Effects of gender on exercise-induced growth hormone release

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Abstract:

We examined gender differences in growth hormone (GH) secretion during rest and exercise. Eighteen subjects (9 women and 9 men) were tested on two occasions each [resting condition (R) and exercise condition (Ex)]. Blood was sampled at 10-min intervals from 0600 to 1200 and was assayed for GH by chemiluminescence. At R, women had a 3.69-fold greater mean calculated mass of GH secreted per burst compared with men (5.4 ± 1.0 vs. 1.7 ± 0.4 μg/l, respectively) and higher basal (interpulse) GH secretion rates, which resulted in greater GH production rates and serum GH area under the curve (AUC; $1,107 \pm 194$ vs. 595 ± 146 μg · l^{-1} · min, women vs. men; $P = 0.04$). Compared with R, Ex resulted in greater mean mass of GH secreted per burst, greater mean GH secretory burst amplitude, and greater GH AUC ($1,196 \pm 211$ vs. 506 ± 90 μg · l^{-1} · min, Ex vs. R, respectivley; $P < 0.001$). During Ex, women attained maximal serum GH concentrations significantly earlier than men (24 vs. 32 min after initiation of Ex, respectively; $P = 0.004$). Despite this temporal disparity, both genders had similar maximal serum GH concentrations. The change in AUC (adjusted for unequal baselines) was similar for men and women (593 ± 201 vs. 811 ± 268 μg · l^{-1} · min), but there were significant gender-by-condition interactive effects on GH secretory burst mass, pulsatile GH production rate, and maximal serum GH concentration. We conclude that, although women exhibit greater absolute GH secretion rates than men both at rest and during exercise, exercise evokes a similar incremental GH response in men and women. Thus the magnitude of the incremental secretory GH response is not gender dependent.

Keywords: sex steroids | deconvolution analysis | endocrinology | human

Article:

Growth hormone (GH) release at rest is greater in young women than in comparably aged men (5, 27, 28). By deconvolution analysis, daily GH secretion rates are as much as 1.5- to 2.5-fold higher in young women than in age-matched men, and this gender difference is accounted for by

a twofold greater mass of GH secreted per burst (28). In addition, increases in basal interpulse GH secretion rates characterize young women (28). Studies in rats (17) have shown a more nearly continuous GH release pattern in females, whereas males have a more pulsatile GH release pattern. Recently, Pincus et al. (18) reported analogously more orderly GH release patterns in men than women. The gender difference in GH release patterns may be caused by increased GH-releasing hormone (GHRH) responsiveness or by reduced somatostatin inhibitory tone in women (28).

Aerobic exercise is a powerful physiological stimulus of GH release (11, 15, 20, 26, 34-36). Although the mechanisms underlying exercise-induced GH release have not been elucidated fully, they likely include GHRH release, somatostatin withdrawal, natural ligand release [i.e., GH-releasing peptide (GHRP)], or a combination of these hypothalamic responses. Although a single previous study reported that men and women have similar qualitative patterns of exercise-induced GH release (15), gender differences in the magnitude of the GH secretory response to aerobic exercise have not been investigated by frequent sampling and deconvolution techniques. Gender differences in exercise-induced GH release could result in different patterns of circulating GH during exercise; these patterns may influence metabolism during and after an aerobic exercise bout. Because exercise is a potent stimulus for GH release, we hypothesized that exercise would override the gender differences observed at rest and that the exercise-induced GH release would be similar in men and women. Given the foregoing issues, the purpose of the present study was to examine gender differences in deconvolution-estimated GH secretion rates during acute constant-load aerobic exercise.

METHODS

Eighteen healthy subjects [9 men (age, 25 ± 1.5 yr; ht, 178 ± 1.0 cm; wt, 75.1 ± 1.8 kg) and 9 women (age, 25 ± 1.0 yr; ht, 169 ± 2.0 cm; wt, 66.5 ± 3.1 kg)] voluntarily participated in this study. All subjects underwent a detailed medical history and physical examination and provided written informed consent as approved by the Human Investigation Committee at the University of Virginia. Subjects were not taking any medications or hormones and were habitual exercisers (20–30 min of aerobic exercise, 3–4 times/wk).

Body density was measured by hydrostatic weighing (12). Residual lung volume was measured by using an oxygen-dilution technique (37). Each subject was weighed in air on an Accu-weigh beam scale accurate to 0.1 kg and weighed underwater on a Chatillon autopsy scale accurate to 10 g. Percent body fat was calculated by using the equation of Brozek et al. (1).

