Acute exercise improves glucose and triglyceride metabolism in young and older adults following high-fat, high-carbohydrate meal intake

Stephanie P. Kurti\textsuperscript{1,2}, Hannah Frick\textsuperscript{1,2}, William S. Wisseman\textsuperscript{1}, Steven K. Malin\textsuperscript{3,4}, David A. Edwards\textsuperscript{4}, Sam R. Emerson\textsuperscript{5}, Elizabeth S. Edwards\textsuperscript{1,2}

\textsuperscript{1}James Madison University, Department of Kinesiology-Human Performance Laboratory, 261 Bluestone Drive, MSC2302, Harrisonburg, VA, 22807

\textsuperscript{2}Department of Kinesiology-Morrison Bruce Center, 261 Bluestone Drive, MSC2302, Harrisonburg, VA, 22801

\textsuperscript{3}Rutgers University, Department of Kinesiology and Health, Division of Endocrinology, Metabolism and Nutrition, 70 Lipman Drive, New Brunswick, NJ, 08901

\textsuperscript{4}University of Virginia, Department of Kinesiology, Charlottesville, VA, 22904

\textsuperscript{5}Oklahoma State University, Department of Nutritional Sciences, 301 HSCI/NRD Stillwater, OK 74078

**Corresponding author:** Stephanie P. Kurti, 261 Bluestone Drive, MSC2302, Harrisonburg, VA, 22807, Email: kurtisp@jmu.edu

**Shortened titled:** Acute exercise improves postprandial glucose and triglycerides in young and older adults

\textsuperscript{NS}

This peer-reviewed article has been accepted for publication but not yet copyedited or typeset, and so may be subject to change during the production process. The article is considered published and may be cited using its DOI

10.1017/S0007114521002208

The British Journal of Nutrition is published by Cambridge University Press on behalf of The Nutrition Society
Abstract
A single high-fat, high-carbohydrate meal (HFHC) results in elevated postprandial glucose (GLU), triglycerides (TRG) and metabolic load index (MLI; TRG (mg/dL) + GLU (mg/dL)) that contributes to chronic disease risk. While disease risk is higher in older adults (OA) compared to younger adults (YA), the acute effects of exercise on these outcomes in OA is understudied. Twelve YA (age 23.3 ± 3.9 yrs, n = 5 M/7 F) and 12 OA (age 67.7 ± 6.0 yrs, n = 8 M/4 F) visited the laboratory in random order to complete a HFHC with no exercise (NE) or acute exercise (EX) condition. EX was performed 12 hours prior to HFHC at an intensity of 65% of maximal heart rate to expend 75% of the kcals consumed in HFHC (Marie Callender’s Chocolate Satin Pie; 12 kcal/kgbw; 57% fat, 37% CHO). Blood samples were taken at 0, 30, 60, 90 minutes, and then every hour until 6 hours post-meal. TRG levels increased to a larger magnitude in OA (Δ~61 ± 31%) compared to YA (Δ~37 ± 34%, p < 0.001), which were attenuated in EX compared to NE (p < 0.05) independent of age. There was no difference in GLU between OA and YA after the HFM, however EX had attenuated GLU independent of age (NE: Δ~21 ± 26%; EX: Δ~12 ± 18%, p = 0.027). MLI was significantly lower after EX compared to NE in OA and YA (p < 0.001). Pre-prandial EX reduced TRG, GLU and MLI post-HFHC independent of age.

Keywords: Aging, physical activity, triglycerides, postprandial lipemia, postprandial glycemia
Introduction

Older adults have increased risk of developing life-style related diseases such as CVD and type 2 diabetes and hyperlipedemia, with diagnosed CVD in over 70% of individuals between the ages of 60 and 79 (1). While several factors in older adults’ influence disease risk, dietary intake including energy-dense, nutrient-poor foods that are high in saturated fats and sugar are thought to promote metabolic and vascular impairment. Indeed, even a single high-fat, high-carbohydrate meal (HFHC) meal that raises postprandial lipemia (PPL, e.g. triglycerides) and glucose (PPG) results in insulin resistance as well as impaired endothelial function (2). HFHC meals induce these cardiometabolic derangements due to, in part, a collective substrate toxicity that exacerbates oxidative stress/inflammatory mediated mechanisms, a concept referred to as the “metabolic load index” (MLI) (3). Since up to 40% of individuals with coronary artery disease have normal plasma lipid concentrations in the fasting state (4), additional work is warranted to understand treatments that influence HFHC mediated effects on post-prandial metabolic health.

