Oxidative Stress Response in Trained Men following Repeated Squats or Sprints

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ABSTRACT

BLOOMER, R. J., M. J. FALVO, A. C. FRY, B. K. SCHILLING, W. A. SMITH, and C. A. MOORE. Oxidative Stress Response in Trained Men following Repeated Squats or Sprints. Med. Sci. Sports Exerc., Vol. 38, No. 8, pp. 1436–1442, 2006. Purpose: The purpose of this investigation was to measure the oxidative stress response to similarly matched work bouts of squat and sprint exercise. Methods: Twelve anaerobically trained men performed six 10-s sprints and, on a separate occasion, repeated barbell squats to approximately equal the amount of work performed during the sprints. Blood lactate, heart rate, and perceived exertion was measured before and following each exercise bout. Muscle soreness, muscle force, and creatine kinase activity was determined preexercise and through 48 h of recovery. Desmin cytoskeletal protein was determined via muscle biopsy of the vastus lateralis before and at 24 h following each exercise. Plasma protein carbonyls (PC) and malondialdehyde (MDA) were measured as biomarkers of oxidative stress. Results: Heart rate and perceived exertion was not different between exercise sessions (P > 0.05), although lactate was higher following sprinting compared with squatting (P = 0.002). Muscle soreness was greater for squatting than sprinting (P = 0.003) and reached a peak immediately postexercise for both sessions (P = 0.0003). Muscle force was unaffected by either exercise session (P > 0.05), and creatine kinase activity was elevated to a similar extent following both sessions. Desmin-negative fibers were virtually nonexistent after either exercise bout, indicating no loss of this cytoskeletal protein. Neither PC nor MDA was affected by the exercise (P > 0.05). Conclusion: These results suggest that in anaerobically trained men, the oxidative stress and muscle injury response to similarly matched anaerobic exercise bouts is minimal, and not different between exercise modes. Furthermore, when compared with previous literature on untrained subjects, the response is significantly attenuated, possibly because of adaptations occurring as a result of chronic, strenuous anaerobic training. Key Words: FREE RADICALS, SPRINTING, SQUATTING, REACTIVE OXYGEN SPECIES

An increase in macromolecule oxidation has been demonstrated following both aerobic and anaerobic exercise of sufficient intensity (4,15,22). It has been suggested that the generation of reactive oxygen and nitrogen species (RONS) and subsequent macromolecule oxidation, leading to a state of oxidative stress, occurs in response to aerobic exercise in large part due to a disturbance in electron transport with the corresponding increase in oxygen consumption. In relation to anaerobic exercise however, other pathways of RONS generation exist (4,13), including ischemic reperfusion conditions, xanthine and NADPH oxidase production, prostanoid metabolism, phagocytic respiratory burst activity, disruption of iron containing proteins, and altered calcium homeostasis. Moreover, the form of anaerobic work (e.g., sprinting vs resistance exercise) may dictate the extent of RONS generation, as resistance exercise often induces muscle injury giving rise to many of the before mentioned RONS generating systems.

Anaerobic exercise, in particular sprint and resistance exercise, is commonly used by athletes and other individuals who regularly train in an attempt to improve speed, strength, and athletic performance. Although this form of exercise is useful for these goals, it often results in an acute state of oxidative stress, in particular in individuals who are untrained (4). Such a condition may prove harmful over time, as chronic oxidative stress has been linked a variety of disease states. Strenuous sprint exercise has demonstrated either an increase (1,18,26) or no change (12) in oxidative stress biomarkers following exercise. Although, Groussard and colleagues (12) did demonstrate an increase in lipid radicals detected by electron spin resonance spectroscopy following a single 30-s cycle sprint. In relation to resistance exercise, oxidized macromolecules have been reported to increase following either full body (19,24) or squat exercise (3), with differences in the magnitude and time course of elevation noted between studies. Specifically, oxidation end products may be elevated 24–48 h following the exercise stimulus, suggesting that skeletal muscle injury and the subsequent biochemical responses to such injury may further contribute to macromolecule oxidation. This is underscored by the observation of oxidative stress in the days following high force eccentric exercise, which is accompanied by skeletal muscle injury (7,16,23).

To date, no study has measured oxidative stress in response to both resistance and sprint exercise within the same subjects. Due to the vast difference in the protocols used to induce oxidative stress, as well as the variety of
assays that have been employed, comparing the response across individual sprint and resistance exercise investigations has proven difficult.

