in BLA [17]. One study recorded the effects of regular exercise in the BLA and found that the density of 5-hydroxytryptamine 2A receptor (5-HT<sub>2A</sub>) increased, which is known to have an anti-depressive effect [18]. Regular exercise also decreased adenosine A<sub>2A</sub> receptors in the BLA which are known to blunt its anxiolytic effects [19]. 5-HT<sub>2A</sub> receptor is also known to produce anti-inflammatory effects. Furthermore, the 5-HT<sub>2A</sub> receptor was also associated with increased BDNF levels in the various other regions of the brain and has shown to increase the learning [20]. However, studies have also shown that an increase of BDNF in the amygdala induces anxiety-like behaviors [11]. Malfunctioning of the amygdala leads to the development of the bipolar disorder [21]. Some studies report that bipolar disorder leads to an increase in the amygdala volume and decrease in the hippocampal volume [22], whereas one study says that it leads to a reduction in the amygdala volume [23]. One reason for

the conflicting ideas could be the age factor. The latter study was focused on adolescents, while the former had a mean age of around 35 years.

### C. Overexpression of BDNF in the Hippocampus

Although we have so far shown the positive effects of BDNF on learning, examining the effects of overexpression of BDNF and learning, the researches show that overexpression of BDNF hinders learning by stimulating inhibitory pathways. It also found that too little of the BDNF also negatively impacts learning [72]. To validate this claim, several experiments were conducted. One of which was the passive avoidance test, testing short term and long term memory. This test included two types of mice, the wild type (WT) mice without the overexpression of BDNF, and the transgenic mice with overexpression of BDNF. In this test, the apparatus consisted of bright lightroom and the darkroom. This test was done in two cases. The first consisted of a bright room with double the size of the darkroom, whereas in the second case, the size of the rooms was almost the same. Both the mice were left in the bright room, and the door to the darkroom was opened. As the mice moved to a dark room, they experienced a small electric shock. Their latency to enter the darkroom was assessed in 3 cases to test their short term and long term memory: In the initial condition when they first entered the darkroom and experienced shock, 24 hours after the initial condition which meant the 24 hour recall, and after 10 days of the training, which corresponded to long term memory. The results showed that the wild type mice had higher latency before entering the darkroom as compared to the transgenic mice proving that overexpression of BDNF hinders in learning.

In order to see the effects of overexpression on the short term memory, we included one more case consisting of equal-sized rooms and tested their latency after 1 hour of training. The results again showed that overexpression of BDNF was the culprit in both short term and long term memory formation. Also, we have earlier seen BDNF results in decreased bodyweight so that we might assume its overexpression results in a significant decrement in the body weight. However, the study has also shown that overexpression of BDNF provides only a small contribution towards the overall weight loss. It has also been noted that decreased BDNF levels do not correspond to increased body weight. Hence overexpressed BDNF proves to be not so beneficial apart from acting as an anti-depressant [1].

### D. Paradoxical Nature of BDNF in Both Brain Regions

Recent studies have also found that BDNF plays different roles depending on the region it is expressed. Taking into account the intervention of stress, researchers exposed mice to chronic and acute stress and measured changes in the BDNF levels in the BLA and hippocampus. Under acute stress, no changes were observed in the BDNF in the hippocampal region, but under chronic stress, there was a decrement in the BDNF levels. In the BLA, under both acute and chronic stress conditions, BDNF levels showed increment as there was

increased spine density and dendritic growth. Moreover, under acute stress, these increased levels stayed up for at least ten days after 24 hours of stress exposure. In the case of chronic stress, BDNF levels stayed up for 21 days. In the hippocampus, the BDNF showed up-regulation after ten days and 21 days for acute and chronic stress, respectively. They were able to postulate these results as they did two tests on the 21st day: two-tone shock pairing and open field test, in which they showed enhanced fear conditioning due to long freezing time and decreased exploration in the open field [11]. This study also proves the fact that BDNF is transient; that is, it requires continuous stimulation order to stay at high or low levels [24], [25]. Discussion of stress is relatable over here because recent studies have shown that stress as short as lasting for few hours impairs cell to cell communication, which is vital for learning. It is also to be noted that depression leads to decreased hippocampal volume hence affecting neural plasticity [26].

Earlier, we looked at the overexpression of BDNF in the hippocampus. Further research shows that the overexpression of BDNF in the hippocampus is, in fact, the expression of BDNF in BLA. A study found that the BDNF over-expressing mice mentioned earlier showed increased BLA spine density, an indicator of expression of BDNF. This effect was also proved in an experiment in which there was increased BDNF mRNA after 1 hour of exposure to intermittent water-immersion stress [27].

The fact that BDNF expression in the amygdala leads to impairment may mislead to believe that BLA plays only a negative role in memory formation through the hippocampus. However, extensive studies have also demonstrated that BLA is sometimes essential for memory consolidation related to emotions in the hippocampus. This was seen in the inhibitory avoidance test, that inducing histone deacetylase inhibitors in the dorsal hippocampus that enhance memory depends on the functionally intact BLA [28].

## IV. DIET AND THE BRAIN PLASTICITY

The food we eat also plays a significant role in the process of learning. A study analyzed the effects of a western diet rich in saturated fats and refined sugar (High Fat Sugar (HFS) diet) on the brain structure. They found that two months of HFS diet was enough to reduce the hippocampal BDNF levels and hence impacted the spatial memory consolidation. Another relationship that the study demonstrated was consumption of HFS to be inversely related to the BDNF levels [29]. The nutrient dense food and the unhealthy food both impact the hippocampal volume in independent ways. Lower intake of nutrient dense food and higher intake of unhealthy food decrease the hippocampal volume [30]. Another study divided rats into two groups: one fed with corn starch diet and the other with a high sucrose diet for 20 days. On the 21st day, the rats were either given their usual meal or opposite meal, and their oxytocin activity was observed. They found out that chronic sucrose intake interferes with the activity of the anorexigenic oxytocin system, hence reducing satisfaction.

