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.

syndrome. For example, molecular mimicry with heat shock proteins (HSPs) was reported as a potential pathogenic mechanism of Guillain-Barré syndrome after SARS-CoV-2 infection.[5] HSPs might also promote a superantigen response, which contributes to the para-infectious response.

To establish association and potential causality, researchers should endeavor to collect data from as many cases with COVID-19 as possible from whom SARS-CoV-2 is presumed as the single or joint cause, on the basis of accurate and timely diagnosis of SARS-CoV-2 infection.

We declare no competing interests.

*Hai-Feng Li, Hong-Jun Hao, Xiang-Jun Chen
drlhf@163.com

Department of Neurology, Xuanwu Hospital, Capital Medical University, Beijing 100053, China (H-FL); Department of Neurology, Peking University, First Hospital, Beijing, China (H-JH); and Department of Neurology, Huashan Hospital, Fudan University, Shanghai, China (X-JC)

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Authors' reply

We read with interest the Correspondence by Hai-Feng Li and colleagues on our proposed definitions for COVID-19-associated neurological disease.[1] We thank the authors for recognising the importance of collecting cases together with accurate diagnostic evidence to elucidate disease mechanisms.

Any case criterion for a neurological syndrome associated with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection must incorporate a definition of acute SARS-CoV-2 infection, criteria for the diagnosis of the neurological syndrome itself, and an attempt to link the two in a temporal relationship, excluding other potential causes. The definition of acute SARS-CoV-2 infection must also reflect rapidly evolving diagnostic approaches.

In our proposed definition for SARS-CoV-2-associated Guillain-Barré syndrome, we selected a pragmatic definition of acute COVID-19 infection, reflecting the WHO definition of confirmed infection.[2] However, we accept that the timing of the infection onset is a challenge. A 6-week interval between viral symptoms onset and neurological disease is somewhat arbitrary, but from our knowledge of other infections triggering Guillain-Barré syndrome, a longer delay than this would cast the association into doubt. In patients without symptoms of SARS-CoV-2 infection but with positive RT-PCR or antibody testing, the true date of infection is even more difficult to elucidate.

We agree that it is important to exclude influenza as a potential trigger of Guillain-Barré syndrome, and viral symptoms might be difficult to distinguish. Epidemiological data can be informative, especially as the incidence of respiratory pathogens changes with the seasons around the world. RT-PCR testing for influenza and other respiratory viruses could be done alongside SARS-CoV-2 testing when possible. We advise caution in interpreting the results of studies using positive serum antibody testing for the diagnosis of influenza, which can be vulnerable to cross-reactivity and poor inherent test accuracy. Additionally, the study by Kong and colleagues[3] cited by Li and colleagues' Correspondence did not report co-infection, but rather early cases of COVID-19 in Wuhan, China, that were detected through the national influenza surveillance programme; existing influenza surveillance networks have been used for sentinel testing and to look for potential signs of community transmission worldwide, as supported by the WHO Global Influenza Surveillance and Response System. Furthermore, influenza-like illness is a syndromic definition and does not imply influenza to be the causative illness; its description aligns closely with the "acute respiratory infection" definition used to prompt testing for COVID-19 in earlier WHO and national guidelines.[4]

Our group has shown previously that, in patients with new neurological disease and evidence of more than one infection, there are additional challenges in thinking about causality, particularly when the results are from specimens collected outside the CNS.[5,6]

For the **WHO Global Influenza Surveillance and Response System** see https://www.who.int/influenza/gisrs_laboratory/en/

TS was an advisor for the GlaxoSmithKline ebola vaccine programme, chaired the Siemens diagnostics clinical advisory board and healthineers clinical advisory board, and also has a pending patent test for bacterial meningitis based on a blood test (GB 1606537.7; April 14, 2016). BS reports non-financial support from UK National Institute for Health Research through its Global Health Research Group on Brain Infections, outside the submitted work. All other authors declare no competing interests.

