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SHORT COMMUNICATION

Theodore J. Angelopoulos · Rebecca Lewis
Thanassis Jamurtas · Chris Schumann

Significant changes in VLDL-Triacylglycerol and glucose tolerance in obese subjects following ten days of training

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Abstract We characterized the effect of ten days of training on lipid metabolism in 6 [age 37.2 (2.3) years] sedentary, obese [BMI 34.4 (3.0) $kg \cdot m^{-2}$] males with normal glucose tolerance. An oral glucose tolerance test was performed prior to and at the end of the 10 d of training period. The duration of each daily exercise session was 40 min at an intensity equivalent to ~75% of the age predicted maximum heart rate. Blood measurements were performed after an overnight fast, before and at the end of the 10 d period. Plasma triacylglycerol was significantly ($p < 0.05$) reduced following exercise training (2.15 ± 0.29 vs. 1.55 ± 0.28 $mmol \cdot l^{-1}$). Very low density lipoprotein-triacylglycerol was also significantly ($p < 0.05$) reduced (1.82 ± 0.3 vs. 1.29 ± 0.29 $mmol \cdot l^{-1}$). No significant changes in high density lipoprotein-cholesterol were observed as a result of training. Following training fasting plasma glucose and fasting plasma insulin were significantly reduced [Glucose: 5.9 (0.2) $mmol \cdot l^{-1}$ vs. 5.3 (0.22) $mmol \cdot l^{-1}$ ($p < 0.05$); Insulin 264.3 (53.8) $\rho \cdot mol \cdot l^{-1}$ vs. 200.9 (30.1) $\rho \cdot mol \cdot l^{-1}$, $p = 0.05$]. The total area under the glucose curve during the OGTT decreased significantly ($p < 0.05$). These preliminary data suggest that short-term exercise, without concomitant loss of body mass, induces favorable changes in plasma triacylglycerol, and very low density lipoprotein-triacylglycerol and glucose tolerance but has no effect on high density lipoproteincholesterol.

Key words Exercise · Obesity · Lipoproteins · Glucose tolerance

T.J. Angelopoulos · R. Lewis · C. Schumann
Laboratory of Applied Physiology,
The University of Southern Mississippi,
Bot 5142 Hattiesburg Mississippi 39406, USA

T. Jamurtas
Department of Exercise and Sports Science,
University of North Carolina at Greensboro,
Greensboro, NC 27412, USA

Introduction

The significance of elevated plasma triacylglycerol (TAG) level as a factor for coronary heart disease (CHD) remains controversial despite the fact that many large studies have indicated that the TAG level is a univariate predictor of coronary risk (Austin 1989). High density lipoprotein-cholesterol (HDL-c), however, is inversely related to CHD (Stampfer et al. 1991). The obese state has been recognized to accentuate such established risk factors for atherosclerotic disease as dyslipidemia, hypertension and insulin resistance. Regular exercise is the cornerstone of the treatment of many dyslipidemias, insulin resistance and obesity. Specifically, regular aerobic exercise has been shown to lower atherosclerotic risk through an increase in HDL-c and a reduction in plasma TAG and body mass (Thompson et al. 1991; Weintraub et al. 1989). Such changes in lipids, however, could be partly mediated by mass loss since mass loss and regular exercise have been shown to produce favorable changes in plasma TAG and HDL-c (Weintraaub et al. 1989; Katzel et al. 1995). We have previously shown that short-term exercise brings about significant changes in TAG and HDL-c without concomitant mass loss in sedentary non-obese individuals (Angelopoulos et al. 1993; Seip et al. 1995). It is not known, however, whether acute training without concomitant loss in body mass, would result in similar changes in the atherosclerotic profile of obese individuals. The present study reports the effects of a short-term exercise regime that resulted in no change in body mass in obese sedentary males, while it did induce favorable changes in the TAG profile without concomitant changes in HDL-c.

