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Resistance exercise attenuates postprandial metabolic responses to a high-fat meal similarly in younger and older men

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ABSTRACT

This study examined whether an acute bout of resistance exercise (RE) attenuated postprandial responses to a high fat meal (HFM) similarly in younger versus older adult men, and probed relationships among skeletal muscle mass (SMM), age, the metabolic load index (MLI) response, and the improvement in the MLI elicited by RE versus CON. Eleven younger (24 ± 4 y) and 9 older (61 ± 5 y) men completed RE or control (CON) the night prior to a HFM. Before and 1, 3, and 5 hours after the HFM, blood triglycerides (TG), glucose (GLU), MLI, and cholesterol concentrations were quantified. Following a 7 ± 1 -day washout period, participants returned and completed the opposite condition. Independent of age, TGs were 32.1 ± 27.1 mg/dL and 52.7 ± 26.8 mg/dL lower in RE than CON at 3 and 5 hours, respectively. MLI was also 24.3 to 56.9 mg/dL lower in RE than CON from 1 to 5 hours post-meal independent of age. The TG and MLI area under the curves (AUCs) were 15% to 31% lower in RE than CON. The GLU response was greater in the older than younger men at 1 to 5 hours post-meal. Moreover, the average GLU response was 5.6 ± 2.5 mg/dL lower in RE versus CON and was inversely related to SMM across the sample ($r = -0.615$). However, age, volume, or SMM were not related to the MLI_{AUC} , nor to the improvement elicited by RE. Therefore, although the older men displayed a greater postprandial glucose response than the younger men, RE attenuated the postprandial metabolic response to a HFM similarly in younger and older men.

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1. Introduction

Cardiovascular disease (CVD) is the leading cause of death in the United States, accounting for approximately 1 of every 3 deaths in 2018 [1]. Moreover, approximately 50% of Ameri-

cans, or more than 120 million, are living with CVD or dealing with the after-effects of a stroke [1]. While these statistics are staggering, the impact of CVD is expected to grow worldwide, with projections indicating that it will likely claim over 40 million lives in the year 2030 [2]. However, as noted by the

Abbreviations: ANOVA, analysis of variance; AUC, area under the curve; CON, control; CVD, cardiovascular disease; GLU, glucose; HDL-C, high-density lipoprotein cholesterol; HFM, high fat meal; LDL-C, low-density lipoprotein cholesterol; MLI, metabolic load index; PPL, postprandial lipemia; RE, resistance exercise; SMM, skeletal muscle mass; TG, triglycerides; Total-C, total cholesterol; VOL, resistance exercise volume.

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World Health Organization and the Centers for Disease Control Prevention, “any major reduction in deaths and disability from CVD will come from prevention, not cure” [2].

The underlying pathology of CVD is atherosclerosis, and the key event initiating atherogenesis is the retention of low-density lipoprotein (LDL) cholesterol and cholesterol-rich lipoproteins in arterial walls [3]. A primary factor promoting CVD is thought to be consistent exposure to elevated blood lipids. Indeed, most individuals in the developed world spend the majority of their day in a postprandial state, and postprandial lipemia (PPL) is directly associated with circulating concentrations of remnant chylomicrons, which can infiltrate arterial walls [4]. Postprandial lipemia has also been reported to promote CVD via endothelial dysfunction, an increase in blood coagulability, an increase in systemic inflammation and reactive oxygen species production [5,6]. Consequently, PPL has been reported to be a significant predictor of CVD risk and mortality, even after adjusting for potential confounders [4,7–9]. Therefore, it is important to identify effective approaches for lowering PPL that, when performed habitually, may prevent CVD development.

Aging has been identified as a universal contributor to metabolic decline [10], is associated with an exacerbated PPL response [11], and is a well-established, traditional risk factor for cardiometabolic disease [12]. Data from the Framingham Heart study suggest that, while age is an independent contributor to CVD development, it may also be partly modifiable, in that age also tends to reflect the length of exposure to other CVD risk factors [13]. Thus, the influence of age on CVD risk is not uniform among individuals and is dependent on the burden of accompanying CVD risk factors [13]. Aging is also associated with a loss of skeletal muscle mass (SMM) and an alteration of the intrinsic qualities of skeletal muscle (eg, reduced capillary density, mitochondrial dysfunction and decreased oxidative capacity, etc), traits which have been increasingly recognized as important contributors to metabolic health [14–16]. Indeed, aging is associated with metabolic inflexibility [17], or a diminished ability to alter substrate metabolism in response to changes in substrate availability, which may be mediated, in part, at the level of skeletal muscle [18]. Further, it has been reported that aging is accompanied with decreases in whole body fat oxidation, which are primarily associated with decreases in fat-free mass [19]. Therefore, the identification of methods to reduce PPL is extremely important across the age-span, and methods that may simultaneously improve SMM and quality may be particularly attractive.

While aerobic exercise is often the mode of choice when prescribing exercise to promote cardiovascular health and has been shown effective for lowering PPL [20–22], resistance exercise (RE) also appears to be efficacious for this purpose. For example, Petitt et al [23] reported that RE lowered the PPL response to a high-fat meal (HFM), although an isocaloric bout of aerobic exercise did not. Similarly, Zafeiridis et al [24] reported that low- and high-volume RE similarly lowered the PPL response, while Signhal et al [5] reported that equal volume moderate- and high-intensity RE elicited similar reductions in PPL following a HFM. However, it is important to note that a few studies have also reported that RE has no effect on PPL [25,26]. It is also notable that aged adults display

blunted metabolic responses to exercise training, so it cannot be assumed that the beneficial response previously observed in younger adults will also apply to older adults. For example, in the only study to our knowledge to examine the influence of RE on PPL following a HFM in older adults, Correa et al [27] reported that neither a low- or high-volume RE session improved PPL in post-menopausal women. Thus, additional work is needed to understand the efficacy of RE for lowering PPL, especially in older men.

As noted in a recent meta-analysis [28], the cardiometabolic health benefits of RE are understudied, and no previous studies have examined the efficacy of RE for reducing PPL in older adult men. Resistance exercise also has important health benefits for aging adults that include improvements in skeletal muscle mass and function [29] and, perhaps, weight management [30]. Thus, while aerobic exercise has long been the mode of choice, if RE is similarly or more effective, RE may represent another exercise mode on the exercise prescription menu effective for not only improving skeletal muscle function, but also for improving cardiometabolic health-related outcomes. Defining these effects is important because it enhances the variety of modes available for this purpose, allows for increasingly tailored exercise prescription, and may ultimately improve exercise adherence over the long-term, which is arguably the most important factor with regard to the realized health-benefits of any exercise program.

The primary purpose of this study was to determine whether an acute bout of RE attenuates postprandial metabolic responses to a HFM similarly in younger versus older adult men, which we assessed using a 2-day model in which younger and older participants consumed a standardized snack and either rested passively or completed resistance exercise the night of day one, and consumed a HFM on day 2 to examine the post-prandial metabolic response. A secondary purpose was to probe relationships among SMM, age, RE volume, the metabolic response to the HFM, and the improvement in the metabolic response elicited by RE versus CON. We hypothesized that older men would exhibit a more pronounced postprandial lipemic and glycemic response to the HFM than younger men, and that, while an acute bout of RE performed the night prior to the HFM would decrease the PPL response in both younger and older men, RE would be less effective for attenuating the response in the older men.

