

Acute high-intensity exercise with low energy expenditure reduced LDL-c and total cholesterol in men

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Abstract A reduction in LDL cholesterol and an increase in HDL cholesterol levels are clinically relevant parameters for the treatment of dyslipidaemia, and exercise is often recommended as an intervention. This study aimed to examine the effects of acute, high-intensity exercise ($\sim 90\%$ VO_{2max}) and varying carbohydrate levels (control, low and high) on the blood lipid profile. Six male subjects were distributed randomly into exercise groups, based on the carbohydrate diets (control, low and high) to which the subjects were restricted before each exercise session. The lipid profile (triglycerides, VLDL, HDL cholesterol, LDL cholesterol and total cholesterol) was determined at rest,

and immediately and 1 h after exercise bouts. There were no changes in the time exhaustion (8.00 ± 1.83 ; 7.82 ± 2.66 ; and 9.09 ± 3.51 min) and energy expenditure (496.0 ± 224.8 ; 411.5 ± 223.1 ; and 592.1 ± 369.9 kJ) parameters with the three varying carbohydrate intake (control, low and high). Glucose and insulin levels did not show time-dependent changes under the different conditions ($P > 0.05$). Total cholesterol and LDL cholesterol were reduced after the exhaustion and 1 h recovery periods when compared with rest periods only in the control carbohydrate intake group ($P < 0.05$), although this relation failed when the diet was manipulated. These results indicate that acute, high-intensity exercise with low energy expenditure induces changes in the cholesterol profile, and that influences of carbohydrate level corresponding to these modifications fail when carbohydrate (low and high) intake is manipulated.

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Introduction

Regular physical activity is often recommended as a non-pharmacological intervention to decrease the risk of cardiovascular heart disease (CHD), since it alters lipoprotein profiles by increasing the high-density lipoprotein cholesterol (HDL-c) levels and reducing the low-density cholesterol (LDL-c) (Hubinger et al. 1995). However, several studies have shown that the effect of exercise is dependent on intensity. Therefore, there is a specific exercise intensity where modifications in lipoprotein profiles occur following acute exercise (Kokkinos and Fernhall 1999). For example, Ferguson et al. (1998) reported a reduction in triglycerides

(TG) and LDL-c concentrations and an increase in HDL-c concentrations after exercise sessions at 70% VO_{2max} and with energy expenditures higher than 1.100 kcal. Although exercise intensity and duration are important in inducing alterations of lipoprotein profiles, little is known about the effect of high-intensity exercise ($\sim 90\%$ VO_{2max} , for example) on lipoprotein metabolism.

Moreover, different strategies beyond exercise intensity manipulation, such as diet manipulation, are also used to maximise the beneficial effects of exercise on the lipid profile. Some studies have investigated the effect of combining lipid (i.e. omega-3 fatty acid n-3 FA) supplementation and exercise training on the treatment of lipaemia (Thomas et al. 2004, 2007). These studies suggest that either an n-3 fatty acid supplementation or an exercise session can affect total HDL-C and subfractions, but they do not alter LDL-C or subfractions. In addition, the combination of n-3 fatty acid and exercise may have additional effects on total HDL-C and LDL-C subfractions as compared to either treatment alone in active young men. However, although the effects of fat diets on lipid profiles have been well documented in the literature (Thomas et al. 2004, 2007; Bortolotti et al. 2007; Peoples et al. 2008; Simopoulos 2008), the effects of acute, high-intensity exercise associated with different carbohydrate intake levels on lipid profiles in healthy subjects are unknown. Given the lack of studies investigating the effect of both CHO diets and high-intensity exercise, we aimed to compare the effects of high-intensity exercise ($\sim 90\%$ VO_{2max}) and an intervention diet (control, low and high status carbohydrate) on the lipid profile. We hypothesised that high-intensity exercise combined with carbohydrate intervention may induce beneficial effects on lipoprotein profiles in healthy men.

