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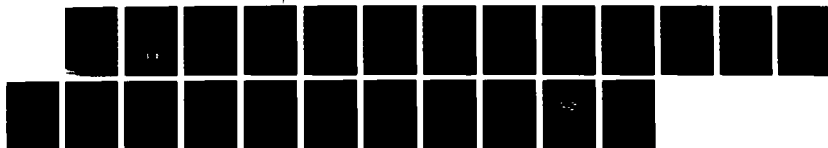
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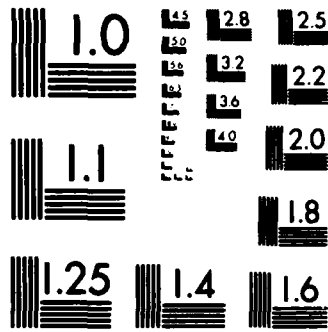
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Report no. M59-87

Endocrine Responses to Resistance Exercise

Running Head: Endocrine Responses

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Endocrine Responses to Resistance Exercise

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Abstract

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The purpose of this brief review is to examine resistance training responses of selected hormones related to acute stress and growth promoting actions. Hormonal mechanisms appear to be involved with both short-term homeostatic control and long-term cellular adaptations. Few studies have modeled the exercise stimulus in resistance training to determine the role of different exercise variables to the hormonal response. A variety of resistance exercise protocols result in increases in peripheral hormonal concentrations. It appears that single factor variables such as the intensity (% of RM) of exercise and amount of muscle mass utilized in the exercise protocol are important determinants of hormonal responses. The volume (sets x repetitions x intensity) of exercise, also appears to be an important determinant of hormonal response. Still, little is known with regard to other single and multiple factor variables (e.g., rest period length) and their relationships to peripheral hormonal alterations. Collectively, such information will allow greater understanding concerning the nature of the exercise stimulus and its relationship to training adaptations resulting from heavy resistance exercise. *Keywords: luteinizing hormone; follicle stimulating hormone; adrenal cortex, testosterone*

INTRODUCTION

It has been established that heavy resistance training results in muscle tissue growth and strength development. Acute resistance exercise provides strong stimuli which activate the neuroendocrine system. When attempting to understand these neuroendocrine responses, the diversity of both the physiological roles of each hormone and the specific exercise stimulus must be considered. Hormonal mechanisms most assuredly help mediate both short-term homeostatic control and long-term cellular adaptations to resistance exercise and training.

Few studies have tried to model the exercise stimulus in resistance training when examining hormonal responses. This is necessary, if we are to identify the specific exercise variables which alter peripheral circulating concentrations of hormones (25). The type of exercise (e.g. leg press or bench press), the order of exercise (e.g. large muscle group vs small muscle group), the rest period lengths, the load (i.e. % one repetition maximum), and number of sets are variables which might all effect the endocrine responses to resistance exercise. The specific combination of these variables in different resistance training programs may be related to the effectiveness of the exercise stimulus in eliciting training adaptations. This brief review examines the resistance training responses of selected hormones related to acute stress and growth promoting actions.

ANDROGENS

The differences in muscular development between males and females have been attributed to the anabolic actions of the male sex hormone testosterone (2,15). Primary and secondary sex characteristics are also dependent upon testosterone's androgenic actions.

Hypothalamic gonadotropin releasing hormone stimulates the secretion of both anterior pituitary gonadotrophins, luteinizing hormone (LH) and follicle-stimulating hormone (FSH). LH provides the primary stimulus for production of testosterone in the interstitial cells of Leydig in the testes. FSH appears to potentiate the effectiveness of LH in testosterone production. Testosterone is also produced in small amounts from cells in the zona reticularis of the adrenal cortex. In females this may represent a major source of testosterone.

