Resistance Training Reduces Subclinical Inflammation in Obese, Postmenopausal Women

MELODY D. PHILLIPS, ROBERT M. PATRIZI, DENNIS J. CHEEK, JOSHUA S. WOOTEN, JAMES J. BARBEE, and JOEL B. MITCHELL

Exercise Physiology Laboratory, Texas Christian University, Fort Worth, TX; Nursing Research Laboratory, Texas Christian University, Fort Worth, TX; Institute for Women’s Health, Texas Woman’s University, Denton, TX; and JPS Health Network, Division of Primary Care Sports Medicine, Arlington, TX

ABSTRACT

PHILLIPS, M. D., R. M. PATRIZI, D. J. CHEEK, J. S. WOOTEN, J. J. BARBEE, and J. B. MITCHELL. Resistance Training Reduces Subclinical Inflammation in Obese, Postmenopausal Women. Med. Sci. Sports Exerc., Vol. 44, No. 11, pp. 2099–2110, 2012. Purpose: Aerobic exercise is frequently prescribed to reduce inflammatory-related disease (cardiovascular disease and diabetes) risk. Resistance training (RT), however, may be key to maximizing anti-inflammatory benefits of consistent exercise. We examined the influence of RT on inflammatory biomarkers in obese, postmenopausal women. Methods: Twenty-three women (65.6 ± 2.6 yr; body mass index, 33 kg·m⁻²) underwent 12 wk of RT (3 sets, 10 exercises, 3 × per week, 8–12 repetition maximum (RM), resistance exercise (EX), N = 11) or social interaction intervention (SI, stretching, knitting, health lectures, 2 × per week, control group (CON), N = 12). Both before (BT) and after (AT) RT or SI, blood was collected before (PR), immediately (PO), 2 h (2H), and 24 h (24H) after a single resistance exercise bout (RE) in EX and at the same time points in nonexercise, resting CON. For all time points, blood was analyzed for IL-6, leptin, and lipopolysaccharide (LPS)-stimulated tumor necrosis factor-α (TNF-α) (LPS-TNF) and IL-10 (LPS-IL10). PR samples were also examined for C-reactive protein, TNF-α, and adiponectin, and mRNA expression of toll-like receptor 4 (TLR4) and MC1R. Subcutaneous adipose tissue was extracted BT and AT and analyzed for mRNA expression of monocyte chemotactic protein-1, leptin, CD68, and TLR4. Results: RT improved strength (44%) and reduced circulating C-reactive protein (−33%), leptin (−18%), and TNF-α (−29%) with no change in body composition. IL-6 decreased after SI in CON (−17%). LPS-TNF increased after SI or RT (CON +26%, EX +67%, respectively), whereas LPS-IL10 decreased in CON (−28%) but increased in EX (−20%). RT did not influence inflammatory biomarker gene expression in whole blood or subcutaneous adipose tissue. A single RE bout augmented LPS-TNF and LPS-IL10 at 24H in EX, particularly AT. Conclusion: RT reduced markers of subclinical inflammation in circulation in obese, postmenopausal women in the absence of changes in body composition. Chronic RT also enhanced response to endotoxin challenge both at rest (PR) and 24 h after an acute RE bout (24H). Key Words: CRP, IL-6, TNF-α, LEPTIN, IL-10, LIPOPOLYSACCHARIDE (LPS), ADIPOSE TISSUE

A significant body of literature exists regarding the influence of aerobic exercise training on subclinical systemic inflammation in various populations including those who are obese and/or elderly (1,4,9,23,35,42). Less is known about consistent resistance training (RT) or acute resistance exercise in those who are at risk for inflammatory-related diseases or conditions such as the metabolic syndrome. Obesity contributes significantly to the development of the metabolic syndrome and to diseases such as Type II diabetes mellitus (T2D) and cardiovascular disease (CVD) (32). Age- and inactivity-related diseases (CVD, T2D, and osteoporosis) are associated with elevations in biomarkers of inflammation such as C-reactive protein (CRP) and a variety of inflammatory-related cytokines, adipokines, and other acute phase reactants (26). Moreover, elevated interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and leptin have been linked to obesity, insulin resistance, endothelial dysfunction, and CVD (25,30,40).

Obesity alone, in the absence of overt disease, is frequently accompanied by increased levels of inflammatory markers and adipokines, and low-level systemic inflammation appears to increase with age. A group with a significant risk of developing inactivity-related diseases is postmenopausal women (33). The reduction in circulating sex hormones that occurs with menopause is commonly associated with increased pro-inflammatory activity (33). Furthermore, menopause is associated with increased adiposity predisposing women to obesity, which contributes to systemic inflammation because adipose tissue is known to act as an
endocrine organ secreting a variety of adipokines that are pro-inflammatory, including leptin, TNF-α, IL-6, and CRP (33). In addition, adiponectin, an anti-inflammatory adipokine (43,48), is reduced with increasing adiposity and insulin resistance (40). Weight gain and adiposity are associated with macrophage accumulation in adipose tissue and increased expression of pro-inflammatory proteins (47); therefore, the inflammatory profile of adipose tissue is heightened in the obese state (4,47). Activated macrophages release cytokines and chemokines including monocyte chemotactic protein-1 (MCP-1), which is significantly correlated with body mass index (BMI) (10). In fact, a significant portion of some pro-inflammatory adipokines released from adipose tissue can be attributed to infiltrating monocytes/macrophages (47). Diet and exercise reduce macrophage infiltration, evidenced by reductions in CD68, and expression of pro-inflammatory cytokines (6,7).

Macrophages and monocytes respond to some inflammatory stimuli via the pattern recognition receptor toll-like receptor 4 (TLR4), which, with CD 14, makes up the binding receptor for lipopolysaccharide (LPS) from Gram-negative bacteria. Stimulation of macrophages and monocytes via TLR4 induces a potent inflammatory response, and TLR4 activation is greater in obese persons compared with non-obese controls (39). The significance of these data is that the pro-inflammatory milieu in adipose tissue of obese individuals promotes insulin resistance, endothelial dysfunction, and atherosclerosis (28) and does so partly through TLR4 (32).

