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The response of growth hormone to acute exercise has been shown to be altered by training. The present study examined the time course of GH adaptation to 3-weeks of sprint exercise training on a cycle ergometer. Eight, recreationally active, male subjects (age = 26.8 ± 4.3 yrs; height = 1.77 ± 0.05 m; weight = 77.79 ± 7.35 kg; lean body mass = 62.0 ± 7.1 kg) completed a 2-hour resting profile and a 2-hr acute sprint profile, followed by three weeks of training with additional acute sprint tests at the end of each week. Blood samples were taken every 15 minutes [Q15] during rest and more frequently [Q1-Q10] during exercise. Body composition was assessed by DXA [Prodigy Advanced, GE Lunar]. Training consisted of 4-6 repetitions of 30-second maximal sprints relative to body mass, three times per week. Integrated 2 hr serum GH area under the curve (AUC) was calculated using trapezoidal integration. Peak power (PP) increased 7.5% while time to peak power (TTP) decreased 58.0 % ($P < 0.05$) over the 3-week training period. The mean of 2 hr GH AUC per unit lean body mass (LBM), did not significantly decrease ($P = 0.82$) during the 3-week sprint training protocol, although a trend was observed for GH AUC to decrease from the first acute sprint test to the second acute sprint test (8.45 ± 3.0 ng/ml vs. 3.59 ± 1.21 ng/ml, respectively; $P = 0.90$). While this trend was similar to previously reported findings, we conclude that 3-weeks of sprint training does not significantly alter the GH AUC response to acute sprint exercise despite significant increases in power output during this same time frame.

THE GROWTH HORMONE ADAPTATION

TO SHORT TERM SPRINT

EXERCISE TRAINING

By

Jason K. Smith

A Thesis Submitted to
the Faculty of The Graduate School at
The University of North Carolina at Greensboro
in Partial Fulfillment
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Approved by

_____
Committee Chair

To my little brother,

May you always dare to dream.

APPROVAL PAGE

This thesis has been approved by the following committee of the Faculty of The Graduate School at the University of North Carolina at Greensboro.

Committee Chair _____

Committee Members _____

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Date of Acceptance by Committee

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Date of Final Oral Examination

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CHAPTER I

INTRODUCTION

Background

Human growth hormone (hGH) is a peptide hormone secreted from the anterior pituitary gland. GH is not one single hormone, it is in fact a family of over 100 different molecules all sharing similar characteristics (Wideman, Weltman et al. 2002). GH is secreted in a pulsatile fashion with the highest concentrations occurring after sleep and exercise, if the exercise is of adequate intensity and duration (Godfrey, 2003; Sutton, 1976). The exact mechanisms that elevate GH release in response to exercise are not known, however it is thought that it may be a result of; direct neural stimulation, feedback from circulating insulin-like growth factors (IGF), circulating catecholamines, blood lactate, nitric oxide (NO), and/or blood pH changes (mainly H^+ ion accumulation) (Godfrey, 2003; Gordon, 1994). GH release also acts as a glucose sparing agent by increasing free fatty acid concentration (FFA) in the blood (Quabbe, 1972). In addition to having an effect on substrate utilization, GH release has also been proposed to inhibit protein catabolism and lead to muscle hypertrophy and tissue remodeling in response to exercise (Godfrey, 2003; Kraemer, 2005).

Previous research (Stokes, Nevill et al. 2004) showed that after 6 weeks of supervised sprint training a decrease in peak GH of over 40% was seen (10.3 ± 3.3 vs. 5.8

± 2.5 μg/l; $P < 0.05$) while no changes were seen in a group of control subjects. We believe this may occur earlier than 6 weeks much like the muscle metabolic adaptations that have occurred in other sprint training studies (Burgomaster, Hughes et al. 2005; Gibala, Little et al. 2006). Burgomaster and colleagues (2005) found that 6 sessions of sprint training with 1-2 rest days between each session significantly increased peak power output during the 6^{th} training session ($P < 0.05$) compared to the first training session (Burgomaster, Hughes et al. 2005). In the same study sprint training showed an increased endurance capacity but no increases in VO_2peak.

Similar to most training studies, we expect sprint performance to improve over the three week training program. We also believe there will be an altered growth hormone response to sprint training. Therefore the purpose of this study is twofold; first to identify the characteristics (magnitude, duration etc), of the altered growth hormone release in response to sprint exercise training and secondly, to measure the time course of this adaptation to sprint exercise training.

Specific Aims

1. To determine the time course of a 3-week sprint training protocol on the acute growth hormone response to a single maximal 30-second sprint.
2. To the time course peak power output and VO_{2max} changes from baseline to the end of the 3-week sprint training protocol.

Hypotheses

1. There will be a significant attenuation in peak growth hormone concentration in response to an acute maximal 30-second sprint after sprint training.
    a. A significant attenuation of peak growth hormone concentration in response to acute sprint exercise will not be observed after 1 week of sprint training.
    b. A significant attenuation of peak growth hormone concentration in response to acute sprint exercise will not be observed after 2 weeks of sprint training.
    c. A significant attenuation of peak growth hormone concentration in response to acute sprint exercise will be observed after 3 weeks of sprint training.
2. There will be a significant attenuation in growth hormone area under the curve (AUC) in response to an acute maximal 30-second sprint after sprint training.
    a. A significant attenuation of growth hormone AUC in response to acute sprint exercise will not be observed after 1 week of sprint training.
    b. A significant attenuation of growth hormone AUC in response to acute sprint exercise will not be observed after 2 weeks of sprint training.
    c. A significant attenuation of growth hormone AUC in response to acute sprint exercise will be observed after 3 weeks of sprint training.
3. Three weeks of sprint training will significantly increase peak power output compared to baseline.

4. It is not expected that a significant increase in VO_2peak compared to baseline after 3-weeks of spring training will occur.

CHAPTER II

REVIEW OF THE LITERATURE

The purpose of this section is to review the previously reported literature on growth hormones and sprint exercise that used cycling as the modality. This section includes reviews on; growth hormone release during exercise, factors affecting GH release during exercise, the role of GH after exercise, acute sprint cycle exercise and sprint training.

Overall Health Implications

GH has been linked to many health related benefits, those of which are absent in those whom are GH deficient. Patients exhibiting GH deficiency in conditions such as hypothalamic or pituitary disease experience decreased lean muscle mass, increased body fat (especially abdominal obesity), reduced exercise capacity, insulin resistance, dyslipidemia, and impaired heart function (Wideman, Weltman et al. 2002). GH administration in deficient adults resulted in lean muscle mass increases, fat mass decreases, reduced abdominal obesity, along with an increase in exercise capacity. Age and body composition are also important determinants in GH production. GH production and release decreases approximately 14% per decade after the age of 40 (Wideman, Weltman et al. 2002). Many of the same conditions apparent in GH deficient individuals

are also present in the aging population. GH administration to older adults has been reported to result in the same changes that occur in the GH deficient population (Wideman, Weltman et al. 2002).

Growth Hormone and Exercise

Sutton and Lazarus (1976) attempted to validate the efficacy of exercise as a stimulus for the release of endogenous GH, by comparing exercise to pharmacological stimuli known to increase GH release. The exercise stimuli included 20 minutes of constant load cycling on a cycle ergometer at 300 (25-33%), 600 (40-66%), and 900 kpm/min (75-90% of VO_2max). Other stimuli used to induce GH release were arginine, L-DOPA, insulin hypoglycemia, and sleep. The highest GH elevations were obtained following the most intense exercise with GH concentrations reaching 35.1 ± 6.3 ng/ml after 900 kpm/min of exercise, similar to the GH concentrations attained w ith insulin induced hypoglycemia (36.0 ± 5.0 ng/ml). Low intensity exercise was not sufficient to produce pronounced elevations in GH leading investigators to theorize that a minimum intensity threshold may need to be reached to produce significant GH elevations in the blood (Viru 1985). Thus, using exercise as a stimulator for GH release gives practitioners a safer alternative to measure the pituitary reserve of GH in deficient patients than insulin induced hypoglycemia, assuming an appropriately quantified exercise intensity and duration can be prescribed (Sutton and Lazarus 1976).

Role of Exercise Intensity & Duration

Prior to work by Pritzlaff and colleagues (1999); debate still existed about the role intensity played in the release of GH during acute exercise, since previous research

suggested that a 'threshold of intensity' was likely required to significantly increase GH release (Sutton and Lazarus 1976; Felsing, Brasel et al. 1992; Pritzlaff, Wideman et al. 1999). Pritzlaff and colleagues examined the impact of exercise intensity on exercise induced GH release, and hypothesized that there would be an attenuation in GH release until lactate threshold (LT) was reached. Constant load exercise was performed for 30 minutes at 5 different exercise intensities [2 below, 2 above and one at LT]. Results indicated that GH release increased with increasing exercise intensity. However, an exercise induced GH response occurred prior to LT, in opposition to what the authors had postulated. Therefore, it appears that there is a direct linear relationship between exercise intensity and the growth hormone response (Pritzlaff, Wideman et al. 1999), and a threshold for GH release at the lactate threshold as postulated by Felsing (Felsing, Brasel et al. 1992) does not appear to be substantiated.

