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

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Abstract

A single high-fat, high-carbohydrate meal (HFHC) results in elevated postprandial glucose (GLU), triglycerides (TAG) and metabolic load index (MLI; TAG (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. TAG 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 TAG, GLU and MLI post-HFHC independent of age.

Key words: **Ageing: physical activity: TAG: postprandial lipaemia: postprandial glycaemia**

Older adults (OA) have increased risk of developing life style-related diseases such as CVD and type 2 diabetes and hyperlipe-daemia, with diagnosed CVD in over 70 % of individuals between the ages of 60 and 79 years[1]. While several factors in OA influence disease risk, dietary intake including energy-dense, nutrient-poor foods that are high in saturated fats and sugar is thought to promote metabolic and vascular impairment. Indeed, even a single high-fat, high-carbohydrate (HFHC) meal that raises postprandial lipaemia (PPL, e.g. TAG) 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 postprandial 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 postprandial responses in OA[9]. This is clinically problematic since OA have been reported to have higher PPL and PPG with a HFHC meal compared with a younger group, even when physically active[14]. Therefore, we sought to examine the PPL, PPG and MLI in young adults (YA) and OA 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 with 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 twelve YA (23·3 (sd 3·9) years, n 5 M/7 F) and twelve OA (67·7 (sd 6·0) years, n 8 M/4 F). All participants were free of CVD, metabolic and renal diseases as assessed by the 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 and the International Physical Activity Questionnaire to determine chronic PA level in MET-min/week. All of the procedures were approved by the Institutional Review Board at James Madison University (protocol no. 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 randomised between: HFHC with no exercise (NE) or an acute bout of exercise (EX) performed 12 h prior to a HFHC (Fig. 1), with condition separated by at least 7 d. 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 05.00 and 09.00 hours 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). Body mass was assessed using a standard physician's scale (Dymo Pelouze model 4040, Newell Brands), and in turn, BMI was calculated. Subjects underwent a dual-energy x-ray absorptiometry scan (GE Lunar iDXA, Fairfield) to measure lean body mass and body fat. Subjects then sat up and rested for 5 min to assess brachial artery blood pressure using an automatic sphygmomanometer (ProBP 3400 Welch Allyn). Two measurements were taken and averaged as detailed in the American College of Sports Medicine 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), and values were averaged for analysis.

Incremental exercise test

The incremental exercise test was performed on a cycle ergometer (VIAsprint 150P) to determine VO_2peak. Metabolic and ventilatory data were recorded through the entire test, and 30-s averages were used for analyses (Vmax Encore, Vyaire Medical). The protocol began with a 5-min warm-up at a self-selected cadence of >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 30 min. The starting output for the test and increment by which output was increased every minute was determined by the warm-up workload (online Supplementary Table 1). Heart rate (Polar Lake Success) and rating of perceived exertion were recorded in the last 10–20 s of each stage. Subjects were verbally encouraged by investigators throughout the entire test until they reached volitional fatigue or inability to maintain >50 rpm.

Pre-prandial exercise bout

In the EX trial, the acute exercise bout was performed 12 h prior to the HFHC challenge on a cycle ergometer and corresponded to 60–70 % of their heart rate peak. Subjects were required to exercise until they achieved a total energy expenditure of 75 % of the energy content 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 h for OA 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:

$$\frac{mass\ in\ kg * 12.5\ kcals * 0.75}{absolute\ VO2 * 4.825L\ O2/kcal * 0.65}$$

High-fat, high-carbohydrate meal 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 min of rest, two baseline blood pressure measurements were taken with at least 30 s 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) and kept patent with 0·9 % NaCl. The HFHC meal (Marie Callender's Chocolate Satin Pie (57 % fat, 39 % carbohydrate, 4 % protein); Conagra Brands) was standardised to 12 kcal/kg BW and consumed within a 20-min 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 min postprandial until the 2-h mark, after which point draws were performed every hour up to 360 min. Glucose (GLU) was
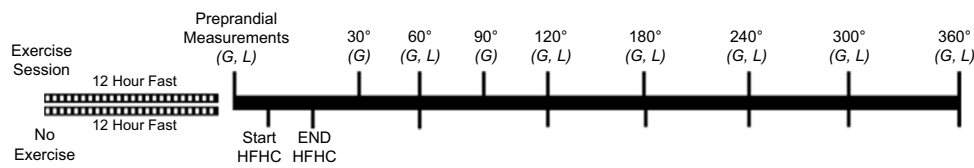
Fig. 1. Experimental design in the exercise (EX) and no exercise (NE) condition. The acute bout of exercise was performed 12 h prior to the HFHC, and the same protocol was replicated in both conditions. Pre-prandial measurements were taken before the HFHC meal, and then hourly for blood lipids (L) and glucose (G). G was also assessed at 30, 90 and 120 min postprandially.

