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A single bout of whole-body resistance exercise augments basal VLDL-triacylglycerol removal from plasma in healthy untrained men

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Running head: Resistance exercise and VLDL-TAG kinetics

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ABSTRACT

A single bout of prolonged aerobic exercise lowers plasma triacylglycerol (TAG) concentrations the next day by increasing the efficiency of very low-density lipoprotein (VLDL)-TAG removal from the circulation. The effect of resistance exercise on VLDL-TAG metabolism is not known. Therefore, we evaluated VLDL-TAG kinetics by using stable isotope labeled tracers in 8 healthy untrained men (age: 25 ± 1 years, body mass index: 24.5 ± 0.6 kg/m²) in the post-absorptive state in the morning, on two separate occasions: once after performing a single 90-min bout of strenuous isokinetic resistance exercise (3 sets \times 10 repetitions, 12 exercises at 80% of maximum peak torque production, with a 2-min rest interval between exercises) on the preceding afternoon and once after an equivalent period of rest. Fasting plasma VLDL-TAG concentration the morning after exercise was significantly lower than the morning after rest (0.23 ± 0.04 vs. 0.33 ± 0.06 mmol/l, respectively, $P=0.001$). Hepatic VLDL-TAG secretion rate was not different ($P=0.31$), but plasma clearance rate of VLDL-TAG was significantly higher (by $26 \pm 8\%$) after exercise than rest (31 ± 3 vs. 25 ± 3 ml/min, respectively, $P=0.004$), and the mean residence time of VLDL-TAG in the circulation was significantly shorter (113 ± 10 vs. 144 ± 18 min, respectively, $P=0.02$). Fasting plasma free fatty acid and serum beta-hydroxybutyrate concentrations were both significantly higher after exercise than rest ($P<0.05$), whereas plasma glucose and serum insulin concentrations were not different ($P>0.30$). We conclude that, in healthy untrained men, a single bout of whole-body resistance exercise lowers fasting plasma VLDL-TAG concentration by augmenting VLDL-TAG removal from plasma. The effect appears to be qualitatively and quantitatively similar as that reported previously for aerobic exercise.

INTRODUCTION

Increased fasting plasma triacylglycerol (TAG) concentrations are associated with higher risk for coronary heart disease, independently of other major lipid risk factors [1]. Postabsorptive hypertriacylglycerolemia typically results from an imbalance between the rate of very low-density lipoprotein (VLDL) TAG secretion from the liver and the rate of VLDL-TAG removal from the circulation [2,3]. It was demonstrated recently that a single bout of prolonged aerobic exercise (90-120 min at moderate intensity) decreases basal VLDL-TAG concentration the next day by increasing the rate of whole-body VLDL-TAG clearance [4,5]. This effect is probably mediated by the secretion of fewer but TAG-rich VLDL particles after exercise [4], which are hydrolyzed faster by lipoprotein lipase (LPL) in vitro [6] and removed faster from the circulation in vivo [7]. In addition, possible transient increases in LPL activity in skeletal muscle (but not adipose tissue) after exercise [8] may further augment TAG clearance [9,10].

Although it is well established that single bouts of prolonged (≥ 90 min), moderate-intensity aerobic exercise significantly reduce basal plasma TAG concentrations the following day in a manner that depends on the total energy expenditure of exercise [11-14], there is currently no consensus regarding the effects of resistance exercise. Petitt *et al.* [15] showed that a single resistance exercise bout (total energy expenditure ~ 1.7 MJ) lowered fasting plasma TAG concentrations the next day in recreationally weight-trained men and women, and the effect was actually greater than that of an aerobic exercise bout of similar energy expenditure. However, others have failed to observe a TAG-lowering effect in the fasting state after single bouts of resistance exercise ranging in total energy expenditure from ~ 0.6 MJ to 2.6 MJ [16-18], and Burns *et al.* [19] found increased postprandial plasma TAG concentrations immediately after resistance exercise, though the effect was transient and disappeared ~ 8 hours following meal ingestion.

The effect of a single bout of resistance exercise on fasting plasma TAG concentration is therefore not clear. Furthermore, all previous studies examined TAG metabolism in response to high-fat meals [15-19]; there is currently no information regarding the effects of resistance

exercise on hepatic VLDL-TAG secretion and plasma clearance rates, the net balance of which ultimately determines plasma VLDL-TAG concentration [2,3]. Therefore, we evaluated the effects of a single bout of whole-body isokinetic resistance exercise on basal VLDL-TAG kinetics in the postabsorptive state in healthy, untrained young men.

METHODS

Volunteers and preliminary testing

Eight young men (age: 25.3 ± 0.8 years, body weight: 78.3 ± 3.5 kg, height: 178 ± 3 cm, body mass index: 24.5 ± 0.6 kg/m², body fat: $18 \pm 1\%$; means \pm SEM) participated in the study. All volunteers were non smokers and their body weight was self-reported stable (± 1 kg) for at least two months before the study. They were healthy, as indicated by comprehensive history, physical examination and standard blood tests. All volunteers were recreationally active but untrained (participated in moderate intensity physical activities less than twice per week) and were instructed to refrain from vigorous exercise for at least two weeks before the study. The protocol was approved by the Human Studies Committee of Harokopio University, Athens, Greece, and written informed consent was obtained from all participants.

