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A Note on Molecular and Cellular Mechanisms of Depression

Paul Albert*

Department of Biomedical and Clinical Sciences, Linköping University, Sweden

Commentary

Depression is a complex illness that has a significant impact on one's health. The specific brain mechanisms underlying the development of depression remain obscure due to the wide range of clinical symptoms displayed by affected individuals. Although it is impossible to phenocopy every symptom of human sadness in rodents, the preclinical field has had a lot of success modelling some of the most common affective and neurovegetative depressive symptoms, such as social withdrawal, anhedonia, and weight loss. Individual depression symptoms may be caused by adaptations in specific cell populations, and new techniques have increased our ability to monitor and control specific cell types.

Some of the most current preclinical findings on the molecular and neurophysiological pathways in reward circuitry that underpins the manifestation of depressive symptom-related behavioural components. Inflammation is thought to play a role in the pathophysiology of depression. Microglia plays a crucial role in neuroinflammation as tissue-specific macrophages in the central nervous system (CNS). During neuroinflammation, resident microglia becomes activated to either a pro-inflammatory (M1) or anti-inflammatory (M2) phenotype. Neurons in the CNS convey their state to microglia and can control microglial activity, while microglia modulates neuronal processes, including neuroplasticity.

The molecular processes governing microglia-neuron communication, which include intracellular and extracellular signalling pathways, are likely to be complicated and important for future research into the pathophysiology of depression. The goal of this study is to highlight the common cellular and molecular mechanisms underlying microglial activation and abnormal neuroplasticity in depression, as well as their significance in depression aetiology. Depression is a major health concern in Western nations, with a high prevalence and a significant social cost. It's linked to atrophy and dysfunction in the corticolimbic regions of the brain that control mood and emotion. It's been suggested that changes in neurotrophins cause decreased neuroplasticity, which could be linked to the onset and progression of depression. As a result, emerging data suggests that antidepressant medication may improve neuronal and synaptic plasticity by increasing trophic signalling.

Current antidepressants, on the other hand, have a delayed beginning of action and are ineffective. As a result, a better knowledge of the molecular and cellular mechanisms involved in the pathophysiology of depression and the action of antidepressants could lead to the development of new, faster-acting, and more effective treatments. The current evidence on the role of neurotrophic factors in the pathogenesis and therapy of depression is summarised here. Furthermore, we believe that future antidepressant development should be founded on the neurotrophin idea. Deciphering the pathophysiology of depression is a difficult task. Not only are depressive syndromes different in terms of etiologies, but symptoms like guilt and suicidality are impossible to replicate in animal models. Other symptoms, on the other hand, have been accurately predicted, and these, along with clinical data, are revealing information about the neurobiology of depression. Certain features of depression are caused by maladaptive stress-induced neuroplastic alterations in certain brain circuits, according to recent studies combining behavioural, molecular, and electrophysiological approaches. They also suggest that knowing the mechanisms of stress resilience adds a crucial new dimension to the creation of antidepressant medications that are fundamentally different.

In the 1960s, the serotonin (5-HT) hypothesis of depression was proposed. It was once thought that the sickness was caused by a lack of serotonin in the brain, which could be addressed with antidepressant medicines. Recurrent mood disorders are now widely regarded as brain diseases caused by a complex interaction of genetic, other biological, and environmental variables that develop over time. All fields of neuroscience are actively engaged in attempts to elucidate the pathophysiology of depression and the mechanisms underpinning the success of antidepressant treatments, from genes to behaviour, molecules to cognition, and experimental to clinical. This is the first of two special issues of Philosophical Transactions B that aims to provide an overview of current developments in the subject, with a focus on cellular and molecular mechanisms and how their unravelling opens up new research avenues [1-5].

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***Address for Correspondence:** Paul Albert, Department of Biomedical and Clinical Sciences, Linköping University, Sweden, E-mail: Paul.albert@liu.se*

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