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Neuropeptide Y Receptor Gene Expression in the Primate Amygdala Predicts Anxious Temperament and Brain Metabolism.

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Abstract

BACKGROUND:

Anxious temperament (AT) is identifiable early in life and predicts the later development of anxiety disorders and depression. Neuropeptide Y (NPY) is a putative endogenous anxiolytic neurotransmitter that adaptively regulates responses to stress and might confer resilience to stress-related psychopathology. With a well-validated nonhuman primate model of AT, we examined expression of the NPY system in the central nucleus (Ce) of the amygdala, a critical neural substrate for extreme anxiety.

METHODS:

In 24 young rhesus monkeys, we measured Ce messenger RNA (mRNA) levels of all members of the NPY system that are detectable in the Ce with quantitative real time polymerase chain reaction. We then examined the relationship between these mRNA levels and both AT expression and brain metabolism.

RESULTS:

Lower mRNA levels of neuropeptide Y receptor 1 (NPY1R) and NPY5R but not NPY or NPY2R in the Ce predicted elevated AT; mRNA levels for NPY1R and NPY5R in the motor cortex were not related to AT. In situ hybridization analysis provided for the first time a detailed description of NPY1R and NPY5R mRNA distribution in the rhesus amygdala and associated regions. Lastly, mRNA levels for these two receptors in the Ce predicted metabolic activity in several regions that have the capacity to regulate the Ce.

CONCLUSIONS:

Decreased NPY signaling in the Ce might contribute to the altered metabolic activity that is a component of the neural substrate underlying AT. This suggests that enhancement of NPY signaling might reduce the risk to develop psychopathology.

KEYWORDS:

Anxiety, behavioral inhibition, depression, prefrontal cortex, rhesus macaque, stress

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