Effects of resistance training on selected indexes of immune function in elderly women

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Effects of resistance training on selected indexes of immune function in elderly women. J. Appl. Physiol. 86(6): 1905-1913, 1999.—Women aged 67–84 yr were randomly assigned to either resistance exercise (RE; n = 15) or control group (C; n = 14). RE group completed 10 wk of resistance training, whereas C group maintained normal activity. Blood samples were obtained from the RE group (at the same time points as for resting C) at rest, immediately after resistance exercise, and 2 h after exercise before (week 0) and after (week 10) training. Mononuclear cell (CD3^+^, CD3^+^CD4^+^, CD3^+^CD8^+^, CD19^+^, and CD3^+^CD16^+^CD56^+^) number, lymphocyte proliferative (LP) response to mitogen, natural cell-mediated cytotoxicity (NCMC), and serum cortisol levels were determined. Strength increased significantly in RE subjects (%change 8-repetition maximum = 148%). No significant group, exercise time, or training effects were found for CD3^+^, CD3^+^CD4^+^, CD3^+^CD8^+^, or CD3^+^CD16^+^CD56^+^ cells, but there was a significant exercise time effect for CD3^+^CD16^+^CD56^+^ cells. LP response was not different between groups, across exercise time, or after training. NCMC was increased immediately after exercise for RE subjects at week 0 and for RE and C groups at week 10. The week 0 and week 10 NCMC values were above baseline for both RE and C groups 2 h after exercise. In conclusion, acute resistance exercise did not result in postexercise suppression of NCMC or LP, and 10 wk of resistance training did not influence resting immune measures in women aged 67–84 yr.


Loss of strength in the elderly has been identified as an important ingredient contributing to a decline in independence and a corresponding heightened dependence on the health-care system (35). Loss of independence may occur due to the role that strength plays in the performance of functional activities of daily living and in the prevention of falls (38). It has been demonstrated that increased strength may improve work capacity and balance (26) in the elderly. It follows that such changes may prolong independent functioning, decrease the occurrence of falls, and result in an enhanced quality of life.

The positive effects of resistance training on musculoskeletal function in young adults are well known, and recent reports have documented similar positive adaptations in the elderly (11, 15). Resistance training has been reported to increase strength (8, 11, 15, 16), muscle size (5, 8, 11), and resting metabolic rate (33) and functional capacity (11, 26) in older individuals. Fiatarone et al. (11) reported a 174% increase in strength, concomitant with a 9% increase in muscle size, in female nonagenarians after only 8 wk of resistance training.

The influence of exercise on host defense in young adults has been equivocal (3, 4, 19, 22, 27, 30). Whereas it has been reported that a moderate-intensity endurance training program improved natural killer cell activity in adult women (25), a single bout of heavy exercise may have a transient immunosuppressive effect (4). For example, T-cell proliferative response decreased as endurance exercise intensity increased (19), leaving open the possibility that an acute bout of intense resistance exercise could elicit similar effects on host defense, especially during the early days of training.

Rincon et al. (32) recently reported that 3 mo of exercise (60 min, 3 days/wk) resulted in a decline in natural killer cell cytotoxic activity in six frail men deemed at risk for falls, compared with seven controls. In recent research conducted in our laboratory, neither elderly nor young women had a decline in natural killer cell function after a single bout of resistance training (37).

Chronic endurance training has been shown to produce a significant elevation in resting natural killer cell activity in elderly subjects (9); however, investigators from a recent study on resistance exercise training showed no effect on resting immune parameters (31). It has not been determined how the elderly respond to a single bout of resistance exercise before and after a period of resistance training. Acute resistance exercise has been reported to increase serum cortisol and epinephrine (18, 24), which are potential modulators of the immune system. In addition, high-intensity endurance exercise has been reported to suppress postexercise immune responses (16). Therefore, we hypothesized that acute resistance training would result in a similar downregulation of immune function in this population. Because the initiation of a rigorous resistance training program may prove to be a significant stressor for elderly subjects, the potential positive consequences of strength development could be compromised by impaired host defense. The purpose of this investigation was to examine the effects of both acute resistance exercise and a 10-wk resistance training program on...
METHODOLOGY

Subjects. Subjects were recruited from seniors centers in Toledo, OH, and the surrounding communities and also from newspaper advertisements. Potential subjects were asked to complete a medical history and exercise questionnaire and return it by regular mail. These questionnaires were used as a preliminary screening tool.

