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The aim of the current study was to examine the influence of exercise intensity on systemic oxidative stress (OS) and endogenous antioxidant capacity. Non-smoking, sedentary healthy adult males (n = 14) participated in two exercise sessions using an electronically braked cycle ergometer. The first session consisted of a graded exercise test to determine maximal power output and oxygen consumption (V_{O2max}). One week later, participants undertook 5-min cycling bouts at 40%, 55%, 70%, 85% and 100% of V_{O2max} , with passive 12-min rest between stages. Measures of systemic OS reactive oxygen metabolites (dROM), biological antioxidant potential (BAP), heart rate (HR), V_{O2} , blood lactate and rating of perceived exertion were assessed at rest and immediately following each exercise stage. Significant ($P < 0.05$) differences between exercise bouts were examined via repeated measures ANOVA and post hoc pairwise comparisons with Bonferroni correction. Increasing exercise intensity significantly augmented HR ($P < 0.001$), V_{O2} ($P < 0.001$), blood lactate ($P < 0.001$) and perceived exertion ($P < 0.001$) with no significant effect on dROM levels compared with resting values. In contrast, increasing exercise intensity resulted in significantly ($P < 0.01$) greater BAP at 70% (2427 ± 106), 85% (2625 ± 121) and 100% (2651 ± 92) of V_{O2max} compared with resting levels ($2105 \pm 57 \mu\text{mol Fe}^{2+} / \text{L}$). **The current results indicate that brief, moderate-to-high-intensity exercise significantly elevates endogenous antioxidant defences, possibly to counteract increased levels of exercise-induced reactive oxygen species. Regular moderate-to-high-intensity exercise may protect against chronic OS associated diseases via activation, and subsequent upregulation of the endogenous antioxidant defence system.**

One thing I think this study shows is that exercise is a kind of hormesis, upregulating stress defense mechanisms.