Attenuation of Long Term Synaptic Plasticity Following Application of a Restraint Stressor

J. Harry Blaise, Senior Member, IEEE

Abstract—We assess the impact of an acute restraint stress on the persistence of long term potentiation, a cellular model widely assumed to underlie learning and memory. Results indicate a restraint stress attenuates long term potentiation. This finding may have clinical implications in terms of acute stress possibly promoting deficits in cognitive functions in populations already at risk for stress-related neurological disorders.

I. INTRODUCTION

The basolateral amygdala (BLA), a brain region which is involved in emotional memories and the stress response, has strong synaptic connections with the hippocampal dentate gyrus (DG), which is implicated in learning and spatial memory. Together the BLA-DG neural circuit assigns emotional significance to memories of particularly salient events and experiences by regulating long term synaptic plasticity [1-3]. Previously, we showed chronic early-life stress resulted in significantly greater long-term potentiation (LTP, a cellular mechanism for memory formation and consolidation) compared to age-matched controls [1]. The aim of the present study is to assess whether an acute stress (restraint for 30 minutes) has any impact on long term plasticity of the BLA-DG neural circuit.

II. MATERIALS AND METHODS

Rats (2-3 months old) were chronically implanted with a stimulating electrode in the BLA and a recording electrode in the DG. Following a one-week postsurgical recovery period, LTP was induced in the DG using a standard burst tetanization protocol (100-pulse, 5-Hz theta-burst stimulation, TBS) applied to the BLA. Ninety minutes after LTP induction animals were subjected to a period of 30 minutes of restraint stress.

III. RESULTS AND DISCUSSION

Representative field potential traces are shown in the inset of Figure 1. Our results indicate restraint stress attenuates LTP suggesting this type of acute stress has a profoundly disruptive effect on synaptic mechanisms thought to underlie learning and memory processes (Fig. 1). Moreover, they are consistent with previous reports of long-lasting alterations in structural and functional plasticity of neural circuits involved in emotion and the regulation of the stress response in organisms that experienced chronic stress [2, 3]. Finally, these findings may have clinical implications in terms of acute stress promoting deficits in cognitive functions in populations already at risk for major depressive and post-traumatic stress disorders [4].

REFERENCES


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J. H. Blaise is with the Department of Engineering and the Neuroscience Program, Trinity College, Hartford, CT 06106, USA (e-mail: harry.blaise@trincoll.edu).