Aerobic Exercise Training Prevents Impairment in Renal Parameters and in Body Composition of Rats Fed a High Sucrose Diet.

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Abstract

Objective: This study aimed to evaluate the effect of swimming training (T) on the renal system and body composition parameters in young animals treated with a high sucrose diet (SUD) during 12 weeks.

Results: The SUD impaired the physical performance, increased the body adiposity index (BAI), Lee index (LI) and retroperitoneal adipose tissue (RAT) weight, proteinuria, plasma creatinine and number renal cells nuclei, decreased urinary volume and creatinine besides creatinine clearance. The T reversed the increased the BAI, LI, RAT weight, plasma and urinary creatinine, creatinine clearance and number renal cells nuclei. This study found that eight weeks of swimming physical training protected renal function and restored normal glomerular filtration rate (GFR) values. Swimming training also contributed to prevention of the onset of a renal inflammatory process and caused a decrease in the risk of development of obesity promoted by SUD decreasing the body composition parameters (BAI, LI, and RAT weight).

Introduction

A high sugar level is a factor that has been linked to an increase in kidney disease and association with some risk factors, including diabetes, gout, metabolic syndrome [1–4] and the development of obesity. Besides an increase in oxidative stress and nitric oxide inactivation via reactive oxygen species, high sugar also induces high level of creatinine and blood urea nitrogen [5]. Sucrose, specifically, can promote an increase in proteinuria and a higher percentage of abnormal glomeruli in obese rats [6].

Physical exercise is recognized to promote alterations in renal parameters in experimental animal models [7–9] in a chronic manner thus providing renal-protective effects in high sugar diet models and neutralizing the accumulation of renal triglycerides associated with this diet, which is partially mediated by upregulation of fatty acid β-oxidation [7].

Data have shown that a metabolic re-programming process subsequent to early-life insults, i.e. maternal life or childhood, occurs leading to development of chronic diseases [10, 11]. The use of food rich in refined sugar has been shown to exponentially raise sugar levels in children and adolescents since the total caloric intake and the prevalence of metabolic syndrome are increasing continuously in these populations [12].

A balanced diet and regular physical exercise are of great relevance in preventing obesity and cardiovascular and kidney diseases, justifying the development of studies that assess the beneficial effects of regular physical training in childhood that will contribute to the decrease in deleterious effects of consuming a diet rich in simple carbohydrates.

Thus, this study aimed to evaluate the effect of swimming training (T) on the renal system and body composition parameters (body adiposity and Lee indices [BAI and LI, respectively] and retroperitoneal adipose tissue [RAT] weight) in animals treated with SUD.

Methods

Animals: 38 male Wistar rats, age 21 days (50g) (come from the CCA-UFOP) divided randomly, into 4 groups (4 animals/box): (1) sedentary rats fed with standard diet (S-SD), n = 14; (2) T rats fed with standard diet (T-SD), n = 9; (3) sedentary rats fed with high sucrose diet (S-SUD), n = 05; and (4) T rats fed with SUD (T-SUD), n = 10; marked on the tail; allocated in a room at a temperature of 24 ± 2 °C and 12-hour light/dark cycle (7 am - 7 pm). *Body weight assessments:* realized at weeks 1, 4, 5, 7, and 11. *Food intake assessments:* realized at weeks 1, 5, and 10. *Approval of the study:* by the Ethics Committee for Animal Use, UFOP (Protocol 45/2014). The experimental design scheme was shown in figure S2.

Diets: SD - commercial rodent feed in pellets (Nuvilab CR1 Quimtia®) and SUD as previously published [13] administered for 12 weeks from weaning.

Exercise training and evaluation of endurance capacity: *Local* - collective glass tank of CCA-UFOP with warm water at 28 ± 2 °C and 45 cm depth. *Adaptation period:* 15 min (first day) increased by 15min each day until reaching 60 min (fourth day) (*adapted from* [14]). *Maximal test:* at the 4th and 12th (24 h after the last training section) weeks as proposed by others [15]. Fatigue point was *adapted from* [16]. *Training:* swimming for 8 weeks. The first 4 weeks didn't use a workload, but that was added at the fifth week (60% of that obtained in maximal test) as *adapted from* [15].