Exercise attenuates PPL and PPG following HFHC in some (5,6), but not all studies (7) (8) (9). Although several factors (e.g. training status, exercise intensity or duration, meal timing around exercise, etc.) may explain the disparity between these prior studies on affecting metabolic load (10) (11) (12)(13), the majority of this work has been conducted in young to middle-aged adults. In fact, there are very few studies investigating the impact of exercise on post-prandial responses in older adults (OA) (9). This is clinically problematic since OA have been reported to have higher PPL and PPG with a HFHC meal compared to a younger group, even when physically active (14). Therefore, we sought to examine the PPL, PPG and MLI in young and older adults following a HFHC and secondarily determine whether an acute bout of exercise would attenuate these responses. Our hypothesis was three-fold: (1) PPL, PPG and MLI would be higher in OA compared to YA, (2) an acute bout of exercise would attenuate PPL, PPG and MLI in OA and YA and (3) there would be an interaction between age and acute exercise on PPL, PPG and MLI.
Methods

Subjects

Twenty-four subjects were recruited to participate in the study from the local Harrisonburg, Virginia community, including 12 YA (23.3 ± 3.9 yrs, n = 5 M/7 F) and 12 OA (67.7 ± 6.0 yrs, n = 8 M/4 F). All participants were free of cardiovascular, metabolic and renal diseases as assessed by health history questionnaire. Subjects were not currently consuming any antioxidant supplements, anti-inflammatory agents, or medications (e.g. statins, hypertensives, anti-diabetic agents, etc.) that would interfere with the primary outcomes of the study. Subjects also completed a physical activity readiness questionnaire (PAR-Q), and the International Physical Activity Questionnaire (I-PAQ) to determine chronic PA level in MET-minutes/week. All of the procedures were approved by the Institutional Review Board at James Madison University (protocol #19-0747) in accordance with the Declaration of Helsinki, and participants signed informed consent documents.

Experimental Design

Subjects visited the laboratory for an initial consult and were then randomized between: HFHC with no exercise (NE) or an acute bout of exercise (EX) performed 12 hours prior to a HFHC (Figure 1), with condition separated by at least 7 days. Subjects did not go longer than 3 weeks between trials and body mass was assessed at each session to ensure subjects were weight stable. The HFHC was consumed at the same time between conditions for each subject; this start time ranged from 5 am and 9 am among subjects. Subjects were also instructed at the initial visit on completing a food log in which they were required to replicate prior to both of their HFHC challenges.

Anthropometry and Blood Pressure

On each subject’s initial visit, height was measured with a portable stadiometer (Charder Model HM 200P, Charder Electronic Co Ltd., Taichung, Taiwan). Body mass was assessed using a standard physician’s scale (Dymo Pelouze model 4040, Newell Brands, Hoboken, NJ) and, in turn, body mass index was calculated. Subjects underwent dual-energy x-ray absorptiometry (DEXA) scan (GE Lunar iDXA, Fairfield, CT) to measure lean body mass and body fat. Subjects then sat up and rested for 5 minutes to assess brachial artery blood pressure (BP) using an automatic sphygmomanometer (ProBP 3400 Welch Allyn, Skaneateles Falls, NY). Two measurements were taken and averaged as detailed in the American College of Sports
Accepted manuscript

Medicine (ACSM) guidelines (15). Waist circumference was then measured two times by the same investigator at the narrowest part of the waist with a Gulick tape measurer (Creative Health Products, Ann Arbor, MI) and values were averaged for analysis.

Incremental Exercise Test

The incremental exercise test was performed on a cycle ergometer (VIAsprint 150P) to determine VO\(_{2\text{peak}}\). Metabolic and ventilatory data were recorded through the entire test and 30-sec averages were used for analyses (Vmax Encore, Vyaire Medical, Lake Forest, IL). The protocol began with a five-minute warm-up at a self-selected cadence of \(\geq 50\) revolutions per minute and self-selected power. The power output was either increased or decreased during the warm-up every minute until subjects identified a workload that they perceived as sustainable for approximately thirty minutes. The starting output for the test and increment by which output was increased every minute were determined by the warm-up workload (Supplementary Table 1). Heart rate (HR) (Polar Lake Success, NY) and rating of perceived exertion (RPE) were recorded in the last 10-20 seconds of each stage. Subjects were verbally encouraged by investigators throughout the entire test until they reached volitional fatigue or inability to maintain \(\geq 50\) rpm.

