Brief Report

Lower Postprandial Oxidative Stress in Women Compared With Men

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ABSTRACT

Background: Previous studies indicate that oxidative stress is increased following intake of a high-fat meal, mediated in large part by the triglyceride (TG) response to feeding as well as fasting oxidative stress values. It has been suggested that women may process TG more efficiently after high-fat meals, based on the antilipidemic properties of estrogen. It has also been reported that women present with lower fasting oxidative stress values than do men. It is possible that women experience attenuated postprandial oxidative stress compared with men.

Objective: The purpose of this study was to compare the postprandial TG and oxidative stress response after a lipid meal in healthy men and women.

Methods: This study was conducted at The University of Memphis in Memphis, Tennessee, from October to December 2008. Blood samples were collected before (in a 10-hour fasted state), and at 1, 2, 4, and 6 hours after ingestion of a lipid load (heavy whipping cream at 1 $g \cdot kg^{-1}$). Blood samples were analyzed for TG, malondialdehyde (MDA), hydrogen peroxide (H_2O_2), and nitrate/nitrite (NOx). The AUC was calculated for each variable and results were compared using a *t* test. Effect-size calculations were performed using Cohen's *d*.

Results: Samples from 10 men and 10 women, aged 18 to 47 years (17 subjects aged <37 years), were compared. AUC data were not significantly different for TG (mean [SEM] 330 [48] vs 354 [34] $mg \cdot dL^{-1} \cdot 6h^{-1}$ for men and women, respectively; effect size = 0.09) or NOx (165 [25] vs 152 [17] $\mu mol \cdot L^{-1} \cdot 6h^{-1}$ for men and women; effect size = 0.09). However, significant differences were noted for MDA (10.7 [1.3] vs 6.1 [0.5] $\mu mol \cdot L^{-1} \cdot 6h^{-1}$ for men and women, respectively; $P = 0.002$; effect size = 0.61) and H_2O_2 (154 [23] vs 86 [8] $\mu mol \cdot L^{-1} \cdot 6h^{-1}$ for men and women; $P = 0.013$; effect size = 0.53).

Conclusions: These data indicate that women experience lower oxidative stress than do men, with regard to MDA and H_2O_2, after ingestion of a lipid load in the form of heavy whipping cream. Considering the strong association between oxidative stress and cardiovascular disease, lower postprandial oxidative stress may be one mechanism associated with decreased risk of cardiovascular disease in women compared with men. Further research is needed to confirm this hypothesis. (*Gend Med.* 2010;7:340–349) © 2010 Excerpta Medica Inc.

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INTRODUCTION

A condition of oxidative stress occurs when the production of reactive oxygen and nitrogen species (RONS) exceeds antioxidant defenses.[1] This scenario has been observed in a number of human studies involving acute feeding of either glucose- or lipid-rich meals and is characterized by a transient (eg, 2–6 hour) increase in blood biomarkers of oxidative stress,[2] including but not limited to isoprostanes, protein carbonyls, malondialdehyde (MDA), hydrogen peroxide (H_2O_2), and nitrate/nitrite (NOx). The elevation in such biomarkers can represent oxidation of cellular lipids and proteins, and has been implicated in a variety of human diseases[3] as well as in the aging process.[4] Although the generation of RONS occurs as a consequence of cellular metabolism[5] and is a necessary component for normal physiological functioning, RONS production beyond what the system is capable of rendering inactive via antioxidant defense may be problematic.[6,7]

The ingestion of high-fat meals has been reported to increase RONS production and result in excessive macromolecule oxidation.[2,8] It has been suggested that because many individuals consume frequent daily meals rich in dietary fat (in particular, saturated and trans fatty acids), they experience a prolonged postprandial state, which may be a significant risk factor for atherogenesis.[9,10]

