

1 Arterial stiffness and wave reflection responses following heavy and moderate 2 load resistance training protocols.

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22 **Abstract**

23 This study compared the acute effects of resistance training between a moderate (ML) and a
24 high loading (HL) intensity (12RM vs 4RM, respectively), with the same intensity of effort on
25 arterial stiffness and wave reflection in young healthy adults.

26 Eleven healthy adults (age 36.4 ± 6.8 years) performed two resistance training (RT) protocols,
27 ML and HL, in a randomised order. Both RT sessions consisted of three sets of deadlifts and
28 three sets of bench press, with two minutes rest between sets and exercises. Loading intensity
29 was 12RM and 4RM for the ML and HL condition, respectively. Measurements of pulse wave
30 velocity (PWV) and pulse wave analysis (PWA; e.g., augmentation index) were collected at
31 baseline, immediately post and 15 minutes post training.

32 ML elicited significantly greater increases in carotid-femoral PWV (from 6.4 ± 0.3 to 7.3 ± 0.5),
33 and augmentation index normalised to 75 bpm (from -5.1 ± 1.1) than HL (all $p < 0.05$).

34 These findings demonstrate that an acute bout of RT performed to volitional failure using
35 lower loads and higher repetitions impose a greater workload on the arterial and
36 cardiovascular system in comparison to a RT scheme with heavier loads and lower repetitions.

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50 **1. Introduction**

51 Arterial stiffness (AS) is an independent predictor of cardiovascular disease (CVD) and is
52 strongly associated with hypertension and future cardiovascular events [1]. Regular
53 participation in resistance training (RT) has been endorsed by current guidelines as a
54 promising intervention for the prevention and treatment of CVD [2]. Yet, the effects of RT on
55 AS are not fully understood and remain to be ascertained [3]. Interestingly, increases [4],
56 decreases [5] and no changes [6] in AS have been reported following both acute and chronic
57 RT interventions. Differences in the loading characteristics among the prescribed RT protocols
58 (i.e., loading intensities, proximity to failure) may account for the discrepancies observed [3].

59 Loading intensity is a well-researched training variable particularly in the field of muscular
60 hypertrophy and strength [7], yet there is a paucity of research directly examining the effects
61 of different loading intensities on AS. One of few studies to directly investigate loading and AS
62 compared the effects of heavier [75% - 90% 1-repetition maximum (1RM)] vs lighter load (30%
63 - 50% 1RM) RT on AS and reported a reduction in AS regardless of the load lifted (Au et al.,
64 2017). Elsewhere, Werner et al. (2021) reported no differences in AS after a 12-week RT
65 programme performed using high (80 - 90% 1RM) or moderate (50% - 70% 1RM) loading
66 intensities. Nitzsche et al. (2016) reported acute increases in AS following low (30% 1RM) and
67 moderate (50% 1RM) intensities but not following a higher loading intensity (70% 1RM) in
68 young healthy adults. These findings contrast a recent meta-analysis which suggested that
69 loading intensity is the key variable determining arterial responses to RT [8]. It was noted that
70 low to moderate loading intensities (< 70% 1RM) tend to decrease AS, whereas high loading
71 intensities (> 70% 1RM) seem to increase AS particularly in young individuals [8]. Nonetheless,
72 the lack of control over proximity to failure in previous reports may be a confounding factor
73 when interpreting these findings [6, 9].

74 Proximity to failure operationalised as 'intensity of effort' [10], appears to be a major
75 determinant of cardiovascular [11] and metabolic [12] responses to RT. Notably, loading
76 intensity in the study by Nitzsche et al. (2016) was prescribed in relative terms (i.e., as a
77 percentage of 1RM) with a fixed number of repetitions (i.e., 3 sets of 10 repetitions at 70% of
78 1RM), despite previous evidence suggesting large variability in the number of repetitions that
79 can be performed at given relative load by different individuals [13]. That said, it has recently
80 been suggested that standardisation of effort allows for a more appropriate comparison of
81 other training variables, such as load, since it ensures a more uniform stimulus and establishes
82 parity among the conditions or groups under investigation [14].

