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Title: **Effects of two stress models on serotonin transporter of rat lymphocytes**

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Abstract: Modifications produced at lymphocyte serotonergic system could be associated to immune alterations observed in chronic stressed rats and depressive patients. The dysregulation of the hypothalamic-pituitary-adrenal axis and the increase of blood glucocorticoid levels reported in these patients might be involved in such changes. To investigate the effects of stress on some functional aspects of serotonin transporter (5-HTT) from rat lymphocytes we applied two stress models to adult male rats: 1) physical restraint stress 5 hours/day for 5 days or 2) intraperitoneal injection of 2,5 mg/kg/day of reserpine for 3 days with the aims to: a) measure the proliferative response of lymphocytes to the mitogen concanavalin A (Con A) in the presence or in the absence of fluoxetine and b) calculate affinity constants (Kd), number of binding sites (Bmax) and Hill coefficients (n_H) from [^{3}H]-paroxetine binding using lymphocytes membranes. Morning serum corticosterone concentration was measured with an EIA kit of DSL. Corticosterone values in restrained: 544±52 ng/ml and controls: 232±74 ng/ml ($p<0.05$), reserpine treated: 467±94 ng/ml and controls: 322±39 ng/ml. Lymphocytes were isolated by Ficoll-Hypaque density gradients and differential adhesion to plastic. Cell proliferation was measured with a tetrazolium salt. In the restraint stress group, fluoxetine reduced basal proliferation at 5 μM in controls and at 25 μM in restrained. In the presence of Con A, fluoxetine had an antiproliferative effect at 5 μM in controls and at 10 μM in restrained. In the reserpine treated group, fluoxetine had a basal antiproliferative effect at 5 μM in controls and in treated rats. In the presence of Con A, fluoxetine had an antiproliferative effect at 5 μM in controls and at 10 μM in treated rats. In both stress groups it was observed a significant increase in Bmax, Kd and n_H respect to controls. These parameters changes indicate an elevation of the number of binding sites with a concomitant decrease in the affinity for the ligand and a loss of cooperativity for the binding to 5-HTT of lymphocytes from stressed rats, and are probably related to the differential sensitivity to fluoxetine observed in culture assays. Glucocorticoids might be involved in these alterations, although other unknown mechanisms could participate either.

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