Acute impact of submaximal resistance exercise on immunological and hormonal parameters in young men

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Acute impact of submaximal resistance exercise on immunological and hormonal parameters in young men

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In this study, we examined the acute effects of submaximal resistance exercise on immunological and hormonal parameters in 7 resistance-trained and 10 non-resistance-trained males. The participants, who were aged 29.5 ± 7.1 years (mean ± s), performed submaximal resistance exercise at 75% of their one-repetition maximum. Blood samples were taken before, during, immediately after, and 30, 60 and 120 min after exercise and analysed for leukocyte subpopulations and stress hormones. Total leukocytes, neutrophils and monocytes increased during exercise, reaching their maximum 2 h after exercise. Lymphocytes increased during exercise, T-helper cells returned to resting values after exercise, and natural killer cells and T-suppressor cells decreased below resting values. The CD4/CD8 ratio decreased during exercise but increased during recovery. The resistance-trained participants tended to have lower T-helper cell counts before, during and immediately after exercise and a lower CD4/CD8 ratio during recovery than the non-resistance-trained participants. Plasma cortisol correlated positively with leukocytes during exercise (r=0.572, P<0.05), but negatively with T-helper cells 30 and 60 min after exercise (r=-0.573, P<0.05; r=-0.642, P<0.01, respectively). Our results indicate that resistance exercise leads to acute changes in leukocyte counts, despite moderate hormonal changes, independent of training status. Regular resistance exercise might lead to decreased T-helper cell counts and a lower CD4/CD8 ratio, which could increase susceptibility to infections.

Keywords: cortisol, leukocyte, noradrenaline, recovery, resistance exercise, submaximal exercise.

Introduction

Although physical exercise is known to have many beneficial effects in humans (Sato, 2000; Boreham and Riddoch, 2001), several studies have reported that physical exercise can induce changes in immunological parameters (Nieman et al., 1994; Gleeson et al., 1995a; Shinkai et al., 1996; Bishop et al., 1999; Shephard and Shek, 1999). Depending on exercise mode, intensity, duration and recovery, immunosuppression can occur in athletes and might contribute to a higher incidence of upper respiratory tract infections (Nieman et al., 1998). Most researchers have used endurance exercise to examine exercise-induced immune modulation. Only a few studies have been published regarding the acute or chronic effects of resistance exercise on immunological parameters (Nieman et al., 1995b; Kraemer et al., 1996; Rall et al., 1996; Miles et al., 1999). However, these studies did not consider recovery time (Kraemer et al., 1996) or were designed especially to induce muscle damage (Miles et al., 1999) or to meet the needs of a particular patient group (Rall et al., 1996). Some research has used unusual exercise loads – for example, 8–10 sets of one exercise – which do not reflect practical exercise conditions (Nieman et al., 1995b; Kraemer et al., 1996). The immune modulatory effects of a submaximal resistance exercise circuit have not previously been investigated. Submaximal resistance exercise is recommended for maintaining health, for older and obese adults, for individuals with rheumatoid arthritis and osteoporosis (American College of Sports Medicine, 1993, 1998a,b; Beyer et al., 1996) and is performed regularly by many recreational athletes. Despite the lower oxygen demands compared with endurance exercise, resistance exercise also induces muscle damage, oxidative stress (McBride et al., 1998) and hormonal changes, all of which are known to modulate the immune system (Nieman, 1994; Shek et al., 1995; Kraemer et al., 1996; Suzuki et al., 1996). The aims of this study were two-fold: (1) to examine the acute impact of submaximal resistance exercise on

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immunological and hormonal parameters during exercise and recovery; and (2) to assess the responses of resistance-trained and non-resistance-trained individuals to submaximal resistance exercise.

**Methods**

**Participants**

Altogether, 17 males (7 resistance-trained, 10 non-resistance-trained) aged 29.5 ± 7.1 years (mean ± s) were included in the study. The non-resistance-trained participants had not performed resistance exercise or weightlifting at regular intervals for 6 months or more before the study. Five members of the non-resistance-trained group reported that they were sedentary; the other five reported that they performed endurance exercise (i.e. running, cycling) 2–3 times a week. The members of the resistance-trained group had performed resistance exercise or weightlifting at least three times a week for 6 months or more before the study. Five resistance-trained participants also reported that they took part in endurance exercise 2–3 times a week. The participants were informed about the aims, nature and potential risks of the study and provided written informed consent to take part. The study protocol was approved by the Ethics Committee of the Medical Faculty, University of Vienna. The characteristics of the participants are shown in Table 1.

