International Journal of EXERCISE SCIENCE

Original Research

The Effects of Machine-Weight and Free-Weight Resistance Exercise on Hemodynamics and Vascular Function

ERICA M. MARSHALL†1, JASON C. PARKS†1, YU LUN TAI‡1,2, and J. DEREK KINGSLEY‡1

[1]Cardiovascular Dynamics Laboratory, School of Health Sciences, Kent State University, Kent, OH, USA; [2]Department of Health & Human Performance, University of Texas-Rio Grande Valley, Brownsville, TX, USA

†Denotes graduate student author, ‡Denotes professional author

ABSTRACT

International Journal of Exercise Science 13(2): 526-538, 2020. The purpose of this study was to examine hemodynamic and vascular responses between machine-weight and free-weight exercise. Resistance-trained individuals were assigned to a machine-weight (n = 13) or free-weight (n = 15) group. Groups completed two visits consisting of their assigned exercise condition and a control (CON). A 2 x 2 x 3 repeated measures ANOVA was used to test the effects of group across condition and time on the hemodynamic parameters [cardiac output (CO), heart rate (HR), total peripheral resistance (TPR), mean arterial pressure (MAP), and stroke volume (SV)]. A 2 x 2 x 2 repeated measures ANOVA was used to test the effects of group across condition and time on the hemodynamic variable, forearm vascular conductance (FVC), as well as on vascular measures [forearm blood flow (FBF), blood flow peak, and total reactive hyperemia (RH)]. Main effects were analyzed using pairwise comparisons. The results of the present study demonstrate that both machine-weight and free-weight exercise produce similar ($p > 0.05$) alterations in hemodynamics and vascular function. Specifically, during recovery both groups demonstrated significant ($p \leq 0.05$) increases in measures of hemodynamics such as CO, HR and FVC, as well as significant ($p \leq 0.05$) decreases in TPR, MAP, and SV. Measures of vascular function such as FBF, blood flow peak, and total RH were also significantly ($p \leq 0.05$) increased during recovery. Therefore, this study suggests that either machine weight or free-weight exercise may induce acute hemodynamic and vascular benefits, which may reduce the risk of cardiovascular disease (CVD) and CVD events.

KEY WORDS: Mean arterial pressure, total peripheral resistance, forearm blood flow, reactive hyperemia, endothelial function, strength exercise

INTRODUCTION

It is currently recommended that a resistance exercise (RE) regimen include both single-joint and multi-joint exercises in order to increase muscular strength (2). For this, individuals that are resistance training may choose to include machine-weight and free-weight exercise, respectively. However, though machine-weight and free-weight exercises are suggested to produce similar increases in muscular strength, they have different patterns of muscular recruitment, such that free-weight exercise generally recruits a greater amount of muscle mass

compared to machine-weight exercise (24). These greater amounts of muscle mass utilized during free-weight exercise may positively alter the hemodynamic response and measures of vascular function (25) to a greater degree compared to that of machine-weight exercise. This may be important for RE prescription such that it is not only beneficial to muscular fitness, but it may also confer positive changes in cardiovascular health. Literature suggests that positive alterations in measures of hemodynamics and vascular function may reduce the occurrence of cardiovascular disease (CVD) related events (e.g. hypertension, stroke, coronary and peripheral artery disease) (9, 12). Currently, no studies have compared the hemodynamic and vascular responses between these two RE modalities, which may be due to methodological differences in terms of exercise volume associated with each modality. Nevertheless, studies have reported that various combinations of machine-weight and free-weight exercise, and free-weight exercise alone, produce positive alterations in measures of hemodynamics and vascular function (3, 8, 26).

