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Major Depressive Disorder can also be Triggered by Pharmacological Agents or Drug Abuse

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DESCRIPTION

Major depression is a heterogeneous disorder that affects 1 in 5 people and is the leading cause of disability worldwide. MDD is now considered to be a multifactorial disease with various causes, including genetic susceptibility, stress and other pathological processes. Multiple studies have developed multiple theories that attempt to explain the development of MDD. However, none of these hypotheses are comprehensive and none can explain all cases, mechanisms and symptoms of MDD. Nonetheless, we believe that these hypotheses represent multiple pieces of the same large puzzle, as they all share some common paths. This review therefore highlights the common mechanisms and relationships of all major depression theories and briefly describes these theories and their strengths and weaknesses in order to bring them together and present the current picture.

The symptoms of MDD are associated with structural and neurochemical defects in brain regions. The behavioural symptoms of depression are extensive, covering emotional, motivational, cognitive, and physiological domains, and include abnormal reward-related cognition, and memory impairment. MDD is now thought to be a multifactorial disease with a variety of causes and triggers, including genetic susceptibility, stress and other pathological processes such as inflammation. For example, genetic factors can in some cases contribute to or even cause the development of depression. Some mutations and polymorphisms can affect the response of receptors to neurotransmitters or biologically active substances, which, in turn, could affect the resistance of the brain's chemical balance to stressors. Some mutations and polymorphisms can affect the response of receptors to neurotransmitters or biologically active substances, which, in turn, could affect the resistance of the brain's chemical balance to stressors. These factors can influence both general risk of disease and individual susceptibility to environmental influences. The neurotropic hypothesis of depression states that the cause and ethology of depression is a disruption in the functioning of the brain's neurotropic system, and that antidepressant treatment may reverse deficiencies in this system and reduce depressive symptoms. We hypothesize that it can be explained by facts. Studies on this hypothesis suggest that brain-derived neurotropic factors are involved in neurogenesis in regulating neuronal differentiation and growth, and that they may influence behaviour through control of neurogenesis. There is a focus on other regulators of neuroplasticity. It has been suggested that adult neurogenesis may enhance glucocorticoid-mediated negative feedback on the HPA axis and promote stress tolerance. Decreased neurogenesis may therefore underlie the development of depression-like symptoms in stressful situations. Overall, we emphasize that depression is a heterogeneous disorder that includes a wide range of subtypes (melancholic, atypical, psychotic, etc.) and has distinct characteristics in terms of symptoms, neurobiology, physiology and endocrine function. From the above literature, it is clear that many symptoms associated with depression are most likely the result of abnormalities in various aspects of normal neuronal function, from the molecular level to the neural circuitry. There may be several subtypes of MDD with different etiologist. The observation that classical antidepressants work only in a subset of patients suggests that depressed patients exhibit abnormalities in different neural processes, or different parts of the same complex mechanism consisting of an extensive network of interconnected pathways is shown. A complete baseline assessment of depressive symptoms before treatment enables the creation of a patient-specific profile and aids in the development of more effective treatment plans.

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CONFLICT OF INTEREST

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