The Effect of Eight Weeks Swimming Training on Hepatic Enzymes and Hematological Values in Young Female

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Abstract
The aim of this study was to determine the effect of eight weeks swimming training on Hepatic Enzymes and Hematological values in young female. Twenty-one healthy female were selected by convenience sampling method and were randomly assigned in the two groups: The test (n=15) and control (n=6) groups. The exercise protocol included long-term swimming training lasted for eight weeks and 3 sessions per week and every session lasted for 60 to 90 minutes with intensity of 65-85 percent of maximum heart rate reserve. Blood samples were taken to measure serum hepatic enzymes and hematological values before and after swimming training period. Data were analyzed by parametric (Paired and Independent-Samples t-test) and nonparametric (Wilcoxon and Mann-Whitney U test) for compared within and between groups; and the level of significance was set at P< 0.05. The level of WBC, RBC, Hb and Hct in exercise group towards the end of period of the training increased significantly. Also, there were no significant differences between groups in the levels of WBC, Hb, PLT, AST and ALT. The variance between group RBC and Hb were significant. Although, the levels of serum AST and ALT levels reduce at the end of the eight weeks swimming training, but these changes did not significantly. The liver function parameters, AST and ALT were decreased after eight weeks swimming training. These findings highlight the importance of imposing restrictions on swimming training and during clinical studies. Future examinations are now essential to clarify the effectiveness of exercise on various parameters.

Keywords: Hematological Values; Hepatic Enzymes; Swimming Training

1. Introduction
The liver is the main organ for conversion of one chemical species to a different and this interconversion is that the main route for making ready medicine for excretion from the body. The metabolism of medication will result in the formation of chemically reactive intermediates that may play a major role within the induction of hepatic injury. It is necessary that potentially hepatotoxic effects of recent medicine are recognized early throughout drug development. Therefore, in phase it clinical trials, observance of liver perform parameters is necessary. The occurrence of asymptomatic elevations in liver perform tests could be a problem throughout all phases of drug development. An asymptomatic raising of, for instance, liver transaminases throughout clinical trials might be drug related, however other factors, like exercise [1] and diet [2],
might also have had this effect. A liver is termed “fatty liver” if lipids account for more than five percent of its weight. The mechanisms for the event of liver disease are varied. A decrease within the hepatic oxidation of fatty acids as results of mitochondrial dysfunction can lead to microvesicular statuses. Another mechanism is association with an imbalance between fat uptake and secretion, with high insulin/glucagon ratio condition cause macrovesicular statuses. Fatty liver are often the consequence of many illness, involving alcohol excess, nonalcoholic steatohepatitis, hepatitis C infection, metabolic disorders, medication effects and nutritional disorders [3].

Most patients with liver disease are asymptomatic, and also the condition is typically discovered due to hepatomegaly or mild abnormalities of serum aminotransferase or alkaline phosphatase levels found on a routine physical examination. One controlled study demonstrated that a weight reduction program (combined diet and exercise) can improve liver function test results and liver histology in patients with nonalcoholic steatohepatitis. With a weight loss of 4.5 to 6.8 kg, liver transaminase levels often return to normal [4]. Investigators in another study found a correlation between high fat and oil consumption and elevated liver transaminase levels. They concluded that a low-fat diet and exercise could minimize hepatic steatosis [5].

Jabbar (2010) Found that resistance training in sedentary males lead to the no significant differences in Aspartate aminotransferase (AST) and Alanine aminotransferase (ALT) activities between creatine training and placebo groups before or after the eight weeks training [6]. Therefore, they concluded that eight weeks resistance training period along with creatine monohydrate ingestion does not have adverse effects on these hepatic cellular damage indices. Rezaeeshirazi et al (2011) reported that an eight-week period of aerobic exercise has made no significant changes in the physiological variables such as hepatic enzymes (ALT, AST and ALP) [7]. Sreenivasa (2006) in the study on Sixty-five (mean age 38.7 ± 9.5 years; 46 [78%] males) out of 94 patients diagnosed with NASH that participated in their study, found that Moderate intensity aerobic exercise helps in normalizing ALT levels in patients with NASH [8]. Takato (1997) Indicated that restricted diet and exercise therapy such as walking and jogging (for a trial period of 3 months) in twenty-five obese patients with fatty liver, are useful means of improving blood biochemical data and histological findings in liver tissues related to fatty liver [9].

