Arterial stiffening following eccentric exercise-induced muscle damage

Jill N. Barnes, Justin R. Trombold, Mandeep Dhindsa, Hsin-Fu Lin, and Hiroyumi Tanaka

Cardiovascular Aging Research Laboratory, Department of Kinesiology and Health Education, University of Texas at Austin, Austin, Texas

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Barnes JN, Trombold JR, Dhindsa M, Lin H, Tanaka H. Arterial stiffening following eccentric exercise-induced muscle damage. J Appl Physiol 109: 1102–1108, 2010. First published July 29, 2010; doi:10.1152/japplphysiol.00548.2010.—Acute inflammatory responses are linked to a transient increase in risk of a cardiovascular event, and this risk may be mediated by a concomitant reduction in vascular function. Humans experience an acute inflammatory response as a consequence of infection, injury, or muscle damage. We measured macrovascular function before and after eccentric exercise to determine whether muscle damage from unaccustomed exercise has an unfavorable effect on the large elastic arteries. A total of 27 healthy sedentary or recreationally active men (age 18–38 years) participated in either bilateral leg press eccentric exercise or unilateral elbow flexor eccentric exercise. Postexercise muscle damage was confirmed by significant reductions in isometric strength and increases in muscle soreness (P < 0.05). Carotid-femoral pulse-wave velocity was significantly elevated 48 h after leg exercise (808 ± 31 vs. 785 ± 30 cm/s; P < 0.05) and arm exercise (790 ± 28 vs. 755 ± 24 cm/s; P < 0.05). There were no changes in mean arterial pressure. C-reactive protein was elevated after leg exercise but not after arm exercise. The increase in carotid-femoral pulse wave velocity 48 h after arm exercise was associated with muscle strength (r = −0.47; P < 0.05) and creatine kinase concentrations (r = 0.70; P < 0.01). We concluded that eccentric exercise in both small and large muscle mass translates to transient, unfavorable changes in central macrovascular function and that the increase in central arterial stiffness after small muscle eccentric exercise is associated with indicators of muscle damage.

METHODS

SUBJECTS

A total of 27 healthy men, aged 18–38 years, were recruited from the University of Texas at Austin and surrounding community. Eleven subjects were recruited for protocol 1, and 16 subjects were recruited to complete protocol 2. All participants were nonsmoking, nonobese, sedentary or recreationally active (no participation in any structured resistance or endurance training within the past 3 mo; limited to <2 days of exercise per week), and had no orthopedic injury that would have inhibited the ability to complete the eccentric exercise trial. Recruited participants had no history of hypertension (<140/90 mmHg) or kidney dysfunction and were not using lipid-lowering, anti-inflammatory steroid medication or antioxidant supplements. All participants gave written informed consent, and all procedures were reviewed and approved by the Institutional Review Board at The University of Texas at Austin.

EXPERIMENTAL DESIGN

Protocol 1. To test the effect of large muscle mass damage on macrovascular function, we used a randomized, crossover experimental design with 2 trials lasting 4 days. In the exercise trial (ECC), eccentric bilateral leg press exercise was used to evoke muscle damage; the other trial was a sham control. The trials were random-
ized and separated by at least 10 days. Approximately 4 days before the first experimental testing day, cardiovascular measurements were taken to familiarize the subjects with the testing procedures (see General Procedures for description of measurements). Subjects completed a total of three isometric force familiarizations before the first trial to minimize any training or learning effects. Familiarizations were separated by at least 24 h and completed at least 72 h before the first experimental testing day. Each familiarization consisted of four unilateral isometric knee extension contractions, four trials on the dominant leg, with two at 90° and two at 45°. In addition, subjects completed a leg press one-repetition maximum, as determined using the standard American College of Sports Medicine protocol.

