

Research article

Cardiorespiratory characteristics and cholesterol responses to a single session of heavy leg press exercise

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Abstract

The effect of resistance exercise on blood lipids is not clear yet. The purpose of this study was to examine the cholesterol responses to a heavy resistance leg press exercise emphasizing on the eccentric movement 24 and 48 hours following exercise and to quantify the cardiorespiratory responses of the exercise bout in an attempt to clarify the exercise characteristics that may be responsible for the effects of heavy resistance exercise on blood lipids. Nine healthy, untrained male volunteers aged 27.2 ± 1.1 yrs (76.2 ± 2.5 kg, 1.79 ± 0.02 m) performed a session of heavy RE emphasizing on the eccentric movement consisting of eight sets of inclined leg presses at six repetition maximum with 3-min rest intervals. Venous blood samples were obtained at rest (control) and 24 and 48 hours following exercise. Average $\dot{V}O_2$ at rest was 4.0 ± 0.4 ml·min⁻¹·kg⁻¹, during exercise 19.6 ± 0.2 ml·min⁻¹·kg⁻¹ and during the 180 sec recovery period between sets 12.5 ± 0.2 ml·min⁻¹·kg⁻¹. RER values decreased with the progression of the exercise and were significantly lower during the last four sets compared with the first four sets of the exercise session. Resting heart rate was 67 ± 2 bpm, and maximum heart rate during exercise was 168 ± 1 bpm. Serum creatine kinase was significantly elevated on day 1 (1090 ± 272 U·L⁻¹, $p < 0.03$) and peaked on day 2 (1230 ± 440 U·L⁻¹, $p < 0.01$). Total cholesterol, HDL cholesterol and calculated LDL cholesterol concentration did not change significantly following with exercise. This protocol of heavy resistance exercise has no effect on TC or cholesterol sub-fraction concentration 24 and 48 hours following exercise which may be due to the low energy expenditure of the exercise and/or to the gender of the participants.

Key words: Muscle damage, energy expenditure, total cholesterol, HDL, oxygen uptake.

Introduction

The use of resistance exercise (RE) to improve health-related fitness has increased considerably over the past few years (Centers for Disease Control and Prevention, 2006). The American College of Sports Medicine has recommended RE as an effective means of improving muscular strength and endurance, fat-free mass, and bone mineral density (Pollock et al., 1998). Also, RE is recommended by the American Heart Association as complementary to aerobic exercise to reduce cardiovascular disease risk factors (Williams et al., 2007). Previous studies have shown that RE can reduce postprandial lipemia 16 h postexercise (Pafili et al., 2009; Petitt et al., 2003; Zafeiridis et al., 2007), but the effect of RE on total cholesterol (TC) and cholesterol sub fractions concentration remains controversial. There is limited evidence concerning the acute effect of RE on TC, but most studies have

failed to show an effect (Hill et al., 2005; Jurimae et al., 1990; Magkos et al., 2008; Wallace et al., 1991). However, when eccentric contractions are performed and muscle damage ensues, TC has been reported to decrease not immediately after, but in the following one to four days post-exercise (Nikolaidis et al., 2008; Paschalis et al., 2010; Shahbazzpour et al., 2004; Smith et al., 1994). All these authors partly attributed this to the extraction of cholesterol from the blood in order to assist in the resynthesis of disrupted muscle cell membranes.

However, the exact mechanisms are not known and it remains speculative whether this effect is due to muscle damage or to the exercise energy expenditure. When examining the relevant literature, one problem in comparing the effects of different RE protocols on blood lipids is that the characteristics of the exercise are not adequately quantified and described and also vary significantly between studies (Durstine and Haskell, 1994). Even though there is evidence that the effect of RE on blood lipids depends on exercise intensity and volume (Lira et al., 2009; Tucker and Silvester, 1996), the few studies that examined the effect of eccentric RE on TC and cholesterol sub-fraction concentration (Nikolaidis et al., 2008; Paschalis et al., 2010; Shahbazzpour et al., 2004; Smith et al., 1994). have not measured energy expenditure or other cardiorespiratory responses. Also two of the aforementioned studies have not controlled (Smith et al., 1994) or inadequately controlled (Shahbazzpour et al., 2004) the participants' diet the days preceding the blood sampling and therefore their results cannot be safely attributed to the effect of exercise or to a possible change in participants' dietary intake.

Therefore, the purpose of this study was to examine the cholesterol responses to a heavy leg press exercise session emphasizing on the eccentric movement previously shown to reduce fasting TAG 40 hours postexercise (Pafili et al., 2009). Since the effects of exercise on TC may not be obvious earlier than 24 h following exercise (Durstine and Huskel, 1994; Nikolaidis et al., 2008; Paschalis et al., 2010) we present unpublished data regarding the effects of a heavy resistance exercise emphasizing on the eccentric movement protocol on total cholesterol and cholesterol sub-fraction concentration up to 48 h post exercise. Another purpose of the study was to quantify and present the cardiorespiratory responses of the exercise bout.

