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Exercise attenuates the increase in plasma monounsaturated fatty acids and high-density lipoprotein cholesterol but not high-density lipoprotein 2b cholesterol caused by high-oleic ground beef in women

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ABSTRACT

We hypothesized that dietary monounsaturated fatty acids (MUFA) and exercise increase high-density lipoprotein cholesterol (HDL-C) by independent mechanisms, so there would be additive effects between a single, intensive session of exercise and high-MUFA ground beef on HDL-C and blood risk factors for cardiovascular disease. Seventeen postmenopausal women completed a 2-way crossover design in which they consumed five 114-g ground beef patties per week for two 6-week periods separated by a 4-week washout (habitual diet) period. The ground beef patties contained 21% total fat with either 9.97 (low-MUFA) or 12.72 (high-MUFA) g total MUFA. Blood was taken at entry, at the end of each 6-week diet period, after the 4-week washout period, and 24 hours after aerobic exercise sessions (75% VO_{2peak}, 2.07 MJ). After the ground beef intervention, the high-MUFA ground beef increased plasma palmitoleic acid and oleic acid, low-density lipoprotein (LDL) particle density, HDL-C, and HDL_{2b}-C (all $P < .05$), whereas the low-MUFA ground beef increased LDL density. After the washout (habitual diet) period, the single exercise session increased serum LDL cholesterol, HDL-C, and HDL_{2a} and decreased TAG and oleic acid. After the low-MUFA ground beef diet, exercise increased LDL size and HDL density and decreased LDL density and very low-density lipoprotein cholesterol, but had no effect on HDL-C fractions. After the high-MUFA ground beef intervention, exercise decreased palmitioleic acid, oleic acid, HDL-C, and HDL_{2a}-C, but not HDL_{2b}-C. Contrary to our hypothesis, the effects of exercise and a high-MUFA diet were not additive; instead, exercise attenuated the effects of the high-MUFA ground beef on HDL-C and plasma MUFAs. The differential effects of high-MUFA ground beef and exercise on HDL_{2a}-C and HDL_{2b}-C indicate that diet and exercise affect HDL-C by different mechanisms.

Abbreviations: AHA, American Heart Association; BMI, body mass index; CETP, cholesterol ester transferase protein; CVD, cardiovascular disease; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; Lp(a), lipoprotein a; MUFA, monounsaturated fatty acids; RLP-C, remnant lipoprotein cholesterol; SCD, stearoyl-CoA desaturase; SFA, saturated fatty acids; TAG, triacylglycerol; TC, total cholesterol; VLDL-C, very low-density lipoprotein cholesterol.

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1. Introduction

Cardiovascular disease (CVD) is the leading cause of death in the United States. A common assessment of CVD risk uses the blood lipid and lipoprotein profile of an individual [1,2]. Although increases in high-density lipoprotein cholesterol (HDL-C), including larger more buoyant HDL particles (HDL_{2b}), have been shown to be cardioprotective [3], a more anthrogenic lipoprotein profile consists of higher circulating low-density lipoprotein cholesterol (LDL-C), with small dense LDL particles being more athrogenic than larger LDL subfractions [4]. Additional lipid-rich lipoproteins related to increased CVD risk include very low-density lipoprotein cholesterol (VLDL-C), remnant lipoprotein (RLP), intermediate density lipoprotein, and lipoprotein a (Lp[a]) [5–7]. Blood CVD risk factors can be ameliorated with aerobic exercise training and/or single bouts of intensive training [8–14]. As little as 30 minutes of moderate-intensity exercise slows the progression of CVD in women [15], and a meta-analysis of the literature concluded that plasma concentrations of total cholesterol (TC), triacylglycerols (TAGs), and LDL-C fall and HDL-C rise with aerobic exercise [13]. Similarly, the Stanford Five-City Project demonstrated a significant correlation between physical activity score and HDL-C [16].

There is an increased risk of CVD in postmenopausal women due, in part, to reduced circulating estrogen and an overall reduction in physical activity. Although women have higher HDL-C and TC and lower LDL-C and TAG than do men [17], HDL-C concentrations decrease in women after menopause. The postmenopausal decrease in HDL-C after menopause is accompanied by more dense, smaller HDL particles [18] and an increase in LDL-C, TC, VLDL-C, TAG, and body mass index (BMI), contributing to an increased risk for CVD [12,17,18]. Although Wooten et al [19] reported that a single bout of aerobic exercise decreased HDL-C in premenopausal women, others have reported that a single session of aerobic exercise increased HDL-C in postmenopausal women [11,20,21].

Dietary fat intake, which dictates plasma fatty acids and influences lipoprotein metabolism, is also known to influence the risk for CVD. The previous recommendation of the American Heart Association (AHA) was to consume a low-fat diet, especially low in saturated fatty acids (SFAs). At that time, the AHA recommended that calories from fat be replaced by carbohydrate, which may have increased the risk of CVD by increasing plasma TAG and Lp(a) and decreasing HDL-C and LDL particle size [22–24]. These findings resulted in the reevaluation of dietary SFA and their effects on CVD [25]. Although the recommendation for a reduction in dietary SFA remained, the AHA recognized that diets that provided up to 40% of dietary energy in the form of unsaturated fat were as heart healthy as low-fat diets [26,27].

