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RESEARCH ARTICLE

Comparison of the acute metabolic and cardiovascular effects of electrical stimulation and voluntary exercise

Divya Goyal[1], Aksh Chahal[2], Aashi Bhatnagar[1], Vishakha[1], Sheetal Malhan[1], Vishwajeet Trivedi[1*]

Abstract

Despite being marketed as a fat reduction tool, the effects of electrical stimulation on blood lipid profiles remain underexplored. This study aimed to compare the acute effects of electrical stimulation and voluntary exercise on heart rate, respiratory rate, blood pressure, and blood lipid parameters, including cholesterol, triglycerides, and HDL levels. Ten healthy males (mean age: 24.6 ± 1.35 years) participated in a randomized crossover study. Each underwent 20 minutes of either electrical stimulation (faradic current at 50 Hz, 3 seconds stimulation, 6 seconds rest, applied bilaterally to quadriceps) or cycling (60–70% of maximal heart rate), with a 7-day washout period between sessions. Pre and post-intervention measures included heart rate, respiratory rate, blood pressure, cholesterol, triglycerides, and HDL levels. Electrical stimulation significantly altered post-treatment values for cholesterol (183.6 ± 10.94 *vs.* 185.5 ± 9.70 mg/dL), triglycerides (127.62 ± 29.52 vs. 128.5 ± 29.07 mg/dL), HDL (40.30 ± 4.69 *vs.* 39.17 ± 5.23 mg/dL), and heart rate (65.60 ± 7.79 *vs.* 61.40 ± 8.67 beats/min) compared to pre-treatment values ($p < 0.05$). Similar changes were observed in the cycling group. These findings suggest that electrical stimulation has the potential to influence blood lipid profiles, comparable to moderate-intensity cycling, highlighting its potential as an alternative intervention and warranting further research.

Keywords: Electrical stimulation, Bicycle ergometry, Blood lipid profile, HDL, Triglycerides, Heart rate, Exercise.

Introduction

The inability to control body weight or over fat is fraught with serious dangers and exposes the over-fat individual to a good deal of discomfort and difficulty. Weight control or fat reduction is not a simple task. It is a challenge that many people are unable to cope with. With the increasing popularity of exercise, the Indian public is confronted with an increasing number of exercise devices for which the manufacturing companies make remarkable claims. Many of these devices are based on the technique of electrical muscle stimulation, which has been practiced for a long time in electrotherapy. However, the results of these electronic gadgets and the mechanism by which they may bring about fat reduction are rarely investigated. Healthy skeletal muscle undergoes numerous histochemical, physiological and morphologic changes when subjected to prolonged periods of increased activity. Increased muscular activity may result from either increased exercise or chronic electrical stimulation. The changes that occur in healthy muscles in response to increased exercise are the same as those that occur during prolonged electrical stimulation. Little is known about the contribution of plasma-free fatty acids and intramuscular triacylglycerol as substrates for energy production during prolonged electrical stimulation of skeletal muscle. One study that examined electrical stimulation and fatty acid metabolism concluded that at least a portion of intracellular triacylglycerol in skeletal muscle is constantly being synthesized and hydrolyzed during electrical stimulation (Hopp and Palmer, 1990). It is important to investigate whether the electrical muscular stimulation has the potential to bring about fat reduction.

[1]Department of Physiotherapy, SoHAS, G D Goenka University, Gurugram, Haryana, India.

[2]Department of Physiotherapy, Galgotias University, Noida, Uttar Pradesh, India.

***Corresponding Author:** Vishwajeet Trivedi, Department of Physiotherapy, SoHAS, G D Goenka University, Gurugram, Haryana, India, E-Mail: Physiovishu87@gmail.com

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This study aimed to compare the acute metabolic and cardiovascular effects of electrical stimulation and voluntary exercise.

Materials and Methods

This study employed a repeated measures experimental design involving the same subjects. Ten healthy adults were recruited for the study, with an average age of 24.6 ± 1.35 years (range: 20–29 years) and a mean VO_2 max of 50.35 ± 10.05 mL/kg/min. These participants were selected from an initial cohort of 45 participants. Subjects were included in the study based on their adherence to predetermined inclusion and exclusion criteria and volunteered for the research.

