



Behavioral deficits and exaggerated feedback control over raphe-hippocampal serotonin neurotransmission in restrained rats

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Abstract:

Serotonin (5-hydroxytryptamine, 5-HT), acting *via* the hippocampus, is thought to be critical for the neuroadaptation that alleviates the adverse effects of stress on emotion and behavior. It was hypothesized that a decrease in raphe-hippocampal serotonin neurotransmission caused by exaggerated feedback inhibition of 5-HT synthesis and release significantly contributes to stress-induced behavioral deficits. Acute exposure to 2 h of restraint stress increased 5-HT metabolism in the cortex and raphe region but had no such effect in the hippocampus. Exposure to 2 h of restraint stress elicited anxiety-like behavior, which was monitored in the light-dark transition test the next day. Animals sacrificed 24 h after termination of the stress period exhibited a decrease in the concentration of 5-HT and its metabolite 5-hydroxyindoleacetic acid (5-HIAA) in the hippocampus but not in the cortex and raphe. 8-Hydroxy-2-di-n-propylaminotetralin (8-OH-DPAT) injected at doses of 0.125, 0.25 and 0.5 mg/kg decreased 5-HT metabolism in the raphe, cortex and hippocampus of restrained and unrestrained animals, and the decreases in the raphe and hippocampus, but not those in the cortex, were greater in restrained than unrestrained animals. Exaggerated feedback control over raphe-hippocampal serotonin neurotransmission may be involved in the inability of the organism to cope with increased stress and elicits behavioral depression.

Key words:

light-dark transition test, 5-HT_{1A} receptor, cortex, hippocampus, raphe, 8-OH-DPAT, feedback control
