Running-Induced Alterations in Growth Hormone, Prolactin, Triiodothyronine, and Thyroxine Concentrations in Trained and Untrained Men and Women

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This study examined whether gender and/or training were related to the exercise-induced changes in plasma concentrations of growth hormone (GH), prolactin (PRL), triiodothyronine ($T_3$), and thyroxine ($T_4$). Twenty subjects (male and female 10 km runners; untrained males and females) ran on a treadmill for 30 min at 80% of previously determined maximum heart rate. Blood samples were taken through an indwelling catheter from an antebrachial vein at 0, +15, and +30 min of the test and 30 min of recovery. Rectal temperature rose significantly ($p < .01$) at +15 and +30 min with concomitant rise in GH concentration, but PRL, $T_3$, and $T_4$ were not affected by the exercise. We concluded that a 30-min run at 80% of maximum heart rate is associated with higher concentrations of GH but not of PRL, $T_3$, and $T_4$. Neither training state nor gender affected the aforementioned results.

Key words: growth hormone, prolactin, gender, training, running, hormone

The effects of exercise on hypothalamic–pituitary function have been of interest to numerous investigators (Bullen et al., 1984; Dulac et al., 1987; Karagiorgos, Garcia, & Brooks, 1979). However, disparities and questions have emerged from previous investigations, especially concerning the effects of gender and training on anterior pituitary hormones. Growth hormone (GH), for instance, has been reported to have a greater response to exercise in trained subjects (Bunt, Boileau, Bahr, & Nelson, 1986), whereas others have reported a lesser GH response in trained subjects (Hartley et al., 1972; Sutton, Young, Lazarus, Hickie, & Maksytyis, 1969). There is some evidence that gender differences exist in untrained subjects. GH response has been observed from less than 20 min of exercise, with greater responses found in females (Bunt et al., 1986; Sidney, Shephard, Webster, & Wood, 1974). Prolactin (PRL) is another anterior pituitary hormone that is released in response to stress and has a molecular structure very similar to that of GH. The response of PRL in males is not as well documented as in females (Zaworonok, Hudson, & Orban, 1987); moreover, both increases and decreases in response to exercise have been reported (Viru, 1985). For females, a postexercise increase in PRL concentrations in response to 60 min of cycling exercise has been established; however, prior to the training the PRL increase was not observed (Bullen et al., 1984). Additionally, exercise may induce release of thyroid-stimulating hormone (TSH) from the anterior pituitary (Schmid et al., 1982). TSH release could affect thyroid gland secretion of triiodothyronine ($T_3$) and thyroxine ($T_4$). Several investigators have focused on the effect of exercise on $T_3$ and $T_4$ concentrations. For men, it has been shown that bench stepping at a low intensity accompanied by a heat load was associated with a reduction in $T_3$ concentrations (Epstein, Udassin, & Sack, 1979). Resting $T_3$ and $T_4$ concentrations have been examined after women completed different volumes of training (Boyden, Pamentier, Stanforth, Rotakis, & Wilmore, 1982). Higher training volumes resulted in increased circulating levels of $T_3$ and $T_4$, whereas lower volumes of training showed the opposite effect.

Both gender and training may affect various endocrine responses through a variety of possible mechanisms. For example, a greater GH response to exercise in women could result from gender-affect 17β-estradiol concentrations (Frantz & Rabkin, 1965) or lower cardiorespiratory fitness levels (training effect) (Bunt et al., 1986). Female PRL response to exercise could be enhanced by breast motion produced during exercise (Prior,
Jensen, Yeun, Higgins, & Brownlie, 1981). A lower training-induced PRL response to exercise could occur as well, because higher body temperatures may increase PRL concentrations (Calbo, 1983) and trained individuals would experience an attenuated rise in body temperature. In addition, it has been suggested that training may cause a reduction in percent body fat in women that may be associated with a reduction in the $T_v/T_4$ ratio (Boyden et al., 1982).

In an earlier article we reported the effects of treadmill running on plasma beta-endorphin, corticotropin, and cortisol levels in 10-km runners (Kraemer, Blair, Kraemer, & Castracane, 1989). The present study continues the endocrinological investigation of those same runners in a descriptive effort to determine whether gender and training state affect GH, PRL, $T_v$, and $T_4$ responses to treadmill running.