Exclusion criteria were carefully defined to ensure homogeneity and reliability of the results. Subjects were excluded if they had any musculoskeletal injuries at the time of the experiment, were on medication, or had cardiorespiratory impairments. These criteria ensured that only healthy individuals capable of safely participating in the interventions were included in the study. The study received approval from the DRB, Malout Institute of Physiotherapy, Punjab.

Pre-Experiment Procedures

Subjects attended a pre-participation screening three days before the experiment. During the screening, they completed a screening questionnaire and provided written informed consent for voluntary participation in the study. On the same day, the maximal oxygen uptake (VO_2 max) of each subject was measured using the Queen's College Step Test (Shamsi *et al*., 2011). Subjects were instructed to abstain from alcohol or any other substances that could cause physiological alterations for three days prior to the experiment. Additionally, they were required to fast overnight for 8 to 9 hours before the experiment and were allowed to eat breakfast only after its completion.

Stimulating Electrode Setup

Before the application of stimulating electrodes, the skin was debrided and cleaned with water. All participants were seated while the investigator marked the positions for the electrodes. The exact electrode positions were marked with a pen, so that the investigator replicates the position of the electrode on every participant. Before placement, a layer of conductive gel was applied over the stimulating electrode surface, and after being placed on the marked points, they were applied to the leg by the same investigator and secured with an elastic bandage (Figure 1). The cathode of the stimulating electrodes was always positioned distally, whereas the anode was positioned proximally.

Participant Grouping and Counterbalancing

Subjects were randomly divided into two equal groups. One group performed exercise on a cycle ergometer first, followed by electrical stimulation after a one-week gap. The other group underwent electrical muscle stimulation (EMS) first, followed by cycle ergometry. This counterbalancing was implemented to eliminate any order effects.

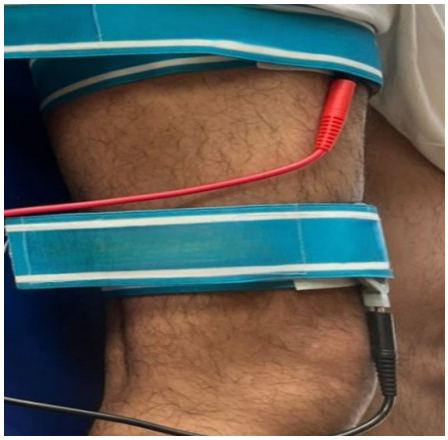

Figure 1: Placement of EMS electrodes

Baseline Measurements

Before the experiment, subjects rested for 15 minutes in a supine position. At the end of the rest period, baseline physiological variables, including heart rate, respiratory rate, and blood pressure, were measured. While in a sitting position, a blood sample was collected from the antecubital vein at the cubital fossa using a 21-gauge needle and a 10 mL syringe for biochemical analysis using a standard laboratory protocol (WHO, 2010).

Cycle ergometry protocol

Cycle ergometer of King company, model no. R-300 was used to perform exercise. Subjects performing cycle ergometry were equipped with a POLAR short-range telemetry strap on their chest and a monitoring watch on their wrist. Exercise intensity was determined using Karvonen's formula, targeting 60 to 70% of the maximal heart rate (Froelicher & Myer, 2000). Participants were instructed to gradually increase their exercise intensity until reaching the target heart rate and to maintain it for 20 minutes (Figure 2). After completing the exercise, physiological parameters were re-measured, and a blood sample was taken 5 minutes post-exercise for biochemical analysis. These values were recorded as post-cycling values.

