Abstract


Subchronic administration of ascorbic acid elicits antidepressant-like effect and modulates cell survival signaling pathways in mice.

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OBJECTIVE: In this study, we examined the ability of subchronic ascorbic acid administration to produce an antidepressant-like effect in the mouse tail suspension test (TST). Moreover, we investigated the effect of this vitamin on hippocampal and cerebrocortical brain-derived neurotrophic factor (BDNF) immunocortex, phosphorylation of protein kinase B (AKT), extracellular signal-regulated kinase (ERK), p38MAPK and c-Jun.

METHODS AND RESULTS: N-terminal kinase (JNK). Fluoxetine (10 mg/kg, positive control, po) or ascorbic acid (0.1 and 1 mg/kg, po), administered once daily for 21 days, produced a significant antidepressant-like effect in the TST. The significant effects obtained in protein immunocortices were: administration of ascorbic acid at 1 mg/kg induced an increase in AKT phosphorylation in cerebral cortex of mice. Ascorbic acid treatment (1 mg/kg), similar to fluoxetine, decreased hippocampal p38MAPK but did not alter ERK or JNK phosphorylation.

CONCLUSION: These results extend the data about the antidepressant-like effect of ascorbic acid by exploring, for the first time, the intracellular pathways involved in its antidepressant properties after subchronic administration.

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