Published in final edited form as:

Metabolism. 2013 February ; 62(2): 244–254. doi:10.1016/j.metabol.2012.08.003.

The effects of sex, metabolic syndrome and exercise on postprandial lipemia

Kimberly A. Cox-York^{1,3}, Teresa A. Sharp^{1,4}, Sarah A. Stotz¹, Daniel H. Bessesen^{1,2}, Michael J. Pagliassotti³, and Tracy J. Horton¹

¹University of Colorado Denver, Aurora CO

²Denver Health Medical Center, Denver CO

³Colorado State University, Fort Collins CO

⁴University of Northern Colorado, Greeley CO

Abstract

Objective—Exercise has been suggested to have cardio protective benefits due to a lowering of postprandial triglycerides (PPTG). We hypothesized that a morning exercise bout would significantly lower PPTG measured over a full day, in response to moderate fat meals (35% energy) in men more so than women, and in metabolic syndrome (MetS) relative to normal weight (NW) individuals.

Materials/Methods—Participants completed two randomized study days; one control and one exercise day (60 min of morning exercise, 60% $\text{VO}_{2\text{peak}}$). Meals were consumed at breakfast, lunch and dinner with the energy expended during exercise replaced on the active day. The areas (AUC) and incremental areas (IAUC) under the curve were calculated for total triglycerides, total cholesterol and other metabolites.

Results—Exercise did not significantly change the PPTG AUC & IAUC overall, nor within, or between, each sex or group (NW and MetS). Exercise induced a 30% decrease in total cholesterol IAUC ($p = 0.003$) in NW subjects. Overall, women had a lower IAUC for PPTG compared to men ($p = 0.037$), with the greatest difference between MetS women and MetS men, due to a sustained drop in TG after lunch in the women. This suggests that PP, rather than fasting, lipid analyses may be particularly important when evaluating sex differences in metabolic risk.

Conclusions—With energy replacement, moderate morning exercise did not result in a significant decrease in PPTG excursions. Exercise did elicit a significant decrease in PP cholesterol levels in NW subjects, suggesting a potential mechanism for the cardio protective effects of exercise.

© 2012 Elsevier Inc. All rights reserved.

Address reprint requests to Kimberly Cox-York, Colorado State University, Department of Food Science and Human Nutrition, 234 Gifford Building, Fort Collins, CO 80523-1571, Phone: 970.397.7148, Fax: 970.491.3875, Kimberly.Cox-York@Colostate.edu.

Authors have no conflicts of interest or financial disclosures.

AUTHOR CONTRIBUTIONS

Dr. Cox-York: manuscript writing, data collection, analysis and interpretation. Dr. Sharp: data collection and analysis. Sarah Stotz: data collection. Dr. Pagliassotti: technical assistance and mentoring. Dr. Bessesen data collection and interpretation, manuscript writing, mentoring. As the primary investigator, Dr. Horton: study design, funding, provided mentoring, and data collection, analysis and interpretation.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Keywords

obesity; triglyceride; cardiovascular disease; sex-based differences; cholesterol

1. Introduction

Cardiovascular disease (CVD) is the leading cause of death in the United States and most developed countries, and dyslipidemia is a major treatable risk factor for CVD. While it is standard practice to evaluate serum lipid levels in the fasted state, the majority of individuals spend most of each day in a fed or postprandial (PP) state. It has been suggested that 40% of all patients with premature coronary artery disease have normal fasting plasma lipids, but impaired clearance of PP lipoproteins a response that appears to be exaggerated in obese subjects.

The association of elevated PP triglycerides (PPTG) with increased risk of CVD, has led to a search for interventions that might minimize the PPTG response, including exercise. Participation in regular exercise has been shown to reduce CVD risk in a wide range of individuals, and there is data to suggest that a single, acute bout of exercise can reduce PPTG levels. Although certain studies report a significant decrease in PPTG with prior exercise others report varying effects on PPTG response based on exercise intensity, duration, and time relative to meal ingestion.

Most studies to date have evaluated PPTG following an acute bout of moderate exercise, performed 12–14 hours before a single high fat test meal. The fat content of these test meals has not been typical of the intake in the general population with both the absolute (grams) and relative (percentage of total kilocalories) fat intake being much greater than would be normally consumed in a single meal. Moreover, as there is evidence of a “second meal” effect on cumulative post prandial lipemia (PPL) for a day, it would be prudent to study PPTG in response to multiple meals. Indeed, while TG were significantly increased in men with >3 components of the metabolic syndrome at baseline and after a single meal, the separation between those with 1–3 components was not obvious until after consumption of a second meal.

Habitual diet, physical activity patterns, sex and estrogen status (i.e. menstrual cycle phase & menopause) are also critical variables that have not always been controlled in previous studies of PPL. Healthy, premenopausal women are generally believed to have a lower PPTG response, and a lower incidence of CVD relative to age-matched men and postmenopausal women. Whether or not exercise lowers PPTGs to the same extent in women as compared to men has not been directly tested within a single study.

Individuals characterized as having metabolic syndrome (MetS) have been reported to have an exaggerated PPTG response relative to normal weight subjects (reviewed in). This is relevant as individuals with MetS have a significantly increased risk of mortality from CVD, as well as increased incidences of CVD, coronary heart disease and stroke. Elevated PPL could contribute to the increased CVD risk of MetS individuals, therefore, evaluating the ability of interventions such as exercise, to lower PPL in these high risk individuals is relevant. Notably, women with MetS have never been tested with regard to the effects of exercise on PP lipemia.

The main aim of this study, was to address the effect of exercise on PPL under conditions that more closely resemble normal daily living, that is, after a single bout of moderate morning exercise, followed by the consumption of 3 moderate fat meals over an entire day.

This was evaluated in normal weight and MetS, men and pre-menopausal women to address the effects of sex and/or CVD risk status on the PPTG response.

The primary hypotheses were that a) exercise would significantly decrease PPTG AUC and IAUC in all groups b) women would have a lower PPTG response than men, and c) subjects with MetS would have a greater PPTG response than NW subjects and that a moderate exercise bout would 'normalize' the MetS PPTG response to a level similar to that seen in NW subjects on a rest day.

2. Subjects and methods

The study was approved by the Colorado Multiple Institution Review Board, and subjects were studied at the Clinical Translational Research Center (CTRC) at the University of Colorado Denver. Written informed consent was obtained from all subjects before participation in the study.

