





Letter to the Editor

Reply Regarding “Correlation Between Post-traumatic Stress Disorder and SARS-CoV-2 Infection”

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In the correspondence of “Correlation between post-traumatic stress disorder and Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection”, three important points were raised, namely: (1) severe SARS-CoV-2 infection (SC2I) may trigger post-traumatic stress disorder (PTSD) by multiple brain-related pathophysiological mechanisms; (2) PTSD may contribute to the pathophysiology of SC2I through inflammatory or neurohormonal pathways; and (3) vaccination may decrease SARS-CoV-2 infectivity and the severity of resultant disease, thus, affecting both exposure and outcome.

These points are valid. All of these influences are possible, moreover, they are certainly probable. When Genome-Wide Association Studies (GWAS) data become available as part of more phenotypically detailed SARS-CoV-2 datasets, we will certainly follow up with analyses aimed at the proposed mechanistic dissection of the effects of SC2I on neuropsychiatric outcomes.

For now, we acknowledge that the conclusions of our MR study [1] are necessarily limited to the genetic components of each of the studied exposures, although its environmental components remain beyond its scope [2]. Causal inferences presented in our paper should be used as part of triangulation efforts covering multiple sources of evidence rather than a statement in support of unidirectional influence to the exclusion of any effect exerted by SC2I on subsequent illnesses of a neuropsychiatric nature.

Author Contributions

FZ integrated the comments and responded. AB and FZ consulted the literatures. HC, YS, and AB drafted the manuscript. FZ, YS, LF, and HC revised the manuscript. All authors read and approved the final manuscript.

Ethics Approval and Consent to Participate

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Conflict of Interest

The authors declare no conflict of interest.

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