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*Original Research*

# The Effects of Caffeine and Resistance Exercise on Pulse Wave Reflection in Resistance-Trained Women

Sarah G. Kearney†[1], Therese M. Smith*[2], Meredith C. Paskert Joplin†[3], Erica M. Marshall†[4], Jason C. Parks†[5], Stacie M. Humm†[3], Michelle A. Kern*[2], J. Derek Kingsley‡[6]

[1]Exercise Science, Grand Valley State University, Allendale, MI, USA; [2]Exercise Science and Exercise Physiology, Kent State University, Kent, OH, USA; [3]Exercise Science, Lake Erie College, Painesville, OH, USA; [4]Exercise Science, Florida Southern College, Lakeland, FL, USA; [5]Kinesiology, State University of New York at Cortland, Cortland, NY, USA; [6]Exercise and Sport Science, Keiser University, West Palm Beach, FL, USA

*Denotes student investigator, †Denotes early-career investigator, ‡Denotes established investigator

## Abstract

***International Journal of Exercise Science 18(6): 1310-1320, 2025.*** https://doi.org/10.70252/RWDY4284 Caffeine, alone or in conjunction with an acute bout of resistance exercise (RE), increases measures of pulse wave reflection (PWR), with most studies focusing on men. Therefore, the purpose of this study was to investigate caffeine's effects on measures of PWR alone and following an acute bout of RE in young, healthy, resistance-trained women. Eleven resistance-trained women completed an acute bout of RE using free-weights with repetitions to failure for squat and bench press with caffeine (4mg/kg) and a placebo. Measures of PWR were measured at Rest, 50 minutes following ingestion (caffeine or placebo), immediately following the acute bout of RE and after 10 minutes of recovery. There were no differences ($p > 0.05$) between caffeine and placebo conditions for measures of PWR at any time point. Aortic pulse pressure (APP), augmentation pressure (AP), augmentation index (AIx), augmentation index normalized at 75bpm (AIx@75), systolic pressure time index (SPTI), and left ventricular wasted energy (ΔEw) significantly increased ($p < 0.01$) following the acute bout of RE for up to 10 minutes in both conditions while diastolic pressure time index (DPTI) significantly decreased ($p < 0.01$). There was no change in aortic systolic blood pressure (ASBP), aortic diastolic blood pressure (ADBP), time of the reflected wave (Tr) and subendocardial viability ratio (SEVR) following the acute bout of RE in either condition ($p > 0.05$). Collectively, this study suggests that a caffeine dose of 4mg/kg does not alter measures of PWR beyond the individual influence of an acute bout of RE in resistance-trained women.

Keywords: Cardiovascular, augmentation index, supplementation, placebo, subendocardial viability ratio

## Introduction

Measures of pulse wave reflection (PWR) include the augmentation index (AIx), the AIx at 75bpm (AIx@75), augmentation pressure (AP), and subendocardial viability ratio (SEVR). These

measures may be used to predict cardiovascular events and have been shown to be superior to brachial blood pressure (BP).[1,2] In addition, it has been demonstrated that changes in measures of PWR may be risk factors for the development of cardiovascular disease.[1,2] Measures of PWR, primarily the AIx and AP, are a direct result of increases in arterial reservoir pressure, contraction and relaxation of the heart, and forward traveling waves in the aorta,[3–5] while SEVR is an indirect measure of myocardial perfusion and left ventricular workload.[6] It has been demonstrated that cardiovascular incidences may increase in relation to changes in the AIx mediated by increases in left ventricular afterload and wasted left ventricular energy (ΔEw).[7,8] Therefore, measurements of PWR can be used to noninvasively examine how the cardiovascular system may be impacted by an acute stressor. In response to an acute bout of resistance exercise (RE) measures of PWR are significantly increased.[9–12] However, the majority of these studies were in men, with women included, but did not have sufficient power to evaluate sex differences. Previous data have demonstrated that young, healthy women may have a significantly increased resting AIx when compared to men.[13–15] Furthermore, it has also been suggested that young, healthy women have an increased resting AIx@75 and ΔEw,[16] with significantly reduced SEVR,[17] compared to men. The differences between the sexes on measure of PWR may be mediated by estrogen, as well as the shorter arterial tree in women. Since women have a shorter arterial tree compared to men this in turn may result in a faster heart rate (HR), such that the reflected wave would return during, or closer to, diastole.[13]