Table 1. GH response to submaximal endurance exercise

Study	Participants	Method	Effect on Acute GH release (µg/l)
Wideman et al. (2006)	Men (27 ± 4)	Cycling 70% VO_2peak 30, 60, or 120 min	Peak GH (13.3 ± 1.3, 22.4 ± 5.3, 24.3 ± 5.3)

Role of Growth Hormone after Exercise

Pritzlaff and colleagues (2000) examined the role GH and catecholamines play in substrate utilization during and after exercise. The researchers followed up on previous studies indicating a shift in substrate utilization to fat oxidation post-exercise (Wolfe, 1990; Bahr, 1991). Subjects exercised for 30 minutes at intensities of 25 and 75% of the difference between O_2 uptake at rest and at LT, LT, and 25 and 75% of the difference

between O_2 uptake at LT and VO_{2peak}. During recovery, fat expenditure increased with each increase in exercise intensity as did both peak GH and integrated GH concentration (IGHC). Regression analysis for each subject revealed a statistically significant relationship between recovery fat oxidation and both peak GH and IGHC. The results of their study infer that as the intensity of exercise increased so does the post-exercise fat oxidation, peak [GH], and IGHC. This suggests that GH may be partially responsible for utilizing more fat from adipose tissue after exercise and preventing post-exercise hypoglycemia (Pritzlaff, 2000).

Enevoldsen and colleagues (2007) examined the role GH plays in post-exercise fat oxidation by inhibiting GH release through octreotide infusion. Subjects came into the lab fasted and were studied for one hour at rest, one hour after beginning octreotide infusion, during one hour of treadmill running at 50% VO_2max, and for four hours post-exercise. Adipose tissue glycerol and FFA output increased during exercise in both control and octreotide groups and returned to baseline levels within 60 minutes of cessation of exercise. Arterial GH concentration increased with exercise in the control group only, reaching peak values about 30-45 minutes into exercise and decreasing steadily after cessation of exercise until baseline levels were re-established at 60 minutes post-exercise. An increase was seen in adipose tissue glycerol and FFA release beginning at 60 minutes post-exercise and this time delay for increased FFA oxidation was also noted in other studies (Gravholt, Schmitz et al. 1999; Hansen, Gravholt et al. 2002; Moller, Gjedsted et al. 2003; Djurhuus, Gravholt et al. 2004; Enevoldsen, Polak et

al. 2007). This article supports the idea that GH is involved in the enhanced fat-oxidation observed in the post-exercise recovery period (Enevoldsen, Polak et al. 2007).

Sprint Exercise

Stokes and colleagues (2002a) considered the role exercise duration plays in the GH response to a single bout of maximal sprint cycle exercise. They examined the duration of the GH response after 6 and 30 seconds of maximal cycling exercise using a

Table 2. Summary of GH response to acute sprint cycling

Study	Subjects	Exercise	Effect on Acute GH release
Stokes (2002a)	9 health males 23 (1) yrs	6s and 30s, 7.5% of body weight	30s sprint resulted in GH peak of 18.5 ± 3.1 $\mu g \cdot l^{-1}$ GH AUC of 1808 ± 90 $\mu g \cdot l^{-1}$
Stokes (2002b)	10 healthy males 24.5 (1.1) yrs	30s maximal sprint at either 7.5% (fast) or10% (slow) pedaling rates	Peak [GH] was higher in the fast trial vs. the slow trial (40.8 ± 8.2 vs. 20.8 ± 6.1 mU/l respectively; $P < 0.05$) GH AUC for fast vs. slow was also significant (1,697 ± 367 vs. 933 ± 306 $min \cdot mU^{-1} \cdot l^{-1}$; $P = 0.05$) respectively
Stokes (2003)	11 healthy males 24.6 (3.7) yrs	examined reproducibility of GH response to 30s sprint on cycle ergometer.	no significant difference in mean GH concentrtion or mean power output between trials. 725 ± 84 vs. 721 ± 84 W between the first and second trial respectively GH AUC 270.9 ± 296.7 vs 273.8 ± 285.5 $min \cdot \mu g \cdot l^{-1}$ respectively
Stokes (2005)	8 healthy males 23 (2) yrs	30s maximal sprint against 7.5% of body weight with either 60 or 240 min of rest	For first sprint Peak GH of 14.4 ± 9.6 µg/l 60 min GH AUC 460 ± 348 $min \cdot \mu g \cdot l^{-1}$

resistance equivalent to 7.5% of the subject's body mass. There were no differences between the power outputs of the participants in the first 6s of either the 6s or 30s trials. Serum GH concentrations were elevated after both trials, however the magnitude of the

elevation after the 30s sprint was much greater than the 6s sprint. Serum GH peaked at 40 min post exercise in both trials and remained elevated for 60 min after the 6s sprint and 90-120 min after the 30s sprint. The 30s sprint produced elevations in GH to 18.5 ± 3.1 μg/l at peak, an increase of 530% from baseline, whereas the 6s sprint produced a GH concentration of only 4.0 ± 1.5 μg/l at peak concentration or 217% above baseline concentration. This represented a 450% greater maximal GH concentration during the 30s vs. the 6s trial. The results from this study suggest that duration of a maximal sprint exercise bout or perhaps, more specifically, the amount of muscle mass used and work produced has an effect on both the amplitude and duration of the GH response (Stokes, 2002a).

Stokes and colleagues (2002b) also examined the GH response to maximal sprint cycling at different pedaling rates. The authors were interested in seeing if the GH response was different for varied pedaling rates and if repeated bouts of sprint exercise attenuated the GH response. The authors cited studies that showed simliar metabolic responses following maximal cycling at fast and slow pedaling rates [140 and 60 rpm respectively](Jones, McCartney et al. 1985). Therefore Stokes and colleagues believed that if metabolic responses determined the exercise induced GH response, the two conditions would yield similar results. Subjects in this study completed two 30s maximal sprints separated by 60 min of recovery. Resistance for the subjects was set at either 7.5% (fast) or 10% (slow) of their body weight to elicit different pedaling rates. Between the two trials there was no difference in peak or mean power output, but peak power output was slightly lower in the second sprint regardless of resistance (fast or slow).

Peak GH response after the initial fast sprint was more than twice as great as that after the initial slow sprint (37.7 ± 6.0 vs. 17.6 ± 3.7 mU/l respectively; $P < 0.05$) and mean GH area under the curve (AUC) during 60 minutes of recovery from sprint 1 was also elevated in the fast vs. slow trial (1,697 ± 367 vs. 933 ± 306 $min \cdot mU^{-1} \cdot l^{-1}$ respectively; $P = 0.06$). There was no increase in serum GH concentration after the second sprint in either trial although the rate of GH clearance did slow after the second sprint. The exact mechanism for the increase in serum GH concentration due to faster pedaling rates is not known, but was suggested to be related to muscle mass recruited (Stokes, Nevill et al. 2002). Additionally the increased GH concentration could be the result of increased frequency of neural firing during the fast trial as there was significant correlation between mean pedal revolutions and GH AUC ($r = 0.59$, $P < 0.01$ for sprint 1) as well as peak pedal revolutions ($r = 0.48$, $P < 0.05$ in sprint 1). These findings contradict those of Kanaley *et. al.* (1997), that reported subesequent 30 min aerobic exercise bouts following a similar 60 minute passive recovery period, to produce distinct GH pulses. However, the aerobic exercise study reported much lower GH AUC after exercise than that observed in the sprinting study (Kanaley, Weltman et al. 1997; Stokes, Nevill et al. 2002).

Stokes and colleagues (2005) further investigated the attenuation of the GH response after a second bout of sprint exercise. Subjects completed a 30s sprint on a cycle ergometer followed by either 60 or 240 minutes of passive recovery before attempting another 30s maximal sprint. Results indicated that the GH response did not increase after the second sprint after the 60 minute recovery trial. During the 240 minute

trial, GH concentration was back to baseline by 120 minutes and the second sprint produced a second increase in serum GH concentration that was smaller in magnitude than the initial response. The authors infered that the amount of recovery time influenced the amount of GH released, but similar GH outputs were seen the next day. This suggests that a short term negative feedback loop exists in the hypothalamic-pituitary axis and plays a role in the control of GH release. It appears that in some circumstances, the negative feedback loop can override the exercise stimulus for GH release. (Stokes, Nevill et al. 2005). In contrast, several studies have reported the discrete ability of exercise to breakthrough this negative feedback loop (Kanaley et al 1997, Veldhuis et al 2004), but these studies used constant load aerobic exercise as the stimulus.

