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ACUTE TESTOSTERONE AND CORTISOL RESPONSES TO HIGH POWER RESISTANCE EXERCISE

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This study examined the acute hormonal responses to a single high power resistance exercise training session. Four weight trained men ($X \pm SD$; age [yrs] = 24.5 ± 2.9 ; hgt [m] = 1.82 ± 0.05 ; BM [kg] = 96.9 ± 10.6 ; 1 RM barbell squat [kg] = 129.3 ± 17.4) participated as subjects in two randomly ordered sessions. During the lifting session, serum samples were collected pre- and 5 min post-exercise, and later analyzed for testosterone (Tes), cortisol (Cort), their ratio (Tes/Cort), and lactate (HLA). The lifting protocol was 10×5 speed squats at 70% of system mass (1 RM + BW) with 2 min inter-set rest intervals. Mean power and velocity were determined for each repetition using an external dynamometer. On the control day, the procedures and times (1600–1900 hrs) were identical except the subjects did not lift. Tes and Cort were analyzed via EIA. Mean $\pm$ SD power and velocity was 1377.1 ± 9.6 W and 0.79 ± 0.01 m $\cdot$ s⁻¹ respectively for all repetitions, and did not decrease over the 10 sets ($p < 0.05$). Although not significant, post-exercise Tes exhibited a very large effect size (nmol $\cdot$ L⁻¹; pre = 12.5 ± 2.9 , post = 20.0 ± 3.9 ; Cohen's D = 1.27). No changes were observed for either Cort or the Tes/Cort ratio. HLA significantly increased post-exercise (mmol $\cdot$ L⁻¹; pre = 1.00 ± 0.09 , post = 4.85 ± 1.10). The exercise protocol resulted in no significant changes in Tes, Cort or the Tes/Cort ratio, although the Cohen's D value indicates a very large effect size for the Tes response. The acute increase for Tes is in agreement with previous reports that high power activities can elicit a Tes response. High power resistance exercise protocols such as the one used in the present study produce acute increases of Tes. These results indicate that high power resistance exercise can contribute to an anabolic hormonal response with this type of training, and may partially explain the muscle hypertrophy observed in athletes who routinely employ high power resistance exercise.

Key words: endocrine, squats, weight training.

Resistance exercise has been shown to induce an array of acute and chronic responses in human skeletal muscle. A potentially beneficial acute response is the effect of resistance exercise on circulating levels of both anabolic and catabolic hormones. For an individual seeking to improve strength and increase lean body mass, optimizing these endocrine responses appears to be a contributing factor [1]. Furthermore, the ratio of testosterone and cortisol has repeatedly been associated with the physiological stresses applied to the body [2–8]. While much has been elucidated concerning the acute resistance exercise variables and the associated endocrine responses, few data are available concerning the role of resistance exercise power and the immediate hormonal responses.

Resistance exercise has been shown to acutely increase circulating levels of total testosterone in adult males [1, 5, 7, 9–14]. Because some studies have shown no increase after resistance exercise [1, 9, 15], it is apparent that the response is dependent on a number of factors such as the amount of muscle mass involved [13], the intensity [11, 16, 17], volume [5], inter-set rest periods [7], age [10], and training experience [8]. Generally, protocols using large muscle

mass, multi-joint exercises with high relative intensities are effective for increasing acute testosterone concentrations [3–8, 10, 18–22].

Acute increases have also been demonstrated for cortisol [5, 6, 8, 16, 23, 24], although some investigations have found no response [13, 25, 26]. Although plasma volumes shifts certainly account for some of the resistance exercise-induced cortisol kinetics, circulating concentrations are influenced by numerous other factors [27]. While cortisol has been shown to acutely increase after large volumes of high power, large muscle mass exercises, the relative intensity must be great enough to elicit such a response [5, 6, 8, 12, 13, 17, 19, 21–24, 26, 28–30].

