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**The effects of different run training programs
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adrenocorticotropin and cortisol
to maximal treadmill exercise**



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Running Head: *beta*-endorphin, ACTH and cortisol responses

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ABSTRACT

The purpose of this study was to examine the effects of three different run training programs on plasma responses of Beta-endorphin (β -EP) adrenocorticotropin (ACTH) and cortisol to maximal treadmill exercise. Subjects ($n=30$) were randomly assigned to one of three training groups: sprint intervals (SI), endurance (E) or combination (C). Training was monitored for 10 weeks and maximal treadmill exercise tests were administered pre- and post training. Blood samples were obtained before, immediately after, and 5 and 15 min following the maximal exercise tests. All groups significantly ($p<0.05$) increased maximal oxygen consumption values at 8 and 10 wk of the training period. Significant exercise-induced increases in plasma β -EP, ACTH, cortisol and blood lactate were observed for both pre- and post-training tests. The SI group demonstrated significant post-training increases in β -EP, ACTH, cortisol and peak 5 min post-exercise blood lactate concentrations in response to maximal exercise. No training-induced hormonal changes were observed for the E group. The C group exhibited significant post-training decreases in plasma β -EP, ACTH and blood lactate concentrations in response to maximal exercise, while resting and post-exercise plasma cortisol measures were significantly increased in the post-training test. These data suggest that different run training programs produce differential effects on plasma β -EP, ACTH and cortisol in response to maximal exercise, and these responses may be linked to anaerobic metabolic factors.

Key words: endogenous, opioid peptides, anaerobic, maximal oxygen consumption, proopiomelanocortin blood lactate

Previous studies have generally established that an acute bout of exercise stimulates hormonal responses in the hypothalamic-pituitary-adrenal axis. A wide range of studies have reported acute increases in plasma concentrations of Beta-endorphin (β -EP), adrenocorticotropin (ACTH) or cortisol in response to high intensity submaximal, maximal or supramaximal exercise (2-8, 10, 14-23, 27-32). To date, these studies have only examined acute exercise responses of either one or two of these hormones.

Cross-sectional investigations have tried to discern possible adaptive changes in these hormonal responses to exercise training. No differences have been demonstrated between trained and untrained subjects for relative submaximal or maximal exercise responses of plasma β -EP and ACTH (17, 31). However, lower hormonal responses to exercise have been observed in trained subjects when evaluated in absolute terms of oxygen consumption (31). Cross-sectional data from Farrell et al. (17) demonstrates that endurance trained subjects produce significantly higher plasma concentrations of β -EP and ACTH than untrained subjects in response to a treadmill exercise intensity of 110% of $\dot{V}O_{2\max}$. They suggest that this may represent a possible adaptive mechanism to extreme levels of exercise stress.

Longitudinal studies have provided conflicting results concerning training induced alterations of these hormones. Carr et al. (4) have reported that endurance training facilitates the exercise induced responses of β -EP and ACTH. In contrast, Buono et al. (3) recently demonstrated a significant reduction in the ACTH response to an absolute submaximal exercise intensity following a 12 wk endurance training program. Other studies have been unable to demonstrate any training related changes (8, 26). To our knowledge, no study has examined plasma β -EP and ACTH and cortisol maximal exercise responses to training. Due to the need for more longitudinal training data, this study was undertaken. It was our hope that further insights

regarding possible adaptive changes in the hypothalamic-pituitary-adrenal axis, due to exercise training, might be realized. Thus, the primary objectives of this study were to: 1) examine the acute exercise responses of plasma β -EP, ACTH and cortisol to maximal exercise and 2. evaluate these maximal exercise-induced hormonal response patterns consequent to three different run training programs.