Positive alterations in measures of hemodynamics have been reported following an acute bout of RE consisting of both machine-weight and free-weight exercise (8, 10), as well free-weight exercise alone (11, 29). Specifically, in the work by Collier, et al. (8) an acute bout of full-body RE resulted in a significant increase in cardiac output (CO) and forearm vascular conductance (FVC), with a reduction in total peripheral resistance (TPR) and no change in mean arterial pressure (MAP). In another study, De Freitas, et al. (10) reported that MAP was reduced following an acute bout of full-body machine-weight and free-weight exercise. Tai, et al. (27) also reported an increase in CO and a reduction in TPR, as well as a significant reduction in MAP, following an acute bout of full-body, free-weight exercise. Additionally, Fahs, et al. (11) reported a reduction in TPR following an acute bout of upper-body RE using only free-weights. Collectively, these studies suggest that RE performed with both machine-weight and free-weight exercise, or free-weight exercise alone, seem to have a positive effect on measures of hemodynamics. However, no studies have directly compared the hemodynamic responses between machine-weight and free-weight exercise, which may be different.

These positive alterations in hemodynamics may produce favorable responses in the vasculature. As previously mentioned, studies that have reported positive alterations in hemodynamics have also demonstrated improvements in measures of vascular function including: increased forearm blood flow (FBF), blood flow peak, and total reactive hyperemia (RH) (8, 11, 27). Improvements in these measures may reduce the risk of vascular dysfunction, which generally refers to a decrease in the production, or availability of potent vasodilators, thereby accelerating the formation of atherosclerotic plaques that are largely responsible for the development of CVD-related events (9). To date, the specific effects of machine-weight and free-weight exercise on vascular function are unclear. This is important as the modalities may produce different responses in terms of vascular function.

Therefore, the purpose of the present study was to compare machine-weight and free-weight exercise on measures of hemodynamics and vascular function in young, healthy, resistance-trained individuals. We hypothesized that in regard to hemodynamics, there would be acute increases in CO and FVC, with reductions in TPR, stroke volume (SV), and MAP after free-

weight exercise, and that these responses would be greater than the machine-weight exercise. We also hypothesized that there would be significant increases in all measures of vascular function during recovery from machine-weight and free-weight exercise, but that these responses would be increased following free-weight compared to machine-weight exercise.

METHODS

Participants

Twenty-eight young (18-30 yrs of age), healthy individuals (16 men; 12 women) self-reported that they had been engaging in RE for at least 3 days per week for a minimum of 2 years. Exclusion criteria included being a smoker, obese, orthopedic issues, cancer, known cardiovascular, or metabolic disease, uncontrolled hypertension (resting brachial blood pressure (BP) > 140/90 mmHg), use of medications or supplements that are known to affect HR, BP, or vascular function as assessed via a medical questionnaire. This research was carried out fully in accordance to the ethical standards of the International Journal of Exercise Science (21). Informed consent was obtained from all individuals and the study was approved by the Institutional Review Board.

Protocol

During Visit 1, individuals were assessed for anthropometrics and body composition followed by muscular strength assessment of either machine-weight (Machine-Weight: n = 13, Men: 8, Women: 5) or free-weight exercise (Free-Weight: n = 15, Men: 8, Women: 7) based upon random group assignment. Height and weight were measured using a stadiometer and a beam balance platform scale, respectively (Detecto 448; Cardinal Scale Manufacturing, Web City, MO, USA). Body composition was measured by seven site skinfold measurement (Lange; Beta Technology, Santa Cruz, CA, USA). Generalized skinfold equations were used to determine body density appropriately for men (17) and women (18). The Brozek equation was used to calculate percent body fat (7).

Prior to the 1RM, individuals warmed-up on a cycle ergometer (Schwinn Air Dyne; Boulder, Colorado) for 5-min. For the machine-weight group, muscular strength was assessed by the 1-repetition maximum (RM) in the order of leg press, latissimus dorsi pulldown, leg extension, chest press, and seated leg curl. For the free-weight group, the 1RM was assessed in the order of squat, bench press, and deadlift. During the 1RM, individuals were given 5 attempts following a warm-up with 50% of their body-weight, based on recommendations from the National Strength and Conditioning Association (14). The highest resistance moved through a full range of motion between the two maximal strength testing days was used to determine the resistance load for the acute bout of exercise. Muscular strength was re-verified 72 hours later during Visit 2.

