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Serum Chemistry and Hematological Adaptations to 6 Weeks of Moderate to Intense Resistance Training

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ABSTRACT

This study examined immune cell and blood chemistry changes occurring in trained weightlifters after 1 week of rest followed by 6 weeks of Olympic-style resistance exercise. Blood was drawn weekly after 1 day of rest at the same time and on the same day of the week for 7 weeks. Lymphocyte numbers increased in weeks 5 through 7. Sodium concentration rose above entry levels in week 2, remained elevated, and peaked in week 5. Direct bilirubin dropped below baseline values in the final week. Chloride and alkaline phosphatase concentrations increased as training progressed. Chloride, potassium, albumin, CO_2, and alkaline phosphatase concentrations peaked in weeks 4 through 6. Serum creatinine was elevated in weeks 2 through 5. Data indicate that resistance training induces changes in immune cell count and blood chemistry that remain within, or near, normal clinical values. It appears that resistance training does not induce immunosuppression or negatively affect hepatic or renal function.

Key Words: weight training, hematology, exercise, blood markers

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Introduction

A single bout of intense aerobic exercise induces a variety of changes in the chemical and hematological properties of blood (10). Although these properties have been described in great detail, the type, as well as magnitude, of response from this mode of exercise varies greatly. Higher aerobic intensities tend to lead to larger disturbances (reviewed in [11]). Resistance exercise can also be extremely intense, requiring effort at a work rate greater than that seen at 100% of $\dot{V}O_2$max. It has been well documented in a number of physiological systems that the structural and functional changes seen with aerobic training differ from those occurring with resistance exercise. Therefore, we cannot presume that aerobic exercise experiments and their results can describe the responses and adaptations expected from participation in regular anaerobic training. Although intensity of exercise may be an integral stimulus of change, few studies have attempted to describe the serum chemical and hematological changes resulting from either acute or chronic resistance exercise. In what is likely the first systematic study of the hematological response to acute weight training, Nieman et al. (12) described selected immune and hematological responses to the squat exercise performed for multiple sets to failure. Apart from immune-related measures, there is scattered and equivocal data regarding other acute hematological variables. A few researchers have addressed serum chemistry responses to acute anaerobic training (sprint or weight training). Similarly, studies describing chronic serum chemistry adaptations after regular weight training are sparse, use widely varying training protocols, and assay for different chemistries. Data regarding chronic hematological adaptations to regular resistance training are sparse in current scientific databases, and the extant literature focuses primarily on previously untrained or recreational exercisers. No research to date has examined the acute or chronic effects of Olympic-style weight training on serum chemistry or immune parameters in well-trained competitive weightlifters. The dearth of related literature can be demonstrated by examination of 2 major reviews of exercise immunology (13, 18). None of these very comprehensive reviews included a single citation of the effects of resistance training. This points strongly to a lack of information regarding this important area of fitness and performance.

Table 1. Subject characteristics (mean ± *SD*).

Characteristic	Entry	End
Body mass (kg)	89.0 ± 27.9	90.2 ± 29.0
% Fat	15.8 ± 6.62	15.9 ± 6.8
Lean body mass (kg)	74.0 ± 19.4	74.8 ± 19.2
Snatch (kg)	94.6 ± 25.9	96.7 ± 21.3
Clean & jerk (kg)	118.7 ± 26.8	123.3 ± 26.1
Back squat (kg)	158.7 ± 39.0	168.0 ± 40.0
Training history (sessions per wk)	3.8 ± 3.1	—
Age (y)	28.3 ± 6.3	—

The purpose of this project was threefold: (a) to test the hypothesis that variations in serum chemistry and immune cell counts with Olympic-style resistance exercise would be similar to, or greater than, those with intense aerobic exercise. Such data could provide insight into whether athletes who train and compete in very intense anaerobic sports are more or less likely to develop exercise-induced immunosuppression; (b) to provide additional descriptive data regarding human beings' blood chemistry and immune response to resistance training; any variations resulting from this rigorous training regimen may be potentially valuable as indices of overreaching or overtraining; and (c) to determine whether Olympic-style weightlifting training in trained weightlifters alters any measured variable in a manner different from those seen in sedentary or recreational populations.

