



Acute Exercise-Induced Growth Hormone is Attenuated in Response to Short-Term, High-Intensity Exercise Training

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ABSTRACT

Ritsche K, Smith J, Mellick P, Wideman L. Acute Exercise-Induced Growth Hormone is Attenuated in Response to Short-Term, High-Intensity Exercise Training. **JEPonline** 2014;17(6):1-12. The aim of this study was to determine if 3-wks of short-term, high-intensity exercise training (HIT) alters growth hormone (GH) release. Nineteen recreationally active males (Mean \pm SD) (age = 24.9 ± 3.9 yrs, BF% = 20.1 ± 7.7) participated in this study. Each subject completed a 2-hr resting profile and a 2-hr acute sprint (AS) profile that consisted of one maximal 30-sec Wingate sprint on a cycle ergometer after 30 min of rest. Blood samples were taken every 15 min [Q15] during rest and more frequently [Q1-Q10] immediately following the sprint. Short-term, HIT consisted of 4-6 repetitions of 30-sec maximal sprints relative to body mass, 3 times \cdot wk⁻¹ with an additional AS profile at the end of each week of training for 3 wks. Peak power (PP) and fatigue index (FI) significantly increased while mean power (MP), minimum power (MinP), time to peak power (TTPP), and total work per sprint (TW) were unchanged after 3 wks of HIT. Total body mass significantly increased and was confirmed by a significant increase in lean mass of the lower extremities. Growth hormone area under the curve (AUC) and peak GH were significantly decreased after the first week of HIT despite no change in time to reach peak GH. One week of HIT significantly decreased GH release, with a simultaneous significant increase in anaerobic power and lean body mass of the lower extremities.

Key Words: GH, Lean Body Mass, Anaerobic, Exercise Performance

INTRODUCTION

Human growth hormone (GH) is one of the seven-peptide hormones produced and secreted from the anterior lobe of the pituitary gland (22). In the plasma, most GH is bound to GH-binding proteins and is taken up by specific GH receptors on target cells and thus, has effects on local tissue such as increased lipid metabolism via increased free fatty acid (FFA) mobilization and decreased triglyceride formation (13,23). Additionally, GH has been linked to the increase in lean muscle mass and strength (8).

Alterations in exercise intensity and duration (equating to increases in workload) have been shown to increase the GH response in a positive linear fashion (24-27). Furthermore, shorter bouts of high-intensity exercise elicit an elevated growth hormone response. Peak GH secretion occurs ~30 to 40 min after the beginning of a sprint exercise and one acute bout of maximal exercise as short as 6 sec (19) can stimulate a significant GH pulse that typically returns to baseline within 90 to 120 min (18-21).

While the effects of acute exercise on GH release are well documented, there is less agreement in the literature about the effect of training on the resting and exercise-induced GH response. This outcome is likely due in part to the various populations and training protocols utilized. Although one training study in elite swimmers has reported an augmented GH response to 18 wks of training (1), most controlled training studies have reported that training attenuates the exercise-induced GH response (3,5,17,28). Previous research (17) has shown that peak GH concentration was decreased by 40% after 6 wks of short-term, high-intensity sprint training and similar reports have indicated that the GH response is attenuated after only 3 wks of training (28).

The primary purpose of this study was to examine the time course and magnitude of the GH adaptation, anthropometric and anaerobic performance changes to 3 wks of high-intensity sprint exercise training on a cycle ergometer using a weekly measure of hormonal responses and exercise performance. We hypothesized that the exercise-induced GH response to an acute sprint (Wingate test) would be blunted with sprint training, but the physiological adaptation would occur much sooner than previously reported. Secondly, sprint interval training has been shown to reduce fat mass and increase fat-free mass in as little as 6 wks of training consisting of 3 days·wk⁻¹ of 4 to 6 30-sec maximal sprints (7). Furthermore, it has been reported that 6 sessions of sprint-training with 1 to 2 rest days between each session significantly increased muscle oxidative potential, increased cycling endurance capacity, and elevated peak power output (2). Similar to the GH adaptation, we believe these anthropometric and anaerobic performance changes may occur earlier than 6 wks with high-intensity sprint training but would have no association with the exercise-induced GH response.