Women may be less susceptible to oxidative stress, owing to the antioxidant properties of estrogen. Mechanistically, estrogen binds estrogen receptors and activates both mitogen-activated protein kinase and nuclear factor-κβ signaling pathways to induce an upregulation in endogenous antioxidants.[11] This increase in antioxidant activity may help to combat oxidative stress when conditions of increased RONS production arise (eg, ingestion of high-fat meals). In support of this, mitochondria from female rats have been found to exhibit higher antioxidant enzyme gene expression compared with male rats, as well as increased mitochondrial glutathione.[12] Using a human model, our research group has recently noted higher total glutathione (mean [SEM] of 1.86 [0.03] vs 1.78 [0.03] mmol · L^{-1}), and reduced blood glutathione (1.65 [0.03] vs 1.56 [0.03] mmol · L^{-1}) and α-tocopherol, in women compared with men, respectively.[13] Moreover, and likely due to this increased antioxidant defense, markers of lipid peroxidation,[14,15] protein,[16] and DNA oxidation[12] have been reported to be lower in females compared with males.

Aside from improving antioxidant defense, estrogen is known to possess antilipidemic properties.[17,18] It is possible that such an effect may allow for greater uptake of triglycerides (TG) into tissue from the circulation; however, this requires confirmation in human subjects consuming high-fat meals. Such an increased uptake may lead to lower postprandial lipemia, which is viewed as favorable, considering that circulating TG is positively correlated with leukocyte superoxide production.[19] Lipemia is also believed to have a direct detrimental effect on endothelial function by inactivating nitric oxide, possibly leading to the toxic reaction product peroxynitrite.[20] This may contribute to the development of atherosclerotic disease.[19,21] Collectively weighing these factors, it is possible that women may be less susceptible to postprandial oxidative stress than are men, which may help to partly explain the increased longevity in women compared with men.[11]

The purpose of the present investigation was to compare the postprandial TG and oxidative stress response after a lipid meal in healthy men and women. We hypothesized that women would experience an attenuated TG and oxidative stress response to feeding compared with men.

SUBJECTS AND METHODS

This study was conducted at The University of Memphis in Memphis, Tennessee, from October to December 2008.

Subjects

Men and women were recruited for this study via word of mouth and through the use of approved recruitment flyers. Sample size was of convenience, based on previous studies of a similar nature. All subjects were very active and involved in a regular program of structured exercise. Each completed a health history prior to enrollment. All subjects were normolipidemic (fasting TG <150 mg · dL^{-1}) and free of major signs and symptoms suggestive

of cardiovascular, metabolic, or pulmonary disease as defined by the American College of Sports Medicine.[22] No participant reported using medications (eg, anti-inflammatory or cardiovascular drugs) or antioxidant supplements during the course of the study. No women were peri- or postmenopausal, and all reported normal (28–35 day) menstrual cycles, did not use oral contraceptives, and reported to the lab for testing during the first 2 days of their menstrual cycle. Anthropometric data were obtained from all subjects using standard procedures, and a 7-site skinfold test was performed using Lange calipers to estimate body density, which was subsequently used to calculate body-fat percentage. Resting heart rate and blood pressure were recorded after a 10-minute rest period. Following the screening procedure, subjects were scheduled for testing and given instructions and data forms related to the recording of dietary data during the 6 days prior to the test meal. All experimental procedures were performed in accordance with the Declaration of Helsinki guidelines and approved by the University Human Subjects Review Board (protocol H08-54). Subjects provided oral and written consent before participating.

Test Meal

All meal testing was performed in the morning following an overnight fast (minimum of 10 hours). Despite our recent finding that small variation in estrogen across a single menstrual cycle within the same sample of women has little effect on postprandial oxidative stress,[23] all women in the present design were tested during the first 2 days of their menstrual cycle. After reporting to the lab, and following a 10-minute period of rest, a premeal blood sample was collected from the subjects. They were then given 15 minutes to consume the test meal, which consisted of heavy whipping cream mixed in water (1 g of fat per kg of body mass, totaling ~9 kcal per kg of body mass). Water was added to the whipping cream in an amount needed to reach a total fluid volume of 1 mL per kcal, considering the volume of whipping cream used. Therefore, a 70-kg subject consumed 70 g of whipping cream = 630 kcal = 630 mL of total fluid volume (water volume = total fluid volume – whipping cream volume). The amount was calculated for each subject's meal/drink using measuring cylinders. Subjects remained in the laboratory (or in close proximity) during the entire 6-hour postprandial period and were allowed to watch movies, work on a computer, or read. No additional meals or calorie-containing beverages were allowed; however, water was allowed ad libitum.

