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High-volume resistance training reduces postprandial lipaemia in postmenopausal women

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(Asked 6 February 2015)

Abstract

The aim of this study was to compare the effects of 11 weeks of low-volume resistance training (LVRT) and high-volume resistance training (HVRT) on muscle strength, muscle thickness (MT), and postprandial lipaemia (PPL) in postmenopausal women. Thirty-six healthy and untrained postmenopausal women (age, 58.9 ± 5.8 years; 68.6 ± 10.3 kg; and BMI, 26.9 ± 4.8 kg · m⁻²) participated in resistance training 3× per week for 11 weeks (HVRT = 12; LVRT = 13; and control group = 11). Biochemical variables, both pretraining and post-training, were evaluated 16 h after the administration of an oral fat tolerance test (OFTT) and metabolic variable during [energy expenditure (EE)] and after training session [excess postexercise oxygen consumption (EPOC)]. Muscle strength (1 RM) and MT were also calculated, and no significant differences were observed between the groups for PPL (mmol · L⁻¹ per 5 h) as measured by glucose, high-density lipoprotein, low-density lipoprotein, and total cholesterol. EE total (EE + EPOC; 6.12 ± 1.21 MJ vs. 2.26 ± 0.85 MJ), resting fat oxidation (5.52 ± 1.69 g · h⁻¹ vs. 4.11 ± 1.12 g · h⁻¹); MT (vastus medialis, 21.4 ± 1.8 mm vs. 18.4 ± 1.2 mm and vastus lateralis 22.3 ± 1.2 mm vs. 20.8 ± 1.3 mm); triacylglycerol (TAG) 0, 1, 2, 4; and 5 h after OFTT, TAG area under the curve (AUC) (5.79 ± 0.42 vs. 7.78 ± 0.68), and incremental AUC (−46.21 ± 14.42% vs. 7.78 ± 4.68%) were all significantly different post-training for HVRT versus LVRT, respectively (P < 0.05). The results of this investigation suggest that HVRT reduces PPL in postmenopausal women.

Keywords: energy expenditure, resistance exercise, menopause, muscle thickness, resting fat oxidation

Introduction

Elevated postprandial triacylglycerol or elevated postprandial lipaemia (PPL) concentrations are associated with the development of cardiovascular disease and other factors for health such as obesity, metabolic syndrome, hypertension, and type II diabetes (Burns & Stensel, 2008; Joon Cho et al., 2008; Petitt, Arngrimsson, & Cureton, 2003; Roche & Gibney, 2000; Zafeiridis et al., 2007), accounting for over 23% of female mortality and being implicated as the world's leading cause of death in women at the onset of menopause (Joon Cho et al., 2008).

Postmenopausal women lose much of the cardio-protective effect of endogenous estradiol through hormonal changes (i.e., decrease of oestrogen), and their incidence of cardiovascular disease rises above that of men (Gill & Hardman, 2000; Mikkola & Clarkson, 2002).

From a metabolic point of view, PPL can be considered as a transient episode of hypertriglyceridemia that occurs several times each day (Chapman, 2007). With evidence suggesting that the magnitude of the plasma triglycerides response to fat intake is a major determinant of low-density lipoprotein (LDL) heterogeneity measured in fasted state, it is important to identify the factors that influence this response, particularly in forms of exercise such as resistance training (Zafeiridis et al., 2007). Rapid removal of triglycerides-rich lipoproteins will reduce the severity of PPL and potentially the atherogenic phenomenon (Hardman, Lawrence, & Herd, 1998).

Skeletal muscle has a major effect on energy expenditure (EE) and is considered to be an...
important determinant of resting metabolic rate (Binzen, Swan, & Manore, 2001; Poehlman & Dvorak, 2000) due to the greater oxygen uptake of skeletal muscle compared to that of other tissues of body. For this reason, it has been suggested that an increased muscle mass following resistance training may reduce multiple risk factors for cardiovascular disease (Poehlman, Dvorak, DeNino, Brochu, & Ades, 2000). Resistance training is known to be a strong inducer of insulin sensitivity improvement (Poehlman & Dvorak, 2000; Tsetsonis, Hardman, & Mastana, 1997), to decrease fasting triglycerides, and to markedly increase fat oxidation rate at rest and 24 h after a single exercise bout (Binzen et al., 2001; Treuth, Hunter, Weinsier, & Kell, 1995). As resistance training is considered to be naturally intermittent, it may therefore induce prolonged postexercise oxygen consumption, EE and a greater use of fat during recovery (Binzen et al., 2001; Tremblay et al., 1990; Treuth et al., 1995).

