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The effect of direct transcranial current stimulation of the dorsolateral prefrontal cortex with aerobic training on food craving, attentional bias to food, and cardiometabolic indices in obese men

Hamid Khazaei[1], Bahman Hassanvand[1]*

[1] *Department of exercise physiology, Khor.C, Islamic Azad University, Khorramabad, Iran.*

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Abstract

Introduction: The present study aimed to investigate the effect of direct transcranial stimulation of the dorsolateral frontal cortex with aerobic exercise on food craving, attentional bias to food, and cardiometabolic indices in obese men.

Methods: The research type was a quasi-experimental study with a pre-test-post-test design. The subjects included 24 obese men who were selected purposively and according to the inclusion criteria. The subjects were randomly assigned to two groups: real-time stimulation of the posterior lateral frontal cortex with aerobic exercise and sham stimulation with aerobic exercise. In the pre-test and post-test, participants completed a food craving and attentional bias questionnaire. The moderate-intensity endurance training program was 8 weeks long, with 3 sessions per week. Then, blood sampling was performed 10 hours later. Data analysis was performed using paired t-tests and analysis of covariance.

Results: The results showed that real stimulation with aerobic exercise had a significant effect on reducing cravings and food attention bias in obese men ($P<0.01$). However, sham stimulation with aerobic exercise had no significant effect on these variables ($P<0.05$). Also, these conditions resulted in a decrease in fasting glucose, insulin resistance, total cholesterol, triglycerides, and LDL, and a greater increase in HDL in obese men ($P<0.05$).

Conclusions: The results showed that real stimulation with aerobic exercise had a significant effect on reducing cravings and food attention bias in obese men ($P<0.01$). However, sham stimulation with aerobic exercise had no significant effect on these variables ($P<0.05$). Also, these conditions resulted in a decrease in fasting glucose, insulin resistance, total cholesterol, triglycerides, and LDL, and a greater increase in HDL in obese men ($P<0.05$).

Key Words: Non-invasive brain stimulation, dorsolateral prefrontal cortex , food craving, attentional bias, cardiometabolic syndrome.

Corresponding author: Bahman Hassanvand, Assistant Professor, *Department of exercise physiology, Khor.C, Islamic Azad University, Khorramabad, Iran. Email:* Hasanvand121@iau.ac.ir

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1. Introduction

Obesity, as a chronic and complex disease, has become one of the major health problems worldwide. It is associated with a significant increase in the risk of cardiometabolic diseases such as type 2 diabetes, hypertension, and dyslipidemia. To the extent that it is called the syndrome of today's world. This condition not only severely affects the quality of life of individuals, but also imposes a heavy economic burden on health systems. Also, according to reliable statistics, the prevalence of obesity is increasing day by day. In addition to genetic and environmental factors, food cravings and impaired appetite regulation play a key role in the development and persistence of obesity. In the meantime, food cravings, which means a strong desire to eat food, have been considered by researchers as an important factor in causing obesity, and past results also indicate the involvement of this factor in the occurrence of obesity. Therefore, it seems that better knowledge in this field can help provide weight control programs (8). On the other hand, due to the complexity of the neural mechanisms involved in food craving, the need for multidimensional management strategies is more evident (9, 10). Recent studies have shown that certain areas of the brain, especially the dorsolateral prefrontal cortex, are involved in the control of eating behavior, decision-making, and cognitive regulation of food craving (11). In the past, the role of this area in various mechanisms, including working memory, cognitive flexibility, decision-making, impulsivity, problem solving, and many other cases, has been clarified. In this context, it has been well established that there is an inverse relationship between the activity of the dorsolateral prefrontal cortex and food craving, and that increased activity in it will lead to excessive food craving and impulsive decision-making. The term impulsivity also refers to the tendency to act with minimal control and thought, and its role in intensifying food craving, irregular dieting, and overeating leading to obesity has been proven. Previous results also confirm the impairment of cognitive activity related to the dorsolateral prefrontal cortex in obese individuals, which will lead to an increase in food cravings and the inability to control impulses (2). Therefore, research related to food cravings and obesity has often focused on this brain area. Another psychological factor that causes obesity is attentional bias towards food (3, 4). Attentional bias towards food actually means that when someone is exposed to several stimuli at the same time, their attention is drawn to a specific stimulus such as food (1). In support of this, it has been shown that obese individuals show a greater attentional bias towards food, and changes in brain activity in areas related to impulsivity and compulsive behaviors have been observed in these individuals (2). Therefore, it seems that attentional bias towards food is common in obese individuals (3). Therefore, it is likely that many obese individuals are involved in a type of emotional eating disorder that will predispose them to overeating (4). This disorder is characterized by the duality of a feeling of lack of control and overeating with repeated episodes of excessive consumption (5). On the other hand, the positive effects of physical exercise on obesity control have been well established, and the positive effects of physical activity on physiological and psychological factors unrelated to obesity are clear. In support of this, previous results have reported the positive regulatory role of physical activity on weight management through its effects on food cravings and cognitive inhibition (8, 12). Although all types of physical activity are useful in controlling obesity, it has been determined that the most appropriate training method for this purpose is endurance training programs. For example, previous results have shown the effect of brisk walking and moderate aerobic exercise in reducing food cravings (11). Therefore, it seems that this is related to increased activity in the PFC and dorsolateral prefrontal cortex, which leads to improved mechanisms involved in appetite control and overeating (6). Some sources mention tDCS as a tool for controlling cognitive behaviors, as it is used in many cases of mental and neurological disorders (7, 8), so this approach has also been considered for controlling dysfunctional behavior related to food (9). tDCS improves motor function and motor skills by modulating the resting membrane potential of neurons, increasing synaptic plasticity and strengthening the functional connectivity of neural networks. Therefore, this method seems to be involved in improving learning and memory. Other effects include increasing regional cerebral blood flow, strengthening the functional connectivity of the brain network and increasing the secretion of excitatory neurotransmitters. Therefore, today, the wide applications of tDCS in sports science and medicine are common. As it has been shown, this will improve muscle capabilities and aerobic metabolism, reduce fatigue and improve cognition. It is also of great value for athletes, the elderly, and patients with neurological injuries (15, 16). tDCS involves the stimulation of the cerebral cortex using a constant low-amperage electrical current to the cerebral cortex through scalp electrodes. The excitability of the cerebral cortex is increased by anodal stimulation and decreased by cathodal stimulation (10, 11). Therefore, in recent years, noninvasive brain stimulation methods such as tDCS have been considered as a promising tool for modulating neural activities and improving cognitive functions. tDCS is able to change the excitability threshold of neurons and thereby affect the neural networks associated with appetite control and eating behavior. Now, considering the critical role of the dorsolateral prefrontal cortex in regulating food craving and the known benefits of aerobic exercise in obesity management, this study aimed to investigate the combined effect of transcranial direct stimulation of the prefrontal cortex and transcranial direct stimulation of the prefrontal cortex.