Stokes and colleagues (2003), examined the reproducibility of the GH response to a 30s maximal sprint on a cycle ergometer. Subjects performed two trials of a single 30s cycle ergometer sprint separated by 7 days. The authors found no significant difference in mean power output between the two sprint trials (725 ± 84 vs. 721 ± 84 W, P = 0.81), even though some subjects had an increase in peak GH output during the second sprint there was no significant difference in mean GH concentration between trials. There was a large interindividual variability in the GH output between subjects but this is decidedly normal (Gordon et al 1994, Wideman et al 2002). However, the authors in this study were more interested in the intra-individual variability in GH output with respect to sprint exercise. Area under the curve (AUC) was not found to be statistically different between trials and the test-retest correlation was found to be statistically significant (r = .97). This study supports previous work that there is a large interindividual variability in the GH

response to acute exercise (Raynaud, Capderou et al. 1983; Wideman, Weltman et al. 1999; Stokes, Nevill et al. 2002), but was the first to show it with sprint exercise. This study shows that despite the interindividual variation in the exercise-induced GH response, the intraindividual GH response to sprint exercise on a cycle ergometer was highly reproducible (Stokes, Nevill et al. 2003).

Gordon and colleagues (1994) examined the effect of acid-base balance on the serum concentration of GH in the blood after acute high-intensity cycling exercise. Subjects were either given a placebo or a solution of $NaHCO_3$ and were asked to perform a maximal sprint exercise bout of 90 seconds at 0.49 N (0.05kg)/kg body weight. As a result of $NaHCO_3$ solution ingestion, blood pH was lower in the placebo group at all points except baseline and serum GH concentrations were higher in the placebo group at 15, 20, and 30 minutes post-exercise. The authors believed that although it is not the only factor, $[H^+]$ may have an effect on the release of GH after acute exercise (Gordon, Kraemer et al. 1994).

Thus, acute sprint exercise has been shown by multiple studies to be a valid and predictable stimulus for growth hormone release in men. It seems to have a larger amplitude than submaximal aerobic exercise, which most likely results in the attenuation of GH release when multiple bouts of sprint exercise are employed. However, few studies have examined the long term effects of sprint training on the GH response to sprint exercise.

Sprint Training

Stokes and colleagues (2004) were the first to examine the acute GH response to sprint cycling after sprint training. Subjects completed two 30s maximal sprints on a cycle ergometer against a load equivalent to 7.5% of their body mass separated by 60 minutes of rest. The training involved three supervised training sessions per week that were either speed or speed-endurance training. During speed sessions the participants sprinted against a resistance of 4% body mass to develop speed while during speed-endurance sessions subjects pedaled against a resistance of 11% body mass in an attempt to develop lean muscle mass and strength. After 6 weeks of training there was a decrease in the peak GH response to sprint exercise (10.3 ± 3.1 vs 5.8 ± 2.5 μg/l, $P < 0.05$). This study reported that a 6 week sprint training program can decrease the GH response to sprint exercise with between 3 and 5 days rest between training a testing sessions (Stokes, Nevill et al. 2004).

Burgomaster and colleagues (2005) examined the effects of sprint training on muscle oxidative potential, VO_{2peak}, and time to fatigue while cycling at an intensity equivalent to 80% of VO_{2peak}. The authors utilized a two week training protocol with 1-2 days of rest in between sprint training sessions. Training protocols consisted of 4-6 maximal 30s sprints with a resistance of 7.5% body weight. After training, cycle endurance capacity increased significantly as well as anaerobic work capacity and muscle oxidative capacity. This study demonstrates that sprint cycle training was effective in improving sprint as well as endurance performance (Burgomaster, Hughes et al. 2005).

Table 3. Summary of response to sprint cycle training

Study	Subjects	Exercise	Chronic Training Effects
Stokes (2004)	8 Males Recreationally active age = 24 (1.1)	Examined acute GH response to sprint cycling before and after 6 weeks of training Two 30s sprints separated by 60 min rest 30s training sessions pedaling against either 4% or 11% body mass	There was a decrease in peak GH response to sprint exercise (10.3 ± 3.1 vs 5.8 ± 2.5 µg/l)* AUC (µg/l) 567 (158) vs 256 (121)* PP (W) 1395 (83) vs 1470 (73) PP-corr (W) 18.0 (0.8) vs 19.2 (0.6) MP (W) 656 (40) vs 692 (29) MP-corr (W) 8.5 (0.3) vs 9.0 (0.1)
Burgomaster (2008)	10 males and 10 females untrained <2x/wk (5 of each per group)	30s at 7.5% body weight 4-6x 3 day/wk for 2 weeks.	Post-training cycle endurance capacity as well as anaerobic capacity increased significantly

Stokes et al. (2004) have shown a clearly defined response of GH to six weeks of sprint training, but it is the only study investigating the effects of sprint training on GH release and no midpoint training measures were taken so the time course of changes is not known. Studies have also shown that sprint training can influence both endurance capacity and power output through skeletal muscle adaptation (Burgomaster, Hughes et al. 2005; Gibala, Little et al. 2006). However, there is still no information as to the time course of the training adaptation within these studies. Thus, the proposed study will investigate the GH time course adaptation within the first 3-weeks of a sprint training protocol.

CHAPTER III

METHODS

Subjects

Eight recreationally active males (age = 26.8 ± 4.3 years) completed this study and baseline characteristics of the subjects are presented in Table 4. Subjects participated in less than 10 hours of recreational (swimming, basketball, jogging, cycling etc.) activity per week and were not involved in any sprint (interval) training. Subjects had a BMI between 18 and 30 and were non-smokers [had not smoked within the previous 6 months]. Subjects gave written informed consent to participate in this study as approved by the Institutional Review Board committee of the University of North Carolina at Greensboro. For safety reasons subjects with two or more risk factors for cardiovascular disease as defined by the American College of Sports Medicine were excluded from participation in this study.

Table 4. Baseline subject characteristics

Subj. Characteristic	mean (SD)
Age	26.8 (4.3)
Height	177 (5.0) cm
Weight	77.8 (7.35)
VO_2max	47.03 (7.26)
% body fat	17.72 (4.76)
BMI	24.7 (1.74)

Baseline and Post-Study Measurements

Individuals who met the inclusion criteria attended a preliminary screening & data collection session. Subjects will gave written informed consent (See appendix) to

participate in this study as approved by the Institutional Review Board at the University of North Carolina at Greensboro. Height was recorded to the nearest centimeter using a stadiometer and weight was recorded to the nearest 0.1 kg using an electronically calibrated scale (Seca, Vogel and Halke; Hamburg, Germany).

Subjects filled out a detailed medical history form and a physical activity history form (see appendix). Subjects with two or more risk factors for cardiovascular disease as defined by the American College of Sports Medicine were excluded from participation in this study. Subjects completed a brief familiarization session on the cycle ergometer (Lode Excalibur Sport. Lode BV, Groningen, The Netherlands), so they can experience cycling on electronically braked equipment and get bike set up position. This entire preliminary session took no more than 30 minutes.

Total and regional body composition were assessed using a whole body dual energy X-ray absorptiometry scan (DXA) [Lunar –Prodigy Advance Plus] at either the preliminary data collection session or at the baseline measurement session dependent on the availability of the DXA schedule. Subjects were asked to wear loose fitting clothing and to remove glasses and all jewelry. Subjects were asked to lay still and flat on the x-ray table, while scans were performed in fan beam mode (scan time ~10-20 min). Bone area (BA), bone mineral density (BMD), bone mineral content (BMC), fat mass (FM) and fat-free mass (FFM) were measured for the whole body and regional areas. All scans and analysis were completed by a trained DXA technician.

Subjects completed a standardized VO_{2peak} protocol on the electronically braked cycle ergometer. Briefly, subjects were asked to do a warm-up on the bike for 2 minutes

at 25-50 Watts. VO_{2peak} protocol consists of beginning the initial three 2-minute stages at 50, 100, and 150 W respectively. After this, the work was increased 50 W every minute until volitional fatigue. VO_{2peak} corresponded to the highest value achieved during any 30s collection period. Subjects then completed an active recovery at 50 Watts for 2 minutes. Oxygen consumption and carbon dioxide production were measured using open circuit spirometry (TrueOne 2400 Metabolic Measurement System. ParvoMedics, Inc. Sandy, Utah) calibrated to known gases and a Polar heart rate monitor was used to assess heart rate during the entire protocol. The entire testing process took 20-30 minutes depending on the subject's fitness level.

Baseline Resting Blood Sampling

Prior to initiating the training protocol, subjects entered the exercise physiology lab after an overnight fast (8-12 hrs) to complete a baseline 2 hour blood profile. A catheter was inserted into an arm vein by a trained technician and blood was taken every 15 minutes for 2 hrs, with more frequent sampling near the time that exercise would occur during the exercise-stimulated trials. Patency was maintained by displacing the blood in the catheter with isotonic saline. Blood samples were taken with the subject in a seated position at 0, 15, 30, 31 (time of exercise), 35, 45, 60, 75, 90, 105, and 120, minutes. Blood samples were collected in red-top vacutainers (10 ml) and a total of 110 ml or 110 cc of blood was collected over the 2 hr time frame. Blood samples were analyzed by GH ELIZA 07BC1033 (MP Biomedicals, Solon, Ohio). After completion of the resting hormonal profile, participants were scheduled for their baseline exercise profiles.