Anthropometric and body composition measurements were conducted approximately one week before the study. Body weight and height were measured to the nearest 0.1 kg and 0.5 cm, respectively, and body mass index was calculated. Total body fat mass and fat-free mass were determined by dual-energy X-ray absorptiometry (model DPX-MD, Lunar Corp., Madison, WI, software version 3.6) [20].

Peak oxygen consumption (VO₂ peak) was determined by a submaximal incremental brisk walking test (modified Balke treadmill protocol) [21]. Subjects warmed up for 5 minutes and were familiarized with the treadmill (Technogym Runrace, Italy). Expiratory gases were collected by using a breath-by-breath gas analyzer (SensorMedics, Vmax229D, Yorba Linda, CA). After warm-up, treadmill speed was kept constant and grade was increased by 2% every 3

minutes. The test was terminated at 80% of heart rate reserve, and VO_2 peak was predicted from the oxygen consumption / heart rate relationship [21]. All volunteers were familiarized with the isokinetic resistance exercise protocol on a separate day, and three maximum intensity repetitions were performed to determine peak torque production for each exercise.

Experimental protocol

Volunteers performed two time-matched trials (rest and exercise) approximately one week apart, in a randomized counterbalanced design. For the exercise trial, they were admitted to the laboratory on the evening before the isotope infusion study and performed a single bout of resistance exercise on an isokinetic dynamometer (BIODEX System 3 Pro, Shirley, NY). The exercise protocol was designed so as to involve whole-body resistance exercise, consisting of 3 sets of 10 repetitions at 80% of maximum peak torque production, for a total of 12 exercises (Table 1). All participants had visible contact with a bar chart on the monitor that indicated performance for each repetition and obtained verbal feedback to achieve the target. The exercise protocol was performed for each limb separately and the resting period between exercises was 2 minutes. The order in which the individual exercises were performed was the same for all participants. Total exercise duration was 90 ± 1 min. Heart rate was monitored continuously by a telemetric heart rate monitor (Polar Accurex Plus, Finland). Energy expenditure was measured by indirect calorimetry using a face mask throughout exercise, as done in previous studies examining the effect of resistance exercise on TAG metabolism [15,18]. Despite that the short and intermittent duration of resistance exercise invalidates the typical assumptions of indirect calorimetry, since the respiratory quotient (RQ) is consistently ≥ 1 , using a fixed caloric cost for the volume of O_2 consumed during exercise, as done by other investigators [16,19], yielded slightly higher (by $\leq 15\%$) estimations for the total energy expenditure of our exercise session; such a difference is not likely of physiological importance. Calibration of the O_2 and CO_2 was performed once before the start of the exercise bout and at 45 minutes during the 2-minute resting period. For the time-matched resting trial, participants were requested to refrain from any form of physical activity and remain rested at home.

Stable isotope infusion

The next morning, volunteers arrived at the laboratory with minimal physical activity and after having fasted overnight. One catheter was inserted into a forearm vein to administer stable isotope labeled tracers and a second catheter was inserted into a contralateral hand vein for blood sampling. The latter was kept warm with a heating pad. Catheters were flushed with 0.9% NaCl solution after each blood sampling. Subjects were allowed to relax and get used to the catheters for approximately 1 hour. At 14 ± 1 h after completion of the exercise trial or the equivalent period of rest (time = 0), a baseline blood sample was taken to determine fasting plasma and serum metabolite concentrations and background tracer-to-tracee ratio (TTR) of glycerol in VLDL-TAG. A bolus of [1,1,2,3,3- $^2\text{H}_5$]glycerol (75 $\mu\text{mol/kg}$ body wt; Goss Scientific Instruments Ltd., Essex, UK), dissolved in 0.9% NaCl solution, was then administered through the catheter in the forearm vein, and blood samples were obtained at 15 min after tracer injection and then every hour for 6 h, to determine glycerol TTR in VLDL-TAG. Indirect calorimetry was performed using a ventilated canopy (Sensormedics, Vmax229D, Yorba Linda, CA) for 15 min before catheter insertion and every hour after tracer injection. The first 5 min served as a habituation period. Oxygen consumption (VO_2) and carbon dioxide production (VCO_2) were measured for the remaining 10 min to estimate resting metabolic rate (RMR) [22] and whole-body fat oxidation rate [23]. For the entire duration of the studies, volunteers remained fasted in the laboratory in a sitting position. Water consumption was allowed ad libitum.

Diet assessment and control

Upon entry into the study, volunteers received instructions on how to record food and beverage intake and provided a detailed recording of all nutrient intake for the 3 days before the first trial, including the last meal (dinner) consumed after the completion of the exercise or resting session (whichever came first). They were then instructed to reproduce the same diet for the 3 days preceding the second trial. None of the subjects reported any deviation from the dietary plan. Food journals were analyzed by using nutrition analysis software (Nutritionist Five, FirstDataBank, San Bruno, CA). No alcohol or caffeine intake was permitted for two days before each isotope infusion study. Also, no caloric consumption was permitted during the exercise session or the equivalent period of rest. Dinner was completed within 2 h after cessation of the

exercise or resting sessions. Thereafter, volunteers remained fasted for ~12 h before starting the tracer infusion the next morning.

