Resistance exercise at variable volume does not reduce postprandial lipemia in postmenopausal women

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Abstract The aim of this study was to investigate the acute effects of resistance exercise sessions (RESs) performed at different levels of high-volume resistance exercise (HVRE) and low-volume resistance exercise (LVRE) on postprandial lipemia (PPL) in postmenopausal women. Thirty-nine healthy unconditioned postmenopausal women (59.5 ± 4.8 years of age, body mass 69.6 ± 9.1 kg, height 157.9 ± 7.2 cm, BMI 27.6 ± 4.1 kg m⁻², waist circumference 76.1 ± 9.7 cm, $\text{VO}_{2\text{max}}$ 18.7 ± 1.4 mL kg⁻¹ min⁻¹) were assigned to a LVRE ($n=12$), HVRE ($n=14$), and control group (CG, $n=13$). Experimental groups performed one RES involving eight exercises. The HVRE group performed three sets with a maximum of 15 repetitions, and the LVRE group performed one set with a maximum of 15 repetitions. Approximately 16 h after a RES, all of the groups were

given an oral fat tolerance test (OFTT). During the RES, we evaluated the energy expenditure (EE) of the resistance session and excess postexercise oxygen consumption (EPOC); following the RES and the OFTT, we evaluated lipid profiles (total cholesterol, HDL, LDL, and triglycerides). While the study groups did not demonstrate significant differences in lipid profiles, the total energy expenditure (EE+EPOC) of the session exercise treatments was significantly higher for HVRE than for LVRE (0.60 ± 0.12 and 0.31 ± 0.11 MJ, respectively, $p < 0.001$). Different levels of resistance exercise do not lower basal triglyceride concentration and postprandial lipid profile parameters at approximately 16 h following resistance exercise in untrained postmenopausal women.

Keywords Energy expenditure · Resistance exercise · Triglycerides · Postprandial lipemia · Menopause

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Introduction

Postprandial lipemia (PPL) has been associated with cardiovascular disease (CVD) (Shannon et al. 2005; Zafeiridis et al. 2007). CVD risk is reduced among premenopausal women, presumably due to the cardioprotective effects of endogenous estradiol, the lack of which confers upon men a risk factor for the development of CVD (Burns et al. 2005). Postmenopausal women lose much of the cardioprotective effect of endogenous estradiol through hormonal changes (i.e., decrease of estrogen), and their incidence of cardiovascular

disease rises above that of men (Kanaya et al. 2003). Globally, CVDs are responsible for 23 % of deaths among women over 60 years of age (Takagi and Umemoto 2011). Loss of muscle mass and reduced activity of the enzyme lipoprotein lipase (LPL) are closely related to the increased PPL observed in postmenopausal women (Petitt et al. 2003). Studies on postprandial lipid metabolism have suggested that increased PPL is directly associated with atherosclerosis progression via the deposition of postprandial lipoproteins on the arterial surface (Mamo et al. 1998) or indirectly through the contribution of PPL to the predominance of LDL and VLDL cholesterol (Aldred et al. 1994). Thus, interventions such as resistance training (RT) that are designed to reduce the risk for coronary heart disease are particularly relevant for postmenopausal women.

The skeletal muscle has a major effect on energy expenditure and is considered to be an important determinant of resting metabolic rate (Mamo et al. 1998) due to the greater oxygen uptake of the skeletal muscle compared to that of other tissues (Ballor and Poehlman 1992; Henry et al. 2003). Accordingly, it has been suggested that RT and the consequent increase in muscle mass can reduce multiple risk factors for cardiovascular disease (Hurley et al. 1988; Poehlman et al. 2000; Smutok et al. 1993). RT is known to induce a marked improvement in insulin sensitivity (Tsetsonis et al. 1997), decrease fasting triglycerides (Yarasheski et al. 2001), and markedly increase the rate of fat oxidation rate at rest and 24 h following a single RT episode (Treuth et al. 1995). This increase in fat oxidation suggests that resistance exercise may be particularly useful in reducing lipemia both at rest and in the postprandial period. Lower levels of lipemia observed following aerobic exercise are due to energy expenditure and occur as a result of muscle mass used and the energetic pathways involved during aerobic exercise that is important for women in menopause (Gill and Hardman 2000). Few studies have investigated the effects of different levels of RT in reducing PPL, and no study has examined these effects in postmenopausal women (Burns et al. 2005; Burns and Stensel 2008; Petitt and Cureton 2003; Zafeiridis et al. 2007). The only study that examined the acute effect of resistance exercise performed at different volume levels on PPL reported no dose–response relationship (Shannon et al. 2005; Zafeiridis et al. 2007). Thus, opportunities exist to examine the effects of different RT regimens on PPL with consideration of other exercise-related effects such as metabolic

and hormonal changes, energy deficits, and physiological disorders.

