Hormone and Recovery Responses to Resistance Exercise with Slow Movement

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Abstract: This study examined acute hormone and recovery responses to resistance exercise with slow movements. Six men performed three types of exercise regimens (five sets of knee extension exercise): (1) high-intensity resistance exercise with normal movement (HN; 1 s for lifting action, 1 s for lowering action), (2) low-intensity resistance exercise with slow movement (LS; 3 s for lifting action, 3 s for lowering action), and (3) low-intensity resistance exercise with normal movement (LN; 1 s for lifting action, 1 s for lowering action). The intensity in the first set was set at approximately 80% of 1RM for HN and 40% of 1RM for LS and LN. In the HN and LS, the subjects performed each exercise set until exhaustion. In the LN, both intensity and number of repetitions were matched with those for LS. The total work volume in the HN showed approximately double the value of LS and LN (P < 0.05). Electromyography (EMG) data indicated that LS showed sustained EMG signals throughout the exercise. During the exercise, the HN and LS showed lower muscle oxygenation levels. After the exercise, LS caused significantly greater norepinephrine and free testosterone responses (delta value) than in the HN and LN (P < 0.05). However, no significant difference was observed in the recovery of maximal isometric strength, isokinetic strength, and jump performance between the HN and LS. These results indicate that slow movements during the resistance exercise are important for the enhancement of hormonal responses, especially catecholamine and free testosterone, but they do not affect muscle strength recovery.

Key words: anabolic hormone, catabolic hormone, muscle strength, muscle oxygenation.

Resistance exercise is a potent stimulus for enhancing endocrine activities, causing acute regulations of hormone secretions [1]. The magnitude of exercise-induced increases in growth hormone (GH) and testosterone are dependent on various factors, including rest period length [1], exercise intensity [2], exercise volume [3], contraction type [4], and other types of prior exercise workout [5, 6]. Although muscle growth can occur in the absence of elevations of circulating anabolic hormones [7], muscular hypertrophy and strength gain after prolonged training might be related, at least in part, to exercise-induced increases in anabolic hormones [1].

In general, a training intensity of greater than 65% of one repetition maximum (1RM) is considered the minimum intensity to induce muscular hypertrophy and strength gain [8]. However, resistance exercise at high intensity would not be appropriate for some individuals, including sedentary, frail, and elderly people. Therefore it is necessary to develop effective regimens with reduced mechanical loads. Evidence from several studies indicates that low-intensity (20%–50% 1RM) exercise with moderate vascular occlusion markedly increases acute GH secretion [9–11] and considerable muscular hypertrophy [12]. However, resistance exercises with vascular occlusion require special equipment and careful monitoring of blood flow and occlusive pressure. Alternatively, a low-intensity resistance exercise (~50% 1RM) with slow lift and tonic force generation has been shown to enhance GH secretion [13], and muscular hypertrophy, and strength gains [14]. Moreover, a low-intensity (~50% 1RM) exercise with combined slow movements and a short interset rest has been reported to increase muscular size and strength in middle-aged women [15]. The mechanisms of slow-movement exercise for inducing muscular hypertrophy remain unclear, but an augmented response of GH [13] might be partly related. Endogenous testosterone and cortisol have also been thought to affect the muscular adaptations following resistance training [1]. However, the responses of these hormones to resistance exercise with slow movements are not understood.

Strenuous resistance exercise temporarily reduces maximal strength [16] and voluntary neural activation [17] and causes subsequent muscle damage [18]. Based on the principal of resistance exercise, the next training
session should be performed under conditions in which complete recovery has been obtained [16]. Therefore information about the recovery processes of muscle function would be important in designing an appropriate training schedule. Some studies have focused on the recovery of muscle function after sessions of multiple resistance exercises [16], eccentric exercise [19], and prolonged endurance exercise [20]. However, no studies have examined the effects of low-intensity resistance exercise with slow movements on the recovery of muscle function. In general, the rate of recovery is affected by the intensity and volume during the exercise [21]. Therefore the slow-movement exercise with light load might cause a faster recovery of muscle strength than traditional resistance exercise using a heavier load.