**Method**

**Subjects**

The subjects of this investigation were the same as those in a previous study (Kraemer et al., 1989) (see Table 1). The research protocol was approved by the Investigative Review Board of Texas Tech Health Sciences Center, Amarillo, TX, and all participants gave written informed consent to participate. The subjects were divided into four groups: 5 male and 5 female 10-km runners, 5 male and 5 female untrained subjects. Subjects were free from known disease. Trained subjects had personal records of 45 min or better for a 10-km run and trained a minimum of 56 km per week. All untrained participants had not exercised on a regular basis for at least 1 year. One 10-km runner and one untrained subject were taking oral contraceptives. The resting hemoglobin values for all subjects were within the normal range. All subjects maintained normal dietary habits and state of physical conditioning during the study.

**Procedures**

The subjects reported to the laboratory and were given a Bruce treadmill test (American College of Sports Medicine, 1991) to exhaustion to determine maximum heart rate (Table 1). The leveling of heart rate at or near functional capacity was the criterion for determining maximal heart rate. Subjects were encouraged to exert maximal effort during the test.

Subjects again reported to the laboratory for a 30-min treadmill run at 80% of maximum heart rate. This exercise test was chosen because it had an adequate duration and intensity for eliciting an endocrine response but was not too strenuous for the untrained subjects to complete. No food or beverage other than water was consumed 2-6 hours prior to the run. A catheter was inserted into an antecubital vein of the subject’s left arm prior to the initiation of exercise and kept patent with a heparin lock. Blood samples were taken without stasis 30 min before and immediately before treadmill running (-30, 0), 15 min into the run (subjects continued to run during the blood draw) (+15), immediately after the run (subjects rested during blood draw) (+30), and after 50 min of recovery (R30). The first four blood samples were taken in an upright position that represented the normal body position of a runner before, during, and after a competitive run. Subjects rested after the run in a seated position, and that position was maintained during the last blood sampling. Heart rate was monitored continuously; treadmill running was maintained at 80% of maximal heart rate by adjusting treadmill speed and grade. Auscultatory blood pressure was assessed before, during, and after the exercise. To obtain auscultatory blood pressure at +10 and +20 min during the exercise test while maintaining 80% of maximal heart rate, it was necessary to increase the treadmill grade and reduce the speed for some of the subjects. A telethermometer was used to monitor rectal temperature with a rectal probe inserted approximately 100 mm into the rectum.

A 7-ml blood sample was immediately put into a chilled glass tube containing EDTA and then placed on ice; 10 ml of untreated whole blood were collected in a separate tube. A 3-ml aliquot was put into an EDTA tube at room temperature for hemoglobin analysis. Plasma for GH analysis and serum for PRL, total $T_v$, and total $T_4$ analysis were separated from whole blood by centrifugation (3000 g for 12 min, 4°C for EDTA samples) and stored at -70°C until assayed.

**Radioimmunoassays**

Plasma or serum levels of GH, PRL, total $T_v$, and total $T_4$ were measured using commercially available reagents (Diagnostic Products Corp., Los Angeles, CA). Serum was stored at -70°C until assayed for GH, PRL, total $T_v$, and total $T_4$. All samples from an individual subject were

| Table 1. Weight, age, total treadmill time from maximum heart rate test, maximum heart rate, and exercise heart rate of subjects at 80% of maximum heart rate |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|
|                                | Trained         | Untrained       |                 |                 |
|                                | Males           | Females         | Males           | Females         |
| Weight (kg)                    | 65.0 (6.9)      | 57.0 (7.8)      | 87.2 (18.5)     | 67.8 (10.5)     |
| Age (years)                    | 32.2 (4.7)      | 30.4 (2.7)      | 29.6 (6.5)      | 28.8 (4.7)      |
| $HR_{max}$ ($b/min^{-1}$)      | 194.6 (8.3)     | 184.0 (14.8)    | 196.4 (14.8)    | 194.8 (8.7)     |
| $HR_{exp}$ ($b/min^{-1}$)      | 155.6 (6.7)     | 148.0 (2.0)     | 157.0 (12.3)    | 155.2 (6.9)     |

*Note: Values are means ± (SD) of n = 5 in each group.*

$HR_{exp}$ = exercise heart rate at 80% of maximal heart rate.
assayed within the same assay run. Intra assay coefficient of variation was less than 5% for each assay.

