Effect of concurrent training on the Serum Paraoxonase-1(PON-1) activity and Lipid Profile in obese men

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ABSTRACT: The purpose of this study was to investigate the effects of concurrent training on serum paraoxonase-1 (PON1) activity and lipid profile in obese men. Sixteen obese men volunteered to participate in this research. They were randomly assigned into two groups: concurrent training group (n=8) and control group (n=8). Duration of training was 4 weeks, 3 sessions per week and each session lasted 70 min. Concurrent training group carried out exercises at 60-75 percent of heart rate reserve. In order to evaluate the effect of concurrent training on PON1 activity and lipid profile, the t-test were used. Statistical significance was accepted if P<0.05. Correlation coefficient was used to determine associations between variables. Results show significant increase in PON1 activity and significant decrease in total serum cholesterol (p ≤ 0.05) but other lipid parameters of this research including LDL, HDL, VLDL, TG levels was not significant compared with control group. In addition, in obese men changes levels of paraoxonase1 have significant relationship with HDL, but there was not significant relation with the other lipid indexes of this research. However, HDL-c, HDL-c/Total cholesterol ratios and maximal oxygen uptake (VO2max) significantly increased (P<0.05) and body mass index (BMI) significant decreased (P<0.05) due to concurrent training. HDL function associated with PON1 activity has been consistently associated with cardiovascular and other diseases. The result of this study showed that after four weeks of concurrent training on lipid and weight changes had the desired effect and as well as significantly decrease was in enzyme paraoxonase 1 and lower cholesterol concentration.

Keywords: Concurrent training, Lipid Profile, LDL, obese men, Paraoxonase

INTRODUCTION

Human serum paraoxonase (PON1) is located on one of the high density lipoprotein cholesterol (HDL-c) subfractions which contain another components including APO A-I and clusterin (Mackness, 1995: 243-53). PON1 is synthesized by the liver, and is stored in large amounts in mammals. It has been shown that PON1 is an enzyme with two type activity including paraoxonase activity (measurable toward paraoxon) (LaDU B. N, 1984).

It is suggested that the activity toward paraoxon (PON) is more variable and is sensitive to different modulating factors (Beltowski J and et al. 2002). PON1 is a member of a family of proteins that also includes PON2 and PON3 (Gyorgy PS and et al. 1998). PON1 is synthesized primarily in the liver and a portion is secreted into the plasma, where it is associated with HDL (Senti M, 2000). One natural physiological function of PON1 appears to be the metabolism of toxic oxidized lipids of both LDL as well as HDL particles (Pols MA. 1995:381-8).

Other studies have confirmed and extended this finding that PON1 prevents the formation of oxidized LDL and phospholipids once they are formed. PON1 also protects HDL phospholipids from oxidation (Beltowski J and et al. 2002). These actions suggest a role of PON1 in preventive of cardiovascular diseases and atherosclerosis. PON1 has also been shown to metabolize a number of drugs and pro-drugs via its lactonase activity (Beltowski J and et al. 2002).

Earlier studies had indicated that the plasma paraoxonase activity in human populations exhibited a polymorphic distribution, and individuals with high, intermediate or low paraoxonase activity could be identified...
ON1 activity and atherosclerosis, and it is believed that PON1 probably has a role in the antiatherogenic properties of HDL-c. In other words, PON1 can increase power of HDL-c in metabolizing lipid peroxides and reducing the extension of atherosclerotic lesions (Liggy P, 2002; H. Noto, 2001). Other reports have also pointed out that low density lipoprotein cholesterol (LDL-c) and a number of other oxidized phospholipids can be counted as appropriate physiological substrates for serum PON1 and that PON1 prevents the oxidative changes of lipoproteins, especially LDL-c (Durrington PN, 2001). However, current reports pointed out that a high amount of HDL-c and a low amount of LDL-c cannot solely guarantee cardiovascular health (Senti M, 2000; Pols MA, 1995). These results refocus attention on the many aspects of HDL biology that are not captured by serum measurements of HDL, such as its paraoxonase 1 (PON1) activity, which itself is inversely associated with cardiovascular and other human diseases (Tomas M, 2002; McArdle WD, 2001). PON1 is a liver-produced glycoprotein enzyme bound to the surface of HDL whose activity is consistently correlated with atherosclerotic vascular disease and end-organ damage (Durrington PN, 2001; Tomas M, 2002).

