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1 Original Research Article

2 JOURNAL OF PHYSIOLOGY AND BIOCHEMISTRY

3 **Resistance Training Status Modifies Inflammatory Response to**
4 **Explosive and Hypertrophic Resistance Exercise Bouts**

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26

27 Abstract

28

29 The purpose of the present study was to examine the immediate and prolonged immune response in
30 circulating cytokine and adipocytokine concentrations after two different resistance exercise bouts:
31 hypertrophic (HYP1, 5 x 10, 80 % of 1RM) and maximal explosive (POW1, 10 x 5, 60 % of 1RM)
32 resistance exercise bouts and how 12 weeks of resistance training (RT) modifies these responses
33 (HYP2, POW2). Eight men completed the study. RE-induced interleukin-6 (IL-6), interleukin-1 β (IL-
34 1 β), interleukin-1 receptor antagonist (IL-1ra), monocyte-chemoattractant protein-1 (MCP-1), leptin,
35 resistin, and adiponectin were measured before (PRE) and immediately (POST0), 24 (POST24) and 48
36 (POST48) hours after RE bouts before and after RT. In the untrained state IL-6 increased immediately
37 after RE in HYP1 ($p = 0.002$) and in POW1 ($p = 0.003$) whereas no changes were observed after RT.
38 Similar results were observed in IL-1 β , whereas conversely IL-1ra increased only after RT in HYP2
39 and POW2 ($p < 0.05$). Resistin increased before RT in HYP1 and in POW1 ($p = 0.011$ and $p = 0.003$,
40 respectively), but after RT significant responses were not observed. Interestingly, in HYP2 MCP-1
41 increased significantly at POST24 ($p = 0.009$) and at POST48 ($p = 0.032$) only following RT. The
42 present study shows that RT modifies RE-induced cytokine responses towards an anti-inflammatory
43 direction.

44

45

46

47 Keywords: initial response; hypertrophic resistance exercise; power; cytokines; inflammation

48 **INTRODUCTION**

49

50 Skeletal muscle is the largest organ in the human body and ground-breaking work during the last
51 decade has demonstrated that skeletal muscle is an active endocrine organ releasing a host of cytokines
52 [1]. In recent years, studies reporting the acute effects of exercise on cytokines have been published
53 quite extensively, whereas the specific time-course of the effect of different resistance exercise
54 protocols on cytokines remains unclear [2]. Cytokines are glycoproteins involved in the regulation and
55 modulation of the immune response and they are produced by a broad range of cells, including immune
56 cells, skeletal muscle, connective tissue, and adipose tissue cells [3]. Resistance exercise (RE) is a
57 potent activator of the immune system demonstrated by the changes in circulating pro-inflammatory
58 and anti-inflammatory cytokine concentrations after exercise bouts [4]. For instance, a single bout of
59 resistance exercise of moderate to high intensity has been shown to promote a transient increase in pro-
60 inflammatory interleukins IL-6 and IL-1 β as well as in the circulating levels of C-reactive protein
61 (CRP), which is associated with a later increase in the levels of the anti-inflammatory IL-1 receptor
62 antagonist (IL-1ra) [4, 5]

63

64 Far less is known about the effects of resistance exercise bout on so-called adipocytokines, e.g. leptin,
65 adiponectin, and resistin. Adipocytokines are hormones that were first discovered to be secreted by
66 adipose tissue and to regulate both energy metabolism and appetite. More recent findings on the
67 ubiquitous expression of their receptors and on their cellular effects have revealed that adipocytokines
68 also are involved in the regulation of a variety of biological functions related to immune responses and
69 inflammatory diseases [6]. Previous studies have demonstrated that a single heavy resistance exercise
70 bout exerts a specific acute effect on circulating adipocytokine concentrations and the immediate
71 response appears to be dependent on the duration and intensity of the exercise, as well as on training
72 status and background [7-9].

73

74 Previous studies have suggested that exercise training can significantly alter the acute inflammatory
75 responses to high intensity resistance exercise and recovery processes after the resistance exercise bout.
76 Murton et al. [10] highlighted that the response to the first RE bout in participants with no background
77 in resistance training (RT) is significantly different and has more inter-subject variability compared to

78 the second resistance exercise. Izquierdo and colleagues [4] reported a significantly greater
79 inflammation-responsive cytokine IL-6 response followed by a significantly enhanced response in the
80 anti-inflammatory IL-1ra after a heavy resistance training intervention compared to the response before
81 training. Cross-sectional studies have also suggested that training background affects the acute cytokine
82 responses to a RE bout [9]. Thus, the overall effects of long-term RT appears to attenuate the acute
83 inflammation response but there are mixed findings on the effect of RT on specific markers [11].

