Effect of Exercise on Inflammatory Cytokines, Lipid Profile and Glycemic Control in Normal and Obese Rats

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Abstract

Background: Obesity is becoming more and more linked to a subacute inflammatory state that plays a key role in the pathogenesis of many diseases. Different types of physical activities are believed to affect both obesity and inflammatory conditions. The purpose of this study was to investigate the possible role of acute and chronic swimming training on plasma levels of pro/anti-inflammatory cytokines.

Methods: Wistar rats were divided into two main groups: Normal Weight control group (NW), and Obese group (OB). Each of the two groups was further subdivided into three subgroups (Sedentary, S; Acute Exercise, AE and Chronic Exercise, CE). Acute exercise rats performed a single bout of exercise while chronic exercise rats performed swimming exercise for one hour/day, 5 days/week for 8 weeks. At the end of the study period, all animals were decapitated to obtain blood samples. Blood samples were tested for insulin, glucose, Total Cholesterol (TC), High-Density Lipoprotein (HDL), Triglycerides (TGs), Interleukin-6 (IL-6), Interleukin-10 (IL-10), Tumor Necrosis Factor-α (TNF-α), C-Reactive Protein (CRP), and adiponectin levels. Also, Homeostatic Models for Insulin Resistance (HOMA-IR) and B cell function (HOMA-B) were calculated.

Results: Single session of acute exercise in normal-weight animals significantly increased IL-6 and IL-10 levels with no effect on other parameters. While in obese animals it induced a significant reduction in insulin, glucose, HOMA-IR, levels and a significant increase in adiponectin levels.

Chronic exercise for 8 weeks in normal weight animals significantly decreased insulin, HOMA-B, HOMA-IR, TNF-α and significantly increased IL-6, IL-10, and adiponectin. While in obese animals it induced a significant reduction in all glycemic and lipid profiles. Moreover, it significantly decreased IL-6, TNF-α, and significantly increased adiponectin levels.

Conclusion: Chronic and acute exercise has a significant effect on inflammatory mediators and this effect is more profound with chronic exercise and in obese more than normal weight animals.

Key Words: Obesity – Exercise – Inflammatory cytokines.

Introduction

OVERWEIGHT or obesity, when accompanied with a sedentary lifestyle may cause or at least predisposes to as much as 80% of common chronic diseases, such as diabetes mellitus, cancer and several cardiovascular diseases [1]. Obesity is a risk factor for cardiovascular disease and is associated with an increased risk of morbidity and reduced life expectancy [2]. In addition, obesity drives diabetic and vascular mortality [3]. There is a remarkably high increase in the incidence of the metabolic syndrome. The concept “metabolic syndrome refers to a combination of disorders including glucose intolerance, central obesity, dyslipidemia and hypertension [4].

Reports investigating various markers of inflammation in different population groups have confirmed an association between low-grade systemic inflammation on one hand and the metabolic syndrome, type 2 diabetes, and atherosclerosis [5].

Moreover, there is growing evidence that adipose tissue plays a strong integral role in the development of cardio-metabolic disease. Chronic low-grade inflammation could be the missing link between adipose tissue, metabolic syndrome and disease outcomes [7].

Inflammatory cytokines are classified into proinflammatory as tumor necrosis factor-α (TNF-α) and C-Reactive Protein (CRP), anti-inflammatory as adiponectin and IL-10 (IL-10), while IL-6 is classified as both pro and anti-inflammatory. Changes in the levels of this pro and anti-inflammatory cytokines usually accompany the metabolic syndrome [8].
Obesity is usually accompanied by endothelial dysfunction which results in vascular wall damage, accumulation of phagocytes in the subintimal space and a subsequent release of inflammatory cytokines [9]. Peripheral blood mononuclear cells also demonstrate increased inflammatory cytokine expression in the presence of obesity [10].

Regular physical exercise has been reported to be an effective method for the treatment of some chronic diseases including obesity [11], type 2 diabetes [12] and ischemic heart disease [13]. Physical exercise can be regarded as a form of physical stress. Other types of clinical physical stressors such as trauma, burn, and sepsis can induce a pattern of hormonal and immunological responses that have similarities to that of exercise. Moreover, it has been reported that exercise induces an acute phase response, which has some similarities to the acute phase response to sepsis and trauma [14].