On the morning of day 1, measurements were taken 30 min before (Pre) ECC or sham control and 90 min and 24, 48, and 72 h after (Post) ECC or sham control protocol. For the ECC trial, 6 sets of 10 repetitions of bilateral eccentric-only inclined leg press exercise (Cybex International, Medway, MA) were performed at 110% of each subject’s one-repetition maximum (295 ± 20 kg) with 3–4 min of rest between sets. For each repetition, subjects were instructed to maximally resist the weight of the carriage until the knee joint flexion reached 90°, and the investigators lifted the weight to the starting position. This eccentric-only leg press protocol was used in previous studies and sufficient to cause muscle damage and inflammation (27, 43, 50). For the sham control trial, subjects sat quietly in the same weight room for 25 min.

Protocol 2. A separate group of subjects was recruited to complete a muscle-damaging eccentric protocol that used a smaller muscle mass. Protocol 2 induced damage to the elbow flexors and followed the same timeline as protocol 1 but without the sham control condition. After the Pre measurements, subjects performed 2 sets of 20 unilateral eccentric elbow flexion contractions on a Biodex isokinetic dynamometer (Cybex International, Medway, MA), with each contraction lasting 3 s with one repetition performed every 15 s and 4 min of rest in between sets (7, 8, 15, 24, 44). After the exercise protocol, measurements were taken at 90 min and 24, 48, and 72 h Post ECC (Fig. 1). Subjects completed one complete familiarization and three strength familiarizations before the first experimental testing day. Each familiarization trial consisted of four maximal isometric contractions, two at 30° and two at 45° above complete elbow extension.

General Procedures

In both protocols, subjects abstained from alcohol and caffeine in the 12 h before they arrived to the laboratory and were instructed to keep dietary intake and physical activity constant. Additionally, subjects arrived to the laboratory after an overnight fast or after a light snack. If subjects elected to eat in the morning before arriving, they were instructed to eat the same light snack, every morning, for the duration of the protocol. Because both legs were exercised in protocol 1, leisure-time physical activity was restricted to 48 h before and throughout the entire duration of each trial.

Central and peripheral hemodynamics. Subjects rested in the supine position for 10 min before brachial and ankle blood pressures, heart rate, and arterial stiffness were measured with an automated previously validated device (Colin VP-2000, Colin Medical Instruments, San Antonio, TX) (9, 51). Arterial stiffness was determined by pulse-wave velocity measured between carotid and femoral artery (cfPWV). Data were collected over a 30-s interval followed by 2–3 min of quiet rest, and the average of two to four intervals was calculated. The day-to-day coefficient of variation for cfPWV in our laboratory is 3 ± 3%.

Blood analyses. A 10-ml blood sample was obtained from the antecubital vein to determine markers of muscle damage and inflammation. All blood samples were obtained before isometric strength testing. Serum C-reactive protein (CRP) (Alpha Diagnostics, San Antonio, TX) and interleukin-6 (IL-6) levels were assessed in duplicate with the use of a high-sensitivity ELISA (Quantikine, R&D Systems, Minneapolis, MN). Creatine kinase (CK) was used as a marker of muscle damage (11) in protocol 2 and assessed by the UV-kinetic method (Teco Diagnostics, Anaheim, CA).

Indicators of muscle damage. Muscle soreness and isometric strength were measured as indexes of muscle damage. Subjects rated the soreness of the exercised knee extensors (protocol 1) or elbow flexors (protocol 2) based on perception of soreness during day-to-day activities and unloaded knee extension or elbow flexion using a visual analog scale of 0–10 as previously described (5, 8). To measure the reduction in maximal isometric strength in both protocols 1 and 2, each respective joint was stabilized and connected to a load cell (CL101–500, Omega Engineering, Stamford, CT). Subjects performed maximal isometric contractions at two joint angles similar to the familiarization procedure. Isometric strength was recorded as an average of the peak torque at each joint angle.