Screening and exclusion criteria. After the preliminary medical screening, potential subjects were asked to report to the laboratory, and they were given a detailed explanation of the risks, stresses, and potential benefits of the study before they signed informed consent. A family practice physician specializing in sports medicine examined each potential subject, and a complete medical history was obtained before participation. Subjects were screened for dementia by using the MiniMental Status Scale (14) and were excluded from participation if dementia was evident. In addition, subjects meeting the exclusion criteria of the American College of Sports Medicine (1) were not allowed to participate. Other exclusion criteria were arthritis, being bedridden within 3 mo of the study, central or peripheral nervous system disorders, stroke, use of antidepressant medications, acute or chronic infection, major affective disorder, human immunodeficiency virus infection or autoimmune disorders, metabolic disorders (type I diabetes mellitus), oral steroid use, cigarette or smokeless tobacco use, regular aerobic training or resistance training within previous 3 mo, surgery within the previous 3 mo, and caffeine consumption in excess of four cups of coffee per day (or equivalent).

Before the study, a lower extremity musculoskeletal exam was performed to identify musculoskeletal or flexibility limitations that would interfere with the completion of the training protocol. Each subject was also asked to perform a “get-up and go” test (20), which involved rising from a chair, walking 15 m, turning, and returning to sit in the chair. Subjects who were unable to complete this task were excluded from participation.

Cardiac screening and testing. After the physical examination, each subject performed a submaximal treadmill test with blood pressure and electrocardiogram monitoring. After a 3-min warm-up at 2 miles/h, the workload was increased by one metabolic equivalent every 2 min until the subjects’ heart rate reached 85% of age-predicted maximal heart rate.

Potential subjects were taught the proper lifting techniques for leg-extension exercise. After this demonstration, each subject performed a one-repetition maximum (1RM) and an eight-repetition maximum (8RM) test for each exercise. The 1RM and 8RM were defined as the weight that could be lifted no more than one time or no more than eight times, respectively, using “acceptable form.” Acceptable form was defined as the subject performing the leg-extension resistance exercise using the specified muscle groups and without using momentum or changes in body position to help apply force.

Blood pressure and electrocardiogram were monitored during each trial of the session for all subjects. The subjects rested for 5 min between exercises and for 10 min between exercise sets.

Subjects cleared for participation were randomly assigned to either a resistance exercise training (RE, n = 15) or control group (C, n = 14). Both groups participated in a 1-wk period of acclimation to resistance training, after which the C group remained inactive and the RE subjects continued to train for 10 wk.

Acclimation to resistance training. Subjects in RE and C groups were acclimated to the following resistance training exercises for 1 wk: leg extension, leg curl, hip extension, hip flexion, hip adduction, hip abduction, and ankle plantar flexion and dorsiflexion. Focus was placed on lower extremity exercises, because gait and balance were also being analyzed in these subjects. Approximately 5–10 min of either cycle ergometry or treadmill walking and a period of stretching preceded resistance exercise sessions. On Monday, Wednesday, and Friday during the acclimation week, each subject completed three sets of eight repetitions for each exercise at 50% of 1RM. On Friday of the acclimation week, the RE and C subjects’ 1RM was assessed on leg extension, leg curl, and plantar-flexion exercises, and the 8RM was assessed for every exercise.

