Effects of acute exercise on high density lipoprotein cholesterol and high density lipoprotein subfractions in moderately trained females

P M Gordon, S Fowler, V Warty, M Danduran, P Visich, S Keteyian

Abstract

Increases in high density lipoprotein cholesterol (HDL-C) levels have previously been reported after moderate exercise bouts lasting less than two hours in men. Little information exists, however, on HDL-C responses after moderate duration exercise in women. Post-exercise HDL-C modifications may appear differently in women because of higher baseline HDL-C concentrations and differences in lipolytic activity. To determine the influence of exercise on acute HDL-C responses in women, 12 trained premenopausal women (22 (4) years old; mean (SD)) who ran 24-48 km a week exercised on a motor driven treadmill at 75% $\text{VO}_{2\text{MAX}}$ until 3.34 MJ (800 kcal) were expended (72 (9) min). Subjects were all tested during the early follicular phase of their menstrual cycle. Fasting blood samples were obtained before exercise (baseline), immediately after (IPE), one hour after (1 h PE), 24 hours after (24 h PE), and 48 hours after (48 h PE) exercise. Plasma was analysed for HDL-C, HDL₂-C, and HDL₃-C. A significant increase in HDL-C was observed 48 h PE ($p < 0.05$). HDL₃-C increased IPE ($p < 0.01$) but returned to baseline at 1 h PE. In contrast, HDL₂-C was not significantly different from baseline at any time point. The rise in HDL-C, however, was attributed to an increase in both HDL₂ and HDL₃. Moreover, at 48 h PE, the increase in HDL-C correlated highly with changes in HDL₂-C ($r = 0.92$). Thus it appears that exercise of moderate duration can elicit similar post-exercise increases in HDL-C in women to those previously reported in men. However, the changes in HDL subfractions leading to the rise in HDL-C may be different in women.

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Risk for coronary heart disease is inversely related to serum concentrations of high density lipoprotein cholesterol (HDL-C).^{1,2} Habitual exercise training reduces the risk of coronary heart disease, in part, through an increase in HDL-C.³ Similarly, some of the HDL-C modifications observed with endurance training can be partially induced from a single exercise bout.⁴⁻⁶ Previous investigations

have identified increases in HDL-C after protracted endurance events such as a marathon or triathlon in men,^{7,8} women,^{9,10} or both.¹¹ However, investigations using shorter exercise bouts (less than two hours) have yielded equivocal results, in part, because of the lack of control for potentially confounding variables.³

Post-exercise HDL-C responses may be influenced by diet composition, training status, baseline HDL-C, a previous bout of exercise, and lack of or an incomplete estimation of plasma volume shifts.³ When these extraneous variables were controlled, increases in HDL-C after shorter exercise bouts were observed in moderately trained men,^{5,6} but there is little information on women. Changes in HDL-C responses unique to women after short duration exercise might be confounded because of the use of oral contraceptive agents,^{3,12} different phases of the menstrual cycle,^{12,13} gender specific lipolytic activity,¹⁴ and high baseline HDL-C levels.¹⁵

This study was undertaken to provide a controlled evaluation of the acute effects of moderate duration exercise on HDL-C and HDL-C subfractions in trained premenopausal women.

Methods

SUBJECTS

Twelve women runners were recruited from Mankato State University and the surrounding community to participate in this investigation. They were selected on the basis of the following criteria: 18-35 years of age (mean (SD) age 22 (4) years); running 24-48 km a week for a minimum of six months (35.7 (9.1) km/week); stable weight for at least six months (56.6 (7.1) kg); no use of medications or oral contraceptives; no current use of alcohol; no medical conditions that would preclude them from participation. Mean percentage body fat, determined using hydrostatic weighing and the Siri equation,¹⁶ was 14.3 (6.6)%. All participants reported regular menstrual cycles (27-32 days) for the previous six months. Each participant signed a written informed consent approved by the Institutional Review Board for Human Subjects. Subjects were given a health screening and oriented to running on a motor driven treadmill before participating.

