ABSTRACT

This modern age is experiencing increased cardiovascular diseases and higher mortality rates due to inert lifestyles and bad nutrition habits along with stress. Apo lipoproteins A and B are among the most important prognosticating factors of cardiac diseases. Therefore, the present study attempts to study the effects of a three-month college football practice on changes in Apo proteins A and B in inactive young men. Through a public call, 30 subjects were selected randomly from male college students and were divided into two control and experimental groups of 15. The experimental group participated in 3 sessions of exercising per week and the control group had not practice at all. 24 hours before the first and 24 hours after the last exercise session, 10 cc blood samples were taken from both groups after 12 hours of fasting from the left arm vessel. These samples were centrifuged to separate the serum and kept at -70°C to be analyzed at a laboratory. In order to compare Apo proteins A and B of the posttest in both groups, covariance analysis was utilized. Results revealed that physical activities in the form of three-month college football practice change Apo protein A and B levels in blood serum in pre and posttests of experimental group significantly. Yet, the study failed to find significant differences in the level of Apo protein A and B of control and experimental groups.

INTRODUCTION

Lipoproteins are formed by particles called lipids and proteins including free cholesterol, esterified cholesterol, triglyceride and phospholipids along with quadruple lipids (Song et al., 2012). The proteins of lipoprotein structure are called Apo lipoprotein and Apo protein (Niklas et al., 2009). Sixty percent of HDL lipoproteins with high density are generally made up of Apo proteins; however, the percentage falls to modest 1% in chylomicrons (Holme et al., 2007). One or more types of Apo lipoprotein exists in each lipoprotein. Apo lipoprotein B is built in liver and is discharged into plasma as a part of VLDL. It acts in transferring cholesterol to tissues and is a positive risk factor for coronary heart diseases. High density lipoproteins are formed in liver and a high ratio of them consists of Apo lipoprotein A_{1} and A_{II} (Ghanbari Niaki, 2013). Apo lipoprotein A is the main protein in HDL particles. It is also responsible for transferring the extra cholesterol from tissues and the primary lipoproteins inside intestines. Like HDL, Apo lipoprotein A is a negative risk factor for heart attack and coronary diseases (Paoli et al., 2013; Tsekouras et al., 2008). In HDL, the main Apo protein is distinguished with A and in LDL the major Apo protein is shown with B (Segrest et al., 2013). An important prognosticating factor for cardiovascular diseases is the ratio of Apo lipoprotein B to A so that it must be less than 0.5 (Mercedes, 2009). One of the important chemical tasks played by Apo proteins is their role as Enzyme co-factors. For instance, Apo protein A is the enzyme co-factor of lecithin cholesterol acyl transferase (Mestek, 2009; Parish et al., 2009) and plays the role of lipid carrying protein (like Apo protein D as a carrier) HDL. Finally, it plays the role of ligand in linking lipoproteins with receptor molecules on cellule membrane of different tissues like Apo protein B_{100} and E for LDL cell receptors and Apo protein A, for HDL cell receptors (Annie et al., 2008; Durheim et al., 2009). This modern age is experiencing increased cardiovascular diseases and higher mortality rates due to inert lifestyles and bad nutrition habits along with stress (Haram et al., 2009; Mestek et al., 2009). Hyperlipidemia is among the
major reasons for people suffering cardiovascular diseases (Song et al., 2012). Several studies have revealed that the main reason for this is the increase in Apo protein B and Apo protein A of blood in these people (Parish et al., 2009; Yourka et al., 2008). Therefore, amount of Apo protein A as an anti-risk factor and the level of Apo protein B as a risk factor in athletes are more significant compared to non-athletes and it is the opposite in case of Apo protein B (Durheim et al., 2008; Kooze Chian et al., 2014). Researchers have reported that high intensity aerobic exercises for 20 minutes with strength training for two periods has a decreasing effect on lipid profiles (Azarbaijani and Abedi, 2012).