Electrical stimulation protocol

Subjects undergoing electrical stimulation lay supine on a plinth during the procedure. The simulator used was the ET-3000 (Z.M.I. Electronics Ltd, Taiwan) (Figure 3), delivering moderate Faradic current with a frequency of 50 Hz and duty cycles of 3 seconds on and 6 seconds off. This protocol aligns with established guidelines for human studies (Allen & Goodman, 2014) and has been shown to produce forces up to 60% of maximal voluntary isometric contraction in

Figure 2: Cycle ergometer

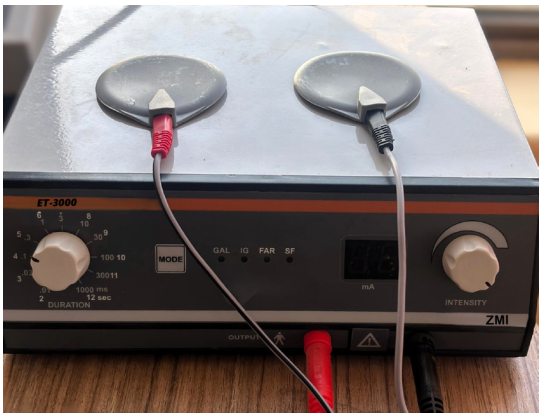

Figure 3: Modality used to stimulate the muscle

quadriceps (Nussbaum *et al*., 2017). The stimulation intensity was adjusted to the tolerable limits of each subject. Surface rubber electrodes were placed bilaterally on the femoral triangle and the distal portion of the quadriceps femoris, secured with elastic band to ensure good contact.

Data Recording

For both interventions, physiological and biochemical data were recorded at baseline, immediately post-intervention, and at specified intervals as per the protocol. The experiment was conducted under standardized conditions to ensure the reliability and validity of the data.

Results

Table 1 shows that the post-treatment heart rate of electrical stimulation (65.60 ± 7.79) bpm was significantly (p <0.05) different from the pre-treatment heart rate score (61.40 ± 8.67) bpm. The post-treatment heart rate of cycling (111.60 ± 9.47) bpm was highly significantly (p <0.001) bpm different from the pre-score (61.60 ± 7.56) bpm. The intergroup comparison showed that the pre-treatment scores of both groups were not different. The post-treatment scores of cycling (111.60 ± 9.47) were highly significantly (p <0.001) different from the post-heart rated scores of electrical stimulations (65.60 ± 7.79).

Table 1: Comparison of pre and post-treatment heart rate (beats/min) in electrical stimulation and cycling groups

Group (n = 10)	*Pre-treatment*		*Post-treatment*			
	Mean	*SD*	*Mean*	*SD*	*t*	*P*
Electrical stimulation	61.40	8.67	65.60	7.79	2.43*	
Cycling	61.60	7.56	111.60	9.47	12.72***	
	t = 0.11		t = 10.98***			

* = $t_{(9,0.05)}$ 2.26 **= $t_{(9,0.01)}$ 3.26; ***= $t_{(9,0.001)}$ 4.78

Table 2 shows that the post-treatment blood cholesterol of electrical stimulation (183.6 ± 10.94) mg/dl was significantly (p <0.05) different from the pre-treatment blood cholesterol level score (185.5 ± 9.70) mg/dl. The post-treatment blood cholesterol level scores of cycling (185.23 ± 11.99) mg/dl were highly significantly different from the pre-treatment cholesterol score of cycling (186.8 ± 12.15) mg/dl. No significant differences were observed between the groups in pre-treatment and post-treatment cholesterol levels.

Table 3 shows that the post-treatment triglyceride level of electrical stimulation (127.62 ± 29.52) mg/dl was highly significantly (p <0.01) different from the pre-treatment triglyceride scores of electrical stimulations (128.5 ± 29.07) mg/dl. In contrast, no such difference was observed in the cycling group. No significant differences were observed between the groups in pre-treatment and post-treatment triglyceride levels.

