

Exercise Training and Growth Hormone 運動訓練與生長激素

Lo Cho Yim

Department of Physical Education

Hong Kong Baptist University, HONG KONG

盧灶炎

香港浸會大學體育學系



Abstract

The present paper attempts to discuss the relationship between the Growth Hormone and exercise intensity. Growth Hormone causes growth, enhances fat utilization and glycogen deposition, decreases use of glucose, etc. The high intensity training physical training may increase the release of Growth Hormone.

摘要

本文章嘗試探討生長激素與運動訓練的關係。生長激素能促進人體生長、增加蛋白質的合成、和減低糖原的消耗。在高運動強度的訓練時，人體分泌生長激素會增加，此增長也有利蛋白質的合成。

Introduction

Growth hormone (GH), also called somatotrophic hormone or somatotropin, is a small peptide containing 191 amino acids in a single chain and has half-life in the blood of less than 20 minutes. It is known that GH causes growth, enhances fat utilization and glycogen deposition, decreases use of glucose, diminishes uptake of glucose and increases protein synthesis (Guyton & Hall, 1996). The increase in blood GH with exercise was initially reported by Roth et al. in 1963 (Karagiorgos et al., 1979). Since then, the GH response to exercise has been studied extensively. Sutton et al. (1976) documented that comparing exercise at 300, 600 and 900 kgm/min (75-90% of the subjects' VO₂ max) for 20 min, the 900 kgm/min level produced the largest amount of GH secretion. Their data also suggested that there was a correlation between blood GH and lactate (LA) levels. Van Helder et al. (1987) demonstrated that GH release has a linear relationship with LA level in resistance training. In a one-year running study, Weltmen et al. (1992) compared the above-lactate threshold (>LT) group to their control at-lactate threshold (=LT) group. They found significant difference in the pulsatile release of GH within these two groups and confirmed that LA level in >LT group had the strongest linear relationship with GH release. Recently, this observation was supported by the research of Chwalbinska-Moneta et al. (1996) in their study of progressive, multistaged exercise. However, when Kragiorgos et al. (1979) examined the GH response to continuous (45%

of VO₂ max) and intermittent exercises, no significant correlation between GH and LA levels was observed although GH and LA levels increased in both protocols. It is concluded that GH response was not related to anaerobiosis or lactate and that LA level was not a determinant factor in the control of GH release to exercise. The studies from other researchers, Kinderman et al. (1982) and Koivisto et al. (1982), supported this conclusion. In view of the contradictory findings of GH with LA correlations, the problem may lie in the GH responses to the intensity of training. Therefore, the aim of this paper is to discuss the effects of the type of exercise and the intensity of training on GH response.

Literature Review

In order to diagnose GH deficiency, Sutton et al. (1976) used exercise, sleep (physiological stimulus), insulin, arginine and L-Dopa (pharmacological stimuli) to stimulate the GH secretion. Eight healthy males, aged 21-24 years, participated in a 6-week study. The subjects rode the cycle ergometer at 3 different intensities, i.e., 300, 600 and 900 kgm/min for 20 minutes in the exercise protocol. The 20 -minute of exercise at 900 kgm/min acquired 70-90% of subjects' VO₂ max and was long enough to attain the above-lactate threshold (>LT). The released GH was supposed to be effective in the blood. Therefore, the intensity and duration of a single exercise bout is pertinent to the diagnosis of GH deficiency. The pharmacological

stimulus methods using insulin, arginine and L-Dopa were also found useful in the diagnosis of GH deficiency. In 1979, Kargiogos et al. used both continuous and intermittent exercises to stimulate GH release. Ten healthy and fit males (20-30 years of age) were given either 40 minutes of continuous cycle ergometer exercise (CE, 45% of VO₂ max) or 20 minute intermittent bouts of exercise (IE, one minute on/off at 2 x of the CE work rate). The mode and duration of exercise in their study appeared to be appropriate to stimulate GH release. However, the exercise intensity of 45% of VO₂ max in CE group may be the reason why there was no significant correlation between GH and LA levels in the blood. Weltmen et al. (1992) studied 21 healthy untrained females in a one-year running program. Several parameters were measured in their studies including the O₂ consumption at the lactate threshold (=LT), fixed blood lactate concentrations of 2.0, 2.5, and 4.0 nM, peak VO₂, maximal VO₂, body composition and pulsatile release of GH. The =LA group (n=9) and >LA group (n=7) completed similar weekly running mileage but at different training intensities. The control group (n=5) did not engage in any running. To avoid the effect of female menstrual cycle (MC) on GH release, for both the =LT and >LT groups, the physiological endocrine assessments were performed during the early follicular phase of the MC (9 days after onset of menses) at baseline and every fourth MC thereafter for the remaining of the 12-14 months of training. The control group was assessed at baseline and also at 12-14 months thereafter. Weltmen et al. (1992) concluded that the mode, intensity and duration of exercise affect the GH release.