The purpose of this study was to demonstrate acute hormonal responses and the recovery of muscle function after resistance exercise with slow movements. Electrical activity and muscle oxygenation levels during exercise were also investigated. These responses were compared to those produced by conventional high-intensity resistance exercise. We hypothesized that resistance exercise with slow movements stimulates testosterone response along with GH and shows a faster recovery of strength.

METHODS

Subjects. Six healthy men (mean ± SE: 24.3 ± 0.4 years; height, 173.7 ± 1.9 cm; body mass, 69.2 ± 1.6 kg; % fat, 19.3 ± 1.0%) participated in this study. All subjects were physically active and well accustomed to heavy-resistance exercise. They were informed about the experimental procedure and the purpose of this study. Subsequently, their written informed consent was obtained. The study was conducted in full accordance with the statement of protection for human subjects in the Declaration of Helsinki.

Exercise regimen. The subjects visited the laboratory four times during the experimental period. During the first visit, the value of one-repetition maximum (1RM) for the bilateral knee extension exercise was measured based on normal procedures in our laboratory [6, 22]. A determination of 1RM began with a few minutes of stretching the quadriceps femoris muscle, followed by warm-up sets consisting of 10 repetitions. The load was increased until subjects were unable to perform a lift. Subsequently, the subjects performed exercise with slow movements (lifting action for 3 s, lowering action for 3 s) at 40% of 1RM and with normal movements (lifting action for 1 s, lowering action for 1 s) at 80% of 1RM. These practices were performed to confirm the number of repetitions in two exercise regimens. The subjects also performed practices of maximal muscular strength and countermovement jump measurements for familiarization. During visits 2–4, the three experimental trials were conducted.

In random order, all subjects participated in the three trials: (1) high-intensity resistance exercise with normal movements (HN trial; 1 s for lifting action, 1 s for lowering action), (2) low-intensity resistance exercise with slow movements (LS trial; 3 s for lifting action, 3 s for lowering action), and (3) low-intensity resistance exercise with normal movements (LN trial; 1 s for lifting action, 1 s for lowering action). These trials were performed from 8:00 until noon after overnight fasting. A movement speed for the LS trial was chosen in accordance with a previous study using low-intensity resistance exercise with slow movement [14]. The resistance exercise consisted of five sets, with 1-min rest periods between sets. The exercise intensities in the first and second sets were set respectively at approximately 80% of 1RM for the HN trial and 40% of 1RM for the LS and LN trials. From sets 3–5, the load was decreased by about 10% of the 1RM every set. In the HN and LS trials, the subjects performed each set of exercise until exhaustion. They were instructed to exercise at constant speed and frequency with the aid of a metronome. In the LN trial, the subjects performed the exercise at the same relative intensity and with the number of repetitions in each set as those for the LS trial. The range of joint motion in each set was from 90° to 0° (0° at full extension). The resistance exercise in each trial was performed at the same time of day to avoid diurnal variations of hormonal responses.