Sample collection and storage

Blood samples were collected in pre-cooled tubes containing EDTA as anticoagulant. Samples were placed in ice immediately, and plasma was separated by centrifugation within 30 min of collection. Aliquots of plasma (~3 ml) were transferred into plastic culture tubes and kept in the refrigerator for immediate isolation of VLDL. The remaining plasma samples were stored at -80°C until measurement of total plasma TAG, total and high-density lipoprotein (HDL) cholesterol, glucose, and free fatty acid (FFA) concentrations. All determinations were performed by using commercially available enzymatic kits (Alfa Wassermann Diagnostics Technologies, West Caldwell, NJ) on an automated analyzer (ACE Schiapparelli Biosystems, Fairfield, NI). The concentration of low density lipoprotein (LDL)-cholesterol in plasma was calculated according to the Friedewald equation [24]. A separate blood sample portion was transferred into tubes with no additives for serum preparation. Serum was stored at -80°C until analysis of insulin with an immunoenzymetric fluorescent method using a commercially available kit (ST AIA-PACK IRI, Tosoh Medics, San Francisco, CA) on an automated analyzer (Tosoh AIA 600II, Tosoh Medics, Inc., San Francisco, CA). Serum beta-hydroxybutyrate (Randox Laboratories Ltd., Antrim, UK), creatine kinase and lactate dehydrogenase (Alfa Wassermann Diagnostics Technologies, West Caldwell, NJ) concentrations were measured by using commercially available enzymatic kits on an automated analyzer (ACE Schiapparelli Biosystems, Fairfield, NI). Whole-body insulin sensitivity was assessed with the homeostasis model assessment of insulin resistance (HOMA-IR) index as: $\text{HOMA-IR} = \text{fasting serum insulin } (\mu\text{U/ml}) \times \text{fasting plasma glucose (mmol/l)} / 22.5$ [25]. Paired samples for each volunteer were analyzed in the same batch. For each trial, a small aliquot of baseline blood samples was used for determination, in triplicates, of haematocrit and haemoglobin concentration by microhaematocrit and cyanomethaemoglobin assays, respectively (Boehringer Mannheim Diagnostics, Mannheim, Germany). Changes in plasma volume between rest and exercise trials were assessed by using the Dill and Costill equation [26].

VLDL preparation and analyses

The VLDL fraction was prepared as previously described [5]. Briefly, ~2 ml of plasma was transferred into Quick Seal Centrifuge Polyallomer Tubes (Beckman Instruments Inc., Palo Alto, CA), overlaid with a NaCl/EDTA solution ($d = 1.006 \text{ g/ml}$) and spun for 3 h at 90000 rpm at 4°C , in an Optima TLX ultracentrifuge equipped with the fixed-angle TLN-100 rotor (Beckman Instruments Inc., Palo Alto, CA). The top layer, containing the VLDL, was removed and collected quantitatively by tube slicing (CentriTube slicer, Beckman Instruments Inc., Palo Alto, CA), and stored at -80°C until analyses. The concentration of VLDL-TAG was measured by using an enzymocolorimetric assay (Alfa Wassermann Diagnostics Technologies, West Caldwell, NJ) and the concentration of VLDL-apolipoprotein B-100 (VLDL-apoB-100) was measured immediately in fresh samples by using an immunoturbidimetric assay (Randox Laboratories Ltd., Antrim, UK) on an automated analyzer (ACE Schiapparelli Biosystems, Fairfield, NI).

Isolation of TAG in VLDL particles

Aliquots (~750 μl) of the isolated VLDL fractions were deproteinized with 3 ml acetone, centrifuged (3000 rpm for 15 min at 4°C), and the supernatant was dried under vacuum (SpeedVac, ThermoSavant, Holbrook, NY), before isolating VLDL-TAG by thin layer chromatography (TLC). Dried samples were resuspended in chloroform : methanol (3:1), spotted on a LK6D silica gel plate (60 \AA silica gel, 250 μm layer thickness; Whatman, Maidstone, UK), and developed with heptane : diethyl ether : formic acid (80:20:2) in an enclosed developing chamber. The TAG band was visualized with 0.01% rhodamine 6G in the TLC lanes, scraped off the TLC plate, and transferred into 13 \times 160 mm test tubes. TAG were extracted from the silica gel with chloroform : methanol (3:1) and dried under vacuum. The dried samples were hydrolyzed by resuspension in 10% acetyl chloride in methanol and incubation for 30 min at 70°C , and then dried under vacuum. Glycerol was derivatized with heptafluorobutyric (HFB) anhydride (30 min incubation at 70°C) and samples were dried under vacuum. Dry HFB-glycerol was dissolved in heptane and stored at -40°C . The TTR of glycerol in VLDL-TAG was determined by gas-chromatography / mass-spectrometry (GC/MS; MSD 5973 system, Hewlett-Packard, Palo Alto, CA), by selectively monitoring the ions at mass to charge ratios (m/z) 467 and 472 [27]. Calibration curve for standards with known isotopic enrichment was used.