Methods

Six sedentary male non-smokers participated on a volunteer basis in this study. Their characteristics were [mean (SE)] as follows: age, 37.2 (2.3) years; height, 182.0 (3.6) cm; mass, 113.1 (8.6) kg; body

mass index (BMI), 34.4 (3.0) kg · m^{-2}; and waist to hip ratio (WHR) .97 (.01). All subjects gave their written consent to participate. Subjects in this investigation served as their own controls. Prior to acceptance into the study subjects were required to undergo initial screening consisting of medical history, anthropometric measurements, oral glucose tolerance test (OGTT) and a graded exercise test. Only healthy individuals with normal glucose tolerance participated in the study. None were taking any medications or hormones. The research protocol was approved by the University of Sourthern Mississippi's Institution Review Board for Human Subjects Research. Subjects exercised for 40 min (30 min on a motor-driven treadmill and 10 min on a cycle ergometer) at an intensity equivalent to ~75% of the age-predicted maximum heart rate for 10 consecutive days. Subjects were told not to change their eating habits and to refrain from alcohol. Diet was assessed during training by a three-day dietary recall. Glucose tolerance was assessed using a 75 g 2 h OGTT, performed after a 12 h overnight fast prior to and approximately 18–20-h after the final exercise bout. Plasma insulin levels (fasting and during the OGTT) were determined using a radioimmunoassay (RIA) kit (ICN, CA). Plasma glucose (fasting and during the OGTT) was analyzed using a Kodak Ektachem DT60^{TM} system. Samples for lipoprotein determination were obtained by venipuncture. Sampling followed an overnight fast and, in the case of post-training samples, was 18–20 h after the most recent exercise bout. Plasma lipoproteins were measured in the Washington University Lipid Research Clinic Core Laboratory, which participates in the lipid standardization program of the Center for Disease Control. For any given subject, pre- and post-training samples were analyzed in the same assay. Plasma lipids and lipoproteins were measured in the Washington University Lipid Research Clinic Core Lab. Apolipoprotein B (apoB) in the LDL subfraction was measured by immunonephelometry (Behring Diagnostics, Somerville, NJ). Haematocrit (Hct) was determined by a microcapillary technique (Van Beaumont, 1972). A Wilcoxon *t*-test was used to evaluate for differences before and after training. Statistical significance was accepted at the $p < 0.05$ level of confidence.

Results

Body mass did not change [113.1 (8.6) vs. 112.9 (8.4) kg] following training. Hct was not different [.43 (0.01) vs. .44 (0.02)] between the blood sampling points (Table 1), suggesting that the venous blood variables were not influenced by changes in vascular volume. Energy expenditure was 1894.1 (122.2) KJ · $session^{-1}$. Mean energy intake and diet composition assessments were based on a three-day dietary recall and were 12,719.00 (447.7) KJ · day^{-1}, and consisted of 40.5 (2.5)% carbohydrate, 18.1 (1.5)% protein, 41.4 (1.6)% fat respectively. Table 1 presents means (±SE) for the lipid variables. TAG and VLDL-TAG were significantly ($p < 0.05$) decreased following 10 d of exercise as compared to pre-participation with values of 2.15 (0.25) vs. 1.55 (0.28) mmol · l^{-1} and 1.82 (0.3) vs. 1.29 (0.29) mmol · l^{-1} respectively. There were no differences ($p > 0.05$) in TC, LDL-C, LDL-apoB, HDL-C HDL_2-C, and HDL_3-C in response to 10 d of exercise as compared to pre-exercise. Figure 1 presents glucose and insulin responses to a 75 g OGTT. The area under the glucose curve decreased 14% from 1,046.8 (93) mmol · l^{-1} · 120 min^{-1} to 905.6 (79) mmol · l^{-1} · 120 min^{-1} ($p < 0.05$), while the area under the insulin curve decreased 19% from 130,298.0 (15,100.5) ρ · mol · l^{-1} · 120 min^{-1} to 105,594.5 (14,007.0) ρ mol · l^{-1} · 120 min^{-1} ($p < 0.05$) following 10 days of exercise. Fasting glucose was lower ($p < 0.05$), [5.9 (0.2) vs. 5.3 (0.2) mmol · l^{-1}] following 10 d of exercise as compared to

Table 1 TAG, VLDL-TAG, TC, LDL-c, HDL-c, HDL-c subfractions, LDL-apoB, and Hct before and after 10 days of training