84

85 It has been proposed that the acute anti-inflammatory (immunosuppressive) effect following a bout of
86 resistance exercise could be beneficial for patients with autoimmunity disease, for senior citizens, and
87 for obese individuals [3]. It is important to identify the specific resistance exercise-induced changes in
88 circulating cytokines as well as the longitudinal effect of resistance training on immediate and
89 prolonged responses after specific resistance exercise bouts in order to better understand possible
90 health benefits, but also the possible health hazards, related to resistance training [12]. Pedersen &
91 Febbraio [13] have linked skeletal muscle contraction to cytokine production. The extent of
92 inflammation response to resistance exercise is affected by the physiological demands of RE,
93 depending on the mode (eccentric and/or concentric muscular contractions), volume (total work of the
94 session), load (weight lifted), and intensity (extent of neuromuscular and metabolic fatigue) of
95 resistance exercise [14]. Many types of resistance exercises can be effectively used to improve
96 muscular fitness and overall health [15]. Muscle strength and the ability of the muscles to develop force
97 rapidly are important performance characteristics, which have also been shown to contribute to health
98 and several tasks of daily life [16]. Since the cytokine responses appear to be related to the intensity of
99 the exercise protocol, it is of interest to examine the cytokine responses of exercise protocols that aim
100 for gains in muscle mass and strength [17], as well as in rapid force development [18, 19].

101

102 If the cytokine responses are related to the amount of muscle mass activated and respective metabolic
103 changes, it is expected that the greatest responses will be observed after hypertrophic resistance
104 exercise. However, in explosive RE, muscles are also activated maximally but with a shorter duration
105 of each repetition, accompanied by a lower metabolic response. Thus, it is not clear whether this type
106 of stimulus is large enough to cause considerable hormonal changes. We expected that the hypertrophic
107 resistance exercise (HYP, 5 x 10, 80 % of 1RM) would induce a significant response to the variables

108 measured [4, 7]. The hormonal responses to a maximal explosive (POW, 10 x 5, 60 % of 1RM) RE
109 bout have been shown to be similar as in HYP but with a lower magnitude [18, 19]. Thus, we expected
110 to observe a significant but lower response in the measured variables after POW RE bout. However,
111 several studies have showed that hormonal responses to same hypertrophic [20] as well as explosive
112 [21] RE are modified by 7-21 weeks of resistance training. Hence, we found it justified to assess
113 whether the inflammatory response is modified after progressive resistance training. Therefore, the
114 purpose of the present study was to examine the immediate and prolonged immune response, by
115 measuring circulating cytokine and adipocytokine concentrations, induced by two different resistance
116 exercise bouts: hypertrophic (HYP, 5 x 10, 80 % of 1RM) and maximal explosive (POW, 10 x 5, 60 %
117 of 1RM) resistance exercise bouts. In addition, we measured how typically preiodized 12 weeks of RT
118 possibly modified these responses.

119

120

121 **MATERIALS AND METHODS**

122

123 **Subjects.** This study was a part of a larger research project (TEKES Finland, Decision No. 70007/13).
124 Eight healthy, slightly overweight, young men were selected for this study (age 31.0 ± 0.9 years, body
125 weight 84.6 ± 1.9 kg, height 1.78 ± 0.04 m, fat percentage 25.3 ± 7.1 %). All participants were
126 physically active on a weekly basis, but none were competitive athletes or had a background in
127 systematic strength training. The subjects' physical activity was characterized by walking, cycling or
128 occasionally participating in team sports at light to moderate intensity and a frequency of $3 \text{ d}\cdot\text{wk}^{-1}$.
129 Participants filled in a health questionnaire prior to participation in the study. All subjects reported that
130 they were non-smokers, free from injury, and were not using any medications. Each subject was
131 informed of the potential risks and discomforts associated with the measurements, and all the subjects
132 gave their written informed consent to participate. The study was conducted according to the
133 Declaration of Helsinki, and ethical approval for the study procedures were granted by the Ethical
134 Committee at the University of Jyväskylä and by the Ethical Committee of the Central Hospital,
135 Jyväskylä.

136 **Study design and experimental resistance exercise bouts.** The study design (A) and experimental RE
137 protocols (B) are presented in Figure 1. The first phase of the study was a four-week long preparatory

138 RT period, during which the subjects were familiarized to RT and underwent pretesting. Subsequently
139 the cross-over study was started and the subjects were randomly assigned to perform HYP1 or POW1
140 RE bout first and then after ten days of recovery, POW1 or HYP1, respectively. Thereafter, they
141 trained for 12 weeks according to supervised progressive RT protocol and did the same RE bouts with
142 the same order as before training (HYP2/POW2). The POW RE bout included 10 sets of 5 repetitions
143 of the concentric phase as fast as possible at 60% of 1 repetition maximum (RM) and the HYP RE bout
144 was 5 sets of 10 repetitions at 80% of 1RM for the leg press (David D210 horizontal leg press device,
145 David Health Solutions Ltd., Helsinki, Finland) exercise. The loads used during the first set were
146 determined from the 1RM load measured during pretesting. The loads were adjusted during the
147 sessions to enable completion of the required repetitions. The inter-set rest period was three minutes in
148 POW and two minutes in HYP. The duration of POW was 32 minutes; whereas HYP was completed in
149 20 min. Total volume (load \times repetitions) in was 7160 ± 272 kg and 7550 ± 427 kg in POW and HYP,
150 respectively. All experiments were conducted at the same time of day (± 1 h) for each subject.

151

152 ***Figure 1 somewhere here***

153

154 **Pretesting.** All subjects participated in a pre-test session, which included anthropometrics and body
155 composition measurement, as well as the 1RM test performed in the leg press device. This bilateral
156 1RM test was used to determine the loads used in each acute RE bout. Three warm-up sets (5×70 –
157 75% , 3×80 – 85% , and 2×90 – 95% of estimated 1RM) with 1 minute of rest between sets were
158 performed before the 1RM trials. Upon verbal instruction, subjects performed a full leg extension (knee
159 angle 180°) from a starting knee angle of below 60° . After each successful completion, the load was
160 increased. Subjects were allowed a maximum of 5 trials. The trial with the highest completed load was
161 accepted as the 1RM.