**Experimental protocol**

One week before testing began, the participants were shown the 10 exercises of the resistance exercise circuit (bench press, leg press, latissimus dorsi pull, leg extension, shoulder press, triceps exercise, ‘crunch’, vertical row, biceps curl and pull-up) and their one-repetition maximum (1-RM) for each exercise was determined (Table 2). For the ‘crunch’ exercise, three-quarters of the repetitions recorded were performed in the main experiment.

For the main experiment, the participants visited the resistance exercise circuit at 07.30 h after an overnight fast and having abstained from alcohol for 24 h. They were asked not to perform any exercise in the 48 h before the experiment. After a warm-up on a bicycle ergometer (15 min at 75 W), each participant began the submaximal resistance exercise circuit at the defined intensity until they became exhausted. The 10 non-resistance-trained and five of the resistance-trained participants completed the circuit twice; the other two members of the resistance-trained group completed the circuit three times. On average, the resistance-trained participants completed 2.3 circuits. Average exercise time (without warm-up) for the non-resistance-trained group was 36.6 min (range 34–41 min); that for the resistance-trained group was 43.1 min (range 35–56 min). Recovery time between the different exercise stations was 1 min. Blood samples were drawn from an antecubital vein using a catheter at the following times: 30 min before exercise, after the first completed circuit, immediately after exercise, and 30, 60 and 120 min after exercise (Fig. 1).

**Biochemical analysis**

Venous blood was collected into heparin-containing vacuum tubes (Vacuette; Greiner, Vienna, Austria). White blood cell counts (neutrophils, lymphocytes and monocytes) and haematocrit were measured using a blood analyser in the Department of Haematology at the General Hospital in Vienna. The determination of lymphocyte subpopulations [T-helper cells (CD3+ and

**Table 1.** Characteristics of the participants (mean ± s)

<table>
<thead>
<tr>
<th></th>
<th>NRT (n = 10)</th>
<th>RT (n = 7)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>28.2 ± 3.9</td>
<td>31.3 ± 10.2</td>
<td>0.394</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.82 ± 0.05</td>
<td>1.82 ± 0.08</td>
<td>0.975</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>81.5 ± 6.0</td>
<td>82.9 ± 10.0</td>
<td>0.729</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>18.6 ± 5.0</td>
<td>14.5 ± 4.5</td>
<td>0.111</td>
</tr>
</tbody>
</table>

**Abbreviations:** RT = resistance-trained, NRT = non-resistance-trained.

**Table 2.** Group differences in one-repetition maximums (kg; mean ± s)

<table>
<thead>
<tr>
<th>Exercise</th>
<th>NRT (n = 10)</th>
<th>RT (n = 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bench press</td>
<td>52 ± 9</td>
<td>111 ± 12</td>
</tr>
<tr>
<td>Leg press</td>
<td>118 ± 22</td>
<td>168 ± 27</td>
</tr>
<tr>
<td>Latissimus dorsi pull</td>
<td>65 ± 10</td>
<td>114 ± 12</td>
</tr>
<tr>
<td>Crunch *</td>
<td>31 ± 8</td>
<td>56 ± 9</td>
</tr>
<tr>
<td>Triceps exercise</td>
<td>34 ± 6</td>
<td>82 ± 8</td>
</tr>
<tr>
<td>Biceps curl</td>
<td>40 ± 9</td>
<td>72 ± 12</td>
</tr>
<tr>
<td>Pull-up</td>
<td>47 ± 7</td>
<td>101 ± 9</td>
</tr>
<tr>
<td>Vertical row</td>
<td>63 ± 13</td>
<td>109 ± 18</td>
</tr>
<tr>
<td>Leg extension</td>
<td>64 ± 14</td>
<td>98 ± 25</td>
</tr>
<tr>
<td>Shoulder press</td>
<td>40 ± 7</td>
<td>82 ± 8</td>
</tr>
</tbody>
</table>

**Abbreviations:** RT = resistance-trained, NRT = non-resistance-trained.

*Maximal number of repetitions.
Values were expressed in nmol l\(^{-1}\) plasma. The noradrenaline measurements were made in duplicate. The intra-assay coefficient of variation for this method was 5.1%.