For these reasons, the purpose of this investigation was to measure the oxidative stress response to similarly matched work bouts of sprint and resistance exercise, while at the same time measuring markers of skeletal muscle injury. In this way, we could determine the degree of macromolecule oxidation following two commonly utilized forms of anaerobic work. We hypothesized that both forms of exercise would result in an immediate postexercise elevation in oxidative stress biomarkers, with the increase possibly following a longer time course for resistance exercise due to the potential impact of muscle injury and further RONS generation. We chose to use cycle sprints as opposed to track (running) sprints, in an attempt to eliminate eccentric muscle actions and subsequent muscle injury. Therefore, we used two common modes of anaerobic work: one that likely would involve muscle injury and one that likely would not. If increases in oxidative stress variables were noted following these two forms of exercise, appropriate treatments (i.e., antioxidant therapy) may be recommended.

METHODS

Subjects

Twelve anaerobically trained men volunteered for this experiment. All subjects were free of orthopedic and metabolic conditions that could have affected their performance or the variables of measurement. All subjects were currently involved in anaerobic exercise with no interruptions in training over the past 6 months. Specifically, subjects performed resistance exercise using moderate rest intervals (e.g., 2–3 min) and moderate volume. No participants were competitive bodybuilders or powerlifters and none performed frequent cycle or track sprints, and are best classified as recreationally trained. Most subjects also performed aerobic exercise on a recreational basis (e.g., moderate-intensity exercise 1–3 d wk⁻¹). Subjects were required to lift ≥ 1.5 times their body mass in the barbell back squat exercise to be considered for enrollment. No subject used antioxidant supplements or tobacco products. Table 1 provides the descriptive characteristics of the subjects. All experimental procedures were performed in accordance with the policy statement of the American College of Sports Medicine on research with human subjects as published by Medicine & Science in Sports & Exercise® and were approved by the university human subjects committee. All subjects provided both verbal and written consent prior to participating.

Screening and Familiarization

One to 2 wk prior to the exercise sessions, anthropometric measurements were obtained on all subjects. Subjects then performed a one-repetition maximum (1RM) test for the barbell back squat exercise using free weights. The maximum amount of weight that could be lifted one time using proper form, depth (posterior thigh parallel to floor), and speed (5-s eccentric phase) was recorded as the 1RM, and was used to calculate the weight to be used during the squat session. The barbell squat was chosen as the resistance exercise due to the large degree of muscle mass recruited, the similar nature of muscle mass activated as compared with sprinting, in addition to the fact that we have previously shown this form of exercise to increase oxidative stress (3).

Following assessment of 1RM, subjects performed a familiarization set of barbell squats for 10 repetitions using a load equal to 50% 1RM. The movement speed was fixed at 5 s for the eccentric phase (via metronome and verbal counting by the investigators) with an explosive concentric phase. The depth of squatting was fixed for each subject (posterior thigh parallel to floor) by placing an adjustable box under the hips that subjects were required to lightly touch during each descent prior to performing the concentric phase. These same procedures were used during the squat session described below. Familiarization for the maximal isometric force assessment, as described below, was performed next. This involved the performance of three submaximal and three maximal attempts. Following these tests, bar displacement was measured for each subject to allow for the calculation of work performed during each repetition of the squatting sessions.

Vertical bar displacement was measured relative to the ground using a linear transducer (Unimeasure, Corvallis, OR) with an accuracy of 0.01 cm. Subjects performed four barbell squats to a predetermined depth. Displacement-time data were sampled at 1000 Hz using a Data Pac 2K acquisition and analysis software program (Runtech, Mission Viejo, CA). Total displacement from starting position to bottom squat position (eccentric phase) was measured. Mean displacement across the four repetitions was recorded for each subject and then the average was entered in the following equation to determine the approximate physiological demand that would be performed for each repetition:

\[
\text{work (kJ)} = 1.33 \times \text{displacement (m)} \times \frac{[\text{BM (kg)} \times 0.88 + \text{load (kg)}]}{9.81}
\]

where 1.33 = concentric + eccentric, BM = body mass, and \(-9.81 \text{ m s}^{-2}\) = acceleration due to gravity.