This leads to overeating and hence, weight gain [31]. Another experiment also showed an active link in the oxytocin and sucrose ingestion, that mice lacking oxytocin expression genes had a higher preference for sugar-rich diet [32].

There are numerous misconceptions involved with diet and health. One of the most common beliefs is that if an individual is healthy but mostly sedentary, he/she will not be affected by the high-fat diet. It turns out that the previously healthy but sedentary individuals, when exposed to a high-fat diet even for one week, performed worse on tasks measuring attention, and speed of retrieval than they had before diet consumption [33].

The process of mastication involves the crushing of food, mixing it with saliva, and the formation of a ball like a bolus that is ingested. There has been a correlation between mastication and memory formation. Long term exposure to soft diet leads to memory impairments. One such study fed chow pellets to the control group and a liquid diet to the experimental group. Afterward, they noted that mice on the liquid diet performed significantly lower than the control on the passive avoidance test. Although the BDNF levels in the hippocampus were found higher in the experimental group, the TrkB receptors (to which the BDNF binds and enhances brain plasticity) were found to be lower. Hence, this resulted in decreased neurons in the hippocampus [34]. However, the opposite of this does not hold: hard food is not the food that leads to the most increase in learning. A study analyzed the effects of the varying hardness of food on the number of pyramid cells (multipolar neurons [35]) and the BDNF levels in the hippocampus. The control group was fed with regular food, 1st group with soft food, and the 2nd with hard food. The control group had the highest number of pyramidal cells and the BDNF levels in the hippocampus compared to the other 2 [36]. Another study put the results of this study into the Eight Arm Radial Maze test, which is related to spatial memory. The results were consistent and found that the control group (the one fed with regular food) showed the highest spatial memory. However, these results do not mean that hard food has detrimental effects [37]. One study showed that the hard food diet and enriched environment leads to the recovery of spatial memory in aged mice by bringing it to the levels of the control group [38].

There are multiple hypotheses people adapt to solving the current issue related to obesity. One study analyzed the effects of eating quantity with the duration it was fed, and eliminated the variable of "healthiness of diet." They either food-deprived the mice or fed it with a western diet and further divided them into subgroups depending on the duration of their diet. The results showed that being food deprived even for 48 hours reduced the BDNF levels. The positive energy balance created by the western diet leads to increased BDNF levels and body weight. A possible reason for this increase could be to suppress the weight gain by the body since it increases the anorexigenic activity suppressing the food intake. This all explains the decrease of BDNF in the underfed group as, in this case, BDNF reduction leads to the activation of orexigenic activity, which promotes appetite consumption. It was also mentioned that this BDNF increase was because of the leptin

hormone. However, after three weeks of western diet consumption, BDNF levels declined because of the development of leptin resistance. The BDNF levels noted in this experiment, however, were neither in the hippocampus and nor in the BLA [39]. Increased leptin levels are also associated with increased depressive symptoms [40].

#### A. Diet and Depression

As we reviewed earlier, the discussion of depression is vital since it directly influences the brain plasticity. Intake of high intakes of fruit, vegetables, fish, and whole grains may be associated with reduced depression risk [41]. One study via questionnaire could trace out that there exists a relation between habitual diet quality and depression-like disorders [42]. To test the relation between the quantity of fat in the diet and the damage done on brain plasticity, one study fed mice either western diet with 40% of the fat or high-fat lard diet with 60% fat. Although both the groups had increased adiposity and fasting blood glucose, only the high-fat lard diet showed symptoms of oxidative stress due to decreased antioxidant response [43]. Increased oxidation stress has been associated with diseases like Alzheimer's disease, and also a decrease in working memory and executive skills [44].

One study on the yeast *S. pombe* revealed that increased glucose uptake activated the pathways that lead to increased stress sensitivity and decreased cell survival. It also promoted aging and inhibited stress defense mechanisms. It also found that decreased glucose intake leads to increased oxidative stress resistance and hence provided increased longevity [45].

Another pathway through which a poor diet leads to inhibitory responses in the brain is through inflammation, which has emerged as an essential factor in mood disorders. Patients who suffer from mood disorder show elevated plasma levels of cytokines such as TNF- $\alpha$ , IL-6, and IL-1 $\beta$  [46]. Other studies have shown that high levels of cytokines such as TNF- $\alpha$ , IL-6, and IL-1 $\beta$  lead to inhibition of neurogenesis in the hippocampus. Interestingly, another study showed that inflammation was not linked to cognitive decline, and is a biomarker for oxidative stress, which does lead to cognitive decline [47].

In order to check whether the diet rich only in sugar or the combination of a diet rich in both fat and sugar produced more detrimental effects on the memory, scientists performed a study on mice test subjects. For this, researchers [48] conducted two experiments, and in both the experiments, they included two tests: object (perirhinal-dependent) and place (hippocampal-dependent) recognition memory. In the first experiment, mice were divided into two groups: one was fed with chow and cafeteria food (with 10% of sucrose solution), or chow food. The mice fed with both the cafeteria food and chow food performed worse on the place recognition but not on the object recognition. In the second experiment, the rats were fed with cafeteria food with or without sucrose and control fed with chow diet with sucrose, and both rats performed worse on the place but not on the object. The study also found that rats that ate cafeteria food consumed five times the food as compared to the control and also showed increased

levels of leptin, exhibited hippocampal inflammation and oxidative stress markers [48]. However, one interesting finding was that the rats who consumed the cafeteria diet without added sucrose did not exhibit a significant increase in the markers even though they had similar BDNF levels. This demonstrated that the diet rich in both fat and sugar had more significant detrimental effects even before the weight gain [48].