**Statistical analysis**

Leukocyte counts were corrected for changes in haematocrit. The group data were tested for normal distribution. Normally distributed groups were compared using independent samples t-tests and Bonferroni correction. Correlations were reported using Pearson’s correlation coefficient and time effects were examined using repeated-measures analysis of variance with the statistical software SPSS/PC 10.0 (SPSS Inc., Chicago, IL). Statistical significance was set at \( P < 0.05 \).

**Results**

Haematocrit changed over time and was significant for the resistance-trained group (Table 3). The effect of time was significant for most leukocytes (Table 3). Changes over time were also observed when the values were adjusted for haematocrit. There was no interaction between training status and time. All leukocytes increased during exercise. In particular, natural killer cell counts increased dramatically (\( \sim 250\% \)) above baseline values; T-helper cell counts increased by only a moderate amount (\( \sim 20\% \)). Neutrophil and monocyte counts remained elevated during recovery, reaching their highest values 2 h after exercise. Lymphocyte counts increased during exercise but fell slightly below baseline values during recovery; this could be attributed to a reduction in the number of T-suppressor (CD8\(^+\)) cells and natural killer cells, but not T-helper (CD4\(^+\)) cells. Due to the redistribution of T-helper and T-suppressor cells, the CD4/CD8 ratio was lower during exercise but higher during recovery.