Although independently, diet and exercise are known to influence CVD risk, the interactive effects of diet fatty acid composition and exercise have not been studied extensively. Nolte et al [28] demonstrated that dietary fat reduction (to 20%-25% energy from fat) decreased HDL-C in men, which was attenuated by an aerobic exercise program. Wooten et al [29] reported the interaction between supplemental n-3 fatty acids (4.55 g/d) and aerobic exercise (3 consecutive days of treadmill walking at 65% VO_{2peak} for 60 minutes) on LDL and HDL particle size. Exercise increased LDL particle size but did not affect HDL particle size. Although n-3 fatty acid supplementation increased HDL_2-C and decreased HDL_3-C [29], there were no additive or synergistic effects of exercise and n-3 fatty acid supplementation.

To our knowledge, there are no published studies that describe the interaction between dietary oleic acid (which increases HDL-C [25]) and exercise. This study was designed to assess the following: (1) the impact of a single bout of exercise on CVD risk factors, (2) the effects of high-oleic ground beef and chub pack ground beef on CVD risk factors, and (3) the combined effects of diet and exercise on CVD risk. Diet and exercise may independently influence risk for CVD. If this is so, then the effects of diet and exercise on CVD humoral risk factors should be additive. Therefore, this study tested the hypothesis that a single, intensive session of exercise would act additively with high-oleic acid ground beef intervention to reduce humoral risk factors for CVD in postmenopausal women, who are at increased risk for CVD after menopause due, in part, to a decrease in HDL-C.

2. Methods and materials

2.1. Approval

The study protocol was reviewed and approved by the Texas A&M University Institutional Review Board for the use of human subjects in research (2008-125), and all participants gave written consent.

2.2. Participants and study design

Twenty-nine postmenopausal women were recruited from the local Bryan/College Station, Texas community. Nineteen women completed the diet portion of the study, whereas 17 completed both the exercise and diet portions. Only those subjects who completed all phases of the study were used in the final analyses. The subjects were women who had experienced natural or surgical menopause and whose last menstrual period was more than 1 year before enrollment of the study. Subjects were able to walk briskly for 20 minutes without chest pain or fatigue; were nonsmokers with no history of CVD, stroke, or diabetes; had normal liver function test results; normal fasting glucose; had normal resting and exercise electrocardiogram result; had a serum TC less than 6.5 mmol/L; and were not taking lipid-lowering drugs (including fish oils) or hormone replacement therapy.

Participants were allotted to 2 groups in a crossover design. The first group was fed low–monounsaturated fatty acid (MUFA) ground beef for a 6-week period and, after a 4-week habitual diet washout period, was rotated to a high-MUFA ground beef diet (Fig.). The second group was fed a high-MUFA test ground beef for a 6-week period and, after the 4-week habitual diet washout period, was rotated to a low-MUFA test ground beef diet. The participants were asked to replace one of their normal meat servings with a test ground beef patty for a total of 5 patties/wk, or 30 patties for each dietary ground beef phase. No restrictions were placed on how the patty was to be

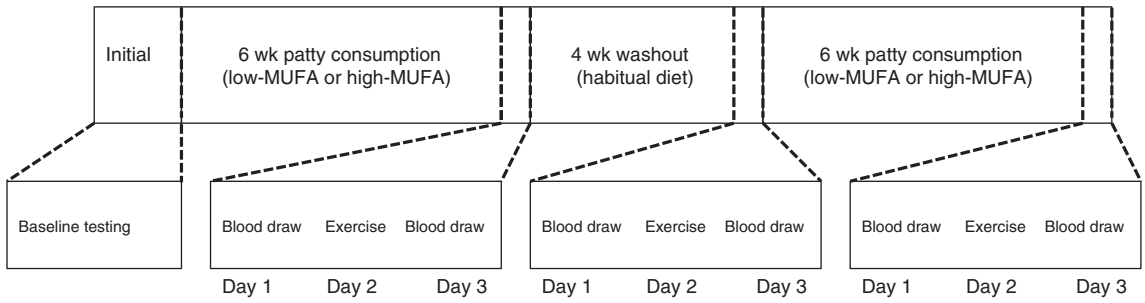

Fig. – Study design shows the relation of patty consumption, blood draws, and acute exercise bouts.

prepared other than one patty should be prepared at a time, and all of the patty should be consumed in one sitting. The subjects were not informed as to which test patty they had been assigned.

At study commencement, each subject underwent a complete history and physical examination by a physician and had an exercise stress test with electrocardiogram and measured VO_{2peak} (Medical Graphics Corp, St. Paul, MN, USA, CPX/D gas analysis system), according to the Bruce protocol [30]. Each subject also received a dual-energy x-ray absorptiometry scan to assess body composition changes at baseline and upon completion of the study.