Electromyography signals and muscle oxygenation during exercise. Electromyography (EMG) signals were recorded from the right vastus lateralis muscle, rectus femoris muscle, and vastus medialis muscle. Bipolar surface electrodes were placed over the belly of the muscle with a constant interelectrode distance of 20 mm. The EMG signals were amplified, then fed into low (10 Hz) cut filter. A near-infrared continuous-wave spectroscopic (NIRcws) monitor (BOMLITR, Omegawave Inc.) was used to measure the muscle oxygenation in the right vastus lateralis muscle during exercise based on the method of a previous study [14]. The wavelengths of emission light were 780, 810, and 830 nm, and the relative concentrations of oxygenated hemoglobin and myoglobin (Oxy-Hb/Mb) in tissues were quantified according to the Beer-Lambert law [23]. The NIRcws signals registered during exercise do not reflect the absolute levels of oxygenation. Therefore the changes of oxygenation in muscles are expressed as relative values to the overall changes in the signal monitored according to the arterial occlusion method [23]. The resting level of Oxy-Hb/Mb was regarded as 100% (baseline), and the minimum plateau level of Oxy-Hb/Mb (by arterial occlusion) was regarded as 0%. A pressure cuff was placed around the proximal area of the right thigh and inflated manually up to 300 mmHg until the minimum plateau level of Oxy-Hb/Mb appeared. The distance between the incident point and the detector was 30 mm.
Blood sampling and analyses. After an overnight fast, the subjects arrived at the laboratory and rested for 30 min prior to the first blood collection. Venous blood samples were obtained from an indwelling cannula in the antecubital vein before the exercise and 5 min, 15 min, and 30 min after the exercise to determine acute hormone responses. Additional blood samples were also obtained at 24 h and 48 h after the exercise to measure serum creatine kinase (CK) activity (Fig. 1). Blood samples for measurements of hormones and enzymes were centrifuged at 3,000 rpm for 10 min to obtain serum or plasma and stored at −85°C until analyses. Concentrations of epinephrine and norepinephrine were measured using HPLC. The inter- and intra-assay coefficients of variation (CV) were 2.7% and 2.0% for epinephrine, and 2.4% and 1.3% for norepinephrine. Serum GH was measured using radioimmunoassay (RIA). The inter- and intra-assay CV were 4.0 and 3.4%. Free testosterone and cortisol concentrations were measured using RIA by normal procedure in our laboratory [24].

Recovery of muscle function after exercise. Maximal isometric and isokinetic strengths of the unilateral knee extension exercise and the countermovement jump (CMJ) height were measured before and immediately after the exercise (2 min after the exercise), and at 3 h, 6 h, 24 h, and 48 h during the recovery period. The subjects were familiarized with the test procedure on several occasions prior to taking measurements. Maximal isometric and isokinetic strengths of knee extension exercise with the right leg were measured using an isokinetic dynamometer (COMBIT, Minato Medical Science Co. Ltd.) based on normal protocol of our laboratory [6]. A subject sat on a chair with the ankle of the right leg attached firmly with a strap to the lever of the dynamometer. A pivot of the lever was aligned accurately with the rotation axis of the knee joint. The requisite axial alignment of the joint angles and dynamometer axes were maintained during the movements. The maximal isometric strength at a knee angle of 80° was then measured. The subjects were instructed to exert maximal force for 3 s. The highest value of 2–3 trials was adopted. Maximal isokinetic strength at 180°/s was also measured. Three repetitions were carried out to determine the peak torque for joint angles ranging from 90° to 0°.

For measurement of the CMJ height, the subjects performed a maximal vertical jump on a platform (CT-916, Takei Scientific Instruments Co. Ltd.) that was connected to a personal computer. The subjects were instructed to perform a maximal jump while placing their hands on the lumbar division to eliminate upper-limb effects. The vertical jump flight time was recorded. From the flight time, the CMJ height was calculated using the following formula [25]:

\[
\text{jump height (m)} = \frac{1}{8} (\text{flight time})^2 \times \text{the gravity constant}
\]

\[
\text{gravity constant} = 9.81 \text{ m/s}^2
\]

Muscle soreness. Muscle soreness was reported using a 100-mm visual analogue scale (VAS), where the subject was instructed that 0 mm indicated “no pain at all,” whereas 100 mm reported “unbearable pain” [18]. The subjects were asked to rate the soreness experienced during each measure by making a mark on a 100-mm line.

Statistical analysis. The data are expressed as means ± SE. A two-way (trial × time) analysis of variance (ANOVA) with repeated measures was used. When ANOVA revealed significant interaction effect (trial × time) or main effect, a one-way ANOVA with repeated measures followed by a Tukey-Kramer test was performed to identify the difference at relevant time points among three trials, and/or the difference over the experimental period (vs. pre-exercise value). The increases in exercise-induced hormone concentration (Δ value) were determined as absolute differences between pre-exercise value and peak value of postexercise. The differences of relative muscle
oxygenation level during each set of exercises and the Δ value were evaluated using the Tukey-Kramer test. *P* < 0.05 was considered significant.