METHODS

Subjects

Nineteen recreationally active male subjects (24.9 ± 3.9 yrs) participated in this study. The subjects were screened prior to participation for contraindications to exercise and factors known to affect GH secretion, including hematological, renal, hepatic, metabolic, and thyroid function. Subjects were excluded if they: (a) had a BMI less than 18 or greater than 30 kg·m⁻²; (b) reported a history of hematological, renal, hepatic, metabolic, or thyroid dysfunction; (c) were currently on a caloric restriction program; (d) participated in more than 10 hrs of recreational activities (swimming, basketball, jogging, cycling, etc.) per week; and/or (e) were involved in any type of sprint training 6 months prior to the study. All subjects were provided a written informed consent in accordance with

the institutional review board at the University of North Carolina at Greensboro and Winston-Salem State University.

Procedures

Experimental Design

Subjects completed three separate laboratory visits prior to starting the training program. During the first visit, total and regional body composition were measured by a trained technician using a whole body dual energy X-ray absorptiometry scan (DXA) (Lunar-Prodigy Advance Plus). Then, each subject completed an exercise protocol familiarization test on an electronically-braked cycle ergometer (Lode Excalibur Sport, Lode BV, Groningen, The Netherlands). After 48 to 72 hrs, the subjects reported back to the laboratory between 7:00 a.m. and 9:00 a.m. after an overnight fast (8 to 12 hrs) to complete a baseline resting 2-hr blood profile.

Resting and Acute Sprint Exercise-Induced GH Profiles

Prior to each GH profile, the subjects were asked to refrain from exercising for the previous 24 hrs. An intravenous catheter was inserted into the forearm by a trained technician. Patency was maintained by displacing the blood in the catheter with isotonic saline. Blood samples were taken on average every 15 min [Q15] for 2 hrs with more frequent sampling near the time that exercise would occur during the exercise trials (0, 15, 30 [sprint], 31 [immediate post-exercise], 35, 45, 60, 75, 90, 105, and 120 min). Blood samples were collected in vacutainers (10 mL) and a total of 110 mL of blood was collected over the 2-hr time frame.

The subjects returned to the laboratory 1 wk later to complete their first 2-hr sprint protocol. The same protocol as the resting GH profile was followed, except the subjects rested for 20 min after catheter insertion prior to beginning a standardized warm-up on the cycle ergometer. The warm-up consisted of pedaling against 60 W of resistance for 4 min, 80 W for 30 sec, and 100 W for an additional 30 sec. Then, the subjects rested for 5 min while the 30-min pre-exercise blood draw was performed. Immediately following the draw, the subjects were instructed to begin pedaling at maximal pedal speed for 2 to 3 sec at which point a resistance load equivalent to 7.5% of each subject's body weight was applied for one maximal 30-sec sprint. Each subject was verbally encouraged to give his maximal effort during the maximal sprint. Immediately after the exercise test, a post-exercise blood sample was taken while the subject remained seated on the ergometer. Immediately afterwards, each subject moved into a chair to rest comfortably for the remaining blood draws.

Short-Term, High-Intensity Exercise Training (HIT)

The training protocol used in this study was based on similar high-intensity protocols published by Burgomaster et al. (2) and Gibala et al. (4). Training began 24 hrs after the completion of the acute exercise testing session. It consisted of 4 to 6 repetitions of 30-sec maximal sprints, 3 times·wk⁻¹ for 3 wks. One day of rest intervened each training session. The first 3 training sessions consisted of four 30-sec repetitions at 7.5% body mass with 4 min of active recovery at 50 W between each repetition. Training sessions 4 to 6 (wk 2) consisted of 5 repetitions, and sessions 7 to 9 (wk 3) consisted of six 30-sec maximal repetitions. During each repetition, each subject was encouraged verbally to provide maximal effort. At the end of each week, 48 hrs after the third training session for the week, subjects completed the acute sprint test protocol outlined previously (including blood draws). At least 48 hrs after the final blood profile, a post-training DXA scan was completed as outlined previously.