Methods

The present study, considering the anticipated objectives, is quasi-experimental research and also a cross-sectional study considering the duration of the research. The present study design is a pre-test-post-test with an experimental group and a control group. The present study was also an applied research in terms of its purpose. The present study was also conducted in the field in terms of its implementation. The subjects were 24 obese male athletes selected through a purposive sampling method and placed in two groups of 12. These individuals were selected according to the research criteria and voluntarily expressed their willingness to participate. Gpower software (version 3.1 and alpha 5%, beta 80% and effect size 0.30) was used to determine the sample size (19). The inclusion criteria for the study were no history of injuries affecting motor function (no sports activity in the past 6 months), completion of an informed consent form, age range between 30 and 40 years, body mass index above 30 kg/m2, medical certificate of absence of any metal object in the body, and absence of any cardiovascular disease. The exclusion criteria for the study were also withdrawal from participation in the study, absence on the day of the study, injury, lack of motivation to continue cooperation, and lack of regular participation in training sessions.

Table 1 The characteristics of the samples are shown in the table below.

BMI (kg/m^2)	Weight (kg)	Height (cm)	Age (years)	Number	Group
31.28±0.97	33.89±5.21	76/3±00/172	05/3±26/37	15	Real tDCS with aerobic exercise
11/1±31/31	55/4±00/93	53/3±46/172	69/2±46/35	15	Sham tDCS with aerobic exercise

One week before the start of the training protocol, the participants were explained how to perform the aerobic exercise program and dorsolateral prefrontal cortex stimulation in an orientation session. After the orientation session and to prevent adverse reactions due to electrical brain stimulation, all samples received short doses of stimulation. Therefore, none of them reported any abnormal reaction up to 48 hours after receiving the stimulation. The subjects were also told to refrain from taking any type of medication, especially blood pressure-lowering drugs, during the study. Then, the subjects were randomly assigned to two groups: 1- real stimulation of the dorsolateral prefrontal cortex with aerobic exercise, and 2- sham stimulation of the dorsolateral prefrontal cortex with aerobic exercise. In the pretest phase, the samples completed a questionnaire related to strong food cravings and attentional bias towards food. The next day, the subjects came to the laboratory 30 minutes before sampling after a 10-hour overnight fast, and after a 15-minute rest, 5 cc of blood was taken from the right arm vein of each

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subject. Then the training phase began. The training phase will last 8 weeks and 3 sessions per week. In each session, the participants went to the gym for real stimulation with aerobic exercise and then sat on a chair in a quiet room and the dorsolateral prefrontal cortex was stimulated (anode F3 and cathode F4) for 20 minutes at an intensity of 2 mA. Aerobic exercise was performed 5 minutes after the stimulation (Montenegro et al., 2012). While the participants were sitting on a chair with aerobic exercise in a quiet room, a 2 milliampere electrical current was applied to the subject after the electrodes were connected. However, after 30 seconds, the electrical current was stopped without informing the subject. Aerobic exercise was performed again 5 minutes after the stimulation (Montenegro et al., 2012). Finally, 48 hours after the last training session, the subjects completed the food craving and attentional bias questionnaires. Then, the next day, the participants were asked to be present in the laboratory 30 minutes before sampling after a 10-hour overnight fast, and after about 15 minutes of rest, 5 cc of blood was taken from the subjects. Blood samples were collected in special tubes and collected in test tubes with EDTA anticoagulant and transported to the laboratory.

Transcranial Direct Current Stimulation (tDCS)

A tDCS device (Neuratism 2, Tehran, Iran) and a 9-volt battery were used to electrically stimulate the brain. The electric current was applied by connecting electrodes with different polarities (anode and cathode) that were placed on the scalp and transmitted a constant electric current from the skull to the brain. In this study, the electrodes were placed inside 35 cm2 sponge pads and the surface of the pads was soaked with 9% sodium chloride solution to increase the conductivity of the electric current and prevent heat build-up. The device was controllable in terms of current intensity, electrode size, and stimulation duration. In the stimulation of the dorsolateral prefrontal cortex, they were placed at F3 and F4 (13). In this study, the duration of the electric current stimulation was 20 minutes and its intensity was 2 mA. In order to prevent any cognitive and motor interference on the effects of stimulation in this study, the subjects were in a resting state when tDCS was applied. They looked around during the stimulation (14). It is worth noting that all stages of this study were conducted under the supervision of a clinical psychologist and instructor licensed by the Organization of the Psychological System