Statistical Analyses

Descriptive statistics were used for the analyses of subject characteristics using SPSS statistical package (version 16.0; Chicago, IL). Because of large between-subject variability at Pre, absolute changes from the Pre measurement in cfPWV, CK, CRP, and IL-6 were calculated for each subject before statistical analysis. Similarly, relative changes in isometric muscle strength and increases in muscle soreness were calculated before analysis. Dependent variables were
analyzed separately within each treatment to determine the time effect using repeated-measures ANOVA or analysis of covariance. A two-way repeated measures ANOVA was used for analyses of time and treatment (ECC/sham control for protocol 1) effects. When necessary, the conditions were compared using a standard one-way ANOVA. Associations were determined by Pearson’s product-moment correlations. Because CRP was not normally distributed, Spearman’s Rho correlations were used to determine significant associations. CRP was analyzed in two ways: delta changes in CRP or a log transformation applied to absolute CRP values before repeated-measures ANOVA. Significance was set a priori at $P < 0.05$.

**RESULTS**

**Protocol 1**

Subject characteristics are presented in Table 1. Subjects completed all familiarization sessions and complied with the food, alcohol, and physical activity restrictions. The ability of ECC protocol to induce muscle damage was documented by a loss in isometric strength and an increase in muscle soreness (Fig. 1).

**Central and peripheral hemodynamics.** Heart rate and brachial blood pressure data are displayed in Table 2. There were small, yet significant changes in heart rate and pulse pressure. Mean cfPWV at Pre was not different between the ECC and sham control trials ($785 \pm 30$ vs. $782 \pm 29$ cm/s; $P < 0.05$).

**Blood markers of inflammation.** Serum CRP concentration was not different between the sham control and ECC trials at Pre ($0.99 \pm 0.45$ vs. $0.60 \pm 0.23$ mg/l). CRP concentration increased significantly at 24 h Post (+0.72 mg/l; $P < 0.05$) during the ECC trial, and, at 24 and 48 h, values of CRP were greater than the corresponding values for the sham control trial ($P < 0.05$; Fig. 3). When a log transformation was applied to absolute CRP values, results at 24 and 48 h were significantly greater than at baseline, and results for the ECC trial were higher than the sham control trial at 24 h (Table 2). IL-6 and IL-8 levels both increased relative to Pre at 24 h (Table 2).

### Table 1. Selected subject characteristics

<table>
<thead>
<tr>
<th>Subject characteristic</th>
<th>Protocol 1</th>
<th>Protocol 2</th>
</tr>
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<tbody>
<tr>
<td>n</td>
<td>11</td>
<td>16</td>
</tr>
<tr>
<td>Age, years</td>
<td>$25 \pm 1$</td>
<td>$24 \pm 2$</td>
</tr>
<tr>
<td>Height, cm</td>
<td>$176 \pm 3$</td>
<td>$178 \pm 1$</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>$76 \pm 4$</td>
<td>$73 \pm 2$</td>
</tr>
<tr>
<td>Body mass index, kg/m$^2$</td>
<td>$25 \pm 1$</td>
<td>$23 \pm 1$</td>
</tr>
</tbody>
</table>

Values are means $\pm$ SE.