Resistanceresistance training regimen. Subjects assigned to RE group completed an additional 10 wk of resistance training while the controls did not resistance train and were asked to maintain their normal activity level for 10 wk. A warm-up and stretching session similar to the one described above was performed before each training session. During the first week, the subjects performed three sets of eight repetitions for each exercise at 70% of 1 RM on Monday, Wednesday, and Friday. During the second week, the intensity was increased to 80% of 1RM. The 1RM was retested at the end of the fifth and tenth weeks. On Friday of each week, the subjects performed their third set to failure. If they were able to perform more than 12 repetitions in the third set, resistance was increased the following week.

Pre-and posttraining responses to resistance exercise. The pretraining experimental trials were conducted on Monday or Wednesday following the acclimation week. Each RE subject performed three sets of leg extension, leg curl, plantar flexion, and dorsiflexion at 80% of 1 RM. The first and second set were eight repetitions, and the third set was performed to voluntary fatigue. Each RE subject also performed two sets of leg abduction, leg adduction, hip extension, and hip flexion at their previously determined 8RM. Leg extension was the final exercise of the session for all subjects. The subjects rested for at least 2 min between each set. The controls did not complete the resistance exercise but sat quietly in the laboratory during the pre- and posttraining experimental trials. Posttraining experimental trials were conducted on Monday or Wednesday following the completion of resistance training. Each RE subject performed the same number of repetitions at the same absolute workload that was used during the pretraining trial. As in the pretraining experimental trials, the controls sat quietly in the laboratory during this time period.

Data-collection schedule. The first blood sample (Pre) was obtained between 0600 and 0800 from RE and C groups after a 15-min supine rest on the day of each experimental trial (pre- and posttraining). The second blood sample was obtained from RE subjects within 1 min after the resistance exercise bout and from resting controls at the same time (Post). After performing low-intensity functional capacity tests, e.g., balance, gait analysis, etc. (data presented elsewhere), the subjects were moved to a quiet room and remained seated until 2 h had elapsed from completion of the resistance exercise session. The final blood sample was obtained at this time (2hPost).

Blood sample treatment and analysis. All blood samples were obtained from an antecubital vein by using a needle and a 12-ml syringe, and blood was dispensed into four evacuated tubes: the first a plain tube, the second containing EDTA, the third containing acid citrate-dextrose, and the fourth containing preservative-free heparin.

Blood in the plain tube was kept on ice, allowed to clot, and the serum was separated and stored at −80°C until analyzed.
for serum cortisol. Blood samples (EDTA) for complete blood count and differential were refrigerated until analyzed on the same day. Lymphocyte proliferation and natural killer cell activity (natural cell-mediated cytotoxicity (NCMC)) assays were performed on heparinized blood (room temperature) within 4 h of sampling. Mononuclear cell populations were determined on acid citrate-dextrose samples stained and fixed within 6 h of sampling.

Mononuclear cell population. Mononuclear cell populations were determined by direct immunofluorescence with the use of the whole blood lysis technique. Monoclonal antibodies, conjugated with phycoerythrin and FITC (Becton-Dickinson), and appropriate controls were added to whole blood aliquots and incubated at room temperature for 15 min. FACS lysing solution (Becton-Dickinson) was added, and the mixture was incubated for an additional 15 min before centrifugation at 400 g for 45 s. The supernatant was removed, and the cells were washed before being fixed with 2% paraformaldehyde.

The absolute number and percentage of cells was determined by monoclonal cells bearing the cell surface markers CD3 (T cells), CD4 (CD3 + CD4 + helper/inducer cells), CD8 (CD3 + CD8 + suppressor/cytotoxic cells), CD56 (CD3 + CD56 + natural killer cells). Mononuclear cell populations analyses were only performed on the Pre samples of the controls and at all time points for RE subjects. White blood cell count and differential were determined by an electronic counter (Coulter STKS).

