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outcomes, decrease hospital stays, and increase quality and duration of life. However, cost savings can be offset by the necessary upfront investments in quality improvement of a structured network. Substantial investment is needed to build disease-specific country or regional networks in Europe. The Dutch and the Lombardy networks are natural candidates for further comparison and testing.

We declare no competing interests.

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Authors’ reply

We thank our colleagues for their comments on our novel network model for reshaping care for people with Parkinson’s disease or other chronic neurological conditions.1 David Grimes and colleagues discuss the feasibility of implementing Parkinson’s disease-specific training of community-based allied health professionals across different health-care settings. Their proposed approach is compatible with our model and is a feasible implementation route to ascertain adequate care delivery by allied health professionals. An important part of the integrated network model is to establish links between Parkinson’s disease specialists and generalists working in the community.1 We agree that using local resources and adapting the specific characteristics of the network, such as number of trained professionals, accordingly is essential. We were encouraged to note that specialised allied health-care programmes, which were originally developed in the Netherlands, could be transferred successfully to a different health-care setting in another country with modifications dictated by the regional availability of resources.2

Alberto Albanese and Daniela Calandrella draw attention to the Lombardy network, which is a promising illustration of several key components of our proposed model: to deliver integrated care, to ascertain continuity of care, and to enable access to appropriate care.3 However, they might have misinterpreted some other components of our model. The role of a personal care manager in the model is fulfilled by a medical professional (eg, a Parkinson’s disease nurse).3,4 The specific characteristics of the network, including the number of hubs, depend on the prevalence of specific diseases and the travel distances in a country. For example, the approximately 50 000 people with Parkinson’s disease in the Netherlands are served not by a single hub but by at least six of these hubs. These hubs are centres of expertise where knowledge should help to remotely support local health-care professionals, allowing them to deliver optimal care nearby the patient. Treatment should be administered as close to home as possible and visits to hubs should remain an exception—eg, for those with a difficult diagnostic trajectory or those in need of complex advanced treatments.

Our colleagues inadvertently create the impression that allied health training is the sole, or most important, part of the integrated care model, whereas it is only one component of a much broader and multifaceted approach that also includes self-management, a personal care manager adopting a proactive approach, and access to specialised services in a remotely situated hub when needed. In line with the recommendation of Grimes and colleagues, an extreme focus on patient participation is at the core of every element of this approach.1

We agree with Albanese and Calandrella that we should reach international agreement about relevant outcomes to evaluate the cost-effectiveness of different integrated care models, allowing for benchmarking and enabling a crucial process of learning from the differences. We also agree with Grimes and colleagues that the COVID-19 pandemic has created unique opportunities to rapidly implement key elements of the integrated care model. In many ways, the ongoing pandemic already acts as a catalyst to narrow existing gaps between community-based generalists and disease-specialists through virtual peer-to-peer consultations.5 The now widely deployed telemedicine approaches will improve the collaboration between tertiary expert centres and general neurologists working in community hospitals, enabling harmonisation of high-level care for people with Parkinson’s disease.

BRB currently serves as co-editor in chief for the Journal of Parkinson’s Disease, serves on the editorial board of Practical Neurology and Digital Biomarker. BRB has received honoraria from serving on the scientific advisory board for AbbVie, Biogen, and Union Chimique Belge. BRB has received speaker fees from AbbVie, Zambon, Roche, GE Healthcare, and Bial, and has received grants from the Netherlands Organisation for Scientific Research, Michael J Fox Foundation, Union Chimique Belge, AbbVie, Stechting Parkinson Fonds, Hersenstichting Nederland, Parkinson’s Foundation, Verly Life Sciences, Horizon 2020, and Parkinson Vereniging. EJH received funding from the UK National Institute of Health (NIH) Research, the Gatsby Foundation, British Geriatrics Society, and Parkinson’s UK. EJH has also received speaker and consultancy fees from Profile, Medics, and Luye and received travel support from Bial AbbVie and Ever pharma. MSO serves as a consultant for the Parkinson’s Foundation, and has received research grants from the US NIH, Parkinson’s Foundation, the Michael J Fox Foundation, Parkinson Alliance, Smallwood Foundation, Bachmann-Strauss Foundation, Tourette Syndrome Association, and University of Florida Foundation. MSO’s deep brain stimulation research is supported by NIH National Institute of Nursing Research (R01NR014852) and NIH National Institute of Neurological Disorders and Stroke (R01NS051608). MSO has received royalties for publications with Demos, Manson, Amazon, Smashwords, Books4Patients, Perseus, Robert Rose, Oxford, and Cambridge for movement disorder books. MSO is an Associate Editor for New England Journal of Medicine Journal Watch Neurology. MSO has participated in continuing medical education.
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E Ray Dorsey, Michael S Okun, Mark Ellul and colleagues propose case definitions for the association of COVID-19 with neurological diseases. We would like to discuss the practicality of their definitions and the potential causality behind the associations, through the example of Guillain-Barré syndrome. Guillain-Barré syndrome can be easily differentiated from neurovirulent neuropathies, such as West Nile virus-associated neuropathy, and there is surveillance on its incidence in several countries, which renders Guillain-Barré syndrome a candidate for assessing the association between infection and neurological disease.

Neurological disease occurring in the 6-week interval after acute infection is considered evidence for autoimmune association. However, typical acute respiratory symptoms are not always indicators of acute severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection; atypical presymptomatic or asymptomatic presentations of SARS-CoV-2 infection can occur before the onset of Guillain-Barré syndrome. Should we adopt the WHO-confirmed COVID-19 case definition as the definition of SARS-CoV-2 infection? Moreover, should we use the screening date of a positive SARS-CoV-2 result as the onset of acute SARS-CoV-2 infection in all patients with Guillain-Barré syndrome without typical COVID-19 symptoms?

A possible association differs from a probable association in the evidence of other commonly associated causes. Although Ellul and colleagues discuss evidence from other viruses as the cause of Guillain-Barré syndrome, influenza was not listed in their proposed case definitions for COVID-19-associated neurological disease. There is robust evidence on influenza-like illnesses as triggers for Guillain-Barré syndrome, and vaccination against influenza might reduce the risk of influenza-associated Guillain-Barré syndrome. Early data showed a co-infection with influenza virus in about 50% of hospitalised patients with COVID-19. Co-infection of SARS-CoV-2 also exists in influenza-like illness. Therefore, influenza should be included among the possible causes of Guillain-Barré syndrome. Moreover, evaluation of the safety of a SARS-CoV-2 vaccine will face the same questions on whether Guillain-Barré syndrome is related to the infection itself or to the vaccine. Thus, information on the exact infectious agent is crucial for defining the adverse events of SARS-CoV-2 vaccines.

A pathogenic mechanism to link COVID-19 and Guillain-Barré syndrome has not yet been described. However, a molecular mimicry mechanism, autoimmune response against aberrant modification of nervous tissue by infection, and para-infectious immune dysfunction are common explanations for their potential association. We suggest that all these mechanisms should be investigated in COVID-19-related Guillain-Barré syndrome.