Grass-Fed and Non-Grass-Fed Whey Protein Supplementation and High-Intensity Eccentric Exercise Do Not Affect Arterial Stiffness and Systemic Hemodynamics in Resistance-Trained Individuals

Original Research

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Abstract

Introduction: Dairy products from pasture-raised grass-fed cows are known for their higher anti-inflammatory and antioxidant content, which may enhance vascular modulation compared to products from grain-fed cows. Given that eccentric muscle loading (EML) and the resulting exercise-induced muscle damage (EIMD) may temporarily disrupt vascular homeostasis, this study tested the hypothesis that whey protein from pasture-raised grass-fed cows (PRWP) would improve markers of vascular recovery compared to conventional whey protein (CWP) or a non-protein control (NPC).

Methods: A randomized, double-blind, placebo-controlled study was used to test 39 resistance-trained participants (66% male, PRWP, $n = 14$; CWP, $n = 12$; NPC, $n = 13$). EIMD was induced via an eccentric barbell back squat protocol, followed by vascular assessments (peripheral/central blood pressure and carotid-femoral pulse wave velocity using applanation tonometry) at 24, 48, and 72 hours post-EML.

Results: No between-group differences in blood pressure or vascular measures were observed pre-EML. However, a significant interaction was observed 48 hours post-EML, with diastolic blood pressure (DBP) elevated in the PRWP group (PRWP, 77 ± 11 mm Hg; CWP, 66 ± 9 mm Hg; NPC, 67 ± 10 mm Hg; $P = 0.011$). Pulse wave velocity did not differ significantly between groups ($P = 0.598$), visit ($P = 0.753$), or group-by-visit interaction ($P = 0.418$). No additional differences in blood pressure or vascular assessments were observed post-EML.

Conclusions: Unexpectedly, PRWP increased DBP 48 hours post-EML, without any observed benefit in vascular recovery markers. Participants' training backgrounds may have reduced the sensitivity to detect EML-induced vascular disruptions.

Key Words: EIMD, squat, cardiovascular

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Introduction

Acute, unaccustomed exercise, including eccentric loading, can induce structural damage to muscle tissue, thereby reducing performance (1). While much research has characterized the link between muscle soreness and impaired muscle function, a related consequence also features transient vascular perturbations (2–7). Prior work suggests that exercise-related muscle damage may upregulate sympathetic outflow, as evidenced by elevated blood pressure and/or arterial stiffness (8,9). Since exercise adaptation balances progressive overload with sufficient recovery, determining optimal post-exercise strategies to enhance performance is of widespread interest.

Post-exercise whey protein supplementation is one such strategy that may enhance recovery and performance (10). Consistent with this premise, a recent meta-analysis of 40 exercise trials indicated that whey protein supplementation helps preserve maximal strength and attenuates creatine kinase concentrations following resistance exercise (11). In addition to promoting muscle recovery, several studies have shown that whey-derived peptides possess antihypertensive properties that are thought to act by inhibiting angiotensin-converting enzyme (ACE) (12,13). Given that ACE converts angiotensin I to angiotensin II, a potent vasoconstrictor, inhibiting ACE via whey protein may provide antihypertensive effects (14). Furthermore, ACE degrades bradykinin, a potent vasodilator; therefore, whey protein may increase bradykinin levels, thereby exerting a vasodilatory effect (15). Along these lines, Oliveira and colleagues showed improvement in endothelium-dependent dilation 30 min following a single serving of whey protein in physically active adults (16). However, most commercially available whey protein supplements are derived from milk from conventional grain-fed cows. According to the literature, milk from pasture-raised, grass-fed cows contains higher levels of omega-3 polyunsaturated fatty acids, vitamins, minerals, and phytonutrients (15–17) than conventional milk. More specifically, research has found that whey peptides (α-lactalbumin and kappa-casein) exhibit potency and mechanisms of action similar to those of commonly prescribed antihypertensive drugs regarding ACE inhibition (17,18). Furthermore, the digestion of bioactive peptides from whey exerts anti-inflammatory and antioxidant effects (12). However, these products can vary in bioactive peptide composition (19). Research has yet to examine the potential physiological effects of whey protein supplementation from pasture-raised cows on vascular perturbations arising following eccentric muscle loading (EML) exercise.

Based on the above rationale, the current work examined the acute effects of pasture-raised whey protein (PRWP) versus conventional whey protein (CWP) supplementation on peripheral and central hemodynamics and arterial stiffness following EML in healthy, resistance-trained individuals. We hypothesized that PRWP would reduce peripheral and central blood pressure and arterial stiffness to a greater extent than CWP during the recovery period following EML exercise.

Methods

Participants

This work is part of a past investigation examining whey protein consumption and muscle damage recovery (19). A total of 39 participants participated in the present study. Participants who were free of metabolic, cardiovascular, and musculoskeletal diseases and had no dairy allergies were eligible to participate. To qualify for the study, participants had to self-report participating in ≥3 days/week of resistance training for ≥3 months. Only participants with low cardiovascular disease risk and no contraindications to physical exercise were allowed. All participants performed a barbell back squat with an estimated one-repetition maximum (1RM) of ≥1.5 and ≥1.25 times their body mass for male and female participants, respectively [obtained during visit 1 (V1)] (20,21). A single questionnaire administered during V1 was used to identify contraceptive usage of female participants; six (n = 3 in placebo group, n = 2 in CWP group, and n = 1 in PRWP group) utilized contraceptives, while seven (n = 1 in placebo group, n = 2 in CWP group, and n = 4 in PRWP group) were not using contraceptives. The Indiana University Institutional Review Board approved procedures (#12559) on September 28, 2021; the study protocol was registered under ClinicalTrials.gov (identifier: NCT05100459), and written informed consent was obtained from all participants before enrollment.