Water Intake, Urinary Volume and Water Balance Measurements in 24h:

Rats were allocated in metabolic cages (Tecnoplast® SPA) during the 12-week period (CCA-UFOP); weighed (SF−400 scale), individually housed for a period of 24 h with free access to water and food. Measurements of urine volume and water intake were realized. Samples of urine 1.5 mL were obtained, labeled, and frozen at −20 °C. Calculation of water balance was done according to the equation:

Equation 1:

Water Balance (mL/24h) = (Water Intake [mL/24h]− Urinary Volume [mL/24h]).

Euthanasia: 48 h after the last T session. *Collected materials:* Adipose tissues inguinal, retroperitoneal and epididymal (IAT, RAT and EAT, respectively), blood and kidneys. Blood was centrifuged (CENTRIBIO 80-2 B scale) at 3000 rpm for 10 min to separate the plasma and then maintained at a temperature of −20 °C. *Details:* Supplementary Material (Table S2).

Determination of the LI:

LI was measured according to [17]. Animals' body mass and the nasoanal length [17], were calculated using the formula below:

Equation 2:

$$\frac{\sqrt[3]{BM\ (g)}}{NL\ (cm)}$$

Determination of the BAI:

BAI was measured according to [19]. The EAT, IAT, and RAT were removed and weighed (BEL precision scale), and used in the following equation:

Equation 3:

$$\frac{EAT\ x\ IAT\ x\ RAT\ (g)}{BW\ (g)}\ x\ 100$$

Evaluation of RAT:

RAT analyse provide an assessment of the risk of developing cardiovascular diseases [20].

Plasma and Urine Creatinine Concentration and Urine Protein:

Samples - plasma and urine; *kit* - commercially available kit (Labtest, Belo Horizonte/MG, Brazil) – by colorimetric modified Jaffé approach. Calculation of creatinine clearance (CrCl):

Equation 4:

$$CrCl\ (mL/min) = \frac{[Urine\ Creatinine\ (mg/dL)x\ Urine\ Volume\ (mL/24h)]}{[Serum\ Creatinine\ (mg/dL)\ x\ 1440\ (min)]}$$

Proteinuria was determined with the use of pyrogallol red technique using a commercially available kit (Bioclin, Belo Horizonte, Brazil), $n = 38$ rats. Results were expressed in mg/dL.

Renal Histology: *Collected material* - right kidneys; *Stage and calculation* - as described by others [21, 22]. *Equipment* - light microscope (Leica DM5000). *Analysis* - Analysis and Image Processing Software Leica Qwin (Germany) [23].

Data and Statistical Analysis: *Normality test* - Kolmogorov-Smirnov. *Statistical tests used* - One-way or two-way analysis of variance (ANOVA) and Tukey's post-test for multiple comparisons following ANOVA. *Software used* - GraphPad Prism 7.0 for Windows (GraphPad Software, San Diego California USA). Data were expressed as mean ± standard deviation of mean and differences between pairs of means were

considered significant when the probability of type I error was less than 5% ($p < 0.05$). The data were analyzed blindly by the researchers involved.

Results

Confirmation of the effectiveness of the T employed: SUD affected the physical performance at the 12th week since only the T-SD group had a longer exhaustion time with an increase of 16.9% (Table 1).

Table 1
Exaustion times and hydrics parameters for SD and SUD rats, sedentary and trained.

