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The effect of exercise intensity on post resistance exercise hypotension in trained men.

Running Head: Exercise intensity and hypotension

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Abstract

The occurrence of post resistance exercise hypotension (PEH) is controversial and the influence of resistance exercise intensity on PEH remains unknown. This study examined blood pressure (BP) 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 ± 5.9 years) performed an acute bout of low (40% of 1 repetition maximum) and high intensity (80% 1 repetition maximum) resistance exercise, matched for total work, separated by 7 days and performed in a counterbalanced order. Systolic (SBP and diastolic (DBP) blood pressure, mean arterial pressure (MAP) and HR were assessed pre-exercise, following completion of each exercise resistance exercise (3 sets of back squat, bench press and deadlift) and every 10minutes post resistance exercise for a period of 60minutes. Results indicated a significant intensity X time interaction for SBP (P = .034, partial $\eta^2 = .122$) and MAP (P = .047, partial $\eta^2 = .116$) whereby SBP and MAP at 50minutes recovery and 60minutes recovery were significantly lower following high intensity exercise (P = .01 for SBP and P = .05 for MAP in both cases) compared to low intensity exercise. There were no significant main effects or interactions in regard to DBP (all P>0.05). HR data indicated a significant main effect for time (F 9, 135 = 2.479, P = .0001, partial $\eta^2 = .344$). Post-hoc multiple comparisons indicated that HR was significantly higher post squat, bench press and deadlift exercise compared to resting HR and HR at 40, 50 and 60 minutes recovery (all P = .03). The present findings suggest that an acute bout of high intensity, but not low intensity, resistance exercise using compound movements can promote post exercise hypotension in trained men.

Keywords: Strength; Systolic Blood Pressure; Diastolic Blood Pressure; Heart Rate; Exercise Prescription
Introduction

A considerable amount of evidence has shown that regular exercise lowers both systolic and diastolic blood pressure (6, 15, 22). The exact mechanisms by which exercise leads to a reduction in 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 7mmHg with a time course of up to 22 hours after cessation of aerobic exercise and under ambulatory conditions (6, 13, 16, 19). Although the PEH responses to aerobic exercise have been well described, 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). More, 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 is not sufficient to gain a conclusive picture of how different facets of resistance exercise (e.g., intensity, training status of participants) influence PEH. Thus, further studies are required so more effective prescription of resistance exercise to elicit PEH can be targeted.

Experimental and meta-analytical work has shown that resistance exercise may reduce both SBP and DBP in normotensive and hypertensive populations (6, 7, 8, 16). The most recent of these meta-analysis confirmed that both dynamic and isometric resistance exercise has a beneficial effect on BP in individuals with optimal BP or prehypertension (8). Studies have also suggested that PEH occurs after resistance exercise but that the
magnitude and duration of the BP lowering effects are less than occurs with aerobic exercise (11, 12, 26, 28). During resistance exercise, blood pressure increases (20). On cessation of a resistance exercise bout authors have reported that BP remains elevated (4, 12), is lower (2, 9, 11) or is equal to (21, 29) pre exercise values. The equivocal nature of this literature may be due to a number of reasons including the exercise protocol employed, 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 a key mechanism 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 post exercise BP responses to resistance exercise, controlling for total work done, in trained males. We hypothesised that both low and high intensity resistance exercise would elicit post exercise hypotensive effects, congruent with the prior work on this area using a similar intensity, matched for work protocol (28) but that in the current 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 three separate occasions separated by at least 7 days and in 2 separate phases. In the first phase participants attended the human performance laboratory were familiarized with the equipment, exercise execution production procedures and underwent 1 repetition maximum testing (1RM) on the exercise tests being used in the study. The second phase of the study comprised two visits to the laboratory undertaken in a counterbalanced order in which SBP, DBP and heart rate (HR) were assessed before resistance exercise, on completion of each
exercise and after (every 10 minutes for 60 minutes) 2 resistance exercise sessions. The resistance exercise sessions comprised a low (40% 1RM) and high (80% 1RM) intensity sessions with total work being kept the same. Subjects were also instructed to avoid participation in any structured exercise training in the 48 hours prior to each experimental condition, to maintain the same dietary patterns in the 24 hours prior to 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 minimise any effect of circadian variation on the results obtained. The dependant variables were SBP, DBP and HR with the independent variable being exercise intensity and post exercise recovery time.