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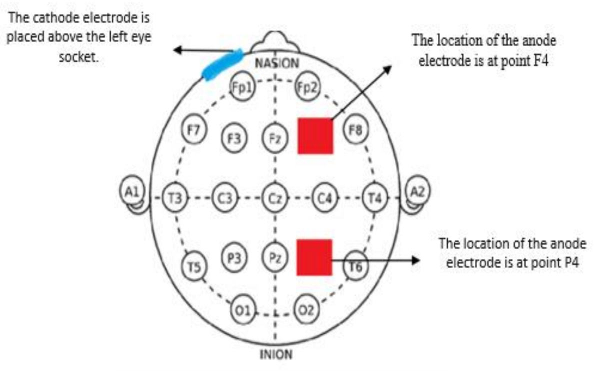
Table 2 Aerobic exercise protocol

Cool-down	Main exercises	Warm-up	week
Ten minutes of stretching exercises focusing on the muscles involved in the activity	40 %of maximum oxygen consumption and 30 minutes duration	Ten minutes of gentle stretching and warming up	First
	45 %of maximum oxygen consumption and duration 30 minutes		Second
	50 %of maximum oxygen consumption and duration of 30 minutes		Third

	55 %of maximum oxygen consumption and duration 35 minutes		Fourth
	60 %of maximum oxygen consumption and 40 minutes duration		Fifth
	65 %of maximum oxygen consumption and 40 minutes duration		Sixth
	70 %of maximum oxygen consumption and 45 minutes duration		Seventh
	70 %of maximum oxygen consumption and 45 minutes duration		Eighth

Figure 1. International 20-10 Brain Scoring System



Also, in this study, the Food Craving Questionnaire was used, which consists of 39 items on a 6-point Likert scale. It is worth noting that Kachouei and Ashrafi (2016) showed that the Persian version of this scale has good validity and reliability for measuring food cravings in the Iranian population. This questionnaire is an instrument designed to assess "food cravings" or in other words, "love for food." This craving can include various aspects, such as enjoying eating, thinking about food, or even the feelings that a person experiences after eating food. In this Sepida Benito questionnaire, if it is a standard and valid version, there is usually a scoring key. This key will indicate what score each option has. In this key, I strongly disagree = 1 point, I disagree = 2 points, I neither agree nor disagree = 3 points, I agree = 4 points, I strongly agree = 5 points. After the score for each question is determined, all the points are added together. The total sum shows the final score of the individual in this questionnaire.

Food Attention Bias Test

This tool was developed and validated by Afzali et al. (2021) (15). In total, 300 images of

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different foods and 300 neutral images were selected and graphically homogenized to create a dot-probe task for food attention bias. Previously, the content validity of this test was measured using 300 selected images of food and non-food items displayed to 50 students with optimal mental health. Finally, 200 images of emotional stimuli (food) and 200 neutral images were selected based on the scores given to each image.

To measure blood variables, a 5 ml venous blood sample was taken from each subject at 9 am. The samples were centrifuged and stored at -20°C until the variables were measured. HDL was measured after precipitation of apolipoprotein B with phosphotungstic acid using an autoanalyzer and Pars Azmoun kits. Total cholesterol was also measured by enzymatic colorimetric method with cholesterol esterase using an autoanalyzer and Pars Azmoun kits. Finally, to measure LDL values, the Friedewald equation was used according to the following equation.

LDL=TC - (HDL+TG/K) (K=5(

Triglyceride values were measured by enzymatic colorimetric method with glycerol phosphate oxidase. Fasting blood sugar (FBS) was also measured using the glucose oxidase method in the laboratory by the relevant technician. In addition, insulin resistance index was measured by measuring insulin levels using a Biosource laboratory kit and using an immunoradiometric assay and a gamma counter device.

This study was conducted with the ethics code (IR.IAU.B.REC.1403.045) and clinical trial code (IRCT20210617051606N6) approved by Boroujerd Azad University.

In order to analyze the data, descriptive statistics methods were used to calculate central and dispersion indices. The Shapiro-Wilk test was used to check the normality of the data. The Levene test was used to check the equality of variance of the variables in question. The regression slope test was used to check the collinearity of the data. In the inferential statistics section, analysis of covariance test was used for intergroup comparison and dependent t-test for intragroup effects. It should be noted that data analysis was performed using SPSS statistical software version 26 at a significance level of 0.05.

3. Results

3.1. Values of research variables

Table 5. Mean values ± standard deviation of research variables.

Post-test	Pre-test	variable
133/66±19/15	153/46±19/97	Food cravings real stimulation group with exercise
152/20±17/44	154/53±23/15	Intense food cravings in sham stimulation group with exercise
6/20±2/36	9/46±3/68	Real stimulus group bias with practice
8/86±3/11	9/86±3/64	Food bias of sham stimulation group with exercise
80/20±5/70	88/46±4/42	Glucose in the real-life exercise (stimulation group (mg/dl
87/60±4/50	93/46±4/77	Glucose in sham-stimulated (group with exercise (mg/dl
1/06±0/26	2/10±0/34	Insulin resistance group real stimulation with exercise
1/33±0/25	2/07±0/34	Insulin resistance in the sham-stimulated group with exercise
2/99	149/13±6/82	Total cholesterol in the real exercise stimulation group (mg/dl)
2/33	153/66±5/86	Cholesterol in sham-stimulated group with exercise (mg/dl)
100/40±6/33	108/06±4/33	Triglycerides in the actual

		exercise stimulation group (mg/dl)
106/00±6/63	111/00±5/23	Triglyceride sham exercise stimulation group (mg/dl)
104/86±4/15	112/13±4/24	LDL group actual stimulation with exercise (mg/dl)
113/86±5/93	116/46±5/37	LDL in sham-stimulated group with exercise (mg/dl)
63/80±7/24	50/80±4/90	HDL group actual stimulation with exercise (mg/dl)
55/93±6/40	50/00±6/91	HDL in sham-stimulated group with exercise (mg/dl)

Craving for food

According to the data in Table 4, it can be seen that real stimulation of the dorsolateral prefrontal cortex with aerobic training has a significant effect on the craving for food in obese men (P=0.014, T=2.8). Also, the craving for food in obese men showed a significant decrease in the result of real stimulation of the DLPFC with aerobic training in the pre-test from the post-test. However, sham stimulation of the DLPFC with aerobic training had no significant effect on the craving for food in obese men (P=0.672, T=/.43). Also, there was no significant difference between the groups in craving for food with an effect size of 0.22 (P=0.010, F=/.775). On the other hand, the group of real stimulation of the dorsolateral prefrontal cortex with aerobic training showed a greater decrease in craving for food compared to the group of sham stimulation of the dorsolateral prefrontal cortex with aerobic training (P<0.05).

