STRESS AND PHAGOCYTOSIS: ROLE OF CORTICOSTERONE AND CATECHOLAMINES IN PHAGOCYTOSIS BY POLYMORFONUCLEAR LEUCKOCYTES

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When homeostasis of an organism is threatened, a stress response is induced with the activation of the Hypothalamic-Pituitary-Adrenal axis that results in the corticosterone secretion (mice) and the activation of the sympathetic nervous system and liberation of catecholamines. In this study, we investigated in mice, the effect of cold stress (4hours at 4ºC) on the phagocytic capacity of blood neutrophils and the action of corticosterone and of catecholamines in phagocytosis mediated by two types of receptors. We found that neutrophils submited to a stress condition showed a decrease in the phagocytosis mediated by Fc? and complement receptors compared with cells from control mice. Experiments in vitro, demonstrated that both hormones, were capable of reducing phagocytosis mediated by these receptors. Experiments with a drug, RU 486, capable of blocking the intracellular glucocorticoid receptor did not interfere with the effects induced by stress or the treatment in vitro with corticosterone. The effects observed with corticosterone could possibly be attributed to a nongenomic action of this hormone, since it is not blocked by actinomycine-D and cycloheximide. These results might be an important factor of modulation of the phagocytic process with implications in the mechanisms of defence and possibily in other physiologic processes.
SUPPORT: CNPq, FAPESP and FAEPA