Subjects also completed a peak oxygen consumption ($\dot{V}_{O_2\,peak}$)-lactate threshold (LT) test on a cycle ergometer. Initial power output was 40 W for women and 60 W for men, and the power output (PO) was increased 15 W every 3 min until volitional fatigue. Metabolic measures were collected by using standard open-circuit spirometric techniques (metabolic cart 2700Z; Sensormedics, Yorba Linda, CA). Heart rate was determined by electrocardiogram. An indwelling venous cannula was inserted in a forearm vein, and blood samples were taken at rest and during the last 15 s of each stage for the measurement of blood lactate concentration (model 2700; YSI Instruments, Yellow Springs, OH). The LT was determined from the blood lactate-PO

relationship (34). The PO for the constant-load aerobic exercise sessions (CLPO) was calculated as follows

$$CLPO = PO \text{ at } LT + 0.50(PO \text{ at } VO_2 - PO \text{ at } LT)$$

Subjects reported to the General Clinical Research Center (GCRC) on two other occasions (rest and aerobic exercise). Subjects were asked not to exercise within 24 h of the admission. Admissions for rest and aerobic exercise were randomized and were scheduled at least 2 days apart; women were studied during the early follicular phase (*days 2–8*) of the menstrual cycle. Volunteers received a standardized constant meal, based on body weight, at 1700 h the evening before the study. The total calories for the meal were calculated by using 0.33 × 37 kcal/kg for women and 0.33 × 38.5 kcal/kg for men; this amount included an activity factor (3a). The nutrient composition of the meal was 55% carbohydrate, 30% fat, and 15% protein. After subjects fasted overnight, venous cannulas were placed at 0500, and blood samples were withdrawn at 10-min intervals between 0600 and 1200. Each time subjects were admitted, blood samples were obtained at 0600 for later measurements of serum insulin-like growth factor (IGF)-I, total and free testosterone, and estradiol concentrations. During admission for aerobic exercise, subjects exercised for 30 min (from 0810 to 0840) at their predetermined PO. Subjects rested quietly in their rooms before and after aerobic exercise as well as during the nonexercise admission.

GH concentrations in all serum samples (0600–1200, both admissions) were measured in the GCRC Core Laboratory by using an ultrasensitive (0.002 µg/l threshold) chemiluminescence assay (Nichols, San Juan Capistrano, CA). The chemiluminescent assay detects predominantly the 22-kDa form of GH. The cross-reactivity for 20 kDa GH (methionated) in this assay is 33.8%. The addition of recombinant GH binding protein (rGHBP) in the physiological range resulted in a 10–20% reduction in measured GH; this indicates that rGHBP competes modestly with the assay antiserum for binding sites on the GH molecule. Intra-assay coefficient of variation (CV) for the GH assay was 6.0%, whereas the interassay CV was 9.9%. Total testosterone, free testosterone, and estradiol concentrations were measured in the GCRC Core Laboratory by using a solid-phase radioimmunoassay (RIA) (Coat-a-Count; Diagnostic Products, Los Angeles, CA). The intra-assay CVs were 6.9, 3.8, and 3.9% for total testosterone, free testosterone, and estradiol, respectively, whereas the interassay CVs were 10.3, 4.2, and 9.5%, respectively. IGF-I was measured by RIA (Nichols). The intra-assay CV for IGF-I was 6.7%, and the interassay CV was 13.6%.

Total GH area under the curve (AUC) was calculated by using the trapezoidal rule (31). The change in AUC (ΔAUC) was calculated thereafter by subtracting the AUC for the resting admission from the AUC for the aerobic exercise admission for each subject.

A multiple-parameter deconvolution method was employed to derive quantitative estimates of attributes of GH secretion from the measured serum GH concentrations. The subject-specific monoexponential half-life of apparent metabolic removal of endogenous GH was estimated concurrently (30). The procedure for deconvolution entails prefitting via an automated waveform-independent technique (PULSE2), in which regions that contain significant secretion impulses of undefined waveform are identified successively within the time series by their ability

to significantly reduce the total fitted variance by F ratio testing (10). Peak locations from PULSE2 were used as estimates in the multiparameter deconvolution analysis, which followed a set fitting pathway, as previously described (4, 33).

To avoid overdetermination of peaks (Nyquist concept), GH peaks that were closer than 20 min (2 sampling intervals apart) were eliminated from the file, and the data were refit. In addition, any peaks that were outside the sampling window (0–370 min) by more than one sample interval (10 min) were eliminated from the file. A pulse of GH secretion was approximated algebraically by a Gaussian distribution of secretory rate (30). Basal secretion (time invariant) was estimated concurrently, as previously described (33). GH secretory pulses were considered significant if the fitted amplitude (maximal value attained within the computed secretory event) could be distinguished from zero with 95% statistical confidence. The half duration of the GH secretory pulse (defined as the duration in minutes of the calculated secretory burst event at half-maximal amplitude), GH half-life of elimination, and GH distribution volume were assumed to be constant throughout any one study period for an individual. The mass of GH secreted per pulse was estimated as the area of the calculated secretory pulse (in µg/l distribution volume) (30). The endogenous pulsatile GH production rate was defined as the product of the number of GH secretory pulses and the mean mass of GH secreted per pulse.