	Initial	After 10 d of exercise
TAG (mmol · l^{-1})	2.15 ± 0.29	1.55 ± 0.28*
VLDL-TAG (mmol · l^{-1})	1.82 ± 0.3	1.29 ± 0.29*
TC (mmol · l^{-1})	4.59 ± 0.28	4.12 ± 0.40
LCL-c (mmol · l^{-1})	2.83 ± 0.25	2.65 ± 0.29
HDL-c (mmol · l^{-1})	0.84 ± 0.08	0.86 ± 0.08
HDL_2-c (mmol · l^{-1})	0.016 ± 0.005	0.03 ± 0.01
HDL_3-c (mmol · l^{-1})	0.83 ± 0.08	0.84 ± 0.07
LDL-apoB (mmol · l^{-1})	1.65 ± 0.14	1.50 ± 0.17
Hct	.44 ± .01	.43 ± .02

*$p < 0.05$ indicates mean was different post-training as compared to pre-training value

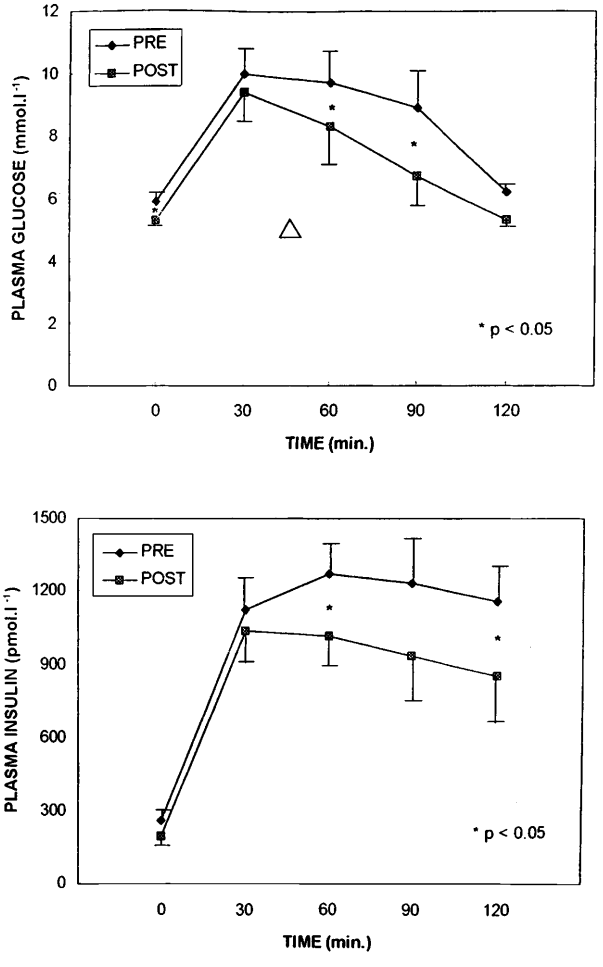

Fig. 1 Means and SEs for glucose and insulin during OGTT. *indicates that at this time point during the OGTT means are different ($p < 0.05$) between pre-training and post-training. Δ indicates that the total area under the glucose curve during the OGTT was different ($p < 0.05$) between pre-training and post-training

pre-exercise. Fasting insulin was also lower following training as compared to pre-training [264.3 (53.8) $\rho \cdot \text{mol} \cdot l^{-1}$ vs. 200.9 (30.1) $\rho \cdot \text{mol} \cdot l^{-1}$], ($p = 0.05$). Glucose concentration and insulin response at 60 min (during the OGTT) were reduced following 10 d of exercise as compared to pre-exercise with values 9.7 (0.9) $\text{mmol} \cdot l^{-1}$ vs. 8.3 (1.2) $\text{mmol} \cdot l^{-1}$ and 1,271.4 (134.9) $\rho \cdot \text{mol} \cdot l^{-1}$ vs. 1,017.4 (116.2) $\rho \cdot \text{mol} \cdot l^{-1}$, respectively. Glucose concentration at 90 min (during the OGTT) was also reduced following 10 d of exercise as compared to pre-exercise with values 8.9 (1.3) $\text{mmol} \cdot l^{-1}$ vs. 6.7 (0.8) $\text{mmol} \cdot l^{-1}$. Glucose concentration and insulin response at 120 min (during the OGTT) were reduced following 10 d of exercise as compared to pre-exercise with values 6.2 (0.4) $\text{mmol} \cdot l^{-1}$ vs. 5.3 (0.2) $\text{mmol} \cdot l^{-1}$ ($p = 0.07$) and 1,156.6 (185.9) ρ $\text{mol} \cdot l^{-1}$ vs. 855.3 (184.4) $\rho \cdot \text{mol} \cdot l^{-1}$ ($p < 0.05$) respectively.