2.1. Study population

Two subject groups were recruited (Table 1) including NW healthy subjects (13 men and 13 women) and moderately obese subjects with MetS (9 men and 9 women) from the University of Colorado and surrounding area. Inclusion criteria for both groups were 20–45 years old, non-smoking, stable body weight (± 3 kg over 6 months), and less than 2h/wk moderate physical activity. Women were eumenorrheic and not using steroidal contraceptives, nor were they pregnant or lactating. Subjects were excluded for a thyroid stimulating hormone of <0.5 or >5.0 μ U/ml, anemia (hemoglobin < 13.5 g/dl women or < 14.5 g/dl men), diabetes, past or present history of CVD, and any other significant hormonal or metabolic abnormality. An apoE genotype of E2/E2 was also grounds for exclusion. NW subjects were also excluded for TG >200 mg/dl, a blood glucose <65 or > 110 mg/dl, insulin > 20 μ U/ml, total cholesterol >200 mg/dl and LDL cholesterol >160 mg/dl.

Subjects with MetS were each required to have at least 3 of the following 5 characteristics: fasting glucose, 100–140 mg/dl; fasting TG, > 150 mg/dl; waist circumference > 102 cm for men and >88 cm for women, blood pressure of >130 / >85 mmHg and HDL-C, < 40 mg/dl men and < 50 mg/dl women (National Cholesterol Education Program Adult Treatment Panel III criteria). MetS subjects were required to have a BMI ≥ 32 kg/m² to minimize the likelihood of other co-morbidities and body weight and body composition differences between groups, and within subjects in the MetS group.

2.2. Screening assessments

Subjects were screened for study inclusion with a health and physical examination, including blood chemistry and a fasting lipid profile, before which subjects were asked to refrain from alcohol and exercise and to consume a low carbohydrate diet for 48 hrs. An attempt was made to recruit NW men and women with similar fasting TGs. Women had samples drawn in the follicular phase of their menstrual cycle.

A 4-day dietary record (3 weekdays, 1 weekend day) was also completed. Subjects were excluded if the percentage of total EI derived from nutrients was within 33% of the test meal nutrient composition, to avoid including individuals for whom the period of pre-study dietary control, and test meals, would be a drastic change from their habitual diet.

2.3. Preliminary assessments

If subjects qualified for the study, they completed the following assessments prior to the main study days.

Resting metabolic rate—Indirect calorimetry (Sensormedics 2900 metabolic cart system, Sensormedics, Yorba Linda, CA) in the morning, following a 10–12 hr fast as previously described. Results were used to calculate EI for the controlled diet phase of each study as well as the meals consumed on each study day.

Peak oxygen uptake test (VO_{2peak})—Subjects were studied in the morning after an overnight fast having abstained from any planned exercise the day prior. Peak oxygen uptake was determined using a graded treadmill (Quinton, Cardiac Science Corp, Bothell WA) test using the Bruce Protocol (NW subjects) or modified Bruce protocol (MetS subjects). Heart rate was continually monitored (12-lead ECG) with blood pressure measured in the final minute of each work load. Respiratory gas exchange was monitored continuously during the test via indirect calorimetry. The test was terminated when the subject reached volitional exhaustion or if there was any clinical abnormality noted in the ECG or blood pressure. Peak oxygen uptake was calculated from the final minute of oxygen consumption prior to the test termination.

Body composition and anthropometry—Total body, abdominal (ABFAT) and leg fat were measured with dual energy X-ray absorptiometry (DXA) and waist and hip circumferences were measured as previously described.

Visceral adiposity—Visceral fat (VFAT) and subcutaneous fat (SCFAT) were determined by computed tomography (CT) from a single transverse image centered at the L4–L5 disk.

Menstrual cycle phase in eumenorrheic women—Only women with regular menstrual cycles over the preceding 6 months (cycle length 22–36 days) were recruited. Information on cycle length was collected at screening and thereafter. Studies were conducted in the follicular phase between days 5–11 of a typical 28 day cycle. For longer/shorter cycles, study days were adjusted accordingly.

2.4. Preparatory Dietary and Exercise Control

Subjects consumed a controlled diet for 5 days prior to each study day and no other food was permitted. The diet was prepared by the adult CTSC and the composition was identical to that of the meals consumed on the study days. Daily EI on the controlled diet was calculated by multiplying the subjects' RMR by an activity factor that reflected the subjects' reported activity level: 1.50 x RMR for a no exercise or 1.6–1.7 for a moderate exercise day, respectively. Subjects were requested not to perform planned exercise, other than activities of daily living, for 60 hrs prior to study days.

2.5. Experimental Protocol

Subjects were studied twice in random order. One study included a morning bout of exercise and the other did not, with the following details.

Study Timeline(Figure 1)—Subjects were admitted to the CTSC the evening prior to each study day, given their evening meal, and spent the night on the CTSC. They were fasted from 22:00, with only water or non-caloric/non-caffeinated beverages permitted. The following morning an intravenous catheter was placed in an antecubital vein at 6:30 a.m.. At 7:15 a.m. on the exercise day, subjects walked on a treadmill for 60 mins, at 60% VO_{2peak} . Respiratory gas exchange was measured twice for 15–20 minutes during the exercise bout. On the non-exercise day, this same time was spent resting during which two 15–20 minute measurements of respiratory gas exchange were made.

Subjects remained resting in bed on the CTRC during the day except to use the bathroom or stretch their legs briefly. Subjects rested for 30 minutes prior to any blood sampling. At the end of the first study day (~10:30 pm), subjects had the option of staying a second night on the CTRC, or were allowed to leave and asked to return the next morning for a final fasting blood draw at 7:30 a.m. This routine was repeated for their second test day.

Study day meals—Total EI was calculated to meet energy requirements on the corresponding day and was divided as follows; 25%, 35% and 40% of total daily EI at breakfast, lunch and dinner, respectively. The composition of each meal was 34% fat, 15% protein and 51% carbohydrate, and cholesterol intake was 128 mg/1000 kcal. EI was designed to maintain energy balance as follows: rest day, $1.3 \times \text{RMR}$; exercise day, $1.3 \times \text{RMR} + \text{net energy expenditure (EE) during the exercise} + \text{estimated excess post-exercise EE (10\% of net EE in kcal/min} \times 30 \text{ mins)}$. A factor of $1.3 \times \text{RMR}$ was used as subjects spent the majority of the day resting in bed.

