Change in high-density lipoprotein cholesterol in response to acute resistance exercise

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Abstract

Several studies have reported improvements in blood lipid profiles following acute exercise bouts and following endurance training. The aim of this study was to determine the acute effects of resistance exercise on blood lipid variables in male and female adults. Thirty-three normal healthy subjects were divided into male (12 persons) and female (21 persons) groups. All subjects performed maximal resistance exercise (MRE) test. This test consisted of 3 sets of 7 exercises encompassing upper and lower body limbs at an intensity corresponding to 15 repetition maximum. Venous blood samples were obtained before and immediately after MRE test and analyzed for total cholesterol (TC), triglyceride (TG) and high density lipoprotein cholesterol (HDL-C). Blood TC and TG were unchanged whereas HDL-C rose significantly in response to MRE in both groups. Statistically, no gender differences in lipid profiles were observed at rest or in response to MRE test. It is concluded that maximal resistance exercise acutely increases HDL-C in male and female subjects.

Keywords: weight training, total cholesterol, triglyceride, high density lipoprotein cholesterol

Lipids and lipoproteins play a major role in the cascade of events leading up to the manifestations of atherosclerosis as it relates to coronary heart disease.\(^1\) It has been well documented that a variety of personal characteristics and environmental factors influence the composition of plasma lipids and lipoproteins, including age, gender, body composition, dietary intake, alcohol, smoking, medication used and physical exercise.\(^2\)

The effects of physical exercise on total cholesterol (TC), triglycerides (TG), and high density lipoprotein cholesterol (HDL-C) have been the subject of several studies.\(^3\)\(^4\) Prospective study indicated that individuals who undertake exercise on a regular basis, such as athletes, have low plasma TC and TG levels and a high concentration of HDL-C.\(^5\) However, whether this is due to differences in physical activity per se, or is a result of differences in other factors is unclear.

Resistance exercise has become a popular physical fitness activity, as evidenced by the increasing number of private and public weight-raining facilities. The inclusion of resistance exercise equipment for recreational lifters in various types of physical and cardiac rehabilitation program is widespread.\(^6\) The effects of resistance exercise on muscular strength, cardiorespiratory endurance, and body composition have been extensively studied and widely reported. Little attention, however, has been devoted to explore the effects of this kind of exercise on blood lipid variables in males and females. Therefore the present study was designed to examine the acute effects of resistance exercise on TG, TC, and HDL-C in adult male and female.
METHODS

Subjects

The study was conducted at the Division of Sport Sciences, Manchester Metropolitan University, England from January to March 1995. Thirty-three volunteer normal healthy subjects participated in this study. Experiments were carried out on 12 males and 21 females. Subjects were recruited by means of internal advertisements in various campus publications of the Crewe and Alsager Faculty, Manchester Metropolitan University. After being fully informed of the risks and stresses associated with the experiments, each volunteer signed an informed consent which have been approved by the Manchester Metropolitan University Ethics Committee. When subjects reported to the laboratory for testing, body mass and percentage body fat were determined. Percentage body fat was estimated from skin fold measurements as described by Durnin and Womersley.

Maximal resistance exercise testing

Subjects reported to the laboratory in pairs and were fed a standardized breakfast (50 g cereal and 100 ml of skimmed milk; energy 946 kJ, carbohydrate 52 g, protein 3.6 g, and fat 0.4 g). Maximal resistance exercise testing was conducted at the same time of day (08:00-12:00 hours).

The maximal resistance exercise test consisted of the completion of three sets of seven different exercises using resistance (weight) corresponding to 15 repetition maximum (15 RM). The 15-RM strength was determined for each exercise to obtain measures of maximal resistance exercise volume for the upper and lower body parts. The 15 RM was defined as the maximal weight that could be lifted 15 times through the full range of motion employing the correct techniques. Resistance exercises performed were bench press, leg press, latissimus pull down, prone leg curl, shoulder press, leg extension and standing biceps curl. Rest periods interspaced exercises (30 s) and sets (120 s). Exercise volume (kg) was calculated by multiplying the number of sets by the number of repetitions times the weight lifted per repetition.

Blood sampling and analytical procedures

Blood sampling and treatment of blood samples were carried out at the Physiology Laboratory, Division of Sport Sciences, the Manchester Metropolitan University, England by the researcher.