NCMC and lymphocyte proliferation. The function of natural killer cells and lymphocytes was assessed by using NCMC (i.e., natural killer cell activity) and lymphocyte proliferative response to mitogen assays, respectively, using the whole blood techniques of Baron et al. (2) and Fletcher et al. (13). NCMC was measured in triplicate by using four concentrations of cultured K562 cells (American Type Culture Collection) preincubated with 51Cr (2). After a 4-h incubation (37°C, 5% CO2) 100 µl of chilled medium were added to each well to stop the assay, and the plates were centrifuged (10 min at 400 g). The supernatant (100 µl) was transferred to polystyrene plates and counted for 5 min in a gamma counter (Gamma Trac 1191, Tm Analytic). NCMC was calculated in two ways: 1) as percent cytotoxicity and 2) at a 1:1 effector-to-target ratio. The 1:1 effector-to-target ratio calculation required the information from the flow cytometry analysis (natural killer cell number). Because flow cytometry data were only available for the Pre sample for controls, the 1:1 effector-to-target ratios were calculated for C group at rest (Pre) and for RE group at all time points. NCMC values expressed as percent cytotoxicity were calculated without flow cytometry data and were available at all time points for C and RE groups.

Lymphocyte proliferative response to mitogen assays (13) were performed by making triplicate cultures from a 1:5 dilution of whole blood. The cultures were incubated at 37°C for 72 h in an atmosphere of 5% CO2, at two concentrations of concanavalin A (ConA; 10 and 40 µg/ml). Four hours before harvest onto glass-fiber filter paper (Skatron Cell Harvester), the cells were pulsed with 1 µCi/well of [3H]thymidine. Each filter disk was placed into scintillation vials with 5 ml of scintillation fluid and counted for 1 min (BetaTrac 6895, Tm Analytic).

Serum cortisol. Serum cortisol was determined on all blood samples in duplicate, via solid-phase radioimmunoassay (129) using a commercially available kit (Diagnostic Products, Los Angeles, CA). The sensitivity of the assay is reported by the company to be 5.5 nmol/l, and the average intra-assay coefficient of variation is 4.3% for a range of concentrations (85.5–938.1 nmol/l).

Table 1. Descriptive data for RE training and C groups before and after 10 wk of resistance training by RE group

<table>
<thead>
<tr>
<th>Week 0</th>
<th>Week 10</th>
<th>Week 0</th>
<th>Week 10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>72.6 ± 3.5</td>
<td>72.9 ± 4.9</td>
<td>72.6 ± 3.5</td>
</tr>
<tr>
<td>Height, cm</td>
<td>155.5 ± 6.6</td>
<td>157.9 ± 4.6</td>
<td>155.5 ± 6.6</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>62.0 ± 12.0</td>
<td>62.7 ± 12.6</td>
<td>65.4 ± 5.8</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>25.6 ± 4.4</td>
<td>25.7 ± 4.5</td>
<td>26.3 ± 2.0</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>29.5 ± 7.2</td>
<td>27.8 ± 7.0</td>
<td>30.6 ± 5.9</td>
</tr>
</tbody>
</table>

Values are means ± SD. RE, resistance exercise-trained group; C, control group; BMI, body mass index; week 0, pretraining; week 10, posttraining.

RESULTS

Subjects. Random assignment to groups resulted in similar age, height, weight, body mass index, and body fat between RE and C subjects. There were no differences in these data before vs. after training. The descriptive data for the subjects can be found in Table 1.

Strength changes with training. In the RE group, strength increased significantly for all exercises, as evidenced by the pre- to posttraining 1RM and 8RM differences (Tables 2 and 3). There were also significant differences between RE and C groups for pretraining 1RM values of leg extension (Table 2) and 8RM values of leg extension, hip flexion, hip extension, and hip adduction (Table 3).

Lymphocyte pheno-type number. There were no significant group, exercise time, training time, or interaction effects for CD3+, CD3+CD4+, and CD3+CD8+ cell number (Table 4). In addition, there were no significant group, training time, or interaction effects for CD3–CDBCD56–CD16+ (natural killer cells, Table 5).

