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Hormones in Short-Term Exercises: Resistance and Power Exercises

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RESISTANCE AND POWER EXERCISES involve intense neuromuscular activity and subsequent muscle contraction. The central motor commands originate from the motor cortex, and consist of high-frequency nervous charge sent by axons of pyramidal neurons to spinal motoneurons. According to Kjaer (24), these central motor commands increase endocrine activity. It is apparent that the motor cortex can activate hypothalamic autonomic centers and neurosecretory neurons via a collateral nervous pathway.

Resistance and power exercises evoke hormonal responses despite the short duration of muscle effort. There is also no doubt that hormonal responses evoked by resistance and power exercises are necessary for long-term adaptation of skeletal muscles. However, there are some questions regarding the function of these hormonal responses, and the significance of preexercise hormone levels for preconditioning the neuromuscular system.

Hormonal Responses in Resistance Exercises

Resistance exercises evoke hormonal responses even when a limited number of motor units are involved. Three consecutive bouts of static handgrip at 30% of maximal voluntary contraction, using 2 hands alternatively and without rest intervals (total duration of the isometric exercise work 9 minutes), caused a significant increase in the plasma vasopressein concentration (40). The maximal values appeared after the first 3 minutes. Then a moderate decline appeared, but the vasopressin concentration remained significantly elevated over the initial level up to the end of the exercise. The concentration of growth hormone did not change during the first 6 minutes of effort. Immediately and 10 minutes after the effort, the growth hormone concentration was increased in most of the persons. Plasma corticotropin and cortisol levels did not increase significantly, although at the end of the exercise, a trend for an increase appeared. Contrary to these results, Few et al. (12) found significant increases in corticotropin and cortisol concentrations while holding a 20-kg weight for 5 minutes in 1 hand. The difference in the results can be explained by the larger muscle groups and higher percentage of maximal voluntary strength involved in the latter experiment. Few et al. (12) also observed a cortisol response 5 minutes after the effort that lasted 15–20 minutes, although the corticotropin level was increased only at the end of the effort. Several researchers have also observed a sympathoadrenal response after several isometric efforts (14, 25, 47, 54). A catecholamine response has also been observed within the first minute of handgrip at 30% of maximal voluntary strength, in men but not in women (44). Collectively, this evidence supports the fact that hormone responses are triggered by intense muscle contraction.

Several studies dealt with hormonal changes induced by resistance training sessions. A significant testosterone increase was found after 30 minutes of resistance training in males but not in females (55). When 8 exercises for various muscles were performed with workloads of either 5 repetition maximum (5RM) (rest intervals 3 minutes) or 10RM (rest intervals 1 minute), testosterone concentration increased in males but not in females, with prior recreational resistance training experience (28, 31, 33). In contrast, Cumming et al. (8) found a 20% increase of testosterone level in women after a session consisting of 6 isokinetic resistance exercises. Although the lutropin level increased by 60%, it is unlikely that it was the physiological stimulus for testosterone production because females produce this hormone as a byproduct of steroid biosynthesis in the adrenal glands. Accordingly, those females who did not exhibit a cortisol response failed to show an increase in testosterone concentrations. The other female subjects showed parallel increases in testosterone and cortisol concentrations (8).

In a study involving 2 workload protocols (10RM versus 5RM), Kraemer and coworkers (31) observed greater increases in blood testosterone after the 4 exercises using the 10RM (i.e., lower weight, greater total work) protocol as compared with 5RM (i.e., higher weight, lower total work). At the end of sessions, no statistically significant difference in testosterone levels was found between the 2 protocols (31). Volek et al. (53) also observed increased testosterone concentrations using a bench press protocol of 5 sets to failure with 10RM load (7.4%) and a jump squat protocol of 15 sets with 10 repetitions at 30% of 1RM squat (15.1%) The significance of training session workload was confirmed by Cotshalk et al. (7). A 3-set heavy-resistance protocol resulted in a greater increase in testosterone concentration than a 1-set exercise protocol.

Besides the characteristics of exercise, duration of rest intervals also has a significant effect on hormonal respones. After a 30-minute intensive single-circuit weight training session (work-to-rest ratio 30 seconds:30 seconds at 70% 1RM), male university students exhibited testosterone levels 24% higher than the initial values (23).