Protocol

Our previous work (19) reported that participants underwent five visits in this double-blind, randomized, isocaloric placebo-controlled trial. Designed to maximize ecological validity, this experiment tested the application of consuming different whey protein supplements (i.e., PRWP and CWP) or a non-protein control (NPC), three times per day, on peripheral and central hemodynamics and arterial stiffness during the 72-hour recovery period following eccentric exercise, in free-living, young and healthy, resistance-trained participants. The peripheral and central hemodynamic response to EML included peripheral and central blood pressure, while arterial stiffness measures included carotid-

femoral pulse wave velocity and wave reflection parameters. These measures were performed during visit 2 (pre-EIMD; V2), visit 3 (24 h post-EIMD; V3), visit 4 (48 h post-EIMD; V4), and visit 5 (72 h post-EIMD; V5) (Figure 1).

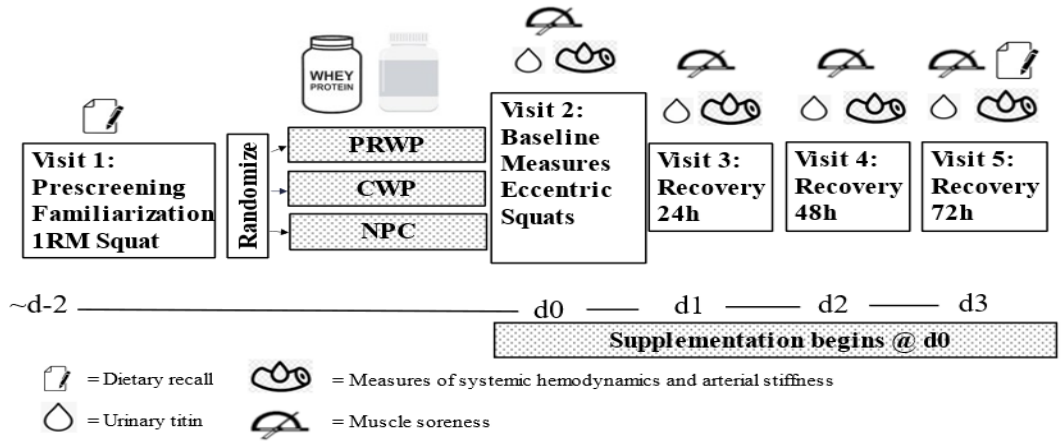

Figure 1. Schematic of the randomized, double-blind, and parallel group study design. d, days (time from baseline visit); 1RM, one repetition maximum (estimated); CWP, conventional whey protein; PRWP, pasture-raised whey protein; NPC, non-protein control. (19).

During visit 1 (V1), participants were familiarized with oscillometric blood pressure and applanation tonometry procedures. Following familiarization, an estimated 1RM back squat test was performed using the Berger prediction equation (22), as previously explained (19). As described before (19), dietary intake recall data were collected and analyzed using the Web-based Automated Self-Administered 24-Hour (ASA24) Dietary Assessment Tool (23). Subjects were told to consume their habitual diets throughout the study period.

Visits 2 through V5 happened ± 1 h of the day to account for diurnal variation in peripheral and central hemodynamics and arterial stiffness. Participants were instructed to refrain from anti-inflammatory/pain medications, rigorous exercise, weight training, vitamins, ergogenic nutraceuticals (e.g., vasodilators), alcohol, and any recovery modality (e.g., foam rolling) for 48 h before V2 and for the duration of the study. In addition, participants were asked not to eat or consume caffeine for 180 minutes before V2-V5; this restriction was based on the consensus document by Van Bortel and colleagues on measuring aortic stiffness using carotid-femoral pulse wave velocity (24). Before hemodynamic measures during V2-V5, participants filled out a health history update and a supplementation compliance questionnaire. Following completion of the questionnaires, hydration was quantified using urine specific gravity (USG) measured by refractometry; USG < 1.020 indicated euhydration (25). If participants had a urine specific gravity greater than 1.020, they received 300 mL of water. Urine was also collected to assess muscle damage from eccentric squats via the N-terminal titin fragment (26). Stature was measured with a stadiometer (Seca® 213, Hamburg, Germany), and body mass was measured with a Tanita MC-780U (Tanita Corp., Tokyo, Japan). Participants completed a muscle soreness assessment using a visual analog scale after anthropometric measurements. During V2 (baseline), hemodynamic and arterial stiffness measures were obtained 10 minutes before the maximal isometric voluntary contraction (MIVC) test. After five minutes of recovery from the MIVCs, participants underwent a series of eccentric barbell back squats to induce muscle damage.

Supplementation

Following the EML protocol, participants were allocated to consume either PRWP (130 calories, 25 g protein), CWP (130 calories, 25 g protein), or a non-protein-containing iso-caloric shake (control; 130 calories, 32 g carbohydrates, 0 g protein) beverage. Participants took their designated supplements in the mid-morning, immediately post-study visit (V2-V4), and before bedtime on the days following the EML bout. This supplementation period extended over four

days, covering visits V2-V5. Both whey protein samples were analyzed for protein content by mass spectrometry, as reported in our previous work (19).