Pre-prandial Exercise Bout

In the EX-trial, the acute exercise bout was performed 12 hours prior to the HFHC challenge on a cycle ergometer and corresponded to 60-70% of their HR\(_{\text{peak}}\). Subjects were required to exercise until they achieved a total energy expenditure of 75% of the calories that would be consumed during the HFHC challenge (Equation 1). This protocol has been chosen to administer a true-to-life bout of moderate intensity exercise, however for a slightly longer duration than in our previous research in which there were no effects of exercise on postprandial metabolic outcomes (7)(16). Subjects were not permitted to exercise longer than 2-hours for older adults for safety and logistical reasons. However, only two OA were cut short on exercise and all other subjects completed their complete exercise bout.

Equation 1:

\[
\frac{mass \ in \ kg \times 12.5 \ kcal/s}{absolute \ VO2 \times 4.825L \ 02/kcal} \times 0.65
\]

HFHC Challenge

Subjects were instructed to replicate diet, sleep, and refraining from vigorous physical activity/habitual exercise the day prior to their HFHC sessions. After an overnight fast, subjects
arrived at the laboratory and were seated in a reclining phlebotomy chair. After about 5 minutes of rest, two baseline BP measurements were taken with at least 30 seconds of rest between each recording and averaged for analysis. An indwelling catheter was then inserted into a forearm vein via a 22-gauge needle (Fisher Scientific, Waltham, MA) and kept patent with 0.9% NaCl. The HFHC meal (Marie Callender’s Chocolate Satin Pie [57% fat, 39% carbohydrate, 4% protein]; Conagra Brands, Chicago, IL) was standardized to 12 kcal/kg BW and consumed within a 20-minute period following the baseline blood draw. Subsequent timings for blood draws are based upon the time at which the subject finished the HFHC meal. In addition to baseline, blood samples were drawn every 30 minutes postprandial until the two-hour mark, after which point draws were performed every hour up to 360 minutes. Glucose (GLU) was assessed at baseline, 30, 60, 90, 120, 240 and 360 minutes and triglycerides (TRG), total cholesterol (TC) and low as well as high density lipoproteins (LDL and HDL, respectively) were assessed at baseline and hourly following the HFHC meal to 360 minutes. LDL was calculated using the Friedewald equation.

Statistical Analyses

An a priori sample size calculation was performed based on previous research\(^\text{17}\) with the difference in postprandial TRG change between OA and YA as the main outcome. The difference is postprandial TRG change between OA and YA was 68 ± 34.8 mg/dL (Cohen’s \(d = 0.96\)). With a power of 0.80 and a two-sided \(\alpha = 0.05\), 8 subjects were required per group to observe differences in TRG between the OA and YA. We recruited 12 participants to each group to ensure statistical power. Data were analyzed using IBM SPSS Statistics v26.0 (IBM Corporation, Armonk, NY). Metabolic load index (MLI) was calculated as a summation of the TRG + GLU responses at each hourly measurement as previously described\(^\text{18}\). All data were analyzed for skewness, kurtosis, and normality using the Shapiro-Wilk test. Glucose and LDL were not normally distributed and were log transformed. A repeated measured ANOVA including all time points was used with time and condition (NE, EX) as the within-subjects’ factors and age as the between-subjects factor. When sphericity failed, the Greenhouse-Geiser correction factor was utilized. Post-hoc analyses were performed when there were significant effects with a Bonferroni correction. Secondary analyses for area under the curve (AUC), incremental AUC (iAUC), and peak values were calculated for TRG and GLU using GraphPad Prism (Graphpad Software, Inc; La Jolla, CA). Significance was set a priori at \(p < 0.05\).
Results

Subject Characteristics

There were no significant differences in body mass (p = 0.17), BMI (p = 0.72), body fat (p = 0.15) or android fat (p = 0.06) by age group, however older adults were taller compared to younger adults (p = 0.02, Table 1). The kilocalorie content of the HFHC meal consumed by YA was not statistically different from the OA (856.8 ± 207.1 kcals and 968.8 ± 180.9 kcals, respectively, p=0.17).

Maximal and Submaximal Exercise Characteristics

While relative VO2peak was significantly lower in OA compared to YA (p = 0.005) due to body mass, absolute VO2 was similar between OA and YA (p > 0.99, Table 1). Watts achieved at peak exercise was not different in the OA and YA (p = 0.97), but peak HR was significantly lower in older versus young adults (p < 0.01). Moreover, there were no significant differences in the exercise duration or energy expenditure in kcals (p = 0.07 and p = 0.17, respectively; Table 2).

Fasting Substrates

Older adults had significantly higher TC (age effect: p < 0.01) and LDL-C (age effect: p < 0.01) compared to younger adults (Table 3). EX condition did not affect fasting GLU, TRG and cholesterol in young or older adults the following morning (all p’s > 0.05). In addition, all fasting metabolic outcomes were below clinical values for diagnosis of type 2 diabetes (19) or hyperlipidemia (20).