	Standard Diet (SD)		Sucrose Diet (SUD)	
	Sedentary (S-SD)	Trained (T-SD)	Sedentary (S-SUD)	Trained (T-SUD)
Initial ET (s)	720 ± 44 (n = 04)	774 ± 73 (n = 09)	819 ± 74 (n = 05)	770 ± 74 (n = 10)
Final ET (s)	709 ± 53 (n = 04)	853 ± 92* (n = 09)	791 ± 47 (n = 05)	782 ± 73 (n = 10)
Water Intake (mL/24h)	29.5 ± 5.7 (n = 14)	33.9 ± 4.2 (n = 09)	29.0 ± 2.2 (n = 05)	30.9 ± 1.9 (n = 10)
Urinary Volume (mL/24h)	14 ± 2.4 (n = 14)	9 ± 3.9 (n = 09)	8 ± 1.4* (n = 05)	14 ± 5.9 (n = 10)
Water Balance (mL/24h)	15.8 ± 5.9 (n = 14)	24.6 ± 6.4* (n = 09)	21.1 ± 3.5 (n = 05)	17.3 ± 5.5+ (n = 10)

Exaustion times before (Initial ET) and after (Final ET) were realized on the 4th and 12nd (24 hours after the last training section) weeks. It was used the parametric test, two-way ANOVA followed by Tukey's post-test; $p < 0.05$. *different from S-SD group. Mensurements of water intake and urinary volume were realized on the metabolic cages, after 24h. It was used the parametric test, two-way ANOVA followed by Tukey's post-test; $p < 0.05$. *different of S-SD group; +different of T-SD group.

Effects of T on body composition: At the 11th week, SUD caused a decrease of 23.8% in the body weight gain and an increase in the BAI (69%), LI (10.5%) and RAT weight (28.6%) as shown in Fig. S1, panels A to C. T caused a decrease of 15.2% in body weight gain only in the T-SD (Table S1) and a decrease in BAI (81%), LI (15.8%), and RAT weight (55.1%) in T-SUD, decreasing only the BAI (54.9%) and RAT weight (26.5%) in T-SD.

Effects of the T on 24-hours parameters hydric: SUD caused a decrease of 42.9% the urinary volume, while and T restored these values to normal levels (Table 1). However, T caused an increase of 36% in water balance in the T-SD (Table 1).

Effects of the T on the renal biochemical parameters: SUD led to a 28.7% increase in proteinuria (panel A, Fig. 2), and a 22.9% increase in plasma creatinine (Fig. 1, panel B). SUD also led to a 46.2% and 75% decrease in urinary creatinine and creatinine clearance, respectively (Fig. 1, panels B and D, respectively). T protected the kidney against SUD-induced modifications of plasma and urinary creatinine and creatinine clearance.

Effects of the T on renal structure: SUD promoted a small increase of 31.5% in cell nuclei (Fig. 2 panel C, third quadrant left side, and panel G) and T caused a reduction of 31.1% about this parameter.

Discussion

This study found that eight weeks of swimming physical training protected renal function and restored normal GFR values. Swimming training also contributed to prevention of the onset of a renal inflammatory process and caused a decrease in the risk of development of obesity promoted by SUD decreasing the body composition parameters (BAI, LI, and RAT weight).

Although some authors have not observed impairment in the physical performance of animals fed a diet rich in simple carbohydrates [24], SUD contributed negatively to the physical performance of the animals in our study. After 12 weeks or T the exhaustion time was higher only in animals of the T-SD group.

Sugar seems to increase parameters, such as BAI [19, 25–27], LI [27], and RAT weight [28, 29] that contribute to the development of cardiovascular diseases [26]. In our study we observed that all these parameters were increased by SUD. Although, T caused a reduction in BAI in the T-SUD (81%) and T-SD (54.9%) groups and LI and RAT weight in the T-SUD group although some studies haven't shown the effects of physical exercise in reducing the BAI, LI and RAT weight [13, 20, 25, 30] promoted by high sugar content. A possible explanation for these exercise-associated effects is energetic homeostasis tends to been maintained in mammals through a balance between energy intake and expenditure, thus influencing body weight [31] doing the T have an important role in controlling these factor [32, 33].