Methods

Subjects

Six healthy men, non-smoking and physically active [age 28.5 (6.8) years, height 178.0 (4.5) cm, weight 73.8 (8.1) kg and VO_{2max} 47.1 (8.5) ml kg^{-1} min^{-1}], participated in this

study. The protocol, benefits and risks were explained before written consent was obtained. The study procedures were previously approved by the Ethics Committee of the School of Physical Education and Sport of the University of São Paulo.

Experimental design

The subjects performed an incremental test, a control test and two experimental cycling tests to exhaustion (Fig. 1).

On the first visit, the subjects were submitted to a progressive test conducted on an electromagnetically braked cycle ergometer (Ergo Fit 167, Pirmansens, Germany) in order to determine VO_{2max} , and first (LT1) and second (LT2) lactate thresholds. On the second visit, after 8- to 12-h overnight fasting and 48 h after the incremental test, the subjects exercised until exhaustion at an intensity of approximately 75% of the difference between LT2 and the maximal workload (control test). On the third visit, after 5 days, the subjects performed an exercise protocol, to deplete muscle glycogen levels, which consisted of 90 min of cycling at a power output required for 50% of the difference between LT₁ and LT₂, followed by six bouts at 1 min each at 125% VO_{2max} , interspersed with 1-min rest periods (Gollnick et al. 1973, 1974; Heigenhauser et al. 1983). During the following 2 days, starting immediately after the depletion protocol, a diet providing 10% of energy in the form of CHO was consumed (low-CHO diet: 10% CHO, 35% fat and 55% protein). Thereafter (fourth visit), the subjects exercised until exhaustion at the same intensity employed in the control test. After an interval of 1 week, the subjects performed again the CHO depletion protocol to reduce muscle glycogen stores (fifth visit), before embarking on an 80% CHO diet (high CHO diet: 80% CHO, 5% fat and 15% protein). After 2 days on the high CHO diet, subjects performed the second experimental trial (sixth visit) to fatigue, at an intensity of approximately 75% of the difference between LT₂ and the maximal workload (high CHO diet).

The experimental conditions (low- or high-CHO diet) were applied in a counterbalanced order and the subjects were not informed of the time to exhaustion until the study had been concluded. The experimental high-intensity

Day 1 (visit 1)	Day 2	Day 3 (visit 2)	Day 4-7	Day 8 (visit 3)	Day 9	Day 10 (visit 4)	Day 11-17	Day 18 (visit 5)	Day 19	Day 20 (visit 6)
Incremental test	No exercise or food manipulation	Control high intensity exercise	No exercise or food manipulation	Depletion CHO protocol followed for 10% CHO diet	No exercise. 10% CHO diet.	Experimental high intensity exercise	Washout (No exercise or food manipulation)	Depletion CHO protocol followed for 80% CHO diet	No exercise. 80% CHO diet.	Experimental high intensity exercise

Fig. 1 Schematic representation of the experimental design. The sequence from diet manipulation was counterbalanced

exercises were performed in the morning period after 8–12 h of overnight fasting, similar to the control test. All diets throughout the study periods were isocaloric (2096.3 ± 63.1 kcal). It has been previously demonstrated that the consumption of diets with high- or low-CHO content during 2 days can restore or reduce the endogenous CHO availability, respectively (Heigenhauser et al. 1983; Gridale et al. 1990; Glass et al. 1997). The diets were modulated by taking into consideration the body mass and food preferences using previous 5-day records of food intake.

Incremental exercise test

Each subject performed an incremental exercise test until volitional fatigue, in which the work rate was started at 50 W and increased by 20 W for every 3 min at a cycling frequency of 60–70 rpm. At the end of each stage, 25 μ l of blood was drawn from the earlobe and immediately analysed to determine the blood lactate concentration (YSI 1500 Sport, Yellow Springs Instruments, Yellow Springs, OH). The first (LT_1) and second (LT_2) lactate breakpoints were determined by linear regression analysis (Ribeiro et al. 1986). The VO_{2max} was defined as the highest oxygen uptake obtained during the last 30-s interval during the incremental test. The maximal workload was determined as the highest workload reached with a pedal frequency between 60 and 70 rpm. When subjects were not able to complete the last stage, the maximal workload was calculated from the following equation (Kuipers et al. 1985):

$$W_{max} = LCS + (TLIS/180 \times 20)$$

where W_{max} is the maximal workload (in watts), LCS is the workload in the last complete stage performed by the subject, and TLIS is the time in seconds sustained by the subject in the last incomplete stage.