However, the effect of exercise on the circulating levels of inflammatory cytokines is still controversial. Some studies reported that exercise induced a significant increase in adiponectin, and a significant decrease in CRP [15] a significant decrease in CRP, IL6 and IL8 [16]. In contrast, other studies reported that exercise did not result in any significant changes in these inflammatory cytokines [17,18].

This study was designed to demonstrate the effects of acute and chronic swimming exercise on glycemic control, lipid profile and some inflammatory cytokines levels (adiponectin, C-reactive protein, Interleukin-6, IL-10 and tumor necrosis factor-a) in normal weight and high-fat-diet-induced obese rats.

Material and Methods

Experimental animals:

In the period from 15th April 2016 to 21 July 2016, this study was performed on 48 male albino rats in the Faculty of Medicine, Zagazig University. Rats were 12-15 weeks old weighing 200-250gm. And were obtained from the Animal House of Faculty of Veterinary Medicine, Zagazig University. Animals were kept in clean wire cages at a comfortable temperature with 12-hour light/dark cycles and with free access to water.

Diet and induction of obesity:

After a two weeks’ adaptation period the animals were randomized into two main groups: Standard diet group (NW, n=24) and high-fat diet group (OB, n=24). Each main group was further divided into three equal subgroups.

Group I: Normal Weight (NW): Normal weight animals were fed standard commercial rat chow for four weeks. This group was further subdivided into three subgroups:
- Group IA (N=8): Normal Weight Sedentary group (NWS).
- Group IB (N=8): Normal Weight Acute Exercise (NWAE).
- Group IC (N=8): Normal Weight Chronic Exercise (NWCE).

Group II: Obese (OB): Obese animals were fed specially formulated highly palatable High Fat Diet (HFD) to induce obesity (HFD consisted of (by weight) 33% standard rat chow, 33% Nestle® condensed milk, 7% sucrose, and 27% water, for four weeks [19]. This group was further subdivided into three subgroups:
- Group IIA (N=8): Obese Sedentary group (OBDS).
- Group IIB (N=8): Obese acute exercise (OBAE).
- Group IIC (N=8): Obese Chronic Exercise (OBCE).

Verifying obesity:

At the end of the experimental period, Body Mass Index (BMI) for each animal was calculated by the equation: BMI=Body weight (gm)/Naso-Anal length$^2$ (cm$^2$). This index can be used as an indicator of obesity where rats can be considered obese if BMI is more than 0.68gm/cm$^2$ [20].

Rats in HFD fed group that did not gain weight after 4 weeks of the start of HFD were considered resistant and excluded.

Swimming exercise:

Acute exercise: Swimming was performed plastic pool (90 X 45 X 45cm) filled with water maintained at a temperature of 36±2ºC. The water depth, 35cm, was set so that the rats could not rest by supporting their tails on the bottom of the pool. Each rat had a weight attached (5% body weight) to its tail for the duration of the swim. This weight is supposed to give the Maximal Lactate Steady State level (MLSS), (around the maximal aerobic level) [21]. The exercise was a one (lasting 30 minutes) acute swimming exercise. Experimental animals were made to swim at the end of the study and before decapitation.

Chronic exercise: In these groups, animals swam for one hour each day for five days a week for four weeks. The swimming period started for 30 minutes then it was increased gradually. Each
rat had a weight attached (5% body weight) to its tail for the duration of the swim [21].

**Sedentary groups:** Rats in non-exercising groups (Ia and Iia) were not subjected to swimming exercise protocol, but were placed in shallow water during the period of exercise in the chronic exercise groups [22].

**Biochemical analysis:**

**Sampling of blood:**

At the end of the study protocol, all animals were anesthetized, sacrificed by decapitation and blood was obtained, centrifuged at 3000rpm for 10 minutes. The serum was separated then stored at –20°C in dark containers till the time of analysis.

**Insulin and glucose:**

Glucose and insulin concentrations were measured by commercial assay kits (BioSource Europe S.A., Belgium).

**Lipid profile:** Total cholesterol, High-Density Lipoprotein, (HDL), and triglycerides were measured using commercial kits (BioSource Europe S.A., Belgium). Low-Density Lipoprotein, (LDL) and Very Density Lipoprotein, (VLDL) were calculated with the Friedewald equation [23].

**Adipokines:**

Adiponectin, IL-6, IL-10, TNF-α, and CRP levels were measured using commercial kits (Sigma-Aldrich, St. Louis MO, USA).