Caffeine is commonly consumed as a "pre-workout" supplement and ergogenic aid taken prior to an acute bout of RE and has been documented to improve performance measures by reducing the onset of fatigue, as well as increasing acute muscular endurance, power and strength.[3,7,13,18] While caffeine may be beneficial for performance, the literature suggests that caffeine alone negatively impacts the cardiovascular system via measures of PWR,[10,15,19] as does an acute bout of RE in the absence of caffeine.[1,2,20] This may be a cause for concern as 70% of adults in the United States regularly consume a caffeinated supplement, with 30% of those doing so to improve exercise performance.[14] With this, the concern is that while RE performance may improve, caffeine in conjunction with an acute bout of RE could further alter measures of PWR beyond their independent effects.

Augmentations in measures of PWR have been shown to last upwards of 3 hours post caffeine ingestion.[10,15,19] These changes include an increase in aortic systolic (ASBP), diastolic (ADBP) blood pressure, AP, AIx and AIx@75,[15,17,19,21] as well as a decrease in the time of the reflected wave (Tr).[15] In the absence of caffeine, an acute bout of RE has also been documented to increase AP, AIx, AIx@75, and ΔEw as well as significantly decrease Tr.[1,2,20] The negative impact on these variables via caffeine and an acute bout of RE could result in greater left ventricular workload and reduced aortic compliance.[15,20] Therefore, caffeine in conjunction with an acute bout of RE could potentially pose a greater threat to the cardiovascular system, however, the literature to support this is sparse.

The few studies that have demonstrated the cardiovascular response to caffeine and an acute bout of RE have primarily included men,[9,11,22,23] with caffeine doses of 2-6mg/kg. Thus, the effects on women are unclear.[7] Additionally, the cardiovascular variables collected are limited to measures of hemodynamics (brachial BP and HR),[7,9,11,22,23] with none, to our knowledge,

investigating measures of PWR. Therefore, the purpose of this study was to examine the effects of caffeine, alone and in conjunction with an acute bout of RE, on measures of PWR in young, resistance-trained women. We hypothesized that 50 minutes following caffeine ingestion alone and in conjunction with an acute bout of RE would lead to: (a) significant increases in measures of PWR including aortic systolic (ASBP) and diastolic blood pressure (ADBP), aortic pulse pressure (APP), AP, AIx, AIx@75, systolic pressure time index (SPTI) and $\Delta E_w$; and (b) reductions in measures of PWR including Tr, diastolic pressure time index (DPTI) and SEVR, immediately and 10 minutes following acute RE when compared to a placebo.

## Methods

This study was a double-blind, placebo controlled cross-over design that aimed to compare the effects of caffeine alone and in conjunction with an acute bout of RE on measures of PWR. Participants completed five visits to the laboratory on separate days, which included an orientation session. Orientation involved the participants completing a Physical Activity Readiness Questionnaire (PARQ+) to determine study eligibility, an informed consent, and a Health History Questionnaire. The second visit involved measuring anthropometrics and maximal strength, and the third session was maximal strength verification. The final two sessions were counterbalanced and consisted of an acute bout of RE with caffeine (4mg/kg) and an acute bout of RE with placebo, separated by a minimum of 72 hours and a maximum of 10 days. To collect data prior to noon, and to account for circadian rhythm, participants arrived at the laboratory between 6 am and 10 am. Participants were instructed to abstain from strenuous physical activity, alcohol, and caffeine for 24 hours, and food for three hours. Measures were assessed at rest (Rest1), 50 minutes following the ingestion of the caffeine and placebo (Rest2), immediately-post acute bout of RE (Rec1), and 10 minutes following the acute bout of RE (Rec2).