Sprint Test Protocol

Subjects were asked to return to the exercise physiology lab at least 48 hrs after the baseline measurements have been completed (but no longer than 2 weeks). A catheter was inserted into an arm vein by a trained technician and blood was taken as outlined in the previous section. After 20 minutes, subjects began a standardized submaximal warm-up. Warm-up consisted of pedaling at 60 watts (W) for 4 min, pedaling at 80 W for 30s, and pedaling at 100 W for another 30s. Subjects were then told to rest for 5 minutes before the 30s maximal sprint. During the sprint test subjects completed one maximal 30s sprint against a load equivalent to 7.5% of their body mass. Subjects were instructed to begin pedaling at maximal pedal speed for 2-3 seconds at which point the resistance was applied. Heart rate was assessed throughout exercise using a Polar heart rate monitor and heart rate was recorded every 5 seconds during exercise. Immediately after the test, a post-exercise blood sample was taken while the subject remained seated on the ergometer. After the immediate post-exercise sample was taken, the subject moved into a chair to rest comfortably for the remaining blood draws. Subjects were instructed to give maximal effort during each of the test trials and verbal encouragement was provided by the research team. Total work completed, peak and average power output were calculated from the Wingate test.

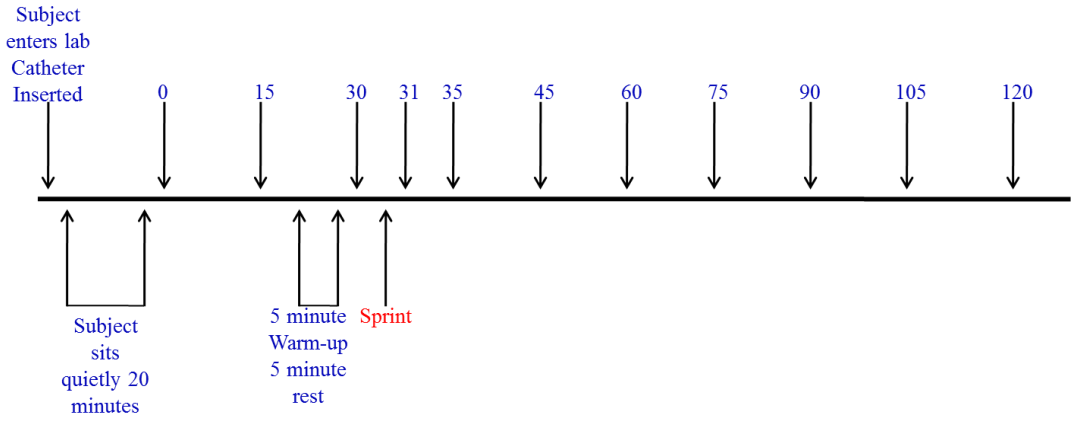

Figure 1. Timeline of acute sprint

Training

At least 36 hours after completion of all baseline testing sessions, subjects began the 3-week training protocol. Training protocol is based upon the 2-week training protocol developed by Burgomaster and colleagues (2005). Training consisted of 3 sessions per week with one rest day in between sessions. The first 3 training sessions consisted of four 30s repetitions at 7.5% body mass with 4 minutes of active recovery at 50 W between each repetition. Training sessions 4-6 [week 2] consisted of 5 repetitions, and sessions 7-9 [week 3] consisted of six 30s maximal repetitions. During each repetition subjects were encouraged verbally to provide maximal effort. At the end of each week, 2 days after the third training session for the week, subjects completed the sprint test protocol outlined previously (including blood draws). At least 24 hrs after the final blood profile, a post-training VO_{2max} test was completed as outlined previously.

Table 5. Timeline of subject visits to laboratory

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
		DXA VO_2max		2 hr resting profile		
Test Sprint #1	4 x 30s		4 x 30s		4 x 30s	
Test Sprint #2	5 x 30s		5 x 30s		5 x 30s	
Test Sprint #3	6 x 30s		6 x 30s		6 x 30s	
Test Sprint #4		DXA VO_2max				

Statistical Analysis

Descriptive data (mean ± SEM) was calculated for subject characteristics such as HR, power, height, weight, and age.

Total area under the curve (AUC) for GH was calculated using trapezoidal integration. Mean GH concentration, peak GH concentration attained and AUC were assessed to determine the serum GH response to sprint exercise. Training was expected to result in increased work during each of the weekly sprint trials. Therefore, total GH area under the curve was normalized for work completed during the sprint trial (GH response = AUC/W). The effect of training on all dependent variables was assessed using a one-way ANOVA with repeated measures. Independence of observations and normality were tested and when these assumptions were met, sphericity was assumed and no correction factor was used. When assumptions were violated, corrections were utilized as indicated in the Results.

GH Assay

This protocol utilized an HGH ELISA from MP Biomedicals (Solon, OH). Specimens were frozen at -80 C until being prepared for assay. Serum was then thawed for 1 hr prior to assay procedure. Due to use of manual pipetting only 32 wells were utilized for each assay run to keep pipetting time to under 3 minutes as recommended by assay insert. All samples were run in duplicate. Absorbance was run at 450 nm in a microtiter plate reader. Specimens were compared to dublicate values and reference standards to determine sample concentration. Assay was sensitive to a detectible concentration of 0.5 ng/mL.

CHAPTER IV

RESULTS

Maximal Oxygen Consumption

VO_2max testing was completed pre and post training to determine the effects of sprint training on maximal aerobic capacity. Neither absolute nor relative VO_2max was increased as a result of 3 weeks of sprint training (3.68 ± 0.77 L/min vs. 3.73 ± 0.82 L/min, $P = 0.53$, and 47.03 ± 7.26 ml/kg/min vs. 47.46 ± 7.98 ml/kg/min, $P = 0.66$, respectively). Maximum heart rate (184.63 ± 8.11 vs. 184 ± 6.52, $P = 0.72$) and RPE (17.88 ± 1.64 vs. 18.25 ± 1.04, $P = 0.5$), did not differ significantly from pre to post training. However, the maximum workload attained during the VO_2max test did increase significantly from pre to post-training (318.75 ± 59.39 vs. 343.75 ± 62.32, P = 0.033).

Body Composition

During the 3 week sprint training period, fat mass decreased (17.73 ± 4.76 vs. 16.81 ± 4.93, $P = .036$), while LBM increased (62.05 ± 7.13 vs. 62.90 ± 6.85, $P = 0.049$), with no significant changes in total body mass; (78.79 ± 7.35 vs. 78.13 ± 7.42, $P = 0.402$). Thigh girth was also unchanged in response to training (54.34 ± 3.41 vs. 54.19 ± 3.21, $P = 0.86$).

Training Related Changes

Within subject differences in mean total work output during training was assessed with a repeated measures ANOVA (Table 6), and the results indicate that there were no

significant difference between the mean total work output for each week [F (2, 14) = 1.278, p = 0.309]. However, a linear trend for decreased mean work completed across the 3 weeks of training did approach significance (p = .07)

Table 6. Total and mean of total work (KJ) by week. The total work for each individual sprint during the training week was averaged to attain the weekly mean.

	Mean	Std. Deviation	Weekly Total Work	Std. Deviation
Week 1	15.10	3.015	179.63	34.135
Week 2	15.01	3.089	225.23	46.330
Week 3	14.84	2.747	267.43	48.794

Mean peak power during each week of training was also assessed (Table 7). In this case, the assumption of sphericity was violated and a Huynh-Feldt correction was used. There were no differences in the mean peak power output across the 3 weeks of training [F (1.8, 12.8) = 2.498, p = .12].

Table 7. Mean and standard deviation for peak power during each week of training

	Mean	Std. Deviation
Week 1	1021.48	177.31
Week 2	1047.87	177.68
Week 3	1053.99	186.73

Likewise, the weekly average of the mean power output for each training week was not different across the three weeks of training [F (2, 14) = .836, p = .45] (Table 8).

Table 8. Weekly average of the mean power output for each training bout for the 3-weeks of training.

	Mean	Std. Deviation
Week 1	501.58	98.13
Week 2	500.44	102.97
Week 3	494.89	90.23

Analyzing only the data from the sprint with the highest peak power from each training session to remove the effect of added sprints yielded similar results. The mean peak and mean power did not change across the three weeks of training [$F(1.2, 8.3) = 2.355$, $p = .16$ and $F(2, 14) = 0.984$, $p = .398$, respectively] (Table 9).

Table 9. Mean and peak power average for training week using only the sprint with highest peak power for each training day. Means are presented as Means (SD). n = 8.

	Mean Power	Peak Power
Week 1	585.00 (104.80)	1166.63 (172.57)
Week 2	605.75 (113.90)	1166.96 (192.60)
Week 3	578.54 (117.31)	1199.46 (174.41)

Acute Sprint Trials

Differences in the mean power attained during each acute sprint trial were assessed by repeated measures ANOVA. The assumption of sphericity was violated, thus, a Huynh-Feldt correction was used. Mean power was unchanged across the acute sprint trials [$F (2.7, 19.1) = .669$, $p = 0.568$] (Table 10).

Table 10. Mean power and standard deviation during each acute sprint (N=8).

