LOWER BODY FIRST RESISTANCE EXERCISE SEQUENCING AMPLIFIES POST EXERCISE HYPOTENSION IN ACTIVE WOMEN

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Abstract

Purpose. Exercise order is one of several manipulable variables in resistance training, yet its acute effect on blood pressure remains underexamined, particularly in women. This study compared two strength training sessions performed in opposite exercise orders and measured the resulting post exercise hypotension (PEH) in active women.

Methods. Fourteen active normotensive women (38 ± 6.1 years, 58 ± 8.7 kg, 158 ± 7.9 cm) completed two strength training sessions in a randomized crossover design. Both sessions used the same four compound free weight exercises (bench press, hex bar squat, shoulder press, stiff leg deadlift) at 75% of one repetition maximum for three sets of ten repetitions with 90 second rest intervals. EO1 progressed from upper to lower body; EO2 progressed from lower to upper body. Systolic (SBP) and diastolic (DBP) blood pressure, mean arterial pressure (MAP) and heart rate (HR) were recorded at rest and at 1, 10, 20 and 30 minutes post exercise. Data was analyzed with two way repeated measures ANOVA.

Results. Significant group and time effects were found (group $F_{1,13} = 4.44$, $p \leq 0.05$; time $F_{4,10} = 43.17$, $p < 0.0001$). EO2 lowered SBP at 20 and 30 minutes, DBP from 10 through 30 minutes, and MAP at 20- and 30-minutes relative to rest, with moderate to large effect sizes at 30 minutes. Between sessions mean reductions favored EO2 for SBP (−3.7 ± 2.4 mmHg, p = 0.028), DBP (−3.8 ± 1.1 mmHg, p = 0.001) and MAP (−3.6 ± 1.1 mmHg, p = 0.002). HR rose after both sessions with no between session difference (p = 0.504).

Conclusion. Starting a compound strength training session with lower body exercises produced a larger and longer post exercise reduction in blood pressure than starting with upper body exercises. Lower body first sequencing may be the preferable arrangement when an acute blood pressure lowering response is the goal.

Keywords: Post Exercise Hypotension; Exercise Order; Resistance Training; Blood Pressure; Mean Arterial Pressure; Non-Communicable Disease; Cardiovascular Disease; Gender Equality; Premature Mortality

INTRODUCTION

Shifts in diet and physical activity over recent decades have raised the burden of non-communicable disease worldwide. Cardiovascular disease sits at the center of that burden, and elevated blood pressure is among its strongest modifiable contributors (Kong et al., 2022). In Malaysia, ischemic heart disease was again the leading cause of death in 2024, accounting for 17,421 deaths, or 13.0% of medically certified deaths, the first year these statistics were compiled under ICD 11 (Department of Statistics Malaysia, 2025). Ischemic heart disease also affects middle aged adults, becoming the leading cause of death among people aged 41 to 59 (5,380 deaths, 17.6%), and Selangor, where the present study was conducted, recorded the highest state count at 3,038 deaths. Controlling modifiable risk factors, particularly elevated blood pressure, is essential for reducing premature mortality associated with non-communicable diseases. This objective aligns with Sustainable Development Goal Target (SDG) 3.4, which aims to reduce premature mortality from non-communicable diseases by one-third by 2030. (United Nations, 2015; World Health Organization, 2006).

Regular exercise lowers resting arterial pressure and is recommended as a first line, non-pharmacological strategy for managing hypertension. Part of this benefit comes from an acute drop in blood pressure that follows a single exercise bout, termed post exercise hypotension (PEH). A reduction as small as 3 mmHg in resting pressure lowers the risk of stroke and coronary disease, so a session that reliably produces a meaningful and sustained PEH carries clinical value (Tipton, 2011). A meta-analysis of 30 acute resistance exercise trials (62 interventions, N = 480) found post session reductions of roughly 1 to 4 mmHg in people with normal pressure and up to about 12 mmHg in those with elevated pressure, confirming that strength training produces a reliable, if modest, hypotensive response (Farinatti et al., 2021).

