The article *"Astrocyte-mediated hippocampal damage in the pathogenesis of dysexecutive syndrome following COVID-19"* presents a narrative review of the neurobiological mechanisms underlying cognitive impairment in long-COVID. It focuses on how SARS-CoV-2 affects the hippocampus, a brain region central to memory, emotional regulation, and executive functioning.

The authors argue that neuroinflammation is a key driver of post-COVID cognitive symptoms. SARS-CoV-2 infection activates astrocytes and microglia, disrupts the blood–brain barrier, and induces cytokine-mediated toxicity. These processes impair neurogenesis and neuroplasticity in the hippocampus, leading to structural and functional alterations.

Astrocytes play a central role in this model. Normally responsible for maintaining neural homeostasis and supporting synaptic activity, their dysregulation contributes to neuronal damage and altered brain signaling. The review integrates evidence from clinical observations and experimental studies showing hippocampal hypometabolism and degeneration in COVID-19 patients.

Clinically, these mechanisms manifest as dysexecutive syndrome, characterized by deficits in attention, planning, working memory, and cognitive flexibility. Patients often also report fatigue, apathy, and depressive symptoms. The authors highlight the importance of distinguishing neuroinflammation-driven mood symptoms from primary psychiatric disorders, as this has implications for diagnosis and treatment.

The review also discusses emerging evidence on the protective role of COVID-19 vaccination, suggesting it may mitigate neuroinflammatory processes and support hippocampal recovery. However, the authors emphasize that current evidence remains limited and longitudinal studies are needed.

Overall, the paper underscores the importance of targeting neuroinflammation and promoting neuroplasticity in managing long-COVID cognitive symptoms. It calls for integrated therapeutic approaches, including pharmacological strategies and cognitive rehabilitation, to address the persistent neurological sequelae of SARS-CoV-2 infection.