Postprandial Metabolic Responses

While TRG increased from baseline to 6-hours (time effect; p < 0.01) among all groups, TRG were significantly greater in older versus young adults (age effect; p < 0.01). There was a significant effect of exercise on TRG in OA and YA (Figure 2). However, there was no difference between age or effect of exercise on TRG tAUC (p=0.294), iAUC (p = 0.487), or peak TRG response (p=0.085).

There was a significant increase in GLU shortly after the meal, but concentrations returned to baseline within the assessed post-prandial period (p < 0.001, Figure 3). Exercise lowered the GLU response in both YA and OA compared to the non-exercise control (p = 0.027). In fact, there was significantly lower GLU response in the OA-EX condition compared to the
YA-NE condition (p = 0.014). However, there was no difference between age or effect of exercise on glucose tAUC (p = 0.356), iAUC (p = 0.910), or peak GLU response (p = 0.09).

MLI increased across time-points among all subjects (time effect; p < 0.001). There was a significantly higher MLI in O-NE compared to the Y-NE (p = 0.008) and Y-EX (p < 0.001), driver by greater postprandial TRG (Figure 4). The OA-EX also had a significantly greater MLI compared to the YA-EX (p=0.049). However, MLI was reduced with acute exercise in OA-EX and YA-EX compared to OA-NE and YA-NE (p < 0.001). There was no difference in postprandial MLI for tAUC (p = 0.135), iAUC (p = 0.478) or peak MLI (p = 0.228) between age or exercise. All postprandial metabolic outcomes for YA in NE and EX and the OA in NE and EX conditions are displayed in Table 4.

There was no change across time-points for TC from baseline to 6-hours (p = 0.836), however, TC was significantly lower by condition (p<0.001, Figure 5A). TC was higher in older versus younger adults (age effects; p < 0.001). In fact, there was also a significantly higher postprandial TC response in the O-NE condition compared to the YA-NE and YA-EX conditions (p = 0.020 and p = 0.031, respectively). For LDL-C, there was no change across time-points from baseline to 6-hours (p = 0.205, Figure 5B), however there was a significant effect of condition where OA-EX and YA-EX had lower LDL compared to OA-NE and YA-NE (p < 0.001). LDL-C was also higher in older compared to younger adults. Peak LDL tended to be higher in older compared to younger adults independent of exercise (p = 0.052). Similarly, HDL-C was unchanged across time (p = 0.943, Figure 5C), but was significant by condition (p=0.016). HDL was also significantly higher in the older compared to younger adults.
Discussion

In the present study, older adults had elevated post-prandial TRG, but not glucose, compared with young adults. In turn, older adults had a higher MLI across time compared to younger adults. This finding confirms previous literature that regardless of chronic PA level there are independent effects of aging on postprandial substrate responses (14). Secondarily, EX 12 hours prior to the HFHC meal lowered postprandial TRG, glucose and MLI in both young and older adults. These findings support exercise as an intervention to impact both glucose and TRG, although the absence of a greater attenuation in postprandial TRG in older adults compared to younger adults is noteworthy given overall concentrations were higher.

Postprandial lipid responses with and without acute exercise

Our study supports previous findings that PPL (namely TRG) are elevated in older adults compared to younger adults. However, we believe that the fitness level (i.e. absolute VO2peak) and health status of the older adults that composed our study population may have been why postprandial TRG did not exhibit a larger attenuation post-exercise. The findings that even healthy, fit older adults have elevated postprandial TRG is important because postprandial TRG may better predict the risk of cardiac event or CVD development than fasting lipids (21) (22). Therefore, identifying methods to attenuate this larger TRG response is critical to minimize deleterious health outcomes.

Interestingly, the impact of acute exercise on postprandial TRG levels in young adults has mixed findings, with true-to-life exercise durations and intensities between 30 minutes and an hour not consistently showing reductions in postprandial TRG (7,8,23). Many factors such as exercise timing (10) (12), caloric replacement after the exercise bout (16) (24) (25), energy expenditure relative to the test meal (26) (27), and mode of exercise (28) are likely important considerations for the effect of exercise on reductions in lipemia post-meal. While there is conflicting literature on the effect of exercise timing, duration, and energy expenditure relative to the test meal in healthy young adults, more consistent findings report acute exercise reduces adverse metabolic outcomes in untrained, inactive or diseased populations. Specifically, 90 minutes of moderate intensity preprandial exercise attenuated postprandial TRG in untrained adults (29) and adults diagnosed with hyperlipidemia or other metabolic diseases (5). In the present study, an acute bout of preprandial exercise expending 75% of the calories consumed in a HFHC meal was sufficient in reducing postprandial TRG and glucose in healthy, active young and older individuals. This prescription
though may not be possible for untrained or inactive older adults. Although exercise bouts of shorter duration, even when bouts are dispersed throughout the day, may be sufficient to elicit reductions in postprandial glucose in inactive adult males (30), there are no studies to our knowledge examining metabolic responses (i.e. TRG, GLU and MLI) to a HFHC meal after acute preprandial exercise in inactive older adults specifically. Additional work is warranted to understand the optimal dose of an acute preprandial exercise bout in untrained or inactive older adults.

