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Commentary

Multi-Scale Neural Signatures of Stress-Related Disorders

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ABOUT THE STUDY

The extent of the common and different neural mechanisms underlying Major Depressive Disorder (MDD), anxiety, and stress-related disorders is still unknown. We compared the neural signs of these disorders in 5,405 UK Biobank patients and 21,727 healthy controls. The difference between resting functional connectivity of MDD and maximum case control of cortical thickness was discovered, followed by anxiety and stress-related disorders. Neuronal characteristics of MDD and anxiety disorders were highly consistent with changes in frontal striatum connectivity, whereas stress-related disorders showed a clear pattern. By controlling the genetic risk of cross-disorder, there was some increase in the similarity between the functional neuronal characteristics of stress-related disorders and both MDD and anxiety disorders. In case and healthy controls, decreased connectivity within the network and increased connectivity between frontal and standard modes were associated with decreased cognitive performance. These results provide evidence of significant impairment of neural circuit function in MDD and anxiety disorders compared to stress disorders. However, cognitive impairment appears to be independent of diagnosis and depends on circuit function.

Depression and anxiety are often caused by stressful life events and therefore share the etiology of stress-related disorders defined by the development of major stressors or traumas (DSM5). More specifically, post-traumatic stress disorder (PTSD) is characterized by hypervigilance during recurrent flashback to stressful events, and stress adjustment disorder is a depressive symptom in response to severe stressors.

Multimodal studies of functional connectivity, brain structure, cognitive function, and genetic risk have shown that the signs of MDD and anxiety disorders are very consistent and different from stress-related disorders. The shared polygene risk described some of the brain connectivity and structural similarities between MDD and anxiety, but alleviated the concordance of stress-related disorders and MDD neural signs. We also identified disorders in the pair of speed, attention, fluency, and related learning that are common to all the disorders investigated. Dimensional analysis focused on MDD, anxiety disorders, and stress-related disorders identified increased internetwork connections and decreased intra-network connections in the frontal parietal default mode network as neural correlations for cognitive decline.

Several neurocognitive processes may underlie the duplication of neural signs of MDD and anxiety disorders, including executive function. Common neural mechanisms are found in the limbic circuits involved in prefrontal cortex regulation and mood and emotional processing in default mode. Stress-related disorders, on the other hand, appear to have distinct neuronal characteristics with differences in parahippocampal structure and default mode connectivity that may be associated with maladaptive stress responses and memory formation increase.

We found a high level of similarity to the neural signs of MDD, an anxiety disorder (single and comorbid) that is different from stress-related disorders. Our results are consistent with the diagnostic classification of MDD and anxiety disorders as internalizing disorders (DSM 5). Stress-related disorders showed higher-order dysfunction profiles that were very similar to MDD and anxiety disorders, but their neural signs showed little similarity, especially in areas of cortical thickness. Although comorbidities of cross-disorder are considered therapeutic challenges, the identified neurobiological substrates provide a default mode and connections within and between the frontal parietal networks that provide cognitive dysfunction. It may represent a promising target for a particular intervention.

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