2. Methods and materials

2.1. Participants

Eleven healthy younger (mean $\pm$ SD, age = 24.3 ± 4 y) and 9 healthy older men (60.8 ± 5 y) participated in this study. Prior to enrollment, subjects signed an informed consent form and completed a health and exercise history questionnaire. Participants must have (1) been between the ages of 18 and 35 years or 55 years and older, (2) had a BMI >18.4 kg/m² but <30.0 kg/m², (3) could not have been a smoker in the past year, (4) could not have been diagnosed with cardiovascular or metabolic disease, (5) could not have had any current or ongoing neuromuscular disorders or disease, (6) could not

currently have been taking medications known to affect energy metabolism such as lipid lowering (ie, statins) or insulin sensitivity (ie, metformin) medications, and (7) could not have been clinically diagnosed with stage 3 hypertension. Participants were recruited by word of mouth, via flyers placed on campus, and via approved emails to the target population affiliated with the university. This investigation was approved by and carried out in accordance with the university's Institutional Review Board for the protection of human subjects (IRB Approval #: ED-17-135, Approved May 1, 2018).

2.2. Experimental design

A randomized, cross-over design was used in the present study, where participants were required to visit the laboratory on 5 occasions. A power analysis was conducted to detect a moderate within-between interaction effect ($f = 0.3$) on the area under the curve of the PPL response with 90% power at an alpha level of 0.05, and with a correlation between repeated measures of 0.8, which were based on the studies by Emerson et al [11] and by Pettit et al [23]. Our analysis indicated that a total sample size of 14 should be sufficient to detect an interaction, and we over recruited by approximately 30% to be conservative.

During an initial visit, participants were screened and consented, familiarized, and then performed 6 repetition maximum (6RM) testing for each of the 8 exercises that were used during the RE condition. Following the resistance exercise familiarization, participants returned for visits 2 to 5 and completed a RE and control (CON) condition separated by 7 ± 1 days in randomized order (Fig. 1). For each condition,

a 2-day model was used in which RE or passive rest (eg, CON) was performed on the first day, similar to models used previously [27,31]. Immediately prior to the RE and CON condition, all participants consumed a standardized snack as the last meal of the day. The following morning after a 12-hour overnight fast, a HFM was administered. Participants were instructed to refrain from any vigorous physical activity, exercise, and alcohol consumption for 48 hours prior and from caffeine for 24 hours prior to the start of each condition.

2.3. Body composition

Upon arrival to the laboratory and following the overnight fast, participants' body composition was measured via multi-frequency bioelectric impedance analysis (seca 514, Hanover, MD, USA) in accordance with the manufacturer's recommendations and as described previously [32]. Body composition was assessed while participants were fasted, euhydrated, and after having avoided strenuous exercise for the previous 48 hours.

2.4. Six repetition maximum testing and 1 repetition maximum prediction

Before testing, participants performed 5 minutes of light aerobic exercise on a cycle ergometer (Monark, Ergomedic 828 E, Vansbro, Sweden) as a general warm-up. Following a specific warm-up using a light weight for the exercise being tested, each participant performed 6RM testing for the 8 exercises used in the RE protocol. For each exercise in the protocol, the 6RMs were used to predict a 1-RM using a previously validated equation [33].

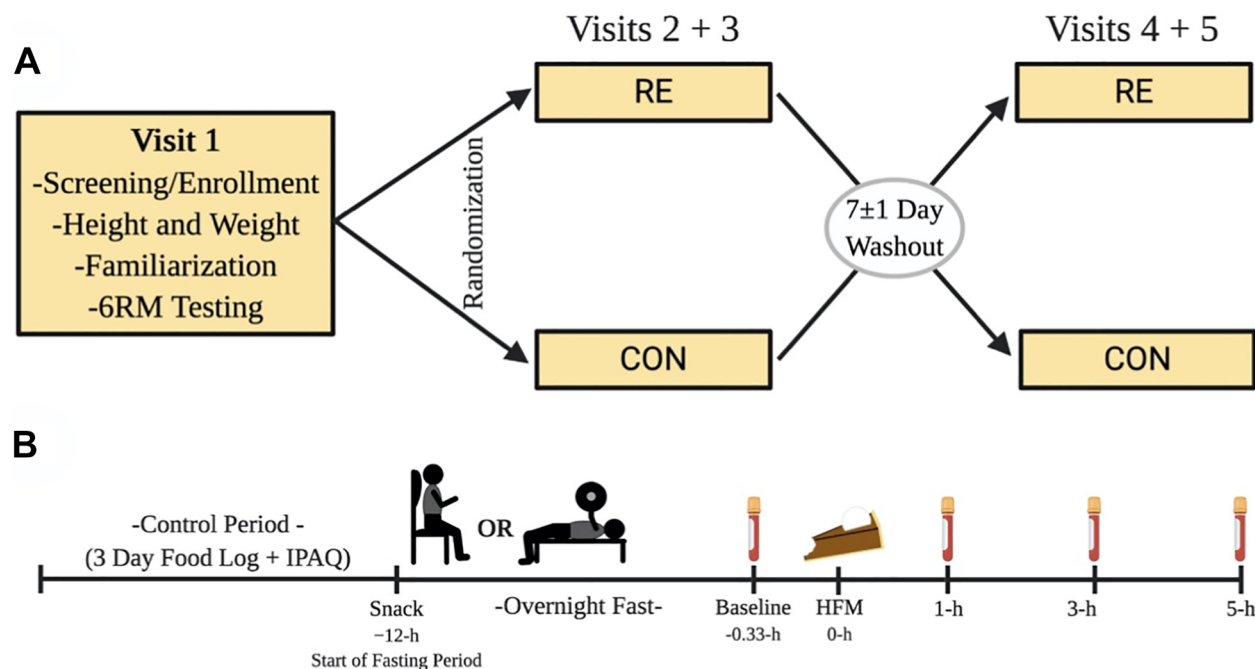


Fig. 1 – Experimental design. Schematics illustrating the (A) general study design, and (B) the experimental procedures during the RE and CON conditions (ie, visits 2–5). The blood tubes indicate blood draws used to quantify metabolic markers.

2.5. Resistance exercise protocol

The prescribed load during the RE condition was 70% 1RM of predicted 1RM and the participants completed 12 repetitions for all exercises. However, if the participant was unable to complete 12 repetitions, the weight was lowered by 2.5% to 5% to allow the participant to complete 12 repetitions for subsequent sets. The exercises were performed in circuit fashion with 1 minute of rest provided between exercises, and the participants completed 3 complete circuits with 2 minutes of rest provided after the completion of each circuit. The order of the exercises was as follows: bench press, body weight squats, seated rows, leg extensions, lateral pull-downs, leg curls, bicep curls, and lateral raises. Lifting volume for each exercise was calculated as:

$$\text{Volume} = (\text{load} \times \text{reps})_{\text{SET1}} + (\text{load} \times \text{reps})_{\text{SET2}} + (\text{load} \times \text{reps})_{\text{SET3}} \quad (2)$$

The volume completed for each exercise was then summated to calculate a total lifting volume ($\text{VOL}_{\text{Total}}$), which was expressed in arbitrary units (a.u.). All resistance exercise was performed the evening before the HFM tolerance test (ie, ~6:00–8:00 pm).

