Neuroinflammation and Mood: Dissecting the Role of Eotaxin-1 in Depression

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Depression is a complex, multifactorial disorder with a pathophysiology that is still not fully understood. Recent research suggests that neuroinflammation and disruption of the blood-brain barrier may play a significant role in the development of depressive symptoms. Chemokines, particularly eotaxin-1, have emerged as a key component linking peripheral immune activation to central nervous system changes. Elevated levels of eotaxin-1 have been associated with reduced neurogenesis, cognitive decline, and depression-like behavior. Stress and infections can damage the blood-brain barrier, allowing peripheral chemokines such as eotaxin-1 to enter the brain or be locally expressed, potentially triggering neuroinflammation. Overall, findings suggest that chemokine signaling following blood-brain barrier disruption may play a crucial role in the pathogenesis of depression and could offer potential targets for therapeutic intervention.
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INTRODUCTION

Major depressive disorder (MDD) is a condition influenced by both genetic predisposition and environmental stressors (Buch and Liston, 2021; Park et al., 2019; Penner-Goeke and Binder, 2019). While its pathomechanism remains incompletely understood, growing evidence implicates neuroinflammatory processes in its development (Beurel et al., 2020; Charo and Ransohoff, 2006). In particular, the disruption of blood-brain barrier (BBB) integrity may be a key mediator linking peripheral inflammation to central nervous system (CNS) dysfunction and depressive symptoms.

Different stressors, including psychological stress and infections, can impair BBB function (Chai et al., 2014; Gal et al., 2023, 2024), potentially allowing peripheral chemokines to enter the CNS or triggering their production within the brain (Del Guerra et al., 2013). Among these, the role of chemokines in modulating neuroimmune interactions and neuronal function has gained attention; however, the exact molecular pathways remain to be fully revealed.

CHEMOKINES AND NEUROIMMUNE SIGNALING

Chemokines are a family of structurally related, low-molecular-weight cytokines that direct immune cell migration through chemotaxis. They play crucial roles in immune system monitoring, inflammation, tissue development, and repair (Charo and Ransohoff, 2006;

Zlotnik and Yoshie, 2012). Their effects are mediated by binding to G protein-coupled receptors on target cells, initiating intracellular signaling pathways that alter cell behavior and promote leukocyte extravasation to sites of injury or infection (Réaux-Le Goazigo et al., 2013).

Based on conserved cysteine motifs, chemokines are classified into four subfamilies: CC, CXC, CX3C, and XC. Within the CC subfamily, Eotaxins, comprising eotaxin-1 (CCL11), eotaxin-2 (CCL24), and eotaxin-3 (CCL26), are outstanding for their strong eosinophil-attracting capacity. These ligands primarily act via the CCR3 receptor, expressed not only on eosinophils and basophils but also on certain Th2 cells and CNS-resident cells such as astrocytes, microglia, and neurons (Menzies-Gow et al., 2002; Baruch et al., 2013).

CHEMOKINES IN THE CENTRAL NERVOUS SYSTEM

Chemokines and their receptors are constitutively expressed in the CNS, where they contribute to neurodevelopment, synaptic transmission, and neurogenesis (de Haas et al., 2007; Teixeira et al., 2018). Through neuron-glia signaling, chemokines influence neuroplasticity, cell survival, and the immune response. Their dysregulation has been implicated in various neuropsychiatric disorders, including depression (Yuan et al., 2019).

Among these, Eotaxins, especially CCL11, have emerged as key players in neuroinflammation and neurodegeneration (Ivanovska et al., 2020; Pease and Williams, 2013). Eotaxin production is induced by cytokines such as IL-4 and IL-13, and while traditionally associated with allergic responses, their presence in the brain suggests a broader function in CNS pathology.

Elevated CCL11 levels have been linked to autoimmune diseases, vascular inflammation, and neuropsychiatric conditions, including depression and age-related cognitive decline (Teixeira et al., 2018). Given its ability to affect BBB integrity and activate glial cells, CCL11 is a strong candidate for mediating inflammatory pathways in MDD.