Blood Sampling and Analysis

Blood samples were allowed to clot at room temperature for 30 min. Samples were then centrifuged at 3000 rev·min⁻¹ for 15 min at 4°C. Serum was extracted and pipetted into microcentrifuge tubes and stored at -80°C until subsequently analyzed. Growth hormone concentration at all time points was

determined in duplicate using a human GH enzyme-linked immunosorbant assay (MP Biomedicals, Solon, OH). The minimum detectable dose of this assay was $0.5 \mu\text{g}\cdot\text{L}^{-1}$. The intra-assay variance was 2.2 to 2.9%. To eliminate inter-assay variance, all samples from a single subject were assayed within the same plate.

Statistical Analyses

Paired samples *t*-tests were used to evaluate changes in body composition pre-training versus post-training. A repeated measures ANOVA with Greenhouse-Geisser correction was used to assess whether there were differences in anaerobic performance during the acute sprints at the end of each week of training compared to pre-training. When the data were non-normally distributed, a Wilcoxon signed-rank test was used to adjust for skewedness. Friedman nonparametric tests for several related samples with Wilcoxon *post-hoc* follow ups and appropriate Bonferroni corrections were conducted to assess if there were differences among the mean ranks of the mean GH area under the curve (AUC), peak GH, time to peak GH and total GH concentrations during each sprint x time point in order to determine how the individual GH profiles fluctuated on a weekly basis as a result of HIT. Mean GH AUC was calculated using the trapezoidal integration method. The AUC was calculated as previously described by Stokes et al. (20). The level of statistical significance was set at $P \leq 0.05$. All statistical analyses were performed using PASW for Windows, version 22.0 (Chicago, Illinois, USA). All results are expressed as means \pm SEM, unless otherwise noted.

RESULTS

Body Composition

Body mass index and total percent body fat were unchanged despite an increase in total body mass pre-training versus post-training (Table 1). Results indicate that the increase in total body mass was a result of the increase in total lean body mass with no change in total body fat accumulation. Regional body composition analysis revealed that total leg mass was increased and that this change was the result of an increase in the total lean mass of the legs without a change in total fat content of the legs. Upper body mass content distribution remained unchanged pre-training versus post-training.

Table 1. Pre-Training vs. Post-Training Changes in Body Composition.

Variable	Pre-Training	Post-Training	P	Adjusted P \ddagger
BMI ($\text{kg}\cdot\text{m}^{-2}$)	26.1 \pm 2.6	26.2 \pm 2.6	0.402	
Total Body Mass (kg)	86.9 \pm 13.4	87.9 \pm 13.4	0.007 \dagger	
Arms Mass (kg)	10.6 \pm 1.7	10.4 \pm 1.7	0.071	
Legs Mass (kg)	29.9 \pm 4.7	30.7 \pm 5.0	0.000 \dagger	
Total Body Fat %	20.1 \pm 7.7	19.9 \pm 8.4	0.406	
Arms Body Fat %	12.7 \pm 5.7	12.6 \pm 6.2	0.586	0.542
Legs Body Fat %	19.9 \pm 7.4	19.5 \pm 7.7	0.089	
Total Lean Body Mass (kg)	65.8 \pm 8.3	66.7 \pm 8.4	0.020*	
Arms Lean Mass (kg)	8.8 \pm 1.6	8.6 \pm 1.5	0.134	
Legs Lean Mass (kg)	22.5 \pm 2.9	23.3 \pm 3.2	0.000 \dagger	
Total Body Fat (kg)	17.4 \pm 8.6	17.4 \pm 9.3	0.957	0.904
Arms Fat (kg)	1.3 \pm 0.7	1.3 \pm 0.7	0.112	
Legs Fat (kg)	5.7 \pm 3.0	5.9 \pm 3.0	0.176	0.212

* $P < 0.05$; $\dagger P < 0.01$. \ddagger When the data were non-normally distributed, P values were adjusted for skewedness (>1.0) using the nonparametric Wilcoxon sign-ranked test. Values are mean \pm SD.

Exercise Performance

The training protocol used in this study increased workload for three successive weeks by increasing the total number of sprints per training day by one per week. As expected, results indicated that the total work per training week increased each week ($P = 0.000$) (Figure 1d).