Statistics

A Sex (male, female) x Training (trained, untrained) x Time (-30, 0, +15, +30, R30) repeated measures ANOVA was conducted for each dependent variable. Tukey’s least significant differences tests were applied where appropriate. All comparisons were considered statistically significant at $p < .01$. The probabilities were adjusted for the Geisser–Greenhouse degrees of freedom (Greenhouse & Geisser, 1959).

Results

A Sex x Training x Time repeated measures ANOVA was conducted for GH, PRL, $T_3$, $T_4$, and systolic blood pressure. No significant interactions for GH were found. Only one of the main effects, time, $F(4, 60) = 14.58$, $p < .01$, was significant (see Figure 1). The significant time effect was followed with a Tukey’s least significant difference test. GH levels were significantly higher ($p < .01$) at +15 and +30 than resting and recovery values for all subjects (Figure 1, inset).

No significant interactions or main effects (sex, training, and time) were observed for PRL (see Figure 2). No significant interactions were found for $T_3$. There was a significant time effect, $F(4, 64) = 5.09$, $p < .01$ (see Figure 3). The $T_3$ concentrations at 0, +15, +30, and R30 were all higher ($p < .01$) than the -30 concentrations; however, 0 (min) concentrations were not significantly different than +15 and +30 concentrations for $T_3$.

No significant interactions were found for $T_4$. There was a significant time effect, $F(4, 64) = 3.68$, $p < .01$. The $T_4$ concentrations at 0, +15 were significantly higher than -30, (see Figure 4); however, 0 (min) concentrations were not significantly different than +15 and +30 concentrations for $T_4$.

No significant interactions were found for systolic blood pressure, but a significant time effect, $F(3, 48) = 100.94$, $p < .01$, was found. Mean systolic blood pressure for all subjects rose significantly at +10 and +20 (Figure 5).

Discussion

A significant rise in GH, $T_3$, and $T_4$ occurred over time for all subjects, whereas PRL increases were not significant. The absence of any gender effects, training
effects, or interaction indicated that GH, PRL, T₄, and T₃ responses were similar for the subjects regardless of gender or training status. It has been suggested that the response of growth hormone to exercise is associated with the training level of the individual (Bunt et al., 1986; Hartley et al., 1972; Rennie & Johnson, 1974); however, results are conflicting. Some investigators have found that exercise at 60% of maximal oxygen consumption (VO₂ max) produced higher GH concentrations in trained men relative to untrained men (Bunt et al., 1986). To the contrary, other investigators have reported higher GH concentrations in untrained men in comparison to trained men after cycling at 100% VO₂ max; no GH training effect was observed at 75% of VO₂ max (Hartley et al., 1972). There were no significant training differences found for GH in the present study. In contrast to Wheeler, Wall, Belcastro, and Cumming (1984), no significant differences were detected between the resting PRL concentrations of trained and untrained men in the present study. Perhaps a higher intensity would have resulted in a greater PRL response.

It has been shown that reduced blood glucose and insulin concentrations in men enhanced the increase in GH and PRL concentrations following submaximal exercise (Johannessen, Hagen, & Galbo, 1981). Additionally, GH appeared to be more sensitive than PRL to changes in glucose and insulin. However, it has been demonstrated that when insulin concentrations are elevated by infusion during prolonged exercise, GH concentrations are not reduced (Galbo, Holst, & Christensen, 1979). Although insulin was not measured in the present study, it is not likely that reduction of insulin concentrations in this study caused GH release. GH concentrations were elevated at 15 min, well before insulin levels have been shown to be significantly reduced from exercise at 60% of VO₂ max (Gymleberg, Rennie, Hickson, & Holloszy, 1977). In addition, conditions that do not produce insulin and glucose reduction (e.g., surgery) are associated with elevation of GH (Noel, Suh, Stone, & Franz, 1972).