Methods

Participants

Nine male volunteers (age: 27.2 ± 1.1 yr, body mass: 76.2 ± 2.5 kg, height: 1.79 ± 0.02 m) took part in the study, which was approved by the institutional ethics committee. Participants gave written consent after being informed about the procedures, the risks involved, and their right to terminate participation at will. All procedures in this study were in accordance with the Helsinki declaration of 1975, as revised in 1996. Participants were nonsmokers, without known history of cardiovascular disease, and were not taking any medication or nutritional supplements known to affect lipid or carbohydrate metabolism. In addition, participants were selected only if they had refrained from regular RE for the past 12 months. None of the participants performed more than $2 \text{ h}\cdot\text{wk}^{-1}$ of recreational physical activities, (e.g., walking, cycling, swimming) as reported in personal interviews.

Study design

Each participant took part in two conditions separated by at least one week in random order. In the control condition participants consumed a standardized meal at 8:00 AM and seven hours later (15:00 AM) a baseline blood sample was obtained for the measurement of cholesterol and cholesterol sub-fraction concentration. On a separate day, one week later, the resistance exercise protocol was performed. The exercise protocol was timed so that it would finish at 15:00 AM. This was done so that the 24 and 48 h post-exercise blood samples were obtained at the same time of the day as in the control condition (15:00 AM) and after the consumption of the standardized meal at 8:00 AM on both post-exercise days.

Resistance exercise protocol

To avoid the accustomization of the participants with eccentric exercise and the ensuing muscle damage, i.e. the so called "repeated bout effect", (Clarkson and Hubal, 2002), the load corresponding to 6 RM was calculated for each individual using the fat-free thigh cross sectional area (CSA). On the day of the resistance exercise trial, the lean thigh cross sectional area was estimated from mid thigh circumference and the corresponding anterior thigh skinfold thickness, using a previously validated regression equation developed by Housch et al. (1995):

$$\text{CSA} = (4.68 \times \text{mid thigh circumference (cm)} - (2.09 \times \text{anterior thigh skinfold (mm)}) - 80.99.$$

Then, the individual 6 RM load was calculated from CSA using the regression equation for untrained individuals developed by Dolezal et al. (2000):

$$\text{Load} = 1.231 \times \text{CSA} + 173.664.$$

The predicted 6-RM weight was used for the first set and the participant was instructed to perform as many repetitions as possible. If the number of repetitions was different from six, then the load for the next set was adjusted according to the equation provided by Brzycki (1993):

$$1 \text{ RM} = \frac{\text{weight (kg)}}{1.0278 - (0.0278 \times \text{number of repetitions})}$$

Care was taken so that the eccentric phase of the movement was emphasized.

The resistance exercise protocol was preceded by a standardized warm-up (5 min jogging on a treadmill at $8 \text{ Km}\cdot\text{h}^{-1}$ and 5 min of stretching). Participants performed eight sets of six repetitions of leg presses that emphasized the eccentric movement the 6- RM load calculated from their CSA on a 45° inclined leg press machine. For each repetition, they were instructed to lower the weights in 4 s and to perform a forceful push when reversing from the eccentric to the concentric movement. This was done to maximize the eccentric load. A metronome was used to ensure that the 4 s phase was timed. A three minute break was used between sets. Two spotters ensured that the required repetitions were executed in all sets by assisting during the concentric phase when the participant was unable to lift the weight. The mechanical stops of the machine were adjusted so that knee angle (between femur and tibia) at the bottom of the downward movement was 60° . This exercise protocol has been used previously (Dolezal et al., 2000).

Throughout the exercise and for 3 min into the recovery, expired air was analyzed breath-by-breath using (Cosmed K4b² portable metabolic unit) and data were averaged every 5 s by the spirometer software. The spirometer was calibrated before each exercise protocol.

Cardiorespiratory measurements and energy expenditure estimation

For the estimation of energy expenditure, aerobic plus anaerobic energy were summed, while protein contribution was ignored. Aerobic energy was calculated using an equivalent of 21.1 kJ per litre of oxygen consumed during exercise and 19.6 kJ per litre of oxygen consumed during recovery, while anaerobic energy was estimated by converting the increase in lactate concentration (from rest to the peak value) to oxygen equivalents assuming that one $\text{mmol}\cdot\text{l}^{-1}$ of lactate is equal to $3 \text{ ml O}_2 \text{ kg}^{-1}$ body mass, oxygen values was then converted to kilojoules as 21.1 kJ per liter of O_2 (Scott, 2006).

Heart rate was continuously recorded using a Polar S610i heart rate monitor, and the recorded data were analysed with Polar Precision Performance SW Software (Version 4.00.024, Copyright Polar Electro Oy 2003, www.polar.fi).

Blood sampling and analysis

Duplicate 20 μL capillary blood samples were taken before exercise, in the middle of the rest period between sets 4 and 5, immediately after the last set and three minutes into recovery. Samples were deproteinized in 0.3 M perchloric acid and lactate was measured enzymatically at a later date (Sigma Diagnostics, St Louis, MO, USA).