83 High and low loading intensities may elicit similar hypertrophic responses [7], however high
84 loads may induce superior neural adaptations [15]. Notably, muscular strength exhibits a
85 stronger inverse relationship with AS than muscle mass alone across a wide age (20 -75y) span
86 [16]. Thus, identifying the potential impact of loading intensity on AS would assist in
87 determining the most effective RT prescription for improved vascular adaptations.

88 To date, there is no study that has directly compared the effects of load on AS while
89 standardising proximity to failure. Thus, the purpose of this study was to compare indices of
90 AS between a strength-type RT scheme using heavier loads (4RM) and lower repetitions with
91 a hypertrophy-type RT protocol using moderate loads and repetitions (12RM) while
92 maintaining intensity of effort constant. Based on the available evidence, it was hypothesised
93 that the high load session would increase AS, whereas the moderate load would reduce or not
94 significantly affect AS.

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96 **2. Methods**

97 **2.1 Participants**

98 Eleven young healthy participants (5 male, 6 female) volunteered to participate in this study.
99 Participants were classified as recreationally active who had been participating in resistance
100 training (e.g., with free weights or weight machines) at least once a week for the last 6 months.
101 All participants were in a good state of health, non-hypertensive and free of any cardiovascular,
102 musculoskeletal, and metabolic disease. All female participants voluntarily reported that they
103 were tested during the follicular phase of their menstrual cycle, although previous research
104 indicates minimal influence of different menstrual cycles on AS measured by cfPWV [17].
105 Participants were not taking any prescribed medications known to affect vascular function
106 during the study period. Prior to participating in the study and after being informed of the
107 study's procedures, all participants provided written informed consent. The study was
108 approved by the Faculty of Health and Wellbeing Research Ethics Committee at the University
109 of Winchester.

110 **2.2 Sample size calculation**

111 Sample size was calculated using G*Power 3.1 (version 3.1.9.7, Heine University, Düsseldorf,
112 Germany). The effect size was estimated based upon a between-group (e.g., heavier vs lighter
113 load) partial eta squared (η_p^2) effect size of 0.114 reported for acute cfPWV in a previous study
114 [18]. Thus, for a repeated measures within factors design, with an effect size $F = 0.35$, a

115 significance level of 0.05, a power of 0.80, and a correlation among repeated measurements
116 of 0.70 [19], a sample size of 11 participants was found to be sufficient to show a difference
117 between higher and lighter load RT conditions, while accounting for a 10% drop-out.

118 **2.3 Study design**

119 Participants took part in a randomised cross over design and reported to the research facility
120 on four separate occasions. The first two visits consisted of anthropometric measurements
121 (i.e., height and body mass), and a familiarisation session in which participants were
122 introduced to the experimental procedures and RM testing, to determine individual 4RM and
123 12RM training loads for the deadlift and bench press exercise. Loading prescription of the two
124 RT sessions was based upon the 'repetition continuum' also known as the 'strength-endurance
125 continuum' [20]. According to the repetition continuum strength gains are maximised by a low
126 repetition strategy with heavy loads (1 to 5 repetitions per set with 80% to 100% of 1RM) while
127 a moderate repetition scheme with moderate loads (e.g., 8–12 repetitions per set at 60%–80%
128 of 1RM) maximises hypertrophic adaptations [21]. In the third and fourth visits, participants
129 were randomly assigned RT protocol using either a 4RM (High Load; HL) or 12RM (Moderate
130 Load; ML). Participants refrained from strenuous physical activity for 24 hours and large meals
131 and caffeine consumption for at least 4 hours prior to testing [22]. The research process is
132 illustrated in Figure 1.

133 **2.4 RM Testing protocol and Experimental sessions**

134 **RM testing protocol (visit 1 and 2)**

135 The maximum amount of weight lifted for 12 and 4 repetitions with proper form in the deadlift
136 and bench press exercise was recorded as the participant's 12RM and 4 RM load respectively.
137 This was determined following the guidelines proposed by the National Strength and
138 Conditioning Association [23]. All RM loads were determined in no more than 5 attempts with
139 rest periods of 3 minutes between the trials.