When subjects reported to the laboratory for testing, they were 12-hour fasted and had consumed no alcohol for the preceding 24 hour. Subjects were instructed to refrain from activity 12 hour preceding tests. They were requested to rest in supine position for 30 minutes followed by the removal of a 5 ml venous blood sample. Further blood sample were removed immediately after the maximal resistance exercise test. Blood was collected on chilled plastic tubes containing anticoagulant (EDTA). Aliquots of whole blood were used for lactate determination in duplicate (YSI 1500 L-Lactate Analyzer, Yellow Springs Instruments, Ohio). A small portion of blood was placed in a heparinized micro-hematocrit capillary tubes for the determination of hematocrit in triplicate (Hawksley method). Blood hemoglobin was measured in duplicate (HemoCue, Helsingborg, Sweden) and changes in plasma volume were determined from hematocrit and hemoglobin values. Total cholesterol and TG were determined in whole blood, while HDL-C was determined in plasma (Reflotron, Boehringer Mannheim, Germany).

Based on 12 determinations for single blood sample the coefficients of variation for TC, TG, and HDL-C were 1.9%, 1.4% and 2.9%, respectively. Accuracy of lipid measurements was ascertained using control sera. All lipid variables results after exercise were corrected for plasma volume changes.

Statistical analysis

The statistical analysis of the data were carried out using analysis of variance (ANOVA) with repeated measurements to compare metabolic, physiological as well as lipid variable at rest and in response to the maximal resistance exercise test between male and female groups. The alpha level of p < 0.05 was the minimum level required to reject the null hypothesis. The calculations were performed with the CSS statistical package (USA).

RESULTS

Physiological and metabolic responses

The mean and standard error (SE) values of plasma volume changes in response to the MRE test in males and females are presented in Table 1. Plasma volume feel by a value of -13.5% and -13.8% after performing the MRE test in males and females, respectively.

The result of blood lactate levels at rest and in response to the MRE test in males and females are presented in Table 2. Statistically no differences in the resting mean
values of blood lactate were found between males and females. The mean values of blood lactate increased significantly (p<0.05) after performing the MRE in both groups with a significantly (p<0.05) higher response in males compared to that observed in females.

Table 1. Physical characteristics of the subjects.

<table>
<thead>
<tr>
<th></th>
<th>Males (N=12)</th>
<th>Females (N=21)</th>
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<tbody>
<tr>
<td>Age (years)</td>
<td>22.1 ± 7.3</td>
<td>19.9 ± 5.2</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>64.4 ± 8.1*</td>
<td>56.4 ± 6.1</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>15.4 ± 1.1*</td>
<td>27.2 ± 1.6</td>
</tr>
<tr>
<td>Plasma volume loss</td>
<td>-13.5 ± 0.7</td>
<td>-13.8 ± 1.2</td>
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</table>

*significantly (p) higher than that observed in females.

Table 2. Mean(SE) values of blood lactate, total cholesterol (TC), triglyceride (TG) and high density lipoprotein cholesterol (HDL-C) levels at rest and in response to the maximal resistance exercise test in males.

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>Post MRE</th>
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<tbody>
<tr>
<td>Blood lactate (mmol L⁻¹)</td>
<td>Males 0.80 ± 0.30</td>
<td>6.30 ± 0.95*</td>
</tr>
<tr>
<td></td>
<td>Females 1.00 ± 0.46</td>
<td>5.10 ± 1.02*</td>
</tr>
<tr>
<td>TC (mmol.L⁻¹)</td>
<td>Males 3.80 ± 0.67</td>
<td>3.44 ± 0.73</td>
</tr>
<tr>
<td></td>
<td>Females 3.84 ± 0.67</td>
<td>3.67 ± 0.70</td>
</tr>
<tr>
<td>TG (mmol.L⁻¹)</td>
<td>Males 1.18 ± 0.21</td>
<td>1.07 ± 0.15</td>
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<tr>
<td></td>
<td>Females 1.40 ± 0.30</td>
<td>1.23 ± 0.33</td>
</tr>
<tr>
<td>HDL-C (mmol.L⁻¹)</td>
<td>Males 0.91 ± 0.18</td>
<td>1.17 ± 0.23*</td>
</tr>
<tr>
<td></td>
<td>Females 0.95 ± 0.17</td>
<td>1.16 ± 0.24*</td>
</tr>
</tbody>
</table>

*significantly (p) higher than that observed at rest.

Lipid variables at rest and in response to the maximal resistance exercise test

All of the thirty-three subjects showed normal lipid values at rest. The mean values of TC, TG, and HDL-C values for rest and in response to the MRE test in males and females are presented in Table 3.

Blood TC and TG values at rest or in response to MRE were unchanged (p>0.05) in male and female groups. No significant differences (p>0.05) in the resting mean values of plasma HDL-C concentration were found between males and females. The mean values of HDL-C showed, after data were adjusted for plasma volume reductions, a statistically significant (p<0.05) increase on after performing the MRE test in males and females.