Consistent exercise training (aerobic and/or resistance) has reduced markers of inflammation in various tissues including CRP (1,2,9,14,23,41,46), IL-6 (2,12,23,25,35), TNF-α (2,23,25,35), leptin (2,15,38), and TLR4 (16,25), whereas adiponectin (2,15) and the anti-inflammatory cytokine, interleukin-10 (IL-10) (2), have increased after training. Many scientists conducting longitudinal, prospective studies have used aerobic exercise or a combination of aerobic and resistance exercise (1,9,12,41) in elderly women, we hypothesized that RT would reduce gene expression of melanocortin 3 receptor (MC3R) or if relationships exist between their expression and exercise-induced improvements of traditional markers of inflammation.

Cross-talk occurs among tissues such as muscle, adipose, hepatic, blood, and others via cytokines, chemokines, and acute phase reactants. Exercise-induced alterations in this cross-talk likely influence low-level systemic inflammation; therefore, it is of value to characterize the effects of both acute and chronic exercise on various inflammatory responses (from tissue, blood, and in vitro culture) concomitantly to better understand the physiology behind the benefits of RT. It is particularly important to understand the effects of RT in obese persons or those at risk for the metabolic syndrome because RT is becoming recognized as a nonpharmacological therapeutic modality in the treatment and prevention of obesity and related disorders (44). In addition to the actual tissue adaptations that may be produced by consistent RT on resting variables, it is also key to evaluate the acute responses to resistance exercise because, in many cases, acute exercise produces lasting effects that persist for a day or more. As a result of the acute exercise-induced responses, a chronic beneficial effect may occur that is not truly a long-term adaptation but instead is the persistent effect of multiple acute bouts that are chained together over days and weeks of regular exercise. It is also important to study the acute responses to RT because the nature of the acute response itself may be altered as a result of prolonged, regular exercise.

The purpose of this project was to investigate the influence of 12-wk resistance exercise training (RT) at 8 RM on inflammatory markers associated with inactivity-related diseases in obese, postmenopausal women, a population at risk for the metabolic syndrome. A second purpose was to examine the acute effects of a single bout of resistance exercise on selected inflammatory markers before and after training. On the basis of previous observations in normal weight, young, and elderly women, we hypothesized that RT would result in positive changes in inflammatory markers in circulation and LPS-stimulated cytokine production in vitro. In addition, we hypothesized that RT would reduce gene expression of inflammatory markers in blood and subcutaneous adipose tissue (SCAT). We also hypothesized that an acute bout of resistance exercise would increase plasma IL-6 immediately postexercise and result in a suppression of LPS-stimulated cytokine production during recovery. Finally, we hypothesized that RT would reduce muscle contraction-induced IL-6 release after a single exercise bout.

**METHODS**

Participants and screening. Participants were obese, postmenopausal women (60–70 yr; BMI, 30–40 kg m−2; N = 23) that had not participated in regular exercise for the previous 6 months (Table 1). Postmenopausal was defined...
as not experiencing a menstrual cycle for five or more years as a result of natural or surgical menopause. Participants reviewed and signed an informed consent form and obtained written approval from their personal physician before all pretesting procedures. Participants underwent a medical screening by our study physicians, which included a review of their medical history and a physical examination to identify musculoskeletal or flexibility limitations and a screening for dementia.

Exclusion criteria included the following: use of hormone replacement therapy or selective estrogen receptor modulators, severe arthritis, bedridden within 3 months of the study, central or peripheral nervous system disorders, previous stroke, acute or chronic infection, major affective disorder, HIV infection or autoimmune disorders, metabolic disorders (Type I diabetes mellitus or T2D), smokers or smokeless tobacco users, regular aerobic or RT within the previous 6 months, oral steroid use, or surgery within the previous 3 months. Regular nonsteroidal anti-inflammatory drug users were asked to refrain from taking their medication until after the experimental trials on test days. Potential participants with contraindications to exercise testing or characterized as high risk, based on the American College Sports Medicine guidelines, were excluded from participation. This study was approved by the institutional review boards at Texas Christian University (TCU) and JPS Health Network in Fort Worth, TX.

**Preliminary testing and group assignment.** Body mass was measured to the nearest 0.1 kg. BMI (kg m\(^{-2}\)) was calculated by dividing body mass (kg) by height squared (m\(^2\)). In addition, body density was estimated using a seven-site skinfold procedure, and percentage body fat was estimated from body density using the Siri equation. Skinfold measures were performed by three female technicians. The same technician performed a participant’s skinfold measures both BT and AT in 85% of cases. Before the study, the female technicians trained together to ensure repeatability among technicians and to reduce intertechnician variability.

Those cleared for participation completed a submaximal, graded exercise test on a treadmill up to 80% of their age-predicted maximum heart rate as a prescreening procedure. Participants exercised under continuous ECG and blood pressure monitoring with a study physician present. Participants were randomized while attempting to match for age, BMI, and medication use into either a resistance exercise (EX) or a nonexercise control group (CON). Number of participants taking medications related to the metabolic syndrome were approximately equal in each group: statins for hypercholesterolemia (EX = 5, CON = 5), nonsteroidal anti-inflammatory drug (EX = 5, CON = 6), and hypertension (EX = 4, CON = 7). Hypertension medications included primarily angiotensin-converting enzyme inhibitors and thiazide diuretics. Two CON participants were taking cardioselective β-blockers. The number of participants having had a hysterectomy was equal between groups (EX = 6, CON = 6).

After an overnight fast (10 h), approximately 400 mg of SCAT was obtained from the abdominal region at the level of the umbilicus using a minimally invasive percutaneous miniliposuction method (3). Tissue was cleaned with saline and snap frozen in liquid nitrogen.

**Acclimation.** Both groups completed a week-long acclimation period to the following resistance exercises: chest press, “lat” pull-down, seated rows, shoulder press, leg abduction, leg adduction, chest flys, leg press, and leg curl, and leg extension. The participants performed three acclimation exercise sessions on consecutive days. On acclimation day 1, participants were taught proper lifting techniques and each subject’s 8 RM was assessed for each of the 10 resistance exercises. On the second acclimation day, participants performed three sets of each exercise at 50% of their estimated 1 RM. During the first two sets, participants performed eight repetitions, and the last set was to volitional fatigue (failure to complete a repetition using proper form through a full range of motion). Eight RM for all exercises was reassessed on acclimation day 3. Nonexercise CON participants were reacclimated, and 8 RM was reassessed for each person after the 12-wk intervention. Acclimation and 8-RM testing was conducted in CON before and after the social interaction intervention to ensure that strength between groups was similar at baseline and to account for seasonal variations on activity level, which may affect strength. All acclimation and resistance exercise sessions were preceded by 5–10 min of cycle ergometry or walking on an indoor track and were performed in the TCU Recreation Center using Nautilus (Vancouver, WA) and Cybex equipment (Medway, MA) under the supervision of trained exercise technicians.