EXPERIMENTAL DESIGN

On day 1 subjects underwent a graded exercise test on a motor driven treadmill to determine $\text{VO}_{2\text{MAX}}$. After a 15 minute period of rest, participants then ran on the treadmill to deter-

School of Medicine,
West Virginia
University
P M Gordon

Department of Human
Performance, Mankato
State University
S Fowler

Department of Clinical
Chemistry, University
of Pittsburgh
V Warty

Lutheran General
Hospital, Chicago, IL
M Danduran

Human Performance,
Central Michigan
University, Detroit, MI
P Visich

Henry Ford Hospital,
Detroit, MI, USA
S Keteyian

Correspondence to:
Dr P M Gordon, West
Virginia University School of
Medicine, Division of
Exercise Physiology, PO Box
6116, Morgantown, WV
26506-6116, USA.

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Table 1 Four day dietary composition. Values are mean (SD)

	Day 1	Day 2	Day 3	Day 4	Average
Total energy (Kcal)	1523 (356)	1718 (286)	1674 (317)	1605 (410)	1630 (338)
Carbohydrate (%)	59 (9)	65 (11)	68 (10)	66 (11)	65 (10)
Fat (%)	25 (9)	22 (7)	20 (9)	20 (8)	22 (8)
Protein (%)	16 (2)	13 (3)	12 (4)	14 (2)	14 (3)
P/S Ratio	0.6 (0.3)	0.7 (0.2)	0.7 (0.2)	0.7 (0.2)	0.7 (0.2)

P/S ratio = polyunsaturated/saturated fat ratio.

mine the work rate corresponding to 75% $\text{VO}_{2\text{MAX}}$.

One to two weeks later, on day 2, subjects ran on a motor driven treadmill at 75% $\text{VO}_{2\text{MAX}}$ until 3.34 MJ (800 kcal) was expended. Each subject was scheduled to perform the experimental trial during the early follicular phase (days 5–10) of their menstrual cycle since minimal lipid and lipoprotein variation occurs during this phase.^{12–17} On day 2 subjects underwent a standard warm up run, at which time the treadmill speed was gradually increased until the appropriate power output (75% $\text{VO}_{2\text{MAX}}$) was reached. This was accomplished by increasing the treadmill speed to 70, 85, and 100% of the predetermined running speed every other minute. The treadmill speed was adjusted to maintain the target intensity throughout the exercise trial. This was done if VO_2 varied ± 2 ml/kg per minute from the target intensity.

During the exercise trial, cardiovascular and respiratory metabolic measures were recorded every minute. However, once steady state was ensured, subjects were allowed to remove the respiratory apparatus for seven minutes after every ten minutes of respiratory gas measurement. Respiratory gas measurements were obtained using a MedGraphics CPX metabolic system. Energy expenditure was calculated using the steady state respiratory exchange ratio and absolute VO_2 .¹⁸ Cool water was provided to subjects every ten minutes during the exercise trial.

To avoid dietary interaction with lipid measurements, each subject was asked to maintain their normal dietary habits and complete a four day food diary beginning two days before the experimental trial. Daily nutrient intakes were analysed by the Food Processor II nutritional software package from ESHA Research Inc.

BLOOD SAMPLING AND STORAGE

Venous blood samples were obtained at the following time points: baseline and immediately (IPE), one hour (1 h PE), 24 hours (24 h PE), and 48 hours (48 h PE) after exercise. All blood samples were taken after a 12 h fast from an antecubital vein with the subject in the seated position. Subjects abstained from exercise for 48 hours before the baseline measurement, to avoid any influence on baseline. Blood samples were collected into a 10 ml heparinised tube. Plasma was analysed for total cholesterol, triglycerides, HDL-C, HDL₂-C, and HDL₃-C. The plasma samples were separated by low speed centrifugation and stored at -70°C until analysis.

