The Acute Immune Response to Exhaustive Resistance Exercise


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Ten young male adults (mean age 46.9 ± 1.2 yrs) with 9.2 ± 1.4 years of weight training experience and the ability to parallel squat at least 1.5 times their body mass were selected as subjects. The exercise session consisted of sets of 10 repetitions at 65% 1-RM of the parallel leg squat, with a cadence of one rep every 6 sec and 3 min rest between sets, to muscular failure. The average subject lifted a total of 9711 ± 1576 kg during 58 ± 14 reps for a total work output of 72.5 ± 10.5 kJ before muscular failure occurred. Mean oxygen consumption during exercise was 1.58 ± 0.06 L/min at 42.5 ± 2.0 % peak VO2. A strong leukocytosis, lymphocytosis, and lymphocytopenia, similar to what has been reported following high-intensity cardiorespiratory exercise, were measured following leg squat exercise. Con A-stimulated lymphocyte proliferation (unadjusted) rose 50% above preexercise levels (p = 0.07), but when these data were adjusted on a per T cell (CD3+) basis, no change from rest was observed. Natural killer cell cytotoxic activity (NKCA), when adjusted on a per NK cell (CD56+) basis, was decreased about 40% below preexercise levels for at least 2 h post-exercise. No significant increase in cortisol was seen after exercise, although norepinephrine and epinephrine increased moderately (465% and 133%, respectively), immediately following exercise. The data demonstrate that leg squat exercise to muscular failure results in a very similar immune response to that associated with intense endurance exercise, despite a lower mean oxygen consumption and only a moderate hormonal response.

Introduction

Cardiorespiratory exercise, especially when the intensity is high, stimulates the neuroendocrine system to a significant degree, causing significant perturbations in the functional capacity and concentrations of the various cells of the immune system (26–29). The extent and duration of the alterations in leukocyte subset counts and function appear to be related to the exercise-induced changes in epinephrine and cortisol (5, 6, 17, 24, 25, 28, 37). The concentrations of epinephrine and cortisol begin to increase strongly when the exercise intensity rises above 60% VO2max, and attain their highest levels following near-maximal intensity exercise sessions (5, 6, 17, 37).

Most studies have shown that during weight training exercise at a measured percent of maximum heart rate, the aerobic demand or percent VO2max is less than for endurance exercise (3, 16). Although mean heart rates are usually between 60–100% of maximum during weight lifting, oxygen consumption averages only 35–60% of aerobic power. The mechanism underlying the higher heart rate during weight training exercise compared to endurance exercise at the same oxygen consumption is unknown, but it may be related in part to enhanced sympathetic activity as reflected in relatively higher concentrations of norepinephrine and epinephrine (16, 23).

We are unaware of any published reports on the acute immune response to a single bout of weight training exercise. We propose that this is an excellent model to test the assumption that relative intensity of exercise (or percent VO2max) determines the magnitude of the acute immune response (24, 25). It is our hypothesis that although the oxygen consumption during weight lifting is relatively low when compared to endurance exercise, the high sympathetic nervous system response, as indicated by increased levels of catecholamines and heart rate, should lead to large alterations in circulating concentrations and function of various leukocyte subsets. The purpose of this study was to measure the acute response of the immune system to an exhaustive bout of leg squat exercise along with metabolic indicators and hormone concentrations, to allow comparison with previous work conducted in our laboratories on high- and moderate-intensity aerobic exercise (24, 25).

Key words: Weight training, immune system, lymphocyte, natural killer cell activity, lymphocyte proliferative response, cortisol, epinephrine
Methods

Subjects

Ten young male adults with at least 2 years of weight training experience and the ability to parallel squat with at least 150% of their body mass were selected as subjects. Each gave written informed consent prior to participation in the study.