Variations in the volume of resistance exercise sessions may produce different biochemical and metabolic responses. Higher energy expenditures appear to promote greater biochemical responses, as demonstrated by the increased activity of LPL. Thus, the prescription of resistance exercise is recommended for the improvement of quality of life and prevention of several disorders (Zafeiridis et al. 2007) as cardiovascular disease in postmenopausal women (Kanaya et al. 2003). Furthermore, the effects of different volumes of a single resistance exercise session and the subsequent effects of training at different volumes warrant study. In addition, RT as a component of a health and fitness program is recommended by health organizations such as the American College of Sports Medicine (2009) and the American Diabetes Association (Cefalu 2012) to improve quality of life and weight management as well as to prevent various diseases. Thus, the present study was designed to investigate the acute effects of RT performed as high-volume resistance exercise (HVRE) and low-volume resistance exercise (LVRE) on PPL in postmenopausal women.

Methods

Participants Thirty-nine postmenopausal women (59 ± 4 years of age, body mass 69.6 ± 9.1 kg, height 157.9 ± 7.2 cm, body mass index (BMI) 27.6 ± 4.1 kg m⁻², waist circumference 76.1 ± 9.7 cm, maximal oxygen uptake (VO_{2max}) 18.7 ± 1.4 mL kg⁻¹ min⁻¹), who had not engaged in regular and systematic RT for at least 1 year before the study, participated. The low VO_{2max} was believed to be due to the participants' menopausal status, high BMI, and sedentary lifestyles. The study excluded nonsmokers and individuals with diabetes, dyslipidemia, and/or a history of severe endocrine, metabolic, cardiovascular, and neuromuscular diseases. Participants were recruited by advertisement in a widely read local newspaper. Before the experiment, the participants were carefully informed about the study design, especially regarding the potential risks and discomfort related to the procedures. The participants then gave their written informed consent. The study protocol complied with the Declaration of Helsinki and was approved by the Ethics Committee of the Federal University of Rio Grande do Sul.

Experimental design Thirty-nine volunteers were divided into low-volume resistance exercise (LVRE=12, one set), high-volume resistance exercise (HVRE=14, three sets), and a control group (CG=13). The LVRE and HVRE groups performed eight exercises: bench press, biceps curl, triceps free weights, one-arm dumbbell row, leg press, knee extension, knee flexion, and abdominal crunch. All of the exercises were performed for 15 maximum repetitions (RMs) using the heaviest possible weight for the designated number of repetitions (Shimano et al. 2006). Participants were familiarized with all exercises by the 15-RM test. The exercise cadence was 2 s for the concentric phase and 2 s for the eccentric phase. A 40-s time interval was used between sets and exercises. The duration of each resistance exercise session (RES) was approximately 15 min for LVRE and 45 min for HVRE. All of the participants underwent lipid profile analysis 1 and 2 weeks prior to the first session, and all participants were familiarized with the resistance exercises 1 week before the study onset. At two separate times, participants performed a 1-RM test on a knee extensor exercise machine.

Participant characteristics Before the RES, participants underwent preliminary laboratory testing to collect anthropometric data and to obtain $\text{VO}_{2\text{max}}$ determinations. Body mass and height were recorded for the calculation of BMI [body mass (kg)/height (m^2)]. Waist circumference was measured at the most narrow point between the iliac crest and the last rib. Thigh circumference was measured at the point of the largest muscular volume for the right thigh. The skinfolds were measured at seven sites (triceps, subscapula, pectoral, axilla, abdomen, suprailiac, and thigh) using a Lange caliper (Beta Technology Inc., Cambridge, MD, USA) according to the method recommended by the International Society for the Advancement of Kinanthropometry. All of the skinfold measurements were performed by a single examiner with contingency coefficient (CC) reliability >0.8 . The skinfolds were also used to calculate the body fat percentage (Marfell-Jones 2006).