The acute effect of resistance exercise on the testosterone/cortisol (Tes/Cort) ratio is less clear. Increasing testosterone, decreasing cortisol, or both, would appear to result in a more anabolic environment for skeletal muscle. Typically, the acute hormonal response to a single resistance exercise training session is a relatively greater molar response of cortisol compared to testosterone. The net result is that the Tes/Cort ratio decreases immediately after most resistance exercise protocols [6, 27], although some studies

have failed to demonstrate acute changes in this ratio following resistance exercise [22]. On the other hand, high stress training by elite weightlifters has been shown to negatively affect this ratio at rest [3, 4, 18, 19, 28, 30], while well designed periodized programs are capable of increasing the Tes/Cort ratio [29].

While many studies have examined the acute hormonal response to resistance exercise, most investigations have focused on more traditional movements using heavy loads and relatively slow velocities. Few investigations to date have examined the effect of a high power training session incorporating lighter loads and higher velocities. Volek et al. [13, 26] found significant increases in circulating testosterone after jump squat sessions with a 30% 1 RM load. cortisol, however, did not exhibit an increase. In another study, testosterone and cortisol were increased in response to an acute bout of moderate to high intensity snatches, which would be considered a power exercise, in elite junior weightlifters [6]. However, when explosive resistance exercise was compared with sub-maximal and maximal load resistance exercise, testosterone was found to increase only when using the heaviest loads and slower velocities [17]. Because of the limited research regarding high power resistance exercise and hormonal responses, the purpose of this study was to examine the effect of a high power resistance training (HPRT) protocol on circulating levels of total testosterone, cortisol, and the Tes/Cort ratio.

METHODS

Subjects included trained men ($n = 4$, mean $\pm$ *SD*, age = 24.5 ± 2.9 years, weight = 96.9 ± 10.6 kg, height = 182 ± 5 cm, 1 RM barbell squat = 129.3 ± 17.4 kg) who volunteered to participate in the study. At the onset of the study, all participants had been involved in a resistance training program for at least the past year and were capable of performing a 1 RM squat equal to $1.0 \times$ body weight. Prior to participation, all subjects were informed of the protocols and risks associated with the study and signed informed consent documents approved by The University Institutional Review Board.

A randomized, repeated measures design was used to examine the acute effects of a high power resistance training (HPRT) session on serum concentrations of testosterone, cortisol and the Tes/Cort ratio. Each subject served as his or her own control, and the order of the sessions was randomized (see Fig. 1). Subjects reported to the lab on three separate occasions. The purpose of the first session was to brief the participants on the procedures and risks associated with the protocol, obtain written consent, determine barbell squat 1 RM, and familiarize participants with the speed squat movement. One RM strength for the parallel barbell squat was determined using the methods of Kraemer and Fry [31]. Subjects were required to reach a depth of parallel throughout the study, defined as the

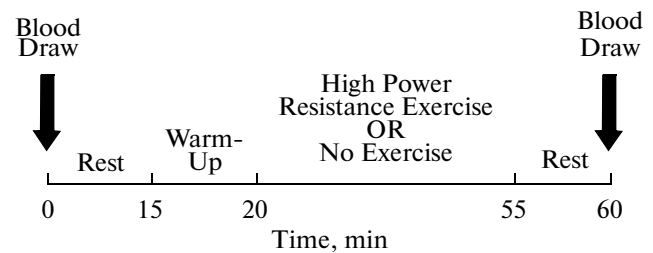


Fig. 1. Study time line.

posterior surface of the thigh being parallel with the floor. A high bar position (barbell on the superior aspect of the trapezius) was also used throughout the study. One RM strength was determined from the most weight that could be lifted one time using correct form. After determining 1 RM, familiarization consisted of 5 sets of 5 repetitions of speed squat with a load of 70% 1 RM system mass (1 RM barbell load + BM). Subjects were instructed to exert a maximal effort and to accelerate through the entire concentric portion of the speed squat. Verbal cues in regard to form, depth, and speed of movement were given throughout the familiarization session.

