Rest-Interval Length Affects Leukocyte Levels During Heavy Resistance Exercise

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ABSTRACT. Mayhew, D.L., J.P. Thyfault, and A.J. Koch. Restinterval length affects leukocyte levels during heavy resistance exercise. J. Strength Cond. Res. 19(1):16-22. 2005.-We sought to determine the effect of varying rest intervals on leukocyte levels during heavy resistance exercise. Nine college men completed 2 exercise bouts of 10 sets of 10 repetitions at 65% 1 repetition maximum (1RM) leg press with 1- (1MIN) or 3-minute (3MIN) rest intervals, respectively. Blood collected at rest (PRE), immediately postexercise (POST), and 1.5 hours postexercise (1.5H) was analyzed for leukocyte levels. Data were analyzed using a 2 imes 3 repeated measures analysis of variance. A greater PRE-POST lymphocytosis (+83% vs. +16%, p = 0.002) and monocytosis (+47% vs. +15%, p = 0.005) was observed following 1MIN vs. 3MIN. Serum creatine kinase (CK) activity was increased to a greater extent 24 h postexercise following the 1minute rest protocol (p = 0.022). CK was correlated (r = 0.611) to the PRE-POST lymphocytosis. We conclude that short rest intervals increase the extent of postexercise lymphocytosis and monocytosis, when total work is kept constant.

KEY WORDS. weight training, immune function, muscle damage

INTRODUCTION

igorous endurance exercise has been shown since 1902 to elicit changes in circulating leukocyte counts following exercise (22). The transient exercise-induced changes in these counts are often characterized by a biphasic response, with increased leukocyte levels (due to neutrophilia, lymphocytosis, and monocytosis) observed during and immediately after exercise and a subsequent lymphopenia and a sustained neutrophilia during the 30 minutes to 6 hours following recovery. The magnitude and duration of each response depends on the nature, intensity, and duration of exercise, with changes typically resolving within 6 hours (25). Historically the majority of investigators have sought to elucidate the immunological effects of exercises with a primarily aerobic component (9, 27, 31, 38, 39). The responses noted are similar to those found in sepsis (37), although less pronounced.

Investigators studying the immune response to endurance exercise have come to recognize that the pattern of altered leukocyte subset trafficking (the process by which different subsets of leukocytes migrate differentially into different tissues) is dependent on the duration and intensity of the exercise (26). Mechanisms behind the fluctuations in leukocyte trafficking are thought to include stress hormones and cytokine concentrations, changes in body temperature, hydration status, and increased blood flow (26). Particular attention has been given to the effects of cortisol (38), epinephrine (14), and norepinephrine (13) on cell trafficking changes experienced during exercise recovery. Thus, increased exercise duration and intensity increase the magnitude of elevation of these neuroendocrine factors leading to alterations in circulating leukocyte subsets.

Changes in leukocyte subset trafficking following resistance exercise have been less studied. The results of available studies display a large degree of inconsistency due to the wide variability in resistance exercise protocols utilized (5, 8, 15, 16, 23, 28). Therefore, it has been difficult to establish the thresholds of intensity and duration needed to elicit leukocyte distribution responses. Typically, significant leukocytosis with lymphocytosis, monocytosis, and neutrophilia has occurred immediately in response to exercise (5, 8, 15, 23, 28), while lymphocytes (15, 28) and eosinophils (28) have occasionally shown a drop below baseline levels during the postexercise recovery period. However, while some studies have reported no acute changes in cortisol secretion in response to resistance exercise (5, 8, 23, 28), others have reported changes (15, 16).

Resistance exercise comprises a number of factors: the specific exercise movement, the weight used, the repetition cadence, and the rest interval utilized between sets. Differences in rest-interval length have been shown to influence the neuroendocrine response to resistance exercise when other factors were kept constant. Specifically, the classic work of Kraemer and colleagues (17-19) has demonstrated that resistance exercise with short (60 second) rest intervals leads to markedly greater increases in circulating epinephrine, norepinephrine, cortisol, and growth hormone than exercise bouts with longer rest intervals. Norepinephrine, epinephrine, cortisol, and growth hormone have all been implicated as causative agents behind the redistribution of circulating leukocyte levels following exercise (reviewed in 33). In this study, an attempt was made to characterize the immunological changes that occur as a result of varying only rest-interval length between 2 isolated heavy resistance exercise sessions.