Randomization and Blinding
The randomization sequence was stratified by gender (male and female) and lifting ability (moderate and high), yielding blocks of six participants per treatment group. High lifting ability for males was defined as an estimated 1RM ≥ 1.75 times body mass (27), while for females, it was described as an estimated 1RM ≥ 1.5 times body mass (21). Another person, not affiliated with this investigation, labeled the protein and control supplements in blinded containers.

Peripheral and Central Hemodynamics
After recording their body mass, participants rested for 5 minutes in the supine position on a standard athletic table. Before the five-minute rest period, participants were fitted by the research team with a standard brachial and femoral blood pressure cuff connected to an automated oscillometric device (ATCOR SphygmoCor®; Naperville, IL, USA). All blood pressure measurements were performed in duplicate and averaged for subsequent analysis.

Arterial Stiffness and Wave Reflection
Measures of wave reflection were assessed using a brachial arm cuff equipped with a strain gauge (ATCOR SphygmoCor®, Naperville, IL, USA) (28). Using pulse wave analysis via the ATCOR system, augmentation index (AIx), central augmentation pressure (AP), AIx normalized for heart rate at 75 BPM (AIx75), subendocardial viability ratio (SEVR), and reflection magnitude (%) were calculated (29). Brachial artery compression waveforms were acquired by partially inflating a cuff over the brachial artery. The previously mentioned variables were derived from the brachial pressure waveform, using a generalized transfer function and proprietary digital signal processing to create a central pressure waveform (30,31). Therefore, all parameters were automatically calculated using the transfer function in the ATCOR SphygmoCor® software (32). The mean of two high-quality measurements, determined by the software, was used at each time point.

The ATCOR SphygmoCor® device measured carotid-femoral pulse wave velocity using applanation tonometry (33). Following the pulse wave analysis measurement, a femoral blood pressure cuff and carotid tonometer were applied simultaneously to obtain pressure waveforms at the carotid and femoral artery sites. The SphygmoCor® software uses physical distance measurements between sites to calculate pulse wave velocity in milliseconds. A measuring tape calculates the surface distance from the suprasternal notch to the carotid and femoral recording sites. The recording site was marked with indelible ink during the first hemodynamic assessment (V2) to ensure consistency across all subsequent visits. The mean of the two measurements that met the ATCOR SphygmoCor® software standards at each time point was used.

Maximal Isometric Voluntary Contraction (MIVC)
As detailed in our previous work (19), three five-second (100% effort) MIVCs with one-minute rest were completed. Knee extensor force during voluntary contractions was used to quantify muscle damage and measured with a calibrated load cell (model Z Tension Load Cell; Dillon, Fairmont, MN, USA). The load cell was fixed to a table and connected to a noncompliant cuff attached just superior to the ankle malleoli of the subject's right leg. The load cell height was adjusted for each subject to maintain a line of action parallel to the applied force. The noncompliant strap was attached to the load cell for force measurement and connected to a custom amplifier (Hector Engineering Co., Inc., Ellettsville, IN, USA). The signal was ultimately sampled at 2000Hz and analyzed using AcqKnowledge Software v5.0 (BIOPAC Systems, Goleta, CA, USA). The highest force value was used for analysis.

Eccentric Muscle Loading Protocol
As previously described (20), the EML stimulus consisted of 10 sets of 10 repetitions of barbell back squats at 60% of their estimated 1RM, determined during V1. The tempo, for each squat, was a four-second eccentric contraction, no pause, and a one-second concentric contraction (34). This barbell back squat protocol has been used in previous studies to elicit EIMD indicators in trained individuals (19,34,35). Heart rate and ratings of perceived exertion (RPE) were recorded using a chest strap and 10-point RPE scale throughout the squat protocol. Immediately following the EML protocol, the MIVC protocol was repeated to confirm EIMD occurred in response to the squat protocol. Moderate muscle damage was characterized by a decrease in quadriceps muscle strength of ≥20% following EIMD (36). If a 20% or more significant reduction in MIVC force did not happen, two further squat sets were completed until this threshold was met. Ten of the 39 subjects required additional barbell back squats.

Urinary Titin
As previously detailed (19), urine samples were collected to quantify urinary titin levels using a solid-phase sandwich enzyme-linked immunoassay (ELISA) (Human Titin N-Fragment ELISA® Kit, #27900, Immuno-Biological Laboratories, Inc., Minneapolis, MN, USA).

Muscle Soreness
Muscle soreness was evaluated utilizing a visual analog scale (VAS). Participants performed a standing bodyweight squat (no external load) to a 90-degree knee angle (as confirmed by the research team), holding the bottom position for three seconds. This scale ranges from 0 mm (indicating no muscle soreness) to 100 mm (representing the most severe muscle soreness).

Statistical Analysis
Summary statistics, including mean, standard deviation, median, and range, were calculated for all variables of interest (IBM SPSS). Baseline participant characteristics (i.e., stature and body mass) across groups (NPC, CWP, and PRWP) were compared using a one-way analysis of variance. The consistency of participant body mass and hydration for V2-V5 was assessed by calculating the intraclass correlation coefficient. The impact of supplementation on blood pressure and pulse wave analysis parameters (i.e., PWV, AIx, SEVR) was evaluated using linear mixed models, with time (pre, 24, 48, and 72 h post-EML) and treatment (NPC vs. CWP vs. PRWP) as fixed factors, including the interaction between them, and participant identification as the random effect. Model assumptions were evaluated, and the normality of the residuals was confirmed. Estimated marginal means from the models, utilizing Bonferroni's adjustment for multiple comparisons, were employed to test for differences between groups at V2-V5. The statistical significance level was set at $\alpha = 0.05$. Data are expressed as mean ± SD and 95% confidence intervals (CI).