It is well known that energy-restricted diet and exercise are very useful therapy for obese patients [10]. These treatments result in the reduction of body weight, blood pressure and serum lipids, and may also improve the fatty liver which accompanies obesity. However, there are few reports demonstrating a relationship swimming exercise and enzymes of liver. In the present study, we investigated the effect of eight weeks swimming training on Hepatic Enzymes and Hematological values in young female.

2. Methodology
2.1 Subjects
This study was semi-experimental; which compared two groups with pre-test and post-test designs. Furthermore, its plan was confirmed by Research Assembly of Physical Education and Sport Sciences Faculty of Ferdowsi University of Mashhad life save federation. During first stage, the subjects of this study were twenty-one healthy females who randomly assigned into the experimental (n=15) and control (n=6) groups. Before starting the program, written informed consents were taken from all subjects. The levels of health and physical activity of the subjects were determined using general practice physical activity questionnaire, physical activity readiness questionnaire and medical survey (including electrocardiogram and blood pressure tests) by a specialist physician [11]. The subjects were nonsmokers, received no drugs and had no metabolic disease and physical impairment affecting their performance. During the second stage, their height was measured in centimeters using a height determiner and their weight was calculated using a digital scale produced by a German company called Beurer (PS07-PS06). The percent of body fat (BPF) was calculated using a body compound determiner (model In-body-720 made in Korea) and based on a method called bioelectrical impedance. All of these measurements were carried out while the volunteers had stopped eating or drinking 4 hours prior to their test, and their bladder, stomach, and bowels were empty.

2.2 Exercise protocol
The swimming training program was designed according to in guideline of American College of Sports Medicine [12] and was performed by professionally qualified physical instructors (an experienced coach) from the health clubs Sports and Health Pioneer East, Mashhad, Iran. Under the supervision of exercise physiologists who assessed and individualized the exercise program for volunteers and supervised their performance during exercise, each participant was trained to achieve a target heart rate reserve (HRR). The exercise protocol included aerobic exercise training
lasted for eight weeks and 3 sessions per week and every session lasted for 60 to 90 minutes and with intensity of 65-85 percent of maximum heart rate reserve (MHRR). According to the MHRR for every single athlete was respectively calculated based on Karvonen equation (1) and was also controlled during exercise by a heart rate monitor (made in Finland–Polar) [13].

\[ \text{Target heart rate} = \frac{\%60 \text{ or } \%70 + \frac{\text{resting pulse}}{220} \times \text{age}}{220} \times \text{age} \]

The aerobic training program consisted (20 minutes of warm up exercise, 50 to 60 minutes of aerobic exercise and 10 minutes of cool down exercise) with 65-85 % HRR during each training session. The first to fifth sessions of swimming exercise the HRR was 65% and for each session the heart rate was added to the end of a swimming exercise sessions. So that in the sessions of sixth to tenth (70%HRR), eleventh to fifteenth sessions (75% HRR), sixteenth to eighteenth sessions (80% HRR) and nineteenth to twenty forth sessions intensity of swimming exercise was maintained in 85% HRR.

2.3 Blood sampling

Blood samples in all related studies were collected by venipuncture for forearm vein after at least 15 minutes of sitting at rest or in the supine position. Blood sample were poured into a tube containing K2EDTA and mixed for 15 min before analysis. After centrifuging samples in plastic capillary tubes using Haemato Spin Centrifuge device. Aspartate aminotransferase (AST) and Alanine aminotransferase (ALT) were determined using the Olympus AU400 Chemistry Analyzer.

2.4 Statistical analysis

All statistical analyses were performed with SPSS version 15. The average and standard deviation of data were calculated after checking the data distribution normaly using Kolmogorov-Smirnov test and Homogeneity of variance method. The result of Kolmogorov-Smirnov test in some parameters was not normal. Therefore, data were analyzed by parametric (Paired Samples, Independent-Samples t-test) and nonparametric (Wilcoxon and Mann-Whitney U test) for compared within and between groups; and the level of significance was set at P< 0.05.

3. Results

The average, standard deviation and results coming from within the group’s changes of levels WBC, RBC, Hb, Hct, PLT, AST and ALT of swimmers is presented in table 2. According to the (Table 2), our results show increase in WBC, RBC, Hb and Hct levels in exercise group towards the end of period of the training significantly (P<0.05). Also, there were no significant differences between groups in the levels of WBC, Hb, PLT, AST and ALT. Results showed a variance between group RBC and Hb were significant (P<0.05). The levels of serum AST reduce in exercise group; however, these changes did not significantly (P>0.05).