162 **Training.** Subjects were asked to maintain individual habitual physical activity (e.g., light walking,
163 cycling, and occasional team sports) throughout the study period. All prescribed training in the study
164 was consistently supervised by qualified instructors. The training was designed to reflect a program
165 **designed** for physically active populations according to recommendations outlined by the American
166 College of Sports Medicine [15]. The detailed training program (hypertrophic-strength-group) has been
167 previously reported by Hulmi et al. [22]. Briefly, in the preparatory 4 week's RT period before POW1 /

168 HYP1, see above, the subjects exercised using whole-body workouts two times per week to standardize
169 training status, to minimize the effects of stressors related to unaccustomed exercise, and to overcome
170 strong neural and learning adaptations known to occur within the first few weeks of RT. Training loads
171 were 50–80 % of one repetition maximum (1 RM) increasing throughout the preparatory RT period.

172 The **veritable** 12-week progressive RT program was started after POW1 / HYP1 and divided into three
173 different blocks. Each block consisted of four weeks of RT. The first block consisted only of
174 hypertrophic RE sessions. In the second block 75 % of the sessions were hypertrophic and 25 % were
175 maximal-strength sessions, and in the last block 25 % were hypertrophic and 75 % maximal-strength
176 RE sessions. The subjects did on average nine exercises in each session, 2–3 sets per exercise. Bilateral
177 leg press, bilateral knee extension, and bilateral knee flexion exercises were performed during each RE
178 session.

179 **Body composition.** Whole body composition was estimated before and after RT by Dual X-ray
180 Absorptiometry (DXA, LUNAR Prodigy, GE Medical Systems) after an overnight fast and 48 hours
181 without training. Abdominal fat was calculated manually defining a range of interest confined cranially
182 by the upper end plate of the first lumbar vertebra, laterally by the ribs and caudally by the iliac crest
183 [23] at PRE. This customized range was then copied to the DXA scans at wk4 and wk16, respectively.

184 **Nutrition.** Dietary intake was recorded over three weekdays and one weekend day during the training
185 period. The subjects were instructed to follow the same diet before all the acute exercise bouts. The
186 breakfast before RE bouts was standardized and served at the laboratory.

187 **Subjective muscle Soreness.** Muscle Soreness was rated on a visual analogic scale (VAS) of 0 (= no
188 pain) to 100 (= maximum pain) in millimeters for the overall muscle soreness of the quadriceps
189 muscles at PRE, POST24, and POST48.

190 **Blood samples and analyses.** Blood lactate was measured to determine the metabolic effect of work
191 performed in RE bouts. Blood samples were obtained from the fingertip and collected into capillary
192 tubes (20 µL), which were placed in a 1 mL hemolyzing solution and analysed automatically after the
193 completion of testing according to the manufacturer's instructions (EKF diagnostic, C-line system,
194 Biosen, Germany). To assess the immediate and prolonged (up to 48 hours) impact from exercise
195 protocols, blood samples were collected pre-exercise (PRE) and during recovery as follows:
196 immediately (POST0), 24 hours (POST24) and 48 hours (POST48) after the exercises.

197

198 Venous blood samples were drawn from the antecubital vein into EDTA tubes (Venosafe, Terumo,
199 Belgium). Haemoglobin and hematocrit were determined with Sysmex KX-21N (TOA Medical
200 Electronics Co., Ltd., Kobe, Japan) and plasma volume change was determined after exercise at POST0
201 from haemoglobin and hematocrit concentrations using the equation by Dill & Costill [24]. Plasma
202 glucose (GLU) was measured using the KONELAB 20XTi analyser (Thermo Fisher Scientific Inc.,
203 Waltham, MA, USA).

204

205 The serum samples were held for 15 min at room temperature before being centrifuged for 10 min at
206 2000 x g (Megafuge 1.0 R, Heraeus, Germany). The serum was kept at -80°C until analysed. High-
207 sensitivity C-Reactive Protein (hsCRP), Creatine Kinase (CK), cortisol (COR), and interleukin -1 beta
208 (IL- β) in serum samples were analysed from using the Immulite 1000 and immunoassay kits (Immulite,
209 Siemens, IL, USA). The detection limits and inter-assay coefficients of variation, respectively, were 0.1
210 mg·L⁻¹ and 10 % for hsCRP, 3.9 pg·mL⁻¹ and 5.9 % for CK, 5.5 nmol·L⁻¹ and 7.9% for COR and 1.5
211 pg·mL⁻¹ and 2.8 % for IL-1 β .

212

213 The EDTA-treated samples were centrifuged for 10 min at +4°C with 2000 x g (Megafuge 1.0 R,
214 Heraeus, Germany). The plasma was kept at -80°C until analysed. Concentrations of interleukin-6 (IL-
215 6), monocyte chemoattractant protein-1 (MCP-1), interleukin-1 receptor antagonist (IL-1ra),
216 adiponectin, leptin, and resistin were determined by enzyme-linked immunosorbent assay (ELISA)
217 with commercial reagents (R&D Systems, Europe Ltd, Abingdon, UK). The detection limits and inter-
218 assay coefficients of variation, respectively, were 0.2 pg·mL⁻¹ and 1.8 % for IL-6, 3.9 pg·mL⁻¹ and 5.0 %
219 for MCP-1, 31.3 pg·mL⁻¹ and 2.0 % for IL-1ra, and 0.78 ng·mL⁻¹ and 2.2 % for adiponectin, 15.6
220 pg·mL⁻¹ and 4.0 % for resistin and 15.6 pg·mL⁻¹ and 5.1 % for leptin.

