Swimming Exercise Did Not Ameliorate the Adverse Effects of High-Sugar Diet in Young Rats

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ABSTRACT

Carmo MAV, Pinto ABG, Queiroz KB, Sá RG, Silva ME, Lima WG, Oliveira EC, Becker LK. Swimming Exercise Did Not Ameliorate the Adverse Effects of High-Sugar Diet in Young Rats. JEPonline 2017;20(3):177-183. The purpose of this study was to investigate the effects of a swimming exercise (SE) in young rats fed with a high-sugar diet (HSD). Rats fed with a standard diet or a HSD were simultaneously subjected to SE or not (sedentary group) for 8 wks. The results showed that the HSD decreased calorie intake and body weight, but increased body adiposity index, total cholesterol, HDL-cholesterol, and LDL-cholesterol. The SE reduced the total cholesterol and LDL-cholesterol. Interaction between HSD and SE was detected based on body adiposity index measurements, on VLDL-cholesterol and triglycerides levels, and on citrate synthase activity. Blood pressure and heart rate did not change significantly among the different groups. The HSD negatively affected the majority of the biological parameters while the SE improved body adiposity index and the levels of some lipoproteins. In conclusion, the SE did not attenuate the adverse effects of HSD in young rats.

Key Words: Blood Pressure, High-Sugar Diet, Swimming Exercise, Young Rats
INTRODUCTION

Children and adolescents often consume high-sugar foods (cakes, cookies, etc.). Added sugars are consumed very frequently; the most common sources of added sugars in the United States are soft drinks (14). Sucrose is widely used in processed foods, and its high usage can be a causative factor for increased blood pressure. This hypothesis is supported by meta-analyses of randomized controlled trials suggesting that sugar is strongly related to the increase in blood pressure in humans (23).

Regular physical exercise is beneficial to one’s health because it helps to prevent obesity, hypertension, dyslipidemia, and cardiovascular complications (17,21), and it reduces blood pressure (18). Several diet models (high-fat, high-sugar, or a combination of each) highlight the important role of exercise in reducing their side effects. Exercise has been shown to reduce the accumulation of fat in the liver in sucrose-enriched choline-deficient diet (non-alcoholic fatty liver disease) (1). Additionally, exercise restored pancreatic function and autonomic nervous system activity in obese rats, which were reduced by a high-fat diet (13). The combination of high-sugar consumption and regular exercise attenuates hypertension in rats (20).

It is crucial to start exercising at an early age to avoid the deleterious metabolic effects induced by different diets. Low-intensity swimming exercise after weaning considerably inhibits the obesity induced by neonatal treatment with monosodium L-glutamate in mice. Exercise helps reduce hyperglycemia, maintains insulin sensitivity, and protects against deterioration effects in tissues (2). Voluntary post-weaning exercise completely reversed the metabolic effects of maternal obesity in chow-fed offspring, by reducing adiposity, plasma leptin, and triglyceride levels (19).

Since children are more physically active by nature, it is commonly thought that the adverse effects associated with the ingestion of high-sugar foods would be attenuated. However, there are few studies focusing on the possible association of high-sugar diets with physical exercise at a young age. Therefore, this study aims to evaluate the effect of regular physical exercise on biological parameters of young rats fed with a HSD.

METHODS

Animals
Wistar rats (21 days old) were randomly divided into four groups: sedentary and exercised rats fed with a standard chow diet (S-STD, n = 10), (E-STD n = 10); sedentary and exercised rats fed with HSD (S-HSD n = 10), (E-HSD n = 10). The Ethics Committee for Animal Use of the Federal University of Ouro Preto approved the study (25/2013).

Diet and Nutritional Parameters
The animals were fed a HSD (68% carbohydrates) that consisted of 33% standard chow (Nuvilab, Curitiba, Brazil), 33% condensed milk, and 7% sucrose by weight (the remainder was water) (9). The control groups were fed with a standard chow (STD). The composition of each diet has been previously published (11). Rats had their calorie intake and body weight measured once a week during the experimental period. The weekly food intake was multiplied by the energy density for the STD (12.22 kJ/g) and the HSD (13.31 kJ/g), to
calculate the energy intake. The Lee index was used to evaluate the development of obesity. It was calculated as cubic root of body weight (g) divided by the naso-anal length in millimeters × 10 (15).

