S20-03

NEUROGENETICS OF EMOTION PROCESSING IN MAJOR DEPRESSION

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Converging lines of research suggest that major depression is characterized by abnormalities in different stages of emotion processing (e.g., automatic emotion processing, emotion regulation, or reward processing). Using functional neuroimaging, the neurobiological correlates of these emotion processing biases have been intensively investigated during the last decade. Depressed patients appear to be characterized by a hyper-responsive amygdala to negative stimuli, dysfunctional prefrontal activity during emotion regulation and blunted activity of the ventral striatum in response to positive cues or reward.

We present a series of studies investigating genetic underpinnings of these neurobiological abnormalities frequently observed in depression. We demonstrate a consistent pattern of serotonergic and neuropeptide genes that bias automatic amygdala responsiveness towards subliminally presented negative stimuli. Other genes were shown to impact prefrontal engagement during conscious emotion processing, e.g. genetic variants in the interleukin 1 beta gene. Further polymorphisms, e.g. in the endocannabinoid system rather seem to influence striatal responsiveness to positive social cues.

We conclude that the neurobiological abnormalities in major depression which are linked to biased emotional processes are strongly under genetic influence. The investigation of neurogenetic pathways to different aspects of depression psychopathology might help to better characterize patients according to their neurobiological dysfunctions and might ultimately lead to individualized therapy plans.

https://doi.org/10.1016/S0924-9338(11)73787-7 Published online by Cambridge University Press