Because the squat is performed in a fixed position, work from the lower extremities (knee to foot) was not considered in this equation (29). As described elsewhere, physiological

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
</tr>
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<tbody>
<tr>
<td>Age (yr)</td>
<td>23 ± 3</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>175 ± 5</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>82 ± 12</td>
</tr>
<tr>
<td>Percent body fat</td>
<td>13 ± 5</td>
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<tr>
<td>Squat one-repetition maximum (kg)</td>
<td>134 ± 45</td>
</tr>
<tr>
<td>Anaerobic training exercise (h wk⁻¹)</td>
<td>5.2 ± 2.4</td>
</tr>
<tr>
<td>Leg training exercise (h wk⁻¹)</td>
<td>2.5 ± 1.4</td>
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work was performed only on 88% of the body (10) and the concentric and eccentric components were collectively equal to 1.33 rather than 2, because less work is done during eccentric muscle actions (9). In this way, the amount of physiological work per repetition could be calculated and used to determine the total of number of repetitions needed to be performed in order to equal the total amount of work done during the sprint cycling session.

Following the squat familiarization, subjects rested for 10 min and then performed a familiarization trial for the cycle sprint. This included a maximal effort performed on a Lode Excalibur Sport cycle ergometer (Lode B.V. Medical Technology, Groningen, The Netherlands) at a torque factor equal to 0.7 times body weight. The duration of the cycle sprint was 10 s, and subjects warmed up at a low intensity (100 W) for 3 min before completing the sprint. These same procedures were used during the sprint cycling session described below.

Testing

For both exercise sessions, subjects reported to the lab in the morning following an overnight fast (8–12 h). Subjects were instructed not to perform any strenuous exercise for the 48-h period preceding the sprint cycling and squat sessions. All subjects performed the sprint cycling sessions first in order to avoid any potential disruption in the activated muscle tissue that may have occurred and persisted if subjects were to perform the squat session first. It was believed that the concentric only nature of the sprint exercise would not lead to any significant muscle injury, especially in our sample of trained men.

Following a warm-up period of 5 min, subjects performed six 10-s maximal sprints, with 3 min of recovery between each. Subjects were instructed to pedal as fast as possible throughout the 10-s periods and were provided with verbal encouragement by the investigators during each sprint. Following each sprint, subjects pedaled lightly against minimal resistance until they approached the next bout. They were encouraged by the investigators during each sprint. Following the squat familiarization, subjects rested for 10 min and then performed a familiarization trial for the cycle sprint. This included a maximal effort performed on a Lode Excalibur Sport cycle ergometer (Lode B.V. Medical Technology, Groningen, The Netherlands) at a torque factor equal to 0.7 times body weight. The duration of the cycle sprint was 10 s, and subjects warmed up at a low intensity (100 W) for 3 min before completing the sprint. These same procedures were used during the sprint cycling session described below.

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Subjects performed the squat exercise 14 d after the sprint session in sets carried to a point of momentary muscular failure using 70% 1RM, with 3 min of rest between each set. The number of repetitions was determined based on the total amount of work performed during the sprint session. Using this method, the number of sets performed by subjects was 6.4 ± 0.28 (mean ± SEM) and the number of repetitions was 40 ± 2. The total physiological work performed was equal to 46,310 ± 2,809 kJ. Prior to the squat, subjects warmed up by performing one set of five repetitions using a load equal to 40, 50, and 60% 1RM. Subject form (both speed and depth of squatting) was monitored throughout, and subjects were provided the same verbal encouragement as during the sprint sessions. Heart rate and RPE was recorded immediately at the end of each set of squats.

Blood Collection and Analysis

Prior to both exercise sessions and following a 10-min quiet rest period, a 7-mL blood sample was taken via vacutainer from an antecubital vein. Additional blood samples were taken immediately, 30 min, 24 h, and 48 h following exercise. Blood samples were analyzed for whole-blood lactate (only preexercise, immediately, and 30 min postexercise) using an Accutrend portable lactate analyzer (Roche Diagnostics, Mannheim, Germany). Hematocrit and hemoglobin were measured to correct for plasma volume shifts following the procedures of Dill and Costill (11). The remainder of whole blood was separated immediately to plasma and stored in multiple aliquots at −80°C to be used for the measurement of creatine kinase activity and oxidative stress biomarkers.

Creatine kinase activity was measured spectrophotometrically using a reagent purchased from StanBio Labs (CK-NAC, UV-Rate). Protein carbonyls (PC), a measure of protein oxidation, were analyzed using an ELISA according to the procedures recommended by the manufacturer (Zentech Technology, Dunedin, New Zealand). Malondialdehyde (MDA), a measure of lipid peroxidation, was analyzed using a commercially available colorimetric assay (Northwest Life Science Specialties, Vancouver, WA), using the modified method described by Jentzsch et al. (14).