The purpose of this study was to determine the effect of rest-interval length on leukocyte subset trafficking during heavy resistance exercise while controlling for total work output. We hypothesized that a shorter rest interval would yield greater leukocytosis, monocytosis, and lymphocytosis immediately postexercise and a lymphopenia during exercise recovery due to a greater rise in cortisol and other neuroendocrine factors in the circulation.

METHODS

Experimental Approach to the Problem

The purpose of the present study was to evaluate the impact of rest-interval length on leukocyte levels following

TABLE 1. Physical characteristics of the nine weight-trained men.^{*}

Variable	Mean \pm SEM
Age (y)	22.2 ± 0.3
Height (cm)	180.7 ± 2.5
Body mass (kg)	77.05 ± 3.06
Percent fat (%)	8.89 ± 0.75
1RM Leg press (kg)	$263.4~{\pm}~56.6$
Training experience (y)	5.9 ± 2.2

* 1RM = one repetition maximum.

acute resistance exercise, when controlling for total work. We sought to control for total work output by completing the 1-minute bout 1 week prior to the 3-minute bout with all subjects. Therefore, the same work output (volume load) in each set could be controlled between bouts, while providing adequate recovery between the two. While this study design does not control for the effects of maturation, through an analysis of previously collected data (15) we determined that no significant differences exist in leukocyte responses to identical resistance exercise bouts performed 1 week apart.

The exercise stimulus selected was modified from previously published work (15, 16, 28): using a large muscle mass resistance exercise to provoke an immune response. The fact that the current protocol consisted of a single exercise performed for high volume at a relatively low percentage of 1 repetition maximum (1RM) may make the data harder to generalize to whole body workouts. The impetus behind the chosen exercise stimulus was twofold: a high volume of exercise has been previously shown to produce fluctuations in leukocyte levels (15, 28) and using a single exercise allowed for expediency in testing 1RM.

Subjects

Nine recreationally weight-trained college men completed an exercise history questionnaire composed by the authors. All subjects were currently participating in resistance exercise at a frequency ≥ 3 days per week, with at least 1 day per week devoted to resistance exercise for the legs that included the performance of the leg press exercise. Physical characteristics of all participants are presented in Table 1. Each volunteer participated in 2 testing sessions and were asked to refrain from their habitual resistance exercise or other strenuous physical activity for 48 hours prior to each session. The University's Institutional Review Board approved all aspects of data collection employed in this study, and all subjects provided written informed consent.

Screening

During the subjects' first visit to the lab, anthropometric data were collected. Height was determined to the nearest 0.5 cm using a wall-mounted stadiometer, body mass was recorded to the nearest 0.01 kg using a calibrated digital scale, and percent fat was estimated using a Lange skinfold caliper (Beta Technology Inc., Cambridge, MA) and a 3-site skinfold equation (11). Next, the subjects' 1RM leg press was determined according to the procedures outlined by Stone and O'Bryant (40). Leg presses were performed on a Pro Star PS617 machine (Orthotech Sports Medical Equipment, Collinsville, IL) in a full range of motion. For this study, a full range of motion was defined as legs moving from full extension to femur and tibia at 90° and back to full extension. Subjects had to achieve a 1RM leg press ≥ 2 times their body mass in order to qualify for participation in the study.

Exercise Bouts

The first exercise bout took place 7 days after the pretesting. Subjects reported to the laboratory after having fasted for 3 hours. After 2 warm-up sets of 10 repetitions of leg presses each at 40% and 50% of 1RM, respectively, all participants completed an exercise bout consisting of 10 sets of 10 repetitions at 65% of 1RM leg press. During the first exercise bout, 1-minute rest intervals were given between each set. If a subject was unable to complete the assigned 10 repetitions during a set, the resistance was lowered by 4.54 kg for the next set.