Blood sampling and analysis—All biochemical analyses were performed by standard methods in the CTRC Core Laboratory. Estradiol and progesterone were measured at baseline. Total TG, cholesterol, FFA, adiponectin, glucose and insulin were measured at all time points. Total TG concentrations in plasma were corrected for free glycerol concentrations. It was assumed that all acylglycerols detected by the assay were triacylglycerols. HDL-C was measured every 2 hours and fasting LDL-C was calculated by difference (Freidwald equation). The homeostasis model assessment of insulin-insulin resistance (HOMA-IR) was calculated using fasting values from the study day as well as the following day ($\text{Glucose (mg/dl)}/\text{insulin } (\mu\text{U/ml})/405$).

2.6. Statistical analysis

Based on previous studies, it was estimated that 20 subjects (10 male and 10 female) would provide greater than 80% power to detect a) a difference of 15%–30% between the daily post-prandial TG IAUC on the control vs exercise day, for males and females separately and b) a difference between men and women, in the control vs exercise change in TG response, in the order of 20–55%. It was estimated that 40 subjects (10 NW men and 10 NW women and 10 MetS men and 10 MetS women) would provide greater than 80% power to detect a) a difference of between 11%–21% in the daily post-prandial TG IAUC on the control vs exercise day *within* those with or without MetS and b) a difference of between 13–29% in the control vs exercise daily post-prandial TG IAUC in those versus without MetS.

Data were analyzed with SPSS for Windows 16& 17 (SPSS, Chicago, IL) and GraphPad Prism 5 for Windows (GraphPad Software, San Diego, CA). Area under the PP curve (AUC) was calculated for plasma TG measures using the trapezoidal rule and incremental area under the curve (IAUC) was calculated by subtracting the corresponding days' -75 min value from each point used to determine the AUC. AUC and IAUC results were analyzed with univariate ANOVA.

Differences by day, sex and group (NW vs MetS) were examined along with any 2 or 3 way interactions. Significant differences identified by ANOVA were analyzed post-hoc by t-test. Repeated measures ANOVA was used to test for significant differences in the pattern of PP responses for all variables. As expected, there were a number of significant group differences in demographic data (weight, BMI, total fat and fat-free mass). However, only age was significantly correlated with the primary outcome variable of TG AUC and IAUC and other secondary variables. Therefore, age was used as a covariate in all analyses. Data are presented as mean values $\pm$ SEM. Statistical significance was set at $p = 0.05$.

3. Results

3.1. Subject demographics

Table 1 displays subject characteristics. The NW women were significantly older than the NW men ($p=0.04$), whereas no sex difference in age was observed for the MetS group. As a group, the MetS subjects were significantly older than the NW subjects ($p < 0.001$).

MetS subjects had a higher BMI than NW subjects ($p < 0.001$), and NW men had a higher average BMI than NW women ($p = 0.01$), but there was no sex difference in the MetS group. MetS subjects had significantly greater percent body fat ($p<0.05$) than NW subjects, and women had a significantly greater percent body fat than men within each group ($p<0.01$). Analysis of CT data by ANOVA gave a significant sex x group interaction ($p = 0.04$) for VFAT with the MetS men having significantly more VFAT than MetS women ($p = 0.05$). A significant sex x group effect ($p = 0.02$) was also observed for SCFAT, due to MetS women having a significantly greater SCFAT than MetS men ($p = 0.03$). No sex differences existed in SCFAT and VFAT within the NW group. By DEXA, MetS subjects had significantly more trunk and leg fat than NW subjects ($p < 0.02$) and women had more trunk and leg fat than men ($p < 0.001$).

3.2. Fasting plasma values

Fasting data (Table 2) are expressed as the average of the T-75 min time point from each rest and exercise day. Predictably, the MetS group had a significantly poorer fasting metabolic profile compared to the NW group. Women had significantly higher HDL-C levels as compared to their respective male counterparts. Fasting adiponectin was higher in NW women than NW men, but no sex differences were observed within the MetS group. In contrast, MetS women had significantly higher HOMA-IR values than MetS men whereas no sex difference in HOMA-IR was observed within the NW group. There were no differences in total or LDL cholesterol between the sexes or groups.

3.3. Exercise

Relative exercise intensity was equivalent among subjects; on average 60% of VO_2 peak, or 29 ml $\text{O}_2/\text{kg FFM}/\text{min}$. In terms of absolute VO_2 this corresponded to 2010 ± 66 ml/min in NW men, 1351 ± 56 ml/min in NW women, 1835 ± 63 ml/min in MetS men and 1375 ± 72 ml/min in MetS women. Consequently, the energy cost of the exercise was 579, 391, 550 and 412 kcals, respectively; with men having a higher total exercise EE than women ($p < 0.001$) but no difference relative to FFM.

3.4. Energy intake

By design to maintain energy balance, energy intake (kcal/day) was significantly greater on the exercise (E) day than the rest (R) day for all groups ($p < 0.001$): NW women E = 2075 ± 42 , R = 1731 ± 41 ; NW men E = 2776 ± 72 , R = 2221 ± 46 ; MetS women E = 2411 ± 65 , R = 2004 ± 69 ; MetS men E = 2911 ± 99 , R = 2393 ± 84 .

3.5. Postprandial TG response

As depicted in Figure 2, there was no significant effect of exercise on the PPTG response over the day in either the NW or MetS groups, in either sex, nor in the group as a whole. Repeated measures ANOVA revealed a time x group x sex effect for PPTG (Figure 2, $p < 0.001$). A subsequent repeated measures ANOVA of just the MetS group verified a significant time x sex effect ($p = 0.032$) which was not seen in the NW group ($p = 0.28$). When data was expressed as either AUC or IAUC, there was also no significant effect of exercise by sex or group and no interaction. Therefore, AUC and IAUC were averaged for

the exercise and rest days for the following calculations. Collectively, the men exhibited a 34% higher PPTG response than women expressed as IAUC (5.086×10^4 vs 3.358×10^4 mg/dl/840 mins, $p = 0.037$). There was a significant overall effect of group as the MetS group had a 44% higher PPTG AUC ($196,259 \pm 11494$ vs $100,964 \pm 5750$) and a 55% higher IAUC (58768 ± 6473 vs 16487 ± 2582) than the NW group ($p < 0.001$ for both).