Furthermore, the ALT levels presented that in table 3. Although, the ALT levels reduce at the end of the eight weeks swimming training, but these changes did not significantly (P>0.05).

**Table 1.** Shows means for age, height, weight and subject’s background in sports.

<table>
<thead>
<tr>
<th>Variables</th>
<th>M±SD*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>26.93±1.53</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>164.33±5.16</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>61.43±16.53</td>
</tr>
</tbody>
</table>

**Table 2.** Values of hematological indices and hepatic enzymes before and after eight week of swimming training (Mean±SD) *

<table>
<thead>
<tr>
<th>Variables</th>
<th>Groups</th>
<th>Pre-test Mean±SD*</th>
<th>Post-test Mean±SD*</th>
<th>p**</th>
<th>p***</th>
</tr>
</thead>
<tbody>
<tr>
<td>White blood count</td>
<td>Exercise group</td>
<td>5706.67±855.62</td>
<td>6742.13±1257.42</td>
<td>0.04*</td>
<td>0.18</td>
</tr>
<tr>
<td>(x 106/m3)</td>
<td>Control group</td>
<td>6150.00±898.33</td>
<td>6133.33±915.78</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Red blood count</td>
<td>Exercise group</td>
<td>4.73±0.29</td>
<td>5.37±0.47</td>
<td>0.00*</td>
<td>0.00*</td>
</tr>
<tr>
<td>(x 106/m3)</td>
<td>Control group</td>
<td>4.83±0.31</td>
<td>4.70±0.20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemoglobin (gm/dl)</td>
<td>Exercise group</td>
<td>13.90±0.82</td>
<td>14.48±1.22</td>
<td>0.00*</td>
<td>0.17</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>13.38±0.92</td>
<td>13.61±0.81</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>Exercise group</td>
<td>41.78±2.09</td>
<td>44.27±3.13</td>
<td>0.00*</td>
<td>0.00*</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>41.00±2.49</td>
<td>40.75±1.70</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Platelets (1000)</td>
<td>Exercise group</td>
<td>259133.42±533.79</td>
<td>267367.71±57908.37</td>
<td>0.37</td>
<td>0.21</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>236500.00±42522.32</td>
<td>225500.00±45715.42</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AST (U/L)</td>
<td>Exercise group</td>
<td>16.80±5.89</td>
<td>15.40±5.04</td>
<td>0.15</td>
<td>0.28</td>
</tr>
<tr>
<td></td>
<td>Control group</td>
<td>13.17±3.31</td>
<td>13.50±3.72</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Data presented as mean ± standard deviation  ** Paired sample t-test  *** Independent samples t-test
C levels within the different stems should show a physiological variable can be identified those twenty days intensive exercise did not cause any changes at HCT levels. Once literature was scanned about HCT parameters, Boyali et al., (2006) reported a significant increase at HCT values [23] and Mashiko et al., (2004) reported a significant decrease in WBC levels of experimental group in camping amount. % , it couldn’t be found a significant increase in WBC levels from blood samples after regular aerobic exercise too. Mashiko (2004) reported a significant decrease in WBC levels of experimental group in camping amount [19]. It is thought that the results of these will increase and reduces was caused by experimental design.