Blood Sampling and Biochemistry

For each subject, ~10 mL of venous blood was taken from a forearm vein via needle and collection tube by a trained phlebotomist at the following times: premeal (0 hour) and 1, 2, 4, and 6 hours postmeal. Blood collected in tubes containing EDTA was centrifuged immediately at 4°C and the plasma stored in multiple aliquots at –80°C until analyzed. Blood collected in tubes with no additive was allowed to clot for 30 minutes at room temperature and then centrifuged at 4°C. The serum was stored in multiple aliquots at –80°C until analyzed.

TG was analyzed in serum following standard enzymatic procedures as described by the reagent provider (Thermo Electron Clinical Chemistry, Thermo Fisher Scientific Inc., Waltham, Massachusetts). The %CV of this assay in our lab is <5%. MDA was analyzed in plasma using previously described methods[24] and commercially available reagents (Northwest Life Science Specialties, LLC, Vancouver, Washington). The %CV of this assay in our lab is <6%. H_2O_2 was analyzed in plasma using the Amplex Red reagent method as described by the manufacturer (Molecular Probes, Invitrogen Corporation, Eugene, Oregon). The %CV of this assay in our lab is <5%. As a surrogate marker of nitric oxide, the NOx metabolites were analyzed in plasma using a commercially available assay (Caymen Chemical Company, Ann Arbor, Michigan). The %CV of this assay in our lab is <6%.

Dietary Records and Physical Activity

Subjects were instructed to maintain their usual diet and physical activity levels, and to record all food and drink consumed during the 6 days prior to the test meal day. Diet records were analyzed using the Food Processor SQL, version 9.9 (ESHA

Research Inc., Salem, Oregon) for total calories, macronutrients, and antioxidant micronutrients, because dietary antioxidant intake can influence oxidative stress.[4] Subjects were asked to avoid physically stressful tasks during the 24-hour period preceding the test meal. This was done to control for any acute effects of physical activity on postprandial oxidative stress.[25,26]

Statistical Analysis

AUC was calculated for each variable using the trapezoidal method (AUC_G) as described in detail by Pruessner et al.[27] All variables were then analyzed using a *t* test. Effect-size calculations were performed using Cohen's *d*. Premeal values for all variables were compared using contrasts. The data are presented as mean (SEM). Pairwise correlations were made between all variables. All analyses were performed using JMP statistical software, version 4.0.3 (SAS Institute Inc., Cary, North Carolina). Statistical significance was set at $P \leq 0.05$.

RESULTS

Healthy men (n = 10) and women (n = 10) between the ages of 18 and 47 years participated in this study. Seventeen of the subjects were <37 years of age; 1 man and 2 women were in their forties (aged 41, 46, and 47 years, respectively). With the exception of 1 man and 2 women who were black, all other participants were white. Subject characteristics are presented in **Table I**.

Blood samples from the men and women were compared. Significant differences between men and women were noted for height, weight, body fat, waist:hip ratio, and systolic blood pressure (all, $P < 0.05$; **Table I**). Regarding dietary variables, a significant difference was noted between men and women for carbohydrate intake, with men consuming more carbohydrate ($P = 0.009$; **Table II**). No other significant differences were noted in dietary intake.

Regarding biochemical variables, AUC data were not significantly different between men and women for TG (effect size = 0.09; **Figure 1**) or NOx (effect size = 0.09; **Figure 2**). However, significant differences were noted for MDA ($P = 0.002$; effect size = 0.61; **Figure 3**) and H_2O_2 ($P = 0.013$; effect size = 0.53; **Figure 4**), with women exhibiting lower values than men. No significant differences were noted between men and women for premeal TG ($P = 0.34$), MDA ($P = 0.12$), H_2O_2 ($P = 0.66$), or NOx ($P = 0.07$) (data not shown).