	Mean	Std. Deviation
Mean Power AS1	603.13	119.50
Mean Power AS2	602.13	114.66
Mean Power AS3	608.38	105.60
Mean Power AS4	586.13	109.19

Significant differences in peak power across each acute sprint trial were observed [F (2.6, 18.3) = 4.385, p < .05] (Table 11). A Huynh-Feldt correction factor was used, since the assumption of sphericity was violated. Post-hoc comparisons revealed that peak power increased significantly as a result of training in acute sprint trial 1 compared to acute sprint trial 4 (1160.63 ± 135.39 vs. 1247.25 ± 140.04, $P < .05$). Given the significant correlation of lean body mass on peak power (Pre; r= .826, p = .011, Post; r = .876, p = .004), the results were adjusted to correct for LBM. A paired samples t-test was performed and showed that a significant difference was observed even when peak power was corrected for lean body mass ($p < 0.05$, Table 11).

Table 11. Average peak power and peak power corrected for by lean body mass and standard deviations across all acute sprint trials. N=8

	Mean(SD)	Mean-corr (SD) in W/kg LBM
AS1	1160.63 (135.39)	8.51 (0.55)
AS2	1212.88 (149.99)	
AS3	1180.88(167.70)	
AS4	1247.25(140.04)	9.03 (0.49)

When analyzed by repeated measures ANOVA time to peak power (TTP) was not significantly different across the four acute sprint trials, although the P value trended

toward a significantly shorter TTP after training[F (1.2, 8.4) = 3.42, p = 0.096] (Table 12).

Table 12. Mean TTP and standard deviation across all acute sprint trials (s).

	Mean	Std. Deviation
TTP AS1	1.725	1.377
TTP AS2	0.825	0.328
TTP AS3	0.775	0.311
TTP AS4	0.725	0.385

Analysis of the total work completed (KJ) during each acute sprint trial, revealed that there were no significant differences [F(2.7, 13.8) = .67, p = .568] (Table 13).

Table 13. Total work in KJ and standard deviation of each acute sprint trial (N=8)

	Mean	Std. Deviation
AS1	18.09	3.58
AS2	18.06	3.44
AS3	18.25	3.17
AS4	17.58	3.28

When analyzed by repeated measures ANOVA maximum heart rate across all acute sprint trials tended to decrease, but the changes were not significant [Wilks Lambda = 0.552, F = 1.355, p = 0.357].

The average GH profile for the resting baseline trial and for each acute sprint trial is depicted in Figure 1. The GH AUC for each of the four acute sprint tests and the resting baseline was assessed using repeated measures ANOVA. The assumption of sphericity was violated in all analyses related to GH AUC and a Greenhouse-Geisser correction was used for all these analyses. Despite that the GH AUC for AS1 was greater

than the other 3 trials, the results indicate that the GH AUC did not differ significantly for any of the trials [F (1.5, 10.8) = 3.452, p = 0.078, eta^2 = .55] (Table 14).

Table 14. Mean and standard error of the GH AUC and correction factors for the five 2 hour profiles. All values are mean (SEM)

	GH AUC	GH AUC per LBM	GH AUC per KJ	GH Peak	Time to GH Peak
Rest	166.02 (61.39)	2.57 (.90)		3.36 (1.51)	42.63 (12.12)
AS 1	530.13 (185.77)	8.45 (3.00)	28.93 (10.39)	8.42 (2.95)	54.38 (7.99)
AS 2	226.15 (81.85)	3.59 (1.21)	12.65 (4.14)	4.25 (1.68)	41.88 (6.68)
AS 3	213.86 (110.47)	3.28 (1.59)	11.12 (5.26)	2.94 (1.25)	38.13 (9.35)
AS 4	205.83 (97.26)	3.18 (1.41)	10.46 (4.95)	4.12 (1.78)	48.88 (5.43)

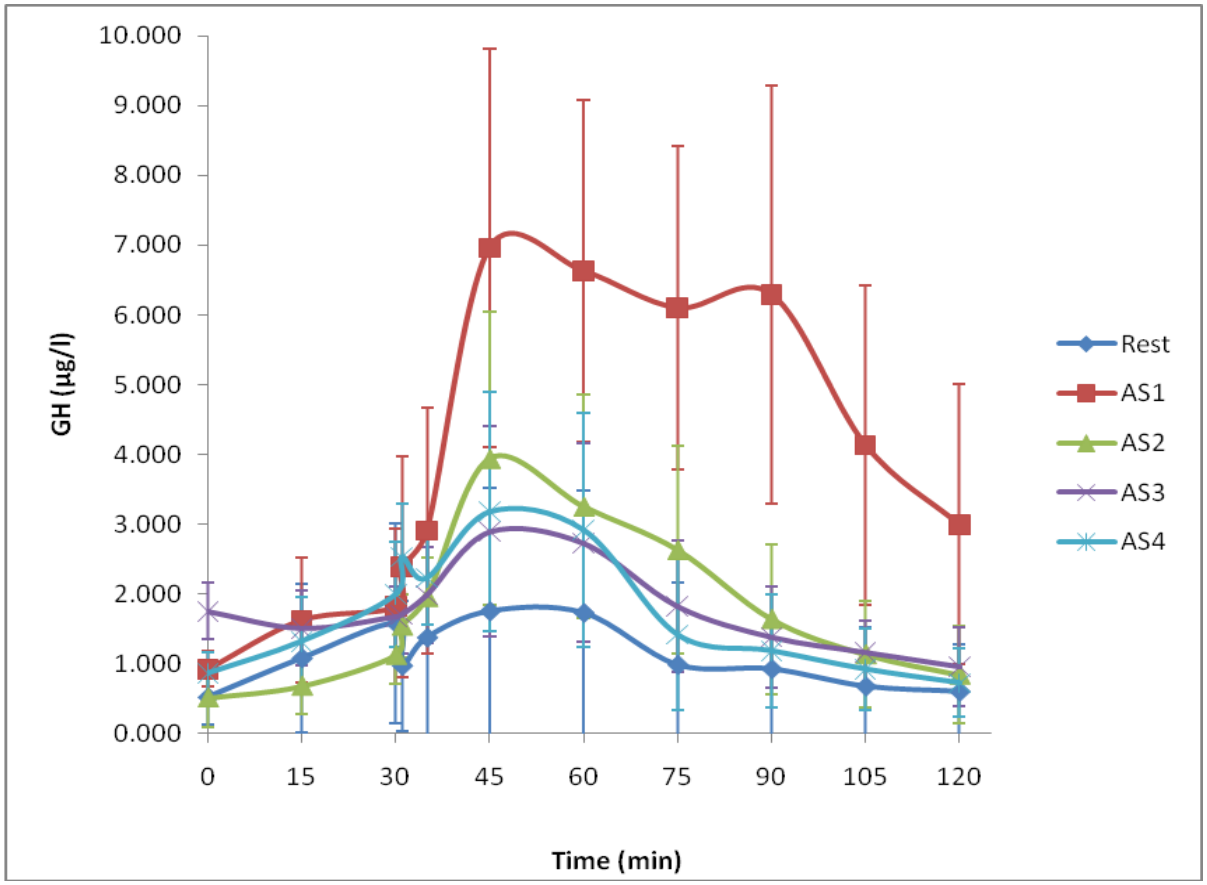

Figure 2. Mean (±SEM) profile of GH output in response to each of the 4 acute sprint trials.

Because GH AUC is influenced by LBM and total work completed we normalized GH release based on these factors. Normalizing GH AUC for LBM [F (1.461, 10.226) = 3.453, p = 0.082, eta^2 = .33] did not alter the initial findings related to GH AUC. Normalizing GH AUC for total work completed (KJ) approached, but did not reach, significance [F (1.12, 7.84) = 4.194, p = 0.073, eta^2 = .38].

Peak GH showed a trend toward a decrease across the four acute sprint trials. Sphericity was violated and a Greenhouse-Geisser correction was used (F = 4.439, p =

0.059, eta^2 = 0.39). Time to peak (GH TTP) did not differ significantly across sprint trials (F = 2.513, p = .173)

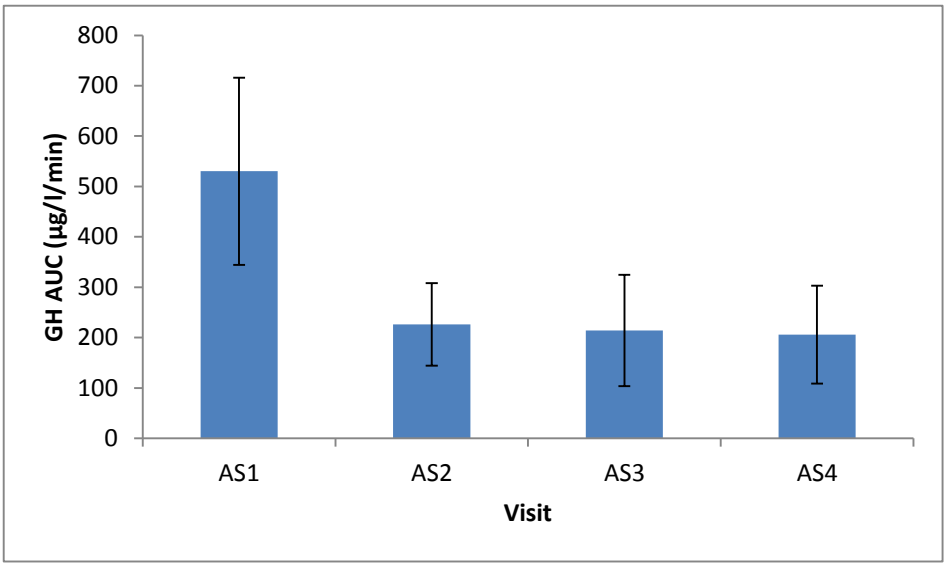

Figure 3. Mean 2 hour GH AUC for each of the five acute sprint profiles.