**Intervention.** EX completed 12 wk of resistance exercise training (RT) on nonconsecutive days (3 sets of 10 exercises, 3 d wk\(^{-1}\), at 8 RM). The first two sets included eight repetitions, and the last set was to fatigue. All exercises were performed in pairs (listed in the sequence mentioned previously) where each set of each pair was alternated until three

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**TABLE 1. Descriptive data.**

<table>
<thead>
<tr>
<th>Variable</th>
<th>BT (EX = 11)</th>
<th>AT (EX = 11)</th>
<th>CON (AT = 12)</th>
<th>AT (CON = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>64.8 ± 2.4</td>
<td>—</td>
<td>66.4 ± 2.8</td>
<td>—</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>161.3 ± 5.5</td>
<td>—</td>
<td>159.6 ± 7.7</td>
<td>—</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>83.6 ± 8.8</td>
<td>83.2 ± 8.5</td>
<td>86.0 ± 13.4</td>
<td>86.1 ± 12.8</td>
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<tr>
<td>Percentage body fat</td>
<td>35.1 ± 2.7</td>
<td>34.8 ± 2.3</td>
<td>36.4 ± 3.3</td>
<td>36.0 ± 3.0</td>
</tr>
<tr>
<td>BMI (kg m(^{-2}))</td>
<td>32.2 ± 3.3</td>
<td>32.0 ± 3.5</td>
<td>33.7 ± 4.4</td>
<td>33.7 ± 4.0</td>
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<tr>
<td>Fat-free mass (kg)</td>
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<td>54.9 ± 7.0</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
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<td>29.0 ± 4.3</td>
<td>31.6 ± 6.9</td>
<td>31.2 ± 6.5</td>
</tr>
</tbody>
</table>

Descriptive data before (BT) and after (AT) 12 wk of RT in the exercise group (EX) or social interaction in the CON. Values are mean ± SD.
sets of the two exercises were completed (e.g., pair one = chest press and “lat” pull-down). This protocol was followed for all resistance exercise training sessions and for experimental trials. During training, if the participant was able to perform 12 or more repetitions on the third set, the resistance was increased on the following exercise day. CON participants did not undergo RT but reported to the laboratory twice per week for 1-h supervised “control” activities (stretching, health education and safety talks, and activities such as playing chess or knitting) to control for potential psychosocial effects of consistent social interaction. All participants were asked to maintain their normal diet during the training period and were reminded of this weekly. During training, three EX participants complained of joint discomfort during upper body exercises, so those exercises causing discomfort (chest press, shoulder press, and seated row) were replaced with alternate exercises (cable press, abdominal crunches, and back extensions).

**Experimental trials.** Participants completed an experimental trial at least 1 wk after the last acclimation or exercise session both before (BT) and after (AT) the training or control interventions. The experimental trials consisted of either a single resistance exercise bout (EX group) or a resting control trial (CON group) where CON participants sat quietly in the laboratory. The resting control trial in CON was performed to account for diurnal variations. Blood samples were obtained immediately preexercise (PR), within 4 min postexercise (PO), and then 2 (2H) and 24 (24H) h postexercise (same time of day for resting CON participants). Participants reported to the laboratory, after an overnight fast (10 h), between 0530 and 0730 and rested quietly for at least 15 min before the PR blood sample was obtained. EX then performed 3 sets of the 10 exercises at their 8 RM for each exercise, whereas CON rested quietly in the laboratory for the control trial. At least 1.5 min of recovery was allowed between sets with 2-min recovery between exercises. The total duration of the exercise bouts, including 5–10 min of walking warm-up, was approximately 75 min. After the exercise bout (EX group) or resting control session (CON group), all participants rested quietly in the laboratory for 2 h and were given water ad libitum. Total volume load (TVL) was calculated for each experimental trial (no. of repetitions × no. of sets × resistance [kg]) in EX.

The miniliposuction procedure was repeated after the intervention period (AT), 48 h after the last exercise session in EX and 1 wk after the 8 RM assessment in CON. Participants were asked to refrain from alcohol consumption 24 hours prior to any blood or tissue sampling and recorded all food consumed during the 24 h before the BT experimental trial, which represented a “normal” day’s diet. After the 12-wk intervention, they were given a copy of their food logs and asked to consume the same meals and record dietary intake during the 24 h before the AT experimental trial to help standardize macronutrient and caloric intake.

**Blood sample treatment and analysis.** During the experimental trials, blood was drawn into prechilled tubes containing ethylenediaminetetraacetic acid and room temperature ethylenediaminetetraacetic acid and sodium heparin tubes. One milliliter of whole blood from prechilled tubes was stored at −80°C for gene expression analysis. The prechilled tubes were centrifuged (1500g, 4°C, 10 min), and the plasma was stored at −80°C until analyzed for IL-6, TNF-α, leptin, adiponectin, and CRP. IL-6 and leptin were assayed BT and AT at all time points (PR, PO, 2H, and 24H), whereas TNF-α, adiponectin, and CRP were examined at PR only. Total leukocyte number and a five-part differential count were measured at all time points using an ACT-Diff 5 Beckman-Coulter hematology analyzer (Hillaeh, FL).

Cytokine production was assessed by diluting 0.5 mL of whole blood from room temperature sodium heparin tubes into 4.5-mL culture medium (RPMI supplemented with penicillin (100 U/mL) and streptomycin (100 μg/mL)) (Sigma Diagnostics, St. Louis, MO). Two milliliters of diluted whole blood samples were placed in 24-well flat-bottomed plates and stimulated with the polyclonal activator LPS (LPS from Salmonella enteritidis; final concentration, 25 μg/mL; Sigma Diagnostics). After a 24-h incubation (37°C, 5% CO2), the culture plates were centrifuged (800g, 4°C, 10 min) and supernatants were harvested and stored at −80°C until further analysis. TNF-α (LPS-TNF) and interleukin-10 (LPS-IL10) supernatant concentrations from LPS-stimulated whole blood cultures were analyzed for all time points using enzyme-linked immunosorbent assay (ELISA) (Invitrogen, Carlsbad, CA). Supernatants were diluted in assay solution before ELISA analysis (TNF-alpha, 1:11; IL-10 1:2) so that sample concentrations would fit the standard curve. LPS-stimulated cytokine concentration in supernatant (pg/mL) was corrected for number of monocytes in each culture well to give final cytokine production (femtograms per monocyte). Plasma IL-6 and TNF-α concentrations were assayed using high-sensitivity ELISA kits (R&D Systems, Minneapolis, MN), CRP (Invitrogen), leptin (R&D Systems), and adiponectin (Millipore, St. Charles, MO) were also assessed using ELISA as per manufacturer’s instructions.