Calculation of VLDL-TAG kinetic parameters

The fractional catabolic rate (FCR, pools/h) of VLDL-TAG was determined by using the monoexponential approach, after logarithmically transforming the raw TTR data [28]. The total rate of hepatic VLDL-TAG secretion was calculated as: total VLDL-TAG secretion ($\mu\text{mol}/\text{min}$) = $\text{FCR} \times C \times \text{PV} / 60$, where C is the concentration of VLDL-TAG in plasma ($\mu\text{mol}/\text{ml}$) and PV is the plasma volume (ml), which was calculated as 55 ml per kg of fat-free mass [29]. It was assumed that VLDL-TAG volume of distribution is equal to PV since VLDL particles are essentially restricted in the plasma compartment [30]. The rate of whole-body VLDL-TAG plasma clearance (ml/min), which is an index of the efficiency of VLDL-TAG removal from the circulation, was calculated by dividing the rate of VLDL-TAG disappearance from plasma ($\mu\text{mol}/\text{min}$), which equals VLDL-TAG secretion rate under steady state conditions, by the concentration of VLDL-TAG in plasma ($\mu\text{mol}/\text{ml}$). The mean residence time of VLDL-TAG in the circulation (min) was calculated as $60 / \text{FCR}$.

Statistical analysis

Data were analyzed using SPSS 11.0 (SPSS Inc., Chicago, IL). All variables were normally distributed according to the Shapiro-Wilks test; hence comparisons between rest and exercise were performed by using parametric procedures, and results are presented as means \pm SEM. The Student's t-test for paired samples was used to evaluate metabolic and kinetic parameters between rest and exercise. Analysis of covariance for repeated measures was performed to test the stability of total plasma and VLDL-TAG concentrations and VO_2 throughout the 6-h kinetic study for both trials, taking into account inter-correlation between time points. Covariates as potential cofounders were, age, fat-free mass, body mass index and VO_2 peak. Statistical significance was set at $P \leq 0.05$. Based on a previous reproducibility study, a sample size of 8 would allow for detecting physiologically significant differences in VLDL-TAG secretion and clearance rates of ≥ 20 -25% in magnitude, at $\alpha = 5\%$ and with 80% power [31].

RESULTS

Body composition, VO₂ peak, dietary intake and exercise energy expenditure

Average fat mass and fat-free mass were 14 ± 1 kg and 64 ± 3 kg, respectively. Absolute and relative VO₂ peak were 3.4 ± 0.3 l/min and 42.8 ± 2.8 ml/kg·min, respectively. Dietary energy intake during the 3 days preceding each trial averaged 8.2 ± 0.9 MJ/day ($48 \pm 4\%$ from carbohydrate, $36 \pm 6\%$ from fat, and $16 \pm 2\%$ from protein), and the last meal (dinner) consumed after the completion of the exercise and resting sessions provided 3.9 ± 0.5 MJ (119 ± 1 g carbohydrate, 34 ± 7 g fat, and 35 ± 7 g protein). Average oxygen consumption during the resistance exercise session was 10.9 ± 0.8 ml/kg·min, at an average heart rate of 125 ± 5 bpm (including the resting intervals between the exercise sets). Mean RQ during exercise averaged 1.02 ± 0.01 . The gross energy expenditure of exercise was 1.63 ± 0.17 MJ, whereas exercise net energy expenditure (i.e., above resting values) was 1.19 ± 0.15 MJ. Mean work production for each exercise and total work production of the entire exercise bout, measured by the isokinetic dynamometer, are shown in Table 1.

Metabolic rate and substrate oxidation during fasting

Basal VO₂ (exercise: 262 ± 10 ml/min and rest: 252 ± 10 ml/min, $P = 0.25$) and hence RMR (exercise: 1764 ± 80 kcal/day and rest: 1706 ± 65 kcal/day, $P = 0.16$) were not significantly different the morning after the two interventions. Likewise, there was no effect of evening exercise compared with rest on basal RQ (0.75 ± 0.01 and 0.77 ± 0.02 , respectively, $P = 0.32$) and whole-body fat oxidation rate (0.093 ± 0.012 and 0.078 ± 0.009 g/min, respectively, $P = 0.12$).

Substrate and insulin concentrations during fasting

Plasma volume changes between the two trials were minimal ($2 \pm 1\%$, $P = 0.14$). Evening resistance exercise, compared with rest, did not affect serum insulin and plasma glucose concentrations and therefore whole-body insulin sensitivity (HOMA-IR index), nor plasma total, HDL-, and LDL-cholesterol and serum lactate dehydrogenase concentrations (Table 2). Plasma FFA, serum beta-hydroxybutyrate and creatine kinase concentrations were significantly higher the morning after exercise than rest, whereas total plasma TAG, VLDL-TAG and VLDL-apoB-

100 concentrations were significantly lower; the ratio of VLDL-TAG to VLDL-apoB-100 concentrations was significantly increased by exercise (Table 2).

Basal VLDL-TAG kinetics

In both trials, plasma VLDL-TAG concentrations remained constant throughout the 6-h sampling period (Figure 1), indicating a metabolic steady state. The time-courses of glycerol TTR in VLDL-TAG and logarithmically transformed TTR data are shown in Figure 2. The FCR of VLDL-TAG was significantly higher after exercise than rest (0.56 ± 0.05 and 0.46 ± 0.05 pools/h, respectively, $P = 0.007$). Exercise did not affect the rate of hepatic secretion of VLDL-TAG ($P = 0.31$), but increased VLDL-TAG plasma clearance rate by $26 \pm 8\%$ ($P = 0.004$) compared with rest (Figure 3). The mean residence time of VLDL-TAG in the circulation was significantly shorter after exercise than rest (113 ± 10 and 144 ± 18 min, respectively, $P = 0.02$).