The change between peak GH and pre-exercise levels significantly differed across the four acute sprint trials (F = 5.115, p = .040, eta^2 = .422).

CHAPTER V

DISCUSSION

The present study showed that three weeks of sprint exercise training can; 1) increase peak power output during a 30 second wingate test, 2) decrease percent body fat and 3) increase lean body mass.

Prior to any sprint training, GH AUC in response to an acute sprint has been reported to be 270.9 ± 296.7 (Stokes et al. , 2003) and 567 ± 158 min·µg·l^{-1} (Stokes et al, 2004), compared to 530.13 ± 525.45 prior to training in our study. It should be noted that the recovery period after the 30-second sprint was 60 minutes in the previous two studies, while it was 90 minutes in our study. Weltman et al. (1977), showed that GH adaptation to acute constant load cycling had already occurred after 3 weeks of training and was similar when re-assessed after 6-weeks of training (pre- 238 ± 145 min·µg·l^{-1}; wk 3- 138 ± 106 min·µg·l^{-1}; wk 6- 130 ± 145 min·µg·l^{-1}). However, the intensity of the training sessions and acute sprint tests remained at the same absolute level and thus, did not represent the same relative intensity as prior to training. It is important to note that the current sprint training study required maximal effort for each acute trial and therefore represented a consistent relative intensity of 100%. In addition, the work load was adjusted for even minor changes in body weight to minimize changes in relative intensity. Thus the intent of our study was specifically to address the time course of adaptation in

GH output in response to sprint training in the first 3 weeks of the training cycle. While the GH response to sprint exercise has been well documented (Stokes, Nevill et al. 2002; Stokes, Nevill et al. 2002; Stokes, Nevill et al. 2003; Stokes, Nevill et al. 2004; Stokes, Nevill et al. 2005) and has been reported to be reproducible (Stokes 2003) the current study is the only one to track the week to week time course of adaptation in GH output in response to sprint training. Stokes and colleagues (Stokes, Nevill et al. 2004) investigated the GH responses to 3 and 6 weeks of sprint training, without investigating the weekly changes in GH output with training. Similar to previous reports utilizing acute sprint exercise, we observed an increase in GH output (Figure 1), after as little as 30 seconds of sprinting (Stokes, Nevill et al. 2003; Stokes, Nevill et al. 2005), but the interindividual variability in the GH response was still large. Peak GH in previous sprint studies ranged between 10.7 ± 11.1 and 15.0 ± 14.8 $\mu g \cdot l^{-1}$, compared to a peak GH of 8.42 ± 8.34 $\mu g \cdot l^{-1}$ in our study (means ± SD). With training, the peak GH values in our study tended to decrease about 4 $\mu g \cdot l^{-1}$ (8.42 ± 8.34 vs 4.12 ± 5.05 $\mu g \cdot l^{-1}$), which is consistent with the decreases in peak GH observed by Stokes et al (2003), who reported a decrease of about 4.5 $\mu g \cdot l^{-1}$ (10.3 ± 3.3 vs 5.8 ± 2.5 $\mu g \cdot l^{-1}$) after 6 weeks of training. This corresponds to a mean decrease in peak GH concentration of 50% in response to 3 weeks of sprint training, compared to about a 40% decrease in peak GH concentration in the Stokes study after 6 weeks of training.

The present study saw a 61% decrease in mean GH AUC from AS1 to AS4 compared to a 55% ($P < 0.05$) decrease in mean GH AUC in the Stokes study. The value in the present study was not significant, possibly due to the large interindividual

variability between subjects or the small (n = 8) sample size, but is similar to what has been reported previously with sprint training. Even with these minor differences it appears that after 3 and 6 weeks of sprint training exercise-induced GH outputs are lower and remain relatively similar during the 3 to 6 week timeframe. Therefore it appears that adaptation in GH output begins to occur in the initial 3 weeks of training.

GH peak results in this study are comparable to those found in acute resistance exercise studies which found peak GH levels of 5-25 $\mu g \cdot l^{-1}$ with levels returning to baseline values around 90 minutes post-exercise (Wideman, Weltman et al. 2002). This also agrees with a study by Kraemer et al. (1998), using a heavy resistance exercise protocol that consisted of 4 sets of a 10 repetition maximum squat exercise and elicited a peak GH response of about 8.5 $\mu g \cdot l^{-1}$ at 30 minutes post exercise (Kraemer, Hakkinen et al. 1998). This study utilized a large muscle mass exercise (squats) that is similar to the muscle mass activated in the present study when completing all out maximal sprint exercise on a cycle ergometer. The Kraemer study also noted that while GH levels were elevated at cessation of exercise, they were not significantly different from baseline. This finding is similar to what we noted in the current study with sprint exercise, but is completely different than the GH profiles noted with endurance exercise. This difference may be due to the short duration of the actual exercise performed in these aforementioned resistance and sprint exercise protocols, compared to endurance exercise protocols. Even though the endurance exercise was only 10 minutes, it was still 20 fold longer than the sprint exercise (10 min/0.5 min) and while the total amount of time for the resistance exercise was actually longer than the endurance exercise utilized in the Weltman

investigation, resistance exercise is intermittent and this may have altered the peak GH response. In the Weltman (2008) study the GH response occurs near or immediately after the termination of exercise in a 10 minute endurance exercise bout. It appears that this delay from the onset of exercise is similar to the delay in peak GH concentrations seen in our study. The 24-hour AUC does not differ in the Weltman study between the 3x10 min and 30 min continuous bouts which were both at the same intensity.It appears that significant elevations in GH concentrations past the immediate post exercise measure are not as likely in short duration protocols. The longer duration protocols may produce more elevations in GH due to the increased amount of work performed over the exercise period. This may explain why shorter duration protocols do not see a GH response that is as elevated as those of longer duration.

One notable finding is the change in difference from pre-exercise and peak GH levels across the acute sprint trials while total work remained the same. There could be several factors related to this occurrence most likely an adaptation to the activity or more sensitivity to circulating GH. This does note a significant change in the level of GH released in response to an acute sprint trial while also taking into account GH levels pre-exercise.

Another notable and somewhat surprising finding from the current investigation was that lean body mass increased with a concomitant decrease in fat mass and no change in total body mass after only 3 weeks of sprint training. Exercise induced GH release leads to protein sparing and increased lipolysis which could facilitate the maintenance of lean mass and decreases in fat mass (Frisch 1999). Increases in lean body mass also assist

in maintaining resting metabolic rate which may lead to decreases in body fat (McMurray and Hackney 2005). The high intensity and longer total duration of the training bouts [> 30 minutes in total duration and ~ 3 minutes of cumulative all out maximal exercise], would have elicited a marked GH response that was likely greater than that of the acute sprint tests. The anaerobic nature of these short training bouts most likely led to an accumulation of catecholamines in the blood and recruitment of primarily Type II motor units which as stated in Consitt (2007) is purported to stimulate an increase in GH release. In addition Kanaley (2004) noted GH release increased the release and turnover of free fatty acids post-exercise in men and GH deficient men who were administered GH pre-exercise. Pritzlaff (2000) also found that the increased usage of fat as a fuel source during recovery was positively correlated with increase in exercise intensity.

Although the changes in body composition were small they were still significant even with a small sample size. Five of 8 subjects decreased total fat mass and increased total lean mass, without significant changes in total body mass. Of note is the usage of a DXA scanner in measurement of body composition in the current study. The DXA is the gold standard in measurement of bone mineral density but there may be issues in the efficacy of soft tissue analysis. For confirmation of the results found in this study it is suggested that a similar study with a larger sample size be performed to compare results.

The intense nature of the training utilized in this study brings to the forefront the concept of overtraining as a mediator of the results. Although assessing overtraining markers is beyond the scope of the current study, some of the subjects made comments about their fatigue levels which lead the authors to believe they may have been

experiencing overtraining syndrome. Future studies should look into the concept that stress hormones may have played a role in the body composition changes found in this study. In a study of overtrained runners, Barron et al. (1985) noted decreases in the GH response to insulin hypoglycemia. Urhausen et al. (1998) studied endurance athletes for 1.5 years and found a decrease in exercise-induced GH release in response to an endurance test at 110% of the subjects' anaerobic threshold. Blood was taken immediately post as well as 5 and 10 minutes post-exercise. In our study the immediate and 5 minute post values did not significantly differ across all acute sprint trials. The 15 minute post-exercise value trended toward significance (Greenhouse-Geisser; $F = 3.67$, $P = 0.083$) across all trials. It is possible these subjects were experiencing overtraining syndrome but analysis of other markers of overtraining syndrome must be addressed to make this conclusion.

