RESEARCH ARTICLE

The effect of swimming and running exercises on oxidant-antioxidant and lipid profiles in streptozotocin-induced diabetic rats

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Abstract

Background: Lifestyle changes in Diabetes Mellitus (DM) positively affect blood glucose and all risk factors. This study aims to determine the effect of swimming and running exercises on oxidant-antioxidant and lipid profiles in streptozotocin (STZ)-induced type 1 diabetic and non-diabetic rats.

Methods: We included forty-eight adult male Wistar albino rats in this study, and we randomly classified them into six groups (eight per group). The groups were organized as Control Sedentary, Control Exercise-swimming, Control Exercise-running (CE-r), Diabetes Sedentary (DS), Diabetes Exercise-swimming (DE-s), and Diabetes Exercise running (DE-r). Half of these rats were subjected to experimental diabetes via STZ. We evaluated total oxidant capacity (TOC), total antioxidant capacity (TAC), superoxide dismutase (SOD), and as lipid parameters: high-density lipoprotein (HDL), low-density lipoprotein (LDL), and triglycerides. The rats were sacrificed at the end of the four weeks.

Results: We found a significant difference between DE-s and DE-r groups in terms of TOC ($p = 0.043$) and SOD ($p = 0.030$). The highest TAC was found in the CE-r group, and the highest TOC was found in the DS group. Exercise significantly reduced LDL levels. There was no significant difference between the DE-s and DE-r groups ($p = 0.084$) for lipid profiles (HDL).

Conclusion: Based on the lower TOC (oxidant) and higher SOD (antioxidant) levels in the diabetic running group, these results suggest that running may be more beneficial than swimming for diabetics. HIPPOKRATIA 2023, 27 (4):148-154.

Keywords: Diabetes mellitus, moderate intensity exercise, oxidant-antioxidant, lipid profile

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Introduction

Diabetes Mellitus (DM) is one of the most severe diseases affecting the general population worldwide. The global diabetic population is estimated to rise by 2030 to 578 million and by 2045 to 700 million[1]. DM is a metabolic disorder marked by defects in carbohydrate, fat, and protein metabolism with multiple etiologies resulting from impairments in insulin secretion, insulin action, or both[2]. In addition to being a chronic metabolic disorder, DM is a disease that leads to oxidative stress, which has a critical role in the development of microvascular and cardiovascular diabetic complications[3]. Evidence is mounting that lifestyle changes can significantly reduce the incidence of DM. Benefits of exercise include improvements in blood pressure, cardiovascular health and quality of life, a satisfactory lipoprotein profile, increased insulin sensitivity, decreased insulin requirements, and reduced body weight. Even in experimental DM models, exercise is emphasized to positively alter glucose metabolism, protect pancreatic β-cell integrity, increase insulin sensitivity[4], and make critical changes in triglyceride (TG) and total cholesterol levels[5]. Exercise's therapeutic benefits are well established in patients with metabolic syndrome or DM[6]. People with DM pursue the benefits of exercise, but DM management is complex. For these patients, understanding exercise physiology in health, specifically the control of metabolism and fuel mobilization, highlights the problems in managing DM through exercise[7]. Regular aerobic exercise has been reported to reduce the production of free oxygen radicals, thereby strengthening antioxidant defenses and reducing DNA damage[8]. It has been argued that in diabetic rats, swimming may have a positive effect on lipid metabolism[9] and may lead to an important recovery of antioxidant enzymes and metabolic parameters[10]. The study by Hung et al[11] found that treadmill exercise lowered TG levels in diabetic rats, and another study found that exercise positively affected elevated lipid levels in diabetic rats[12]. Similarly, despite elevated MDS levels after running exercises in rats, superoxide dismutase (SOD) levels increased and antioxidant

defenses improved[13]. It was reported that aerobic exercises performed by rats on a treadmill decreased oxidative stress and increased SOD and glutathione peroxidase[14]. Therefore, many researchers have begun using running or swimming exercises in rats and rodent models to study various physiological and metabolic responses. However, it is seen that the number of studies using two different exercises in the same study is limited. Consequently, it is necessary to rule out the effects of two different types of exercise (swimming and running). Therefore, the current study aimed to evaluate the effect of two distinct exercise types (swimming and running) on oxidant-antioxidant metabolism and lipid profiles in type 1 diabetic rats.

