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Cortisol is related to acute leukocytosis in maximal but not in hypertrophic dynamic resistance exercise

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ABSTRACT

Introduction. Exercise induces immune changes that are multifactorial and include neuroendocrine factors. Acute resistance exercise is followed by marked increases in adrenaline, cortisol, growth hormone, and other factors that have immunomodulatory effects. The purpose of the present study was to investigate the relationship between leukocytosis and hormone responses to two different resistance exercises, low volume high load (gains in maximal strength, MAX) and high volume medium load (gains in muscle mass, HYP).

Methods. Using a cross-over design twelve healthy men participated in bilateral leg press exercise consisting of 5 sets of 10 RM and 15 sets of 1 RM. The inter-set rest period was 3 minutes for MAX and 2 minutes for HYP. Venous blood samples were taken at baseline, immediately after (P0) and 15 (P15) and 30 (P30) minutes after the exercise. Basic blood count was analyzed using Sysmex KX-21N (TOA Medical Electronics Co., Ltd., Kobe, Japan). Serum cortisol (COR), testosterone (TES), and growth hormone (GH) concentrations were analyzed by an immunometric chemiluminescence method (Immunlite R 1000, DPC, Los Angeles, USA)

Results. Both exercises induced significant acute leukocytosis ($p < 0.001$). Leukocytosis was significantly higher after HYP ($p < 0.01$). COR and TES increased significantly after HYP ($p < 0.01$) but not in MAX. GH increased significantly ($p < 0.05$) in both exercises and stayed elevated at P30 in HYP. There was a significant negative correlation between acute leukocytosis and cortisol at P0 in MAX ($R = -0.622$, $p = 0.031$) but not in HYP $r = 0.287$ ($p = 0.366$). Significant correlations between TES, GH and leukocytes were not observed.

Conclusions. Clearly, manipulation of the rest period and load in resistance exercise alters endocrinal as well as immunological responses. Hypertrophic resistance exercise triggered significantly stronger immunological as well as endocrinal responses. In line with the previous studies (e.g. Kraemer et al. 1996) cortisol did not correlate with leukocytes nor with leukocyte subgroups in HYP. It might be that cortisol acts as an anti-inflammatory agent in MAX, however in HYP leukocytosis appears to be related to additional physiological mechanisms e.g. muscle damage and metabolic demands, which might explain why we did not observe the same in HYP. When considering recovery from resistance exercise the immune system should be monitored in addition to hormones.