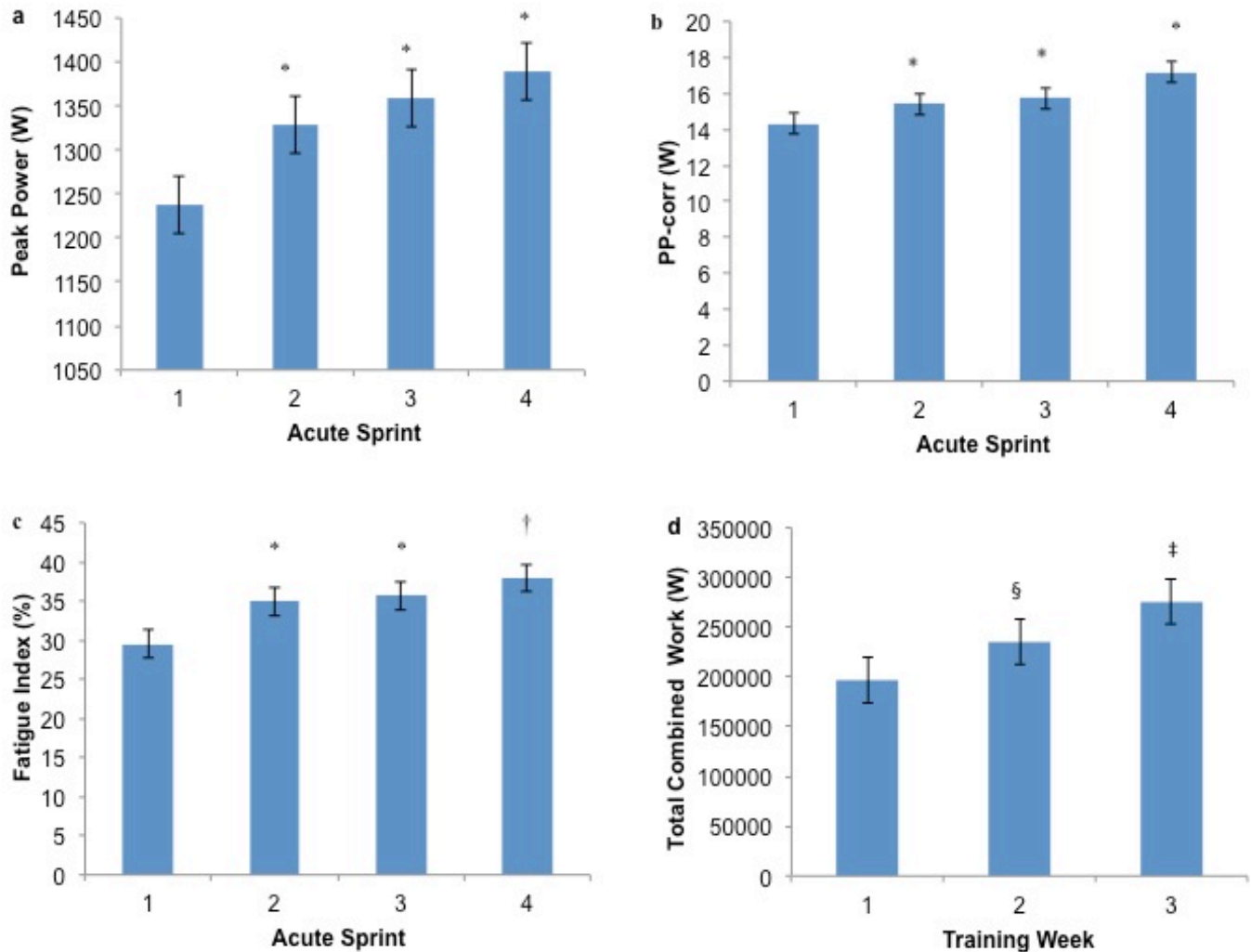


Figure 1. (a) Peak Power; (b) Peak Power-Corrected for Subjects' Body Mass; and (c) Fatigue Index during Each 30-sec Maximal Cycle Ergometer Acute Sprint (AS) Before and After 3 wks of HIT; and (d) Total Combined Workload of Every Sprint during Each Training Week. Values are mean \pm SD. *Greater than AS1 ($P < 0.01$). †Greater than AS1-AS3 ($P < 0.05$). ‡Greater than training weeks 1 and 2 ($P < 0.01$). §Greater than training week 1 ($P < 0.01$).

