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Oral Presentation

Pathophysiology of Anxiety Disorders

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Abstract

The most important risk factors for anxiety disorders include genes, early life stress, and current stress. These factors do not act independently but interact with each other throughout human development through examples such as epigenetic modifications and complex forms of learning. The neural substrate of pathological anxiety includes hyperactivity in the amygdala and other limbic brain regions, and decreased activity of the anterior cingulate and prefrontal cortex. This pattern of abnormal brain activity points to an impaired balance between subcortical emotional centers and higher cortical centers that are responsible for cognitive control. The molecular underpinnings of this unbalance are dysfunctions of neurotransmitter and neuropeptide systems responsible for the connectivity and communication between brain regions. GABA is the major inhibiting neurotransmitter in the human brain. The rapid and robust anxiolytic effects of benzodiazepines, which are positive allosteric modulators at the $GABA_A$ receptor, has encouraged imaging and genetic studies on the GABA neurotransmitter system. The anxiolytic effect of drugs that increase monoamines (e.g., serotonin, norepinephrine and dopamine) in the synaptic cleft, have made the monoaminergic systems a prime target of anxiety disorder research. Neuropeptides with particularly strong links to anxiety include the corticotropin-releasing factor, cholecystokinin, neuropeptide Y and oxytocin. This talk will give an overview on the current knowledge on the neurobiology of anxiety disorders and discusses the findings with respect to preventative approaches, pharmacotherapy, psychotherapy and future clinical research of anxiety disorders.

Keywords: Anxiety Disorders, GABA Receptor, Amygdala, Epigenetic.

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