2.3. *Production of test patties*

Low-MUFA ground beef patties (MUFA/SFA = 0.86) were made from chub pack ground beef purchased from a local retail outlet. Chub pack ground beef is the least expensive beef available at retail and contains 20% to 30% total fat. Samples of chub pack ground beef from all retail outlets in the Bryan (College Station, TX, USA) were evaluated, and ground beef with 20% fat was used in this study. High-MUFA patties (MUFA/SFA = 1.43) were premade from lean and fat trim of full-blood Akaushi cattle and were processed and packaged at HeartBrand Beef (Yoakum, TX, USA; Table 1). Beef from Akaushi cattle is naturally enriched with oleic acid (18:1n-9) [31,32], and these cattle were fed grain-based diets that further increased the oleic content of beef [31,33]. Patties were individually vacuum packed, quick frozen, and boxed by diet type. The beef was supplied to the participants in the form of 114-g raw ground beef patties. The frozen, vacuum-packaged ground beef patties for an entire diet period (30 patties) were delivered to the participants on or before the first day of the diet period.

2.4. *Exercise component*

All participants completed exercise questionnaires to establish habitual energy expenditure and exercise energy expenditure and duration. At the completion of each diet phase and once in the washout period, the participants completed a blood sampling and aerobic exercise protocol. The women were asked to abstain from any physical exercise for at least 3 days and fast for 12 hours before reporting to the laboratory for their resting, preexercise blood sample. This coincided with 1 day after each 6-week ground beef intervention period and the last day of the 4-week washout period.

Subjects returned to the laboratory on the following day to complete the submaximal, experimental exercise session. Specifically, the subjects were asked to walk on a motor-driven treadmill at 75% of their predetermined VO_{2peak} for the duration required to expend 2.07 MJ of energy. Heart rate was monitored continuously (Polar heart rate monitor), and expired gases were measured every 10 minutes of exercise with a portable metabolic system (Medical Graphics Corp, VO2000) to ensure that the prescribed intensity and energy expenditure were maintained. The speed and grade of the treadmill were adjusted as necessary to maintain the required intensity and caloric expenditure. Fasting blood samples were again obtained for postexercise analysis 24 hours after the single exercise session.

2.5. *Collection and handling of blood samples*

Blood samples were collected at 7 intervals: 1 day before the first phase of the dietary ground beef (low-MUFA or high-

Table 1 – Fatty acid content of low-MUFA and high-MUFA ground beef patties

Fatty acid (g/patty)	Low MUFA	High MUFA
Myristic (14:0)	0.74 ± 0.02	0.58 ± 0.01 **
Myristoleic (14:1(n-5))	0.18 ± 0.01	0.24 ± 0.01 **
Palmitic (16:0)	6.06 ± 0.14	5.32 ± 0.14 *
Palmitoleic (16:1(n-7))	0.64 ± 0.01	1.00 ± 0.02 **
Stearic (18:0)	4.46 ± 0.07	2.67 ± 0.13 **
18:1*trans*-9	0.11 ± 0.01	0.11 ± 0.01
18:1*trans*-10	1.10 ± 0.06	0.83 ± 0.07 *
trans-Vaccenic (18:1(*trans*-11))	0.38 ± 0.03	0.15 ± 0.01 **
Oleic (18:1n-9)	8.60 ± 0.13	10.59 ± 0.04 **
Cis-Vaccenic (18:1(n-7))	0.35 ± 0.01	0.54 ± 0.01 **
Linoleic (18:2(n-6))	0.31 ± 0.04	0.34 ± 0.02
α-Linolenic (18:3(n-3))	0.01 ± 0.01	0.02 ± 0.01
18:2*cis*-9, *trans*-11	0.02 ± 0.01	0.04 ± 0.01
18:2*trans*-10, *cis*-12	0.01 ± 0.01	0.02 ± 0.01 *
Total MUFA [a]	9.97 ± 0.14	12.72 ± 0.07 **
Total *trans*-fatty acids	1.59 ± 0.02	1.09 ± 0.08 **
MUFA/SFA [b]	0.86 ± 0.01	1.43 ± 0.04 **

Data are means ± SEM (n = 3). Ground beef contained an average of 21% total fat, or 24 g total fat per patty. Neither EPA, 20:5(n-3), nor DHA, 22:6(n-3) was detectable in the ground beef patties.

[a] MUFA, *cis*-9 MUFAs.

[b] Ratio of total *cis*-9 MUFA to total SFA.

* $P < .05$, low MUFA vs high MUFA.

** $P < .01$, low MUFA vs high MUFA.

MUFA) intervention (onset), 6 weeks after initiation of phase 1 (preexercise), 24 hours after acute exercise at the end of phase 1, at the end of the 4-week washout phase, 24 hours after acute exercise at the end of the washout period, 6 weeks after initiation of the second phase of the dietary ground beef (low-MUFA or high-MUFA) intervention (preexercise), and 24 hours after acute exercise at the end of the washout period (Fig.). With the subject seated at a quiet rest, all blood samples were drawn without stasis from an antecubital vein into vacutainer tubes containing either EDTA (plasma) or a clot activator (serum). Plasma and serum were isolated by centrifugation at 1500 × *g* for 30 minutes at 4°C and then stored at –80°C.