**RESULTS**

**Exercise performance**

In the first set of exercises, the relative exercise intensity to the 1RM and number of repetitions were 79.1 ± 1.2% (relative exercise intensity) and 11.5 ± 1.0 (number of repetitions) in the HN trial, and 43.4 ± 1.1% and 11.8 ± 0.8 in the LS and LN trials, respectively. Moreover, the relative exercise intensity (average values) and total work volume throughout five sets of exercise showed significantly greater values in the HN trial (67.3 ± 2.1%, 4,067 ± 146 J) than in the LS and LN trial (36.4 ± 1.3%, 1,957 ± 80 J, *P* < 0.05).

**Electromyography signals and muscle oxygenation during exercise**

Figure 2 shows a typical example of EMG signals during three types of exercise. Their patterns were different according to exercise intensity and movement speed in each type of regimen. In the HN trial, the magnitude of the EMG signal was much greater than in the LS and LN trials because of the difference in load. However, the magnitude

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**Fig. 2.** Typical examples (from the same subject) showing knee angle and EMG from the right vastus lateralis, rectus femoris, and vastus medialis muscles during high-intensity exercise with normal movement (HN), low-intensity exercise with slow movement (LS), and low-intensity exercise with normal movement (LN). Records indicate from the second lifting movements in the first set for LS and the second to fourth lifting movements for both HN and LN.

**Fig. 3.** Relative muscle oxygenation level of the right vastus lateralis muscle during each set of exercises. The baseline (rest condition) and minimum muscle oxygenation (arterial occlusion condition) levels of hemoglobin and myoglobin (Oxy-Hb/Mb) are defined as 100% and 0%, respectively. Values (means ± SE) indicate average values during each set of exercises.
of the signals was not constant during the exercise, indicating the existence of a relaxing phase. The LS trial with lower intensity and slower movements showed a small but constant EMG signal during the exercise. In the LN trial, the exercise intensity was identical to that in the LS trial, but intermittent EMG activities were found during the exercise. Therefore the LN trial would be performed with ballistic actions at the switching period of lifting (concentric) and lowering (eccentric) phases because of faster movements.

During each set of exercises, the relative muscle oxygenation level of the vastus lateralis muscle showed a rapid reduction as the exercise repetitions started. Figure 3 shows average values of the relative muscle oxygenation level during three types of exercises. The HN and LS trials showed lower values than the LN trial, but no significant difference was observed among the three trials. Further, the average values of minimum oxygenation levels during the exercises showed no significant difference among the trials (HN, 37.5 ± 5.1%; LS, 33.1 ± 5.9%; LN, 38.4 ± 2.4%, NS).

Acute hormonal responses

Figure 4 shows changes in plasma epinephrine and norepinephrine concentrations in three trials. In the HN and LS trials, plasma epinephrine increased significantly after the exercises ($P < 0.05$), with no significant difference between the two trials. In the LN trial, no significant difference was observed after the exercise. Exercise-induced change in epinephrine concentration (delta value) showed a significantly higher value in the LS trial than in the LN trial ($P < 0.05$). The concentration of norepinephrine increased significantly after the exercises in the HN and LS trials, though the LN trial showed no increase. Delta values showed significantly higher values in the LS trial (478 ± 83 pg/ml) than in the HN (303 ± 85 pg/ml) and LN trials (70 ± 46 pg/ml, $P < 0.05$).

Figure 5 shows changes in serum GH, free testosterone, and cortisol concentrations in the three trials. Pre-exercise values of GH were slightly but significantly higher in the LS trial than in the HN and LN trials ($P < 0.05$). The LS trial showed marked increases in GH after the exercise ($P < 0.05$), whereas no significant increase was observed in the HN and LN trials. Consequently, the delta values showed a higher value in the LS trial than in the HN and LN trials, and a significant difference was observed between the LS and LN trials ($P < 0.05$).

