ACETYL SALICYLIC ACID INCREASES THE RESPONSE OF ESCITALOPRAM IN A RAT MODEL OF DEPRESSION

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Escitalopram (ESC) is the S(+) enantiomer of citalopram, the most selective serotonergic antidepressant available today. Several clinical and preclinical studies have shown that the antidepressant activity of citalopram resides in its S(+) enantiomer.
Preclinical and clinical studies have recently shown that Escitalopram possesses a faster onset of action compared to other antidepressants. It has been reported that Escitalopram is able to normalize the sucrose intake in the chronic mild stress model of depression after one week of treatment, thus showing a faster effect in reversing anhedonia, a key symptom of the depressive disorder reproduced in this animal model.
Nevertheless, the clinical spectrum of major depression is more complicated and includes other aspects different from anhedonia, like behavioral despair.
We previously demonstrated that the combination of acetylsalicylic acid (ASA) with Fluoxetine (FLX) is able to accelerate and potentiate the effect of the antidepressant in the chronic escape model of depression. These results, together with preliminary clinical data in major depressed non-responder patients, suggest that ASA might accelerate the onset of action of SSRIs.
Therefore, aim of the study was to compare the effect of one week of combined treatment with Escitalopram plus ASA vs. ESC alone in the chronic escape deficit model of depression. This behavioral model is based on the modified reactivity of rats to external stimuli induced by exposure to unavoidable stress and allows evaluating the capacity of a treatment to revert the condition of escape deficit.
Our study demonstrated that the response after one week of treatment was present in about 50% of the animals receiving ESC (10 mg/kg/day) alone and in about 75% of the rats receiving ESC plus ASA (45 mg/kg/day).
These results suggested that the co-administration of ASA with ESC increased the response to treatment in reverting the behavioural despair induced by stress in rats. Future biochemical analyses will be made in order to identify possible targets of the combination.