With literature suggesting that postexercise oxygen consumption and EE are influenced pretraining by the menstrual cycle phase, care must be taken when considering the metabolic response to resistance training in premenopausal women. These findings are important due to metabolic (postexercise oxygen consumption, EE, and basal metabolic rate) and biochemical (lipids) variables having a direct dose–response relationship with the volume of resistance training (Binzen et al., 2001; Matsuo, Saitoh, & Suzuki, 1999).

The volume of resistance training plays a significant role in strength, morphological, metabolic, and biochemical changes (Shimano et al., 2006). The optimal number of sets for a resistance training programme (one vs. three or low volume vs. high volume) remains controversial, however, and the majority of studies indicate no significant difference for increases in either strength (Krieger, 2009) or muscle thickness (MT) (Radaelli et al., 2013). To the best of our knowledge, only three studies – Burns, Corrie, Holder, Nightingale, and Stensel (2005), Burns and Stensel (2008), and Zafeiridis et al. (2007) – have reported the effect of different training volumes on changes in PPL after a single session of strength exercise. However, none of these studies evaluated the response of a regular or systematic programme of resistance training on triglycerides concentrations during PPL, or its relationship with increases in strength and muscle mass. Furthermore, the aforementioned studies have conflicting results when comparing different training volumes, with none of the studies cited finding differences between low- and high-volume resistance exercise (RES).

Indeed, it seems that moderate-intensity low-volume resistance training (LVRT) substantially reduces the overall time period of muscle recovery when compared to high-volume resistance training (HVRT) (Burns et al., 2005; Burns & Stensel, 2008; Shannon et al., 2005; Zafeiridis et al., 2007). Nonetheless, all of the above studies are an acute response to a single session of RES and we could not find any study published that has evaluated the effects of chronic training on PPL in postmenopausal women. Therefore, the objective of this study was to compare the effects of 11 weeks of both LVRT and HVRT PPL, EE, basal metabolic rate, postexercise oxygen consumption, muscle strength, and MT in postmenopausal women.

**Methods**

**Participants**

Thirty-six healthy and untrained postmenopausal women (age 58.9 ± 5.8 years; body mass 68.6 ± 10.3 kg; height 158.5 ± 3.2 cm; body mass index (BMI) 26.9 ± 4.8 kg · m⁻², 14 with overweight and 1 obese; waist circumference 79.8 ± 7.7 cm; peak oxygen uptake (VO₂peak) 21.2 ± 2.0 mL · kg⁻¹ · min⁻¹) who had not engaged in regular and systematic resistance training for at least 1 year participated in the study. The study excluded individuals with a history of severe endocrine, metabolic, cardiovascular, neuromuscular diseases; diabetes; dyslipidaemia; and smokers. Participants were recruited through advertisements in a widely read local newspaper. Prior to participation, participants were fully informed of the design of the study and the possible risks and discomforts related to the procedures in order to provide informed written consent. The study protocol complied with the Declaration of Helsinki and was approved by the Ethics Committee.

**Study design**

This was a longitudinal study that lasted 11 weeks of training. Thirty-six volunteers were divided randomly and by electronic method, according to BMI, into HVRT group (=12, five with overweight; three sets), LVRT group (=13, five with overweight and one obese; one set), and a control group (CG = 11, four with overweight; no physical training throughout the duration of the study). Both LVRT and HVRT groups were required to perform 15 maximum repetitions (RM, defined as the heaviest possible weight that could be maintained for the designated number of repetitions), and the load was adjusted during the training period for attempted and error (15 RM) in eight individual exercise routines (bench press, biceps curl, triceps halter, one arm row back, leg press, knee extensor, and knee flexion and abdomen crunch) three times a week during the training period. A 40-s time...
interval was used between sets and exercises, resulting in the duration of each training session lasting approximately 15 min for LVRT and 45 min for HVRT. Lipid profiles of all participants’ blood samples were analysed two weeks or one week before the first evaluation. One week before the first evaluation, all participants were also familiarised with resistance training exercise and performed a one maximum repetition (1 RM) test on a knee extensor exercise machine in two different moments. All variables were analysed preresistance and postresistance training.