Material and Methods

Animals

In this study, we utilized adult male Wistar albino rats (n =48) weighing approximately 250-300 g obtained from the Experimental Animal Unit of Mersin University. The Local Ethics Committee of Mersin University Animal Experiments approved the study (decision No 09, date: 11/02/2019). In determining the number of animals to be used in the experiment, the Type I error value was 0.05, and the power analysis was 80 %. The experimental animals are divided into six groups: control sedentary (CS), control exercise-swimming (CE-s), control exercise-running (CE-r), diabetes sedentary (DS), diabetes exercise-swimming (DE-s), and diabetes exercise-running (DE-r). Rats were fed *ad libitum* with standard rodent diets (20-22 % crude protein, 2,600-2,650 kcal/kg energy, 4-5 % crude fat, 5-7 % crude cellulose) in a plastic rat cage, in the experimental animal unit, at 23 ± 2 °C ambient temperature, in 50 ± 10 % relative humidity environment for 12/12 hours night/day.

Diabetes Mellitus Induction

Streptozotocin (STZ) is an antibiotic that destroys pancreatic islet beta cells and is widely used experimentally to create a type 1 DM (T1DM) model. In the study, we administered a single intraperitoneal streptozotocin dose of 45 mg/kg to the rats, which induced a T1DM diabetic state three days after STZ administration. Fasting blood glucose levels were measured with a Gluco-Meter (PlusMED) using capillary blood collected from the tail tip of the rats. It was checked whether a diabetic state was induced or not. Blood glucose levels of all the animals in this group were higher than 250 mg/dl, and it was confirmed that all the rats became diabetic[15, 16].

Exercise Protocols

Swimming Exercise Protocol

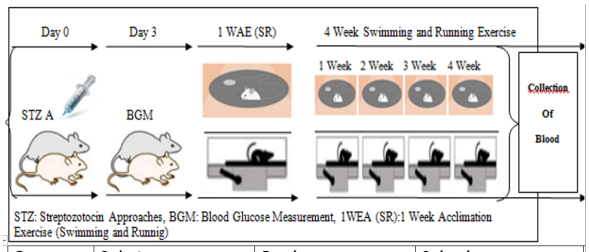
We conducted the swimming exercise protocol in two phases: adaptation (one-week duration) and training. On the first day of the adaptation phase, rats performed 10 minutes of exercise in a Morris water tank (water 22-25 °C), and each day, adaptation was prolonged by 10 minutes until the rats could swim for 30 minutes (except for the sedentary and running groups). After one week of swimming adaptation, the exercise phase-commenced. In the exercise phase, the animals in the CE-s (swimming; 30 min/day, 3 day/4 weeks) and DE-s (swimming; 30 min/day, 3 day/4 weeks) groups performed the exercises at the same time of the day[17].

Treadmill Exercise Protocol

Similarly, we conducted the running exercise protocol in two phases: adaptation and training. The adaptation phase lasted one week. On the first day, the rats ran on a treadmill (animal treadmill) for 10 minutes. Two rats were made to run at the same time for adaptation, and the adaptation period was prolonged for 10 minutes each day until the rats could run for 30 minutes (except for the sedentary and swimming groups). The exercise phase commenced after one week of the adaptation phase. In the exercise phase, the animals in the CE-r (running; 30 min/day, 3 day/4 weeks) and DE-r (running; 30 min/day, 3 day/4 weeks) groups performed the exercises at the same time of the day[18,19]. Training took four weeks (a total of 90 minutes/week) (Figure 1a, Figure 1b).

Blood Sampling and Analyses

Many studies have shown that ketamine/xylazine anesthesia does not make a significant difference in oxidative stress. Blood samples collected under intracardiac



Groups	Sedentary groups	Running groups	Swimming groups
Control groups	Control sedentary(n:8)	Control running(n:8)	Control swimming(n:8)
DM groups	DM sedentary (n:8)	DM running(n:8)	DM swimming(n:8)

Figure 1a: The experimental setup of this study and specifications of the exercise (swimming and running) planning.