One week later, subjects returned for either the high power resistance training session (HPRT) or the control session (CON). The second session was performed 1 week later, with the order of the session conditions randomized. Subjects reported to the lab between 1600 h and 1900 h for both sessions to minimize any effects of diurnal variation on hormonal concentrations. On the HPRT day, subjects performed a general 5-minute warm-up before commencing the HPRT protocol. After warming up, 10 sets of 5 speed squat repetitions were performed using 70% of the system 1 RM mass. Two minutes of rest separated each set. Barbell velocity was measured for each repetition using a linear position transducer attached to the right side of the bar (Fitrodyne; Fitronics, Bratislava, Slovakia). Velocity was calculated as the first derivative of position with respect to time. When barbell velocity dropped below 90% for at least two repetitions of a set, the barbell weight was decreased 5 kg to permit barbell velocities to be maintained. As a result, speed of movement was maintained throughout all sets for all subjects. Force was calculated as the product of the system mass and acceleration due to gravity. Average power was calculated as the product of force and average velocity.

Serum hormonal and blood lactate responses to this exercise protocol were determined from antecubital venous blood samples. Serum and whole blood samples were collected 15 minutes pre-exercise and 5 minutes post-exercise using a 21 ga needle and VacutainerTM with no preservatives, while an additional whole blood sample was collected immediately post exercise. The entire time line for data collection sequence is graphically illustrated in Figure 1. Samples were allowed to clot at room temperature, and were

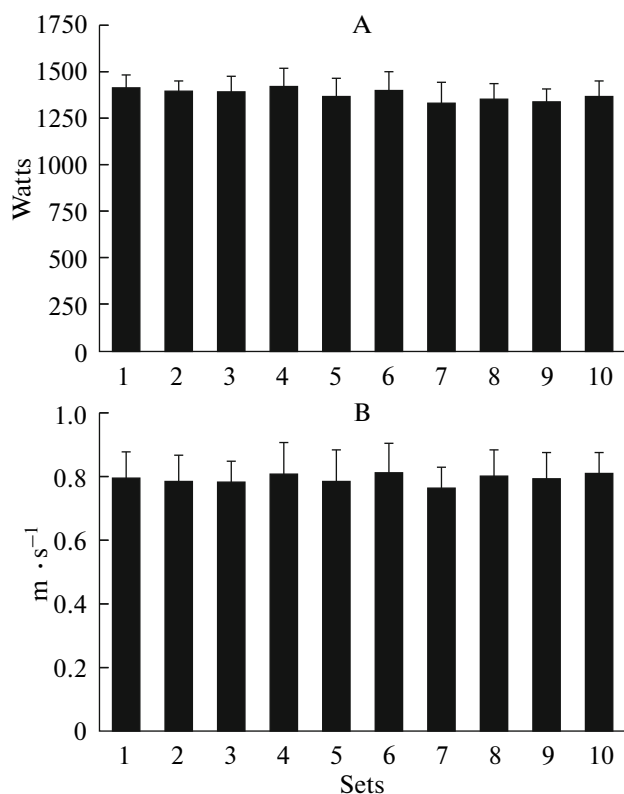


Fig. 2. Power and barbell velocity ($X \pm SD$) for each set of the speed squat training protocol. No significant differences occurred across the ten sets ($p > 0.05$).

then centrifuged for 15 minutes at $1500 \times g$. Aliquots of the resulting serum were stored at -80°C until analyzed for either total testosterone or cortisol. Elisa technology was used to determine circulating concentrations for these hormones (Diagnostic Systems Laboratories, Webster, TX). Intra-assay coefficients of variation for both hormones were $<3.4\%$. Plasma volume shifts for this exercise protocol were calculated for each post-exercise blood sample and were determined to be $<10\%$ [32], and were not great enough to solely account for the hormonal response patterns of this study. For that reason, serum hormone concentrations were not corrected for plasma volume shifts in this investigation; thus all statistical analyses were performed on hormonal values based on actual measured circulating concentrations. Whole blood was analyzed for lactate concentrations 5 min pre-exercise and immediately following exercise. The control session consisted of the same procedures as HPRT with the only difference being that subjects rested quietly for approximately 45 minutes instead of performing the HPRT exercise.