### B. The Ideal Diet

One of the most beneficial diets reported by several types of research is the Mediterranean (Med) diet. The main components of the Med diet include daily consumption of vegetables, fruits, whole grains, and healthy fats; weekly intake of fish, poultry, beans and eggs; moderate portions of dairy products; and limited intake of red meat [49]. One of the studies found out that continued adherence to the Med diet leads to a protective trend against ischemic stroke, mild cognitive impairment, dementia, and particularly Alzheimer's disease. They also reported that even moderate adherence to this diet was accompanied by reduced risk for depression and cognitive impairment. However, protection against strokes was only marginal in this case [50]. Another study published that the Med diet in older women did provide an overall moderate increase in cognition, but most importantly, it prevented cognitive decline with age [51]. Not only limited to this, but this diet has also shown to reduce the symptoms of metabolic syndrome, which is caused by increased oxidative biomarkers. Hence it also reduces oxidative damage [52]. With this big array of benefits, some studies have also looked at its benefits by combining the Med diet with other diet products. One study combined this diet with nuts and studies its effect on type 2 diabetes (DM2), which is most commonly caused by a sedentary lifestyle. They compared their results to the group, which was only fed with Med diet found out that Med diet with nuts produced significant results in the reduction of depression-like symptoms in the DM2 patients [53]. A similar study studied the combination of Med diet with olive oil that had profound effects on diabetes, obesity, as well as depression. Not limited to this, this combination also provides various other properties like acting as antioxidants, anti-inflammatory response, and discloses antithrombotic

properties. Since olive oils are composed of unsaturated fats, they are also recommended in diabetic treatments as a low-fat alternative [54]. Out of the two combinations mentioned above (Med diet+nuts and med diet +olive oil), a study measured the plasma BDNF levels in both the cases with just low fat diet as the control group on humans suffering from depression. They found that although both the groups with the Med diet had increased BDNF levels, the Med diet+nut combination produced significantly higher results, and hence is the better combination [55].

Some of the other diets that have also shown beneficial effects include diet rich in vitamin E. One study fed mice high-fat diet with or without vitamin E. Although both of the groups did show high oxidative stress, it was reduced to a great extent in the vitamin E group, but the group on high fats showed impairments in the behavior tests. Also, BDNF was normalized in the group on the vitamin E [56]. Another way is to incorporate omega-three fatty acids in the diet. It has shown to normalize the BDNF levels even after Mild Fluid Percussion injury, which increases oxidative stress (and suppresses BDNF expressing pathways) and hence prevents learning disability [57]. Flavonoids, antioxidant-rich berries, resveratrol, and a polyphenol that are found in red grapes and other fruits also stimulate neurogenesis, decrease oxidative stress and pro-inflammatory process [58]. Another new combination diet that produces profound effects on neurogenesis and BDNF increase is by combining polyphenol and polyunsaturated fatty acids. Not only this, but it also protects from cognitive decline with aging and neurodegenerative diseases like Alzheimer's Disease [59]. Lastly, there is another substance called glutathione, which can also be beneficial to include in the diet as it promotes neurogenesis (low levels of glutathione correspond to a decline in the executive functions [60]). Glutathione can also be used in the treatment of bipolar patients, which involves high oxidative stress due to the malfunction of BLA [61]. Its levels can be naturally increased by consumption of foods like vitamin C rich food, and sulfur-rich foods like broccoli, Brussels sprouts, cauliflower, kale, watercress, and mustard greens [62].

TABLE II  
DIFFERENT TYPES OF DIETS AND THEIR EFFECTS

Types of diet	Oxidative Stress	inflammation	obesity	BDNF	Alzheimer's disease	Depression	Other
Med	+	+	0	+	+	+	
Med+olive oil	+	+	+	+	+	+	Antithrombotic properties
Med+nuts	+	+	+	+	+	+	
Vitamin E	+	+	0	+	+	+	
Omega 3 fatty acid	+	+	-	+	+	+	
Flavonoids (in berries)	+	+	0	+	+	+	improves executive function
Polyphenol like Resveratrol (in red wine)	+	N/A	N/A	N/A	+	+	
Glutathione	+	+	N/A	+	+	+	
Polyphenol+unsaturated fatty acid	+	+	+	+	+	+	
Fat rich diet	-	-	-	+/-	-	-	
Sugar rich diet	-	-	-	-	-	-	
Fat+sugar	-	-	-	-	-	-	

+ = positive effects, - = negative effects, 0 = no effect, N/A = not applicable.

Since many of the studies have discussed the positive relation between unhealthy food and poor learning, the reverse is not necessarily true, that is healthy food may not always result in a cognitive increase. A study analyzed the med food eaters were not found to be protective against cognitive decline, but a high intake of monounsaturated fats was predictive of mild cognitive impairment [63].