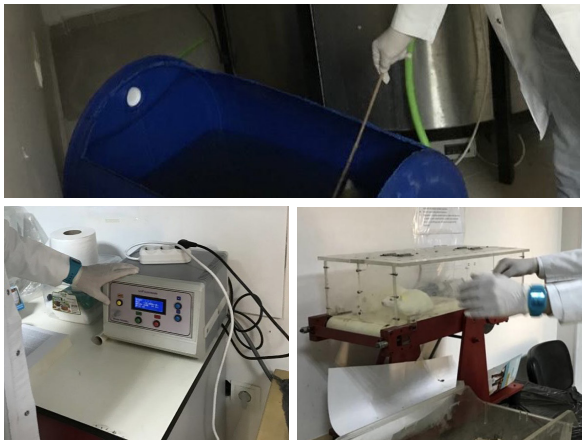


Figure 1b: Images demonstrating the performance of the running and swimming exercises in the experimental setups.

ketamine/xylazine anesthesia were centrifuged at 1,500x g for 10 minutes and stored at -80 °C. We utilized the blood samples of all experimental groups to measure levels of total antioxidant capacity (TAC), total oxidant capacity (TOC) and superoxide dismutase (SOD), low-density lipoprotein (LDL), TG, and high-density lipoprotein (HDL)[20].

Measurements Related to Biochemical Analysis

TOC and TAC were analyzed using Rel Assay Diagnostics kits (Rel Assay, Mega Tıp, Gaziantep, Turkey). The kits had Trolox Equivalent Antioxidant Capacity (TEAC) to analyze samples. The ABTS method is based on antioxidants blocking the absorbance of radical cation. Samples (serum) were collected from the rats and analyzed in the laboratory using the kits[21,22].

We measured SOD activity as per the previously described method after obtaining whole blood with EDTA. A glass spectrophotometer cuvette was filled with 2,450 µl of the test mixture (0.3 mM xanthine, 0.6 mM EDTA, 150 µM NBT, 0.4 M Na_2CO_3, 1.2 g/L BSA), 500 µl supernatant, and 50 µl xanthine oxidase, and then they were mixed. It was incubated for an average of 20 minutes. After the addition of 100 µl of 0.8 mM $CuCl_2$, the reaction was terminated.

The HDL, LDL, and TG quantitation kit was used to determine the concentrations of HDL, LDL, and TG in serum samples. This kit separates first the serum HDL, LDL, and TG, and subsequently, the cholesterol concentration of each is determined by a dual enzyme assay, resulting in a colorimetric (570 nm)/fluorometric (λex =535/λem =587 nm) product. We used the Rel brand assay kits and Mindray brand BS300 model fully automated biochemical analyzer.

Statistical Analyses

Six groups in the study (swimming, running, and sedentary) were created separately for the experimental and control groups. We calculated the number of rats in each group to be at least three. However, 48 rats (eight rats in each group) were included in the animal experiment to account for the loss that may occur during the experimental phase. We utilized the Shapiro-Wilk-W test to calculate the distribution's normality and determined that it was not normally distributed. Descriptive statistics are presented via arithmetic means, standard deviations, and graphs. We used the Kruskal Wallis H, and Mann Whitney U tests for intra-group comparisons. We used multiple comparison tests (post hoc) to determine which group produced the difference when we found a significant difference. We considered a p-value <0.05 significant for detecting differences from statistical analyses and within-group differences.

Results

Total antioxidant capacity

For pre-study data, values from the sedentary control groups were used as a reference. For post-study data, we used intracardial sera obtained from the blood of the rats. TAC levels differed between the experimental and control groups ($p < 0.001$). When the groups were compared to the CS group (as reference), we found TAC values to be decreased ($p < 0.001$). TAC levels were higher in the DE-r group than in the DE-s group, but the difference was not significant ($p = 0.825$) (Table 1).

Total oxidant capacity

The experimental and control groups' TOC values obtained from rats significantly differed ($p < 0.001$). When we compared the groups to the CS group (as reference) in terms of TOC values (Figure 2 and Table 1), TOC values were found to be increased ($p = 0.024$). The average TOC value in the DS group was the highest of all groups, while the average TOC level in the CE-s group was the lowest. When we compared the DE-s and DE-r groups, a significant difference was found in TOC values, and the DE-r group had lower TOC values ($p = 0.043$).