Discussion

The evaluation of GH secretory reserve is important for the assessment of children with short stature and adults with suspected hypopituitarism. Provocative tests are usually necessary because base-line level of GH is normally low and can not be used to distinguish between the normal and GH-deficient conditions (Greenspan, 1974). The study by Sutton et al. (1976) confirmed that a single acute exercise bout in cycling ergometry at 900 kgm/min (75-90% of VO₂ max) for 20 min was an appropriate provocative test for GH secretion. However, cautions should be taken, if the lactate threshold is used as the indication for the exercise intensity to induce GH release. Karagiogos et al. (1979) examined 10 healthy and fit young males (20-30 years of age) in continuous and intermittent cycling of 40 min duration and found no significant correlation between GH and LA levels in the blood. This finding was supported by the studies done by Kinderman et al. (1982) and Koivisto et al. (1982). In contrast, the study by VanHelder et al. (1984) showed there was a correlation between blood lactate concentration and GH release during exercise. Their observation was further supported

by Chwalbinska-Monta et al. (1996) in their progressive multistaged exercise study. They found that there was close correlation ($r=0.64$, $p(0.001)$) between GH and lactate levels. This finding was further explained by the study of Weltman et al. (1992). They reported that the pulsatile release of GH was increased significantly in untrained females after one year of running at the above-lactate threshold. Obviously, these results differ from those of Karagiogos et al. and could be explained by the smaller sample size ($n=10$) and the low intensity of the exercise (45% of the minimum load of VO₂ max) in their study and the better fitness of the subjects recruited by Karagiogos et al.

It is known that GH secretion is very important for the growth in children. In one study, young males were divided into two groups to perform resistance training with one group given GH and the other without GH (Robergs & Roberts, 1977). Those who received GH showed the largest increase in lean body mass. Another study conducted by Fryburg et al. (1991) showed that GH increased lean body mass when given to GH-deficient adults. Apparently, GH has its own effect on protein synthesis which is different from that of IGF-I. The timing of high release of GH discovered by Sutton et al. (1976) provided information for appropriate timing of protein compensation. Recent studies show that highly trained athletes and athletes involved in intensive exercise training need extra protein intake to compensate the protein catabolism in their body (Robergs & Roberts, 1997). The finding of maximum GH release at 5, 10 or 15 min after exercise discovered by Sutton et al. provides a better timing for protein supplementation. In 1992, Weltmen et al. reported that chronic endurance training increases the pulsatile release of GH even after the exercise. This indicates that running, the easiest and less costly type of exercise can be helpful in clinical situations where pulsatile secretion of GH is deficient. However, this increased release occurs only when training is above the LT level. It seems that GH secretion is more related to the peak exercise intensity rather than the duration of exercise or total exercise output (Van Helder et al., 1984). This may be explained by the hypothesis that GH release is closely linked to the degree of exertion and probably motor center activities in the brain. This hypothesis is supported by the observation that GH level did not rise in individuals with spinal cord injury during involuntary electrically induced exercise with paralyzed muscles when no motor center activities were present. However, GH level did rise when exercise was conducted with the non-paralyzed muscles (Hoffman-Goetz, 1996).

Conclusion

GH release has a close correlation with LA level and exercise intensity. Thus, the intensity of exercise is a determinant

factor in GH secretion. For the chronic training, only those reaching above the LT level will increase the pulsatile release of GH in 24 hours. However, for acute exercise, a single bout of continuous or intermittent resistance exercise or weight lifting can increase GH secretion and maximize GH release during recovery. These observations provide better timing for protein supplementation. However, further studies are needed to understand GH response to chronic intermittent training and the functions of GH and IGH-I in muscular protein synthesis.

Reference

- Chwalbinska-Moneta, J., et al. (1996). Threshold increases in plasma growth hormone in relation to plasma catecholamine and blood lactate concentrations during progressive exercise in endurance-trained athletes. *Eur. J. Appl. Physiol.* 73: 117-120.
- Fryburg, D.A. et al. (1991). Growth hormone acutely stimulates forearm muscle protein synthesis in normal humans. *Am. J. Physiol.* 260E: 499-504.
- Guyton, A.C. & Hall, J.E. (1996). Textbook of medical physiology (9th ed.). W.B. Saunders.
- Greenspan, F.S. & Baxter, J.D. (1994). Basic and clinical endocrinology (4th ed.). Appleton & Lange.
- Hoffman-Goetz, L. (1996). Exercise and Immune function. CRC press.
- Karagiorgos, A. et al. (1979). Growth hormone response to continuous and intermittent exercise. *Med. Sci. Sports* 11: 302-307.
- Kinderman, W. et al. (1982). Catecholamines, growth hormone, cortisol, insulin and sex hormones in anaerobic and aerobic exercise. *Eur. J. Appl. Physiol.* 49: 389-399.
- Kiovisto, V. & Hendler, R. (1982). Influence of physical training on fuel-hormone response to prolonged low intensity exercise. *Metabolism* 31: 192-197.
- Robergs, R.A. & Roberts, S.O. (1997). Exercise physiology: exercise, performance and clinical applications. Mosby-Year Book.
- Sutton, J. & Lazarus, L. (1976). Growth hormone in exercise: comparison of physiological and pharmacological stimuli. *J. Appl. Physiol.* 41: 523-527.
- VanHelder, W.P. et al. (1984). Growth hormone responses during intermittent weight lifting exercise in men. *Eur. J. Appl. Physiol. Occup. Physiol.* 53: 31.
- VanHelder, W.P. et al. (1987). Regulation of growth hormone during exercise by oxygen demand and availability. *Eur. J. Appl. Physiol. Occup. Physiol.* 56: 628-632.
- Weltmen, A. et al. (1992). Endurance training amplifies the pulsatile release of growth hormone: effects of training intensity. *J. Appl. Physiol.* 72: 2188-2196.