Seven days after the first exercise bout, subjects completed a second exercise bout consisting of an identical resistance exercise bout of 10 sets of 10 repetitions at 65% of 1RM leg press. However, on this occasion, 3-minute rest intervals were given between sets. Total work output was kept constant between the 2 bouts by having the subjects complete the same number of repetitions at the same resistance in each respective set during each session (i.e., if a subject did not complete all of their assigned repetitions during the first session, the repetitions for the corresponding set on the second session were kept identical). The 1-minute bout always preceded the 3-minute bout by 1 week, because failure was more likely to occur during the 1-minute bout. Each subject performed all of his testing sessions at the same time of day (1100–1400 hours) to control for diurnal fluctuations in cortisol.

Blood Collection and Analysis

During both the second and third test sessions, blood samples were collected preexercise (PRE), immediately postexercise (POST), and 1.5 hours (1.5H) postexercise for both treatments. Additionally, a blood sample was collected 24 hours after the completion of each test session (24H). All blood samples were taken from an antecubital vein using a vacutainer with the subject in the supine position. A trained phlebotomist performed all blood draws using a sterile needle and vacutainer collection tubes. Subjects rested in a supine position for 10 minutes prior to the PRE, 1.5H, and 24H samples in order to normalize leukocyte distribution. For the POST sample collection, subjects exited the leg press machine and immediately lay supine on a nearby gymnastics mat. Blood samples from PRE, POST, and 1.5H were collected into 2 vacutainers: a 4-ml ethylenediaminetetraacetic acid (EDTA) tube for the analysis of hemoglobin, hematocrit, and complete blood counts (CBC), and a 4-ml heparinized tube for the determination of plasma cortisol. Additionally, at the PRE and 24H time points, more blood was collected into a 10-ml nontreated serum collector tube for the determination of creatine kinase activity.

Leukocyte Subsets

Blood collected in EDTA tubes at PRE, POST, and 1.5H was sent to a clinical hematology laboratory (Northeast Missouri Regional Hospital, Kirksville, MO) for the determination of CBCs. The CBC included total leukocyte number, the leukocyte subsets (lymphocytes, neutrophils, basophils, eosinophils), and hemoglobin and hematocrit. Leukocyte counts were corrected for plasma volume shift



FIGURE 1. Leukocyte levels following 10×10 leg presses compared between 1-minute and 3-minute rest intervals. Data shown are mean \pm *SEM*. * indicates $p \leq 0.025$ between-treatments difference from PRE measure. # indicates within-treatments difference from PRE measure.



FIGURE 2. Lymphocyte levels following 10×10 leg presses compared between 1-minute and 3-minute rest intervals. Data shown are mean \pm *SEM*. * indicates $p \leq 0.025$ between-treatments difference from PRE measure. # indicates within-treatments difference from PRE measure.

using hemoglobin and hematocrit as described by Dill and Costill (3).

Plasma Cortisol

Blood collected in heparinized tubes at PRE, POST, and 1.5H was immediately centrifuged at 4° C and 1,600g. The plasma was then harvested and frozen for later analysis. Plasma cortisol was assayed using a competitive solid phase ¹²⁵I radioimmunoassay technique (Diagnostic Products Corporation, Los Angeles, CA). Intra-assay coefficient of variation (CV) for plasma cortisol was 2.4%.

Serum Creatine Kinase

Blood samples collected at PRE and 24H into untreated serum collector tubes were allowed to clot for 15 minutes. Then the tubes were centrifuged for 10 minutes at 1,600g; the resulting serum was then harvested and frozen for later analysis. Serum creatine kinase (CV = 8.0%) was



FIGURE 3. Monocyte levels following 10×10 leg presses compared between 1-minute and 3-minute rest intervals. Data shown are means \pm *SEM*. * indicates $p \leq 0.025$ between-treatments difference from PRE measure. # indicates within-treatments difference from PRE measure.

determined using a commercially available kit (Sigma Chemical Co., St. Louis, MO) and a Beckman DU 530 spectrophotometer (Beckman Instruments, Fullerton, CA).