Several strength training variables shape the magnitude and duration of PEH, including training volume, load intensity, the number of sets, the amount of muscle mass recruited the rest interval between sets, and exercise order (Simão et al., 2012; Figueiredo et al., 2015a, 2015b, 2016). Of these, exercise order has received the least attention even though it influences the efficiency, safety and effectiveness of a session when prescribed alongside load, volume and rest (Simão et al., 2012). Figueiredo et al. (2013) compared upper to lower to upper sequences after a strength session in normotensive men and found no PEH, although heart rate variability shifted toward sympathetic dominance under the upper to lower order. Studies manipulating rest interval, load and set number have produced PEH of varying size (De Salles et al., 2010; Figueiredo et al., 2015a, 2015b, 2016), but direct comparisons of exercise order remain limited.

Most of this evidence comes from men. Women are underrepresented in PEH research, even though strength training lowers post session pressure in this group too. For instance, one study found that a single multi joint session using large muscle groups elicited PEH in normotensive trained women (Freire et al., 2017), and conventional strength training reduced systolic pressure during recovery in normotensive strength trained women (Monteiro et al., 2023). Whether the order of exercises alters that response in women is unclear, and the few order studies in women have examined the sequence of aerobic and resistance work rather than the arrangement within a resistance session (Garcês et al., 2025). This study compared two strength training sessions that used identical exercises in opposite orders, one beginning with upper body and the other with lower body movements, and measured SBP, DBP, MAP and HR over 30 minutes of recovery. We hypothesized that beginning with lower body, large muscle mass exercises, combined with short rest intervals, would produce a larger and longer PEH than beginning with upper body exercises.

LITERATURE REVIEW

Post exercise hypotension and its clinical significance

Post exercise hypotension refers to the prolonged fall in arterial pressure below pre-exercise resting values that continue once a single exercise bout has ended (Tipton, 2011). Its clinical interest rests on a straightforward observation that even with a reduction of only 3 mmHg in resting pressure lowers the population level risk of stroke and coronary disease, so any session that reliably produces a meaningful and lasting reduction carries therapeutic value (Tipton, 2011). However, the magnitude of the response is not fixed and appears to depend strongly on baseline values. Pooled evidence from acute resistance exercise trials points to reductions of roughly 1 to 4 mmHg in people with normal pressure and considerably larger falls, up to about 12 mmHg, in those with elevated pressure (Farinatti et al., 2021). This gradient explains why most applied work has concentrated on hypertensive and prehypertensive samples, leaving the normotensive response, and especially the response of trained individuals, comparatively under characterized.

Mechanisms underlying the response

The mechanisms that generate post exercise hypotension are only partly resolved, and the resistance exercise literature has increasingly questioned the assumption that autonomic withdrawal is the main driver. In a systematic review and meta-analysis, Farinatti et al. (2021) reported that the hypotension following resistance exercise is not mediated by autonomic control, as sympathetic activity tends to rise and parasympathetic activity tends to fall during recovery, which is the reverse of what an autonomic withdrawal model would predict. Interest has consequently moved toward peripheral vascular mechanisms, chiefly a reduction in vascular resistance driven by local vasodilation. The quantity of active muscle mass appears central to this route, since greater recruitment is associated with larger reductions in resistance, partly through nitric oxide mediated increases in blood flow (Polito & Farinatti, 2009). Direct support comes from work in hypertensive women, in whom resistance sessions that raised plasma nitrite produced clear systolic and diastolic hypotension (Martins et al., 2024). Exercise intensity shapes the haemodynamic profile further by reducing plasma volume and altering cardiac output during recovery (Rezk et al., 2006), while the slow restoration of baroreflex sensitivity after exercise (Niemelä et al., 2008) helps account for why the response can persist well beyond the immediate post exercise window.