Peak power (PP) and peak power corrected to the subjects' body mass (PP-corr) increased after HIT ($P = 0.002$ and $P = 0.013$, respectively). *Post-hoc t-test* comparisons for paired samples revealed that PP and PP-corr increased after just the 1st wk of training ($P = 0.002$ and $P = 0.002$, respectively) with no additional improvements in PP or PP-corr following the additional 2 wks of training (Figure 1a/b). However, PP and PP-corr increased 12% and 20% respectively, over 3 wks of HIT. Mean power (MP) ($P = 0.280$) and mean power corrected to the subjects' body mass (MP-corr) ($P = 0.282$), as well as the time to reach peak power (TTPP) ($P = 0.741$), and minimum power throughout the 30-sec sprint tests (MinP) ($P = 0.359$) were all unchanged with training (Table 2). Fatigue index (FI) increased 28% after training ($P = 0.003$) and *post-hoc t-test* comparisons revealed that FI increased after each week

of training (Figure 1c). However, despite the increase in PP and PP-corr, the total work (TW) during the single acute 30-sec sprint at the end of each week was unchanged during HIT ($P = 0.280$) (Table 2).

Table 2. Anaerobic Performance.

Variable	Sprint 1	Sprint 2	Sprint 3	Sprint 4
MP (W)	657 ± 190	664 ± 170	649 ± 129	609 ± 141
MP-corr (W)	7.5 ± 1.4	7.7 ± 1.4	7.6 ± 1.2	6.9 ± 1.9
PP (W)	1237 ± 163	1329 ± 241*	1359 ± 302*	1389 ± 283*
PP-corr (W)	14.3 ± 1.2	15.4 ± 1.8*	15.7 ± 2.6*	16.2 ± 5.3*
MinP (W)	394 ± 203	350 ± 120	360 ± 112	323 ± 129
TTPP (sec)	1.4 ± 0.9	1.7 ± 1.2	1.7 ± 1.1	2.4 ± 3.6
FI (%)	30 ± 7	35 ± 10*	36 ± 12*	38 ± 13†
TW (W)	19704 ± 5695	19926 ± 5086	19462 ± 3863	18272 ± 4215

Mean power (MP), mean power corrected for each subject's body mass (MP-corr), peak power (PP), peak power corrected for each subject's body mass (PP-corr), minimum power (MinP), time to reach peak power (TTPP), fatigue index (FI) and total work per sprint (TW) during each acute sprint (AS) throughout the 3-wk training period. *Greater than AS1 ($P < 0.01$). †Greater than AS1-AS3 ($P < 0.05$). Values are mean ± SD.

Growth Hormone

Exercise elicited a GH response immediately following one acute 30-sec sprint on a cycle ergometer, regardless of HIT ($P = 0.000$) (Figure 2).