Some studies have shown that a sugar can have influence (or not) in parameters like water intake [8, 34, 35], urinary volume [8], and water balance [8] in rodents. In this study, SUD didn't affect water intake in any group and caused a decrease in urinary volume since that T restored it to normal levels. The T-SD group had a higher water balance, demonstrating that swimming promotes changes in the hydric balance depending on the diet to which it was assigned. When aligned with SD, this model seems to have had a higher expenditure of water by other means than urinary excretion. Therefore, when swimming was undertaken by the SUD group, these differences do not appear. There are no studies that demonstrate how changes in water balance occur in response to sugar overload in rodents and outline the mechanisms by which this process happens. Studies that investigate this parameter are necessary to

clarify many of the physiological changes that may occur in rodents fed with sugar overload in response to changes in hydric parameters of these animals, since these issues may be related to some compensation of the physiological system to maintain homeostasis, mainly of the renal system.

It is known that the overload sugar can modifying the following parameters: (1) proteinuria [35, 36], (2) plasma and urinary creatinine [35], and (3) creatinine clearance (used as estimate of the GFR) [35, 37]. In our study, SUD caused an increase in proteinuria, but T didn't reverse this situation. SUD also caused an increase in plasma creatinine and decrease urinary creatinine and creatinine clearance. T led to a reversal in GFR alterations by bringing this parameter back to normal levels corroborating with others [7].

Sugar has also been seen as a villain that can cause renal inflammation, as noted in other study [38]. In our research, SUD caused an increase in cell nuclei suggesting an increase in the number inflammatory infiltrates. T protected against these SUD-induced changes (decreasing this parameter in 31.1%), thus corroborating the results presented by others authors [39]. All of the other parameters were not affected neither by SUD nor for T (Fig. 2, panels E, F, H, and I).

It was observed that damage to kidney function appeared earlier than structural renal damage in response to excess sugar in the SUD. High glucose-induced oncotic pressure alterations didn't initially modify the structure of the glomerulus but did modify renal function probably by increasing the excretion of urinary proteins, thus increasing proteinuria and decreasing GRF [40]. In addition, the time of exposure to the diet and age of animals (young animals) could have provided protection for the renal structure conferred by mechanisms of adaptation to sugar overload.

Collectively, our study demonstrated that T prevented SUD-induced impairment of kidney functional parameters and was efficient in preventing an SUD-induced increase adipose tissue deposits.

Limitations

We lacked investigate the cardiovascular parameters complementing the data aiming to observe the effects of sugar overload also on these parameters, being able to make a direct relationship between renal parameters, body composition and cardiovascular parameters in response to SUD and T.

Abbreviations

UFOP: Federal University of Ouro Preto; T: swimming training; SUD: high sucrose diet; S-SD: sedentary standard diet; S-SUD: sedentary SUD; T-SD: trained standard diet; T-SUD: trained SUD; BAI: body adiposity index; LI: Lee index; RAT: retroperitoneal adipose tissue; EAT: epididymal adipose tissue; IAT: inguinal adipose tissue; RAT retroperitoneal adipose tissue; CrCl: creatinine clearance; GFR: glomerular filtration rate; CCA-UFOP: Center of Animal Science of UFOP.

Declarations

Information regarding

Criteria for the inclusion of animals in the study; Criteria for the exclusion of animals in the study; Animals excluded of the study; Handling of the animals; Monitoring of the animals; Number of animals per experimental procedures; Justification of the choice of experimental procedures; Sample calculation; Euthanasia, were described in Table S2 in the Supplementary Material.

Ethics approval and consent to participate

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

- **JAS:** Design of experimental protocols; performed the experiments; analyzed data; interpret results of experiments; prepared figures; drafted manuscript; approved final version of the manuscript.
- **ABGP:** Design of experimental protocols; performed the experiments; analyzed data; interpret results of experiments; prepared figures; drafted manuscript; approved final version of the manuscript.
- **ECO:** edited and revised manuscript; approved final version of the manuscript.

- **DBC:** edited and revised manuscript; approved final version of the manuscript.
- **NLT:** Performed histology; helped in experiments; edited and revised manuscript; approved final version of the manuscript.
- **WGL:** Performed histology; edited and revised manuscript; approved final version of the manuscript.
- **LKB:** Conception and design the research; analyzed data; helped in experiments; interpret results of experiments; prepared figures; drafted manuscript; edited and revised manuscript; approved final version of manuscript.

