Effect of Muscle Oxygenation during Resistance Exercise on Anabolic Hormone Response

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ABSTRACT


Purpose: The mechanisms that underlie the affect of acute program variables on muscle growth and strength development for strength/power athletes have been of great interest. This investigation examined the affects of two different resistance exercise protocols on muscle oxygenation, and the anabolic hormonal response to such exercise.

Methods: Eleven experienced resistance-trained male athletes performed four sets of the squat exercise using either a low-intensity, high-volume (LI; 15 repetitions at 60% one-repetition maximum [1-RM]) or high-intensity, low-volume (HI; 4 repetitions at 90% 1-RM) load. Venous blood samples were obtained before (Pre), immediate (IP), 20- (20P), and 40-min (40P) postexercise. Continuous-wave near-infrared spectroscopy was used to measure oxygen desaturation during exercise.

Results: No differences in muscle deoxygenation were seen between LI and HI. However, time-dependent postexercise reoxygenation was significantly different between the two exercise sessions (35.3 ± 17.4 s vs 24.5 ± 14.3 s in LI and HI, respectively). Testosterone and growth hormone (GH) concentrations were significantly elevated from Pre at IP, 20P, and 40P in both LI and HI. GH concentrations were higher (P < 0.05) for LI than at HI at 20P and 40P.

Conclusion: Muscle oxygen recovery kinetics appeared to be influenced by differences in the intensity and volume of exercise, and delayed reoxygenation appears to affect the GH response to exercise.

Key Words: TESTOSTERONE, GROWTH HORMONE, WEIGHT TRAINING, NEAR-INFRARED SPECTROSCOPY, MUSCLE ISCHEMIA

During aerobic exercise, blood flow and oxygen delivery to working muscle is generally increased to meet the demands of exercise (23). However, as exercise intensity increases, intramuscular forces become elevated, resulting in an attenuation of exercise hyperemia (4,5,24,26). As a consequence, increases in lactic acid and intramuscular oxygen desaturation are observed. The relationship between restricted blood flow, lactic acid, and oxygen desaturation within the exercising muscle has been demonstrated (9,24). Although it is generally recognized that exercise resulting in high force production (i.e., resistance exercise) will result in a reduction of blood flow to exercising muscle (22), studies examining this mode of exercise are limited. Moreover, most studies investigating blood flow in the context of resistance exercise have used a static model (i.e., isometric exercise), despite the fact that a majority of resistance-training programs utilize dynamic movements with large variations in training intensity and volume.

The manipulation of these training variables appears to have important implications on the physiological response to resistance exercise. Kraemer and colleagues (13,14) have shown that hormonal responses to acute heavy resistance exercise are sensitive to changes in these variables (e.g., rest intervals, exercise intensity, and volume) in both men and women. Specifically, significantly higher testosterone concentrations have been observed when rest periods between sets are reduced (3 to 1 min), or when the intensity of exercise is decreased (5 repetition maximum [RM] to 10-RM) and volume is increased (14). Similarly, circulating immunoreactive growth hormone concentrations increase in response to an acute bout of resistance exercise, and appear to be greatest when training protocols of moderate intensity (10-RM vs 5-RM) and short rest periods (1 min vs 3 min) are utilized (13). The elevations in anabolic hormonal response to manipulations of the acute program variables may explain, in part, the variability in muscle hypertrophy observed with different resistance training programs. For example, bodybuilders typically incorporate high-volume, moderate-intensity training programs with short rest periods.

Recent research has suggested that muscle ischemia caused by vascular occlusion appears to enhance the hormonal response to exercise (30). The extent and duration of
ischemia may ultimately determine the extent of tissue damage (8) that occurs consequent to a specific stimulus, such as heavy resistance exercise. In addition, muscle damage that occurs as a result of heavy resistance exercise is thought to be requisite for muscle hypertrophy and increases in strength (16). Thus, it would appear that resistance-training programs designed to invoke a greater extent and duration of muscle ischemia would result in increased muscle hypertrophy. Therefore, the purpose of this study is to compare the effects of a high-intensity, low-volume with a low-intensity, high-volume exercise paradigm on muscle oxygenation during resistance exercise. In addition, the relationship between muscle oxygenation and the anabolic hormonal response to resistance exercise was examined. The hypothesis of this study is that exercise programs that elicit a greater degree of muscle ischemia will result in a greater anabolic hormonal response to exercise.