DISCUSSION

In the present study, we examined basal VLDL-TAG kinetics the morning after a single, evening bout of strenuous resistance exercise or an equivalent period of rest in healthy, untrained young men. We found that resistance exercise lowered fasting plasma VLDL-TAG concentrations by ~30% compared with rest, due to enhanced VLDL-TAG removal from the circulation (by ~25%), resulting in ~30 min shorter residence time of VLDL-TAG in the bloodstream. This effect was independent of changes in glucose and insulin concentrations and insulin sensitivity (i.e., HOMA-IR index). Resistance exercise did not affect the rate of VLDL-TAG secretion from the liver, even though it greatly augmented FFA availability (as indicated by the higher FFA concentrations), likely due to increased hepatic fatty acid oxidation (as indicated by the higher beta-hydroxybutyrate concentrations) and perhaps also use of FFA for tissue TAG repletion. These results suggest that the hypotriacylglycerolemic effect of acute resistance exercise is qualitatively and quantitatively similar to that reported previously for endurance exercise [4,5].

Single bouts of prolonged (≥ 90 min), moderate-intensity aerobic exercise lower fasting plasma TAG concentrations the following day, provided that sufficient energy be expended during

exercise [11-14]. It has long been recognized [32] that the hypotriacylglycerolemic effect of endurance exercise manifests acutely, ~12 hours after the cessation of a single exercise bout, lasts for 1-2 days and does not result from metabolic adaptations to repeated exercise sessions (i.e., training) [33,34]. Resistance/weight training also favourably modifies several risk factors for coronary heart disease [35], but the effects of acute resistance exercise on TAG metabolism are not well understood. Results from previous studies using a similar protocol to ours are inconsistent: a single bout of resistance exercise of approximately the same duration (~90 min) and total energy expenditure (1.7-2.3 MJ) has been reported to either decrease [15] or not affect [16] fasting and postprandial plasma TAG concentrations the next morning. Others reported unchanged fasting but reduced postprandial TAG concentrations, when measurements were made ≥ 14 h after the cessation of resistance exercise sessions of even shorter duration (40-50 min) and total energy expenditure (0.75-1.4 MJ) [18], whereas an increase in postprandial plasma TAG concentrations was observed when measurements were made immediately postexercise [19].

The reasons for these inconsistent results are not clear, nor can they be inferred from our study. One possible explanation may be differences in energy balance between the rest and exercise trials. It was recently shown that the hypotriacylglycerolemic effect of aerobic exercise in the fasting and postprandial states is abolished when the energy expended during exercise is replaced by overfeeding [36], and single bouts of resistance exercise varying in duration from 20 to 90 min and total energy expenditure from 0.6 to 2.6 MJ do not affect fasting and postprandial TAG concentrations in the face of increased caloric intake [17]. Hence the inherent inability to accurately quantify the energy expenditure of resistance exercise by means of indirect calorimetry [15,18] or otherwise [16,17,19] and possible differences in dietary energy intake during the days preceding each trial might have contributed to the reported inconsistent results. Clearly, the role of energy balance and its components (exercise energy expenditure and dietary energy intake) in mediating the effects of exercise on TAG metabolism warrant further investigation. In the present study, controlling of the diet for the 3 days preceding the two tracer infusions (by recording all food and beverage intake before the first trial and replicating this diet before the second trial) ensured that there were no major differences in energy content and macronutrient composition of the diet, including the last meal (dinner) consumed after the

completion of the exercise and resting sessions. Hence subjects were in a negative energy balance during the exercise compared with the resting trial, by the net energy expenditure of the exercise session (i.e., by ~1.2 MJ). And, we did observe a significant reduction in fasting plasma TAG and VLDL-TAG concentrations the morning after exercise than rest; the main purpose of our study was to describe the mechanisms for the hypotriacylglycerolemic effect of acute resistance exercise.

Our results indicate, for the first time in humans *in vivo*, that increased VLDL-TAG removal rate from the circulation mediates the TAG-lowering effect of resistance exercise. We cannot ascertain the site/tissue responsible for this effect, since VLDL-TAG clearance refers to all possible routes of VLDL-TAG removal from plasma. This includes hydrolysis by LPL (and possibly also hepatic lipase), transfer of TAG to other lipoproteins (e.g., HDL) via neutral lipid exchange, conversion of VLDL to lipoproteins of higher density, i.e., intermediate density lipoproteins (IDL) and LDL, as well as removal of the whole VLDL particle from plasma via interaction with hepatic and/or peripheral receptors [37]. Our kinetic data do not allow for distinguishing between these routes. Previous studies have shown that a single, prolonged bout of moderate-intensity aerobic exercise (90-120 min at 60% of VO_2 peak) enhances the efficiency of VLDL-TAG removal from plasma without affecting hepatic VLDL-TAG secretion [4,5] and reduces the hepatic secretion of VLDL-apoB-100 [4], indicating that the liver after exercise secretes fewer but TAG-rich (and therefore possibly also larger) VLDL particles [4]. This is consistent with our observation that resistance exercise increased the ratio of VLDL-TAG to VLDL-apoB-100 concentrations. There is evidence from several *in vivo* studies in humans [37,38] and animals [7,39] suggesting that the removal of TAG from the core of TAG-rich, large VLDL particles is more efficient than TAG removal from TAG-poor, small VLDL, possibly because increasing TAG content (and size) of lipoprotein particles enhances their susceptibility to hydrolysis by LPL [6]. We observed a significant decrease in VLDL-apoB-100 concentration after resistance exercise compared with rest, which may have resulted from reduced hepatic secretion of VLDL-apoB-100 (i.e., secretion of fewer VLDL particles), as shown previously after aerobic exercise [4]. Hence secretion of TAG-rich VLDL after resistance exercise may underlie the augmented plasma clearance rate of VLDL-TAG via enhanced whole-body LPL-

mediated lipolysis; this mechanism does not presuppose any changes in LPL activity of tissues after exercise.