Finally, looking at the effects when the diet factor combined with exercise, the study divided mice into three groups based on the diet they received (high fat vs. regular vs. high protein) and further divided these groups into whether they had access to the running wheel or not. Results clearly showed that high protein with the exercising group showed the most benefits in BDNF production. In the second experiment, however, they tested the quantity of the protein that gave the most benefits and proved that 1.5% of leucine (an amino acid) in the diet significantly induced BDNF as compared to the group receiving a dose of 3% [64].

#### V. CONCLUSION

- High sucrose diet leads to decreased BDNF.
- Increased western diet leads to an increase in BDNF, but also it leads to an increase in leptin, which is associated with increased depressive symptoms and hence, poor memory.
- Stress or depression-like symptoms lead to decreased BDNF levels in the hippocampus.
- Regular exercise leads to more BDNF levels, and the amount of BDNF levels depends on the intensity of exercise.
- Different exercises lead to an improvement in different memory types, and hence performing mixed exercises of mixed intensity exercises is recommended.
- Only a few types of exercises lead to increased BDNF in the amygdala, one of which is a spinning wheel and not treadmill running. Regular exercise also increases the stress receptors of serotonin, which are known to mediate BDNF levels.
- Overexpression of BDNF in the hippocampus, though acts as an anti-depressant; it hinders in both long term and short term memory formation. However, this overexpressed BDNF does not hinder with behavioral performance. This overexpression also diminishes the anorexigenic effect of BDNF.
- BDNF is known to have a paradoxical nature under various degrees of stress. In the BLA under both chronic and acute stress, BDNF levels stay high, whereas in the hippocampus under chronic stress, they stay low. Hence the overexpression of BDNF in the hippocampus is the expression of BDNF in the BLA.
- The expression of BDNF in BLA also plays positive roles like stress reduction by increasing and also plays a vital role in cued fear conditioning. Inducing histone deacetylase inhibitors in the dorsal hippocampus improves memory that is also dependent on BLA.
- A western diet rich in high sugar and saturated fats leads to a decrease in hippocampal volumes. Also, long term

high sugar intake leads to increased appetite intake hence leading to weight gain. This increase in the appetite consumption also suppresses the BDNF since the presence of BDNF is an indicator of lower appetite consumption.

- The consumption of a high-fat diet by healthy but sedentary individuals has the same detrimental effect of decreased concentration as that of an unhealthy individual who is sedentary.
- Soft and liquid diet leads to reduced BDNF levels if consumed for a long. The hard diet is also not the best-recommended diet. A regular diet is a best-recommended diet aiding in neurogenesis.
- Staying food-deprived for longer durations is not found beneficial since it reduces the BDNF levels. Also, increasing the western diet increases BDNF because of leptin but only up to limited time to decrease the food intake. After that limit, the leptin resistance is developed, and hence, the BDNF decreases. Since leptin leads to hippocampal inflammation, an increased quantity of diet is also detrimental.
- Detrimental effects of unhealthy food are directly related to their quantity of intake.
- Inflammation and oxidative stress both produce detrimental effects; however, inflammation does not lead to a reduction in executive functions, whereas oxidative stress does. However, inflammation leads to activation of cytokines that inhibit neurogenesis
- Sugar, when combined with fats, leads to enhanced leptin production, contributing to increased inflammation in the hippocampus and oxidative responses than fats alone. This also means fat+sugar combination leads to lower BDNF expression.
- Med diet is considered the ideal diet for the prevention of dementia, Alzheimer's disease, cognitive impairment, depression-like symptoms, and also reduces oxidative stress and inflammation.
- Combinations of the med diet with various other diets also produce enhanced benefits than the med diet alone. For example, for treating DM2, the combination of the med diet with nuts seems the most beneficial. Hence this combination must also enhance BDNF levels to have performed the function of an appetite suppressor. Combining a med diet with olive oil can be an alternative to low-fat food and also produces various benefits like reducing oxidative stress and also antithrombotic properties. However, in neurogenesis, Med diet+ nuts is a better combination.
- Other products that are considered beneficial hence recommended include vitamin E rich diet, omega three fatty acid, flavonoids, antioxidant-rich berries, resveratrol, and polyphenol. Combining polyphenol and unsaturated fats produced enhanced BDNF levels.
- Another substance called Glutathione, whose deficiency causes impairment in executive functions, can also be used to fix the bipolar disorder by increasing the hippocampal volume and decreasing the amygdala

volume in middle-aged people. Glutathione can also increase the amygdala volume (in adolescents) depending on the age group.

- There is also a possibility that an unhealthy diet, which increases the BDNF due to leptin, also increases the BDNF in the BLA as it induces stress.
- The bidirectional link between an unhealthy diet and stress levels does not exist.
- A combination of a healthy diet, when combined with regular exercise, produces additive benefits on the BDNF.
- The quantity of the healthy diet also matters and is not directly related to the increase in the BDNF. For example, 1.5% of leucine amino acids produce better results than 3% of it.

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#### REFERENCES

[1] Praveen Saroha, Mridul Sharma, "Comparative Study on the Effects of Exercise and Sleep on the Process of Learning", UGC Sponsored 3rd National Conference in "UGC Sponsored 3rd National Conference on Latest Trends in Health and Physical Education book ISBN: 978-81-7524-879-3" pp 78,pg 280-pg282.

[2] <http://scitechconnect.elsevier.com/what-is-brain-plasticity-why-so-important/>

[3] <https://www.healthline.com/nutrition/10-benefits-of-exercise#section10>.