Our sample size was based on the outcomes of our original research aim (muscle damage and performance measures), as previously explained (19). Briefly, a power analysis performed with G*POWER 3.1.9.7 (University of Kiel, University of Düsseldorf, and University of Mannheim, Germany) calculated that 30 participants were needed with a power of 0.80, an effect size (f) of 0.25, and an $\alpha = 0.05$ to detect within- and between-group differences. Other studies examining the effects of eccentric exercise on arterial stiffening 24, 48, and 72 hours post-EML had sample sizes of 27 (2), 12 (3), and 20 (37), respectively, which are lower than this study's sample size of 39.

Results
As previously reported in our work (19), baseline characteristics did not differ significantly (Table 1). Eccentric back squats significantly reduced MIVC from pre- to immediately post-back squats ($P < 0.001$), with no between-group difference ($P = 0.722$). The average heart rates for all groups at the end of all squat sets did not differ significantly ($P \geq 0.05$). In addition, RPE at the end of the 5th set did not differ between groups ($P = 0.349$). The mean RPE for the 10th set across the three groups was 9.6 ± 0.7. Supplementation compliance was 100%.

Table 1. Participant characteristics.

	PRWP (*n* = 14, 5F/9M)	**CWP (*n* = 12, 4F/8M)**	**NPC (*n* = 13, 4F/9M)**	***P*-Value**
Age (y)	23 ± 6	23 ± 5	22 ± 4	0.882
Stature (cm)	171 ± 9	173 ± 9	173 ± 10	0.756
Body mass (kg)	75.7 ± 15.0	76.4 ± 15.6	75.6 ± 15.4	0.991
BMI (kg/m^2)	26.0 ± 4.0	25.7 ± 4.2	25.2 ± 3.5	0.878
FFM (kg)	59.3 ± 10.8	60.4 ± 11.0	60.4 ± 11.7	0.956
Body fat (%)	21.5 ± 6.4	21.3 ± 6.4	20.4 ± 6.0	0.876
1RM/body mass	1.64 ± 0.36	1.60 ± 0.17	1.58 ± 0.17	0.817
Training History (days per week)	4.6 ± 1.2	4.3 ± 1.0	4.3 ± 1.0	0.663
Training History (hours per day)	1.6 ± 0.5	1.5 ± 0.7	1.5 ± 0.5	0.950

Values are mean ± SD. F, Females; M, Males; BMI, body mass index; FFM, fat-free mass; 1RM/body mass, estimated one-repetition maximum barbell back squat divided by body mass; PRWP, pasture-raised/grass-fed whey protein; CWP, conventional whey protein; NPC, non-protein control. (19).

The peripheral hemodynamic responses to the EML bout are depicted in Table 2. Brachial systolic blood pressure (SBP) and pulse pressure (PP) were unaffected throughout the recovery period, both within and between groups ($P \geq 0.05$). The PRWP group experienced a significant increase of 5.3 ± 1.7 mm Hg (95% CI, 0.9 to 9.8 mm Hg) in brachial diastolic blood pressure (DBP) between pre-EML and 48h post-EML ($P = 0.011$). As a result, the CWP group had a significantly lower mean DBP of 11.4 ± 3.6 mm Hg (95% CI, 2.6 to 20.2 mm Hg; $P = 0.007$) compared to the PRWP group. Furthermore, the NPC group's DBP was 10.4 ± 3.5 mm Hg (95% CI, 1.8 to 19.0 mm Hg), significantly lower ($P = 0.012$) than the PRWP group at 48h post-EML.

Table 2. Peripheral hemodynamic response to EML.

Variable Group	Time Post-EML, Hours			
	Pre-EML	**24**	**48**	**72**
Brachial				
SBP, mm Hg				
PRWP	128 ± 14	130 ± 15	130 ± 15	130 ± 16
CWP	130 ± 16	125 ± 16	125 ± 14	127 ± 16
NPC	127 ± 9	129 ± 10	126 ± 12	124 ± 9
DBP, mm Hg				
PRWP	72 ± 10	73 ± 12	77 ± 11*	73 ± 9
CWP	69 ± 8	68 ± 7	66 ± 9**	69 ± 7
NPC	69 ± 7	68 ± 9	67 ± 10**	67 ± 6
PP, mm Hg				
PRWP	56 ± 9	56 ± 10	52 ± 9	56 ± 11
CWP	61 ± 10	57 ± 11	59 ± 12	58 ± 12
NPC	58 ± 8	60 ± 9	59 ± 10	57 ± 7

Mean ± SD. *$P < 0.05$ vs. pre-EML. **$P < 0.05$ vs. Pasture-raised/Grass-fed whey protein group within time point. EML, eccentric muscle loading; PRWP, pasture-raised/grass-fed whey protein; CWP, conventional whey protein; NPC, non-protein control; SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure.

The central hemodynamic responses to the EML bout are displayed in Table 3. The CWP group experienced a significant mean reduction in central SBP of 5.7 mm Hg (95% CI, -11.1 to -0.2 mm Hg) at 48h post-EML compared to pre-EML ($P = 0.038$). The PRWP group experienced a significant central DBP rise of 5.4 ± 1.7 (95% CI, 0.9 to 9.8 mm Hg) at 48h post-EML compared to pre-EML ($P = 0.009$). This resulted in the CWP and NPC groups having a significantly lower central DBP at 48h post-EML of 11.6 ± 3.5 (95% CI, 2.9 to 20.3 mm Hg; $P = 0.005$) and 10.0 ± 3.4 (95% CI, 1.5 to 18.5; $P = 0.015$) mm Hg, respectively, compared to the PRWP group. Central pulse pressure and heart rate were unaffected throughout the recovery period ($P > 0.05$). Central mean arterial blood pressure at 48h post-EML was 11.2 ± 4.0 (95% CI, 1.5 to 20.9 mm Hg), significantly lower in the CWP group compared to the PRWP group ($P = 0.018$).