Approximately 72 hours following Visit 2, participants were assessed for hemodynamics and vascular function at rest and following an acute bout of machine-weight, or free-weight exercise, in addition to a control (CON) in a randomized order (Visits 3 and 4). These visits were separated by a minimum of 1 week and were completed at the same time of day (± 1 hour). All testing

during Visit 3 and 4 occurred between the hours of 6am-12pm in order to control for diurnal variation. Women were tested during the early to mid-follicular phase (Day 1-7) of their menstrual cycle determined by the start of their menses. Individuals were at least 3 hours postprandial and were instructed to avoid caffeine, alcohol, and strenuous exercise for at least 24 hours prior to testing. The temperature of the room was constant at approximately 22 ^{o}C. Upon arriving at the laboratory, individuals rested quietly in the supine position prior to the start of hemodynamic and vascular assessment.

Beat-to-beat BP was recorded during all measurements of blood flow via finger photoplethysmography (NexfinCC, BMEYE, Amsterdam, Netherlands). The pressure waveforms obtained were used to assess CO, HR, TPR, MAP, and SV. The Modelflow technique allows the pressure on the index finger to compute an aortic waveform to calculate SV (6, 29). The aortic waveform per beat provides measurement of left ventricular SV, and thus CO through the multiplication of SV and HR (6). Total peripheral resistance was then derived from MAP and CO.

Forearm blood flow, blood flow peak, and total RH were assessed using a mercury-in-silastic strain gauge plethysmography (EC-6; DE Hokanson Inc., Bellevue, WA, USA), which has been shown to be both reliable and valid (19). An explanation of this method is described by Higashi et al. (16). In brief, the arm was elevated above heart level while the individual was supine. Then, the circumference of the widest portion of the left forearm was quantified and the appropriate strain gauge was attached to the forearm and connected to the plethysmograph. Two blood pressure cuffs were placed on the left arm, one on the most proximal portion of the upper left arm over the brachial artery, and the other around the left wrist. The wrist cuff was inflated to 220 mmHg at 1-min prior to, and throughout all vascular measurements. The brachial cuff was then inflated to 50 mmHg for 7-sec and then deflated for 8-sec for a 15-sec cycle using a rapid cuff inflator (EC-20; DE Hokanson Inc.) in order to occlude venous flow. Six measurements were averaged from the plethysmograph in order to determine FBF both at rest and during recovery following the acute bout of exercise, or CON. Once baseline FBF was determined, the brachial cuff was rapidly inflated to induce circulatory occlusion at 220 mmHg for 5-min. One minute prior to the release of the brachial cuff, the wrist cuff was inflated to 220 mmHg. At the end of the 5-min, the brachial cuff was released to induce RH. Blood flow was measured for the next 3-min for a total of 13 measurements using a 15-second cycle consisting of a 7-sec inflation and 8-sec deflation. The highest blood flow reading was recorded as the blood flow peak. All 13 measurements during RH were graphed onto a curve, and the area under the curve was taken as a measure of total RH. Forearm vascular conductance (FVC) of the left arm was calculated by the division of mean FBF and MAP. All vascular data were analyzed using Noninvasive Vascular Program 3 Software Package (DE Hokanson Inc., Bellevue, WA).

During the acute exercise, both machine-weight and free-weight exercise groups completed 3 sets of 10 repetitions at 75% 1-repetition maximum (1RM) with 2-min of rest between sets and exercises. Within 3-min of completing the acute bout of RE individuals returned to the supine position to rest before collection of hemodynamics and vascular measures were repeated. The CON was time-matched to the acute bout of RE. All measurements during recovery were

collected in a similar manner to rest. At rest, measures of hemodynamics and vascular function were assessed at 15-min and 15 to 25-min, respectively. During the recovery, measurements were taken at 15-min (Rec1) and again at 25-min (Rec2) for measures of hemodynamics and from 15 to 25-min (Rec) for measures of vascular function.