2.6. Resistance exercise energy expenditure estimate

A small pilot study was conducted in 3 participants to obtain an estimate of energy expenditure during the RE protocol. To do this, an accelerometer was worn on the right side of the waist (GT3XP-BTLE, Actigraph Pensacola, FL, USA) to obtain accelerometer counts in the x (horizontal) and y (vertical) axes from the beginning of the warm-up through the last set of resistance exercise and energy expenditure was estimated using the equation provided by Rawson and Walsh [34]. The average net energy expenditure was calculated to be 184.9 ± 24.3 kilocalories (kcal) for the RE protocol (4.42 ± 0.44 kcal/min). The RE session took between 40 and 43 minutes to complete.

2.7. Snack

A snack was provided prior to the start of the RE and CON conditions and consumption of the snack marked the start of the 12-hour fasting period. The snack used was a protein shake made with vanilla almond milk (Silk; WhiteWave Foods, Denver, CO, USA) and whey protein powder (Dymatize ISO-100 Whey Protein; Dymatize Nutrition, Dallas, TX, USA). The amount of protein provided was made relative to body weight (0.3 g/kg body mass). On average, the snack provided 132.1 ± 5.4 kcal, 26.3 ± 1.3 g protein, 4.9 ± 0.4 g carbohydrate, and 0.8 ± 0.1 g fat.

2.8. High-fat meal tolerance test

The HFM used in the present study was chocolate pie (Marie Callender's Chocolate Satin Pie; Conagra Brands, Omaha, NE, USA). The amount of pie consumed was similar to a typical serving at a restaurant or social gathering (1–2 servings), and was relative to each participant's body mass (12 kcal/kg body mass; 0.84 g/kg fat, 1.02 g/kg carbohydrate, 0.09 g/kg protein) [11]. All participants were asked to consume the pie within 20 minutes

following the baseline blood draw and were allowed to drink water ad libitum. On average, the HFM provided 1030.8 ± 53.6 kcal, 72.2 g (63%) fat, 87.6 g (34%) carbohydrate, and 7.7 (3%) g protein. There were no differences ($P = .95$) in the total energy or macronutrients consumed during the HFM for the younger versus older men.

2.9. Blood collection and analysis

On day 2 of each condition, the participants arrived to the laboratory between 6:00 and 8:00 am following a 12-hour overnight fast. An indwelling safelet catheter was inserted into a forearm vein using a 24-gauge needle (Exelint International, Redondo Beach, CA, USA) and was kept clear with a constant infusion of 0.9% NaCl solution (~1 drip/second). Blood draws were performed before, and 1, 3, and 5 hours post-HFM. The blood samples were transferred to 6 mL Vacutainer test tube (BD, Franklin Lakes, NJ, USA) coated with EDTA, from which triglyceride (TG), glucose (GLU), total cholesterol (Total-C), and high-density lipoprotein cholesterol (HDL-C) were measured using a Cholestech LDX analyzer (Alere Cholestech, San Diego, CA, USA). If measured TG values were below the lowest detectable limit (<45 mg/dL), 45 was used as the detected TG value. Low-density lipoprotein cholesterol (LDL-C) values were calculated by the investigators using the Friedewald equation. The coefficients of variation for TG, GLU, Total-C, and HDL-C were 2%–4%, 3%–5%, 2%–3%, and 3%–6%, respectively. The metabolic load index (MLI) was also calculated as the sum of circulating GLU and TG [35] at each time point (eg, baseline, 1, 3, and 5 hours) as previously described [35].

2.10. Area under the curve calculations

Within each condition, total area under the curves (tAUCs) and incremental area under the curve (iAUC) values were calculated for TG, GLU, and MLI for each participant. For one older participant, a blood draw was not completed at 3 hours due to a complication with the catheter set-up. Therefore, to calculate AUCs in this participant only, the concentration of each metabolic marker at the 3-hour time point was estimated as the average of the respective 1- and 5-hour concentrations. The tAUC and iAUC were calculated using the trapezoidal rule [36]. The iAUC values were used to elucidate differences in the postprandial metabolic change above fasting concentrations between conditions.

2.11. Dietary and physical activity assessments

Participants were instructed to consume the same foods for the 2 days prior to and on the first day of each experimental condition. To help participants replicate their diet between conditions, dietary records were provided to record the quantity and type of the foods consumed. The information collected in the dietary records for each participant was then entered by a trained investigator into a freely-available online dietary analysis software (<http://www.myfitnesspal.com>, MyFitnessPal LLC, San Francisco, CA) [5,37,38], which was used to calculate the average energy, fat, carbohydrate, and protein consumed by each participant in the 3-day periods

prior to each condition. Participants were also asked to maintain habitual physical activity levels throughout the study, while avoiding strenuous exercise or physical activity for 48 hours prior to the start of each condition. In order to assess physical activity levels during the days leading up to visits 2 and 3, participants filled out the International Physical Activity Questionnaire Long Last 7-days Format (IPAQ) [39].

2.12. Statistical analyses

Independent-samples *t* tests were used to analyze age-related differences in age, height, weight, body mass index, fat mass, SMM, and self-reported aerobic and resistance exercise history. In addition, independent samples *t*-tests were used to examine age-related differences in the resistance exercise load, repetitions, and volumes completed during the RE condition, as well as the absolute macronutrient content of the HFM and snack provided during the experimental conditions. Two-way (age [younger vs. older] × condition [CON vs. RE]) mixed-factor ANOVAs were used to analyze differences in dietary patterns and physical activity performed in the periods leading up to each condition.

As previously mentioned, for one older adult, a blood draw was not completed at 3 hours due to a complication with the catheter set-up. Therefore, 3-way (age [younger vs. older] × condition [CON vs. RE] × time [0 vs 1 vs 3 vs 5 hours]) mixed-effects models were fit to the data using the maximum likelihood method and were used to analyze each of the metabolic markers. Two-way (age [younger vs older] × condition [CON vs RE]) mixed-factor ANOVAs were used to analyze the tAUCs and iAUCs of TG, GLU, and MLI.

To probe significant interactions and main- or fixed-effects, follow-up Tukey- and Sidak-corrected multiple comparisons tests were used. Finally, Pearson's product moment correlation coefficients were used to examine the relationships between age, SMM, VOL_{Total}, the MLI tAUC during the CON and RE conditions versus the intra-individual change in MLI tAUC (Δ MLI_{tAUC}) from the CON to RE condition, as well as the relationship between SMM and the average GLU response. When appropriate, mean differences are reported as the mean difference ± 95% CI. A type 1 error rate of 0.05 was used for all analyses, which were conducted using GraphPad Prism (Version 7.05; GraphPad Software, Inc, La Jolla, CA, USA) and SPSS statistical software (IBM, v. 23, Armonk, NY, USA).

3. Results

3.1. Baseline characteristics and lifestyle controls

The baseline characteristics of the younger and older men are displayed in Table 1. The younger men were 36.5 ± 4.1 y younger ($P < .001$), completed 4.1 ± 2.3 hours more resistance exercise per week ($P = .002$), and had 5.0 ± 3.0 kg more skeletal muscle mass ($P = .003$) than the older men. There were no age-related differences in height, weight, BMI, or fat mass (all $P > .11$).