VO_2max did not increase after 3 weeks of sprint training in the current study, and these findings are consistent with those reported in other sprint training protocols of similar length (Burgomaster, GJF et al. 2006). The present study also noted similar effects with respect to heart rate changes during the VO_2max test. One notable finding in the present study is an increase in workload attained at the same VO_2max. These results suggest that short term sprint training can increase muscle oxidative capacity even in a short time period. This may be a result of reduced lactate accumulation and reduced glycogenolysis like that noted in matched-work exercise after two-weeks of sprint training in the study by Burgomaster, et al ((Burgomaster, Hughes et al. 2005). In addition Burgomaster et al. (2006) found an increase in citrate synthase activity and

stored muscle glycogen posttraining (Burgomaster, GJF et al. 2006). It is believed by the authors that a notable increase in VO_2max would require a longer training program such as the study by Weltman et al (1997) which found increased VO_2max (3.15 ± 0.54 vs. 3.41 ± 0.47 L/min; $P < 0.05$) and increased power output values at VO_2max (223 ± 40 vs. 263 ± 31 W; $P < 0.05$) after 6 weeks of endurance training on a cycle ergometer. While small changes in oxidative capacity may occur in as little as 2 weeks, it is well documented that changes in tissue related to oxygen delivery, such as increased capillary density, take longer to occur (McArdle, 2007).

The concept of interval or high intensity training has been widely studied for the use of performance enhancement for the athletic population. The author believes that high intensity training could be a viable alternative to endurance style aerobic training in the normal and special populations as well. The heavy workload of this particular protocol would prove difficult for most populations. However, adjustments to training intensity via alterations in resistance, duration, repetitions, and sessions per week could allow for more manageable and effective training for other populations. Interval training can provide a more time effective means of exercise for individuals who cannot devote 30-60 minutes to moderate intensity exercise. Additionally as has been shown in the present study, it appears that this type of interval training can improve lean body mass and decrease fat stores which could lead to improved maintenance of body composition in addition to cardiovascular benefits.

The lack of substantial findings for GH variables was likely due to a combination of the large inter-individual variability in GH output and the small sample size of the

current study. Therefore future studies should include a larger sample size in order to minimize the effects of the large inter-individual variability in GH release. Possible further research should include the effects of this type of training on those of aging individuals as well as the sedentary population. High-intensity sprint training if practiced correctly could be a viable alternative to traditional endurance training for the normal population with limited time for exercise.

Possible limitations in the present study include that of subject motivation, homogenous population, and the possibility that the subjects were overtrained. Subjects participated in high intensity exercise during 3 training sessions and 1 acute sprint session each week. It is difficult to quantify the exertion level given by these subjects in each of these training bouts. Subjects could have held back from maximal effort in order to conserve energy for subsequent sprints. Although encouraged to perform maximally each time it is possible they may not have given maximal effort. In this study we utilized a population of young active white males which limits generalization to other populations. Future studies should address gender as well as age differences. Yet another limitation to this study is the possibility that was discussed earlier that the subjects in this study may have been experiencing overtraining syndrome. Overtraining syndrome can result in hormonal disruption as well as performance decrements. One possible method to better tailor the training to the subjects' fitness level to avoid overtraining may be to train subjects based off a percentage of peak power in a pre-training cycle test.

In summary, we reject our hypothesis that a significant attenuation in peak growth hormone concentrations and growth hormone area under the curve would occur in response to an acute maximal 30-second sprint after sprint training. We supported our hypothesis that VO_2peak would not significantly increase in response to training. We also supported our hypothesis that peak power during acute sprint trials would increase significantly in response to training. Suggestions for future training protocols include training at an intensity level that is relative to LBM or VO_2max instead of purely total body mass. In conclusion, regular sprint exercise training may provide a viable alternative to longer durations of aerobic exercise for increasing GH output in some populations. However, the intensity of work required limits the use of sprint exercise in higher risk populations. Perhaps the best use of sprint exercise training is including it as a small part of a regular exercise program for individuals in the general population who are healthy.

WORKS CITED

Burgomaster, K. A., H. GJF, et al. (2006). "Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance." J Appl Physiol **100**: 2041-2047.

Burgomaster, K. A., S. C. Hughes, et al. (2005). "Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans." J Appl Physiol **98**: 1985-1990.

Djurhuus, C., C. Gravholt, et al. (2004). "Additive effects of cortisol and growth hormone on regional and systemic lipolysis in humans." American Journal of Physiology: Endocrinology and Metabolism **286**: E488-E494.

Enevoldsen, L., J. Polak, et al. (2007). "Post-exercise abdominal, subcutaneous adipose tissue lipolysis in fasting subjects is inhibited by infusion of the somastatin analogue octreotide." Clinical Physiol Funct Imaging **27**: 320-326.

Felsing, N., J. Brasel, et al. (1992). "Effect of low and high intensity exercise on circulating growth hormone in men." J Clin Endocrinol Metab **75**: 157-162.

Frisch, H. (1999). "Growth Hormone and body composition in athletes." Journal of Endocrinological Investigation **22**: 106-109.

Gibala, M. J., J. P. Little, et al. (2006). "Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance." Journal of Physiology **575**: 901-911.

Gordon, S. E., W. J. Kraemer, et al. (1994). "Effect of acid-base balance on the growth hormone response to acute high-intensity cycle exercise." J Appl Physiol **76**(2): 821-829.

Gravholt, C., O. Schmitz, et al. (1999). "Effects of a physiological GH pulse on interstitial glycerol in abdominal and femoral adipose tissue." American Journal of Physiology **277**: E848-E854.

Hansen, T., C. Gravholt, et al. (2002). "Dose dependency of the pharmacokinetics and acute lipolytic actions of growth hormone." J Clin Endocrinol Metab **87**: 4691-4698.

Jones, N., N. McCartney, et al. (1985). "Muscle performance and metabolism in maximal isokinetic cycling at slow and fast speeds." J Appl Physiol **59**: 132-136.

Kanaley, J. A., J. Y. Weltman, et al. (1997). "Human growth hormone response to repeated bouts of aerobic exercise." J Appl Physiol **83**: 1756-1761.

Kraemer, W. J., K. Hakkinen, et al. (1998). "Acute hormonal responses to heavy resistance exercise in younger and older men." European Journal of Applied Physiology **77**: 206-211.

McMurray, R. G. and A. C. Hackney (2005). "Interactions of Metabolic Hormones, Adipose Tissue and Exercise." Sports Medicine **35**(5): 393-412.

Moller, N., J. Gjedsted, et al. (2003). "Effects of growth hormone on lipid metabolism in humans." Growth Hormone & IGF Research **13(Suppl. A)**: S18-S21.

Pritzlaff, C. J., L. Wideman, et al. (1999). "Impact of acute exercise intensity on pulsatile growth hormone release in men." J Appl Physiol **87**(2): 498-504.

Raynaud, J., A. Capderou, et al. (1983). "Intersubject variability in growth hormone time course during different types of work." J Appl Physiol **55**: 1682-1687.

Stokes, K. A., M. E. Nevill, et al. (2004). "Effect of 6 weeks of sprint training on growth hormone responses to sprinting." European Journal of Applied Physiology **92**: 26-32.

Stokes, K. A., M. E. Nevill, et al. (2005). "Human growth hormone responses to repeated bouts of sprint exercise with different recovery periods between bouts." J Appl Physiol **99**: 1254-1261.

Stokes, K. A., M. E. Nevill, et al. (2002). "Growth hormone responses to repeated maximal cycle ergometer exercise at different pedaling rates." J Appl Physiol **92**: 602-608.

Stokes, K. A., M. E. Nevill, et al. (2002). "The time course of the human growth hormone response to a 6s and 30s cycle ergometer sprint." Journal of Sports Sciences **20**: 487-494.

Stokes, K. A., M. E. Nevill, et al. (2003). "Reproducibility of the growth hormone response to sprint exercise." Growth Hormone & IGF Research **13**: 336-340.

Sutton, J. and L. Lazarus (1976). "Growth Hormone in exercise: comparison of physiological and pharmacological stimuli." J Appl Physiol **41**(4): 523-527.

Viru, A. (1985). Pancreatic Hormones and the Somatotropin-Somatomedian System. Hormones in Muscular Activity. Boca Raton, CRC Press. **1:** 61-75.

Wideman, L., J. Y. Weltman, et al. (2002). "Growth Hormone release during acute and chronic aerobic and resistance exercise." Sports Medicine **32**(15): 987-1004.

Wideman, L., J. Y. Weltman, et al. (1999). "Effects of gender on exercise-induced growth hormone release." J Appl Physiol **87**: 1154-1162.

APPENDIX A:

RECRUITMENT FLYER

Male Research Participants Needed

Male research participants are needed for a research study investigation the effect of sprint exercise training on hormones & oxidative stress markers

You may be eligible for this study if you:

- Are a male between the ages of 18 and 30
- Currently exercise less than 5 hours per week
- Are willing to exercise 3 times per week on the UNCG campus
- Meet the study's height/weight requirements

You are *not* eligible for this study if you:

- You currently exercise more than 5 hours per week
- You currently train with sprint exercise
- You currently smoke or quit smoking within the past 6 months
- Have diabetes or heart disease

This training study will last approximately 4 weeks and will include a free assessment of body fat percentage and a 3 week supervised training plan in the UNCG Exercise Physiology Lab. Exercise sessions will be three times per week (30 min each) and there will also be 1 testing session per week (2 hrs).