3.6. Other PP response measures

Repeated measures ANOVA revealed significant time x day, time x sex, and time x group, effects ($p < 0.001$ for all) for total cholesterol (Figure 3A& B). Exercise resulted in a 30% decrease in total cholesterol IAUC ($p = 0.003$) in the NW group (Figure 3C), but had no effect on cholesterol in MetS subjects.

Borderline significant overall effects were observed for time x day and for time x sex with respect to apoB IAUC ($p = 0.064$ and 0.056 respectively) with no differences in AUC. Total apoB AUC was 8% higher in the MetS vs NW subjects ($p = 0.004$), but IAUC was not different between groups. There was a decrease in apoB over the day with exercise relative to rest, and in men relative to women (repeated measures ANOVA, $p < 0.002$ for both). An estimate of lipoprotein particle size (PS) is the ratio of TG to total apoB, with a higher ratio suggesting a greater PS. Exercise had no effect on PS by sex or by group, therefore data for PS were combined for the two days (Figure 4). Data suggests that relative to NW subjects, MetS subjects as a group had significantly larger TG particles (IAUC $p < 0.001$).

There was no main effect of exercise on the insulin and PP glucose AUC or IAUC's, nor a main effect of sex. The MetS subjects had higher AUC and IAUC's for both insulin and glucose compared to the NW subjects ($p < 0.001$ for both). Repeated measures ANOVA for insulin established a significant time x sex x group effect ($p = 0.001$), due to a greater insulin response in the MetS women after the breakfast (T-75 to T240; $p = 0.005$) and lunch (T240 to T540; $p = 0.06$) meals relative to the MetS men (Figure 5A). In contrast, the NW men and women responded equivalently. No time x group or time x sex interactions were observed for PP glucose changes. The AUC and IAUC for FFAs were significantly higher on the exercise day vs rest day ($p = 0.03$ and $p = 0.02$, respectively), driven by a significant increase at the end of the exercise bout in all subjects, but a decrease thereafter, showing a characteristic fall with the consumption of meals and rise between meals (Figure 5B). There were no group or sex differences in the total FFA response nor in the pattern of response. The MetS women had higher FFA levels from T180–T360, relative to MetS men, but there was no significant effect when evaluated over the full day via repeated measures ANOVA.

While many between group and sex differences remained, fasting values measured the morning after the main study day did not differ statistically between the exercise and rest conditions.

4. Discussion

In contrast to what was hypothesized, in this population of NW and MetS men and women, a single bout of moderate, morning exercise did not significantly decrease the cumulative PPTG response to mixed meal feeding, measured over an entire day. Possible explanations for the lack of effect include the composition of the diet; timing, length and/or intensity of the exercise bout; and/or the replacement of the energy expended during exercise.

As discussed, data is equivocal in the limited number of studies employing 30–40% fat meals to evaluate the effects of exercise on PPTG, with both a significant reduction in PPTG and also no effect being observed. The fat content of the meals used in the current study was based on the standard intake of most Americans (NHANES III;), with an average of 80

grams of fat over the day. This moderate fat intake may not have induced a large enough PPTG response to facilitate a reductive effect of moderate exercise.

Alternatively, the exercise stimulus itself may not have been great enough to elicit a change in PPTG. In the current study, moderate intensity (60% $\text{VO}_{2\text{peak}}$) exercise of moderate duration (1hr) was employed due to its applicability to the majority of individuals lifestyle and capabilities, and resulted in an average of 484 kcal (NW) and 481 kcal (MetS) expended. This was anticipated to potentially lower PPL based on observations that PPTG were lowered in response to a high fat meal in men with MetS, with an exercise bout resulting in 500 kcal EE, and in overweight/obese men and women with 30 minutes of aerobic exercise the evening before a moderate fat, mixed meal (8%, $p < 0.05$) over an 8 hour period.

Nevertheless, others suggest a stronger exercise stimulus may be required to induce a lowering of PPL. For example, moderate intensity exercise (61–65% $\text{VO}_{2\text{max}}$) reduced the PPTG response to a single meal relative to no exercise control days, whereas low intensity exercise (25–35% $\text{VO}_{2\text{max}}$) did not, despite equivalent exercise duration (90 min) or energy expenditure (1,100 kcal). Furthermore, it has been suggested that at least 600 kcal and up to 1000 kcal must be expended to induce a decrease in measures of PPL. In practical terms, however, expending this amount of energy in a single exercise session would be difficult for the average individual as part of a regular exercise program considering that roughly 1/2 of Americans get less than the recommended 30 minutes of physical activity 3 or more days per week. Given the mixed data on the effects of more practical levels of exercise on PPL we felt it important to evaluate this in both healthy and at-risk individuals.

Perhaps more importantly, the replacement of energy expended during exercise in the current study may have diminished the ability of the intervention to decrease PPL. It may be that the relative energy deficit produced by a bout of exercise is the most important factor in producing a reduction in PPTG. Recent data, published after the completion of the current study, support this contention. For instance, in overweight or obese men a moderate exercise bout (expending ~400 kcal for a 70 kg man) performed 16 hours prior to a test meal, resulted in a lower PPTG (AUC) response, reduced plasma insulin concentrations, and increased fat oxidation as compared to a non-exercise, control condition. When the expended energy was replaced, however, the TG AUC response was no longer significantly different from the control day. Taking into account that the fasting TGs on the day following exercise were lower in the non-energy replaced condition compared to the energy replaced and control conditions (IAUC), the PPTG effect was no longer significant. Harrison et. al. recently reported similar findings with carbohydrate replacement following exercise. This suggests that the main effect of exercise, and its consequent energy deficit, is on fasting TGs and VLDL-production/secretion as opposed to meal lipid metabolism, a common observation in other studies of PP lipemia.

It has been difficult to demonstrate that normal individuals compensate for the energy expended in an acute bout of exercise, and the degree of compensation has been quite variable. In a cohort of overweight/obese men, a single exercise bout resulting in an EE of 700 kcal did not cause an increase in *ad libitum* EI relative to a non-exercise control condition, with free access to a buffet-style breakfast and lunch. (Again, although the exercise decreased the overall TG AUC, there was no difference in IAUC, further evidence that exercise is affecting fasting TG levels and not the post-prandial response *per se*.) However, in overweight and obese men a moderate exercise bout resulted in a *decreased* energy intake of an *ad libitum* meal served immediately following exercise, whereas self-reported intake for the remainder of the study day indicated a significant *increase* in energy intake in the exercise condition. It may be that the ability of exercise to reduce PPTG levels

in free living individuals varies inversely with the degree of caloric compensation per individual. In the current study, subjects had a higher absolute EI in the exercise condition and yet had similar PPTG to the rest condition, indicating that exercise *maintained* PPTG despite the replacement of the energy expended.

