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Activation of the ventral medial prefrontal cortex during an uncontrollable stressor reproduces both the immediate and long-term protective effects of behavioral control.

Amat J, Paul E, Watkins LR, Maier SF.

Source

Department of Psychology and Center for Neuroscience, University of Colorado, Boulder, CO 80309-0345, USA. amat@psych.colorado.edu

Abstract

DRN - Serotonin

The degree of behavioral control that an organism has over a stressor determines the behavioral and neurochemical sequelae of the stressor, with the presence of control preventing the typical outcomes that occur when the stressor is uncontrollable (e.g. failure to learn, exaggerated fear, dorsal raphe nucleus (DRN) 5-HT activation). Furthermore, an experience with a controllable stressor blocks the consequences of later uncontrollable stressors ("immunization"). These effects of control have been argued to be mediated by control-induced activation of ventral medial prefrontal cortex (mPFCv) output to the DRN. The experiments that have led to this interpretation have all involved the inactivation of the mPFCv with muscimol, showing that inactivation during the stressor eliminates the stressor-resistance produced by control, with the controllable stressor now acting as if it were uncontrollable. The present experiments in rats employed the opposite strategy, activating the mPFCv during the stressor. mPFCv microinjection of picrotoxin during the stressor eliminated the DRN 5-HT activation that normally occurs during the uncontrollable stressor, as well as the escape learning deficit and exaggerated fear that normally follows uncontrollable stress. Furthermore, mPFCv activation during an initial exposure to an uncontrollable stressor led the uncontrollable stressor to produce behavioral and neurochemical immunization when the subjects were later exposed to an uncontrollable stressor. That is, the conjoint activation of the mPFCv and exposure to an uncontrollable stressor led the uncontrollable stressor to act as if it were controllable. These results provide strong support for the argument that behavioral control produced stress-resistance by activating the mPFCv.

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