Resistance training variables that modulate the response

Exercise order belongs to a wider set of programmable resistance training variables that shape the magnitude and duration of the hypotensive response, among them training volume, load intensity, the number of sets, the rest interval between sets, and the amount of muscle mass recruited (Simão et al., 2012). Each has been studied in turn. Varying the number of sets alters both blood pressure and heart rate variability after a session (Figueiredo et al., 2015a), as does load intensity (Figueiredo et al., 2015b). Rest interval length influences the response in older and prehypertensive men, with longer intervals tending to favor a larger systolic effect (De Salles et al., 2010; Figueiredo et al., 2016), and muscle mass interacts with set number to determine the size of the fall (Polito & Farinatti, 2009). Training modality is also an important consideration because traditional and high-speed resistance training formats appear to produce different blood pressure responses in older adults (Coelho-Júnior et al., 2022), and time efficient superset arrangements have been weighed against conventional sets, with early reports suggesting hypotension only after the higher volume traditional method (Bentes et al., 2017). A recent meta-analysis tempers that conclusion, finding broadly comparable blood pressure responses across superset and traditional prescriptions despite the time supersets save (Zhang et al., 2025). Taken together, these studies imply that total work and rest interval, rather than session format itself, govern the response, which leaves exercise order as a plausible but still untested lever.

Exercise order as an under studied variable

Against this background, exercise order has received the least empirical attention, despite its bearing on the efficiency, safety and effectiveness of a session once load, volume and rest have been set (Simão et al., 2012). The few experimental tests have been equivocal. Figueiredo et al. (2013) compared upper to lower to upper sequences in normotensive men and found no hypotension under either order, although heart rate variability shifted toward sympathetic dominance when the session opened with upper body work. Lemos et al. (2018) manipulated both the order of upper body exercises and the rest interval in trained men and reported that the larger systolic effect tracked the 90 second interval rather than the sequence, so their result speaks more to rest interval than to order. The resulting literature makes the independent contribution of exercise order hard to isolate, because order has typically been confounded with rest interval, total work or training status. Whether ordering compound movements by the body region trained first changes the response, and whether any such effect appears in women, remains unresolved.

Post exercise hypotension in women

A further weakness of the existing evidence is its reliance on male samples. Women are underrepresented in post exercise hypotension research, even though resistance training lowers post session pressure in this group too. A single multi joint session built around large muscle groups was enough to elicit hypotension in normotensive trained women (Freire et al., 2017), and conventional strength training reduced systolic pressure during recovery in a comparable sample (Monteiro et al., 2023). Sex differences in the cardiovascular response to posture, such as the altered hemodynamics of prolonged sitting (Gotshall et al., 1994), caution against assuming that findings from men transfer directly. The small body of order research in women has, moreover, examined the sequencing of aerobic and resistance work within a combined session rather than the arrangement of exercises inside a resistance session, reporting that order changed the blood pressure response in postmenopausal women (Garcês et al., 2025). The within session ordering of resistance exercises in women therefore remains largely untested, and it is this gap that the present study was designed to address.

METHODS

Fourteen active Participants

women aged 20 to 50 years with prior strength training experience volunteered. A pre study power analysis was conducted to determine the minimum sample size required to detect the expected effect with adequate statistical power (G*Power 3.1) with power = 0.91, α = 0.05 and an effect size of 0.35 returned a required sample of 11, so the recruited sample exceeded 80% statistical power (Figueiredo et al., 2015a). Inclusion criteria were, non-smoker, no metabolic disease, no skeletal injury, and no medication affecting cardiovascular response. Participants were either normotensive or hypotensive and trained in a structured program on more than one day per week. Each was informed of the procedures, possible risks and benefits, completed the Physical Activity Readiness Questionnaire (PAR-Q) and the International Physical Activity Questionnaire (IPAQ), and signed informed consent before participating.

Experimental design

The study used a randomized crossover design across five laboratory visits at a fitness center in Selangor, Malaysia. Visit 1 covered screening, anthropometry (height, body mass), resting blood pressure and familiarization. Visits 2 and 3 established one repetition maximum (1RM) for each exercise, with a retest 72 h later to confirm reliability (Baechle & Earle, 2000). Visits 4 and 5 were the two experimental strength training sessions, performed in counterbalanced order and separated by at least 72 h. Participants avoided caffeine, alcohol and strenuous activity before testing days.

One-repetition maximum testing

1RM was determined for bench press, hex bar squat, shoulder press and stiff leg deadlift, all performed with free weights. Testing began with a warmup at 40 to 60% of estimated maximum, after which load was increased progressively until the heaviest successfully completed lift was recorded, within three to five attempts and with 3 to 5 minutes rest between attempts (Kraemer & Fry, 1995). Standardized instructions, technique cues and verbal encouragement were given throughout. The retest 72 h later established reliability, and the range of motion followed Simão et al. (2012).