### *Participants*

Participants were 11 healthy, resistance-trained women (Table 1). Participants self-reported having performed RE for at least three days a week for one year, as well as their daily caffeine intake, via the Health History Questionnaire. All participants were between 18–30 years of age, had a body mass index (BMI) between 18.5–24.9kg/m$^2$, and were free from cardiovascular, pulmonary, or metabolic disease. In addition, all participants were non-smokers (< 6 months), non-hypertensive (resting brachial systolic blood pressure < 130 mmHg or brachial diastolic blood pressure < 80 mmHg), had no musculoskeletal problems, and were not pregnant or planning to become pregnant. Aside from caffeine, participants reported that they were not taking any medications or supplements that may have affected the outcomes of the study. All data were collected during the early to-mid follicular phase of the menstrual cycle (days 1–9) based on self-report of the participant's last menstrual cycle. All participants signed an informed consent prior to the collection of any data. This research project was approved by the Kent State University Institutional Review Board and was carried out fully in accordance with the ethical standards of the *International Journal of Exercise Science*[24].

**Table 1.** Participant Characteristics (N = 11)

| | |
|---|---|
| Age (yr) | 24 ± 4 |
| Height (m) | 1.7 ± 0.1 |
| Weight (kg) | 67.2 ± 11.4 |
| BMI (kg/m$^2$) | 23.8 ± 3.0 |
| Daily Caffeine Intake (mg/day) | 236 ± 173 |

*Note.* BMI, body mass index. Data are presented mean ± SD

*Protocol*

The present study utilized the National Strength and Conditioning Association one repetition maximum (1RM) guidelines to assess maximal strength on the squat and bench press.[25] Participants began with a five-minute warm-up on a cycle ergometer prior to beginning the 1RM protocol. The initial weight of the 1RM protocol was set at 50–70% of the participant's perceived capacity with the weight being gradually increased by 10–20% for the squat and 5–10% bench press. Three minutes of rest was allotted after each squat and bench press attempt. The maximum amount of weight that was moved through a full range of motion was recorded as the 1RM (Table 1), with no more than five maximal attempts allotted per participant. Seventy-two hours later, participants returned to the laboratory for verification of their 1RM. The ICC for the bench press was 0.97 and was 0.95 for the squat.

For visits four and five, upon arrival to the laboratory, participants rested in a supine position for 10 minutes. Following the 10-minute rest, the brachial pulse wave was assessed to determine measures of PWR using a valid transfer function.[12] The pulse wave captured from the SphygmoCor device (AtCor Medical, SphygmoCor EXCEL Technology, Sydney, Australia) was used to estimate aortic BP (ASBP and ADBP), and the augmentation of the aortic pressure wave form. APP was then calculated via the difference between ASBP and ADBP. Augmentation of the aortic pressure wave is mediated by reflection of the peripheral pressure wave, which is generated by left ventricular ejection.[26] The AIx was calculated through the division of the AP and APP and expressed as a percentage.[4,26] The AIx@75 was calculated in a similar manner where a heart rate of 75bpm is utilized in order to control for the inverse relationship between the AIx and heart rate.[27] The Tr, was calculated as the transit time from ventricular ejection to the arrival of the reflected wave.[4] The SEVR was calculated as the ratio between DPTI, the time spent in diastole, and SPTI, the time spent in systole[4] and is indicative of coronary blood flow. Lastly, $\Delta E_w$ was calculated as follows to determine the wasted energy exerted by the left ventricle, where 1.333 is the conversion factor for mmHg/s to dyne-seconds/cm$^2$: $\Delta E_w = [(\pi / 4) \times (AP \times \Delta Tr) \times 1.333$.[5]

Following Rest1, participants consumed 4 mg/kg of caffeine (Blackburn Distributions, Burnley Lancashire, England) or a placebo that was mixed with 12 oz of flavored water (Crystal Light Lemonade, Chicago, Illinois, USA) and consumed over a five-minute period. For the next 40 min, participants watched a Netflix (Netflix, LosGatos, CA, USA) wildlife documentary series in the seated position. Forty minutes post-ingestion, the participants returned to the supine position, rested for 10 minutes, and PWR measurements were repeated at 50 minutes post-ingestion (Rest2). It has been demonstrated that 50 minutes is sufficient for caffeine to have an effect and reach a plateau that remains stable for at least 50 minutes.[8]