140 **Experimental sessions (visit 3 and 4)**

141 Two RT sessions comprised the experimental protocol, one for each condition (HL and ML).
142 Both RT sessions consisted of three sets of deadlifts and three sets of bench press. Rest
143 duration was two minutes between sets, and two minutes between exercises, for both
144 conditions. Participants were guided by the researcher to adopt a repetition tempo of
145 approximately 1s concentric, 2s eccentric and 1s isometric pause between concentric and
146 eccentric actions. Intensity of effort was standardized between the experimental conditions

147 and during both sessions exercises were performed to volitional failure [i.e., the exercise was
148 terminated once trainees determined they could not complete further repetitions if attempted
149 [24]]. If a participant reached momentary failure [i.e., the inability to perform another
150 concentric repetition despite attempting to [25]] before performing the expected number of
151 repetitions (i.e., 12 or 4) in a single set, the set was terminated without any further attempts
152 to complete the required number of repetitions. Loading intensity was set at 12RM and 4RM
153 for the ML and HL condition respectively.

154 **2.5 Haemodynamic Measurements**

155 Haemodynamic measurements were collected three times during the experimental
156 procedure: before training (baseline), immediately post (Post) and 15 minutes post training
157 (15Post). All measurements were collected in a supine position. The first haemodynamic data
158 collection was conducted after 10 minutes of rest in a supine position [22]). Following that,
159 participants completed the warm-up routine as described above, and then completed their
160 randomly assigned RT protocol. Participants returned to the supine position immediately post
161 the acute RT protocol while data collection equipment was applied and remained supine until
162 completion of 15Post assessments.

163 All haemodynamic measurements were performed using a validated oscillometric cuff-based
164 device (Vicorder, Skidmore medical, Bristol, UK), that calculates PWV by simultaneously
165 recording the upstroke of the femoral and carotid pulsations [26]. Two inflatable cuffs were
166 used to measure Carotid-femoral PWV (cfPWV), one placed around the neck over the carotid
167 artery and the other around the thigh over the femoral artery. CfPWV was calculated by
168 dividing the pulse wave travel distance by the pulse transit time between the two recording
169 sites. Transit time was determined by the software using an in-built cross-correlation algorithm
170 and travel distance length was defined as the distance from the suprasternal notch to the mid
171 upper thigh cuff, as indicated by the manufacturer. Augmentation index is an indicator of AS
172 which reflects the augmentation of systolic blood pressure by reflection of the peripheral pulse
173 wave. Alx was calculated as the ratio between the augmentation pressure (AP) and the central
174 pulse pressure (Cpp) and was expressed as a percentage ($Alx = AP/Cpp \times 100$). Alx was
175 normalised to 75 beats per minute (bpm) to reduce its reliance on HR ($Alx@75$), as previously
176 suggested [27]. Measurements of subendocardial viability ratio (SEVR), an index representing
177 myocardial perfusion, which is calculated as the ratio between diastolic pressure time index
178 (DPTI) and systolic pressure time index (SPTI) [28]; and of central and peripheral blood

179 pressures were performed using a cuff placed around the upper arm over the brachial artery
180 and assessed using device-specific pulse wave analysis [29].

181 **2.6 Statistical Analysis**

182 All descriptive data are expressed as means \pm standard deviation. Normality of distribution
183 was assessed by the Shapiro-Wilk test and visual inspection. A two-way (Condition x Time)
184 repeated measured analysis of variance (ANOVA) was conducted to evaluate the effects of
185 condition, time and the interaction between them for: cfPWV; Aix; Alx@75; central systolic
186 blood pressure (cSBP); central diastolic blood pressure (cDBP); peripheral systolic (pSBP);
187 peripheral diastolic (pDBP); AP; mean central arterial pressure (cMAP); and SEVR. Violations
188 of sphericity were adjusted for using the Greenhouse-Geisser correction. Post-hoc pairwise
189 comparisons with a Bonferroni correction were conducted when Condition by Time
190 interactions were detected. Analysis of the effect size was conducted using the partial Eta
191 squared (η_p^2). The magnitude of effect size was interpreted as trivial (< 0.01), small (0.01-0.06),
192 moderate (> 0.06-0.14), and large (> 0.14) [30]. Significance was set at $p < 0.05$. Data were
193 analysed using SPSS version 28 statistical software (SPSS Inc, Chicago, IL, USA). A linear mixed-
194 effects model (Jamovi, version 2.3.28) was also employed to evaluate the effect of the
195 intervention on cfPWV (primary outcome), controlling for cMAP as a covariate [22]. Random
196 intercepts were included for each subject to account for repeated measurements over time.
197 Fixed effects in the model included Condition, Time, and the interaction between Condition
198 and Time, while cMAP was included as a covariate to account for its effect on cfPWV. Residual
199 plots were examined to verify model assumptions of normality and homoscedasticity.