Aortic augmentation pressure (AP), augmentation index (AIx), augmentation index normalized to a heart rate of 75 bpm (AIx75), subendocardial viability ratio (SEVR), and reflection magnitude (%) were not altered during the recovery period within groups ($P > 0.05$). There was no significant difference in AP with respect to group ($P = 0.992$), recovery period visits ($P = 0.902$), or group by visit interaction ($P = 0.163$). There was no significant difference in AIx with respect to group ($P = 0.949$), recovery period visits ($P = 0.907$), or group by visit interaction ($P = 0.316$). There was no significant difference in AIx75 with respect to group ($P = 0.630$), recovery period visits ($P = 0.652$), or group by visit interaction ($P = 0.543$). There were no significant differences in SEVR with respect to group ($P = 0.106$), recovery period visits ($P = 0.148$), or group by visit interaction ($P = 0.835$). There were no significant differences in reflection magnitude percentage with respect to group ($P = 0.923$), recovery period visits ($P = 0.289$), or group by visit interaction ($P = 0.674$).

The pulse wave velocity response to the eccentric exercise bout and supplementation is presented in Figure 2. There was a small, yet significant time effect reduction in pulse wave velocity (PWV) heart rate of all groups at the 48h visit compared to the 24h visit (-2.6 ± 1.0 bpm; 95% CI, -5.3 to -0.01 bpm, P = 0.048). There was no significant difference ($P \geq 0.05$) in PWV heart rate and pulse transit time at any time point during recovery between groups. PWV was not significantly different between groups ($P = 0.598$), visit ($P = 0.753$), or group by visit interaction ($P = 0.418$).

Table 3. Central hemodynamic response to EML.

Variable		Time Post-EML, Hours		
Group	**Pre-EML**	**24**	**48**	**72**
Central SBP, mm Hg				
PRWP	111 ± 13	113 ± 14	112 ± 14	112 ± 15
CWP	112 ± 14	107 ± 14	106 ± 11*	108 ± 13
NPC	108 ± 8	109 ± 9	108 ± 10	106 ± 9
Central DBP, mm Hg				
PRWP	73 ± 10	74 ± 11	78 ± 11*	75 ± 9
CWP	71 ± 8	69 ± 7	67 ± 8**	70 ± 7
NPC	70 ± 7	69 ± 9	68 ± 10**	68 ± 6
Central PP, mm Hg				
PRWP	37 ± 7	38 ± 8	35 ± 6	38 ± 9
CWP	42 ± 7	38 ± 9	39 ± 8	39 ± 8
NPC	38 ± 6	40 ± 6	39 ± 7	38 ± 6
Central MAP, mm Hg				
PRWP	88 ± 11	89 ± 13	92 ± 12	89 ± 11
CWP	85 ± 10	83 ± 9	81 ± 9¥	84 ± 9
NPC	85 ± 7	85 ± 9	83 ± 9	82 ± 6
Heart Rate, bpm				
PRWP	63 ± 8	62 ± 9	63 ± 8	63 ± 9
CWP	58 ± 12	59 ± 8	57 ± 9	58 ± 9
NPC	66 ± 8	65 ± 8	61 ± 8	63 ± 7

Mean ± SD. *$P < 0.05$ vs. pre-EML. **$P < 0.05$ vs. Pasture-raised/Grass-fed whey protein group within time point. ¥$P < 0.05$ vs. Pasture-raised/Grass-fed whey protein group within time point. EML, eccentric muscle loading; PRWP, pasture-raised/grass-fed whey protein; CWP, conventional whey protein; NPC, non-protein control; SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure; MAP, mean arterial pressure.