**Exercise**
The exercised rats swam in collective tanks containing water at 31 ± 1 °C, 1-hr·d⁻¹ and 5 d·wk⁻¹. For the 1st 2 wks, the rats swam without a workload. During the 3rd and 4th wks, the rats swam supporting a lead weight tied to the tail base that created a workload of 2% of the rat’s body weight, well above the aerobic/anaerobic metabolic transition that occurs at a workload of approximately 5% of the rat’s body weight (3). During the 5th wk and until the end of the experiment, the workload was weekly adjusted to 3% of the rat’s body weight, still above the aerobic/anaerobic metabolic transition.

**Arterial Pressure Measurements**
Blood pressure and heart rate were measured in awake rats 48 hrs after the last exercise section and 12 hrs after their last meal using a pressure transducer coupled to an acquisition system (PowerLab System; ADInstruments, Dunedin, New Zealand). The data were analyzed by LabChart 7 software for Windows (16).

**Statistical Analyses**
Statistical analyses were performed using Graph Pad Prism. The KS test was used to verify the normality of the data, which were expressed as mean ± standard deviation. We also used analysis of variance (two-way ANOVA). P values ≤0.05 were considered statistically significant.

**RESULTS**

**Main Effects of Diet or Exercise**
The HSD induced lower calorie intake and reduced body weight. However, body adiposity index, total cholesterol, HDL and LDL levels were increased. Exercise reduced total cholesterol and LDL levels (Table 1).

**Table 1. Parameters Measured for Rats Fed with an STD or an HSD Subjected to Swimming Exercise or Not for an 8-Wk Period.**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>S-STD</th>
<th>E-STD</th>
<th>S-HSD</th>
<th>E-HSD</th>
<th>Two-Way ANOVA (P values)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Diet</td>
<td>Training</td>
<td>Interaction</td>
</tr>
<tr>
<td>Calorie intake (kJ)</td>
<td>367.10 ± 58.03</td>
<td>352.83 ± 56.73</td>
<td>265.05 ± 44.68</td>
<td>264.51 ± 38.39</td>
<td>p = 0.0001</td>
</tr>
<tr>
<td>Body weight (g)</td>
<td>329.90 ± 44.08</td>
<td>323.00 ± 31.08</td>
<td>270.10 ± 17.27</td>
<td>267.00 ± 21.74</td>
<td>p &lt; 0.0001</td>
</tr>
<tr>
<td>Body Adiposity index</td>
<td>1.01 ± 0.22</td>
<td>0.90 ± 1.15</td>
<td>1.70 ± 0.44</td>
<td>1.41 ± 0.22</td>
<td>p &lt; 0.0001</td>
</tr>
<tr>
<td>Total cholesterol (mg·dL⁻¹)</td>
<td>76.89 ± 9.76</td>
<td>65.51 ± 9.96</td>
<td>111.90 ± 15.98</td>
<td>91.12 ± 20.22</td>
<td>p &lt; 0.0001</td>
</tr>
<tr>
<td>HDL cholesterol (mg·dL⁻¹)</td>
<td>22.43 ± 2.80</td>
<td>21.75 ± 1.47</td>
<td>32.17 ± 11.24</td>
<td>29.19 ± 8.10</td>
<td>p = 0.0029</td>
</tr>
<tr>
<td>LDL cholesterol (mg·dL⁻¹)</td>
<td>46.47 ± 13.00</td>
<td>36.35 ± 5.11</td>
<td>64.19 ± 10.48</td>
<td>43.63 ± 10.24</td>
<td>p = 0.0020</td>
</tr>
</tbody>
</table>