It has been suggested in the literature that testosterone may act in an anticatabolic or anabolic role in the muscle. The majority of evidence now indicates that testosterone acts as an anabolic hormone through a direct interaction with a cytoplasmic receptor leading to the typical migration of the hormone-receptor complex to the nucleus, resulting in RNA synthesis and muscle protein accumulation (17). Dramatic differences are apparent in this mechanism when examining skeletal muscle and reproductive tissue. In skeletal muscle there is typically preferential binding of testosterone rather than dihydrotestosterone to the muscle receptor(17).

Several mechanisms have been proposed to explain exercise induced increases in peripheral circulating testosterone including: increased hemoconcentration, reduction in the clearance rates from hepatic and extra hepatic (e.g. adipose tissue) sites, and stimulation of β receptors in the testis (10,13,24,17). Differences in response may be due to age, health and fitness levels (33,40). Also, the effects of an anticipatory rise prior to exercise must be evaluated in some research designs: resting testosterone values range from 14.0 to 28.0 nmol \cdot L⁻¹ (48). It is possible, due to the long half life of testosterone originating from three metabolic pools, that significant effects could be realized from exercise induced increases (29). Previous studies have demonstrated

that both high intensity aerobic and anaerobic exercise induces increases in serum testosterone concentrations (10,13,27,28,43).

Examining heavy resistance exercise Fahey and co-workers (16), utilizing the deadlift exercise with a high intensity (i.e.5RM) and low volume (i.e. one exercise and five sets) exercise protocol, demonstrated an increased post-exercise serum testosterone value regardless of the skill level of the subject. Conversely, Guezennec and co-workers (19) found no changes in serum testosterone values pre-to post-exercise when a moderate intensity (i.e. 8 repetitions at 70% 1RM) and low volume (i.e. one exercise and seven sets) bench press exercise protocol was utilized. Volume effects were suggested in a study by Weiss and co-workers (46) who observed that a moderate intensity (i.e. 80% 1RM) and moderate volume (i.e. 3 sets of 4 exercises) exercise session resulted in a post-exercise increase in serum testosterone concentrations. These studies suggest that three exercise variables may effect the serum testosterone concentrations following an acute bout of resistance exercise. These include the intensity of the exercise (i.e % 1RM), the volume of the exercise (i.e. mass x repetitions x sets) and the amount of muscle tissue used in the exercise protocol. Thus, there may be a threshold intensity, volume and muscle mass necessary to produce increases in testosterone concentrations. The impact of other single exercise variables (e.g. rest period length) of other combinations of variables (e.g. low intensity and high volume) on testosterone concentrations remains to be determined.

Evidence suggests that resting testosterone concentrations may remain unaltered by resistance training. One exception to this finding was demonstrated by Stone and co-workers (40) in older (42 ± 12 yrs) untrained males where increases were observed over 12 wks of resistance training. The significance of this observation remains to be established.

Whether testosterone acts in an anticatabolic role to produce its anabolic actions in the muscle cell by displacement of cortisol from the glucocorticoid receptor is debatable. Still, the testosterone/cortisol ratio has been used as an indicator of changes in the body's anabolic-androgenic activity. All resistance training programs may not result in an increased testosterone/cortisol ratio. Hakkinen and co-workers (21) reported an increased resting testosterone/cortisol ratio after 16-20 weeks of heavy resistance training. A subsequent decrease was observed over 12 weeks of detraining. Conversely, Guezennec and co-workers (19) observed no increases in the resting testosterone/cortisol ratio over 16 weeks of strength training. Since both studies observed increases in strength over the training period, the precise factors which effect the testosterone/cortisol ratio remain to be established, as well as the exact contribution made by testosterone in any anticatabolic role in skeletal muscle. Still, Hakkinen et al. (21) was able to demonstrate significant relationships between maximal isometric leg extensor strength and testosterone/cortisol ratios during the last four weeks of a 24 week training program. Recently, Hakkinen and co-workers (20) have shown testosterone/sex hormone binding globulin ratios to be related to maximal strength weightlifting performances.

