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# THE EFFECT OF EXERCISE INTENSITY ON POSTRESISTANCE EXERCISE HYPOTENSION IN TRAINED MEN

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## ABSTRACT

Duncan, MJ, Birch, SL, and Oxford, SW. The effect of exercise intensity on postresistance exercise hypotension in trained men. *J Strength Cond Res* 28(6): 1706–1713, 2014—The occurrence of postresistance exercise hypotension (PEH) after resistance exercise remains unknown. This study examined blood pressure and heart rate (HR) responses to an acute bout of low- and high-intensity resistance exercise, matched for total work, in trained males. Sixteen resistance-trained males ( $23.1 \pm 5.9$  years) performed an acute bout of low- (40% of 1 repetition maximum [1RM]) and high-intensity resistance exercise (80% 1RM), matched for total work, separated by 7 days and performed in a counterbalanced order. Systolic blood pressure (SBP) and diastolic blood pressure (DBP), mean arterial pressure (MAP), and HR were assessed before exercise, after completion of each exercise resistance exercise (3 sets of back squat, bench press, and deadlift) and every 10 minutes after resistance exercise for a period of 60 minutes. Results indicated a significant intensity  $\times$  time interaction for SBP ( $p = 0.034$ , partial  $\eta^2 = 0.122$ ) and MAP ( $p = 0.047$ , partial  $\eta^2 = 0.116$ ) whereby SBP and MAP at 50-minute recovery and 60-minute recovery were significantly lower after high-intensity exercise ( $p = 0.01$  for SBP and  $p = 0.05$  for MAP in both cases) compared with low-intensity exercise. There were no significant main effects or interactions in regard to DBP (all  $p > 0.05$ ). Heart rate data indicated a significant main effect for time ( $F_{(9, 135)} = 2.479$ ,  $p = 0.0001$ , partial  $\eta^2 = 0.344$ ). Post hoc multiple comparisons indicated that HR was significantly higher after squat, bench press, and deadlift exercise compared with resting HR and HR at 40-, 50-, and 60-minute recovery (all  $p = 0.03$ ). The present findings suggest that an acute bout of high intensity, but not low intensity, resis-

tance exercise using compound movements can promote PEH in trained men.

**KEY WORDS** strength, systolic blood pressure, diastolic blood pressure, heart rate

## INTRODUCTION

A considerable amount of evidence has shown that regular exercise lowers both systolic blood pressure (SBP) and diastolic blood pressure (DBP) (7,15,17,22). The exact mechanisms by which exercise leads to a reduction in blood pressure (BP) is unclear (14,28) although it has been suggested that this may be a consequence of the summation of the BP reductions that occur during the immediate recovery periods after exercise referred to as postexercise hypotension (PEH) (14). Studies have consistently described PEH after bouts of aerobic exercise reporting a magnitude of BP reductions from 5 to 7 mm Hg with a time course of up to 22 hours after cessation of aerobic exercise and under ambulatory conditions (7,13,16,19). Although the PEH responses to aerobic exercise have been well described, the evidence for a BP lowering effect as a consequence of resistance exercise is scarce (8), this is despite authors suggesting this is a key research need (14,28). Moreover, there is a greater emphasis on prescribing resistance exercise or a combination of resistance and aerobic exercise in populations with both normal BP and hypertension (22), thus it is important to understand the acute effects of resistance exercise on BP. The literature to date does not adequately describe how the acute resistance exercise program variables (i.e., intensity) or participant characteristics (i.e., training status, resting BP classification) influence PEH. Thus, further studies are required so that more effective prescription of resistance exercise to elicit PEH can be targeted.