METHODS

Initially, thirty healthy active male and female volunteers were randomly assigned (balanced gender design) to one of three training groups. Each subject gave informed written consent to participate in the study. Subjects were not taking any medications and had no history of any endocrine disorders. Due to attrition, there was an unequal number of subjects in each group at the end of the training period. Subject characteristics were as follows (mean $\pm$ 1 SD): sprint interval group (SI) [n=8], age 26.5 ± 5.0 yrs, height 172.8 ± 7.6 cm, and weight 70.1 ± 9.4 kg; endurance group (E) [n=10], age 23.1 ± 4.1 yrs, height 172.4 ± 11.5 cm, and weight 68.1 ± 13.6 kg; combined group (C) [n=7] age 21.9 ± 3.1 yrs, height 167.0 ± 9.9 cm, and weight 69.1 ± 11.6 kg. No significant differences were observed between groups for these physical characteristics.

A ten week training program was undertaken by each subject. All subjects were familiarized with all training and monitoring procedures prior to the start of training. All training sessions were individually monitored on a measured track and recorded. The training programs used for each group were as follows: the SI group trained 3 days $\cdot$ week $^{-1}$ and performed two sets of interval sprints separated by 5 min of rest. Each set consisted of four 20 sec maximal interval sprint bouts of exercise with 1 min rest between intervals. Heart rates were monitored before and after each sprint and mean exercise intensity after the second set was determined to be 185.25 ± 3.1

beats $\cdot$ min $^{-1}$ which equated to 96.7% of their directly determined maximal heart rate; the E group trained 3 days $\cdot$ week $^{-1}$ and ran as far as they could in 30 min. Subjects were encouraged to increase this distance over the training period. Heart rate was again monitored before and after each run and the mean exercise intensity at 15 min into run was determined to be $80.0 \pm 1.9\%$ of their treadmill $\dot{V}O_{2\max}$; the C group trained 6 days $\cdot$ week $^{-1}$ and performed the exercise protocols of the SI and E groups on alternate days. Heart rate determination during the exercise sessions showed that the mean exercise intensities were 182.57 ± 3.21 beat $\cdot$ min $^{-1}$ which equated to $93.4 \pm 2.4\%$ of their directly determined maximal heart rate and $79.7 \pm 4.7\%$ of their treadmill $\dot{V}O_{2\max}$ for SI and E exercise sessions respectively. Exercise intensities are reported from mid-exercise values to avoid any end of run training kicks which may inflate exercise intensities.

Prior to any testing, subjects were familiarized with all experimental procedures. Each subject performed an incremental $\dot{V}O_{2\max}$ test on a motor driven treadmill (6). Exercise intensities for these tests were pre-determined for each subject so that volitional exhaustion occurred in the same amount of time (≈ 5 min), after a 5 min warm up at 40-50% of $\dot{V}O_{2\max}$. Thus, the time of the test over training and between subjects was similar. This was considered important to help partial out durational effects on hormonal responses to the incremental maximal treadmill exercise test. Maximal oxygen consumption ($\dot{V}O_{2\max}$) was determined using an open circuit semiautomated sampling system (35). Oxygen uptake was monitored every 30s during the test. The $\dot{V}O_{2\max}$ was determined according to criteria previously used (25, 33). During the test heart rate was monitored via ECG.

All testing was performed at identical times for each subject. Prior to the test subjects refrained from food for 8 hr and exercise for 24 hr prior to the test. Blood samples were obtained from a superficial antecubital arm vein utilizing a syringe and three way stop-cock fitted to an indwelling teflon cannula. Pre-exercise blood sampling occurred following a 20 min equilibration period. Immediate post-exercise [within the first minute] (IP) and 5 and 15 min post-exercise samples were also taken. Blood was collected and gently mixed in pre-cooled plastic syringes containing an appropriate preservative (EDTA, 7.2 mg/5 ml whole blood). Blood to be used for subsequent radioimmunoassay (RIA) was centrifuged at $2000 \times g$ at 4°C for 15 min and plasma was immediately frozen to -80°C until analyzed with only one thaw prior to analysis. Hemoglobin and hematocrit were determined in triplicate via a 5880 micro Coulter Counter. Whole blood lactate was measured in triplicate using a 23L-Lactate Analyzer (Yellow Springs Inc.). Plasma volume shifts were calculated from the changes in hematocrit and hemoglobin (13).

