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.

Olfactory dysfunction and COVID-19

In their meta-analysis, published in *The Lancet Psychiatry*, Jonathan Rogers and colleagues[1] highlighted common neuropsychiatric symptoms in patients with severe acute respiratory syndrome (SARS-CoV) and Middle East respiratory syndrome coronavirus (MERS-CoV) compared with COVID-19 and emphasised the need to recognise these potential problems in the management of COVID-19. Sommer and Bakker[2] called for caution when making direct comparisons between these coronaviruses because chronic sequelae of COVID-19 are still unknown and confounding factors cannot be excluded.

We would like to draw attention to an important factor that was not alluded to in the meta-analysis.[1] Olfactory dysfunction (anosmia and hyposmia) has a strikingly high prevalence (60–70%) in patients with severe acute respiratory syndrome 2 (SARS-CoV-2) compared with other coronaviruses.[3] Olfactory dysfunction was not reported as a symptom during past SARS-CoV and MERS-CoV outbreaks. In our community care facilities, we have seen patients with SARS-CoV-2 and pure olfactory dysfunction without nose block or other signs of respiratory infections. These individuals were anxious about permanently losing their sense of smell, and in some cases their sense of taste. The anxiety is understandable because olfactory dysfunction usually occurs early during infection with SARS-CoV-2 and can be severe, with some patients experiencing severe dyspnoea.[3]

In animal models, disruption of olfactory pathways and experimental removal of the olfactory bulb can lead to neurochemical and behavioural changes seen in depressive states that are reversible with antidepressant drugs.[4] In humans, olfactory dysfunction has been reported in patients with depression and cognitive impairment.[4,5] In a study of 6783 adults, better olfactory performance was associated with better cognitive performance.[5]

The potential neuropsychiatric burden associated with cranial nerve problems, such as olfactory and gustatory dysfunctions, is still largely unexplored in the COVID-19 pandemic. Olfactory dysfunction can lead to both short-term and long-term neurological and neuropsychiatric complications that need to be investigated. Recognising neuropsychiatric sequelae of olfactory dysfunction and other neurological complications, such as stroke as a result of COVID-19, and facilitating closer longitudinal follow up of patients with structural or functional brain damage will improve their quality of care and mental wellbeing.

We declare no competing interest.

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Psychiatric and neuropsychiatric syndromes and COVID-19

The systematic review and meta-analysis by Rogers and colleagues[1] of acute and post-illness psychiatric and neuropsychiatric presentations of individuals with suspected or laboratory-confirmed coronavirus infection was much needed. However, it does not address one of the key susceptible groups with high rates of neuropsychiatric symptoms—people with dementia.

People with dementia have an increased risk of delirium,[2] and might also be particularly sensitive to the potential neurotropic effect of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).[3] 98% of patients with dementia experience neuropsychiatric symptoms over the course of their disease.[4] These symptoms might be exacerbated during the acute or post-illness phases of infection with SARS-CoV-2 as a result of the virus itself and related social and environmental effects. Importantly, inappropriate management of neuropsychiatric symptoms in people with dementia could lead to substantial excess morbidity and deaths.

It is essential to gather further evidence regarding the effect of delirium on individuals with dementia who are infected with SARS-CoV-2, the broader impact and management of neuropsychiatric symptoms, and the different approaches to physical distancing. Optimising management and preventing inappropriate and potentially harmful management strategies are all the more urgent given people with dementia are at an increased risk of mortality and strokes associated with the antipsychotic medications[5] that are too often used for managing delirium and neuropsychiatric symptoms.

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.

We declare no competing interests.

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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