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Commentary

Implication and Developments of Social Phobia

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DESCRIPTION

The symptoms of this disorder include severe anxiety, an elevated heart rate, diaphoresis, and other symptoms of autonomic arousal. The person with this disorder fears being embarrassed or humiliated by their conduct in social and/or performance circumstances. These physical symptoms may exacerbate anxiety, frequently resulting in a conditioned fear reaction that feeds their worry when in public.

An individual with a history of extreme shyness or social inhibition in childhood may or may not encounter a rapid beginning of social phobia, which frequently manifests after a stressful event or embarrassing social experience. If social anxiety is severe enough to impair social or professional functioning, it is regarded as a condition. True social phobics go to considerable efforts, frequently to their own damage, to avoid social situations. Due to the egodystonic nature of the dread of embarrassment, socially anxious people are bothered by their symptoms.

Recent concepts about cognition and studies into brain connectivity and function are changing our understanding of the pathophysiology of social phobia. Emotion regulation was the topic of a functional connectivity study with 174 participants, 78 of whom had social anxiety disorder. The findings demonstrated that effective regulation in the presence of negative stimuli involved activating the Prefrontal Cortex (PFC) and reducing amygdala reactivity, and that greater symptom severity was associated with decreased activation of the Dorsal Anterior Cingulate Cortex (DACC) and decreased functional connectivity between the amygdala and ventrolateral prefrontal cortex.

The "Clark and Wells cognitive model of social phobia," one of the cognitive theories useful in understanding the aetiology of social anxiety, hypothesises that self-focused attention, unfavourable observer-perspective images of oneself, and safety behaviours maintain anxiety in subjects with social anxiety and that this anxiety is associated with observer-perspective imagery and safety-seeking behaviour in adolescence. Such self-centered negative thoughts, however, are not linked to self-reported social anxiety.

There are additional theories that examine the effectiveness of medications used to treat social anxiety. Since serotonergic reuptake inhibitors are effective at reducing symptoms, serotonergic functioning may be involved. Similar to this, other researchers think that the efficacy of propranolol therapy supports an adrenergic origin. Studies have indicated that the amygdala's heightened response to adverse social stimuli in social anxiety may be due to neurocircuitry involving the amygdala, a region engaged in fear. Treatment implications include the need to avoid advancing therapy too rapidly, setting off extreme anxiety, or terminating therapy too soon. An initial strategy to improve the capacity to tolerate modest levels of anxiety is the presence of a caregiver who can model adaptive behaviour.

Premature infants with very low birth weights (600-1250 g) may also be more likely to develop social anxiety disorder later in life. This may be because of abnormalities in the uncinate fasciculus, a major white matter tract that connects the frontal cortex to the amygdala and other limbic temporal regions.

One multisite investigation investigated the possibility of a genetic link between treatment response and particular genetic loci. The change in FKBP5 DNA methylation was nominally associated with treatment response, as individuals who showed the greatest reduction in severity decreased in percentage DNA methylation during treatment compared with individuals with one or more FKBP5 risk alleles who had little or no decrease or an increase in percentage DNA methylation. Treatment response was not associated with specific genetic loci, FKBP5, GR polymorphisms, or pretreatment percentage DNA methylation, however.

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