SOD values

SOD levels obtained from rats demonstrated a significant difference between the experimental and control groups ($p < 0.001$). When the groups were compared to the CS group in terms of SOD values (Figure 2 and Table 1), the CE-s group had the highest SOD level. In diabetic groups, we found the highest SOD level in the DE-r group. When comparing the DE-s and DE-r groups, a significant difference in SOD levels was detected ($p = 0.030$).

Lipid Profiles

We found the LDL levels in rats to be different between the experimental and control groups ($p = 0.001$). When we compared the groups to the CS group in terms of LDL levels (Figure 3a and Table 2), the DS group had the highest LDL level ($p < 0.001$). When comparing the DE-s and DE-r groups to the CS group, the LDL value also decreased significantly compared to the DS group ($p < 0.001$). We found no significant difference in LDL levels between the DE-s and DE-r groups ($p = 0.618$). TG obtained from rats differed significantly between the experimental and control groups ($p < 0.001$). When we compared the groups with the CS group in terms of TG levels (Figure 3b and Table 2), it was found that DM increased TG levels ($p = 0.020$). When comparing DE-s and DE-r groups to the CS group, TG levels were high and considerably similar to the DS group ($p = 0.014$). No significant difference was found between the DE-s and DE-r groups in terms of TG value ($p = 0.636$). HDL levels differ significantly between the experimental and control groups ($p = 0.007$). DM reduced HDL value when comparing the groups with the CS group (as reference) in terms of HDL level (Figure 3c and Table 2). HDL decreased in the diabetic groups. We observed no significant difference between the DE-s and DE-r groups regarding HDL levels ($p = 0.084$).

Table 1: Different exercise approaches among diabetic rats demonstrate a difference in oxidant-antioxidant values among the experimental groups.

Group	TAC (mmol/L)		Kruskal Wallis H	
	Mean	Sd.	z	p
CS	1.53	0.23		
CE-s	1.66	0.24		
CE-r	1.64	0.33		
DS	1.28	0.18		
DE-s	1.18	0.14	24.799	0.001
DE-r	1.20	0.21		
Group	**TOC (µmol/L)**			
	Mean	**Sd.**	**z**	**p**
CS	30.17	9.64		
CE-s	20.08	7.54		
CE-r	24.50	16.65		
DS	33.90	15.41		
DE-s	31.37	8.63	12.920	0.024
DE-r	22.18	7.89		
Group	**SOD (U/ml)**			
	Mean	**Sd.**	**z**	**p**
CS	195.13	14.29		
CE-s	195.53	11.98		
CE-r	190.12	12.44		
DS	174.52	17.80		
DE-s	173.36	18.56	13.392	0.020
DE-r	195.02	17.34		

Values are presented as mean and standard deviation. Sd: standard deviation, TAC: Total antioxidant capacity, TOC: Total oxidant capacity, SOD: Superoxide dismutase, CS: Control Sedentary, CE-s: Control Exercise-swimming, CE-r: Control exercise-running, DS Diabetes Sedentary, DE-s: Diabetes exercise-swimming, DE-r: Diabetes exercise Running.

Table 2: Different exercise approaches among diabetic rats demonstrate a difference in lipid profile values among the groups.

Group	LDL (mg/dl)		Kruskal Wallis H	
	Mean	Sd.	z	p
CS	16.25	4.86		
CE-s	15.87	4.54		
CE-r	14.42	1.90		
DS	32.25	5.44	20.279	0.001
DE-s	15.62	3.15		
DE-r	16.37	2.72		
Group	**TG (mg/dl)**			
	Mean	**Sd.**	**z**	**p**
CS	63.87	23.70		
CE-s	45.70	10.60		
CE-r	44.28	7.01		
DS	89.37	14.10	31.028	0.000
DE-s	90.12	13.45		
DE-r	94.12	19.20		
Group	**HDL (mg/dl)**			
	Mean	**Sd.**	**z**	**p**
CS	145.87	18.65		
CE-s	151	25.45		
CE-r	149.28	15.26		
DS	121.37	34.34	16.04	0.007
DE-s	115.37	22.51		
DE-r	117.37	15.84		

Values are presented as mean and standard deviation. Sd: standard deviation, LDL: Low density lipoprotein, TG: Triglycerides, HDL: High density lipoprotein, CS: Control Sedentary, CE-s: Control Exercise-swimming, CE-r: Control exercise-running, DS Diabetes Sedentary, DE-s: Diabetes exercise-swimming, DE-r: Diabetes exercise Running.