Plasma β -EP, ACTH and cortisol were determined using RIA procedures. All samples (run in duplicate) for each RIA were measured in the same assay to avoid run to run assay variations. Furthermore, all sample identification was decoded only after analyses were completed (i.e. blinded analysis procedure). Determinations of different plasma immunoreactivity values were accomplished with the use of a Beckman 5500 gamma counter and on-line data reduction system. The RIA used for plasma β -EP has been previously described in detail (28). Cross-reactivity with Beta-lipotropin is less than 5%. The intra-assay coefficient of variation was less than 3.9%. Plasma ACTH was measured by a double antibody RIA (Diagnostic Products Corp., Los Angeles, CA), sensitive to $1.54 \text{ pmol} \cdot \text{L}^{-1}$. Intra-assay coefficient of variation was less than 4.2%. Plasma cortisol was measured using a solid phase RIA (Diagnostic Products Corp., Los Angeles, CA) and was sensitive to $8.28 \text{ nmol} \cdot \text{L}^{-1}$. The intra-assay coefficient of variation was less than 3.8%.

Statistical evaluation of the data was accomplished using a three-way analysis of variance (ANOVA) with repeated measures or 3x2 ANOVA (groups x pre-post) where appropriate. Subsequent Tukey post-hoc tests were utilized to determine pair-wise differences. Statistical significance was chosen as $p < 0.05$.

RESULTS

Table 1 presents the effects of the three different training programs on $\dot{V}O_{2\max}$ values. Pre-training values were not significantly different among groups. All three groups significantly increased $\dot{V}O_{2\max}$ above pre-training values at 8 and 10 wks, and there were no significant group differences at these measurement time points. Also, no significant differences were observed for total treadmill test time between tests or groups.

The pre-and post-training plasma β -EP responses to maximal, treadmill exercise are presented in Figure 1. All three training groups demonstrated significant exercise-induced elevations in plasma concentrations of β -EP at all three recovery time points both pre-and post-training. Significant increases pre- to post-training were observed at IP and 5 min post-exercises for the SI group. No training-induced hormonal changes were observed for the E group. The C group demonstrated a significant pre- to post-training decrease at 5 min post-exercise.

Figure 2 shows the pre- and post-training plasma ACTH responses to maximal, treadmill exercise. Again, all three groups experienced significant exercise-induced increases in plasma ACTH at all three post-exercise time points, both pre- and post-training. The SI group showed significant pre-to post-training increases at the IP timepoint. The E group failed to demonstrate any training-induced hormonal changes. The C group demonstrated a significant pre- to post-training decrease IP exercise.

The pre- to post-training plasma cortisol responses to maximal treadmill exercise are presented in Figure 3. Significant pre-training exercise-induced elevations in plasma cortisol concentrations were observed at 5 and 15 min post-exercise for the SI group and at 15 min post-exercise in the post-training tests. Pre- to post-training changes were observed for the SI group at the pre- and 5 min post-exercise measurement time points. Exercise-induced increases were observed at 15 min post-exercise during both pre- and post-training testing but no training related changes were observed for the E group. The C group followed a pattern similar to the SI group, with exercise-induced increases occurring post-exercise, at 5 and 15 min during pre-training tests and at IP, 5 and 15 min post-exercise during post-training tests. Training-induced increases were also demonstrated at the pre-exercise and IP measurement timepoint for the C group.

Blood lactate responses 5 min following maximal treadmill exercise are shown in Figure 4. All three groups demonstrated significant increases at 5 min post-exercise, during both pre- and post-training testing. Post-training, the SI group demonstrated a significant increase in blood lactate concentration 5 min post-exercise. No changes in 5 min post-exercise blood lactate responses were observed for the E group pre- to post-training. The C group demonstrated a significant decrease pre- to post-training for 5 min post-exercise blood lactate concentrations.

No differences were observed between pre- and post-training tests for the percentage change in plasma volume. The values for pre- and post-training respectively, were (mean $\pm$ SD): SI = $(-9.13 \pm 3.41\%$ and $-8.92 \pm 5.42\%)$, E = $(-10.11 \pm 3.92\%$ and $-9.52 \pm 5.1\%)$, C = $(-10.12 \pm 4.21\%$ and $-9.87 \pm 4.92\%)$.