**Real-time reverse transcription polymerase chain reaction.** Total RNA was isolated from SCAT using TRIzol and from whole blood using TRIzol LS as per the manufacturer’s instructions, and followed by column purification (SCAT: RNeasy lipid tissue mini kit; whole blood: RNeasy mini kit; Qiagen, Valencia, CA). Real-time reverse transcription polymerase chain reaction was completed on each sample in duplicate using TaqMan gene expression assays for the melanocortin 1 receptor (MC1R, Hs00267168_s1), TLR4 (Hs00152939_m1), leptin (Hs00174877_m1), MCP-1 (CCL2, Hs00234140_m1), and CD 68 (Hs00154355_m1; Applied Bioscience, CA). Gene expression of MC1R and TLR4 was examined in whole blood while that of TLR4, leptin, MCP-1, and CD68 were examined in SCAT. Samples were analyzed on the ABI 7500 platform using the one-step method. All Ct values were normalized to 18S (Hs99999901_s1), and fold change (FC) was calculated using the delta delta Ct method. Relative FC for each variable was
calculated as each subject’s AT Ct value relative to their BT Ct. Melanocortin receptor 3 (MC3R) gene expression in whole blood was also assessed in a subgroup (N: EX = 9, CON = 10) of our participants and has been reported elsewhere [20].

**Statistical analysis.** Statistical analyses were conducted using a two (group × training) or three-factor (group × training × exercise time point) ANOVA with repeated measures on the within factors. The between-subjects factor was group (EX and CON), and the within factors were training time point (before (BT) and after (AT) training/intervention), and exercise time point (PR, PO, 2H, 24H). A Shapiro–Wilk test was used to ensure normality of the data. Mauchly’s test of sphericity was also used. If the sphericity test confirmed that the covariance assumption was not satisfied, the Huynh–Feldt adjustment was used to correct degrees of freedom. When the ANOVAs detected significant main effects (P < 0.05), pairwise comparisons using Bonferroni adjustments were used to determine where the differences were located. When the ANOVAs identified significant interactions (P < 0.05), differences were located using the Student Newman–Keuls test post hoc. Pearson product–moment correlations were used to compare relationships between strength and dependent variables. Although appropriate adjustments for multiple comparisons were made for each analysis, it should be mentioned that there is an increased risk of a Type I error when examining multiple variables in a small sample size, particularly in multiple correlational analyses. Statistical analyses were performed using IBM SPSS statistical analysis software (version 19.0, Chicago, IL). Descriptive information is expressed as mean ± SD and dependent variables are presented as means ± SE. Threshold for significance was set at P < 0.05.

**RESULTS**

**Body composition, strength, and circulating biomarkers.** Twelve weeks RT had no significant effect on body mass or body composition (Table 1). Eight RM did not differ between groups before the intervention. It increased significantly for each of the 10 exercises after training in EX (P < 0.01; mean = 44%; range, 22%–67%), whereas there was no change in CON strength. TVL during the exercise trials was significantly greater AT (13,000 ± 2269 kg) compared with BT (9637 ± 1978 kg; P < 0.01, EX). RT significantly reduced circulating CRP and TNF-α in EX compared with sedentary CON (Figs. 1A, B). Neither RT (EX) nor social interaction (CON) influenced adiponectin concentration (Fig. 1C).

For circulating leptin concentration, we observed a main effect of time (P = 0.002) and a group × training interaction (P = 0.047, Fig. 2A). Leptin decreased significantly from PR to 2H but returned to PR levels at 24H in both groups. RT significantly reduced mean leptin concentration for EX (BT, 40 ± 5; AT, 33 ± 5 ng mL⁻¹) compared with CON (BT, 40 ± 5; AT, 41 ± 4 ng mL⁻¹).

ANOVA revealed a training × time × group interaction (P = 0.043) for plasma IL-6 (Fig. 2B). Acute RE increased IL-6 in EX at PO, and it remained elevated at 2H both before and after RT. IL-6 was lower in EX compared with CON at 24H before training (Fig. 2B). IL-6 increased (P = 0.045) in CON until it was significantly greater at 2H compared with PR before training. After the intervention, IL-6 was significantly higher at 2H compared with PO and 24H in CON. CON experienced a significant decrease in mean IL-6 after training (BT = 3.0, AT = 2.5 pg mL⁻¹). There was a tendency for a decrease in PR IL-6 for EX (P = 0.057) and for PR IL-6 to be lower than that of CON (P = 0.051) after training.

**LPS-stimulated cytokine production and leukocytes.** After adjusting for monocyte number per culture well in whole blood cultures, we observed a group × training (P = 0.042) and a training × time interaction (P = 0.023, Fig. 3A) for LPS-stimulated IL-10 production (LPS-IL10). Post hoc analyses revealed that mean LPS-IL10 was greater in EX compared with CON after the intervention (EX: BT = 0.621 ± 0.11; AT, 0.756 ± 0.09; CON: BT, 0.747 ± 0.9; AT, 0.541 ± 0.08 fg per monocyte) and decreased from BT to AT in CON.
In addition, mean 2H LPS-IL10 was less than PO before the intervention and less than PR after the intervention (Fig. 3A).

There was a main effect of training for LPS-TNF, corrected for monocyte number (BT = 34.01, AT = 49.83 fg per monocyte, \( P = 0.001 \)), and a tendency for a training \( / \) time \( / \) group interaction (\( P = 0.055 \); Fig. 3B). There was a significant time \( / \) group interaction (Fig. 3C, \( P = 0.002 \)) where LPS-TNF significantly increased by 2H in EX and remained elevated at 24H when it was significantly greater than CON. LPS-TNF increased in CON at 2H but was not different from PR at 24H (PR, 6.2\( \pm \)0.41; PO, 6.45\( \pm \)0.40; 2H, 7.0\( \pm \)0.45; 24H, 6.17\( \pm \)0.42 \( 1 \times 10^6 \) cells\( / \)mL\(^{-1} \)). Leukocyte number increased in EX at PO and remained elevated at 24H when it was significantly greater than CON. LPS-TNF increased in CON at 2H but was not different from PR at 24H (Fig. 3C).