Methods

Experimental Approach to the Problem

Six trained weightlifters were evaluated weekly for immune cell numbers, serum chemistry concentrations, and weightlifting performance for 6 weeks of training after 1 week of enforced rest. The training protocol consisted of a 2-week period of increasing volume and intensity, followed by 2 weeks of maximal intensity and high volume, and finally a 2-week taper of volume and intensity that culminated in an official USA Weightlifting competition. A repeated-measures design was used, with control values being established after the initial week of rest.

Subjects

The subject pool consisted of 6 apparently healthy male volunteers with similar histories of physical activity, weight training, and competition (past 6 months in continuous training; all have competed in weightlifting; all qualified for a USA Weightlifting National Event). Subject characteristics are presented in Table 1. All subjects completed a health questionnaire and screening and signed an informed consent form before entering the study. Health histories were evaluated according to the criteria set forth by the American College of Sports Medicine (1). Experimental protocols were consistent with and approved by the Human Subject Research Committee at Midwestern State University (HSRC#02070001).

Table 2. Imposed load characteristics of training program.*

Week of training	Training days per wk	Mean intensity in % of 1RM	Repetitions per set	Weekly repetitions†
1	0	0	0	0
2	3	75	2	60
3	4	85	2	112
4	5	90+	2 or 1	188
5	5	90+	2 or 1	158
6	4	88	1	56
7	2	85	1	24

* 1RM = 1 repetition maximum.

† Does not include warm-up repetitions; weeks 4 and 5 had 7 planned training sessions; In weeks 4 and 5, a relative daily 1RM was attempted twice in each snatch or clean and jerk.

Training Protocol

The total duration of the study was 7 weeks (1 week of enforced rest followed by 6 weeks of training). Documentation of exact training programs for Olympic weightlifters is sporadic and incomplete in the scientific literature. The training load imposed during the experiment was a variation on published research that included well-defined training regimens and was also based on training programs for competitive weightlifters proposed and used by elite weightlifting coaches (7, 8, 17, 19). The program is presented in Tables 2 and 3.

Hematology and Chemistry

All venipuncture was performed by a medical technologist (American Society of Clinical Pathologists) at the same hour and on the same day weekly for the duration of the study to control for diurnal variations. Blood samples were obtained in a fasting state (8 hour)

Table 3. Training program.

Week	Monday	Tuesday	Wednesday	Friday	Saturday
1	Off	Off	Off	Off	Off
2	Power snatch Power clean & jerk Back squat Abdominals	Off	Hang power snatch Hang power clean Romanian deadlift Abdominals	AM Blood sample Off	Snatch Power clean Jerk Back squat Abdominals
3	Power snatch Clean Push press Back squat Back extension	Off	Snatch Power clean Jerk Front squat Abdominals	AM Blood sample Power snatch Power clean Press Romanian deadlift	Snatch Clean & jerk Back squat Abdominals
4	AM Back squat Snatch pull Clean pull PM Snatch Clean & jerk Abdominals	Snatch Clean & jerk Back squat Abdominals	Power snatch Press Front squat Abdominals	AM Blood sample AM Snatch Push press Clean PM Back squat Snatch pull Clean pull Abdominals	Snatch Jerk Front squat Abdominals
5	AM Back squat Snatch pull Clean pull PM Snatch Clean & jerk Abdominals	Snatch Clean & jerk Back squat Abdominals	Power snatch Press Front squat Abdominals	AM Blood sample AM Snatch Push press Clean PM Back squat Snatch pull Clean pull Abdominals	Snatch Jerk Front squat Abdominals
6	Snatch Clean & jerk Back squat Abdominals	Off	Snatch Jerk Front squat Abdominals	AM Blood sample Power snatch Power clean Press Abdominals	Snatch Clean & jerk Back squat Romanian deadlift Abdominals
7	Snatch Clean & jerk Back squat Abdominals	Off	Snatch Clean & jerk Front squat Abdominals		Competition