Post-absorptive (7 hours after a standardized meal) blood samples were obtained in the control condition as well as after 24 and 48 hours after exercise. Samples (5 ml each) were taken from an antecubital vein and were placed in non-heparinized tubes to clot for 1 h. Serum was stored at -20°C and was later analyzed for triglyceride, total cholesterol (TC), high density lipoprotein cholesterol (HDL-C) and creatine kinase (CK) using an automated analyzer (ACE[®] Clinical Chemistry System, Alfa Wasserman, Inc, NJ, USA). Biochemical parameters were measured in duplicate. Intraassay coefficients of variation

were as follows: lactate 3.1%, TAG, 1.9%; TC, 1.0%; HDL-C, 1.6% and CK, 2.0%. LDL was calculated according to the Friedewald formula (Friedewald et al, 1972).

Control of diet and exercise

To avoid the effect of previous diet and exercise on blood lipid values participants were asked to refrain from any form of physical exercise for 2 d before each condition, and to record their diet for 2 d prior to the first blood sample. This diet was replicated for 2 d before the other condition 1 wk later. In the exercise condition that required blood sampling for two consecutive days (24 and 48 h following exercise), food intake for the rest of the first day following exercise was prescribed individually so that the total daily energy and macronutrient intake for each participant was the same as in the previous day. By doing this, energy intake and diet composition were standardized for the day before each blood sample. Additionally, the last meal before each data collection day was administered according to body mass 7 hours before blood sampling. The cholesterol content of that meal was 0.42 mg/kg body mass.

Statistical analysis

Data were analysed using one-way ANOVA and differences between means were located using Tukey's post hoc tests. Data was analysed using STATISTICA for Windows StatSoft, Inc. (Release 5.0, Tulsa, OK). Significance was accepted at the $p < 0.05$ level, and data are presented as mean \pm SE, unless otherwise stated.

Results

The total exercise duration was 25.4 ± 0.2 min. The average weight lifted per contraction was 2.80 ± 0.21 kg/kg body mass while the average angular velocity of the knee joint movement during the eccentric phase was $30^\circ \cdot s^{-1}$ (as calculated from the total change in knee angle (from 180° to 60°) in the timed period of 4 s.) Total exercise energy expenditure (from the beginning to the end of exercise) was 136.72 ± 7.11 Kcal (7.86 Kcal \cdot min $^{-1}$). Average energy expenditure during the 8 sets of exercise, the subsequent eight 3 min recovery periods and exercise + recovery is shown in Figure 1. The average lactate contribution to energy expenditure during exercise was 13.63 ± 1.70 Kcal.

The average oxygen consumption and the average metabolic energy equivalents (METs) during rest, the 8 sets of exercise, the subsequent 3 min recovery periods and exercise + recovery are shown in Figure 2. Since pure exercise duration per set was only about 30 s and the recovery period per set was 3 min, the arithmetic means of pure exercise + recovery oxygen consumption and MET's differ from the exercise + recovery values. The time course of oxygen uptake, CO₂ output and RER during the whole exercise session is shown in Figure 3. At this point it must be noted that the protocol of the study required the participants to perform as many repetitions as possible against a calculated 6RM load for the first exercise test. In fact, the number of repetitions performed in the first set was 13.6 ± 2.8 and this increased the duration of the first set to about 59.5 sec. Then the load for the

next sets was readjusted to the actual individual 6RM. The increased number of repetitions performed in the first set, resulted in significantly higher oxygen uptake and CO₂ production during that set (Figure 3). Thereafter oxygen consumption remained unchanged throughout the test protocol (Figure 3). However, RER values decreased with the progression of the exercise and were significantly lower during the last four sets compared with the first four sets of the exercise session both during exercise (1.12 ± 0.06 vs. 1.00 ± 0.05 for first and last four sets respectively, $p < 0.01$), and recovery (1.48 ± 0.07 vs. 1.15 ± 0.05 for first and last four sets respectively, $p < 0.01$).

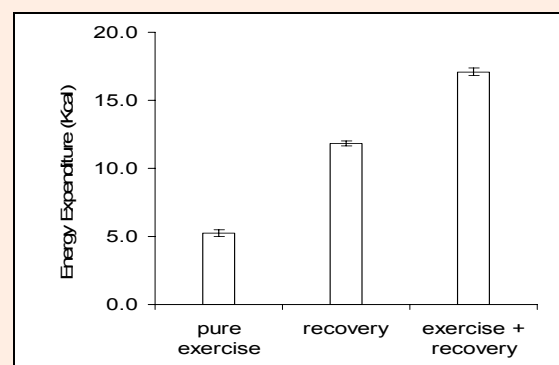


Figure 1. Average energy expenditure during the eight sets of exercise (pure exercise), the subsequent eight 3 min recovery periods and exercise + recovery. Values are means (\pm SE). (n = 9).

Average heart rate during the exercise session is shown in Figure 4. Rest average HR was 67 ± 2 bpm. Maximum heart rate during exercise was 168 ± 1 bpm. During exercise periods average HR was 152 ± 2 bpm, while during recovery periods average HR was 133 ± 1 bpm. The intensity of exercise as determined by % predicted HR max (as calculated from the formula: HR max = $220 - \text{age (yrs)}$), was $78.2 \pm 2.1\%$ HR max.