Discussion

The purpose of the present investigation was to determine the effects of acute training on plasma TAG and HDL-c in obese sedentary subjects with normal glucose tolerance. The results demonstrated: 1) TAG and VLDL-TAG were significantly reduced following exercise training, 2) no significant changes in HDL-c were observed following training, 3) Glucose tolerance was improved following 10 d of daily exercise.

Chronic exercise training has been shown to decrease TAG and VLDL-TAG in sedentary individuals. Houmard et al. (1994) showed a significant reduction in plasma TAG and VLDL-TAG in middle-aged obese subjects in response to 14 weeks of training. Després et al. (1990), also reported a significant reduction in VLDL-TAG in young overweight men following 100 days of training. It could be argued, however, that these changes in plasma TAG and VLDL-TAG may have been at least partly due to significant reductions in mass observed in both studies (Houmard et al. 1994; Despres et al. 1990), since it has been shown that mass loss results in decreased plasma TAG and VLDL-TAG (Weintraub et al. 1989; Katzel et al. 1995). The reports regarding the effects of short-term exercise without concomitant weight loss, however, appear to be conflicting. Rogers et al. (1988) reported a significant reduction in plasma TAG following 7 days of training in overweight men. Cononie et al. (1994), however, reported no significant changes in Tg and VLDL-TAG following seven days of exercise in overweight elderly men and women. Our study showed significant reductions in TAG and VLDL-TAG following 10 days of daily exercise. We have shown that short-term exercise raises the plasma activity lipoprotein lipase (LPL), a protein bound to capillary endothelium, whose main function is to metabolize TAG in circulating lipoproteins. Furthermore, it has been shown that insulin levels affect VLDL particle synthesis and secretion (DeFronzo et al. 1991) and regulate LPL activity (Kiens et al. 1989). In our study, fasting insulin and insulin response to a glucose challenge decreased by 24% and 19% respectively. It is reasonable then to speculate that the changes in TAG and VLDL-TAG observed in the present study were at least partly mediated by changes in insulin and LPL activity. Of particular significance was the observation that HDL-c did not change following this training protocol despite the dramatic reductions in TAG and VLDL-TAG. A reduction in TAG has been associated with favorable HDL-c changes. Although the underlying biochemical mechanism behind the exercise induced increase in HDL-c is not completely understood, evidence suggests that the enzymes LPL, hepatic triglyceride lipase (HTGL), lecithin:cholesterol acyl-transferase (LCAT), and cholesterol ester transfer protein (CETP) play a key role (Seip et al. 1993). The present study was not designed to address enzymatic changes following this training protocol. Further investigations, however, should examine the acute effects of exercise on these enzymes in the obese state.

Chronic exercise training has been previously shown to improve glucose tolerance and insulin response to a glucose load in untrained individuals (Holloszy et al. 1986). The reports regarding the effects of short-term exercise on glucose tolerance and insulin however, are conflicting. Some studies have reported significant improvements in glucose tolerance (Cononie et al. 1994), whereas others have failed to show significant differences in either glucose tolerance or insulin responsiveness (Gulve et al. 1995). As shown in previous studies exercise mediates its effects on glucose tolerance by means of short-term increase in insulin sensitivity and responsiveness of skeletal muscle. It appears, however, that the disparity between results of the above mentioned studies could be due to differences in the exercise training protocol employed. In our study the areas under the glucose and insulin curves during the OGTT decreased 14% and 19% respectively and our observations are consistent with the data published by Cononie et al. (1994).

In conclusion, our preliminary data indicate that short-term exercise induces favorable changes in TAG and VLDL-TAG in sedentary middle-aged obese individuals. Such salutary changes in plasma and VLDL-TAG are not due to mass loss but could be mediated by favorable changes in glucose tolerance and insulin responsiveness. The lack of favorable changes in HDL-c despite the changes in TAG warrants further investigation.

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