Statistical Analyses

All data are presented as mean \pm *SE*. Leukocyte subsets and cortisol were compared between treatments using a 2×3 (condition \times time) repeated measures analysis of variance (ANOVA). Post hoc analyses were accomplished by using paired *t*-tests in comparison with the PRE value, with a Bonferroni adjustment (p = 0.025). Creatine kinase was compared using a 2×2 (condition \times time) repeated measures ANOVA. Relationships between creatine kinase, cortisol, and the exercise-induced changes in leukocyte subsets were determined using Pearson's product moment correlations. Statistical significance was set at $p \leq 0.05$.

RESULTS

Exercise Volume

Volume load (including warm-up sets) was kept identical for both the 1-minute and 3-minute rest conditions, at $16,281 \pm 1,309$ kg. Only 5 of the 9 subjects were able to complete all of the assigned repetitions at 65% 1RM.

Leukocyte Levels

Significant time effects were observed for total leukocytes, lymphocytes, monocytes, and neutrophils (p < 0.001) under both exercise conditions. Significant treatment × time interactions were found for both lymphocytes (p = 0.003; observed power = 0.999; eta² = 0.544) and monocytes (p = 0.006; observed power = 0.881; eta² = 0.495), which contributed to the overall leukocyte interaction (p = 0.030; observed power = 0.667; eta² = 0.370). Post hoc analyses indicated significantly greater PRE-POST elevation for leukocytes (p = 0.018; Figure 1), lymphocytes (p = 0.001; Figure 2), and monocytes (p =0.018; Figure 3) in the 1-minute rest interval when compared to the 3-minute rest interval. No significant treatment × time interactions were noted for neutrophils (p



FIGURE 4. Neutrophil levels following 10×10 leg presses compared between 1-minute and 3-minute rest intervals. Data shown are mean \pm *SEM*. # indicates within-treatments difference from PRE measure.



FIGURE 5. Serum creatine kinase activity at PRE and 24H following 10×10 leg presses compared between 1-minute and 3-minute rest intervals. Data shown are mean \pm *SEM.* * indicates $p \leq 0.05$ difference between treatments.

= 0.576; observed power = 0.128; eta² = 0.071; Figure 4). No other significant differences existed between or within treatments with respect to leukocyte numbers.

Serum Creatine Kinase and Plasma Cortisol

Significant elevations from PRE were observed in creatine kinase (Figure 5) at 24H for both treatments (p < 0.001), with a significantly higher creatine kinase elevation, following the 1-minute rest interval than following the 3-minute rest interval (p = 0.022). Cortisol levels did not increase significantly from rest for either treatment (time effect p = 0.698, treatment \times time effect p = 0.197; Figure 6).

No correlations were found between plasma cortisol and any of the observed alterations in circulating leukocyte subsets. A moderately strong correlation (r = 0.611; p = 0.007) was found between 24H creatine kinase levels and the PRE-POST lymphocytosis.



FIGURE 6. Plasma cortisol response to 10×10 leg presses compared between 1-minute and 3-minute rest intervals. Data shown are mean \pm *SEM*.

DISCUSSION

These data indicate that rest-interval length affects leukocyte levels even when controlling for total work. In both the 1- and 3-minute rest-interval conditions lymphocytosis, monocytosis, and neutrophilia, as well as overall leukocytosis, were observed at POST, with lymphocytes and monocytes returning to resting levels by 1.5H. A sustained neutrophilia was observed from POST to 1.5H under each condition. The overall effect of lymphocyte, monocyte, and neutrophil movement at 1.5H left leukocytes at a sustained elevation when compared to PRE levels. However, lymphocytes (+83% for 1 minute vs. +16%for 3 minutes) and monocytes (+47% for 1 minute vs. +15% for 3 minutes) were elevated above PRE values to a greater degree at POST following the 1-minute rest bout when compared to the 3-minute rest bout. Therefore, it appears rest-interval length is more influential in eliciting lymphocytosis and monocytosis than in driving other leukocyte trafficking changes during heavy resistance exercise.