A repeated measures ANOVA was used to compare mean serum concentrations of testosterone, cortisol, Tes/Cort and HLa between pre and post exercise for each condition ($p < 0.05$). When appropriate, Cohen's D effect sizes were calculated. All values are reported as mean $\pm SD$.

RESULTS

Mean power and velocity were calculated for all sets, and the individual means for each set were averaged for the entire group. Mean power across all sets was 1377.1 ± 30.4 W, while mean velocity across all sets was 0.79 ± 0.01 m · s⁻¹. Figure 2 shows mean power and mean velocity for each set. Lactate and hormonal responses for both HPRT and CON are shown in Figure 3. Post HPRT blood lactate levels were significantly higher than pre HPRT, pre CON and post CON ($p < 0.05$). Blood lactate levels did not change significantly between pre and post measures during CON. There was no significant difference between pre and post values for total testosterone, cortisol, or the testosterone/cortisol ratio for either condition. However, the change in serum testosterone concentration between pre and post exercise for HPRT resulted in an extremely large Cohen's D effect size of 1.27.

DISCUSSION

Of particular interest is the acute testosterone response observed following the HPRT stimulus. Previous studies of the endocrine responses to heavy resistance exercise have often reported very large increases in circulating cortisol [4, 5, 7, 12, 21–24, 27]. While testosterone concentrations have been observed to increase post resistance exercise, the increases in cortisol have consistently been greater [4–7, 12, 21–24, 27, 30]. The results of the present study, however, should not be entirely unexpected since previous examinations of high power resistance exercise (i.e., weightlifting) have also exhibited large acute testosterone responses [6, 19, 21, 27, 28, 30]. Additionally, other examples of increased acute testosterone have utilized lifting protocols that incorporated large muscle mass exercises [6] and moderately high volumes of exercise [10] as in the present study. When relative intensities (i.e., % 1 RM) have been too low, little to no acute testosterone increases have been reported [22]. Although short inter-set rest intervals may contribute to an acute testosterone response [7], this is not likely to be a major contributing factor in the current study since rest intervals were held constant at two minutes.

Contrary to a number of previous resistance exercise studies [3, 4, 7, 16, 19, 21, 28–30], no acute cortisol response was observed following this high power protocol. As mentioned previously, the acute molar cortisol response is usually larger than the testosterone response [7]. Resistance exercise session characteristics that appear to contribute to this include large muscle mass exercises, high power output, large training volumes, and/or full body training protocols [6, 7, 27]. When inter-set rest intervals are decreased, the cortisol response can also be considerable [7]. In light of the current results and these previous reports of acute cortisol responses, the training volume, and inter-set