To our knowledge, this is the first study to examine the impact of acute exercise in fit older adults compared to a younger group with similar absolute fitness levels and other subject characteristics. The only other recent investigation, to our knowledge, elucidated the effect of moderate-intensity exercise on reducing postprandial TRG in middle-aged adults and older adults (mean age: 58 y/o) (9). Specifically, 90 minutes of exercise 12 hours prior to a HFM was not sufficient in attenuating any metabolic outcomes (TC, LDL, HDL, TRG or glucose) in the middle-aged and older adults who were of mixed physical activity levels. It was speculated that the chronic physical activity level of the subjects and their age may partially explain the findings. Our findings, however, demonstrate that while the older adults in both studies exhibited larger postprandial TRG compared to a control group, a single exercise bout was able to lower TRG in the present study. We did not design this study to determine the mechanism by which exercise lowered TRG, but we suggest that exercise may have raised skeletal muscle LPL activity (31). LPL activity is the rate-limiting step for TRG clearance in the circulation and provides a direct mechanism to regulate TRG concentrations during the postprandial period (32), therefore increasing LPL activity may decrease PPL. Also, acute exercise could impact PPL through reductions in the appearance of TRG-rich lipoproteins from the liver (5), which has been previously found to account for up to 70% of the TRG attenuation (33).

In aging humans, there is reduced TRG clearance rates (34) which may come from several mechanisms, which include but are not limited to; the decrease in LPL activity with age (35) and age-related changes in liver physiology including increased liver steatosis (36), with evidence that this increase in liver fat is accompanied by an increase in circulating TRG (37). While several factors may explain the results of the present study, lowered LPL activity specifically in older adults could explain why older adults had an elevated postprandial TRG response compared to younger adults due to reduced fatty acid utilization and TRG lipolysis. In addition, it is possible
that with lowered LPL activity and lower TRG clearance, older adults were less responsive to the acute bout of exercise than expected, but still more responsive than in previous work using a similar age group. Therefore, exercise contributes to lower TRG, but it is likely dependent on the intensity of the exercise relative to the test meal and the fitness levels of the subjects included.

The acute exercise used in the present study also had an effect by condition and not by age on TC and LDL-C. The findings of the present study also align with work by Bittel and colleagues who reported that a single bout of resistance training reduced lipemic responses to a mixed meal in obese prediabetic men without significantly altering postprandial VLDL iAUC (38). Yet there was a reduction in meal-derived fatty acid incorporation into chylomicrons and triglyceride-rich lipoprotein-TRG. In the present study, we have also reported no changes in LDL-C iAUC, yet reduced LDL-C in the EX + HFHC meal condition. Previous examinations have suggested that an exercise bout that created an energy deficit of >1100 kcal will elicit changes in LDL and HDL (26) (39). Our study adds to the existing literature by providing a possible exercise prescription to attenuate TRG, glucose, TC and LDL-C with a true-to-life exercise bout averaging ~643 kilocalories expended in young adults and ~730 kilocalories expended in older adults.

Glucose responses in OA and YA and the impact of acute exercise

An interesting finding in the present study was the lack of any significant differences in glucose levels between younger and older adults. Glucose levels typically increase with age (40). A confounder often to this observation is fitness, and it is possible the similar postprandial glucose responses in younger and older adults may be due to their similar VO2peak levels. Also, since there were no differences in BMI or central adiposity, which are highly associated with insulin resistance (41), it is reasonable that there were similar glucose responses in older and younger adults. Acute exercise has been shown in some (42) (43), but not all studies (9) to lower circulating blood glucose in adults at risk for chronic disease. Herein, we report that acute EX lowers PPG in younger and older adults. The reason exercise lowered PPG in the current study is beyond the scope of this work, but it may relate to hepatic glucose production, skeletal muscle glucose uptake or elevated insulin sensitivity (44). Since there was no effect on fasting glucose levels, it would suggest that hepatic glucose production was unaffected by exercise in the present work. Subsequently, further work is needed to clarify whether peripheral insulin action and/or pancreatic function is improved uniquely in young versus older adults.
Taken together, the glucose and other metabolic results in the present study align with recent work by Ramirez-Velez et al., who investigated the impact of exercise training on postprandial lipid and glucose responses (45). While the authors utilized various exercise training protocols in a group of inactive younger adults approximately 30 years of age, they reported that postprandial glucose was attenuated after an exercise training program, whereas there were trends present for the attenuation of postprandial TRG. Considering the group utilized in the present study were fit older adults compared to a younger adult group with similar fitness, it is not surprising that metabolic outcomes align, with a consistent reduction of glucose. The lowered glucose in the younger group also drove a reduction in the metabolic load index. Thus, there was a lower metabolic load post-meal experienced after exercise in the younger adults compared to the older adults.