The findings of the present study differ markedly from those reported by Kraemer et al. (16). Kraemer and colleagues reported a significant overall leukocytosis, with no significant changes in leukocyte subsets in response to an exercise protocol of 8 sets of 10RM leg presses with rest intervals of either 1 or 3 minutes. A possible reason for these discrepant findings between the work of Kraemer and colleagues and the current data was that the former data were analyzed according to the cortisol response to exercise, rather than the length of rest interval. Another possible reason for the discrepancy may be differences in the subjects' training status. Kramer et al. (16) reported that their subjects were "recreationally weight trained" and that the subjects' 10RM weight ranged from 75-90% of their tested 1RM, values that were unreported. With these criteria, each of the subjects Kraemer et al. tested completed all of their assigned repetitions during both 1-minute and 3-minute rest intervals (16). Subjects in the present study were required to achieve a 1RM leg press of at least 2 times their body mass and then asked to complete 10 sets of 10 repetitions at 65% of their 1RM. Only 5 of the 9 men recruited were

able to complete all of their assigned repetitions. Thus, due either to differences in subjects' training status (i.e., different 1RM between samples) or perhaps the differences in exercise volume (2 additional sets in the present protocol), the present study provided a relatively greater challenge to the subjects tested than was provided during the work of Kraemer et al. (16).

Cortisol has been strongly implicated as a causative agent behind fluctuations in immune cell trafficking following endurance exercise (2). However, in the present study, no correlation existed between cortisol and either overall leukocyte levels or any leukocyte subpopulation at any time point. Thus, cortisol had no immediate effect on leukocyte trafficking as observed through circulating blood. These findings are consistent with others who found no influence of cortisol on leukocyte trafficking in response to resistance exercise (15, 16). The absence of 1.5H postexercise lymphopenia when no changes in cortisol concentrations during exercise were noted concurs with previous reports (38), lending permissive support to the argument that cortisol is associated with the delayed lymphopenia and monocytopenia (33) during exercise recovery. Significant cortisol elevation and subsequent lymphopenia were absent, possibly due to any combination of the following: (a) the relatively short duration of the resistance exercise protocol, because cortisol secretion is dependent mainly on the duration of exercise (33); (b) the time of day: cortisol varies diurnally and is known to peak in early morning and drop throughout the day (38); or (c) subjects participating in the study had only fasted for 3 hours, and cortisol is inversely related to blood glucose concentrations (30). Therefore, even the vigorous resistance exercise utilized in this study did not elicit significant cortisol elevation and a concomitant 1.5H postexercise decline in circulating leukocyte subpopulations.

It has been proposed that the neutrophilia observed several hours after strenuous exercise in many studies is at least in part perpetuated by increased corticosteroid secretion (33). However, the 1.5H neutrophilia observed in this study was not significantly related to cortisol secretion. Therefore, other untested mechanisms were responsible for the continued increase in postexercise neutrophils. Other researchers suggest that the lingering effects of catecholamines (13) and growth hormone (12) may contribute to this phenomenon. These observations do not exclude cortisol as an agent of neutrophilia when it is secreted in physiologically significant concentrations; they simply contend that other mechanisms contribute to neutrophilia independently of plasma cortisol concentration changes.

Muscle damage, as measured by creatine kinase, was significantly greater 24H after the 1-minute rest-interval bout than after the 3-minute bout and was positively correlated to the lymphocytosis observed in both conditions. The correlation findings from the present study are consistent with other reports (1, 35) that have found muscle damage associated with a rise in the lymphocyte subpopulations of NK cells and CD8⁺ T cells. The finding that short rest intervals lead to elevated serum CK, independent of the total amount of work performed, has not been reported previously to our knowledge. However, because of our research design, in which the 1-minute rest-interval condition always preceded the 3-minute rest-interval condition, these findings should be viewed with caution. Indeed, several investigators have reported an attenuated serum CK response to a second bout of exercise performed 1 week after an earlier, identical bout (6). Nonetheless, because of the training status of our subjects, who were accustomed to resistance exercise in general and the leg press exercise specifically, it is possible that the observed greater rise in serum CK activity was due to restinterval length rather than treatment order. This possibility is strengthened by the fact that CK activity was related to the POST lymphocytosis under both exercise conditions.