200

201 **3. Results**

202 Characteristics of the study participants are displayed in Table 1.

203 There were no significant differences between the experimental conditions at baseline (BL) for
204 any of the variables analysed (all $p > 0.05$).

205 Haemodynamic data are shown in Table 2. Significant Condition and Time interactions were
206 observed for cfPWV ($\eta_p^2 = 0.47$), Alx ($\eta_p^2 = 0.4$), and AP ($\eta_p^2 = 0.42$) (all $p < 0.05$). Post-hoc
207 analysis revealed that changes from BL to Post for cfPWV, Alx and AP were significantly higher
208 in the ML compared to HL. In the ML condition, cfPWV, was significantly greater than BL at
209 both Post and 15Post, but significantly decreased from Post to 15Post ($p < 0.05$). Alx and AP
210 showed a similar trend and were significantly greater than BL at both Post and 15Post following

211 the ML condition (all $p < 0.05$). In the HL condition no significant differences in cfPWV from BL
212 were observed, while Alx and AP significantly increased between BL and Post only. cMAP had
213 no significant main effect on cfPWV ($p = 0.14$), therefore a similar statistical Condition by Time
214 interaction was observed for cfPWV when controlling for cMAP ($p < .001$) (Figure 2). There
215 were no significant Condition by Time interactions for all other variables (Table 2).

216 Main effects of Condition were detected for Alx@75 ($\eta_p^2 = 0.45$), HR ($\eta_p^2 = 0.44$) and SEVR
217 ($\eta_p^2 = 0.49$) (all $p < 0.05$), in which Alx@75 and HR were significantly higher and SEVR was
218 significantly lower in ML compared to HL.

219 Main effects of Time were detected for Alx@75 ($\eta_p^2 = 0.34$), cSBP ($\eta_p^2 = 0.45$), cDBP ($\eta_p^2 =$
220 0.43), pDBP ($\eta_p^2 = 0.41$), HR ($\eta_p^2 = 0.32$) and SEVR ($\eta_p^2 = 0.52$). Alx@75, cSBP, and HR
221 significantly increased from BL to Post. cDBP and pDBP significantly decreased from BL to Post,
222 while SEVR significantly decreased throughout the recovery period ($p < 0.05$). There were no
223 significant Condition by Time interactions, or main effects for pSBP and cMAP (all $p > 0.05$).

224

225 **4. Discussion**

226 This study compared acute changes in indices of AS and central haemodynamics between two
227 RT protocols with a high (HL) or moderate (ML) loading intensity (4RM vs 12RM), while keeping
228 intensity of effort, repetition duration and rest interval constant. Contrary to our hypothesis,
229 ML promoted significantly greater increases in AS and pulse wave reflection, and significantly
230 reduced myocardial perfusion when compared to the HL condition. These findings suggest that
231 an acute bout of RT performed to volitional failure using a moderate loading intensity of 12RM
232 places a greater demand on the arterial and cardiovascular system as opposed to a RT protocol
233 with a higher loading intensity of 4RM.