assessed at baseline, 30, 60, 90, 120, 240 and 360 min, and TAG, total cholesterol (TC) and LDL and HDL were assessed at baseline and hourly following the HFHC meal to 360 min. LDL was calculated using the Friedewald equation.

Statistical analyses

An *a priori* sample size calculation was performed based on the previous research[17] with the difference in postprandial TAG change between OA and YA as the main outcome. The difference in postprandial TAG change between OA and YA was 68 (SD 34·8) mg/dl (Cohen's $d = 0.96$). With a power of 0·80 and a two-sided $\alpha = 0.05$, eight subjects were required per group to observe differences in TAG between the OA and YA. We recruited twelve participants to each group to ensure statistical power. Data were analysed using IBM SPSS Statistics version 26.0 (IBM Corp.). MLI was calculated as a summation of the TAG + GLU responses at each hourly measurement as previously described[18]. All data were analysed for skewness, kurtosis and normality using the Shapiro–Wilk test. Glucose and LDL were not normally distributed and were log-transformed. A repeated-measure 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 utilised. *Post hoc* analyses were performed when there were significant effects with a Bonferroni correction. Secondary analyses for AUC, incremental AUC (iAUC) and peak values were calculated for TAG and GLU using GraphPad Prism (GraphPad Software, Inc.). 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, OA were taller compared with YA ($P = 0.02$, Table 1). The kilocalorie content of the HFHC meal consumed by YA was not statistically different from the OA (856·8 (SD 207·1) kcals and 968·8 (SD 180·9) kcals, respectively, $P = 0.17$).

Maximal and submaximal exercise characteristics

While relative VO$_2$peak was significantly lower in OA compared with YA ($P = 0.005$) due to body mass, absolute VO$_2$ 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 heart

Table 1. Subject demographics
(Mean values and standard deviation)

	YA (*n* 12/5 M, 7 F)		OA (*n* 12/8 M, 4 F)	
	Mean	SD	Mean	SD
Age (years)	23·4	3·8	67·4	5·0*
Height (cm)	167·5	8·1	176·0	8·9*
Weight (kg)	71·4	17	80·7	15·1
BMI (kg/m^2)	25·3	5·0	25·8	3·1
Total body fat (%)	28·2	8·7	33·1	6·5
Android body fat (%)	30·1	13·1	40·3	9·7

* Significantly different from YA ($P < 0.05$).

Table 2. Exercise data
(Mean values and standard deviation)

	YA (*n* 12/5 M, 7 F)		OA (*n* 12/8 M, 4 F)	
	Mean	SD	Mean	SD
VO$_2$peak (l/min)	2·3	0·4	2·3	0·8
VO$_2$peak (ml/kg per min)	33·4	5·3	28·3	6·7*
Peak power (Watts)	199·2	42·1	198·3	67·9
Peak heart rate (Bpm)	187·9	11·7	156·6	8·5*
Exercise duration (min)	88·1	15·2	105·6	25·7
Exercise bout expenditure (kcals)	642·6	155·4	726·7	135·6

* Significantly different from YA ($P < 0.05$).

rate peak was significantly lower in OA *v.* YA ($P < 0.01$). 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

OA had significantly higher TC (age effect: $P < 0.01$) and LDL-cholesterol (age effect: $P < 0.01$) compared with YA (Table 3). EX condition did not affect fasting GLU, TAG and cholesterol in YA or OA 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 hyperlipidaemia[20].

Postprandial metabolic responses

While TAG increased from baseline to 6 h (time effect; $P < 0.01$) among all groups, TAG were significantly greater in OA *v.* YA (age effect; $P < 0.01$). There was a significant effect of exercise on TAG in OA and YA (Fig. 2). However, there was no difference between age and effect of exercise on TAG tAUC ($P = 0.294$), iAUC ($P = 0.487$) or peak TAG response ($P = 0.085$).