METHODS

Subjects. Eleven experienced resistance-trained men (20.8 ± 1.3 yr; 96.2 ± 14.4 kg, 182.4 ± 7.3 cm) served as subjects for this investigation. After an explanation of all procedures, risks, and benefits each subject gave his written informed consent to participate in this study. The Institutional Review Board of the College approved the research protocol. Each subject had 6.9 ± 0.8 yr of resistance training experience (range 6–8 yr), had a 1-RM squat of 184.5 ± 16.5 kg, and was free of any musculoskeletal and endocrine disorders. In addition, all subjects reported being free of anabolic steroid use during the previous year and were not taking any other ergogenic supplementation during the 6 wk before the study.

Testing procedure. Each subject reported to the Human Performance Laboratory on three different occasions. During the initial visit, subjects performed a 1-RM strength test for the squat exercise. Each subject performed a warm-up set using a resistance that was approximately 40–60% of his perceived maximum, then performed three to four subsequent attempts to determine the 1-RM with 3–5 min of rest between each lift. During the next two sessions, each subject performed a resistance exercise protocol consisting of five sets of the parallel squat exercise. The first set was used as a warm-up set. For each testing session both intensity and volume of exercise differed. Subjects exercised using either a light-intensity, high-volume (LI) (15 repetitions with 60% of 1-RM), or high-intensity, low-volume (HI) (4 repetitions with 90% of 1-RM) exercise protocol during each testing session. The exercise protocol was randomly assigned to prevent an order effect. Rest intervals between sets were 3 min for both exercise sessions. Each testing session was separated by at least 72 h and occurred at the same time of day to account for diurnal variation in hormonal measurements.

Hemoglobin/myoglobin muscle tissue deoxygenation procedure. A 16-channel continuous wave near infrared spectroscopy (cwNIRS; NIMS, Philadelphia, PA) was used to measure changes in hemoglobin (Hb)/myoglobin (Mb) muscle tissue oxygenation/deoxygenation (oxy/deoxy-Hb/Mb) in the capillary bed of the vastus lateralis muscle at rest, during each set, and for 3 min postexercise. After each training session, cuff ischemia (CI) was performed to obtain maximal oxy/deoxy-Hb/Mb. Linearity of NIRS technology has been previously validated in other studies (7,19), and validity for cwNIRS has been recently established (15). The NIRS unit included a probe consisting of two LED light sources and eight photodiode detectors. An 11- × 7-cm optical probe with a 3-cm source-to-detector separation was positioned on the lateral aspect of the right vastus lateralis at a location equidistant between the greater trochanter of the femur and the muscle’s distal connection to the quadriceps tendon. The location of the probe was recorded by noting distances from the anatomical landmarks to ensure identical probe placement during all trials. Wavelengths of 730 and 850 nm were used to monitor relative change in oxy/deoxy-Hb/Mb. Oxy-Hb/Mb of muscle tissue was estimated by changes in the difference in signal strength at those wavelengths. Data acquisition sampling frequency was set at approximately 2 Hz. Relative oxy/deoxy-Hb/Mb (% oxy/deoxy-Hb/Mb) for each exercise period was calculated by taking the difference between baseline at rest and the greatest oxy/deoxy-Hb/Mb that occurred during exercise, and dividing by the maximal oxy/deoxy-Hb/Mb obtained during CI. To obtain CI, a thigh cuff was placed proximal to the probe and inflated to 260 torr for 8 min or until a plateau for deoxy-Hb/Mb was reached. CI was administered while subjects were seated in a semirecumbent position with the instrumented leg extended horizontally and supported. Deoxy-Hb/Mb values from the baseline, exercise, and CI were obtained by taking 10-s averages.