Statistical Analysis

Characteristics between the groups were analyzed using independent samples t-tests. RE volume was calculated as resistance x sets x reps. A 2 x 2 x 3 repeated measures ANOVA was used to test the effects of group (machine-weight, free-weight) across condition (acute bout of exercise, CON) with the repeated factor of time (Rest, Rec1, Rec2) on hemodynamic parameters [CO, HR, TPR, MAP, SV]. A separate 2 x2 x 2 repeated measures ANOVA was used to test the effects of group across condition with the repeated factor of time (Rest, Rec) on the hemodynamic parameter FVC. A 2 x 2 x 2 repeated measures ANOVA was used to test the effects of group across condition with the repeated factor of time (Rest, Rec) on vascular measures [FBF, blood flow peak, and total RH]. Total RH was calculated using GraphPad Prism 5.0 (GraphPad, La Jolla, CA) using the trapezoidal rule. Significant interactions were analyzed using pairwise comparisons. Adjustment for multiple comparisons was made using a Bonferroni correction. Partial eta squared (η_p^2) was used to assess the effect size of each dependent variable. Significance was accepted *a priori* at $p \leq 0.05$. Values are presented as mean ± standard deviation (SD). All statistical analyses were completed using IBM SPSS version 23 (Amrok, NY, USA). Our sample size was based on pilot data in our laboratory that was collected under identical conditions using seven healthy, resistance-trained individuals. We determined an effect size of 1.3 for the dependent variable, FBF, which estimated a sample size of 11 individuals in each group. This was with an alpha of 0.05 and a power of 80%.

RESULTS

Group characteristics are presented in Table 1. The groups were similar ($p > 0.05$) for age, height, weight, BMI, percent fat, lean mass, and fat mass. However, as expected, the groups were significantly different ($p \leq 0.05$) for RE volume performed during the acute bout of exercise. There were no significant ($p > 0.05$) differences between the machine-weight and free-weight groups for any of the dependent variables at Rest or during recovery from the acute bout of exercise.

Table 1. Subject characteristics

	Machine-Weight (n = 13)	Free-Weight (n = 15)
Age (yr)	23 ± 2	22 ± 2
Height (m)	1.7 ± 0.1	1.7 ± 0.1
Weight (kg)	76.9 ± 16.0	76.1 ± 14.0
BMI (kg/m^2)	25.2 ± 2.4	26.1 ± 3.9
Percent Fat (%)	18.5 ± 6.7	19.0 ± 10.9
Lean Mass (kg)	67.1 ± 15.3	65.9 ± 10.7
Fat Mass (kg)	15.0 ± 6.2	14.8 ± 9.6
Workload (kg)	16741 ± 17000ψ	7476 ± 2033

Note: BMI – Body Mass Index. Data presented are mean ± SD. ψSignificantly different from free-weight group ($p \leq 0.05$).

Hemodynamics are presented in Table 2. There were no significant 3-way interactions for hemodynamics. There were significant condition-by-time interactions for CO ($F_{1,26} = 47.02$, $p < 0.001$, $\eta_p^2 = 0.64$), HR ($F_{1,26} = 190.24$, $p < .0001$, $\eta_p^2 = 0.88$), TPR ($F_{1,26} = 50.7$, $p < .0001$, $\eta_p^2 = 0.66$), and MAP ($F_{1,26} = 11.58$, $p < 0.001$, $\eta_p^2 = 0.31$). Cardiac output and HR were increased during Rec1 and Rec2 compared to Rest following the acute bout of machine-weight and free-weight exercise and the CON. Furthermore, HR decreased from Rec1 to Rec2 following the acute bout of machine-weight and free-weight exercise compared to the CON. Additionally, TPR decreased during Rec1 and Rec2 compared to Rest following the acute bout of machine-weight and free-weight exercise and compared to the CON. Mean arterial pressure decreased from Rest to Rec1 following CON. Following the acute bout of machine-weight and free-weight exercise, MAP was decreased during Rec1 and Rec2 compared to Rest and the CON.

There was a significant main effect of condition ($F_{1,26} = 8.9$, $p = 0.006$, $\eta_p^2 = 0.26$) and time ($F_{1,26} = 9,1$, $p < 0.001$, $\eta_p^2 = 0.26$) for SV. Stroke volume was decreased at Rest and during Rec1 following the acute bout of machine-weight and free-weight exercise and also when compared to CON. Additionally, following the acute bout of machine-weight and free-weight exercise, SV was increased from Rec1 to Rec2. Lastly, there was a significant condition-by-time interaction for FVC ($F_{1,26} = 52.85$, $p < 0.0001$, $\eta_p^2 = 0.67$) such that it differed from the CON and was increased during recovery from the acute bout of machine-weight and free-weight exercise compared to Rest.