BLOOD ANALYSIS

All blood assays were performed in duplicate in a Centers for Disease Control reference

laboratory by the same technician. Interassay variation was prevented by analysing all samples from each subject in a single batch run. Cholesterol¹⁹ and triglycerides²⁰ were measured using enzymic methods on a Cobas Bio (Roche) Centrifugal Analyzer. HDL-C was determined after precipitation of lower density lipoproteins using heparin/ MnCl_2 .²¹ The methods of Delong *et al*²² were used to calculate low density lipoprotein cholesterol (LDL-C) and very low density lipoprotein cholesterol (VLDL-C). High density lipoprotein (HDL) subfractions were determined using HDL 2/3 precipitation methods.²³ Packed cell volume and haemoglobin were measured using a Coulter Counter model STKS from Coulter Electronics Inc. Changes in plasma volume were determined by the methods of Dill and Costill.²⁴

STATISTICAL ANALYSIS

Results are all means (SD). Lipid and lipoprotein variables were analysed using one way analysis of variance with repeated measures. All values were corrected for changes in plasma volume. A Tukey HSD post hoc test was utilised to test for differences between means. Values for each time point were compared with baseline values. $p < 0.05$ was required for statistical significance.

Results

On day 2 subjects exercised for 72 (9.8) min at 73.4 (2)% of $\text{VO}_{2\text{MAX}}$ (41.1 (3.4) ml/kg per min). The subjects' total energy expenditure was 3.35 (0.01) MJ (801.4 (3.4)) kcal, completing a total distance of 12.2 (2.1) km. The subjects' average daily diet consisted of 6.81 (1.4) MJ (1630 (338)) kcal and was comprised as follows: 65 (10)% carbohydrate, 14 (3)% protein, and 22 (8)% fat. The mean polyunsaturated to saturated fat ratio was 0.7 (0.2). Diet composition was unchanged over the course of the experiment (table 1).

UNCORRECTED FOR PLASMA VOLUME

Significant differences in lipid and lipoproteins, uncorrected for changes in plasma volume, were observed over time (table 2). HDL-C at IPE was increased above baseline values ($p < 0.01$) but returned to baseline at 1 h PE. Similarly, HDL₃-C was increased above baseline values at IPE ($p < 0.01$). However, no significant increase was observed in HDL₂-C. Plasma triglyceride values were significantly lower than baseline at 24 h PE ($p < 0.05$) and 48 h PE ($p < 0.05$). As expected, VLDL-C replicated plasma triglyceride values ($p < 0.05$). No significant differences were observed between baseline and any other sampling point for total cholesterol or LDL-C.

CORRECTED FOR PLASMA VOLUME

Once corrected for post-exercise plasma volume shifts, significant lipid and lipoprotein changes were observed which differed from the uncorrected values. The calculated loss in plasma volume IPE was 4 (6)% ($p < 0.05$) (table 3). When the shift in plasma volume was

Table 2 Uncorrected plasma lipid and lipoprotein responses after exercise in moderately trained females. Values are mean (SD)

	Before exercise Baseline	After exercise			
		IPE	1 h PE	24 h PE	48 h PE
TC	3.88 (0.6)	4.13 (0.8)	4.03 (0.7)	3.81 (0.8)	3.94 (0.9)
TG	1.06 (0.7)	1.14 (0.6)	0.97 (0.5)	0.82 (0.5)*	0.81 (0.4)*
LDL-C	2.08 (0.6)	2.09 (0.7)	2.08 (0.7)	2.02 (0.7)	2.09 (0.8)
VLDL-C	0.42 (0.3)	0.43 (0.2)	0.36 (0.2)	0.3 (0.18)*	0.3 (0.14)*
HDL-C	1.46 (0.3)	1.62 (0.3)*	1.56 (0.4)	1.53 (0.3)	1.54 (0.3)
HDL ₂ -C	0.67 (0.3)	0.64 (0.3)	0.7 (0.4)	0.7 (0.27)	0.73 (0.3)
HDL ₃ -C	0.8 (0.14)	0.98 (0.2)**	0.84 (0.16)	0.75 (0.13)	0.81 (0.13)

All values are given as mmol/l. To convert cholesterol to mg/dl divide by 0.02586. To convert triglycerides to mg/dl divide by 0.01129.