Table 3. Metabolic outcomes measured in fasting participants
(Mean values and standard deviation)

| | | YA (*n* 12/5 M, 7 F) | | | | OA (*n* 12/8 M, 4 F) | | | |
| | | NE | | EX | | NE | | EX | |
Fasting values	Optimal values (mg/dl)	Mean	SD	Mean	SD	Mean	SD	Mean	SD
TAG (mg/dl)	<150	107·3	38·8	98·3	29·3	117·5	30·6	109·8	22·5
Glucose (mg/dl)	<100	115·0	7·9	110·3	10·3	118·5	18·7	112·7	16·0
MLI (mg/dl)	N/A	222·3	40·7	208·6	30·5	236·0	38·4	222·4	29·5
TC (mg/dl)	<200	139·9	17·2	151·3	21·8	184·3	38·1*	177·7	40·7*
LDL-cholesterol (mg/dl)	100–129	63·8	11·6	77·4	18·4	101·8	29·5*	97·6	33·9*
HDL-cholesterol (mg/dl)	40–60	53·9	16·1	54·3	14·7	58·9	16·6	57·8	17·7

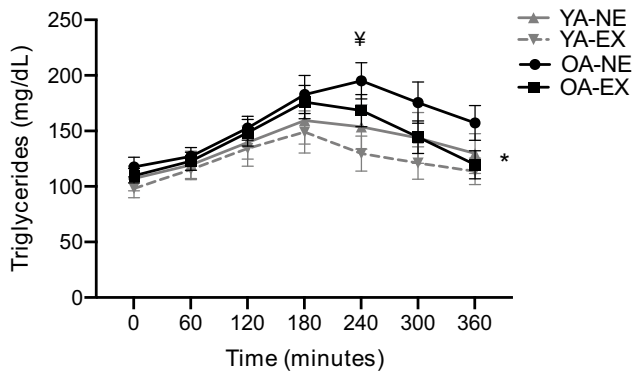* Significantly different from YA (*P* < 0·05).

Fig. 2. TAG responses across time in older adults (OA) (black lines) and younger adults (YA) (grey lines) over postprandial period in the no exercise (NE) and exercise (EX) conditions. There was a greater TAG responses in OA compared with YA (¥; *P* < 0·05). However, there was a lower TAG in the exercise condition compared with the non-exercise condition, specifically in OA-NE compared with YA-EX (*; *P* < 0·05).
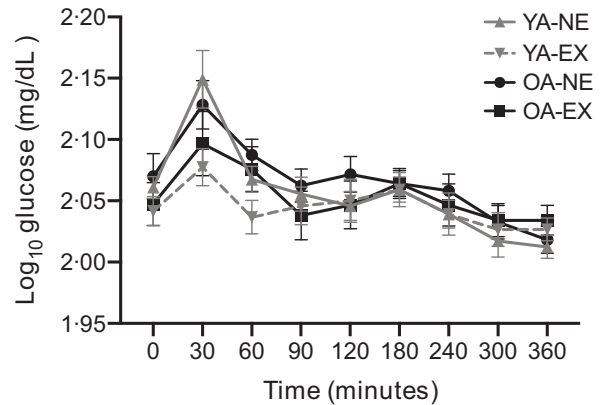

Fig. 3. Glucose responses across time in older adults (OA) (black lines) and younger adults (YA) (grey lines) over the postprandial period in the no exercise (NE) and exercise (EX) conditions. There was an attenuation of postprandial glucose post-exercise in the OA and YA (*; *P* < 0·05).

There was a significant increase in GLU shortly after the meal, but concentrations returned to baseline within the assessed postprandial period (*P* < 0·001, Fig. 3). Exercise lowered the GLU response in both YA and OA compared with the non-exercise control (*P* = 0·027). In fact, there was significantly lower GLU response in the OA-EX condition compared with the YA-NE condition (*P* = 0·014). However, there was no difference between age and 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 OA-NE compared with the YA-NE (*P* = 0·008) and YA-EX (*P* < 0·001), driven by greater postprandial TAG (Fig. 4). The OA-EX also had a significantly greater MLI compared with the YA-EX (*P* = 0·049). However, MLI was reduced with acute exercise in OA-EX and YA-EX compared with 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 and 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 h (*P* = 0·836); however, TC was significantly lower by
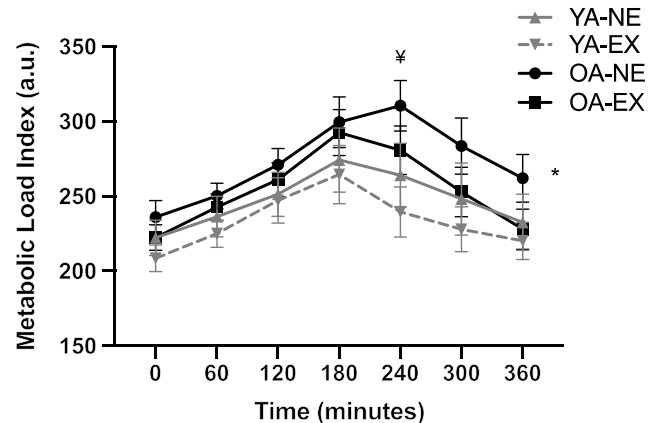