234 Results of the present study are in partial agreement with a previous study assessing acute AS
235 responses following RT protocols performed at different loading intensities [18]. Similarly to
236 the current study, where cfPWV increased 14% immediately post ML exercise, Nitzsche et al.
237 (2016) reported a 13.8% and an 8% increase in cfPWV immediately post RT consisting of three
238 sets of 30 reps at 30% of 1RM load (i.e., low load) and three sets of 20 reps at 50% of 1RM
239 load (i.e., moderate load), respectively. Likewise, the protocol classified by Nitzsche et al.
240 (2016) as the 'high load' (i.e., 3 sets of 10 reps at 70% of 1RM) did not induce any significant
241 changes in cfPWV, a finding that was also supported in the current study. Contrary to the
242 present study in which all sets were performed to volitional failure in an attempt to standardise

243 effort, intensity of effort was not controlled in the study by Nitzsche et al. (2016) where
244 participants performed a fixed number of 10 repetitions. It has been argued that prescribing
245 an arbitrary number of reps at a relative load based on a percentage of the maximal load an
246 individual can lift (i.e., % of 1RM) limits comparisons within and between RT studies, since
247 individual differences in muscular endurance among the studied population are completely
248 disregarded [14]. Indeed, research has reported a great variability in the number of repetitions
249 that can be performed by different individuals at the same relative load [13], indicating a
250 different intensity of effort among them at the same loading intensity. Considering that
251 cardiovascular responses seem to rely on the effort exerted during RT [31], it could be difficult
252 to say whether differences between conditions in Nitzsche et al. (2016) can be attributed
253 exclusively to the different loads used, since heterogenous levels of effort may have blunted
254 the study's findings.

255 Results of the present study are in contrast to the upheld belief that high loading intensity
256 increases AS. Of note, the ML exercise protocol promoted significantly greater increases in
257 cfPWV, Alx@75, and HR than HL. Specifically, the ML significantly increased cfPWV from BL
258 values at Post (from 6.4 ± 0.3 m/s to 7.3 ± 0.4 m/s) and 15Post (6.8 ± 0.4 m/s). Regardless of
259 load, be it high (>80% 1RM), moderate (60-80% 1RM) or low (<60% 1RM), mixed effects on AS
260 have been reported in the literature following acute RT schemes [4, 5, 18]. Of note, this is the
261 first study investigating the independent effects of load on AS, while maintaining a set of RT
262 variables (e.g., intensity of effort, repetition and rest duration) constant between experimental
263 conditions. Importantly, standardisation of effort adopted in the current study allows for a
264 more appropriate investigation of other RT variables (i.e., load in this instance). This notion is
265 supported by previous meta-analyses examining the effects of single RT variables from a
266 strength/hypertrophy standpoint, as they have only included studies that performed to
267 volitional failure as a means of controlling intensity of effort [7, 32]. Since differences in AS and
268 pulse wave reflection parameters between different loading intensities are not yet fully
269 understood, some speculations can be made based on previous research examining
270 cardiovascular responses between various training loads while having a set of RT standardised
271 between conditions [11, 33]. Vale et al. (2018) reported that RT at high intensity of effort (i.e.,
272 both conditions reps were performed to volitional failure) with lighter loads and higher
273 number of repetitions resulted in greater sympathetic activation in hypertensive
274 postmenopausal women compared to heavier load – lower repetition RT protocol. Similarly,
275 Gjovaag et al. (2016) reported that a 20RM RT session resulted in higher blood-pressure in
276 young healthy adults than a 4RM RT when performed to volitional failure. In accordance,

277 results of the present study indicated a significant greater increase in HR in the ML condition,
278 indicative of sympathetic activation, while cSBP and pSBP also increased to a greater degree
279 in the ML condition, although these changes were not significantly different from the HL.
280 Considering the strong associations between AS with blood pressure and sympathetic activity
281 [34, 35], it can be argued that additional, other than load, RT variables such as volume and
282 time under tension might be related to increased AS following RT [11].

