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activities that could help to explain why different activities differentially activate various mechanisms of action.

We declare no competing interests.

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Neuropsychiatric disorders and COVID-19

We read with interest the Article by Maxime Taquet and colleagues that reports on the incidence rates of anxiety disorders after a COVID-19 diagnosis,[1] and we noted from the appendix that this outcome includes codes F40–F48 of the ICD-10. Given the known occurrence of developing post-traumatic stress disorder after admission to an intensive therapy unit,[2] we wondered if the authors had considered re-analysing their data to assess the incidence of post-traumatic stress disorder in survivors of COVID-19, including both those treated in intensive therapy units and those treated elsewhere?

It is very concerning that 4·72% of patients with COVID-19-related encephalopathy received a first diagnosis of dementia within 6 months. We note that the authors have included F01–F03, G30, G31, and G31·83 when defining dementia, and we wondered whether the authors were able to provide more information on the incidence rates for these different types? We believe that it would be useful to know if a particular type of dementia is observed, both to help understand the pathophysiology and to help assess patients.

In the appendix of the Article, the authors state that "for chronic illnesses, only first diagnoses were counted".[1] In the case of dementia, were patients with a history of mild cognitive impairment (eg, F06·7) or delirium (F05) excluded? This clarification would help to establish whether COVID-19-related encephalopathy is associated with the rapid onset of dementia, or whether these patients were already a group at risk of developing dementia, considering that delirium and mild cognitive impairment can act as markers of vulnerability to dementia and can themselves lead to dementia.[3,4]

We wondered if there were any data reporting on the psychiatric outcomes in patients with asymptomatic COVID-19 (given that it is suspected that these patients account for at least one-third of all COVID-19 cases[5]), or in patients with only mild symptoms? This information would be useful for future planning of mental health services.

Finally, given that the estimated incidence of a neurological or psychiatric diagnosis in the 6 months after a COVID-19 diagnosis was 33·62%, we would propose a number of measures. First, we would suggest that questions about past COVID-19 infection become a standard part of clinical history taking. Second, we would propose that psychiatrists become an integral part of long COVID clinics nationwide. Finally, we would like to introduce the notion of reverse redeployment, in which health-care workers from general medicine might support mental health services, given the expected increased demand after this pandemic.

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Maxime Taquet and colleagues reported an increased incidence of neurological and psychiatric disorders in patients diagnosed with COVID-19 (ie, group 1) compared with two matched control cohorts: patients diagnosed with influenza (ie, group 2) and patients diagnosed with any respiratory tract infection, including influenza (ie, group 3).[1] In my opinion, having two control groups containing patients with influenza is a shortcoming of the study, and patients with influenza in group 3 should have been transferred to group 2. The authors suggested that the potential mechanisms for the association of neurological and psychiatric disorders with COVID-19 include viral invasion of the CNS, hypercoagulable states, neural effects of the immune response, and psychological and other implications of a COVID-19 diagnosis for people with common psychiatric disorders (eg, mood and anxiety disorders).[1] Other mechanisms, however, should be considered. One mechanism is that morbidity could have increased if patients did not attend necessary medical appointments during the COVID-19 pandemic because of decreased access to medical services during lockdown and semi-lockdown periods or patients' anxiety at getting infected.

The authors did not report information about the anti-COVID-19 medications that were given to the patients in group 1 during or after

hospitalisation. Neurotoxic drugs that are used to treat COVID-19 include daptomycin, linezolid, lopinavir, ritonavir, hydroxychloroquine, cisatracurium, clindamycin, tocilizumab, and glucocorticoids.[2] Neuropathy or myopathy in patients with COVID-19 requiring treatment in intensive care units can also result from bedding (ie, compression neuropathy), compartment syndrome, artificial nutrition, infection, electrolyte disorder, or sepsis (ie, critically ill neuropathy or myopathy).[2] Anti-COVID-19 drugs can also be myotoxic, for example, chloroquine is associated with myopathy and myasthenia.[3]

Taquet and colleagues distinguished Guillain-Barre syndrome and nerve root disorders on the basis of ICD codes in the database. However, the ICD system is incomplete: Guillain-Barre syndrome is a classic nerve root disorder and some doctors might encode Guillain-Barre syndrome under nerve root disorders, whereas other doctors might use the ICD code for Guillain-Barre syndrome. Therefore, these two groups should be assessed together.

The authors excluded patients with a diagnosis of COVID-19 or a positive test for SARS-CoV-2 from the control cohorts. However, it is not known whether all patients in the control groups were actively tested for SARS-CoV-2 infection: patients from the control groups need to be SARS-CoV-2 negative to serve as controls, which can be ensured only by systematic PCR tests in each patient.

Major outcomes that were not considered in the evaluation included cerebral vasculitis, venous sinus thrombosis, seizures or epilepsy, cranial nerve affection, myelitis, acute disseminated encephalomyelitis, and headache.[4] Particularly, venous sinus thrombosis should have been included in the evaluation as COVID-19 is associated with an increased risk of thrombosis.[5] The authors also did not consider hyperlipidaemia or atherosclerosis as pre-existing comorbidities: these might worsen during COVID-19 and might contribute to the increased incidence of neuropsychiatric disease.

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We read with interest the Article by Maxime Taquet and colleagues[1] in *The Lancet Psychiatry* that evaluated a large population (n=236 379) for neurological and psychiatric complications of COVID-19 via analysis of diagnostic codes associated with electronic health notes.[1] The highest hazard ratios (HRs) reported were for myoneural junction or muscle disease (ICD-10 codes G70–73), with HR 5·28 (95% CI 3·71–7·53) after COVID-19 versus after influenza and 4·52 (3·65–5·59) after COVID-19 versus after other respiratory tract infection. These striking findings received little discussion in the manuscript. The incidences of specific disorders within this category were not reported and presumably were not available.

Some of our author group are involved in a project providing weekly syntheses of the neurological and psychiatric sequelae of COVID-19.[2] We have not yet found any large-scale publication reporting empirical analysis of specific neuromuscular disease in COVID-19. Hence, we were interested to read these new findings. We speculate that critical illness neuromyopathy might partially account for the findings. Critical illness polyneuropathy and critical illness myopathy commonly co-occur, presenting with limb and respiratory muscle weakness, and delayed weaning from mechanical ventilation. Critical illness polyneuropathy and critical illness myopathy are complications of critical illnesses, particularly sepsis, and including severe COVID-19.[3,4] These complications are likely to adversely affect both short-term and long-term patient outcomes, and are of particular concern given the very high numbers of critically ill patients with COVID-19.[5]

Within the ICD-10 system, critical illness myopathy is likely to be coded as other specified myopathy (G72.8), although there could be other possibilities within the myoneural junction or muscle disease coding (G70–73). Other diagnoses, such as myasthenia gravis, muscular dystrophy, and congenital myopathy, are generally less common. The higher risk of myoneural junction or muscle disease for patients with COVID-19 who are hospitalised versus those who are not (HR 7·76 [95% CI 5·15–11·69]) and those who are admitted to the intensive therapy unit versus those who are not (11·53 [6·38–20·83]) are compatible with critical illness polyneuropathy and critical illness myopathy potentially being an important component of this ICD-10 category.

At face value, this specific finding reported by Taquet and colleagues could indicate an important neuromuscular complication in COVID-19. We recommend that COVID-19-related neuromuscular complications are investigated in more detail. Neuromuscular disorders after COVID-19 might have substantial implications for patient recovery and