A two-way nested ANOVA model was used to analyze condition and gender effects for GH AUC, GH secretion parameters, and concentrations of serum sex-steroids and IGF-I. ANOVA computations were carried out by using the mixed model procedure (Proc Mixed in SAS version 6.12 SAS/STAT Software Changes and Enhancements, 1996). Model parameters were estimated by restricted maximum likelihood (8), and 95% confidence limits were estimated by least significant difference (LSD) criteria (13). All outcomes were analyzed on the natural log scale to attain equal variance among groups. P values presented from the ANOVA are for comparisons made on the log-transformed data. Gender differences in the constant-load exercise data and the time to maximal GH concentration were tested by using an unpaired Student's t-test. Linear regression was applied to investigate the relationship between maximal serum GH concentration and GH half-life. Statistical significance was interpreted as $P \leq 0.05$.

RESULTS

Gender comparisons for total body fat, $\dot{V}_{O_2\,peak}$ and constant-load aerobic exercise data are presented in Table 1. Men had a greater absolute $\dot{V}_{O_2\,peak}$ (in l/min) compared with women, but there was no gender difference in relative $\dot{V}_{O_2\,peak}$ peak (ml $\cdot$ kg^{-1} $\cdot$ min^{-1}), the CLPO, the PO at LT, or the maximal PO obtained. The CLPO expressed as a percentage of the maximal PO was 72%. During the constant-load aerobic exercise, men and women did not differ in the total work completed in 30 min, the final blood lactate concentration, or the total energy expended. Although the $\dot{V}_{O_2\,peak}$ attained in the last 2 min of aerobic exercise was significantly greater in men than women, the end-aerobic exercise oxygen consumption ($\dot{V}_{O_2}$) expressed as a percentage of $\dot{V}_{O_2\,peak}$ was similar in both genders (Table 1).

Table 1. Comparison of total body fat, $\dot{V}_{O_2\,peak}$, lactate threshold, power output, and constant-load exercise results between men and women

	Men	Women
Total body fat, %	14.5 ± 1.9	$22.6 \pm 1.8^*$
$\dot{V}_{O_2\,peak}$, ml·kg^{-1}·min^{-1}	43.4 ± 1.7	39.5 ± 2.2
$\dot{V}_{O_2\,peak}$, l/min	3.3 ± 0.16	$2.6 \pm 0.12^*$
Constant load, W	158 ± 11.5	133 ± 11.0
Lactate threshold, W	99 ± 8.5	87 ± 10.0
Max power output, W	220 ± 15.0	183 ± 13.5
Constant load/max power output, %	71.7 ± 0.69	72.6 ± 1.2
Constant-load total work, kJ	285 ± 20.9	235 ± 19.8
[Blood lactate] at 30 min, mmol/l	6.0 ± 0.7	5.5 ± 0.7
Constant-load peak $\dot{V}_{O_2}$, liters	2.5 ± 0.2	$2.0 \pm 0.1^*$
Constant-load total energy, kcal	354 ± 22.2	293 ± 23.4
End $\dot{V}_{O_2\,peak}/\dot{V}_{O_2}$, %	75.2 ± 4.0	74.2 ± 4.2

Values are means $\pm$ SE; $n = 9$ for men and $n = 9$ for women. $\dot{V}_{O_2}$, O_2 consumption; $\dot{V}_{O_2\,peak}$, peak $\dot{V}_{O_2}$; max, maximal; [blood lactate], blood lactate concentration. $^*P < 0.05$.

The visually evident pattern of the serum GH concentration response during rest and aerobic exercise was similar in men and women (Figs. 1 and 2, respectively). The time to reach the maximal GH concentration during exercise was greater in men than women (men attained peak serum GH concentrations 32 min after the initiation of exercise, whereas women achieved peak GH concentrations 24 min into the 30-min constant-load exercise bout, $P = 0.004$). During aerobic exercise, both men and women responded with increased GH release, and the maximal serum GH obtained was greater in women than men. However, the relative increase in GH concentration observed for men (5.8-fold, 95% CI 3.9–8.6) was significantly greater than the increase observed for women (3.2-fold, 95% CI 2.2–4.8; $P = 0.043$). Linear regression analysis revealed that the maximal GH concentration attained did not significantly influence the GH half-life, as measured by deconvolution analysis.
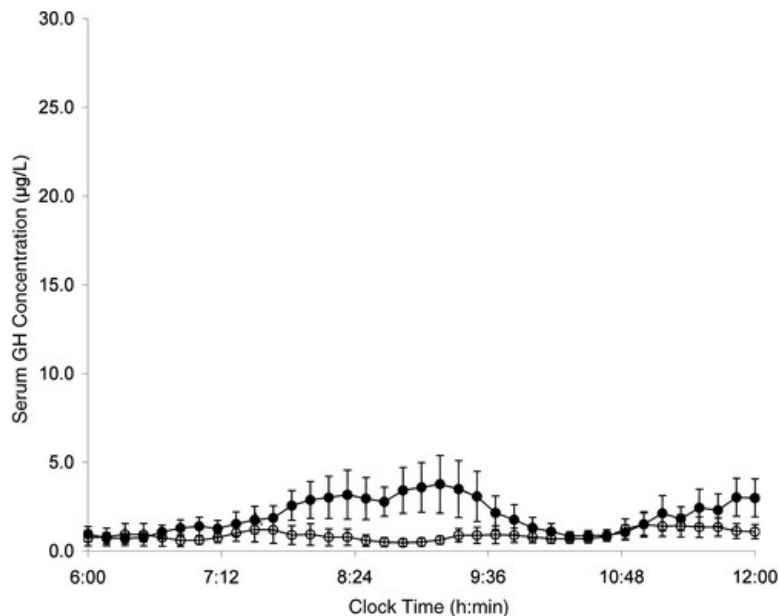

