The Relationship Between Resistance Exercise Induced Testosterone and Cortisol Responses and Steroid Receptor Phosphorylation

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ABSTRACT

The precise contribution of hormones to resistance training adaptations remains unclear. Recently, resistance exercise (RE) has been shown to change phosphorylation of androgen (pAR) & glucocorticoid receptors (pGR). Examining the relationships between the hormonal responses & steroid receptor phosphorylation may elucidate the role of acute hormonal responses to training adaptations. **PURPOSE**: The purpose of this study was to examine relationships between exercise-induced hormonal responses and pGR & pAR. **METHODS**: Resistance trained (RT) (n = 10; age = 21.3±1.7yrs, ht = 175.8±6.8cm, bodymass = 84.5±13.5kg) & untrained (UT) (n = 9; age = 20.8±3.1yrs, ht = 178.7±8.9cm, bodymass = 81.0±14.0kg) men completed an acute RE session of 6 sets of 10 reps, & 4 sets of 10 reps at 75% 1RM of barbell back squats, & knee extension, respectively. Muscle biopsies were obtained at rest, 10+, 30+, 60+, & 180+ minutes post-exercise & analyzed for total AR, pAR at ser81, ser213, ser515, ser650, total GR, and pGR at ser134, ser211, ser226. Testosterone & cortisol samples were obtained before, & up to 45 minutes post-exercise. Pearson correlations were performed to determine relationships between endocrine responses (area-under-curve [AUC]) & changes in total & phosphorylated AR & GR. Significance was determined at p≤0.05. **RESULTS**: The change in total AR at 180+ was correlated with cortisol (Pooled: r = -0.668, *p* = 0.002) & was strongest in RT subjects (RT: r = -0.767, *p* = 0.010). Cortisol was correlated with pARser81 at 60+ (r = 0.601, *p* = 0.006) & 180+ (r = 0.537, *p* = 0.018). Cortisol was correlated with the change in pARser650 at 180+ (r = 0.724, *p* = 0.018) in RT subjects. In UT the changes in pGRser134 & pGRser226 were correlated at 10+ (r = 0.987, *p* = 0.001) & 30+ (r = 0.943, *p* = 0.001). **CONCLUSION**: Cortisol responses were related to AR content, & changes in phosphorylation at sites regulating AR ligand sensitivity, & AR localization. There was a training status-specific relationship in UT subjects between pGR sites that regulate receptor localization, & GR sensitivity to cellular stress. Individualized cortisol responses are strongly related to AR activity and may explain the discrepancy in studies that solely investigated anabolic hormones & training adaptations, since these relationships also appear to be specific to different training statuses.