Noradrenaline concentration increased during exercise; during recovery, it remained slightly elevated above resting values. The changes over time were significant for both groups of participants (Table 3). Noradrenaline concentration correlated with natural killer cell counts during exercise (\( r = 0.630, P = 0.028 \)) and with a decrease in circulating numbers of monocytes after 30 min of recovery (\( r = -0.762, P = 0.004 \)). The response of cortisol to exercise was inconsistent between individuals; therefore, the average cortisol concentration was not significantly affected during exercise or recovery, although a decrease over time was observed (Table 3). Nevertheless, cortisol correlated with haematocrit-corrected cell counts. Cortisol concentration correlated with the increase in total leukocyte counts during exercise (\( r = 0.572, P = 0.026 \)).
<table>
<thead>
<tr>
<th>Groups</th>
<th>Before</th>
<th>During</th>
<th>Immediately after</th>
<th>30 min after</th>
<th>60 min after</th>
<th>120 min after</th>
<th>Time effect</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haematocrit (%)</td>
<td>NRT</td>
<td>40.5±0.9</td>
<td>44.1±1.6</td>
<td>41.9±1.5</td>
<td>42.1±2.9</td>
<td>40.7±2.6</td>
<td>43.4±4.9</td>
<td>0.086</td>
</tr>
<tr>
<td></td>
<td>RT</td>
<td>41.9±2.0</td>
<td>43.8±1.5</td>
<td>43.0±1.8</td>
<td>40.6±1.9</td>
<td>40.1±1.6</td>
<td>40.3±3.2</td>
<td>0.001</td>
</tr>
<tr>
<td>Leukocytes (×10⁶ ml⁻¹)</td>
<td>NRT</td>
<td>5.50±1.70</td>
<td>8.09±2.89</td>
<td>6.99±2.09</td>
<td>5.91±1.95</td>
<td>6.92±1.87</td>
<td>10.08±3.37</td>
<td>0.013</td>
</tr>
<tr>
<td></td>
<td>RT</td>
<td>4.68±1.11</td>
<td>6.66±1.77</td>
<td>6.63±1.49</td>
<td>5.17±0.66</td>
<td>5.68±1.19</td>
<td>7.98±2.48</td>
<td>0.021</td>
</tr>
<tr>
<td>Neutrophils (×10⁶ ml⁻¹)</td>
<td>NRT</td>
<td>2.82±1.04</td>
<td>3.96±1.63</td>
<td>3.72±1.29</td>
<td>3.41±1.17</td>
<td>4.34±1.50</td>
<td>6.95±3.41</td>
<td>0.023</td>
</tr>
<tr>
<td></td>
<td>RT</td>
<td>2.45±0.93</td>
<td>3.64±1.36</td>
<td>3.81±0.98</td>
<td>3.10±0.63</td>
<td>3.62±1.10</td>
<td>5.50±2.53</td>
<td>0.058</td>
</tr>
<tr>
<td>Lymphocytes (×10³ ml⁻¹)</td>
<td>NRT</td>
<td>1.92±0.51</td>
<td>3.04±0.85</td>
<td>2.33±0.57</td>
<td>1.71±0.59</td>
<td>1.74±0.68</td>
<td>2.01±0.86</td>
<td>0.001</td>
</tr>
<tr>
<td></td>
<td>RT</td>
<td>1.61±0.31</td>
<td>2.26±0.96</td>
<td>2.14±0.82</td>
<td>1.50±1.4</td>
<td>1.45±0.10</td>
<td>1.78±0.33</td>
<td>0.122</td>
</tr>
<tr>
<td>T-helper cells (×10³ ml⁻¹)</td>
<td>NRT</td>
<td>808±261</td>
<td>1027±300</td>
<td>861±266</td>
<td>793±312</td>
<td>849±348</td>
<td>910±433</td>
<td>0.212</td>
</tr>
<tr>
<td></td>
<td>RT</td>
<td>596±112</td>
<td>691±142</td>
<td>644±94</td>
<td>626±107</td>
<td>621±102</td>
<td>733±215</td>
<td>0.280</td>
</tr>
<tr>
<td>T-suppr. cells (×10³ ml⁻¹)</td>
<td>NRT</td>
<td>437±162</td>
<td>668±333</td>
<td>509±244</td>
<td>381±146</td>
<td>375±143</td>
<td>428±187</td>
<td>0.008</td>
</tr>
<tr>
<td></td>
<td>RT</td>
<td>416±152</td>
<td>554±274</td>
<td>519±251</td>
<td>388±58</td>
<td>359±36</td>
<td>454±117</td>
<td>0.208</td>
</tr>
<tr>
<td>CD4/CD8 ratio</td>
<td>NRT</td>
<td>1.96±0.53</td>
<td>1.76±0.61</td>
<td>1.88±0.63</td>
<td>2.20±0.68</td>
<td>2.36±0.70</td>
<td>2.20±0.66</td>
<td>0.002</td>
</tr>
<tr>
<td></td>
<td>RT</td>
<td>1.58±0.61</td>
<td>1.47±0.64</td>
<td>1.47±0.60</td>
<td>1.66±049</td>
<td>1.74±0.33</td>
<td>1.66±0.66</td>
<td>0.045</td>
</tr>
<tr>
<td>NK cells (×10³ ml⁻¹)</td>
<td>NRT</td>
<td>218±142</td>
<td>626±337</td>
<td>424±196</td>
<td>143±91</td>
<td>112±66</td>
<td>166±75</td>
<td>0.001</td>
</tr>
<tr>
<td></td>
<td>RT</td>
<td>247±84</td>
<td>581±339</td>
<td>517±303</td>
<td>190±81</td>
<td>166±33</td>
<td>198±47</td>
<td>0.019</td>
</tr>
<tr>
<td>Monocytes (×10³ ml⁻¹)</td>
<td>NRT</td>
<td>475±168</td>
<td>699±260</td>
<td>599±253</td>
<td>536±246</td>
<td>622±288</td>
<td>878±345</td>
<td>0.010</td>
</tr>
<tr>
<td></td>
<td>RT</td>
<td>416±141</td>
<td>545±173</td>
<td>480±131</td>
<td>422±98</td>
<td>445±163</td>
<td>571±213</td>
<td>0.188</td>
</tr>
<tr>
<td>Noradrenaline (nmol l⁻¹)</td>
<td>NRT</td>
<td>1.33±0.57</td>
<td>2.13±0.91</td>
<td>1.89±0.63</td>
<td>1.42±0.68</td>
<td>1.39±0.60</td>
<td>1.45±0.49</td>
<td>0.001</td>
</tr>
<tr>
<td></td>
<td>RT</td>
<td>1.15±0.17</td>
<td>1.92±0.54</td>
<td>1.76±0.51</td>
<td>1.26±0.25</td>
<td>1.33±0.36</td>
<td>1.19±0.25</td>
<td>0.004</td>
</tr>
<tr>
<td>Cortisol (nmol l⁻¹)</td>
<td>NRT</td>
<td>895±448</td>
<td>846±328</td>
<td>1180±609</td>
<td>1001±658</td>
<td>980±759</td>
<td>803±720</td>
<td>0.353</td>
</tr>
<tr>
<td></td>
<td>RT</td>
<td>1146±317</td>
<td>957±402</td>
<td>843±443</td>
<td>760±572</td>
<td>632±612</td>
<td>506±515</td>
<td>0.135</td>
</tr>
</tbody>
</table>

Abbreviations: RT = resistance-trained (n = 7), NRT = non-resistance-trained (n = 10), NK = natural killer.
and at 30, 60 and 120 min of recovery ($r = 0.737$, $P = 0.001$; $r = 0.767$, $P < 0.001$; $r = 0.580$, $P = 0.023$, respectively); with the increase in neutrophil counts during exercise ($r = 0.478$, $P = 0.071$) and at 30, 60 and 120 min of recovery ($r = 0.790$, $P < 0.001$; $r = 0.835$, $P < 0.001$; $r = 0.658$, $P = 0.008$, respectively); and with the increase in monocyte counts at 30 and 60 min of recovery ($r = 0.567$, $P < 0.022$; $r = 0.507$, $P = 0.045$, respectively).