Leukocyte values are shown in Table 2. There was a time \( / \) group interaction (\( P < 0.001 \)) for circulating leukocytes where mean leukocyte number increased in EX at PO and remained elevated at 24H when it was significantly greater than CON. Leukocyte number was significantly greater at PO and 2H in EX compared with CON. The percentage of neutrophils in circulation increased in EX by 2H (PR, 53.0\% \( \pm \)2.3\%; PO, 55.0\% \( \pm \)2.1\%; 2H, 61.6\% \( \pm \)1.6\%; (\( P = 0.011 \)). In addition, mean 2H LPS-IL10 was less than PO before the intervention and less than PR after the intervention (Fig. 3A).

FIGURE 2—Circulating leptin and IL-6 concentration before (BT) and after (AT) 12 wk of RT (EX) or social interaction (CON). PR, preexercise; PO, postexercise; 2H, 2 h postexercise; 24H, 24 h postexercise, or same time points for resting CON. A, Leptin. a = 2H mean different than PR and 24H means (\( P = 0.002 \)), EX AT mean (b) lower than CON AT mean (c) (\( P = 0.047 \)). B, IL-6. a = time point mean greater than PR and 24H in EX, b = greater than PR in CON BT, c = greater than PO and 24H in CON AT, d = EX BT different than CON BT (\( P = 0.043 \)), AT mean (e) lower than BT mean (f) in CON. Means \( \pm \)SE.

FIGURE 3—LPS-stimulated IL-10 and TNF-\( \alpha \) adjusted for number of monocytes before (BT) and after (AT) 12 wk of RT (EX) or social interaction (CON). PR, preexercise; PO, postexercise; 2H, 2 h postexercise; 24H, 24 h postexercise, or same time points for resting CON. A, IL-10. a = 2H mean less than PO BT, b = 2H mean less than PR AT (\( P = 0.023 \)); mean AT in EX (c) greater than mean AT in CON (d), AT mean (d) lower than BT mean (e) in CON (\( P = 0.042 \)). B, TNF-\( \alpha \). AT grand mean (b), greater than that of BT (a). C, TNF-\( \alpha \) (time by group interaction). * Greater than PR within group. * Greater than CON at 24H. Means \( \pm \)SE.
24H, 54.8% ± 2.1%), at which time, it was greater than that of CON (PR, 53.7% ± 1.6%; PO, 55.3% ± 2.1%; 2H, 54.5% ± 1.6%; 24H, 54.2% ± 2.1%; time × group interaction; P < 0.001).

We observed a time × group interaction (P < 0.001) for lymphocyte number where lymphocytes increased in circulation at PO in EX (PR, 1.98 ± 0.16; PO, 2.41 ± 0.16; 2H, 1.94 ± 0.14; 24H, 1.80 ± 0.14 1 × 10⁶ cells/mL⁻¹). Lymphocyte number was significantly elevated at 2H in CON (PR, 2.09 ± 0.16; PO, 2.08 ± 0.16; 2H, 2.39 ± 0.14; 24H, 2.1 ± 0.14 1 × 10⁶ cells/mL⁻¹). Lymphocyte number was significantly greater in EX compared with CON at PO but fell significantly below CON at 2H and 24H. A time × group interaction (P < 0.001) revealed that lymphocyte percentage decreased by 2H in EX such that it was significantly lower than all other time points in EX and also less than CON at 2H (EX: PR, 34.6% ± 2.5%; PO, 33.6% ± 2.1%; 2H, 28.2% ± 1.7%; 24H, 33.5% ± 2.2%; CON PR, 34.8%; PO, 34.1% ± 2.1%; 2H, 35.6% ± 1.7%; 24H, 35.0% ± 2.2%).

The number of circulating monocytes increased in EX at PO where it was significantly different than CON at the same time point (time × group interaction, P = 0.003; EX: PR, 0.46 ± 0.04; PO, 0.53 ± 0.03; 2H, 0.48 ± 0.03; 24H, 0.42 ± 0.04; CON: PR, 0.46 ± 0.04; PO, 0.44 ± 0.03; 2H, 0.44 ± 0.03; 24H, 0.44 ± 0.04 1 × 10⁶ cells/mL⁻¹). A training × time interaction (P < 0.001) indicated a decrease in monocyte number after the intervention at the PR time point only (BT: PR, 0.47 ± 0.03; PO, 0.47 ± 0.02; 2H, 0.45 ± 0.02; 24H, 0.41 ± 0.02; AT: PR, 0.44 ± 0.03; PO, 0.50 ± 0.03; 2H, 0.48 ± 0.03; 24H, 0.45 ± 0.03 1 × 10⁶ cells/mL⁻¹). There was only a main effect of time for monocyte percentage (P < 0.001) where it was significantly lower than PR at PO, further decreasing at 2H, then returning to PR values by 24H (PR, 7.5% ± 0.3%; PO, 7.0% ± 0.2%; 2H, 6.6% ± 0.2%; 24H, 7.3% ± 0.3%).

**Gene expression.** TLR4 mRNA expression in whole blood was not different between groups when expressed as “fold change” (EX: −1.12, N = 7; CON: 1.17, N = 7). RT did not significantly influence gene expression of MC1R in whole blood expressed as FC, nor did it influence that of inflammatory markers in SCAT (leptin, adiponectin receptor, CD68, MCP-1, and TLR4) (data not shown). MC3R gene expression has been previously reported (20). Briefly, we found that RT up-regulated MC3R expression in whole blood by 16-fold (P = 0.035).

**Correlational analyses.** After training, TVL was negatively associated with FC in SCAT TLR4 (r = −0.82, P = 0.013, N = 8) and MCP-1 (r = −0.76, P = 0.028, N = 8), with the higher the TVL, the lower the inflammatory marker. TVL after training was also negatively correlated with circulating leptin (r = −0.80, P = 0.017, N = 8) and IL-6 (r = −0.68, P = 0.032, N = 10), whereas change in TVL was negatively associated with CRP (r = −0.69, P = 0.04, N = 9) after training. TVL after training was positively correlated with whole blood expression of MCR3 FC (r = 0.78, P = 0.023), indicating a link between strength increases and a potential anti-inflammatory modulator. Finally, MC1R FC in whole blood was associated with change in CRP (r = −0.67, P = 0.012, N = 13), and MC3R and MC1R FC were related to LPS-IL10 after training at 2H (r = 0.59, P = 0.015, N = 16) and 24H (r = 0.66, P = 0.015, N = 13), respectively.