Research Design

Height, body mass, body composition, and aerobic power were measured during baseline testing. Body composition was assessed from a 7-site skinfold test (31) and peak VO₂ was determined utilizing a cycling ergometer protocol (34). Oxygen uptake was measured using a SensorMedics MMC Horizon System metabolic cart (Sensor medics, Yorba Linda, CA). Maximal heart rate was measured using the Polar Facemix heart rate monitor (Polar USA, Inc.). Training history was assessed through a questionnaire. At a separate session one week prior to the study, subjects' one-repetition maximum (1-RM) parallel squat was measured as described by Stone and O'Bryant (33).

Subjects reported to the Human Performance Laboratory in a 12-hour fasted condition at 0745. Not having engaged in exercise during the previous day. After resting quietly for 15 minutes, the first blood sample was taken at 0800. Subjects were then fitted with head gear for metabolic measurements and a heart rate monitor (same equipment as baseline testing). Oxygen consumption, respiratory exchange ratio, and heart rate were recorded every minute of the exercise session. At 0815, subject started their leg squat exercise session, with blood samples taken immediately after exercise, and then again after 2 hours of recovery. Subjects were given fluids before, during, and after exercise.

Leg squat exercise to muscular failure

The exercise session consisted of sets of 10 repetitions of the parallel leg squat, with a cadence of one repetition every 6 seconds and 3 minutes rest between sets. Subjects continued the sets of leg squat exercise to muscular failure (inability to stand up with the weight without assistance). Between sets, subjects rested in a seated position. Two warm-up sets of 10 repetitions at 45% and 55% of each individual's 1-RM preceded the exercise sets of 10 repetitions at 65% of 1-RM. Oxygen consumption was measured during each minute of the entire exercise session (warm-up and exercise sets), with heart rates recorded before and after each set. During 2 hours of recovery, subjects rested quietly with some light activity allowed to prevent muscle cramping.

Exercise performance (including the 2 warm-up sets) was determined by counting the total number of repetitions, duration in minutes, weight lifted, total work performed, and energy expenditure until muscular failure occurred. Total work was estimated by measuring with a tape measure the vertical displacement during performance of the parallel squat and multiplying it by the resistance (weight lifted plus 0.67 body mass) and the total number of repetitions. Energy expenditure was determined using oxygen consumption and respiratory ratio data derived from metabolic cart data according to the equation of Weir et al. (38).

Measurements from blood samples

All 3 blood samples were drawn from an antecubital vein with subjects in the seated position. A clinical hematology laboratory performed routine complete blood counts (CBC) using a Coulter STKS instrument (Coulter Electronics, Inc. Hialeah, Florida).

Peripheral blood mononuclear cells were used for immune cell phenotyping for analysis of lymphocyte subsets. The cells were isolated from heparinized blood by density gradient centrifugation with Ficoll and sodium diatrizoate (American Red Cross, Washington D.C.). Lymphocyte phenotyping was accomplished by direct immunofluorescence labelling of cell surface antigens with mouse anti-human monoclonal antibodies conjugated to fluorescein isothiocyanate (FITC) or phycoerythrin (PE) from Becton Dickinson Immunocytometry Systems (San Jose, CA). One million mononuclear cells were stained with 50 μL of the appropriate antibody (T (CD3'), B (CD19'), and NK (CD56')) and incubated for 30 minutes. The cells were then washed, fixed in 0.5 % paraformaldehyde, and analyzed on a Becton Dickinson FACScan 420 flow cytometer to determine percent of total lymphocytes. Absolute numbers were calculated using CBC data (with correction for monocytes).