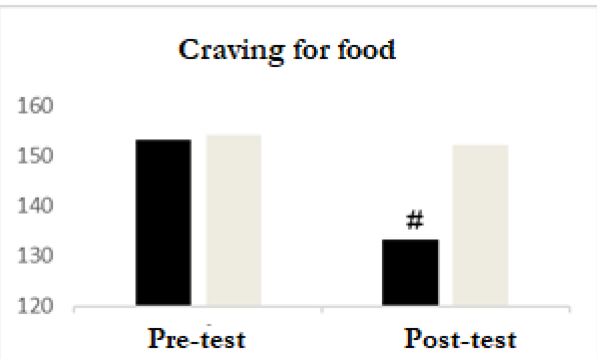

Figure 1. Dark color indicates real stimulation and light color indicates fake stimulation. Also, the # symbol indicates significant differences from pre-test to post-test.

Attentional bias to food

As can be seen in Table 5, real stimulation of the dorsolateral prefrontal cortex with aerobic training has a significant effect on the attentional bias to food in obese men (P=0.001, T=5.70). The results show that the attentional bias to food in obese men due to real stimulation with aerobic training is significantly lower in the pre-test than in the post-test. Also, sham stimulation of the dorsolateral prefrontal cortex with aerobic training has no significant effect on the attentional bias to food in obese men (P=0.495, T=0.70). On the other hand, there is no significant difference between the groups in attentional bias to food with an effect size of 0.19 (P=0.016, F=6.63). Also, the group that received real stimulation of the dorsolateral prefrontal cortex with aerobic training had a statistically lower attentional bias toward food compared to the group that received sham stimulation of the dorsolateral prefrontal cortex with aerobic training, with a mean difference of 2.61 units (P<0.05).

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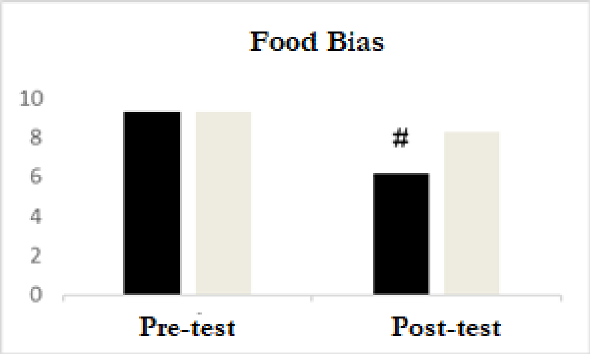

Figure 2. Dark color indicates real stimulation and light color indicates fake stimulation. Also, the # symbol indicates significant differences from pre-test to post-test.

Fasting glucose

As can be seen in Table 4-7, real stimulation of the dorsolateral prefrontal cortex with aerobic training had a significant effect on fasting glucose in obese men (P=0.001, T=6.24). Also, fasting glucose levels in obese men due to real stimulation of the dorsolateral prefrontal cortex with aerobic training in the pre-test were significantly lower than in the post-test. Simulated stimulation of the dorsolateral prefrontal cortex with aerobic training had a significant effect on fasting glucose in obese men (P=0.006, T=3.25). Fasting glucose in obese men due to sham stimulation with aerobic training in the pre-test was significantly lower than in the post-test. But there was no significant difference between the groups in fasting glucose with an effect size of 0.20 (P=0.013, F=7.10). Meanwhile, the glucose levels of the real stimulation group with aerobic exercise were significantly lower than those of the sham stimulation group (P<0.05).

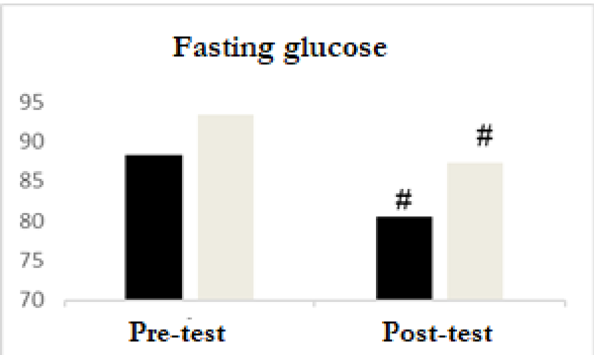

Figure 3. Dark color indicates real stimulation and light color indicates fake stimulation. Also, the # symbol indicates significant differences from pre-test to post-test.

Insulin resistance

As can be seen in Table 4-8, real stimulation of the dorsolateral prefrontal cortex with aerobic training has a significant effect on insulin resistance in obese men (P=0.001, T=0.11). Insulin resistance in obese men was significantly lower due to real DLPFC stimulation with aerobic training in the pre-test than the post-test. Also, sham stimulation of the dorsolateral prefrontal cortex with aerobic training had no significant effect on insulin resistance in obese men (P=0.001, T=6.61). However, its amount was significantly lower due to sham stimulation of the dorsolateral prefrontal cortex with aerobic training in the pre-test than the post-test. While there was no significant difference in insulin resistance between the groups with an effect size of 0.23 (P=0.010, F=7.73). On the other hand, participants who received real stimulation of the dorsolateral prefrontal cortex with aerobic exercise had significantly lower insulin resistance compared to participants in the sham stimulation group (P<0.05).