TC, total cholesterol; TG, triglyceride; LDL, low-density lipoprotein; VLDL, very low density lipoprotein; HDL, high-density lipoprotein; C, cholesterol.

*Significant difference from baseline value ($p < 0.05$).

**Significant difference from baseline value ($p < 0.01$).

Table 3 Corrected plasma lipid and lipoprotein responses after exercise in moderately trained females. Values are mean (SD)

	Before exercise Baseline	After exercise			
		IPE	1 h PE	24 h PE	48 h PE
Plasma volume (%)	—	-4.1 (6)*	-0.54 (7.3)	6.6 (6.8)*	3.9 (7.6)*
TC	3.88 (0.6)	3.96 (0.8)	4.0 (0.8)	4.04 (0.8)	4.06 (0.8)
TG	1.06 (0.7)	1.09 (0.5)	0.95 (0.4)	0.86 (0.5)	0.84 (0.4)
LDL-C	2.08 (0.6)	2.0 (0.7)	2.06 (0.7)	2.13 (0.6)	2.15 (0.7)
VLDL-C	0.42 (0.3)	0.41 (0.2)	0.36 (0.16)	0.33 (0.2)	0.31 (0.15)
HDL-C	1.46 (0.3)	1.56 (0.3)	1.56 (0.4)	1.55 (0.3)	1.59 (0.3)*
HDL ₂ -C	0.67 (0.3)	0.62 (0.3)	0.73 (0.4)	0.76 (0.3)	0.75 (0.3)
HDL ₃ -C	0.8 (0.14)	0.94 (0.2)**	0.83 (0.1)	0.8 (0.1)	0.84 (0.1)

All values are given as mmol/l. To convert cholesterol to mg/dl divide by 0.02586. To convert triglycerides to mg/dl divide by 0.01129.

TC, total cholesterol; TG, triglyceride; LDL, low-density lipoprotein; VLDL, very low density lipoprotein; HDL, high-density lipoprotein; C, cholesterol.

*Significant difference from baseline value ($p < 0.05$).

**Significant difference from baseline value ($p < 0.01$).

accounted for, HDL-C significantly increased above baseline at 48 h PE ($p < 0.05$).

Changes in HDL subfractions after exercise were similar to uncorrected values. HDL₃-C increased IPE ($p < 0.01$) and returned toward baseline at 1 h PE. Furthermore, no significant differences were observed for HDL₂-C over time ($p = 0.10$). No other differences in plasma lipid or lipoproteins were observed between baseline and other sampling points.

Discussion

This study observed an immediate post-exercise increase in HDL-C when uncorrected for plasma volume shifts. However, after adjusting for changes in plasma volume, we detected a significantly different HDL-C response. After exercise, the increase in HDL-C was observed at 48 h PE ($p < 0.05$) (fig 1). These findings support the need to adjust for plasma volume to measure actual post-exercise changes in HDL-C concentrations. Consequently, it appears that short term exercise lasting less than two hours in moderately trained premenopausal women results in a delayed increase in HDL-C concentrations.

Post-exercise increases in HDL-C have previously been reported in women after prolonged exercise bouts lasting more than two hours.^{10–11} However, investigations that have attempted to identify post-exercise HDL-C responses in women after shorter exercise bouts (less than two hours) have yielded equivocal results.^{25–26} Many of the inconsistencies in short term post-exercise lipid and lipo-

Figure 1 High density lipoprotein (HDL) cholesterol (HDL-C) and HDL subfraction (HDL₂-C, HDL₃-C) changes after acute exercise in moderately trained women. Measurements were made before exercise (baseline), or immediately (IPE), one hour (1 h PE), 24 hours (24 h PE), or 48 hours (48 h PE) after exercise. * $p < 0.05$ v baseline value.