*Experimental considerations*

There are several limitations in the present study that are important to consider. First, since the older adults in the study were not active and also not on lipid-lowering medications, this demographic does not represent the majority of older adults in the United States. As a result, the subjects included in this study may limit the generalizability of the findings, and applying these results to older adults with chronic diseases should be done with caution. However, we also view the investigation of this specific population as a strength in the present study, since healthy, relatively fit older adults has not been previously investigated. Aging alone places these individuals at an increased risk for chronic disease development, so understanding and possibly mitigating deleterious responses is of critical importance. Also, the young adults included in this study had higher mean fasting glucose than age-predicted normal values (19). While this suggests that participants on average, did have impaired fasting glucose, repeated measures are needed for clinical diagnosis and impaired fasting glucose is not a disease itself. Also, it remains unclear if exercise would uniquely effect glucose and lipid responses in different cohorts of prediabetes (i.e. impaired fasting glucose and/or impaired glucose tolerance). Further, it is worth noting that fasting glucose levels in our study could be due to the method of glucose assessment. Indeed, it is possible that the Cardiochek Plus PA Analyzer had a positive bias for glucose assessment (46). However, the Cardiochek hand-held device is certified by the Center for Disease Control and Prevention for meeting clinical laboratory reference standards (47) and is acceptable to use in glucose assessment. Next, cycling exercise was performed in the present study and several of the
cited studies administered walking, running and resistance training exercise interventions. Utilizing exercise that incorporates greater muscle mass recruitment may be an important consideration in future work, and also better elucidating how exercise mode may impact postprandial responses. In the present study, we were not able to match the number males and females that were included in the OA and YA due to recruitment issues, specifically in recruiting OA who met the inclusion criteria. However, we do not believe sex influences our findings between OA and YA since there were no time by sex interactions for any primary metabolic outcomes (Supplementary data in Tables 2 and 3). Finally, we tried to make a realistic test meal and exercise protocol to keep a true-to-life aspect of the study design, however participants did not eat after the exercise bout since we have recently reported that it can abolish the effect of exercise on postprandial lipemia \(^{(25)}\). Caloric replacement after acute night-time exercise may be more typical of human behavior, though may interfere with the mechanisms that are responsible for the effect of an exercise bout on PPL and PPG.

**Conclusion**

While older adults have greater postprandial lipemia compared to younger adults who are similar in fitness levels, an acute bout of exercise is effective at reducing postprandial lipemia in older and younger adults. While the older and younger adults did not show differences in postprandial glucose responses, both were reduced with acute exercise. Therefore, an acute bout of exercise lowers the total metabolic load experienced in older and younger adults. However, since there was not a greater attenuation in TRG and glucose in older adults when compared to younger adults, further work should investigate mechanisms underpinning these results to maximize the use of exercise as a therapy to lower type 2 diabetes, dyslipidemia and overall CVD risk.

**Acknowledgements**

**Financial Support**

This research received grant support from the 4-Virginia. Funding for this project was provided by the 4-Virginia Collaborative research grant between James Madison University and the University of Virginia.
Conflicts of Interest
The authors declare that they have no conflicts of interest.

Author Contributions
The authors contributed to the completion of the manuscript in the following ways: S.P.K established the idea and wrote the manuscript, H.F and W.S.W helped to develop the idea, edited the manuscript and provided feedback, S.K.M and D.A.E helped with conceptualization of the project, edited the manuscript and provided vital feedback, S.R.E helped with developing the idea and interpretation of the data as well as manuscript edits and feedback, E.S.E aided in initial conceptualization of the project, editing of the manuscript and provided vital feedback.
Accepted manuscript

References:

1. CDC N (2015) Underlying Cause of Death 1999-2013. CDC WONDER Online Database.
9. Bodell NG & Gillum T 90 Minutes of Moderate-Intensity Exercise does not Attenuate Postprandial Triglycerides in Older Adults. Int. J. Exerc. Sci. 9, 677–684.