Table 2. Hemodynamics at rest and during recovery from a control and machine-weight or free-weight exercise in young, healthy resistance-trained individuals (n = 28).

	CON			Acute Exercise		
	Rest	Rec1	Rec2	Rest	Rec1	Rec2
CO (L/min)						
Machine-Weight	6.8 ± 0.7	6.6 ± 0.9*	6.7 ± 0.8*	6.8 ± 0.9	7.8 ± 1*†	8.1 ± 0.9*†
Free-Weight	6.6 ± 0.8	6.3 ± 0.6*	6.3 ± 0.7*	6.6 ± 0.6	7.8 ± 0.7*†	7.9 ± 0.6*†
HR, bpm						
Machine-Weight	62 ± 10	60 ± 11*	61 ± 10*	65 ± 6	89 ± 9*§†	85 ± 10*†
Free-Weight	61 ± 10	57 ± 9*	57 ± 9*	62 ± 10	89 ± 15*§†	85 ± 14*†
TPR, mmHg/ml/min						
Machine-Weight	0.7 ± 0.1	0.8 ± 0.1*	0.8 ± 0.1*	0.7 ± 0.	0.6 ± 0.1*†	0.6± 0.1*†
Free-Weight	0.8 ± 0.1	0.8 ± 0.1*	0.8 ± 0.1*	0.8 ± 0.1†	0.6 ± 0.1*†	0.6 ± 0.1*†
MAP, mmHg						
Machine-Weight	81 ± 6	87 ± 8*	85 ± 6	82 ± 6	80 ± 6*†	78 ± 5*†
Free-Weight	82 ± 5	84 ± 6*	83 ± 7	82 ± 5	77 ± 6*†	78 ± 5*†
SV, ml/beat						
Machine-Weight	86.2 ± 4.7	85.3 ± 5.8	85.6 ± 4.3	85.1 ± 4.9†	82.3 ± 3.2*§†	84.9 ± 3.3
Free-Weight	87.5 ± 7.7	86.3 ± 7.4	86.7 ± 7.3	85.3 ± 6.2†	82.5 ± 4.2*§†	83.9 ± 4.1

CON – Control; CO – Cardiac Output; HR – Heart Rate; TPR – Total Peripheral Resistance; MAP – Mean Arterial Pressure; SV – Stroke Volume. Data are mean ± standard deviation. *Significantly different from Rest $p \leq 0.05$; †Significantly different from Control $p \leq 0.05$; §Significantly different from Rec2 $p \leq 0.05$.

Blood flow data are presented in Figure 1. There were significant condition-by-time interactions for FBF ($F_{1,26} = 45.94$, $p < .0001$, $\eta_p^2 = 0.64$), blood flow peak ($F_{1,26} = 15.17$, $p = .001$, $\eta_p^2 = 0.37$), and total RH ($F_{1,26} = 44.37$, $p < .0001$, $\eta_p^2 = 0.63$) such that they differed from the CON, and were increased during recovery from the acute bout of machine-weight and free-weight exercise compared to Rest.