High-intensity exercise

The subjects performed a constant workload test until exhaustion at a power output required for a 75% of difference between LT_2 and W_{max} ($93.5 \pm 5.5\%$ of VO_{2max} , i.e., $\Delta 75\%$). We chose to determine the workload of the high-intensity exercise using the lactate breakpoint instead of exclusively VO_{2max} , because the former has shown a more consistent level of metabolic stress amongst different individuals than the latter (Weltman et al. 1989). All subjects were instructed to arrive at the laboratory after 8–12 h of overnight fasting. After a 5-min warm-up at 50 W, the workload was adjusted to $\Delta 75\%$, and the subjects cycled (60–70 rpm) until volitional exhaustion under each of the three conditions (control, high and low). Abstention from strenuous exercise and from consumption of caffeine was

recommended for the previous 48 h. The participants were also asked to avoid strenuous exercise in the 48 h preceding the test and to record food intake for 5 days before the test. After the end of the test, the subjects remained seated quietly in a chair during a recovery period of 1 h. The VO_2 and heart rate were measured continually during the whole test and recovery. A 10 ml blood sample was collected at rest, at exhaustion, and at 60 min of recovery.

Measurement of pulmonary gas exchange

During every test, the breath-by-breath gas exchange was measured using a metabolic measurement system (Quark b2, Cosmed, Rome, Italy). Throughout the test, each subject wore a mask (Hans Rudolph®, Kansas City, MO, USA) connected to the gas analyser system for breath-by-breath measurements of gas exchange. The gas analyser was calibrated according to the manufacturer's specifications before each test (Quark b2 instruction manual).

Energy expenditure calculation

The trapezoidal method was used to calculate the VO_2 area over time during and after high-intensity exercise. Thus, the aerobic energy contribution was calculated by subtracting $VO_{2baseline}$ from the VO_2 area integrated over time. The contribution of anaerobic metabolism was estimated by the oxygen-deficit method. Initially, we fitted the breath-to-breath VO_2 on-transient response using a biexponential model (Eq. 1) (Origin 6.0, Microcal, Massachusetts, USA), as previously suggested by Ozyener et al. (2001). Thus, the oxygen deficit was obtained by integration of the first exponential part (Eq. 2) (Whipp 1994). A caloric equivalent of $20.9 \text{ kJ} \times 1 \text{ O}_2^{-1}$ was considered for the two energy systems (Di Prampero and Ferretti 1999). Total energy expenditure was calculated as the sum of the two energy systems:

$$\dot{V}O_{2(t)} = VO_{2baseline} + A_1 \left(1 - e^{-(t-\delta_1)/\tau_1} \right) + A_2 \left(1 - e^{-(t-\delta_2)/\tau_2} \right) \quad (1)$$

$$\text{Oxygen deficit} = A_1 \times t_1 \quad (2)$$

where $VO_{2(t)}$ is the oxygen uptake at time t , $VO_{2baseline}$ is the oxygen uptake at baseline, A is the amplitude, δ is the time delay, τ is the time constant, and 1 and 2 denote the fast and the slow components, respectively.