47. Laboratory Quality Assurance and Standardization Programs: Clinical Laboratory Certification Program, Centers for Disease Control and Prevention. 
Table 1. Subject Demographics

<table>
<thead>
<tr>
<th></th>
<th>YA (n = 12 / 5 M, 7 F)</th>
<th>Mean</th>
<th>SD</th>
<th>OA (n = 12 / 8 M, 4 F)</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>23.4 ± 3.8</td>
<td></td>
<td></td>
<td>67.4 ± 5.0*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height (cm)</td>
<td>167.5 ± 8.1</td>
<td></td>
<td></td>
<td>176.0 ± 8.9*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>71.4 ± 17</td>
<td></td>
<td></td>
<td>80.7 ± 15.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body mass index (BMI) (kg/m²)</td>
<td>25.3 ± 5.0</td>
<td></td>
<td></td>
<td>25.8 ± 3.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Body Fat (%)</td>
<td>28.2 ± 8.7</td>
<td></td>
<td></td>
<td>33.1 ± 6.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Android Body Fat (%)</td>
<td>30.1 ± 13.1</td>
<td></td>
<td></td>
<td>40.3 ± 9.7</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Significantly different from YA (p<0.05)
Table 2. Exercise Data

<table>
<thead>
<tr>
<th></th>
<th>YA (n = 12 / 5 M, 7 F)</th>
<th>OA (n = 12 / 8 M, 4 F)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>VO$_{2\text{peak}}$ (L/min)</td>
<td>2.3 ± 0.4</td>
<td></td>
</tr>
<tr>
<td>VO$_{2\text{peak}}$ (mL/kg/min)</td>
<td>33.4 ± 5.3</td>
<td></td>
</tr>
<tr>
<td>Peak Power (Watts)</td>
<td>199.2 ± 42.1</td>
<td></td>
</tr>
<tr>
<td>Peak Heart rate (bpm)</td>
<td>187.9 ± 11.7</td>
<td></td>
</tr>
<tr>
<td>Exercise Duration (min)</td>
<td>88.1 ± 15.2</td>
<td></td>
</tr>
<tr>
<td>Exercise Bout Expenditure (kcals)</td>
<td>642.6 ± 155.4</td>
<td></td>
</tr>
</tbody>
</table>

*Significantly different from YA
(p<0.05)
Table 3. Metabolic outcomes measured in fasting participants

<table>
<thead>
<tr>
<th>Fasting values</th>
<th>Optimal Values</th>
<th>YA (n = 12 / 5 M, 7 F)</th>
<th>OA (n = 12 / 8 M, 4 F)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(mg/dL)</td>
<td>NE</td>
<td>EX</td>
</tr>
<tr>
<td>----------------</td>
<td>----------------</td>
<td>-----</td>
<td>-----</td>
</tr>
<tr>
<td>TRG (mg/dL)</td>
<td>&lt;150</td>
<td>107.3 ± 38.8</td>
<td>98.3 ± 29.3</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>&lt;100</td>
<td>115.0 ± 7.9</td>
<td>110.3 ± 10.3</td>
</tr>
<tr>
<td>MLI (mg/dL)</td>
<td>N/A</td>
<td>222.3 ± 40.7</td>
<td>208.6 ± 30.5</td>
</tr>
<tr>
<td>TC (mg/dL)</td>
<td>&lt;200</td>
<td>139.9 ± 17.2</td>
<td>151.3 ± 21.8</td>
</tr>
<tr>
<td>LDL-C (mg/dL)</td>
<td>100-129</td>
<td>63.8 ± 11.6</td>
<td>77.4 ± 18.4</td>
</tr>
<tr>
<td>HDL-C (mg/dL)</td>
<td>40-60</td>
<td>53.9 ± 16.1</td>
<td>54.3 ± 14.7</td>
</tr>
</tbody>
</table>

*Significantly different from YA (p<0.05)
Table 4. Postprandial metabolic outcomes