221

222 **Statistical Analyses.** Conventional statistical methods were used for the calculation of means and
223 standard deviations and standard errors. Before applying further statistical methods, the data was
224 checked for sphericity and normality. If a specific variable violated the assumptions of parametric tests
225 then log-transformation was used. For IL-6, adiponectin and leptin the log-transformation provided
226 sufficient remedy for normality or homogeneity of variance. Absolute changes were analyzed via two-
227 way repeated analysis of variance for time (PRE, POST0, POST24, POST48), training (before, after)

228 and interaction (time \times training) effects. This was followed by one-way repeated measures ANOVA on
229 each RE bout to examine a main effect of time. If a main or interaction effect was observed at $p \leq 0.05$,
230 the change from pre-values to POST0, POST24 and POST48 was compared between type or time using
231 paired t-tests. Effect sizes (ES) are given as Cohen's d with an effect size of 0.20-0.50 being considered
232 small, 0.50-0.80 medium, and >0.80 large. Data was analyzed using PASW statistic 18.0 (SPSS,
233 Chicago, IL, USA). The level of statistical significance was set at $p \leq 0.05$.

234

235 **RESULTS**

236

237 Descriptive statistics of the anthropometric characteristics and 1RM are presented in Table 1. After RT
238 whole-body fat-free mass was increased significantly ($p < 0.05$), whereas body weight, whole-body fat
239 mass and abdominal fat mass stayed unaltered after RT. 1RM increased significantly (+13%, $p=0.032$).
240 Table 2 shows that RT significantly ($p<0.05$) suppressed the immediate increases in lactate and glucose
241 and prolonged those of creatine kinase and muscle soreness induced by HYP. A significant increase in
242 cortisol was observed before RT in HYP ($p < 0.001$), whereas after RT this was not observed. POW
243 reduced average circulating concentrations before and after RT, but the decrease was statistically
244 significant only after RT ($p<0.05$).

245

246 ***Table 1 somewhere here***

247

248 ***Table 2 somewhere here***

249

250

251

252

253 Effects of HYP and POW RE on the circulating levels of inflammatory markers before and after RT
254 intervention shown in Table 3. The most notable changes were seen in MCP-1 and resistin (Figure 1).
255 A significant time \times training effect was observed in circulating MCP-1 concentration in HYP ($p =$
256 0.002 , $ES = 0.924$) (Figure 1a): after RT in HYP2 a significant increase in MCP-1 was observed during
257 recovery at POST24 ($p = 0.009$) and at POST48 ($p = 0.032$). An increasing trend in circulating MCP-1
258 concentration was seen also at POST24 and POST48 in POW2 but that effect did not reach statistical
259 significance.

260

261 There was a significant main effect of time ($p=0.005$, $ES = 0.560$) and time \times training ($p = 0.028$, $ES =$
262 0.732) in circulating resistin levels in POW (Figure 1b). A significant increase in resistin concentration
263 from PRE to POST0 was observed before RT in POW1 ($p = 0.003$, $ES = 0.997$), however after RT in
264 POW2 such significant response was not observed ($p = 0.102$). In HYP, a similar effect, although
265 smaller in quantity, in circulating resistin concentration was found: there was an increase in HYP1 from

266 PRE to POST0 before RT ($p = 0.011$, $ES = 0.789$) whereas resistin stayed statistically unaltered by
267 HYP2 after RT ($p = 0.248$) (Figure 1b).

268

269 ***Figure 2 somewhere here***

270

271 Neither of the RE bouts, before or after RT, elicited significant changes in circulating CRP
272 concentrations (Table 3). IL-6 response was significantly affected by RT in HYP (time \times training
273 interaction, $p = 0.048$, $ES = 0.534$) and in POW (time \times training interaction, $p = 0.013$, $ES = 0.808$). A
274 significant increase in IL-6 was observed at POST0 in HYP1 ($p = 0.002$, $ES = 0.719$) and in POW1 (p
275 $= 0.003$, $ES = 0.878$) before training whereas no effect was observed after training (Table 3).

276

277 A significant main effect of time in circulating IL-1 β was observed in HYP ($p = 0.022$, $ES = 0.775$) and
278 in POW ($p=0.043$, $ES=0.496$) (Table 3). Significant increases from PRE to POST0 were observed in
279 IL-1 β in HYP1 before RT ($p = 0.019$, $ES = 0.795$) whereas after RT statistically significant changes
280 were not observed. Similarly in POW1 before RT a significant increase was observed in circulating IL-
281 1 β concentration from PRE to POST0 ($p = 0.048$), whereas after RT significant changes were not
282 observed.

283

284 Circulating IL-1ra remained unaltered before RT in HYP1 and in POW1 but after RT the circulating
285 IL-1ra concentration increased significantly from PRE to POST0 in HYP2 ($p = 0.048$) and in POW2 (p
286 $= 0.024$), see Table 3.