The $\text{VO}_{2\text{max}}$ was determined by the breath-by-breath method using an open-circuit spirometry system (metabolic cart, CPX/D, MGC, Saint Paul, MN, USA). The progressive cycle ergometer test (The Bike, Cibex, Ronkonkoma, NY, USA) produced an initial workload of 25 W with a prompt increase of 25 W min^{-1} at 2 min until exhaustion with a recovery period of 3 min at 25 W. The pedaling cadence was maintained between

60 and 70 r min^{-1} . The heart rate was monitored using short-range telemetry (S610, Polar Electro Oy, Finland). Participants were verbally encouraged to perform at maximum effort during the test. The test lasted for 6–8 min in accordance with the recommendations of the American College of Sports Medicine (2009) for postmenopausal women and was halted at any time if the participant was unable to maintain pedal rotation, reached a plateau in the oxygen uptake (VO_2) curve, or reached a heart rate (HR) near the maximum predicted (220 minus age). The data obtained from the $\text{VO}_{2\text{max}}$ test performed by the breath-by-breath method were graphically recorded for ventilation and respiratory exchange ratio as a function of VO_2 and time during the test (Cunha et al. 2011; Dekerle et al. 2003; McConnell and Copestake 1999). Ergospirometer data were obtained by visual inspection of graphs by three independent observers.

Muscle strength evaluation (1 RM) The knee extensor 1-RM test of the dominant lower limb was performed by a knee extensor exercise using the same “extension chair” equipment employed for the resistance exercise sessions (Taurus, Brazil, resolution 0.1 kg). To control the movement speed during the test, a Quartz metronome with a 1-Hz resolution was used. The cadence of the knee extension exercise was 2 s for the concentric phase and 2 s for the eccentric phase (for test details, see Correa et al. (2012)). No significant differences were found between 1-RM tests at baseline for all groups evaluated: HVRE averaged 27.2 ± 4.1 SD, 1.14 kg standard error (SE); LVRE averaged 27.0 ± 2.81 SD, 0.78 kg SE; and CG 27.6 ± 4.65 SD, 1.42 kg SE. On retesting, HVRE averaged 27.5 ± 3.1 SD, 1.43 kg SE; LVRE averaged 27.4 ± 2.81 SD, 0.89 kg SE; and CG 27.5 ± 6.05 SD, 1.40 kg SE. Cronbach’s alpha for HVRE, LVRE, and CG was 0.79, 0.75, and 0.94, respectively.

Experimental procedure Preliminary assessments were performed in the morning following a 12-h overnight fast. Starting time was maintained constant among participants. Dietary records were collected for the 3 days preceding the experiment. To accurately detail and standardize the description of food portions, a portfolio with photos was prepared for the participants. No differences in energy and macronutrient intake occurred between all three groups (Table 3). Participants were required to refrain from exercise for 48 h prior to testing and were

asked to refrain from caffeine, alcohol, and strenuous exercise 24 h prior to evaluation visits.

Upon arrival at the laboratory, basal metabolic rate (BMR) data were collected for 30 min via continuous indirect calorimetry (metabolic cart, CPX/D, MGC, Saint Paul, MN, USA) with the subject in a supine position. Data collected during minutes 20–25 of the rest period were averaged and used to represent the resting metabolic activity (Bianco et al. 2011).

On the day before the oral fat tolerance test (day 1), subjects returned to the laboratory between 3:30 p.m. and 5:30 p.m. and performed one set of exercises (for LVRE), three sets (for HVRE), and no exercise (for CG). Dietary records were obtained for the 2 days preceding the pre- and postprandial lipemia protocol. Immediately after the exercise session completion, the subject returned to the darkened room and resumed a supine position for 30 min of recovery for excess post-exercise oxygen consumption (EPOC) measurements (Fig. 1). Data collected during minutes 20–25 of the rest period were averaged and used to represent the resting metabolic activity (Bianco et al. 2011). The values for energy expenditure (EE) in RES (HVRE and LVRE) were obtained by summing the product of the average VO_2 per minute by respiratory exchange ratio (RER) during each part of the protocol (RES for HVRE=45 min, LVRE=15 min, and EPOC=30 min for both) and subtracting the participant's total BMR, i.e., the number of kilocalories that subjects would have consumed if they had been at rest during the RES. The