Subjects

Following institutional ethics committee approval and written informed consent, 16 male participants aged 19-36 years (mean age ± S.D. = 23.1 ± 5.9 years) agreed to take part. The participants were all regular exercises, undertaking approximately 12 hours per week of structured exercise including resistance exercise training (Mean years ± S.D. of years engaged in structured exercise training = 4.1 ± 2.8 years) 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 pre-season preparatory period. A priori power analysis indicated that a sample size of 13 was needed for an effect size of .25 at 95% power with a P value of .05. The subjects were instructed not to consume alcohol, coffee or chocolate from 6.00 pm the night before the experimental sessions. Subjects were excluded if they a) smoked; b) if resting SBP was 140 mmHg or DBP was 90 mmHg; 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 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 three 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. All participants had experience performing resistance exercises in general and these exercises in particular. However, prior to commencing the 1RM testing, each exercise, with proper lifting technique, was demonstrated to each participant. 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 following 20 minutes rest. Two measurements were taken on each arm in the first instance with the average of the two 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 non-dominant arm during experimental trials. Moreover, the resting BP was compared to pre-exercise BP measurement taken in the first experimental trial evidencing an appropriate intraclass correlation coefficient between the two trials (R = .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. Prior to any measurements being taken, participants were asked whether they had had any significant life events which would have caused undue stress in time prior to or in between experimental trials. No
participant reported that this was the case. Participants were also asked to confirm that they had had what they considered to be their normal duration and quality of sleep the night before each trial.

In the two subsequent sessions participants rested in the seated position for 20 minutes where resting BP and HR was assessed. They then participated in 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 in 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 in order 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 each experimental condition was completed, participants walked approximately 10 metres to a quiet room where they rested in a seated position for 60 minutes. Air temperature was kept between 20 and 22 degrees C.

**Lifting Procedures**

All exercises were performed using a 20kg Eleiko bar (Eleiko Sport AB, Halmstad, Sweden), Pullum Power Sports lifting cage and Olympic lifting platform (Pullum Power Sports, Luton, UK). All lifts were completed in accordance with protocols previously described, by Earle and Baechle, for the back squat, bench press and deadlift (10). 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 beats min⁻¹. This cadence resulted
in one complete repetition every 4 s with concentric and eccentric phases comprising 2 s each.

**Blood pressure and heart rate assessment**

Systolic (SBP) and diastolic (DBP) blood pressure and heart rate was 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: (2DBP+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 whilst post exercise 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 post session BP assessment was completed by a separate investigator to the one taking post-exercise BP measurement within each session to ensure accurate blinding of those taking post exercise measures to the experimental session just completed.

**Statistical Analysis**

Any changes in SBP, DBP, MAP or HR were analysed using a series of 2(Exercise intensity) X 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 blood pressure 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. Prior
to data analysis data were checked to ensure they met all the requirements for use of parametric statistical techniques. Statistical Package for Social Sciences (SPSS, Version 20, 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 X time interaction for SBP ($F_{9, 135} = 2.094$, $P = .034$, partial $\eta^2 = .122$). Post-hoc analysis indicated that SBP at 50minutes recovery and 60minutes recovery was significantly lower following high intensity exercise ($P = .01$ in both cases) compared to low intensity exercise (See 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 X time ($F_{9, 135} = 1.97$, $P = .047$, partial $\eta^2 = .116$). Similar to results for SBP, post-hoc analysis indicates that MAP was significantly lower at 50minutes recovery and 60minutes recovery was significantly lower following high intensity exercise ($P = .05$ in both cases) compared to low intensity exercise (See Figure 2). In regard to HR, data indicated a significant main effect for time ($F_{9, 135} = 2.479$, $P = .0001$, partial $\eta^2 = .344$). Post-hoc multiple comparisons indicated that HR was significantly higher post squat, bench press and deadlift exercise compared to resting HR and HR at 40, 50 and 60 minutes recovery (all $P = .03$ or better, See Figure 3). When data were rerun using 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 ± 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, but not low intensity, resistance exercise decreased systolic BP 50 minutes following the exercise bout. There was no effect of resistance exercise on diastolic BP and both low and high intensity resistance exercise resulted in comparably elevated heart rate during exercise which returned to resting levels with 60 minutes post exercise. This finding is in keeping with previous studies (3, 4, 9, 11, 19) that have shown post exercise hypotension in normotensive participants following resistance exercise. The magnitude of fall in SBP reported in prior studies varies considerably (ranging from 3 – 20 mmHg). In the present study the fall in SBP was only significant in the high intensity resistance exercise condition with the decrease seen at 50 and 60 minutes recovery comparable with that seen in some prior studies of post resistance exercise hypotension (14, 28, 30) and similar to those reported following aerobic exercise (22) with this decrease being apparent at 50 and 60 minutes post exercise. However, the results reported in the presented study are in contrast to other studies that have reported decreases in SBP post resistance exercise in lower intensity resistance exercise sessions such as that reported by Focht et al. (12) at an intensity of 50% 1RM and data reported by Rezk et al. (28) at an intensity of 40%1RM.