287

288 Following RT the circulating leptin concentrations were significantly higher at PRE before both
289 loadings. In HYP1 a significant reduction in circulating leptin concentration was observed at POST24
290 ($p<0.05$) and in HYP2 at POST0 ($p<0.05$). Pre-exercise circulating adiponectin concentration was
291 significantly higher after RT in HYP2 ($p = 0.048$) and in POW2 ($p = 0.026$) and stayed unaltered after
292 RE bouts.

293 **Table 3 somewhere here**

294

295

296 **DISCUSSION**

297

298 The purpose of the present study was to examine the immediate and prolonged immune response, by
299 measuring circulating cytokine and adipocytokine concentrations, induced by two different resistance
300 exercise bouts: hypertrophic (HYP, 5 x 10, 80 % of 1RM) and maximal explosive (POW, 10 x 5, 60 %
301 of 1RM) resistance exercise bouts, and how 12 weeks of RT may modify these responses. We
302 expected HYP RE to elicit greater responses in the selected cytokines. However, the differences
303 between the loadings were cytokine-specific. Nevertheless, the pro- and anti-inflammatory responses
304 were modified differently: The pro-inflammatory IL-6, IL-1 β and resistin response was blunted,
305 whereas anti-inflammatory IL-1ra response was enhanced in both HYP and POW as a consequence of
306 training. Interestingly, the prolonged MCP-1 response was enhanced after RT in HYP, possibly as a
307 marker of muscle regeneration. The present data also demonstrated that progressive RT increases
308 muscle strength and alters immediate and prolonged response of cytokine and adipocytokine
309 concentrations to HYP and POW similarly. Interestingly, RT affected responses to POW similarly even
310 though the training did not include explosive type of training. This could indicate that the effect of
311 training on immediate prolonged cytokine response is more dependent on overall training status than on
312 specific RE training background.

313

314 Regular exercise reduces the risk of chronic metabolic and cardiorespiratory diseases, and this
315 reduction has been linked to the anti-inflammatory effect of exercise [25]. It has been suggested that
316 this anti-inflammatory effect of exercise is mediated via the introduction of an anti-inflammatory
317 environment following each bout of endurance or resistance exercise [25]. The present study showed
318 increased IL-6 and IL-1 β levels immediately post-exercise before RT but not after RT, whereas
319 circulating IL-1ra increased only after RT in both resistance exercise bouts. This modified IL-6
320 response is in-line with the study by Izquierdo et al.[4], which showed exercise-induced IL-6 response
321 only in the initial phase of resistance training. Contracting skeletal muscle has been proposed to be the
322 main source of increased IL-6 in circulation during and following exercise but also connective tissue,
323 brain, and adipose tissue contribute to the exercise-induced increased IL-6 levels [1]. IL-6 production
324 in the muscle cells is increased when glycogen is compromised suggesting that IL-6 has a role as an
325 energy sensor in the exercising muscle; and IL-6 has been shown to enhance basal and insulin-

326 stimulated glucose uptake in muscle cells and to its favorable effects on energy metabolism, and its
327 anti-inflammatory effects, IL-6 has also pro-inflammatory effects in inflammatory diseases and in
328 connection to obesity and metabolic syndrome. Therefore, the observed adaptation of the IL-6 response
329 to heavy exercise induced by resistance training as observed in the present study can be regarded as a
330 beneficial form of acclimatization considering that exercise training has also been found to increase IL-
331 6 receptor expression and IL-6 sensitivity in skeletal muscle [26]. The present study also demonstrated
332 a significant increase in IL-1 β concentrations before RT in HYP and POW, whereas after RT the
333 response was blunted. IL-1 has been reported to enhance the secretion of hypothalamic corticotropin-
334 releasing factor, which further stimulates glucocorticoid release [20]. In the present study, the increase
335 in IL-1 β was accompanied by an increase in cortisol before RT in HYP, and neither were increased
336 following RT supporting a possible link between those two. Nevertheless, in POW we did not observe
337 a significant cortisol response before RT, however, a significant increase in IL-1 β was observed. To
338 summarize, in the present study after RT, a blunted IL-6 response, increased IL-1ra concentration, and
339 suppressed IL-1 β response were observed regardless of the type of RE bout. These observations lead to
340 a hypothesis that RT enhances anti-inflammatory effects and that might suppress the immediate pro-
341 inflammatory response at the cellular level. Such an anti-inflammatory response within the circulation
342 may provide positive metabolic changes through increased fat oxidation and glucose uptake [13, 27].
343 In-line with the observed responses in HYP, the present study observed a significant increase in blood
344 glucose before RT, whereas after RT blood glucose response was suppressed and a similar blunted
345 response was observed in cortisol.