For More information please contact:
Jason K. Smith
Department of Exercise & Sport Science
University of North Carolina at Greensboro
(704) 609-6367
Jksmith3@spartan.uncg.edu

THE UNIVERSITY of NORTH CAROLINA
GREENSBORO

Jason Smith 704-609-6367 Jksmith3@spartan.uncg.edu

Jason Smith 704-609-6367 Jksmith3@spartan.uncg.edu
Jason Smith 704-609-6367 Jksmith3@spartan.uncg.edu
Jason Smith 704-609-6367 Jksmith3@spartan.uncg.edu
Jason Smith 704-609-6367 Jksmith3@spartan.uncg.edu
Jason Smith 704-609-6367 Jksmith3@spartan.uncg.edu
Jason Smith 704-609-6367 Jksmith3@spartan.uncg.edu
Jason Smith 704-609-6367 Jksmith3@spartan.uncg.edu
Jason Smith 704-609-6367 Jksmith3@spartan.uncg.edu
Jason Smith 704-609-6367 Jksmith3@spartan.uncg.edu

APPENDIX B:

CONSENT FORM

UNIVERSITY OF NORTH CAROLINA AT GREENSBORO
CONSENT TO ACT AS A HUMAN PARTICIPANT

Project Title: The hormonal & oxidative stress adaptation to short term sprint exercise training

Primary Investigator: Laurie Wideman, PhD
Project Directors: Jason K. Smith or Kevin Ritsche

Participant's Name: _____

Why are we doing this study?

The purpose of this study is to compare how hormones [i.e. growth hormone] and oxidative stress markers change in response to acute exercise following short term (3 weeks) sprint exercise training.

Who is eligible to participate?

We are looking for individuals between the ages of 18 and 30, who are currently exercising between 3 and 5 hours per week. You must be a non-smoker and not taking medications that are known to alter hormones. You cannot have any injuries to your lower body that would prevent you from participating in this study.

What are we asking you to do?

If you agree to participate in this study, these are the things that we will be asking you to do:

- Come into the UNCG Exercise Physiology Laboratory to fill out a medical history questionnaire, have preliminary screening measurements and practice riding the stationary bicycle. This session will take ~ 30 minutes.
- Go to the Stone Building to have a bone scan completed to assess how much body fat you have. This will take ~ 30 minutes, including transit time between buildings.
- Perform a bike test that will get progressively harder to get your VO_{2max} value (VO_{2max} is the amount of oxygen your body uses to create energy). This will take ~20-30 minutes depending on how fit you are.
- Return to the lab at least 24 hrs later and have a resting blood profile done, which will take 2 hrs. The total blood taken will be just under half a cup [110 ml].
- At least 48 hrs later, return to the lab to have an exercise blood profile completed. This will require you to do a 30 second maximal sprint on the bike and we will collect blood samples before and after exercise. This will take ~2.5 hrs.

- Come in for 9 training sessions lasting about 30-45 minutes every other day for a total of 3 weeks (3 sessions each week).
- Come in for additional exercise blood profiles after each week of training [i.e. after week 1, 2 and 3 of training]. Each these sessions will take ~2.5 hrs.
- Repeat the bike test for your VO_{2max} on a separate day.

The total amount of blood that will be taken during the course of the entire study is 550 ml [110 ml x 5 sessions] or just over half a quart [2 1/3 cups].

What are the possible risks and discomforts related to this study?

There are minor risks that are possible as a result of participating in this study. These include muscle fatigue and dizziness during and after the exercise, abnormal changes in heart function, and, in very rare instances, heart attack (non-fatal or fatal) may also occur during the exercise test. However, the incidence of sudden cardiac death during vigorous exertion in healthy adults is extremely rare and is estimated at one death per year for every 15,000 to 18,000 individuals [ACSM 2006]. If you experience any pain during exercise you should immediately notify the researcher. In the unlikely event of an emergency, the researcher will provide Cardio-Pulmonary Resuscitation (CPR) and/or administer an Automatic External Defibrillator (AED) if appropriate and will call 911 for emergency assistance.

Infection is possible when blood samples are taken, but the risk of infection will be minimized through the use of sterile techniques by a trained technician. Only slight discomfort should occur. You should feel slightly more pain than a mosquito bite when the catheter is placed. Bruising may occur following catheter placement and may result in mild-to-moderate soreness to the touch for several days. You will be exposed to a small amount of radiation from the DXA scan that is equivalent to 1/10 the exposure from a routine chest x-ray, and less than the exposure from a dental x-ray.

What are the potential benefits of doing this study?

There is no direct benefit to you for participating in the study, except that you will receive 9 supervised training sessions.

Benefits gained by the researchers will be an increase in knowledge about the effect of sprint exercise training on hormonal and oxidative stress responses.

How will your confidentiality be maintained?

You will be assigned a subject number and all data will be identified by this number. The list connecting your name to this number will be kept in a locked file. Your name will not be used in any report. All information that is obtained during this study will be accessible only to the research staff. All de-identified data will be stored on the principal investigator's personal computer.

What happens if you get injured during the study?

There is no compensation for any physical injury that may result from your participation.

What happens if you have questions before or after you are involved in the study?

The University of North Carolina at Greensboro Institutional Review Board, which ensures that research involving people follows federal regulations, has approved the research and this consent form. Questions regarding your rights as a participant in this project can be answered by calling Mr. Eric Allen at (336) 256-1482. Questions regarding the research itself will be answered by Laurie Wideman, PhD at 334-3234, Jason K. Smith at (704) 609-6367 or Kevin Ritsche at 473-7293. Any new information that develops during this project will be provided to you if the information might affect your willingness to continue participation in the project.

What if you want to stop your participation in the study?

You are free to refuse to participate or to withdraw your consent to participate in this research at any time without penalty or prejudice; your participation is entirely voluntary.

What does signing this paper mean?

By signing this consent form you agree that you understand exactly what we are asking you to do, how long it will take, and any risks and benefits involved in this research.

By signing this form, you are affirming that you are 18 years of age or older and are agreeing to participate in the project described to you by _____. A copy of this consent form will be provided to you for your records.

_____ _____

Participant's Signature Date

APPENDIX C:

PHYSICAL ACTIVITY QUESTIONNAIRE

Physical Activity Questionnaire
Fall 2008

Name: _____ Date: _____

Question #1: In general, compared to other persons your age, rate how physically fit you are:

1	2	3	4	5	6	7	8	9	10
Not at all fit				Somewhat fit					Extremely fit

Question #2: How often do you engage in resistance training?

□ 5 or more times per week □ 4 times per week □ 3 times per week
□ 2 times per week □ 1 time per week □ Never/only occasionally

Question #3: How much hard physical work is required on your job?
□ a great deal □ a moderate amount □ a little □ none

APPENDIX D:

MEDICAL QUESTIONNAIRE

MEDICAL QUESTIONNAIRE

The purpose of this questionnaire is to determine if you have any physical limitations that may exclude you from participation in this investigation. All information will be kept completely confidential.

A. Personal Information

Name/Last: _____ First: _____ Middle Initial: _____
Date of Birth: _____ Gender: _____

Address/Street: _____

City: _____ State: _____ Zip Code: _____

Phone (H): _____ Phone (w): _____

Phone (cell): _____ E-mail: _____

Best way to reach you and when?_____

1. When was your last physical exam? _____

2. Please list any serious or chronic illnesses of which you are aware._____

3. Please list any allergies to medications, foods, or other substances. _____

4. Please list any medication you have been on or presently take

Type	Dosage/Frequency	How Long?	Why?

B. Medical History

1. Illnesses---Please check if you have had any of the following:

Illness	**Present**	**Past**	**Dates**
Heart attack			
Anemia			
Asthma			
Epilepsy			
Lung disease			
Stroke			
Gout			
Diabetes			
Hypoglycemia			
Rheumatic fever			
Heart murmur			
Hernia			

2. Symptoms---During the last 12 months, have you experienced:

Condition	Yes	No
High blood pressure		
Swelling of hands and feet		
Pain or cramps in legs		
Orthopedic problems		
Musculoskeletal problems		
ECG abnormalities		
Blurred vision		
Chest pain/pressure		
Shortness of breath		
Unusual fatigue		

Dizziness/light headed		
Significant weight change		
High cholesterol		
Numbness in limbs or face		

3. Hospitalizations---List the dates and the reasons for hospitalizations for any significant illness.

	Date	Diagnosis
1.		
2.		
3.		

C. Family History

1. Is your father living? Yes___No___If not, age at death and cause.
2. Is your mother living? Yes___No___If not, age at death and cause.
3. Has you father, mother, grandparents, or siblings had:

Condition	**Yes**	**No**	**Who?**
High blood pressure			
Stroke			
Heart attack (<50 yrs)			
Heart attack (>50 yrs)			
Diabetes			
Cardiovascular disease			
Other			