

## Aging Adults with COVID-19 Infection and Alzheimer's Disease

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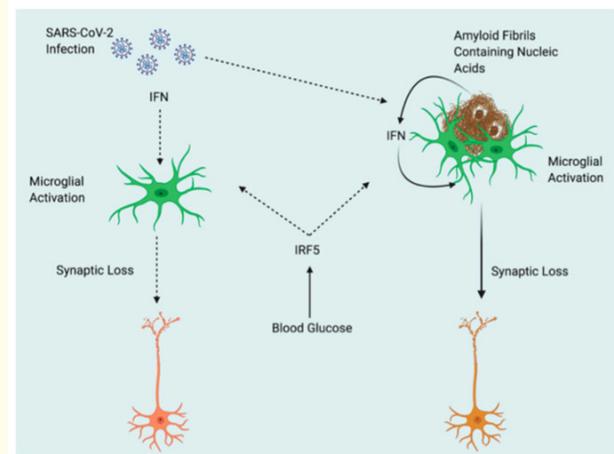
Received: September 22, 2022

Published: October 01, 2022

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SARS-CoV-2 (COVID-19) demonstrated decrease in the consciousness level [1]. Patients with Alzheimer's disease (AD) experienced longer lockdown periods to protect themselves from being infected with COVID-19 or to recover from COVID-19 infection [2,3] that contributed to direct results (worse behavioral interferences and more severe neuropsychiatric symptoms) [3]. People with the mid-60's of ages frequently present the most frequent type of AD that is not produced one gene only (apolipoprotein E gene (APOE)) [4]. Several hypothetically potential pathways are entry via the olfactory system [5,6], blood-brain barrier (BBB) [7,8], and COVID-19-infected-immune cell infiltration [9-11]. Smooth muscle cells in the brain blood vessels express angiotensin-converting-enzyme-2 receptors same as in the pulmonary alveolar cells [12]. Diabetes type 2 (T2D) is a risk factor for both AD and SARS-CoV-2 (COVID-19) via increased activity of interferon-regulatory-factor 5 (IRF5) (Figure 1) [13].

In conclusion, currently, there is lacking proved data that SARS-CoV-2 (COVID-19) is associated with AD development despite several proved studies of cognitive impairment in SARS-CoV-2 (COVID-19) patients. This question is urgently needed.



**Figure 1:** Demonstrating potential Interactions Between “SARS-COV-2” and AD and possibly predisposed by Type 2 diabetes (T2D). Elevations in blood glucose resulting from T2D may exacerbate pathology in both AD and COVID-19, or comorbidity of the two, through increased activity of interferon regulatory factor 5 (IRF5). Type I interferons (IFN) mediate inflammation after viral infection and in response to nucleic acid containing amyloid fibrils, finally contributing to synaptic loss. Amyloid fibrils may entrap viral particles, contributing to further enhancement of IFN response. Solid line arrows indicate proven mechanisms, dotted line arrows indicate theoretical mechanisms [13].

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