resting VO_2 rate was approximately 0.3 L min^{-1} for both groups and was multiplied by the rate recovery time to produce the equivalent of rest ($0.3 \text{ L min}^{-1} \times 45 \text{ min} = 13.5 \text{ L}$) using an adopted caloric equivalent (CE) of 4.92 kcal L^{-1} of oxygen consumed for the calculation of EE in EPOC. For the calculation of EE in the RES, an adopted CE of 5.05 kcal L^{-1} of VO_2 was used, a value for which there is consensus in the literature due to the large increase in CO_2 production that occurs during the RES due to hyperventilation and thus generating RER values above 1 (Ratamess et al. 2007). The values were obtained from the sum of the product of the mean VO_2 per minute by the CE in this recovery period, subtracting the total value of the BMR of the participants or the number of kilocalories that the subjects would have consumed during the period of the RES and EPOC had they not performed the RES (Ratamess et al. 2007; Thornton and Potteiger 2002). The HVRE and LVRE showed an average VO_2 of 0.74 and 0.67 L min^{-1} , respectively; RER for EE of 0.88 and 0.89 , respectively; RES of 0.42 and 0.34 L min^{-1} , respectively; and RER of 0.87 and 0.81 , respectively, for EPOC. All VO_2 data were converted to megajoules (MJ), whereby $1 \text{ kcal} = 0.0041868 \text{ MJ}$.

Oral fat tolerance test On day 2, an oral fat tolerance test (OFTT) was administered after a 12-h overnight fast, approximately 16 h postexercise protocol of RES+EPOC. The 5-h OFTT began at 07:30 a.m. and ended at 12:30 p.m. Upon 07:30 a.m. arrival at the

Fig. 1 Experimental design of the OFTT protocol. The participants underwent a session of resistance exercises (RES) with evaluation of energy expenditure (EE) and excess postexercise oxygen consumption (EPOC) between 3:30 p.m. and 5:00 p.m.; a standard meal was consumed at 08:00 p.m.; participants then fasted for 12 h and arrived at the laboratory 7:30 a.m. to 8:00 a.m.

the following morning for the first blood sample; a fatty meal was consumed between 8:00 a.m. and 8:30 a.m.; and postprandial blood samples were collected at 08:00 a.m., 08:30 a.m., 09:30 a.m., 10:30 a.m., 11:30 a.m., and 12:30 p.m. to assess postprandial lipemia (PPL)

laboratory, participants rested for 5 min while a fasting baseline blood sample was collected. Participants were given 5–10 min to consume the meal, and no gastrointestinal discomfort or nausea was reported (Fig. 1). The test meal consisted of a whole milk “milk shake” (milk, ice cream, and sour cream) given the rapid gastric absorption of this mixture (60 % lipids, 30 % carbohydrate, and 10 % protein). The energy provided by the meal was calculated individually to cover the half of EE of BMR (12-h overnight fast). For the calculation, the volume of oxygen consumed by each individual was converted into metabolic equivalents (METs). Based on the assumption that one MET is equivalent to $1.0 \text{ kcal kg}^{-1} \text{ h}^{-1}$ (Ainsworth et al. 2000), the energy expenditure of each participant was calculated using the individual’s body mass (in kilograms). The meals contained a high amount of fat (mean $\text{MJ} \pm \text{SD}$: 2.9 ± 0.4 , 2.7 ± 0.6 , and 2.6 ± 0.8 for HVRE, LVRE, and CGs, respectively), with no significant difference in energy or macronutrient values for test meals between groups. The energy and macronutrient contents 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.

Blood samples were analyzed to determine the levels of glucose (GLU), total cholesterol (TC), HDL, LDL, and triglycerides (TAG). The blood samples were collected at baseline, 1, 2, 3, 4, and 5 h using a disposable sterile cannula inserted at the antecubital region of the arm. The cannula was maintained with nonheparinized saline solution ($9 \text{ mg mL}^{-1} \text{ NaCl}$). A 10-mL volume was obtained for each blood sample (Burns et al. 2005; Burns and Stensel 2008). The ambient temperature (22°C) and the relative air humidity (65 %) were controlled for all tests.

Blood biochemistry Blood samples for TC, HDL, LDL, and TAG analyses were stored in tubes without anticoagulants; blood samples for GLU analysis were stored in tubes with fluoride anticoagulant. These samples were centrifuged at $3,500 \text{ r min}^{-1}$ ($1,400g$) at 4°C for 10 min. The respective supernatants were aliquoted and frozen at -75°C for future analysis. The concentrations of TC, HDL, LDL, TAG, and GLU were analyzed using an automated enzymatic colorimetric method (cobas® c111 analyzer, Roche Diagnostics, NY, USA). Total area under the curve (AUC) was calculated for TAG using the trapezoidal method as previously described

(Shannon et al. 2005) using the MATLAB software (version 7.14).