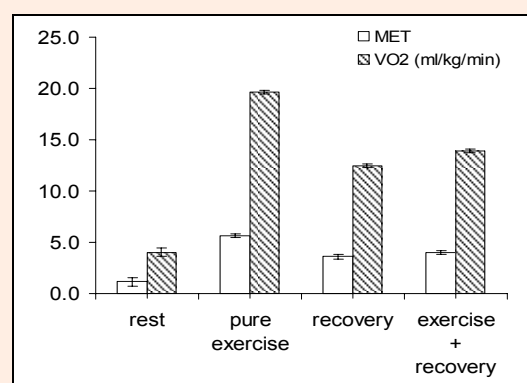


Figure 2. Average oxygen consumption and metabolic equivalents (MET) during rest, the 8 sets of exercise (pure exercise), the subsequent 3 min recovery periods and exercise + recovery. Values are means (\pm SE). (n = 9).

CK concentration was significantly higher on both days after exercise compared with the control value (197 ± 57 vs. 1090 ± 272 , $p < 0.03$ and 1230 ± 440 U \cdot L $^{-1}$ $p <$

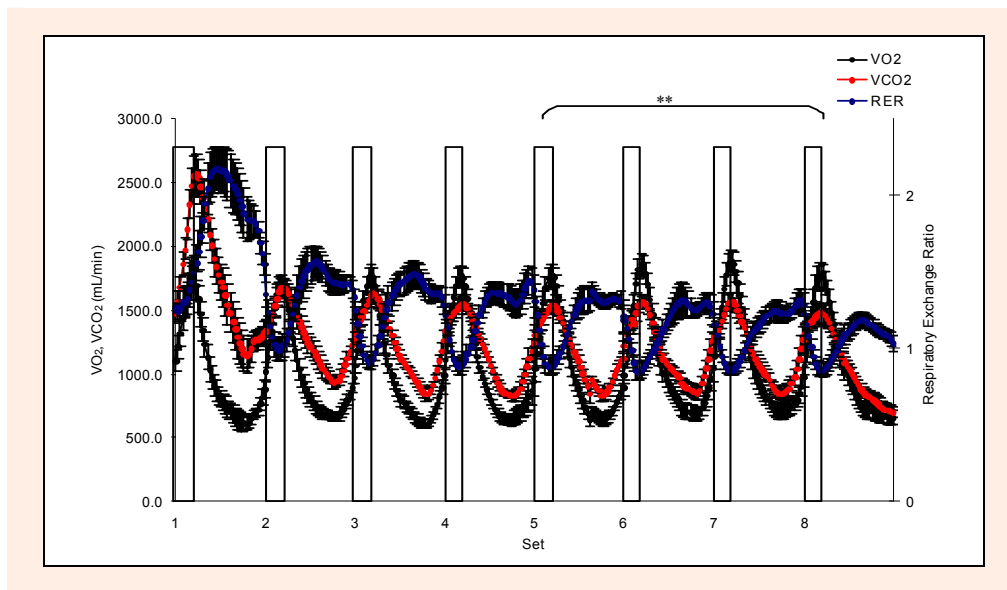


Figure 3. Average VO₂, VCO₂ and RER during the exercise session. Vertical bars show exercise periods. Values are means ± SE. (N=9), ***P* < 0.01 from the first four sets (RER).

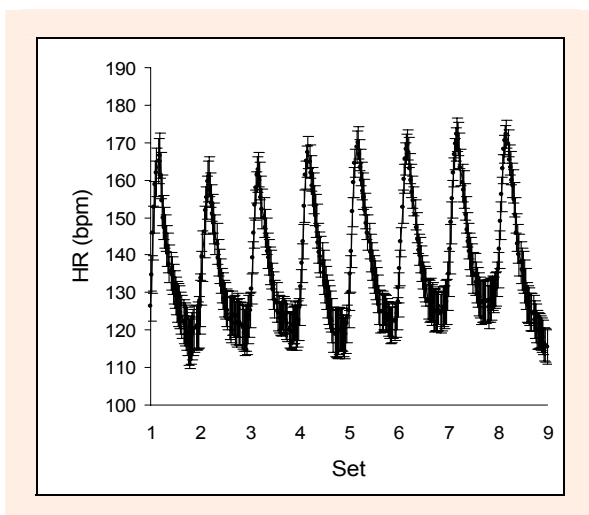


Figure 4. Average heart rate (bpm) during the exercise session. Values are means (± SE). (n = 9).

0.01, for control, 24h and 48h following exercise, respectively).

TC, HDL cholesterol and calculated LDL cholesterol concentration did not change significantly over the 48 hours following exercise (Figure 5).

Discussion

The main findings of this study were that this type of heavy strength exercise increased energy expenditure from aerobic sources to relatively high levels, considering the actual work done. Furthermore, total cholesterol and its sub-fractions HDL-C and LDL-C were not affected by this exercise protocol that caused considerable muscle damage.