Limited data showing no changes in testosterone concentrations following heavy resistance exercise have been reported for high school age males (16). Fahey (16) postulated that this was attributed to a possible lack of experience and motivation. It is also possible that the exercise stimulus in this situation was not of adequate intensity or volume to elicit increases for this age group.

The majority of studies reported have not shown increases in serum testosterone concentrations for females following heavy resistance exercise or training (16,23,46,47).

In a recent study by Cumming et al. (12) who addressed previous research design problems (e.g. anticipatory rise), a significantly higher resting baseline for total testosterone was observed for trained subjects compared to inactive controls. The resting and exercise responses in this study may have been due to better basal quantification and an exercise protocol which utilized short rest periods (1 min),² multi-exercise protocol with high intensity and volume. Still, the significantly higher resting and exercise induced concentrations of testosterone in males are factors hypothesized to contribute to differences in muscular hypertrophy in males and females.

The possible compensatory response and role of other hormones (e.g. growth hormone and somatomedians) in females to heavy resistance exercise, and subsequent muscle tissue growth has yet to be examined. Estrogen responses to heavy resistance exercise remain unknown. There have been few reports of the effects of estrogens on muscle tissue growth (17). To date, a direct anabolic role for estrogens in muscle tissue growth remains to be established (14,17).

GROWTH HORMONE AND SOMATOMEDINS

The secretion of growth hormone (GH) is controlled by the hypothalamus (32). The hypothalamus contains growth hormone-releasing hormone (GH-RH) and growth hormone inhibiting hormone, somatostatin. The synthesis, metabolism and release of hypothalamic hormones are thought to be regulated by a number of neurotransmitters (e.g. dopamine, serotonin, norepinephrine). Thus, modulation of GH-RH and somatostatin release controls GH secretion. Many other factors have been shown to influence GH release including gonadal, thyroid and adrenal hormones (32). Whether it is via a direct or indirect mechanism of action is unclear. Endogenous opioid peptides exert another level of control and can influence GH release by acting on

hypothalamic neurotransmitters or by acting directly on somatostatin or GH-RH secreting neurons (6,38). Physiological stimuli such as exercise, sleep, diet and stress all can stimulate GH release via hypothalamic interactions (8,9,11,28,32).

For over 50 years it has been realized that GH is essential for normal growth of skeletal muscle. In addition, GH has been shown to be involved in a wide variety of biological actions related to maintaining the body's normal structure and metabolic function (18,38,39). However, when examining skeletal muscle development it is not possible to attribute all anabolic effects of GH in muscle to a stimulation of cell proliferation especially in post-mitotic mature muscle (17). One must also keep in mind the importance of multiple hormone interactions related to skeletal muscle growth when evaluating endocrine ablated and hormone treated animal studies (17).

Of particular interest is the question whether GH acts directly or via a somatomedin-mediated mechanism in skeletal muscle. Somatomedians appear to be highly involved in regulatory feedback and GH release (1,5). It has also been demonstrated that a number of other tissues, besides liver, can produce somatomedins (22). Somatomedins are active in the stimulation of anabolic processes in muscle and the majority of evidence indicates that GH acts through somatomedin activity (17). Insulin can act through both somatomedin and insulin receptors to enhance muscle tissue growth (17). It appears that anabolic actions of insulin on muscle may be mediated by a decreased rate of tissue proteolysis. Direct and indirect interactions between insulin's potential anabolic actions and the catabolic actions of glucocorticoids remains to be fully elucidated. Furthermore, the relative contributions and time course of insulin and somatomedin activity to exercise induced muscle tissue growth remains

to be demonstrated. Some problems have arisen with interpretation due to insulin's crossreactivity with somatomedin receptors. No data are available which have examined responses of somatomedins to resistance exercise.

Only limited data are available regarding the responses of circulating concentrations of GH to resistance exercise (31,37,45). The exact contribution of exercise-induced alterations GH to muscle tissue growth and the importance of heavy resistance exercise as an exercise modality used to elicit these changes remains to be determined.