**Adherence and attrition.** After enrollment, three EX participants and two CON participants discontinued the study because of time constraints, leaving 23 women who completed the study (EX = 11, CON = 12). Adherence was excellent in both groups. All EX participants completing the study attended 36 resistance exercise training bouts (100%). The average attendance for the CON (control intervention sessions) was 90%.

**DISCUSSION**

Twelve weeks of moderate–high-intensity RT (8–12 RM) improved whole-body strength (44%) reduced circulating CRP (−33%), leptin (−18%), and TNF-α (−29%) and increased LPS-stimulated IL-10 production (20%) in the absence of detectable changes in body composition. Furthermore, an acute exercise resistance bout resulted in an elevation in

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**TABLE 2.** Leukocyte data (absolute number and percentage) before and after 12 wk of RT in the exercise group (EX) or social interaction in the CON.

<table>
<thead>
<tr>
<th>Leukocyte Type</th>
<th>Before Training/Social Interaction</th>
<th>After Training/Social Interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PR/PO 2H 24H</td>
<td>PR/PO 2H 24H</td>
</tr>
<tr>
<td>Leukocyte EX</td>
<td>6.37 ± 0.38 7.53 ± 0.37 7.17 ± 0.47</td>
<td>5.91 ± 0.54 7.37 ± 0.47 7.27 ± 0.50</td>
</tr>
<tr>
<td>Leukocyte CON</td>
<td>6.22 ± 0.36 6.32 ± 0.43 6.77 ± 0.42</td>
<td>6.10 ± 0.39 6.59 ± 0.43 7.17 ± 0.55</td>
</tr>
<tr>
<td>Neutrophil EX</td>
<td>3.29 ± 0.29 4.10 ± 0.28 4.35 ± 0.33</td>
<td>2.98 ± 0.33 3.86 ± 0.43 4.34 ± 0.37</td>
</tr>
<tr>
<td>Neutrophil CON</td>
<td>3.26 ± 0.29 3.36 ± 0.28 3.55 ± 0.33</td>
<td>3.08 ± 0.28 3.63 ± 0.34 3.93 ± 0.37</td>
</tr>
<tr>
<td>Lymphocyte EX</td>
<td>2.03 ± 0.07 2.35 ± 0.16 1.89 ± 0.14</td>
<td>1.94 ± 0.18 2.47 ± 0.18 1.98 ± 0.17</td>
</tr>
<tr>
<td>Lymphocyte CON</td>
<td>2.12 ± 0.07 2.05 ± 0.14 2.31 ± 0.14</td>
<td>2.05 ± 0.18 2.10 ± 0.18 2.48 ± 0.17</td>
</tr>
<tr>
<td>Monocyte EX</td>
<td>0.48 ± 0.04 0.53 ± 0.03 0.47 ± 0.03</td>
<td>0.43 ± 0.05 0.53 ± 0.05 0.50 ± 0.06</td>
</tr>
<tr>
<td>Monocyte CON</td>
<td>0.47 ± 0.04 0.42 ± 0.03 0.43 ± 0.02</td>
<td>0.44 ± 0.04 0.46 ± 0.03 0.46 ± 0.03</td>
</tr>
</tbody>
</table>

Values are mean ± SE.

PR, immediately before acute exercise in EX or resting control trial in CON; PO, immediately after exercise or control trial; 2H, 2 h after exercise or control trial; 24H, 24 h after exercise or control trial.
LPS-stimulated TNF-α and IL-10, particularly after 12 wk RT, which may indicate improved immunoreactivity. These results indicate a reduction in subclinical inflammation and in risk of CVD (26) via RT alone, with no change in body composition, and support our first hypothesis.

**Chronic RT adaptations in circulating inflammatory biomarkers.** In contrast to previous reports (9,38), we found that with consistent whole-body RT at 8–12 RM, weight loss is not obligatory to experience significant reductions in subclinical inflammation associated with inactivity-related diseases in obese, postmenopausal women. The exercise group entered the study with a mean CRP of 1.2 mg L⁻¹, which indicates a moderate risk (1–2.9 mg L⁻¹) for CVD (27). After training, CRP fell into the low-risk category (<1.0 mg L⁻¹) accompanied by decreases in circulating leptin and TNF-α concentration.

Most authors examining the anti-inflammatory effects of exercise have used aerobic exercise as the primary exercise mode (1,5,7,9,11,12,23,42,46). Less work has been done with RT alone, but evidence suggests that resistance exercise may be a key to maximizing the anti-inflammatory effects of consistent training (2,14,21). Several authors have reported aerobic exercise-induced reductions in CRP concomitant with weight loss or abdominal fat loss (2,46). In other well-controlled studies, however, where aerobic exercise training (with or without fat loss) has failed to reduce CRP (11,14,42), resistance exercise either alone (current study; 14) or in combination with aerobic exercise (2,41) has reduced it.

Although RT appears to decrease CRP and leptin (14,21), it has been reported that RT has no effect on circulating TNF-α (4,8,13), which is in contrast to the current and previous results (35). We found that 10 wk of RT at 8 RM in nonobese elderly women reduced circulating TNF-α by 37% (35), which is supported by our current findings of a 29% reduction in obese postmenopausal women after 12 wk of RT using a similar RT protocol. RT also reduces TNF-α mRNA expression and protein levels in skeletal muscle in elderly subjects (19,25). These findings highlight the importance of using RT as a training modality to improve both local muscular and systemic biomarkers of inflammation, particularly in elderly persons. In fact, in studies where RT-induced reductions in TNF-α were observed, participants were postmenopausal or elderly (mean age range, 65–81 yr) (19,25,35). Immunosenescence contributes to elevations in subclinical inflammation (31) and may interact with training-induced changes in inflammatory profile.

There were no significant RT-induced changes in circulating adiponectin or IL-6. Previously, adiponectin increased after 6 months of RT in elderly, overweight men (15). Our disparate results may be due to exercise intensity or training duration (13,15). The same may be true for changes in plasma IL-6 (37). We observed a 12% reduction (nonsignificant, \( P = 0.057 \)) in preexercise IL-6 after training for EX. The increase in TVL was inversely correlated with IL-6 linking improvements in strength and work capacity to decreases in circulating IL-6 concentration. A longer training period, however, may be required to elicit statistically detectable changes in adiponectin and IL-6 (37).