**Insulin sensitivity:**

Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) index was calculated as fasting insulin (mU/L) X fasting glucose (mmol/L)/22.5 [24].

\[ \text{HOMA-IR} = \frac{\text{Insulin} \times \text{Glucose}}{22.5} \]

Homeostatic Model Assessment of beta cell function (HOMA-β) index, HOMA-β was calculated as (insulin (mU/L) X 20/fasting glucose (mmol/L) – 3.5) X 100 [24].

\[ \text{HOMA-β} = \frac{\text{Insulin} \times 20}{\text{Fasting glucose} – 3.5} \%

**Statistical analyses:**

Results are presented as a mean ± standard deviation. Statistical analysis was performed using the IBM-Statistical Package for the Social Sciences (IBM-SPSS), Version 22 (SPSS Inc., Chicago, IL, United States). Means were compared by one-way Analysis of Variance (ANOVA) followed by the LSD post hoc test. \( p \)-values <0.05 were considered statistically significant.

**Results**

**Glycemic control parameters:**

The effects of acute and chronic exercise on body weight, insulin level, blood glucose level, Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) and beta cell function (HOMA-B) are shown in (Table 1).

In normal weight groups, it was found that single session of acute exercise (NWAE) had no significant effect on serum insulin level, blood glucose level, homeostatic model of insulin resistance and beta cell function when compared to control normal weight group (NWS). However chronic exercise for 8 weeks (NWCE) induced a significant reduction in serum insulin \((p<0.001)\), HOMA-IR \((p<0.020)\) and HOMA-B \((p<0.018)\) but this was not accompanied by significant changes in blood glucose level.

In obese groups, the final body weight was significantly higher than their matching normal weight groups \((p<0.001)\). Also, all obese groups showed a significant increase in serum insulin levels, blood glucose levels, HOMA-IR and HOMA-B when compared to their matching normal weight groups. Moreover, a single session of acute exercise in obese rats (OBAE) induced a significant reduction in serum insulin level \((p<0.001)\), blood glucose levels \((p<0.004)\), HOMA-IR \((p<0.001)\) when compared to control obese group (OBS). Chronic exercise for 8 weeks (OBCE) induced a significant reduction in serum insulin \((p<0.001)\), blood glucose level \((p<0.001)\) HOMA-IR \((p<0.001)\) and HOMA-B \((p<0.001)\).

**Lipid profile:**

The effects of acute and chronic exercise on Total Cholesterol (TC), High-Density Lipoprotein (HDL), Low-Density Lipoprotein (LDL), Very Low-Density Lipoprotein (VLDL) and Triglycerides (TGs) in blood are shown in (Table 2).

In normal weight groups, it was found that both single session of acute exercise (NWAE) and chronic exercise for 8 weeks (NWCE) had no significant effect on serum levels of Total Cholesterol (TC), High-Density Lipoprotein (HDL), Low-Density Lipoprotein (LDL), Very Low Density Lipoprotein (VLDL) and Triglycerides (TGs) when compared to normal weight control group (NWS).
In obese groups, it was found that single session of acute exercise (OBAE) had no significant effect on serum levels of Total Cholesterol (TC), High-Density Lipoprotein (HDL), Low-Density Lipoprotein (LDL), Very Low Density Lipoprotein (VLDL) and Triglycerides (TGs) when compared to control normal weight group (OBS). However, chronic exercise for 8 weeks (OBCE) induced a significant reduction in serum levels of Total Cholesterol (TC) \((p<0.001)\), High-Density Lipoprotein (HDL) \((p<0.002)\), Low-Density Lipoprotein (LDL) \((p<0.001)\), Very Low Density Lipoprotein (VLDL) \((p<0.001)\) and Triglycerides (TGs) \((p<0.001)\) when compared obese control group (OBS).

Adipokine levels:

The effects of acute and chronic exercise on serum adiponectin, C-Reactive Protein (CRP), Interleukin-6 (IL-6), IL-10 and Tumor Necrosis Factor Alpha (TNFα) are shown in (Table 3).

In normal weight groups, it was found that single session of acute exercise (NWAIE) induced a significant increase in IL-6 \((p<0.001)\) and IL-10 \((p<0.001)\) while, it had no significant effect on adiponectin, C-Reactive Protein (CRP) and Tumor Necrosis Factor Alpha (TNFα) levels when compared with control sedentary group. On the other hand, chronic exercise for 8 weeks (NWCE) induced a significant increase in adiponectin level \((p<0.001)\), a significant reduction in, but no significant effects were found as regard the levels of IL-6, TNFα, and CRP.