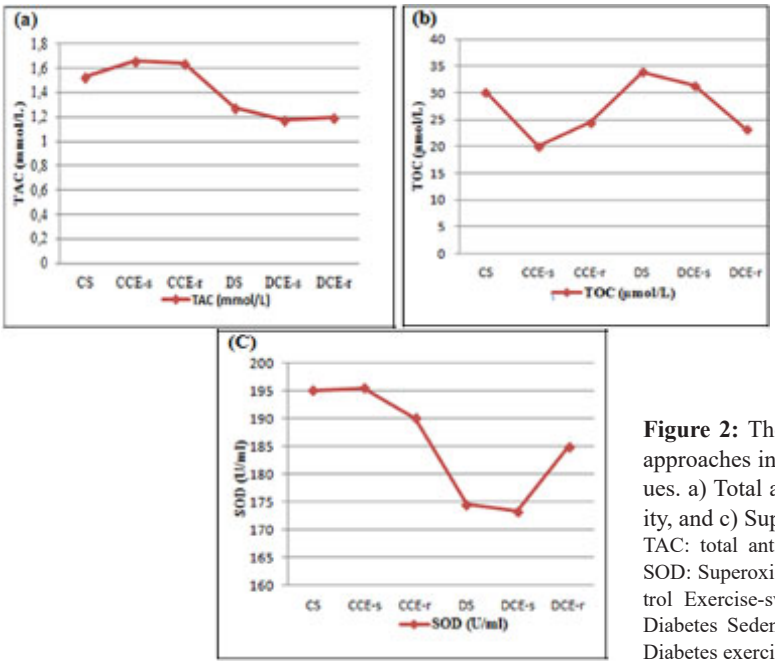


Figure 2: The effect of swimming and running exercise approaches in Diabetic rats upon oxidant-antioxidant values. a) Total antioxidant capacity, b) Total oxidant capacity, and c) Superoxide dismutase.
TAC: total antioxidant capacity, TOC: Total oxidant capacity, SOD: Superoxide dismutase, CS: Control Sedentary, CE-s: Control Exercise-swimming, CE-r: Control exercise-running, DS: Diabetes Sedentary, DE-s: Diabetes exercise-swimming, DE-r: Diabetes exercise Running.

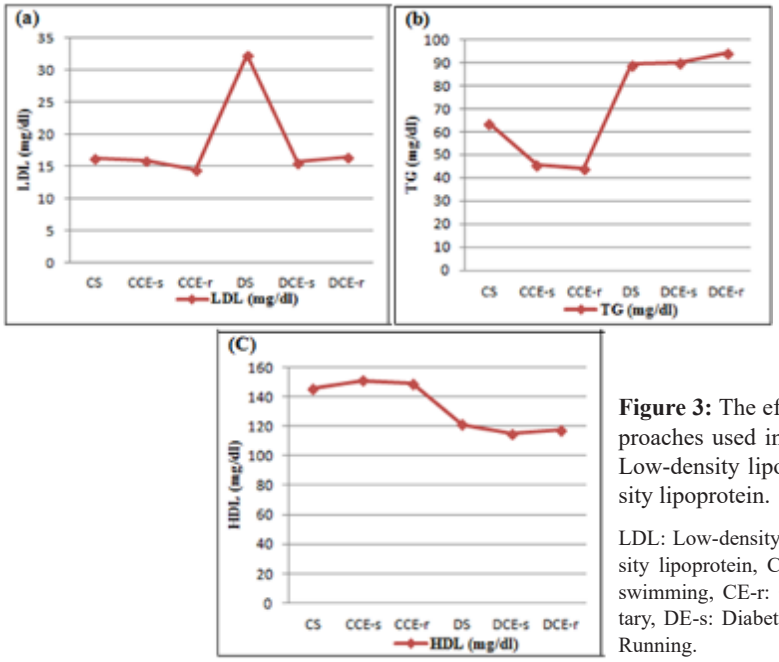


Figure 3: The effect of swimming and running exercise approaches used in diabetic rats upon lipid profile values. a) Low-density lipoprotein, b) Triglyceride, and c) High-density lipoprotein.

