

PSYCHOLOGICAL STRESS EFFECT ON SUPEROXIDE RELEASE IN HUMAN NEUTROPHILS.

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There is evidence that stressful events can modify the immune function, suggesting a relationship between nervous, endocrine and immune systems. Experimental and clinical evidence show that neutrophils play an important role in the mechanism of tissue injury in immune complex diseases through the generation of reactive oxygen species. Our objective was to study the influence of psychological stress on post-graduation students in the capacity of their blood neutrophils to release superoxide anion (SR) when stimulated by nonphagocytosable surfaces. The tests were performed when the students were subjected to the final examination of their thesis. SR was evaluated by the cytochrome-C reduction assay. Neutrophil suspensions were incubated with a *micropore*-filter coated with immune complex for 1h and the absorbance of the supernatant measured. Stress group showed higher SR when compared to control in same research schedule ($P<0,02$). Plasma cortisol levels, evaluated by radioimmunoassay, were significantly higher on stress group ($P<0,02$) when compared to control. This indicates that, under stress situations, the cortisol level might have a modulation effect on the SR by neutrophils. These results gives further reasons for investigating the *in vitro* effects of cortisol on SR, and could provide a better comprehension about the mechanism of development of immune complex diseases and the conditions that modulate their susceptibility and progression.

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