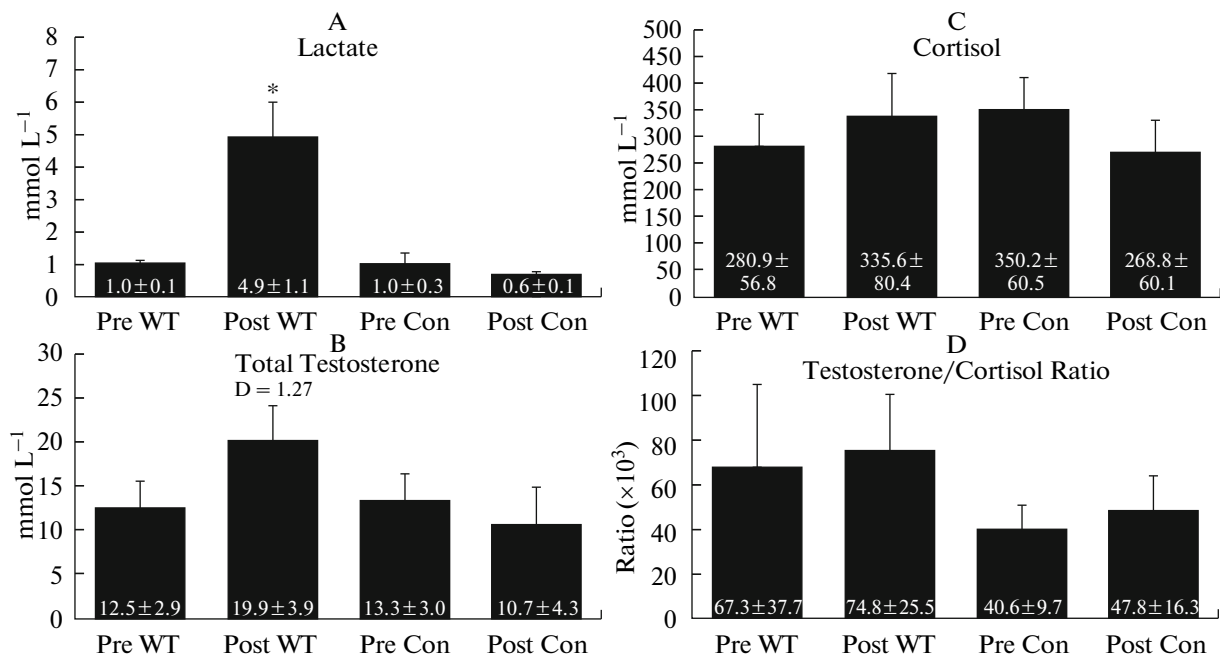


Fig. 3. Lactate and hormonal responses ($X \pm SD$) before and after the high power resistance exercise and control sessions. * different from pre ($p < 0.05$). The Cohen's D value for total testosterone represents a very large effect size.

rest intervals were not great enough to elicit a cortisol response. This information may be helpful when designing a resistance exercise protocol where the catabolic environment needs to be minimized.

Although sometimes more difficult to interpret, the Tes/Cort ratio has been shown to typically decrease immediately post resistance exercise [3, 4, 19, 21, 28–30]. Additionally, decreases in resting values for this ratio correspond to increases in the training stress [2, 16, 18, 21, 29, 30]. Although sometimes used as an indicator of overtraining, this is not always the case, so interpretation of this ratio must be limited to use as simply an indicator of training stress [3, 4, 19]. The present results indicate that the Tes/Cort ratio did not acutely change due to 10 sets of 5 speed squats at 70% system 1 RM loads. At first, it would seem that the ratio should have decreased post-exercise. However, we speculate that the strong role of high power exercise on inducing a testosterone response may have caused this ratio to remain unchanged. It must also be remembered, however, that the cortisol response was negligible. This suggests that although this exercise required maximal effort for power production, it was not very fatiguing in the typical sense of metabolic fatigue, thus resulting in the hormonal profile observed. As such, individuals performing a training session such as used in the present study do not appear to accumulate an inordinate amount of fatigue, either metabolically or mechanically.

Besides the hormonal variables measured, the acute lactate response was also determined. Previous studies have indicated that high power and high relative intensity resistance exercise will produce a marked lactate response. Short inter-set rest intervals will also result in elevated lactate levels [6, 7, 20]. The lactate values observed in the present study indicate only a moderate metabolic stress where the anaerobic glycolytic system is not highly taxed. This may help explain why the stress hormone response was negligible using this lifting protocol.

CONCLUSIONS

Several important implications for the practitioner result from these data. First, acute increases of testosterone may be possible if the resistance exercise power is great enough. Physiologically, it is possible that the elevated testosterone observed is part of a critical adaptation process for this type of exercise. Secondly, indicators of training stress (i.e., cortisol, Tes/Cort ratio) indicate the type of resistance exercise used in the present study is not unduly stressful when presented in a single training session. This is further supported by the modest lactate response observed. While this type of high power resistance exercise may eventually be stressful, it is not due to an anaerobic glycolysis challenge. Finally, variations of this type of training may be especially appropriate when a training taper is being employed to prepare for impending competition. If appropriate, not only is high mechanical power being produced, but the hormonal profile

resulting may contribute to a proper peak for the individual.

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