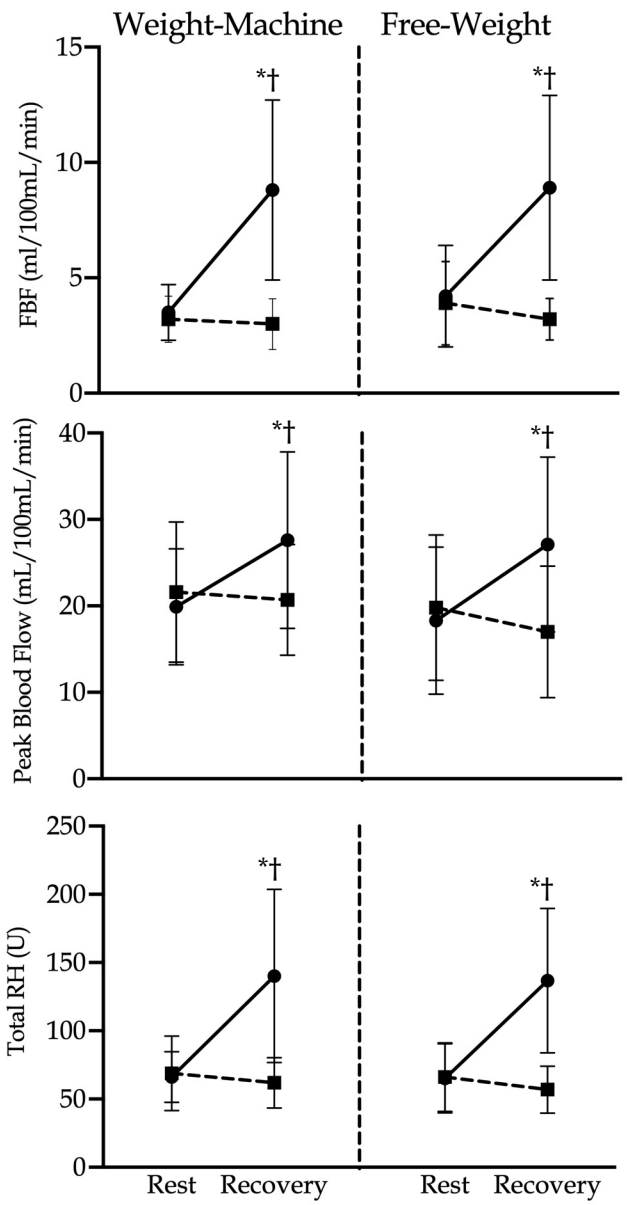

Figure 1. Changes in A) forearm blood flow (FBF), B) blood flow peak, and C) total reactive hyperemia (RH) at rest and during recovery from the control (CON) and acute resistance exercise (RE) consisting of machine-weight or free-weight acute RE in young, healthy, resistance-trained individuals (n = 28). Solid lines represent the acute exercise condition. Dashed lines represent the CON. Data are mean ± standard deviation. *$p \leq 0.05$, significantly different from Rest; †$p \leq 0.05$, significantly different from CON.

DISCUSSION

This study investigated the effects of machine-weight and free-weight exercise on hemodynamics and vascular function in young, healthy resistance-trained individuals. The primary findings of the present study are that machine-weight and free-weight exercise produce similar hemodynamic and vascular responses. Specifically, both exercise modalities demonstrated similar increases in measures of hemodynamics such as CO, HR and FVC, as well

as similar decreases in TPR, MAP, and SV. Measures of vascular function such as FBF, blood flow peak, and total RH also increased similarly between exercise modalities, and across time. Therefore, contrary to our hypothesis, our findings suggest that both machine-weight and free-weight exercise produce similar, beneficial alterations in measures of hemodynamics and vascular function.

The results of this study suggest that both machine-weight and free-weight exercise produce favorable alterations in measures of hemodynamics. These results are similar to other studies that have utilized an acute bout of machine-weight exercise in combination with free-weight exercise (8, 10). Collier, et al. (8) reported an increase in HR and CO, with a concomitant reduction in TPR, following an acute bout of exercise performed on the bench press, bent over row, leg extension, leg curl, shoulder press, biceps curl, and close grip bench for 3 sets of 10 repetitions at the 10RM. However, these authors did not report a significant reduction in MAP, as was observed in the present study. These differences may be due to the fact that hemodynamic measures were assessed at 40-min into recovery compared to 15-min. On the other hand, De Freitas, et al. (10) did report a reduction in MAP at 10-min following an acute bout of exercise consisting of a combination of machine-weight and free-weight exercise on the leg press, leg extension, leg curl, bench press, T-bar row, and biceps curl. Each RE was performed for 3 sets each, for a total of 18 sets at 65% of the 1RM in young, recreationally active men. But, this reduction in MAP appeared to be larger compared to the present study. The present study demonstrated a 2mmHg reduction in MAP at 15-min into recovery while De Freitas, et al. (10) reported a reduction of 10mmHg at 10-min during recovery. The greater reductions in MAP in the study by De Freitas, et al. (10) compared to the present study may be due to differences in exercise volume, where individuals were asked to perform 3 sets of 6 different exercises. In the present study, individuals were asked to perform 3 sets of 10 repetitions for 5 different exercises, or 3 sets of 10 repetitions for 3 different exercises for the machine-weight and free-weight exercise, respectively. It is suggested that the volume of RE performed, typically results in a larger reduction in MAP during recovery (22). It is likely that the present study utilized a lower exercise volume, which may account for these differences. But this is speculation as De Freitas, et al. (10) did not report the number of repetitions performed.

