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Molecular Underpinnings of Cognitive and Emotion Processing in Mood Disorders

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Mood disorders have been frequently associated with limbic hyper-responsiveness to emotionally negative stimuli as measured with functional magnetic resonance imaging (fMRI). Such aberrations of limbic, and particularly amygdala activity during emotion processing appear to be associated with negative cognitive processing biases implicated in the onset and maintenance of depression. In a series of studies, the authors investigated molecular underpinnings of pathological amygdala function using different fMRI paradigms designed to target automatic and controlled stages of emotion processing. Strong effects of selected candidate genotypes and amygdala responsiveness as well as amygdala-prefrontal coupling were observed across independent samples of depressed patients and healthy control subjects, supporting the notion of genetically influenced brain activation patterns associated with mood disorders. The frequently investigated 5-HTTLPR polymorphism was linked to amygdala hyper-responsiveness to negative but not positive facial expressions in paradigms assessing automatic amygdala responsiveness by means of subliminally presented stimuli. In later, controlled stages of emotion processing, genetic variations in COMT, MAO-A, Neuropeptides, and pro-inflammatory cytokines were associated with amygdala activity and amygdala-prefrontal coupling, pointing to dysfunctions in emotion regulation circuitries. In forthcoming studies, gene x environment interaction and epigenetic markers such as DNA methylation will inform about the degree of genetic and environmental influence on brain activation patterns in mood disorders.

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