346

347 MCP-1 is a potent chemotactic and activating factor for macrophages, inflammation, and skeletal
348 muscle regeneration [28]. MCP-1 response has been studied mostly after eccentric exercise [29, 30]. To
349 our knowledge, only two other studies have examined changes in plasma MCP-1 after acute traditional
350 RE. Our previous study found a significant decrease 30 minutes after hypertrophic RE [7] whereas
351 Wells and colleagues [31] reported a significant increase in circulating MCP-1 immediately following
352 damaging RE. The present study added longer tracking into recovery with two measurement points to
353 the existing data; namely 24 and 48 hours after the present RE. Interestingly, MCP-1 significantly
354 increased after resistance training in HYP 24 hours after the RE. Hubal et al. [30] showed that MCP-1
355 mRNA levels were significantly elevated after muscle-lengthening lower body exercise and the

356 response was enhanced in repeated bouts. Furthermore, the immunohistochemistry analysis in their
357 study showed that MCP-1 was localized with resident macrophages and satellite cell populations,
358 which link MCP-1 to muscle regeneration. Skeletal muscle has an intrinsic protective mechanism to
359 adapt after muscle damaging exercise to resist future muscle damage [32]. Deyhle et al. [32] measured
360 intracellular MCP-1 after initial lengthening contraction and 27 days after the first bout and suggested
361 that the muscle or the immune system becomes sensitized to the initial bout of damaging exercise such
362 that inflammatory cell infiltration into the muscle is enhanced upon a repeated bout of damaging
363 exercise. In the present study a significant level of muscle damage, as assessed by CK increase, was
364 observed after all RE bouts, which could be one of the mechanisms that lead to increased MCP-1,
365 however, the mechanisms behind the enhanced MCP-1 response after RT remains unclear. The present
366 study demonstrates that not only the initial response to unfamiliar resistance exercise differs from the
367 later responses but also that RT significantly affects the MCP-1 response.

368

369 Adipocytokines are released from the adipose tissue and have been associated strongly not only with
370 metabolism but also with inflammation [33]. Previous studies have reported no effect [34] or acute
371 decreases [7, 9] on circulating resistin concentration following RE. Interestingly, regardless of the lack
372 of total or abdominal fat loss, there was a beneficial significant increase in pre-exercise leptin and
373 adiponectin concentrations. The present study observed a significant immediate increase in circulating
374 resistin concentration immediately after both HYP and POW bouts before but not after RT. This is in-
375 line with the observation by Varady and colleagues [9] that resistance training background modifies the
376 resistin response. However, they reported a significant reduction after RE bout in resistin in the
377 subjects with RT background whereas no significant response was observed in participants with
378 sedentary and running background. The mechanism of the resistin response has been hypothesized to
379 be related to metabolic demand of exercise, which could explain the blunted response in HYP.
380 However, our data does not fully support this hypothesis as the lactate after RT immediately after HYP
381 was an average of $10.1 \pm 3.4 \text{ mmol}\cdot\text{L}^{-1}$ and no significant response in resistin levels was observed
382 whereas in POW before RT with the average lactate of $3.0 \text{ mmol}\cdot\text{L}^{-1}$ a significant increase in resistin
383 levels was observed. Hence, in addition to the metabolic stress other mechanisms have to be involved.
384 Skeletal muscle cells have been shown to release resistin [35]. In addition, mechanical stress has been
385 shown to enhance the expression of resistin in cardiomyocytes [36]. Thus, one origin of increased

386 resistin concentration could be the skeletal muscle as it has been shown with IL-6 [1]. Another
387 mechanism could be related to the loading of joints. It has been shown that adipokines are produced
388 also in joints and have a role in joint diseases such as osteoarthritis [37]. Vuolteenaho et al. [38]
389 reported a significant effect of marathon running on circulating resistin concentrations and one may
390 hypothesize that it could be related to the cartilage degradation or strenuous muscle exercise.
391 Interestingly, in the present study a significant immediate increase in resistin concentration was
392 observed only before training in both resistance exercise bouts. Especially maximal explosive
393 resistance exercise bout characterized by explosive muscle contraction, produces stress on tendons and
394 joints [39]. While speculative, the blunted resistin response after RT may suggest that RT could elicit
395 protective mechanisms in cartilage, which could be observed as a reduced immediate resistin response
396 to resistance exercise bout.

397

398 Limitations of this study should be noted. The most notable limitation of the present study was the
399 small sample size. In addition, the mechanisms by which resistance training alters the inflammatory
400 response to an acute resistance exercise bouts remain to be explored. Serial blood samples and
401 additional muscle biopsies are needed to investigate the series of events initiated by resistance exercise
402 bouts on the cytokine kinetics after traditional RE bouts. Hence, the present study cannot go further
403 than to state that the acute pro- and anti-inflammatory response is altered by resistance training towards
404 an anti-inflammatory direction. It is also possible that the nutritional status of the participants had an
405 effect on the acute cytokine responses as the nutrition on the day before the RE bout was not strictly
406 controlled [13]. It is notable that the exercise protocols in this study were intense, which must be taken
407 into account when evaluating the present results. The present study showed that the pro-inflammatory
408 response in novice trainers is blunted by the resistance training. Generally, a repetition range between 8
409 and 12 RM is used for novice training and 1-12 RM in a periodized fashion for individuals with
410 resistance training experience are recommended [15]. Our findings support the recommendation.
411 However, to ensure optimal health and fitness gains, resistance training should be undertaken with
412 proper preparation, guidance and surveillance.

413

414

415 **Conclusion**

416

417 12-weeks of resistance training blunted IL-6, IL-1 β and resistin responses in the circulation in both
418 HYP and POW, whereas the response of the IL-1ra was enhanced. In addition, enhanced MCP-1
419 response in HYP was observed only after the RT intervention. This study emphasizes the importance of
420 reporting training background when investigating immediate cytokine and adipocytokine responses
421 after resistance exercise. The improvement in the anti-inflammatory and the blunted pro-inflammatory
422 response achieved in the present study by resistance training may be an effective means for reducing
423 systemic low-grade inflammation and thus improve the future health trajectory of young men.