Several scholars believe that the level of Apo protein B in blood plasma of men and women decreases significantly after a course of endurance training (Niklas et al., 2009; Arthur, 2009; Amy et al., 2014). In another study on the levels of HDL lipoprotein and Apo proteins A and B in elderly women doing a course of long-term aerobic exercises, it was revealed that blood HDL and Apo protein B decreases significantly, yet; the leve of Apo protein A did not change significantly (Kim and Jung, 2014). Besides, Parish et al., (2009); Ghorbanian et al., (2013) investigated athlete and non-athlete individuals and demonstrated that levels of HDL and Apo protein A in athletes were more than non-athletes. In this regard, Arazi et al., (2012) studied the response of blood factors to a course of resistance exercises with various levels of severity in male students revealed that the amount of HDL and Apo protein A in both athlete groups increased and the level of LDL and Apo protein B in athletes decreased.

Another study on active and inactive students revealed that active students experienced a significant decrease in Apo protein B, LDL, total cholesterol, and serum resting heart beat and BMI of body composition in obese girls after physical activities (Habibzadeh and Rahmani Nia, 2011). However, Amy et al., (2008) revealed that above mentioned variables do not change significantly in men and women after long term aerobic physical activities. As it can be seen, the results are very contradictory. For instance, Haram et al., (2009) reached absolutely different results. Sheikh al-Islami Vatani (2011), in another study, demonstrated that endurance training without losing weight increased HDL by 10 to 20% and Apo protein by 9 to 36%. On the other hand, triglyceride decreased by 7%.

Moreover, Fontana et al., (2007) investigated the effects of aerobic exercises on cardiovascular risk factors and found no significant change in Apo proteins A and B. Accordingly, Segrest et al., (2013) failed to find any significant effect for aerobic and endurance training on Apo proteins A and B. In spite of all these studies and the contradictory results they have obtained and lack of a proper training plan, there is still the need for further studies and numerous ambiguities are yet to be clarified. Apart from their gender, numberless students spend at least four years in universities and decent physical education classes along with proper training programs will guarantee a healthy lifestyle away from cardiovascular and pulmonary diseases. Thus, the present study aims to find an answer to the question that if a college training program affects the level of Apo proteins A and B.

**METHODOLOGY**

The present quasi-experimental study was conducted with two control and experimental groups of 15 subjects. The statistical population of the study included all 855 male students aged 23± 2 entering the university in September 2014. The statistical population was called on and 52 students opted to participate in the study. In order to keep the homogeneity 30 students were selected as the statistical sample via physical activity questionnaire and were put into two groups of control and experimental with 15 subjects in each.

**Training method**

Training program in this study included a three-month college football training that was conducted during the first semester with three sessions of 90 minutes in a week. Each session the severity and the length of the exercise was fixed (Table 1). The sessions were held between 4 and 6 pm in the indoor stadium of Shahid Madani University of Azerbaijan.

In order to find the intensity of running in experimental group, maximum heart rate and heart rate were calculated through Adams formula (2012) to be 190 to 195 beat per minute.

\[
\text{Age} - 220 = \text{HR}_{\text{max}} \\
\% \text{HRR} = \frac{\text{HR}_{\text{max}} - \text{HR}}{\text{HR}_{\text{max}}} \\
\% \text{THR} = \frac{\text{HR} + 0.70 \times (\text{HR}_{\text{max}} - \text{HR})}{\text{HR}_{\text{max}}}
\]

**Table 1. One session of the three month college football training with three session a week during one semester**

<table>
<thead>
<tr>
<th>Type</th>
<th>Stretching</th>
<th>Aerobic</th>
<th>up joints</th>
<th>Warming</th>
<th>skills</th>
<th>Game of football</th>
<th>recovery</th>
<th>Whole session</th>
</tr>
</thead>
<tbody>
<tr>
<td>Length</td>
<td>10</td>
<td>15</td>
<td>1</td>
<td>1</td>
<td>40</td>
<td>5</td>
<td>90</td>
<td></td>
</tr>
<tr>
<td>Intensity</td>
<td>0.7</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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**Taking blood samples from subjects**

48 hours before blood sampling, necessary advice was given to the subjects including fasting, not taking any medicine and not doing any physical activity before sampling. 10 cc of blood sample was taken from subjects 24 hours before the first training session and 24 hours after the final session from left arm vessel of all subjects in both groups using sterilized tubes containing anti-clotting EDTA chemical. The subjects were fast for 17 hours. The temperature in blood sampling site was 23-25°c and after sampling the samples were centrifuged by experts and taken to Pasteur Laboratory in a subzero temperature.