For the caffeine and placebo conditions, participants began with a five-minute warm-up on the cycle ergometer followed by six to eight repetitions at 50% 1RM on the squat. Next, participants completed 2 sets of 10 repetitions at 75% 1RM on the squat, and the third set performed at 70% 1RM with repetitions to failure. Once the squat exercise was completed, the same protocol was followed for the bench press. If participants were unable to complete the 10 repetitions on any set, then the number of repetitions completed was recorded. Two minutes of rest was allowed between each set and each exercise. Following the acute bout of RE, participants returned to the laboratory for recovery measures with measurements taken immediately post (Rec1) and 10 minutes following the acute bout of RE (Rec2).

*Statistical Analysis*

An a priori power calculation determined that 11 participants were needed based on a Cohen's d of 1.3, and power at 80%[7]. All results are presented as a mean ± standard deviation with statistical significance set *a priori* at $p \leq 0.05$. Prior to running any analyses all data were evaluated for normality using Shapiro-Wilk tests and all data met requirements for normality. A 2 x 4 Repeated Measures Analysis of Variance (ANOVA) was used to examine the differences across condition (caffeine, placebo) on the repeated factor of time (Rest1, Rest2, Rec1, Rec2) for measures of PWR (ASBP, ADBP, APP, AP, AIx, AIx@75, Tr, SPTI, DPTI, SEVR, and ΔEw). If Mauchly's test indicated that the assumption of sphericity had been violated, a Huynh-Feldt correction was used. If the ANOVA indicated a significant interaction, pairwise comparisons were used for post-hoc analysis. Adjustments for multiple comparisons were made with Bonferroni correction. Partial eta squared ($\eta^2$) was used as a measurement of effect size. A small effect size is 0.20, medium is 0.50, and large is 0.80.[28] Confidence intervals (95% CI) are provided for pairwise comparisons where appropriate. Data analysis was completed using IBM SPSS Version 26 (Armonk, NY, USA).

## Results

There was no significant difference ($p>0.05$) in total RE volume between caffeine and placebo conditions for squat (caffeine: 2800 ± 598 kg; placebo: 2799 ± 662kg) and bench press exercise (caffeine: 1130 ± 379 kg; placebo: 1109 ± 344 kg). Measures of PWR are presented in Tables 2 and 3. There were no significant ($p > 0.05$) condition by time interactions for any PWR measures. There were no significant ($p > 0.05$) main effects of time for ASBP ($\eta^2p = 0.13$), ADBP ($\eta^2p = 0.10$), Tr ($\eta^2p = 0.17$), or SEVR ($\eta^2p = 0.30$).There were significant ($p < 0.01$) main effects of time for APP, AP, AIx, AIx@75, SPTI, DPTI, and ΔEw. There was a significant ($F_{[3, 30]} = 15.03$, $p < 0.001$, $\eta^2p = 0.60$) increase in APP during Rec1 and Rec2 compared to both Rest1 and Rest2 in both conditions. Additionally, changes in AP, AIx, and AIx@75 (AP: $F_{[1, 12]} = 6.67$, $p = 0.02$, $\eta^2p = 0.40$; AIx: $F_{[1, 24]} = 47.31$, $p < 0.001$, $\eta^2p = 0.83$; AIx@75: $F_{[1, 30]} = 80.61$, $p < 0.001$) were similar between conditions, such that Rec1 and Rec2 were significantly greater than Rest1 and Rest2. SPTI and DPTI were significantly (SPTI: $F_{[2, 22]} = 26.39$, $p < 0.001$, $\eta^2p = 0.73$; DPTI: $F_{[1, 30]} = 37.11$, $p < 0.001$, $\eta^2p = 0.79$) different at Rec1 compared to Rest1 and Rest2 in both conditions. Specifically, SPTI was significantly greater while DPTI was significantly reduced during Rec1 and Rec2 when compared to Rest1 and Rest2. Lastly, ΔEw was significantly ($F_{[2, 18]} = 13.23$, $p < 0.001$, $\eta^2p = 0.59$) greater during Rec1 and Rec2 compared to Rest1 and Rest2 in both conditions.