Finally, PON1 activity appears to plays a role in maintaining the endothelial-atheroprotective effects of HDL. PON1 has broad substrate specificity and is protective against exposure to toxic organophosphorus insecticides (Gyorgy PS, 1998). It has been shown that PON1 genotype has an essential role in the PON1 response to exercise training (Tomas M, 2002).

In addition, it has been shown that the effects of physical training on the lipid profile are dependent on PON1 polymorphism (Senti M, 2000). Results of the few reported studies show that concurrent training will inhibit the activity of PON1 (H. Noto, 2001; Mackness, 2001). In addition, it is determined that PON1 activity did not demonstrate significant difference between active and obese non-smokers (Britez F, 2000). While it has found that in well trained rugby players, the activity of the anti-oxidant enzymes such as PON1, was higher than the obese group (Malin R, 2001; Pols MA, 1995). However, the question still remains as to what is the effect of concurrent training on PON1 activity and what is the interaction between this kind of physical exercise and lipid profile in individuals? These are the questions posed in this study.

MATERIAL AND METHODS

Sixteen males aged 39-43 participated in this research. They were randomly divided into two groups: concurrent training (CT-group) (n=8; 101±20/41±2/172±6 mean weight/age/height), and control (n=8; 104±12/41±2/174±5 mean weight/age/height) group. Participants had no previous record of cardiovascular disease, tobacco smoking, and regular physical activity. This information was collected using a health condition questionnaire and Baecke questionnaire of habitual physical activity [15], respectively. Also, dietary intake of subjects during the 2 months of our study was recorded through a 24-hr dietary recall questionnaire. Dependent variables were evaluated in two phases including pre-test and post-test. Duration of training was 4 weeks and participants carried out exercise training for 3 sessions per week and each session lasted 70 minutes. CT groups performed concurrent training (at 60-75 percent of heart rate reserve) respectively. The training heart rate was calculated by “Karvonen method” for every subject (Gyorgy PS, 1998). Concurrent training included combination of endurance and resistance training.

PON1 activity and lipoprotein profile of serum were measured by kinetic enzymatic reaction and CHOD-PAP enzymatic method, respectively. Serum samples were taken 24 hours before the start of protocol and 24 hours after the last session of exercise in post-test. Total cholesterol (TC), and HDL-c concentrations were measured by CHOD-PAP enzymatic method with a Pars Azmun kit (Benitez S, 2002). LDL-c concentration was calculated with the Friedwald formula for serum samples with TG values less than 400 mg/dl (Pols MA, 1995). We estimated the maximal oxygen uptake (VO2 max) by the Fax protocol on the ergometer (5 Min, 150 Watt intensity and 60 RPM speed) (Benitez S, 2002).

In order to evaluate the effect of concurrent training on each of the dependent variables. The t-test were used. Statistical significance was accepted if P<0.05. Correlation coefficient was used to determine associations between variables.

RESULTS AND DISCUSSION

Research finding has shown that concurrent training causes significance increased in weight, body mass index and whole body power (P<0.05) also concurrent training cause’s significance decrease in PON1 (P<0.05). Comparison of cholesterol mean (mg/dl) in pre test and posttest has shown that difference means are meaningful decrease in concurrent group (P=0.001). By considering of PON1 means difference in pre and post test was meaningful in concurrent group (P=0.01) (figure 1). Mean dependent variables including PON1 ,TG, cholesterol, LDL, VLDL, HDL activity values are presented in Table 1. Measuring of TG, LDL, VLDL and HDL has shown that didn’t have significant change in concurrent group. (P=0.05). The results of this research have
shown that there is relationship between PON1 and HDL (P=0.04), but Studies have shown that there is didn’t have significant relationship between PON1 and cholestrol, TG,LDL, VLDL.