[4] <https://globalsportmatters.com/health/2018/10/30/over-1-in-4-people-across-the-world-dont-get-enough-exercise-study-says/>

[5] <https://www.healthline.com/health/fitness-exercise-eating-healthy>

[6] <https://www.sahealth.sa.gov.au/wps/wcm/connect/public/content/sa+health+internet/healthy+living/is+your+health+at+risk/the+risks+of+poor+nutrition>

[7] <https://www.who.int/news-room/fact-sheets/detail/healthy-diet>

[8] <https://medicalxpress.com/news/2019-04-globally-deaths-poor-diet.html>

[9] <https://www.sciencedirect.com/topics/psychology/basolateral-amygdala>

[10] <https://en.wikipedia.org/wiki/Hippocampus>

[11] Lakshmiarasimhan H, Chattarji S., "Stress leads to contrasting effects on the levels of brain derived neurotrophic factor in the hippocampus and amygdala", *PLoS One*. 2012;7(1):e30481. doi: 10.1371/journal.pone.0030481. Epub 2012 Jan 17.

[12] Felice N. Jacka, Nicolas Cherbuin, Kaarin J. Anstey, Perminder Sachdev, and Peter Butterworth, "Western Diet is Associated with a Smaller Hippocampus: A longitudinal investigation," Published online 2015 Sep 8. doi: 10.1186/s12916-015-0461-x, Sydney, 2015.

[13] Peter Curzon, Nathan R. Rustay, and Kaitlin E. Browman, "Cued and Contextual Fear Conditioning for Rodents" in "Methods of Behavior Analysis in Neuroscience. 2nd edition" chapter 2.

[14] Nicola D Hanson, Michael J Owens, and Charles B Nemeroff, "Depression, Antidepressants, and Neurogenesis," *Neuropsychopharmacology*. 2011; 36:2589–2602. doi: 10.1038/npp.2011.220.

[15] Tzu-Wei, Lina Yao-Hsiang, Shiha Shean-Jen, Chenbcd Chi-Hsiang, Lienb Chia-Yuan, Changb Tung-Yi, Huangc Shun-Hua, Chenaf Chauiyng, J. Jenae Yu-Min Kuo, "Running exercise delays neurodegeneration in amygdala and hippocampus of Alzheimer's disease (APP/PS1) transgenic mice",

[16] A.M. Poulos K.M. Christian R.F. Thompson, "Procedural Learning: Classical Conditioning", <https://doi.org/10.1016/B978-012370509-9.00121-2>

[17] Tzu-Wei Lin, Shean-Jen Chen, Tung-Yi Huang, Chia-Yuan Chang, Jih-Ing Chuang, Fong-Sen Wu, Yu-Min Kuo, Chauiyng J. Jen, "Different types of exercise induce differential effects on neuronal adaptations and memory performance", in "Neurobiology of Learning and Memory"

Volume 97, Issue 1, January 2012, Pages 140-147

[18] Schreiber, R., Melon, C. & De Vry, J. *Psychopharmacology* (1998) 135: 383. <https://doi.org/10.1007/s002130050526>

[19] Leem YH, Jang JH, Park JS, Kim HS, "Exercise exerts an anxiolytic effect against repeated restraint stress through 5-HT2A-mediated suppression of the adenosine A2A receptor in the basolateral amygdala", *Psychoneuroendocrinology*. 2019 Oct; 108:182-189. doi: 10.1016/j.psyneuen.2019.06.005. Epub 2019 Jun 12.

[20] De-guo Jiang, Shi-li Jin, Gong-ying Li, Qing-qing Li, Zhi-ruo Li, Hong-xia Ma, Chuan-jun Zhuo, Rong-huan Jiang, M.D, and Min-jie Ye, Ph.D, "Serotonin regulates brain-derived neurotrophic factor expression in select brain regions during acute psychological stress" *Neural Regen Res*. 2016 Sep; 11(9): 1471–1479 doi: 10.4103/1673-5374.191222.

[21] Lara C. Foland-Ross, PhD, John O. Brooks, III, PhD, MD, Jim Mintz, George Bartzokis, MD, Jennifer Townsend, BA, Paul M. Thompson, PhD and Lori L. Altshuler, MD, "Mood-state effects on amygdala volume in bipolar disorder", *J Affect Disord*. 2012 Aug; 139(3): 298–301. Published online 2012 Apr 21. doi: 10.1016/j.jad.2012.03.003

[22] Brambilla P1, Harenski K, Nicoletti M, Sassi RB, Mallinger AG, Frank E, Kupfer DJ, Keshavan MS, Soares JC, "MRI investigation of temporal lobe structures in bipolar patients", *J Psychiatr Res*. 2003 Jul-Aug; 37(4):287-95.

[23] Jessica H. Kalmar Ph.D, Fei Wang M.D., Ph.D. Lara G. Chepenik M.D., Ph.D., Fay Y. Womer M.D., Monique M. Jones B.S., Brian Pittman M.A., Maulik P. Shah M.D., Andrés Martin M.D., M.P.H.R. Todd Constable Ph.D., Hilary P. Blumberg M.D, "Relation Between Amygdala Structure and Function in Adolescents With Bipolar Disorder", in "Journal of the American Academy of Child & Adolescent Psychiatry" Volume 48, Issue 6, June 2009, Pages 636-642.

[24] Conrad, C. D., Magariños, A. M., LeDoux, J. E., & McEwen, B. S. (1999). Repeated restraint stress facilitates fear conditioning independently of causing hippocampal CA3 dendritic atrophy. *Behavioral Neuroscience*, 113(5), 902-913. <http://dx.doi.org/10.1037/0735-7044.113.5.902>.