Further, the hemodynamic responses following free-weight exercise in our study are similar to others who have also utilized a protocol consisting exclusively of free-weights (4, 11, 27). Specifically, Fahs, et al. (11) also reported an increase in HR and a reduction in TPR, but no change in MAP following free-weight exercise consisting of 4 sets of 5 repetitions at 80% 1RM on the bench press, followed by 4 sets of 10 repetitions at 75% 1RM on the biceps curl in young, healthy men at 15-min during recovery. Additionally, Tai, et al. (27) also reported that 3 sets of 10 repetitions at 75% on the squat, bench press, and deadlift significantly increased HR, CO, and FVC with concomitant decreases in MAP, TPR, and SV at 15-min during recovery in resistance-trained individuals. However, while researchers in the present study, and Tai, et al. (27), observed a reduction in MAP, Fahs, et al. (11) reported no change in MAP. Their observations of no change may be due to differences in terms of exercise modality, which only included upper-body exercise, compared to the present study and Tai, et al. (27), that utilized full-body exercise. It has been suggested that a larger, active muscle mass results in a greater reduction in

MAP following RE, which may explain these differences (25). Nevertheless, based on these data it is clear that an acute bout of RE has a profound impact on hemodynamics.

These studies demonstrating positive alterations in measures of hemodynamics have also reported improvements in measures of vascular function, which are similar to the present study. Specifically, the present study observed increases in vascular measures such as FBF, blood flow peak, and total RH, which is supported by previous work (8, 11, 28). In the previously mentioned study by Collier, et al. (8), they reported a significant increase in blood flow peak and total RH at 60-min following an acute bout of machine-weight and free-weight exercise. However, it appears that vascular measures were different at Rest, and in response to the acute exercise bout, compared to the present study. Collier, et al. (8) reported an increase in blood flow peak of 5 mL/100/min at 60min into recovery. The present study demonstrated an 8mL/100ml/min increase, and a 9mL/100mL/min at 15-min during recovery in the machine-weight group, and in the free-weight group, respectively. Additionally, Collier, et al. (8) also reported an increase in total RH of 40 units at 60-min into recovery. In the present study, total RH was increased 74 units at 25-min during recovery following the machine-weight exercise, and 72 units following free-weight exercise. Differences in terms of magnitude of vascular measures following the acute RE modalities may be due to differences in time of assessment. The present study measured blood flow at 25-min into recovery, while Collier, et al. (8) measured it at 60-min. This difference in the time of the measurements likely explains the reduced response in total RH in the work by Collier, et al. (8) compared to the present study as it is likely that vascular measures such as blood flow peak and RH had already begun to recover in the study by Collier, et al. (8). Additionally, differences in vascular measures following the acute RE modalities may be due to both the order and nature of the exercises utilized in the studies. In the present study, individuals performed the leg curl, or the deadlift, last for the machine-weight and free-weight exercise, respectively. Both exercises incorporate substantial grip force. In the study by Collier, et al. (8) the abdominal crunch exercise was performed last. Since grip intensity is suggested to have a positive relationship to blood flow (5), this may explain these differences. Nevertheless, the present study, and other studies, demonstrate that an acute bout of resistance exercise mediates an increase in vascular function. This may have implications for those that are resistance training, such that acute machine-weight or free-weight exercise may induce acute increases in vascular measures, or blood flow.

