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Effects of Intensive Training on Prolactin Responses to Submaximal Exercise in Males

A.C. HACKNEY, R.L. SHARP, W.S. RUNYAN, and R.J. NESS

The purpose of this study was to determine if serum prolactin responses to submaximal exercise were affected by 8 weeks of intensive training (5 d/wk, 90 min/d 65-200% VO_{2}max). Nine males performed 90 minute continuous exercise bouts (cycle ergometry; 65% VO_{2}max) at the end of 1, 4, and 8 weeks of training. Blood samples were obtained pre-training, and pre-, post-exercise. Significant differences were not seen in pre- and post-exercise prolactin levels at weeks 1 and 4. However, at week 8 the post-exercise prolactin was significantly greater than the pre-exercise levels (6.8 ± 0.9 vs 3.8 ± 1.0 ng/ml−1, P<0.05). When post-exercise serum prolactin levels were expressed as a percentage of pre-exercise concentrations, changes of −30.6%, +101.1%, and +233.3% were observed at weeks 1, 4, and 8, respectively. Baseline prolactin levels (pre-exercise) were found to decrease significantly from pre-training and week 1 to week 8 (12.3 ± 1.2 and 11.2 ± 1.1 vs 3.8 ± 0.9 ng/ml−1, respectively). The findings suggest intensive training results in a relative augmentation of the post-exercise prolactin response; however, this effect seems to be due primarily to the training induced lowered resting prolactin levels.

INDEX DESCRIPTORS: Hormones, Aerobic-anaerobic Training, Stress

Serum prolactin has been shown to increase significantly with submaximal and maximal exercise in both men and women (Noel et al., 1972; Brisson et al., 1980; Galbo et al., 1981). Brisson and associates have found that, in women, the magnitude of the prolactin response with exercise is associated with training (Brisson et al. 1980). That is, trained females have a greater prolactin response to submaximal exercise than do untrained females. Such a response has not been shown in males. However, putative challenge studies comparing trained and untrained males have shown significantly greater prolactin release at rest in trained males (Hackney et al., 1987; Smallridge et al., 1985). This finding has also been reported for trained females (Boyden et al., 1982). The purpose of this study was to examine the effects of 8 weeks of intensive training on the prolactin responses to submaximal exercise in males.

MATERIALS AND METHODS

Nine moderately-trained young males (22.0 ± 0.7 yr, mean ± SE) gave informed consent for participation in their study. Their physical characteristics were: height, 178.6 ± 2.0 cm; weight, 74.7 ± 2.5 kg; and percentage body fat, 10.4 ± 1.4% (hydrostatically determined). The training involved 5 days a week of cycle ergometry and was designed to mimic that typically conducted by competitive athletes. The sessions consisted of both high-intensity (70% - 200% of maximal oxygen uptake [VO_{2}max] interval (4 days/week) and moderate-intensity (65% VO_{2}max) continuous (1 day/week) exercise. The total duration of each training bout was 90 minutes per day. The interval sessions were partitioned into 70% aerobic, 20% anaerobic, and 10% sprinting training. Intensities for each of these components were set at 70% VO_{2}max for aerobic training, 85-100% VO_{2}max for anaerobic training, and 200% VO_{2}max for sprint training. To assure an adequate training stimulus, each subject's VO_{2}max was reassessed and new training workloads prescribed at 2-week intervals throughout the study. Due to the training, the VO_{2}max values of the subjects increased from 50.7 ± 1.4 ml·kg⁻¹·min⁻¹ at pre-training to 57.0 ± 1.5 ml·kg⁻¹·min⁻¹ at 8 weeks. No significant changes were observed in any of the physical characteristics.

At the end of weeks 1, 4, and 8, fasting (8 hours after last meal) blood samples were withdrawn via veni-puncture immediately before and after the 90 minute (65% VO_{2}max) continuous exercise bouts. Sowers et al. (1977) have suggested blood sampling via veni-puncture may produce an endocrine "stress response" in subjects. However, all our subjects were knowledgeable of the sampling procedure and had had veni-punctures on numerous occasions. Therefore, it was felt such a "stress response" was unlikely to present a confounding effect on results. Each subject's 90 minute exercise bouts took place on the same day, near the end of each week and began at the same time of day (afternoon). Activity and dietary habits were replicated for the 24 hours prior to each 90 minute ride. Prior to the study, a pre-training blood sample (8 hours after the last meal) was taken at the same time and day of the week as the start of the 90 minute exercise bouts. This served as a pre-training, rest control. Only a single sample was taken as unpublished work from our laboratory had suggested afternoon prolactin concentrations are relatively stable in males.