LDL: Low-density lipoprotein, TG: Triglyceride, HDL: High-density lipoprotein, CS: Control Sedentary, CE-s: Control Exercise-swimming, CE-r: Control exercise-running, DS: Diabetes Sedentary, DE-s: Diabetes exercise-swimming, DE-r: Diabetes exercise Running.

Discussion

DM is a metabolic disease due to hyperglycemia caused by insulin deficiency resulting from β cell damage in the Langerhans islets of the pancreas[23]. We aimed to demonstrate the effects of running and swimming exercises performed to prevent diabetic complications on oxidant-antioxidant and lipid profiles in experimental STZ-induced type 1 DM rats.

Experimental studies suggest that oxidative stress is critical in both types of DM pathogenesis[24]. Free radicals and oxidative stress are among the major mediators of beta cell destruction and death caused by autoimmune destruction of beta cells in T1D or glucotoxicity and insulin resistance in type 2 DM[25]. The present study found a significant difference in TAC values between the CS and DS groups. Due to the adverse effects of DM, the TAC value decreased. Both immobility and DM were associated with decreased TAC, indicating that DM negatively affected total antioxidant capacity. The study by Alzoubi et al, which aimed to treat short-term and long-term memory impairment in rats through swimming exercise, concluded that swimming exercise was an indicator of reduced oxidative stress[26]. This study found a difference between DE-s and DE-r groups regarding TAC values. We found the mean TAC value higher in the CE-r group than in the DE-s group. It has been reported that acute exercise could not stop the increase in liver lipid peroxidation in STZ-induced diabetic rats; however, regular treadmill running reduced lipid peroxidation and hyperglycemia[27]. Similarly, Temiz et al suggested that swimming exercises in rats increased lipid peroxidation in the liver, skeletal muscles, and brain.

In this study, it was observed that DM elevated the TOC level[28]. The DS group had the highest mean TOC value compared with the rest of the groups, whereas the CE-s group had the lowest. When we compared the DE-s and DE-r groups, there was a significant difference in the TOC levels, and we found that the TOC levels were lower in the DE-r group. As a result, running exercises performed in rats with DM decreased the TOC level and resulted in a beneficial effect. Qiao et al reported that swimming exercises done in rats resulted in the highest SOD activity in both skeletal muscle and heart tissue[29]. Similarly, Lima et al found in wistar rats an increase in SOD activity following an exhaustive exercise performed after swimming exercise[30]. In this study, when compared with the CS group, the CE-s group had the highest SOD levels, and DM reduced the SOD value. Previous studies have reported low SOD activity associated with failure of the antioxidant system. In addition, increased SOD enzyme activity caused by superoxide may be reduced due to oxidative damage[31]. Freitas et al found on Wistar rats, the SOD levels increased after high-intensity running exercise, and antioxidant defenses were improved[13]. In the present study, among the diabetic groups, the DE-r group had the highest SOD level. However, when we compared the DE-s and DE-r groups, no significant difference in SOD levels was found. Yet, in the diabetic groups, running exercise made a more positive contribution to SOD levels compared to swimming exercise. Delfan et al studied the adaptations to different exercise stimuli in the skeletal muscle of diabetic rats and also found that the increase in the "work-rest" ratio during high-intensity interval training had a significant impact on skeletal muscle mitochondrial adaptations. Therefore, they concluded that manipulations of the work-rest ratio could improve mitochondrial adaptations in diabetic patients[32]. In our study, the increase in TAC and the high SOD level is considered to have been achieved due to mitochondrial adaptation, been abundant in skeletal muscle during running exercise.Therefore, running exercise can be effective in

preventing the accumulation of free radicals in the cell during aerobic respiration, catalyzed by the mitochondrial electron transport system.