### Table 2. Selected hemodynamic, muscle damage, and inflammatory markers for protocol 1 (involving large muscle mass)

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
<th>24 h Post</th>
<th>48 h Post</th>
<th>72 h Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>cfPWV, cm/s</td>
<td>782 $\pm$ 29</td>
<td>808 $\pm$ 25</td>
<td>779 $\pm$ 30</td>
<td>786 $\pm$ 24</td>
<td>796 $\pm$ 25</td>
</tr>
<tr>
<td>Control</td>
<td>785 $\pm$ 30</td>
<td>790 $\pm$ 24</td>
<td>793 $\pm$ 28</td>
<td>808 $\pm$ 31*</td>
<td>793 $\pm$ 30</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>55 $\pm$ 3</td>
<td>51 $\pm$ 3*</td>
<td>57 $\pm$ 3</td>
<td>59 $\pm$ 3</td>
<td>59 $\pm$ 3*</td>
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<td>ECC</td>
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<td>58 $\pm$ 1</td>
<td>57 $\pm$ 3</td>
<td>61 $\pm$ 3</td>
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<td>113 $\pm$ 2</td>
<td>112 $\pm$ 3</td>
<td>113 $\pm$ 3</td>
<td>114 $\pm$ 3</td>
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<tr>
<td>Control</td>
<td>114 $\pm$ 3</td>
<td>112 $\pm$ 3</td>
<td>113 $\pm$ 2</td>
<td>115 $\pm$ 2</td>
<td>114 $\pm$ 3</td>
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<td>Mean blood pressure, mmHg</td>
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<td>79 $\pm$ 2</td>
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<td>82 $\pm$ 2</td>
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<td>83 $\pm$ 2</td>
<td>82 $\pm$ 2</td>
<td>85 $\pm$ 2</td>
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<td>Diastolic blood pressure, mmHg</td>
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<td>62 $\pm$ 2</td>
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<tr>
<td>Control</td>
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<td>Pulse pressure, mmHg</td>
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<tr>
<td>Control</td>
<td>52 $\pm$ 2</td>
<td>49 $\pm$ 2*</td>
<td>50 $\pm$ 2</td>
<td>52 $\pm$ 1</td>
<td>50 $\pm$ 2</td>
</tr>
<tr>
<td>CRP, mg/l</td>
<td>0.99 $\pm$ 0.45</td>
<td>0.72 $\pm$ 0.38</td>
<td>0.44 $\pm$ 0.20</td>
<td>0.68 $\pm$ 0.30</td>
<td></td>
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<tr>
<td>Control</td>
<td>0.60 $\pm$ 0.23</td>
<td>0.57 $\pm$ 0.22</td>
<td>1.32 $\pm$ 0.37*</td>
<td>1.20 $\pm$ 0.33*</td>
<td></td>
</tr>
<tr>
<td>IL-6, pg/ml</td>
<td>1.03 $\pm$ 0.17</td>
<td>0.69 $\pm$ 0.10</td>
<td>0.83 $\pm$ 0.09</td>
<td>0.97 $\pm$ 0.17</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>0.91 $\pm$ 0.12</td>
<td>0.91 $\pm$ 0.10</td>
<td>0.91 $\pm$ 0.15</td>
<td>0.71 $\pm$ 0.09</td>
<td></td>
</tr>
</tbody>
</table>

Values are means $\pm$ SE. cfPWV, carotid-femoral pulse wave velocity; CRP, C-reactive protein; ECC, eccentric exercise trial; Pre, preexercise; Post, postexercise. *$P < 0.05$ vs. Pre; †$P < 0.05$ vs. control.
concentration did not change significantly during either trial (Table 2).

Protocol 2

The muscle damage caused by the ECC was documented by a loss in isometric strength and an increase in muscle soreness (see Table 3). Isometric strength was reduced and muscle soreness rating was increased 24, 48, and 72 h Post (P < 0.05 vs. Pre).

Central and peripheral hemodynamics. Supine heart rate and brachial blood pressures did not change from Pre for results 24, 48, and 72 h Post (Table 3). The cfPWV at Pre was 755 ± 24 cm/s. At 48 h Post, cfPWV was significantly (P < 0.05) greater than at Pre (+34.6 cm/s; Fig. 4).

Blood markers of muscle damage and inflammation. CK concentration increased significantly at 24 h Post (P < 0.05 vs. Pre; †P < 0.05 vs. control).