Table 4 shows that the post-treatment HDL level of electrical stimulation (40.30 ± 4.69) mg/dl was highly significantly (p <0.05) different from the pre-treatment HDL scores of electrical stimulations (39.17 ± 5.23) mg/dl. The post-treatment HDL scores of cycling (38.5 ± 6.06) mg/dl

Table 2: Comparison of pre and post-treatment blood cholesterol level (mg/dl) in electrical stimulation and cycling groups

Group	*Pre-treatment*			*Post-treatment*				
	Mean	*SD*	*SE*	*Mean*	*SD*	*SE*	*t*	*P*
Electrical stimulation	185.50	9.70	3.07	183.60	10.94	3.46	2.91	*
Cycling	186.80	12.15	3.84	185.23	11.99	3.79	4.21	**
	T = 0.29			t = 0.34^{NS}				

* = $t_{(9,0.05)}$ 2.26 **= $t_{(9,0.01)}$ 3.26; ***= $t_{(9,0.001)}$ 4.78

Table 3: Comparison of pre and post-treatment triglyceride (mg/dl) in electrical stimulation and cycling groups

Group	*Pre-treatment*			*Post-treatment*				
	Mean	*SD*	*SE*	*Mean*	*SD*	*SE*	*t*	*P*
Electrical stimulation	128.50	29.07	9.19	127.62	29.52	9.34	3.42**	
Cycling	125.40	23.42	7.41	123.15	21.199	6.96	2.024	
	T = 0.26			t = 0.38				

* = $t_{(9,0.05)}$ 2.26 **= $t_{(9,0.01)}$ 3.26; ***= $t_{(9,0.001)}$ 4.78

Table 4: Comparison of pre and post-treatment HDL (mg/dl) in electrical stimulation and cycling groups

Group	*Pre-treatment*			*Post-treatment*				
	Mean	*SD*	*SE*	*Mean*	*SD*	*SE*	*t*	*P*
Electrical stimulation	39.17	5.23	1.65	40.30	4.69	1.48	3.45	**
Cycling	37.57	6.15	1.94	38.50	6.06	1.92	3.63	**
	t=0.54			t = 0.66				

* = $t_{(9,0.05)}$ 2.26 **= $t_{(9,0.01)}$ 3.26; ***= $t_{(9,0.001)}$ 4.78

were highly significantly ($p < 0.05$) different from the pre-treatment HDL scores of cycling (37.57 ± 6.15) mg/dl. No significant differences were observed between the groups in pre-treatment and post-treatment HDL levels.

Figure 4 illustrates the difference in the mean HDL levels between the pretest and posttest for both treatment groups.

Pre-treatment

The electrical stimulation group had an average HDL level of 39.17 mg/dl, which was higher than the cycling group (37.57 mg/dl).

Post-treatment

The HDL level in the electrical stimulation group increased to 40.3 mg/dl, showing a significant improvement. The cycling group also showed an increase in HDL levels, rising to 38.5 mg/dl, though the improvement was less pronounced compared to electrical stimulation. Both interventions positively impacted HDL levels, which are a marker of cardiovascular health. However, electrical stimulation appears to be slightly more effective in enhancing HDL levels than cycling in this present study. This suggests that electrical stimulation could be a more impactful intervention for improving HDL, potentially offering better cardiovascular benefits.

Discussion

In the present study significant post-treatment increase in heart rate in both the groups was found, but the extent of increase in heart rate was more in the cycling (81%; $p < 0.001$) than in electrical stimulation (6%; $p < 0.05$). This signifies that cycling induces more demand on the cardiac function than electrical stimulation. The increased workload is because of a large number of muscle group participants (Pinfildi *et al*., 2018) in cycling compared with electrical stimulation, where