and followed 1 day of rest and recuperation. The antecubital vein was located by sight and palpation. Once located, the area was cleansed and sanitized using 15% isopropyl alcohol. A 22G needle fitted to a Vacutainer hub was inserted into the antecubital vein, and 2 tubes of blood were acquired (22 ml total). Tube #1, a standard 10-ml serum separator tube (Becton-Dickinson, Franklin Lakes, NJ), was used to collect serum; tube #2 was a 5-ml hematology tube with tripotassium ethylenediaminetetraacetic acid (EDTA) additive (Becton-Dickinson). The needle was then withdrawn, a sterile cotton pad was placed over the insertion site, pressure was applied to the site, and a bandage was placed over the site to prevent any possible compromise of the insertion site. Each subject and each sample was assigned a unique identification number, and the code key was kept confidential until all analyses were completed. Tube #1 samples were separated into packed cell and serum components, then chemistry and enzyme assays were performed in duplicate on the serum using colorimetric assays on a COBAS Mira+ analyzer (Hoffmann-La Roche, Ltd., Basel, Switzerland). Ethylenediaminetetraacetic acid–treated samples were used for cell-count and cell-type determinations using a JT2 automated cell counter (Beckman-Coulter, Inc., Fullerton, CA). Cell counts were also performed in duplicate. Weekly assays were performed after all equipment was calibrated using

Table 4. Hematology means ± *SD* and norms.*

Measure	Norm	Week 1	Week 3	Week 5	Week 7
Hematocrit	36–50%	44.18 ± 1.28	45.33 ± 0.63	45.60 ± 1.84	45.60 ± 1.18
Hemoglobin	7.8–10.5 mmol·1^{-1}	9.41 ± 0.29	9.60 ± 0.17	9.84 ± 0.39	9.62 ± 0.29
Erythrocytes	4.1–5.6 million per mm^3	4.82 ± 0.15	4.99 ± 0.18	5.05 ± 0.23	5.02[1] ± 0.14
Leukocytes	4.0–10.5 million per mm^3	6.23 ± 1.62	6.42 ± 1.33	5.67 ± 1.02	6.23 ± 1.13
Lymphocytes	14–46%	35.62 ± 5.21	33.17 ± 4.34	38.90[3] ± 7.15	34.97[5] ± 5.23
Monocytes	4–13%	4.47 ± 1.40	4.72 ± 1.79	3.23 ± 0.95	3.08 ± 0.92
Granulocytes	42.2–75.2%	59.92 ± 6.40	62.12 ± 5.55	57.87 ± 7.03	61.95 ± 5.88
Platelets	140–415 thousand per mm^3	234.33 ± 34.31	250.00 ± 44.03	256.50 ± 16.77	249.33 ± 32.24

* Superscript numbers indicate weeks in which there is a significant difference to the value presented when analyzed with a repeated-measures analysis of variance with post hoc Student's *t*-tests.

identical clinical standards to ensure validity, reliability, and repeatability.

Performance Assessment

Performances in the competitive Olympic lifts (snatch and the clean & jerk) and back squat were assessed periodically throughout the study. Entry values for 1 repetition maximum (1RM) for the lifts were derived from the subject's most recent official competitive results. Results older than 60 days were not considered valid. Verification of entry 1RM values took place on the last day of week 2 of the training period. All training and performance testing was done with International Weightlifting Federation–certified and calibrated elite barbells (York Barbell Co., York, PA). Final testing was completed under competition conditions in an event sanctioned by USA Weightlifting.

Statistical Analyses

All serum chemistry and hematological results were analyzed with a repeated-measures analysis of variance with post hoc paired Students *t*-tests of significant results. A p value of ≤ 0.05 was considered significant.

Results

Hematology

Lymphocyte numbers increased in count between weeks 3 and 5 ($p = 0.009$), then returned to lower, near baseline counts in week 7 ($p = 0.05$). A near significant reduction in monocyte numbers that persisted throughout weeks 5 through 7 ($p = 0.0545$) was seen. The low monocyte-counts in weeks 5 through 7 were below normal clinical values. A steady increase in erythrocyte count was seen between entry and week 7 ($p < 0.034$). Mean values of the results are presented in Table 4.