Participants’ characterisation

Before the resistance training programme, participants attended the laboratory for a preliminary session where anthropometric data were collected and the VO\text{peak} test was performed. Body mass and height were recorded for the calculation of BMI (body mass (kg)/height (m\textsuperscript{2})). Skinfolds were measured using a skinfold caliper (Harpenden Scientific Model, Brand Cescorf, Porto Alegre, Brazil); bone diameters were measured by caliper and anthropometer (Cescorf, Porto Alegre, Brazil), using tape measure perimeters (Sanny, São Bernardo do Campo, São Paulo); and weight and height were measured by scale and stadiometer (Tanita, Pinheiros, São Paulo). The markings of the places and the technique of taking skinfold followed the standards of the International Society for the Advancement of Kinanthropometry (ISAK). Calculations of body composition were performed using the methodology of five components (Marfell-Jones, Olds, & Stewart, 2006 – ISAK). This methodology measures 39 benchmarks, being used for this work, the following variables are checked: (1) body mass; (2) height; (3) skinfolds (triceps, subcapular, biceps, iliac crest, supra spinal, abdominal, thigh, and medial calf); (4) circumferences (head, arm, chest, waist, thigh maximum, average thigh, calf, hip, forearm, and ankle); (5) bone diameters (biacromial, thorax transverse, ante-roposterior chest, bi-iliocrystal, bi-epicondylar humerus, bi-styloid of wrist, hand, bi-condylar femur, and bi-malleolar); and (6) bone lengths (acromion-radial-radial styloid, middle-dactilóidea estilóidea, ilium spinal bench, trochanteric bank, trochanter, tibial, lateral tibial bank, medial-medial tibial malleolus, leg length, and sitting height). The muscle mass in percentage was calculated by the following equation: muscle mass (%) = body mass – (fat mass + bone + residual weight)/100. Prior to the project, all measurers were found to meet the ISAK criteria for technical errors of measurement of <5% for skinfolds and <1% for all other variables.

Indirect calorimetry

The following criteria were used to determine maximal capacity for oxygen consumption during the test: the participant reached volitional fatigue, respiratory exchange ratio (RER) ≥1.15, Heart-rate (HR)\textsubscript{rest} ≥ 95% of age-predicted maximum HR (220 – age), and/or a plateau in oxygen consumption with increasing load. The VO\text{peak} was determined using the breath-by-breath method with an open circuit spirometry system (Metabolic Cart, CPX/D, MGC, Saint Paul, MN, USA). The progressive cycle-ergometer test (The Bike, Cybex, Ronkonkoma, NY, USA) consisted of an initial 3 min workload of 25 W followed by a constant-rate increase of 25 W · min\textsuperscript{-1} until exhaustion. The pedalling cadence was maintained between 60 and 70 r · min\textsuperscript{-1} throughout the test and a recovery period of 3 min at 25 W was performed by all participants following achievement of VO\text{peak}. Heart rate was monitored using short-range telemetry (S610, Polar Electro Oy, Kempele, Finland) and participants were verbally encouraged to perform the maximum effort during the test. The test lasted for 6–8 min in accordance with the recommendations of the American College of Sports Medicine (Garber et al., 2011) for postmenopausal women and was halted at any time if the participant presented an inability to maintain the pedalling cadence, displayed a plateau in the curve of the VO\textsubscript{2}, or a HR near the maximum predicted (220–age) (Cunha et al., 2011).

Muscle strength evaluation

The knee extensor 1 RM test of the dominant lower limb was performed through a knee extensor exercise with the same “extension chair” equipment used for the training sessions (Taurus, Brazil, resolution 0.1 kg). Was evaluated the quadriceps muscle because this muscle is what is the most affected the effects of aging and sarcopenia in elderly women (Correa et al., 2014). Movement speed during the test was controlled using a QUARTZ metronome with a 1-Hz resolution (for test details, see Correa et al. 2012). There were no significant differences between 1 RM tests at baseline (test 26.2 ± 4.1 kg; retest 26.8 ± 2.8 kg), and the intra-class correlation coefficient was 0.91.

Muscle thickness

Before and after the training period, the MT of the knee extensors was measured using B-mode ultrasound (Philips, VMI, Lagoa Santa, Brasil). Measurements were taken from each participant following 15 min of rest in the supine position. Post-test MT was assessed 3–5 days after the last training session in order to prevent errors in MT calculations due to swelling (Radaelli et al., 2013). A 38-mm 7.5-MHz
linear probe was placed on the skin perpendicular to the tissue interface. The probe was coated with a water-soluble transmission gel to provide acoustic contact without depressing the dermal surface. MT was determined in the vastus lateralis (VL), rectus femoris (RF), and vastus medialis (VM). The probe was positioned according to previous studies (Korhonen et al., 2009; Radaelli et al., 2013) and all images were digitised and later analysed using a specialist software (ImageJ, National Institute of Health, Bethesda, MD, USA, version 1.37). The intra-observer technical error utilised was the most commonly used measure of precision, which is the square root of measurement error variance <1% for all MT evaluation.

**Experimental procedure**

Preliminary assessments were performed in the morning following an overnight fast, with starting time held constant within participants. Dietary records were collected for the 3 days preceding the pretraining and post-training test, with participants requested to follow the same meal plan over both 3-day periods (Table II). In order to allow participants to accurately detail and standardise the description of food portions, a portfolio with photos was prepared (Becker et al., 2012). Participants were required to refrain from exercise for the 48 h preceding testing and asked to refrain from caffeine, alcohol, and strenuous exercise 48 h prior to the main visits.