Guezennec et al. (15) observed no significant testosterone response after 6 series of 8 bench presses at 70% of 1RM or maximal number of repetitions at the same workload. The measurements were repeated each month for 4 months, but results were the same. Because the subjects were male weight-trained athletes, the lack of testosterone response might be related to previous training adaptation to the test exercises. In studies on elite athletes, Häkkinen and Pakarinen (16) found that 20 sets of squats at 1RM did not increase concentrations of total and free testosterone, whereas testosterone levels rose significantly when 10 sets of 10 repetitions were performed at 70% of 10RM. In weightlifters, increased testosterone concentration has been found after 4 series of 6 squats at 90–95% of 6RM as well as after 4 series of 9–10 squats at 60–65% of 6RM (45). Long-term resistance training may promote testosterone responses, at least in adolescent athletes. The same weightlifting protocols increased testosterone concentrations in 17-year-old juniors who had more than 2 years training experience but not in subjects with less training experience (30).

Häkkinen et al. (19) measured hormone responses in elite athletes performing two strength training sessions per day. Both testosterone and cortisol levels decreased after the first session, but testosterone levels increased after the second session.

Acute strength-training sessions have been shown to increase the levels of growth hormone and insulin-like growth factor I (somatomedin-C) in males as well as females (31, 33). The increased production of insulin-like growth factor I may or may not be triggered by growth hormone. A 10RM load (with 1-minute rest intervals) resulted in more pronounced increases of growth hormone concentrations than a 5RM load (with 3-minute rest intervals). However, differences in insulin-like growth factor response were not detected in males. In female subjects, insulin-like growth factor I level rose secondarily 60 minutes after a session consisting of a 5RM protocol (with 3-minute rest intervals) but not after a protocol of 10RM (with 1-minute rest intervals) (31,33). A study of 8-station heavy-resistance exercise (3 sets of repetitions at 10RM with 1-minute rests) showed that the pronounced immediate response of growth hormone is not associated with changes in insulin-like growth factor I concentrations during or immediately after a 24-hour recovery period (26).

Vanhelder and coworkers (50) confirmed the dependence of growth hormone responses on resistance training workload. A significant response was observed when 7 series of 7 squat lifts were performed at 80% of maximal leg strength but not when 7 series of 21 squat lifts were performed at 30% of the previous load.

Häkkinen and Pakarinen (16) studied elite athletes and found no change in growth hormone levels after 20 sets of squat-lifts at 1RM. Concentration of the hormone increased significantly after 10 sets of 10 repetitions at 70% of 10RM.

Cotshalk et al. (7) found a greater growth hormone response after a 3-set heavy-resistance exercise session when compared with a 1-set exercise protocol.

Cortisol response is often found in heavy-resistance training sessions (7, 29, 30, 34, 51). However, a certain threshold in workload exists to evoke the cortisol response (27, 28). In students, a high-intensity strength training session caused a pronounced increase in cortisol concentration associated with increased levels of corticotropin, growth hormone, and aldosterone (23). The aldosterone response indicated the necessity for homeostatic regulation, obviously due to intense perspiration while exercising.

Increased levels of ß-endorphin have also been also detected after resistance exercise sessions (27, 30). The ß-endorphin response depends on the total workload of the session (28).

Strength exercises also activate the sympatho-adrenal system as indicated by increased levels of epinephrine and norepinephrine. Kraemer et al. (34) reported these changes after a 10-station heavy-resistance exercise protocol (3 sets of 10RM with 10 seconds rest between sets and 30 or 60 seconds rest between exercises). In addition to norepinephrine and epinephrine, cortisol levels also rose. According to the results of Guezennec et al. (15), strength exercises can affect catecholamine responses in the absence of changes in cortisol and testosterone levels.

The training effects on basal hormone levels have been studied in a limited number of experiments. Most of the data show that during 4–6 months, strength training does not significantly alter basal levels of testosterone, lutropin, follitropin, prolactin, growth hormone, and insulin (18, 20). Likewise, McCall et al. (39) did not find significant changes in basal levels of testosterone, growth hormone, and insulin-like growth factor I in college students as a result of a high-volume resistance training. Guezennec et al. (15) also did not find changes in cortisol, epinephrine, and norepinephrine levels, whereas Häkkinen et al. (20) reported a decrease in cortisol concentration. Peronnet et al. (42) also reported increases in epinephrine and norepinephrine levels.