*Tom Solomon, Laura Benjamin, Bhagteshwar Singh, Suzannah Lant, Mark A Ellul
tsolomon@liverpool.ac.uk

National Institute for Health Research Health Protection Research Unit in Emerging and Zoonotic Infections, Institute of Infection, Veterinary and Ecological Sciences, University of Liverpool, Liverpool L69 7BE, UK (TS, BS, SL, MAE); The Walton Centre National Health Service Foundation Trust, Liverpool, UK (TS, MAE); Queen Square Institute of Neurology, University College London, London, UK (LB); and Christian Medical College, Vellore, India (BS)

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Antipsychotic prescribing to people with dementia during COVID-19

Antipsychotic drugs are used to treat agitation, aggression, and psychosis in dementia when alternative strategies have failed. Their use has been reduced because of concerns about safety and limited efficacy.[1] The English National Health Service publishes monthly data on patients registered with a dementia diagnosis in England, including those who have been prescribed an antipsychotic.[2] From March, 2020 (470 292), to April, 2020 (453 377), the number of registered people with dementia fell by 3·60%. There was a similar 3·34% reduction when comparing April, 2019 (469 025) with April, 2020 (453 377). According to the Office of National Statistics, 17 316 patients died in England in April, 2020, with "dementia and Alzheimer's disease" recorded on their death certificate.[3] This number of deaths was nearly three times more than expected, compared with the 5-year mean for April (appendix).

See **Online** for appendix

Although the absolute number of antipsychotic prescriptions for people with dementia decreased this year from March (45 554) to April (45 286), May (43 374), June (42 664), and July (42 964), reductions in the overall number of registered patients meant that the proportion of patients who have been prescribed antipsychotics substantially increased. Similar to the overall number of people with dementia, the proportion of patients who have been prescribed antipsychotics had tended to be constant, between 9·28% and 9·47%, throughout 2018 and 2019. In March, 2020, this percentage increased to 9·69% (95% CI 9·60–9·77) and in April to 9·99% (95% CI 9·90–10·08); it was 9·80% (95% CI 9·67–9·85) in May, 9·66% (95% CI 9·57–9·75) in June, and 9·74% (95% CI 9·65–9·83) in July. Rates in March, April, and May, 2020, were substantially higher than in the same months in 2018 (increased by 4·40%, 6·95%, and 5·22%, respectively) and 2019 (increased by 4·28%, 7·34%, and 4·87%, respectively).

These data support anecdotal reports of increased antipsychotic prescribing to people with dementia during the COVID-19 pandemic.[4] People with late-stage dementia and those within care facilities, who would be the group most likely to be prescribed antipsychotics, were over-represented among the additional deaths in April, 2020, and the effects of loss from the register by death of at least 20 000 such people would have been expected to reduce the proportion of patients receiving these drugs.[5] The register does not record specific indications for antipsychotic prescribing, and it is possible that some of the increase related to delirium management or palliative care, although most of the increase was probably in response to worsened agitation and psychosis secondary to COVID-19 restrictions (eg, care-home residents confined to their bedrooms, cessation of communal activities and family visits). Longer follow-up will show whether systems of caring for people with dementia can adapt to the continued threat of COVID-19 without increased use of antipsychotic drugs and whether we can continue to reduce the use of these drugs when the risks of infection have passed.

RH reports grants from the English National Institute of Health Research, outside the submitted work; and is Trustee of the charity Alzheimer's Research UK. AB reports being National Clinical Director for Dementia at the National Health Service England, and receiving personal fees from National Health Service England, personal fees from International Journal of Geriatric Psychiatry, personal fees from various lectures and talks, personal fees from occasional court reports, other from Driver and Vehicle Licensing Authority, outside the submitted work. LS reports grants and personal fees from Eli Lilly, personal fees from Avraham Pharmaceuticals, personal fees from Boehringer Ingelheim, grants and personal fees from Merck, personal fees from Neurim, personal fees from Neuronix, personal fees from Cognition, personal fees from Eisai, personal fees from Takeda, personal fees from vTv Therapeutics, grants and personal fees from Roche/Genentech, grants from Biogen, grants from Novartis, personal fees from Abbott, grants from Biohaven, grants from Washington University of St. Louis, grants from Dominantly Inherited Alzheimer Network Trial Unit of the National Institute on Aging, and personal fees from Samus, outside the submitted work.

**Robert Howard, Alistair Burns, Lon Schneider*
robert.howard@ucl.ac.uk

Division of Psychiatry, University College London, London W1T 7NF, UK (RH); School of Psychiatry and Behavioural Sciences, University of Manchester, Manchester, UK; and University of Southern California, Los Angeles, California, USA (LS)

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Introducing the Society of Black Neurologists

The Society of Black Neurologists (SBN) was founded on Dec 7, 2018, to foster discussion, mentorship, and camaraderie among Black neurologists. SBN founders Shaun Smart and Andrew Spector recognised the absence of any active groups dedicated to supporting Black neurologists. The project initially started as a small Facebook group, but the SBN has now more than 200 members worlwide and has organised in-person events and webinars.

For more on **The Society of Black Neurologists** see https://www.facebook.com/blackneurologists