The mitogenic response of lymphocytes was determined in whole blood culture using suboptimal mitogen doses (32, 40). Heparinized venous blood was diluted 1:10 with RPMI 1640 supplemented with 10% heat-inactivated fetal calf serum (FCS), penicillin (100 U/mL), streptomycin (100 μg/mL), 200 mM L-glutamine, and 5 x 10⁻⁴ M 2-mercaptoethanol. Concanavalin A (Con A) was prepared in RPMI 1640 media, at a concentration of 1 mg/mL, and then further diluted with RPMI 1640-FCS media to the suboptimal concentration of 20 μg/mL. One hundred microliters of diluted blood were dispensed into each of triplicate wells of a 96-well round-bottom microtiter plate. To each of these were added 100 μl of the suboptimal concentration of Con A. Control wells received FCS-supplemented media instead of mitogen. After 72 h incubation at 37°C, the cells were pulsed with 1 μCi of thymidine (methyl-³H) (New England Nuclear, Boston, MA) prepared with RPMI 1640-FCS. After pulsing the plate was incubated for an additional 4 h. The cells were harvested and the radionucleotide incorporation was assessed by liquid scintillation counting using Optiphase 'HiSafe 3' (1KB Scintillation Products, Gaithersburg MD) as the scintillation cocktail. Con A results are presented as both unadjusted (counts per min, cpm x 10⁴, with the control count subtracted out) and adjusted (cpm per T (CD3') cell, calculated by determining the number of T cells in each assay well based on the lymphocyte count and T cell percentage).

Natural killer cell cytotoxicity (NKCA) was assessed by the chromium release assay (2, 39). Mononuclear cells (effector cells) were separated from heparinized whole blood as described above. Chromium labeled K562 target cells were added (1 x 10⁴) to each of the wells containing effector (mononuclear) cells to yield 40:1, 20:1, 10:1, and 5:1 effector to target (E:T) ratios. The assay was performed in triplicate in V-bottom microtiter plates (Costar, Cambridge, MA). The microtiter plates were then incubated for 4 hours at 37°C in a 5% CO₂ incubator. At the end of the incubation, supernatants were harvested onto Skatron harvesting frames (Skatron, Sterling, VA), and the level of radioactivity was measured in a Packard Cobra auto-gamma
counter. The percent lysis was calculated using the mean counts per minute of the triplicate values for each E:T ratio, and the NKCA lytic units were calculated as the number of effector cells required to lyse 20% of 10,000 target cells, with results reported as the number of lytic units contained in 10^7 cells (2). The NKCA lytic units were also adjusted on a per-NK cell basis with the following formula:

\[
\text{NKCA lytic units} = \frac{[5 \times \text{NK cells} \times (10^7 \text{ effector cells - monocytes})]}{}
\]

For example, if NKCA lytic units were 50 when the proportion of monocytes was 20% (of the total mononucleated cells) and NK cells was 14%, this would represent 4.46 \times 10^5 lytic units per NK cell. This adjustment was made to account for the exercise-induced change in proportion of NK cells and monocytes. To represent the NKCA lytic units per liter of blood, the per-NK cell NKCA lytic units was multiplied by the number of NK cells in a liter of blood.

Plasma cortisol was assayed using a competitive solid-phase radioimmunoassay technique (Diagnostic Products Corporation, Los Angeles, CA). For plasma catecholamines, blood samples were drawn into chilled tubes containing EDTA and glutathione (#RPN352 Vacuumtainer tubes, Amersham Corporation, Atrlington Heights, IL), centrifuged, and the plasma stored at \(-80^\circ \text{C}\) until analysis. Plasma concentrations of norepinephrine and epinephrine were determined by high pressure liquid chromatography (HPLC) with electrochemical detection as we have described previously (25).

Plasma volume changes were determined using the method of Dill and Costill (7). Lactate concentrations were analyzed in duplicate using a Sigma lactate kit (Sigma Diagnostics, St. Louis, MO) and a Bausch and Lomb Spectronic 21 spectrophotometer (Bausch and Lomb, Rochester, NY).

### Statistical analysis

Leukocyte and lymphocyte subset response, lymphocyte proliferative response, NKCA, lactate, and hormone values were analyzed using one-factor repeated-measure analysis of variance with each level of the factor (immediate post- and 2-hour postexercise) compared with preexercise values using paired t-tests. Statistical significance was set at the p \(<0.05\) level, and values are expressed as mean \(\pm \text{SE}\).

### Results

Subject characteristics are summarized in Table 1. The average subject had over 9 years of weight lifting experience (range of 3.5 to 15 years), and was able to parallel leg squat with 19 times their body mass. However, none of the subjects were elite strength-power athletes. None of the subjects engaged in regular vigorous cardiorespiratory endurance exercise, and group peak \(\text{VO}_2\) can be categorized as average using published norms (31).