Table 2. One-repetition maximum for RE training and C groups before and after 10 wk of resistance training for RE and pretraining for C groups

<table>
<thead>
<tr>
<th>Exercise</th>
<th>Week 0</th>
<th>Week 10</th>
<th>Week 0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leg extension, kg</td>
<td>16.2 ± 0.9*†</td>
<td>24.8 ± 1.4</td>
<td>22.8 ± 1.1†</td>
</tr>
<tr>
<td>Leg curl, kg</td>
<td>9.7 ± 0.8*</td>
<td>19.4 ± 1.2</td>
<td>11.1 ± 1.1</td>
</tr>
<tr>
<td>Plantar flexion, kg</td>
<td>18.4 ± 2.1*</td>
<td>35.6 ± 1.9</td>
<td>19.8 ± 2.1</td>
</tr>
</tbody>
</table>

Values are means ± SE. *Significant difference between pre- and postraining values for RE; †significant difference between RE and C groups; P < 0.05.
but there was a significant exercise time effect in the RE group. Natural killer cell number increased postexercise compared with preexercise and 2hPost at both 0 and 10 wk of training (Table 5). Exercise also had a significant influence (exercise time effect) on the CD3+/CD56−/CD8− ratio. Post values for CD4+/CD8− cells were lower than Pre and 2hPost values for both RE- and C groups; p < 0.05.

Lymphocyte proliferative response to mitogen and NCMC. Lymphocyte proliferative responses to mitogen were assessed at both 10 and 40 µg/ml concentration of ConA. There were no significant group, exercise time, or interaction effects for lymphocyte proliferation at either dose of ConA. There was a significant training time effect for 10 µg/ml ConA (Fig. 1), such that the week 10 values were higher for both RE and C groups compared with the week 0 values at Pre, Post, and 2hPost. There was not a similar significant training time effect for 40 µg/ml ConA.

There was a significant group-by-time interaction effect for NCMC expressed as percent cytotoxicity (Fig. 2). Post NCMC values were significantly higher than Pre and 2hPost values for RE than for C subjects on weeks 0 and 10. In contrast, the week 0 2hPost values for NCMC were not different between RE and C groups, and the week 10 Post and 2hPost NCMC values were significantly lower for RE subjects than were C values measured at the same time point. For the C subjects, 2hPost NCMC was significantly higher than baseline (Pre) on week 0 and week 10 for RE and C subjects. For the C subjects, 2hPost NCMC was significantly higher than baseline (Pre) on week 10 and week 0, but at Post, only the week 10 value was significantly higher than Pre (Fig. 2).

There were no significant group or interaction effects for NCMC expressed as a 1:1 effector-to-target ratio (Fig. 3). There was a significant training time effect, i.e., the week 10 resting sample was higher for both RE and C groups compared with the week 0 sample. There was also a significant time (exercise) effect, such that the postexercise value was higher at both week 0 and week 10. The NCMC returned to baseline by the 2hPost value at week 0 but not at week 10.

Serum cortisol. There were no significant group, training time, or interaction effects for serum cortisol. However, serum cortisol was significantly reduced across exercise time in both RE and C. Serum cortisol was significantly lower Post and 2hPost compared with Pre. Because there were no significant group or interaction effects, there was apparently no effect of acute resistance training on serum cortisol levels (Fig. 4).