Blood sampling and analysis

The blood samples (10 ml) were immediately transferred into two 5-ml vacutainer tubes (Becton Dickinson, BD, Juiz de Fora, MG, Brazil) containing EDTA for plasma

separation. The tubes were centrifuged at 3.000 g for 15 min at 4°C, and plasma samples were stored at -80°C until analysis. Triglycerides, HDL cholesterol, and total cholesterol were assessed through commercial enzymatic kits (Labtest®, São Paulo, Brazil). VLDL and LDL cholesterol were calculated according to Friedewald et al. (1972). Plasma glucose concentration was analysed using the enzymatic colorimetric method (Biotécnica, São Paulo, Brazil). Blood lactate concentration was measured using an automatic analyser (YSI 1500 Sport, Yellow Springs Instruments, Yellow Springs, OH). Serum insulin was quantified using commercial kits RIA (DPC®, Brazil).

Statistical analyses

The data distribution was previously checked by the Shapiro–Wilk’s test, and the data are reported as means and standard deviations. The differences in the blood parameters and VO_2 were accessed by repeated measures 3×3 (time \times diet) ANOVA with covariance structure adjustment, and the confidence interval was adjusted by the Bonferroni test. The differences in the time to exhaustion, energy expenditure during exercise and total oxygen uptake during recovery between diets were accessed by one-way ANOVA, followed by the Bonferroni post hoc test. In order to verify the magnitudes of the effects of diets, the effect size to each time of measurement was calculated. Criteria used for interpreting the magnitudes of effects were: trivial 0.0–0.2; small 0.2–0.6; moderate 0.6–1.2; large 1.2–2.0; very large >2.0 (Cox et al. 2008). The analysis was carried out using SPSS (15.0) software, and the significance level was set at $P < 0.05$.

Results

Table 1 shows general data parameters. There is no significant difference in the time to exhaustion (8.00 ± 1.83 ;

7.82 ± 2.66 ; and 9.09 ± 3.51 min) or in the energy expenditure (561.8 ± 242.9 ; 473.6 ± 248.6 ; and 665.2 ± 408.5 kJ) amongst the different conditions (control, low and high, respectively). In addition, the total oxygen uptake during the recovery was not significantly different amongst the conditions (4.7 ± 2.3 ; 5.9 ± 3.9 ; and 5.9 ± 2.3 L for control, low and high, respectively).

The VO_2 data in Table 2 showed significant differences in the exhaustion time (47.18 ± 9.1 ; 41.98 ± 8.03 ; and 45.63 ± 10.7 mL kg^{-1} min^{-1}) when compared with rest (4.85 ± 0.69 ; 5.12 ± 1.71 ; and 4.60 ± 0.64 mL kg^{-1} min^{-1}) and after 1 h of recovery post-exercise (5.03 ± 2.67 ; 4.29 ± 1.68 ; and 4.68 ± 1.09 mL kg^{-1} min^{-1}), for all conditions (control, low and high, respectively), but no significant effect was observed between the diet conditions ($P < 0.05$). Similarly, the lactate concentration showed significant differences in the exhaustion time (7.09 ± 1.20 ; 5.82 ± 1.46 ; and 6.54 ± 3.45 mmol L^{-1}), when compared with rest (0.48 ± 0.35 ; 0.61 ± 0.19 ; and 0.62 ± 0.38 mmol L^{-1}) and after 1 h of recovery post-exercise (1.47 ± 0.71 ; 1.17 ± 0.34 ; and 1.25 ± 0.72 mmol L^{-1}) for all conditions ($P < 0.05$), but no diet effect was observed.

Table 3 shows that acute exercise of high intensity induced significant effects on total cholesterol and LDL-c levels. In the control diet condition, the total cholesterol was reduced in exhaustion and 1 h post-exercise when compared with the rest period (rest 4.64 ± 0.91 ; exhaustion 2.77 ± 0.69 ; 1 h post-exercise 2.68 ± 1.04 mmol L^{-1}), as well as the LDL-c (rest 3.47 ± 0.52 ; exhaustion 1.66 ± 1.00 ; 1 h post-exercise 1.71 ± 1.09 mmol L^{-1}) concentration ($P < 0.05$). However, a similar time effect was not observed when subjects consumed low- or high-carbohydrate diets. The glucose and insulin concentrations were not significantly different in relation to carbohydrate status or time ($P > 0.05$).

