Acta Psychopathologica
ISSN: 2469-6676

Open access Commentary

Immune Dysregulation in Bipolar Disorder: Exploring the Pathophysiological Link

Daniel Cheeow*

Department of Educational Psychology, University of Cape Town, Denmark

DESCRIPTION

Bipolar disorder, a chronic and severe mental health condition characterized by alternating periods of mania and depression, has long been studied for its complex etiology and multifaceted nature. In recent years, growing evidence has suggested a significant link between immune dysregulation and bipolar disorder, shedding light on the biological underpinnings of this mental illness. Patterns of immune dysregulation in bipolar disorder offer a promising avenue for understanding the pathophysiology of the disorder and potentially identifying novel therapeutic targets. Immune dysregulation in bipolar disorder involves both the innate and adaptive immune systems. The innate immune system provides the first line of defense against pathogens and is characterized by the activation of various inflammatory cells and cytokines. The adaptive immune system, on the other hand, provides a more specialized response through the activation of lymphocytes. Dysregulation in these systems has been implicated in the onset and progression of bipolar disorder. One of the key findings in this area is the presence of elevated levels of pro-inflammatory cytokines in individuals with bipolar disorder. Cytokines are signaling proteins that play a crucial role in regulating immune responses. Studies have consistently reported increased levels of cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-alpha), and interleukin-1 beta (IL-1 beta) in patients with bipolar disorder, especially during mood episodes. These cytokines can cross the blood-brain barrier and influence brain function, potentially contributing to the neuroinflammation observed in bipolar disorder. Neuroinflammation, a hallmark of immune dysregulation in bipolar disorder, involves the activation of microglia, the brain's resident immune cells. Activated microglia release pro-inflammatory cytokines and other neurotoxic substances that can damage neurons and synapses, leading to altered brain function. This process is thought to contribute to the mood instability and cognitive impairments commonly seen in bipolar disorder. Neuroimaging studies have provided further evidence of neuroinflammation in bipolar disorder, showing increased activation of microglia in various brain regions. In addition to elevated pro-inflammatory cytokines, bipolar disorder has been associated with alterations in other immune markers. For example, some studies have reported abnormal levels of C-reactive protein (CRP), a marker of systemic inflammation, in individuals with bipolar disorder. Elevated CRP levels have been linked to an increased risk of cardiovascular disease, which is notably higher in patients with bipolar disorder compared to the general population. This suggests that immune dysregulation in bipolar disorder may have broader health implications beyond the central nervous system. The genetic basis of immune dysregulation in bipolar disorder is also an area of active research. Genome Wide Association Studies (GWAS) have identified several genetic variants associated with both bipolar disorder and immune system function. For instance, polymorphisms in genes encoding cytokines and their receptors have been linked to an increased risk of developing bipolar disorder. These findings support the notion that genetic factors contribute to the immune abnormalities observed in this condition. Environmental factors, such as infections and stress, are also believed to play a role in immune dysregulation in bipolar disorder. Infections, particularly during early life, can have lasting effects on the immune system and increase the risk of psychiatric disorders, including bipolar disorder. Chronic stress, a known trigger for mood episodes, can activate the hypothalamic-pituitary-adrenal axis and lead to the release of glucocorticoids, which in turn can modulate immune responses and promote inflammation.

ACKNOWLEDGEMENT

None.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

Received:	01-July-2024	**Manuscript No:**	IPAP-24-21258
Editor assigned:	03-July-2024	**PreQC No:**	IPAP-24-21258 (PQ)
Reviewed:	17-July-2024	**QC No:**	IPAP-24-21258
Revised:	22-July-2024	**Manuscript No:**	IPAP-24-21258 (R)
Published:	29-July-2024	**DOI:**	10.36648/2469-6676-10.07.64

Corresponding author Daniel Cheeow, Department of Educational Psychology, University of Cape Town, Denmark, E-mail: chee.daniel@gmail.com

Citation Cheeow D (2024) Immune Dysregulation in Bipolar Disorder: Exploring the Pathophysiological Link. Act Psycho. 10:64.