Fig. 1. Mean serum growth hormone (GH) response patterns for men (○) and women (●) during rest; $n = 9$ in each group. Values are means $\pm$ SE.
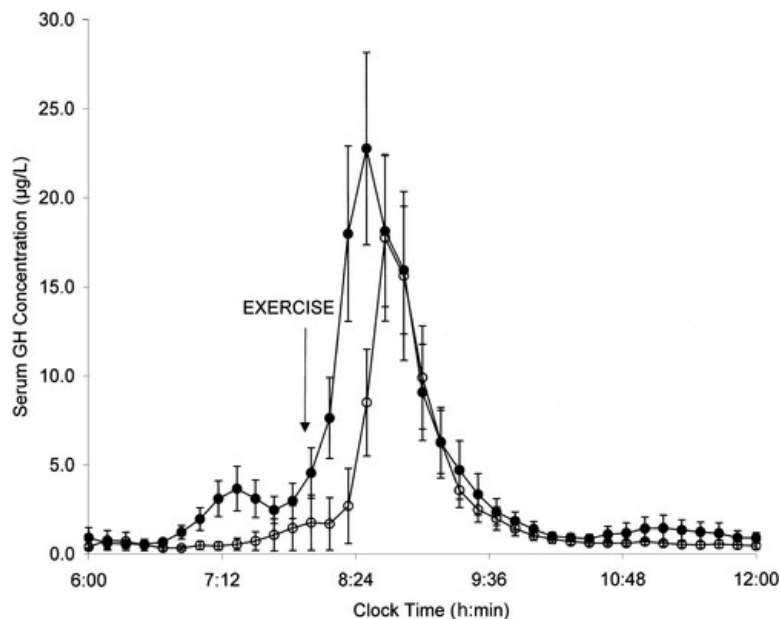
Fig. 2. Mean serum GH response patterns for men (○) and women (●) during exercise; $n = 9$ in each group. Values are means ± SE.

Figure 3 shows GH AUC and ΔAUC for men and women at rest and in response to aerobic exercise. GH AUC increased from 506 ± 90 μg · l^{-1} · min during the resting admission to $1,196 \pm 211$ μg · l^{-1} · min during the aerobic-exercise admission (pooled gender data; $P < 0.001$). Women had greater GH AUC compared with men [$1,107 \pm 194$ vs. 595 ± 146 μg · l^{-1} · min, respectively (pooled rest/aerobic exercise data); $P = 0.042$]. The ΔAUC for women was 811 ± 268 μg · l^{-1} · min and the ΔAUC for men was 593 ± 201 μg · l^{-1} · min ($P = 0.53$).
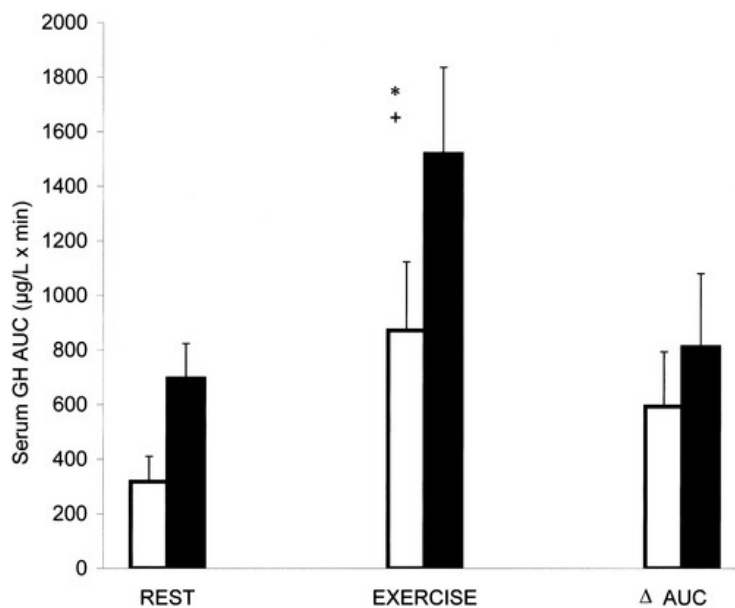