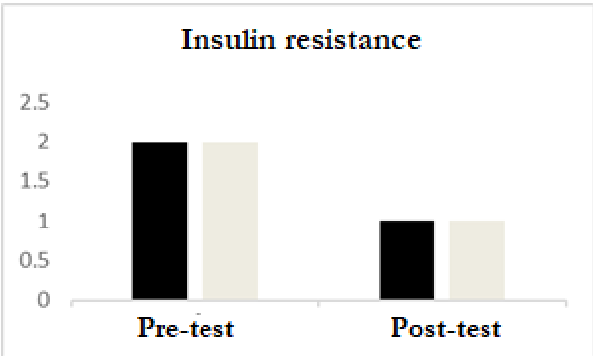

Figure 4. Dark color indicates real stimulation and light color indicates fake stimulation. Also, the # symbol indicates significant differences from pre-test to post-test.

Total cholesterol

As can be seen in Table 9, real stimulation of the dorsolateral prefrontal cortex with aerobic training has a significant effect on cholesterol in obese men (P=0.010, T=2.99). The cholesterol of obese men as a result of real stimulation of the dorsolateral prefrontal cortex with aerobic training in the pre-test was significantly higher than that of post-test. Also, sham stimulation of the dorsolateral prefrontal cortex with aerobic training had no significant effect on cholesterol in obese men (P=0.032, T=2.33). (P=0.032). So that the cholesterol of obese men as a result of sham stimulation of the dorsolateral prefrontal cortex with aerobic training in the pre-test was significantly lower than that of post-test (153.66). Also, there was no significant difference in cholesterol between the groups with an effect size of 0.11 (P=0.041, F=3.66). While real stimulation of the dorsolateral prefrontal cortex with aerobic exercise significantly reduced cholesterol compared to participants in the sham stimulation group (P<0.05).

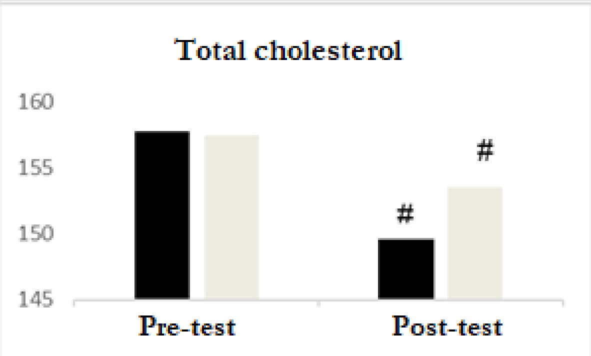

Figure 5. Dark color indicates real stimulation and light color indicates fake stimulation. Also, the # symbol indicates significant differences from pre-test to post-test.

Triglycerides

As can be seen in Table 10, real stimulation of the dorsolateral prefrontal cortex with aerobic training has a significant effect on triglycerides in obese men (P=0.005, T=3.31). Triglycerides in obese men were significantly lower in the pre-test than in the post-test (40.100) as a result of real stimulation of the dorsolateral prefrontal cortex with aerobic training. Also, sham stimulation of the dorsolateral prefrontal cortex with aerobic training had a significant effect on triglycerides in obese men (P=0.039, T=8312). However, triglycerides in obese men were significantly lower in the pre-test than in the post-test as a result of sham stimulation of the dorsolateral prefrontal cortex with aerobic training. While there was no significant difference between the groups in triglycerides with an effect size of 0.23 (P=0.008, F=8.07). So that the group that received real stimulation of the dorsolateral prefrontal cortex with aerobic training had significantly lower triglycerides compared to the participants in the sham stimulation group (P<0.05).

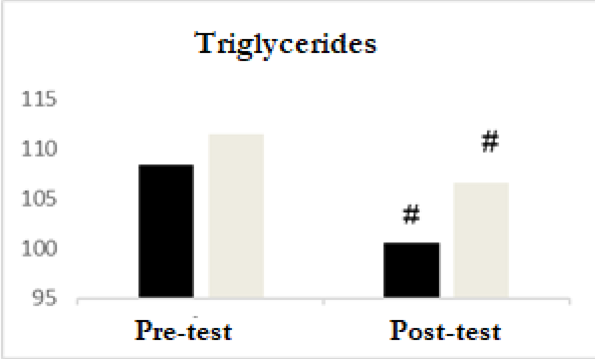

Figure 6. Dark color indicates real stimulation and light color indicates fake stimulation. Also, the # symbol indicates significant differences from pre-test to post-test.

LDL

As can be seen in Table 11, real DLPFC stimulation with aerobic training had a significant effect on LDL in obese men (P=0.001, T=4.93). The LDL level of obese men was significantly lower due to real DLPFC stimulation with aerobic training in the pre-test than the post-test. Also, sham DLPFC stimulation with aerobic training had a significant effect on LDL in obese men (P=0.019, T=3.02). Also, LDL in obese men was significantly reduced due to sham DLPFC stimulation with aerobic training in the pre-test (116.46) than the post-test (113.86). While there was no significant difference between groups in LDL with an effect size of 0.50 (P=0.001, F=26.96). However, the real dorsolateral prefrontal cortex stimulation group with aerobic training was significantly lower compared to the sham dorsolateral prefrontal cortex stimulation group (P<0.05).

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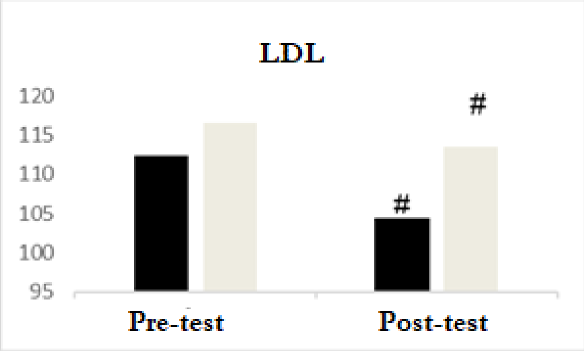

Figure 7. Dark color indicates real stimulation and light color indicates fake stimulation. Also, the # symbol indicates significant differences from pre-test to post-test.