Unexpectedly, plasma IL-6 decreased after the intervention in the CON subjects. Given that CON activities included talks on diet and exercise, it is possible that some CON participants changed their lifestyle (diet, physical activity) to improve health, even though they were counseled to maintain their “normal” diet and physical activity pattern. It is also possible that consistent social interaction for 12 wk resulted in a decrease in IL-6. Socialization associated with CON group activities may have improved sense of coherence and other psychosocial factors, which, in turn, appear to influence immune function and inflammation (22). Our findings may have also been due to seasonal variations.

**Acute response of circulating inflammatory biomarkers to a resistance exercise bout.** Acute resistance exercise resulted in the expected, exercise-induced leukocytosis at PO (Table 2) where total leukocytes and neutrophils remained elevated at 2H. Lymphocyte number, however, was significantly lower in EX compared with CON at 2H and 24H, indicating the known lymphopenia response to exercise during recovery. There was no acute effect of a single resistance exercise bout on leptin, but we observed diurnal variation as circulating leptin decreased in both groups by the 2H time point. Leptin is known to exhibit a diurnal or circadian rhythm and to be influenced by meal timing. It appears that only exercise of very high intensity, long duration, or that which generates an energy expenditure greater than 800 kcal acutely influences leptin concentration (4,5).

The single resistance exercise bout increased plasma IL-6 at PO, which remained elevated up to 2 h postexercise (EX group). This response was expected because contraction stimulates release of IL-6 from the muscle fiber. The percentage increase in postexercise IL-6 was 40%–60% (BT and AT), which is in agreement with percentage increases reported in elderly women (34,35), but somewhat less than that reported in young men (36) after similar resistance exercise protocols. In contrast to our hypothesis, we did not observe a training-induced reduction in IL-6 response to muscle contraction as previously reported in nonobese elderly women (35); however, the current subjects were younger and had significantly more adipose tissue than those in the previous study. Obesity may influence training adaptations in muscle IL-6 release during contraction. Although high levels of IL-6 at rest are known to be associated with CVD and insulin resistance, acute elevations from muscle contraction are thought to exert anti-inflammatory actions. When examining the responses illustrated in Figure 2, IL-6 in EX appears to be trending downward at the 2H time point both BT and AT while that of resting CON continues to increase. In addition, IL-6 in EX was significantly lower than that of CON at 24H BT, indicating a potential residual effect (suppression of resting IL-6) of the last exercise bout. These observations could indicate an acute, exercise-induced reduction of low-level systemic inflammation possibly related to exercise-induced stress hormones, changes in circulating substrate availability,
cell receptor expression, or IL-6 release from muscle exerting an anti-inflammatory effect. There has been debate as to whether muscle contraction-induced IL-6 is indeed anti-inflammatory. More work is necessary to clarify the anti-inflammatory actions of muscle contraction-induced IL-6.

**LPS-stimulated cytokine response to RT.** In contrast to our first hypothesis and previous findings (35), LPS-TNF significantly increased after the interventions in both groups (35). Obese persons frequently experience a hyper-sensitivity to endotoxin, which increases the atherosclerotic process and contributes to low-level systemic inflammation. Obesity, however, may also result in impaired innate immunity (18,45). Because both groups experienced increases in LPS-TNF, when looking at LPS-TNF response alone, we might conclude that seasonal variation may account for the changes or that psychosocial factors (22) may boost monocyte TNF-α response to endotoxin challenge. It is interesting to note, however, that the increased LPS-TNF in EX was more than twice that of CON (CON +26%, EX +67%).

Of more significance than the increase in LPS-TNF alone, however, is the concomitant reduction in LPS-IL-10 found in CON (−28%) while that of EX increased (+20%). IL-10 is a strong anti-inflammatory cytokine, and its production is stimulated by other inflammatory cytokines, particularly IL-6, or activation of the melanocortin system. LPS-IL-10 increased in EX while inflammatory biomarkers (circulating TNF-α, CRP, and leptin) were reduced, indicating an overall state of reduced subclinical inflammation. In CON, the increased LPS-TNF was accompanied by a significant decrease in LPS-IL-10, suggesting a reduced anti-inflammatory response, whereas the improved responsiveness of monocytes (increased TNF-α and IL-10) in EX may reflect a more robust response of the innate immune system and better regulation of the inflammatory/anti-inflammatory balance. Because systemic inflammation is elevated in obese persons, some may develop a sort of endotoxin tolerance such that training alleviates the tolerance resulting in improvements in response to LPS (18,45), which is most likely linked to the observed decreases in circulating markers of inflammation. Regardless, RT not only prevented the decrease in LPS-IL-10 as seen in CON but also resulted in significantly greater LPS-IL-10 in EX compared with CON after training.

The acute resistance exercise bout appeared to have had no significant effect on LPS-IL-10. A time effect, however, revealed that in both groups and training time points (BT and AT), there was a decrease at the 2H time point. It was accompanied by an apparent increase in LPS-TNF at 2H observed in both groups (Fig. 3A, B). Similar results have been observed previously (29,35) and may indicate a tendency toward a pro-inflammatory pattern as fasting or time progresses throughout the morning hours (29,35). This observation may be related to the diurnal increase in circulating leukocytes observed over time (from PR to 2H) in CON. The lack of a resistance exercise-induced blunting of LPS-TNF up to 2H postexercise is in contrast to previous findings in non-obese elderly women and to our third hypothesis indicating that age or the obese state may alter the acute effects of resistance exercise on LPS-stimulated cytokine production (34,35).

The morning after the resistance exercise bout (24H), we observed a significant increase in LPS-TNF along with a tendency for an increase in LPS-IL10 (P = 0.055) reflecting residual effects of the last exercise bout in EX. A significant portion of the training-induced increase in LPS-IL10 appears to be led by the 24H time point value in EX. Furthermore, the elevation of LPS-TNF at 24H in EX was predominantly driven by the AT value. These results indicate that the acute (residual response) to resistance exercise at 24H is more robust after 12 wk RT. It is unlikely that this sensitivity to LPS at 24H AT was due to heightened inflammation consequent to muscle damage. If muscle damage significantly contributed to the increase in 24H LPS-cytokine production, we would have expected to see a more pronounced response before training also. The acute improvements in LPS responsiveness may be due to exercise-induced variations in neuroendocrine factors, or other immune-related changes such as the acute increases in IL-6 or other cytokines, shifts in monocyte subpopulations, or alterations in expression of melanocortin receptors or the LPS receptor, TLR4.