VanHelder and co-workers (45) and Lukaszewska and co-workers (31) have shown that it is possible to stimulate increased serum concentrations of GH following an acute resistance training exercise session. It would appear from these two studies that the intensity (i.e. the mass lifted) of the exercise is an important factor for production of increased serum concentrations. This was demonstrated by an absence of change when 28% of a 7RM was used to perform 21 repetitions in 30 sec for seven sets of a leg press exercise (45). Conversely, heavier loads ranging from 85% of a 7RM (45) to 70-85% of a 1RM (31) in an olympic lift resulted in increased serum concentrations of GH following exercise (1.8 baseline to $4.5 \pm 0.4 \mu\text{g} \cdot \text{L}^{-1}$ and $6.2 \text{ -- } 3.9 \text{ to } 44.0 \text{ -- } 29.0 \text{ ng} \cdot \text{L}^{-1}$, respectively). The effects of higher volumes of exercise (i.e. set x repetitions x loads) and/or higher intensities (i.e % 1RM) remains to be determined. Also, the effect of resistance exercise on concomitant somatomedin production is yet unknown. It has been proposed that both lactate and/or the oxygen deficit may have regulatory roles for GH responses to resistance training (45). It has also been demonstrated that hypoxia, changes in body temperature during the exercise session, fitness, changes in clearance rates and hemoconcentrations may also effect GH and thus somatomedin responses

(7.8.37.41.42.44). More research will be needed to determine the exact contribution of different exercise variables and their relationships to observed hormone concentrations and training adaptations.

Only scant data are available concerning changes in serum GH values with resistance training. Again, Lukaszewska and co-workers (31) showed no differences in immediate-post exercise serum GH concentrations over 3 years following a 30 min Olympic weight lifting session (i.e. 18-25 snatch lifts at 70-85% max). Mean differences were observed in 30 min post-exercise values. These changes may be related to differences in the subjects age, absolute loads used or possible circadian differences in GH pulsatile frequency and amplitude. Considerably more research is needed to evaluate the meaning of GH alterations in peripheral blood and how it relates to long-term training adaptations.

ADRENAL HORMONES

The adrenal glands were first described by Eustacchio in 1563 and their functions remained relatively unknown until the 1800's when Addison described symptoms of individuals who had diseased adrenals (3). By the early 1900's the concept of "split functions" for the adrenal gland became a viable hypothesis. Over the past 30 years further elucidation of adrenal function and hormonal content, including endogenous opioid peptides (i.e. enkephalins), have been realized.

Cortisol

The adrenal cortex has long been connected with "stress" responses. Over 50 years ago, Selye reported enlargement of the adrenal cortex and development of a

"stress syndrome" resulting from a variety of noxious agents. Stressful events including exercise cause release of adrenocorticotrophic hormone (ACTH) from the anterior pituitary. Secretion of ACTH is regulated primarily by corticotropin releasing factors, and may also be influenced by catecholamines and other hormones. It stimulates the synthesis of corticosteroids in the adrenal cortex. In turn, elevated cortisol levels in the blood provide negative feedback on ACTH release from the pituitary thus creating a major cybernetic regulatory pathway (3).

Increased serum cortisol concentrations have been observed following acute bouts of resistance exercise. Similar changes have been observed for various heavy resistance exercise routines (12,19,26). Studies have also demonstrated the absence of any changes (19,31). In all of these studies the effects of blood drawing methods, circadian differences, measurement time course and psychological factors cannot be ruled out as possibly masking the effects of the resistance exercise on the magnitude of cortisol changes observed.

The catabolic actions of glucocorticoids in skeletal muscle have become established (3). It also appears there may be considerable variation in glucocorticoid effects on proteolytic enzymes in different muscle fiber types. The modulation of these actions are also related to many other factors including hormonal interactions (e.g. insulin, testosterone, growth hormone etc.) and diet. The final result is a net gain or loss of muscle proteins (17).