The dietary intake and physical activity data for the younger versus older men are displayed in Table 2. There were no significant group × condition interactions for average

Table 1 – The mean ± 95% CI baseline characteristics of the younger and older participants

	Younger	Older
Age (y)	24.3 ± 2.4	60.8 ± 3.9*
Aerobic exercise history (y)	4.4 ± 3.0	7.7 ± 9.3
Aerobic exercise frequency (h/wk)	2.9 ± 2.0	1.5 ± 1.9
Resistance exercise history (y)	7.8 ± 2.9	6.8 ± 8.5
Resistance exercise frequency (h/wk)	5.0 ± 1.9	1.0 ± 1.5*
Height (cm)	178.4 ± 4.4	176.2 ± 3.3
Weight (kg)	87.0 ± 7.5	84.7 ± 5.3
Body mass index (kg/m ²)	27.3 ± 2.0	27.3 ± 1.4
Fat mass (kg)	19.9 ± 4.6	24.4 ± 3.7
Skeletal muscle mass (kg)	33.9 ± 2.6	28.9 ± 1.4*

* indicates an age-related difference; $P \leq .05$.

carbohydrate ($F_{(1,17)} = 1.09$; $P = .31$), fat ($F_{(1,17)} = 0.06$; $P = .94$), protein ($F_{(1,17)} = 0.58$; $P = .46$), or energy ($F_{(1,17)} = 0.72$; $P = .41$) intake, nor in overall physical activity ($F_{(1,17)} = 0.01$; $P = .94$). However, the participants were characterized as physically active based on their daily physical activity levels, which were ≥ 577 MET·min·day⁻¹ in both the younger and older men.

3.2. Resistance exercise characteristics

The younger men utilized greater loads than the older men for 7 of the 8 exercises, and therefore completed 3849 ± 2617 a.u. greater VOL_{Total} during the RE condition ($P = .006$).

3.3. Metabolic markers

The age × condition × time interaction for TG was not significant ($F_{(3,53)} = 0.21$; $P = .89$, Fig. 2A). However, there was a significant condition × time interaction ($F_{(3,53)} = 7.41$;

Table 2 – The mean ± 95% CI self-reported average daily energy (kcal), carbohydrate (g), fat (g), and protein (g) intakes over the 3 day period and average daily physical activity over the 7 day period prior to each condition in the younger and older men; there were no age × condition interactions (all $P \geq .31$)

	Resistance exercise	Control
Younger		
Energy (kcal/d)	2073.7 ± 199.2	2093.8 ± 160.9
Carbohydrates (g/d)	208.8 ± 42.1	210.2 ± 44.4
Fat (g/d)	86.3 ± 22.0	92.2 ± 17.4
Protein (g/d)	110.1 ± 20.3	106.7 ± 21.9
Physical activity (MET·min·day ⁻¹)	685.1 ± 259.5	656.2 ± 248.9
Older		
Energy (kcal/d)	1953.5 ± 691.3	1802.3 ± 475.7
Carbohydrates (g/d)	251.8 ± 101.5	217.5 ± 72.7
Fat (g/d)	66.2 ± 22.7	73.0 ± 21.2
Protein (g/d)	87.5 ± 25.7	68.8 ± 17.9
Physical activity (MET·min·day ⁻¹)	619.4 ± 197.7	577.4 ± 179.7

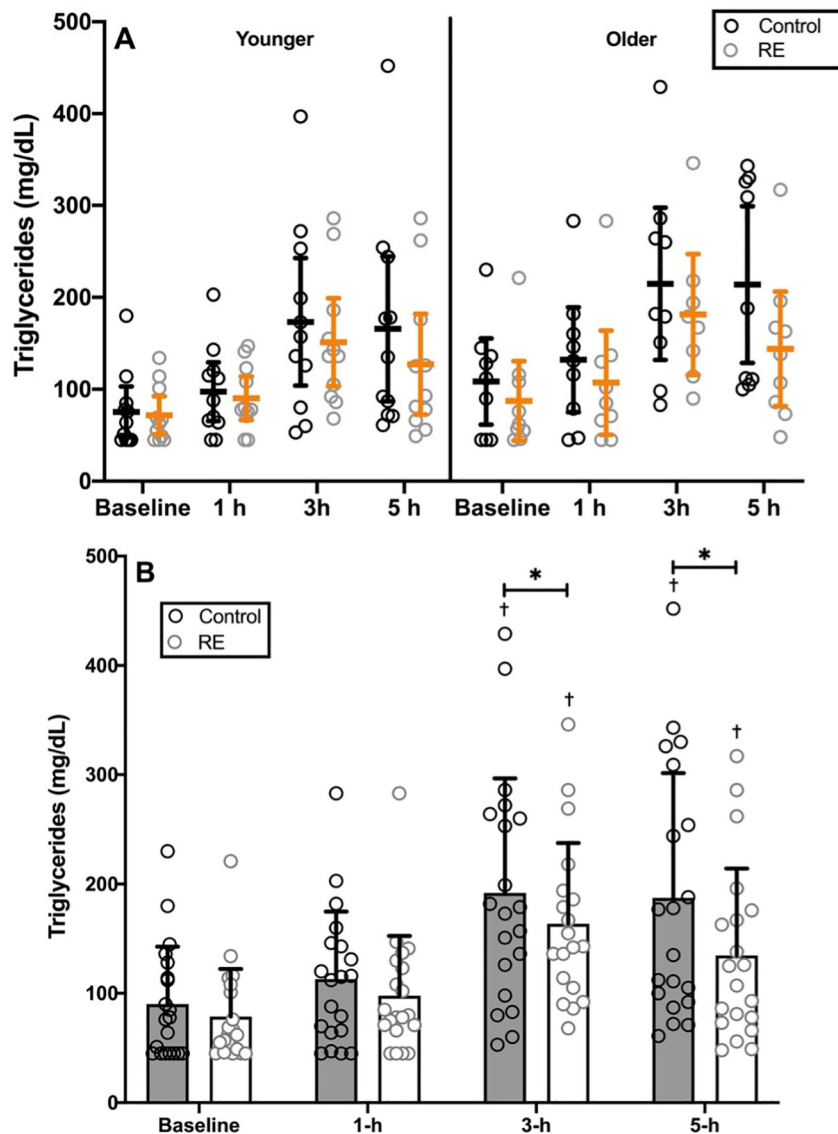


Fig. 2 – Postprandial triglyceride responses. The (A) triglyceride responses during the control (black open circles, black mean $\pm$ 95% CI error bars) and resistance exercise (gray open circles, orange mean $\pm$ 95% CI error bars) conditions in the younger and older men. Panel (B) shows the condition $\times$ time interaction, where * denotes a significant difference between conditions (CON > RE, $P < .05$) and † denotes a significant difference from baseline and 1 hour ($P < .05$).

$P < .001$). The TG concentrations increased from baseline and 1 to 3 and 5 hours in both the RE and CON conditions independent of age (Fig. 2B). However, TG concentrations were 32.1 ($\pm$ 27.1) mg/dL lower at 3 hours ($P = .014$) and 52.7 ($\pm$ 26.8) mg/dL lower at 5 hours ($P < .001$) during the RE versus CON condition (Fig. 2B).