Our study showed significant increase in RBC, Hct and mean hemoglobin concentration after the long swimming training. Halson (2003) reported increase of HGB parameters in trained people [20] and Patlar and Keskin (2007) emphasized decreases and increases in HGB levels after exercise [21]. Yeh (2006) and Umit (2004) haven't found a significant difference at RBC levels of each of the groups after and before exercise program; there are some evidences about exercises done in different intensities that have effects on RBC levels. Once literature was scanned about HCT parameters, Boyali et al., (2006) reported a significant increase at HCT values [23] and Mashiko et al., (2004) identified those twenty days intensive exercise did not cause any changes at HCT levels [19]. Whereas an increase at RBC levels within the different studies, a significant increase could not be found at HGB, HCT and PLT levels (2010). It is thought that these decreases and increases were caused by exercise protocols that applied the sector-specific. Alterations of the hematological variables can influence physical performance. Also, Performance in exercise depends on each the amount of hemoglobin and therefore the development of ability of carrying oxygen [24]. Intensive exercise can cause significant differences in hemoglobin values [25]. Oxygen from red blood cells in tissues is connected to hemoglobin which provides the oxygen to active tissues. Organism’s oxygen demand increases throughout training. In parallel to the present increase, the circulatory and respiratory systems should show a physiological adaptation. Then the oxygen would like of tissues and therefore the amount of oxygen of the cardiovascular system increases [26]. Especially, evaluation of blood volume in young is complex. There are conflicting results in literature regarding changes in blood volume per unit body mass increases with age. Women have lower hemoglobin concentration values and it would lead to reduced oxygen carrying capacity [27]. In fact, exercise intensity and duration are two necessary components in exercise or regular training. Therefore, it is potential that duration and intensity of this training protocol were enough to have an effect on hematological variables. Our study showed declines in AST and ALT concentration after the long-term swimming training. This finding was supported by Sreenivasa et al., (2006) and Davoodi et al., (2012) [28]. Davoodi et al., (2012) in their study found that eight weeks selected aerobic exercise lead to the reduces in serum AST and ALT in experimental group rather than control group. The findings from the present study are inconsistent with those reported in the literature. Nie et al., (2011) found statistically a significant increase at AST and ALT levels after exercise [29], also in other study, it was reported a significant increase at AST and ALT values of athletes running ultra-marathon after and before competition [30]. In excessive muscle forced exercise-induced, AST and ALT levels in blood can raise in muscle damages. Membrane permeability, changes which cause these enzymes to leak into serum, occur related to muscle damage exercise-induced [31]. Furthermore, Aerobic activity stimulates lipid oxidation and hinders lipogenesis.

4. Discussion and Conclusion
Changes occur in metabolism depending on the intensity and severity of exercise; changes may be in blood values before and after exercise [14]. Variations were found among sedentaires and female athletes in some parameters in study done to look at the effects of chronic exercise on some hematological and hepatic enzymes parameters. The results of this study reveal that the level of WBC increased significantly. The findings from this study are consistent with those reported in the literature. Patlar (2010) reported a significant increase in WBC levels after four weeks of chronic submaximal exercise period [15]. The findings of this study are inconsistent with studies demonstrating that WBC not changed in response to exercise. Thus, in studies examining the effects of exercise done on chronic WBC values, Yeh (2006) found that twelve weeks of exercise didn’t cause any significant changes in wbc levels [16]. Within the same way, Banfi et al., (2006) reported that WBC levels were similar before [17] and after camp and in study done by Ergun (2006) [18], it couldn’t be found a significant increase in WBC levels from blood samples after regular aerobic exercise too. Mashiko (2004) reported a significant decrease in WBC levels of experimental group in camping amount [19]. It is thought that the results of these will increase and reduces was caused by experimental design.

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inside the liver [32-33]. This is often mediated by the activation of AMPK pathway. This enzyme is activated when the ratio of AMP to ATP in tissues as in an aerobics. Studies show that a reduced or absent activity of hepatic SCD-1 is crucial to the activation of AMPK. Recent animal studies have found a significant reduction of SCD-1 activity after aerobic exercise [33]. AMPK remains activated after the completion of aerobic activity in liver, adipose tissue and muscle. In liver it reduces lipogenesis through direct inactivation of ACC enzyme and activation of MCD. It also reduces related gene expressions of lipogenic enzymes; ACC and FAS. Activation of MCD in turn reduces the amount of Malonyl-CoA that is an inhibitor of CPT-1. The latter enzyme regulates fatty acid transfer to the mitochondria and thus stimulates hepatic lipid oxidation [33].

As a result, aerobic training can cause increased insulin sensitivity and hepatic lipid oxidation and also reduced activity and inhibition of lipogenic enzymes. All of them contribute to a reduction of hepatic fat [34]. This can explain the significant reduction of blood serum ALT concentration of patients within the group with added aerobic training.

After eight swimming training, hematologic parameters showed differences when compared to resting values. Besides, there have been no significant variations in ALT and AST levels after eight weeks swimming training. As a result, several changes in metabolism occur after long-term exercise. Several other factors play a major role, such as adaptation to exercise, adaptation of cardiovascular, physical and physiological balance in hematological levels. It may be said that more comprehensive studies on examine training should be done to determine the relation among hematologic and hepatic enzymes values and sportive performance.

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