<table>
<thead>
<tr>
<th>Exercise</th>
<th>Week 0</th>
<th>Week 10</th>
<th>Week 0</th>
<th>Week 10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leg extension, kg</td>
<td>11.8 ± 1.2</td>
<td>21.1 ± 1.1</td>
<td>15.9 ± 1.0†</td>
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<tr>
<td>Leg curl, kg</td>
<td>6.8 ± 0.5</td>
<td>16.2 ± 1.0</td>
<td>7.8 ± 0.8</td>
<td></td>
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<tr>
<td>Plantar flexion, kg</td>
<td>12.7 ± 1.3</td>
<td>31.4 ± 1.9</td>
<td>15.1 ± 1.9</td>
<td></td>
</tr>
<tr>
<td>Hip flexion, kg</td>
<td>5.2 ± 0.4</td>
<td>13.4 ± 0.6</td>
<td>7.3 ± 0.6†</td>
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</tr>
<tr>
<td>Hip extension, kg</td>
<td>6.2 ± 0.5</td>
<td>15.7 ± 0.7</td>
<td>7.9 ± 0.6†</td>
<td></td>
</tr>
<tr>
<td>Abduction, kg</td>
<td>6.5 ± 0.6</td>
<td>13.2 ± 0.7</td>
<td>7.8 ± 0.6</td>
<td></td>
</tr>
<tr>
<td>Adduction, kg</td>
<td>8.0 ± 0.5</td>
<td>20.5 ± 0.9</td>
<td>12.4 ± 1.4†</td>
<td></td>
</tr>
<tr>
<td>Dorsiflexion, kg</td>
<td>11.4 ± 1.6</td>
<td>31.7 ± 2.3</td>
<td>12.3 ± 1.7</td>
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</tbody>
</table>

Values are means ± SE. *Significant difference between pre- and posttraining values for RE; †significant difference between RE and C groups; p < 0.05.

Table 4. Number of CD3+, CD3+ CD4+, and CD3− CD8− cells in peripheral blood of RE trained and C groups

Table 5. Number of CD3− CD16− CD56−, CD4+/CD8− ratio, and CD19+ cells, in peripheral blood of RE trained and C groups

DISCUSSION

Intense exercise is frequently associated with postexercise alterations in percentage and number of blood lymphocyte phenotypes and suppression of natural killer cell activity and lymphocyte proliferative responses to mitogen (3, 27, 34, 36). Because suppression is most often observed after intense or prolonged endurance exercise, we hypothesized that high-intensity resistance exercise might elicit similar responses in
elderly exercisers. Postresistance exercise suppression of NCMC has been reported previously by two groups of investigators using young males as subjects (7, 24). Because the elderly are reported to have impaired cellular immune function (23), we were concerned about the potential of an acute bout of resistance exercise to negatively impact the immune system.

There have been few studies completed to date that have examined the immune responses to endurance training in elderly subjects, and these findings have been equivocal (9, 23). Therefore, a second purpose of this investigation was to examine whether 10 wk of resistance training would elicit changes in resting immune function, as measured by phenotypic and functional tests. In summary, acute resistance exercise did not negatively affect immune function either before or after a 10-wk period of resistance training. Additionally, 10 wk of resistance training, while elicitting substantial increases in muscular strength, did not positively influence the immune system in these elderly women compared with inactive controls.

Acute exercise. The finding that CD3⁺, CD3⁺CD4⁺, and CD3⁺CD8⁺ cell numbers were unchanged after an acute bout of resistance exercise is supported by other work from our laboratory in which young men were used (7). Conversely, Neiman et al. (24) found that there was a transient increase in CD3⁺ cell number following intense resistance exercise in young males. A consistent finding between the present study and other investigations examining the immune responses to resistance exercise (7, 24, 37) was a transient increase

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Fig. 1. Values are means ± SE. Lymphocyte proliferative response to mitogen for concanavalin A at 10 µg/ml. A: data for resistance exercise (RE) training group; B: data for nonexercising controls (C) at same time points. Week 0, prereresistance training; week 10, postresistance training; Pre, rest; Post, immediately postexercise (or at the same time point for C); 2hPost, after 2 h of recovery (same time point for C); cpm, counts/min. Like symbol pairs denote a significant difference between week 0 and week 10 means at that exercise time point, i.e., week 10 means were significantly different from week 0 at Pre, Post, and 2hPost; P < 0.05.