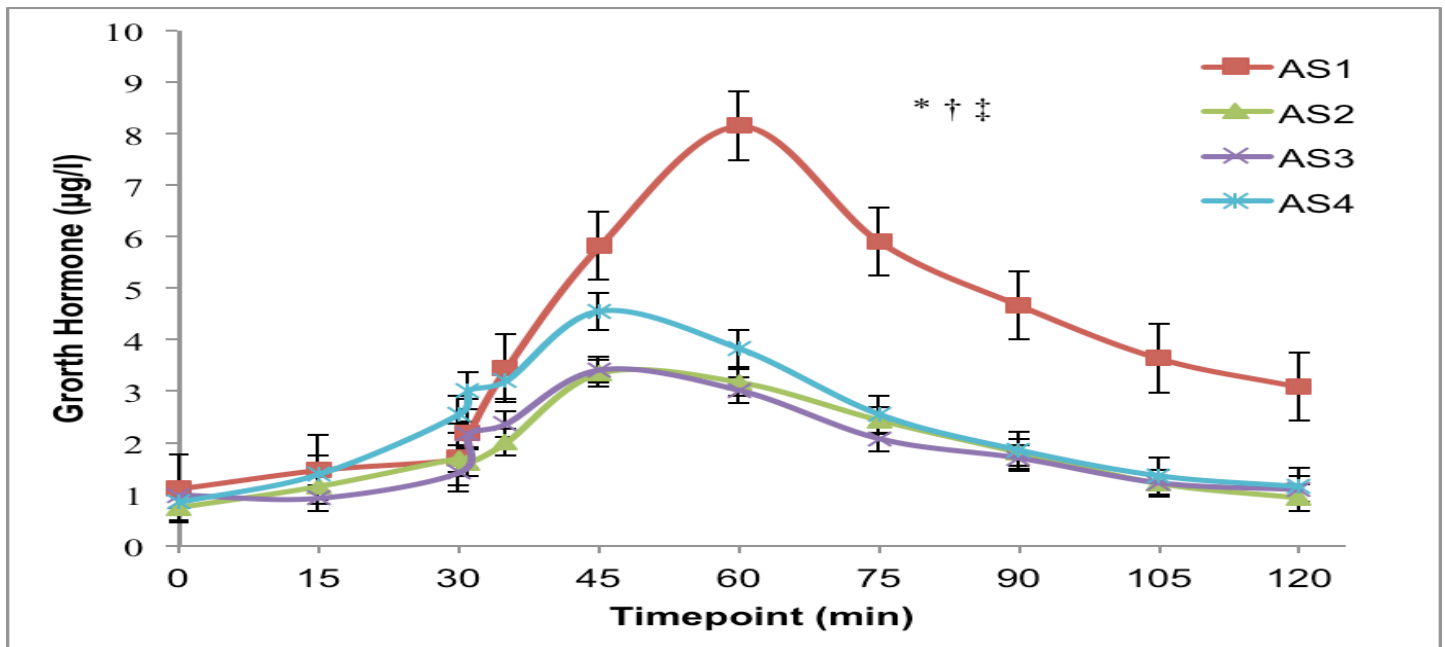


Figure 2. Exercise-Induced Growth Hormone ($\mu\text{g}\cdot\text{L}^{-1}$) during a 2-hr Profile that Included one 30-sec Sprint at Min-30. Profiles were collected before (AS1) and during three consecutive weeks of sprint training at the end of each week (AS2 – AS4). *Exercise produced a significant GH response regardless of training (main effect of exercise, $P < 0.01$). †GH AUC was significantly greater during AS1 compared to all other acute sprint GH profiles (main effect of group, $P < 0.01$). ‡GH concentration was significantly elevated at timepoints 45 to 120 during AS1 compared to AS2-4 (exercise-training interaction effect, $P < 0.01$).

However, the exercise-induced GH response was attenuated after only 1 wk of HIT ($P = 0.048$) (Table 3). More specifically, GH AUC was greater during sprint 1 (AS1) compared to the acute sprints (AS2 – AS4) after each week of HIT ($P = 0.002$ [AS2], $P = 0.002$ [AS3], and $P = 0.014$ [AS4]). Even though total workload was similar between all sprints before and after the end of each training week (Table 2), the exercise-induced GH response divided by total work output (KJ) was still attenuated with training overall ($P = 0.045$) and was greater during AS1 compared to AS2 – AS4 ($P = 0.001$ [AS2], $P = 0.003$ [AS3], and $P = 0.010$ [AS4]). Peak exercise-induced GH also decreased overall ($P = 0.036$) and was greater during AS1 compared to after the first and second weeks of HIT (AS2 – AS3) ($P = 0.002$ [AS2], $P = 0.003$ [AS3]) but similar to AS4 after the final week of HIT ($P = 0.064$). Regardless of the attenuated GH AUC, HIT had no effect on the time to reach peak exercise-induced GH release after each week of HIT ($P = 0.180$).

Table 3. Exercise-Induced Growth Hormone Before and After 3 wks of HIT.

Variable	Sprint 1	Sprint 2	Sprint 3	Sprint 4
GH AUC ($\mu\text{g}\cdot\text{L}^{-1}\cdot\text{min}^{-1}$) * ‡	505.2 \pm 112.2	232.4 \pm 66.3	224.2 \pm 67.4	286.7 \pm 112.8
GH AUC/KJ ($\mu\text{g}\cdot\text{L}^{-1}\cdot\text{min}^{-1}\cdot\text{W}$) * ‡	27.1 \pm 6.1	11.8 \pm 3.1	10.7 \pm 2.9	14.3 \pm 4.8
Peak GH ($\mu\text{g}\cdot\text{L}^{-1}$) * §	8.9 \pm 2.0	4.0 \pm 1.1	3.6 \pm 1.0	5.2 \pm 1.7
Time to Peak GH (min)	30.0 \pm 3.8	20.6 \pm 5.0	27.1 \pm 6.2	16.5 \pm 3.6

*Friedman nonparametric tests for several related samples indicated a difference across all four sprints ($P < 0.05$).