DISCUSSION

Plasma volume changes in response to the maximal resistance exercise test

The maximal resistance exercise test resulted in a 13.5% and 13.8% reduction in plasma volume in males and females, respectively. The reduction in plasma volume during exercise could be attributed to an increase in arterial blood pressure which promotes filtration of plasma into the interstitial spaces. Knowlton et al.⁹ reported that elevation in mean arterial pressure is highly correlated with changes in plasma volume during weight-lifting. Plasma volume shifts are also influenced by osmotic gradients. During exercise the breakdown of glycogen, the production of lactate, and the accumulation of other metabolites in the active muscle increase intracellular osmolarity, which may then result in fluid fluxes from interstitial to intracellular and from vascular to interstitial space.¹¹

The effect of the maximal resistance exercise test on blood lactate levels

Statistically, there were no gender differences in resting blood lactate concentration. As expected, blood lactate concentration increased significantly (p<0.01) after performing the maximal resistance exercise test. This occurred both in males and females, with males having significantly greater blood lactate concentrations than females. This increase in blood lactate is indicative of the predominance of anaerobic glycolysis as an important source of energy for the re-synthesis of adenosine triphosphate (ATP) during resistance exercise. Blood lactate concentrations found after the maximal resistance exercise test in the present study are similar to those reported in study incorporating protocol with similar report.¹²

The acute effects of the maximal resistance exercise test on blood lipid variables

The resting values of blood lipid parameters (total cholesterol [TC], triglycerides [TG], and high-density lipoprotein cholesterol [HDL-C]) observed in the present study were within the normal range for healthy subjects.⁴

Total cholesterol showed statistically no significant change in response to the resistance exercise test, thus in keeping with previous studies employing endurance
exercise protocols, and low and high-volume resistance exercise. Using circuit weight-training, Lee et al. found a significant decrease in TC concentrations after completing a session of bench press, parallel squat, leg extension, and seated row.

In agreement with previous endurance exercise studies, TG concentration was unaltered after performing maximal resistance exercise. Similarly, Wallace et al. and Lee et al. demonstrated no change in TG levels after low and high-volume resistance exercise sessions and circuit weight-training, respectively. Unlike intramuscular stores of TG, which have been shown to contribute significantly to energy metabolism, the importance of blood-borne TG to energy metabolism during exercise has not been established and is still an issue of debate. Kuusi et al. used ultracentrifugation as a fractionation procedure for the separation of lipoproteins and demonstrated that the distribution of TG among different lipoproteins was altered in response to exercise, with no significant change in total TG level. It could be suggested, therefore, that TG played a negligible role during the exercise trials employed in the present study.

Studies of the effect of exercise on HDL-C have yielded contradictory results: HDL-C either increased or remained unaltered. Some reports showed an increase in HDL-C in response to acute submaximal exercise at 60% VO_2max or prolonged exercise for 120 minute at 30% VO_2max. In contrast, others have observed no significant increase in HDL-C after submaximal exercise with varying intensities. In the present study, HDL-C increased significantly after performing the maximal resistance exercise test. These findings are in keeping with previous studies demonstrating that acute high-volume, but not low-volume, resistance exercise alters serum lipid variables; such as an increase in lecithin cholesterol acetyl transferase activity, and HDL-C. Therefore, it is reasonable to suggest that a relationship exist between resistance exercise severity and HDL-C response. The mechanism responsible for the increase in high density lipoprotein cholesterol in response to resistance exercise is not known. Potential factors for the acute change in high density lipoprotein concentration may include their synthesis or degradation. Although no information was obtained in the present study to explain the mechanism by which resistance exercise alters HDL-C, other investigators have suggested that exercise might increase lipoprotein lipase. In human, endurance exercise of long duration resulted in an increase in lipoprotein lipase activity with a concomitant increase in HDL-C. This enzyme catalyses the breakdown of TG in lipoproteins. The breakdown products of this process then enter the blood as a precursor for the formation of HDL-C. Little information is known about lipoprotein lipase responses following resistance exercise. It is probable that resistance exercise, similar to endurance exercise, increases lipoprotein lipase and this may be one of the factors responsible for the increases in HDL-C.

Statistically no gender differences were observed in lipid profiles at rest or in response to the maximal resistance exercise test. Although these results disagree with some reports, they are partly in keeping with Lamon-Flava et al. who demonstrated a similar HDL-C increase in male and female athletes after a triathlon race. It should be noted, however, that Lamon Flava et al also found a significant decrease in TG after the same race in both males and females. The latter finding contrasts with the results of the present study. Other author have shown a significant decrease in TC concentrations in females but not in males after 40 minute cycling. It is apparent that comparison of the results of the above studies is difficult due to differences in exercise procedures, experimental protocols and subjects studied.

It was established that maximal resistance exercise test, similar to endurance exercise has a favorable effect on blood lipid profile, as indicated by an increase in HDL-C. Although no information was obtained in the present study to explain the mechanism by which resistance exercise alters HDL-C, other investigators have suggested that exercise might increase lipoprotein lipase.

Acknowledgments

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REFERENCES