How cortisol changes interact with different resistance training programs requires more clarification. The potential use of this hormone or a specific ratio with another hormone (e.g. testosterone/cortisol or insulin/cortisol) to indicate potential "overtraining syndromes" or indicate the training status of the individual also requires further study.

Catecholamines

Walter Cannon recognized the potential roles and rapid responses of the sympathomedullary system to acute physiological stress. Physical activity, including acute resistance exercise, results in a release of epinephrine and norepinephrine from the adrenal medulla and sympathetic neurons (30). In addition to typical epinephrine and norepinephrine increases, Kraemer and co-workers observed significant (26) increases in dopamine values following a high intensity, low-rest bodybuilding type exercise protocol. This observation was attributed to the possible inability to convert dopamine to norepinephrine due to limitations in enzymatic conversion (4) under such severe exercise conditions (<1 min rest, plasma lactate >20 mmol \cdot L $^{-1}$). Changes in catecholamine metabolism in response to resistance exercise appear to be primarily related to the force of muscular contraction, the amount of muscle tissue stimulated, and the frequency of force application (i.e. amount of rest between sets and repetitions). Exercise protocols, which utilize multi-exercise and high intensity resistance exercise, produce catecholamine concentrations similar to heavy anaerobic sprint and cycle exercise which are greater than those values reported consequent to aerobic activities.

Few studies have examined the effects of catecholamines on skeletal muscle growth. It has been observed that epinephrine may produce additive effects on rate limiting reactions in protein metabolism (17).

Resistance exercise training had been shown to reduce the magnitude of epinephrine response to a single bench press exercise protocol, while lactate production increased (19). No training changes were observed for plasma norepinephrine despite significant strength gains. Whether similar results would be observed if larger muscle

groups were involved, or if multi-exercise sessions were used, remains to be determined.

FUTURE TRENDS

It is quite obvious that a great deal of research is still required to fully understand the mechanisms which explain circulating hormonal responses to resistance exercise and training. The net effects of hormone release are related to complex interactions with other hormones, cell receptors, nuclear acceptors, external factors and the type of resistance exercise stimulus. The relationship between peripheral hormonal alterations and resistance training adaptations, related to muscle strength and a variety of tissue changes, need to be examined. Both in vivo and in vitro studies appear necessary. Collectively, such information will allow greater understanding of the physiological mechanisms involved in the adaptation to heavy resistance training.

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Disclaimers

The views, opinions, and or findings contained in this report are those of the author(s) and should not be construed as any official Department of the Army position, policy or decision unless so designated by other official documentation.

REFERENCES

1. Abe, H., M.E. Molitch, J.J. Van Wyk, and L.E. Underwood. Human growth hormone and somatomedin D suppress the spontaneous release of growth hormone in unanesthetized rats. Endocrinology 113:1319-1324, 1983.
2. Alen, M. and K. Hakkinen. Physical health and fitness of an elite bodybuilder during 1 year of self-administration of testosterone and anabolic steroids: a case study. Int. J. Sports Med. 6:24-29, 1985.
3. Allaben, William T. The adrenal cortex. In: Handbook of Endocrinology. G. H.Gass and H.M. Kaplan (Eds.) Boca Raton, FL: CRC Press, Inc., 1982, pp. 189-228.
4. Axelrod, J., T.D. Reisine. Stress hormones: their interaction and regulation. Science 224:452-460, 1984.
5. Berelowitz, M., M. Szabo, S. Firestone, L.A. Froehman, and L.Chu. Somatomedin-C mediates growth hormone negative feedback by effects on both the hypothalamus and the pituitary. Science 212: 1279-1281, 1981.
6. Borer, K.T., D.R.Nicoski, and V. Owens. Alteration of pulsatile growth hormone secretion by growth-inducing exercise: involvement of endogenous opiates and somatostatin. Endocrinology 118: 844-850, 1986.
7. Bratusch-Marrain, P., O. Bjorkman, J. Wahren and W. Waldhausi. Hepatic disposal of endogenous growth hormone and prolactin in man. J. Clin. Invest. 9: 257-260, 1979.
8. Buckler, J.M.H. The relationship between exercise, body temperature and plasma growth hormone levels in a human subject. J. Physiol. 214: 25P-26P, 1971.
9. Bunt, J.C., R.A. Boileau, J.M. Bahr, and R.A. Nelson. Sex and training differences in human growth hormone levels during prolonged exercise. J. Appl. Physiol. 61:1796-1801, 1986.
10. Cadoux-Hudson, T.A., J.D. Few, and F.J. Imms. The effect of exercise on the production and clearance of testosterone in well trained young men. Eur. J. Appl. Physiol. 54:321-325, 1985.
11. Coiro, V., R. Volpi, P. Muzzetto, et al. Cholinergic-muscarinic receptors participate in growth hormone secretion induced by lysine-8-vasopressin in man. Horm. Metab. Res. 17:316-317, 1985.
12. Cumming, D.C., S.R.Wall, M.A. Galbraith and A.N. Belcastro. Reproductive hormone responses to resistance exercise. Med. Sci. Sports Exerc. 19:234-238, 1987.
13. Dohm, G.L. and T.M. Louis. Changes in androstenedione, testosterone, and protein metabolism as a result of exercise. Proc. Soc. Exp. Biol. Med. 158(4): 622-625, 1978.