Recovery kinetics and rates after each exercise session were also determined using the cwNIRS. Half-time recovery (T1/2) was ascertained by determining the time necessary to achieve to 50% of maximal reoxy-Hb/Mb after exercise cessation (4).

Blood measurements. During each experimental session blood samples were obtained preexercise (PRE), immediately postexercise (IP), 20 (20P), and 40 min (40P) postexercise. All blood samples were obtained using a 20-gauge Teflon cannula placed in a superficial forearm vein. The cannula was maintained patent using an isotonic saline solution placed in a three-way stopcock with a male luer lock adapter. PRE blood samples were drawn after a 15-min equilibration period before exercise. IP blood samples were taken within 30 s of exercise cessation. All blood samples were drawn with a plastic syringe while the subject was in a seated position.

After collection, blood was transferred to a Vacutainer® tube containing SST® Gel and Clot Activator. The blood was allowed to clot at room temperature and subsequently centrifuged at 1500 g for 15 min. The resulting serum was placed into separate 1.8-mL microcentrifuge tubes and frozen at −80°C for later analysis of total testosterone and 22-kDa growth hormone.

Biochemical and hormonal analyses. Serum total testosterone and 22-kDa growth hormone concentrations were determined using enzyme immunoassay and enzyme-linked
immunosorbent assay, respectively (Diagnostic Systems Laboratories, Webster, TX). Determinations of serum immunoreactivity values were made using a SpectraMax340 Spectrophotometer (Molecular Devices, Sunnyvale, CA). To eliminate interassay variance, all samples for a particular assay were analyzed in the same assay run. All samples were run in duplicate with a mean intra-assay variance of < 10%. The detection limit of the testosterone and growth hormone assays were 0.14 nmol·L⁻¹ and 0.3 µg·L⁻¹, respectively. Lactate concentrations were determined with an Analox GM7 enzymatic metabolite analyzer (Analox Instruments U.S., Lunenburg, MA).

Statistical analysis. Data were analyzed using a two-way ANOVA. In the event of a significant F-ratio, post hoc comparisons using the Bonferroni method were applied to determine pairwise differences. In addition, paired Student’s t-tests were used to analyze the area under curve (AUC), which was calculated by using a standard trapezoidal technique. A criterion alpha level of P ≤ 0.05 was used to determine statistical significance. A sample size of 11 subjects provided > 80% statistical power at an α level of 0.05 (two-tailed).

RESULTS

During LI all subjects completed the 15 required repetitions per set except one subject who completed only 12 repetitions during his last set of exercise. During HI, 9 of the 11 subjects were able to complete the four repetitions required per set. One subject was only able to complete three repetitions in his last set, and another subject was only able to complete 5 of the 16 total repetitions that were required.

The duration of exercise per set was significantly (P < 0.05) greater during LI (41.6 ± 6.6 s) than in HI (21.4 ± 3.6 s). However, the extent of muscle deoxygenation (expressed as a percent of maximal desaturation obtained during CI) between the two exercise sessions was not statistically significant. During LI muscle deoxygenation reached 72.7 ± 18.0%, whereas HI muscle deoxygenation reached 79.9 ± 13.4%.

After exercise, muscle reoxygenation appeared to be delayed during both training sessions. However, the duration of the delay was significantly (P < 0.05) different between HI and LI (24.5 ± 14.3 s and 35.3 ± 17.4 s, respectively). Once reoxygenation began the T1/2 recovery was not different between the two training sessions (50.2 ± 15.5 s and 51.7 ± 16.8 s in HI and LI, respectively).

Lactate concentrations at IP were significantly (P < 0.05) elevated above resting levels after both HI and LI testing conditions. However, lactate concentrations for LI was significantly (P < 0.05) higher at that time point than for HI (15.3 ± 2.3 mmol·L⁻¹ and 9.7 ± 3.3 mmol·L⁻¹, respectively).

The serum testosterone response to the two training sessions is shown in Figure 1. Testosterone concentrations were significantly (P < 0.05) elevated from PRE at IP, 20P, and 40P in both HI and LI. However, no significant differences were seen between exercise sessions. AUC analysis also failed to demonstrate any significant difference between exercise sessions.