‡*Post hoc* Wilcoxon signed-rank tests indicated that the GH AUC sprint profile 1 was greater than GH AUC sprint profiles 2, 3, and 4 ($P < 0.01$). ‡*Post hoc* Wilcoxon signed-rank tests indicated that the GH AUC sprint profile 1 was greater than GH sprint profiles 2, 3 ($P < 0.01$), and 4 ($P < 0.05$). §*Post hoc* Wilcoxon signed-rank tests indicated that the GH sprint profile 1 was greater than GH sprint profiles 2 and 3 ($P < 0.01$), but not 4 ($P = 0.064$). Values are mean \pm SEM.

Growth hormone concentration was elevated 120-min post-exercise compared to rest following the AS1 ($P = 0.014$), 105-min after the 1st wk of HIT (AS2, $P = 0.020$), 90-min after the 2nd wk of HIT (AS3, $P = 0.036$), and 105-min after the 3rd wk of HIT (AS4, $P = 0.014$). However, HIT also altered the exercise-induced GH recovery profile. Fifteen minutes after the onset of exercise (timepoint 45) during AS1 prior to HIT, the GH concentration was elevated compared to all 3 subsequent acute sprints (AS2 – AS4) following 3 wks of HIT ($P = 0.002$) and remained elevated for the remainder of the 120-min profile.

DISCUSSION

The major findings of this study indicate that exercise-induced GH release in response to short-term, high-intensity exercise training is attenuated in as little as 1 wk of training. This attenuation in GH release occurred: (a) even after sequentially increasing workload each week of training; and (b) in concert with the increase in lean mass of the lower extremities and greater peak power anaerobic performance.

Our findings are in agreement with several other studies that have reported an attenuated GH response to exercise training (3,5,10,17,28). Because the literature seldom states the time lapse between the last training session and the testing sessions, it is often difficult to dissociate chronic from acute effects of exercise on the outcome variables. This is particularly true of GH, since repetitive bouts of exercise with short recovery periods and inadequate rest between training days have been shown to attenuate GH release (3,5,17,18,28). Based on prior observations (9,18) there is

an optimal recovery period (>120 min) between exercise sessions for maximal exercise-induced GH release. In the current study, a single 30-sec acute exercise sprint occurred at the end of each week 48 hrs after the previous training day. Therefore, we suggest that the attenuated GH response observed in the current study was the result of chronic exercise training and that any effect of acute exercise was negligible.

The attenuated GH response seen with high-intensity exercise training may be linked to increased tissue sensitivity to GH. Due to the chronic and repetitive presence of GH that continually feeds back to the pituitary and hypothalamus during training, the magnitude of the GH response may fluctuate in response to a given exercise stimulus (i.e., a greater exercise intensity to elicit a greater GH response). Another possible mechanism causing the attenuated GH response might be the enhanced negative feedback of IGF-1 on GH release (15). Additionally, increased affinity of GH binding proteins (GHBP) and GH receptor desensitization further displays the complexity of the hypothalamic-pituitary axis. The level of plasma GHBP is inversely related to the GH concentration and the percentage of GH bound to GHBP can vary from 10% to 80% over a 24-hr period (22).

In a chronic fluctuating hormonal environment, such as repetitive high-intensity training that coincides with troughs of GH release, it is possible that a shift in GH binding kinetics may be related to changes in GH production, clearance, and secretory patterns. For example, the mass of GH secreted during exercise after 6 weeks of training decreased 37% of distribution volume and the half-life of GH disappearance decreased 23% (28). Additionally, GH parameters such as maximal GH peak height, incremental GH peak amplitude, GH peak area, nadir GH concentration, and 24-hr integrated serum GH have been reported to increase in women who trained above the lactate threshold for 1 yr (25). This signifies the dose response relationship that training may have on GH secretion parameters. This is also important considering that pulsatile secretion determines more than 85% of the daily GH AUC release and that the pulsatile release of GH is more effective at producing a biological response than continuous release in certain tissues like bone, muscle, and liver where GH plays a metabolic role (22).