No significant differences were observed for TG, VLDL and HDL-c (Table 3) in time or between different diet conditions.

Table 1 Time to exhaustion, Energy expenditure, O_2 uptake during the recovery period

Subjects	Control	Low	High	Effect size	Qualitative descriptor
Time to exhaustion (min)					
	8.00 ± 1.83	7.82 ± 2.66	9.09 ± 3.51	0.29	Small
Energy expenditure (kJ)					
Exhaustion	$496.0 \pm 224.8\#$	$411.5 \pm 223.1\#$	$592.1 \pm 369.9\#$	0.27	Small
1 h	93.67 ± 46.93	118.4 ± 78.8	119.4 ± 46.5	0.21	Small
O_2 uptake during the recovery (L)					
	4.70 ± 2.3	5.9 ± 3.9	5.9 ± 2.3	0.20	Small

Results are expressed as mean value \pm SD

Significantly different from 1 h of recovery under the same intensity ($P < 0.05$)

Table 2 Physiological and performance variables

Subjects	Control	Low	High	Effect size	Qualitative descriptor
VO₂ (mL/kg per min)					
Rest	4.85 ± 0.69	5.12 ± 1.71	4.60 ± 0.64	0.21	Small
Exhaustion	47.18 ± 9.1*	41.98 ± 8.03*	45.63 ± 10.7*	0.23	Small
1 h	5.03 ± 2.67	4.29 ± 1.68	4.68 ± 1.09	0.17	Trivial
Lactate (mmol/L)					
Rest	0.48 ± 0.35	0.61 ± 0.19	0.62 ± 0.38	0.21	Small
Exhaustion	7.09 ± 1.20*	5.82 ± 1.46*	6.54 ± 3.45*	0.25	Small
1 h	1.47 ± 0.71	1.17 ± 0.34	1.25 ± 0.72	0.21	Small
Glucose (mmol/L)					
Rest	4.20 ± 1.33	4.57 ± 0.61	4.02 ± 0.97	0.24	Small
Exhaustion	4.43 ± 1.51	4.48 ± 0.80	4.64 ± 0.72	0.09	Trivial
1 h	3.80 ± 1.66	4.73 ± 0.46	4.27 ± 0.28	0.47	Small
Insulin (μU/mL)					
Rest	3.43 ± 1.62	3.70 ± 0.55	4.45 ± 0.38	0.51	Small
Exhaustion	2.52 ± 0.55	3.29 ± 1.17	3.82 ± 0.85	0.62	Moderate
1 h	3.43 ± 1.04	3.56 ± 0.77	3.04 ± 0.61	0.27	Small

Results are expressed as mean value ± SD

* Significantly different from the rest and 1 h of recovery under the same conditions ($P < 0.05$)

Table 3 Lipids profile in serum

Subjects	Control	Low	High	Effect size	Qualitative descriptor
TG (mmol/L)					
Rest	1.12 ± 0.48	1.08 ± 1.08	1.33 ± 0.64	0.15	Trivial
Exhaustion	0.93 ± 0.57	0.42 ± 0.17	0.82 ± 0.47	0.55	Small
1 h	0.88 ± 0.61	0.70 ± 0.24	1.01 ± 0.46	0.29	Small
VLDL (mmol/L)					
Rest	0.22 ± 0.09	0.21 ± 0.21	0.26 ± 0.13	0.15	Trivial
Exhaustion	0.18 ± 0.11	0.08 ± 0.03	0.16 ± 0.09	0.54	Small
1 h	0.17 ± 0.12	0.14 ± 0.04	0.20 ± 0.09	0.31	Small
Cholesterol (mmol/L)					
Rest	4.64 ± 0.91	3.30 ± 1.08	3.50 ± 2.78	0.37	Small
Exhaustion	2.77 ± 0.69†	3.07 ± 0.46	3.47 ± 0.95	0.43	Small
1 h	2.68 ± 1.04†	2.09 ± 0.22	2.37 ± 1.48	0.26	Small
LDL-c (mmol/L)					
Rest	3.47 ± 0.52	2.18 ± 1.19	2.14 ± 1.66	0.55	Small
Exhaustion	1.66 ± 1.00†	1.89 ± 0.76	2.67 ± 0.71	0.53	Small
1 h	1.71 ± 1.09†	1.19 ± 0.28	1.35 ± 1.22	0.25	Small
HDL-c (mmol/L)					
Rest	0.94 ± 0.76	0.90 ± 0.41	1.09 ± 1.04	0.11	Trivial
Exhaustion	0.92 ± 0.80	1.08 ± 0.45	0.63 ± 0.29	0.36	Small
1 h	0.79 ± 0.50	0.76 ± 0.39	0.81 ± 0.51	0.04	Trivial

