Acute stress facilitates long-lasting changes in cholinergic gene expression

By ***soreq***
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Kaufer, D, Friedman A, Seidman S, Soreq H. 1998.

Abstract:

Acute traumatic stress may lead to post-traumatic stress disorder (PTSD), which is characterized by delayed neuropsychiatric symptoms including depression, irritability, and impaired cognitive performance. Curiously, inhibitors of the acetylcholine-hydrolysing enzyme acetylcholinesterase may induce psychopathologies that are reminiscent of PTSD. It is unknown how a single stressful event mediates long-term neuronal plasticity. Moreover, no mechanism has been proposed to explain the convergent neuropsychological outcomes of stress and of acetylcholinesterase inhibition. However, acute stress elicits a transient increase in the amounts released of the neurotransmitter acetylcholine and a phase of enhanced neuronal excitability. Inhibitors of acetylcholinesterase also promote enhanced electrical brain activity, presumably by increasing the survival of acetylcholine at the synapse. Here we report that there is similar bidirectional modulation of genes that regulate acetylcholine availability after stress and blockade of acetylcholinesterase. These calcium-dependent changes in gene expression coincide with phases of rapid enhancement and delayed depression of neuronal excitability. Both of these phases are mediated by muscarinic acetylcholine receptors. Our results suggest a model in which robust cholinergic stimulation triggers rapid induction of the gene encoding the transcription factor c-Fos. This protein then mediates selective regulatory effects on the long-lasting activities of genes involved in acetylcholine metabolism.

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