Several lines of evidence suggest that exercise has beneficial effects on adipose tissue through increased mitochondrial function and lipolysis, as well as increased insulin sensitivity with exercise[33]. Aerobic exercise induces a protective adipose tissue phenotype against oxidative stress in adipose tissue. When evaluated regarding adipose tissue biology, the results indicate that exercise is essential in developing metabolic capacity and increasing defense against oxidants in adipose tissue[34]. It has been reported that in male rats exposed to forced swimming, insulin sensitivity is worsened, leading to glucose intolerance and insulin resistance. However, a beneficial effect on lipid metabolism may be achieved[9]. In this study, the DS, compared to the CS group, had the highest LDL level, and DM increased LDL values. When we compared the DE-s and DE-r groups with the CS group, the LDL levels also decreased considerably compared with the DS group, suggesting that exercise benefits lipid profiles in DM. No significant difference in LDL levels was found between the DE-s and DE-r groups. Badole et al found higher cholesterol and TG levels in the sedentary DM group compared to the sedentary control group[35], while Hung et al reported that the treadmill exercise decreased TG levels in diabetic rats[11]. In this study, DM was found to increase TG levels compared to the CS group. When we compared the DE-s and DE-r groups with the CS group, the DS group had the highest TG level ($p = 0.020$). We found no significant difference in TG levels between the DE-s and DE-r groups, possibly due to the shorter duration of exercise. Compared with the CS group, the CE-s group had the highest HDL level, and DM reduced HDL level. HDL levels decreased in the diabetic groups. Lee et al argued that treadmill exercise in DM did not increase the HDL level but positively affected the lipid profile of diabetic rats[36]. In this study, when the CE-s group and DE-s groups were compared, the CE-s group had a higher HDL level. There was no significant difference in HDL levels between the DE-s and DE-r groups. It was found that swimming and running exercises performed in DM did not increase the HDL level. Therefore, regular exercise in the long term may lead to a slight reduction in fat, body weight, total cholesterol, TG, and LDL cholesterol. On the other hand, it may lead to an increase in HDL cholesterol. However, as seen in the current study, DM was found to have an adverse effect on lipid profile.

We compared the effects of swimming and running exercise on the adverse complications of DM. In diabetic rats, neither running nor swimming exercise significantly differed in LDL, TG, and HDL levels. However, it has been found that both exercises benefit from reducing LDL levels. In terms of SOD, a significant difference was found between the DE-s and DE-r groups ($p = 0.020$), showing that running exercise will be more beneficial in preventing oxidative stress in rats with DM.

Swimming and running exercises used in diabetic rats positively reduced TOC levels of oxidant-antioxidant capacity. Regarding TOC, when the DE-s and DE-r groups are examined, the lower TOC value in the DE-r group also confirms that running exercise can be preferred to swimming in diabetics ($p = 0.043$). To the best of our knowledge, this is the first study to compare running and swimming exercises by assessing lipid profile and oxidant and antioxidant capacity. In DM, studies should also be done on oxidant-antioxidant and lipid profiles in general, with longer exercise durations.

Limitations of the study

In this study, it was not possible to evaluate oxidant, antioxidant parameters and lipid profiles by collecting sufficient amounts of blood at different times during the experiment. Because DM has reduced body resistance in DM rats, the metabolic effects of running and swimming exercises may be altered by the stress of blood sampling and may even be considered a cause of death. Since we compared the effects of different exercises in DM rats, histological results could add to the results. However, the experiment did not include this in its initial design and was out of our possibilities.

Conclusion

Our study compared the effects of swimming and running exercise on the adverse complications of DM. In diabetic rats, neither running nor swimming exercise significantly differed in LDL, TG, and HDL levels. However, we found both exercises to benefit by reducing LDL levels. In terms of SOD, there was a significant difference between the DE-s and DE-r groups. This shows that running exercise is more beneficial in preventing oxidative stress in rats with DM.

Swimming and running exercises in diabetic rats positively reduced TOC levels in assessing oxidant-antioxidant capacity. Regarding TOC, when examining the DE-s and DE-r groups, the lower TOC value in the DE-r group also confirms that running exercise can be preferred to swimming in diabetics. To the best of our knowledge, this is the first study comparing running and swimming exercises by assessing lipid profile and oxidant and antioxidant capacity. In the case of DM, studies with longer exercise durations should also be conducted to prove the positive effects of exercise on oxidant-antioxidant and lipid profiles in general.

Conflict of Interest

Authors declare no conflicts of interest.

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