There was no significant age $\times$ condition $\times$ time interaction for GLU ($F_{(3,53)} = 0.53$; $P = .67$, Fig. 3A). However, there was a significant age $\times$ time interaction ($F_{(3,53)} = 4.21$ $P = .01$, Fig. 3B) and a fixed effect for condition ($F_{(1,18)} = 16.95$; $P < .001$, Fig. 3C). In the younger men, GLU decreased from baseline to 1 ($P < .001$) and 3 hours ($P < .001$), but increased modestly at 5 hours such that GLU was no longer different from baseline ($P = .19$). In the older men, GLU did not change across time (all $P > .99$). Consequently, although GLU was not different

in the younger versus older men at baseline ($P = .69$), it was lower (all $P < .02$) in the younger than older men at 1, 3, and 5 hours. Furthermore, GLU concentrations were 5.6 ($\pm$ 2.5) mg/dL lower ($P = .0001$) in the RE than CON condition independent of time or age.

Similar to TGs, there was no age $\times$ condition $\times$ time interaction for MLI ($F_{(3,54)} = 0.07$; $P = .98$, Fig. 4A), but there was a condition $\times$ time interaction ($F_{(3,54)} = 5.68$; $P = .002$). The MLI increased from baseline and 1 to 3 and 5 hours (all $P < .001$) in both the RE and CON conditions (Fig. 4B). However, MLIs were 24.3 mg/dL lower at 1 hour ($P = .006$), 38.3 mg/dL lower at 3 hours ($P < .001$) and 56.9 mg/dL lower at 5 hours ($P < .0001$) during the RE versus CON condition (Fig. 4B).

For Total-C, there was no significant age $\times$ condition $\times$ time interaction ($F_{(3,53)} = 0.38$; $P = .77$), nor any lower order

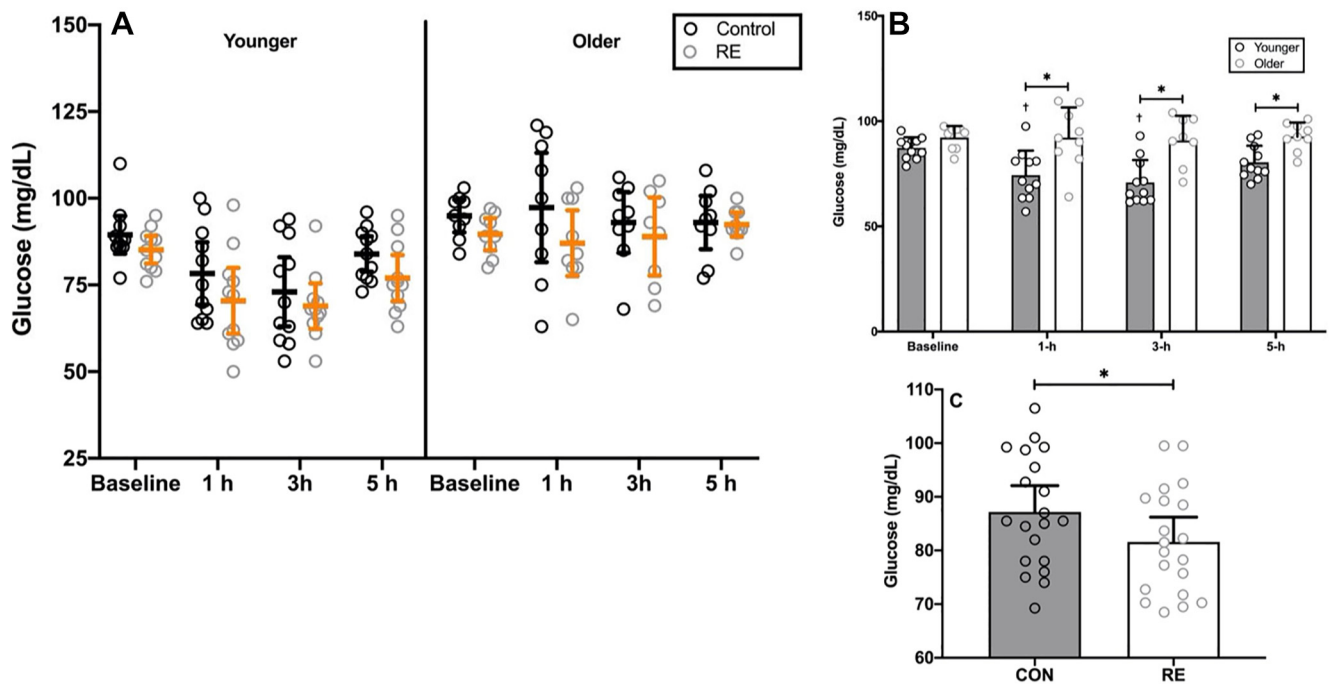


Fig. 3 – Postprandial glucose responses. The (A) glucose responses during the control (black open circles, black mean $\pm$ 95% CI error bars) and resistance exercise (gray open circles, orange mean $\pm$ 95% CI error bars) conditions in the younger and older men. Panel (B) shows the age $\times$ time interaction, where * denotes a significant difference between age groups (Older > Younger, $P < .05$) and † denotes a significant difference from baseline in the younger men ($P < .05$). Panel (C) shows the condition fixed effect, where * denotes that glucose was lower in RE than CON ($P < .05$).

interactions or fixed effects (all $P \geq .076$). For LDL-C, there was no age $\times$ condition $\times$ time interaction ($F_{(3,53)} = 0.53$; $P = .67$), but there was a condition $\times$ time interaction ($F_{(3,53)} = 3.02$; $P = .038$). In the CON condition, LDL-C decreased from baseline and 1 to 3 and 5 hours (all $P < .001$); whereas in the RE condition, LDL-C decreased from baseline and 1 to 3 hours (both $P \leq .014$), but not 5 hours (both $P \geq .12$). However, there were no differences in LDL-C at baseline, 1, 3, or 5 hours between conditions (all $P \geq .23$). For HDL-C, there was no age $\times$ condition $\times$ time interaction ($F_{(3,53)} = 0.79$; $P = .51$), but there was a fixed effect for time ($F_{(3,54)} = 4.87$; $P = .005$). HDL-C decreased from baseline to 3 (-2.8 mg/dL; $P = .014$) and 5 hours (-3.2 mg/dL; $P = .005$) when collapsed across age and condition.

3.4. Total and incremental area under the curves

For TG and MLI tAUC, there were no age $\times$ condition interactions ($F_{(1,18)} = 1.87$ and 1.75 , respectively; $P = .19$ and 0.20 , respectively); however, there were condition main effects for both ($F_{(1,18)} = 12.74$ and 17.21 , respectively; both $P < .01$, Fig. 5A and C). The TG tAUC was $154.0 (\pm 93.3)$ mg/dL·h lower and the MLI tAUC was $178.0 (\pm 94.1)$ mg/dL·h lower in the RE than CON condition. For GLU tAUC, there was also no age $\times$ condition interaction ($F_{(1,18)} = 0.01$; $P = .91$), but there were age ($F_{(1,18)} = 18.0$; $P < .001$, Fig. 6B) and condition ($F_{(1,18)} = 16.91$; $P < .001$, Fig. 6A) main effects. The GLU tAUC was $71.7 (\pm 45.0)$ mg/dL·h greater in the older than younger men and was $28.7 (\pm 14.1)$ mg/dL·h lower in the RE than CON condition.