**Table 2.** Aortic Pressure and Augmentation at Rest, Post-Ingestion, Immediately Following Acute Resistance Exercise, and Following 10 Minutes of Recovery in Resistance-Trained Women (N = 11)

| | | ASBP (mmHg) | ADBP (mmHg) | APP (mmHg) | AP (mmHg) | AIx (%) | AIx@75 (%) |
|---|---|---|---|---|---|---|---|
| Caffeine | Rest1 | 98 ± 6 | 67 ± 6 | 31 ± 4 | 3 ± 3 | 9.6 ± 10.2 | 1.3 ± 11.5 |
| | Rest2 | 102 ± 7 | 72 ± 6 | 31 ± 3 | 2 ± 3 | 7.2 ± 10.0 | -0.7 ± 11.2 |
| | Rec1 | 107 ± 9 | 68 ± 6 | 39 ± 6*§ | 11 ± 8*§ | 27.8 ± 16.2*§ | 28.9 ± 19.8*§ |
| | Rec2 | 104 ± 9 | 67 ± 5 | 37 ± 7*§ | 8 ± 5*§ | 22.2 ± 12.2*§ | 23.6 ± 17.0*^§ |
| Placebo | Rest1 | 98 ± 5 | 67 ± 4 | 31 ± 5 | 2 ± 3 | 6.8 ± 8.5 | 1.7 ± 9.1 |
| | Rest2 | 99 ± 7 | 68 ± 6 | 30 ± 5 | 1 ± 4 | 2.3 ± 11.6 | -5.7 ± 13.9 |
| | Rec1 | 104 ± 7 | 68 ± 5 | 36 ± 3*§ | 10 ± 3*§ | 26.5 ± 8.9*§ | 32.3 ± 10.8*§ |
| | Rec2 | 121 ± 7 | 70 ± 13 | 36 ± 7*§ | 14 ± 27*§ | 19.1 ± 9.1*§ | 22.6 ± 12.6*^§ |

*Note.* APP, aortic pulse pressure; ADBP, aortic systolic blood pressure; AIx, augmentation index; AIx@75, augmentation index normalized at 75bpm; AP, augmentation pressure; ASBP, aortic systolic blood pressure; Rec1, immediately following acute RE; Rec2, 10 minutes following Rec1; Rest1, resting assessment; Rest2, 50 minutes following ingestion. Data are presented as mean ± SD. *Significantly different from Rest1 ($p < 0.01$); ^Significantly different from Rec1 ($p < 0.01$) §Significantly different from Rest2 ($p < 0.01$).

**Table 3.** Ventricular Workload at Rest, Post-Ingestion, Immediately Following Acute Resistance Exercise, and Following 10 Minutes of Recovery in Resistance-Trained Women (N = 11)

| | | Tr (ms) | SPTI (mmHg*ms) | DPTI (mmHg*ms) | SEVR (%) | ΔEw (dyn/cm²) |
|---|---|---|---|---|---|---|
| Caffeine | Rest1 | 146.1 ± 5.7 | 1848.5 ± 267.0 | 2868.0 ± 281.3 | 160.0 ± 33.5 | 775.6 ± 690.1 |
| | Rest2 | 149.0 ± 4.3 | 1994.8 ± 366.7 | 3011.7 ± 338.5 | 158.5 ± 39.7 | 598.7 ± 699.5 |
| | Rec1 | 142.5 ± 4.5 | 2766.6 ± 579.6*§ | 2392.2 ± 301.6*§ | 92.9 ± 33.9 | 2489.7 ± 1621.8*§ |
| | Rec2 | 144.4 ± 4.0 | 2669.2 ± 536.6*§ | 2310.4 ± 381.5*§ | 92.8 ± 35.2 | 1784.8 ± 1150.4*§ |
| Placebo | Rest1 | 144.7 ± 5.6 | 1952.0 ± 313.1 | 2786.5 ± 207.0 | 147.8 ± 34.2 | 610.3 ± 666.8 |
| | Rest2 | 148.3 ± 8.0 | 1879.6 ± 413.2 | 2934.7 ± 249.3 | 167.2 ± 48.8 | 347.3 ± 654.8 |
| | Rec1 | 142.3 ± 2.2 | 2772.2 ± 490.2*§ | 2306.7 ± 336.8*§ | 93.5 ± 37.1 | 2404.3 ± 726.5*§ |
| | Rec2 | 153.3 ± 24.0 | 2386.4 ± 905.9*§ | 2267.8 ± 423.0*§ | 90.8 ± 33.2 | 2303.4 ± 2743.2*§ |

