Resistance exercise decreases β-endorphin immunoreactivity

E. F. Pierce PhD, N. W. Eastman PhD, R. W. McGowan PhD, H. Tripathi PhD*, W. L. Dewey PhD* and K. G. Olson*

Department of Health and Sport Science, University of Richmond, Virginia; and *Medical College of Virginia, Virginia, USA

Previous research investigating the response of plasma β-endorphins (β-EP) to resistance exercise has resulted in equivocal findings. To examine further the effects of resistance exercise on β-EP immunoreactivity, 10 male and 10 female college-age students participated in a series of controlled isotonic resistance exercises. The session consisted of three sets of eight repetitions at 80% of one repetition maximum (1-RM) for each of the following exercises: (1) bench press; (2) lateral pull-downs; (3) seated arm curls; and (4) military press. Blood plasma was sampled both before and after the lifting routine and β-endorphin levels were determined by radioimmunoassay. A Student's t test for paired samples indicated that mean(s.e.) plasma β-endorphin levels after exercise (10.5(1.3) pg β-EP ml⁻¹) were significantly decreased as compared with pre-exercise (control) levels (16.8(1.2), P < 0.05). While the mechanism(s) contributing to the decrease in immunoreactivity is unclear, it may be the result of the synergistic effect of β-EP clearance during rest intervals and changes in psychological states between sampling.

Keywords: Resistance exercise, β-endorphins

Endurance exercise has been consistently shown to increase serum levels of β-endorphin¹⁻⁴. Limited research examining the effects of resistance exercise, however, has resulted in equivocal findings. Elliot et al.⁵ examined the β-endorphin response of resistance trained subjects to an acute bout of isotonic weightlifting exercises. The authors reported a significant increase in plasma β-endorphin response as compared with basal levels following a 45-min bout of isotonic weightlifting exercise. In contrast with these findings, Melchiona et al.⁶ studied the plasma β-endorphin immunoreactivity of both elite athletes and untrained subjects exposed to a high intensity isometric knee extension regimen. Despite profound muscle discomfort as well as significant decrements in strength observed during the exercise, no significant increases in plasma β-endorphin were observed following the exercise. Finally, Petraglia et al.⁷ investigated the effects of acute competition on the plasma β-endorphin responses of elite track and field performers. While athletes participating in running events ranging in distance from 100 m to 10 000 m showed significant increases in plasma β-endorphin levels after their respective events, no significant postexercise increases were reported for athletes competing in the discus event. Finally, Pierce et al.⁸ reported a failure for resistance exercise to produce a significant change in β-endorphin immunoreactivity among US football players.

Generalizations from the studies noted above are further complicated by differences in methodology such as lifting protocol and the duration of recovery intervals. The purpose of the present study was to examine further the effects of a controlled protocol of resistance (weightlifting) on plasma β-endorphin immunoreactivity.

Method

Ten men recreational weightlifters (mean(s.e.) age 20.7(0.56) years; mean(s.e.) weight 79.9(2.2) kg; mean(s.e.) height 182.3(2.5) cm) and ten women basketball players (mean(s.e.) age 19.4(0.41) years; mean(s.e.) weight 68.6(1.7) kg; mean(s.e.) height 176.2(2.0) cm) volunteered to participate in the study. Each subject signed an informed consent document approved by the Human Investigation Committee of the University of Richmond. The participants were briefed on sampling and testing procedures after which blood was sampled from the group by venipuncture, placed on ice, then plasma was separated by centrifugation at 1500 g for 10 min in a refrigerated centrifuge. The plasma was then stored in 1-ml aliquots at −70°C. Immediately after the first venipuncture, each subject began participation in a series of isotonic exercises using Nautilus (Lake Helen, Florida) resistance machines. This equipment was used (as opposed to ‘free’ weights) in order to standardize muscle movements and to control the duration of recovery intervals between exercises. The exercise session consisted of three sets of eight repetitions at 80% of one repetition maximum (1-RM) (or until muscular failure occurred) for each of the following exercises: (1) bench press; (2) lateral pull-downs; (3) seated arm curls; and (4) military press. One repetition maximum (1-RM) for each of the four exercises was determined 24 h before the experimental session at the same time of day to avoid
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diurnal variation. Each exercise was preceded by a progressive warm-up consisting of eight repetitions at 40% and 60% of 1-RM. Recovery intervals between exercises were standardized at 3 min, and completion of the exercises required approximately 45 min.