283 Potential mechanisms that might also explain the differences in arterial responses between
284 loading intensities include improved vascular function, through increased nitric oxide (NO)
285 expression and endothelial progenitor cell (EPC) mobilization [36, 37]. Guzel et al. (2007)
286 reported that NO production was greater in healthy males following a high load (80-95% 1RM)
287 compared to a low load (20-35% 1RM) RT session. Elsewhere, Ribeiro et al. (2017) examined
288 the impact of acute RT at several loading intensities (i.e., 60%, 70% and 80% 1RM) on the
289 mobilisation of circulating EPCs, which may indicate improved endothelial function. The
290 authors reported a dose-response relationship, with the highest loading intensities promoting
291 the highest increases in EPCs both immediately and 6 hours after the RT session. Future
292 research is needed to confirm if these findings translate into improvement in AS following RT
293 and to solidify these hypotheses.

294 Data presented herein demonstrated that measures of pulse wave reflection were significantly
295 affected by the RT protocols. Changes in Alx and Alx@75 were significantly greater in ML than
296 HL. Specifically, Alx@75 significantly increased (from -5.1 ± 3.2 to $0.5 \pm 7.6\%$) in ML and (from
297 -5.7 ± 4.3 to $-2.9 \pm 5.8\%$) after the HL session. Such findings are consistent with previous
298 research reporting increases in Alx@75 (from 4.4 ± 8.1 to $23.0 \pm 11.7\%$) following acute RT [4].
299 Nonetheless, increases in Alx@75 have also been observed in time-matched to RT sedentary
300 controls [38] or following acute aerobic and HIIT training [39], despite being well
301 acknowledged that aerobic and HIIT interventions reduce AS [40]. Along this line, it can be
302 speculated that such effects may represent a physiological stress response that may not
303 necessarily translate to harmful long-term adaptations. That assumed, despite raising blood
304 pressure, it has been recently proposed that wave reflections attenuate increases in blood flow
305 that may protect against flow pulsatility to the microcirculation [41]. Yet, this is still speculative
306 so future studies are needed to validate this notion.

307 In the present study, cSBP significantly increased post-exercise. These findings are in
308 agreement with some [38, 42] but not all previous studies [4, 43]. In addition, the current data
309 did not show any changes in pSBP in any time point for both conditions, supporting previous

310 assumptions that cSBP responses occur independent of changes in pSBP and AS [44]. Of note,
311 cDBP significantly decreased immediately post-exercise. Exact causes of this decrease remain
312 unclear, as it is usually shown that that cDBP remains unchanged up to 10 minutes after acute
313 RT [4, 38], although significant decreases have also been reported [42]. A similar trend was
314 observed for SEVR which significantly decreased after both conditions throughout recovery.
315 Given that myocardial perfusion occurs mostly during diastole, it has been suggested that a
316 reduction in cDBP may result in a decrease in coronary blood flow [45]. Although previous
317 studies reporting decreases in SEVR values after acute RT [4, 38] have not observed any parallel
318 reductions in cDBP. Furthermore, since the extent of myocardial perfusion is primarily
319 determined by DPTI, which in turn is mediated by increases in HR, it should be noted that
320 increases in HR post-exercise were observed, and although significantly different from
321 baseline, these increases were moderate with mean values reaching at 75.5 ± 16 bpm. Given
322 the paucity of available data, future research is needed to elucidate potential relationships and
323 to ascertain whether these transient changes in cDBP and SEVR may represent harmful long-
324 term implications. Nevertheless, data from the current study indicate that RT performed at
325 higher loading intensities promote a more favourable effect on coronary haemodynamics,
326 potentially representing a safer RT approach especially for populations at risk.

327

328 Some limitations of the current study warrant consideration. It should be noted that volume
329 load (i.e., set x repetitions x weight lifted) of ML was twice that of HL. Although volume load
330 could have been equated between conditions, lighter loads inherently result in greater volume
331 load than heavier load RT protocols [46]. Thus, whilst scientifically valid, this approach would
332 have limited ecological validity and transfer to real training settings. For the same reason, the
333 Valsalva manoeuvre was not controlled in this study despite its association with AS [47], since
334 use of it may be unavoidable and often beneficial when lifting heavy loads or performing
335 repetitions close to momentary failure [48]. In addition, in the case of some participants the
336 specific repetition tempo prescribed could not be adopted during the final repetitions.
337 Nonetheless, repetition velocity is usually reduced as a set approaches the point of failure,
338 indicating the interdependence of RT variables [32]. Lastly, the current sample consisted of
339 both males and females, thus sex-related differences in vascular responses to RT might be a
340 confounding factor. Despite these limitations, the current study adequately controlled a set of
341 RT variables (i.e., intensity of effort, rest duration) to investigate the independent effects of
342 load on AS. In addition, the present study was structured as a within-subjects design thus
343 minimising inter-individual variability in physiological responses to RT[49].