Chemistry

Sodium concentrations rose steadily after the commencement of training and peaked after week 5 of training ($p = 0.0001$). Values fell in week 6 ($p = 0.0001$) and remained near baseline in week 7. Potassium concentrations remained constant in weeks 2 and 3, increased coincidentally with workload in weeks 4 and 5 ($p = 0.0284$ and $p = 0.0083$, respectively), remained elevated in week 6 ($p = 0.0291$), and returned to baseline by week 7. Chloride concentrations began to increase in week 3, continued to rise, and peaked in week 4 ($p = 0.0003$), then returned to near week-2 values in week 6. Concentrations did not return to initial levels even during week 7, a period of substantial work reduction. Carbon dioxide levels showed a gradual rise during the study until concentrations peaked in week 6 ($p = 0.014$), and they immediately returned to baseline levels in week 7. Creatinine levels increased over baseline in week 2 and remained significantly elevated for the first 3 weeks of training ($p = 0.0117$). Concentrations fell to near baseline values in weeks 6 and 7. Direct and total bilirubin concentrations underwent a nonsignificant elevation followed by consecutive reductions in concentration to lower than baseline levels in weeks 6 and 7 ($p = 0.0310$ and $p = 0.0174$, respectively). Albumin concentrations remained steady during weeks 2 through 4, rose significantly over the values in earlier training weeks in week 5 ($p = 0.0170$), and remained mildly elevated in week 7 ($p = 0.0278$). Increases in week 3 creatinine and week 5 sodium, albumin, and creatinine all resulted in values nominally above normal clinical values. Aspartate aminotransaminase concentrations dropped nonsignificantly in week 2, began rising in week 3, had risen to concentrations significantly higher than those in week 2 by week 4 ($p = 0.0340$), and were still mildly elevated over week 2 ($p = 0.0409$) but not baseline values in week 7. Alkaline phosphatase levels rose significantly immediately on commencement of training ($p = 0.0056$). Concentrations remained elevated over baseline values throughout the study period and were the highest during weeks 6 and 7 ($p = 0.0030$). Mean values of results are presented in Table 5.

Table 5. Serum chemistry means ± *SD* and norms.

Chemistry	Norms	Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7
Sodium	135–148 mmol·L^{-1}	141.7 ± 1.8	144.2[1] ± 1.8	145.0[1,2] ± 1.5	145.0[1] ± 1.55	148.3[1,2,3] ± 1.86	143.8[1,4,5] ± 1.7	143.5[5] ± 2.51
Carbon dioxide	24.9–33.1 mmol·L^{-1}	27 ± 2.9	28 ± 1.3	27.3 ± 1.6	28 ± 1.2	29.5 ± 2.2	30.1[2,3,4,5] ± 1.3	25.7[2,3,4,5,6] ± 1.0
Chloride	96–109 mmol·L^{-1}	101.8 ± 2.14	102.2 ± 3.43	105.8[1,2] ± 2.14	108.0[1,2,3] ± 1.1	106.5[1,2] ± 1.97	104.2[4] ± 1.97	105.5[1,2,4] ± 1.38
Potassium	3.5–5.3 mmol·L^{-1}	4.3 ± 0.21	4.15 ± 0.23	4.33 ± 0.29	4.67[1,2] ± 0.42	4.62[1,2,3] ± 0.15	4.58[1,2,3] ± 0.3	4.4 ± 0.24
Blood urea nitrogen	0.2–9.3 mmol·L^{-1}	5.78 ± 2.07	5.43 ± 1.71	5.78 ± 1.61	5.25 ± 1.43	5.43 ± 1.36	6.07 ± 1.75	6.07 ± 1.89
Bilirubin (direct)	0.0–5.1 μm·L^{-1}	2.39 ± 1.03	3.42 ± 0.86	3.08 ± 1.03	2.74 ± 0.68	3.25 ± 1.03	2.74[5] ± 0.51	1.71[2,3,4,5,6] ± 1.03
Bilirubin (total)	1.7–20.5 μm·L^{-1}	13.17 ± 2.74	14.88 ± 3.59	13.17 ± 3.25	12.48 ± 2.22	16.76 ± 4.96	10.43[5] ± 2.39	10.09[1,2,5] ± 1.54
Albumin	0.6–0.8 mmol·L^{-1}	0.75 ± 0.03	0.75 ± 0.06	0.74 ± 0.05	0.74 ± 0.03	0.83[2,3,4] ± 0.08	0.72[5] ± 0.03	0.77[4,5] ± 0.03
Creatinine	53.0–132.6 μmol·L^{-1}	123.76 ± 8.84	132.60[1] ± 8.84	141.44[1] ± 17.68	132.60[1] ± 8.84	141.44[1] ± 17.68	123.76 ± 8.84	132.60 ± 17.68
Glucose	3.6–6.3 mmol·L^{-1}	4.99 ± 0.39	4.92 ± 0.42	5.20 ± 0.51	5.38 ± 0.44	4.60 ± 0.76	4.71 ± 0.36	4.87 ± 0.54
Alanine aminotransaminase	7–56 IU·L^{-1}	29.7 ± 14.9	24.7 ± 11.1	26.7 ± 8.5	38.3 ± 27.4	31.5 ± 13.7	30.7 ± 14.4	25 ± 10.1
Aspartate aminotransaminase	5–40 IU·L^{-1}	22.8 ± 4.9	20.5 ± 3.3	25.2 ± 7.4	29.7 ± 9.2	25.7 ± 7.3	22 ± 5.3	22.5 ± 3.7
Alkaline phosphatase	38–126 IU·L^{-1}	58.8 ± 11.7	67.0[1] ± 11.2	71.7[1] ± 10.3	72.5[1,2] ± 13.7	71.7[1] ± 11.4	81.3[1,2,3,4] ± 11.7	76.2[1,2,3] ± 12.5