When combining data for both men and women, significant correlations were noted between TG and MDA ($r = 0.55$; $P < 0.001$), TG and H_2O_2 ($r = 0.52$; $P < 0.001$), TG and NOx ($r = 0.67$; $P < 0.001$), MDA and NOx ($r = 0.66$; $P < 0.001$), MDA and H_2O_2 ($r = 0.71$; $P < 0.001$), and NOx and H_2O_2 ($r = 0.63$; $P < 0.001$). When investigating the correlations separately, the results were as follows

Table I. Descriptive characteristics of men and women. Values are mean (SEM).

Variable	Men (n = 10)	Women (n = 10)	*P*
Age, y	27 (2)	29 (3)	0.755
Height, cm	180 (3)	165 (1)	<0.001
Weight, kg	81 (6)	59 (3)	0.004
BMI, $kg \cdot m^{-2}$	25 (1)	22 (1)	0.077
Body fat, %	15 (2)	21 (2)	0.030
Waist:hip ratio	0.82 (0.02)	0.63 (0.07)	0.015
Resting heart rate, beats/min	66 (6)	59 (3)	0.293
Resting SBP, mm Hg	124 (4)	107 (3)	0.001
Resting DBP, mm Hg	71 (2)	66 (2)	0.119

BMI = body mass index; SBP = systolic blood pressure; DBP = diastolic blood pressure.

Table II. Dietary variables of men and women during the 6 days prior to a lipid meal. Values are mean (SEM).

Variable	Men (n = 10)	Women (n = 10)	*P*
Kilocalories	2419 (153)	2026 (172)	0.105
Protein, g	87 (6)	101 (17)	0.440
Carbohydrate, g	314 (27)	214 (21)	0.009
Fat, g	82 (9)	74 (7)	0.465
Vitamin C, mg	93 (25)	79 (20)	0.664
Vitamin E, mg	13 (5)	5 (2)	0.162
Vitamin A, IU	5992 (1872)	4684 (1268)	0.570