Upon arrival at the laboratory, supine basal metabolic rate data were collected for 30 min via continuous indirect calorimetry (Metabolic Cart, CPX/D, MGC, Saint Paul, MN, USA), with data collected during 20–25 min of the rest period averaged and used to represent the resting metabolic activity (Becker et al., 2012; Cunha et al., 2011). The select time refers to the moment of the O$_2$ stabilisation consumption values, which was around 5 min after the beginning of the test.

On day 1, following resting, baseline measurements of O$_2$ consumption were measured continuously during the RES session of postexercise oxygen consumption and EE (ergospirometry); participants returned to the laboratory between 3:30 pm and 5:00 pm in order to perform one set (LVRT) or three sets (HVRT) of each exercise. Each set consisted of 15 repetitions (or failure) at around 65% 1 RM. A 40-s transition time was provided between each exercise and set. Immediately upon completion of the exercise session, the participant returned to the darkened room and remained in a supine position for 30 min of recovery for EPOC measurements (Figure 1). All metabolic variables were measured with a breath-by-breath method and outlier’s data were excluded. Data points within the Medical Graphics Corporation tests were excluded if the respiratory quotient was 0.6 or 1.2, the VT was 150 mL, or the VO$_2$ or VCO$_2$ were 50 mL · min$^{-1}$. Collected data were then averaged as the middle five of seven breaths.

**Metabolic response**

To account for the fact that RES reduces blood pH, increases CO$_2$, and changes the buffering of acids (thereby rendering RER inaccurate; Binzen et al., 2001; Ratamess et al., 2007), EPOC and EE were calculated as the EE per minute, estimated by multiplying absolute VO$_2$ (l ·min$^{-1}$) by 3.5 to transform to metabolic equivalents (METs), and then multiplying by 5.0 kcal · L$^{-1}$. Total RES session EE (for each RT group), EPOC, and total EE(EE + EPOC) were calculated by multiplying EE per minute by the protocol length and EPOC (L) by 5.0 kcal · L$^{-1}$, respectively. Area under the curve (AUC) and incremental area under the curve (IAUC) were calculated for the relative VO$_2$ response during exercise using a standard trapezoidal technique. Resting fat oxidation was calculated from the VO$_2$ and the Table of Zuntz (Petitt & Cureton, 2003).

![Figure 1. Experimental design of the OFTT protocol. The participants underwent a session of resistance exercises (RESs), pre and post 11 weeks of resistance training, with evaluation of energy expenditure (EE) and excess postexercise oxygen consumption (EPOC) between 3:30 pm and 5:00 pm. A standard meal was consumed at 8:00 pm; participants then fasted for 12 h and arrived at the laboratory between 7:30 am and 8:00 am [basal metabolic rate (BMR)] the following morning for the first blood sample. A fatty meal was consumed between 8:00 am and 8:30 am, and postprandial blood samples were collected at 8:00 am, 8:30 am, 9:30 am, 10:30 am, 11:30 am, and 12:30 pm to assess postprandial lipaemia (PPL).](https://example.com/figure1.png)
Oral fat tolerance test

On day 2, an oral fat tolerance test (OFTT) was administered following a 12-h overnight fast (approximately 15 h postexercise), beginning at 7:30 am and lasting for 5 h (until 12:30 pm). Upon arrival at the laboratory, participants rested for 5 min while a baseline (12-h overnight fasting) blood sample was collected. Participants were given 5–10 min to consume the meal, with no one reporting gastrointestinal discomfort or nausea at any stage. The test meal consisted of a whole “milkshake” (milk, ice cream, and sour cream), chosen due to the rapid gastric absorption of this compound (60% lipids, 30% carbohydrate, and 10% protein) (Figure 1). The energy provided by the meal was calculated individually to cover the EE throughout the basal metabolic rate. For the calculation, the volume of oxygen consumed by each individual was converted into METs. Based on the assumption that 1 MET is equivalent to 1.0 kcal · kg⁻¹ · h⁻¹, the EE of each participant was calculated by adding the values of body mass (kg). The high-fat meals were the same pretraining and post-training for both groups and had a mean MJ ± SD of 0.28 ± 0.04, 0.26 ± 0.06, and 0.28 ± 0.08 for HVRT, LVRT, and CGs, respectively, with no significant difference in the values of the test meals between groups. The energy and macronutrients’ content of the meal were calculated with the assistance of DietWin Professional Software (Brubins CAS, Porto Alegre, Brazil). Water was provided ad libitum during the trials (Becker et al., 2012).