Results are expressed as mean value ± SD

† Significantly different from the rest under the same conditions ($P < 0.05$)

Discussion

Our results indicate that, although high-intensity exercise generated low energy expenditure, it induced a reduction in LDL-c and total cholesterol levels. However, these alterations in lipoprotein profiles induced by high-intensity exercise disappear when carbohydrate levels are changed by 2 days of a low- or high-CHO diet.

High-intensity exercise, performed at intensities above the second lactate breakpoint, but below the VO_{2max} , is characterised by a biexponential increase in VO_2 , which progressively increases until VO_{2max} values are reached at exhaustion (Barstow et al. 1996). It has been demonstrated that muscle glycogen depletion prior to moderate exercise has no effect on VO_2 at the end of exercise (Carter et al. 2004). However, previous studies have shown that glycogen depletion of the type I fibres increases the VO_2 during high-intensity exercise (Bouckaert et al. 2004), whilst a reduction in type II fibre glycogen content results in a decreased amplitude of the VO_2 slow component. We found no difference in VO_2 amongst the diet conditions, which could be expected because our protocol to assess muscle glycogen depletion was designed to reduce the glycogen content of both fibre types; therefore, a possible increase in VO_2 due to the type I fibre depletion could have been compensated for by a reduction in glycogen of the type II fibres (Krustrup et al. 2004; Carter et al. 2004; Bouckaert et al. 2004).

The blood lactate at the end of maximal incremental exercise was still reduced after muscle glycogen depletion when compared with the control condition (Podolin et al. 1991). We did not find differences between the conditions regarding blood lactate accumulation. In addition, the blood lactate values at exhaustion were similar to those demonstrated in other studies incorporating a similar intensity of exercise (Lima-Silva et al. 2009).

We observed that acute, high-intensity exercise during a control diet reduces LDL cholesterol and total cholesterol concentrations in exhaustion, and the reduction remains after 1 h of recovery when compared to the rest period. However, these alterations were not evident in high- and low-carbohydrate levels. Aellen et al. (1993) reported that exercise intensity acts as an important modulator of the beneficial effects of exercise on lipoprotein profiles, since high-intensity training (above the anaerobic threshold in cycling) failed to induce beneficial alterations in the lipoprotein profiles, especially in the anti-atherogenics (reduces LDL and increases HDL). Thus, given that short-term/high-intensity contractions performed during acute, high-intensity exercise widely rely on anaerobic metabolism, such a mechanism does not seem to be the predominant modulator of the lipid profile. However, in a recent study, Tsekouras et al. (2008) examined the effect of high-

intensity intervals of aerobic training on VLDL-TG secretion in men. They observed that subjects who had trained by running on the treadmill for 8 weeks at 90% VO_{2peak} had a reduced rate of VLDL-TG secretion, suggesting that high-intensity exercise induced changes in lipid profiles. Our data are consistent with these results, whereby high-intensity exercise in the control condition diet reduces total and LDL cholesterol. Therefore, high-intensity exercise may be a strategy for improvement of lipoprotein metabolism in healthy subjects, provided that the supply of carbohydrates is appropriate.

Energy expenditure was found to be similar under conditions of different carbohydrate levels (control, low and high). Indeed, this finding excludes this effect to influence the lipoprotein profile (Ferguson et al. 1998; Kokkinos and Fernhall 1999).