Correlations

To gain insight into potential mechanisms for the increase in cfPWV at 48 h, we examined associations between markers of muscle damage and inflammation. There were no significant associations between changes in muscle soreness or inflammatory markers and macrovascular function for either protocol. The increase in cfPWV observed in protocol 2 (arm exercise) was positively associated with the corresponding changes in isometric strength and CK (Table 4 and Fig. 6). When isometric strength was added as a covariate in protocol 1, the increase in cfPWV at 48 h was no longer significant (P = 0.19). In protocol 2, the increase in cfPWV remained significant when CK (P < 0.05) but not isometric strength (P = 0.08) was added as covariate.

DISCUSSION

The most salient findings of the present study are that eccentric exercise, in both large and small muscle mass, transiently increased central arterial stiffness. In addition, the change in central arterial stiffness after small muscle mass exercise was positively associated with indicators of eccentric exercise-induced muscle damage.

To our knowledge, this is the first study to investigate the potential association between exercise-induced muscle damage, inflammation, and macrovascular function. On the basis of previous studies suggesting that experimentally induced acute inflammation reduces endothelial function (3, 19) and increases arterial stiffness (58), we sought to determine whether local muscle damage, a common event that many people experience, would translate to unfavorable changes in vascular function. Eccentric exercise has previously been shown to increase circulating inflammatory markers (6, 18, 44), cause postexercise microvascular dysfunction to the injured muscle in rats (16, 21), and increase vascular resistance to the nonexercised muscle in humans (45). Our working hypothesis was that eccentric exercise causes focal disruption of the sarco-

Table 3. Selected hemodynamic, muscle damage, and inflammatory variables for protocol 2 (involving small muscle mass)

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
<th>24 h Post</th>
<th>48 h Post</th>
<th>72 h Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>cfPWV, cm/s</td>
<td>755 ± 24</td>
<td>779 ± 26</td>
<td>758 ± 26</td>
<td>790 ± 28*</td>
<td>767 ± 24</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>59 ± 2</td>
<td>55 ± 2</td>
<td>58 ± 2</td>
<td>57 ± 1</td>
<td>57 ± 2</td>
</tr>
<tr>
<td>Systolic blood pressure, mmHg</td>
<td>117 ± 2</td>
<td>116 ± 2</td>
<td>118 ± 2</td>
<td>116 ± 2</td>
<td>118 ± 2</td>
</tr>
<tr>
<td>Mean blood pressure, mmHg</td>
<td>86 ± 2</td>
<td>85 ± 2</td>
<td>86 ± 2</td>
<td>84 ± 1</td>
<td>85 ± 2</td>
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<tr>
<td>Diastolic blood pressure, mmHg</td>
<td>65 ± 2</td>
<td>65 ± 2</td>
<td>66 ± 2</td>
<td>63 ± 1</td>
<td>64 ± 1</td>
</tr>
<tr>
<td>Pulse pressure, mmHg</td>
<td>52 ± 2</td>
<td>51 ± 2</td>
<td>52 ± 2</td>
<td>53 ± 2</td>
<td>55 ± 2</td>
</tr>
<tr>
<td>Isometric strength, %</td>
<td>100 ± 0</td>
<td>75 ± 3*</td>
<td>77 ± 3*</td>
<td>80 ± 3*</td>
<td>86 ± 2*</td>
</tr>
<tr>
<td>Muscle soreness, %</td>
<td>0 ± 0</td>
<td>41 ± 7*</td>
<td>82 ± 5*</td>
<td>82 ± 6*</td>
<td>56 ± 8*</td>
</tr>
<tr>
<td>CRP, mg/l</td>
<td>1.40 ± 0.36</td>
<td>1.25 ± 0.35</td>
<td>1.73 ± 0.59</td>
<td>1.28 ± 0.44</td>
<td>0.90 ± 0.24</td>
</tr>
<tr>
<td>IL-6, pg/ml</td>
<td>0.92 ± 0.19</td>
<td>1.91 ± 0.90</td>
<td>1.17 ± 0.28</td>
<td>0.88 ± 0.24</td>
<td>1.10 ± 0.26</td>
</tr>
</tbody>
</table>

