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REASSESSMENT OF THE UNDERLYING MECHANISMS THAT CONTRIBUTE TO THE NEUROLOGICAL DISORDERS LINKED TO LONG-TERM COVID-19

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We read with great interest the article by Graban and his colleagues [1] regarding the neurological effects of long COVID-19. A possible explanation for the neurological difficulties was proposed by the authors as cell fusion. Furthermore, the authors highlight that there is a considerable percentage of people feeling weariness and cognitive dysfunction, which includes lower attention span, short-term memory loss, and overall memory impairment. Moreover, recent scientific findings suggest that there are increasingly severe problems that follow these symptoms. There are considerable disparities in the intelligence quotient (IQ) between those who have been infected with SARS-CoV-2 and those who have not contracted the illness. Even a little illness can result in a decrease in IQ of around three points [2]. Nevertheless, it is important to note that most of these investigations were carried out in the first stage of the pandemic before the availability of vaccinations or antiviral medications and the permanence of these cognitive deficits is likewise uncertain. Research has previously demonstrated that certain individuals have structural brain abnormalities and other brain imaging abnormalities that are commensurate with the changes observed in the brains of typical individuals during around seven years of ageing [3]. Despite the protective blood-brain barrier, inflammation can compromise its integrity, leading to the infiltration of proinflammatory cytokines. This phenomenon may help elucidate some symptoms observed following a COVID-19 infection [4]. One further thing that has been discovered is that people who have been infected with SARS-CoV-2 have abnormally low levels of cortisol, which is a hormone that is necessary for maintaining healthy cognitive functioning. The reduction in serotonin levels caused by COVID-19 infection and inflammation induced by type I interferon happens through three mechanisms: decreased absorption of tryptophan, the compound that precedes serotonin, in the intestines; increased activation of platelets and decreased platelet count, affecting serotonin storage; and increased serotonin turnover mediated by monoamine oxidase (MAO). Decreasing serotonin levels in the peripheral areas of the body obstructs the functioning of the vagus nerve, resulting in decreased reactions and memory in the hippocampus. These findings offer a potential rationale for the neurocognitive symptoms linked to the continuous existence of a virus in long COVID-19, which might also apply to other post-viral disorders. As

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a result, a reduction in peripheral serotonin hampers the activity of the vagus nerve, leading to a decrease in the functioning of the hippocampi and memory. These findings offer a potential explanation for the neurocognitive symptoms that are linked with viral persistence in long COVID-19 [5–8]. In addition to this, it can have a detrimental effect on memory and attention, as well as create persistent fatigue and alter normal sleep habits.

Article information and declarations

Author contributions

All authors contributed equally to the creation of the manuscript.

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Conflict of interest

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Supplementary material

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