424

425

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433

434 **Conflict of interest statement**

435 The authors report no conflicts of interest. The authors alone are responsible for the content and writing
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437

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TABLE LEGENDS

Table 1. Anthropometric characteristics of the participants. (mean \pm SD) *=significant difference to pre-training value $p < 0.05$.

Table 2. Lactate (Lac), creatine kinase (CK), muscle soreness (DOMS), glucose and cortisol responses to hypertrophic (HYP) and explosive (POW) RE before and after the 12-week resistance training (RT) intervention. *=significant difference to pre-exercise value in the corresponding RE bout, #=significant difference to corresponding value before resistance training in the corresponding RE bout (mean \pm SD * $p < 0.05$, ** $p < 0.001$, *** $p < 0.0001$, # $p < 0.05$).

Table 3. High-sensitive C-reactive protein (hs-CRP), Interleukin-6 (IL-6), Interleukin 1 beta (IL-1 β), Interleukin-1 receptor antagonist (IL-1ra), adiponectin, leptin and resistin responses to hypertrophic (HYP) and maximal explosive (POW) resistance exercises (RE). A significant time \times training interaction was observed in IL-6 in HYP ($p=0.048$) and in POW ($p=0.013$).*=significant difference to pre-exercise value in the corresponding RE bout, #=significant difference to corresponding value before resistance training in the corresponding RE bout (mean \pm SD * $p < 0.05$, ** $p < 0.001$, *** $p < 0.0001$, # $p < 0.05$).

LEGENDS TO THE FIGURES

FIGURE 1. Study design (A) and experimental protocols (B). 1RM=repetition maximum, POW=power and HYP=hypertrophic resistance exercise bout, RT=resistance training, PRE = before loading, POST0 = immediately after exercise, POST 24H = 24 hours after the loading, POST48H = 48 hours after the loading.

FIGURE 2. MCP-1 (2a, 2b) and resistin (2c, 2d) responses to hypertrophic (HYP, left) and explosive (POW, right) resistance exercises before (HYP1/POW1) and after (HYP2/POW2) RT (mean \pm SD). A significant time \times training interaction was observed in MCP-1 in HYP ($p=0.002$) and in resistin in POW ($p=0.028$). (* $p<0.05$, ** $p<0.01$, *** $p<0.001$ when compared to pre value in the corresponding RE bout).

Table 1.

| Variable | Week 0 | Week 12 | Δ -% |
|---------------------------|-----------------|------------------|-----------------|
| Body weight (kg) | 84.6 \pm 5.09 | 83.8 \pm 4.85 | -0.8 \pm 2.9 |
| Height (m) | 1.78 \pm 0.04 | 1.78 \pm 0.04 | - |
| BMI (kg·m ⁻²) | 26.8 \pm 1.37 | 26.6 \pm 1.24 | -0.2 \pm 0.9 |
| Body fat mass (kg) | 21.6 \pm 6.8 | 19.8 \pm 5.8 | -6.6 \pm 13.8 |
| Abdominal fat mass (kg) | 2.9 \pm 1.5 | 2.8 \pm 1.0 | -3.2 \pm 6.4 |
| Fat-free mass (kg) | 59.6 \pm 0.53 | 61.6 \pm 0.63* | 3.3 \pm 0.3* |
| 1 RM (kg) | 225 \pm 34.5 | 255 \pm 30.1* | 13.8 \pm 7.6 |

Table 2

| | | PRE | POST0 | POST24 | POST48 |
|-----------------------------------|---------|-----------|----------------|---------------|------------|
| HYP RE | | | | | |
| Lac (mmol·L ⁻¹) | Pre RT | 1.5 ± 0.4 | 13.0 ± 4.1*** | na | na |
| | Post RT | 2.2 ± 0.5 | 10.1 ± 3.4***# | na | na |
| CK (pg·mL ⁻¹) | Pre RT | 250 ± 230 | 280 ± 730 | 362 ± 240*** | 239 ± 120 |
| | Post RT | 140 ± 36 | 150 ± 360# | 279 ± 150***# | 229 ± 91* |
| DOMS (mm) | Pre RT | 2.4 ± 6.4 | na | 42 ± 19** | 44 ± 22** |
| | Post RT | 0.9 ± 1.5 | na | 33 ± 25*# | 22 ± 25# |
| Glucose (mmol·L ⁻¹) | Pre RT | 5.1 ± 0.4 | 6.2 ± 0.4** | na | na |
| | Post RT | 5.3 ± 0.6 | 5.6 ± 0.5# | na | na |
| Cortisol (nmol·mL ⁻¹) | Pre RT | 400 ± 170 | 562 ± 130*** | 380 ± 140 | 330 ± 110 |
| | Post RT | 400 ± 120 | 450 ± 120# | 470 ± 110 | 450 ± 140 |
| POW RE | | | | | |
| Lac (mmol·L ⁻¹) | Pre RT | 1.9 ± 0.9 | 3.0 ± 1.1* | na | na |
| | Post RT | 2.2 ± 0.9 | 3.1 ± 1.8 | na | na |
| CK (pg·mL ⁻¹) | Pre RT | 150 ± 75 | 140 ± 27 | 310 ± 100** | 240 ± 75* |
| | Post RT | 130 ± 80 | 110 ± 59 | 330 ± 94** | 250 ± 65** |
| DOMS (mm) | Pre RT | 1.3 ± 2.4 | na | 20 ± 23 | 10 ± 12 |
| | Post RT | 3.9 ± 5.7 | na | 22 ± 21 | 14 ± 25 |
| Glucose (mmol·L ⁻¹) | Pre RT | 5.7 ± 1.0 | 5.6 ± 1.3 | na | na |
| | Post RT | 5.3 ± 0.7 | 5.7 ± 0.3 | na | na |
| Cortisol (nmol·mL ⁻¹) | Pre RT | 370 ± 49 | 310 ± 70 | 420 ± 120 | 428 ± 93 |
| | Post RT | 410 ± 58 | 340 ± 55* | 490 ± 120 | 371 ± 130 |