In the present study, PON1 did affected with concurrent training. However, we found significantly increase in the PON1 and estimated VO2max, and inversely significantly decrease in BMI after concurrent training at 60-75 percent of maximal reserve heart rate. In other hand, it is determined that although lipoprotein profile, VO2max and BMI of CT-group modified beneficially, but they did not receive to the significance levels. Data concentering the possible effect of exercise training on the anti-oxidant enzymes activity such as PON1 are controversial, as differences and no changes in these parameters comparing active and obese individuals have been described (Tomas M ,2002 ;Britez F , 2000 ).

It has been found that after performing 4 weeks of concurrent training people who carry the R allele showed reduction of PON1 activity, and people who carry the Q allele showed an increase (both significant), while in total group (Q & R allele) changes were not significant (Tomas M ,2002). In another study, the activity of the anti-oxidant enzymes such as PON1, was higher in well trained concurrent than to control group. Duration of our training protocol was 4 weeks and comparison between concurrent and our control subjects is not probably reasonable. It has been found that serum PON1 activity were significantly different (P<0.01) between subjects after chronic exercise (daily, for 6±2.5 year) and acute exercise (jogging-race and concurrent upstairs, 3 day/ week, and for 3 months) (McArdle WD , 2001).

Results of above mentioned studies implicate that making changes in PON1 activity probably need to a long-term and chronic exercise–related stimulation. In addition, it has been shown that PON1 activity is not significantly different comparing active and obese non-smokers, whereas it was significantly higher in active than non–active smokers (Beltowski J ,2002). Besides, some researchers found no significant differences in the physical exercise level (None, 1-5 h/week, and >5 h/week) in leisure time when participants were classified according to PON1 tertiles (high, medium, and low activity) (Durrington PN ,2001). These results (Beltowski J ,2002; Gyorgy PS ,1998) are consistent to our research based on lack of significant different of PON1 between trained and non-trained subjects (both non- smokers). Other reports showed that physical activity in males with the R allele was associated with the increase of HDL-c and the reduction of triglyceride (Malin R.2001). Similarly, we found in our research that HDL-c concentration, and HDL-c/TC ratio showed increase in CT-group, and there were inclination to decrease of holestrol concentration in concurrent group. Generally, the existing results suggest that although some type of physical exercise has not direct and significant effects on PON1 activity, improved physical fitness and adjust lipoprotein profile brings a control of cardiovascular risk factors in the people with low PON1 activity level (AA phenotype) (Tomas M ,2002).

It has been suggested that acute exercise training can inhibit temporarily PON1 activity and chronic exercise training could attenuate this inhibitory effect of training on PON1 activity (H. Noto,2001). Decrease of rats PON1 activity due to both single race and parathionmetyl has been confirmed by another study (Senti M,2000).In our study intensity of concurrent exercise training did not affect the PON1 activity, but it seems that PON1 activity usually decreases due to acute exercise which carry out on higher than the lactate threshold (an concurrent exercise) and to have an improved physical fitness can attenuate this inhibitory effect. We observed the level of Vo2max increased in CT-group and this implies that improved physical fitness could guarantee maintenance of PON1 activity. Furthermore, concerning the reduction of PON1 activity after acute exercise, our results suggest that performing of concurrent training(up to 60 percent of maximal reserve heart rate) does not decreasingly effect on PON1 activity in people with AA phenotype. In other words, this kind of physical activity has beneficial effects on the risk factors and can at least maintain PON1 activity in acute conditions(Britez F ,2000). To the best of our knowledge, our results constitute the first published report on the interaction between PON1 activity and physical exercise in Iranian descent. We found that our participants have lower PON1 activity than people in Western countries. The expression of PON1 enzyme activity in the serum is under genetic control and the polymorphism of the PON gene has been extensively recognized in different communities as a potential genetic determinant of PON1 activity (Benitez S ,2002). In view of variability of genetic makeup of different populations, the activity of PON1 shows great interethnic variability. European populations have been shown to have a bimodal distribution of PON1 activity, but in some non-European populations, low PON1 activity has been observed together with a unimodal curve of activity (Senti M,2000; Pols MA,1995).