It was recently shown that GH will rise in response to submaximal ergocycling with no change in PRL (Barreca et al., 1988); however, the workload, which was not clearly defined, appeared to be lower than that of the present study. Trained subjects in the same study showed elevated PRL concentrations during an exhaustive exercise bout, whereas untrained subjects did not; however, the authors conducted a large series of t-tests, which may have accounted for some of the significant findings.

Bunt et al. (1986) found greater GH levels in women than in men at rest but no gender effect for treadmill running at 60% of VO₂ max. Although the exercise intensity (which was approximately 70% of VO₂ max) and exercise heart rates (Kraemer et al., 1989) in the present study were higher than in the study by Bunt et al. (1986), no gender effects were found for GH or PRL.

Rectal temperature, reported in our earlier study (Kraemer et al., 1989), was significantly elevated, with concomitant increases in GH but not PRL. It has been shown that exercise in the heat is associated with elevated GH levels (Favela, Hagen, & Mager, 1985). We and others (Karagiorgos et al., 1979; Kraemer et al., 1989) have found no significant correlation between rectal temperature and GH concentrations; however, GH release in response to elevated body temperature may occur at different temperatures for each individual, and the amount of GH release may not be a graded temperature response.

There is evidence that submaximal exercise in the heat may be associated with inhibited production of T₃ with a concomitant elevation of rT₃ concentrations (Epstein et al., 1979). Although exercisers in the present study ran in room air temperature conditions, their workload was high enough to elevate rectal temperature.

Figure 4. Alteration of total thyroxine concentrations before, during, and after a 30-min run at 80% of maximal heart rate. Data represent M ± SEM, n = 5 for each group. The left and right panels depict T₃ concentrations in males and females, respectively. Training status is depicted by closed symbol (trained) and open symbol (untrained).

Figure 5. Alteration of mean systolic blood pressure before, during, and after a 30-min run at 80% of maximal heart rate. Data represent M ± SEM, n = 5 for each group. The left and right panels depict systolic blood pressures in males and females, respectively. Training status is depicted by closed symbol (trained) and open symbol (untrained).
to levels similar to those in the investigation by Epstein et al. (1979). The T₃ and T₄ concentrations in the present study were not significantly higher at 15 and 30 min of exercise than the 0-min samples. This is in agreement with a previous study (Premachandra, Winder, Hickson, Lang, & Holloszy, 1981) in which it was demonstrated that a brief strenuous swimming or moderate cycling exercise had a very small effect or no effect on thyroid hormone concentrations after accounting for exercise-induced hemoconcentration.

In the present study elevated resting (time 0) T₃ and T₄ levels were observed for males and females, trained and untrained. It has been shown that anticipation of muscular exercise may stimulate the pituitary-thyroid axis (Galbo, 1983), which may explain changes observed in resting concentrations of T₃ and T₄.

Although the exercise workload in the present study cannot be precisely quantified in terms of percentage of VO₂max, the very strong relationship between exercise and heart rate has long been established. Using this relationship, the workload of the present study would correspond to 70 ± 8% VO₂max (Taylor, Haskell, Fox, & Blackburn, 1969).

In conclusion, a 30-min run at 80% of maximum heart rate is associated with higher concentrations of GH but not PRL, T₃, and T₄. Neither training state nor gender affected the aforementioned results. This descriptive study sets the stage for future research to examine the metabolic consequences and interactions of these hormonal changes in response to running. The net hormonal effects in response to exercise depend on many factors, including complex interactions with other hormones, hormonal concentrations in the blood, cell receptor up-regulation or down-regulation, and clearance rates.

References


Authors' Notes

We acknowledge the technical help of Sharon Andrus in the radioimmunoassay laboratory at Texas Tech University Health Sciences Center in Amarillo, TX. We are also indebted to Dr. Jim Woodyard, Director of Killgore Research Center, West Texas State University, Canyon, TX, for support and assistance. This research was funded by an organized research grant from the Killgore Research Center at West Texas State University in Canyon, TX. Address all correspondence to R. R. Kraemer, Exercise Physiology Lab, Dept. of HPE, Southeastern Louisiana University, Hammond, LA 70402.