The characteristics of this exercise program were typical of a maximal strength training session with heavy load (6 RM) and long rest periods (3 min) (Kraemer and Ratamess, 2004; Kraemer et al., 1987). The METs

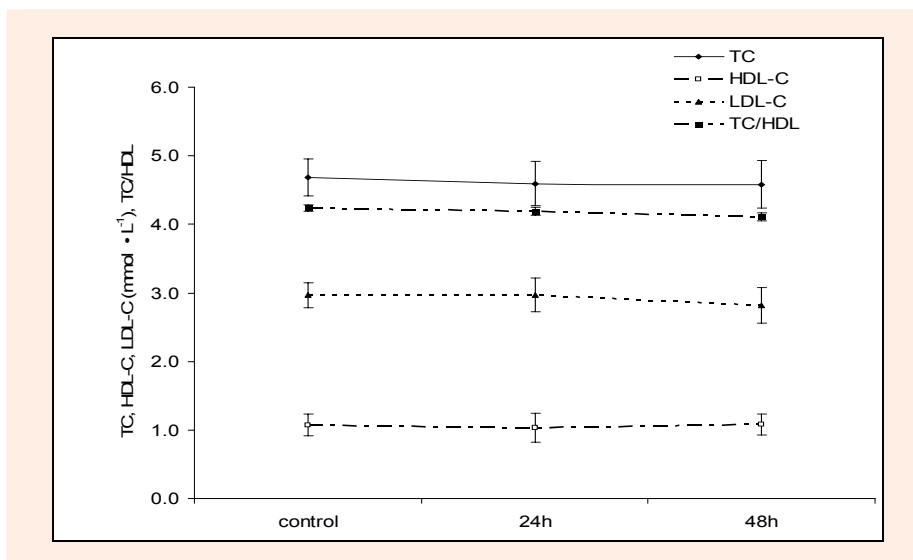


Figure 5. TC, HDL-C and calculated LDL-C concentration in the control condition and 24 and 48 h post-exercise. Values are means (± SE). (n = 9).

expended during exercise were equivalent to bicycling at <10mph (Ainsworth et al., 2000), and according to the oxygen uptake, exercise may be considered as “moderate intensity” according to ACSM's guidelines for aerobic exercise (Haskell et al., 2007). However, due to the nature of resistance exercise, HR responses were significantly higher than those of aerobic exercise of similar oxygen uptake (Braun et al., 2005). According to the energy calculations from VO_2 and blood lactate, this heavy RE session increased energy expenditure to relatively high levels, considering that only 50 repetitions were performed during the 8 exercise sets. It must be noted that the largest part of the aerobic energy expenditure was done during the recovery periods where oxygen uptake remained elevated.

Although a previous report from our laboratory showed a decrease in postprandial lipemia 16 hours after this exercise protocol (Pafili et al., 2009), TC concentration remained unchanged at both 24 and 48 h postexercise compared with rest. These results are in agreement with the results of previous studies of resistance exercise (Hill et al., 2005; Jurimae et al., 1990; Magkos et al., 2008; Wallace et al., 1991), and with results from studies of eccentric resistance exercise (Nikolaidis et al., 2008; Paschalis et al., 2010) as far as 24 h postexercise values are concerned. However, in the study of Nikolaidis et al. (2008) TC concentration was significantly reduced 48 h postexercise following the first exercise bout. This was attributed to a possible use of blood cholesterol for the repair of damaged muscle membranes, and this argument was reinforced by the fact that when the same exercise was performed again after 24–30 days there was no change in TC and this was accompanied with much less muscle damage, i.e. the “repeated bout effect” (Nikolaidis et al., 2008). In the other study by the same research group the more pronounced effect of the exercise on TC concentration in overweighted participants were also attributed to greater muscle damage (Paschalis et al., 2010).

The hypothesis of muscle membrane repair using blood cholesterol is supported by recent studies that examined the gene expression profiling in human skeletal muscle during recovery from eccentric exercise. These studies show that following eccentric exercise a number of genes involved in lipid metabolism are activated, some of them potentially promoting cholesterol synthesis in order to fix the disrupted membranes (Mahoney et al., 2008). However, muscle damage in the present study as well as in the other relevant studies was quantified with indirect indices, and thus the results may not be directly comparable. The indirect index of plasma CK concentration was lower in the present study compared with the study of Paschalis et al. (2010), but greater compared with the study of Nikolaidis et al. (2007). Therefore, a safe conclusion cannot be drawn regarding the possible effect of the degree of muscle damage on the serum concentration of TC and its sub-fractions. Furthermore, the possible effects of energy expenditure on TC concentration can not be compared with previous studies, since none of them measured energy expenditure.

Another factor that may have affected the responses of the participants in the present study is the

longer rest interval employed (3 min) compared with the other studies that have used shorter rest intervals: 2 min Smith et al. (1994), 1 min Shahbazpour et al. (2004), 2 min Nikolaidis et al. (2008) and Paschalis et al. (2010). Rest-interval length could affect the lipoprotein response to exercise since it has been shown that RE with shorter rest intervals affects more the post exercise leukocyte levels (Mayhew et al., 2005), and the hormonal responses to exercise (Bottaro et al., 2009; Buresh et al., 2009), compared to the same exercise with longer rest intervals. The increase in leukocyte levels after exercise is considered a part of the inflammatory response to injured muscle cells (Nieman, 1997) and there is evidence to suggest that it is intermediated by exercise-dependent secretion of stress hormones (Suzuki et al., 1999). Both inflammation and increased hormone secretion following exercise have been shown to affect lipoprotein levels (Frey et al., 1983; Khovidhunkit et al., 2004), so a greater increase in both these parameters could also mean a greater effect on blood lipoproteins.