Muscle Soreness and Force Assessment

At all blood collection time points, measurements of muscle soreness and force were made. Subjects completed a subjective rating of muscle soreness felt during body weight only squatting using a 10-cm visual analog scale as previously described (8). Isometric squat measurements were performed within a modified power rack, customized to accommodate one-inch hole spacing for individual bar height adjustments. Subjects were positioned in a manner by which the knee joint angle was 140°. The joint angle, foot placement, and bar height were recorded and reproduced for subsequent testing sessions. In addition, subjects were asked to perform tests with the same footwear throughout testing.

Subjects were in constant foot contact with a modified commercial-grade floor scale (Rice Lake Weighing Systems; Rice Lake, WI). Encased within the floor scale were four load cells providing vertical ground reaction force data. A 0- to 10-V dc signal from the scale was channeled through an analog-to-digital conversion system (Measurement Computing; Middleboro, MA) and interfaced with a Pentium IV computer. Data acquisition and analysis was performed on
Datapac2K2 software (Runtech, Mission Viejo, CA). Force was sampled at 1000 Hz and low-pass digitally filtered using a fourth-order Butterworth filter (20 Hz).

Each subject was instructed to contract as fast and as hard as possible during maximal attempts. Two warm-up trials of 50 and 75% effort were conducted prior to performing three maximal attempts held for 3–5 s. Subjects were given 1 min of rest between maximal attempts. Peak rate of force development was determined by taking the first derivative of the force signal with respect to time. Average rate of force development is the change in force divided by the change in time for the rise phase of the force–time curve (27).

**Muscle Biopsies**

Muscle biopsies of the vastus lateralis were performed 2 wk before (baseline) and 24 h following both exercise bouts. Biopsies were only obtained on 8 of the 12 subjects. Loss of immunostaining of the intermediate filament protein desmin was determined on all samples as a marker of cytoskeletal disruption as previously suggested (2). In addition, muscle fiber type was analyzed to determine if selected fiber populations were more prone to desmin loss.

Biopsy procedures were identical to methods previously described (27). Briefly, muscle tissue samples (80–100 mg) were extracted from the vastus lateralis muscle, oriented in tragacanth gum, frozen in isopentane cooled by liquid nitrogen to −159°C, and stored at −80°C. To ensure adequate sample sizes, large pieces were obtained using a double-chop method combined with suction. A mean of 866 ± 66 (mean ± SEM) fibers were obtained from these biopsies. The frozen biopsy samples were thawed to −20°C and serially sectioned (12 μm thick) for fiber type and desmin analyses.

Desmin analysis. These procedures were modified from those reported by Barash et al. (2). Briefly, muscle cross-sections were fixed in a 4% formaldehyde solution, followed by incubation in a blocking solution. Sections were then labeled with a 1:100 dilution of a monoclonal 1° antibody (NCL-DES11, Vector Laboratories, Burlingame, CA), followed by incubation in an avidin-biotin solution (Vectastain Elite ABC, Vector Laboratories). Desmin-positive fibers were visualized with a 3,3-diaminobenzadine substrate solution for peroxidase (SK-4100, Vector Laboratories), dehydrated via a series of ethanol rinses, and mounted on slides for analysis.

Fiber type analysis. Routine myofibrillar adenosine triphosphatase (mATPase) histochemical analysis was performed using a preincubation pH value of 4.3 (5) to determine the types I and II fiber type distributions. Computerized images of the fiber sections were analyzed using Scion Image for Windows (Beta 4.0.2). When compared with the desmin stained sections, this procedure would permit determination of fiber type–specific desmin degradation.

**Dietary Records**

All subjects were instructed to maintain their normal diet during the study period and to complete daily food records to allow for nutrient intake assessment between the days surrounding each protocol. Records were kept for the 3 d prior to each exercise session and for the 2 d following each session. All diet records were analyzed by the same investigator for total kilocalories, protein, carbohydrate, fat, vitamin C, vitamin E, and vitamin A intake using Diet Analysis Plus software (ESHA Research, Salem, OR).