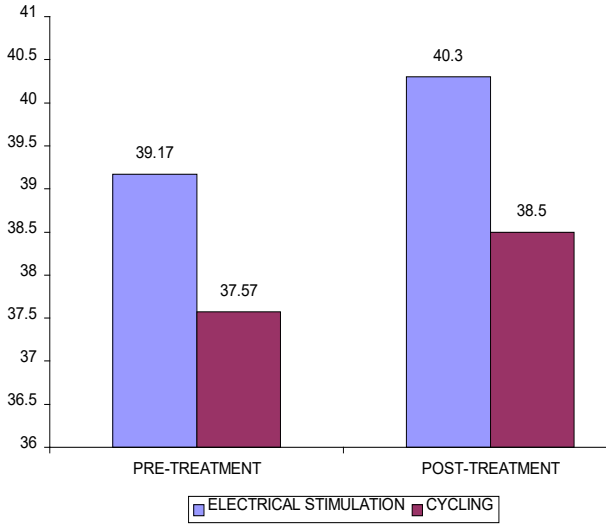

Figure 4: Difference in the mean HDL levels

only the quadriceps were stimulated. Moreover, in cycling, the contraction portion of the contraction relaxation cycle is quite prolonged and the peak loads are approximately twice the load setting (Joyner & Casey, 2015), whereas during electrical stimulation, the isometric contraction lasted for only 3 sec in a cycle of 3:9 sec on-off ratio. The finding on electrical stimulation is consistent with the findings of Taylor *et al*., (2018), who observed that electrical stimulation of lower limb muscles produced little physiological strain compared to concurrent voluntary exercise. The increase in H.R. in response to E.S. can be explained physiologically as a probable volume loading of the heart brought about by an increased venous return. Such an increase in effective circulating blood volume is likely to be due to a diminished venous reservoir consequent on a 'milking' of the deep peripheral thigh veins during rhythmic electrical stimulation-

induced muscle contraction. The H.R. response to E.S. could be a function of the forceful contractions eliciting a more powerful muscle reflex, which has been the modular of the cardiovascular response to exercise (Kang & Hyong, 2014). Another reason for the significantly high heart rate response in cycling can be cited in the isometric work of the arm muscle to hold the handle of the static cycle during cycling. This increases the resistance for the blood flow, which causes considerable ventricular afterload, leading to increased myocardial contractility (Muir *et al*., 2020). The present study showed no significant changes in blood triglyceride levels following exercise. Exercise acutely reduces triglycerides. This effect was first noted in 1982 when Cullinane *et al*., described acute triglyceride reduction following exercise in hypertriglyceridemic men. Grzebisz- Zatońska *et al*., (2021) reported reductions in triglyceride in cross-country skiers after 8 to 9 hours of exertion. This exercise effect appears to increase with energy expenditure and does not require a threshold of exertion (Drenowatz *et al*., 2015). The fuel reserves in a typical young adult male come from two main sources: (1) from triglyceride in fat cells, adipocytes (major) and (2) from intramuscular triglyceride. Triglycerides are stored directly within the muscle fiber in close proximity to mitochondria more in slow-twitch than in fast-twitch muscle fibers (Watt & Cheng (2017). During low-intensity exercise most of the fatty acids oxidized are derived from plasma fatty acid. With increasing exercise intensity, the relative contribution also increases and can represent only half of all fat oxidized. The present study used cycling for 20 minutes at 60 to 70% of VO_2 max, which can be considered a moderate-intensity exercise. Cycling is a form of generalized exercise, which creates lots of physiological demands. Adipose tissue release of free fatty acids and their subsequent use in light and moderate exercise increase directly with blood flow through adipose tissue and active muscle (Mika *et al*., 2019). Free fatty acids catabolism increases principally in slow twitch muscle fibers whose ample blood supply and large numerous mitochondria make them ideal for fat breakdown. During low-moderate intensity exercise, circulating triglycerides carried in lipoprotein complexes also provide an energy source. Harrison *et al*., (2012) reported an increase in skeletal muscle lipoprotein lipase activity following exercise. This alteration in lipoprotein lipase activity may facilitate energy utilization during or after exercise and mediate a decrease in blood triglyceride levels. Lipoprotein lipase facilitates a cell's uptake of fatty acids for use either as energy or for resynthesis (re-esterification) of triglycerides stored in muscle and adipose tissues (Kumari *et al*., 2021). It is reported that exercise training-induced increases in the activity level of skeletal muscle and adipose tissue lipases including biochemical and vascular adaptations in the muscles themselves, contribute to enhance fat use for energy during moderate exercise.