It must also be noted that insulin resistance observed after eccentric exercise (Asp et al., 1996; Kirwan et al., 1992; Pafili et al., 2009) could increase blood cholesterol, since it has been associated with increased cholesterol synthesis and decreased cholesterol absorption in normoglycemic men (Pihlajamäki et al., 2004). This may counterbalance the possible reduction of TC due to the repair of damaged muscle membranes.

Another interesting possible explanation for the differences between our study and the studies of Nikolaidis et al. (2008) and Paschalis et al. (2010) is the interaction between resistance exercise and gender of the participants. Both those studies used female participants whereas in the present study the participants were males. Recent reviews (Tambalis et al., 2009) and meta-analyses of previous studies (Kelley and Kelley, 2009) suggest that the only studies that have shown an effect of RE training on blood TC concentration had female and elderly participants. Since there are studies that have shown different responses between men and women as far as the acute effects of eccentric RE are concerned (Rinard et al., 2000; Sewright et al., 2008), and even though hormonal responses have not been measured in response to eccentric resistance exercise in women, evidence from aerobic exercise studies suggest that men and women have different hormonal responses to exercise (Davis et al., 2000), so gender may be one important factor for changes in blood TC with heavy RE.

Previous studies have shown an increase of HDL concentration 24 h following RE only when exercise was of high volume and of longer duration than the present study (e.g. 90 min, Wallace et al., 1991). This fact, combined with evidence from aerobic studies showing that exercise energy expenditure needs to be at least 350 Kcal in order to have an effect on HDL concentration (Thompson et al., 2001) suggests that the energy expenditure of our protocol was not sufficient to affect HDL concentration.

Conclusion

In conclusion, a small volume of heavy resistance exer-

cise that caused muscle damage had no effect on TC and cholesterol subfraction concentration 24 and 48 hours after exercise, even though it has previously been shown to reduce postprandial triglyceride concentration 16 hours following exercise. This is in contrast with the few studies that reported a decrease in TC in two up to five days after muscle damaging exercise. The lack of an effect of exercise on TC and HDL-C in the present study may be due to a lower degree of muscle damage, lower energy expenditure or a possible effect of gender on the TC responses. Our results point out the necessity of future studies to control for exercise characteristics and take into account factors such as participants' gender, diet and hormonal responses that could affect the cholesterol response to exercise, in order to draw a sound conclusion regarding the effects of resistance exercise on blood lipids.