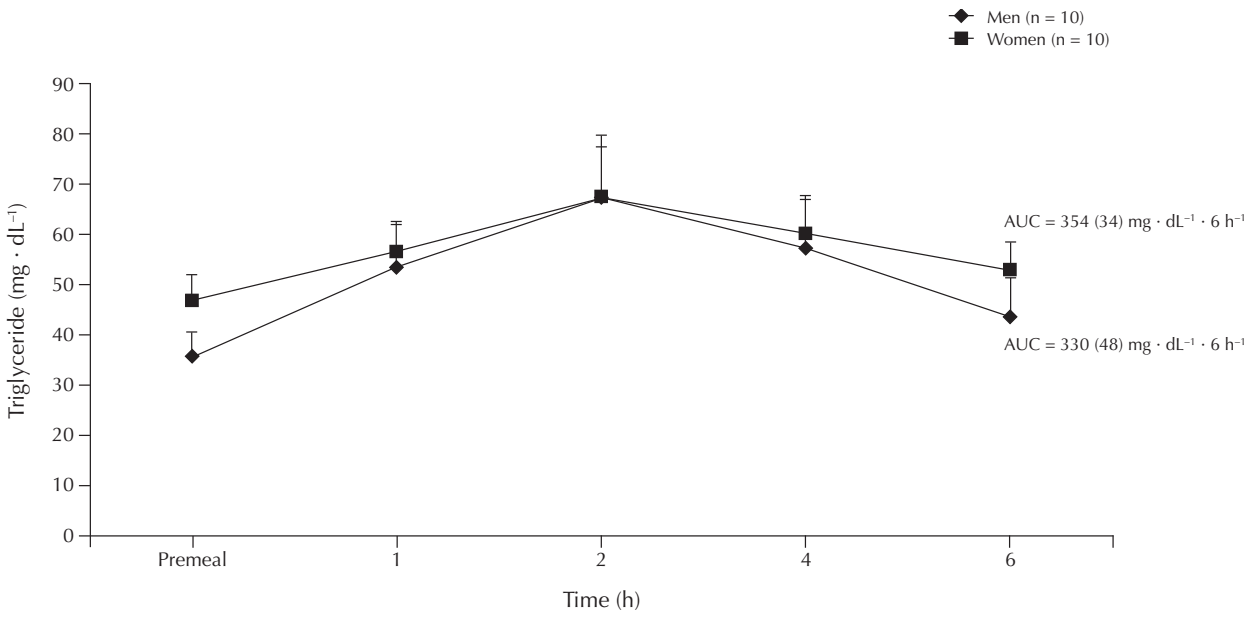

Figure 1. Serum triglycerides before and after intake of a lipid meal in men and women. Values are mean (SEM). No statistically significant difference noted for AUC ($P = 0.689$).

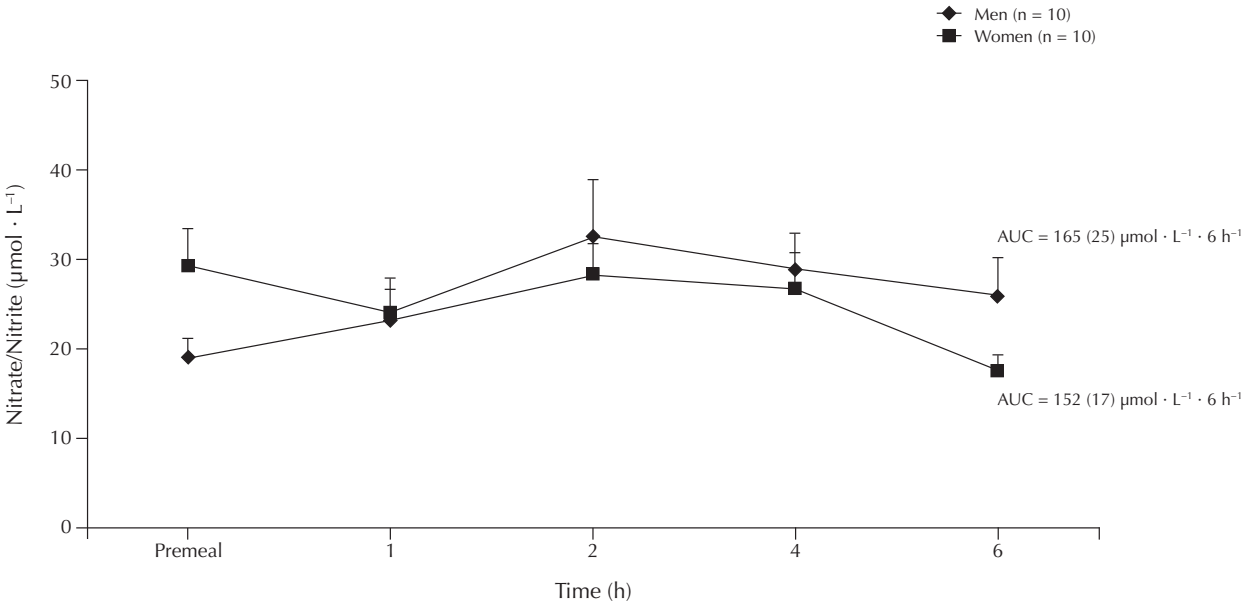

Figure 2. Plasma nitrate/nitrite before and after intake of a lipid meal in men and women. Values are mean (SEM). No statistically significant difference noted for AUC ($P = 0.670$).

for men: TG and MDA ($r = 0.72$; $P < 0.001$), TG and H_2O_2 ($r = 0.71$; $P < 0.001$), TG and NOx ($r = 0.79$; $P < 0.001$), MDA and NOx ($r = 0.83$; $P < 0.001$), MDA and H_2O_2 ($r = 0.70$; $P < 0.001$), and NOx and H_2O_2 ($r = 0.87$; $P < 0.001$). The results were as follows for women: TG and MDA ($r = 0.69$; $P < 0.001$), TG and H_2O_2 ($r = 0.32$; $P = 0.02$), TG and NOx ($r = 0.49$; $P < 0.001$), MDA and NOx ($r = 0.52$; $P < 0.001$), MDA and H_2O_2 ($r = 0.44$; $P = 0.001$), and NOx and H_2O_2 ($r = 0.05$; $P = 0.73$).