<table>
<thead>
<tr>
<th></th>
<th>YA (n = 12 / 5 M, 7 F)</th>
<th>OA (n = 12 / 8 M, 4 F)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NE</td>
<td>EX</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Triglycerides</td>
<td></td>
<td></td>
</tr>
<tr>
<td>tAUC (mg/dL x 6 hr)</td>
<td>831.4 ± 347.5</td>
<td>752.5 ± 263.9</td>
</tr>
<tr>
<td>iAUC (mg/dL x 6 hr)</td>
<td>201.1 ± 136.1</td>
<td>186.5 ± 121.8</td>
</tr>
<tr>
<td>Peak (mg/dL)</td>
<td>162.5 ± 80.1</td>
<td>133.6 ± 45.9</td>
</tr>
<tr>
<td>Glucose</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Log10 tAUC (mg/dL x 6 hr)</td>
<td>16.5 ± 0.20</td>
<td>16.4 ± 0.20</td>
</tr>
<tr>
<td>Log10 iAUC (mg/dL x 6 hr)</td>
<td>-0.01 ± 0.30</td>
<td>0.03 ± 0.20</td>
</tr>
<tr>
<td>Log10 Peak (mg/dL)</td>
<td>2.16 ± 0.07</td>
<td>2.11 ± 0.04</td>
</tr>
<tr>
<td>Metabolic Load Index</td>
<td></td>
<td></td>
</tr>
<tr>
<td>tAUC (mg/dL x 6 hr)</td>
<td>1499.0 ± 359.4</td>
<td>1415.0 ± 270.0</td>
</tr>
<tr>
<td>iAUC (mg/dL x 6 hr)</td>
<td>164.9 ± 162.2</td>
<td>163.8 ± 153.1</td>
</tr>
<tr>
<td>Peak (mg/dL)</td>
<td>283.4 ± 72.8</td>
<td>271.8 ± 62.8</td>
</tr>
<tr>
<td>Total Cholesterol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>tAUC (mg/dL x 6 hr)</td>
<td>851.7 ± 102.5</td>
<td>862.9 ± 89.9</td>
</tr>
<tr>
<td>iAUC (mg/dL x 6 hr)</td>
<td>82.3 ± 231.7</td>
<td>-44.8 ± 76.4</td>
</tr>
<tr>
<td>Peak (mg/dL)</td>
<td>138.8 ± 31.1</td>
<td>128.9 ± 19.4</td>
</tr>
<tr>
<td>LDL-Cholesterol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Log10 tAUC (mg/dL x 6 hr)</td>
<td>10.6 ± 1.0</td>
<td>10.9 ± 0.6</td>
</tr>
<tr>
<td>Log10 iAUC (mg/dL x 6 hr)</td>
<td>0.7 ± 2.8</td>
<td>-0.4 ± 0.3</td>
</tr>
<tr>
<td>Log10 Peak (mg/dL)</td>
<td>1.9 ± 0.0</td>
<td>1.9 ± 0.1</td>
</tr>
<tr>
<td>HDL-Cholesterol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>tAUC (mg/dL x 6 hr)</td>
<td>306.6 ± 77.3</td>
<td>309.2 ± 74.1</td>
</tr>
<tr>
<td>iAUC (mg/dL x 6 hr)</td>
<td>-16.9 ± 25.9</td>
<td>-16.8 ± 33.9</td>
</tr>
<tr>
<td>Peak (mg/dL)</td>
<td>56.9 ± 14.5</td>
<td>57.3 ± 14.6</td>
</tr>
</tbody>
</table>

There were no significant differences in tAUC, iAUC and peak responses for any of the metabolic outcomes, *p < 0.05.
Figure 1. Experimental design in the EX and NE condition. The acute bout of exercise was performed 12 hours prior to the HFHC, and the same protocol was replicated in both conditions. Preprandial measurements were taken before the HFHC meal, and then hourly for blood lipids (L) and glucose (G). G was also assessed at 30, 90 and 120 minutes postprandially.
Figure 2. TRG responses across time in OA (black lines) and YA (grey lines) over postprandial period in the NE and EX conditions. There was a greater TRG responses in OA compared to YA (Y; p < 0.05). However, there was a lower TRG in the exercise condition compared to the non-exercise condition, specifically in OA-NE compared to YA-EX (*; p < 0.05).
Figure 3. Glucose responses across time in OA (black lines) and YA (grey lines) over the postprandial period in the NE and EX conditions. There was an attenuation of postprandial glucose post-exercise in the OA and YA (*; p < 0.05).
Figure 4. Metabolic Load Index in OA (black lines) and YA (grey lines) over the postprandial period in the NE and EX conditions. There was a greater MLI response in OA compared to YA (¥; p < 0.05). There was lower metabolic load index after exercise, driven by a lower response in YA-EX compared to OA-EX (*; p < 0.05).
Figure 5A. Cholesterol responses across time in OA (black lines) and YA (grey lines) over the postprandial period in the NE and EX conditions. TC was lower in the EX compared to the NE condition (*; p < 0.05). There was also greater TRG in the OA compared to the YA (¥; p < 0.05). LDL (5B) was significantly higher to the OA compared to the YA (¥; p < 0.05). There was also a significant difference by condition in exercise compared to non-exercise (*; p < 0.05). HDL (5C) was significantly higher to the OA compared to the YA (¥; p < 0.05). There was also a significant difference by condition in exercise compared to non-exercise (*; p < 0.05).