Despite the lack of change in the primary outcome (PPTG), some interesting secondary results were observed in this study. Exercise significantly reduced total cholesterol in the NW subjects ($p=0.003$). The current analysis cannot determine the exact cause of this reduction, or why it was not observed in MetS subjects, but this result could be considered anti-atherogenic.

Despite similar fasting total TG levels, we observed a significantly lower PPTG response in women than men, as has been reported. What is novel about the current results, is that this sex difference was driven by the significantly lower PPTG response (IAUC) of MetS women compared to MetS men. A recent study reported that 8 h after a high fat meal, MetS men had a significantly higher TG concentration than MetS women, which the authors suggest is likely due to differences in TG clearance. The women in the current study began to separate from the men at 6 h post-meal, however, we are not able to distinguish the contribution of appearance and/or clearance of TGs to this observation. The reduction in TGs in MetS women was preceded by a significant increase in insulin IAUC with the breakfast and lunch meals, consistent with their reduced insulin sensitivity (expressed as HOMA-IR score) relative to the MetS men and NW subjects. The higher insulin excursion could have resulted in an increase in lipoprotein lipase (LpL) activity and increased TG uptake by tissues.

This study has a number of strengths that make the results pertinent. Firstly, the study design included tight control of subject recruitment and testing criteria: women were tested in the same phase of the menstrual cycle, men and women were matched for fasting TG, and there was careful selection of subjects with MetS. A 5-day controlled dietary lead-in normalized proximal measures between subjects. Another major strength is the inclusion of three, moderate fat, mixed meals administered over an entire 24 hour period, which allowed for the evaluation of the effect of exercise on the normal diurnal accumulation of circulating TGs, as well as the observation of a significant separation in PPTG in the MetS subjects. However, we acknowledge that the conditions chosen for this study are not those that would indisputably demonstrate that exercise decreases PPTG. Due to the involved nature of the study and strict recruitment criteria, the number of subjects is relatively low; a larger sample size may have allowed for better resolution of exercise-induced PPTG response. In addition, had the energy expended with exercise not been replaced, there might have been an observable exercise effect, although the relevance of energy replacement is debatable with respect to application to a real-life scenario.

In conclusion, in the context of this well controlled study, a moderate bout of morning exercise did not lower PPTG in NW or MetS men or women. Our data suggests that, in order for exercise to significantly lower PPTG, subjects must consume very high fat meals, expend substantial energy, and/or not compensate for the energy expended in activity via increasing EI. Even if these conditions are met, there may be an energy expenditure threshold beyond which the TG-lowering effect of exercise plateaus.

Although PPTG did not change, exercise did decrease total cholesterol in the NW subjects, supporting the anti-atherogenic potential of exercise. Our study is the first to report the effect of exercise on PPTG in MetS women. Most importantly, the study underscores the heterogeneity in PP lipid responses between subjects of different sexes and metabolic states. In particular, the MetS women had a very different PP metabolic profile compared to either

NW subjects or MetS men, despite a similar baseline. Our data further supports the contention that the fasting state does not necessarily predict the PP state, and that repeated PP measures may be of equal, if not greater, importance in evaluating cardio-metabolic risk. Overall, these data reinforce the mandate for tailored prevention and treatment strategies for CVD and related conditions.

Acknowledgments

We thank the study participants and their families, UC Denver Clinical and Translational Research Center (CTRC), Metabolic Kitchen and Core Laboratory, and the Energy Balance Laboratory of the Colorado Nutrition Obesity Research Center.

SUPPORT: American Heart Association Grant-in-Aid #0355429Z, NIH DK071155, The Colorado Nutrition Obesity Research Center NIH P30-DK048520 and the Colorado Clinical Translational Research Center, NIH MO1-RR000051.

Abbreviations

ABFAT	abdominal fat
AUC	area under the curve
BMI	body mass index
CT	computerized tomography
CTRC	clinical translational research center
CVD	cardiovascular disease
DXA	dual energy X-ray absorptiometry
EE	energy expenditure
EI	energy intake
FFA	free fatty acids
FFM	fat free mass
HDL-C	high density lipoprotein cholesterol
IAUC	incremental area under the curve
LDL-C	low density lipoprotein cholesterol
MetS	metabolic syndrome
NW	normal weight
PP	post prandial
PPL	post prandial lipemia
PPTG	post prandial triglycerides
RMR	resting metabolic rate
SCFAT	subcutaneous fat
TG	triglyceride
VFAT	visceral fat