2.6. Fatty acid composition of plasma and ground beef

Fatty acids were measured in plasma, and fatty acid composition and total fat of the ground beef patties were measured. Total lipid was extracted and methylated as described by others [32,34,35]. Fatty acid methyl esters were analyzed with a Varian gas chromatograph (model CP-3800 fixed with a CP-8200 autosampler; Varian Inc, Walnut Creek, CA, USA). Separation of fatty acid methyl esters was accomplished on a fused silica capillary column CP-Sil88 (100 m × 0.25 mm [i.d.]; Chrompack Inc, Middleburg, the Netherlands) with helium as the carrier gas (1.2 mL/min). Oven temperatures began at 150°C and were increased to 160°C at a rate of 1°C/min. The oven temperature rose further to 167°C at a rate of 0.2°C/min. The temperature increased a rate of 1.5°C/min to a final temperature of 225°C where it was held for 26 minutes. Injector and detector temperatures were at 270°C. Individual fatty acid methyl esters were identified using genuine standards (Nu-check Prep, Inc [Elysian, MN, USA] and Sigma-Aldrich Co, St. Louis, MO, USA) and expressed as a mmol/100 mmol total fatty acids in plasma or as g/beef patty. Total fat content of the dietary ground beef patties was measured by CEM Corp's SMART Trac Moisture and Fat Analysis system [36].

2.7. Clinical assessments

Frozen aliquots of serum were sent to Spectracell Laboratories, Inc (Houston, TX, USA) for analyte and lipoprotein-lipid analyses using an analytical ultracentrifugation process. A complete "Lipoprotein Particle Profile" test was performed using the lipoprotein subgroup particle number analysis method. This lipoprotein particle separation procedure uses a patented method (Patent No.: US 7,856,323 B2) with a continuous gradient generated by analytical ultracentrifugation. The lipoprotein particles were stained with a fluorescent dye and then separated in the gradient over a range of d = 1.000-1.300 g · cm^3. After separation, the fluorescence of the lipoprotein particles was measured in an high-performance liquid chromatography–type flow system and normalized to a cholesterol scale with a proprietary algorithm. Values for each lipoprotein subgroup at their specific densities were determined using a multiple Gaussian fit/integration routine [37]. The coefficient of variation for this analysis using known standards is 2% to 3%. All blood variables expressed as concentrations were adjusted for plasma volume shifts that occurred in response to acute exercise, as described previously [38,39].

2.8. Diet records

Once during each of the 2 ground beef intervention phases and once during the washout period, participants completed a 4-day record (to include 1 weekend day) [40,41]. Participants were instructed to record all items consumed including supplements. The diet records were analyzed for nutrient composition to establish baseline observations and encourage compliance with the total patty consumption requirement. The records were analyzed using Nutritionist Pro (Axxya System, Strafford, TX, USA). Participants were instructed to resume their habitual meat consumption and maintain their habitual exercise levels during the washout period.

2.9. Statistical analyses

On the basis of data from our previous studies in which HDL-C was decreased 0.14 mmol/L by high-SFA ground beef [40] and increased 0.07 mmol/L by high-MUFA ground beef [41], compared with control (habitual) diets, power calculations were conducted to estimate the required sample size. Analyses used the following assumptions: power was set at 0.8, and α was set at .05. It was estimated that a sample size of 16 was sufficient to test the hypothesis that high-MUFA ground beef and/or exercise would increase HDL-C. Data were tested for unequal variance by the Breusch-Pagan/Cook-Weisberg test for heteroscedasticity (SAS version 9.1.2, Cary, NC, USA) to test the null hypothesis that the error variances were all equal.

Plasma and serum values after the dietary interventions and exercise sessions are means for n = 34 (entry plus washout) or n = 17 (postdietary intervention or postexercise). Entry values were compared with post–ground beef intervention values by paired t test (SuperAnova; Abacus Concepts Inc, Berkeley, CA, USA). Preexercise values after the 4-week washout or 6-week dietary interventions were compared with their respective postexercise values by paired t test. Associations among serum analytes were assessed using Pearson correlation coefficients. P values were considered significant at $P < .05$.

The only plasma fatty acids that were affected by either diet or exercise were palmitoleic acid (16:1(n-7)) and oleic acid (18:1(n-9)); other plasma fatty acids were not affected by diet or exercise and are not reported in tabular form. Serum analytes that were not affected by diet or exercise were as follows (baseline values, means ± SEM, n = 17): insulin (42.4 ± 1.9 pmol/mL), homeostatic model assessment score (1.45 ± 0.14), LDL III-C (0.50 ± 0.01 nmol/L), LDL-IV-C (0.17 ± 0.01 nmol/L), intermediate density lipoprotein cholesterol (0.69 ± 0.02 mmol/L), HDL_3-C (0.68 ± 0.01 mmol/L), RLP cholesterol (RLP-C; 0.95 ± 0.18 mmol/L), and Lp(a) cholesterol (0.66 ± 0.07 mmol/L). These data are not reported in tabular form.

3. Results

3.1. Study entry and final characteristics and energy expenditures

Body weights, body fat (as determined by dual-energy x-ray absorptiometry), and BMI did not change over the 16-week

course of the study (Table 2). Physical activity records reflected no significant changes in physical activity preexercise and postexercise (9.8-10.1 MJ/d). During the single exercise session, energy expenditure averaged 2.09 MJ (by design), and exercise duration averaged 78 minutes.