Fig. 3. Serum GH area under the curve (AUC) during rest and exercise and change in AUC (ΔAUC) for men (open bars) and women (solid bars). * Main effect for gender, $P < 0.05$; + main effect for condition, $P < 0.05$.
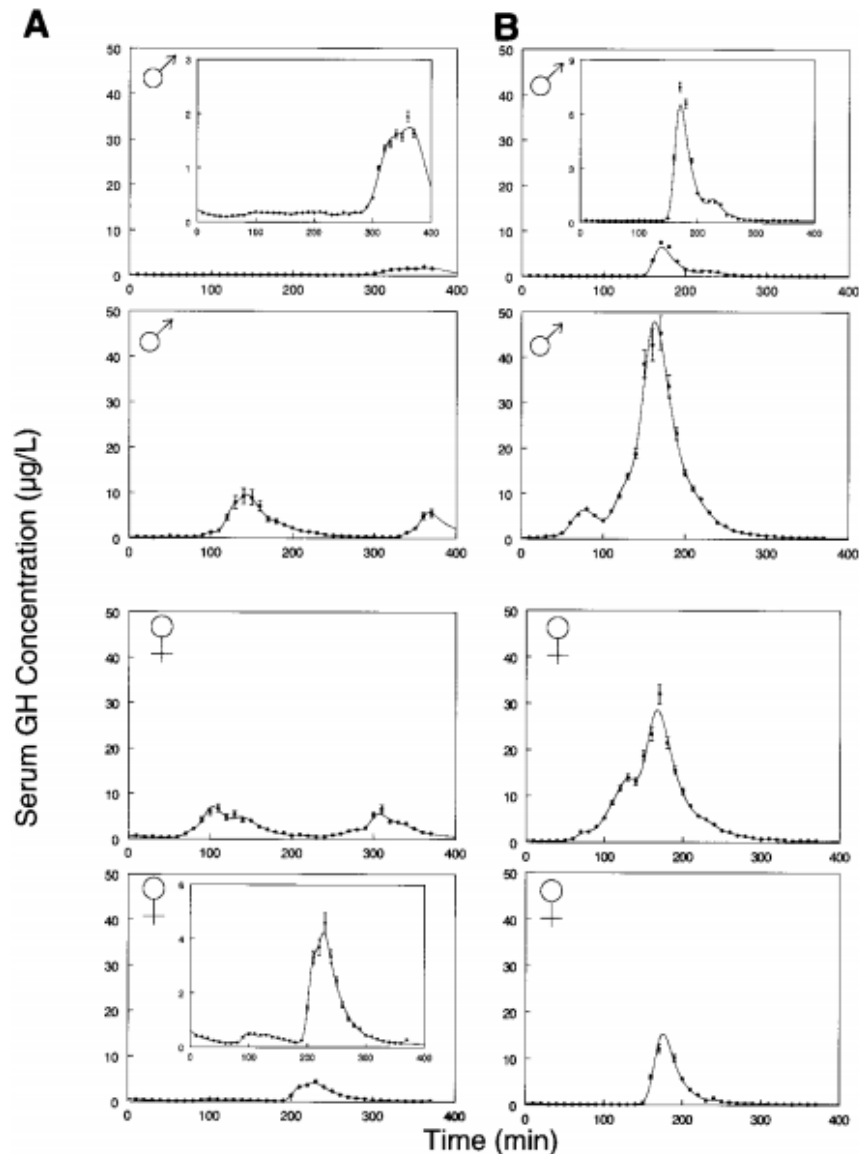
Fig. 4. Representative GH concentration curves from 2 men (*top*) and 2 women (*bottom*) during rest (*A*) and exercise (*B*). *Insets:* blow-ups of curves.

Representative GH concentration curves for two men and two women are depicted in Fig. 4, whereas Fig. 5 shows the GH secretion profiles from deconvolution for the same subjects. Table 2 shows specific GH secretion measures for men and women during rest and exercise. GH basal (interpulse) secretion was significantly greater in women than in men (0.012 ± 0.002 vs. 0.006 ± 0.001 $\mu g \cdot l^{-1} \cdot min^{-1}$, respectively; $P = 0.043$) and was not condition dependent (i.e., resting and aerobic exercise admissions had similar GH basal secretion). There were no significant differences in GH half-life or half duration of GH burst. Although women had greater pulsatile GH production rates compared with men at rest (34.9 ± 8.3 vs. 10.5 ± 3.1 $\mu g \cdot l^{-1} \cdot 6$ h, for women and men, respectively) and during exercise (63.3 ± 12.7 vs. 36.9 ± 9.2 $\mu g \cdot l^{-1} \cdot 6$ h, for women and men, respectively), the relative increase in pulsatile GH production rate between rest and exercise was greater in men (3.5-fold, 95% CI 2.6–5.7) compared with women (1.8-fold,