Blood sample analyses

Blood samples were analysed to determine the levels of total cholesterol, glucose, HDL, LDL, and triglycerides. Baseline blood samples were collected in the antecubital region of the arm via the use of a disposable sterile cannula, followed by further measurements 1, 2, 3, 4, and 5 h into both trials (Figure 1). The cannula was kept patent throughout with a non-heparinised saline solution (9 mg · mL⁻¹ NaCl), and each blood sample collected corresponded to a volume of 10 mL (Burns et al., 2005; Burns & Stensel, 2008). The ambient temperature (22°C) and the relative air humidity (65%) were controlled in pretraining and post-training trials. Blood samples were stored in tubes with ethylenediamine tetraacetic acid for total cholesterol, HDL, LDL, and triglycerides, and in tubes with a fluoride anti-clotting agent for glucose analysis. Samples were centrifuged at 1400 g at 4°C for 10 min, and the resultant supernatants were aliquoted and frozen at −75°C for later analysis. Concentrations of total cholesterol, glucose, HDL, LDL, and triglycerides were analysed using the enzymatic colorimetric automatic method (cobas c111 analyser, Roche®, Nutley, NJ, USA). Total AUC with respect to baseline was calculated for triglycerides using the trapezoidal method as previously described (Burns & Stensel, 2008; Shannon et al., 2005). Total AUC and IAUC were analysed using Matlab software (version 7.14).

Data analysis

Descriptive statistics were calculated and are presented as mean ± SD. Data normality distribution was assessed using the Kolmogorov–Smirnov test. Levene’s homogeneity of variance test and the assumption of sphericity were satisfied for all analyses. For preintervention and postintervention comparisons between groups, a 2-way repeated measures analysis of variance (ANOVA) was used (3 groups × 2 times), with subsequent Bonferroni post hoc tests. The Student’s t-test for paired samples was used to compare the main effect of time (intervention) for parametric data. In addition, when appropriate, one-way ANOVA was performed to compare the relative changes (Δ%) between groups for triglycerides AUC and IAUC. Sample size was calculated using G*Power software (version 3.0.1) and it was determined that a sample size of n = 36 participants would provide a statistical power greater than 0.80 for all variables. The significance level for all statistical tests was P < 0.05 and all analyses were performed using SPSS statistical software version 19.0.

Results

Participant’s characteristics are listed in Table I. No statistical significant differences were found between groups in pretraining and post-training analysis with regard to height, body mass, muscle mass, BMI, waist and thigh circumference, maximal oxygen uptake, and basal metabolic rate.

EE, postexercise oxygen consumption, and EE total (EE + EPOC) during each exercise training session were significantly higher for HVRT versus LVRT in pretraining (5.01 ± 1.44 MJ vs. 1.82 ± 1.02 MJ) and post-training (6.12 ± 1.21 MJ vs. 2.26 ± 0.85 MJ), respectively (P < 0.001). However, no significant difference was found between groups in EE, EPOC, and EE total, pre- and post-RT programme (Table I). Resting fat oxidation was significantly higher ~16 h in post-training after the HVRT session than both LVRT (22%; P = 0.042) and CG (37%; P = 0.001), but not different among treatments at pretraining (Table I).

There were no differences in energy and macronutrient intakes during pretraining or post-training interventions in both groups (Table II).
Table I. Participants’ characterisation and metabolic response, pre and post 11 weeks of resistance training.