<table>
<thead>
<tr>
<th>Variable (10^4/l)</th>
<th>Pre-exercise</th>
<th>Post-exercise</th>
<th>2-h post-exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leukocytes</td>
<td>6.65 \pm 0.33</td>
<td>11.97 \pm 0.79**</td>
<td>10.17 \pm 0.57**</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>3.63 \pm 0.33</td>
<td>6.00 \pm 0.57**</td>
<td>7.89 \pm 0.57**</td>
</tr>
<tr>
<td>Monocytes</td>
<td>0.63 \pm 0.04</td>
<td>1.26 \pm 0.10**</td>
<td>0.74 \pm 0.05*</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0.20 \pm 0.04</td>
<td>0.28 \pm 0.07</td>
<td>0.09 \pm 0.01**</td>
</tr>
<tr>
<td>Basophils</td>
<td>0.03 \pm 0.01</td>
<td>0.05 \pm 0.01**</td>
<td>0.04 \pm 0.01*</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>2.16 \pm 0.13</td>
<td>4.34 \pm 0.33**</td>
<td>1.42 \pm 0.12**</td>
</tr>
<tr>
<td>** T cells (CD3^+)**</td>
<td>1.78 \pm 0.11</td>
<td>2.82 \pm 0.23**</td>
<td>1.28 \pm 0.11**</td>
</tr>
<tr>
<td>** NK cells (CD56^+)**</td>
<td>0.48 \pm 0.06</td>
<td>1.56 \pm 0.22**</td>
<td>0.26 \pm 0.03**</td>
</tr>
<tr>
<td>** B cells (CD19^+)**</td>
<td>0.28 \pm 0.03</td>
<td>0.48 \pm 0.05**</td>
<td>0.24 \pm 0.02**</td>
</tr>
</tbody>
</table>

*p < 0.05 versus preexercise value; **p < 0.01 versus preexercise value

Leg squat performance indicators are outlined in Table 2. Including the two warm-up sets, the average subject lifted a total of 9,711 kg during 98 parallel leg squat repetitions before muscular failure occurred. Although total work output averaged 5,154 kJ, direct measurement of oxygen consumption during the leg squat session indicated that 1,159 kJ were expended, giving a 22.5% net efficiency rating. Excluding the two warm-up sets, subjects averaged 1,58 liters of oxygen consumption per minute or 42.5% of peak \(\text{VO}_2\). Ending heart rates averaged 88% of maximum. The average pre- and postexercise heart rates of the exercise sets (65% - 1RM) were 126 ± 5 and 170 ± 4 beats/min, respectively. Preexercise lactates were 1.7 ± 0.2 mmol/l before climbing to 12.0 ± 3.3 mmol/l immediately postexercise, and then falling to 2.1 mmol/l after 2 hours of recovery.

Plasma volume was not significantly reduced by the exercise session, which was probably due to the relatively large quantity of fluid that was ingested by the subjects. Total leukocytes were increased 80% above preexercise levels immediately postexercise, and were still elevated 53% 2 hours later (Table 3). This leukocytosis was nearly evenly divided between neutrophils and lymphocytes, with some contribution made by monocytes. The lymphocyte count doubled after exercise before falling 34% below preexercise levels 2 hours later: The lymphocytosis was evenly divided between T and NK cells.
with a small contribution made by B cells. Immediately following exercise, NK cells showed the largest percentage increase, with postexercise levels 225% above preexercise. The lymphocytopenia was two-thirds T and one-third NK cells.

Con A-stimulated lymphocyte proliferation rose 50% above preexercise levels after the bout of leg squat exercise (P = 0.07), but when these data were adjusted on a per T cell (CD3+) basis to account for the varying number of T cells initially present in the whole blood assay wells, no alteration in proliferative response was recorded (Table 4).