95% CI 1.2–2.7; $P = 0.009$). In both women and men, the increase in pulsatile GH production rate observed during exercise was due to an increase in mean mass of GH secreted per burst, as the number of GH peaks was significantly reduced by exercise (6 vs. 5, $P = 0.024$). As was observed with pulsatile GH production rate, the relative increase in mean mass of GH secreted per burst between rest and exercise was greater in men (5.1-fold, 95% CI 3.2–9.2) compared with women (2.2-fold, 95% CI 1.4–3.5; $P = 0.016$). Mean GH burst amplitude was greater in women compared with men ($P = 0.027$) and during exercise compared with rest ($P < 0.001$). A trend was observed for a greater relative increase in mean GH burst amplitude between rest and exercise in men (6.6-fold, 95% CI 3.6–12.0) compared with women (3.1-fold, 95% CI 1.7–5.6; $P = 0.077$).
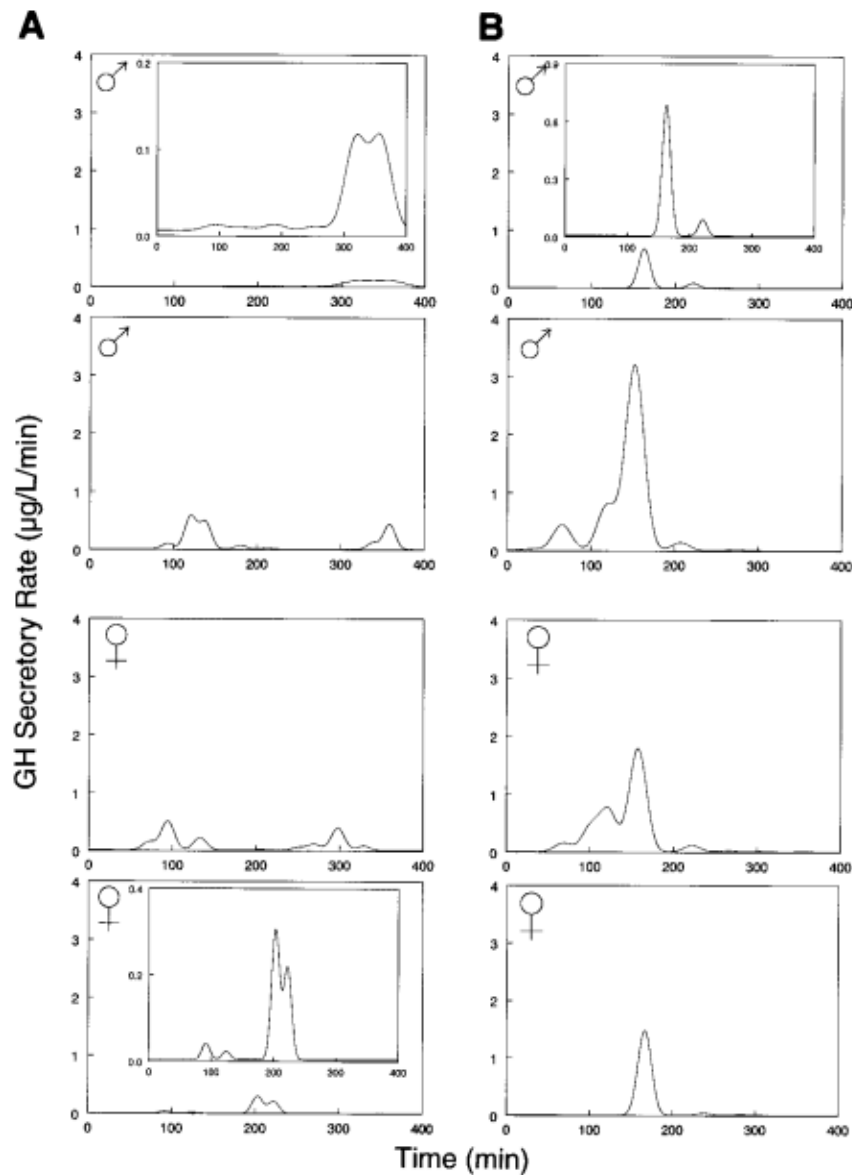

Fig. 5. Representative GH secretion profiles during rest (*A*) and exercise (*B*) from same 2 men (*top*) and 2 women (*bottom*) as in Fig. 4. *Insets:* blow-ups of curves.

