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Peer Review

Review of: "COVID-19 and Alzheimer's Disease, Closely Related?"

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This short communication explores a timely and biologically plausible hypothesis linking COVID-19 and Alzheimer's disease (AD), focusing on shared genetic susceptibility (APOE4, OAS1), neuroinflammatory pathways, and the potential amyloidogenic properties of the SARS-CoV-2 spike protein. The integration of infectious, immunological, and neurodegenerative frameworks is conceptually coherent and reflects current interest in post-COVID neurological sequelae.

The discussion of APOE4 as a shared risk factor for both severe COVID-19 and AD is well grounded in emerging literature. Likewise, the inclusion of OAS1 and interferon-responsive transcriptional networks provides mechanistic depth and strengthens the argument for overlapping immune-genetic vulnerability. The proposal to longitudinally follow COVID-19 patients with anosmia—particularly APOE4 carriers—for early cognitive changes is clinically relevant and hypothesis-generating.

However, the manuscript remains largely speculative and would benefit from clearer differentiation between established associations and mechanistic extrapolations. For example, while the spike protein's amyloidogenic potential has been demonstrated in vitro, direct clinical or neuropathological evidence linking this phenomenon to AD-like pathology in vivo remains limited. Additionally, the conclusion could be framed more cautiously, emphasizing that current data suggest biological convergence rather than a confirmed causal relationship.

Overall, this article provides a concise and thought-provoking perspective on potential neurodegenerative consequences of COVID-19. Greater emphasis on longitudinal clinical data and neuropathological validation would further strengthen its scientific impact.

Declarations

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