In Obese Groups, it was found that single session of acute exercise (NWAIE) induced a significant increase in adiponectin \((p<0.001)\), IL-6 \((p<0.001)\) and IL-10 \((p<0.001)\) levels, a significant reduction TNFα levels \((p<0.029)\), while it has no significant effects on CRP levels when compared with obese control group. Chronic exercise for 8 weeks (OBCE) induced a significant increase in adiponectin \((p<0.001)\) and IL-10 \((p<0.001)\) levels, a significant reduction in IL-6 \((p<0.001)\), and TNFα \((p<0.001)\) levels, while it has no significant effects on CRP level when compared with obese control group.

### Table (1): Effect of acute and chronic exercise on body weight, insulin, and glucose levels and homeostatic models of beta cell functions and insulin sensitivity in normal weight and obese rats.

<table>
<thead>
<tr>
<th></th>
<th>Initial weight (g)</th>
<th>Final weight (g)</th>
<th>Insulin (mU/L)</th>
<th>Glucose (mmol/L)</th>
<th>HOMA-B</th>
<th>HOMA-IR</th>
</tr>
</thead>
<tbody>
<tr>
<td>NWS</td>
<td>227.13±15.870</td>
<td>244.00±12.762</td>
<td>8.19±0.342</td>
<td>5.84±0.205</td>
<td>70.59±8.684</td>
<td>2.12±0.078</td>
</tr>
<tr>
<td>NWAIE</td>
<td>225.38±16.097</td>
<td>253.00±18.807</td>
<td>6.61±0.690</td>
<td>4.91±0.353</td>
<td>98.36±23.905</td>
<td>1.45±0.217</td>
</tr>
<tr>
<td>NWCE</td>
<td>230.13±11.457</td>
<td>240.10±29.964</td>
<td>3.80±0.598</td>
<td>5.47±0.373</td>
<td>39.39±6.944</td>
<td>0.93±0.184</td>
</tr>
<tr>
<td>OBDS</td>
<td>230.00±19.413</td>
<td>311.40±29.233*</td>
<td>21.46±3.792*</td>
<td>7.26±1.029*</td>
<td>119.21±31.241</td>
<td>7.02±0.088*</td>
</tr>
<tr>
<td>OBAE</td>
<td>231.88±17.635</td>
<td>318.00±30.776*</td>
<td>12.59±2.899*</td>
<td>6.25±0.943*</td>
<td>101.10±41.250</td>
<td>3.53±1.204*</td>
</tr>
<tr>
<td>OBCE</td>
<td>233.38±12.501</td>
<td>296.80±26.462*</td>
<td>6.30±0.835*</td>
<td>5.49±0.639*</td>
<td>69.02±21.820</td>
<td>1.52±0.178*</td>
</tr>
</tbody>
</table>

Data expressed as mean ± SD. NB: Normal Weight Sedentary group. OBAE: Obese Acute Exercise group.

### Table (2): Effect of acute and chronic exercise on Total Cholesterol(TC), High-Density Lipoproteins (HDL), Low-Density Lipoproteins (LDL), Very Low-Density Lipoproteins (VLDL) and triglycerides in normal weight and obese rats.

<table>
<thead>
<tr>
<th></th>
<th>TC (mg/dl)</th>
<th>HDL (mg/dl)</th>
<th>LDL (mg/dl)</th>
<th>VLDL (mg/dl)</th>
<th>TGs (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NWS</td>
<td>58.50±8.124</td>
<td>33.13±5.194</td>
<td>14.30±5.664</td>
<td>11.07±1.406</td>
<td>55.38±7.029</td>
</tr>
<tr>
<td>NWAIE</td>
<td>54.75±5.676</td>
<td>32.38±4.658</td>
<td>12.13±4.833</td>
<td>10.25±1.696</td>
<td>51.25±8.481</td>
</tr>
<tr>
<td>OBDS</td>
<td>143.21±27.826*</td>
<td>69.72±16.945*</td>
<td>49.57±13.603*</td>
<td>23.92±3.187*</td>
<td>119.59±15.936*</td>
</tr>
<tr>
<td>OBAE</td>
<td>138.63±25.483*</td>
<td>70.46±10.801*</td>
<td>45.90±15.562*</td>
<td>22.72±3.557*</td>
<td>111.33±17.787*</td>
</tr>
</tbody>
</table>

Data expressed as mean ± SD. NB: Normal Weight Sedentary group. OBAE: Obese Acute Exercise group.