Statistical analyses

All data were analyzed using the Statistical Package for Social Sciences (SPSS) version 19.0 (SPSS Inc., Chicago, IL, USA). Normality of data was first assessed using the Shapiro–Wilk test. The AUC was calculated for blood parameters versus time curve by trapezoidal rules. One-way analysis of variance test was used to determine differences between groups. When appropriate, post hoc tests were applied using the Bonferroni test. For hypothesis testing, the 95 % level of confidence was predetermined as the minimum criterion to denote a statistical difference ($p < 0.05$). Data are expressed as means $\pm$ SD.

Results

The subjects’ characteristics are listed in Table 1. No significant differences were observed between HVRE, LVRE, and CGs prior to RES for height, body mass, body composition, muscle mass, body mass index, waist and thigh circumference, $\text{VO}_{2\text{max}}$, BMR, and muscle strength. The EEs (EPOC; Table 1) and total EE (EE + EPOC) of session exercise treatments were significantly greater for HVRE ($0.60 \pm 0.12 \text{ MJ}$) than for LVRE ($0.31 \pm 0.11 \text{ MJ}$) ($p < 0.001$).

Table 2 demonstrates no significant lipid profile differences between groups (TAG, GLU, TC, HDL, and LDL) at 2 weeks and 1 week prior to RESs. No significant differences in fasting levels of GLU (Fig. 2a) ($p = 0.077$), TC (Fig. 2b) ($p = 0.089$), HDL (Fig. 2c) ($p = 0.068$), LDL (Fig. 2d) ($p = 0.137$), TAG (Fig. 3a) ($p = 0.095$), or AUC TAG (Fig. 3b) ($p = 0.097$) were observed between groups following RESs.

Discussion

The main finding of this study is that high- and low-volume resistance exercises performed approximately 16 h before administration of an OFTT do not decrease basal TAG, lipid profile, or total serum TAG as measured by AUC after a single RES in postmenopausal women. These results are significant regarding

Table 1 Participant characteristics and metabolic responses

	HVRE, <i>n</i> =12	LVRE, <i>n</i> =14	CG, <i>n</i> =13
Variables			
Body mass (kg)	65.4±10.8	70.1±9.9	67.6±6.01
Body fat (%)	39.3±4.6	37.8±5.1	36.3±5.1
Muscle mass (%)	33.1±4.1	37.1±3.8	36.2±2.8
BMI (kg m ⁻²)	25.4±4.1	26.1±3.6	24.7±2.6
Waist circumference (cm)	79.6±9.2	82.4±7.4	78.8±6.5
Thigh circumference (cm)	50.2±9.2	53.4±7.8	50.8±9.5
Waist–hip ratio	0.79±0.06	0.78±0.05	0.81±0.05
Metabolic responses			
VO _{2max} (mL kg ⁻¹ min ⁻¹)	18.8±1.6	18.4±1.7	17.5±1.4
BMR (MJ)	5.62±1.09	5.49±1.14	5.52±1.13
EE (MJ)	0.25±0.12 [#]	0.10±0.08	
EPOC (MJ)	0.24±1.12 [#]	0.08±0.02	

CG control group, BMI body mass index, VO_{2max} maximal oxygen uptake, BMR basal metabolic rate, EE energy expenditure, EPOC excess postexercise oxygen consumption

[#] Significant difference between high-volume resistance exercise (HVRE) and low-volume resistance exercise (LVRE) (*p*<0.001)

menopause onset as postmenopausal women lose much of the cardioprotective effect of endogenous estradiol, and their incidence of cardiovascular disease rises above that of men (Kanaya et al. 2003). Additionally, any intervention that reduces coronary heart disease risk is particularly relevant for postmenopausal women. In this study, postmenopausal women demonstrated homogeneity with regard to body composition (Table 1), lipid profile (Table 2), and dietary intake (Table 3). To our knowledge, this is the first report to evaluate PPL in postmenopausal women under a protocol of variable-

volume RESs and to demonstrate no significant dose–response reduction in TAG (Fig. 3a) and AUC (Fig. 3b) between groups.

Resistance exercise produces an increased energy demand that is met by increased utilization of muscle and liver glycogen and intramuscular TAG (Petitt et al. 2003; Shannon et al. 2005), thus resulting in glycogen and intramuscular TAG depletion (Burns et al. 2005; Burns and Stensel 2008). Exercise is followed by a period of increased postexercise free fatty acid mobilization (Singhal et al. 2009), LPL activity (Petitt et al.