*Note.* DPTI, diastolic pressure time index; ΔEw, left ventricular wasted energy; SEVR, subendocardial viability ratio; SPTI, systolic pressure time index; Rec1, immediately following acute resistance exercise; Rec2, 10 minutes following Rec1; Rest1, resting assessment; Rest2, 50 minutes following ingestion; Tr, time of the reflected wave. Data are presented as mean ± SD. *Significantly different from Rest1 ($p < 0.01$); §Significantly different from Rest2 ($p < 0.01$).

## Discussion

This study aimed to investigate the effects of caffeine, alone and in conjunction with an acute bout of RE on measures of PWR in resistance-trained women. Results concluded that there were no significant differences for measures of PWR, between caffeine and placebo conditions. However, in both conditions, measures of PWR were altered following the acute bout of RE with APP, AP, AIx, AIx@75, and SPTI significantly elevated and DPTI significantly reduced up to 10 minutes. Based on these data, 4mg/kg of caffeine in conjunction with an acute bout of RE had no additional effects on measures of PWR in resistance-trained women.

Fifty minutes following ingestion of 4mg/kg of caffeine, the current study demonstrated no change in any measure of PWR at rest, which does not match the hypothesis. Previous literature reported changes in measures of PWR, such as AP, AIx, and Tr for upwards of 3 hours following

caffeine ingestion alone.[10,15,19] For instance, Karatzis et al.[15] administered 80 mg of caffeine in young, healthy men and women and showed a significant increase in AP and AIx at 90- and 120-minutes following caffeine ingestion, and a decrease in Tr. Karatzis et al[15] noted that the increase in AIx could be impacted by the decrease in heart rate, since AIx and heart rate tend to have an inverse relationship.[27] However, the present study controlled for heart rate, by reporting AIx@75, and still found no change between caffeine and placebo conditions. Similar results to Karatzis et al[15] were reported with higher absolute dosages of caffeine, 250 mg,[19] and 300 mg of caffeine,[29] respectively, and both reported increased AP and AIx. Participants in the present study consumed a relative dose of 4mg/kg of caffeine, averaging to an absolute dose of 269mg per person, which is similar to the literature. Though these studies demonstrate the unfavorable effects caffeine of varying doses has on measures of PWR, the present study suggests that this may not always be the case.

It can also be postulated that other factors such as body positioning and individual differences could have played a role in the lack of cardiovascular responses at rest regarding measures of PWR. Participants from the present study remained in an upright seated position following caffeine ingestion for 50 minutes while watching a nature documentary, then returned to the supine position for 10 minutes for post-ingestion measurements at rest. Alternatively, Karatzsis et al[15] asked participants to immediately return to the supine position following caffeine ingestion for a total of 120 minutes and reported AP and AIx significantly increased after 90 minutes. Similarly, participants in a study by Vlachopoulus et al[19] remained supine and demonstrated a significant increase in AP and AIx after 30 minutes and did not return to baseline levels after 180 minutes. Therefore, it is possible that body positioning can influence this physiological response, such that an extended duration in the supine position following caffeine ingestion (30 - 120 minutes), rather than the seated position, could result in alterations in measures of PWR.[15,19] Though, it may be uncommon for individuals to consume caffeine and immediately lie down for an extended duration, especially when consuming to use in conjunction with exercise, making it difficult to compare these supine results to a real-world scenario. However, this is conjecture, and further research should be considered.