References

1. Genest JJ, McNamara JR, Salem DN, Schaefer EJ. Prevalence of risk factors in men with premature coronary artery disease. *Am J Cardiol*. 1991; 67:1185–1189. [PubMed: 2035438]
2. Gealekman O, Guseva N, Hartigan C, Apotheker S, Gorgoglione M, Gurav K, Tran KV, Straubhaar J, Nicoloso S, Czech MP, et al. Depot-specific differences and insufficient subcutaneous adipose tissue angiogenesis in human obesity. *Circulation*. 2011; 123:186–194. [PubMed: 21200001]
3. Brunaldi K, Huang N, Hamilton JA. Fatty acids are rapidly delivered to and extracted from membranes by methyl-beta-cyclodextrin. *Journal of lipid research*. 2010; 51:120–131. [PubMed: 19625735]
4. Nordestgaard BG, Benn M, Schnohr P, Tybjaerg-Hansen A. Nonfasting triglycerides and risk of myocardial infarction, ischemic heart disease, and death in men and women. *JAMA*. 2007; 298:299–308. [PubMed: 17635890]
5. Sarwar N, Danesh J, Eiriksdottir G, Sigurdsson G, Wareham N, Bingham S, Boekholdt SM, Khaw KT, Gudnason V. Triglycerides and the risk of coronary heart disease: 10,158 incident cases among 262,525 participants in 29 Western prospective studies. *Circulation*. 2007; 115:450–458. [PubMed: 17190864]
6. Blair SN, Morris JN. Healthy hearts--and the universal benefits of being physically active: physical activity and health. *Ann Epidemiol*. 2009; 19:253–256. [PubMed: 19344864]
7. Mitchell JA, Bornstein DB, Sui X, Hooker SP, Church TS, Lee CD, Lee DC, Blair SN. The impact of combined health factors on cardiovascular disease mortality. *Am Heart J*. 2010; 160:102–108. [PubMed: 20598979]
8. Hardman AE, Lawrence JE, Herd SL. Postprandial lipemia in endurance-trained people during a short interruption to training. *J Appl Physiol*. 1998; 84:1895–1901. [PubMed: 9609782]
9. Herd SL, Hardman AE, Boobis LH, Cairns CJ. The effect of 13 weeks of running training followed by 9 d of detraining on postprandial lipaemia. *Br J Nutr*. 1998; 80:57–66. [PubMed: 9797644]
10. Malkova D, Hardman AE, Bowness RJ, Macdonald IA. The reduction in postprandial lipemia after exercise is independent of the relative contributions of fat and carbohydrate to energy metabolism during exercise. *Metabolism*. 1999; 48:245–251. [PubMed: 10024090]
11. Tsetsonis NV, Hardman AE. Reduction in postprandial lipemia after walking: influence of exercise intensity. *Med Sci Sports Exerc*. 1996; 28:1235–1242. [PubMed: 8897379]
12. Zhang JQ, Thomas TR, Ball SD. Effect of exercise timing on postprandial lipemia and HDL cholesterol subfractions. *J Appl Physiol*. 1998; 85:1516–1522. [PubMed: 9760349]
13. Zhang JQ, Ji LL, Nunez G, Feathers S, Hart CL, Yao WX. Effect of exercise timing on postprandial lipemia in hyper triglyceridemic men. *Can J Appl Physiol*. 2004; 29:590–603. [PubMed: 15507695]
14. Katsanos CS, Grandjean PW, Moffatt RJ. Effects of low and moderate exercise intensity on postprandial lipemia and postheparin plasma lipoprotein lipase activity in physically active men. *Journal of applied physiology*. 2004; 96:181–188. [PubMed: 12949025]
15. McDowell MA, Briefel RR, Alaimo K, Bischof AM, Caughman CR, Carroll MD, Loria CM, Johnson CL. Energy and macronutrient intakes of persons ages 2 months and over in the United States: Third National Health and Nutrition Examination Survey, Phase 1, 1988–91. *Advance data*. 1994;1–24.
16. Silva KD, Wright JW, Williams CM, Lovegrove JA. Meal ingestion provokes entry of lipoproteins containing fat from the previous meal: possible metabolic implications. *European journal of nutrition*. 2005; 44:377–383. [PubMed: 15526208]
17. Jackson KG, Walden CM, Murray P, Smith AM, Lovegrove JA, Minihane AM, Williams CM. A sequential two meal challenge reveals abnormalities in postprandial TAG but not glucose in men with increasing numbers of metabolic syndrome components. *Atherosclerosis*. 2012; 220:237–243. [PubMed: 22019448]
18. Tentor J, Harada LM, Nakamura RT, Gidlund M, Castilho LN, Cotta de Faria E. Sex-dependent variables in the modulation of postprandial lipemia. *Nutrition*. 2006; 22:9–15. [PubMed: 16226013]

19. Ni H. Prevalence of self-reported heart failure among US adults: results from the 1999 National Health Interview Survey. *American heart journal*. 2003; 146:121–128. [PubMed: 12851619]
20. van Beek AP, de Ruijter-Heijstek FC, Erkelens DW, de Bruin TW. Menopause is associated with reduced protection from postprandial lipemia. *Arterioscler Thromb Vasc Biol*. 1999; 19:2737–2741. [PubMed: 10559019]
21. Sarti C, Gallagher J. The metabolic syndrome: prevalence, CHD risk, and treatment. *J Diabetes Complications*. 2006; 20:121–132. [PubMed: 16504841]
22. Galassi A, Reynolds K, He J. Metabolic syndrome and risk of cardiovascular disease: a meta-analysis. *Am J Med*. 2006; 119:812–819. [PubMed: 17000207]
23. Horton TJ, Pagliassotti MJ, Hobbs K, Hill JO. Fuel metabolism in men and women during and after long-duration exercise. *J Appl Physiol*. 1998; 85:1823–1832. [PubMed: 9804587]
24. Bruce RA, Blackmon JR, Jones JW, Strait G. Exercising Testing in Adult Normal Subjects and Cardiac Patients. *Pediatrics*. 1963; 32(SUPPL):742–756. [PubMed: 14070531]
25. Pietrobelli A, Wang Z, Formica C, Heymsfield SB. Dual-energy X-ray absorptiometry: fat estimation errors due to variation in soft tissue hydration. *The American journal of physiology*. 1998; 274:E808–816. [PubMed: 9612238]
26. Pouliot MC, Despres JP, Lemieux S, Moorjani S, Bouchard C, Tremblay A, Nadeau A, Lupien PJ. Waist circumference and abdominal sagittal diameter: best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. *The American journal of cardiology*. 1994; 73:460–468. [PubMed: 8141087]
27. Horton TJ, Drougas HJ, Sharp TA, Martinez LR, Reed GW, Hill JO. Energy balance in endurance-trained female cyclists and untrained controls. *J Appl Physiol*. 1994; 76:1936–1945. [PubMed: 8063653]
28. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clinical chemistry*. 1972; 18:499–502. [PubMed: 4337382]
29. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia*. 1985; 28:412–419. [PubMed: 3899825]
30. Gill JM, Murphy MH, Hardman AE. Postprandial lipemia: effects of intermittent versus continuous exercise. *Med Sci Sports Exerc*. 1998; 30:1515–1520. [PubMed: 9789852]
31. Poapst M, Reardon M, Steiner G. Relative contribution of triglyceride-rich lipoprotein particle size and number to plasma triglyceride concentration. *Arteriosclerosis*. 1985; 5:381–390. [PubMed: 4015510]
32. Murphy MH, Nevill AM, Hardman AE. Different patterns of brisk walking are equally effective in decreasing postprandial lipaemia. *International journal of obesity and related metabolic disorders: journal of the International Association for the Study of Obesity*. 2000; 24:1303–1309. [PubMed: 11093292]
33. Pfeiffer M, Wenk C, Colombani PC. The influence of 30 minutes of light to moderate intensity cycling on postprandial lipemia. *European journal of cardiovascular prevention and rehabilitation: official journal of the European Society of Cardiology, Working Groups on Epidemiology & Prevention and Cardiac Rehabilitation and Exercise Physiology*. 2006; 13:363–368.
34. Tsetsonis NV, Hardman AE. Effects of low and moderate intensity treadmill walking on postprandial lipaemia in healthy young adults. *Eur J Appl Physiol Occup Physiol*. 1996; 73:419–426. [PubMed: 8803501]
35. Mestek ML, Plaisance EP, Ratcliff LA, Taylor JK, Wee SO, Grandjean PW. Aerobic exercise and postprandial lipemia in men with the metabolic syndrome. *Medicine and science in sports and exercise*. 2008; 40:2105–2111. [PubMed: 18981938]
36. Ho SS, Dhaliwal SS, Hills A, Pal S. Acute exercise improves postprandial cardiovascular risk factors in overweight and obese individuals. *Atherosclerosis*. 2011; 214:178–184. [PubMed: 21081229]
37. Ferguson MA, Alderson NL, Trost SG, Essig DA, Burke JR, Durstine JL. Effects of four different single exercise sessions on lipids, lipoproteins, and lipoprotein lipase. *J Appl Physiol*. 1998; 85:1169–1174. [PubMed: 9729596]