Table 2 Fasting blood lipid profiles calculated for all three groups

Fasting blood sample analyte	HVRE, <i>n</i> =12			LVRE, <i>n</i> =14			CG, <i>n</i> =13		
	Pre 2 weeks	Pre 1 week	Cronbach's alpha	Pre 2 weeks	Pre 1 week	Cronbach's alpha	Pre 2 weeks	Pre 1 week	Cronbach's alpha
TAG (mmol L ⁻¹)	1.02±0.24	1.06±0.22	0.97	1.03±0.25	0.97±0.23	0.93	0.96±0.16	1.03±0.13	0.79
GLU (mmol L ⁻¹)	4.66±1.53	4.92±1.55	0.95	4.21±1.96	3.93±2.12	0.85	4.71±1.5	4.55±1.23	0.97
TC (mmol L ⁻¹)	5.36±0.97	5.27±0.86	0.98	6.11±0.98	5.32±1.08	0.97	5.23±0.98	5.21±0.97	0.96
HDL (mmol L ⁻¹)	1.65±0.28	1.73±0.38	0.93	1.61±0.62	1.60±0.62	0.96	1.58±0.56	1.63±0.23	0.94
LDL (mmol L ⁻¹)	2.91±0.73	3.11±0.63	0.95	2.49±0.64	3.01±0.75	0.65	3.03±0.56	2.93±0.47	0.84

The lipid profile was evaluated on two occasions (pre 2 weeks and pre 1 week) before commencement of the OFTT protocol. The reliability (pre 2 weeks and pre 1 week before RES) for each of the blood markers was evaluated with Cronbach's alpha

HVRE high-volume resistance exercise, LVRE low-volume resistance exercise, CG control group, TAG triglycerides, GLU glucose, TC total cholesterol, HDL high-density lipoprotein, LDL low-density lipoprotein, RES resistance exercise session

Fig. 2 Serum lipid profile, **a** glucose (GLU), **b** total cholesterol (TC), **c** high-density lipoprotein cholesterol (HDL), and **d** low-density lipoprotein cholesterol (LDL). Abbreviations: HVRE (—■—) high-volume resistance exercise, LVRE (---□---) low-volume resistance exercise, and CG (---●---) control group. The time points

(0=baseline, 1, 2, 3, 4, and 5h) represent blood drawn at 08:00 a.m., 08:30 a.m., 09:30 a.m., 10:30 a.m., 11:30 a.m., and 12:30 p.m., respectively, according to the OFTT protocol. Values are means±SD represented by vertical bars

Fig. 3 a TAG concentrations in the fasting state resistance exercise session, **b** for 5 h after consumption of a high-fat meal in the high-volume resistance exercise (HVRE, —■—), low-volume resistance exercise (LVRE, ---□---), and control group (CG, ---●---). The time points (0=baseline, 1, 2, 3, 4, and 5h) represent blood drawn at 08:00 a.m., 08:30 a.m., 09:30 a.m., 10:30 a.m., 11:30 a.m., and 12:30 p.m., respectively, according to the OFTT protocol. Values are means ±SD represented by vertical bars

Table 3 Energy and macronutrient intake in experimental groups

Characteristic	HVRE	LVRE	CG
Energy intake (kcal)	2,002.28±326.02	1,877.33±761.76	2,032.83±426.02
Carbohydrate (%)	52.13±10.79	54.4±11.23	53.17±14.92
Carbohydrate (g)	252.17±64.25	253.34±119.43	258.44±84.22
Carbohydrate (g kg ⁻¹)	4.03±1.18	3.82±1.89	4.03±1.18
Protein (%)	16.31±5.52	17.3±4.55	16.31±5.52
Protein (g)	81.71±39.36	78.8±28.57	81.71±29.36
Protein (g kg ⁻¹)	1.27±0.63	1.21±0.57	1.33±0.61
Fat (%)	31.91±8.12	28.3±10.14	31.91±8.14
Fat (g)	71.88±34.83	56.96±26.51	71.33±32.3
Fat (g kg ⁻¹)	1.13±0.51	0.91±0.42	1.16±0.55

Data are expressed as mean±SD

HVRE high-volume resistance exercise, LVRE low-volume resistance exercise, CG control group

2003), and GLU uptake (Gill et al. 2002; Petitt et al. 2003) to facilitate repletion of muscle glycogen (Gill and Hardman 2000) and intramuscular TAG (Gill and Hardman 2000; Tsetsonis et al. 1997; Zafeiridis et al. 2007).

