

Neural Signatures of Major Depressive, Anxiety, and Stress-Related Disorders

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ABOUT THE STUDY

Major Depressive Disorder (MDD) and anxiety disorders (i.e., generalized anxiety disorder, panic disorder without agoraphobia) are highly comorbid psychiatric disorders, with shared epidemiologic and developmental features and a shared genetic basis and are among the leading causes of disability worldwide. Depression and anxiety are often triggered by stressful life events, thus sharing the aetiology of stress-related disorders that are defined by occurrence of a severe stressor or trauma. More specifically, Posttraumatic Stress Disorder (PTSD) is characterized by hyperarousal states during recurring flashbacks to the stressful event, while stress adjustment disorder is characterized by depressive symptoms in response to a severe stressor.

Task-based functional MRI findings point to disrupted emotional processing and executive dysfunction, exemplified by disrupted cognitive control, across a variety of disorders, including, but not limited to MDD and anxiety disorders. Similarly, gray matter reductions have been shown in the insular and anterior cingulate cortices across mood, anxiety, and psychotic disorders. Further, greater similarity of deviations in brain structure was found for disorders with greater genetic similarity. Such transdiagnostic phenotypes aim to capture the shared neurocognitive basis of symptoms presenting across disorders and could have utility in improving psychiatric nosology.

Inferior prefrontal and insular cortex, the inferior parietal lobule and the putamen are hypoactivated in task-based Functional Magnetic Resonance Imaging (FMRI) paradigms across MDD, anxiety disorders, and stress-related disorders, implicating inhibitory control and salience processing as shared neural phenotypes underlying mood, anxiety, and stress-related disorders. Impairments in executive functions such as inhibitory control over emotional reactivity and negative mood may capture a transdiagnostic dimension of psychopathology. Executive function is also impaired by anxiety, which reduces cognitive flexibility and working memory and impairs attentional control. While some evidence also points towards executive dysfunction in PTSD, psychological theories of posttraumatic stress typically emphasize the effects of the traumatic event on memory.

Signatures of MDD and anxiety disorders were highly concordant and distinct from stress-related disorders. Shared polygenic risk explained a small proportion of the similarity in brain connectivity and structure between MDD and anxiety, but moderated the concordance of neural signatures for stress-related disorders and MDD. We further identified impairments in processing speed, attention, fluid intelligence, and paired associate learning shared across all disorders under investigation. A dimensional analysis focusing on MDD, anxiety disorders, and stress-related disorders identified increased between-network and decreased within-network connectivity of the frontoparietaldefault mode networks as a neural correlate of poorer cognitive function.

The strong similarity of the neural signatures of the MDD and anxiety disorders groups supports previous studies of shared neural signatures of mental disorders. Controlling for polygenic risk slightly decreased disorder similarity between MDD and anxiety disorders but increased the similarity between stressrelated disorders and MDD or anxiety disorders. Since PRSs are representative of trait-like genetic risk for MDD, we conclude that a small proportion of similarity between MDD and anxiety disorders is explained by genetic risk for these disorders. By contrast, stress-related disorders are transient conditions, less affected by genetic risk than MDD or anxiety disorders.

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