For TG and MLI iAUC, there were no age $\times$ condition interactions ($F_{(1,18)} = 0.40$ and 0.23 , respectively; $P = .54$ and

0.64 , respectively); however, there were condition main effects for both ($F_{(1,18)} = 13.53$ and 13.40 , respectively; both $P = .002$, Fig. 5B and D). The TG iAUC was $91.6 (\pm 51.9)$ mg/dL·h lower and the MLI iAUC was $95.4 (\pm 54.3)$ mg/dL·h lower in the RE than CON condition. For GLU iAUC, there was no age $\times$ condition interaction ($F_{(1,18)} = 0.12$; $P = .74$), but there was a main effect for age ($F_{(1,18)} = 9.18$; $P = .007$, Fig. 6C). The GLU iAUC was $56.4 (\pm 39.2)$ mg/dL·h lower in the younger than older men.

3.5. Relationships among metabolic response improvement, skeletal muscle mass, and volume

Table 3 contains the Pearson correlation matrix for the MLI responses, SMM, VOL_{Total} and age. In brief, SMM and VOL_{Total} were related ($r = 0.89$, $P < .001$), MLI_{tAUC} during the CON and RE conditions were related ($r = 0.91$, $P < .001$), and the ΔMLI_{tAUC} was related ($r = 0.72$, $P < .001$) to the MLI_{tAUC} in the CON condition. In addition, age was inversely related to SMM ($r = -0.64$, $P = .003$) and VOL_{Total} ($r = -0.63$, $P = .003$). There were no other significant relationships.

4. Discussion

To our knowledge, this is the first study to examine the influence of an acute bout of RE on the post-prandial metabolic response to a HFM in younger versus older adult men. The primary finding of this study is that RE decreased postprandial metabolic responses similarly in younger and

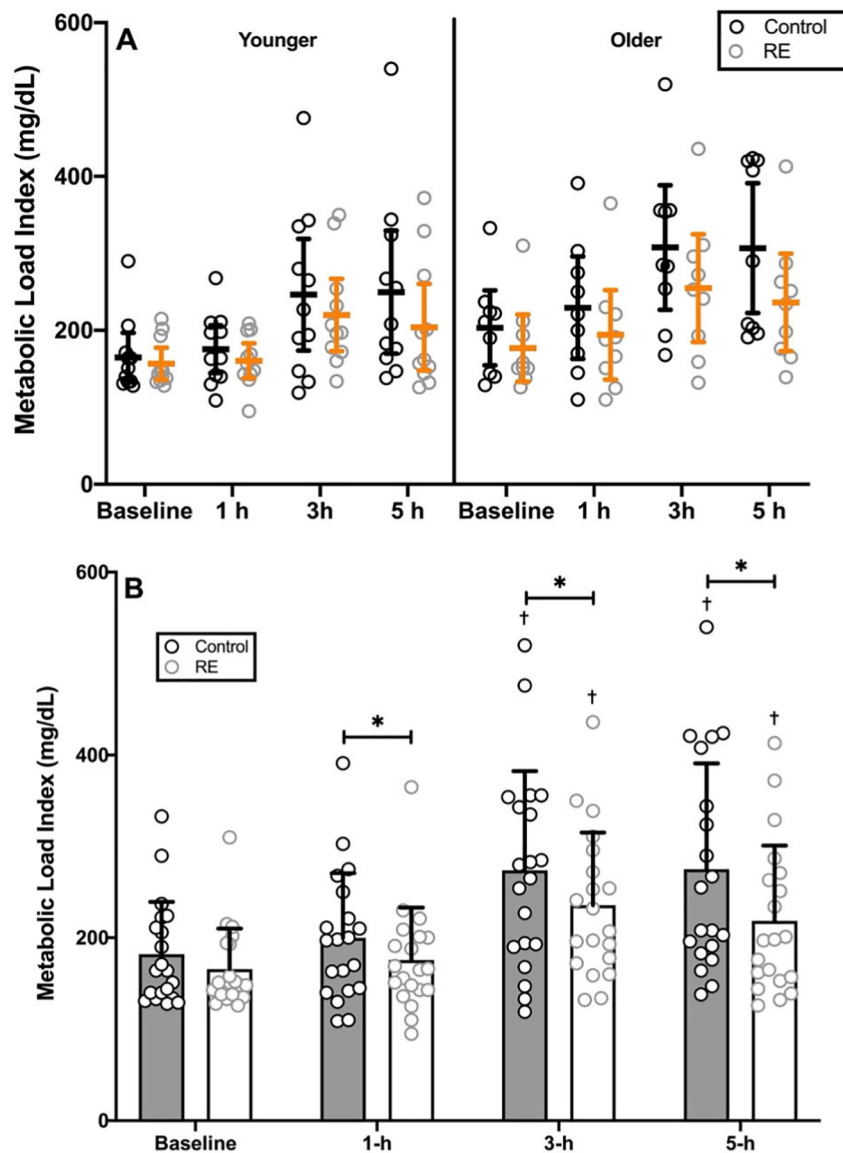


Fig. 4 – Metabolic load index responses. The (A) metabolic load index responses during the control (black open circles, black mean $\pm$ 95% CI error bars) and resistance exercise (gray open circles, orange mean $\pm$ 95% CI error bars) conditions in the younger and older men. Panel (B) shows the condition $\times$ time interaction, where * denotes a significant difference between conditions (CON > RE, $P < .05$) and † denotes a significant difference from baseline and 1 hour ($P < .05$).

older healthy men, as indicated by similar RE-mediated reductions in TG and MLI concentrations 3 to 5 hours and 1 to 5 hours post-meal independent of age, respectively. Furthermore, the TG and MLI tAUCs and iAUCs were 15% to 31% lower in the RE than CON condition. Thus, due to the lowered TG and MLI iAUC values, it appears that the benefits of RE extended beyond changes in fasting levels of these metabolic markers in our sample. Consequently, we accepted our hypotheses that the post-prandial glucose response would be greater in older than younger men and that RE would be effective for lowering PPL, but rejected the hypotheses that the PPL response would be greater in the older than younger men and that RE would be less effective for this purpose in older adults. These findings are novel and important because they indicate that RE may be

safely prescribed to improve PPL independent of age and, chronically, may help prevent age-related declines in cardiometabolic function.

Several previous studies have examined the influence of acute RE on PPL in young adults but have reported conflicting results [5,23–26]. It has been suggested that the differences among these studies were due to differences in the protocol as it is related to the volume, intensity, or timing of resistance exercise prior to the high-fat meal [5]. In the present study, we utilized an exercise protocol that resulted in an average net caloric expenditure of 0.77 MJ (eg, 185 kcal) and 201.6 resistance exercise workload units, which was between the low- (gross energy expenditure = 0.76 MJ; 144 U) and high-volume resistance exercise (gross energy expenditure = 1.4 MJ; 288 U) protocols used previously by Zafeiridis et al [24] and