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\(p<0.001\)
that both acute and chronic aerobic exercise induced
those authors adopted a strenuous treadmill exercise
tura et al., who reported no significant changes in insulin and
glucose levels following acute exercise and Boaventura
results agree with Newsom et al., [25]
In this study, in normal weight animals both
single session of acute aerobic exercise did not affect
serum insulin level, blood glucose level, HOMA-IR and HOMA-B, while chronic exercise training for 8 weeks induced a significant reduction in serum insulin, HOMA-IR and HOMA-B without affecting fasting glucose levels reflecting a significant improvement in insulin sensitivity.
[26], and chronic exercise [27] but those authors adopted a strenuous treadmill exercise session for 3 hours which may be the cause of the difference.
In this study, in obese rats, it was found that both acute and chronic aerobic exercise induced a significant reduction in serum insulin level, blood glucose level and HOMA-IR when compared to control obese group (OBS) also, reflecting an improved insulin sensitivity. These results agree with some authors who reported that a single bout of moderate exercise [25,28], and chronic exercise [29] induced a significant improvement in insulin sensitivity in obese subjects.
Discussion
Obesity is a worldwide health problem linked to many chronic morbid diseases such as diabetes mellitus and cardiovascular disorders [1]. Obesity is believed to cause changes in the endothelial functions as well as, changes in the activity of components of the immune system with a subsequent release of inflammatory cytokines [9].
This improvement in insulin sensitivity can be explained by the reports that exercise increases content and activity of glucose transporter (GLUT4) in skeletal muscles [30], increase the content and activity of hexokinase [31], and increase insulin clearance by the liver [32]. In contrast, other authors reported that both acute [33] and chronic [26,34] exercise protocols had no effects on blood glucose level, insulin level, and insulin sensitivity.
In this study, in normal weight animals both single session of acute aerobic exercise and chronic exercise for 8 weeks had no effects on lipid profile parameters when compared to control group, these results agree with the reports that neither acute exercise [33] nor chronic exercise [35] had any significant effects on lipid profile in normal weight subjects. In contrast, other authors reported a significant reduction in plasma lipids following chronic exercise [36].
Moreover, a single session of acute aerobic exercise in obese rats had no significant effects on lipid profile parameters which agree with Nelson and Horowitz [33]. In contrast, it was reported that exercise acutely reduces TGs and elevates HDL [37]. The reduction was not immediate but occurs 18-24h after exercise which may explain the difference from this study.
In this study, it was found that chronic exercise for 8 weeks in obese rats induced a significant reduction in all tested lipid profile parameters. These results agree with Boaventura et al., [26]. These results can be explained by the findings in an earlier study that exercise may enhance utilization and degradation of triglycerides and cholesterol, which increases the uptake of low-density lipoprotein and lowers plasma lipid level [38]. In contrast, Resaland et al., [39] reported that exercise

Table (3): Effect of acute and chronic exercise on plasma adiponectin, Interleukin 6 (IL-6), Interleukin 10 (IL-10), C-Reactive Protein (CRP) and Tumour Necrosis Factor Alpha (TNF-α) in normal weight and obese rats.

<table>
<thead>
<tr>
<th></th>
<th>Adiponectin (ng/ml)</th>
<th>IL-6 (pg/mL)</th>
<th>IL-10 (pg/mL)</th>
<th>CRP (pg/mL)</th>
<th>TNF-α (pg/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NWS</td>
<td>8.47±0.946</td>
<td>40.78±5.573</td>
<td>3.03±0.199</td>
<td>27.94±3.227</td>
<td>3.14±0.400</td>
</tr>
<tr>
<td>NWS</td>
<td>9.02±0.722</td>
<td>208.66±25.016</td>
<td>4.41±0.470</td>
<td>26.29±4.121</td>
<td>2.93±0.480</td>
</tr>
<tr>
<td>NWCE</td>
<td>9.91±0.600</td>
<td>26.13±3.884</td>
<td>5.32±0.305</td>
<td>24.59±3.032</td>
<td>2.80±0.507</td>
</tr>
<tr>
<td>OBDS</td>
<td>3.16±0.619</td>
<td>98.88±4.856</td>
<td>1.77±0.369</td>
<td>36.40±3.374</td>
<td>7.69±0.881</td>
</tr>
<tr>
<td>OBAE</td>
<td>3.92±0.595</td>
<td>316.50±35.857</td>
<td>3.30±0.334</td>
<td>34.87±3.906</td>
<td>6.88±0.987</td>
</tr>
<tr>
<td>OBCE</td>
<td>6.70±0.853</td>
<td>42.88±11.667</td>
<td>4.29±0.315</td>
<td>33.39±2.608</td>
<td>4.85±0.838</td>
</tr>
</tbody>
</table>