References

1. Johnson, R.J., et al., *Potential role of sugar (fructose) in the epidemic of hypertension, obesity and the metabolic syndrome, diabetes, kidney disease, and cardiovascular disease.* Am J Clin Nutr, 2007. **86**(4): p. 899-906.

2. Gross, L.S., et al., *Increased consumption of refined carbohydrates and the epidemic of type 2 diabetes in the United States: an ecologic assessment.* The American Journal of Clinical Nutrition, 2004. **79**(5): p. 774-779.

3. Neilson, E.G., *The Fructose Nation.* 2007. **18**(10): p. 2619-2621.

4. Malik, V.S., et al., *Sugar-Sweetened Beverages and Risk of Metabolic Syndrome and Type 2 Diabetes: A meta-analysis.* Diabetes Care, 2010. **33**(11): p. 2477-2483.

5. Roberts, C.K., et al., *Oxidative stress and dysregulation of NAD(P)H oxidase and antioxidant enzymes in diet-induced metabolic syndrome.* Metabolism, 2006. **55**(7): p. 928-34.

6. Velasquez, M.T., et al., *Diabetic glomerulopathy in the SHR/N-corpulent rat: role of dietary carbohydrate in a model of NIDDM.* 1995. **38**(1): p. 31-38.

7. Hu, G., et al., *Chronic exercise provides renal-protective effects with upregulation of fatty acid oxidation in the kidney of high fructose-fed rats.* American Journal of Physiology-Renal Physiology, 2020. **318**(3): p. F826-F834.

8. Muller, C.R., et al., *Aerobic exercise training prevents kidney lipid deposition in mice fed a cafeteria diet.* Life Sciences, 2018. **211**: p. 140-146.

9. Preuss, H., et al., *Sugar-Induced Blood Pressure Elevations Over the Lifespan of Three Substrains of Wistar Rats.* Journal of the American College of Nutrition, 1998. **17**: p. 36-47.

10. Gluckman, P.D., M.A. Hanson, and A.S. Beedle, *Non-genomic transgenerational inheritance of disease risk.* Bioessays, 2007. **29**(2): p. 145-54.

11. Inadera, H., *Developmental origins of obesity and type 2 diabetes: molecular aspects and role of chemicals.* Environmental Health and Preventive Medicine, 2013. **18**(3): p. 185-197.

12. Bremer, A.A., M. Mietus-Snyder, and R.H. Lustig, *Toward a Unifying Hypothesis of Metabolic Syndrome.* PEDIATRICS, 2012. **129**(3): p. 557-570.

13. Barbosa De Queiroz, K., et al., *Physical activity prevents alterations in mitochondrial ultrastructure and glucometabolic parameters in a high-sugar diet model.* PLOS ONE, 2017. **12**(2): p. e0172103.

14. Seo, T.B., et al., *Involvement of Cdc2 in axonal regeneration enhanced by exercise training in rats.* Medicine and science in sports and exercise, 2006. **38**(7): p. 1267-1276.

15. Botezelli, J.D., et al., *Exercise counteracts fatty liver disease in rats fed on fructose-rich diet.* Lipids Health Dis, 2010. **9**: p. 116.

16. Casimiro-Lopes, G., et al., *Maternal prolactin inhibition during lactation affects physical performance evaluated by acute exhaustive swimming exercise in adult rat offspring.* Horm Metab Res, 2012. **44**(2): p. 123-9.

17. Bernardis, L.L. and B.D. Patterson, *Correlation between 'Lee index' and carcass fat content in weanling and adult female rats with hypothalamic lesions.* J Endocrinol, 1968. **40**(4): p. 527-8.

18. Carroll, J.F., W.J. Zenebe, and T.B. Strange, *Cardiovascular function in a rat model of diet-induced obesity.* Hypertension, 2006. **48**(1): p. 65-72.

19. Oliveira, M.C., et al., *Acute and sustained inflammation and metabolic dysfunction induced by high refined carbohydrate-containing diet in mice.* Obesity (Silver Spring), 2013. **21**(9): p. E396-406.

