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Neuroscience 2015

Presentation Abstract

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Presentation Title:	Time-dependent changes induced by acute stress in function and architecture of excitatory synapses in prefrontal and frontal cortex
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Presentation time:	Tuesday, Oct 20, 2015, 8:00 AM -12:00 PM
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Topic:	++C.16.c. Mood disorders: Vertebrate animal models
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Abstract:	Stressful life events impact on brain and bodily function and represent major risk factors for stress-related neuropsychiatric disorders. The response to stressful events can promote adaptive plasticity and improved cognition, when the physiological stress response is efficiently activated and inactivated in due time, or maladaptive and harmful effects, when the response is overused or dysregulated. In turn, the outcome of a maladaptive stress response can be associated with the triggering of brain, systemic and metabolic disorders. Chronic stress has been shown to induce reduction of density of synapses and dendrites in prefrontal and frontal cortex (PFC/FC), with concomitant impairments in neuronal activity and cognitive functions. Instead, the early and rapid effects of acute stress on synaptic function and plasticity are often opposite, with enhancement of glutamate release/transmission, increased number of spines and synapses, enhancement of synaptic strength. However, the delayed effects of acute stress have not been investigated, although this could give crucial information on the time-dependent changes in the brain stress response. We have previously characterized the synaptic effects of acute footshock (FS)-stress, which induces enhancement of glutamate release/transmission

in PFC/FC, due to the increase of the readily releasable pool (RRP), in turn mediated by rapid non-genomic corticosterone action at synapses (Mol. Psy., 19:433-443, 2014). Here we have analyzed the effects of acute FS-stress in the PFC/FC of rats at different times after completion of the stress protocol. We found that acute stress induced early and sustained increase of RRP over time in excitatory perforated synapses, while the number of non-perforated and axo-spinous synapses was increased (without changes in vesicle pools). The total number of synaptic spines was increased up to 24 h, while apical dendrites showed decreased density 2 weeks after acute stress (with no significant changes at earlier times). In behavioral tests for working memory, FS-stress improved performance 2 h after stress and impaired it after 24 h. Changes in glutamate release, RRP, number of synapses and spines are blocked or attenuated by prior chronic treatment with the antidepressant desipramine. The different glutamatergic modifications in functional and morphological plasticity suggest a bi-phasic process, during which the stress response in PFC/FC may turn from early increased excitatory activation into its opposite. The identification of these points and the players involved in the switch are crucial for the understanding of the dynamics of stress-related pathology.

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SYNAPTIC PLASTICITY

GLUTAMATE TRANSMISSION

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