In elite weightlifters, a 1-year follow-up study did not reveal significant changes in blood levels of cortisol or testosterone. Only lutropin concentration increased during the first 4 months and remained at this level throughout the year. However, during the first 2 weeks of a 6-week intensive preparation period for competition, testosterone levels decreased and remained at reduced levels until the beginning of competition. Cortisol levels increased slightly during the first 2 weeks but decreased thereafter (17). When elite athletes were studied during a 2-year period, an increase in basal testosterone levels was reported. Cortisol levels changed in parallel with testosterone and, therefore, the testosterone/cortisol ratio did not change (19).

Leg strength training did not change the epinephrine and norepinephrine responses to maximal bicycle exercise (42). Comparison of cyclists and weightlifters during incremental exercise showed that epinephrine and norepinephrine responses are more pronounced in weightlifters, and similar to those observed in untrained persons (38). In principle, this specific difference in training effects was confirmed by Kraemer et al. (35).

During high-intensity resistance training resulting in a decrement in 1RM strength, exercise-induced testosterone levels increased slightly while cortisol levels decreased and growth hormone and peptide F levels did not change (13).

In summary, heavy resistance exercises activate endocrine functions. Responses of testosterone, growth hormone, insulin-like growth factor I, cortisol, and catecholamines have been found. These responses depend on the session protocol, particularly, workload and rest-interval duration. Training experience increases the likelihood of a testosterone response in junior athletes. In females, the testosterone response may not appear.

■ Hormonal Responses in Power Exercises

Hormonal aspects of exercises with high rates of force application in high-speed, cyclic movement have been studied by Kraemer and colleagues. The test exercises were performed at 100, 73, 55, and 36% of maximal leg power (average duration 6 seconds, 16 seconds, 47 seconds, and 3 minutes 31 seconds, respectively). All intensities were greater than those eliciting peak $\dot{V}O_2$ for the individual subjects. Using a Teflon cannula inserted into the vein, the first blood sample was obtained immediately after the end of exercise without any delay. A small but significant increase of norepinephrine concentration was found when 100% of maximal leg power was applied, although the duration of activity was only 6 seconds. Norepinephrine responses increased during longer duration, lower power exercise bouts. Epinephrine levels increased approximately 2.5-fold immediately after exercise at 55% of maximal power output and 47 seconds duration.

Table 1
Action on 1-Minute Continuous Jumping on Levels of Various Hormones (4)

	Before exercise	After exercise
Corticotropin (pg/mL)	31.8 ± 18.1	44.3 ± 20.9*
Cortisol (ng/mL)	181 ± 31	206 ± 41*
Testosterone total (ng/mL	5.8 ± 0.97	6.5 ± 1.18*
Testosterone free (pg/mL)	16.5 ± 3.3	18.6 ± 3.2*
Growth hormone (ng/mL)	2.4 ± 0.30	2.7 ± 0.61
Insulin-like growth factor I (ng/mL)	106 ± 42.5	117.5 ± 59.4
Thyrotropin (µU/mL)	1.07 ± 0.37	1.28 ± 0.43*
Free triiodothyronine (pg/mL)	2.85 ± 0.68	3.66 ± 0.75*
Free thyroxine (ng/mL)	12.2 ± 1.7	15.9 ± 2.1
SHBG (nmol/mL)	22.2 ± 16.3	26.8 ± 16.0
Prolactin (ng/mL)	6.18 ± 1.63	6.12 ± 1.69

Mean ± SD are given. SHBG = steroid hormones binding globulin.
* Statistically significant change according to paired *t* test.

Similar epinephrine responses also occurred after exercise at 36% of maximal leg power (36). Significant increases in ß-endorphin and corticotropin levels were observed immediately after exercise at 36% maximal leg power, as well as 5 and 15 minutes postexercise. Increased plasma cortisol levels were also observed following exercise at 36% maximal power, but only 15 minutes postexercise (37). Thus, these data show that brief, high-intensity (power) exercises are capable of evoking hormonal responses.