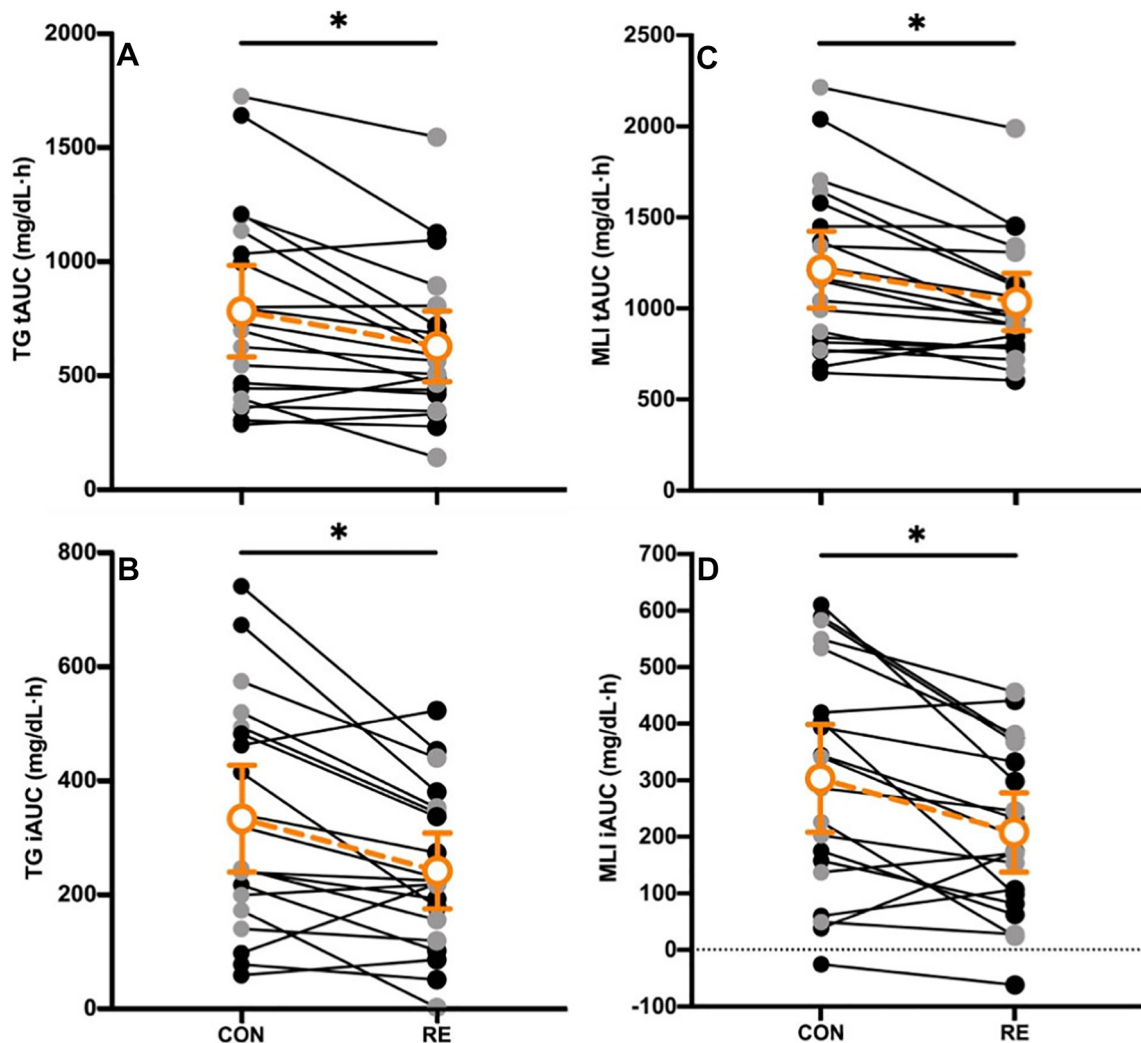


Fig. 5 – Triglyceride and metabolic load index AUC responses. The mean $\pm$ 95% CI (denoted by the orange, open circles and error bars) total and incremental area under the curves (AUCs) for (A and B) triglycerides (TG), and (C and D) the total metabolic load index (MLI) responses (collapsed across age) during the control (CON) and resistance exercise (RE) conditions. Individual responses are also depicted. Black circles indicate younger men, whereas gray circles indicate older men. * indicates CON > RE; $P < .05$.

similar to the moderate-intensity resistance exercise protocol (gross energy expenditure = 1.57 MJ; 192 U) used by Singhal et al [5]. However, it is notable that our protocol took approximately half the total time to complete (~40 min) because it was performed in circuit fashion. Nevertheless, we observed RE-mediated reductions in TG tAUC and iAUC of 20% and 27%, respectively, which were similar to the 20% to 24% reductions in TG tAUC observed by Zafeiridis and colleagues [24] and the 26% to 35% reductions in TG tAUC observed by Singhal et al [5]. Furthermore, the reduction in PPL caused by RE was independent of age-group. This improvement, however, was not related to either SMM or to RE volume, which were greater in the younger than older adults. Instead, it has previously been suggested that age-related reductions in metabolic function may be strongly mediated by decreases in physical activity [40–42]. Hamilton et al [43] suggested that age-related decreases in skeletal muscle lipoprotein lipase (LPL) activity may result in decreased triglyceride metabolism in older adults, but that contractile activity (ie, physical activity) may help counteract

these declines. In the present study, both the younger and older men were completing nearly 600 MET·min·day⁻¹ of physical activity, and there was no difference in physical activity level between them, which may provide a probable explanation for the lack of differences observed in both the PPL response and the improvement observed following RE in the older versus younger men [44]. Thus, our study adds to the body of literature suggesting that acute RE can be used to attenuate lipemic responses by approximately 20% to 30% in the postprandial period, and that this benefit extends to similarly to healthy, physically active older men.

In the present study, RE also reduced average GLU concentrations in the younger and older men by approximately 7%. Our finding of decreased GLU concentrations following an acute bout of RE is unique among studies that have examined the influence of RE on postprandial metabolic responses to a HFM. This novel finding may be partially explained by both the intensity of exercise and the timing and composition of the last meal participants consumed prior to the initiation of fasting. There is