Data expressed as mean ± SD. OBAE: Obese Acute Exercise group.
NWS: Normal Weight Sedentary group.
SDAE: Normal Weight Acute Exercise group.
SDCE: Normal Weight Chronic Exercise group.
OBS: Obese Sedentary group.
OBCE: Obese Chronic Exercise group.

* : Significant vs. non-obese control group.
# : Significant vs. obese control group.
¥ : Significant vs. non-obese acute exercise group.
Ο : Significant vs. obese acute exercise group.
training had no significant effect on lipid parameters in obese subjects.

In this study, in normal weight animals a single session of exercise Induced a significant increase in both IL-6 and IL-10 levels while it had no significant effects on other tested inflammatory adipokines when compared with control sedentary group. These results agree with Petersen and Pedersen [40] who reported a consistent increase in both IL-6 and IL-10 with exercise. In contrast, Rodrigues et al., [41] reported no statistically significant difference between inflammatory expression including IL-6 at rest and at 1h and 24h after an exercise test.

Chronic exercise for 8 weeks in normal weight animals, induced a significant increase in adiponectin level and IL-10, a significant reduction in TNFα level, however, it has no significant effects on, IL-6 and CRP levels when compared with normal weight control group. These results partially agree with some earlier studies [39,42].

In this study, in obese animals, it was found that a single session of exercise induced a significant increase in adiponectin, IL-6 and IL-10 levels, a significant reduction in TNFα level, while it has no significant effects on, or CRP levels when compared with obese control group. Similar results were obtained with chronic exercise for 8 weeks but it was accompanied by a significant reduction in IL-6 level compared to no effect in single session group.

These results indicate that acute exercise in obese animals is mainly accompanied by an increase in both the proinflammatory IL-6 and the anti-inflammatory IL-10, while chronic exercise in either normal weight or obese animals, enhances the increase of anti-inflammatory cytokines adiponectin and IL-10 and a reduction or blunting the increase of pro-inflammatory IL-6 and TNFα. These results agree with several reports [43-45].

Several mechanisms have been proposed to explain how exercise first, chronic exercise improve adipose tissue hypoxia and reduce local adipose tissue inflammation, which reduces pro-inflammatory cytokine expression and increased anti-inflammatory protein expression in adipose tissue [46]. Second, regular exercise increases blood flow and laminar shear stress and reduces endothelial expression and the release of adhesion molecules [47]. Third, chronic exercise may also reduce the number of pro-inflammatory monocytes which are major producers of pro-inflammatory cytokines, such as TNFα [48]. All these explanations were reported to be independent of the exercise-induced weight loss [45]. In contrast, one study reported no statistically significant changes in inflammatory cytokines levels following acute exercise in obese patients [40], no change in adiponectin levels following long-term exercise [17], and a significant reduction in CRP levels following chronic exercise [18].

Conclusion:

This study showed that chronic exercise produced a significant improvement of insulin resistance both in normal weight and obese animals, while it significantly reduced plasma lipids in obese animals. Moreover, it induced a significant increase in adiponectin and IL-10 levels, and a significant reduction in IL-6, and TNFα levels in obese rats. On the other hand, acute exercise significantly increased IL-6 and IL-10 levels. These results suggest that the beneficial effects of exercise are best expressed with chronic and not acute exercise and these changes are more profound in obese animals. However, due to the limitation of a small number of studied animals more research is needed to through more light on the effect of exercise on inflammatory mediators.

Conflict of interest:

The author declares no known conflict of interests.