[25] Hui Yu, and Zhe-yu Chen, "The role of BDNF in depression on the basis of its location in the neural circuitry", *Acta Pharmacol Sin*. 2011 Jan; 32(1): 3–11. Published online 2010 Dec 6. doi: 10.1038/aps.2010.184

[26] University of California - Irvine. "Short-term Stress Can Affect Learning and Memory." *ScienceDaily*, 13 March 2008. <[www.sciencedaily.com/releases/2008/03/080311182434.htm](http://www.sciencedaily.com/releases/2008/03/080311182434.htm)>.

[27] Govindarajan A, Rao BS, Nair D, Trinh M, Mawjee N, Tonegawa S, Chattarji S, "Transgenic brain-derived neurotrophic factor expression causes both angiogenic and antidepressant effects", *Proc Natl Acad Sci U S A*. 2006 Aug 29;103(35):13208-13. Epub 2006 Aug 21.

[28] Martina Blank, Arethusa S. Dornelles, Aline Werenciz, Luciana A. Velho, Diana F. Pinto, Ana Cláudia Fedi, Nadja Schröder, Rafael Roessler, "Basolateral amygdala activity is required for enhancement of memory consolidation produced by histone deacetylase inhibition in the hippocampus", in *Neurobiology of Learning and Memory* Volume 111, May 2014, Pages 1-8.

[29] Molteni R, Barnard RJ, Ying Z, Roberts CK, Gómez-Pinilla F, "A high-fat, refined sugar diet reduces hippocampal brain-derived neurotrophic factor, neuronal plasticity, and learning", *Neuroscience*. 2002; 112(4):803-14.

[30] Felice N. Jacka, Nicolas Cherbuin, Kaarin J. Anstey, Perminder Sachdev, and Peter Butterworth, "Western diet is associated with a smaller hippocampus: a longitudinal investigation", *BMC Med*. 2015; 13: 215. Published online 2015 Sep 8. doi: 10.1186/s12916-015-0461-x

[31] Anaya Mitra, Blake A. Gosnell, Helgi B. Schiöth, Martha K. Grace, Anica Klockars, Pawel K. Olszewski, and Allen S. Levine, "Chronic sugar intake dampens feeding-related activity of neurons synthesizing a satiety mediator, oxytocin", Published in final edited form as: *Peptides*. 2010 Jul; 31(7): 1346–1352. Published online 2010 Apr 22. doi: 10.1016/j.peptides.2010.04.005.

[32] Amico JA, Vollmer RR, Cai HM, Miedlar JA, Rinaman L, "Enhanced initial and sustained intake of sucrose solution in mice with an oxytocin gene deletion", *Am J Physiol Regul Integr Comp Physiol*. 2005 Dec; 289(6):R1798-806. Epub 2005 Sep 8.

[33] Edwards LM, Murray AJ, Holloway CJ, Carter EE, Kemp GJ, Codreanu I, Brooker H, Tyler DJ, Robbins PA, Clarke K, "Short-term consumption of a high-fat diet impairs whole-body efficiency and cognitive function in sedentary men.", *FASEB J*. 2011 Mar;25(3):1088-96. doi: 10.1096/fj.10-171983. Epub 2010 Nov 24.

[34] Okihara H, Ito J, Kokai S, Ishida T, Hiranuma M, Kato C, Yabushita T, Ishida K, Ono T, Michikawa M, "Liquid diet induces memory