Studies conducted by Ferguson et al. (1998) indicate a threshold of energy expenditure that could promote changes in the lipoprotein profile. Their data suggest that moderate-intensity exercise sessions that cause expenditure of more than 1.100–1.500 kcal, performed at approximately 70% of oxygen consumption, had a greater effect on HDL cholesterol. In the present study, high-intensity exercise promoted a total low energy expenditure (124.4 ± 22.5 kcal to combined situations), although it induced changes in LDL-c and total cholesterol levels. Another possible mechanism responsible for modulating the reduced total cholesterol and LDL-c is the pathway of reverse cholesterol (Leaf 2003). This route removes cholesterol from the circulation and distributes it to the peripheral tissues and liver. Actually, both acute and chronic aerobic exercise increase the activity of lecithin:cholesterol acyltransferase (L-CAT), the enzyme responsible for the cholesterol ester transfer to the HDL-c, and reduce the activity of the plasmatic cholesterol ester transfer protein (CETP), the enzyme responsible for transferring the ester in HDL cholesterol to other lipoproteins (Campaigne et al. 1993; Seip et al. 1993; Ferguson et al. 1998; Grandjean et al. 2000; Durstine et al. 2002). It can be speculated that decreased concentrations of total cholesterol and LDL-c in the plasma might be attained through the exchange of cholesterol ester among tissues and lipoproteins to the HDL-c (Leaf 2003). However, this mechanism may have been impaired by low- and high-carbohydrate levels during exercise at high intensity, because no changes in total cholesterol and LDL-c were found. The precise mechanisms by which carbohydrate levels (low and high) fail to modulate the concentration of HDL total cholesterol and LDL-c need to be better examined.

We did not observe changes in HDL-c concentrations, but it should be emphasised that the HDL-c particle concentration is a measurement of the total cholesterol content

alone, carried in plasma HDL. However, HDL particles are composed of a collection of different molecular lipoprotein species containing cholesterol, triglycerides, apolipoproteins (apos) and phospholipids that vary in composition and function (Fruchart et al. 1993). A limitation of the present study was that we did not measure the HDL subparticle composition. Park and Ransone (2003) reported the existence of the threshold intensity of acute aerobic exercise (expending 350 kcal) necessary to promote a significant increase in HDL.

The global rise in obesity has stimulated considerable public interest in the use of very low-carbohydrate, high-carbohydrate, and high-fat (LC) diets, such as the 'Atkins diet', to promote weight loss (Astrup et al. 2004). Volek et al. (2005) described favourable and consistent outcomes in many carbohydrate-restricted diet studies and showed in their review that this diet may well reduce TG and total cholesterol and increase HDL. In the present study, we observed that diets low and high in carbohydrates consumed prior to the high-intensity exercise impaired the benefits observed in the control diet. Indeed, the study conducted by De Bock et al. (2008) showed that adaptations to short-term endurance training on protein transporter fat in the skeletal muscle were inhibited in the group that had carbohydrate intake. However, when combined with acute exercise, the diet fails to increase the benefits. Nonetheless, in spite of the present data, the chronic effects of such interventions are still uncertain and deserve further investigation.

Therefore, high-intensity exercise, even with low energy expenditure, may be a strategy to benefit lipoprotein metabolism, which is in line with previous findings regarding moderate aerobic exercise (Park and Ransone 2003; Lira et al. 2008; Magkos et al. 2007, 2008). Although such changes may be related to the aforementioned mechanisms, we cannot rule out the possibility that other factors may contribute to these findings, such as changes in hormonal concentration in plasma and/or enzymatic activity.

In summary, our results indicate that: although acute high-intensity exercise generated low energy expenditure, it induced a reduction in LDL-c and total cholesterol levels. However, these alterations in lipoprotein profiles induced by acute high-intensity exercise disappeared when carbohydrate levels were changed in 2 days with a low- or high-CHO diet.

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