References


تأثير التمرين على السيتوكينات الإلتهابية،
وصورة الدهون والتحكم في نسبة السكر في الدم
في الجرذان البضاء والمصابين بالمسمة

وقد تم تسبيب المجموعة بلحم في زيادة إحتمالية التعرض إلى الأمراض المزمنة الشائعة، مثل داء السكري وأمراض القلب والأوعية الدموية. وقد أظهرت الدراسات التي تتDestructor في مختلف دقات الإجهاد واستقرار السيتوكينات الإلتهابية وجود إرتباط بين حالة الإجهاد العامة ذات الدورة المنخفضة التي تسبب أجزاء الجسم المختلفة من جدا وعديد من الأمراض مثل السمنة ومتلازمة التمثيل الغذائي. وداء السكري من النوع الثاني، واستقرار الدهون. ومع ذلك، فإن تأثير ممارسة الرياضة على مستويات السيتوكينات الإلتهابية لا يزال مثيرا للجدل. وقد تم تقييم هذه الدراسة لإثبات أثر ممارسة السباحة الحادة والمسمية على السيطرة على نسبة السكر في الدم، صورة الدهون وبعض مستويات السيتوكينات الإلتهابية في الجرذان ذات الوزن الطبيعي أو المصابين بالمسمة.

مواد وطرق البحث: أجريت هذه الدراسة على 48 جرذانبيض ذات أوزان تتراوح من 200 إلى 450جم. وقسمت الجرذان عشوائياً إلى مجموعتين رئيسيتين: 1- مجموعة النظام الغذائي القاسي و2- مجموعات النظام الغذائي على الدهون (24 في كل مجموعة) وبعد ذلك تم تقسيم كل مجموعة بحثية إلى ثلاث مجموعات فرعية منسقة (3- مجموعة خالية، 2- مجموعة ممارسة ثابتة، و1- مجموعة ممارسة ثابتة مزمنة لمدة ثلاثية أسابيع).

وفي نهاية فترة التجربة، تم تقييم الجرذان التضخيمية بها وأخذت عينات الدم قياس مستويات الأنسولين والسكر ومنهما تم حساب معامل مقاومة الأنسولين، ومعدل وظائف خلايا بيتا، تم قياس مستوى الدهون في الدم مثل الكولسترول الكلي، الدهون الدماغية على الكثافة، الدهون المنخفضة الكثافة والدهون الثلاثية. كما تم قياس مستويات بعض السيتوكينات الإلتهابية مثل إنترليكون-1، إنترليكون-6، إنترليكون-7، إنترليكون-11، إنترليكون-12، إلخ، إنترليكون-13، إنترليكون-14، إنترليكون-15، إلخ.

النتائج: أسفرت جلسة واحدة من التمارين الحادة في الحيوانات ذات الوزن الطبيعي عن حدوث زيادة ذات دلالة إحصائية في نسبة إنترليكون-1 و إنترليكون-6. بينما لم ي Nóفع أي تغيير في الباقى المؤثرات التي تم قياسها أثناء الدورة. بينما أسفرت جلسة واحدة من التمارين الحادة في الحيوانات ذات الوزن الطبيعي عن حدوث إنخفاض ذو دلالة إحصائية في مستويات الأنسولين والجلوكوز، ومعامل مقاومة الأنسولين، وكذلك زيادة ذات دلالة إحصائية في مستوى إنترليكون-1.

استمر التمرين المزمن لمدة 8 أسابيع في الحيوانات ذات الوزن الطبيعي عن حدوث إنخفاض ذو دلالة إحصائية في مستويات الأنسولين، معامل وظائف خلايا بيتا، ونسبة الدهون. أما مستويات إنترليكون-6، إنترليكون-12، إنترليكون-13، إنترليكون-14، إنترليكون-15، فإنه تم التمرين المزمن لمدة 8 أسابيع في الحيوانات المثبطة عن حدوث إنخفاض ذته دلالة إحصائية في جميع مجم.

نصيحة السكر في الدم، وعالية على ذلك، فقد أدت أيضاً إلى إنخفاض ذو دلالة إحصائية في مستويات إنترليكون-6، إنترليكون-12، إنترليكون-13، إنترليكون-14، إنترليكون-15، إلخ.

الاستنتاجات: ممارسة التدريبات البينية سواء الحادة أو المزمنة له تأثير ذو دلالة إحصائية على السيتوكينات الإلتهابية، وكأنها كان أثر ضخم في الحيوانات البينية. بعد أن في الحيوانات ذات الوزن الطبيعي.