**Gauging device**

Gauging cholesterol, triglyceride, and HDL, LDL and VLDL variables was conducted using CoBAS miRAS Auto analyzer with photometry method made by Rosche Company in Switzerland and via pathology kits of Pasteur Laboratory. Randox kit with spectrophotometer method used by RA 1000 auto analyzer made in England to measure Apo A1 and Apo B.

**Statistical method**

Descriptive statistics were used to analyze data for mean and standard deviation. Due to the difference in Apo proteins of the subjects in their pre and posttests along with comparing Apo proteins A and B in the post test and Apo proteins A and B in both control and experimental groups, covariance analysis was used. Apo protein levels of the pretest for each individual were taken as covariate to correct the mean of the groups and increase its precision through decrease error probability.

The statistical model used is as follows:

\[ Y_i = B_0 + B_1 X_{i1} + T_i + E_i \]

\( X_i \) = Apo protein variable in pretest \( \mu_i \) (covariate with variable)

\( E_i \) = random error \( T_i \) = fixed effect of group I (post test) (pretest) \( B_0 \) = intercept

\( B_1 \) = regression coefficient and total mean equals: \( \mu = B_0 + B_1 \mu_0 + T \), where \( \mu_0 \) is the mean of \( x \) covariate.

In order to test the difference in Apo protein in pretest and post test pendent samples statistical method was applied for two samples before and after the experiment were dependent to each i.e. subjects gauged in pretest were the same people gauged in post test. The difference in Apo protein changes in control and experimental groups were tested through a multiple regression model where the grouped variable was taken as a binomial variable with 0 and 1 assigned for control and experimental groups respectively. The multiple regression model used was as follows:

\[ Y_i = B_0 + B_1 X_{i1} + B_2 X_{i2} + B_3 X_{i3} + E_i \]

In this regard, Apo proteins A and B are given to \( B_1 \), \( B_2 \), \( B_3 \) and \( B_0 \) mg/dl regression parameters and Apo proteins in pretest \( X_i \). The number defined for groups was \( X_{i0} \) for control group it was 1 and for the experimental group it was 0. The mutual effect of Apo proteins on group pretest was \( X_{i1} \) and \( X_{i2} \) and for the random error, it was \( E_i \). Calculated regression for experimental group was as follows:

\[ E(y)_1 = (B_0 + B_1) + (B_2 + B_3) X_{i2} \]

And for the control group, it was:

\[ E(y)_0 = B_0 + B_1 X_{i1} \]

**RESULTS**

Descriptive data from tests are presented in table 2. Yet, results found in other tables are demonstrating Apo protein A and B of the subjects in control and experimental groups.

**Table 2. Mean of minimum squares of Apo protein A in control and experimental groups with standard error and level of probability**

<table>
<thead>
<tr>
<th>Groups</th>
<th>Sd</th>
<th>Apo protein A Lsmeans (mg/dl)</th>
<th>sig</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>1.1185</td>
<td>119.783</td>
<td>0.0001</td>
</tr>
<tr>
<td>Experimental</td>
<td>1.1185</td>
<td>127.951</td>
<td></td>
</tr>
</tbody>
</table>

**Table 3. Mean of minimum squares of Apo protein B in control and experimental groups with standard error and level of probability**

<table>
<thead>
<tr>
<th>Groups</th>
<th>Sd</th>
<th>Apo protein B Lsmeans (mg/dl)</th>
<th>sig</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>1.0256</td>
<td>108.438</td>
<td>0.0006</td>
</tr>
<tr>
<td>Experimental</td>
<td>1.0256</td>
<td>102.362</td>
<td></td>
</tr>
</tbody>
</table>

The difference between control and experimental groups based on \( P = 0.0006 \) in the level of Apo protein B is significant.