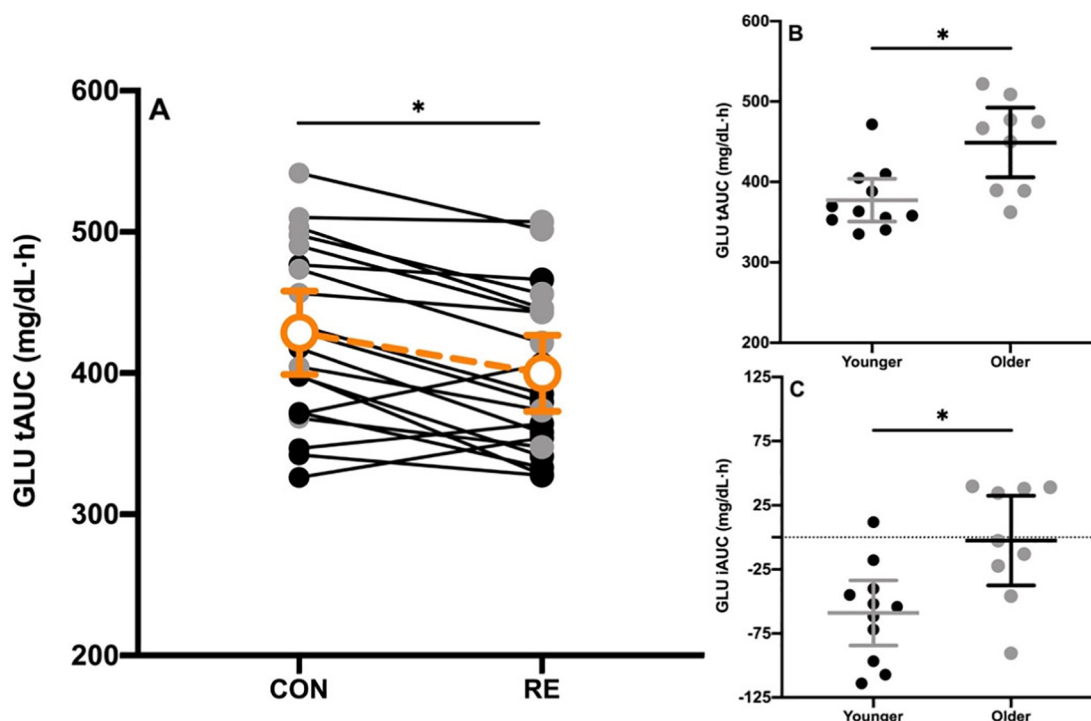


Fig. 6 – Glucose AUC responses. (A) The mean $\pm$ 95% CI (denoted by the orange, open circles and error bars) total area under the curve (AUC) for glucose during the control (CON) and resistance exercise (RE) conditions, where * indicates a significant difference between conditions (CON > RE, $P < .05$). Individual responses are also depicted. Black circles indicate younger men, whereas gray circles indicate older men. Panels (B and C) show the age main effects for glucose total AUC (tAUC) and incremental AUC (iAUC), respectively. * indicates significant difference between age-groups (older > younger, $P < .05$).

evidence that muscle glycogen has a regulatory role in substrate metabolism, where exercise-induced glycogen depletion increases fatty acid oxidation [45]. In addition, Trumbold et al [46] and Harrison et al [47] reported that the post-exercise carbohydrate deficit, rather than energy expenditure, influenced fat oxidation rates and, subsequently, postprandial TG responses

Table 3 – The relationships among age, total resistance exercise volume (VOL_{Total}), skeletal muscle mass (SMM), the metabolic load index AUC during the control (CON MLI_{tAUC}) and resistance exercise (RE MLI_{tAUC}) conditions, and the change in the MLI_{tAUC} from the CON to RE condition (ΔMLI_{tAUC})

	Age	VOL_{Total}	SMM	CON MLI_{tAUC}	RE MLI_{tAUC}	ΔMLI_{tAUC}
Age	1.000					
VOL_{Total}	–	1.000				
SMM	0.631		1.000			
CON MLI_{tAUC}	0.270	–0.179	–	1.000		
RE MLI_{tAUC}	0.198	–0.135	–	0.910	1.000	
ΔMLI_{tAUC}	0.275	–0.176	–	0.720	0.366	1.000
			0.077			

A bolded value indicates a significant relationship ($P \leq .003$).

following an overnight fast. Therefore, the composition of the post-exercise meal likely contributes to exercise-induced improvements in fat oxidation rates and postprandial TG responses. In the present study, the last meal consumed by the participants was a low-carbohydrate protein shake immediately prior to the RE or CON session. The combination of both the resistance exercise employed, which likely resulted in significant glycogen depletion [48] and, the snack that, although likely provided enough energy to offset the energy cost of the RE session did not offset the carbohydrate used during exercise, may have both had a cumulative influence on the observed decreases in both GLU and TG. Additional studies are needed to understand this interplay among meal composition, timing, and the postprandial metabolic benefits of RE.

We also observed an interaction among age and time for GLU in the present study. In the younger men, GLU decreased from baseline to 1 and 3 hours postprandially, whereas GLU did not change throughout the postprandial period in the older men. Therefore, glucose concentrations were lower at 1, 3, and 5 hours postprandially in the younger than older men. The GLU tAUC and iAUC were also 72 and 56 mg/dL·h lower in the younger men, respectively. These age-related differences in glucose responses are expected given that age-related reductions in glucose tolerance have been reported previously [49–51]. Skeletal muscle is considered the major site for glucose disposal. Therefore, differences in the amount and/or quality of skeletal muscle mass in our younger

versus older subjects may be a potential explanation for this age effect [52]. Indeed, skeletal muscle mass was 5.0 ± 3.0 kg lower in the older than younger men, and skeletal muscle mass was inversely associated ($r = -0.615$; $P = .004$) with the average glucose response (collapsed across age, condition, and time) in the present study.

Due to the interplay among carbohydrate and lipid metabolism, it has recently been suggested that postprandial glycemic and lipemic responses be considered together via quantification of the MLI, because it is likely a better indicator of the overall metabolic challenge an individual is experiencing [35]. In the present study, MLI tAUC and iAUC were 14.7% (-178 mg/dL·h) and 31.4% (-95.4 mg/dL·h) lower in the RE than CON condition. While we are unable to pinpoint the specific mechanism responsible for these improvements, it is plausible that RE substantially depleted intramuscular energy stores [53], increased fatty acid oxidation [23,54], enhanced the removal rate of very-low density lipoprotein TG [55] via increased lipoprotein lipase activity [23,56], altered insulin sensitivity [57,58], or some combination of each. Regardless of the mechanisms, however, the notable reductions in MLI AUCs following acute RE may have important clinical ramifications.

There are several limitations to this study, the primary of which include the following. First, while we conducted body composition and physical activity assessments, neither represent the respective gold standard assessment. However, both are widely employed, have been shown valid in various populations, and because they did not represent the primary outcome of the present study, we believe they were sufficient for the purposes of this study. Second, we were unable to speak to mechanisms beyond those currently reported in the manuscript. Follow-up examinations are necessary to directly measure the effects of RE on postprandial metabolism in skeletal muscle of younger versus older adults using methods similar to those employed by Baugh et al [18]. Third, our participants were relatively homogenous apart from age and were physically active. While this provided an additional level of control, it is also not clear to what degree these results can be applied to inactive older adults.

Overall, our data add to the current body of literature suggesting that acute RE is effective for attenuating postprandial metabolic responses. Most importantly, our data are the first to suggest that this benefit is also extended to older men. Because postprandial metabolic derangements independently predict CVD incidence and mortality, these findings have important implications. Specifically, while the benefits of RE for musculoskeletal health, weight management, and quality of life are widely acknowledged by organizations such as the American Heart Association [59] and American College of Sports Medicine [20], the cardiometabolic benefits are less well-understood and generally un-acknowledged. Furthermore, the fact that the present improvements in postprandial responses were observed following a ~40-minute RE session (whereas greater than 60 or 90 minutes of moderate- or low-intensity aerobic exercise may be needed to observe consistent improvements) also has particular importance from an exercise prescription and adherence perspective. Thus, these data may add to the foundation on which public health messages regarding resistance exercise as a primary preventative intervention for cardiometabolic disease are formed.

CRediT authorship contribution statement

Nathaniel D.M. Jenkins: Conceptualization, Methodology, Formal analysis, Resources, Writing – original draft and review & editing, Visualization, Supervision, Funding acquisition. **Nile F. Banks and Emily M. Rogers:** Investigation, Data curation, Writing – review & editing. **Christina M. Sciarillo, Nicholas A. Koemel, and Ryan J. Colquhoun:** Investigation, Writing – review & editing. **Sam R. Emerso:** Conceptualization, Methodology, Resources, Writing – review & editing, Supervision, Funding acquisition.

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