Following an acute bout of RE, the present study found no difference in measures of PWR across the caffeine and placebo conditions, thus negating the original hypothesis. However, data from the present study demonstrated that a full-body acute bout of RE using free weights in resistance-trained women results in significant increases in AP, AIx, and AIx@75 for up to 10 minutes. Although this statistically demonstrated that this was not influenced by caffeine ingestion, these results align with previous literature on responses to an acute bout of RE alone. Similarly, Kingsley et al[20] and Tai et al[1] reported similar results in resistance-trained men and women, with Tai et al[1] attributing this increase in AIx to the decrease in Tr, therefore a faster return time of the reflected wave leading to an increase in AP and thus altering AIx. However, in the current study there was no change in Tr at any time point for either condition. Therefore, it could be suggested that the timing of the reflected wave influenced AIx. This could, in turn, result in decreased compliance of the aorta and thus an increase in arterial reservoir pressure[1].

Further, there were significant alterations in left ventricular function following an acute bout of RE in both conditions, which supported the hypothesis. Changes in these measures within the

literature are limited. However, Tai et al[1] reported an increase in SPTI, a decrease in DPTI, and an increase in ΔEw following an acute bout of RE. Therefore, it could be assumed there was also an overall decrease in SEVR for at least 10 minutes during recovery. In the current study, there was also a significant increase in SPTI, decrease in DPTI, and increase in ΔEw. Although SEVR decreased following acute RE in the present study, demonstrating reductions in coronary perfusion, it was not statistically significant. Therefore, the present study demonstrated that following an acute bout of RE, with caffeine or placebo, results in greater time spent during systole and decreased time during diastole. The increased AP resulted in greater ΔEw, such that the added pressure from the reflected wave resulted in greater left ventricular wasted energy. Again, further research is required to understand how caffeine can further influence these results in resistance-trained women.

Contrary to the hypothesis, the current study found no change in aortic BP or APP following the acute bout of RE in either condition, such that ASBP, ADBP, and APP did not increase as originally hypothesized. This result is intriguing such that even in the absence of acute RE, caffeine consumption alone has been reported to increase aortic BP, even with variations in dosage and types of caffeine consumption (i.e., caffeine supplement powder, caffeine gum, or coffee/espresso drinks).[15,17,19,29] These previous studies, with caffeine consumption ranging from 80 to 300 mg, reported significantly greater aortic BP with caffeine compared to placebo conditions[15,19] at rest. However, despite a similar caffeine dosage (269 mg on average) combined with acute RE in the present study, it remains unclear why comparable increases in aortic BP were not observed. Previous findings on aortic BP response following acute RE are also mixed, as some reported no change in either ASBP or ADBP[1,20] following acute RE, while others reported a significant increase.[2,6] The contrast in findings could be due to variations in acute RE protocols, such that Kingsley et al[17] and Tai et al[1] both utilized similar protocols of full-body acute RE at 75% 1RM, whereas DeVan et al[6] performed 8 to 12 repetitions of full-body acute RE at 50% 1RM following by one set of repetitions to failure. Therefore, alterations in aortic BP likely resulted from greater acute RE intensities. Future studies should utilize previous protocols of similar intensity, number of repetitions, and exercise selection to further clarify the effect of acute RE alone and in conjunction with caffeine on aortic BP and APP.

There are a few limitations to the present study. Although this study controlled for menstrual cycle, the results could be influenced by hormone differences in women over the lifecycle. Participants' caffeine intake was not controlled, only quantified, which could affect outcomes based on habitual use. In addition, unassessed sleep quality and exercise protocol may may have influenced results. Lastly, while the study met power requirements based on Cohen's *d*, the modest sample size may limit generalizability.

In conclusion, both caffeine and placebo conditions resulted in similar effects on measures of PWR at rest, immediately and 10 minutes following an acute bout of RE in resistance-trained women. Although caffeine did not further alter measures of PWR following acute RE, acute RE alone did which is consistent with the literature. Further, measures of aortic BP did not change at any time point. Additional research is warranted given the relationship of these variables to cardiovascular disease and cardiovascular disease-related events, such as arrhythmia. Lastly,

studies should continue to investigate the effects of caffeine on measures of PWR as utilizing caffeine as an ergogenic aid continues to rise in resistance-trained women.

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Corresponding author: Sarah G. Kearney; kearnesa@gvsu.edu