* Superscript numbers indicate weeks in which there is a significant difference to the value presented when analyzed with a repeated-measures analysis of variance with post hoc Student's *t*-tests.

Discussion

The present study investigated whether intense, Olympic-style resistance training for 6 weeks would induce changes in immune status similar to those seen in intense aerobic exercise training. Data obtained here did not demonstrate changes in immune cell counts until week 5 of the experiment, when a small but significant lymphocyte cell number increase was seen coincident with the end of the 2 most intense weeks of training. The change was transient, and baseline values were regained by the end of the training taper in week 7. Further, the increase in lymphocyte numbers remained within clinical norms. Nieman et al. (12) previously described selected immune responses to the squat exercise performed for multiple sets to failure. Their data showed marked leukocytosis comprising an increase in neutrophils, monocytes, basophils, and lymphocytes. Similarly, Fry et al. (4) noted that acute bouts of interval training also induced leukocytosis comprising increases in lymphocytes, monocytes, and neutrophils. In a longer-duration study, Kayashima et al. (9) examined the hematological response to extreme physical exertion (aerobic exercise while carrying 30–40 kg) for 4 weeks. They found prominent leukocytosis comprising increases in neutrophils and monocytes. Rall et al. (14) measured peripheral blood mononuclear cell numbers before and after 12 weeks of twice-per-week progressive resistance training in young and elderly populations. Their results were similar to those of the present study in that changes representative of immunosuppression were not seen. Bermon et al. (2) investigated the effects of 8 weeks of 3 sessions-per-week strength training in elderly and previously sedentary subjects and found no effects on the distribution of lymphocyte subsets. Similar results were also found by Gallagher et al. (6) after 8 weeks of moderate intensity training in previously untrained collegiate men. Previous and current data suggest that alterations in immune cell counts may not be effected with moderate-intensity weight training, rather maximal-intensity training as used in weeks 4 and 5 or exhaustive protocols such as used by Nieman et al. (12) may be required to induce changes in immune cell count. None of these immune cell count data support the contention that high-intensity resistance exercise might cause immunosuppression as seen after exhaustive aerobic exercise. If immune suppression does occur with severe resistance training, these data would suggest that alterations in immunocompetency would be manifested as changes in immune cell function rather than as changes in numbers or cell type.