Fig. 4. Metabolic Load Index in older adults (OA) (black lines) and younger adults (YA) (grey lines) over the postprandial period in the no exercise (NE) and exercise (EX) conditions. There was a greater MLI response in OA compared to YA (¥; *P* < 0·05). There was lower metabolic load index after exercise, driven by a lower response in YA-EX compared with OA-EX (*; *P* < 0·05).

condition (*P* < 0·001, Fig. 5(a)). TC was higher in OA *v.* YA (age effects; *P* < 0·001). In fact, there was also a significantly higher postprandial TC response in the OA-NE condition

Table 4. Postprandial metabolic outcomes
(Mean values and standard deviation)

	YA (n 12/5 M, 7 F)				OA (n 12/8 M, 4 F)			
	NE		EX		NE		EX	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
TAG								
tAUC (mg/dl × 6 h)	831·4	347·5	752·5	263·9	970·6	260·0	874·5	228·5
iAUC (mg/dl × 6 h)	201·1	136·1	186·5	121·8	269·6	151·5	226·9	139·6
Peak (mg/dl)	162·5	80·1	133·6	45·9	199·6	63·1	180·2	59·6
Glucose								
Log10 tAUC (mg/dl × 6 h)	16·5	0·20	16·4	0·20	16·6	0·20	16·5	0·20
Log10 iAUC (mg/dl × 6 h)	−0·01	0·30	0·03	0·20	−0·01	0·50	0·06	0·20
Log10 Peak (mg/dl)	2·16	0·07	2·11	0·04	2·16	0·06	2·13	0·02
Metabolic load index								
tAUC (mg/dl × 6 h)	1499·0	359·4	1415·0	270·0	1664·0	253·6	1556·0	234·5
iAUC (mg/dl × 6 h)	164·9	162·2	163·8	153·1	249·1	152·6	165·6	47·8
Peak (mg/dl)	283·4	72·8	271·8	62·8	321·9	56·8	301·8	35·8
Total cholesterol								
tAUC (mg/dl × 6 h)	851·7	102·5	862·9	89·9	1051·0	204·6	1022·0	207·8
iAUC (mg/dl × 6 h)	82·3	231·7	−44·8	76·4	−54·7	70·9	−44·5	129·9
Peak (mg/dl)	138·8	31·1	128·9	19·4	173·3	39·5	164·3	42·9
LDL-cholesterol								
Log10 tAUC (mg/dl × 6 h)	10·6	1·0	10·9	0·6	11·4	0·9	11·5	0·8
Log10 iAUC (mg/dl × 6 h)	0·7	2·8	−0·4	0·3	−0·5	0·3	−0·4	0·5
Log10 Peak (mg/dl)	1·9	0·0	1·9	0·1	2·0	0·0	2·0	0·1
HDL-cholesterol								
tAUC (mg/dl × 6 h)	306·6	77·3	309·2	74·1	349·0	115·5	333·8	103·2
iAUC (mg/dl × 6 h)	−16·9	25·9	−16·8	33·9	−4·5	34·4	−13·3	32·4
Peak (mg/dl)	56·9	14·5	57·3	14·6	64·3	21·3	61·8	20·3

iAUC, incremental AUC.
There were no significant differences in tAUC, iAUC and peak responses for any of the metabolic outcomes.

compared with the YA-NE and YA-EX conditions ($P = 0·020$ and $P = 0·031$, respectively). For LDL-cholesterol, there was no change across time points from baseline to 6 h ($P = 0·205$, Fig. 5(b)); however, there was a significant effect of condition where OA-EX and YA-EX had lower LDL compared with OA-NE and YA-NE ($P < 0·001$). LDL-cholesterol was also higher in OA compared with YA. Peak LDL tended to be higher in OA compared with YA independent of exercise ($P = 0·052$). Similarly, HDL-cholesterol was unchanged across time ($P = 0·943$, Fig. 5(c)) but was significant by condition ($P = 0·016$). HDL was also significantly higher in the OA compared with YA.