20. De Queiroz, K.B., et al., *Endurance Training Increases Leptin Expression in the Retroperitoneal Adipose Tissue of Rats Fed with a High-Sugar Diet.* 2014. **49**(1): p. 85-96.

21. Totou, N.L., et al., *Swimming exercise demonstrates advantages over running exercise in reducing proteinuria and glomerulosclerosis in spontaneously hypertensive rats.* Physiol Int, 2018. **105**(1): p. 76-85.

22. Souza, J.A., et al., *Swimming training improves cardiovascular autonomic dysfunctions and prevents renal damage in rats fed a high-sodium diet from weaning.* Experimental Physiology, 2020. **n/a**(n/a).

23. Molinaro, E.M., L.F.G. Caputo, and M.R.R. Amendoeira, *Conceitos e métodos para a formação de profissionais em laboratórios de saúde.* Vol. 2. 2010.

24. Sam Lee, J., et al., *Interaction of Diet and Training on Endurance Performance in Rats.* Experimental Physiology, 2001. **86**(4): p. 499-508.

25. De Queiroz, K.B., et al., *Endurance training blocks uncoupling protein 1 up-regulation in brown adipose tissue while increasing uncoupling protein 3 in the muscle tissue of rats fed with a high-sugar diet.* Nutrition Research, 2012. **32**(9): p. 709-717.

26. de Queiroz, K.B., et al., *Molecular mechanism driving retroperitoneal adipocyte hypertrophy and hyperplasia in response to a high-sugar diet.* Mol Nutr Food Res, 2014. **58**(12): p. 2331-41.

27. Oliveira, D.T.D., et al., *High-sugar diet leads to obesity and metabolic diseases in ad libitum -fed rats irrespective of caloric intake.* Archives of Endocrinology and Metabolism, 2020. **64**(1): p. 71-81.

28. Malafaia, A.B., et al., *Indução de obesidade com sacarose em ratos.* ABCD. Arquivos Brasileiros de Cirurgia Digestiva (São Paulo), 2013. **26**(suppl 1): p. 17-21.

29. Castellanos Jankiewicz, A.K., et al. *Adipose tissue redistribution caused by an early consumption of a high sucrose diet in a rat model.* Nutricion hospitalaria, 2015. **31**, 2546-2553 DOI: 10.3305/nh.2015.31.6.8935.

30. Carmo, M., et al., *Swimming Exercise Did Not Ameliorate the Adverse Effects of High-Sugar Diet in Young Rats.* Journal of Exercise Physiologyonline, 2017. **20**: p. 177-183.

31. Borer, K.T., *Exercise Endocrinology.* 2nd Edition ed. 2003. 273.

32. Wing, R.R. and J.O. Hill, *Successful weight loss maintenance.* Annu Rev Nutr, 2001. **21**(1): p. 323-41.

33. Zhang, N. and S. Bi, *Effects of physical exercise on food intake and body weight: Role of dorsomedial hypothalamic signaling.* Physiol Behav, 2018. **192**: p. 59-63.

34. Preuss, H.G., et al., *Effects of high sugar diets on renal fluid, electrolyte and mineral handling in rats: relationship to blood pressure.* Journal of the American College of Nutrition, 1994. **13**(1): p. 73-82.

35. Francisqueti, F., et al., *Effect of Gamma-Oryzanol as Therapeutic Agent to Prevent Cardiorenal Metabolic Syndrome in Animals Submitted to High Sugar-Fat Diet.* Nutrients, 2017. **9**(12): p. 1299.

36. El-Bassossy, H.M. and H.A. Shaltout, *Allopurinol alleviates hypertension and proteinuria in high fructose, high salt and high fat induced model of metabolic syndrome.* Transl Res, 2015. **165**(5): p. 621-30.

37. Roysommuti, S., et al., *Excess dietary glucose alters renal function before increasing arterial pressure and inducing insulin resistance.* American Journal of Hypertension, 2002. **15**(9): p. 773-779.

38. Fan, C.-Y., et al., *Betaine supplementation protects against high-fructose-induced renal injury in rats.* The Journal of Nutritional Biochemistry, 2014. **25**(3): p. 353-362.

