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Depression and anxiety during COVID-19

The COVID-19 Mental Disorders Collaborators conclude that, throughout 2020, the pandemic led to a 27·6% increase in cases of major depressive disorders and 25·6% increase in cases of anxiety disorders globally.[1] However, we propose that these prevalence estimates are likely to be substantially inflated. Decades of trauma research has shown that, for most people, negative life events such as bereavement or disaster exposure are typically followed by resilience (minimal effect on symptoms of anxiety, or depression, or both) or recovery (initial short-term increase in symptoms of anxiety, or depression, or both, followed by recovery).[2] This pattern matches what large-scale studies and reviews[3-5] have found in the context of COVID-19. In a meta-analysis of longitudinal cohort studies,[3] there was an acute increase in mental health symptoms at the pandemic onset. Symptoms declined significantly over time and were indistinguishable from prepandemic symptom profiles within a few months of the outbreak.

See Online for appendix

Psychological adaptation matters in the context of the collaborators' study,[1] because the authors' estimates of the COVID-19 impact are based on studies done primarily during the very early phase of the pandemic (data collection for 39 of 48 studies occurred primarily between March and May, 2020; appendix). At that time, symptoms of anxiety or depression were at their most severe and probably represented an acute reaction to an unexpected and unknown emerging crisis. The authors then extrapolated from those immediate reactions to infer how SARS-CoV-2 infection rates and human mobility affected mental health throughout 2020. However, a failure to take into account the short-lived nature of changes in mental health symptoms during the pandemic and the potentially diminishing relationship between indicators of COVID-19 impact and anxiety or depression throughout 2020, means that prevalence estimates might be grossly overestimated in the collaborators' study.

See Online for appendix

We declare no competing interests.

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Authors' reply

We thank Michael Daly and Eric Robinson for their comments on our Article.[1] We share Daly and Robinson's caution against generalising mental disorder prevalence estimates solely from the very early phases of the COVID-19 pandemic. Our methods took this timing into account in several ways. Every estimate that informed our model had corresponding values of the impact of the pandemic (estimates of human mobility and SARS-CoV-2 infection rate) for the period in which the survey was done, whether it was early or later during the course of the pandemic. We extrapolated the change in prevalence (by age, sex, and location) for each day of the year 2020, on the basis of daily estimates of the COVID-19 impact indicators. We then calculated the average daily prevalence for the year 2020 to represent the annual point prevalence for that year. This approach meant that our extrapolated annual point prevalences incorporated the prevalences during the months leading up to the pandemic, the very early phases of the pandemic, the time between waves of infection, and subsequent waves and prevention measures.

Although Daly and Robinson are correct that the most common month of data collection was April, 2020, we also had studies providing data for every month between March, 2020 and January, 2021 (appendix). We had 12 studies reporting on prevalence from June, 2020 onwards. Many of these studies about the later months in 2020 still reported elevated COVID-19 prevalence. For example, data from Knudsen and colleagues[2] suggested a 70% increase in the prevalence of major depressive disorders (via diagnostic interview) in Trondheim, Norway, during August and September, 2020, compared with prepandemic estimates. Also, the Household Impacts of COVID-19 Survey[3] in Australia reported a 71% increase in the prevalence of psychological distress in November, 2020 compared with prepandemic estimates.

The broader literature on the effect of past population shocks also shows substantial increases in mental disorder prevalence. For example, prevalences of major depressive episodes doubled after the 2009 financial crisis in Greece,[4] and increased by more than 50% following the 2008 financial crisis in Hong Kong.[5] Elevated prevalence of depressive and anxiety disorders have also been observed in conflict-affected populations.[6]

However, our methods contain several important caveats, and we acknowledge the large bounds of uncertainty around the prevalence estimates produced. We need more high-quality mental health survey data across many parts of the world throughout 2020 and 2021 to better understand the effect of COVID-19 on the prevalence of mental disorders. Our method and results reflect the best approach and best estimates available, given the limitations and sparsity of available data. We appreciate the work by researchers like Daly and Robinson in doing these surveys during challenging circumstances brought about during the pandemic. We hope to see more work of this kind in the future.

We declare no competing interests.

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Transmission of SARS-CoV-2: still up in the air

Trisha Greenhalgh and colleagues claim that the dominant mode of SARS-CoV-2 transmission is likely to be airborne.[1] However, many of the reasons that are cited as support for airborne transmission do not hold up to scrutiny. We acknowledge that transmission occurs along a spectrum and airborne spread is possible, particularly in crowded and poorly ventilated settings, but the epidemiology and scientific literature do not support airborne spread as the predominant mode of transmission.

First, the notion that asymptomatic or presymptomatic transmission implies an airborne mode of transmission is inaccurate, as asymptomatic and presymptomatic shedding have been described with other respiratory viruses.[2] Similarly, decreased risk of transmission in an outdoor setting has been described with other viruses that are transmitted by the droplet and contact routes.[3]

Second, in many reports of nosocomial infections, health-care workers used incomplete or inappropriate personal protective equipment, such as absence of eye protection, and these reports cannot rule out other modes of transmission. Reports of transmission despite appropriate personal protective equipment also do not consider whether personal protective equipment was doffed appropriately. Most health-care workers make errors during doffing, which has been shown to be associated with self-contamination.[4]

Finally, Greenhalgh and colleagues do not account for the fact that containment measures focusing on prevention of droplet transmission have been effective at bringing the basic reproduction number below 1 in many jurisdictions.[5]

The science is far from settled, and we need studies of improved quality to further understand the role of short-range and long-range aerosol transmission of SARS-CoV-2.

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Authors' reply


Julien Mattia/Anadolu Agency/Getty Images

We welcome the opportunity to clarify the misconceptions that were raised by Alison Lopez and Jocelyn Srigley, which reflect a widely held but fundamentally flawed paradigmatic view among infection control clinicians.

In our Comment,[1] we list the streams of evidence that suggest that the most plausible explanation for mode of transmission is predominantly an airborne method.[2–4] A predominantly droplet mode (ie, spread mainly via coughing and sneezing) cannot explain the epidemiological pattern of this pandemic: transmission is far lower outdoors; asymptomatic or presymptomatic spread is common; superspreading is almost solely indoors; and when comprehensive studies are done, transmission beyond droplet distance of 1·8 m occurs commonly, sometimes with only fleeting exposure.[1–4]

That other respiratory diseases show asymptomatic shedding and are less transmissible outdoors suggests