**Potential mechanisms.** Exercise training has reduced gene and cell surface expression of TLR4 (16,29) in blood, which may be partially responsible for exercise-induced decreases in inflammatory cytokines. We did not see a change in mRNA expression of TLR4 in whole blood or in SCAT. Because we did not examine cell surface expression of TLR4, we must consider that surface expression could have changed in the absence of changes in gene transcription (29). It is possible that the changes in LPS-induced cytokine release are linked to the 16-fold up-regulation of MC3R gene expression in whole blood reported previously (20). We also observed a positive association between MC3R mRNA up-regulation and TVL after training, linking MC3R expression to strength improvements. There was no change in whole blood MC1R expression; however, we did observe an inverse correlation between MC1R FC and change in CRP revealing an association between MC1R and anti-inflammatory properties. In addition, expression of MC3R and MC1R mRNA in whole blood was positively associated with LPS-IL10 after training at 2H and 24H, respectively. When bound by their agonists, the melanocortin receptors on leukocytes exert anti-inflammatory effects including stimulation of IL-10 release. We were the first to examine MC3R response to exercise training in humans (20). Our overall findings indicate that RT-induced increases in melanocortin receptor expression (MC3R—16-fold, P = 0.035; 20) in whole blood may have contributed to training-induced improvements in LPS-IL-10 production and reductions of inflammatory markers in circulation. Furthermore, these results may be associated with alterations in circulating leptin concentration.

Given the significant decrease in circulating leptin observed in this study along with the increase in MC3R expression (20), it is tempting to speculate that the overall,
exercise-induced reduction in systemic inflammation is, at least partially, linked to interactions of leptin and the melanocortin system. Leptin is a pro-inflammatory adipokine and influences inflammatory-related melanocortin system functions. For example, central signaling of leptin has been shown to accelerate peripheral inflammation through the melanocortin system (43). Potential associations between exercise/fitness and melanocortin receptor expression should be further explored.

SCAT response to RT. Our current findings did not support our second hypothesis because SCAT expression of MCP-1, CD68, leptin, and TLR4 did not significantly change in response to RT. Similarly, Klimacikova et al. (21) found that in the absence of body composition changes, 3 months of dynamic strength training improved insulin sensitivity and reduced circulating leptin but did not change SCAT mRNA levels of adiponectin, leptin, IL-1β, IL-6, or TNF-α in obese, middle-age men. It appears that obese, postmenopausal women respond similarly. To explain the observed changes in circulating and LPS-stimulated cytokines, without concomitant changes in gene expression in whole blood or SCAT, we must consider that alterations in mRNA gene expression are not always associated with changes in protein expression (29). In addition, exercise-induced changes in visceral adipose tissue may have occurred as it is known to have more inflammatory activity than SCAT. Furthermore, modifications in skeletal muscle inflammatory profile may have also influenced circulating markers (19) along with changes in monocyte phenotype (27).

Monocyte phenotype in adipose tissue evolved from an M2 (alternatively activated and anti-inflammatory) to an M1 (classically activated and pro-inflammatory) phenotype in response to diet-induced obesity (27). Perhaps RT in the present study induced a similar but reversed shift (M1 to M2) in monocyte phenotype without a change in total monocyte SCAT number. If so, this may partially reconcile the reduction in circulating inflammatory markers with no changes in SCAT CD68. In addition, RT may induce a change in TLR4 responsiveness to its ligands. Supporting this hypothesis are findings that neither TLR2 nor TLR4 expression was different between inflamed and noninflamed visceral adipose tissue (24). Interestingly, however, the responsiveness of the TLR receptors differed between groups (inflamed vs. noninflamed), indicating that alterations influencing receptor function may occur in the absence of total receptor gene expression in response to an inflammatory environment. This finding is pertinent to the current study because up-regulation of TLR signaling has been linked to leptin resistance (17). Alterations in TLR4 responsiveness may be responsible for changes in circulating and LPS-stimulated inflammatory markers, including decreases in leptin. Investigating potential monocyte phenotype shifts and TLR functionality in an exercise model will be necessary to confirm the given hypotheses.

Strength and inflammatory markers. We observed inverse relationships between measures of strength and inflammatory markers (CRP, IL-6, leptin, MCP-1, and TLR4) after training in EX. These data couple reductions in inflammation to “muscle fitness.” Furthermore, improvements in strength were positively associated with up-regulation of MC3R in whole blood. Our findings support those of Donges et al. (14) who reported a significant inverse relationship between change in CRP and change in strength (upper and lower body) after 10 wk of RT. Interestingly, the authors found no associations between change in CRP and measures of aerobic fitness (14). Our findings, along with others, may indicate that “muscle fitness” and improvements in strength are directly related to the anti-inflammatory effects that RT confers upon skeletal muscle. Although we did not measure cytokines in skeletal muscle, local changes induced by RT (19) may be linked to systemic reductions in subclinical inflammation observed here. In fact, it may partially explain why some aerobic exercise training studies have not resulted in reductions in CRP (11,42), yet RT has (current study and Ref. (14)). The stress stimulus from and physiological response to RT are different than those of aerobic exercise and may be necessary to maximize exercise-induced reductions in inflammation.

SUMMARY

This study was not without limitations. The seven-site skinfold technique may not have been sensitive enough to capture subtle changes in body composition, especially in obese women, and particularly in the visceral adipose region. The number of participants in this study was low for correlational analyses. Finally, it would have been more conservative to provide meals for participants before blood and tissue collection rather than to rely on self-reports. Strengths of the study included 1) tightly controlled exercise and control interventions and excellent adherence to the study protocol, 2) exercise regimen worked all major muscle groups (moderate to high intensity) targeting large muscles and/or multijoint movements, and 3) examination of inflammation using several methods/tissues concurrently.

Here, we show that a whole-body resistance exercise training protocol of moderate–high intensity exerts beneficial changes in subclinical inflammation in circulation and in stimulated culture in obese, postmenopausal women, a group at risk for the metabolic syndrome. These findings help to strengthen our understanding of the health benefits of RT as a single intervention and support those of others (2) who reported that physical activity, including RT, is anti-inflammatory, independent of weight loss. These findings are of value because they show that clinically beneficial alterations can occur during RT before observable changes in body composition. Practically, this is a key point to convey to patients and clients in the general population wishing to improve health. Overweight and obese persons should be encouraged to continue consistent exercise, even if they are not experiencing weight loss because health benefits can result even if they are not readily apparent.
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The authors have no conflicts of interest to declare.

The results of the present study do not constitute endorsement by the American College of Sports Medicine.

REFERENCES