Serum-free testosterone concentration increased significantly after exercise only in the LS trial ($P < 0.05$), though no significant increase was observed in the HN and LN trials. The delta values showed a significantly higher value in the LS trial (3.4 ± 0.8 pg/ml) than in the HN (0.9 ± 0.6 pg/ml) and LN trials (–0.4 ± 0.5 pg/ml, $P < 0.05$). In the HN and LS trials, no significant difference was observed in the cortisol concentration after the exercise. The LN trial showed a significant reduction of cortisol concentration after the exercise ($P < 0.05$). The delta values showed no significant difference among the trials.

Recovery of muscle function

Figure 6 shows relative changes in maximal isometric and isokinetic strengths after three types of exercise. In the HN and LS trials, the maximal isometric strength decreased markedly immediately after exercise ($P < 0.05$), with no significant difference between the trials (HN, 75.1 ± 3.2% vs. LS, 74.9 ± 4.5%, NS). The LS trial showed a faster recovery of maximal strength, but no significant difference was observed at any time point between HN and LS trials. Maximal isokinetic strength decreased immediately after exercise in the HN (83.2 ± 3.8%) and LS trials (79.4 ± 5.6%, $P < 0.05$). However, no significant difference was observed at any time point among the trials.

The CMJ height showed significant decreases after the exercise in the HN and LS trials. However, no significant difference was observed at any time point among the trials.
The Journal of Physiological Sciences
Vol. 58, No. 1, 2008

Muscle soreness and CK activity
Before exercise, no subjects reported any muscle soreness or discomfort. At 24 h after exercise, the muscle soreness level estimated by VAS showed significantly greater values in the LS (55.8 ± 9.2 mm) and HN trials (34.0 ± 7.5 mm) than in the LN trial (6.8 ± 5.0 mm, P < 0.05). However, no significant difference was found between the HN and LS trials. No significant difference was observed in serum CK activity before the exercise (the data of two subjects were excluded because of abnormal values, n = 4). The CK activity increased slightly at 24 h and 48 h after the exercise, but no significant difference (vs. pre-exercise value) was observed in any trial.

DISCUSSION
This study demonstrated that the LS trial caused marked enhancements of hormone secretions. In particular, it showed significantly stronger responses of norepinephrine and free testosterone than the HN trial did. However, no significant difference was observed in the recovery rate of muscle function between the LS and HN trials. The primary factor for enhanced hormone secretions in the LS trial is apparently the slow movements during exercise.

The EMG pattern in the HN trial showed greater fluctuations during the exercise (Fig. 2), indicating that the exercise was performed with ballistic actions. Similar EMG patterns were also observed in the LN trial with low-intensity and normal speed movements. On the other hand, the LS trial showed small but constant EMG signals throughout the exercise. In the LS trial, the continuous force output and concomitant sustained restriction of blood flow might cause a marked and persistent reduction of muscle oxygenation level during exercise (Fig. 3). Also, an increase in muscle oxygen consumption during a longer duration of the exercise might affect the decline of the muscle oxygenation level [26]. However, average and minimum levels of muscle oxygenation during the exercise showed no significant difference among the three trials. In a previous study by Tanimoto et al. [14], the low-intensity exercise with slow movements caused a significantly lower minimum oxygenation level than high-inten-
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Fig. 6. Relative changes in maximal isometric (A) and isokinetic strength (B) after exercise. Values are normalized by pre-exercise values. Dotted lines indicate the value of pre-exercise (baseline). Values are means ± SE. *P < 0.05 vs. Pre. †P < 0.05 compared with LN trial. 0 h, immediately after exercise.

ity exercise with normal movements. A reason for different results might be the use of pause during the slow-movement exercise in a previous study. On the other hand, we must keep in mind that the LS trial caused a much longer duration of lower oxygenation level because of the slow movements. Therefore we believe that a sustained exposure of intramuscular hypoxic condition might be a more important factor for exercise-induced hormonal responses.