Indeed, this fat utilization mechanism depends on a dose–response relationship to the volume of resistance exercise (Petitt et al. 2003; Shannon et al. 2005). However, acute postprandial lipid profile measurements did not differ significantly between the two volume levels (Figs. 2 and 3), which appears to indicate that a single RES, regardless of the volume level, has no effect on PPL (Burns et al. 2005; Shannon et al. 2005). For instance, postprandial GLU response was not different among the three groups as the RES was held the day before the PPL protocol; the next day, the RES, this time (i.e., recovery muscle), primarily uses fat (Petitt et al. 2003). The curve of GLU (Fig. 2a) remained unchanged in both groups in accordance with previous studies (Burns et al. 2005; Chapman et al. 2002; Shannon et al. 2005). Furthermore, during the recovery of the muscle, OFTT protocol utilized a high-fat meal (60 % fat); due to the HVRE, it was more effective to use TAG. Also, within 4 h after the RES, carbohydrate absorption by the cells was facilitated by a GLU transporter (GLUT4) (Poehlman et al. 2000), which decreases the concentration of GLU immediately after exercise (Chapman et al. 2002; Gill and Hardman 2000). Thereupon, the increasing use of this GLU related to a large increase in lean mass. Szczypaczewska et al. (1989) reported that bodybuilders exhibited better GLU tolerance when compared to untrained lean and

obese subjects, suggesting that an increase in lean body mass mediated through training, and a reduction in body fat may account for these effects. In addition to GLU, other parameters including TC, HDL, and LDL also did not appear to present significant differences between groups. Previous studies of acute resistance exercise and PPL have not detected any difference in fasting TC (Burns et al. 2005; Zafeiridis et al. 2007), HDL (Shannon et al. 2005), and LDL (Zaman et al. 2012) concentrations between low- and high-volume resistance exercise groups and CG. Moreover, the bulk of evidence derived from the present research and other studies (Burns et al. 2005; Shannon et al. 2005) indicates that resistance exercise does not influence fasting TC, HDL, and LDL concentrations.

The results of this study corroborate with those of Burns et al. (2005) and Shannon et al. (2005) in that, regardless of the volume of an EE session, caloric expenditure related to a single acute bout of resistance exercise does not appear to exert any influence on the reduction of postprandial TAG. The estimated EE during resistance exercises in our study was 0.25±0.12 MJ in the HVRE group and 0.10±0.08 MJ in the LVRE group ($p<0.001$) (Table 1). This EE is considered very low when compared to the aforementioned studies, which may have an implication on the significance of the current study. Notwithstanding, some studies have reported more robust data from acute responses measured in young individuals as reported by Singhal et al. (2009) (EE=1.81 MJ, 3 sets/16 repetitions, rest=2 min, $p=0.042$) and Zafeiridis et al. (2007) (EE=1.40 MJ, 2 sets/12 repetitions, rest=2 min, $p=0.017$). Petitt et al.

(2003) ($EE=1.70$ MJ, 3 sets/10 repetitions, rest=2 min, $p=0.017$) compared an acute session of RT with an aerobic session in young individuals. While those authors did not evaluate different intensities of RT, their results were important because they indicated that HVRE exerted a strong effect on EE and, in turn, on fat metabolism. Interestingly, other authors have shown higher EE in RES as reported by Burns et al. (2005) ($EE=2.30$ MJ, 4 sets/10 repetitions, rest=2 min) and Shannon et al. (2005) ($EE=2.58$ MJ, 5 sets/10 repetitions, rest=1 min) in accordance with the present work ($EE=0.25$ MJ, 3 sets/15 repetitions, rest=40 s) which found no significant differences after an acute RES. Although we employed a smaller number of resistance exercises (eight exercises) in comparison to the aforementioned studies (ten on average), five of the eight exercises used were performed with dumbbells and barbells. The time intervals between sets and exercises were brief, which made the exercise sessions intensive. This phenomenon is evidenced by the high EPOC after RES in HVRE (0.25 MJ), which was significantly greater than LVRE (0.08 MJ) ($p<0.001$) (Table 1), but was not sufficient to reduce PPL. Binzen et al. (2001) estimated that following an acute bout of resistance exercise in women, 45 min of high volume (70–80 % 1 RM, 60-s rest interval), EPOC accounted for 0.13 MJ of energy expended above resting values during a 2-h recovery period. However, the previous study did not investigate the PPL, and according to Binzen et al. (2001), the RES can significantly elevate recovery EE for at least 1 h postexercise and fuel utilization favors fat oxidation. It therefore seems improbable that the energy expended because of EPOC contributed significantly to the attenuation of PPL after RES.