It is hypothesized that changes in epinephrine and norepinephrine, which are positively related to exercise intensity (29), may act on lymphocytes and monocytes to a greater extent than other leukocyte subpopulations because of relatively high plasma membrane concentrations of β -adrenergic receptors (21). Although unmeasured in this study, greater catecholamine secretion likely occurred in response to the 1-minute exercise bout when compared to the 3-minute bout (17–19), thereby possibly driving, at least in part, leukocytosis and monocytosis.

At the present time it is premature to draw meaningful practical or clinical implications from these results. Exercise-induced alterations in peripheral leukocyte counts have been associated with increased upper respiratory tract infection (URTI) (34), while others contend that no correlation exists (30). Similarly, many studies exist outlining the associative nature of increased training volume and incidence of URTI (7, 10). However, it has not been shown that the transient exercise-induced alterations in peripheral cell counts, similar to those observed in this study, are related to overall impaired immune function and consequently to a reduced ability to resist infection. First, Pedersen and Bruunsgard (comment in 36) noted that numerous studies using self-reported incidence of URTI may be confounded by, among other things, the fact that the exercise-induced inflammatory response may produce symptoms similar to those in URTI (cough, runny nose, sore throat) in the absence of true microbial infection. Second, the alterations seen in immune parameters may simply reflect a robust physiological control mechanism capable of withstanding significant component changes without compromising overall host immunity (36). Third, it must be stressed that the present data provide information regarding only the presence of leukocytes in the blood, and do not give any indication as to the activity of these cells. The cell-type specific redistribution of leukocytes, as seen during exercise, depends on their expression of surface adhesion molecules. Recent studies have begun to examine the influence of exercise on various markers of leukocyte activation, noting that lymphocytes expressing L-selectin decreased (23), while prolactin-receptor expression increased on B lymphocytes (4), and perforin mRNA expression decreased postexercise in natural killer cells (24). Future studies should examine the impact of restinterval length on these cellular variables in addition to simply tracking gross changes in cell numbers before any definitive conclusions regarding the impact of rest-interval length on immune function can be made. It remains to be seen whether the altered immune parameters observed in this and other studies significantly alter the overall host defense mechanisms and, if they do, if the alterations are clinically significant to producing increased susceptibility to URTI. A direct causal link must be established between the transient exercise-induced alterations in immune parameters and impaired host defense before the "open window" theory of immunosuppression is to gain full acceptance (26).

PRACTICAL APPLICATIONS

The key finding of this study is that short rest intervals promote a greater postexercise rise in circulating lymphocytes and monocytes, independent of the total amount of work performed. Additionally, the greater lymphocytosis is related to the extent of muscle damage, which is also greater in magnitude following shorter rest intervals. Strength coaches should be aware that when prescribing short rest intervals to facilitate high-intensity exercise endurance or hypertrophy, they are also more likely to promote disturbances in circulating immune cell traffic. Whether alterations in circulating immune cell numbers are related to an increased risk of illness in athletes is unknown. Nonetheless, these changes are indicative of a greater disturbance in homeostasis following exercise with short rest intervals. Given the relationship between muscle damage and the postexercise rise in circulating lymphocytes, nutritional strategies to minimize muscle damage (i.e. supplementation with β -hydroxy- β -methylbuterate (32) or L-carnitine L-tartrate (20, 41)) may also attenuate postexercise disturbances in leukocyte subset counts. This presents the intriguing possibility that such a nutritional strategy may reduce an athlete's risk of contracting illness. However, it is premature to make any definitive recommendations at this point. Further research into the effects of resistance exercise on immune function and illness is needed.

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