Both experimental and meta-analytical data support that resistance exercise chronically reduces SBP and DBP in normotensive and hypertensive populations (6–8,16). The most recent of these meta-analysis confirmed that both dynamic and isometric resistance exercise have a beneficial effect on BP in individuals with optimal BP or prehypertension (8). Studies have also suggested that PEH occurs after

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**TABLE 1.** Participant baseline characteristics.\*

	Mean	SD
Age (y)	23.1	5.9
Height (cm)	1.78	4.7
Mass (kg)	76.6	8.15
Body mass index (kg·m <sup>-2</sup> )	24.2	4.3
Resting SBP (mm Hg)	122.3	3.3
Resting DBP (mm Hg)	75.9	6.4
MAP (mm Hg)	91.3	4.1
Resting HR (b·min <sup>-1</sup> )	72.6	6.8
Back squat 1RM (kg)	120.3	26.1
Bench press 1RM (kg)	84.4	22.1
Deadlift 1RM (kg)	118.5	44.2

\*SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; MAP = mean arterial pressure; RM = repetition maximum.

resistance exercise but that the magnitude and duration of the BP lowering effects are less than those reported with aerobic exercise (11,12,26,28). During a single bout of resistance exercise, both BP and heart rate (HR) increase (20). On cessation of resistance exercise, authors have reported that BP remains elevated (4,12), is lower (2,9,11), or is equal to pre-exercise values (21,29). The equivocal nature of this literature may be because of a number of reasons including the exercise protocol used, the population assessed (e.g., trained vs. untrained, mixed gender groups), and a failure

to fully account for the intensity of exercise undertaken separate to the physical work completed. This latter point is important as exercise intensity has been cited as one of the mechanisms for the disparity in research findings (12,28), and future studies need to account for the total work completed by participants when comparing the effect of exercise intensity on PEH.

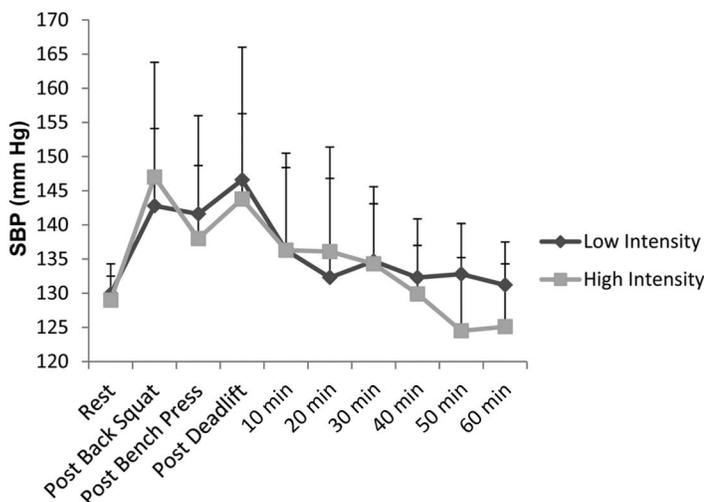
Therefore, the aim of this study was to examine the effect of exercise intensity on postexercise BP responses to resistance exercise, controlling for total work done, in trained males. We hypothesized that both low- and high-intensity resistance exercise would elicit PEH effects, congruent with the previous work on this area using a similar intensity, matched for work protocol (28) but that in this study the magnitude of hypotensive effects would be greater in the high-intensity condition.

**METHODS**

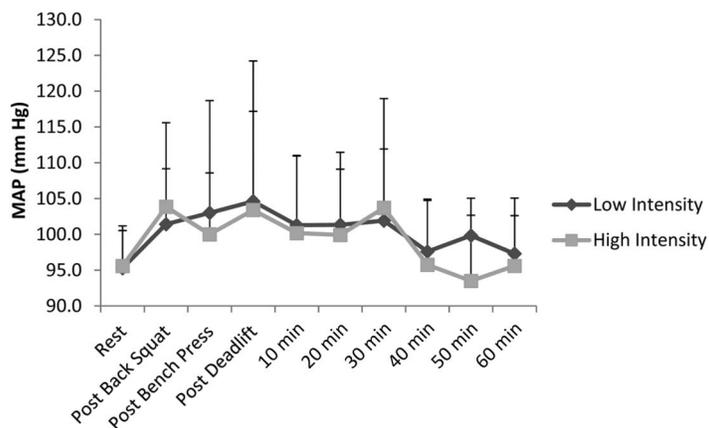
**Experimental Approach to the Problem**