39. Cao, G., et al., *Beneficial Effect of Moderate Exercise in Kidney of Rat after Chronic Consumption of Cola Drinks.* PLOS ONE, 2016. **11**(3): p. e0152461.

40. Guyton, A.C., *Textbook of medical physiology*, Elsevier, Editor. 2006. p. 181-194.
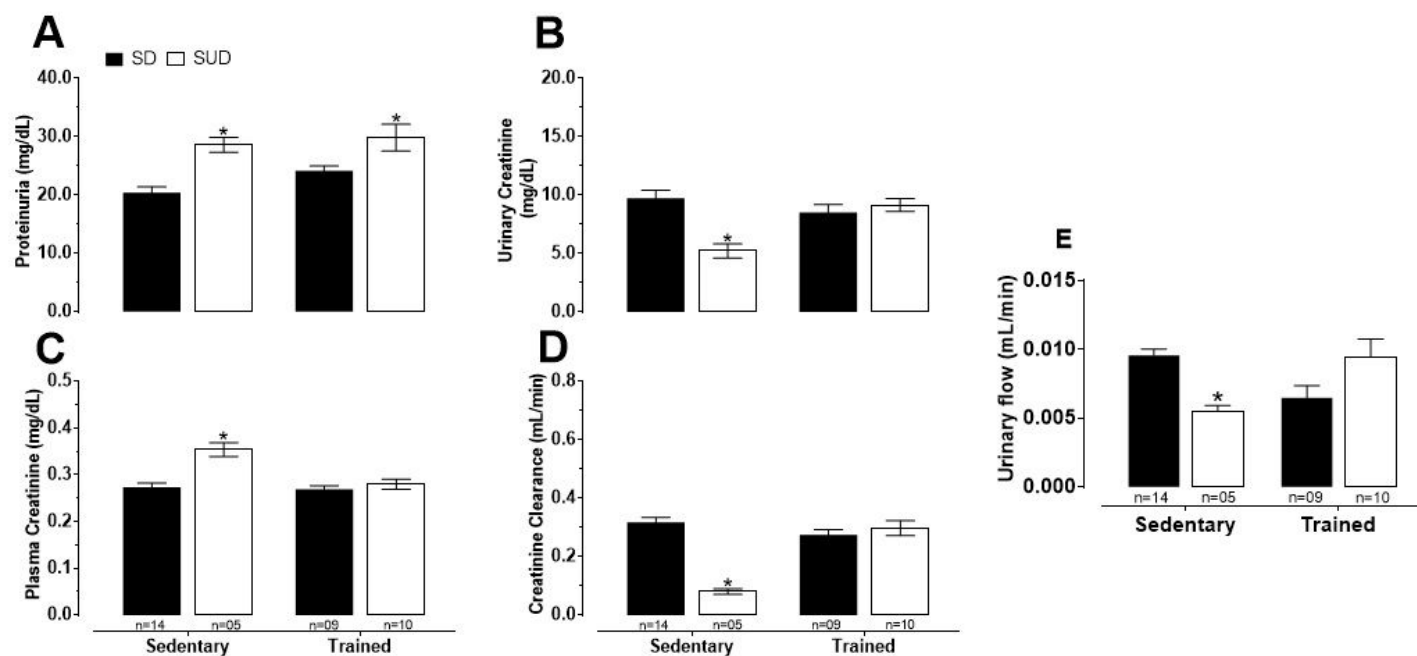
Figures