The study of Petitt et al. (2003) demonstrated a reduction in TAG; however, that study involved males ($n=10$) and females ($n=4$) who had 6 years of weight lifting experience. Zafeiridis et al. (2007) showed an AUC TAG curve that is lower in HVRE and LVRE compared to control, concluding that resistance exercise of 1.40 MJ (four sets, eight exercises, and 12 RMs) or 0.76 MJ (two sets, eight exercises, and 12 RMs) before a high-fat meal reduces the total PPL response. In contrast, none of the participants in the present study were regularly involved in RT. The performance of RT in a regular and systematic fashion, with a buildup of acute resistance exercise episodes, may be effective in reducing TAG. Recently, Davitt et al. (2013) indicated that the reduction in PPL after aerobic exercise session and RES

bouts is not achieved by enhanced clearance of dietary fat but, rather, is achieved by reduced abundance of endogenous fatty acid in plasma TAG. Interestingly, the previous study showed that RES and aerobic exercise session may be equally effective in reducing postprandial plasma TAG concentration and enhancing lipid oxidation when the exercise sessions are matched for duration rather than for EE. Furthermore, previous studies have conflicting results and different RES protocols. Given our results, we do not have strong evidence to support these theories in the reduction of PPL.

A progressive reduction in muscle mass (sarcopenia) due to the aging process (Chapman et al. 2002; Zaman et al. 2012) is usually associated with functional impairment and functional disability and is a direct cause of reduced muscle strength (Correa et al. 2012; Tzankoff and Norris 1977). Furthermore, physical inactivity and loss of muscle mass with aging lead to loss of muscle strength and endurance (Kanaya et al. 2003). This phenomenon is emphasized after menopause and is evidenced by the low values obtained for VO_{2max} with an average of $18.7 \text{ mL kg}^{-1} \text{ min}^{-1}$ among the participants, given that VO_2 consumption represents a measure of functional capacity in the elderly. Moreover, the menopausal process has been associated with a decrease in resting and total EE. Participants in the present study had a lower BMR (approximately 5.67 MJ) when compared with physically active individuals and athletes (Tzankoff and Norris 1977), which may have influenced the poor response to RES in postmenopausal women after an OFTT challenge (Fig. 1).

The American College of Sports Medicine (2009) recommends that RT should be an integral part of an adult fitness program and that RT should be effective in the development and maintenance of muscular strength and endurance, fat-free mass, and bone mineral density. The aforementioned studies (Gill and Hardman 2000; Burns et al. 2005; Petitt et al. 2003; Zafeiridis et al. 2007), showing significant or nonsignificant results, suggest that RT can be an effective strategy for the reduction of plasma lipids, TAG, and cholesterol. Nevertheless, the results of this study provide strong evidence that an acute RES does not effectively reduce PPL in postmenopausal women as has been observed in both young untrained individuals (Burns et al. 2005; Zafeiridis et al. 2007) and those under a specific type of RES used in the current study.

The limitation of the present study was that the EE for HVRE versus LVRE was $\sim 59 \text{ kcal}$ (0.25 MJ) and

~24 kcal (0.10 MJ), respectively, and the total EE (EE+ EPOC) of exercise for HVRE was ~143 kcal (0.60 MJ) while that for LVRE was ~74 kcal (0.31 MJ), substantially lower than those of all other studies on resistance exercise and PPL. In addition, we use different subjects in each group as opposed to performing a crossover-designed trial. Another limitation was the use of 15 RM, which elicits improvements in muscular endurance, combined with the 2-s concentric and eccentric contraction times which can have serious implications towards muscle fiber recruitment and energy utilization pathways (e.g., muscle glycogen). This could be an explanation why our data are not significant. Currently, to our knowledge, the majority of studies in the literature indicate that resistance exercise does reduce PPL. Further research in this population should consider RES, lower repetition (e.g., 5–10 RMs), that have elicited significant attenuation in PPL.

Conclusions

In summary, these results suggest that the volume of resistance exercises that were used in this study does not attenuate basal TAG concentration and lipid profile as measured by a postprandial response approximately 16 h after resistance exercise in postmenopausal women.

Conflict of interest The authors declare that they have no conflict of interest.

Ethics statement All of the participants gave their written informed consent to participate in the study, which was approved by the Human Ethics Committee of the Federal University of Rio Grande do Sul and conformed to current laws for such research conducted in Brazil.

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