EOTAXIN-1 AND DEPRESSION

Elevated peripheral CCL11 levels have been observed in individuals with MDD and are associated with cognitive deficits and mood disturbances. In animal models, CCL11 induces depression-like behaviors and suppresses hippocampal neurogenesis, a region critical for emotion regulation and memory.

The BBB plays a pivotal role in CNS homeostasis by regulating the entry of peripheral substances. Tight junction proteins, such as claudin-5, maintain this barrier. However, chronic stress, infection, or systemic inflammation can downregulate these proteins and compromise BBB integrity (Gal et al., 2023), facilitating the infiltration of proinflammatory mediators, including CCL11 (Kealy et al., 2020).

CCL11 may contribute to depression via:

1. Peripheral-central immune crosstalk. Stress activates peripheral immune responses, increasing IL-6 and CCL11 levels (Maes et al., 2019).
2. BBB disruption. Inflammatory signals weaken tight junctions, allowing immune molecules to enter the brain (Zhang et al., 2025).
3. Glial activation. CCL11 upregulates chemokine receptors on astrocytes and microglia, increasing oxidative stress and altering neuronal function (Parajuli et al., 2015).

These processes collectively impair neuroplasticity, reduce neurogenesis, and contribute to depressive symptomatology.

BBB DISRUPTION, VIRAL INFECTIONS, AND NEUROINFLAMMATION

The blood–brain barrier (BBB) plays a critical role in maintaining central nervous system (CNS) homeostasis by tightly regulating the exchange of molecules between the bloodstream and the brain. Its selectivity is essential for protecting the brain from toxins, pathogens, and peripheral immune cells. However, this protective barrier can be compromised by chronic stress and systemic infections, which have been shown to weaken BBB integrity and increase its permeability, thereby facilitating neuroinflammatory responses and behavioral disturbances (Dion-Albert et al., 2022a; Nazarinia et al., 2022, Gal et al., 2023, 2024).

Viral infections are potent disruptors of BBB function. They can directly infect endothelial cells or indirectly affect the barrier through the release of pro-inflammatory mediators. Increased BBB permeability allows peripheral immune cells, cytokines, and viral particles to access the CNS, where they can activate resident glial cells (microglia and astrocytes), promote cytokine production, and induce oxidative stress. This cascade of neuroimmune interactions can lead to neuronal damage, synaptic dysfunction, and ultimately contribute to the development of cognitive deficits, emotional dysregulation, and psychiatric symptoms, including depression and anxiety (Kempuraj et al., 2024).

Chronic stress can induce a state of immunosuppression, which may impair the elimination of viral pathogens and facilitate their persistence within the host. This prolonged viral presence may lead to a state of chronic low-grade inflammation that alters brain function over time. Such a scenario could create pro-depressogenic condition, especially in genetically vulnerable individuals.

Furthermore, epidemiological evidence suggests that severe viral infections during early life, such as those requiring hospitalization, may significantly increase the risk of developing depression, anxiety, or self-harming behaviors in adolescence (Leone et al., 2022). These long-term effects are thought to result from sustained neuroimmune activation, disrupted neurodevelopment, and potentially enduring epigenetic modifications affecting stress reactivity and emotional regulation later in life.

GENETIC EVIDENCE LINKING INFLAMMATION, BBB INTEGRITY, AND DEPRESSION

Animal models reveal that chronic stress impairs BBB integrity by downregulating claudin-5 expression, increasing permeability and neuroinflammation (Menard et al., 2017; Dion-Albert et al., 2022b). These changes correlate with depressive-like behaviors, reversible by antidepressants. Post-mortem studies of individuals with depression who died by suicide similarly show reduced CLDN5 expression.

Genetic studies further support this link. A CLDN5 polymorphism (rs885985) affects protein isoform expression, potentially disrupting BBB function (Cornely et al., 2017). Similarly, a functional IL6 variant (rs1800795) is associated with higher IL-6 levels under stress, increasing depression risk (Kovacs et al., 2016).