DISCUSSION

The primary finding in this study was that each training program resulted in a somewhat different plasma hormonal response pattern to maximal exercise. To our knowledge this is the first investigation to demonstrate differential training effects on plasma concentrations of β -EP, ACTH and cortisol. A greater understanding of the exercise-induced modulation of these peptides is important in attempting to define their exact physiological functions.

Higher intensity exercise (i.e. $>70\%$ of $\dot{V}O_{2\max}$) has been typically shown to cause acute elevations of β -EP, ACTH and cortisol (2,5,8,15,17,21,34). Consistent with previous studies, all three groups in the present study exhibited acute increases in response to maximal treadmill exercise during both pre- and post-training tests. Furthermore, because exercise duration was the same during pre- and post-training testing, the effect of time on hormonal response patterns was controlled. Exercise time, independent of intensity, is known to influence the response of several hormones in the hypothalamic-pituitary-adrenal axis (21,34). The exact mechanism(s) responsible for these exercise-induced peptide increases remains unknown, but the increases observed were greater than could be explained by plasma volume shifts.

The 10 wk training programs utilized by the SI and E groups each resulted in significant increases in $\dot{V}O_{2\max}$ values, however the hormonal response patterns following training were different. Training specificity may be important in influencing hormone responses (21). In this regard, Farrell et al. (15) have suggested a possible anaerobic influence upon the release of peptides from the proopiomelanocortin (POMC) precursor. In this study, the SI exercise sessions caused significant elevations in blood lactic acid concentrations ($10.6 \pm 0.7 \text{ mmol} \cdot \text{L}^{-1}$). Furthermore, the SI group post-training tests demonstrated significantly higher post-exercise increases in blood

lactic acid levels compared to pre-training values. This was concomitant with the post-exercise increases in β -EP, ACTH and cortisol in the post-training tests for these subjects. Conversely, the E group observed no changes in the 5 min post-exercise blood lactic acid concentrations, nor any alterations in plasma β -EP, ACTH or cortisol, with training. Recently, Buono et al. (3) reported a blunted ACTH response to absolute submaximal exercise following endurance training. Our results support Farrell et al. (17) and clearly demonstrated that a blunting mechanism is not operational during maximal exercise following endurance training. On the other hand, our data support the hypothesis that a program of aerobic exercise training, which also includes a substantial anaerobic component, may influence β -EP, ACTH and cortisol release mechanisms in response to maximal exercise.

The C group utilized both modes of exercise (i.e. sprint intervals and endurance running) in their training program. An increase in $\dot{V}O_{2\max}$ occurred following training along with a significant decrease in 5 min post-exercise blood lactic acid values in response to the maximal exercise. β -EP and ACTH also demonstrated training-induced decreases following maximal exercise, while cortisol increases were observed. The mechanisms explaining these responses remain speculative. However, reductions in blood lactic acid accumulation have been linked to chronic depletion of muscle glycogen stores (9,18), and fasting has been shown to increase resting and exercise plasma cortisol responses (22). Thus, one might speculate that the observed increases in exercise-induced cortisol production concomitant with decreases in maximal blood lactic acid levels, could have been a result of an altered nutritive state of the muscle following training in the C group.

Due to the training intensity and frequency undertaken by the C group, it appears plausible that some degree of overtraining may have been present and, therefore, partially contributed to the hormonal responses observed (34). A previous study has demonstrated the plasma testosterone and cortisol and testosterone/cortisol ratios are significantly altered during intense training and these changes may indicate an "overtrained" state (1). The reductions observed in β -EP and ACTH in this study may indicate an overtrained state, and supports the need for future studies to examine this hypothesis.

In summary, this study examined the effects of three different run training programs on plasma β -EP, ACTH and cortisol release patterns in response to maximal treadmill exercise. Our data suggests that these training programs had differential effects on these hormonal release patterns and support the hypothesis that anaerobic factors may influence β -EP and ACTH release. Furthermore, mechanisms related to possible overtraining influences may have contributed to the training-induced hormonal reductions observed in the C group.