**Table 4. Mean of minimum squares of Apo proteins A and B in pretest of control and experimental group with standard error and level of probability**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (mg/dl)</th>
<th>Sd</th>
<th>t</th>
<th>sig</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apo A1</td>
<td>6.0666</td>
<td>1.9284</td>
<td>3.15</td>
<td>0.0071</td>
</tr>
<tr>
<td>Apo A0</td>
<td>-4.8000</td>
<td>1.1514</td>
<td>-4.17</td>
<td>0.0009</td>
</tr>
</tbody>
</table>
Table 5. Mean, errors, T levels and level of probability of Apo proteins A and B difference in pretest and posttests of experimental group

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (mg/dl)</th>
<th>Sd</th>
<th>t</th>
<th>sig</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apo A1 - Apo A0</td>
<td>-2.0666</td>
<td>0.9878</td>
<td>-2.09</td>
<td>0.0551</td>
</tr>
<tr>
<td>Apo B1 - Apo B0</td>
<td>-2.6000</td>
<td>0.7091</td>
<td>3.67</td>
<td>0.0025</td>
</tr>
</tbody>
</table>

Table 6. Comparing the ratio of Apo proteins B/A in control and experimental groups

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Test step</th>
<th>B/A</th>
<th>B/A</th>
<th>Sig/d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>Pretest</td>
<td>95%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>Posttest</td>
<td>86%</td>
<td>9.3%</td>
<td>decrease</td>
</tr>
<tr>
<td>Experi</td>
<td>Pretest</td>
<td>79%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Experi</td>
<td>Posttest</td>
<td>83%</td>
<td>3.4%</td>
<td>increase 12.7%</td>
</tr>
</tbody>
</table>

Results related to Apo protein A:
Level of probability for B, B, and B was significant (P<0.05) the regression slope for both groups is not similar. The estimated regression for Apo A in control group is:

\[ \text{Apo}_A = 16.153 + 0.850413 \times \text{Apo}_{A0} \]

For experimental group it is:

\[ \text{Apo}_A = 52.7027 + 0.61753 \times \text{Apo}_{A0} \]

Figure 1. Apo protein A changes in both experimental groups depending on the level of Apo protein in pretests

DISCUSSION

Several studies have used the ratio of Apoprotein B to A as an important cardiovascular risk factor (Konstantinos et al., 2009; Parish et al., 2009). Researchers use different methods to change this ratio the most important of which is physical activities. However, findings on this issue are contradictory in many cases for they study different sports. This present study showed that a three-month general physical education two in college affects the level of Apo A of blood serum significantly (Table 2). The findings concur with findings of Greene et al., (2009) on the effects of a 24-week pedaling on Ergometer with 50 and 80% intensities of Vo\textsubscript{2max} on the changes in ratio of Apoproteins B/A. Several other studies finding similar results show that regular physical activities increase levels of HDL and decrease VLDL and LDL levels. This increase in HDL prevents cholesterol depositing in blood vessels (Segrest, 2013; Haram et al., 2009; Jenkins et al., 2010 and Michelle, 2009). Increasing HDL increases the level of Apo A as its major component (Ghorbanian et al., 2013; Song et al., 2012). Moreover, studies approve of the positive effects of physical activities and believe that physical activities significantly increase Apoprotein A and decrease Apoprotein B levels (Arazi, 2012). Furthermore, circular endurance and resistance exercises may have desirable effects on lipid profile, hematology and resting heart rate and offer a proper non-medicinal solution for deterring cardiovascular diseases and disorders induced by obesity in adolescent obese boys.