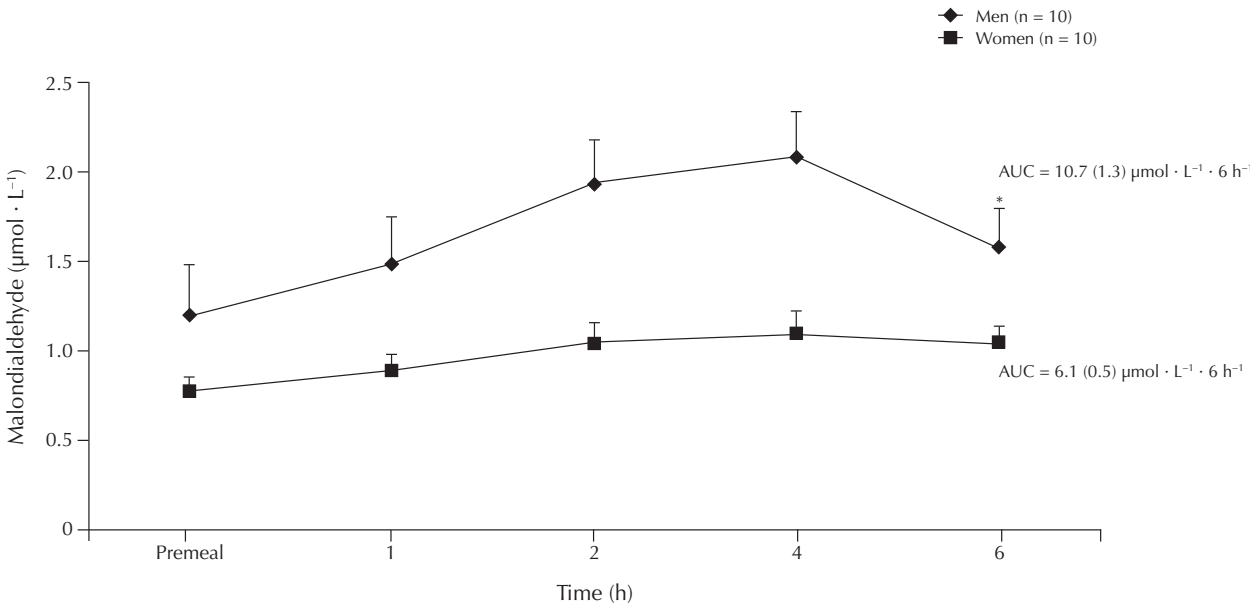

Figure 3. Plasma malondialdehyde before and after intake of a lipid meal in men and women. Values are mean (SEM). *Statistically significant difference noted for AUC ($P = 0.002$).