Values are means ± SE. *P < 0.05 vs. Pre.
mere, microvasculature, and/or excitation-contraction coupling apparatus in the skeletal muscle, inducing a subsequent inflammatory response, thereby altering local and systemic vascular function, either directly or indirectly. In both protocols 1 and 2, central arterial stiffness increased 48 h Post when muscle soreness and the corresponding inflammatory responses were expected to peak (48, 49). It should be noted that, although an increase in pulse-wave velocity was statistically significant, the magnitude of change was rather small (3–5%). This magnitude of change in pulse-wave velocity is similar to studies investigating the effects of acute smoking on arterial stiffness in nonsmokers (3–6%) (22, 29). It remains to be determined whether this magnitude of increase is physiologically or clinically important.

Modest increases in pulse-wave velocity have been reported after vaccination-induced systemic inflammation accompanied by elevations in CRP and IL-6 (58). Aspirin administration attenuated the effect of the vaccination on arterial stiffness, suggesting a cause-and-effect relationship between inflammation and arterial stiffening. Because this previous study lacked sufficient control of subjects’ activity, food intake, or medication use between measurements, the present study was conducted to minimize confounding influences with the idea that the effect of inflammation would be isolated to the injured muscle and would be more pronounced. In the present study, the associations between increases in central arterial stiffness and the corresponding changes in CRP did not reach significance, although there were trends ($r = 0.34–0.44$; $P = 0.156–0.052$). It is possible that muscle damage and inflammation may have a greater physiological effect on microvascular, rather than macrovascular, function. Indeed, a direct effect of inflammation on endothelial function has been reported by several groups (3, 19). The present study was not designed to evaluate the contribution of functional mechanisms (i.e., altered nitric oxide bioavailability, vasoactive hormone, sympathetic adrenergic vasoconstrictor tone, etc.) to the observed increase in central arterial stiffness. Future studies are necessary to characterize the endothelial or microvascular response 48 h after muscle-damaging exercise.

Acute upper body resistance exercise (13) and whole body resistance exercise (10) transiently increase central arterial stiffness immediately after exercise. In addition, high-intensity resistance training (37) is associated with central arterial stiffening. Our present results indicate that central arterial stiffness may remain elevated 1 or 2 days after acute resistance exercise. Arterial stiffening effects of resistance training have been attributed to substantial increases in arterial pressure during weight training exercises (37). However, when the same weight training exercises were repeated over days, increases in blood pressure during the concentric phase of weight lifting exercises can be considerably reduced (26). The present study suggests the eccentric phase may contribute to arterial stiffening, independent of blood pressure changes, by triggering muscle damage, muscle soreness, and inflammation. Indeed, indirect markers of muscle damage (isometric strength and serum CK) were related to the increase in cfPWV after small muscle mass exercise. This indicates that, even without significant increases in systemic inflammatory markers, exercise-induced muscle damage may contribute to arterial stiffening. Therefore, muscle damage and soreness typically associated with a bout of strenuous resistance exercise may be a factor contributing to the unfavorable changes in arterial stiffness that have been reported after high-intensity resistance training (37) and upper body resistance training (39).

Protocol 1 was specifically designed to utilize a large muscle mass with high intensity lower body eccentric contractions. Other groups have demonstrated the effectiveness of strenuous eccentric leg exercise in a comparable population (18, 23, 50), and the feasibility and safety of the exercise protocol were established during pilot work for this study. Protocol 2 was used to determine whether there was a “threshold” of damage necessary for cardiovascular consequences by injuring the elbow flexors, a much smaller muscle mass. With a smaller muscle and the constant resistance from the dynamometer, the