To achieve the aims of this investigation, data assessment took place on 3 separate occasions separated by at least 7 days and in 2 separate phases. In the first phase, participants attended the human performance laboratory where they were familiarized with the equipment, execution of exercises used in the experimental protocol, and underwent 1 repetition maximum testing (1RM) on the exercises included in the study. The second phase of the study comprised 2 visits to the laboratory undertaken in a counterbalanced order in which SBP, DBP, and HR were assessed before resistance exercise, on completion of each exercise and every 10 minutes for 60 minutes after each

resistance exercise session. The resistance exercise sessions comprised a low (40% 1RM) and high (80% 1RM) intensity sessions, and total work were kept the same. Subjects were also instructed to avoid participation in any structured exercise training in the 48 hours before each experimental condition, to maintain the same dietary patterns in the 24 hours before each experimental condition and to report to the laboratory well hydrated. All testing took place in the morning between 9.00 AM and 12.00 PM and at the same time across conditions to minimize any effect of circadian variation on the results obtained. The dependent variables were SBP, DBP, and HR with the



**Figure 1.** Mean ± SD of systolic blood pressure at rest, during and 60 minutes after high- and low-intensity resistance exercise.



**Figure 2.** Mean  $\pm$  SD of mean arterial pressure at rest, during and 60 minutes after high- and low-intensity resistance exercise.

independent variable being exercise intensity and postexercise recovery time.

### Subjects

Following institutional ethics committee approval and written informed consent, 16 male participants aged 19–36 years (mean age  $\pm$  SD = 23.1  $\pm$  5.9 years) agreed to take part. The participants were all performed regular exercises, undertaking approximately 12 hours per week of structured exercise including resistance exercise training (mean years  $\pm$  SD of structured exercise training = 4.1  $\pm$  2.8) and had been actively exercising for at least 12 months before inclusion in the study. The participants came from a team sports background (basketball, soccer, rugby union), and all experimental testing took place within the preseason preparatory period. The subjects were instructed not to consume alcohol, coffee, or chocolate from 6.00 PM till the night before the experimental sessions. Subjects were excluded if they (a) smoked, (b) if resting SBP was 140 mm Hg or DBP was 90 mm Hg, (c) if they used medication that influenced cardiovascular responses or any other substance that could affect the individual performance including antihypertensives, anticoagulants, nonsteroidal anti-inflammatory agents, and herbal supplements, and if (d) they had any muscle, bone, or joint impairment/injury that could impede the execution of the exercises. Baseline participant characteristics are presented in Table 1.

### Experimental Protocol

Each participant attended the human performance laboratory on 3 occasions. The first visit to the laboratory involved a briefing session, measurement of resting BP and HR and determination of each participant's 1RM on the back squat, bench press and deadlift. In general, all of the participants had experience performing resistance exercises, specifically

the exercises included in this study. However, before commencing the 1RM testing, the proper lifting technique for each exercise was demonstrated to the participants. The 1RM was determined according to methods advocated by Kraemer et al. (18) and was used to set the 40 and 80% 1RM intensities undertaken during the proceeding experimental trials. Resting BP measurements were performed in accordance with recommended guidelines for assessment of BP in human (25) in the seated position after 20-minute rest. Two measurements were taken on each arm in the first

instance with the average of the 2 measurements for each arm being used for analysis. There was no significant difference between arm-to-arm measurement of BP ( $p > 0.05$ ). All subsequent measures were taken on the nondominant arm during experimental trials. Moreover, the resting BP was compared with pre-exercise BP measurement taken in the first experimental trial evidencing an appropriate intraclass correlation coefficient between the 2 trials ( $R = 0.826$ ). This was used to confirm BP status of the participants in line with recommended guidelines (5). All measurements were taken in a room/laboratory with only the participant and investigator present to reduce any effect of arousal on BP assessment as much as was practically possible. Before any measurements being taken, participants were asked whether they had experienced any significant life events that would have caused undue stress in time before or in between experimental trials. No participant reported that this was the case. Participants were also asked to confirm that they had what they considered to be their normal duration and quality of sleep the night before each trial.