**Statistical Analyses**

Oxidative stress, muscle soreness, and muscle force data were analyzed using a 2 (exercise session) by 5 (time) ANOVA. Lactate data were analyzed using a 2 (exercise session) by 3 (time) ANOVA. Heart rate and RPE data were analyzed using a 2 (exercise session) by 2 (time) ANOVA. Dietary data were analyzed using a one-way ANOVA. All analyses were performed using JMP statistical software version 4.0 (SAS Institute, Cary, NC). Statistical significance was set at \( P < 0.05 \). The data are presented as mean ± SEM, except for subject characteristics and dietary data, which are presented as mean ± SD.

**RESULTS**

The mean daily calories, protein, carbohydrate, fat, vitamin C, vitamin E, and vitamin A intake did not differ between exercise sessions (\( P > 0.05 \)). These data are presented in Table 2. Blood lactate was not different at rest (1.49 ± 0.09 vs 1.48 ± 0.09 mM·L\(^{-1}\)) between sprinting and squatting sessions, respectively, but was greater immediately (10.3 ± 0.51 vs 7.9 ± 0.54 mM·L\(^{-1}\)) and at 30 min postexercise (6.0 ± 0.56 vs 3.3 ± 0.24 mM·L\(^{-1}\), \( P = 0.002 \)) for sprint cycling compared with squatting. The mean heart rate response (161 ± 2.8 vs 164 ± 2.7 bpm) and RPE (14.9 ± 0.48 vs 15.1 ± 0.58) were not statistically different between sprint cycling and squatting, respectively (\( P > 0.05 \)).

Muscle damage markers. Although no interaction was noted (\( P > 0.05 \)), both an exercise session (\( P = 0.003 \)) and a time main effect (\( P = 0.0003 \)) was noted for muscle soreness, with values greater for squatting than for sprint cycling and reaching a peak immediately postexercise (4.5 ± 0.61 vs 4.3 ± 0.63, respectively). Maximal isometric muscle force was not affected by either session at any time, nor was mean or peak rate of force development (\( P > 0.05 \)). A time main effect (\( P = 0.048 \)) was noted for creatine kinase activity, with values peaking for both exercise sessions at 48 h.
postexercise (squat = 193 ± 33 U·L\(^{-1}\) and sprint = 166 ± 27 U·L\(^{-1}\)) from preexercise values of 101 ± 14 U·L\(^{-1}\) and 115 ± 18 U·L\(^{-1}\) for squat and sprint exercise, respectively. Fiber type analysis indicated that 45.9 ± 1.3\% of muscle fibers were type I, and 54.1 ± 1.3\% were type II. Desmin was virtually unaffected by either exercise bout, with all fibers, both fast and slow, appearing to retain positive staining for desmin. Figure 1 shows a representative sample from baseline and 24 h postexercise for both squatting and sprinting.

**Oxidative stress biomarkers.** There was no difference in preexercise PC between exercise sessions, with values ranging from 0.04 to 0.05 nmol·mg\(^{-1}\) protein. Whereas PC appeared to increase to a slightly greater extent following squat exercise, this did not reach statistical significance (\(P = 0.447\), Fig. 2; standardized effect size = 0.308). For MDA, there was no difference in preexercise values between exercise sessions, with values ranging from 0.7 to 1.0 \(\mu\)mol·L\(^{-1}\). There was a trend for a decrease in MDA following exercise (\(P = 0.0672\)), with no differences noted between exercise sessions (standardized effect size = 0.149, Fig. 3).

**DISCUSSION**

The findings of the present investigation refute our initial hypotheses. That is, oxidative stress biomarkers were not elevated to any significant extent following either exercise bout, and neither exercise bout caused a significant increase...
in markers of muscle injury, with the exception of an acute rise in muscle soreness. Based on these findings, we conclude that in anaerobically trained men, strenuous bouts of either squat or cycle sprint exercise as performed here are not associated with an increase in oxidative stress and have minimal impact on markers of skeletal muscle injury. Therefore, men who are well trained and who perform relatively low-volume strenuous anaerobic exercise should not be concerned about increased oxidative stress because such a condition appears to be nonexistent in this population.

Previous studies using sprint cycling exercise have demonstrated an increase in oxidative stress using recreationally (1,2,6,28) or highly trained subjects (18). Others have reported no increase in oxidative stress biomarkers such as MDA following sprinting (12). Explanations for these conflicting results may include differences in the protocols used to induce an oxidative stress in addition to the actual degree of subject training.