3.2. Nutrient intake

There were no significant differences in energy, protein, or carbohydrate intake among the low-MUFA, high-MUFA, or washout phases ($P \geq .16$; Table 3). During the low-MUFA ground beef interventions, participants consumed a greater amount of total MUFA, oleic acid, and cholesterol ($P < .05$). Total fat, MUFA, and oleic acid intakes were greater ($P < .01$) during the high-MUFA intervention. Intake of polyunsaturated fatty acids, linoleic acid (18:2(n-6)), and α-linolenic acid (18:3(n-3)) was lower during the low-MUFA ground beef intervention than at baseline, but intakes of eicosapentaenoic (20:5(n-3)) and docosahexaenoic acid (22:6(n-3)) were unaffected by the ground beef interventions. Saturated fat intake tended ($P = .07$) to be greater during the low-MUFA and high-MUFA phases than at baseline.

3.3. Diet effects on plasma and serum analytes

Low-density lipoprotein mean density increased after 6 weeks of the low-MUFA and high-MUFA ground beef interventions ($P < .05$; Table 4). Consumption of the high-MUFA ground beef increased plasma palmitoleic acid, oleic acid, and serum HDL_{2b}-C ($P < .05$); the low-MUFA ground beef did not significantly affect any of these analytes ($P > .10$), although numerically they were intermediate between the baseline and high-MUFA values.

Table 2 – Initial and final participant characteristics and habitual energy expenditure, exercise energy expenditure and duration, and mean plasma volume shift after an acute exercise bout

Item	Study period	
	Initial	Final
Age (y)	57.8 ± 1.7	–
Body weight (kg)	67.8 ± 6.2	68.1 ± 6.3
Body fat (%)		
Android fat	44.5 ± 2.7	43.9 ± 2.4
Gynoid fat	49.3 ± 1.4	49.5 ± 1.3
Total fat	41.6 ± 1.8	41.5 ± 1.7
BMI	25.5 ± 1.1	25.7 ± 1.1
	Exercise period	
	Preexercise	Postexercise
Habitual energy expenditure (MJ/d)	10.1 ± 0.3	9.8 ± 0.2
Exercise energy expenditure (MJ)	–	2.09 ± 0.04
Exercise duration (min)	–	78 ± 2
Mean plasma shift (%)	–	2.5 ± 1.0

Data are means ± SEM; n = 17 (initial, final, and preexercise) or n = 51 (postexercise, averaged over the washout, low-MUFA, and high-MUFA periods). There were no significant differences between periods ($P > .20$).

Table 3 – Total daily energy intake and intake of major nutrients for baseline and for test diets of postmenopausal women rotated through ground beefs low in MUFAs (low MUFA) or high in MUFAs (high MUFA)

Nutrient	Baseline	Low MUFA	High MUFA
Total energy (MJ/d)	6.66 ± 0.55	6.64 ± 0.41	6.99 ± 0.47
Protein (g/d)	67 ± 5	75 ± 5	75 ± 5
Carbohydrate (g/d)	189 ± 16	175 ± 15	187 ± 14
Fat (g/d)	44 ± 5	51 ± 3	55 ± 4*
Saturated fat (g/d)	20 ± 2	24 ± 2	24 ± 2
Monounsaturated fat (g/d)	15 ± 2	19 ± 1*	22 ± 2**
Polyunsaturated fat (g/d)	8 ± 1	6 ± 1*	8 ± 1
trans-Fat (g/d)	0.8 ± 0.2	1.1 ± 0.3	1.1 ± 0.2
Cholesterol (mg/d)	202 ± 21	283 ± 40*	240 ± 28
Oleic acid, 18:1(n-9) (g/d)	13 ± 2	17 ± 1*	19 ± 1**
Linoleic acid, 18:2(n-6) (g/d)	6 ± 1	5 ± 1*	6 ± 1
α-Linolenic acid, 18:3(n-3) (g/d)	0.7 ± 0.1	0.4 ± 0.1*	0.7 ± 0.1
Eicosapentaenoic acid, 20:5 (n-3) (g/d)	0.04 ± 0.02	0.07 ± 0.04	0.07 ± 0.04
Docosahexaenoic acid, 22:6 (n-3) (g/d)	0.05 ± 0.02	0.13 ± 0.07	0.13 ± 0.07

Data are means ± SEM (n = 17).
Saturated fat intake tended ($P = .07$) to be greater during the low MUFA and high MUFA phases than at baseline.
* $P < .05$, comparison of low MUFA or high MUFA to baseline, paired *t* test.
** $P < .01$, comparison of low MUFA or high MUFA to baseline, paired *t* test.

3.4. Exercise main effects

The exercise session decreased plasma oleic acid and serum TAG in the washout phase and after the high-MUFA ground

Table 4 – Diet main effects for postmenopausal women after consuming test ground beefs low in MUFAs (low MUFA) or high in MUFAs (high MUFA)