References

- Ainsworth, B.E., Haskell, W.L., Whitt, M.C., Irwin, M.L., Swartz, A.M., Strath, S.J., O'Brien, W.L., Bassett, D.R. Jr, Schmitz, K.H., Emplancourt, P.O., Jacobs, D.R. Jr and Leon, A.S. (2000) Compendium of Physical Activities, an update of activity codes and MET intensities. *Medicine and Science in Sports and Exercise* **32**, S498–S516.
- Asp, S., Daugaard, J.R., Kristiansen, S., Kiens B. and Richter, E.A. (1996) Eccentric exercise decreases maximal insulin action in humans, muscle and systemic effects. *Journal of Physiology* **494**(3), 891–898.
- Bottaro, M., Martins, B., Gentil, P. and Wagner, D. (2009) Effects of rest duration between sets of resistance training on acute hormonal responses in trained women. *Journal of Science and Medicine in Sport* **12**(1), 73–78.
- Braun, W.A., Hawthorne, W.E. and Markofski, M.M. (2005) Acute EPOC response in women to circuit training and treadmill exercise of matched oxygen consumption. *European Journal of Applied Physiology* **94**, 500–504.
- Brzycki, M. (1993) Strength testing—predicting a one-rep max from reps-to-fatigue. *Journal of Physical Education Recreation and Dance* **68**, 88–90.
- Buresh, R., Berg, K. and French, J.J. (2009) The effect of resistive exercise rest interval on hormonal response, strength, and hypertrophy with training. *Journal of Strength and Conditioning Research* **23**(1), 62–71.
- Centers for Disease Control and Prevention. (2006) Trends in strength training—United States, 1998–2004. *Morbidity and Mortality Weekly Report* **55**(28), 769–72.
- Clarkson, P.M. and Hubal, M.J. (2002) Exercise-induced muscle damage in humans. *American Journal of Physical Medicine and Rehabilitation* **81**, S52–S69.
- Davis, S.N., Galassetti, P., Wasserman, D.H. and Tate, D. (2000) Effects of gender on neuroendocrine and metabolic counterregulatory responses to exercise in normal man. *Journal of Clinical Endocrinology and Metabolism* **85**(1), 224–230.
- Dolezal, B.A., Potteiger, J.A., Jacobsen, D.J. and Benedict, S.H. (2000) Muscle damage and resting metabolic rate after acute resistance exercise with an eccentric overload. *Medicine and Science in Sports and Exercise* **32**(7), 1202–1207.
- Durstine, J.L., Grandjean, P.W., Davis, P.G., Ferguson, M.A., Alderson, N.L. and DuBose, K.D. (2001) Blood lipid and lipoprotein adaptations to exercise, a quantitative analysis. *Sports Medicine* **31**(15), 1033–1062.
- Durstine, L.J. and Haskell, W.L. (1994) Effects of exercise training on plasma lipids and lipoproteins. *Exercise and Sports Science Review* **22**, 477–521.
- Frey, M.A., Doerr, B.M., Srivastava, L.S. and Glueck, C.J. (1983) Exercise training, sex hormones, and lipoprotein relationships in men. *Journal of Applied Physiology* **54**(3), 757–762.
- Friedewald, W.T., Levy, R.I. and Fredrickson, D.S. (1972) Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clinical Chemistry* **18**(6), 499–502.
- Haskell, W.L., Lee, I.M., Pate, R.R., Powell, K.E., Blair, S.N., Franklin, B.A., Macera, C.A., Heath, G.W., Thompson, P.D. and Bauman, A. (2007) Physical activity and public health, updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Medicine and Science in Sports and Exercise* **39**(8), 1423–1434.
- Hill, S., Bermingham, M.A. and Knight, P.K. (2005) Lipid metabolism in young men after acute resistance exercise at two different intensities. *Journal of Science and Medicine in Sport* **8**(4), 441–445.
- Housch, D.J., Housch, T.J., Weir, J.P., Weir, L.L., Johnson, G.O. and Stout, J. R. (1995) Anthropometric estimation of thigh muscle cross-sectional area. *Medicine and Science in Sports and Exercise* **27**(5), 784–791.
- Jurimae, T., Karelson, K., Smirnova, T. and Viru, A. (1990) The effect of a single-circuit weight-training session on the blood biochemistry of untrained university students. *European Journal of Applied Physiology and Occupational Physiology* **61**(5–6), 344–348.
- Kelley, G.A. and Kelley, K.S. (2009) Impact of progressive resistance training on lipids and lipoproteins in adults, a meta-analysis of randomized controlled trials. *Preventive Medicine* **48**(1), 9–19.
- Kirwan, J.P., Hickner, R.C., Yarasheski, K.E., Kohrt, W.M., Wiethop, B.V. and Holloszy, J.O. (1992) Eccentric exercise induces transient insulin resistance in healthy individuals. *Journal of Applied Physiology* **72**(6), 2197–2202.
- Khovidhunkit, W., Kim, M.S., Memon, R.A., Shigenaga, J.K., Moser, A.H., Feingold, K.R., and Grunfeld, C. (2004) Effects of infection and inflammation on lipid and lipoprotein metabolism: mechanisms and consequences to the host. *Journal of Lipid Research* **45**, 1169–1196.
- Kraemer, W.J. and Ratamess, N.A. (2004) Fundamentals of resistance training, progression and exercise prescription. *Medicine and Science in Sports and Exercise* **36**(4), 674–688.
- Kraemer, W.J., Noble, B.J., Clark, M.J. and Culver, B.W. (1987) Physiologic responses to heavy-resistance exercise with very short rest periods. *International Journal of Sports Medicine* **8**(4), 247–252.
- Lira, F.S., Zanchi, N.E., Lima-Silva, A.E., Pires, F.O., Bertuzzi, R.C., Santos, R.V., Caperuto, E.C., Kiss, M.A. and Seelaender M. (2009) Acute high-intensity exercise with low energy expenditure reduced LDL-c and total cholesterol in men. *European Journal of Applied Physiology* **107**(2), 203–210.
- Magkos, F., Tsekouras, Y.E., Prentzas, K.I., Basioukas, K.N., Matsama, S.G., Yanni, A.E., Kavouras, S.A. and Sidossis, L.S. (2008) Acute exercise-induced changes in basal VLDL-triglyceride kinetics leading to hypotriglyceridemia manifest more readily after resistance than after endurance exercise. *Journal of Applied Physiology* **105**, 1228–1236.
- Mahoney, D.J., Safdar, A., Parise, G., Melov, S., Fu, M., MacNeil, L., Kaczor, J., Payne, E.T. and Tarnopolsky, M.A. (2008) Gene expression profiling in human skeletal muscle during recovery from eccentric exercise. *American Journal of Physiology, Regulatory and Integrative and Comparative Physiology* **294**, 1901–1910.
- Mayhew, D.L., Thyfault, J.P. and Koch, A.J. (2005) Rest-interval length affects leukocyte levels during heavy resistance exercise. *Journal of Strength and Conditioning Research* **19**(1), 16–22.
- Nieman, D.C. (1997) Immune response to heavy exertion. *Journal of Applied Physiology* **82**(5), 1385–1394.
- Nikolaidis, M.G., Paschalis, V., Giakas, G., Fatouros, I.G., Koutedakis, Y., Kouretas, D. and Jamurtas, A.Z. (2007) Decreased blood oxidative stress after repeated muscle-damaging exercise. *Medicine and Science in Sports and Exercise* **39**(7), 1080–1089.
- Nikolaidis, M.G., Paschalis, V., Giakas, G., Fatouros, I.G., Sakellariou, G., Theodorou, A.A., Koutedakis, Y. and Jamurtas, A. (2008) Favorable and prolonged changes in blood lipid profile after muscle-damaging exercise. *Medicine and Science in Sports and Exercise* **40**(8), 1483–1489.
- Pafili, Z.K., Bogdanis, G.C., Tsetsonis, N.V., and Maridaki, M. (2009) Postprandial lipemia 16 and 40 hours after low-volume eccentric resistance exercise. *Medicine and Science in Sports and Exercise* **41**(2), 375–382.
- Petit, D.D., Sigurborn, A., Arngrimsson, A. and Cureton, K.J. (2003) Effect of resistance exercise on postprandial lipemia. *Journal of Applied Physiology* **94**(2), 694–700.