Several other studies have disproved of this idea (Ghanbari Niaki, 2014 and Poorveghar, 2013). They demonstrated that participating in activities with low intensities and short lengths will not increase Apoprotein A in non-athletes significantly (P=0.01). moreover, the difference in the levels of Apoprotein A in control and experimental groups was not significant (P=0.01), furthermore, some studies have revealed that intense and short activities in young inactive men does not affect weight and body composition and decrease VLDL density.
The present study, on the other hand, found a significant difference in the level of Apoproteins B of control and experimental groups in their pre and posttests (P=0.01). This difference in pre and posttests of the control group may be induced by the 3-month length of the training course (table 3). Various factors may affect the result in this length of time including other physical activities, nutrition and some hereditary factors. As it can be seen, trainings increased Apoprotein A slightly in the experimental group subjects in their posttest compared to the pretest (table 4). Despite the fact that increase in pre and posttests is +6.06666, control group experienced a +2.60000 decrease and this difference in the means of subjects in control and experimental groups have caused T= 3.15 and P= 0.0071. This may show that the training have affected the increase in Apoprotein A in the experimental group sufficiently and significantly (Table 4). This findings concord with findings of Mogharnasi et al., (2014); Poorveghar et al., (2014); Sheikh-al-Islami et al., (2011); Haram et al., (2009); Parish et al., (2009); Arthur S Leon (2009); Michelle et al., (2009) and Nikolas et al., (2009). They concluded that there is a significant difference between means of serum density of Apo lipoproteins A and B in pretest and after aerobic activities. At the same time, a significant difference was observed between mean density of serum triglyceride of the subjects and mean densities of high density cholesterol (HDL-C) and low density cholesterol (LDL-C) in pre and posttests. Accordingly, findings from this study show that in spite of observing increase in the level of Apoprotein A in experimental group, in case of Apoprotein B, like most of the studies mentioned above, no significant difference is seen in the level of Apoprotein B of the control and experimental groups in pre and posttests (P=0.01). Yet, the level of Apoproteins B in this group have decreased by -4.8000 (table 4). This difference in the level of Apoprotein B may have great effects on heart risks and decrease the probable dangers significantly (Mogharnasi et al., 2014; Sheikh-al-Islami, 2011). Data show that these activities significantly affect Apo protein B levels of the subjects under this study (table 4). However, this finding, contradicts with findings of Haram et al., (2009); Fontana et al., (2007) and Amy E et al., (2008). They demonstrated that in spite of the significant difference in the amount of Lipoprotein HDL and Apo protein B of the subjects after long-term training, levels of Apoprotein a does not change significantly. Nonetheless, the ratio of Apo protein B to A is a precise prognosticating factor of damages to heart muscle (Tsekouras et al., 2008; Ghanbari Niaki, 2013). This ratio has increased in subjects of control group by 83%/79%= 0.43 and decreased by 86%/95%= 9.3% in subjects of the experimental group. This ratio was 12.7% in case of A/B ratio of control and experimental groups (table 6). This difference is much higher than its standard level and may pose extreme dangers (Mercedes, 2009 and Ghanbari Niaki, 2013). This shows that despite expectations, these activities cannot be decent activities for decreasing cardiovascular risks (Terrados et al., 2010 and Poorveghar, 2013). Moreover, a significant decrease is observed in the level of Apo protein B (table 6). This finding concords with findings of Ghorbanian et al., (2013); Kim and Jung (2013); Arthur S. Leon (2009) and Matsios et al., (2008). They believe that in order to decrease the level of Apo protein B after long-term physical activities, other factors like, nutrition, number and length of sessions in a week, caloric expenditure, smoking and drugs (which were not under control in the present study) must be considered for caloric expenditure, body fat percentage, heredity and type of training are among the most important factors affecting the level of Apo protein A and B in pre and posttests of studies (Habib Zadeh & Rahman Nia, 2011 and Song et al., 2012). This finding does not concord with findings from studies by Poorveghar et al., (2014); Azarbaijani & Abedi (2012); Arazzi et al., (2012) and Parish et al., (2009). They demonstrated that the difference in mean densities of Apo lipoproteins A and B of serum after intense aerobic exercises. Besides, there is a significant difference in mean density of serum triglyceride of the subjects and mean densities of HDL-C and LDL-C before and after training (P=0.0001). It seems as if these trainings increase secretion of Catecholamine and sympathetic nerves' activity and decrease insulin secretion resulting in increase in growth hormones, cortisol, epinephrine and glucagon.Finally, this provides the ground for freeing fatty acids from fatty tissues (Shahsavar et al., 2011 and Poorveghar, 2014).

CONCLUSION

Overall results reveal that although three months of college football training with three sessions in a week is a regular and classic training program with somewhat low intensity, it increased the level of Apo protein A significantly and this could have positive effects on cardiovascular health. The effect of these trainings on the level of Apo protein B was decreasing and significant. The ratio of Apo B/Apo A gets closer to standard levels after its decrease. In conclusion, both results demonstrate that beneficial changes in biochemical, physiologic and body composition affect cardiovascular condition and decrease the risk of diseases. Change in lifestyle and adding activity to the inactive life could increase calorie expenditure and cardiovascular health condition would improve. In any case, variety of factors affecting Apo proteins A and B and levels of the indices of heart risks,
more precise studies with higher controls over these factors could give better insights into human health.

ACKNOWLEDGMENTS

Hereby the researcher would like to express his gratitude for the collaboration of all the participants in this study.

REFERENCES


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