<table>
<thead>
<tr>
<th>Variables</th>
<th>HVRT, n = 12</th>
<th>LVRT, n = 13</th>
<th>CG, n = 11</th>
<th>P-value, T, G, and T × G</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>65.4 ± 10.8</td>
<td>64.3 ± 10.7</td>
<td>70.1 ± 9.9</td>
<td>68.6 ± 10.1</td>
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<tr>
<td>Body fat (kg)</td>
<td>25.0 ± 2.9</td>
<td>24.0 ± 2.8</td>
<td>26.4 ± 3.1</td>
<td>25.0 ± 3.1</td>
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<tr>
<td>Body fat (%)</td>
<td>39.3 ± 4.6</td>
<td>37.3 ± 3.7</td>
<td>37.8 ± 5.1</td>
<td>36.5 ± 5.2</td>
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<tr>
<td>Muscle mass (kg)</td>
<td>23.0 ± 2.1</td>
<td>25.0 ± 2.0</td>
<td>23.2 ± 1.9</td>
<td>24.0 ± 1.8</td>
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<tr>
<td>Muscle mass (%)</td>
<td>33.1 ± 4.1</td>
<td>35.9 ± 3.8</td>
<td>37.1 ± 3.8</td>
<td>38.1 ± 3.1</td>
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<td>Bone mass (kg)</td>
<td>6.7 ± 0.1</td>
<td>7.1 ± 0.1</td>
<td>6.8 ± 0.1</td>
<td>6.8 ± 0.1</td>
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<tr>
<td>BMI (kg · m⁻²)</td>
<td>25.4 ± 4.1</td>
<td>25.2 ± 3.5</td>
<td>26.1 ± 3.6</td>
<td>26.5 ± 3.8</td>
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<tr>
<td>Waist circumference (cm)</td>
<td>79.6 ± 9.2</td>
<td>78.2 ± 12.2</td>
<td>82.4 ± 7.4</td>
<td>81.2 ± 5.7</td>
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<tr>
<td>Thigh circumference (cm)</td>
<td>50.2 ± 9.2</td>
<td>52.2 ± 4.2</td>
<td>53.4 ± 7.8</td>
<td>54.2 ± 7.7</td>
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<tr>
<td>Waist-hip ratio</td>
<td>0.79 ± 0.06</td>
<td>0.78 ± 0.11</td>
<td>0.78 ± 0.05</td>
<td>0.77 ± 0.04</td>
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<tr>
<td>Metabolic response</td>
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<tr>
<td>VOpeak (mL · kg⁻¹ · min⁻¹)</td>
<td>18.8 ± 1.6</td>
<td>18.9 ± 2.0</td>
<td>18.4 ± 1.7</td>
<td>18.6 ± 2.0</td>
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<tr>
<td>BMR (MJ)</td>
<td>5.6 ± 1.0</td>
<td>5.4 ± 0.9</td>
<td>5.7 ± 0.8</td>
<td>6.1 ± 0.6</td>
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<td>Fat oxidation (g · h⁻¹)</td>
<td>3.32 ± 1.21</td>
<td>5.52 ± 1.69</td>
<td>3.33 ± 0.71</td>
<td>4.11 ± 1.12</td>
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<tr>
<td>EE (MJ)</td>
<td>0.28 ± 0.12</td>
<td>0.28 ± 0.09</td>
<td>0.10 ± 0.08</td>
<td>0.14 ± 0.08</td>
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<tr>
<td>EPOC (MJ)</td>
<td>0.24 ± 1.12</td>
<td>0.27 ± 0.81</td>
<td>0.08 ± 0.02</td>
<td>0.04 ± 0.02</td>
</tr>
</tbody>
</table>

Notes: Values are mean ± SD.

†Significant difference between pre and post 11 weeks of resistance training (P < 0.05).

*Significant difference between high-volume resistance training (HVRT) and low-volume resistance training (LVRT) (P < 0.05).

§Significant difference between HVRT in comparison to LVRT and CG (P < 0.05).

BMI, body mass index; VOpeak, peak oxygen uptake; BMR, basal metabolic rate; EE, energy expenditure; EPOC, excessive postexercise oxygen consumption.

The EE and EPOC were not assessed in CG because this group did not perform the period of training, and the focus of this study is an evaluation of the different volumes of resistance training. T, time (pre and post); G, group (HVRT, LVRT, and CG) and T × G = time × group interaction.
Resistance exercise and postprandial lipaemia

Significant time effects were observed for strength (1 RM; \( P < 0.001 \)), with 11 weeks of strength training leading to improvements in the absolute values of maximal dynamic strength (\( P < 0.001 \)) and MT (VL and VM) for HVRT compared with LVRT and CG (\( P = 0.042 \) and \( P = 0.048 \)). Significant changes occurred for MT (VL and VM) for LVRT in relation to CG, whereas RF MT was significantly different for both HVRT and LVRT when compared to CG (Table III).

The lipid profile (triglycerides, glucose, total cholesterol, HDL, and LDL) in pretraining (i.e., two weeks and one week prerestistance training) showed no significant differences between groups (Table IV).

No significant differences in OFTT amongst groups were observed at any point (0, 1, 2, 3, 4, and 5 h) for glucose (pretraining (Figure 2A) and post-training (Figure 2B)), TC (pretraining, Figure 2C, and post-training, Figure 2D), HDL (pretraining, Figure 2E, and post-training, Figure 2F), or LDL (pretraining, Figure 2G, and post-training, Figure 2H) or for triglycerides in pretraining (Figure 2A). However, post-training triglycerides at 0, 1, 2, 4 and 5 h (Figure 2B) and triglycerides total AUC and IAUC were significantly decreased in HVRT group compared to LVRT and CG (\( P < 0.05 \)) (Figure 3C).

Discussion

The major finding of this study was that 11 weeks of HVRT led to decreases in baseline triglycerides and the total serum triglycerides response (expressed as total AUC and IAUC), ~16 h after an OFTT. Furthermore, resting fat oxidation and MT of both the VL and VM were significantly increased post-training in HVRT. In this sense, to our knowledge, the present study was the first to evaluate postmenopausal women in a protocol of PPL before and after different volumes of RESs.