- Paschalis, V., Nikolaidis, M.G., Giakas, G., Theodorou, A.A., Sakellariou, G.K., Fatouros, I.G., Koutedakis, Y. and Jamurtas, A.Z. (2010) Beneficial changes in energy expenditure and lipid profile after eccentric exercise in overweight and lean women. *Scandinavian Journal of Medicine and Science in Sports* **20**(1), 103-111.
- Pihlajamäki, J., Gylling, H., Miettinen, T.A. and Laakso, M.J. (2004) Insulin resistance is associated with increased cholesterol synthesis and decreased cholesterol absorption in normoglycemic men. *Lipid Research* **45**(3), 507-512.
- Pollock, M.L., Gaesser, G.A., Butcher, J.D., et al. (1998) American College of Sports Medicine position stand on the recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in adults. *Medicine and Science in Sports and Exercise* **30**(6), 975-991.
- Rinard, J., Clarkson, P.M., Smith, L.L. and Grossman, M. (2000) Response of males and females to high-force eccentric exercise. *Journal of Sports Sciences* **18**(4), 229-236.
- Scott, C.B. (2006) Contribution of blood lactate to the energy expenditure of weight training. *Journal of Strength and Conditioning Research* **20**(2), 404-411.
- Sewright, K.A., Hubal, M.J., Kearns, A., Holbrook, M.T. and Clarkson, P.M. (2008) Sex differences in response to maximal eccentric exercise. *Medicine and Science in Sports and Exercise* **40**(2), 242-251.
- Shahbazzpour, N., Carroll, T.J., Riek, S. and Carson, R.G. (2004) Early alterations in serum creatine kinase and total cholesterol following high intensity eccentric muscle actions. *Journal of Sports Medicine and Physical Fitness* **44**(2), 193-199.
- Smith, L.L., Fulmer, M.G., Holbert, D., McCammon, M.R., Houmar, J.A., Frazer, D.D., Nsien, E. and Israel, R.G. (1994) The impact of a repeated bout of eccentric exercise on muscular strength, muscle soreness and creatine kinase. *British Journal of Sports Medicine* **28**(4), 267-271.
- Suzuki, K., Totsuka, M., Nakaji, S., Yamada, M., Kudoh, S., Liu, Q., Sugawara, K., Yamaya, K. and Sato, K. (1999) Endurance exercise causes interaction among stress hormones, cytokines, neutrophil dynamics, and muscle damage. *Journal of Applied Physiology* **87**(4), 1360-1367.
- Tambalis, K., Panagiotakos, D.B., Kavouras, S.A., Sidossis, L.S. (2009) Responses of blood lipids to aerobic, resistance, and combined aerobic with resistance exercise training: a systematic review of current evidence. *Angiology* **60**(5), 614-632.
- Thompson, P.D., Crouse, S.F., Goodpaster, B., Kelley, D., Moyna, N. and Pescatello, L. (2001) The acute versus the chronic response to exercise. *Medicine and Science in Sports and Exercise* **33**(6), S438-S445.
- Tucker, L.A. and Silvester, L.J. (1996) Strength training and hypercholesterolemia: an epidemiologic study of 8499 employed men. *American Journal of Health Promotion* **11**(1), 35-41.
- Zafeiridis, A., Goloi, E., Petridou, A., Dipla, K., Mougios, V. and Kellis, S. (2007) Effects of low- and high-volume resistance exercise on postprandial lipaemia. *British Journal of Nutrition* **97**(3), 471-477.
- Wallace, M.B., Moffatt, R.J., Haymes, E.M. and Green, N. R. (1991) Acute effects of resistance exercise on parameters of lipoprotein metabolism. *Medicine and Science in Sports and Exercise* **23**(2), 199-204.
- Williams, M.A., Haskell, W.L., Ades, P.A., Amsterdam, E.A., Bittner, V., Franklin, B.A., Gulanick, M., Laing, S.T. and Stewart, K.J. (2007) Resistance exercise in individuals with and without cardiovascular disease, 2007 update, a scientific statement from the American Heart Association Council on Clinical Cardiology and Council on Nutrition, Physical Activity, and Metabolism. *Circulation* **116**(5), 572-584.

Key points

- Repeated sets of heavy resistance exercise significantly increase oxygen uptake both during exercise and the following recovery period.
- Even though exercise was of low volume (8 sets x 6 repetitions) the elevated oxygen uptake during the rest intervals in combination with the total exercise session duration (26 min) resulted in aerobic energy expenditure that is equivalent to low to moderate intensity cycling.
- Leg press resistance exercise emphasizing on the eccentric movement that caused muscle damage had no effect on total cholesterol, HDL-C and LDL-C during the two days following exercise in young healthy male subjects.

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