Table 3.

| | | PRE | POST0 | POST24 | POST48 |
|------------------------------------|---------|--------------|--------------|--------------|--------------|
| HYP RE | | | | | |
| hs-CRP (mg·L ⁻¹) | Pre RT | 1.17 ± 0.47 | 1.27 ± 0.53 | 1.55 ± 0.63 | 1.35 ± 0.64 |
| | Post RT | 1.21 ± 0.50 | 1.43 ± 0.56 | 1.46 ± 0.43 | 1.66 ± 0.33 |
| IL-6 (pg·mL ⁻¹) | Pre RT | 0.99 ± 0.24 | 1.67 ± 0.45* | 1.10 ± 0.19 | 1.27 ± 0.38 |
| | Post RT | 1.11 ± 0.32 | 1.27 ± 0.29 | 1.30 ± 0.36 | 1.57 ± 0.40 |
| IL-1ra (pg·mL ⁻¹) | Pre RT | 373 ± 60.1 | 395 ± 78.8 | 350 ± 79.8 | 380 ± 107.1 |
| | Post RT | 370 ± 74.7 | 425 ± 92.2* | 315 ± 49.5 | 364 ± 43.0 |
| IL-1β (pg·mL ⁻¹) | Pre RT | 1.79 ± 0.38 | 2.63 ± 0.58* | 2.15 ± 0.48 | 2.47 ± 0.54 |
| | Post RT | 1.51 ± 0.51 | 2.13 ± 0.60 | 2.52 ± 0.92 | 2.50 ± 0.99 |
| Adiponectin (pg·mL ⁻¹) | Pre RT | 4.00 ± 0.86 | 4.47 ± 1.13 | 3.98 ± 0.92 | 4.78 ± 0.99 |
| | Post RT | 4.50 ± 0.71# | 4.35 ± 0.64 | 4.56 ± 0.89 | 4.33 ± 0.70 |
| Leptin (pg·mL ⁻¹) | Pre RT | 7.21 ± 1.57 | 7.78 ± 1.96 | 5.81 ± 1.23* | 7.57 ± 1.45 |
| | Post RT | 8.73 ± 2.04# | 7.98 ± 1.92* | 8.62 ± 1.87# | 8.39 ± 1.60# |
| | | PRE | POST0 | POST24 | POST48 |
| POW RE | | | | | |
| hs-CRP (mg·L ⁻¹) | Pre RT | 0.89 ± 0.32 | 0.89 ± 0.37 | 1.42 ± 0.60 | 1.72 ± 0.73 |
| | Post RT | 1.52 ± 0.60 | 1.42 ± 0.52 | 1.18 ± 0.33 | 1.22 ± 0.37 |
| IL-6 (pg·mL ⁻¹) | Pre RT | 1.03 ± 0.28 | 1.64 ± 0.48* | 1.20 ± 0.31 | 1.03 ± 0.51 |
| | Post RT | 1.14 ± 0.23 | 1.33 ± 0.27 | 1.20 ± 0.31 | 0.98 ± 0.21 |

| | | | | | |
|------------------------------------|---------|--------------|---------------|--------------|--------------|
| IL-1ra (pg·mL ⁻¹) | Pre RT | 398 ± 94.9 | 386 ± 74.8 | 350 ± 60.9 | 381 ± 74.9 |
| | Post RT | 341 ± 55.2 | 403 ± 64.9* | 344 ± 52.8 | 330 ± 53.3 |
| IL-1β (pg·mL ⁻¹) | Pre RT | 1.28 ± 0.20 | 2.90 ± 0.88** | 1.84 ± 0.49 | 1.56 ± 0.30 |
| | Post RT | 2.17 ± 0.40 | 1.72 ± 0.46 | 2.03 ± 0.68 | 1.22 ± 0.21 |
| Adiponectin (pg·mL ⁻¹) | Pre RT | 3.85 ± 0.88 | 4.69 ± 0.73 | 4.22 ± 0.99 | 4.17 ± 0.93 |
| | Post RT | 4.72 ± 0.67# | 4.49 ± 0.72 | 4.87 ± 0.86 | 4.48 ± 0.69 |
| Leptin (pg·mL ⁻¹) | Pre RT | 6.5 ± 1.70 | 6.63 ± 1.49 | 6.81 ± 1.56 | 7.47 ± 1.72 |
| | Post RT | 8.93 ± 2.17# | 7.88 ± 2.09# | 9.33 ± 2.48# | 8.47 ± 1.86# |

Figures