Strength training sessions

The two sessions used the same four compound exercises and differed only in order. EO1 began with the upper body and alternated toward the lower body in the sequence bench press, hex bar squat, shoulder press, stiff leg deadlift. EO2 reversed this, beginning with the lower body, stiff leg deadlift, shoulder press, hexbar squat, bench press. Each session comprised three sets of ten repetitions at 75% of 1RM with 90 second rest between sets and exercises, preceded by a warmup set of ten repetitions at 50% of 1RM (Lemos et al., 2018; Landers, 1984). No pause was permitted between eccentric and concentric phases, movement velocity was not controlled, and participants were instructed to avoid the Valsalva maneuver.

Blood pressure measurement

SBP, DBP and HR were measured with an automatic oscillometric monitor (Omron HEM-7130, Vietnam), which passed all requirements of the European Society of Hypertension International Protocol 2010 revision, reading within -1.8 ± 5.3 mmHg of mercury for SBP (Takahashi et al., 2015). A baseline reading was taken after 10 minutes of seated rest on arrival. Post exercise readings were taken within 1 min of session completion and at 10 minutes intervals through 30 minutes, following Figueiredo et al. (2015a, 2016). Participants remained seated with the arm supported at heart level, and the cuff was positioned 2 cm above the brachial artery. Mean arterial pressure was derived from SBP and DBP.

Statistical analysis

Analyses were run in SPSS 23.0. A two-way repeated measures ANOVA tested the effects of exercise order (EO1, EO2) and time (rest, 1, 10, 20, 30 minutes) on each dependent variable, with Tukey post hoc comparisons. Data are reported as mean ± standard deviation. Significance was set at $p < 0.05$. Effect sizes were classified using the scale of Rhea (2004) for recreationally trained participants: trivial ≤ 0.35, small 0.35 - 0.80, medium 0.80 - 1.50, large ≥ 1.50.

RESULTS

Participant characteristics

All 14 women completed both sessions. The group was normotensive at rest, with mean resting SBP of 108 ± 10.5 mmHg, DBP of 73 ± 8.2 mmHg, HR of 74 ± 7.3 bpm and MAP of 84 ± 9.0 mmHg. Characteristics appear in Table 1.

Table 1. Participant characteristics (N = 14).

Variable	Minimum	Maximum	Mean ± SD
Age (years)	23.0	48.0	38 ± 6.1
Body mass (kg)	45.0	75.0	58 ± 8.7
Height (cm)	148.0	175.0	158 ± 7.9
Resting SBP (mmHg)	90.0	125.0	108 ± 10.5
Resting DBP (mmHg)	57.0	88.0	73 ± 8.2
Resting HR (bpm)	60.0	82.0	74 ± 7.3
Resting MAP (mmHg)	68.0	101.0	84 ± 9.0

SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; MAP, mean arterial pressure.

Resting values did not differ between sessions ($p = 0.193$). The two-way repeated measures ANOVA returned a significant group effect ($F_{1,13} = 4.44$, $p \leq 0.05$) and time effect ($F_{4,10} = 43.17$, $p < 0.0001$). Between session mean differences for each variable are summarized in Table 2, and the change from rest values with effect sizes appear in Table 3. Figure 1 shows the time course of each variable.

Table 2. Between sessions mean differences (EO1 vs EO2) across the recovery period.

Variable	EO1 mean	EO2 mean	Difference	t	p
SBP (mmHg)	111.5	107.8	−3.7 ± 2.4	3.37	**0.028**
DBP (mmHg)	74.4	70.6	−3.8 ± 1.1	8.37	**0.001**
MAP (mmHg)	86.9	83.3	−3.6 ± 1.1	7.12	**0.002**

Variable	EO1 mean	EO2 mean	Difference	t	p
HR (bpm)	84.8	85.6	+0.8 ± 2.5	0.73	0.504

EO1, upper to lower body order; EO2, lower to upper body order. Bold p-values denote statistical significance (p < 0.05).