It is interesting that the LS trial showed a marked increase in GH concentration after the exercise, whereas the HN trial showed no significant difference. Besides the greater GH response, the norepinephrine response was significantly greater in the LS trial than in the HN trial, though previous study [13] showed smaller response to the slow-movement exercise (LS trial in the present study). It has been suggested that a local accumulation of metabolic subproducts (e.g., lactate and proton) in the working muscle stimulates exercise-induced GH and catecholamine secretions [9, 24]. Although we were unable to measure blood lactate concentration, a previous study [14] showed no difference in lactate response between the low-intensity exercise with slow movements (LS trial in the present study) and the high-intensity exercise with faster movements (HN trial in the present study), suggesting that exercise-induced metabolic stress was similar between the present two trials. On the other hand, reasons for enhanced hormone responses might be smaller, but sustained activation of the motor centers during the slow-movement exercise because increased central command during exercise strongly stimulates the GH and catecholamine secretions [27]. Further research with the monitoring of cardiovascular (e.g., blood pressure) and hormonal responses would be helpful for the elucidation of reasons.

Free testosterone represents the amount of testosterone that is biologically available to cells because 98% of it is bound to binding proteins [28]. Resistance exercises with large muscles have been shown to increase free testosterone concentration [4, 16]. Moreover, circulating testosterone has been shown to affect increases in lean mass and strength during 12 wk of resistance training [29]. The present LS trial showed a significant increase in free testosterone concentration after the exercise, though no significant increase was observed in the HN and LN trials. The finding that low-intensity resistance exercise with slow movements showed that an enhancement of testosterone secretion is novel and has not been reported before. On the other hand, it does not appear that augmented free testosterone response in the LS trial was related to intramuscular metabolite accumulation because several studies have suggested that exercise-induced metabolic stress does not affect testosterone response [4, 24]. Alternatively, testosterone response might be related to direct Leydig cell stimulation by elevated catecholamine concentrations [30]. The factor for altered free testosterone response in the LS trial remains unclear, but augmented secretions of anabolic hormones might play a role in marked muscle hypertrophy in the LS trial after a prolonged training period [14].

It has been shown that resistance exercise using a heavier intensity causes delayed strength recovery [16, 21]. Moreover, several studies demonstrated that a faster velocity exercise caused greater delayed onset muscle soreness (DOMS) [31] and extent of Z-band disruption [32]. Based on these findings, we expected that the HN trial with greater load and faster movements would show a slower recovery of strength than the LS trial. However, no significant difference was observed in the recovery of strength between the HN and LS trials. Also, no significant difference was found in CK activity and muscle soreness levels at 24 and 48 h after the exercises. In the present study, the magnitude of exercise-induced strength loss and muscle damage was much smaller than in previous studies.
using maximal eccentric contractions [18]. Therefore the differences of load and velocity in two types of exercise regimen might have no strong impact on recovery profiles after exercises. From a practical point of view, however, the present results indicate that people can include the slow-lifting exercise regimen into a training schedule with the same frequency as a conventional resistance exercise regimen using a heavier load.

It has been demonstrated that a single workout of eccentric exercise produces an adaptation that reduces muscle damage in subsequent workouts (repeated bout effect) [33, 34]. The present subjects performed three trials during separate periods. Therefore a “repeated workout effect” might pertain. In this study, however, the exercise-induced strength loss and muscle damage were much less than those in previous studies causing repeated bout effects [33, 34]. Therefore the protective effects of repeated exercise trials on muscle damage are inferred to be minor.

In conclusion, the results in the present study indicate that the slow movements during the resistance exercise might be a potent factor for the enhancements of hormonal secretions, especially catecholamine and free testosterone. However, the recovery of muscle strength after the exercises showed no significant difference between the LS and HN trials. The low-intensity exercise with slow movements appears to be a useful regimen, but research with different individuals, including sedentary and elderly people, should be conducted.

The authors are grateful to the subjects who participated in this study. We are also grateful to Masashi Watanabe for assistance with EMG measurements and to Shinji Takahashi for comments on data analysis. The study was supported by grants from the Ministry of Education, Culture, Sports Science and Technology of Japan and from Research Fellowships of the Japan Society for the Promotion of Science for Young Scientists.

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