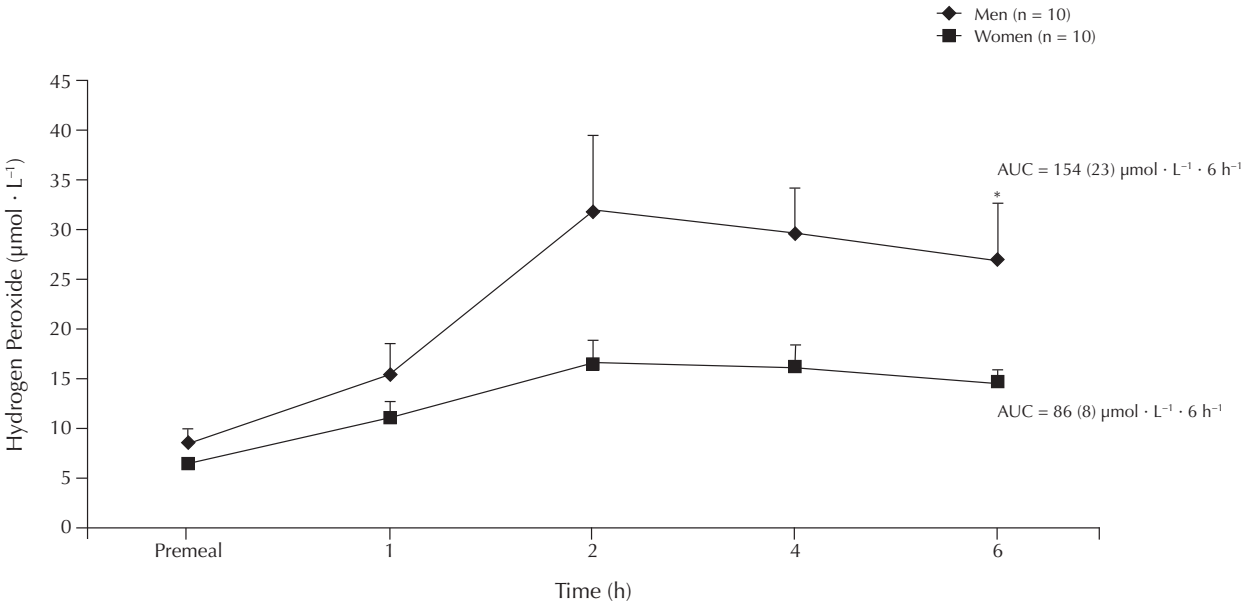

Figure 4. Plasma hydrogen peroxide before and after intake of a lipid meal in men and women. Values are mean (SEM). *Statistically significant difference noted for AUC ($P = 0.013$).

DISCUSSION

Data from the present investigation indicate that, after ingestion of a lipid meal consisting of heavy whipping cream, women are less prone to postprandial oxidative stress than are men. This statement is supported by our finding of significantly lower MDA and H_2O_2 in women compared with men, validated by both AUC *t*-test analysis and effect-size calculations, which were considered moderate to large. These data may have health

implications pertaining to the development of oxidative stress–related disease; however, future study is needed to investigate this possibility.

While subjects were considered to be healthy and relatively young (ie, all subjects but 3 were <37 years of age), most subjects were white. Therefore, it is uncertain as to whether men and women of different racial backgrounds would respond the same way. Further research is needed to investigate this. Also, the subjects in the present study ingested a 100% lipid meal, as opposed to a mixed meal containing lipid, carbohydrate, and protein. Because most individuals regularly consume mixed meals as opposed to pure lipid meals, our data may not be generalizable to most feeding circumstances. This is particularly relevant, as we have recently noted that an isocaloric mixed meal (33% of each nutrient based on kilocalories) does not induce the same magnitude of oxidative stress compared with a pure lipid meal (unpublished findings). These factors should be considered when interpreting our findings.

Although certain anthropometric variables differed between men and women (**Table I**), these are expected differences based on sex, and all variables were considered to be within normal clinical range for both men and women.[22] The one variable that is known to influence oxidative stress (obesity classification) was not different between men and women based on BMI calculation. Body-fat percentage, as expected based on typical differences between men and women, was higher in women compared with men (although comparable between men and women based on known sex differences in body-fat percentage). Therefore, we do not believe that any difference in anthropometric variables influenced our findings.

Likewise, dietary data were not different with the exception of carbohydrate intake. While it is possible that excess carbohydrate intake can result in an increase in fasting oxidative stress values, we do not believe that the differences in carbohydrate intake influenced our postprandial values. This is particularly true considering that the carbohydrate intake relative to body mass was similar for men and women (3.9 g/kg vs 3.6 g/kg of body mass, respectively).