An observation of the present study that may be of interest clinically is the reduction in monocytes during weeks 5 though 7 to levels below clinical norms. Although these reductions were not statistically significant, they were outside clinical limits and may warrant further investigation. There may have been a suppression of immune function, or the intense exercise undertaken may have led to tissue damage and to an increase in cellular debris. This increase in free chemotactic agents outside the vascular system may have simply caused increased movement of monocytes from the vascular compartment into the tissues where, as monocytes, they carried out cleanup and repair duties. This monocytic exodus may be responsible for the reduction in numbers below clinical guidelines.

A second purpose of this study was to provide a fairly comprehensive set of descriptive blood chemistry data for evaluation as potential indicators of training stress. The present study demonstrated that some changes in serum chemistry do occur. Some changes occurred relatively quickly after the commencement of training, and other changes did not occur until well into the experimental period, coincident with or after periods of increased workload (e.g., applied external resistance). In the present study, sodium, creatinine, and alkaline phosphatase concentrations rose significantly within the first week of training. Data such as these are not unexpected. Cohen et al. (3) and Taylor et al. (15) independently demonstrated that sodium concentrations increased after a single bout of sprint training. In their studies, however, the increases in sodium occurred coincident with increases in potassium concentration and decreases in chloride concentrations, findings that were somewhat dissimilar to those of our study.

Studies describing chronic serum chemistry adaptations after longer periods of regular weight training are sparse, use widely varying training protocols, and assay for different chemistries. Apart from the early and persistent changes in sodium and alkaline phosphatase concentrations, concentrations of potassium, chloride, carbon dioxide, albumin, direct bilirubin, and total bilirubin in venous blood all changed in later weeks of this experiment when compared with values obtained after 1 week of inactivity. The electrolytes seemed to exhibit the greatest changes, and the responses seemed to be largely dependent on the severity of the training, with the largest deviations from baseline values occurring after the weeks of maximal volume and intensity training. Attribution of causes for other chemistry changes seen is difficult. Our data showed that high-intensity training did not alter blood urea nitrogen, but creatinine concentrations proved to be higher in weeks 1 through 5 of the experiment, despite all 6 subjects taking supplements of creatine monohydrate (0.03 g per kg body mass) and consuming a self-reported high-protein diet for the duration of the study. One of the more complete previous investigations, a case study of a bodybuilding program's effect on serum chemistry, showed that blood urea nitrogen and creatinine concentrations were higher during 4 weeks of low-intensity training than after 5

weeks of recovery (18), a finding in opposition to that of the present study. This dissimilarity might be attributed to the differing structure of a typical bodybuilding workout as compared with the Olympic-style program researched here. Total and direct bilirubin, markers of hepatic function, were reduced after week 6 of the present experiment, whereas the bodybuilding study of Too et al. (16) showed no changes in bilirubin concentrations as did the work of Gallagher et al. (6). The last significant chemistry findings reported here were (a) an increase in alkaline phosphatase that began in week 2 and continued, reaching the highest levels in weeks 6 and 7, a finding similar to other previous research data (5), and (b) a significant increase in albumin in weeks 5 and 7.

The present study demonstrates that Olympic-style weight training using moderate to maximal intensities performed for several weeks will elicit limited changes in immune cell counts and in serum chemistry. These changes, however, likely represent healthy adaptations to this mode of exercise because neither does immune function appear to be negatively altered nor does renal function, hepatic function, or electrolyte balance appear to be clinically compromised because results largely remain within clinical norms.

Practical Applications

The health of athletes undergoing intense training for competition is a concern of every coach and athlete. It has been proposed that intense training disrupts immune function and renders athletes susceptible to upper respiratory tract infection. This study suggests that intense, Olympic-style weight training does not induce immunosuppression when evaluated by immune cell count. This study also probed for blood markers of training stress that might be exploited as means of identifying overreaching and overtraining syndromes. Although some serum chemistries did seem to be responsive to periods of increased training loads, with the magnitude of results elicited being significantly different, we found that many changes occurred in patterns unrelated to workload or were likely too small to allow for effective use in this manner.

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