Studies (4,17) using short-term sprint or interval training at higher intensities have reported small improvements in anaerobic performance. Most high-intensity training studies (25,28) that reported an increase in exercise performance trained for at least 6 wks, which is similar to Stokes et al. (17) who reported a 6% increase in peak power after 6 wks of training. Interestingly, our subjects had a 12% increase in peak power after only 3 wks of high-intensity training. Improvements in anaerobic performance in a short period of time (typically <3 wks) are most likely the result of enhanced neuromuscular activity (6) that could also explain our increase in peak power. However, Stokes et al. (17) reported that post-exercise plasma ammonia concentrations, reflecting reduced muscle ammonia, decreased with 6 wks of sprint-training. They suggested that this may have improved the balance between ATP hydrolysis and resynthesis during training leading to the small improvements they recorded in mean power.

Additionally, Rodas and colleagues (14) reported significant increases in phosphocreatine (31%) and glycogen (32%) as well as other markers of muscle oxidative capacity from vastus lateralis tissue after only 2 wks of sprint-training. It is possible that subjects in our study had improvements in muscular enzymatic activity, such as improvements in adenylate kinase activity and reduced muscle ammonia, but the time course for these physiological adaptations may take longer than 3 wks to occur. Thus, this might be the reason that we did not see any changes in mean power in our shorter training time frame.

Similar high intensity resistance training programs that focused on lower extremity hypertrophy have reported change in as little as 4 wks (16). Lamont et al. (12) reported an increase in total lean body mass (2.1%) and lower leg lean mass (2.5%) after 6 wks of high-intensity resistance squat training. In the current study, 3 wks of HIT significantly increased overall total body mass 1.2% and total lean body mass (1.2%) with no change in overall fat mass. The increase in total body mass and lean mass coincided with a significant increase in total mass (1.4%) and total lean mass of the lower extremities (3.6%). Therefore, HIT resulted in a significant change in overall mass as a direct result of increased lean mass of the lower extremities that was likely related to the cycling training program.

It has been reported that growth hormone is needed for the acquisition of lean muscle mass and strength (8). However, our results demonstrate that lean mass can increase in the presence of an attenuated GH response during high-intensity training. This may be linked to previous observations suggesting a change in GH secretory dynamics and binding kinetics leading to altered bioavailability (25,28). Additionally, no studies have examined the effect of a high-intensity training program on the subsequent 24-hr GH concentrations or the fact that training can alter GH molecular heterogeneity leading to the secretion of GH molecular mass variants that have higher bioactivity (11). These exercise training-induced factors might all influence physiological adaptations that lead to changes in body composition.

Practical Applications

Exercise program design focusing on maximal effort over short time frames (2 to 3 min·d⁻¹ x 3 d·wk⁻¹) can lead to significant improvements in anaerobic performance and lean body mass after only 3 wks of short term-high intensity training. This is a suitable alternative to time consuming, low-intensity exercise for subjects who are capable of exercising at maximal capacity. Furthermore, the changes in peak power output and lower extremity lean body mass suggest that the training program is suitable training for individuals who are engaged in sporting events that require lower body power output.

CONCLUSIONS

The results of this study indicate that short-term, high-intensity sprint training results in decrements in the exercise-induced GH response after only 1 wk of HIT. This physiological adaption to training occurred in a much shorter time frame than the previous research had documented. Also, the findings suggest that training at higher absolute intensities and continually increasing workloads does not offset the initial decrement in exercise-induced GH release that is observed when HIT is initiated. Improvements in anaerobic performance and lean body mass can occur despite an attenuated GH response during short-term, high-intensity training. More studies need to identify the time course of high-intensity training on GH binding kinetics and secretory parameters. Perhaps, the attenuated GH response to training does not necessarily suggest that less GH is available for biological action in certain tissues, but that the metabolic action of GH is altered as a result of training-induced changes in GH bioavailability and not total GH release.

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