38. Mendes, E. US Health Habits Continue Sharp Winter Decline: Americans' exercise and eating habits worse last month than in same month last year. 2011.
39. Gill JM, Herd SL, Hardman AE. Moderate exercise and post-prandial metabolism: issues of dose-response. *J Sports Sci.* 2002; 20:961–967. [PubMed: 12477005]
40. Burton FL, Malkova D, Caslake MJ, Gill JM. Energy replacement attenuates the effects of prior moderate exercise on postprandial metabolism in overweight/obese men. *Int J Obes (Lond).* 2008; 32:481–489. [PubMed: 17998903]
41. Harrison M, O'Gorman DJ, McCaffrey N, Hamilton MT, Zderic TW, Carson BP, Moyna NM. Influence of acute exercise with and without carbohydrate replacement on postprandial lipid metabolism. *J Appl Physiol.* 2009; 106:943–949. [PubMed: 19112157]
42. James AP, Slivkoff-Clark K, Mamo JC. Prior exercise does not affect chylomicron particle number following a mixed meal of moderate fat content. *Lipids Health Dis.* 2007; 6:8. [PubMed: 17394665]
43. Kolifa M, Petridou A, Mougios V. Effect of prior exercise on lipemia after a meal of moderate fat content. *Eur J Clin Nutr.* 2004; 58:1327–1335. [PubMed: 15054409]
44. Blundell JE, Stubbs RJ, Hughes DA, Whybrow S, King NA. Cross talk between physical activity and appetite control: does physical activity stimulate appetite? *Proc Nutr Soc.* 2003; 62:651–661. [PubMed: 14692601]
45. Hopkins M, King NA, Blundell JE. Acute and long-term effects of exercise on appetite control: is there any benefit for weight control? *Curr Opin Clin Nutr Metab Care.* 2010
46. Farah NM, Malkova D, Gill JM. Effects of exercise on postprandial responses to ad libitum feeding in overweight men. *Med Sci Sports Exerc.* 2010; 42:2015–2022. [PubMed: 20386332]
47. Jokisch E, Coletta A, Raynor HA. Acute energy compensation and macronutrient intake following exercise in active and inactive males who are normal weight. *Appetite.* 2012; 58:722–729. [PubMed: 22142508]
48. Horton TJ, Commerford SR, Pagliassotti MJ, Bessesen DH. Postprandial leg uptake of triglyceride is greater in women than in men. *Am J Physiol Endocrinol Metab.* 2002; 283:E1192–E1202.
49. Adiels M, Olofsson SO, Taskinen MR, Boren J. Overproduction of very low-density lipoproteins is the hallmark of the dyslipidemia in the metabolic syndrome. *Arteriosclerosis, thrombosis, and vascular biology.* 2008; 28:1225–1236.
50. Pedrini MT, Niederwanger A, Kranebitter M, Tautermann C, Ciardi C, Tatarczyk T, Patsch JR. Postprandial lipaemia induces an acute decrease of insulin sensitivity in healthy men independently of plasma NEFA levels. *Diabetologia.* 2006; 49:1612–1618. [PubMed: 16752179]
51. Sadur CN, Eckel RH. Insulin stimulation of adipose tissue lipoprotein lipase. Use of the euglycemic clamp technique. *J Clin Invest.* 1982; 69:1119–1125. [PubMed: 7040473]

FIGURE 1. Study day time line

Following baseline blood sampling and metabolic rate measures, subjects walked on a treadmill (60% $\text{VO}_{2\text{peak}}$) or rested from 0700–0800. Three mixed meals were given over the next 9 hours: breakfast was given at 0830, lunch at 1230 and dinner at 1730; arrows indicate meals. Blood was drawn at regular intervals as indicated.

FIGURE 2. Average PPTG response curves for rest and exercise days for A. normal weight (NW) men and women (n = 13 each) and B. metabolic syndrome (MetS) men and women (n = 9 each)

Following 60 min of rest or exercise (treadmill walking at 60% $\text{VO}_{2\text{peak}}$ period, subjects consumed 3 moderate fat meals (T0, T240 and T840) and plasma TG were measured over 24h. Whole group repeated measures ANOVA: significant time x group x sex effect ($p < 0.001$). MetS repeated measures ANOVA: significant time x sex effect (* $p = 0.032$), which was not seen in the NW group ($p = 0.28$).

FIGURE 3. Total cholesterol for NW and MetS women (A) and men (B)

Following 60 min of rest or exercise (treadmill walking at 60% $\text{VO}_{2\text{peak}}$), subjects consumed 3 moderate fat, mixed meals and total plasma cholesterol was measured over 24h. Repeated measures ANOVA: significant time x group, time x day and time x sex effects ($p < 0.001$ for all). Exercise resulted in a 30% decrease in total cholesterol IAUC (* $p = 0.003$) in the NW group (Figure 3C), but had no effect on cholesterol in MetS subjects.

