
Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

We declare no competing interests.

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We read with great interest the article published in *The Lancet Psychiatry* by Jonathan Rogers and colleagues.[1] As readers with an intensive care background, we noted that the authors state in their discussion that severe acute respiratory distress syndrome (ARDS) is a key feature of COVID-19 illness. This point is contentious, with Gattinoni and colleagues[2] highlighting that although severe COVID-19 might meet the ARDS Berlin criteria, it cannot simply be categorised under this syndrome alone. Clinically, often a dissociation exists between severe hypoxemia and near normal pulmonary compliance, which is not characteristic of ARDS. Treatment pathways might therefore be different to ARDS and it is difficult to draw direct comparison in relation to psychiatric outcomes at 1 year.

We agree that to delineate causation between COVID-19 and subsequent psychiatric outcomes with the current evidence is difficult given the poor quality in methodology in some of the studies cited. A high level of evidence for psychological co-morbidity already existed before COVID-19—eg, post-traumatic stress disorder (PTSD) following general admission to intensive care unit (ICU) has a point prevalence of 34%,[3] which is similar to the overall point prevalence of 32·2% for PTSD in severe acute respiratory syndrome coronavirus 2 cited by the authors.

Although the authors acknowledge that the cause of psychiatric consequences is multifactorial, a more robust exploration of the role of medical interventions is required within an ICU context, including duration of sedation and the results of the severity of illness scoring system such as APACHE II. The type of sedation also has a key role in delirium, with many ICUs in the UK resorting to second-line agents during the COVID-19 pandemic due to resource constraints.[4] These second-line agents include long-acting benzodiazepines and opiates that have longer context-sensitive half-lives compared to first-line. Further qualitative data are required to examine COVID-19-related factors, such as fears that patients can experience on seeing staff in full personal protective equipment and challenges with communication following tracheostomy.

Categorisation of COVID-19 patients admitted to ICU would be useful, given these patients are not a homogenous group; sub-group classification into high dependency care patients and intensive care patients is one suggestion. Our preliminary local experience of using the acute stress disorder scale (which has a reasonable positive predictive value for future PTSD) as a screening tool,[5] suggests that patients with COVID-19 in intensive care might have worse psychological co-morbidity compared with those in high dependency care.

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## Authors' reply

We are grateful for the letters received regarding our Article,[1] which highlight the interest in this field and raise relevant clinical hypotheses. These letters illustrate a fundamental question: does infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) lead to psychiatric and neuropsychiatric morbidity merely because COVID-19 is a severe illness or because of specific factors related to this infection?

There are numerous—but still comparatively rare—examples of specific neuropsychiatric presentations of COVID-19 being collated in an online blog for the *Journal of Neurology, Neurosurgery & Psychiatry*.[2] Yi-Min Wan and colleagues are correct that anosmia requires more