344 **5. Conclusion**

345 The current study suggests that performing RT to volitional failure using a moderate loading
346 intensity (12RM) promotes significantly greater increases in measures of AS and pulse wave
347 reflection in healthy individuals when compared to a high loading intensity 4RM. Collectively,
348 these data suggest that RT protocols with lower loads and higher repetitions impose a greater
349 workload on the arterial and cardiovascular system in comparison to RT schemes with heavier
350 loads and lower repetitions. Findings of the present study may have important implications for
351 the prescription of RT programs particularly from a cardiovascular-health point of view. Future
352 research should examine the potential implication of additional RT variables, such as intensity
353 of effort, under volume equated conditions, in an attempt to elucidate the independent and
354 synergistic effects of various RT variables on AS.

355

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360 The authors reported no conflict of interest relevant to this manuscript.

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364 **DATA AVAILABILITY STATEMENT**

365 The data collected and analysed for this study are available from the corresponding author
366 upon request.

367 **ETHICS STATEMENT**

368 The study was approved by the Faculty of Health and Wellbeing of the University of Winchester
369 (HWB_REC_230811_Karanasios). Experimental procedures were conducted following the
370 approved ethics submission document. All participants received written information
371 explaining the procedures and purpose of the study and gave their written consent prior to
372 data collection.

373 **AUTHOR CONTRIBUTIONS**

374 Conceptualization: EK; Writing/original draft preparation :EK; Investigation/testing: EK; Data
375 analysis: EK, JF, SH; Review and editing: JF, SH, HR-S. All authors have read and approved the
376 final version of the manuscript and agree with the order of presentation of the authors.

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543 **Table 1** Participant characteristics

	ML (12RM)	HL (4RM)
Age (y)	36.4 ± 6.8	
Weight (kg)	67.3 ± 12.0	
Height (cm)	172.8 ± 7.8	
Dead Lift (kg)	56.6 ± 15.0	76.8 ± 16.6
Bench Press (kg)	40.6 ± 10.9	52.4 ± 15.8
Volume (Repetitions)	71.2 ± 0.8	23.8 ± 0.4
Volume load (kg)	3419.6 ± 884.8	1540.6 ± 384.4

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545 Note: DL: deadlift; BP: Bench press; ML: moderate load; HL: high load. Volume load is
546 calculated as : sets x repetitions x load. Data presented are mean ± SD.

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566 **Table 2** Haemodynamic and cardiovascular variables at rest and during recovery from an
 567 acute ML (12RM) and HL (4RM) resistance training protocol.