Figure 1

Proteinuria values (panel A), *different from S-SD group. Urinary creatinine values (panel B), *different from S-SD, T-SD and T-SUD groups. Plasma creatinine values (C), *different from S-SD, T-SD and T-SUD groups. Creatinine clearance values (panel D), *different from S-SD, T-SD and T-SUD groups. Urinary flow (panel E), *different from S-SD group. All measurements were realized after 8th week of T. It was used the parametric test, two-way ANOVA followed by Tukey's post-test; $p < 0.05$. The bars represent group mean data. Differences among the pairs of means are indicated by different signs.
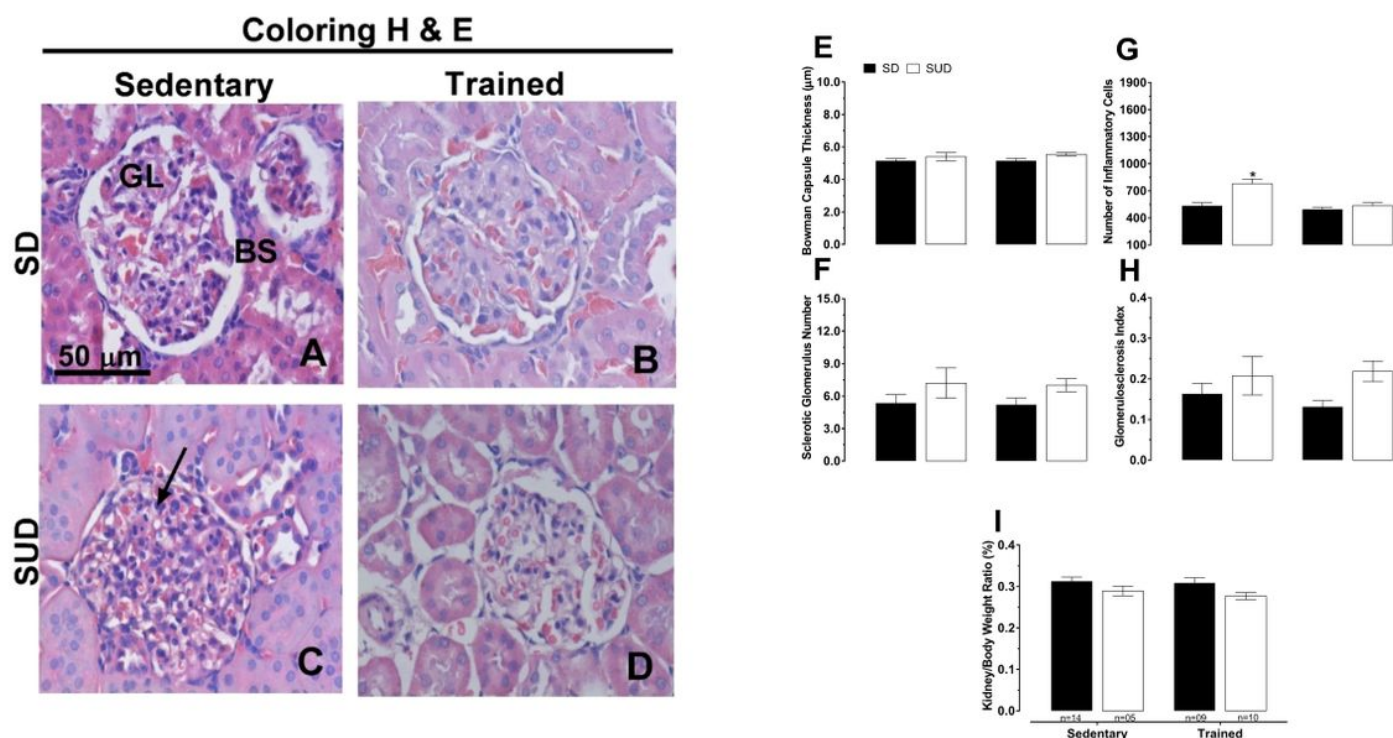
Figure 2

Histological sections stained with hematoxylin and eosin (panels A to D). Bowman Capsule Thickness (panel E), there was no difference among the groups. Sclerotic glomerulus number (panel F), there was no difference among the groups. Number of inflammatory cells values (panel G), *different from S-SD, T-SD and T-SUD. Glomerulosclerosis index (H), there was no difference among the groups. Kidney/body weight ratio values (panel I), there was no difference among the groups. GL – glomeruli, BS – Bowman Space, SD (Standart Diet), SUD (Sucrose Diet). All measurements were realized after 8th week of T. It was used the parametric test, two-way ANOVA followed by Tukey's post-test; $p < 0.05$. The bars represent group mean data. Differences among the pairs of means are indicated by different signs.

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