

Intranasal neuropeptide Y may offer therapeutic potential for post-traumatic stress disorder

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Stress triggered neuropsychiatric disorders take an enormous personal, social and economic toll on society. In the US more than half of adults are exposed to at least one traumatic event throughout their lives. Posttraumatic stress disorder (PTSD) is a debilitating anxiety disorder associated with exposure to a traumatic event outside the range of normal human experience. PTSD typically follows a chronic, often lifelong, course. Patients have diminished quality of life, are more likely to manifest other psychiatric disorders such as depression and six times more likely as demographically matched controls to attempt suicide. Prevention and treatment of PTSD remains a challenge with improved therapies needed to help save billions of dollars in medical care and provide enormous society benefit.

Based on a variety of studies in humans and animals it has been suggested that neuropeptide Y (NPY), a peptide that acts as a neurotransmitter in the brain, has therapeutic potential for PTSD. This naturally occurring peptide is one of the widely expressed inside and outside of the brain with diverse functions. Human studies indicate that NPY is associated with resilience to development of PTSD or helps improve recovery from harmful effects of traumatic stress. Injections into the brain of rodents attenuated some of the <u>behavioral responses</u> related to stress associated <u>neuropsychiatric disorders</u>. However systemic administration of NPY will likely have undesirable side effects, especially on the cardiovascular system.



This study delivered NPY to rats by intranasal infusion, a non-<u>invasive</u> procedure to bypass the <u>blood brain barrier</u>. A single infusion was administered 30 min before or immediately after exposure to single prolonged stress (SPS). Behavioral, neuroendocrine and <u>biochemical analyses</u> were performed 1 to 3 weeks after SPS and compared to untreated controls or to animals infused with the solution without NPY. The SPS-elicited elevation in anxiety, depressive-like behavior and hyper-arousal was reduced in the animals given intranasal NPY, and some of the features were the same as in the animals not exposed to the stress. There was a lower stress triggered rise in plasma stress hormones, such as glucocorticoids and in expression of their receptor in the hippocampus in the NPY treated animals. Intranasal NPY also modulated the response of the brain noradrenergic system to the traumatic stress of SPS.

The results demonstrated, for the first time, that rapid delivery of NPY to the brain by intranasal infusion, before or shortly after exposure to traumatic stress, has a pronounced resilient effect and ameliorates development of PTSD-like symptoms in rats. It provides proof of concept for potential of intranasal NPY for non-invasive prophylactic treatment for individuals likely to be exposed to traumatic stress, such as early responders or military personnel, as well as for early intervention after exposure to traumatic stress.

These research findings will be presented April 21st, 2013 during Experimental Biology 2013 in Boston, MA.

Provided by Federation of American Societies for Experimental Biology

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