protein alterations reported in women may be related, in part, to lack of control over confounding factors. Previous studies evaluating HDL-C responses in women after exercise lasting less than two hours either did not adjust the results for plasma volume²⁵ or failed to correct for the change in red cell volume.²⁶ Consequently, artificial changes in post-exercise lipid and lipoprotein modifications were probably recorded because of shifts in plasma volume. Additional variables that may potentially confound post-exercise HDL-C alterations in women may have also diluted the findings of previous studies.^{25–27} These include the use of oral contraceptives^{3–12–26} and failure to control for the different phases of the menstrual cycle.^{12–13}

Once these potential confounding factors are taken into account, the present findings are similar to those from previous studies utilising a similar research protocol (75% $\dot{V}O_{2\text{MAX}}$ until 3.34 MJ expended) in male subjects,^{5–6} although the delayed increase in HDL-C reported in studies using men occurred at 24 h PE compared with 48 h PE in the present study. Moreover, as in the present investigation, the male subjects had the same training level (24–48 km/week) and similar fitness levels (57.7 (5.2) ml/kg per min for men compared with 56.3 (5) ml/kg per min for women). As expected, however, baseline HDL-C concentrations were much higher ($p < 0.01$) in the women (56.5 (11.5) mg/dl) than the men (41.4 (6) mg/dl). Individuals with higher baseline HDL-C levels are thought to require a larger volume of work to elicit a post-exercise increase.³ Even though the women in the present study have higher baseline HDL-C values, it appears that, given comparable training status and fitness levels, similar post-exercise HDL-C responses occur at this level of energy expenditure.

In the present study, a non-significant increase in HDL-C did occur IPE ($\Delta = 3.9$ mg/dl) which stabilised across sampling time points until reaching significance at 48 h PE ($\Delta = 5$ mg/dl; $p < 0.05$). The rise in HDL-C IPE appeared in the HDL₃-C subfraction ($p < 0.01$). However, we cannot rule out the possibility that the increase in HDL₃-C may also be due, in part, to conversion of HDL₂-C

to HDL₃-C (fig 1). We observed a non-significant decline in HDL₂-C IPE which is not fully understood. Nevertheless, other investigators have reported transient increases in HDL₃-C after shorter duration exercise (less than two hours) in women.^{26,27} Moreover, studies involving short term exercise in men with the same training level and energy expenditure as in the present study have also reported similar post-exercise increases in HDL₃-C.^{5,6,28} However, the time course for post-exercise increases in HDL₃-C appeared different. For example, both Gordon *et al*⁵ and Visich *et al*⁶ did not observe a post-exercise increase in HDL₃-C until 24 h PE.

In contrast, HDL₂-C was not significantly different across sampling points. However, a non-significant increase in HDL₂-C occurred as HDL₃-C began to decline at 1 h PE (fig 1). In fact, by 48 h PE the change in HDL-C was more highly associated with an increase in HDL₂-C ($r = 0.917$; $p < 0.001$) than HDL₃-C ($r = 0.562$; $p < 0.05$). This may exemplify the dynamic environment to which HDL subfractions exist. Berger *et al*²⁹ first proposed that the esterification of free cholesterol by lecithin: cholesterol acyltransferase may be rate limiting, suggesting that additional time may be required for sufficient conversion of HDL₃-C to HDL₂-C to raise HDL₂-C concentrations significantly.²⁷ On the basis of the present findings, we speculate that an acute exercise induced increase in HDL₂-C may be controlled by a rate limiting lipolytic process and therefore may require more time to increase significantly above baseline.