The serum growth hormone response to the two training sessions is shown in Figure 2. Growth hormone concentrations were significantly (P < 0.05) elevated from PRE at IP, 20P, and 40P in both HI and LI. However, growth hormone concentrations were significantly (P < 0.05) higher for LI than HI at 20P and 40P. In addition, AUC for GH was significantly (P < 0.05) higher during LI than in HI.

DISCUSSION

The purpose of this study was to examine the effect of training intensity on muscle oxygenation, and to examine the relationship between muscle deoxygenation and the anabolic hormonal response. The results of this study show no apparent difference between resistance exercise protocols of low-intensity, high-volume and high-intensity, low-volume on muscle deoxygenation. However, the duration of exercise appears to have had a greater influence on the deoxygenation and the delay in postexercise muscle reoxygenation. Although both training intensities result in similar deoxygenation values, exercise performed at the lower intensity was performed for a significantly longer duration. Although previous research has shown that resistance training will reduce tissue oxygenation within contracting skeletal muscle (28), this was the first study to quantify the extent of muscle deoxygenation during such exercise.
Interestingly, the extent of muscle deoxygenation during resistance exercise appears similar to values previously reported in speed skaters skating in a low position (5), alpine skiers during a simulated giant slalom event (26), and sprinters performing the Wingate anaerobic power test (21). Deoxygenation of skeletal tissue during exercise is a function of a change in blood flow and intramuscular pressure (25). Intramuscular pressure is known to increase linearly with increases in the force of muscle contraction, which occurs as a result of increased tension in activated muscle fibers (2,11). As intramuscular pressure increases, blood perfusion to the activated skeletal muscle is reduced. This appears to begin when muscle contraction exceeds 35–50% of its maximal force capability (10). In this study, resistance exercise was performed at 60% and 90% of the subject’s 1-RM and would be expected to cause elevated intramuscular pressures. Thus, the extent of muscle deoxygenation observed during each testing session was not surprising. Even though there appeared to be a tendency for greater tissue deoxygenation after HI than LI, this 7% difference in muscle deoxygenation was not statistically different.

Previous research has reported a moderate correlation \( r = 0.70, P < 0.05 \) between lactic acid concentrations and the rate of muscle deoxygenation during submaximal aerobic exercise (18). However, despite a higher lactate concentration seen after LI \( (P < 0.05) \), there was no significant difference between the two exercise intensities in muscle deoxygenation. In addition, no correlations \( (r < 0.10) \) were observed in this study between muscle deoxygenation and lactate acid concentrations in both LI and HI. Apparently the high intramuscular pressures, as well as the relatively short duration of exercise resulting from heavy resistance exercise, has a much greater impact on muscle deoxygenation than lactate concentrations. It is possible that the higher lactate concentrations seen during LI had a greater impact on muscle reoxygenation postexercise.

A unique aspect to this study was the difference seen in reoxygenation between LI and HI. Despite similar deoxygenation levels during each training session, there was a 44.1% longer delay in the start of reoxygenation after LI compared with HI. The phenomenon of delayed reoxygenation, as a function of intensity and volume during resistance exercise, has not been previously seen. It is likely that the higher lactate accumulation seen during LI facilitated a greater \( O_2 \)Hb dissociation (Bohr effect). In addition, previous research has also demonstrated that \( O_2 \)Mb dissociation will follow a similar pattern of \( O_2 \)Hb dissociation when exercise intensity is maximal (4,19). Considering that subjects were exercising at intensities that approximated their RM for each exercise intensity, it appears that the duration of exercise performed at a maximal effort may be more important than the relative intensity of exercise in affecting muscle oxygen recovery kinetics. In addition, the greater metabolic acidosis seen during LI may have resulted in a \( \alpha_2 \)-adrenoreceptor-mediated constriction of arteriole microvessels. McGillivray-Anderson and Faber (20) have demonstrated that \( \alpha_2 \)-adrenoreceptors on terminal arterioles are highly sensitive to tissue acidosis.

