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ABSTRACT FORM

TITLE	**The activation of the immune/inflammatory system is associated with the stress-induced anhedonia in rats: effect of pharmacological intervention**
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ABSTRACT (less than 500 words)	Despite the increased knowledge of depression neurobiology, an effective improvement in the overall impact of pharmacotherapy is still lacking, possibly because a number of systems that are affected in mood disorders may not be adequately modulated by pharmacological treatments. Indeed, it is known that this complex disorder is characterized by the interaction of many factors of different nature -i.e. genetic, biological, social and environmental- that act in concert to lead to the development of the illness. Currently, there is strong evidence that depression involves alterations of the immune/inflammatory systems, thus pointing to the importance to characterize this association as well as evaluate the potential impact of pharmacological intervention to interfere with such alterations. On these bases, the purpose of our study was to analyze the cerebral expression of several mediators of the immune/inflammatory system in an animal model of depression based on the environmental component of the disease. Specifically, we used the well-established chronic mild stress (CMS) paradigm to develop a validated rat model of depression in order to elucidate the role of inflammation on the generation of the pathological anhedonic phenotype. Moreover, to evaluate the ability of the pharmacological treatment in modulating the behavioral-associated inflammatory alterations, rats exposed to CMS were also chronically treated with the antidepressants imipramine and with the antipsychotic lurasidone. Our findings indicate that the stress-induced anhedonic phenotype is associated to altered expression of specific mediators of immune/inflammatory system and that pharmacological treatment is not only able to normalize the anhedonic phenotype but also the inflammatory changes. These data suggest that immune/inflammatory alteration may contribute to the subject's vulnerability of depression and support the idea that immune/inflammatory may serve as viable therapeutic target for more effective antidepressant drugs.