Fig. 2. Values are means ± SE. Natural cell-mediated cytotoxicity expressed as mean %cytotoxicity for the 4 target cell concentrations for RE trained (A) and C groups (B). Like letters (a, b, c) denote significant differences (interaction) between RE and C groups; P < 0.05. *Significant difference from Pre mean; P < 0.05.
in natural killer cell number (CD3\(^{-}\)CD56\(^{+}\)CD16\(^{+}\)) immediately after exercise.

Both NCMC and lymphocyte proliferative responses to mitogen have been reported to fall below baseline levels for 1–6 h of recovery from either prolonged or intense endurance exercise (27, 36). This phenomenon has also been observed after intense resistance exercise in young males (7, 24), whereby NCMC was significantly lower than baseline after 2 h of recovery. We previously found that NCMC was not suppressed 2 h after resistance exercise in both young and older women (37). We theorized that the volume of exercise might have been insufficient to elicit the postexercise suppression observed in previous studies with resistance exercise in males. Therefore, in the present study, the subjects performed a substantially more rigorous exercise bout (20 sets) and exercised to failure in the final set of four of the eight exercises. Surprisingly, there was still no evidence of postexercise suppression of NCMC in these women. Because these women and the young and elderly women in our previous study (37) were new to resistance exercise, it is possible that they lacked sufficient skill, experience, or motivation, to work at an intensity that would elicit postexercise NCMC suppression. In an effort to maximize the training intensity, 1 wk of acclimation was provided to subjects in both studies, and the 1RM used for calculating intensity was reassessed at the end of the acclimation week. Therefore, we thought it significant that the women in these studies responded differently than the men in previous investigations (7, 24).

The men in the study of Nieman et al. (24) performed with greater absolute workloads than did the women in our study and exercised to muscular failure on a single resistance exercise (squat). However, there are other differences between the two studies that make the disparate immune findings more difficult to explain. For example, the women performed at a higher relative intensity and performed a greater number of total repetitions and sets. This suggests that the postexer-

![Fig. 3](image3.png)

Fig. 3. Values are means ± SE. Natural cell-mediated cytotoxicity (cyt) expressed as 1:1 effector-to-target cell ratio for RE trained (A) and C groups (B). Like letters (a, b, c, d) denote significant differences (time effect) between group means, \( P < 0.05 \). Like symbols (*) denote a significant difference between (training effect) week 0 and week 10 means; \( P < 0.05 \).

![Fig. 4](image4.png)

Fig. 4. Values are means ± SE. Serum cortisol levels for RE trained (A) and nonexercising controls (B). Like letters (a-i) denote a significant difference (time effect) between means; \( P < 0.05 \).
Exercise suppression of NCMC in young males was more likely to be a result of different methodologies for assessing NCMC [whole blood vs. isolated peripheral blood mononuclear cells (PBMC)], greater absolute work, or focus on a single exercise. It is also of interest that our study of resistance exercise in young males (7) employed a protocol most like that of the present study, but the immune system changes more closely paralleled those reported by Nieman et al. (24). Because we have found that young women did not differ substantially from elderly women during and after resistance exercise (37), it would appear that absolute workload might be the most significant factor for inducing postexercise changes in natural killer cell number and/or function. It should be noted that the posttraining tests were conducted at the same absolute intensity in these women and that subsequent studies conducted at the same relative intensity after the women have substantially increased muscular strength could result in changes similar to those previously observed in young males.

Endurance exercise appears to have a suppressive influence on lymphocyte proliferative response to mitogen, with decreases reported during and immediately after exercise and either a return to baseline or continued suppression during the recovery period (12, 19, 27, 36). There were no significant changes from baseline in lymphocyte proliferative response immediately after or during recovery from resistance exercise in the present study. The lymphocyte proliferative data from the few resistance training studies that have been completed to date are conflicting (7, 24, 37). We previously found (7) a significant suppression of lymphocyte proliferation immediately postexercise, which was sustained for 2 h of recovery. Nieman et al. (24) found that ConA-stimulated lymphocyte proliferation was increased by 50% after exhaustive leg squat exercise compared with preexercise values; however, when these data were expressed per CD3+ cell, there was no difference from preexercise values. In our recent study comparing young and elderly women (37), there were no significant changes in lymphocyte proliferation postexercise or during recovery. It would appear that additional research is required to elucidate the lymphocyte proliferative responses to resistance exercise.