The athletic background of these subjects may have also contributed to the postexercise delay in muscle reoxygenation. All of the subjects in this study were anaerobic athletes. Though speculative, it is likely that the fiber composition of these athletes was primarily of the Type II variety. Previous research has shown that such fibers do not demonstrate the level of postexercise hyperemia seen in slow-oxidative and fast-oxidative fibers common in more aerobic athletes (17). Therefore, it is possible that the delay in reoxygenation after high-intensity resistance exercise may also be a function of fiber type composition and associated vasculature density.

Another purpose of this study was to investigate whether differences in muscle oxygenation as a result of exercise intensity will influence the hormonal response to such exercise. Previous studies have indicated that under ischemic conditions the hormonal response to exercise may be enhanced (27,30). However, in these studies the ischemic conditions were examined via vascular occlusion. This is the first study known that has examined the affect of muscle deoxygenation occurring as a natural response to the exercise stress on the hormonal response to resistance exercise. The results attained in this study confirm previous investigations that have demonstrated that growth hormone (27,30), but not testosterone (30), concentrations are influenced by tissue ischemia in actively contracting muscle.

The prolonged ischemic response seen after LI, reflected by the greater delay in muscle reoxygenation, resulted in a greater growth hormone response than that seen after HI. In
addition, lactate concentrations were 58% higher after LI than after HI, presumably a result of the local tissue hypoxia caused by elevated intramuscular pressure. Growth hormone secretion patterns have been shown to be quite responsive to changes in the acid-base balance of muscle (6). Increases in intramuscular acidity can stimulate sympathetic nervous activity through a chemoreceptive reflex that is mediated by intramuscular metaboreceptors (29). Thus, it appears that the duration of acute hypoxia and the accumulation of metabolites during resistance training are important stimuli for the growth hormone response.

The importance of acute program variables such as training intensity and volume on the hormonal response to exercise has been demonstrated (13,14). Although the response of growth hormone in this study supports these previous investigations, the response of testosterone to differences in these training variables did not. Similar to growth hormone, testosterone is responsive to changes in acute program variables (13,14). Generally, resistance exercise of moderate intensity (i.e., 10-RM) appears to induce a significantly greater testosterone response than exercise of higher intensity (i.e., 5-RM) (13,14). However, when the hormonal response was examined using AUC analysis, no significant differences were seen (14). Our results confirm these findings.

Another factor that may have contributed to the similar response of testosterone in this study was the effect of the subject’s training and athletic experience. Previous studies that have examined the effect of acute program variables on the hormonal response to resistance exercise have primarily used recreationally trained subjects (13,14). The subjects of this study were all competitive intercollegiate athletes participating in a strength/power sport. Training experience has previously been shown to result in an attenuated hormonal response to an acute bout of heavy resistance exercise (12). Although this doesn’t explain the similar response of testosterone to the two exercise protocols, it does suggest that the testosterone response to acute resistance exercise is attenuated in highly trained men.

Testosterone concentrations after both LI and HI were significantly elevated from PRE. Nevertheless, it did not appear that the mechanisms responsible for elevated testosterone levels were related to muscle deoxygenation. These results support recent research by Viru and colleagues (30), who also reported that changes in testosterone concentrations were not influenced by muscle ischemia. Though previous studies have suggested that exercise-induced increases in testosterone concentrations are related to direct Leydig cell stimulation by elevated catecholamine concentrations (1) and/or through changes in hormonal clearance rate (3), it appears that the mechanisms responsible for such changes are still not clear.

In summary, although heavy resistance exercise results in significant muscle deoxygenation, training intensity and volume do not seem to impact the extent of deoxygenation. The results of this study do suggest that oxygen recovery kinetics within skeletal muscle are influenced by these training variables. Furthermore, stimulation of growth hormone release during resistance training does appear to be affected by the duration of muscle ischemia and accumulation of metabolites. These same stimuli do not appear to influence changes in testosterone concentrations during such exercise.

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REFERENCES