In the 2 subsequent sessions, participants rested in the seated position for 20 minutes, where resting BP and HR were assessed. Participants then performed resistance exercise at either 40% 1RM or 80% 1RM presented in a randomized order. The exercise sessions consisted of the back squat, bench press, and deadlift exercises and were performed in that order to balance lower- and upper-body exercises. Participants were blinded to which sessions they were going to perform until the beginning of the first exercise session.

In the 40% 1RM condition, participants performed 3 sets of 8 repetitions, and in the 80% 1RM condition participants performed 3 sets of 4 repetitions to ensure that the same total work was completed across exercise intensity conditions. An interval of 45 seconds was used between the sets with an interval of 2 minutes between exercises (28). Once

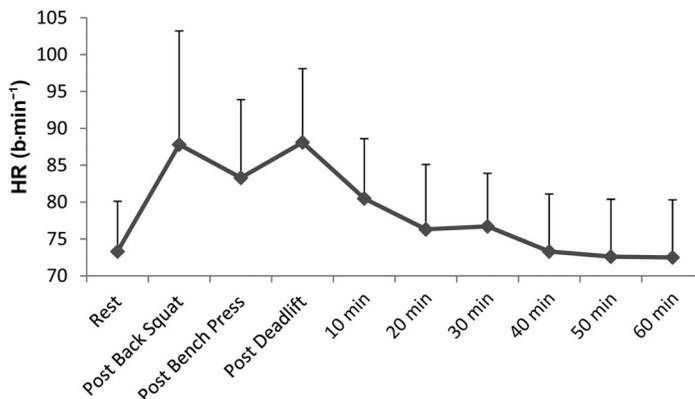


Figure 3. Mean  $\pm$  SD of heart rate ( $b \cdot \text{min}^{-1}$ ) at rest, during and 60 minutes after resistance exercise.

each experimental condition was completed, participants walked approximately 10 m to a quiet room where they rested in a seated position for 60 minutes. Air temperature was kept between 20 and 22° C.

#### Lifting Procedures

All exercises were performed using a 20 kg Eleiko bar (Eleiko Sport AB, Halmstad, Sweden), Pullum Power Sports lifting cage, and Olympic lifting platform (Pullum Power Sports, Luton, United Kingdom). All lifts were completed in accordance with protocols previously described, by Earle and Baechle (10), for the back squat, bench press, and deadlift. A trained researcher/spotter was present during all testing sessions to ensure proper range of motion. Any lift that deviated from proper technique was not counted. During all exercises and across conditions, repetition frequency was paced by a metronome set at 60  $b \cdot \text{min}^{-1}$ . This cadence resulted in 1 complete repetition every 4 seconds with concentric and eccentric phases comprising 2 seconds each.

#### Blood Pressure and Heart Rate Assessment

Systolic blood pressure, DBP, and HR were determined at rest, on completion of each exercise (back squat, bench press, deadlift) and every 10 minutes after completion of each exercise bout for a period of 60 minutes using an automated sphygmomanometer (BoSo Medicus, Jungingen, Germany) and Polar heart rate monitor (RS400, Polar electro, Oy, Kuopio, Finland). Mean arterial pressure (MAP) was calculated from SBP and DBP data using the formula:  $(2\text{DBP} + \text{SBP})/3$ . All participants were instructed to relax the arm during the monitor recording. Talking and moving were not allowed, but reading was permitted. Pre-exercise measurements were taken in a room adjacent to the laboratory, whereas postexercise measurements were taken in the laboratory. All measures were taken in the seated position and in accordance with guidelines described above

(25). Pre-exercise and postsession BP assessment was completed by a separate investigator to the 1 taking postexercise BP measurement within each session to ensure accurate blinding of those taking postexercise measures to the experimental session just completed.

#### Statistical Analyses

Any changes in SBP, DBP, MAP, or HR were analyzed using a series of 2 (Exercise intensity)  $\times$  10 (time period) ways repeated-measures analysis of variance (ANOVA). Where significant differences were found, Bonferroni post hoc pairwise comparisons were

used to determine where the differences lay. Partial eta squared ( $\eta^2$ ) was also used as a measure of effect size. Data also underwent a secondary analysis using resting BP and HR values as covariates in the model to control for any possible effect of resting measures on the dependant variables assessed during experimental conditions. Before data analysis, data were checked to ensure they met all the requirements for use of parametric statistical techniques. A priori power analysis indicated that a sample size of 13 was needed for an effect size of 0.25 at 95% power with a  $p$  value of 0.05. The Statistical Package for Social Sciences (SPSS version 20; SPSS, Inc., Chicago, IL, USA) was used for all analysis, and statistical significance was set, a priori, at  $p = 0.05$ .