Discussion

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

Postprandial lipid responses with and without acute exercise

Our study supports previous findings that PPL (namely TAG) are elevated in OA compared with YA. However, we believe that the fitness level (i.e. absolute VO$_2$peak) and health status of the OA that composed our study population may have been why postprandial TAG did not exhibit a larger attenuation post-exercise. The findings that even healthy, fit OA have elevated postprandial TAG are important because postprandial TAG may better predict the risk of cardiac event or CVD development than fasting lipids[21,22]. Therefore, identifying methods to attenuate this larger TAG response is critical to minimise deleterious health outcomes.

Interestingly, the impact of acute exercise on postprandial TAG levels in YA has mixed findings, with true-to-life exercise durations and intensities between 30 min and an hour not consistently showing reductions in postprandial TAG[7,8,23]. Many factors such as exercise timing[10,12], energetic 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 lipaemia postmeal. While there is conflicting literature on the effect of exercise timing, duration and energy expenditure relative to the test meal in healthy YA, more consistent findings report that acute exercise reduces adverse metabolic outcomes in untrained, inactive or diseased populations. Specifically, 90 min of moderate-intensity
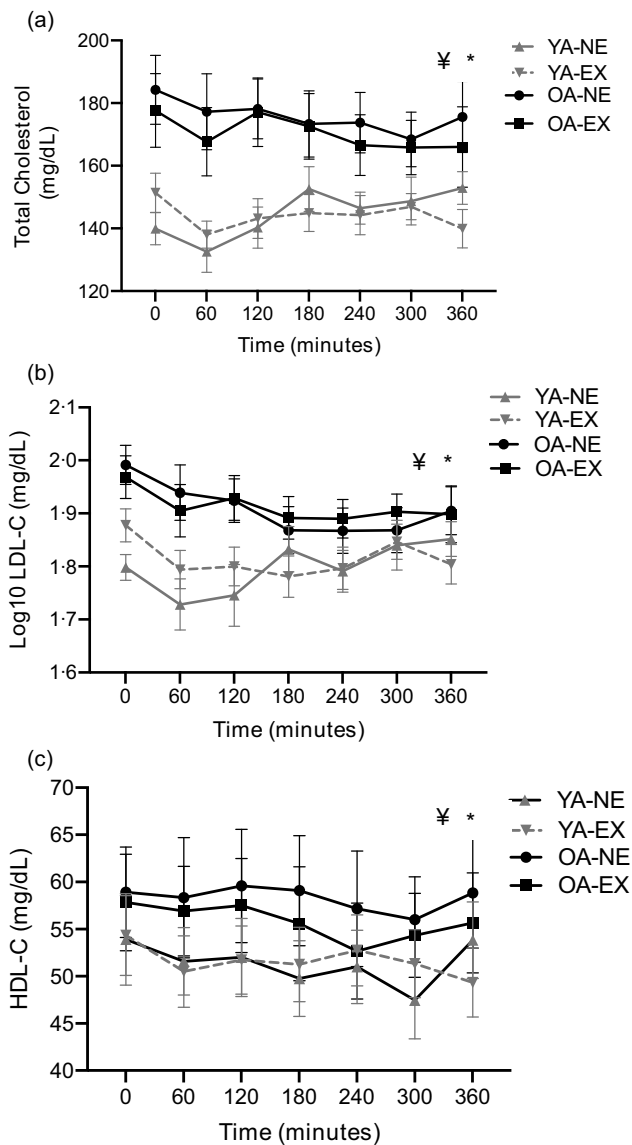
Fig. 5. (a) Cholesterol responses across time in older adults (OA) (black lines) and younger adults (YA) (grey lines) over the postprandial period in the no exercise (NE) and exercise (EX) conditions. TC was lower in the EX compared with the NE condition (*; $P < 0.05$). There was also greater TAG in the OA compared with the YA (¥; $P < 0.05$). LDL (b) was significantly higher to the OA compared with the YA (¥; $P < 0.05$). There was also a significant difference by condition in exercise compared with non-exercise (*; $P < 0.05$). HDL (c) was significantly higher to the OA compared with the YA (¥; $P < 0.05$). There was also a significant difference by condition in EX compared with NE (*; $P < 0.05$).

males[30], there are no studies to our knowledge examining metabolic responses (i.e. TAG, GLU and MLI) to a HFHC meal after acute pre-prandial exercise in inactive OA specifically. Additional work is warranted to understand the optimal dose of an acute pre-prandial exercise bout in untrained or inactive OA.