Cortisol declined significantly over time in both RE and C subjects. Failure of cortisol to rise after high-intensity resistance exercise is not without precedent (18, 24). In fact, Nieman et al. (24) reported no significant increase in cortisol after exhaustive squat exercise in young men and a significant decline in cortisol at the 2-h postexercise time point. These researchers allowed 3-min rest between sets, and Kraemer et al. (18) previously reported that cortisol was significantly increased when 1-min, but not when 3-min, recovery was allowed. In the present study, subjects were required to rest at least 2 min between sets. Berk et al. (4) found that cortisol values obtained 5 min postexercise (simulated marathon) were significantly correlated with natural killer cell activity 1.5 h postexercise. Therefore, the significant Post and/or 2hPost decline in cortisol in the present study may have been responsible for elevated 2hPost NCMC in C and RE groups and could have contributed to minimizing the “exercise effect,” i.e., NCMC differences between RE and C subjects.

Prostaglandin production by activated monocytes has been suggested as one cause of suppression of NCMC during recovery. After observing that a significant reduction in NCMC after exhaustive resistance (squat) exercise was not obfuscated when expressed per natural killer cell, Nieman et al. (24) suggested that “it was very likely” that elevated prostaglandins from activated monocytes and neutrophils were responsible. Rall et al. (31) found that PGE2 production by PBMC was not altered by 12 wk of resistance training in young, elderly, or rheumatoid arthritis patients. Plasma PGE2 was also unaffected by isokinetic concentric and eccentric exercise (10). Cannon et al. (6), on the other hand, reported that damaging eccentric exercise increased the ability of phytohemagglutinin-stimulated PBMC to produce PGE2.

The interplay between numerical redistribution of natural killer cells and prostaglandin production by neutrophils and monocytes and their effects on NCMC suppression during the recovery from exercise have been the subject of considerable debate (21, 29). Because the subjects in this study reported little muscle soreness, it is possible that the magnitude of eccentric work was insufficient to stimulate significant PGE2 production (6). However, since PGE2 was not measured, it may be inappropriate to speculate whether it played a role in the observed postexercise responses. As for numerical redistribution, natural killer cell number had returned to baseline after exercise (2hPost) on both week 0 and week 10; however, NCMC expressed as percent cytotoxicity remained elevated. When NCMC was expressed at a 1:1 effector-to-target ratio, the week 0 2hPost value had returned to baseline, whereas the week 10 2hPost value remained elevated. Thus the contradictory nature of these findings fails to elucidate the contributions of the “numerical redistribution mechanism” to NCMC after intense resistance exercise in elderly women.

Training responses. Rall et al. (31) have completed the only study to date that has examined the influence of resistance exercise training on selected immune system variables. As in the present study, Rall and co-workers found that chronic (12-wk) resistance training did not significantly influence lymphocyte subsets or lymphocyte proliferative response to mitogen. The fact that our week 10 RE and C group values for lymphocyte proliferation were higher than week 0 values suggests that a seasonal variation was responsible for these differences. Rall et al. did not measure NCMC, and there are no other published studies with which to compare our finding that resting NCMC was unchanged after 10 wk of resistance training.

In conclusion, immune function in 67- to 84-yr-old women was not suppressed during the recovery period from a single bout of resistance exercise. In addition, a 10-wk resistance training program did not significantly alter resting indexes of immune function in these
women, and the exercise-induced immune responses were similar before and after resistance training. The present data lead us to suggest that women aged 69–84 yr can substantially improve strength with chronic resistance training without either detrimental or positive effects on selected indexes of immune system function.

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