Item	Baseline	Low MUFA	High MUFA
TAG (mmol/L)	1.21 ± 0.13	1.12 ± 0.11	1.17 ± 0.12
Palmitoleic acid (mmol/100 mmol)	1.04 ± 0.20	1.10 ± 0.23	1.16 ± 0.26*
Oleic acid (mmol/100 mmol)	19.3 ± 0.5	19.5 ± 0.5	20.0 ± 0.6*
LDL-C (mmol/L)	3.07 ± 0.15	3.19 ± 0.14	3.23 ± 0.16
LDL mean density (g/cm^3)	1.029 ± 0.001	1.030 ± 0.001*	1.030 ± 0.001*
LDL mean size (nm)	20.211 ± 0.031	20.171 ± 0.022	20.199 ± 0.024
HDL-C (mmol/L)	1.51 ± 0.06	1.60 ± 0.06	1.62 ± 0.07*
HDL_{2a}-C (mmol/L)	0.23 ± 0.02	0.23 ± 0.02	0.24 ± 0.02
HDL_{2b}-C (mmol/L)	0.64 ± 0.05	0.67 ± 0.03	0.69 ± 0.05*
HDL mean density (g/cm^3)	1.093 ± 0.001	1.092 ± 0.001	1.091 ± 0.001
VLDL-C (mmol/L)	0.31 ± 0.03	0.34 ± 0.04	0.35 ± 0.05

Data are means ± SEM; n = 34 (baseline) or n = 17 (low MUFA or high MUFA). Baseline data were collected at entry and after 4-week washout. No baseline or washout values were different ($P > .25$). Low and high MUFA values were compared with respective baseline values.
* $P < .05$, paired *t* test.

beef intervention ($P < .05$). Low-density lipoprotein cholesterol increased postexercise in the washout phase and after the low-MUFA intervention (Table 5; $P < .05$). Both HDL-C and HDL_{2a}-C were increased by the exercise session during the washout phase but were decreased by the exercise session after the high-MUFA ground beef intervention ($P < .05$). Low-density lipoprotein density and VLDL-C were decreased and LDL mean size and HDL mean density were increased by the exercise session after the low-MUFA ground beef intervention.

3.5. *Simple correlations*

Simple correlations were calculated for measurements taken over the course of the study (Table 6). Low-density lipoprotein cholesterol, VLDL-C, RLP-C, insulin, palmitoleic acid, and oleic acid were positively correlated ($P < .05$) with serum TAG, whereas HDL-C and LDL size were negatively correlated ($P < .01$) with serum TAG. Very low-density lipoprotein cholesterol, RLP-C, HDL density, insulin, palmitoleic acid, and oleic acid were positively correlated ($P < .05$) with serum LDL-C. Very low-density lipoprotein cholesterol, insulin, and oleic acid were negatively correlated with serum HDL-C, whereas LDL density was positively correlated with HDL-C ($P < .05$). Remnant lipoprotein cholesterol, insulin, and oleic acid were positively correlated with VLDL-C ($P < .05$). Insulin and oleic acid were positively correlated with RLP-C, and LDL density was negatively correlated with RLP-C ($P < .05$). Neither HDL density nor LDL density was correlated with serum insulin, palmitoleic acid, or oleic acid.

4. Discussion

The reported energy intake from the dietary records was nearly 40% less than reported energy expenditure. This likely arose from underreporting food intake and overreporting actual physical activity. Regardless, the diet and activity records indicate that neither energy intake nor expenditure changed over the course of the study.

The single session of intense exercise increased the HDL-C concentration during the washout period, in which habitual diets were consumed. This is similar to previous reports that a single session of intense exercise increases HDL-C in postmenopausal women [11,20]. Similarly, the high-MUFA ground beef increased the concentration of HDL-C, consistent with results from our laboratory in which high-MUFA ground beef but not low-MUFA ground beef significantly increased HDL-C in hypercholesterolemic [40] and normocholesterolemic men [41]. Contrary to our initial hypothesis, intensive exercise did not work additively with high-MUFA ground beef consumption to increase HDL-C or to affect any other humoral risk factors for CVD. Rather, the increase in HDL-C elicited by the high-MUFA ground beef was eliminated by the exercise session, thus indicating that exercise and diet increase HDL-C by different and perhaps antagonistic mechanisms.

Increased intakes of myristic and palmitic acid increased LDL-C and HDL-C concentrations, whereas intake of oleic acid (in the form of vegetable oils) decreased LDL-C but increased HDL-C concentrations (reviewed in Kris-Etherton and Yu [25]). In this study, the high-MUFA ground beef increased HDL-C, a change that is associated with increased intake of oleic acid. Dietary intake records indicated that participants consuming the high-MUFA ground beef had the greatest intake of oleic acid.

The increase in LDL-C concentration caused by the single session of exercise has not been reported previously. This change occurred after the washout period and the low-MUFA ground beef period, but not after the high-MUFA ground beef period. Wooten et al [29] reported no effect of a single bout of aerobic exercise on LDL-C concentration or particle size in sedentary, eumenorrheic women. Baumstark et al [42] reported that acute exercise reduced dense LDL subfractions in endurance-trained men. In the current study, LDL III and LDL IV cholesterol concentrations were not affected by diet or

Table 5 – Exercise effects for postmenopausal women after consuming test ground beefs low in MUFAs (low MUFA) or high in MUFAs (high MUFA)