A genome-wide environment interaction study (GWEIS) in over 100,000 UK Biobank participants identified variants in CSMD1 and PTPRD, both involved in BBB maintenance, as interacting with life stress to influence depression (Gal et al., 2024). Another study in ~270,000 individuals found that recent stress interacts with genetic variants in CLDN5 and IL-6, altering BBB function and inflammatory responses, particularly in men (Gal et al., 2023).

Emerging evidence also highlights the role of CCL11, a chemokine with neurotoxic and pro-inflammatory properties, in depression. Although genome-wide association studies have not yet identified a direct link between CCL11 gene and depression, its expression is closely influenced by stress-related pathways, IL-6 signaling, and blood-brain barrier integrity. Elevated CCL11 levels, seen in both human and animal models, are associated with impaired neurogenesis and depressive phenotypes. Its effects are potentiated in conditions of BBB disruption, such as reduced CLDN5 expression, allowing CCL11 to access the brain or become locally upregulated. These interactions suggest a genetically modulated CCL11, BBB, neuroinflammation axis, warranting further investigation.

Taken together, current evidence supports an indirect yet potentially significant relationship between CCL11, CLDN5, and IL6 in depression. All three play key roles in maintaining BBB integrity and regulating neuroinflammation; however, their genetic and mechanistic interactions remain largely unexplored. Understanding their coordinated role, at the genomic, cellular, and systems level, may uncover critical pathways involved in stress-induced depression.

Our future studies aim to investigate the genetic architecture and interaction of CCL11, IL-6, and CLDN5, to clarify their contribution to depression vulnerability and potential sex-specific effects. These findings may further underscore the genetic basis of stress sensitivity and highlight inflammation-related mediators and BBB regulators as promising therapeutic targets.

CONCLUSION

Emerging evidence from molecular, animal, and human studies supports a key role for neuroinflammation and blood-brain barrier dysfunction in the pathogenesis of depression. Among inflammatory mediators, eotaxin-1 has gained increasing attention for its potential to bridge peripheral immune activation with central neuroimmune responses, contributing to impaired neurogenesis and altered neuronal function. These effects are amplified in the context of stress-induced BBB disruption, allowing peripheral chemokines to exert direct influence on the brain.

While current findings highlight the functional relevance of CCL11, the genetic underpinnings of its role in depression remain poorly understood. Future studies may focus on the genetic architecture and polymorphisms of key genes: CCL11, IL-6, and

CLDN5, aiming to clarify how these genes interact and influence neuroimmune signaling and BBB integrity. By integrating genetic, molecular, and systems-level analyses, we hope to advance understanding of the pathways linking stress, immune signaling, and depression, and contribute to the development of targeted, personalized interventions for those at risk.

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Neuroinflammáció és Hangulat: az Eotaxin-1 Depresszióban Betöltött Szerepének Feltárása

A depresszió egy összetett, több tényező által befolyásolt kórkép, amelynek patofiziológiája még mindig nem teljesen tisztázott. Újabb kutatások szerint a neuroinflammáció és a vér-agy gát (BBB) sérülése jelentős szerepet játszhat a depresszív tünetek kialakulásában. A kemokinek, különösen az eotaxin-1, kulcsszereplőként jelennek meg a perifériás immunaktiváció és a központi idegrendszeri változások közötti kapcsolatban. Megemelkedett eotaxin-1 szintet figyeltek meg csökkent neurogenezissel, kognitív hanyatlással és depresszióhoz hasonló viselkedéssel összefüggésben. A stressz és fertőzések károsíthatják a BBB-t, lehetővé téve a perifériás kemokinek, köztük az eotaxin-1 bejutását az agyba, illetve helyi expressziójukat, ami neuroinflammációt idézhet elő. Az összesített eredmények alapján a BBB sérülését követő kemokin jelátvitel kiemelt szerepet játszhat a depresszió patogenezisében, és potenciális terápiás célpontokat is kínálhat.

Kulcsszavak: vér-agy gát, gyulladás, kemokinek, depresszió