Regarding the processing of TG, contrary to our initial hypothesis, we noted no significant difference in TG response between men and women. We believe this may be due to the fact that fasting TG values were low for both men and women, and both groups processed TG relatively efficiently. For example, values increased only slightly and peaked at the 2-hour postingestion time, with a gradual return to baseline by 6 hours postingestion (**Figure 1**). This is in contrast to the significant and prolonged (eg, 6-hour) elevation in TG noted in subjects who are obese,[28] prediabetics,[29] and regular cigarette smokers.[30] In the present study, it appeared that young and healthy men and women processed TG efficiently and that any potential sex differences were masked by the small magnitude and time course of increase. It is possible that the inclusion of men and women with higher fasting TG values, coupled with an impaired ability to process TG in response to feeding (compared with subjects in the present study), would have allowed for differences to be noted between sexes. Future studies should consider these issues.

In reference to our chosen oxidative stress biomarkers, first it should be mentioned that these were included because of their widespread use throughout the feeding and oxidative stress literature. Admittedly, a limitation of this work was the failure to include other measures of oxidative stress such as isoprostanes, protein carbonyls, and 8-hydroxydeoxyguanosine, as well as measures of enzymatic and nonenzymatic antioxidant activity (including paraoxonase activity, which was reported to exhibit sex-specific differences in response after a 14-week period of high-fat feeding in rats[31]). Moreover, although we measured TG, we failed to measure other blood lipids such as total, low, and high-density lipoprotein cholesterol, which may have provided additional insight into explaining the current findings (eg, antioxidant properties of high-density lipoproteins). Finally, with the use of our current biomarkers, we are uncertain as to the specific source of RONS generation, with the exception of our knowledge that the increase in NOx was likely due to an increase in nitric oxide, and the increase in MDA

was likely due to an increase in polyunsaturated fatty acid oxidation. Further research should consider not only the inclusion of additional biomarkers as mentioned, but also an investigation of the specific source of RONS generation after ingestion of high-fat meals (eg, peripheral blood mononuclear cells, such as lymphocytes and monocytes).

Despite these limitations, we believe that our findings provide sound evidence to support our statement that women experience lower postprandial oxidative stress than do men. While this was not the case for NOx, as both men and women experienced a similar response after feeding (**Figure 2**), our data for MDA (**Figure 3**) and H_2O_2 (**Figure 4**) clearly support this. This discrepancy in findings between our 3 oxidative stress biomarkers highlights the importance of including multiple markers within such a research design. In this way, a better overall picture of the oxidative status within the system is provided.[32]

Women experienced very little increase in both MDA and H_2O_2 in response to feeding, despite consuming the same relative amount of lipid compared with men ($1\ g \cdot kg^{-1}$). While the present study is merely observational in nature and does not provide mechanistic evidence for these findings, it is believed based on previous work that higher estrogen levels are responsible for our findings.[33] Considering that women reported to the lab for meal testing when estrogen was theoretically lowest during their menstrual cycle, it is important to note that estrogen concentrations do not necessarily need to be elevated at the time of RONS exposure to allow for enhanced protection. To the contrary, it appears that it is the chronically higher levels of estrogen across the entire menstrual cycle that may serve as the signal for upregulation in endogenous antioxidant defense, which then remains present throughout the entire cycle. This increased antioxidant defense may provide protection against a variety of stressors, including ingestion of a lipid meal, as observed in the present investigation.

CONCLUSIONS

We report that, after ingestion of a lipid meal of heavy whipping cream, women were less prone to postprandial oxidative stress than were men. Considering the strong association between oxidative stress and cardiovascular disease, lower postprandial oxidative stress may be one mechanism associated with decreased risk of cardiovascular disease in women compared with men. Further research is needed to confirm this hypothesis.

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Both authors were responsible for the study design. Kelsey Fisher-Wellman was responsible for data collection, blood collection and processing, and biochemical work. Richard Bloomer was responsible for statistical analyses and manuscript preparation. Both authors read and approved the final manuscript.

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