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Table 1. The effects of three different training programs on maximal oxygen consumption values ($\text{ml} \cdot \text{Kg}^{-1} \cdot \text{min}^{-1}$).

<u>Group</u>	<u>Pre-training</u>	<u>2wks</u>	<u>4wks</u>	<u>8wks</u>	<u>10wks</u>
Sprint Intervals	49.19 (± 6.50)	51.57 (± 5.17)	51.52 (± 5.60)	53.03* (± 5.60)	52.45* (± 5.38)
Endurance	45.89 (± 7.81)	48.06 (± 7.69)	50.85 (± 7.19)	53.77* (± 7.81)	54.31* (± 8.09)
Combined	50.37 (± 9.14)	51.72 (± 10.48)	50.37 (± 9.15)	55.90* (± 10.43)	57.46* (± 9.00)

Mean $\pm$ SD. * = $p < 0.05$ from corresponding pre-training value.

Figure 1. Responses of Plasma Beta-endorphin to maximal exercise for three different run training programs. + = $p < 0.05$ from corresponding resting value. • = $p < 0.05$ from corresponding pre-training value.

PRE
 TRAINING
 POST
 TRAINING

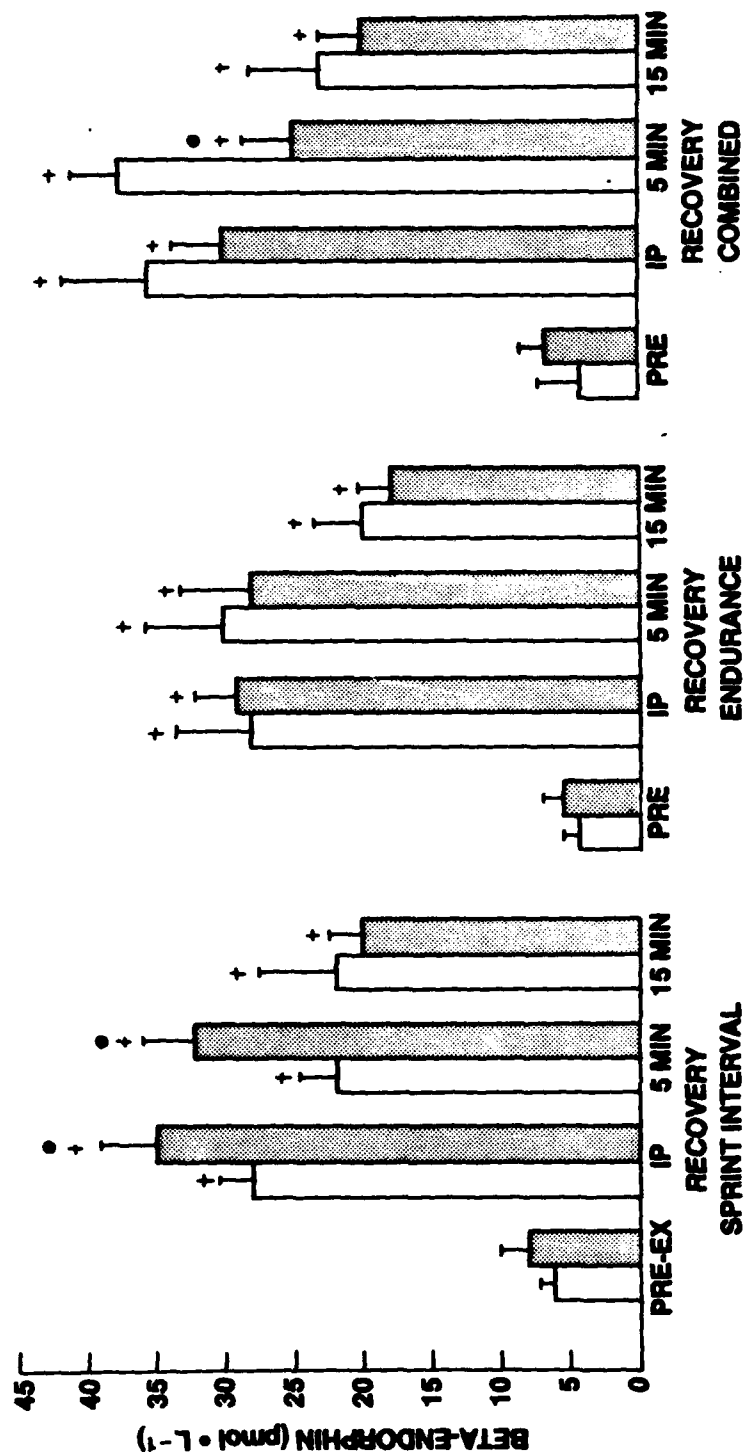


Figure 2. Responses of plasma adrenocorticotropin to maximal exercise for three different run training programs. + = $p < 0.05$ from corresponding resting value, • = $p < 0.05$ from corresponding pre-training value.

ADRENOCORTICOTROPIC HORMONE (pmol • L⁻¹)

PRE TRAINING
POST TRAINING

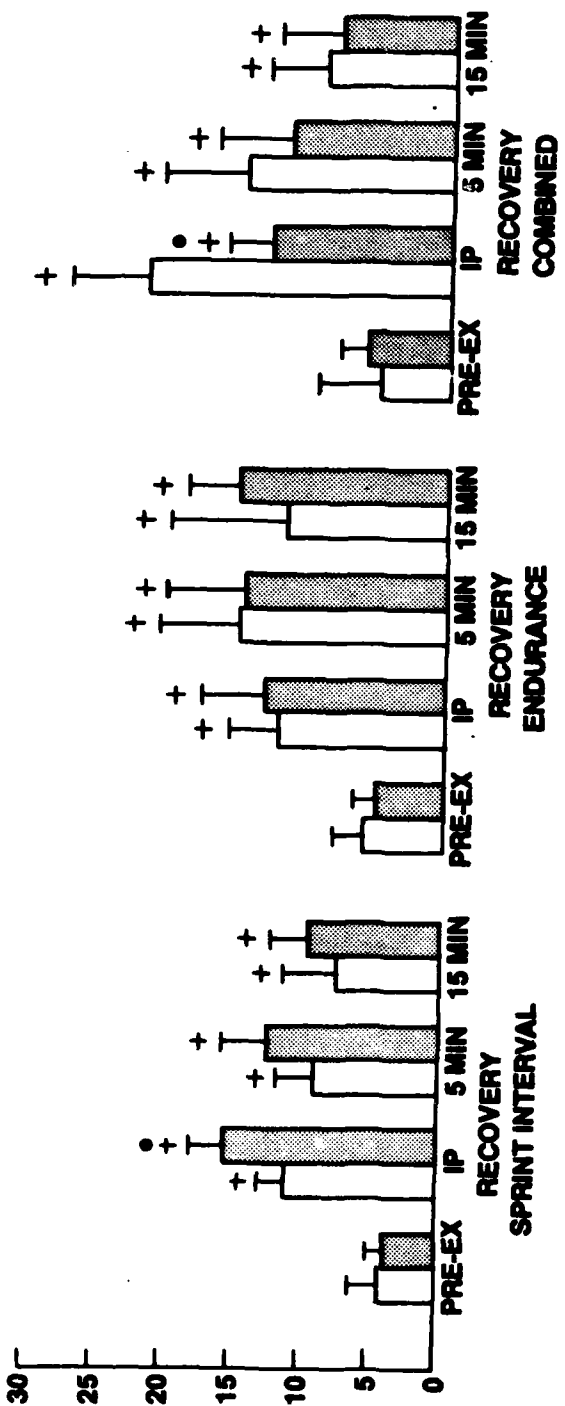


Figure 3. Responses of plasma cortisol to maximal exercise for three different run training programs. + = $p < 0.05$ from corresponding resting value. • = $p < 0.05$ from corresponding pre-training values.