Systolic blood pressure

EO1 produced no significant change in SBP relative to rest at any time point. After EO2, SBP fell significantly versus baseline at 20 minutes ($p < 0.048$) and 30 minutes ($p < 0.005$), reaching a large effect size at 30 minutes (Table 3). The two sessions differed at the 30 minutes time point (mean difference 6.1 ± 8.7 mmHg, $p = 0.022$), and the overall between session reductions favored EO2 (−3.7 ± 2.4 mmHg, $p = 0.028$; Table 2; Figure 1).

Diastolic blood pressure

After EO2, DBP fell significantly versus baseline at 10 minutes ($p < 0.013$), 20 minutes ($p < 0.013$) and 30 minutes ($p < 0.003$), and the reduction persisted through 30 minutes. Effect sizes for EO2 were moderate to large across recovery and large at 30 minutes. The two sessions also differed at 10 minutes ($p = 0.013$), 20 minutes ($p = 0.007$) and 30 minutes ($p = 0.006$), with EO2 producing a larger fall. The overall session reduction was −3.8 ± 1.1 mmHg in favor of EO2 ($p = 0.001$; Table 2).

Mean arterial pressure

After EO2, MAP fell significantly versus baseline at 20 minutes ($p < 0.002$) and 30 minutes ($p < 0.001$) and reached a large effect size by 30 minutes. The two sessions differed at 1 minute ($p = 0.041$), 20 min ($p = 0.002$) and 30 minutes ($p = 0.002$), and EO2 reduced MAP more than EO1 across the recovery period (−3.6 ± 1.1 mmHg, $p = 0.002$; Table 2).

Heart rate

HR rose above rest after both sessions at 10, 20 and 30 minutes, with no significant between session difference ($p = 0.504$; Table 2) and trivial effect sizes throughout. The elevated HR alongside falling blood pressure is consistent with continued baroreflex activity during recovery (Figure 1).

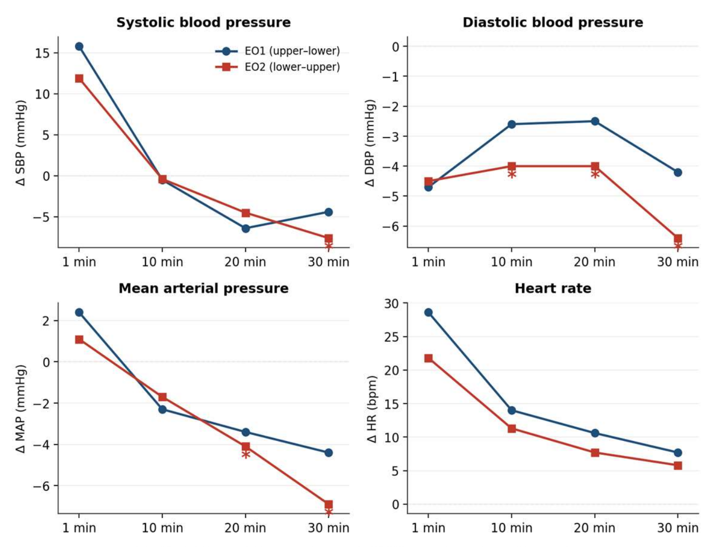

Figure 1. Change from resting baseline in systolic and diastolic blood pressure, mean arterial pressure and heart rate over 30 minutes after each session. EO1 = upper to lower order; EO2 = lower to upper order. Asterisks mark significant between session differences ($p < 0.05$).

Table 3. Resting values and post exercise change (± SD) with effect sizes for EO1 and EO2.