To our knowledge, this is the first study to examine the impact of acute exercise in fit OA compared with 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 TAG in middle-aged adults and OA (mean age: 58 years)[9]. Specifically, 90 min of exercise 12 h prior to a HFM was not sufficient in attenuating any metabolic outcomes (TC, LDL, HDL, TAG 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 OA in both studies exhibited larger postprandial TAG compared with a control group, a single exercise bout was able to lower TAG in the present study. We did not design this study to determine the mechanism by which exercise lowered TAG, but we suggest that exercise may have raised skeletal muscle LPL activity[31]. LPL activity is the rate-limiting step for TAG clearance in the circulation and provides a direct mechanism to regulate TAG 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 TAG-rich lipoproteins from the liver[5], which has been previously found to account for up to 70 % of the TAG attenuation[33].

In ageing humans, there is reduced TAG 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 TAG[37]. While several factors may explain the results of the present study, lowered LPL activity specifically in OA could explain why OA had an elevated postprandial TAG response compared with YA due to reduced fatty acid utilisation and TAG lipolysis. In addition, it is possible that with lowered LPL activity and lower TAG clearance, OA 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 TAG, 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-cholesterol. The findings of the present study also align with the work by Bittel and colleagues who reported that a single bout of resistance training reduced lipaemic 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 TAG-rich lipoprotein-TAG. In the present study, we have also reported no changes in LDL-cholesterol iAUC, yet reduced LDL-cholesterol in the EX + HFHC meal condition. Previous examinations have

pre-prandial exercise attenuated postprandial TAG in untrained adults[29] and adults diagnosed with hyperlipidaemia or other metabolic diseases[5]. In the present study, an acute bout of pre-prandial exercise expending 75 % of the energy content consumed in a HFHC meal was sufficient in reducing postprandial TAG and glucose in healthy, active young and older individuals. This prescription though may not be possible for untrained or inactive OA. 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

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 TAG, glucose, TC and LDL-cholesterol with a true-to-life exercise bout averaging about 643 kcal expended in young adults and about 730 kcal expended in OA.

Glucose responses in older adults and younger adults and the impact of acute exercise

An interesting finding in the present study was the lack of any significant differences in glucose levels between YA and OA. 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 YA and OA may be due to their similar VO$_2$peak 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 OA and YA. 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 YA and OA. 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 YA *v.* OA.

Taken together, the glucose and other metabolic results in the present study align with the recent work by Ramirez-Velez *et al.*, who investigated the impact of exercise training on postprandial lipid and glucose responses[45]. While the authors utilised various exercise training protocols in a group of inactive YA approximately 30 years of age, they reported that postprandial glucose was attenuated after an exercise training programme, whereas there were trends present for the attenuation of postprandial TAG. Considering the group utilised in the present study was fit OA compared with a YA 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 MLI. Thus, there was a lower metabolic load post-meal experienced after exercise in the YA compared with the OA.

Experimental considerations

There are several limitations in the present study that are important to consider. First, since the OA in the study were not active and also not on lipid-lowering medications, this demographic does not represent the majority of OA in the USA. As a result, the subjects included in this study may limit the generalisability of the findings and applying these results to OA 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 OA have not been previously investigated. Ageing 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 YA 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. Utilising 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 (online 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 lipaemia[25]. Energetic replacement after acute night-time exercise may be more typical of human behaviour, though may interfere with the mechanisms that are responsible for the effect of an exercise bout on PPL and PPG.

Conclusion

While OA have greater postprandial lipaemia compared with YA who are similar in fitness levels, an acute bout of exercise is effective at reducing postprandial lipaemia in OA and YA. While the OA and YA 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 OA and YA. However, since there was not a greater attenuation in TAG and glucose in OA when compared with YA, further work should investigate mechanisms underpinning these results to maximise the use of exercise as a therapy to lower type 2 diabetes, dyslipidaemia and overall CVD risk.

British Journal of Nutrition

Supplementary material

For supplementary material referred to in this article, please visit https://doi.org/10.1017/S0007114521002208

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