#### RESULTS

Results from the repeated-measures ANOVA indicated a significant intensity  $\times$  time interaction for SBP ( $F_{(9, 135)} = 2.094$ ,  $p = 0.034$ , partial  $\eta^2 = 0.122$ ). Post hoc analysis indicated that SBP at 50-minute recovery and 60-minute recovery was significantly lower after high-intensity exercise ( $p = 0.01$  in both cases) compared with low-intensity exercise (Figure 1). There were no significant main effects or interactions in regard to DBP (all  $p > 0.05$ ). Data for MAP also indicated a significant intensity  $\times$  time ( $F_{(9, 135)} = 1.97$ ,  $p = 0.047$ , partial  $\eta^2 = 0.116$ ). Similar to results for SBP, post hoc analysis indicates that MAP was significantly lower at 50-minute recovery and 60-minute recovery after high-intensity exercise ( $p = 0.05$  in both cases) compared with low-intensity exercise (Figure 2). In regard to HR, data indicated a significant main effect for time ( $F_{(9, 135)} = 2.479$ ,  $p = 0.0001$ , partial  $\eta^2 = 0.344$ ). Post hoc multiple comparisons indicated that HR was significantly higher after squat, bench press, and deadlift exercise compared with resting HR and HR at 40-, 50-, and 60-minute recovery (all  $p = 0.03$  or better, Figure 3). When data were rerun using Analysis of

**TABLE 2.** Mean (*SD*) of SBP, DBP, MAP, and HR pre, during, and 60 min after low-intensity resistance exercise (40% 1RM) and high-intensity resistance exercise (80% 1RM).\*

	Pre-exercise	Post-back squat	Post-bench press	Post-deadlift	10-min postexercise	20-min postexercise	30-min postexercise	40-min postexercise	50-min postexercise	60-min postexercise
Low intensity										
SBP (mm Hg)	129.9 (4.4)	142.8 (11.3)	140.6 (14.4)	146.6 (19.4)	136.3 (12.1)	132.3 (19.1)	134.7 (10.9)	132.3 (8.6)	132.8 (7.4)	131.2 (6.3)
DBP (mm Hg)	79.9 (8.2)	80.7 (9.3)	84.5 (18)	83.9 (19.6)	83.8 (10.2)	85.8 (9.5)	85.5 (11.7)	80.4 (8.5)	83.6 (7.4)	81.4 (9.1)
MAP (mm Hg)	95.2 (5.3)	101.4 (7.4)	103.2 (15.7)	104.6 (19.6)	101.2 (9.7)	101.3 (10.1)	101.9 (10.1)	97.6 (7.1)	99.8 (5.2)	97.2 (5.3)
HR (b·min <sup>-1</sup> )	74.7 (6.9)	91.1 (16.5)	82.5 (10.6)	127.5 (15.5)	83.6 (8.1)	76.6 (9.7)	76.9 (9.9)	71.6 (9.2)	72.6 (7.6)	72.6 (8.8)
High intensity										
SBP (mm Hg)	129 (3.5)	147 (16.8)	138.7 (10.7)	143.8 (12.5)	136.3 (14.2)	136.1 (10.7)	134.3 (8.8)	129.9 (7.1)	124.5 (10.7)	125.1 (9.2)
DBP (mm Hg)	80.8 (10.8)	70.4 (11.6)	80.6 (17.6)	83.1 (10.9)	81.8 (9.4)	88.5 (11.6)	78.5 (11.9)	78.0 (10.4)	80.1 (12.8)	80.8 (10.8)
MAP (mm Hg)	95.5 (5.6)	103.8 (11.7)	100 (8.6)	103.4 (13.8)	100.1 (10.8)	99.9 (9.2)	103.7 (15.3)	95.7 (9.2)	93.5 (9.2)	95.6 (9.5)
HR (b·min <sup>-1</sup> )	76.0 (8.5)	84.7 (15.6)	84.4 (11.9)	116.3 (10.3)	77.4 (9.2)	76.1 (8.2)	81.5 (9.2)	75.1 (7.9)	73.4 (5.5)	72.6 (7.5)

\*SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; MAP = mean arterial pressure; RM = repetition maximum.