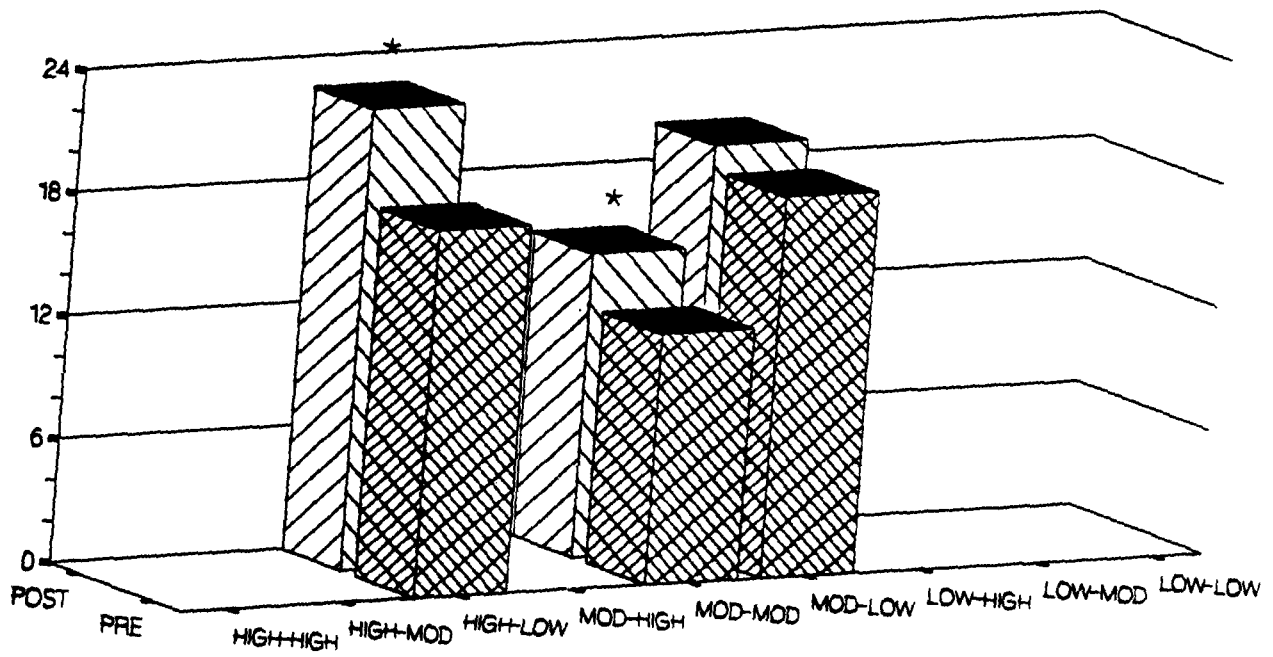
14. Edgerton, R.V. Exercise and the growth and development of muscle tissue. Exercise Sport Science Reviews.
15. Edgerton, V.R., J.J. Garhammer, D.R. Simpson, and D.S. Campion. Case studies of competitive weightlifters taking anabolic steroids. In: Medicine and Sport-Materials of the Soviet-American Symposium. Leningrad:133-141, 1979.
16. Fahey, T.D., R. Rolph, P. Moungmee, J. Nagel, and S. Mortara. Serum testosterone, body composition, and strength of young adults. Med. Sci. Sports. 8:31-34, 1976.
17. Florini, J. R. Hormonal control of muscle cell growth. J. Animal Sci. 61:21-37, 1985.
18. Goldberg, A.L. and H.M. Goodman. Relationship between growth hormone and muscular work in determining muscle size. J. Physiol. 200:655-666, 1969.
19. Guezennec, Y., L. Leger, F. Lhoste, M. Aymonod and P.C. Pasquies. Hormone and metabolite response to weight-lifting training sessions. Int. J. Sports Med. 7:100-105, 1986.
20. Hakkinen, K., A. Pakarinen, M. Alen, H. Kauhanen and P.V. Komi. Relationships between training volume, physical performance capacity, and serum hormone concentrations during prolonged training in elite weight lifters. Int. J. Sports Med. 8:61-65, 1987.
21. Hakkinen, K., A. Pakarinen, M. Alen and P.V. Komi. Serum hormones during prolonged training of neuromuscular performance. Eur. J. Appl. Physiol. 53:287-293, 1985.
22. Han, V.K.M., A.J. D'Ercole and P.K. Lund. Cellular localization of somatomedin (insulin-like growth factor) messenger RNA in the human fetus. Science. 236:193-197, 1987.
23. Hetrick, G. and J. Wilmore. Androgen levels and muscle hypertrophy during an eight-week weight training program for men/women. Med. Sci Sports. 11:102-107, 1979.
24. Jezova, D and M. Vigas. Testosterone response to exercise during blockade and stimulation of adrenergic receptors in man. Hormone Res. 15:141-147, 1981.
25. Knuttgen, H.G. and W.J. Kraemer. Terminology and measurement in exercise performance. J. Appl. Sport Sci. Res. 1:1-10, 1987.
26. Kraemer, W.J., B.J. Noble, M.J. Clark, and B.W. Culver. Physiologic responses to heavy-resistance exercise with very short rest periods. Int. J. Sports Med. 8: (in press).