The above point notwithstanding, there is evidence suggesting that LPL mRNA, protein mass and activity in skeletal muscle, but not adipose tissue, increase transiently within 4-8 h after exercise cessation and remain elevated for some 16-20 h postexercise [40-42] or even longer [43]. This coincides with the time frame of our measurements. Elevated skeletal muscle LPL activity after exercise may therefore account for at least some of the increase in VLDL-TAG removal from the circulation we observed after a single bout of whole-body resistance exercise, by augmenting VLDL-TG clearance across the previously exercised muscles [9,10]. It has been estimated that whole-body skeletal muscle LPL activity accounts for some 10% of whole-body TAG clearance (whole-body adipose tissue LPL activity accounts for ~5%) in fasting humans at rest, but may account for as much as 50% of the clearance of exogenously infused TAG [8]. A single endurance exercise bout may increase skeletal muscle LPL activity by ~70% late into recovery (~18 h after exercise cessation) [43], although it does not enhance basal skeletal muscle (or adipose tissue) blood flow at that time [10]. Dynamic knee extension exercise also increases skeletal muscle LPL activity and the clearance of VLDL-TAG across the exercised muscles at rest [9,42], but data on other types of resistance exercise is not available. Other possible routes for the accelerated VLDL-TAG removal after resistance exercise include enhanced TAG transfer from the core of VLDL to HDL in exchange for cholesterol esters, as well as faster removal of the VLDL particles themselves from the circulation. The first mechanism is unlikely, since enhanced transfer of TAG from VLDL to HDL after exercise would result in TAG-enrichment of HDL particles and thus accelerated clearance of HDL, thereby lowering HDL-cholesterol concentrations [44]. Furthermore, if anything, prolonged exercise has been shown to significantly reduce the concentration and activity of cholesterol ester transfer protein [45], i.e., the major enzyme responsible for the loss of TAG from the core of VLDL in exchange for cholesterol esters from HDL. Faster removal of VLDL particles themselves from the circulation, whether via conversion to lipoproteins of higher density such as IDL and LDL, or via direct uptake from the liver or peripheral tissues through interaction with specific receptors, is also unlikely to have contributed to the increase in whole-body clearance of VLDL-TAG after resistance exercise. Although we did not assess VLDL-apoB-100 kinetics, it has been shown

previously that the plasma clearance rate and the mean residence time of VLDL-apoB-100 (i.e., VLDL particles) in the circulation are not different the morning after single bouts of moderate-intensity endurance exercise lasting 1-2 h or equivalent periods of rest [4,46].

We observed that prior resistance exercise did not affect the rate of VLDL-TAG secretion from the liver. This is consistent with all previous studies examining the effects of endurance exercise on TAG metabolism in the postabsorptive state, which found no differences in hepatic VLDL-TAG secretion the morning after exercise or rest [4,5,46]. Interestingly, resistance exercise significantly increased basal plasma FFA concentrations by ~35%, suggesting that plasma FFA availability was higher after exercise than rest [4,46]. On the other hand, we did not observe any significant changes in glucose and insulin concentrations and whole-body insulin sensitivity (assessed with the HOMA-IR index) after resistance exercise, in accordance with previous reports [15,16,18]. Insulin and the availability of plasma FFA are considered the major regulatory determinants of basal VLDL-TAG secretion by the liver [47,48], but several recent studies provide no evidence in support of this notion under various physiological conditions in humans in vivo [46,49,50]. The fate of the excess FFA after resistance exercise is uncertain. Some may be directed towards storage to replenish tissue TAG stores that were likely depleted by prior resistance exercise [51]. Our data suggest that at least some of the additional FFA are oxidized in the liver, as evidenced by the almost doubling of serum beta-hydroxybutyrate concentrations the morning after exercise than rest. This may have actually prevented an increase in hepatic VLDL-TAG secretion after exercise, by limiting the intrahepatic availability of fatty acids (which would be greater after exercise than rest) for TAG synthesis and secretion. Moreover, we observed a non-significant ~20% increase in whole-body fat oxidation rate after exercise, which did not reach statistical significance perhaps due to type II error [15,52]; hence some of the excess FFA after exercise may be oxidized in peripheral tissues as well. Still, exercise-induced changes in total plasma TAG concentration [53] and VLDL-TAG concentration and kinetics [5] are independent of those in basal whole-body fat oxidation. Overall, our findings suggest that the morning after a single bout of resistance exercise, basal plasma FFA availability is not the primary determinant of hepatic VLDL-TAG secretion, consistent with observations made previously following endurance exercise [4,46].