In regard to diastolic BP the results of this study evidence no significant change in DBP following either high or low intensity resistance exercise. Little change in DBP following resistance exercise is not unusual although some prior studies have reported a significant decrease in DBP following resistance exercise (2, 14). The results of the present 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 heart rate responses reported following both low and high resistance exercise are also common (28) and the lack of significant difference between exercise intensities is also not unexpected due to the matching of resistance exercise sessions for total work completed.
The discrepancy between the findings of the current study and that of prior research in relation to SBP and to a lesser extent DBP may be explained by a number of issues. Prior research on this topic has employed a variety of resistance exercise protocols in their examination of post exercise hypotension. 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 done (28). Thus, it is not surprising that the data regarding the effect of resistance exercise on post exercise hypotension is equivocal. In the present study, a design similar to that used by Rezk et al. (28) was employed. This is important as it ensures that any changes in blood pressure 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 the present study and that of Rezk et al. (28) may therefore be due to the exercise protocols employed. 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 the present study employed a protocol involving 3 resistance exercises using free weights. These exercises were chosen as they were typical of those performed by resistance trained individuals, are total body compound exercises and given the ‘trained’ status of the participants in the present 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 et al. (26) that exercises involving greater muscle mass are desirable when examining the impact of resistance exercise on post exercise hypotension. In the current study a systolic hypotensive effect was evidenced following a relatively smaller number of exercises than used in prior research. However, the exercises used in the present 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 the present study and that of Rezk et al (28) and therefore explain why the difference in magnitude of systolic hypotension was greater in the Rezk et al (28) paper compared to the present 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 the present study were ‘trained’ males and undertook strength and conditioning activities regularly. It may therefore be that training status influences the post exercise 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 post resistance exercise.

In addition to the restricted number of exercises employed in the present study post exercise recovery data was only taken for a 60minute period. Although this recovery duration is not uncommon in the literature (14, 26, 27), other studies have reported post resistance exercise hypotension for up to 90minutes post resistance exercise (28) and in some studies of post aerobic exercise hypotension, blood pressure has been assessed for 9 hours post exercise (24). Therefore, assessment of blood pressure in recovery from exercise for a longer duration than that used in the present study would have been desirable. Moreover, as with any laboratory study, BP reductions may not be accurately reflected during laboratory measurement due to 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 recognise that arousal may have influenced BP measurement immediately post each resistance exercise due to 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 which assesses BP post resistance exercise. Furthermore, in the present study only blood pressure and heart rate were assessed. There is evidence that cardiac sympathetic modulation of the heart is influenced following resistance exercise (28). Therefore, inclusion of measures of heart rate 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. While no study to date appears to have examined post resistance exercise hypotension 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 blood pressure 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. As resistance exercise is regularly prescribed to individuals without known hypertension as a means to reduce cardiovascular disease risk (1) understanding the magnitude of any postexercise hypotensive response can be used to inform planning effective resistance exercise programmes. This is particularly so as the post exercise hypotensive response has been suggested as, at least partly responsible for the lower resting blood pressure 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 post exercise hypotension 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 blood pressure may only be realised following high intensity exercise and therefore high intensity resistance exercise might be prescribed as a means to promote postexercise hypotension. 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 programmes designed to improve health parameters. As resistance exercise is commonly prescribed to individuals without known hypertension to enhance health, the results presented here should be considered when designing resistance training programmes for health benefit with high intensity resistance exercise incorporating compound movements utilised to promote short term post exercise hypotension in normotensive participants.

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References


Figure Captions

Figure 1. Mean ± S.D. of Systolic Blood Pressure (SBP) at rest, during and 60 minutes post high and low intensity resistance exercise

Figure 2. Mean ± S.D. of Mean Arterial Pressure (MAP) at rest, during and 60 minutes post high and low intensity resistance exercise

Figure 3. Mean ± S.D. of Heart rate (BPM) at rest, during and 60 minutes post resistance exercise