HDL

As can be seen in Table 12, real DLPFC stimulation with aerobic training has a significant effect on HDL in obese men (P=0.001, T=4.89). So that the HDL level of obese men due to real stimulation of the dorsolateral prefrontal cortex with aerobic training in the pre-test (50/80) is significantly lower than the post-test. Also, sham stimulation of the dorsolateral prefrontal cortex with aerobic training had a significant effect on HDL in obese men (P=0.048), so that HDL in obese men increased significantly from pretest to posttest (55.93) as a result of sham stimulation of the dorsolateral prefrontal cortex with aerobic training. There was also no significant difference between the groups in HDL with an effect size of 0.29 (P=0.002, F=11.45). However, the group with real stimulation of the dorsolateral prefrontal cortex with aerobic training had significantly higher HDL compared to the participants in the sham stimulation of the dorsolateral prefrontal cortex group (P<0.05).

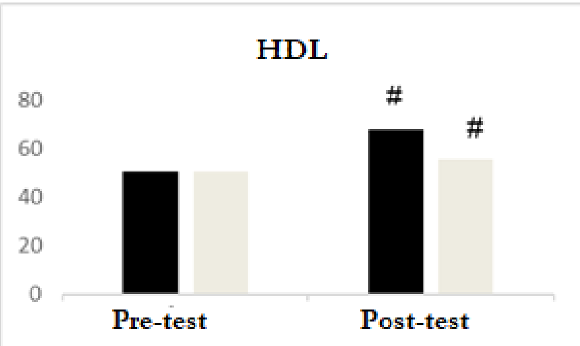

Figure 8. Dark color indicates real stimulation and light color indicates fake stimulation. Also, the # symbol indicates significant differences from pre-test to post-test.

Discussion

The present study aimed to investigate the effect of direct transcranial stimulation of the dorsolateral prefrontal cortex with aerobic exercise on food craving, attentional bias to food, and cardiometabolic indices in obese men. In general, the results of the present study showed that real stimulation of the dorsolateral prefrontal cortex has a significant effect on reducing food craving and attentional bias to food in obese men. However, sham stimulation of the dorsolateral prefrontal cortex has no significant effect on reducing attentional bias to food in obese men. Aerobic exercise also had a significant effect on reducing fasting glucose, insulin resistance, total cholesterol, triglycerides, and LDL, and increasing HDL in obese men. Similarly, real stimulation of the dorsolateral prefrontal cortex with aerobic exercise caused a greater reduction in fasting glucose, insulin resistance, total cholesterol, triglycerides, and LDL, and a greater increase in HDL in obese men, compared to sham stimulation of the dorsolateral prefrontal cortex with aerobic exercise. These results can be discussed in three separate sections. First, real stimulation of the DLPFC significantly reduced food craving and attentional bias in obese men compared to sham stimulation. In line with this finding, Tabasi et al. (2024) reported in a study that transcranial direct current stimulation (tDCS) has a significant effect on food craving in patients with depression and overweight (16). Also, Chimel et al. (2024) showed in another study that tDCS is effective in reducing food craving, binge eating, binge eating frequency, and food consumption in people with binge eating (17). In addition, Arrow et al. (2022) in another study examined the effects of transcranial direct current stimulation on food craving and food consumption in people with obesity and overweight. The results showed that targeting the dorsolateral prefrontal cortex with unipolar montage produced ameliorating effects on food craving (18). However, the results of some studies were inconsistent with the present study, for example, Beaumont et al. (2023) showed that tDCS had no significant effect on food craving, food reward, and subjective appetite in women with mild to moderate binge eating behavior. The reasons for the inconsistency of this study with the present study can be mentioned in this type of stimulation (single session) and the subjects. Because it has been shown that repeated sessions of low-intensity direct current will produce a cumulative effect on cortical excitability, which is not observed in single-session stimulation (19, 20). In confirmation of this, the cumulative beneficial effects of direct current are commonly used to treat neuropsychiatric conditions such as depression and eating disorders (20-22). It has also been well demonstrated in the past that multiple sessions of direct current stimulation produce cumulative and incremental increases in motor cortex excitability (23). And even when direct current is applied to the left dorsolateral prefrontal cortex for only two sessions per day, cognitive skills have been shown to increase (24). One explanation for the improvement in skill acquisition due to repetition is that repeated practice of a given task makes the task easier through a practice effect mechanism (Donovan and Radesevich, 1999). Interestingly, direct current increases cortical excitability when applied to the relevant cortical network. Thus, it seems that both the practice effect and the increased cortical excitability are involved in the enhanced cognitive effects of multiple sessions of tDCS (22). On the other hand, a process called attention bias causes obese individuals to selectively focus on food stimuli and ignore other stimuli. This selective attention ultimately directs the individual's responses to the relevant stimuli, in this case food (16). In this regard, it has been shown that obese individuals were less sensitive to reward and had changes in the activity of different brain regions (striatum and insular cortex, prefrontal cortex and impulse control networks) related to impulsivity and compulsive behavior (2). This leads to an attentional bias towards food. In contrast, the term impulsivity reflects decisions that occur with limited expectations and in such decisions there is a tendency to act hastily, indicating a motivation related to reward. In contrast, compulsion is characterized by repetitive and persistent actions that are not related to an overall goal or reward and can be continued despite adverse consequences (25). Therefore, the factors that cause food cravings are related to the biological neural systems and pathways in the brain. In this regard, it has been shown that direct electrical stimulation of the skull reduces food cravings. Also, neuroimaging in relation to obesity has shown that an imbalance in electrical activity in the prefrontal cortex and limbic system circuits is the main driving force for overeating behaviors (26). Therefore, changes and stimuli caused by tDCS can change the function of the reward system and play a role in attention control in these patients, which ultimately leads to a decrease in appetite and weight gain. In this regard, it has been shown that the three main mechanisms involved in controlling food cravings are: 1) a system consisting of the brain stem, hypothalamus and autonomic nerves that interact with digestive hormones to create a sense of hunger or satiety. 2) a motivational system that works with elements involved in memory and learning to create a desire to eat food 3) a self-regulatory system that regulates appetite based on the individual's life circumstances (27). It is therefore clear that the dorsolateral prefrontal cortex is involved in eating disorders and that its decreased activity can lead to eating disorders (28). In addition, stimulation of glutamatergic neurons is another mechanism of the reward pathway, as stimulation of these neurons is associated with modulation of dopaminergic sensitivity, which ultimately reduces the patient's desire for certain foods (29). Apart from changes in food craving, stimulation of the limbic system can play a role in controlling the symptoms of these patients by increasing cognitive control and reducing appetite (24, 29). Therefore, it is possible that stimulation of these areas by tDCS improves their neural function because tDCS optimizes the function and structure of these neurons. Although the exact anatomical correlates of impulsivity remain unknown, several brain regions are thought to be involved in this process, including the striatum, temporal pole, insula, anterior cingulate cortex, prefrontal cortex, and hippocampus (30). Given the widespread distribution of dopamine receptors in various brain regions and neural circuits, which play an important role in food craving, decision-making, executive function, and impulsivity, this

