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Development/Plasticity/Repair

Chronic Stress Impairs α_1-Adrenoceptor-Induced Endocannabinoid-Dependent Synaptic Plasticity in the Dorsal Raphe Nucleus

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Author contributions: S.H.-D. designed research; S.H.-D. performed research; S.H.-D. and R.-Y.S. analyzed data; S.H.-D. and R.-Y.S. wrote the paper.

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Abstract Full Text Full Text (PDF)

Abstract

Alpha 1-adrenergic receptors (α_1-ARs) control the activity of dorsal raphe nucleus (DRn) serotonin (5-HT) neurons and play crucial role in the regulation of arousal and stress homoeostasis. However, the precise role of these receptors in regulating glutamate synapses of rat DRn 5-HT neurons and whether chronic stress exposure alters such regulation remain unknown. In the present study, we examined the impact of chronic restraint stress on α_1-AR-mediated regulation of glutamate synapses onto DRn 5-HT neurons. We found that, in the control condition, activation of α_1-ARs induced an inward current and long-term depression (LTD) of glutamate synapses of DRn 5-HT neurons. The α_1-AR LTD was initiated by postsynaptic α_1-ARs but mediated by a decrease in glutamate release. The presynaptic expression of the α_1-AR LTD was signaled by retrograde endocannabinoids (eCBs). Importantly, we found that chronic exposure to restraint stress profoundly reduced the magnitude of α_1-AR LTD but had no effect on the amplitude of α_1-AR-induced inward current. Chronic restraint stress also reduced the CB1 receptor-mediated inhibition of EPSC and the eCB-mediated depolarization-induced suppression of excitation. Collectively, these results indicate that chronic restraint stress impairs the α_1-AR LTD by reducing the function of presynaptic CB1 receptors and reveal a novel mechanism by which noradrenaline controls synaptic strength and plasticity in the DRn. They also provide evidence that chronic stress impairs eCB signaling in the DRn, which may contribute, at least in part, to the dysregulation of the stress homeostasis.

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