FIGURE 4. Average TRL particle size (PS) represented by TG:apoB ratio in NW and MetS men and women

Following 60 min of rest or exercise (treadmill walking at 60% $\text{VO}_{2\text{peak}}$), subjects consumed 3 moderate fat, mixed meals and plasma TG and apolipoprotein B (apoB) were measured over 24h. Average of rest and exercise days analyzed by ANOVA: significant time x sex x group interaction (* $p = 0.013$) due to a decrease in TG:apoB ratio over time in MetS women relative to other groups. Overall, relative to NW subjects, MetS subjects as a group had significantly larger TG particles (IAUC $p < 0.001$).

A. MetS Postprandial Insulin Response Curve

B. Postprandial Free Fatty Acids Response Curves

FIGURE 5. Average response curves for (A)MetS insulin, and (B) NW and MetS FFA over 840 minutes

Following 60 min of rest or exercise (treadmill walking at 60% $\text{VO}_{2\text{peak}}$), subjects consumed 3 moderate fat, mixed meals and insulin and free fatty acids (FFA) were measured over 24h. MetS subjects had significantly higher AUC and IAUC's for both insulin and glucose compared to the NW subjects ($p < 0.001$ for both). Repeated measures ANOVA for insulin: significant time $\times$ sex $\times$ group effect ($p = 0.001$), due to a greater insulin response in the MetS women after the breakfast (T-75 to T240; $p = 0.005$) and lunch (T240 to T540; $p = 0.06$) meals relative to the MetS men (7A). FFA were significantly increased with exercise in all groups (* $p < 0.05$).

Table 1

Anthropometric Characteristics of Subjects.

	Normal Weight		MetS	
	Men	Women	Men	Women
N	13	13	9	9
Age (yr) [¶]	26.4 ± 3.6	31.1 ± 7.1*	34.9 ± 2.4	36.3 ± 2.0
Height (m)	1.83 ± .09	1.66 ± .081 [‡]	1.75 ± .03	1.66 ± 0.02
Body weight (kg) [¶]	79.7 ± 9.2	60.3 ± 7.1 [†]	92.8 ± 3.9	79.4 ± 3.0 [†]
BMI (kg/m ²) [¶]	23.7 ± 1.6	21.9 ± 1.9 [†]	30.2 ± 0.9	28.7 ± 0.91
FM (% body weight) ^{¶¶}	20.9 ± 3.8	27.3 ± 6.5 [†]	29.3 ± 1.2	36.0 ± 1.2 [†]
FM (kg) [¶]	16.7 ± 4.2	16.6 ± 5.4	27.2 ± 3.4	28.7 ± 5.3
FFM (kg) ^{¶¶}	62.9 ± 7.1	43.7 ± 4.8 [†]	65.4 ± 7.1	50.2 ± 4.8 [†]
Trunk FM (DXA) ^{¶¶}	22.3 ± 0.01	25.7 ± 0.02	33.2 ± 0.01	38.1 ± 0.04*
Leg FM (DXA) ^{¶¶}	22.3 ± 0.01	31.2 ± 0.02 [†]	26.8 ± 0.01	34.7 ± 0.24 [†]
SagDiam (CT, cm ²) [¶]	19.4 ± 21	17.3 ± 16	25.3 ± 14	23.3 ± 16*
VFat (CT, cm ²) ^{¶¶}	62.6 ± 22	43.4 ± 22	146.8 ± 15	96.4 ± 45*
SCFat (CT, cm ²) [¶]	188.9 ± 67	182.6 ± 61	320.6 ± 58	386.4 ± 89*
VO2 Peak (mL/min) [¶]	3308 ± 394	2231 ± 319 [†]	3163 ± 264	2353 ± 331 [†]
VO2 Peak (mL/kgFFM/mn) [¶]	52.7 ± 1.04	51.1 ± 1.4	48.6 ± 1.4	46.9 ± 1.6

Values = Mean ± SD. **BMI** = body mass index, **FM** = fat mass, **FFM** = fat-free mass, **SagDiam** = sagittal diameter, **VFat** = visceral fat, **SCFat** = subcutaneous fat, **DXA** = dual x-ray absorptiometry, **CT** = computed tomography, VO2 Peak = peak maximal oxygen uptake

p 0.05 =

* p 0.01 = [†] } between sexes within the same group

p 0.001 = [‡]

p 0.05 = [¶] between NW and MetS groups as a whole

p 0.05 ^{¶¶} = between men and women overall

Table 2

Pre-study Fasting Plasma Measures

	Normal Weight		MetS	
	Men	Women	Men	Women
Total TG (mg/dl) [§]	104 ±36	94 ±35	174±47	151 ±53
Total apoB(mg/dl) [§]	78.0 ±14	77.6 ±25	82.8 ±17.6	91.9 ±17.5
Total Cholesterol (mg/dl)	161 ±23	170 ±42	153 ± 29.8	168 ±30.6
FFA (μE/L) [§]	435 ±128	410 ±145	489 ±190.6	502 ±158.5
HDL-C (mg/dl) [¶]	42 ±7.9	47 ±8.8*	31 ±4.7	37 ±3.7‡
LDL-C (mg/dl)	99 ±22	104 ±36	87 ±27.4	101±22.5
Insulin (uU/ml) [§]	5.2 ±2.6	5.3 ±2.6	11.4± 5.1	15.4 ±7.3
Glucose (mg/dl) [§]	87 ±6.0	85 ±6.6	92 ±8.0	92 ±6.8
HOMA-IR [§]	1.12±0.56	1.10 ±0.53	2.60 ±1.3	3.57 ±1.8†
Adiponectin(mg/dl) [§]	6.7 ±3.3	9.1 ±4.1*	3.99 ±1.9	3.2 ±1.7
Estradiol (pg/ml) [¶]	30 ±9.0	84 ±53‡	35 ±8.8	60 ±41.5†
Progesterone (pg/ml)	1.0 ±0.4	0.7±0.4†	0.6±0.15	0.7 ±0.55

Values = Mean ± SD. **TG** = triglycerides, **apoB** = apolipoprotein B, **FFA** = free fatty acids, **HDL-C** = high-density lipoprotein cholesterol, **LDL-C** = low-density lipoprotein cholesterol, **HOMA-IR** = homeostasis model assessment of insulin resistance

p 0.05 = *

p 0.01 = † between sexes within the same group

p 0.001 = ‡

p 0.05 = § between NW and MetS groups as a whole

p 0.05¶ = between men and women overall