NKCA data are summarized in Figs. 1 and 2. Fig. 1 demonstrates that the unadjusted NKCA (lytic units) was significantly decreased (61%) 2 hours after the exercise session. When adjusted on a per NK cell (CD56+) basis to account for exercise-induced alterations in the proportion of NK cells present in the assay wells, however, the immediate-post and 2-hour recovery values were significantly decreased about 40% below preexercise levels.

Table 5 summarizes the changes in cortisol, norepinephrine, and epinephrine following the exercise session. No significant increase in cortisol was measured after exercise. Norepinephrine and epinephrine increased by 465% and 133%, respectively, immediately following exercise, and were back to preexercise levels within 2 hours.

**Discussion**

Numerous studies have now established that vigorous endurance exercise (70–85% VO2 max) is associated with a unique biphasic perturbation of the circulating leukocyte count (10,15,24,36). Immediately postexercise, total leukocytes increase 50–100%, represented evenly by lymphocytes and neutrophils with a small contribution from monocytes. Within 30 minutes of recovery from exercise, however, the leukocyte count dips 30–60% below preexercise levels, remaining low for 3 to 6 hours. Meanwhile, a marked and prolonged neutrophilia can be measured. The data from the present study demonstrate that leg squat exercise to muscular failure results in a very similar response of circulating immune cells despite a lower metabolic and hormonal response.

The extent and duration of the alterations in leukocyte subset counts have usually been attributed to exercise-induced changes in epinephrine and cortisol (24,25). Exercise is known to induce a rapid increase in the density of lymphocytic β2-adrenoceptors, especially as the intensity increases (22). Epinephrine is a potent β2-adrenergic agonist, and it has been associated with substantial increases in the number of circulating lymphocytes (17,37). Of the three major lymphocyte subpopulations (T, B, and NK cells), NK cells are by far the most responsive to endurance exercise (13,25). It is typical for NK cells to increase 150–300% immediately following high-intensity
exercise, and then contribute substantially to the overall lymphocytosis (19,13 18 25 29,30). Circulating numbers of T cells, especially the naive cell subpopulation of T suppressor/cytotoxic cells (CD8 CD45RO); also increase markedly (50–100%) after vigorous endurance exercise while helper/inducer and B cells are relatively unaffected (5,13 19 22,24). However, the effect is transient, and within 30 minutes lymphocytes from each of the subpopulation groups exit the circulation in large numbers under the influence of cortisol, falling 30–60% below preexercise levels. Increases in serum cortisol concentrations have been shown in various infusion studies to induce a strong and sustained neutrophilia while inhibiting the entry of lymphocytes into the circulation and facilitating their egress from the blood to other lymphoid compartments (6,35).

The immune response to exhaustive leg squat paralleled that of high-intensity endurance exercise despite a relatively low mean oxygen consumption (42.5% peak VO2) and a moderate hormonal response. For example, Nieman et al. (25), have reported that immediately following 45 minutes of running at 80% VO2 max or graded treadmill walking at 50% VO2 max, epinephrine rose to 1.29 and 0.568 nmol/L respectively, with cortisol at 926 and 742 nmol/L respectively. These data when compared with the post-exercise epinephrine and cortisol concentrations of 0.77 and 719 nmol/L in the present study indicate a rather moderate hormonal response, similar to the results of others investigating the acute hormonal response to resistance exercise (20,23). Kraemer et al. (20) have reported that cortisol is elevated to a significant degree in response to heavy-resistance exercise only when the rest period length between sets is relatively short (1 but not 3 minutes). According to Kraemer et al. (20), in heavy resistance exercise, the duration of the force production and the length of the rest periods between sets are key variables that influence the hormonal response.