The results of this study are important for a number of reasons. Firstly, the increased serum triglycerides concentrations and associated PPL at the onset of menopause may increase the risk of coronary heart disease in this group when compared to men (Zaman et al., 2012). Therefore, any intervention that reduces coronary heart disease risk is particularly relevant for this group. Secondly, the effects of cyclic hormone concentrations on lipid metabolism may confound the study of premenopausal women (Binzen et al., 2001). Recently, Zaman et al. (2012) preformed a protocol of OFTT, without performing any type of exercise, comparing premenopausal with postmenopausal women. The triglycerides serum was significantly higher in the postmenopausal participants, and the authors concluded that the PPL response indicated
higher risk patterns in postmenopausal women. Furthermore, postmenopausal women are a homogeneous sample as shown in body composition (Table I), dietary (Table II), and pretraining lipids profile (Table III).

The present study showed significant reductions of triglycerides (Figure 3B, \(P = 0.004\)), total AUC (−34.31\%, \(P = 0.012\)), and IAUC (−46.21\%, \(P = 0.001\) (Figure 3C) in the post-training fasting state, a result which may have been due to an increased hydrolysis of circulating triglycerides by increased lipoprotein lipase enzyme activity, or a reduced secretion of very low-density lipoprotein (VLDL) by the liver (Roche & Gibney, 2000). In animals and humans, lipoprotein lipase exhibits a delayed increase in messenger RNA, protein, and activity up to 48 h after the last exercise session. Lipoprotein lipase induction was observed after local contraction of muscles in both rats and humans, suggesting that the response is localised to muscles recruited during exercise (Petitt et al., 2003). Exercise is followed by a period of increased postexercise free fatty acid mobilisation lipoprotein lipase activity and glucose uptake to facilitate repletion of muscle glycogen intramuscular triglycerides (Hardman et al., 1998). Theoretically, it is also possible that the appearance of chylomicrons into the circulation may have been delayed in LVRT and CG. This is unlikely, however, because peak triglycerides concentrations occurred at a similar time point in all groups (i.e., 3 h after OFTT protocol). The uptake of triglycerides from circulating chylomicrons and VLDL is a two-stage process, with triglycerides first hydrolysed by the action of lipoprotein lipase on the capillary endothelium to release free fatty acids, before the movement of these fatty acids into adipocytes to be entrapped and re-esterified (Gill & Hardman, 2000).

An increased utilisation of fatty acids occurs both during RES and in the postexercise state, resulting in muscle glycogen and intra-muscular triglycerides depletion (Gill & Hardman, 2000). Therefore, the
greater muscle usage time during HVRT will have the greatest effect on fat metabolism, with increased postexercise free fatty acid mobilisation and lipoprotein lipase activity acting to facilitate repletion of stores of muscle glycogen and intramuscular triglycerides. In the current study, resting fat
oxidation = 5.52 g · h\(^{-1}\), EE = 0.26 MJ, and total EE + excess postexercise oxygen consumption = 0.61 MJ (\(P = 0.042\) (Table I) were significantly different from triglycerides following 11 weeks of HVRT (3 sets/15 repetitions) when compared to LVRT (1 set/15 repetitions; resting fat oxidation = 4.11 g · h\(^{-1}\), EE = 0.14 MJ, and total EE = 0.18 MJ) and rest (R) = 40 s, suggesting that HVRT is more effective than LVRT for increasing the metabolic response in postmenopausal women. Some authors found significant decrease of triglycerides AUC in an acute response with young individuals as shown by Singhal, Trilk, Jenkins, Bigelman, and Cureton (2009) (EE = 1.81 MJ, 3 sets/16 repetitions, R = 2 min, \(P = 0.042\)) and Zafeiridis et al. (2007) (EE = 1.40 MJ, 2 sets/12 repetitions, R = 2 min, \(P = 0.017\)). Petitt and Cureton (2003) (EE = 1.70 MJ, 3 sets/10 repetitions, R = 2 min, \(P = 0.017\)) compared an acute session of resistance training with an aerobic session in young individuals. Although the authors did not evaluate the effect of different volumes of resistance training, it is important to note that the results indicated that multiple sets of resistance training have a strong effect on EE and consequently reach levels responsible for fat oxidation. Interestingly, other authors such as Burns et al. (2005) (EE = 2.30 MJ, 4 sets/10 repetitions, R = 2 min) and Shannon et al. (2005) (EE = 2.58 MJ, 5 sets/10 repetitions, R = 1 min) have shown a higher EE, yet found no significant values after an acute session of RESs.