Covariance (ANCOVA) controlling for resting SBP, DBP, MAP, and HR, the results from statistical analysis remained the same, and none of the covariates were significant within these models (all  $p > 0.05$ ). Mean  $\pm$  SD of SBP, DBP, MAP, and HR between low- and high-intensity resistance exercise and across time are presented in Table 2.

## DISCUSSION

The main findings of this study are that a single bout of high-intensity resistance exercise, but not low-intensity resistance exercise, decreased SBP 50 minutes after the exercise bout. There was no effect of resistance exercise on DBP, and both low- and high-intensity resistance exercise resulted in comparably elevated HR during exercise, which returned to resting levels within 60 minutes after exercise. This finding is in keeping with previous studies (3,4,9,11,19) that have shown PEH in normotensive participants after resistance exercise. The magnitude of fall in SBP reported in previous studies varies considerably (ranging from 3 to 20 mm Hg). In this study, the fall in SBP was only significant in the high-intensity resistance exercise condition with the decrease seen at 50- and 60-minute recovery comparable with that seen in some previous studies of PEH (14,28,30) and similar to those reported after aerobic exercise (22) with this decrease being apparent at 50- and 60-minute postexercise. However, the results reported in this study are in contrast to other studies that have reported decreases in SBP postresistance exercise in lower intensity resistance exercise sessions such as that reported by Focht and Koltyn (12) at an intensity of 50% 1RM and data reported by Rezk et al. (28) at an intensity of 40% 1RM.

In regard to DBP, the results of this study evidence no significant change in DBP either after high- or low-intensity resistance exercise. Little change in DBP after resistance exercise is not unusual although some previous studies have reported a significant decrease in DBP after resistance exercise (2,14). The results of this study are clearly not congruent with this past research. However, the lack of change in DBP during resistance exercise is similar to other studies (26,27) and can be interpreted as a diastolic hypotensive effect similar to that described by other authors (28).

The HR responses reported after both low and high resistance exercise are also common (28), and the lack of significant difference between exercise intensities is also not unexpected because of the matching of resistance exercise sessions for total work completed.

The discrepancy between the findings of this study and that of previous research in relation to SBP and to a lesser extent DBP may be explained by a number of issues. Previous research on this topic has employed a variety of resistance exercise protocols in their examination of PEH. This has ranged from circuit-based resistance exercise (2), 3 sets of upper and lower body resistance exercises performed at 80% 1RM (14), 3 sets of upper and lower body resistance exercises performed at an intensity of 6RM (27), and 3 sets of

upper and lower body resistance exercises performed at 40 and 80% 1RM but matched for total work performed (28). Thus, it is not surprising that the data regarding the effect of resistance exercise on PEH are equivocal. In this study, a design similar to that used by Rezk et al. (28) was used. This is important as it ensures that any changes in BP that are recorded are as a result of the intensity of exercise performed rather than being a result of increased total work as can be the case in protocols using 3 sets of 10 repetitions at a high and low intensity.