In this study, a decrease in blood triglyceride levels following electrical stimulation was found. Electrical stimulation of short-term duration, as well as that of long-term duration, influences the biochemical and structural components of the muscle. Vromans & Faghri (2017) reported that during prolonged exercise, energy for muscle contraction is derived from both plasma-free fatty acids and intramuscular triglycerides. In contrast, a number of investigators have concluded that during prolonged in situ electrical stimulation, energy for muscle contractions is derived entirely from plasma-free fatty acid (Hargreaves & Spriet, 2020). This conclusion was based on the finding that skeletal muscle triglyceride did not decrease after electrical stimulation. In the *in-situ* electrical stimulation preparation (Hioki *et al*., 2021), triglyceride was hydrolyzed, but a decrease in triglyceride content occurred because the rate of synthesis from plasma-free fatty acid equals the rate of hydrolysis. No established information is available on the intramuscular triglyceride synthesis occurring during *in- situ* electrical stimulation. Menalla *et al*. (2024) reported that *in-vitro* continuous and intermittent electrical stimulation caused a decrease in intramuscular triglyceride content. They concluded that intramuscular triglyceride is a substrate for energy production during continuous and intermittent electrical stimulation. They used isolated flexor digitorum brevis muscle stimulation, which was composed of 90 to 95% fast-twitch oxidative glycolytic fibers and 5 to 10% slow-twitch oxidative fibers (Hargreaves & Spriet, 2020). This fiber population is characterized by a high capacity to utilize lipid as a substrate for energy production (Lattimer & Haub, (2010). In this study the surface electrical stimulation of quadriceps muscle was applied bilaterally using a frequency of 50 Hz, which can be considered as a passive form of exercise. The reduction of blood triglyceride may be due to increased utilization of intramuscular triglyceride during electrical stimulation locally in the muscle. Hopp and Palmer (1990) concluded that intracellular triglyceride is a substrate for energy production during continuous and intermittent electrical stimulation. These findings show that there may be mobilization of triglyceride from blood to contracting muscle to meet the localized depletion in energy stores during electrical stimulation. In addition, during electrical stimulation, the stimulation protocol and the frequency of stimulation can influence this passively induced metabolic process. Houston *et al*. (1982) studied the metabolic effects of short-term electrical stimulation on quadriceps muscles using frequencies of 50 and 10 Hz. He reported that glycogen content in slow-twitch fibers was essentially unchanged following 10 Hz stimulation but moderately reduced in about 40% of slow-twitch fibers following 50 Hz stimulation. This suggests that fatty acids were a primary energy source in the former condition and glycogen in the latter condition. Wakeling *et al*., (2006) state in their' size principles' that slow

motor neurons which innervate slow twitch muscle fibers are recruited during activities where low-tension output is required. Fast motor units containing fast twitch fibers are progressively activated as the demand for muscle tension is increased. This does not get along with our finding of decreased triglyceride as we have used a frequency of 50 Hz which is comparable to high intensity, which would have depleted glycogen stores in the muscle. In our stimulator machine ET-3000, the current we used to be categorized as moderate faradic type current.

Significant decreases in blood cholesterol levels between pre and post-scores with both cycling, as well as electrical stimulation were observed in the current study. Total cholesterol levels have been reported to either increase or remain unchanged Gorgey *et al*., (2015) following electrical stimulation. In this study a decrease in total cholesterol level following exercise was observed. Rosenthal (2000) reported that the initial decrease in total cholesterol immediately after the exercise was due to a fall in LDL cholesterol. It is estimated that, at 18 hours after the exercise, approximately half of the decrease was due to a fall in VLDL cholesterol and half to a fall in LDL cholesterol. VLDLs transport triglyceride to muscle and adipose tissue. Fat utilized by a working muscle is derived not only from plasma-free fatty acids but also from muscle triglycerides. Exercise has been shown to lower plasma triglycerides-rich lipoprotein (Wang *et al*., 2019). A single bout of exhaustive aerobic exercise increases HDL particle changes whose formation may represent exercise-induced catabolism of triglyceride-rich lipoprotein. In our study, we used 20 minutes of cycle ergometry, which is a form of aerobic exercise. Prolonged exercise generally produces a small reduction in total cholesterol. The effect of exercise on total cholesterol is the summation of changes in the various lipoprotein subfractions so that changes in the total cholesterol alone have little physiological significance. Wang & Xu (2017) reported that some of the reduction in LDL-C may be due to the expansion of the plasma volume, which itself a possible beneficial acute exercise effect. Expanded plasma volume decreases blood viscosity and the concentrations of atherosclerotic cardiovascular risk factors, which may reduce their effect on the arterial wall. Further studies are warranted to clarify the role of exercise-induced changes in plasma lipids and lipoproteins. The present study also showed significant differences in blood cholesterol levels following electrical stimulation. We were unable to find studies in the area of electrical stimulation and blood cholesterol metabolism. Cycling is a generalized form of exercise that involves a lot of muscle activity and physiological demands. This can produce metabolic alterations, leading to a decrease in blood cholesterol levels. In the present study, the quadriceps muscles were stimulated for 20 minutes which may be considered as a localized aerobic activity of the muscle. Aerobic exercise reduces the activity of the slow-twitch muscle fibers. This might have led to lipoprotein lipase activity in capillary beds of the quadriceps muscle, where fatty acids are hydrolyzed from circulating lipoprotein for energy utilization, which in turn might have influenced lipoprotein metabolism. Skeletal muscle lipoprotein lipase has become the recent focus of exercise related effects on both plasma lipids and lipoprotein (Bey & Hamilton, 2003). Further studies are warranted to clarify these changes.