27. Kuoppasalmi, K. and H. Adlercreutz. Interaction between catabolic and anabolic steroid hormones in muscular exercise. In: Exercise Endocrinology, K. Fotherby and S.B. Pal (Eds.). Berlin: Walter de Gruyter, 1985, pp. 65-98.
28. Kuoppasalmi, K., H. Naveri, S. Rehunen, M. Harkonen and H. Adlercreutz. Effect of strenuous anaerobic running exercise on plasma growth hormone, cortisol, luteinizing hormone, testosterone, androstenedione, estrone, and estradiol. J. Ster. Biochem. 7:823-829, 1976.
29. Lamb, D.R. Androgens and exercise. Med. Sci. Sports. 7:1-5, 1975.
30. Lehmann, M. and J. Keul. Free plasma catecholamines, heart rates, lactate levels, and oxygen uptake in competition weight lifters, cyclists and untrained control subjects. Int. J. Sports Med. 7:18-21, 1986.
31. Lukaszewska, J., B. Biczowa, D. Bobilewicz, M. Wilk and B. Obuchowicz-Fidelus. Effect of physical exercise on plasma cortisol and growth hormone levels in young weight lifters. Endokrynologia Polska XXVII. 2:149-158, 1976.
32. Martin, J.B. Growth hormone releasing factor. In: Brain Peptides, D. T. Krieger, M.J. Brownstein and J.B. Martin (Eds.). New York: John Wiley and Sons, 1983, pp. 976-980.
33. Remes, K., K. Kuoppasalmi and H. Adlercreutz. Effect of physical exercise and sleep deprivation on plasma androgen levels: modifying effect of physical fitness. Int. J. Sports Med. 6:131-135, 1985.
34. Rennie, M.J., D.M. Park and W.R. Sulaiman. Uptake and release of hormones and metabolites by tissues of exercising leg in man. Am. J. Physiol. 213(3):967-972, 1976.
35. Salvatore, D., A.F. Miranda, S. Khan and K. Gitlin. Somatomedin-C mediates growth hormone negative feedback by effects on both the hypothalamus and the pituitary. Science 212:1279-1281, 1981.
36. Shepard, R.J. and K.H. Sidney. Effects of physical exercise on plasma growth hormone and cortisol levels in human subjects. In: Exercise and Sport Sciences Reviews, J.H. Wilmore and J.F. Keogh (Eds.). New York: Academic Press Inc., 1975, pp. 1-29.
37. Skierska, E., J. Ustupka, B. Biczowa and J. Lukaszewska. Effect of physical exercise on plasma cortisol, testosterone and growth hormone levels in weight lifters. Endokrynologia Polska XXVII 2:159-165, 1976.
38. Sonntag, W.E., L.J. Forman, N. Miki, and J. Meites. Growth hormone secretion and neuroendocrine regulation. In: Handbook for Endocrinology, G.H. Gass and H.M. Kaplan (Eds.) Boca Raton, FL: CRC Press, Inc., 1982, pp. 35-59.
39. Steger, R.W. and J.J. Peluso. Anterior pituitary. In: Handbook of Endocrinology, G.H. Gass and H.M. Kaplan (Eds.), Boca Raton, FL: CRC Press, Inc., 1982, pp. 27-33.