A second blood sample was obtained immediately after the exercise routine according to the order of the initial sample. β-endorphin was extracted from thawed plasma samples with octadecylsil-silica (ODS-silica) cartridges (Sep Pak C18 cartridges, Waters Associates, Milford, Massachusetts, USA), and eluted with 4 ml of 75% acetonitrile containing 0.1% trifluoroacetic acid. The resulting cartridge extracts were concentrated to dryness in a centrifugal vacuum concentrator, reconstituted in radioimmunoassay (RIA) buffer containing 0.1% Triton X-100, then assayed for β-endorphin by RIA according to the method of Hong et al.9. The RIA buffer consisted of 20 mM sodium phosphate (pH 7.5), 0.15 M sodium chloride, 0.1% gelatin, and 0.01% bovine serum albumin. A polyclonal antiserum raised against human β-endorphin was used in the RIA. The antiserum cross-reacts 100% on a molar basis with β-lipotropin, the immediate precursor of β-endorphin, but does not cross-react with the encephalins, the dynorphins, adrenocorticotropic hormone (ACTH), β-melanotropin and α-endorphin. An iodinated (125I) β-endorphin tracer was used in the RIA. Antiserum bound and free β-endorphin tracers were separated after a 24-h incubation at 4°C by addition of a 2% Norrit A charcoal and 0.1% dextran (T70 dextran, Pharmacia Fine Chemicals, Piscataway, New Jersey, USA) suspension followed by centrifugation. All samples were assayed in duplicate with before-lifting and after-lifting samples assayed together in the same RIA. The RIA was sensitive to 5 pg β-endorphin per sample and the binding of plasma extract dilutions paralleled the binding of β-endorphin standards.

An analysis of variance (ANOVA) with repeated measures were used to detect significant differences between control (pre-exercise) and post-exercise levels of plasma β-endorphins. The alpha level was established at P < 0.05.

Results

Results indicated that mean(s.e.) plasma β-endorphin levels after exercise of 10.5(1.3) pg β-EP ml⁻¹ were significantly decreased as compared with pre-exercise levels (16.2(1.2), P < 0.05). Post-exercise β-endorphin response ranged from a 12.1% increase to a 90.9% decrease compared with pre-exercise levels, with a mean decrease of 33.2% observed for the group.

Discussion

The major finding of the present study was a significant reduction in β-endorphin immunoreactivity following resistance exercise. Although a significant reduction in β-endorphin following resistance exercise has not been previously reported, a failure of resistance exercise to produce a significant increase in post-exercise β-endorphin levels has been reported in previous studies6,7. In addition, previous investiga-

tion in our laboratory has demonstrated a failure on the part of resistance exercise to influence levels of β-endorphin significantly among habitually trained athletes. Analysis of the β-endorphin response to resistance training among trained US football players revealed a non-significant decrease in β-endorphin immunoreactivity after the exercise. This is in contrast to the consistent increase in β-endorphin levels previously reported following bouts of endurance exercise1-4. It is tempting to speculate that the intermittent nature of the exercise bout employed in studies examining the effects of resistance exercise on endorphin immunoreactivity might contribute to a tendency towards a decreased β-endorphin response following exercise. In support of this, Farrell et al.8 suggested that the clearance of plasma β-endorphins after exercise might be affected by the higher cardiac outputs observed in trained individuals. This presumably would influence the delivery of blood to β-endorphin catabolizing tissue during the periods of rest which occur in intermittent exercise bouts. However, despite conjecture regarding this mechanism, previous studies using intermittent exercise such as resistance exercise6 and sprint interval running11 with well trained athletes have reported significant increases in β-endorphin levels in comparison with basal values.

While it may be argued that the failure of resistance exercise to produce a significant increase in β-endorphin immunoreactivity may be related to factors such as clearance during recovery, such a mechanism would not explain the significant post-exercise decrease observed in the present study. While the basis for this decrease remains speculative, previous research has established that environmental and situational factors may significantly influence affect12. As affect and levels of plasma β-endorphins have been suggested to be closely related13,14, it may be argued that the decrease in post-exercise β-endorphin levels may have been a function of a change in affective state and/or a decrease in the perceived threat of study procedures such as blood sampling. In support of this is research which has reported that at least part of the increase in β-endorphin immunoreactivity during exercise may be due to psychological stress associated with the perceived threat represented by the protocol15. In addition, procedures such as venipuncture or the cannulating of exercising subjects to obtain blood samples may also influence β-endorphin response16. One might argue, therefore, that the acclimation of subjects to study procedures such as sample procurement (and the accompanying decrease in anxiety) might influence the β-endorphin response following exercise. However, in an analysis published elsewhere17, we reported the relationship between β-endorphin levels at the respective study conditions (control, post-exercise) and affect for the subjects participating in the present study. Results indicated no significant relationship between β-endorphin levels and either total mood disturbance or profile of mood states (POMS) subscores of tension–anxiety, depression–dejection, anger–hostility, vigour–activity, fatigue–inertia and confusion–bewilderment. It is tempting therefore, to conclude that the significant
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decrease in β-EP immunoreactivity observed in the present study is the result of physiological, rather than psychological factors. Given a significant body of research which has associated the β-endorphin response with psychological factors, however, it is ultimately difficult to ignore the possibility of a synergism of both physiological and psychological factors resulting in decreased β-endorphin immunoreactivity following exercise.

In summary, a significant reduction in β-endorphin immunoreactivity was observed following an acute bout of resistance exercise. This is in marked contrast to the significant increase consistently reported following endurance exercise. Future research investigating mechanisms responsible for the differential response of β-endorphin to resistance and endurance exercise is encouraged.

References

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