Item	Washout		Low MUFA		High MUFA	
	Pre-Ex	Post-Ex	Pre-Ex	Post-Ex	Pre-Ex	Post Ex
TAG (mmol/L)	1.24 ± 0.12	1.08 ± 0.11**	1.12 ± 0.11	1.01 ± 0.11	1.17 ± 0.12	1.02 ± 0.07*
Palmitoleic acid (mmol/100 mmol)	1.13 ± 0.25	0.97 ± 0.18	1.10 ± 0.23	1.00 ± 0.21	1.16 ± 0.26	1.01 ± 0.21*
Oleic acid (mmol/100 mmol)	19.8 ± 0.5	18.6 ± 0.4*	19.5 ± 0.5	19.6 ± 0.5	20.0 ± 0.6	18.6 ± 0.4**
LDL-C (mmol/L)	3.05 ± 0.16	3.30 ± 0.16**	3.19 ± 0.14	3.39 ± 0.18*	3.23 ± 0.16	3.27 ± 0.18
LDL mean density (g/cm^3)	1.029 ± 0.001	1.029 ± 0.001	1.030 ± 0.001	1.028 ± 0.001**	1.030 ± 0.001	1.030 ± 0.001
LDL mean size (nm)	20.216 ± 0.028	20.216 ± 0.025	20.171 ± 0.022	20.269 ± 0.027**	20.199 ± 0.024	20.197 ± 0.026
HDL-C (mmol/L)	1.49 ± 0.06	1.59 ± 0.05**	1.60 ± 0.05	1.56 ± 0.06	1.62 ± 0.07	1.51 ± 0.06*
HDL_{2a}-C (mmol/L)	0.20 ± 0.02	0.25 ± 0.02*	0.23 ± 0.02	0.22 ± 0.02	0.24 ± 0.02	0.21 ± 0.01*
HDL_{2b}-C (mmol/L)	0.62 ± 0.05	0.66 ± 0.04	0.67 ± 0.03	0.65 ± 0.04	0.69 ± 0.05	0.67 ± 0.02
HDL mean density (g/cm^3)	1.088 ± 0.005	1.093 ± 0.001	1.092 ± 0.001	1.094 ± 0.001**	1.091 ± 0.001	1.092 ± 0.001
VLDL-C (mmol/L)	0.32 ± 0.03	0.31 ± 0.05	0.34 ± 0.04	0.27 ± 0.03**	0.35 ± 0.05	0.32 ± 0.04

Data are means ± SEM; n = 17. Washout data were collected after 4 weeks of consuming habitual diets, immediately before the exercise session. The low MUFA and high MUFA pre-ex data are the postground beef intervention data from Table 4. Pre-Ex, preexercise; Post-Ex, postexcercise.
* $P < .05$, paired t test.
** $P < .01$, paired t test.

Table 6 – Pearson correlations among plasma metabolites, fatty acids, and lipoprotein fractions in postmenopausal women at baseline, after ground beef interventions, and after an acute session of exercise

Plasma item	TAG	LDL-C	HDL-C	VLDL-C	RLP-C	HDL density	LDL density
TAG	–						
LDL-C	0.32 **	–					
HDL-C	–0.35 **	–0.01	–				
VLDL-C	0.69 ***	0.47 ***	–0.23 *	–			
RLP-C	0.44 **	0.58 ***	–0.11	0.83 ***	–		
HDL density	0.09	0.21 *	–0.17	0.07	0.08	–	
LDL density	0.12	–0.18	0.20 *	0.02	–0.25 *	–0.09	–
LDL, nm	–0.30 **	0.09	0.12	–0.12	0.16	0.12	–0.12
Insulin	0.65 ***	0.30 **	–0.21 *	0.53 ***	0.39 **	0.16	–0.06
Palmitoleic acid	0.23 *	0.45 ***	–0.12	0.21 *	0.09	0.19	0.05
Oleic acid	0.45 ***	0.25 *	–0.28 *	0.37 **	0.22 *	–0.02	–0.10

Values are simple correlation coefficients; n = 119.
* $P \leq .05$.
** $P \leq .01$.
*** $P \leq .001$.

exercise, so we concluded that the increase in total LDL-C represented modification of the larger, less dense LDL fraction. However, the magnitude of increase in LDL-C was small (approximately 0.20 mmol/L, or 8 mg/dL) and, thus, would have little physiological significance.

Small, dense LDLs are considered a risk factor for CVD because this form of LDL is more susceptible to oxidative damage [43,44]. We previously reported that a low-MUFA ground beef intervention decreased LDL particle size in hypercholesterolemic men [40] but had no effect on LDL size in the normocholesterolemic men [41] and postmenopausal women of this study. It is noteworthy that exercise increased LDL size after consumption of the low-MUFA ground beef. Because chub pack ground beef is the least expensive and most commonly consumed form of ground beef in the United States (reviewed in Gilmore et al [41]), the data suggest that a single session of exercise would be most beneficial to sedentary individuals in which ground beef is a significant component of their diet. The increase in LDL particle size caused by exercise was small (approximately 0.10 nm); Kratz et al [45] reported that, relative to a diet high in saturated fat, diets high monosaturated and polyunsaturated oils decreased LDL size by approximately 0.3 nm. It is unknown if changes of this magnitude would significantly influence the risk for CVD.