- impairment accompanied by a decreased number of hippocampal neurons in mice.” *J Neurosci Res*. 2014 Aug;92(8):1010-7. doi: 10.1002/jnr.23383. Epub 2014 Mar 31.
- [35] G.N.Elston, “Specialization of the Neocortical Pyramidal Cell during Primate Evolution”, <https://doi.org/10.1016/B0-12-370878-8/00164-6>
- [36] Jenny Sunariani, Christian Khoswanto, and Wahyuning Ratih Irmalia, “Difference of brain-derived neurotrophic factor expression and pyramidal cell count during mastication of food with varying hardness”, *J Appl Oral Sci*. 2019; 27: e20180182. Published online 2019 Apr 1. doi: 10.1590/1678-7757-2018-0182.
- [37] Wahyuning Ratih Irmalia, Jenny Sunariani, Christian Khoswanto, “Different Food Hardness Affect Memory”, *Journal of International Dental and Medical Research* ISSN 1309-100X Food Hardness Affect Memory <http://www.jidmr.com> Wahyuning Ratih Irmalia, and et al.
- [38] Mendes Fde C, de Almeida MN, Felício AP, Fadel AC, Silva Dde J, Borralho TG, da Silva RP, Bento-Torres J, Vasconcelos PF, Perry VH, Ramos EM, Picanço-Diniz CW, Sosthenes MC, “Enriched environment and masticatory activity rehabilitation recover spatial memory decline in aged mice.”, *BMC Neurosci*. 2013 Jun 28;14:63. doi: 10.1186/1471-2202-14-63.
- [39] Kaitlyn E. Gilland and Edward A. Fox, “Effect of food deprivation or short-term Western diet feeding on BDNF protein expression in the hypothalamic arcuate, paraventricular, and ventromedial nuclei”, *07 APR 2017* <https://doi.org/10.1152/ajpregu.00256.2016>.
- [40] Pasco JA, Jacka FN, Williams LJ, Henry MJ, Nicholson GC, Kotowicz MA, Berk M., “Leptin in depressed women: cross-sectional and longitudinal data from an epidemiologic study.”, *J Affect Disord*. 2008 Apr; 107(1-3):221-5. Epub 2007 Aug 28.
- [41] Lai JS, Hiles S, Bisquera A, Hure AJ, McEvoy M, Attia J, “A systematic review and meta-analysis of dietary patterns and depression in community-dwelling adults.”, *Am J Clin Nutr*. 2014 Jan;99(1):181-97. doi: 10.3945/ajcn.113.069880. Epub 2013 Nov 6.
- [42] Jacka FN, Pasco JA, Mykletun A, Williams LJ, Hodge AM, O'Reilly SL, Nicholson GC, Kotowicz MA, Berk M., “Association of Western and traditional diets with depression and anxiety in women.”, *Am J Psychiatry*. 2010 Mar;167(3):305-11. doi: 10.1176/appi.ajp.2009.09060881. Epub 2010 Jan 4.
- [43] Morrison CD, Pistell PJ, Ingram DK, Johnson WD, Liu Y, Fernandez-Kim SO, White CL, Purpera MN, Uranga RM, Bruce-Keller AJ, Keller JN, “High fat diet increases hippocampal oxidative stress and cognitive impairment in aged mice: implications for decreased Nrf2 signaling.”, *J Neurochem*. 2010 Sep;114(6):1581-9. doi: 10.1111/j.1471-4159.2010.06865.x. Epub 2010 Jul 27.
- [44] Ihab Hajjar, Salim S. Hayek, Felicia C. Goldstein, Greg Martin, Dean P. Jones, and Arshed Quyyumi, “Oxidative stress predicts cognitive decline with aging in healthy adults: an observational study”, *J Neuroinflammation*. 2018; 15: 17. Published online 2018 Jan 16. doi: 10.1186/s12974-017-1026-z.
- [45] A Antoine E. Roux, Alexandre Leroux, Manal A. Alaamery, Charles S. Hoffman, Pascal Chartrand, Gerardo Ferbeyre, Luis A. Rokeach, “Pro-Aging Effects of Glucose Signaling through a G Protein-Coupled Glucose Receptor in Fission Yeast”, Published: March 6, 2009 <https://doi.org/10.1371/journal.pgen.1000408>.
- [46] Helen M. Melo, Luis Eduardo Santos, and Sergio T. Ferreira, “Diet-Derived Fatty Acids, Brain Inflammation, and Mental Health”, *Front Neurosci*. 2019; 13: 265. Published online 2019 Mar 26. doi: 10.3389/fnins.2019.00265.
- [47] Ihab Hajjar, Salim S. Hayek, Felicia C. Goldstein, Greg Martin, Dean P. Jones, and Arshed Quyyumi, “Oxidative stress predicts cognitive decline with aging in healthy adults: an observational study”, *J Neuroinflammation*. 2018; 15: 17. Published online 2018 Jan 16. doi: 10.1186/s12974-017-1026-z.
- [48] Beilharz JE, Maniam J, Morris MJ, “Short exposure to a diet rich in both fat and sugar or sugar alone impairs place, but not object recognition memory in rats.”, *Brain Behav Immun*. 2014 Mar;37:134-41. doi: 10.1016/j.bbi.2013.11.016. Epub 2013 Dec 3.
- [49] <https://www.mayoclinic.org/healthy-lifestyle/nutrition-and-healthy-eating/in-depth/mediterranean-diet/art-20047801>.
- [50] Psaltopoulou T, Sergentanis TN, Panagiotakos DB, Sergentanis IN, Kosti R, Scarmeas N, “Mediterranean diet, stroke, cognitive impairment, and depression: A meta-analysis”, *Ann Neurol*. 2013 Oct;74(4):580-91. doi: 10.1002/ana.23944. Epub 2013 Sep 16.
- [51] Samieri C, Okereke OI, E Devore E, Grodstein F, “Long-term adherence to the Mediterranean diet is associated with overall cognitive status, but not cognitive decline, in women.”, *J Nutr*. 2013 Apr;143(4):493-9. doi: 10.3945/jn.112.169896. Epub 2013 Jan 30.
- [52] Mitjavila MT, Fandos M, Salas-Salvadó J, Covas MI, Borrego S, Estruch R, Lamuela-Raventós R, Corella D, Martínez-González MÁ, Sánchez JM, Bulló M, Fitó M, Tormos C, Cerdá C, Casillas R, Moreno JJ, Iradi A, Zaragoza C, Chaves J, Sáez GT, “The Mediterranean diet improves the systemic lipid and DNA oxidative damage in metabolic syndrome individuals. A randomized, controlled, trial.”, *Clin Nutr*. 2013 Apr;32(2):172-8. doi: 10.1016/j.clnu.2012.08.002. Epub 2012 Aug 31.