In summary, a single bout of strenuous resistance exercise in the evening lowers total plasma and VLDL-TAG concentrations the next morning in healthy, normolipidemic, non-obese, untrained young men. Augmented VLDL-TAG removal from the circulation is the mechanism responsible for the hypotriacylglycerolemic effect of acute resistance exercise, without any changes in VLDL-TAG secretion from the liver. The effects of resistance and endurance exercise on basal VLDL-TAG metabolism appear thus to be qualitatively and quantitatively similar. It is currently not known whether resistance exercise similarly affects VLDL-TAG metabolism in other population groups (e.g., in women), or in high-risk individuals such as the obese patients or those suffering from hyperlipidemia. These questions should be addressed in future investigations. Nonetheless, results from the present study support the premise that resistance exercise may be beneficial for the management of hypertriacylglycerolemia.

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FIGURE LEGENDS

Figure 1. Very low-density lipoprotein-triacylglycerol (VLDL-TAG) concentration in plasma throughout the 6-h sampling period the morning after a single evening bout of resistance exercise and an equivalent period of rest. Values are means \pm SEM. There is a significant effect of trial (exercise versus rest, $P = 0.03$), but no effect of time ($P = 0.29$), or an interaction between trial and time ($P = 0.89$).

Figure 2. Time-course of tracer-to-tracee ratio (TTR) of glycerol in very low-density lipoprotein-triacylglycerol (VLDL-TAG) (*a*) and ln-transformed TTR data (*b*) throughout the 6-h sampling period the morning after a single evening bout of resistance exercise and an equivalent period of rest. Values are means \pm SEM.

Figure 3. Hepatic very low-density lipoprotein-triacylglycerol (VLDL-TAG) secretion rate (*a*) and whole-body VLDL-TAG plasma clearance rate (*b*) the morning after a single evening bout of resistance exercise and an equivalent period of rest. Values are means \pm SEM. * Significantly different from rest ($P = 0.004$).

Table 1. Work production for each exercise and total work production of the entire resistance exercise bout performed on the isokinetic dynamometer

Exercise	Angular velocity (degrees/sec)	Work production (joules)
Leg press	120	8444 ± 654
Leg pull	120	1860 ± 178
Shoulder press	180	1786 ± 287
Chin ups	180	4499 ± 493
Knee extension	120	9417 ± 908
Knee curl	120	5664 ± 977
Chest press	150	3449 ± 335
Upright row	150	2181 ± 202
Hip flexion (horizontal position)	120	5944 ± 1096
Hip extension (horizontal position)	120	4097 ± 317
Shoulder abduction	120	2882 ± 314
Shoulder adduction	120	9102 ± 876
Total work production		59326 ± 986

Values are means ± SEM. Work production for each exercise is presented as the sum of left and right sides for all sets.

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Table 2. Fasting metabolite concentrations the morning after a single evening bout of resistance exercise and an equivalent period of rest

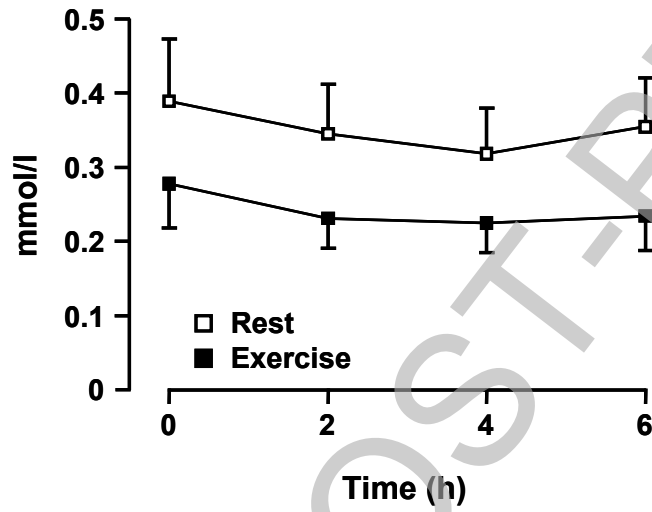
	Rest	Exercise	<i>P</i> -value
Insulin (μ U/ml)	6.2 \pm 0.4	5.9 \pm 0.7	0.66
Glucose (mmol/l)	5.5 \pm 0.2	5.6 \pm 0.1	0.33
HOMA-IR index	1.50 \pm 0.01	1.46 \pm 0.01	0.84
Total cholesterol (mg/dl)	183 \pm 7	172 \pm 7	0.27
HDL-cholesterol (mg/dl)	40 \pm 2	38 \pm 2	0.18
LDL-cholesterol (mg/dl)	127 \pm 5	120 \pm 5	0.35
Creatine kinase (U/l)	127 \pm 22	518 \pm 157	0.04
Lactate dehydrogenase (U/l)	187 \pm 9	206 \pm 7	0.08
Free fatty acids (mmol/l)	0.59 \pm 0.09	0.80 \pm 0.09	0.04
Beta-hydroxybutyrate (mg/dl)	0.23 \pm 0.06	0.44 \pm 0.10	0.03
Total plasma TAG (mmol/l)	0.77 \pm 0.11	0.61 \pm 0.08	0.04
VLDL-TAG (mmol/l)	0.33 \pm 0.06	0.23 \pm 0.04	0.001
VLDL-apoB-100 (mg/dl)	5.5 \pm 1.1	2.4 \pm 0.7	0.01
VLDL-TAG / VLDL-apoB-100	6.0 \pm 0.8	8.4 \pm 1.1	0.04

Values are means \pm SEM.