Serum was separated from all blood samples and stored at −80°C, and later analyzed for prolactin (in triplicate) by using a double antibody radioimmunoassay (RIA) procedure (Clinical Assays Inc., Cambridge MA). For the RIAs the within-assay coefficients of variation (CV) were less than 10%, while the between-assay CV was 11%. Results were statistically analyzed with repeated measures analysis of variance. Specific mean comparisons were made with the Turkey post hoc procedures (Winer, 1971).

<table>
<thead>
<tr>
<th>Table 1. Changes in pre- and post-exercise prolactin concentration (mean ± SE) throughout the 8 weeks of training.</th>
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<td><strong>Time</strong></td>
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RESULTS

Table 1 presents the prolactin concentrations pre-training, and pre-, post-exercise for weeks 1, 4, and 8. All values were found to be within the normal range for the RIA procedure used, as reported by the company (Clinical Assays Inc.).

During week 1, prolactin levels decreased non-significantly pre- to post-exercise (p = 0.12), while at week 4 no change was observed. However, at week 8 the mean post-exercise concentration was significantly greater than the pre-exercise concentration (p<0.05). Furthermore, the week 8 mean resting (pre-exercise) prolactin concentration was significantly lower than the pre-training and week 1 pre-exercise concentrations (p<0.05).

All post-exercise prolactin concentrations were also expressed as the percent change from the respective pre-exercise concentration (post-exercise/pre-exercise × 100) for weeks 1, 4, and 8. Statistical analysis indicated the prolactin change at 8 weeks (mean ± SE; 233.3 ± 101.1%) was significantly greater than at week 1 (−30.6 ± 22.1%); however, the week 4 (+101.1 ± 79.1%) change was not significantly different from either week 1 or 8.

DISCUSSION

Our findings suggest that both resting prolactin levels and the prolactin responses to prolonged submaximal exercise are affected by 8 weeks of intensive exercise training. When examining the relative changes (pre- to post-exercise) it appears training produces an augmented prolactin response to exercise. However, if absolute hormonal concentrations are considered this observation must be qualified. That is, the absolute concentration changes indicate training produces lowered resting prolactin levels, but did not significantly affect the absolute post-exercise response. Boyden et al. (1982) has reported a lowering of resting prolactin levels in women when they are subjected to endurance training (i.e., marathon training). Furthermore, Brisson et al. (1980) has shown a greater prolactin response (percentage change) to submaximal exercise in women with athletic histories versus women with non-athletic histories. However, we are unaware of any previous study suggesting that intensive training alters the resting or exercise responses of prolactin in males.

The finding of lowered pre-exercise prolactin levels agrees with resting endocrine changes typically associated with trained individuals. Resting levels of epinephrine, norepinephrine, cortisol, and testosterone have all been reported to be lower following a training program (Galbo, 1983; Terjung, 1977). The mechanism for the reduction in the pre-exercise, resting prolactin concentrations currently observed is uncertain. Boyden et al. (1982) has proposed that the lowered resting prolactin levels with training may be due to an enhanced post-exercise dopaminergic influence and/or increased short-loop feedback (prolactin-prolactin) sensitivity. We concur with this view; however, from the present data we can only speculate about an exact mechanism. Obviously, more research is necessary to elucidate the cause of this change.

It is generally accepted that most acute endocrine responses to exercise (both absolute and relative) are attenuated by exercise training (Terjung, 1977). The present findings of an increased relative prolactin response at 8 weeks is at variance with this belief. The concentration of hormones in the blood are mainly affected by: 1) plasma volume shifts, 2) changes in metabolic clearance, and/or 3) hormonal production. Plasma volume shifts could not account entirely for the observed changes in prolactin concentrations; our estimates of hemococoncentration (van Beaumont, 1972) showed only small acute shifts occurred (4 - 8.5% decreases), and the effects lessened over the 8-week period. It has also been demonstrated that training results in an improved exercise hepatic blood flow, thereby increasing the metabolic clearance of hormones during exercise (Galbo, 1983). Therefore, an enhanced prolactin production seems a more likely explanation for our findings. We speculate the mechanism of this increased production may be via enkephalin-mediated processes. That is, enkephalins are known to stimulate prolactin secretion (possibly via disruption of the dopaminergic inhibition of prolactin secretion), and exercise increases the circulating levels of this opiate (Grossman et al., 1981; Grossman and Surton, 1985). Whether circulatory enkephalin levels directly represent those within the adenohypophysis is uncertain, but the two would seem to be highly related.

In conclusion, intensive combined aerobic-anaerobic training in males would seem to result in an augmented relative prolactin response to prolonged submaximal exercise and a lowering of resting prolactin levels. Because of our limited sampling size and collection protocol, the mechanism mediating these effects as well as their physiological significance currently remains unclear.

REFERENCES