	Rest	1 minutes	10 minutes	20 minutes	30 minutes
EO1					
SBP (mmHg)	110.6 ± 11.1	+15.8 ± 4.1	−0.5 ± 1.3	−6.4 ± 3.2	−4.4 ± 0.7
ES	-	trivial	trivial	medium	small
DBP (mmHg)	77.2 ± 8.5	-4.7 ± 0.3	-2.6 ± 0.0	-2.5 ± 0.5	-4.2 ± 1.7
ES	-	medium	small	small	medium
MAP (mmHg)	88.4 ± 9.2	+2.4 ± 0.1	-2.3 ± 0.2	-3.4 ± 0.9	-4.4 ± 1.0
ES	-	small	small	small	medium
HR (bpm)	72.6 ± 8.8	+28.6 ± 5.0	+14.0 ± 0.9	+10.6 ± 0.0	+7.7 ± 0.2
ES	-	trivial	trivial	trivial	trivial
EO2					
SBP (mmHg)	108.0 ± 10.9	+11.9 ± 2.5	-0.4 ± 1.2	-4.5 ± 2.3	-7.6 ± 3.7
ES	-	trivial	trivial	medium	large
DBP (mmHg)	74.4 ± 7.9	-4.5 ± 0.4	-4.0 ± 0.2	-4.0 ± 0.0	-6.4 ± 0.7
ES	-	medium	medium	medium	large
MAP (mmHg)	85.6 ± 8.7	+1.1 ± 0.8	-1.7 ± 1.2	-4.1 ± 1.2	-6.9 ± 1.1
ES	-	trivial	small	medium	large
HR (bpm)	76.3 ± 7.8	+21.8 ± 4.8	+11.3 ± 1.9	+7.7 ± 0.6	+5.8 ± 2.6
ES	-	trivial	trivial	trivial	trivial

Post-exercise values are changing relative to rest. Effect sizes follow Rhea (2004): trivial ≤ 0.35; small 0.35-0.80; medium 0.80-1.50; large ≥ 1.50.

DISCUSSION

The main finding is that exercise order changed the acute blood pressure response to an otherwise identical strength training session. Beginning with the lower body (EO2) produced a larger and longer post exercise reduction in SBP, DBP and MAP than beginning with the upper body (EO1). For SBP, PEH emerged only after EO2, at 20 and 30 minutes. For DBP, EO2 produced a fall from 10 minutes that lasted 30 minutes, and for MAP the reduction appeared at 20 minutes and continued to 30 minutes. Effect sizes for EO2 reached the large category by 30 minutes for all three pressure variables. This supports the hypothesis that starting with large muscle mass, lower body exercises favours a greater hypotensive response.

Heart rate rose after both sessions and stayed above rest through 30 minutes, with no difference between orders. Falling blood pressure alongside elevated HR fits the pattern reported in a meta analysis of acute resistance exercise trials, where post session reductions in SBP and DBP occurred together with a rise in sympathetic and a fall in parasympathetic activity rather than the reverse (Farinatti et al., 2021). That pattern argues against a simple model in which withdrawal of sympathetic drive produces the hypotension, and points instead toward peripheral vascular mechanisms. Baroreflex sensitivity also takes time to recover after exercise (Niemelä et al., 2008), so the sustained HR at 30 minutes suggests recovery was incomplete and that a longer observation window might have captured further blood pressure decline and the time taken to return to baseline.

The amount of muscle mass recruited offers the most direct explanation for the order effect. Lower body compound lifts engage larger muscle groups than upper body lifts, and greater active muscle mass during resistance exercise is linked to a larger PEH through reduced vascular resistance, partly via nitric oxide mediated increases in blood flow (Polito & Farinatti, 2009). In hypertensive women, resistance sessions that raised plasma nitrite produced clear systolic and diastolic PEH and adding active recovery between sets brought the hypotension on sooner (Martins et al., 2024), which is consistent with a vasodilatory, flow mediated route. A single multi joint session built around large muscle groups was enough to lower pressure in normotensive trained women (Freire et al., 2017). Placing the lower body exercises first, when participants are freshest and can express the highest mechanical output, plausibly drives a stronger vasodilatory and metabolic stimulus that carries into recovery. Training intensity at the start of a session also reduces plasma volume and raises cardiac contractile effort to maintain output (Rezk et al., 2006), which interacts with the peripheral mechanisms that restore pressure during recovery.

These results differ from Figueiredo et al. (2013), who found no PEH when comparing exercise orders in normotensive men and reported that only heart rate variability shifted with order. Sex, training status and exercise selection differ between the studies, and upper limb musculature carries a higher proportion of fast fibers that accumulate metabolites and impose greater cardiovascular stress, which may partly explain the divergent patterns. The present findings align with the broader literature showing that manipulating strength training variables (rest interval, load, volume, set number, muscle mass) reliably alters PEH (De Salles et al., 2010; Figueiredo et al.,2015a, 2015b, 2016; Polito & Farinatti, 2009). They also extend to within session order the observation, in postmenopausal women, that the sequence of exercise within a combined session changes the blood pressure response, with diastolic pressure falling under only one order (Garcês et al., 2025).