The discrepancy between the findings of this study and that of Rezk et al. (28) may, therefore, be because of the exercise protocols used. In the study by Rezk et al. (28), their resistance exercise protocol involved 6 upper and lower body resistance exercises performed on exercise machines. In contrast, this study used a protocol involving 3 resistance exercises using free weights. These exercises were chosen as they were typical of those performed by resistance trained individuals, were total body compound exercises; and given the “trained” status of the participants in this study, this was thought to provide a more realistic approximation of the types of exercises undertaken by trained individuals as opposed to machine based exercises. This was also congruent with suggestions previously made by Polito and Farinatti (26) that exercises involving greater muscle mass are desirable when examining the impact of resistance exercise on PEH. In this study, a systolic hypotensive effect was evidenced after a relatively smaller number of exercises than used in previous research. However, the exercises used in this study involved compound movements performed using free weights, rather than machines, and as such likely resulted in recruitment of a greater number of muscles (and increased muscle mass) during execution. This difference in terms of protocol and volume of exercises performed might, however, have resulted in differences in the cardiovascular and hemodynamic loads involved in this study and that of Rezk et al. (28) and therefore explain why the difference in magnitude of systolic hypotension was greater in the paper by Rezk et al. (28) compared with this study. Likewise, the participants in the study by Rezk et al. (28) did not “exercise regularly” and were a mixed gender group. Conversely, the participants in this study were “trained” males and undertook strength and conditioning activities regularly. It may, therefore, be that training status influences the postexercise cardiovascular and hemodynamic responses to resistance exercise with larger differences seen in less trained individuals. This point is, however, speculative and further research would be needed to explore this claim. Likewise, gender influences hemodynamic responses in a number of situations (28) and therefore single gender participant groups are needed to fully understand any hypotensive effects after resistance exercise.

In addition to the restricted number of exercises performed in this study, postexercise recovery data were only taken for a 60-minute period. Although this recovery

duration is not uncommon in the literature (14,26,27), other studies have reported PEH for up to 90 minutes after resistance exercise (28), and in some studies of post-aerobic exercise hypotension, BP has been assessed for 9 hours after exercise (24). Therefore, assessment of BP in recovery from exercise for a longer duration than that used in this study would have been desirable. Moreover, as with any laboratory study, BP reductions may not be accurately reflected during laboratory measurement because of short assessment time periods. Ambulatory BP monitoring may have been helpful in providing additional information about how BP is affected during activities of normal living and to what extent resistance exercise might influence these responses. Unfortunately, ambulatory BP assessment was not available to us, but this mode of BP assessment may be fruitful for researchers in future work. We also recognize that arousal may have influenced BP measurement immediately after each resistance exercise because of the different surroundings in which BP was assessed. Although BP assessment was conducted according to recommended guidelines (25), future research should attempt to minimize any effect of arousal on BP measurement. It is difficult to manage such a possibility as increased arousal may be likely in any study that assesses BP after resistance exercise. Furthermore, in this study, only BP and HR were assessed. There is evidence that cardiac sympathetic modulation of the heart is influenced after resistance exercise (28). Therefore, inclusion of measures of HR variability in future studies may be useful in providing a more comprehensive understanding of the cardiac and hemodynamic responses to resistance exercise. Finally, this study examined responses in trained normotensives. Although no study to date appears to have examined PEH in a specifically strength trained population and there is evidence to suggest that BP responses will be similar in normotensives and hypertensives (11), there is a need to examine this topic in hypertensive populations as a means to understand whether free weight compound resistance exercises might be effective in reducing high BP in the short and longer term. Despite this, it is also important to understand how exercise “doses” impact on health related parameters in apparently healthy, relatively fit, trained individuals. Because resistance exercise is regularly prescribed to individuals without known hypertension as a means to reduce cardiovascular disease risk (1), understanding the magnitude of any PEH response can be used to inform planning effective resistance exercise programs. This is particularly so as the PEH response has been suggested as, at least partly responsible for the lower resting BP values recorded in habitually physically active individuals (23).

### PRACTICAL APPLICATIONS

The present findings suggest that an acute bout of high-intensity resistance exercise using compound movements can promote PEH in normotensive regular exercisers. Responses to low-intensity resistance exercise did not

evidence a hypotensive effect. Thus, any potential health effect of resistance exercise on BP may only be realized after high-intensity exercise, and therefore high-intensity resistance exercise might be prescribed as a means to promote PEH. The use of compound movements with free weights also likely resulted in a greater number of muscles being recruited during the resistance exercise session and hence indicates the importance of including such compound movements in exercise programs designed to improve health parameters. Because resistance exercise is commonly prescribed to individuals without known hypertension to enhance health, the results presented here should be considered when designing resistance training programs for health benefit with high-intensity resistance exercise incorporating compound movements used to promote short-term PEH in normotensive participants.

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