40. Stone, M.H., R. Byrd, and C. Johnson. Observations on serum androgen response to short term resistive training in middle age sedentary mates. National Strength and Conditioning Association Journal.
41. Sutton, J.R. Effect of acute hypoxia on the hormonal response to exercise. J. Appl. Physiol.: Respir. Environ. Exercise Physiol. 42: 587-592, 1977.
42. Sutton, J.R. Hormonal and metabolic responses to exercise in subjects of high and low work capacities. Med. Sci. Sports 10: 1-6, 1978.
43. Sutton, J.R., M.J. Coleman, J. Casey, and L. Lazarus. Androgen responses during physical exercise. Br. Med. J. 1:520-522, 1973.
44. Sutton, J.R., J.D. Young, L. Lazarus, J.B. Hickie, and J. Maksvytis. The hormonal response to physical exercise. Austral. Ann. Med. 18: 84-90, 1969.
45. Vanhelder, W.P., M.W. Radomski, and R.C. Goode. Growth hormone responses during intermittent weight lifting exercise in men. Eur. J. Appl. Physiol. 53: 31-34, 1984.
46. Weiss, L.W., K.J. Cureton, and F.N. Thompson. Comparison of serum testosterone and androsteredione responses to wieght lifting in men and women. Eur. J. Appl. Physiol. 50: 413-419, 1983.
47. Westerlind, K.C., W.C. Byrnes, P.S. Freedson, and F.I. Katch. Exercise and serum androgens in women. The Physician and Sportsmed. 15(5): 87-94, 1987.
48. Young, Donald S. Implementation of SI Units for clinical laboratory data. Ann. Intern. Med. 106: 114-129, 1987.

Figure 1: Testosterone responses to resistance exercise as it relates to an intensity-volume continuum (16.19.46). * = $p < 0.05$.

SERUM TESTOSTERONE (nmol/L)



INTENSITY-VOLUME

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