A unique finding of this study is that the high-MUFA ground beef increased HDL_{2b}-C, which returned to baseline values after acute exercise. There is convincing evidence that, of the subclasses of HDL particles, buoyant HDL_{2b} is cardioprotective [46,47]. Patients with premature CVD have reduced HDL_{2b} [47], and families with low HDL_{2b} have increased carotid intima-media thickness (associated with CVD) [46]. Thus, high-MUFA ground beef not only increased HDL-C concentration, it also increased the most cardioprotective subclass of HDL particles. In contrast, exercise increased HDL_{2a}-C without significantly affecting HDL_{2b}-C. Furthermore, although exercise decreased total HDL-C after the high-MUFA ground beef intervention, it did not affect HDL_{2b}-C. The differential effects of high-MUFA ground beef and exercise on HDL_{2a}-C and HDL_{2b}-C provide the most convincing evidence that diet and exercise affect HDL-C by different mechanisms.

By design, the exercise sessions occurred 2 days after the completion of the ground beef interventions. Therefore, we cannot rule out the possibility that some portion of the decline in HDL-C postexercise in the high-MUFA may have been caused by removal of ground beef from the diet. However, the other changes in serum analytes caused by the high-MUFA ground beef (LDL density and HDL_{2b}-C) did not return to baseline after exercise, and HDL_{2a}-C concentrations were decreased by exercise in the high-MUFA group. We conclude that the postexercise decline in HDL-C and plasma MUFA was caused by exercise rather than resumption of their habitual diets.

It is a limitation that this study provided little information about the mechanism of action for the interactions between diet and exercise. Both the low-MUFA and high-MUFA ground beef interventions increased LDL particle density, suggesting that some component of the ground beef increased cholesterol ester transferase protein (CETP) activity. In an earlier study, a diet high in oleic acid decreased CETP activity in women [48], relative to a diet high in palmitic acid, which should decrease the exchange of CE from HDL to VLDL and LDL. However, HDL-C was unaffected by the high-oleic acid diet [48] indicating that in that group of women, CETP activity was not associated with HDL-C metabolism. In the current study, HDL-C concentrations and LDL density were significantly, positively correlated (r = 0.20, $P < .05$); that is, increased HDL-C was associated with a higher LDL particle density. This was clearly evident in the effects of the high-MUFA ground beef, which increased both HDL-C and LDL density. This argues against a role of CETP in regulating HDL-C concentrations, at least in response to the dietary interventions of this study because elevated CETP activity would have concomitantly decreased HDL-C and increased LDL particle density. In addition, a previous study from our laboratory [21] demonstrated no change in CETP activity with acute exercise in postmenopausal women, thus indicating that any changes in HDL-C in response to a single session of intense exercise were unrelated to CETP activity.

We previously reported that a similar, acute exercise session reduced TAG but had no effect on lipoprotein lipase activity in normocholesterolemic, postmenopausal women [21]. This would also seem to rule out lipoprotein lipase as

the basis for the decreases in serum TAG caused by exercise observed in this study. We have demonstrated highly significant, positive correlations between the plasma palmitoleic acid and oleic acid and plasma TAG in men [40,41], and similar results were observed in this study. In addition, palmitoleic acid and oleic acid were positively associated with LDL-C and VLDL-C concentrations, and oleic acid was positively associated with RLP-C and negatively associated with HDL-C concentrations. These correlations among plasma MUFA and lipoprotein cholesterol fractions suggest that decreased hepatic stearoyl-CoA desaturase (SCD) activity was responsible for the decreases in plasma oleic acid. SCD activity supports TAG synthesis in humans and mice [49,50], and hepatic SCD activity is increased by dietary stearic acid (18:0) and depressed by dietary oleic acid in mice [51]. The increase in plasma palmitoleic acid and oleic acid caused by the high-MUFA ground beef may have been caused by increased hepatic SCD activity, but this was not accompanied by changes in serum TAG. However, exercise decreased oleic acid and TAG during the washout and high-MUFA phases, thereby suggesting that exercise may work, in part at least, to depress plasma TAG through a reduction in hepatic SCD activity.

In normocholesterolemic men, high-MUFA ground beef decreased plasma insulin [41], an effect not observed in this study. However, insulin concentrations were significantly, positively correlated with TAG, LDL-C, VLDL-C, and RLP-C and were negatively associated with HDL-C. Schwarz et al [52] reported that insulin promoted hepatic de novo lipogenesis in humans [52], and the high correlation between serum insulin and TAG and VLDL-C concentrations is consistent with their findings. However, we cannot conclude if the significant, negative relationship between insulin and HDL-C indicates a causative role of insulin in HDL metabolism.

In conclusion, this study duplicated the ability of a single session of aerobic, intense exercise to increase HDL-C concentrations in postmenopausal women. In addition, high-oleic acid ground beef increased HDL-C concentrations, as seen in previous studies from our laboratory. Based on the concomitant reductions in oleic acid and TAG postexercise, we conclude that exercise decreases plasma TAG, in part by depressing hepatic SCD activity. Rather than the effects of exercise being additive (as initially hypothesized), the single session of acute exercise attenuated the increase in HDL-C concentrations elicited by the high-oleic acid ground beef. The basis for this antagonism between acute exercise and the dietary-induced increase of HDL-C is unknown, but the differential effects of dietary MUFA and exercise on HDL_{2a}-C and HDL_{2b}-C concentrations indicate that diet and exercise influence HDL-C by different mechanisms.

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