Further, Fahs, et al. (11) also reported increases in FBF, blood flow peak, and total RH following an acute bout of free-weight exercise. Additionally, researchers also reported an increase in total RH of 67 units compared to the 74 units following machine-weight exercise and 72 units following free-weight exercise. These small differences may be due to the fact that Fahs, et al. (11) utilized young, healthy men whereas the present study recruited resistance-trained individuals. It is possible that training status may have mediated these differences in total RH at Rest, as previous research has suggested positive vascular adaptations in response to a RE stimulus (15). On the other hand, Tai, et al. (27) reported vascular measures that appear to be larger compared to the present study with the exception of total RH. Specifically, Tai, et al. (27) reported an increase in FBF of 10mL/100mL/min at 15 to 25-min during recovery, which was double that of the present study. In the present study, which utilized a similar RE protocol and

time of measurement, there was only a 5mL/100mL/min increase during recovery in the free-weight group. The machine weight group also increased by 5mL/100mL/min. Additionally, Tai, et al. (27) reported an increase in blood flow peak of 15mL/100mL/min at 15 to 25-min during recovery, whereas the present study reported an increase of 9mL/100mL/min at 15 to 25-min in the free-weight group. Similarly, the machine-weight group demonstrated an increase in peak blood of 8mL/100mL/min at 15 to 25-min during recovery. It is possible that differences between this study and the present study may be explained by exercise volume, but this was not reported by Tai, et al. (27).

Collectively, the present study suggests that both machine-weight and free-weight exercise results in similar hemodynamic and vascular responses. Specifically, both modalities in this study mediated similar increases in metabolic demand, thus similar alterations in HR, CO, TPR, and MAP as well as vascular measures. Comparable rates of metabolic demand may be due to similar amounts of active muscle mass being recruited (13). It is suggested that RE utilizing a large amount of active muscle mass results in a greater reduction in MAP compared to a smaller active muscle mass (22). Therefore, similar reductions in MAP between machine-weight and free-weight groups in this study may be attributed to the fact that both exercise modalities were full-body in nature, which may have recruited similar amounts of active muscle mass. For example, during the machine weight exercise, since the exercise is unidirectional, there may have been an increase in active muscle mass via the primary muscle. It has been suggested that during machine-weight exercise, the primary muscle may be more directly loaded, which would augment active muscle mass recruitment (23). On the other hand, during free-weight exercise, more synergist muscles may be actively recruited, lending to the increase in active muscle mass.

Lastly, although the free-weight exercise likely recruited several synergist muscles, the workload (i.e. volume) performed in the machine-weight group was substantially greater. It is likely that this large increase in volume may have contributed to the similar alterations in hemodynamics and vascular measures to that of the free-weight exercise. It has been previously reported that hemodynamics are affected by the number of sets performed (i.e. exercise volume) (22). Therefore, machine-weight and free-weight exercise may have similarly altered hemodynamics and vascular measures via volume and intensity dependent mechanisms, respectively.

This study is not without limitations. Specifically, we did not match exercise volume. Future studies may wish to utilize a matched-load design to limit the influence of duration, intensity, and volume of exercise on hemodynamic and vascular responses. Additionally, RE volume was not run as a covariate due to a violation of the assumption of independence between weight-machine and free-weight RE groups (20). Additionally, though women were measured in the follicular phase, estrogen begins to rise in the later portion of this phase (Day 9). It has been suggested that the menstrual cycle phase plays a role in vascular function with greater estrogen levels in the luteal phase contributing to vasodilation (1). Therefore, it is possible women measured in later phases of the follicular cycle may have already began to experience increases in estrogen, which could have influenced the results of this study. Due to the present sample size, sex-specific differences were not examined.

In conclusion, both machine-weight and free-weight RE are associated with similar, improved hemodynamic and vascular function measures. Future studies should investigate similar studies with a crossover design with matched load to determine if these outcomes remain similar. Additionally, studies should continue to investigate other RE modalities, training variables (e.g. sets, repetitions, rest time, etc.), and their effects on hemodynamics and vascular function.

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