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neurotransmitter has received much attention in research on overeating (31). In support of the correlation of eating motivation with the mesolimbic dopaminergic system, it has been shown that the mesolimbic circuits formed by dopamine neurons in the ventral tegmental area send messages to the ventral striatum, the primary brain region associated with food cravings, and the initiation of food consumption is facilitated by an overactive mesolimbic dopaminergic system, which ultimately increases the background of eating habits, the salience of motivation, or the desire for food-related rewards (32). In support of this, previous research results have shown that dopamine levels and activity are reduced in obese individuals (30). This concept is also related to reward-related models or theories that relate to drug addiction (dopamine desensitization theory), obesity (dynamic vulnerability model), and alcohol use disorder (three-stage model). The reward and reward deficiency hypotheses are also two competing dopamine-related ideas discussed in the obesity literature. The former suggests that eating high-energy foods increases reward responsiveness or dopamine signaling, which in turn increases the risk of obesity. The latter suggests that the reverse process occurs (30). In this regard, tDCS application to frontal regions has been shown to induce dopamine release in the striatum, which confirms the effects of tDCS on the dopaminergic and reward systems. Since eating disorders are closely related to substance use disorders, and since both conditions share a common neurobiological basis related to disruption of dopaminergic pathways, tDCS may offer a potential avenue for intervention. Given the dopamine desensitization theory of drug addiction (Bridg et al., 2016), tDCS could be explored as a tool to modulate dopamine release at different stages of BED (33). By targeting the striatum, which is associated with reward processing, tDCS may affect dopamine release and help regulate reward-related mechanisms involved in overeating and substance use. It may therefore potentially counteract the reduced dopamine release and diminished responses associated with chronic overeating. . Similarly, in line with the three-stage model of alcohol dependence, tDCS may be used to target the ventral striatum during the binge/intoxication phase. This could potentially reduce the positive reinforcement processes that contribute to the initial transition to addiction. Furthermore, tDCS may be investigated in addressing compromised dopamine functions during the withdrawal/negative affect phase, potentially reducing sensitivity to rewards and tolerance. The prefrontal cortex is a key regulator of inhibitory control over behavior. In support of these processes, impairments and lower baseline activity in PFC regions have been identified in obese individuals. Furthermore, there are deficits in inhibitory control, particularly in the medial prefrontal cortex, and we see increased activation in response to food cues (34). On the other hand, there are two competing conceptualization systems involved in the impulsive process: a "STOP" system that is impaired in compulsive eating and a "GO" system that is sensitized in compulsive eating (21). tDCS modulates brain activity in the "GO" system. It is possible that this may regulate or reduce the overreactivity to craving stimuli by focusing on areas with hyperreactivity associated with food cues. In the case of compulsive eating, this stimulation method seems to improve inhibitory control and reduce impulsivity by creating a better balance between the "GO" and "STOP" systems. The second part of the findings of the present study indicates that in both groups that underwent aerobic training, training improved fasting glucose, insulin resistance, and lipid profiles in obese men. This finding emphasizes the importance of physical activity, especially aerobic training, in improving cardiometabolic indices. In explaining this finding, it can be said that the possible mechanisms in which insulin resistance and fasting glucose are improved by aerobic exercise are extensive, which could be due to changes in the content and activity of glycolytic and aerobic enzymes, an increase in skeletal muscle GLUT4, and similarly, an increase in the activity of β-hydroxyl coenzyme A dehydrogenase (a key catalyst for lipid oxidation) (35, 36). Another possible mechanism could be a change in the direction of insulin and insulin receptor signaling. Previous studies have shown that insulin signaling at the insulin receptor level is improved by exercise. For example, it has been shown that endurance exercise can increase the expression of insulin receptors on the surface of cell membranes, which increases glucose uptake and utilization by muscles. Regarding the improvement in lipid profile due to aerobic exercise in the present study, it probably indicates that exercise interventions, especially aerobic exercise, can also increase lipolysis and reduce fatty acids in the blood and muscles of the subjects. Possible causes of the improvement in HDL levels include increased production by the liver following changes in the activity of the lipoprotein lipase enzyme and a decrease in hepatic lipase following physical activity (37, 38), and a decrease in LDL levels occurs as a result of weight and body fat reduction, in which the lecithin cholesterol acyltransferase enzyme plays a key role in this process (39). Aerobic exercise also likely increases mitochondrial biogenesis and related oxidative enzymes, which may account for the increased fat consumption and improved lipid profile. Increased lipid levels in obesity may increase insulin resistance, leading to increased blood glucose levels, while exercise improves body fat status by reducing hepatic glucose production, increasing pancreatic insulin secretion, and reducing insulin resistance (40) (Coff et al., 2003). However, the biological mechanisms that may contribute to the improved lipid profile associated with exercise are complex. However, in this regard, it seems that the increase in the enzymes lipoprotein lipase (LPL) and lysine cholesterol acyltransferase (L-CAT)), the decrease in cholesteryl ester transfer protein (CETP) and hepatic triglyceride lipase play an important role in changing the concentration of lipid factors and increase the ability of the muscle to oxidize fatty acids and reduce triglycerides (41, 42). On the other hand, it has been shown that during and after exercise, insulin levels decrease, and it is likely that one of the factors that changes cholesterol is the level of circulating insulin (43, 44). However, the third and most important result of the present study was that the combination of aerobic exercise intervention with tDCS stimulation caused further improvement in attentional bias for food, food craving, and cardiometabolic indices in obese men. In line with this finding, Montenegro et al. (2012) investigated whether transcranial direct current stimulation of the prefrontal cortex associated with aerobic exercise alters appetite sensitivity in overweight adults (6). Findings in overweight individuals suggest that the combination of tDCS in the dorsolateral prefrontal cortex and aerobic exercise produces greater reductions in appetite sensitivity compared with either tDCS or exercise alone. In this regard, there is increasing evidence that the combination of physical and neural activities may have synergistic effects (45, 46). Although physical exercise contributes to plasticity, neural activities also lead to changes in plasticity (46, 47). This combined benefit of physical and neural interventions suggests the combined effect hypothesis, which states that combining physical intervention with neural interventions may provide additional benefits. Therefore, the participants in the present study may have benefited from the synergistic effects of physical and neural training by combining the aerobic exercise intervention with tDCS stimulation and were able to further improve attentional bias towards food, food craving, and cardiometabolic indices. It is hypothesized that the therapeutic potential of tDCS intervention for obesity is based on the modulation of dysregulated top-down control by prefrontal cortex (PFC) function and reward processing in subcortical-limbic structures, mainly the ventral striatum. In recent years, data obtained from neuroimaging and cognitive assessments have provided evidence that food reactivity, especially in obese individuals, is associated with dysregulated activity in prefrontal areas, in particular, the dorsolateral prefrontal cortex. The neuromodulatory potential of the dorsolateral prefrontal cortex may modulate these dysregulated activities in frontal brain regions that are associated with both impaired inhibitory control (e.g., binge eating and purging) and poor cognitive flexibility (e.g., obsessive concerns about eating, weight, and shape) (48). Accordingly, therapeutic strategies that modulate brain activity in the dorsolateral prefrontal cortex may regulate or suppress food cue reactivity, cravings, and appetite. In this regard, the effect of modulating cortical activity using high-frequency rTMS over the left dorsolateral prefrontal cortex in eating disorders and obesity has been tested in many studies, and the temporary anti-craving effect of this modulation