HOMA-IR, homeostasis model assessment of insulin resistance; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TAG, triacylglycerol; VLDL, very low-density lipoprotein; apoB-100, apolipoprotein B-100.

Figure 1

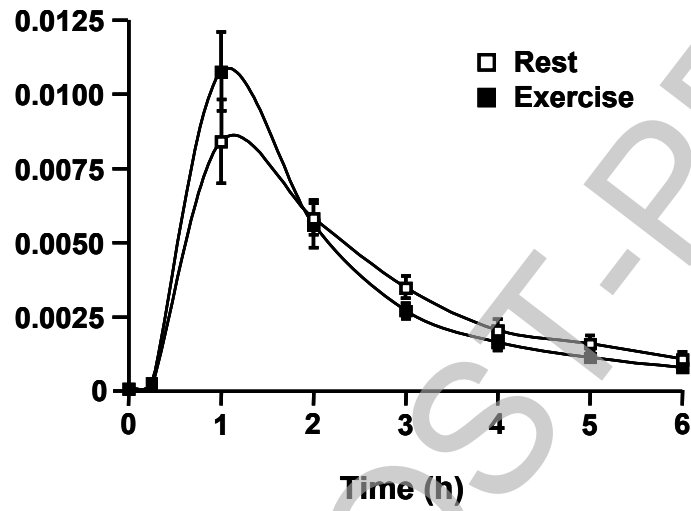
VLDL-TAG concentration



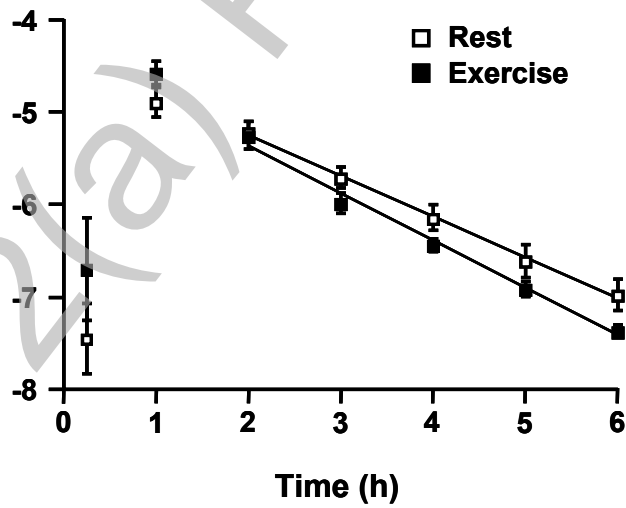
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Figure 2

(a) VLDL-TAG-glycerol TTR



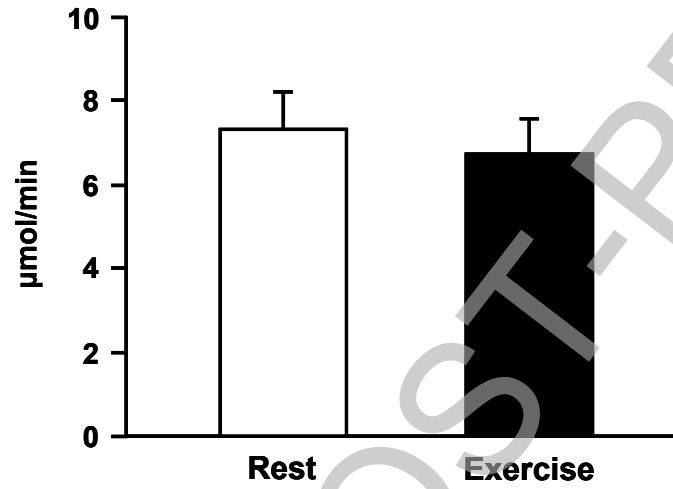
(b) ln VLDL-TAG-glycerol TTR



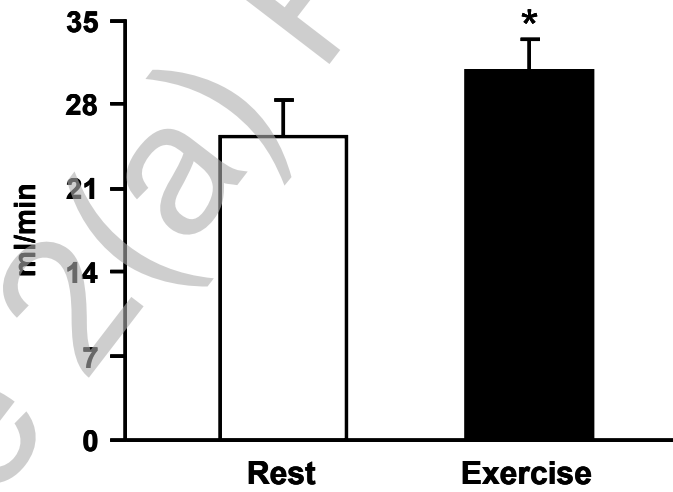
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Figure 3

(a) Hepatic VLDL-TAG secretion rate



(b) VLDL-TAG plasma clearance rate



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