There are several potential reasons why changes in circulating levels of leukocyte subsets following 45 minutes of running at 80% VO2 parallel those found after leg squat exercise to fatigue (with 3 minute rest intervals). Growing evidence suggests that the immune system is at least partly under the control of the sympathetic nervous system, being anatomically linked by a dense innervation of lymphoid tissues (19,22,26,37). Lymphocytes have surface receptors for the sympathetic neurotransmitters, and strong activation of the sympathetic nervous system, which occurs during heavy resistance exercise as reflected by the high norepinephrine concentrations, may induce a rapid up-regulation of lymphocytic adrenoceptors (22,37). Gabriel et al. have demonstrated that most of the increase in circulating number of lymphocytes (and various subsets) occurs within the first 10 minutes of intensive endurance exercise, before epinephrine has had time to rise strongly (13). They propose that various factors in addition to epinephrine may trigger the loss of adhesion to the endothelium, leading to an exercise-induced lymphocytosis. During long-term recovery from leg squat exercise, injured muscle cells may release filament fragments whose peptides are capable of being depicted as chemotactic signals for various cells of the immune system which aid in the regeneration process. Together, these data suggest that sympathetic nervous system activation, increased blood flow, and various soluble factors are important even when the hormonal response is moderate in redistributing cells of the immune system both during and following intensive exercise. Further research is warranted to test this hypothesis under more carefully controlled conditions.

Determination of the proliferative response (blastogenesis) of human lymphocytes upon stimulation with mitogens in vitro is a well-established test to evaluate the functional capacity of T lymphocytes (24). A common finding immediately following high- but not moderate-intensity endurance exercise is that mitogen-stimulated proliferation of separated mononuclear cells is decreased 35–50%, returning to preexercise levels within 2 h (10,11,12,24,36). The decrease in mitogen response is most likely due to the large exercise-induced increase in NK cells that occurs immediately following sessions of vigorous exercise (24). In the present study, no alteration in the Con A-stimulated lymphocyte proliferative response was measured following exhaustive leg squat exercise. Several researchers have provided evidence that physiologic elevation of both cortisol (6) and epinephrine (4,37) inhibit mitogen-induced lymphocyte proliferation. Various monocyte functions are inhibited in the presence of cortisol and since monocytes are important as accessory cells in many of T and B lymphocyte responses, cortisol-induced inhibition of monocyte function indirectly contributes to the decrement in activity of T cells to proliferate in response to mitogens (6). In the present study, the median post-exercise hormonal concentrations probably explain why no significant alteration in Con A-stimulated lymphocyte proliferation was measured.

NK cells are recruited rapidly from peripheral lymphoid tissues to the circulation in large numbers in response to high-intensity cardiorespiratory exercise (13). Investigators have consistently reported that immediately following high-intensity endurance exercise percent specific lysis of cancer target cells (NKCA) is increased by 40–100% before falling 25–35% below preexercise levels by 1-2 h of recovery (1,17,21,25,28,30). Although most researchers agree that the immediate postexercise increase in NKCA is due to the recruitment of NK cells into the circulation, they tend to disagree on the reasons for the transient NKCA decrease during recovery. Although some reason that the drop in NKCA can be ascribed to numerical shifts in NK cells (21,25), others report that prostaglandins from activated monocytes and neutrophils (17,29,30) or elevated hormone levels (26) suppress the ability of NK cells to function appropriately. In the present study, NKCA was significantly decreased after squat exercise to exhaustion even after adjustment on a per NK cell basis. It is very likely that prostaglandins from activated monocytes and neutrophils, as suggested by Pederson et al. (30), are responsible for this finding. As proposed earlier in this article, leg squat exercise to exhaustion may lead to significant muscle cell injury and the release of filament fragments whose peptides are capable of attracting several types of immune cells including neutrophils and monocytes which could release prostaglandins in sufficient quantity to suppress NKCA.

In summary, the data from this study suggest that perturbations in circulating concentrations of the various leukocyte and lymphocyte subsets following a single bout of exhaustive squat exercise are very similar to what has been measured following high-intensity endurance exercise, despite dissimilar metabolic and hormonal responses. T cell function was not significantly altered by squat exercise, but both unadjusted and ad-
justed NKCA was significantly depressed for at least 2 hours. Further research is warranted to determine whether prosta-
glandins from activated monocytes and neutrophils or other
factors, are responsible for the low NKCA following intense leg
squat exercise.

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