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has been demonstrated (49). Another clinical study also reported the beneficial effects of high-frequency rTMS applied to the left DLPFC to modify cue-induced food cravings and binge eating in individuals with bulimic eating disorder (49). This finding was also replicated using bilateral tDCS to the dorsolateral prefrontal cortex (anode over F4/cathode over F3), showing a reduction in self-reported craving for active stimulation compared with sham stimulation (50, 51) (Fergni et al., 2008; Kecik et al., 2014). As discussed earlier, previous studies have shown that prefrontal tDCS modulates food craving (50, 51), providing evidence that food craving is associated with dorsolateral prefrontal cortex activity. For example, Ward and colleagues reported that the combined application of chronic tDCS and aerobic exercise significantly improved several cognitive functions in healthy young adults. Furthermore, combining tDCS with aerobic exercise has been shown to be more effective than either intervention alone in several domains, including reducing pain perception, reducing acute appetite, enhancing executive function, delaying cognitive decline and dementia, and improving information processing speed and executive control (18). The neural mechanisms by which dorsolateral prefrontal cortex stimulation reduces food craving are unknown, although data suggest that the level of activity in the right prefrontal cortex may determine the level of inhibition in downstream circuits that promote overeating (52).

Conclusion

The results of this study showed that combined tDCS and aerobic exercise interventions can be significantly effective in reducing food craving and shifting attentional bias from food stimuli to non-food stimuli in obese men. In addition, this approach also yielded positive results in improving cardiometabolic indices, including blood pressure, glucose levels, blood lipids, insulin levels, and insulin resistance. These findings suggest that combining tDCS as a neuromodulatory method with aerobic exercise can be considered as a novel and complementary therapeutic strategy in the management of obesity and its related complications, especially in the field of controlling eating behaviors and improving metabolic health. However, this study emphasizes the need for further research with larger samples and longer follow-up periods to confirm and generalize these results, as well as to investigate the precise mechanisms of these effects.

Article Message:

The present study suggests that the combination of direct transcranial stimulation of the dorsolateral prefrontal cortex and aerobic exercise appears to be an effective and promising approach to reduce food cravings and significantly improve cardiometabolic indices in obese men. Therefore, our findings suggest that by simultaneously targeting the neural mechanisms associated with appetite control and utilizing the physiological benefits of exercise training, better results can be achieved in managing obesity and reducing the risk of related diseases. Therefore, the present study paves the way for the development of new and more comprehensive therapeutic interventions in the field of public health.

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Conflicts of Interest

The authors of this article declare that there is no conflict of interest in connection with this study, its writing, and its publication. Therefore, all authors assume full responsibility for the data and analyses presented in this article.

Authors' Contributions

All authors have contributed to the initial writing or revision of the article, and all accept responsibility for the accuracy and completeness of the material contained therein with the final approval of the present article.

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