Two related studies warrant a careful comparison because their findings are often summarized loosely. Lemos et al. (2018) varied the order of upper body exercises (large to small vs small to large muscle mass) and the rest interval (40 vs 90 s) in trained men, the larger systolic PEH appeared with the 90 second interval, while the 40 second interval raised cardiac stress, so their result speaks to rest interval length more than to upper versus lower sequencing. Bentes et al. (2017) compared reciprocal supersets with traditional training in 13 men and found PEH only after the traditional method, attributing the difference to the greater total work performed. A recent meta-analysis helps reconcile these findings, reporting that across 19 studies, supersets and traditional sets produced comparable blood pressure responses, despite supersets being more time efficient. (Zhang et al., 2025). Read together, these studies suggest that total work and rest interval, rather than the superset format itself, govern the size of the response, which leaves room for exercise order to act through the muscle mass mechanism seen here.

Several limitations apply. The sample was small (N=14) and drawn from active women aged 20 to 50 at a single site, so the findings may not extend to men, to hypertensive populations, or to other age groups. Participants remained seated throughout the 30 minutes recovery, prolonged sitting reduces venous return and raises peripheral resistance, which can blunt or partly mask the diastolic response (Gotshall et al., 1994). Only one of the six manipulable strength training variables (exercise order) was studied. Day to day variation in participants activity and stress, equipment calibration, and the absence of a longer recovery window also constrain interpretation. The 30-minute observation period may have ended before the full hypotensive response resolved.

FUTURE DIRECTIONS

Four lines of work would build on these findings. First, extending the monitoring window with 24 h ambulatory blood pressure would show whether the lower body first advantage persists into daily life rather than fading within the 30 minutes laboratory period, and would capture the full duration of the response. Second, the effect should be tested in the groups most likely to benefit clinically such as prehypertensive and hypertensive women, postmenopausal women, and older adults, alongside men, to establish how far the order effect generalizes. Third, longitudinal training studies are needed to determine whether the acute order effect translates into lower resting blood pressure over weeks of training and whether it resets baroreflex sensitivity, the mechanism linked to chronic blood pressure control. Fourth, mechanistic studies that measure plasma nitrite, vascular conductance, cardiac output and total work alongside blood pressure would separate the contribution of active muscle mass from that of training volume, since the two covary when order is manipulated (Martins et al., 2024; Farinatti et al., 2021).

Two design refinements would also strengthen future trials. Larger samples with the menstrual cycle phase or hormonal status recorded would address a source of variability specific to studies in women, and time efficient session formats such as supersets, which produce a comparable hypotensive response in less time (Zhang et al., 2025), could be compared head to head with traditional ordering to identify the most practical prescription for blood pressure management.

CONCLUSION

In active women, a strength training session that began with lower body compound exercises produced a larger and more sustained post exercise reduction in systolic, diastolic and mean arterial pressure than one that began with upper body exercises. When the goal is an acute blood pressure lowering response, arranging compound exercises from lower to upper body is the preferable order, a low cost change that needs no extra time or equipment. The reduction in MAP after the lower body first session echoes reports that traditional, large muscle mass resistance formats lower mean arterial pressure where time compressed formats may not (Coelho-Júnior et al., 2022).

Beyond its training implications, the work speaks to two SDGs. By identifying a simple, equipment free way to enlarge the blood pressure lowering effect of a single session, it supports SDG 3 (Good Health and Wellbeing) and specifically target 3.4 on cutting premature mortality from non-communicable disease through prevention (United Nations, 2015). By testing the response in women, who remain underrepresented in exercise and cardiovascular research, it also contributes to the evidence base needed to serve women equitably, in line with SDG 5 (Gender Equality). Translating an acute laboratory effect into population benefit will require the longitudinal and clinical studies outlined above.

DECLARATIONS

Ethics. All participants gave written informed consent after being briefed on procedures, risks and benefits.

Funding. This research received no external funding.

Conflicts of interest. The author declares no conflict of interest.

Generative AI And AI-assisted technologies. While preparing this work, the authors used ChatGPT to do paraphrasing and language refinement. After using this tool/service, the authors reviewed and edited the content as needed and took full responsibility for the content of the publication.

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