Table 4. Correlations with changes in arterial stiffness

<table>
<thead>
<tr>
<th>Protocol 1: ΔcfPWV at 48 h Post</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>ΔMuscle strength</td>
<td>$-0.43$</td>
<td>0.096</td>
</tr>
<tr>
<td>ΔCRP</td>
<td>$0.34$</td>
<td>0.156</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Protocol 2: ΔcfPWV at 48 h Post</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>ΔMuscle strength</td>
<td>$-0.47$</td>
<td>0.040*</td>
</tr>
<tr>
<td>ΔCK</td>
<td>$0.70$</td>
<td>0.002*</td>
</tr>
<tr>
<td>ΔCRP</td>
<td>$0.44$</td>
<td>0.052</td>
</tr>
</tbody>
</table>

CK, creatine kinase. Δ, change. *Significant correlation.
ECC protocol may produce more stress and more pronounced soreness, localized inflammation, and hemodynamic consequences. Although we were focusing on the small vs. large muscle mass in the context of our study, others have demonstrated that upper vs. lower body exercise may have differing responses. For example, acute resistance exercise utilizing only the upper body elevated central arterial stiffness similar to our results (13). Lower body resistance exercise, however, did not change arterial stiffness (17). Together, the fact that protocols 1 and 2 had comparable results with different eccentric stimuli and distinct subject populations suggests that local muscle damage and the amount of mechanical stress relative to muscle size are factors in eccentric exercise-induced central arterial stiffness.

Eccentric exercise to both small and large muscle mass induced considerable muscle damage as evidenced by the decline in isometric strength and elevated muscle soreness that persisted 3 days after exercise. Despite this functional evidence of muscle damage, circulating inflammatory markers were not consistently elevated (the increase in CRP was significant for protocol 1). Previous studies demonstrated increases in CRP, IL-6, or other cytokines as evidence of inflammation from muscle damage (4, 6, 18, 41, 44, 47, 57). A potential explanation for the observed results is that locally induced muscle damage causing inflammation may not be easily detected systematically. Measuring circulating markers of inflammation is an indirect method to quantify the local alterations within and around the damaged skeletal muscle. It is also possible that the standardized blood collection intervals did not coincide with the peak circulating inflammatory markers. However, the small changes in systemic inflammatory markers in this study are in agreement with several others who have used various eccentric exercise protocols and techniques to quantify inflammation (20, 31, 32, 35). Although local inflammation likely occurs after muscle damage, the amount of spillover into the systemic circulation appears to be related to the type of eccentric exercise. Eccentrically biased cycling or downhill running results in a greater increase in serum IL-1β and IL-6 than eccentric contractions (20, 28, 44, 52) that is not explained by the magnitude of muscle damage. Because IL-6 is produced upon muscle contraction (14), dynamic eccentric-biased aerobic exercise will elicit greater hemodynamic changes that possibly contribute to increased “spillover” to the systemic circulation (42).

There are several limitations of this study that warrant discussion. First, we were unable to include a time control trial for protocol 2. However, each subject acted as his own control, and the unexercised arm was used as a control for the strength and soreness measurements. As such, we do not expect large day-to-day changes in any of the variables of interest. Second, we did not collect physical activity data using accelerometers during the follow-up periods. However, subjects were reminded of the restrictions and asked each morning to recall any physical activity since the last visit. Third, we did not measure blood pressure during the ECC protocol. Because eccentric contractions have less of a blood pressure response during exercise and blood pressure was not significantly elevated at 2 h Post, this should not affect our cPWV results observed at 48 h. Fourth, we did not measure CK in protocol 1. Finally, the physiological or clinical significance of our findings is unclear.

We conducted this study in young adults, and it is not known whether the same response can be observed in older adults. In conclusion, eccentric exercise, resulting in muscle damage, translates to transient unfavorable changes in central macrovascular function as assessed by central arterial stiffness. The increase in arterial stiffness after small muscle mass exercise was significantly associated with the reduction in muscle strength and the increase in CK. Our results indicate that eccentric contraction-induced muscle damage may have acute unfavorable cardiovascular effects in young adults. Further research is warranted to evaluate the clinical significance of this phenomenon.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

REFERENCES