It is reported that there is variability as 10 to 40 fold in PON1 activity between populations (Gyorgy PS ,1998). Low PON1 activity observed in our study is consistent to another research performed in Iranian population [1]. The lack of significant interaction between PON1 activity and concurrent exercise in an Iranian group (with AA phenotype) along with low PON1 activity of our subjects probably confirm the concept of racial variability of PON1 activity (Tomas M ,2002).

In addition, some of the inconsistent results are probably related to use of different designs of research (experimental vs. observational studies), and different nature (intensity and duration) of performed exercise training. Further studies are required to elucidate the interactions between exercise training, PON1 phenotype, and cardiovascular risk factors.
The result of this study showed that after four weeks of concurrent training on lipid and weight changes had the desired effect and as well as significantly decrease was in enzyme paraoxonase 1 and lower cholesterol concentration. But it seems the PON1 enzyme in obese men were significant relationship with HDL lipid parameters and other factors not related to research. Thus this protocol can in non-training obese men with inhibits the oxidation of LDL and limiting the HDL in circulation of serum with the goal of prevention cardiovascular disease effectively to be used. Finally, exercise can induced enhanced in the total antioxidant system.

Figure

![Comparison of PON (nM/lit) before and after of training.](image)

Table 1. Results of T-test related to effects of concurrent training on the dependent variables

<table>
<thead>
<tr>
<th>variables</th>
<th>group</th>
<th>Pre test</th>
<th>Post test</th>
<th>T</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>PON1(nM/lit)</td>
<td>concurrent</td>
<td>156</td>
<td>118</td>
<td>3.427</td>
<td>0.01*</td>
</tr>
<tr>
<td></td>
<td>control</td>
<td>125</td>
<td>112</td>
<td>1.488</td>
<td>0.18</td>
</tr>
<tr>
<td>LDL(mg/dl)</td>
<td>concurrent</td>
<td>109.75</td>
<td>105</td>
<td>1.025</td>
<td>0.34</td>
</tr>
<tr>
<td></td>
<td>control</td>
<td>103.14</td>
<td>113.62</td>
<td>0.875</td>
<td>0.412</td>
</tr>
<tr>
<td>HDL(mg/dl)</td>
<td>concurrent</td>
<td>34.75</td>
<td>38.5</td>
<td>0.419</td>
<td>0.038</td>
</tr>
<tr>
<td></td>
<td>control</td>
<td>38.37</td>
<td>37.87</td>
<td>-0.57</td>
<td>0.956</td>
</tr>
<tr>
<td>VLDL(mg/dl)</td>
<td>concurrent</td>
<td>28.27</td>
<td>30.77</td>
<td>0.17</td>
<td>0.87</td>
</tr>
<tr>
<td></td>
<td>control</td>
<td>22.38</td>
<td>34.7</td>
<td>-5.18</td>
<td>0.001*</td>
</tr>
<tr>
<td>cholesterol(mg/dl)</td>
<td>concurrent</td>
<td>195.87</td>
<td>183.25</td>
<td>-5.18</td>
<td>0.001*</td>
</tr>
<tr>
<td></td>
<td>control</td>
<td>198.37</td>
<td>183</td>
<td>2.007</td>
<td>0.085</td>
</tr>
<tr>
<td>TG(mg/dl)</td>
<td>concurrent</td>
<td>184.57</td>
<td>190.12</td>
<td>0.62</td>
<td>0.555</td>
</tr>
<tr>
<td></td>
<td>control</td>
<td>198.37</td>
<td>183</td>
<td>2.007</td>
<td>0.085</td>
</tr>
</tbody>
</table>

Note: * significant effects (P ≤ 0.05) of concurrent training

REFERENCES