	Time Point			Interaction Effect	
	Baseline	Post	15Post	P for Interaction effect	Effect size (η^2_p)
cSBP (mmHg) §					
ML	111.4 ± 11.0	119.9 ± 10.2	111.3 ± 14	0.239	0.13
HL	112.7 ± 10.1	117.6 ± 14.9	115.7 ± 15.6		
<i>Total</i>	112 ± 9.9	118.7 ± 9.9	113.5 ± 13.6		
cDBP (mmHg) §					
ML	60.5 ± 8.5	47.8 ± 6.6	50.2 ± 11.0	0.180	0.15
HL	60.3 ± 8.6	56.6 ± 12.5	56.8 ± 11.7		
<i>Total</i>	60.3 ± 8	52.2 ± 7.3	53.5 ± 8.3		
cfPWV (m/s)					
ML	6.4 ± 0.3	7.3 ± 0.5	6.9 ± 0.4	0.002	0.47
HL	6.6 ± 0.5	6.8 ± 0.3	6.6 ± 0.2		
<i>Total</i>	6.5 ± 0.3	7 ± 0.3	6.7 ± 0.3		
cfPWV-adjusted (m/s)					
ML	6.4 ± 0.3	7.3 ± 0.3	6.9 ± 0.3		
HL	6.6 ± 0.3	6.7 ± 0.3	6.6 ± 0.3	<0.001	
<i>Total</i>	6.5 ± 0.3	7 ± 0.3	6.7 ± 0.3		
AIx (%)					
ML	12.5 ± 5.4	32.5 ± 10.7	17.2 ± 6.2	0.018	0.40
HL	14.3 ± 7.3	23 ± 7.9	14.8 ± 8.9		
<i>Total</i>	13.3 ± 5.6	27.7 ± 7.6	16 ± 7		
AIx@75 (%) †.§					
ML	-5.1 ± 3.3	0.6 ± 7.7	-2.0 ± 6.1	0.210	0.14
HL	-5.7 ± 4.3	-3.0 ± 5.9	-4.3 ± 4.7		
<i>Total</i>	-5.4 ± 3.6	-1.1 ± 6	-3.1 ± 5		
AP (mmHg)					
ML	6.5 ± 3.2	23.5 ± 9.6	11 ± 7.3	0.004	0.42

<i>HL</i>	7.5 ± 3.9	14.2 ± 6.2	9.5 ± 7.5		
<i>Total</i>	6.9 ± 3	18.8 ± 6.3	10.2 ± 6.3		
pSBP (mmHg)					
<i>ML</i>	118.4 ± 8.8	123.2 ± 10.1	116.4 ± 13		
<i>HL</i>	119.5 ± 8.9	121.3 ± 14.1	122.8 ± 12.7	0.131	0.18
<i>Total</i>	118.9 ± 8	122.2 ± 9.3	119.5 ± 11.3		
pDBP (mmHg) §					
<i>ML</i>	60.5 ± 8.5	47.8 ± 6.6	50.2 ± 11		
<i>HL</i>	59.8 ± 8.8	57.4 ± 13.2	56.8 ± 11.7	0.130	0.18
<i>Total</i>	60.1 ± 8.3	52.5 ± 7.6	53.5 ± 8.3		
HR (bpm) †,§					
<i>ML</i>	64.1 ± 6.8	75.5 ± 16.0	70.5 ± 12.6		
<i>HL</i>	62.8 ± 9.0	68.4 ± 12.2	65.8 ± 9.7	0.236	0.13
<i>Total</i>	63.4 ± 7.3	71.9 ± 12.9	68.1 ± 10.6		
MAP (mmHg)					
<i>ML</i>	83 ± 10.8	82.4 ± 8.0	79.6 ± 10.3		
<i>HL</i>	83.5 ± 9.7	85.1 ± 13.1	82.5 ± 12.2	0.782	0.02
<i>Total</i>	83.2 ± 9.9	83.7 ± 8.6	81.1 ± 10.3		
SEVR (%) †,§					
<i>ML</i>	160.6 ± 18.8	128.5 ± 26.9	131.1 ± 36.2		
<i>HL</i>	158.3 ± 18.9	146.8 ± 27.5	138.8 ± 20.2	0.058	0.24
<i>Total</i>	159.4 ± 17.2	137.6 ± 26.5	134.9 ± 26.2		

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569 Note: ML: moderate load; HL: high load; cSBP: central systolic blood pressure; cDBP: central
 570 diastolic blood pressure; cfPWV: carotid-femoral pulse wave velocity; cfPWV-adjusted:
 571 cfPWV adjusted for mean arterial pressure; Aix: augmentation index; AP: augmentation
 572 pressure; pSBP: peripheral systolic blood pressure; pDBP: peripheral diastolic blood pressure;
 573 cMAP: central mean arterial pressure; HR: heart rate; SEVR: subendocardial viability ratio;
 574 Aix@75: augmentation index normalised at 75bpm; Total: means of main effect of time. Data
 575 are displayed as means \pm SD; \ddagger interaction main effect ($p < 0.05$), \dagger condition main effect ($p <$
 576 0.05), \ddagger time main effect ($p < 0.05$).

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