- [53] Almudena Sánchez-Villegas, Miguel Angel Martínez-González, Ramón Estruch, Jordi Salas-Salvadó, Dolores Corella, Maria Isabel Covas, Fernando Arós, Dora Romaguera, Enrique Gómez-Gracia, José Lapetra, Xavier Pintó, Jose Alfredo Martínez, Rosa María Lamuela-Raventós, Emilio Ros, Alfredo Gea, Julia Wärnberg, and Lluís Serra-Majem, “Mediterranean dietary pattern and depression: the PREDIMED randomized trial”, *BMC Med*. 2013; 11: 208. Published online 2013 Sep 20. doi: 10.1186/1741-7015-11-208.
- [54] Pérez-Martínez P1, García-Ríos A, Delgado-Lista J, Pérez-Jiménez F, López-Miranda J., “Mediterranean diet rich in olive oil and obesity, metabolic syndrome and diabetes mellitus.”, *Curr Pharm Des*. 2011;17(8):769-77.
- [55] Mitjavila MT1, Fandos M, Salas-Salvadó J, Covas MI, Borrego S, Estruch R, Lamuela-Raventós R, Corella D, Martínez-González MÁ, Sánchez JM, Bulló M, Fitó M, Tormos C, Cerdá C, Casillas R, Moreno JJ, Iradi A, Zaragoza C, Chaves J, Sáez GT, “The Mediterranean diet improves the systemic lipid and DNA oxidative damage in metabolic syndrome individuals. A randomized, controlled, trial.”, *Clin Nutr*. 2013 Apr;32(2):172-8. doi: 10.1016/j.clnu.2012.08.002. Epub 2012 Aug 31.
- [56] Wu A1, Ying Z, Gomez-Pinilla F., “The interplay between oxidative stress and brain-derived neurotrophic factor modulates the outcome of a saturated fat diet on synaptic plasticity and cognition”, *Eur J Neurosci*. 2004 Apr;19(7):1699-707.
- [57] Wu A1, Ying Z, Gomez-Pinilla F., “Dietary omega-3 fatty acids normalize BDNF levels, reduce oxidative damage, and counteract learning disability after traumatic brain injury in rats.”, *J Neurotrauma*. 2004 Oct;21(10):1457-67.
- [58] Alberto Granzotto and Paolo Zatta, “Resveratrol and Alzheimer’s disease: message in a bottle on red wine and cognition”, *Front. Aging Neurosci*. 14 May 2014 | <https://doi.org/10.3389/fnagi.2014.00095>.
- [59] Valente T1, Hidalgo J, Bolea I, Ramirez B, Anglés N, Reguant J, Morelló JR, Gutiérrez C, Boada M, Unzeta M., “A diet enriched in polyphenols and polyunsaturated fatty acids, LMN diet, induces neurogenesis in the subventricular zone and hippocampus of adult mouse brain.”, *J Alzheimers Dis*. 2009;18(4):849-65. doi: 10.3233/JAD-2009-1188.
- [60] Nisha Singh, Adriane R Rosa, Eleanor Whitaker, Marianne de Britto, Alexander Lewis, Edward Vieta, Grant C Churchill, John R Geddes, and Guy M Goodwin, “Relating mood to plasma glutathione and BDNF levels in patients with bipolar disorder”, *FASEB journal* Vol. 27, No. 1 supplement.
- [61] Ihab Hajjar, Salim S. Hayek, Felicia C. Goldstein, Greg Martin, Dean P. Jones, and Arshed Quyyumi, “Oxidative stress predicts cognitive decline with aging in healthy adults: an observational study”, *J Neuroinflammation*. 2018; 15: 17. Published online 2018 Jan 16. doi: 10.1186/s12974-017-1026-z.
- [62] <https://www.healthline.com/nutrition/how-to-increase-glutathione>.
- [63] Cherbuin N1, Anstey KJ, “The Mediterranean diet is not related to cognitive change in a large prospective investigation: the PATH through Life study”, *Am J Geriatr Psychiatry*. 2012 Jul;20(7):635-9. doi: 10.1097/JGP.0b013e31823032a9.
- [64] Nadine Zeeni, Edwina Abou Haidar, Adriana Azar, Anthony Ghanem, Katherine Bassil, Maya Bassil, and Sama Sleiman, “The Combinatory Effects of Diet and Exercise on BDNF Gene Expression”, *FASEB journal* Vol. 31, No. 1 supplement April 2017
- [65] Catherine L. Davis, Phillip D. Tomporowski, Colleen A. Boyle, Jennifer L. Waller, Patricia H. Miller, Jack A. Naglieri, and Mathew Gregoski, “Effects of aerobic exercise on overweight children’s cognitive functioning”, *Res Q Exerc Sport*. 2007 Dec; 78(5): 510519. doi: 10.1080/02701367.2007.10599450
- [66] Winter B, Breitenstein C, Mooren FC, Voelker K, Fobker M, Lechtermann A, Krueger K, Fromme A, Korsukewitz C, Floel A, Knecht S, “High impact running improves learning.”, *Neurobiol Learn Mem*. 2007 May;87(4):597-609. Epub 2006 Dec 20.
- [67] Coursera online course “Biohacking Your Brain’s Health”, by Prof. Karima Benameur, Department of Neurology, School of Medicine, Emory University.



- [68] S Matura, J Fleckenstein, R Deichmann, T Engeroff, E Füzéki, E Hattingen, R Hellweg, B Lienerth, U Pilatus, S Schwarz, V A Tesky, L Vogt, W Banzer, and J Pantel, "Effects of aerobic exercise on brain metabolism and grey matter volume in older adults: results of the randomised controlled SMART trial", *Transl Psychiatry*. 2017 Jul; 7(7): e1172. Published online 2017 Jul 18. doi: 10.1038/tp.2017.135
- [69] Kirk I. Erickson, Regina L. Leckie, Andrea M. Weinstein, "Physical activity, fitness, and gray matter volume", <https://doi.org/10.1016/j.neurobiolaging.2014.03.034>.
- [70] Thomas Frodl, Veronica O'Keane, "Chapter 9 - Integrating the Stress Systems and Neuroimaging in Depression", <https://doi.org/10.1016/B978-0-12-802456-0.00009-1>.
- [71] Stroth, Sanna & Hille, Katrin & Spitzer, Manfred & Reinhardt, Ralf. (2008). Aerobic endurance exercise benefits memory and affect in young adults. *Neuropsychological rehabilitation*. 19. 223-43. 10.1080/09602010802091183.

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