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The Physiopathological Contributions of Gut Functions and White Adiposity to Neuroinflammation

Aquilina Carbonara*

Department of Neurovascular Pathophysiology, University Complutense of Madrid, 28040 Madrid, Spain

Introduction

Neuroinflammation, the inflammatory response within the Central Nervous System (CNS), has garnered significant attention for its role in various neurological disorders, including Alzheimer's disease, Parkinson's disease, multiple sclerosis and depression. While the CNS has long been the primary focus of neuroinflammation research, growing evidence highlights the critical roles of peripheral systems, such as gut function and White Adipose Tissue (WAT), in modulating this inflammatory response. The gut-brain axis and the adipose-brain axis, once considered distinct entities, are now recognized as interconnected pathways contributing to the complex physiopathology of neuroinflammation. Understanding the mechanisms by which gut functions and WAT influence neuroinflammatory processes could open new avenues for targeted interventions in neurodegenerative and psychiatric disorders [1].

Description

The gut plays a pivotal role in maintaining overall physiological homeostasis, serving as a site for digestion, nutrient absorption and immune modulation. The gut-brain axis represents the bidirectional communication between the gastrointestinal tract and the CNS, mediated through neural, hormonal and immune pathways. Dysregulation of this axis, often stemming from gut dysfunctions such as altered microbiota composition (dysbiosis), increased intestinal permeability, or chronic gastrointestinal inflammation, has been implicated in neuroinflammatory processes.

Gut dysbiosis, characterized by an imbalance in microbial populations, is a primary driver of systemic and neuroinflammation. A healthy gut microbiota produces beneficial metabolites such as Short-Chain Fatty Acids (SCFAs), which regulate the immune system, maintain intestinal integrity and modulate the Blood-Brain Barrier (BBB). However, dysbiosis leads to the production of pro-inflammatory microbial metabolites, including Lipopolysaccharides (LPS), which can translocate into the systemic circulation via a compromised intestinal barrier. These endotoxins activate immune cells, such as macrophages and monocytes, leading to the release of pro-inflammatory cytokines like Tumor Necrosis Factor-Alpha (TNF) and Interleukin-6 (IL-6). Such systemic inflammation can breach the BBB, promoting neuroinflammation and exacerbating neurological pathologies [2,3].

Increased intestinal permeability, often referred to as "leaky gut," is another critical factor linking gut dysfunction to neuroinflammation. The intestinal epithelium, held together by tight junction proteins, forms a physical barrier preventing the translocation of harmful substances into the systemic circulation. Disruption of these tight junctions, triggered by factors like poor diet, stress, or infections, allows microbial products and toxins to enter the bloodstream. This breach elicits an immune response that amplifies systemic inflammation, with downstream effects on the CNS. The subsequent infiltration of immune cells into the brain and activation of microglia, the resident immune cells of the CNS, perpetuates a cycle of neuroinflammation that can damage neurons and disrupt neural circuits.

White adipose tissue, commonly associated with energy storage, also plays a critical role in modulating immune responses and systemic inflammation. As an endocrine organ, WAT secretes various adipokines, including leptin, adiponectin and pro-inflammatory cytokines, which influence both peripheral and central immune activities. In obesity or metabolic syndrome, WAT undergoes pathological remodeling, characterized by hypertrophy of adipocytes, increased macrophage infiltration and the secretion of pro-inflammatory mediators. This state of chronic low-grade inflammation, termed "metaflammation," has profound implications for neuroinflammatory processes. Leptin, an adipokine primarily involved in regulating energy homeostasis, is elevated in obesity and has pro-inflammatory properties. High leptin levels promote the activation of peripheral immune cells and the secretion of inflammatory cytokines, which can cross the BBB and activate microglia [4,5].

Conclusion

Therapeutic strategies targeting gut function and WAT offer promising avenues for mitigating neuroinflammation. Probiotics, prebiotics and dietary interventions aimed at restoring gut microbiota composition have shown potential in reducing systemic and CNS inflammation. For instance, supplementation with SCFA-producing bacteria or dietary fibers can enhance intestinal barrier integrity, reduce systemic LPS levels and modulate microglial activity in the brain. Similarly, interventions targeting WAT inflammation, such as weight loss, exercise and pharmacological modulation of adipokines, may help attenuate neuroinflammatory processes. Combined therapies addressing both gut and WAT dysfunctions could provide synergistic benefits in reducing systemic and CNS inflammation, ultimately improving outcomes in neuroinflammatory diseases.

The physiopathological contributions of gut functions and white adiposity to neuroinflammation underscore the complexity of systemic influences on CNS health. Gut dysfunctions, characterized by dysbiosis, increased intestinal permeability and chronic inflammation, serve as primary drivers of systemic and neuroinflammation. Simultaneously, WAT, particularly in its pathological state during obesity, amplifies systemic inflammation through the secretion of pro-inflammatory adipokines and cytokines. The interplay between these systems, mediated through metabolites, endotoxins and immune signaling pathways, creates a multidimensional framework for understanding neuroinflammatory processes.

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Conflict of Interest

None.

***Address for Correspondence:** Aquilina Carbonara, Department of Neurovascular Pathophysiology, University Complutense of Madrid, 28040 Madrid, Spain, E-mail: carbonaraquilina@ila.es*

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References

1. Ordovas-Montanes, Jose, Seth Rakoff-Nahoum, Siyi Huang and Lorena Riol-Blanco, et al. "The regulation of immunological processes by peripheral neurons in homeostasis and disease." *Trends Immunol* 36 (2015): 578-604.
2. Godinho-Silva, Cristina, Filipa Cardoso and Henrique Veiga-Fernandes. "Neuro-immune cell units: A new paradigm in physiology." *Annu Rev Immunol* 37 (2019): 19-46.
3. Shepherd andrew J., Lorna J. Beresford, Eric B. Bell and Jaleel A. Miyan. "Mobilisation of specific T cells from lymph nodes in contact sensitivity requires substance P." *J Neuroimmunol* 164 (2005): 115-123.
4. Fishman, Renata B. and John Dark. "Sensory innervation of white adipose tissue." *Am J Physiol* 253 (1987): R942-R944.
5. Wozniak, Susan E., Laura L. Gee, Mitchell S. Wachtel and Eldo E. Frezza. "Adipose tissue: The new endocrine organ? A review article." *Dig Dis Sci* 54 (2009): 1847-1856.

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