The protocol used in the present study included six 10-s sprints performed on a cycle ergometer. Thompson et al. (28) used a 90-min shuttle run, involving intermittent sprinting, jogging, and walking. Schiff et al. (26) had subjects perform two sprints until they “felt exhausted,” whereas Marzatico et al. (18) had subjects perform six 150-m sprints on a track, as opposed to the non-weight-bearing cycle ergometer sprints used in the present study. Moreover, with the exception of the Marzatico et al. (18) study, which used sprint-trained athletes, subjects have simply been described as “recreationally active” or “active in sports.” With such a vague description of subjects’ past and current training status, it is difficult to know their potential for adaptations that may allow protection against oxidative stress (21) and muscle injury (20). The majority of subjects in the present study had several years of continuous anaerobic exercise training experience, spending an average of over 5 h wk⁻¹ performing anaerobic exercise and 2.5 h wk⁻¹ on leg exercise alone. Such training may have allowed for adaptations that could have led to the minimal change in oxidative stress, as well as muscle injury, following exercise. We believe that this factor helps explain our results. It should also be noted that although our subject pool was considered “trained,” the level of conditioning may differ from other individuals based on their training history (e.g., set and rep patterns, rest interval length, duration and intensity of training). This needs to be considered when generalizing these findings. In fact, if subjects had been performing sprint exercise regularly as part of their training regimen, they may have been protected more so.

Through pilot testing, it was determined that 10-s sprints were optimal because they allowed subjects to achieve a significant anaerobic stimulus, as evidenced by increased blood lactate, while allowing subjects to actually complete all six sprints. When increasing the sprint duration to even 15 s, we found that many individuals could not complete all six sprints and became ill during the exercise. To avoid this occurrence during testing, we decided to include the 10-s duration. We also decided on this duration because it is similar to a 100-m sprint in a well-trained individual.

As with the sprint exercise, the squat exercise failed to result in any significant increase in oxidative stress (Figs. 2 and 3). We previously reported (3) an increase in PC following repeated squatting using a load equal to that used in the present study (i.e., 70% 1RM). In this study, the increase in PC from preexercise was approximately two-fold at 24 h postexercise, compared with the 1.6-fold increase in the present study. However, the average number of sets performed was 13, roughly double that performed in the present study, with the average number of reps per set, heart rate, and RPE being nearly identical. Thus, in comparing these two studies alone, it appears that in relation to squat exercise, exercise volume may influence the oxidative stress response. This is especially true considering the lack of detectable muscle injury in the present study, which potentially would have been greater given a higher volume of work. Considering the trained status of our subjects, it is likely that greater volume, the inclusion of pure eccentric muscle actions as opposed to traditional concentric/eccentric actions, and/or a greater intensity of stimulus may be required to induce oxidative stress and muscle injury. Although muscle soreness and creatine kinase activity was reported to be elevated following exercise, other more sensitive measures of muscle injury, including desmin cytoskeletal protein and muscle force, were unaffected. This finding should be considered when selecting dependent variables in future research focused on muscle damage resulting from strenuous exercise.

Prior studies using pure eccentric exercise have resulted in either an increase (7,16,23) or no change (6,17,25) in oxidative stress biomarkers. All of these studies have reported an increase in certain markers of muscle injury (e.g., creatine kinase activity, muscle soreness, force decrement), although there are no uniform findings related to these increases and changes in oxidative stress markers. Moreover, in the studies that have demonstrated an increase in muscle injury with a concurrent increase in oxidative stress, a cause and effect relationship has not been established. Due to the lack of increase in oxidative stress biomarkers in the present study, relationships could not be established. As stated previously, it is likely that in a population of anaerobically trained men, the
exercise stress needs to be more extreme to induce increases in oxidative stress and muscle injury. For example, athletes engaged in multiple daily sessions of training may experience a more pronounced response than the subjects in the present study, who were exposed to a relatively low volume of exercise stress. Certainly, more work is needed in this area to determine the association and possible role of muscle injury and oxidative stress within skeletal muscle.

In summary, we failed to demonstrate an increase in oxidative stress in anaerobically trained men. Furthermore, neither squat nor sprint cycling exercise resulted in significant muscle injury. Therefore, we conclude that in this population of well-trained men, strenuous bouts of squat or cycle sprint exercise are not associated with an increase in oxidative stress and have minimal impact on markers of skeletal muscle injury. This may be attributed to the training status of the research subjects, the relatively low-volume nature of the exercise protocols, or a combination of these factors. As such, trained individuals involved in acute, low-volume, strenuous anaerobic exercise should not be concerned about increased oxidative stress because such a condition does not seem apparent in this population.

REFERENCES