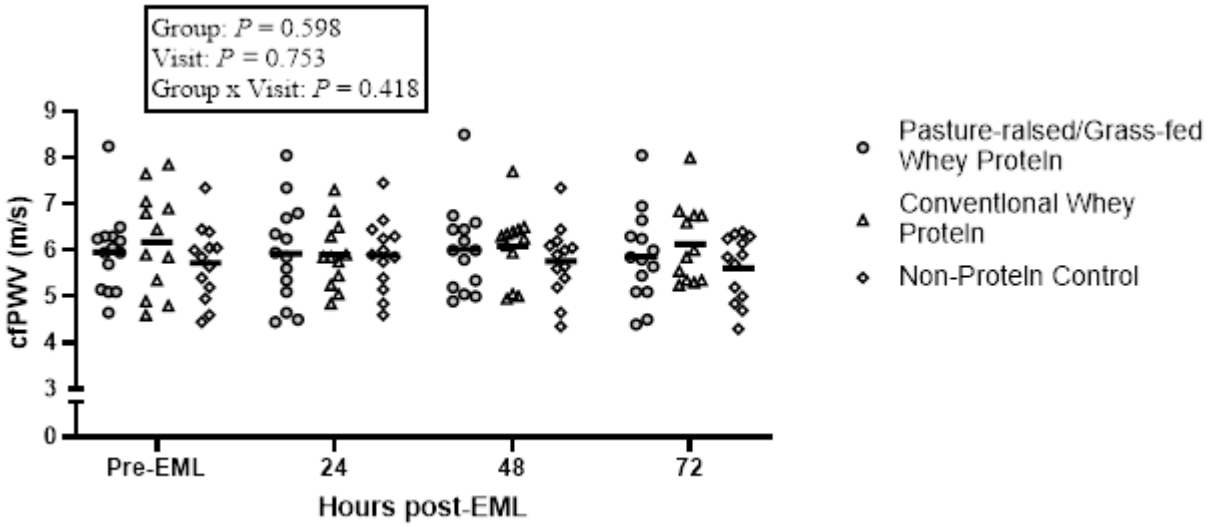

Figure 2. Individual carotid-femoral pulse wave velocity response to eccentric muscle loading. Individual measures of carotid-femoral pulse wave velocity between PRWP, CWP, and NPC at pre-EML, 24 h, 48 h, and 72h post exercise-induced muscle damage. PRWP, Pasture-raised/Grass-fed Whey Protein; CWP, Conventional Whey Protein; NPC, Non-protein control; m/s, meter per second; cf, carotid-femoral; PWV, pulse wave velocity. Thick lines indicate the mean.

As previously reported (19), the overall time effects of skeletal muscle damage, via titin, peaked 24 hours post-EML ($P < 0.001$; Δ, 135.4 ± 32.1%; 95% CI, 49.1 to 221.8%) compared to pre-EML. No significant difference in time was observed between pre-EML and 48 hours ($P = 0.256$). A significant increase in urinary titin across all groups was observed at the 72-hour visit compared with baseline ($P = 0.010$; Δ, 104.0 ± 32.1%; 95% CI, 17.6 to 190.4%). No significant within-group differences ($P > 0.05$) were detected in the NPC and CWP groups throughout the study period. In the PRWP group, the 24-hour visit showed a significant increase of 149.3 ± 53.5% (95% CI, 5.5 to 293.2%) ($P = 0.037$). Figure 3 displays no differences between groups ($P = 0.580$) or visit-by-group interaction ($P = 0.807$).

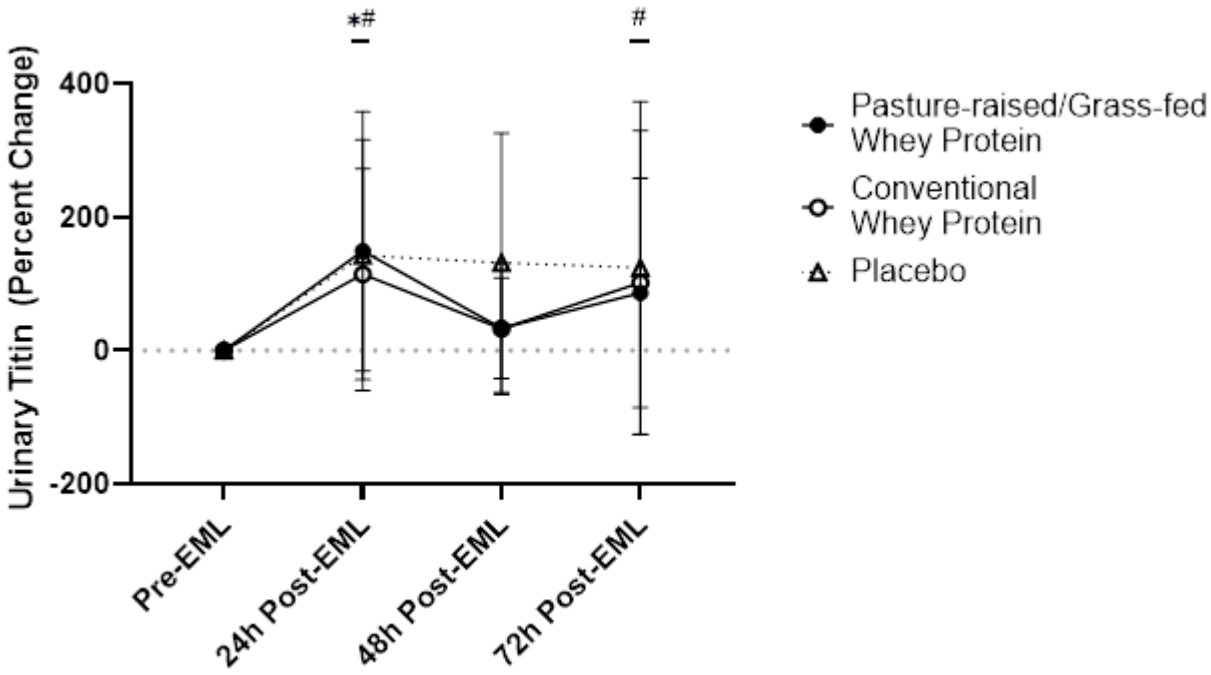

Figure 3. Urinary titin in percent change response to eccentric muscle loading. Measures of urinary titin between PRWP, CWP, and NPC at pre-EIMD, 24 h, 48 h, and 72h EML. PRWP, Pasture-raised/Grass-fed Whey Protein; CWP, Conventional Whey Protein; NPC, non-protein control. *indicates PRWP group significantly increased at 24 h post-EML (19) #indicates significant overall time effect for all groups compared to baseline.

As previously reported (20), muscle soreness increased significantly 24 hours post-EML in all groups (33.8 ± 3.1; 95% CI, 25.5 to 42.0; $P < 0.05$) and remained elevated 48 hours post-EML compared with baseline. No significant difference in overall muscle soreness was found between groups ($P = 0.515$) or in the time-by-group interaction ($P = 0.695$). All groups returned to baseline levels of muscle soreness at the 72-hour visit post-EML.