Table 2. Gender comparisons for calculated GH secretion measures

	Men		Women	
	Rest	Exercise	Rest	Exercise
Basal GH secretion rate, $\mu g \cdot l^{-1} \cdot min^{-1}$*	0.006 ± 0.002	0.007 ± 0.001	0.014 ± 0.004	0.011 ± 0.002
GH burst half duration, min	38 ± 3.4	33 ± 1.9	33 ± 3.1	34.0 ± 1.9
GH half-life, min	16 ± 1.4	14 ± 0.8	14 ± 1.3	14.4 ± 0.8
No. of GH peaks/6 h†	5.8 ± 0.5	4.8 ± 0.8	6.2 ± 0.6	5.3 ± 0.6
Mean mass of GH secreted/burst, $\mu g/l$‡	1.7 ± 0.4	8.7 ± 2.2	5.4 ± 1.0	12.5 ± 2.5
Mean GH burst amplitude, $\mu g \cdot l^{-1} \cdot min^{-1}$*†	0.08 ± 0.02	0.5 ± 0.15	0.2 ± 0.05	0.7 ± 0.16
Pulsatile GH production rate, $\mu g \cdot l^{-1} \cdot 6$ h‡	10.5 ± 3.1	36.9 ± 9.2	34.9 ± 8.3	63.3 ± 12.7
Maximal serum GH, $\mu g/l$‡	3.4 ± 0.8	18.6 ± 4.8	7.1 ± 1.3	24.3 ± 5.0

Values are means $\pm$ SE; $n = 9$ for men and $n = 9$ for women. GH, growth hormone. *Significant gender effect, $P < 0.05$. †Significant condition effect, $P < 0.05$. ‡Significant gender $\times$ condition interaction effect, $P < 0.05$.

The concentrations of serum IGF-I and sex steroids are presented in Table 3. Total and free testosterone concentrations were greater in men than in women ($P < 0.001$ and $P < 0.001$, respectively). There was no difference in the concentration of total or free testosterone during the rest compared with the aerobic exercise admissions ($P = 0.152$ and $P = 0.245$, respectively). Serum estradiol and IGF-I concentrations were similar in men and women ($P = 0.069$ and $P = 0.852$, respectively) and on the aerobic exercise vs. rest days ($P = 0.630$ and $P = 0.577$, respectively).

Table 3. Gender comparisons of serum concentrations of sex steroids and IGF-I

	Men		Women	
	Rest	Exercise	Rest	Exercise
Total testosterone, nmol/l*	26 ± 1.4	24 ± 2.0	1.1 ± 0.1	1.0 ± 0.1
Free testosterone, pmol/l*	88 ± 8.0	92 ± 10.2	9.0 ± 3.9	4.0 ± 0.5
Estradiol, pmol/l	132 ± 10.9	143 ± 16.5	110 ± 25.7	99 ± 15.4
IGF-I, $\mu g/l$	338 ± 18.0	319 ± 19.0	328 ± 32.0	327 ± 22.0

Values are means $\pm$ SE; $n = 9$ for men and $n = 9$ for women; IGF-I, insulin-like growth factor I. *Values for men are significantly greater than those for women, $P < 0.05$.

DISCUSSION

Gender differences in pulsatile GH release exist, but such gender distinctions have been recognized in the resting condition only (7, 18,27, 28). In the present study, we examined gender differences in GH release during rest and constant-load aerobic exercise. Our analyses show that women have greater basal (interpulse) GH secretion and serum GH AUC compared with men, independent of condition (Table 2). The higher serum GH AUC and calculated GH (pulsatile) production rate in women reflected a greater mean mass of GH secreted per burst compared with men during both rest and aerobic exercise. During exercise, augmentation of the mean mass of GH secreted per burst resulted in greater GH AUC and GH production rate, compared with rest in both genders (Table 2). Serum GH AUC (or GH secretion rate) was not affected by estradiol, total or free testosterone, or IGF-I (Table 3).

Gender differences in calculated baseline (resting) GH secretion and serum GH AUC were previously noted by several investigators (7, 18,27). In support of data previously reported by van den Berg et al. (27), who used an immunofluorometric assay, women in the present study had higher basal serum GH concentrations than did men (Table 2), as assessed in a

chemiluminescence assay. The 24-h GH AUC also is higher in young women than in young men, and the magnitude of the difference is approximately twofold (7, 27, 28). Although serum GH AUC in the current study represents data collected for only 6 h, women had significantly greater GH AUC, and the magnitude of the disparity was similar to that reported for 24-h GH AUC (~2-fold). Serum GH concentrations in the present study (Figs. 1 and 2) at rest were uniformly measurable for the first time in both men and women in the ultrasensitive GH assay employed here, unlike earlier RIA (5) or immunofluorometric assays (18, 27).

As expected, aerobic exercise elicited a greater serum GH AUC compared with the resting admission (Fig. 3). This finding supports data from previous studies that indicate that aerobic exercise of appropriate intensity and duration is a powerful, effective, physiological stimulus for GH release (11, 15, 20, 35, 36). However, as documented in other studies (11, 15, 20, 26, 34, 35), the maximal aerobic exercise-induced GH concentration is variable among subjects (Fig. 4). This variability was not gender specific and was not due to the intensity of aerobic exercise, because all subjects worked at the same intensity relative to their LT and maximal exertion (~75% of their individual $\dot{V}_{O_{2\,peak}}$), for the constant-load aerobic exercise session. The biological variability in the GH response to exercise is similar to that observed with multiple other GH stimulation tests (i.e., l-arginine, l-3,4-dihydroxyphenylalanine, clonidine) (5).