Exercise acutely increases HDL-C, which has varied from 4 to 43%. The increase generally parallels the decrease in triglyceride in onset and disappearance, suggesting mediation by similar metabolic changes. The increase in HDL-C reported in our study goes along with these findings. The quality and quantity of exertion required to increase HDL-C acutely is not defined, although changes in moderately fit and well-trained subjects have been reported after expenditure of 350 to 400 and 1000 Kcal, respectively in a single exercise session. Smaller changes may occur with less energy expenditure but require adjustment for expansion in plasma volume. The increase in HDL-C in sedentary subjects after exercise appears to be due to increases in HDL-3 whereas HDL-2 increases in trained individuals (Franczyk *et al*., 2023). Acute changes in APO AI and AII do not occur even with prolonged exertion, indicating that the changes in HDL-C are probably due to enhanced cholesterol delivery to HDL particles (Thompson *et al*., 2001). Exercise acutely increases lipoprotein lipase activity even in untrained individuals exercising for as little as one hour at 80% of maximal heart rate (Wang & Xu, 2017). Thompson *et al*. (2001) hypothesized that exercise acutely depletes intramuscular triglyceride, which stimulates the synthesis or translocation of lipoprotein lipase, which hydrolyzes triglycerides from lower-density lipoprotein with a transfer of excess surface cholesterol to the HDL particle. Exercise acutely reduces triglyceride and increases HDL-C. It is likely that these changes are related to total energy expenditure, but there is insufficient evidence to define whether caloric expenditure intensity of effort of some combination is responsible (Franczyk *et al*., 2023). The present study showed an increase in HDL-C following electrical stimulation. We were not able to find any studies done in this area. This study used a frequency of 50 Hz for 20 minutes on the quadriceps bilaterally. Baskin *et al*. (2015) suggested that slow twitch fiber activity causes preferential utilization of body fat as an energy substrate. Gugliucci (2023) has shown a higher percentage of slow twitch muscle fibers, which have a higher capacity to metabolize fatty acids liberated by lipoprotein lipase from triglyceride-rich lipoproteins, associated with increased plasma HDL-cholesterol levels. Skeletal muscle lipoprotein lipase has become the recent focus of exercise related effects on both plasma lipids and lipoprotein (Matsumoto *et al*.,

2019). Exercise increases skeletal muscle lipoprotein lipase activity. The increase in lipoprotein lipase activity may also have occurred in response to stimulation of the quadriceps muscle. The rise in lipoprotein lipase activity in the capillary beds of the quadriceps muscle, where fat is hydrolyzed from circulatory lipoprotein for energy utilization, might have influenced plasma triglyceride and HDL metabolism. Further studies are warranted to clarify these changes.

Conclusion

From the present study, it is concluded that electrical stimulation and voluntary exercise have comparable biochemical effects as far as lipid metabolism is concerned. Electrical stimulation may have the potential to bring about changes in blood lipid profile, which in turn may influence fat reduction. However, these possibilities require further investigation.

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