Table 4 displays participants' dietary intake before and during recovery (with supplementation). As reported earlier (19), there was no significant difference in dietary intake between groups before supplementation.

During the recovery period (with supplementation), no significant differences between groups were observed for energy consumption ($P = 0.442$), fat intake ($P = 0.398$), protein intake ($P = 0.060$), and fat intake relative to body mass ($P = 0.346$). There were significant differences regarding carbohydrate intake during V2-V5 between both WPC groups (PRWP; $P = 0.014$ and CWP; $P = 0.014$) compared to NPC. Protein intake relative to body mass significantly differed between the CWP and NPC groups ($P = 0.033$), but not between the PRWP and NPC groups ($P = 0.057$). Carbohydrate intake relative to body mass differed between PRWP and NPC groups ($P = 0.049$), but not between CWP and NPC ($P = 0.051$). Aside from the macronutrients provided by the supplements, no significant differences were observed concerning protein ($P = 0.653$), carbohydrate ($P = 0.940$), and fat ($P = 0.202$) consumption relative to body mass during V2-V5.

Table 4. Participants' dietary intake before and during the supplementation period.

	PRWP (n = 14)		CWP (n = 12)		NPC (n = 13)	
	Before	**During**	**Before**	**During**	**Before**	**During**
Energy (kcal)	2,445 ± 704	2,528 ± 645	2,474 ± 1,127	2,613 ± 862	2,675 ± 1,196	3,016 ± 1,445
Protein (g)	140 ± 54	192 ± 37	134 ± 63	208 ± 59	125 ± 62	144 ± 96
Carbohydrate (g)	246 ± 93	226 ± 87*	249 ± 125	236 ± 89*	297 ± 122	343 ± 124
Fat (g)	101 ± 35	96 ± 28	102 ± 50	93 ± 41	113 ± 59	121 ± 84
Protein relative (g·kg^{-1})	1.8 ± 0.6	2.6 ± 0.6	1.7 ± 0.7	2.7 ± 0.6¥	1.6 ± 0.7	1.9 ± 1.1
Carbohydrate relative (g·kg^{-1})	3.3 ± 1.5	3.2 ± 1.5¥	3.2 ± 1.5	3.1 ± 1.2	3.9 ± 1.3	4.5 ± 1.6
Fat relative (g·kg^{-1})	1.4 ± 0.5	1.3 ± 0.4	1.3 ± 0.5	1.2 ± 0.5	1.5 ± 0.7	1.5 ± 0.8

Data are presented as mean ± SD. *PRWP and CWP differ from NPC ($P < 0.05$). ¥ Different from NPC ($P < 0.05$). PRWP, pasture-raised/grass-fed whey protein; CWP, conventional whey protein; NPC, non-protein control. (19).

Discussion

This study tested the hypothesis that PRWP supplementation, compared to CWP supplementation, would reduce arterial stiffness to a greater extent during the recovery period following EML in resistance-trained individuals. To the best of our knowledge, this study is the first to investigate the impact of consuming pasture-raised/grass-fed products on various vascular parameters following high-intensity eccentric exercise in this population.

Firstly, contrary to other studies that used eccentric exercise to induce muscle damage (2,3,7,35,36), the selected muscle-damaging protocol (i.e., barbell back squats) in this study did not adversely affect the primary outcome, arterial stiffening measured by pulse wave velocity, at any post-EML time point across groups. Secondly, supplementation with PRWP and CWP during the recovery period produced similar effects at 24 and 72 hours post-EML; however, in contrast to our hypothesis, they differed at 48 hours post-EML in systemic hemodynamics (DBP). To confirm EIMD induction using the squat protocol, urinary titin was measured during recovery. Studies have demonstrated that muscle damage can initiate cytoskeletal and sarcomeric disruption, including calpain-mediated titin degradation (38). Our findings, indicating elevated urinary titin concentrations up to 72 hours after the recovery period, align with other studies (39–41) showing an increase in urinary titin following eccentric exercise. An overall time effect was observed, with a ~135% increase in urinary titin at 24 hours post-EML. Tanabe and colleagues (42) found a ~81% increase 24 hours after eccentric activity, while Yamaguchi detected a ~172% increase (39). This confirms muscle damage in our study following the barbell back squat protocol. Additionally, muscle soreness was significantly elevated in all groups for up to 48 hours during recovery. This supports our urinary titin data, which suggest that EIMD resulted from the barbell back squat protocol. EIMD is associated with muscle soreness and discomfort 12-72 hours post-eccentric exercise, depending on the stimulus of the muscle-damaging exercise, and is related to disruptions of myofibrillar proteins (36).

Eccentric-dominant exercise has been observed to elevate arterial stiffness through pulse wave velocity at 48- and 72-hours post-EML (2,3,7). An eccentrically based leg press exercise increased central arterial stiffness at 48 hours post-exercise in young adults, an effect the authors deemed unfavorable for cardiovascular outcomes (2). Research has shown that arterial stiffening, as indicated by nearly a 10% increase in cfPWV and muscle soreness, tends to peak 48 hours after eccentric exercise in a mixed-sex population of recreationally active individuals (3). This phenomenon is linked to the heightened inflammatory response following EIMD. For instance, Lin and colleagues (37) used both downhill running and an eccentrically biased leg press exercise, resulting in 17% and 13% increases in cfPWV, respectively, in healthy young individuals. In line with the previously mentioned experiments (2,3,7), several reviews of resistance training and its impact on arterial stiffness have consistently reported an increase in arterial stiffness (43–45).