Repetitive jumping is another method for studying the effect of power exercise on hormone levels. Significant increases in the concentrations of corticotropin (by 39%), cortisol (by 14%), total testosterone (by 12%), free unbound testosterone (by 13%), thyrotropin (by 20%), free triiodothyronine (by 28%), and free thyroxine (by 12%) have been documented immediately after a 60-second period of consecutive vertical jumps (Bosco test). Significant changes in the blood levels of growth hormone, somatomedicin-C, and prolactin were not detected (Table 1). The most pronounced testosterone responses were demonstrated in subjects with higher jumping performance. The increase in serum total testosterone was highly correlated with average power output (r = 0.61) and jumping height (r = 0.66) (4).

It is arguable whether the endocrine system is capable of increasing these blood hormone levels within 1 minute. Of course, the increased hormone levels might not be related to increased secretion, but instead to a possible hemoconcentration (shift of a part of blood plasma from vessels to the interstitial fluid) and/or to a "wash-out" of hormones from the gland caused by increased rate of blood flow. However, the increases of serum concentrations of corticotropin, triiodothyronin, and thyroxine were between 28 and 39%. It is difficult to assume that these changes were related solely to changes in plasma volume because the decrease in plasma volume reached only 15–20% in 1-minute bouts of exhaustive exercise (46).

In practice, power exercises constitute various training sessions. Bosco and collaborators (2) have studied hormonal changes induced by some training variants. Hormones were assessed before and after 5 sessions (Table 2).

A high number of low-power exercises decreased testosterone and increased growth hormone

Table 2
Characteristics of Studied Power Training Sessions (2)

Person	*n*	Number of reps in series	Number of series	Rest between series (min)	% 1RM	% max power	Type of exercises
Male sprinters	6	6+6+4	6	8	80	100	Half squats, full squats
Female sprinters	6	6+6+4	6	8	80	100	Half squats, full squats
Body builders	6	8–12	12	1–2	70–75	65–75	Half squats, leg press, leg extensor
Weightlifters	4	2–3	10	3–5	60–80	100	Snatch, clean, jerk
Weightlifters	4	2–4	20	2–3	50–70	100	Snatch, clean, jerk

levels in bodybuilders, whereas in weightlifters, a large number of high-power exercises in the first weightlifting session increased testosterone concentration without causing a change in growth hormone level. However, when the power output remained close to maximum, but application of force was increased and number of series decreased (in the last weightlifting session), no significant hormone changes were detected.

Sprinters performed exercises at maximal power using 80% 1RM. Although the number of series was not extremely high, the load was subjectively perceived as very high. In men, blood concentrations of lutropin, testosterone, and cortisol decreased in comparison with presession values. It can be suggested that the inversed hormonal responses were related to pronounced fatigue during the session. This possibility was confirmed by a significant decrease of average power in full squats and half squats, as well as by an increased EMG/power ratio in full squats during the session. Females did not demonstrate these changes, but instead demonstrated an increase in EMG/power ratio. Lutropin, testosterone, and cortisol levels did not change in female sprinters (2). The decreased blood testosterone concentration in bodybuilders might also be related to fatigue. A correlation was found between the changes in EMG/power ratio and the decrease in serum testosterone.

In conclusion, short-term exercises founded on high-rate application of muscle forces cause increased blood levels of norepinephrine when exercise duration is only 6 seconds or more. In exercises lasting 40–60 seconds, pronounced increases appear in blood levels of epinephrine, cortisol, ß-endorphine, testosterone, and even thyrotropin and triiodothyronine but not in levels of growth hormone, insulin-like growth factor I, prolactin, and thyroxine. Training sessions consisting of frequent repetitions of power exercices may result in either decreased testosterone and increased growth hormone levels or increased testosterone concentration without changes in growth hormone level. Appearance of these 2 variants seems to depend on the degree of force application. In training sessions of 1-hour duration, testosterone and cortisol levels may decrease as a fatigue phenomenon.