Data expressed as means ± SD; Statistical differences were determined using a two-way ANOVA to examine the effects of diet and/or training and/or interaction, followed by Bonferroni post hoc test; S-STD = sedentary + standard diet; E-STD = exercised + standard diet; S-HSD = sedentary + high-sugar diet; E-HSD = exercised + high-sugar diet.
Interactions Between Diet and Exercise
The interaction between diet and exercise for the Lee index (adiposity index) contributed to equalizing E-HSD to the control group (S-STD) (Figure 1). The HSD increased the VLDL cholesterol levels while exercise had the opposite effect. Therefore, the interaction between the HSD diet and exercise equalized E-HSD to the control group (S-STD) for VLDL values (Figure 1). Diet and exercise interaction did not affect triglycerides levels (Figure 1) because HSD blocked the exercise-induced decrease in triglycerides levels. Citrate synthase activity in the E-HSD group was increased compared to that in the control group (S-STD) (Figure 1), which indicates that the HSD did not prevent the adaptation to physical training.

Figure 1. Interaction of Diet and Exercise Showed by Two Way ANOVA for (1) Lee Index (P = 0.0119), (2) VLDL cholesterol (P = 0.0493), (3) Triglycerides (P = 0.0132) and (4) Citrate Synthase (P = 0.0069) are Represented by Different Letters According to Bonferroni post hoc Test.

Systolic, Diastolic Blood Pressure, and Heart Rate
There were no statistically significant differences in systolic blood pressure (S-HSD = 120.9 ± 16.8 mmHg; E-HSD = 107.5 ± 9.5 mmHg), diastolic blood pressure (S-HSD = 97.1 ± 16.3 mmHg; E-HSD 97.0 ± 11.5 mmHg), and heart rate (S-HSD 394 ± 33.1 beats·min⁻¹; E-HSD 369 ± 43.6 beats·min⁻¹).

DISCUSSION

Animals fed with the HSD did not develop high blood pressure or changes in heart rate. Some studies have reported a positive correlation between sugar consumption and the increase in arterial pressure (4,5) while others found no correlation (8). It is noteworthy that rats used in the present study were young. It is possible that this intervention period of 8 wks was not long enough to induce an alteration in blood pressure.
Studies that provided the HSD to weaning rats observed that the alterations in weight gain were delayed, thus suggest a perturbation in animal growth (9). Previous findings from our group indicate that HSD induced elevated adiposity indices and hyperplasia of brown adipose tissue indicates that dietary stimulus can effectively induce obesity (11). However, consistent with other studies, the S-HSD and E-HSD rats showed high body adiposity index (6,11). Hypertriglyceridemia was observed in animals that received a diet rich in simple carbohydrates owing to hepatic lipogenesis and hepatic VLDL secretion, resulting in elevated serum triglycerides levels (6,10,21).

The lower food intake of the groups that received the HSD may be associated with an increase in plasma levels of leptin (10,21). Leptin is a hormone secreted by adipocytes, and its circulating levels are correlated with the amount of fat stored in the body. Leptin levels increase when an individual adds body fat and, together with other hormones, leptin regulates appetite control (6,12). Physical training had no effect in weight, calorie intake, and Lee index in animals that received HSD. Botezelli et al. (3) also studied the combinatorial effect of the HSD and exercise and showed that exercise did not affect body weight in young rats.

The combination of the HSD and exercise improved markers of non-alcoholic fatty liver disease, indicating the beneficial role of exercise (3). Low intensity exercise was not effective in preventing abdominal adiposity or the increase in plasma and liver triglycerides associated with sucrose consumption (7). On the contrary, studies using high-intensity and high frequency training for rats fed with the HSD showed improvement in cholesterol and glucose homeostasis (3,20). In the current study, exercise training was of submaximal intensity.

The activity of mitochondrial enzymes was used to confirm training-induced elevation of the oxidative capacity of skeletal muscles (22). Regardless of the diet followed, physical exercise on a treadmill improved the oxidative capacity of trained animals when compared with their respective controls (11). In the current study, we observed an increase in the citrate synthase activity in both HSD- and STD-fed trained groups.

CONCLUSIONS

The HSD negatively affected the majority of the biological parameters, except for blood pressure and heart rate. Also, while exercise improved the body adiposity index and the levels of some lipoproteins, it did not prevent the side effects caused by the HSD in young rats.

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