Abstract Title: NEURONAL MAPK/ERK1/ERK2 ACTIVATION IS BLOCKED IN MOUSE BRAIN BY RESTRAINT STRESS

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Abstract: Stress causes many physiological changes and there is great interest in determining the signal transduction pathways involved. However, most examinations of putative signaling pathways have involved in vitro preparations. Here we utilized a mouse restraint stress model that involves both a physical and psychological disturbance of homeostasis. This model was used to examine, in vivo, Jak-STAT and MAPkinase pathways believed to play a role in transducing stress signals. Female C57BL/6J mice were restrained in 2.5 cm I.D. restrainers and killed immediately after restraint by focused microwave irradiation. Phosphorylation states of the extracellular signal-regulated kinases (erk), the JNKs and the p38 stress activated protein kinases as well as STAT3 and CREB were determined by immuno-blotting and chemiluminescence detection using phospho state-specific antibodies. Restraint for 2 hrs caused an 80% decrease in activated p42/44 MAPK in hippocampus, a 67% decrease in cortex and a 60% decrease in striatum. By contrast there was no significant change in hypothalamus at 2 hrs. After 8 hrs, the hippocampal content of phospho p42/44 MAPK in restrained animals had risen to 42% of controls. Hypothalamic phospho p42/44 was equal in restrained and controls while the decrements in cortex and striatum were still approximately 60%. Restraint stress did not change phospho STAT3, CREB, SAPK or p38 MAPkinase in any of the brain areas examined. As the erks serve as a key biochemical integration system, our data suggest that effects of stress may be mediated by a disruption of this key signaling cascade. Future work examining the impact of this in vivo stress model on the upstream regulators (Raf-1, B-Raf, MEK, etc) of the erks may provide clues as to how stress can impact neuronal events (e.g, plasticity) believed to be targeted by stress.

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