In the present study, we found that EE = 0.28 MJ, EPOC (pre = 0.24 MJ and post = 0.27 MJ) (Table I), and total EE = 0.68 MJ post-training in HVRT all contributed to the lower level of triglycerides in HVRT compared with LVRT and CG. Another important factor was the 40-s time interval between sessions and exercises, shorter than that mentioned by the aforementioned authors (Burns et al., 2005; Petitt & Cureton, 2003; Shannon et al., 2005; Singhal et al., 2009; Zafeiridis et al., 2007). This interval makes for a more intense exercise session with higher EE, culminating in a substantial decrease in triglycerides after 11 weeks of resistance training due to its removal from adipose tissue stores and use for energy needs, thus increasing the rate of fat oxidation (Petitt et al., 2003). Recently, Davitt, Arent, Tuazon, Golem, and Henderson (2013) indicated that the reduction in PPL after both aerobic and RES sessions is achieved not by an enhanced clearance of dietary fat, but rather by a reduced abundance of endogenous fatty acid in plasma triglycerides.

Another limitation of these studies mentioned earlier is that they did not assess muscle size. An increased muscle mass is directly linked with increased fat metabolism (Szczypaczewska, Nazar, & Kaciuba-Uscilko, 1989) requiring a higher energy
demand both for the training session itself and for the transition period between muscle recovery and muscle adaptation. The literature supports the idea that different volumes of resistance training may have the same effects on strength and muscle hypertrophy (Krieger, 2009), and the strength gains observed following HVRT in this study were clearly related to an increased MT (VL, VM, and RF) when compared to CG (Table III). In relation to the development of maximal strength, our results agree with previous studies that the observed improvement in strength related to increases in muscle hypertrophy (Radaelli et al., 2013). Correa et al. (2014) showed pretraining results are similar to the present study for MT 1 RM, reinforcing the idea of homogeneity of the sample of postmenopausal women. Morphological adaptations showed an increased MT of the VL, VM, and RF (higher in HVRT) in all resistance training groups, indicating that hypertrophy likely contributed to the increased maximal dynamic strength (Correa et al., 2012, 2014; Radaelli et al., 2013). Withal, the LVRT is effective for increasing strength and muscle hypertrophy in postmenopausal women (i.e., 15 min of strength training can be effective in preventing the loss of strength and muscle mass in this population). Nevertheless, MT, serum triglycerides in the OFTT protocol, and the metabolic responses of EE and EPOC during the session were significantly higher for HVRT. These results suggest that HVRT is fundamental to the increments of morphological, metabolic, and biochemical adaptations, especially for individuals who are at risk of developing cardiovascular disease as in the case of this study of postmenopausal women.

The postprandial glucose response was not different among the three groups, in accordance with the works of Burns et al. (2005), Chapman, Garvin, Ward, and Cartee (2002), and Fenicchia et al. (2004). This may have been due to the fact that as the RES was held the day before the PPL protocol and a high-fat meal (60% fat) was utilised for the OFTT, it was more effective for those performing the HVRT session to use triglycerides, resulting in the use of fat as the primary energy source for muscle recovery the following day (Petitt et al., 2003). The curve of glucose (Figure 2A) remained unchanged in both groups in accordance with previous studies (Burns et al., 2005; Chapman et al., 2002). The lack of changes found in fasting and postprandial glucose in our study suggests that postprandial changes in triglycerides caused by HVRT are unlikely to be mediated by altered glucose. Despite no significant differences being observed in glucose in this study, it cannot be ruled out that longer resistance training interventions (minimum 1 year) may have a significant effect. Szczympczewska et al. (1989) reported that bodybuilders show better glucose tolerance levels compared with untrained lean and obese participants, suggesting that the increase in lean body mass and reduction in body fat mediated through training may account for these effects.

Others parameters such as total cholesterol, HDL, and LDL also did not present significant differences. Previous studies of acute exercise and PPL have not detected any difference in fasting total cholesterol, HDL, and LDL concentrations between exercise and control trials, and most evidence indicates that RES does not influence fasting total cholesterol, HDL, and LDL cholesterol concentration (Burns et al., 2005). The response of LDL is associated with a variation in the serum concentration of triglycerides according to the Friedewald formula, and for this reason, both studies of aerobic and RES rarely show significant differences after an OFTT protocol (Petitt et al., 2003; Petitt & Cureton, 2003).

In conclusion, both LVRT and HVRT are effective for increasing muscle strength. However, HVRT attenuates baseline triglycerides concentration and the total postprandial triglycerides response, increases resting fat oxidation around 16 h later, and increases MT of the knee extensors to a greater magnitude than LVRT. HVRT is a non-pharmacologically effective strategy for the prevention of cardiovascular diseases that are associated with the phenomenon of PPL and may provide other health benefits than those traditionally associated with this type of exercise in postmenopausal women.

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References