The effect of training status on cell counts is shown in Table 3. The resistance-trained participants had similar T-suppressor cell counts to, but lower T-helper cell counts than, the non-resistance-trained participants at all times. This resulted in a lower CD4/CD8 ratio for the resistance-trained than the non-resistance-trained group at all times; the difference was most marked 1 h after exercise. However, after Bonferroni correction, these differences were no longer significant. The increase in monocyte counts 2 h after exercise was much greater in the non-resistance-trained group. After adjustment for haematocrit, the difference was no longer significant. The resistance-trained participants had higher natural killer cell counts immediately after exercise ($r = 0.577$, $P = 0.024$). At 30 and 60 min after exercise, cortisol concentration was negatively related to the observed increase in T-helper cells ($r = -0.573$, $P = 0.020$; $r = -0.642$, $P = 0.007$, respectively), and positively related to the observed decrease in T-suppressor cells ($r = 0.587$, $P = 0.017$; $r = 0.603$, $P = 0.013$, respectively) and natural killer cells ($r = 0.787$, $P < 0.001$; $r = 0.722$, $P = 0.02$, respectively).

**Discussion**

An increased mobilization of white blood cells was observed during exercise. The marked leukocytosis was due to an increase in both the numbers of lymphocytes and neutrophils. These alterations were similar to the changes reported in short-term sprint protocols (Gleson et al., 1995a), endurance protocols (Nieman et al., 1994; Gleson et al., 1995b; Shek et al., 1995; Suzuki et al., 1996; Nagao et al., 2000) and other resistance protocols (Nieman et al., 1995b; Kraemer et al., 1996; Miles et al., 1999). The resistance protocol used in the present study included 10 different exercises for the training of various muscle groups. This contrasts with other protocols reported in the literature, which used 8–10 sets of the same exercise (Nieman et al., 1995b; Kraemer et al., 1996) or focused on eccentric exercise (Miles et al., 1999). It is our belief that this resistance protocol reflects a practical exercise modality, which suggests that immunomodulation occurs not only in experimental studies but also after quite usual exercise (i.e. a resistance training circuit).

The hormonal changes (noradrenaline, cortisol) during resistance exercise were moderate compared with those reported previously for endurance exercise (Meyer et al., 1992). This indicates that factors other than increased concentrations of hormones (e.g. cardiovascular effects) might alter cell distribution during exercise. Nevertheless, significant correlations between noradrenaline, cortisol and total leukocytes during exercise were observed in the present study, suggesting that there is at least some influence of cortisol and noradrenaline on white blood cell counts, as previously suggested (Weicker and Werle, 1991; Mazzeo 1994; Tvede et al., 1994; Brenner et al., 1998). In this study, noradrenaline concentrations were associated with higher natural killer cell counts during exercise, an effect that has been observed before in both endurance (Nagao et al., 2000) and resistance exercise (Stock et al., 1995).

During recovery, we noted different distribution patterns of the leukocyte subpopulations. The greatest increase was in the number of circulating neutrophils. This has also been reported after endurance protocols (Shinkai et al., 1996; Weinstock et al., 1997). During recovery, the blood lymphocyte count decreased slightly below resting values. A much larger decrease has been reported after intense endurance exercise (Frisina et al., 1994; Nieman et al., 1994; Shek et al., 1995). Cortisol has been suggested to be responsible for the decrease in lymphocytes during recovery (Nieman et al., 1997). In the present study, cortisol concentration correlated negatively with all lymphocyte subpopulations (i.e. T-helper, T-suppressor and natural killer cells) during recovery. The decrease in CD4/CD8 after exercise was associated with high cortisol concentrations. According to Kraemer et al. (1996), a clear role for cortisol in changes to the numbers of circulating leukocytes during exercise has yet to be established. In the present study, cortisol concentration correlated positively with the increase in the total number of circulating lymphocytes during exercise. Similar results were reported by Boas et al. (2000). One explanation for this anomaly – an increase in lymphocyte counts during exercise but a reduction in lymphocyte counts after exercise – might be that cortisol induces both lymphocyte mobilization and margination, whereas lymphocyte margination might be delayed.