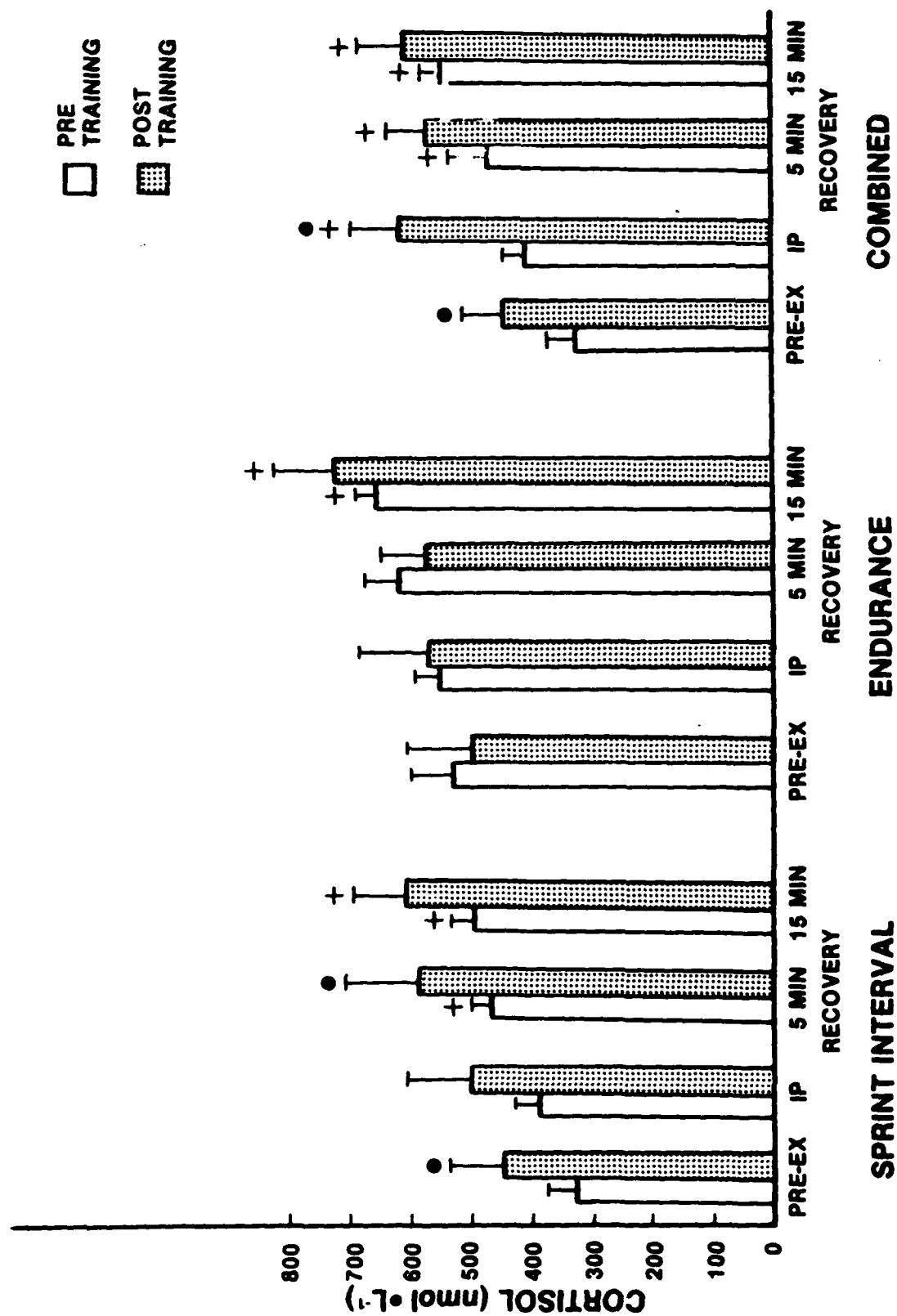
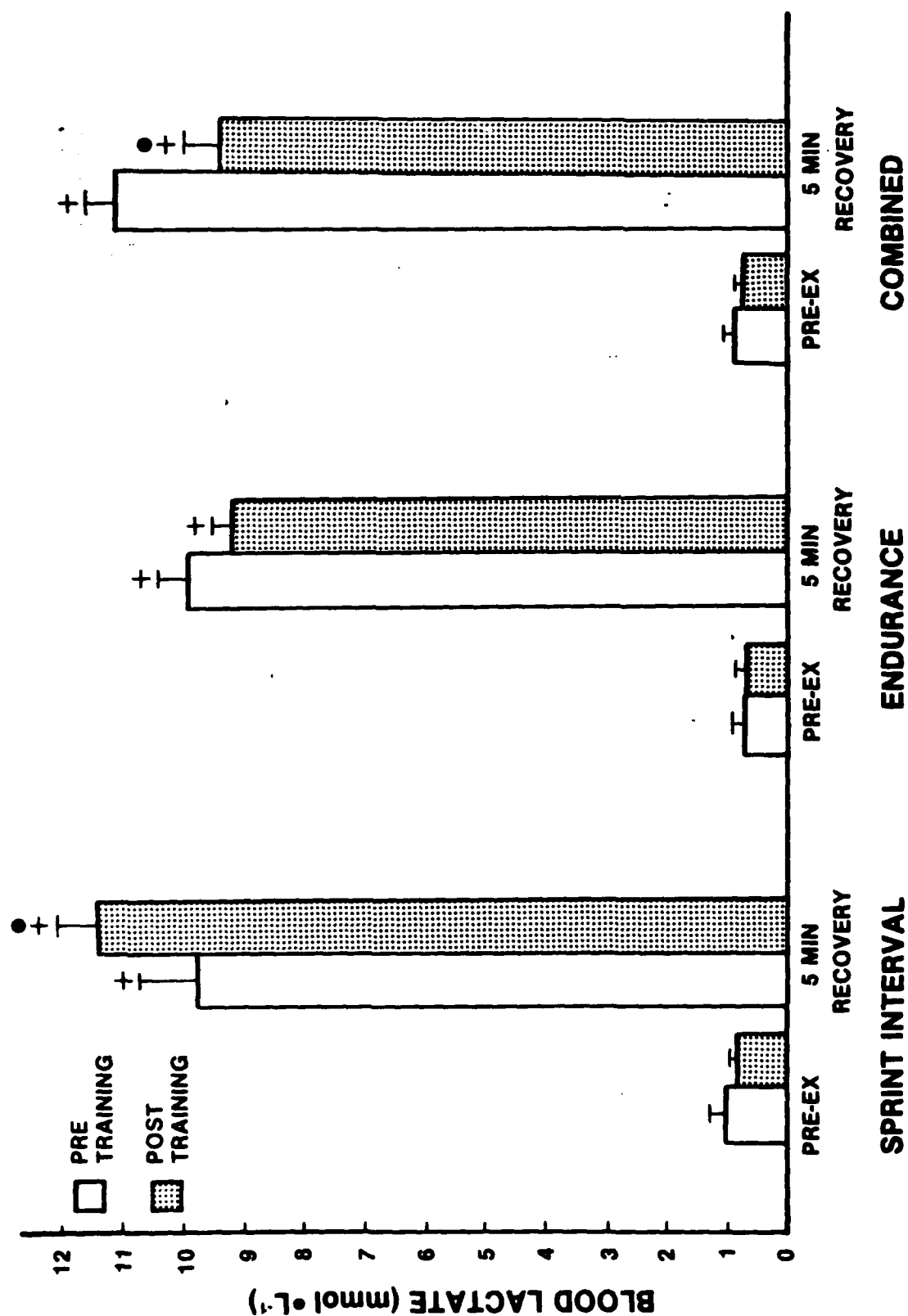


Figure 4. Responses of whole blood lactate to maximal exercise for three different run training programs. + = $p < 0.05$ from corresponding resting value. • = $p < 0.05$ from corresponding pre-training value.



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Disclaimers

The views, opinions, and or findings contained in this report are those of the author(s) and should not be construed as any official Department of the Army position, policy or decision unless so designated by other official documentation.

HUMAN RESEARCH

Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.

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