The underlying biochemical mechanism responsible for acute increases in HDL-C after exercise is probably related to catabolism of triglyceride rich lipoproteins via lipoprotein lipase.⁶ Increased lipoprotein lipase activity after exercise results in chylomicron and VLDL hydrolysis and reductions in plasma triglyceride levels.³ Consequently, surface remnants are converted into nascent HDL-C. However, while HDL-C levels may increase after exercise, a concomitant decline in plasma triglyceride levels has not been consistently observed. Moreover, reductions in plasma triglyceride concentrations after acute exercise appear to be related to the amount of work completed.³ Exercise studies of women taking part in events that require a high energy expenditure—for example, triathlon—have reported decreased plasma triglyceride levels 24 h after exercise.^{9,11} In contrast, shorter durations of exercise may result in only modest decreases in plasma triglyceride²⁷ or no change at all.²⁶ In the present study, once values were corrected for changes in plasma volume, triglyceride levels at 48 h PE were no longer significantly different from baseline ($p = 0.06$). However, the average decline in plasma triglyceride concentrations at 48 h PE was 21% and was similar to observations in men performing the same volume of exercise.⁶ Furthermore, the decline in plasma triglyceride observed in the present study, while not statistically significant, probably has clinical ramifications for HDL-C improvements.

In summary, it appears that an exercise bout of less than two hours in duration can elicit post-exercise increases in HDL-C in moderately trained premenopausal women. Although women have higher baseline HDL-C values than men, a similar delayed increase can be observed provided that training status and amount of exercise performed is equivalent. In addition, post-exercise modifications in HDL subfractions may be more dynamic in women than men. However, the exact mechanisms responsible for these alterations in lipolytic activity requires further investigation.

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Musings of a running “agony aunt”

Although the learned researchers and clinicians who have their offerings published in the *British Journal of Sports Medicine* probably do not realise it, much of their material is relatively unintelligible to the general public. Yet if you scan the book stalls, sports magazines abound, and many include medical and injury columns together with the obligatory “Agony Aunt” to satisfy the need for definitive advice on sport and exercise medicine.

Although it may seem easy for a doctor writer to fill the pages of a magazine with precis of interesting research, cautionary tales, and simple common sense, pitfalls abound. In the early 1980s, when people only wanted to run marathons and any other distance was considered soft, queries came from men with broken legs who had only one ambition in life—to run a marathon—when could they? Doubts begin to surface, however, as one realises that there is a considerable responsibility, both to the reader and their medical advisers, in replying. Should the exfootballer, already missing part of a meniscus, be warned that running high mileages on tarmac could aggravate his knee condition, and what of the lady who vomits at the three mile mark of every run, then can run for ever? Is it pathological or psychological?

Having read the closely written pages of A4, often with detailed training schedules and racing programmes, sometimes with an action photo thrown in, one has to appreciate the desperation with which some of these letters are written. Most have already sought medical advice and met with varying degrees of disinterest, incomprehension, trivialisation, and sometimes—only having received a highly subjective account of the problem—what appears to be an arrogant dismissiveness by the profession that the enquirer should

never have entertained hopes of competing or running after injury. While it is all too easy to criticise the obsession and introspection with which these people view their sport, to many it is not only a hobby and an escape, but their whole social life. Replying tactfully in order not to exacerbate differences between patient and doctor can require the skills of a Kissinger on occasions.

Many have received advice that is totally negative, and seek a second opinion. One has to be guarded in one's answers, for, although the runner whose myopic retina detached in a race really is at risk of a recurrence, the MI victim may benefit his coronary arteries through a graduated training programme, although this really ought to be under the guidance of a cardiologist after full investigation and counselling, most of which is outside the scope of a written and published reply.

The postbag confirms that injured sportsmen feel that they do not receive the urgent priority care that their injuries merit. Whether they are more poorly treated than other sections of the population is another debate. What is undoubtedly true is that they feel there is neither the interest nor very often the local ability to understand and treat their problems. Much of their frustration is the result of a high degree of motivation which tempts some to overdo the therapeutic processes.

Finally, what of the letter I received from a lady about to have a reduction mammoplasty, as the offending objects were slowing her running down. Would this operation affect her centre of gravity and therefore cause injury, she wanted to know? Answers please on a plain postcard!

PATRICK MILROY

Dr Milroy has been the medical advisor to Runners World since 1982.