The T-helper cell counts did not decline below resting values, which is in contrast to intense endurance protocols (Kajiura et al., 1995; Moyna et al., 1996). A negative correlation was observed between cortisol concentration and the increase in T-helper cell counts between baseline and 2 h of recovery. The negative
effect of cortisol on T-helper cell counts has been described previously (Nieman et al., 1997). The T-helper cell counts tended to be lower among the resistance-trained participants during exercise; however, the data for T-helper cell counts in trained versus untrained individuals are conflicting (Baj et al., 1994; Nieman et al., 1995b; Bury et al., 1998).

The T-suppressor cell counts did not increase and the CD4/CD8 ratio did not decrease during recovery, as is usually observed after intense endurance exercise (Nieman, 1994; Greenleaf et al., 1995). A lower CD4/CD8 ratio might contribute to the higher susceptibility of athletes to infections after strenuous exercise, the so-called 'open window'. In the present study, the CD4/CD8 ratio was in fact higher after exercise than before. Although the exercise protocol did not negatively influence the CD4/CD8 ratio, the resistance-trained participants tended to have a lower CD4/CD8 ratio at all times. Resistance exercise, performed on a regular basis, could result in a lower CD4/CD8 ratio, although in our study the ratio remained in the normal range. It is interesting to note that three resistance-trained participants (42%), but only one non-resistance-trained participant (10%), had a CD4/CD8 ratio below 1.2 during recovery. Whether regular resistance exercise decreases T-helper cell counts and the CD4/CD8 ratio, and whether it leads to an increased risk of infection, requires further investigation. Down-regulation of the immune system has been observed in professional athletes during periods of intense training (Baj et al., 1994; Bury et al., 1998), but not in recreational athletes (Nieman et al., 1995a). In our study, the CD4/CD8 ratio correlated negatively with cortisol. The reason for this might be that cortisol is responsible for a reduction in T-helper cells and, to a lesser extent, a reduction in T-suppressor cells, as demonstrated by Shinkai et al. (1996).

Natural killer cell counts decreased during recovery, which has also been reported after endurance exercise (Shephard and Shek, 1999). The decrease in natural killer cell counts correlated positively with cortisol concentration. The results of a meta-analysis (Shephard and Shek, 1999) suggested that resting numbers of circulating natural killer cells are increased in endurance athletes. A significant difference was not observed between the resistance-trained and non-resistance-trained participants in the present study. It would appear that regular endurance exercise, but not resistance exercise, results in higher counts of natural killer cells.

Thirty minutes after exercise, the numbers of circulating monocytes had reverted from their high during exercise to pre-exercise values. This decrease correlated negatively with plasma noradrenaline concentration. Both 60 and 120 min after exercise, the monocyte counts had increased. The increase in monocyte counts during recovery correlated positively with cortisol concentration. Data on the effect of endurance exercise training on resting monocyte counts are conflicting. Both increased (Weinstock et al., 1997) and decreased numbers of circulating monocytes (Bury et al., 1996) have been reported in the literature. No difference in monocyte counts was observed between the resistance-trained and non-resistance-trained participants.

Conclusions

The resistance exercise adopted in this study reflects a common training practice and so is different from earlier studies that used unusual training loads and experimental conditions. Our resistance training protocol altered white blood cell counts during exercise and recovery similar to that observed with endurance exercise independent of training status. The leukocyte counts were modulated by stress hormones, although noradrenaline and cortisol were changed only moderately. Although the resistance exercise was exhausting, it did not result in a decrease in the CD4/CD8 ratio, which normally occurs after intense endurance exercise. However, it did result in a decrease in natural killer cells during recovery. The changes in white blood cell counts were similar in both groups. Although the resistance-trained participants trained more than the non-resistance-trained participants, we believe that this did not confound the results, as the differences in duration and repetitions were minor. Performed regularly, resistance exercise might cause modulation of the immune system, as the resistance-trained participants tended to have decreased T-helper cell counts and a decreased CD4/CD8 ratio, although still within the normal range. This requires further investigation, as it could increase susceptibility to infections in resistance-trained individuals. Unfortunately, epidemiological studies on the infection risk of resistance-trained individuals, which would allow a comparison between experimental and epidemiological data, have yet to be published.

References


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