Significance of Hormonal Responses

The acyclic high-resistance or power efforts are too brief to assume that triggered hormonal responses may have any significance on the performance of the same effort. When these acyclic exercises are repeated, the possibilities for contribution of hormones increase. This raises a question regarding the locus of action of hormones that may be essential for rapid influence on neuromuscular performance. It has been speculated that the rapid hormonal influence on intracellular calcium shifts in muscle fiber or neurons may have significance. Unfortunately, these suggestions still do not have convincing experimental evidence. Nonetheless, 2 real possibilities exist that make links between hormonal responses during resistance or power exercises and performance. These are as follows: 1) triggering long-term adaptation processes and 2) the hormonal preconditioning effect.

The main result of strength training is myofibrillar hypertrophy. This process is founded on induction of synthesis of myosin and actin. Experiments with rats showed that training induced muscle hypertrophy is controlled at transcription, translation, and posttranslation levels, whereas the choice of exercises determines the relative significance of contribution of actions at each control level (1). Therefore, hormonal effects on muscle hypertrophy should vary depending on the exercise used. While various metabolic factors can induce this process, testosterone is a powerful amplifier of the induction of contractile protein synthesis (52). In rats, a pharmacological blockade of testosterone receptors prevented training-induced muscle hypertrophy (22). At the same time, muscular activity increases the number of androgen receptors in rat muscles (21). Control of testosterone action at the receptor level ensures fiber-type specific stimulation of protein synthesis in muscles during the postexercise recovery period. Resistance exercises result in down-regulation of androgen receptors in slow-twitch fibers and up-regulation of these receptors in fast-twitch fibers (10). Consequently, the tissue's susceptibility to testosterone's effect increases selectively during resistance exercise in fast-twitch muscle fibers.

In men, the testosterone effect on muscle strength and protein synthesis has been convincingly demonstrated (29, 49). The testosterone effect is supported by thyroid hormone action at the level of transcription. Growth hormone, growth factors, and insulin stimulate the actualization of the protein synthesis at the translation level (52).

There is evidence that the initial level of testosterone is related to performance in power (explosive strength) exercises (5, 32). Because performance in these exercises is positively related to the percentage of fast-twitch fibers (3), it can be hypothesized that a comparatively high level of blood testosterone is common in people with fast-contracting muscles. In turn, the significance of testosterone for explosive power and maximal speed may be founded on a causal relationship between the pubertal development of fast-twitch fibers and testosterone concentration, as established in animal experiments (11). High-level sprinters have high basal levels of testosterone (6).

Another way to understand the significance of testosterone in power exercises is the preconditioning effect. Testosterone is known to contribute as a predisposition for aggressive behavior (41). By analogy, it is possible to assume the same in regard to performance in explosive power and sprint exercises.

Epinephrine is capable of activating the reticular formation in the brainstem and, thereby, increasing the excitability of nervous centers, including the motor cortex (9, 43). The latter is an essential condition for high-level performance, particulary for performance in brief power exercises. Thus, hormonal responses evoked by strength or power exercises are essential for the metabolic control during the postexercise recovery. However, hormone levels before exercise may have significance for the neuromuscular performance.

Conclusion

Short-term high resistance and power exercises are capable of introducing various hormonal changes. However, most of the evoked hormonal responses appear during the postexercise recovery period. Pronounced hormonal changes also appear during exercise sessions for improved strength or power. These changes (increased levels of testosterone, growth hormone, and insulin-like growth factor I) are related to induction of adaptive protein synthesis during long-term recovery. Hormonal responses with significance for energy metabolism such as epinephrine and cortisol occur simultaneously. The main significance of hormonal responses triggered by resistance or power exercises is the induction of adaptive protein synthesis, and especially of contractile proteins. Another possibility is that hormones (testosterone, epinephrine) are essential for preconditioning the neuromuscular apparatus enabling maximal performance.

The problem of hormonal preconditioning of neuromuscular performance should be considered as an important issue for further studies. Although the long-term adaptive action of testosterone has been convincingly demonstrated, the links between exercise specificity and testosterone anabolic effects, as well as the coactions of testosterone, growth hormone, and growth factors are also matters for future research. Attention should also be paid to the possibility that adaptive protein synthesis during the recovery period may involve nervous tissue (48). ▲

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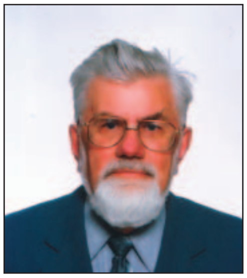

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