A possible explanation for the lack of significant effects on arterial measures (i.e., cfPWV, cfPTT, augmentation pressure, augmentation index, augmentation index at 75 bpm, subendocardial viability ratio, and reflection wave magnitude) resulting from the chosen eccentric exercise regimen could be attributed to the fact that our study's participants were well-trained individuals with a healthy vascular system, rendering them well-adapted to the specific EML protocol. In contrast, previous studies reporting increased arterial stiffness following eccentric exercise were conducted among recreationally active individuals and participants with unknown strength-to-body mass ratios (2,7), potentially leading to adverse vascular effects. Moreover, in physically active individuals, long-term resistance exercise has been shown to reduce arterial stiffness (46). It can be speculated that arterial stiffness may be minimized or prevented, as observed in the repeated-bout effect on muscle recovery measures, such as reduced delayed-onset muscle

soreness (47). This assumption is further supported by the observation that most baseline and post-EML pulse wave velocity values were within normal ranges, underscoring the study participants' vascular health (48).

The diastolic blood pressure findings from both peripheral and central measurements at the 48-hour post-EML contrast with prior research indicating that eccentric exercise does not affect blood pressure (2,3,49). Unexpectedly, the PRWP group exhibited significant elevations in both central and peripheral DBP at this time point. This increase may not represent a true physiological response but instead reflect random variation or a chance finding resulting from multiple statistical comparisons. Additionally, variability in bioactive whey protein peptides such as κ-casein and α-lactalbumin, which have antihypertensive properties via ACE inhibition (20), may have contributed to this outcome, although this remains speculative and warrants further investigation (12). Notably, substantial evidence demonstrates that whey protein supplementation generally lowers DBP (12,50), further complicating the interpretation of these findings. The 48-hour post-EML visit regarding CWP supplementation and its resulting reduction in central SBP supports data on whey protein's ability to lower SBP (51). It is currently unclear why PRWP did not affect SBP at this time point. However, the primary vascular health measure in this experiment was pulse wave velocity, which remained unaffected across all recovery time points, potentially diminishing the significance of the blood pressure findings.

The literature provides ample data supporting the use of whey protein for vascular health (12,16,51–53). Therefore, it was hypothesized that whey protein supplementation would beneficially modulate vascular parameters and/or protect against vascular disturbances post-EML. In healthy individuals, two weeks of five grams per day ingestion of a whey protein-derived bioactive peptide enhanced vascular endothelial function through nitric oxide-dependent and independent mechanisms (52). More acutely, a single 20-gram serving of whey protein in healthy young adults elevated flow-mediated dilation 30 minutes post-ingestion (16).

However, not all data support whey protein's beneficial impact on cardiovascular hemodynamics, as 44 grams of whey protein produced no post-exercise effects in young individuals (54). Most of the literature on whey protein and vascular health focuses on individuals with poor metabolic health (12,55). Hence, data on the effects of whey protein on peripheral and central hemodynamics and arterial stiffness post-EML in resistance-trained individuals are sparse.

In support of this study's findings on the effects of strenuous exercise on various vascular parameters, another research group, using high-intensity functional training in young and active adults, found no impact of this training modality on augmentation pressure, pulse pressure, or augmentation index at 75 bpm (56). Strenuous resistance training may temporarily strain the cardiovascular system (56–58). Still, the present data and other research indicate that this exercise mode is neutral and/or beneficial for the cardiovascular system (45,59,60).

This study has several limitations. We might have overlooked elevated arterial stiffness findings, as other trials use assessment time points less than 24 hours post-EML (i.e., 10 minutes post-exercise) (58,61). The absence of the EML stimulus, which would typically impact pulse wave analysis and pulse wave velocity, was surprising. However, it's worth noting that simple calf raises, which engage significantly less muscle mass than free-weight barbell back squats, have been found to induce systemic vascular dysfunction (6,62,63). Therefore, it seemed reasonable to conclude that the more extensive whole-body movement (barbell back squat) would elicit similar, if not more pronounced, vascular disturbances. Furthermore, the RPE data indicated near-maximal effort for all groups during the EML bout, signifying a strenuous exercise session. Therefore, the unexpected outcome of a strenuously perceived whole-body EML bout (with moderate muscle damage, as evidenced by decreased MIVC, elevated urinary titin, and muscle soreness) and its resulting null effects on the currently used vascular parameters were unexpected. These findings may not be generalizable to untrained individuals or clinical populations, who may exhibit greater vascular vulnerability and respond differently to high-intensity EML exercise Lastly, this study did not assess morphological structure or biomarkers related to the vascular system and/or inflammation. Inflammatory biomarkers are associated with arterial stiffness and EML, as these cytokines impair nitric oxide production and promote elastin degradation (36,64). If implemented, these measures could have provided valuable insights into the interpretation of the results.

Conclusions

In conclusion, the present study's findings indicate that a bout of high-intensity, eccentric-dominant barbell back squats did not negatively affect central or peripheral hemodynamics, nor did it alter arterial stiffness during the recovery period in resistance-trained individuals. Furthermore, supplementation with PRWP or CWP during recovery from EML did not result in significant changes in systemic hemodynamics or arterial stiffness. Future research should examine vascular responses and related biomarkers during the immediate and more acute (i.e., 10 minutes and two

hours) phases of recovery following EML and determine whether earlier supplementation with whey protein confers vascular benefits during these phases.

Conflict of Interest

The authors declare no conflicts of interest.

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