Despite gender differences in the absolute values of GH release during rest and aerobic exercise, the pattern of aerobic exercise-induced GH release was similar in men and women. As previously described in humans (15, 20, 25, 26), both genders attained maximal serum GH concentrations at or near the end of the 30-min aerobic exercise bout, and GH levels returned to baseline within 90 min of the termination of exercise (Fig. 2). We are not aware of any other studies that compare in men and women the pattern of acute exercise-induced, short-term (6 h) GH release. Although Lassarre et al. (15) reported that women and men had similar chronologies of GH release, the authors did not present any data for women. In the present study, women achieved peak serum GH concentration significantly earlier in the exercise bout than did men (24 vs. 32 min). Although women had higher serum GH concentrations at rest and during exercise and attained their peak GH concentration sooner than men, the fold increase in GH concentration was significantly greater in men compared with women (5.8- vs. 3.2-fold). The ΔAUC and the incremental magnitude of the aerobic exercise-induced rise in GH secretion were similar in men and women (Fig. 3). We speculate that the lower absolute GH AUC observed during rest and exercise in men, without detrimental effects on muscle mass and fat patterning, is offset by the combined anabolic effects exerted by testosterone and GH on target tissues.

In the present study, GH AUC and calculated pulsatile GH production rate were greater in women than men. This was attributable to an increased mean mass of GH secreted per burst in women. These results are similar qualitatively to 24-h data reported previously (18, 27), but they represent the first such gender comparison in an ultrasensitive chemiluminescence-based GH assay (29). Mean GH burst amplitude was greater in women compared with men in the present study, akin to previous 24-h observations (28). There was no gender difference in GH secretory pulse number, half duration, or half-life. Previous reports suggest that gender does not affect pulse number (7, 27, 34), and GH half-life has been reported to be gender-independent (23).

The increases in serum GH AUC and calculated GH production rate during aerobic exercise were due to augmentation of the mean mass of GH secreted per burst, which in turn reflects the mean GH secretory burst amplitude. Kanaley et al. (11) also observed a rise in the mean mass of GH secreted per burst (and the mean GH burst amplitude) with repeated acute aerobic exercise. We did not identify a decrease in apparent GH half-life during aerobic exercise compared with rest. Although this finding is similar to that of Kanaley et al., both results differ from other findings (15, 26). This may be due to the fact that the GH half-life observed at rest in the present study was shorter than that observed in other studies and was calculated on the basis of a chemiluminescence assay measurements of basal release (32). However, it falls within the normal range of biologically acceptable GH half-life values measured directly (22).

Because sex steroid hormones and IGF-I have been shown to affect GH release in a variety of ways (2, 7, 14, 28), the concentrations of these hormones were measured in each study session, and the possible contributions to differences in GH release were assessed. As expected, total and free testosterone concentrations were significantly greater in men than in women, but there was no difference within gender in the concentrations on the rest or aerobic exercise day. Estradiol concentrations were similar in men and women, probably because all women were studied in the early follicular phase of the menstrual cycle, when estradiol levels are lowest. Serum IGF-I concentrations were similar in the resting and aerobic exercise admissions as well as in men and women. Aerobic exercise can result in acute GH-independent increases in serum IGF-I levels (2), but IGF-I concentrations in the present study were measured only at *time 0* before aerobic exercise. Although gender differences in aerobic exercise-induced changes in serum IGF-I concentrations might exist, this question was not addressed in the present study.

The actual mechanisms by which aerobic exercise-induced GH secretion occurs are still unclear. In a rat model, with GH assessed in a growth-plate bioassay, afferent neural excitation during exercise signaled release of GH from the pituitary (6). Various neurotransmitters (such as norepinephrine, acetylcholine, and opioids) also have been suggested as playing roles in the control of aerobic exercise-induced GH secretion (3, 16, 24, 27). However, no one mechanism has been proven to be primary. Probably several neurotransmitters are involved (5). However, the final common pathway presumptively involves either increased release of GHRH, decreased release of somatostatin, or a combination of both. The recent cloning of the GHRP receptor (9, 19), and the possibility that an endogenous GHRP-like molecule will be isolated, allow the consideration that the putative GHRP system also can serve to regulate aerobic exercise-induced GH secretion. Thus the mechanisms underlying aerobic exercise-induced GH secretion must be investigated more extensively.

In conclusion, the present study substantiates that 30 min of aerobic exercise at an intensity above the LT constitute an effective physiological stress for GH release in both men and women. The present data show that, regardless of gender differences in baseline serum GH concentrations, basal pulsatile GH secretion (as assessed by deconvolution analysis) and the time to reach the maximal serum GH concentration, the magnitude of the (incremental) increase in GH release that is induced by aerobic exercise is similar in both men and women.

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