

The impact of the COVID-19 pandemic on cognitive decline



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The effect of the COVID-19 pandemic on the general population has been catastrophic. Despite substantial progress in understanding the virology, transmission, and pathogenesis of SARS-CoV-2,¹ many of the long-term consequences of COVID-19 or the restriction measures implemented around the world remain unknown. Evidence for the associated adverse effects of isolation, loneliness,² post-traumatic stress, depression, fear, anger, and confusion is overwhelming.³⁻⁵ However, changes in cognitive and physical functioning due to the COVID-19 pandemic have been less well documented.

Anne Corbett and colleagues⁶ used longitudinal data from the PROTECT study to evaluate the effect of the pandemic on cognition in older adults in the UK, exploring various factors associated with changes in cognitive functioning. The use of computerised neuropsychological data collected before the pandemic and during its first and second years enabled repeated measures to be obtained for the same individuals across the pandemic. Declines in executive function and working memory were observed across the whole cohort in the first year of the pandemic, including in subgroups of individuals with mild cognitive impairment or a history of COVID-19, and the decline in working memory continued into the second year of the pandemic. Although initially thought to cause acute respiratory symptoms, the effects of SARS-CoV-2 on other systems—including the central and peripheral nervous system—is becoming increasingly clear. The new findings from the PROTECT study indicate domain-specific cognitive changes for individuals with a history of COVID-19 that mirrored similar trajectories for those with mild cognitive impairment but with a slightly lower rate of decline. This study also highlights reduced exercise, alcohol use, depression, and loneliness as key risk factors that affected the rates of cognitive decline in the older population during the COVID-19 pandemic. The study was observational in nature and therefore causality cannot be inferred, but it would be of interest to elucidate some of the potential biological mechanisms involved in these associations.

Alcohol intake and lack of exercise could have direct effects on psychological wellbeing and cognitive function; for example, alcohol can inhibit the communication between nerve cells and suppress

the activity of excitatory nerve pathways.⁷ Unhealthy lifestyles are linked to oxidative stress, which is recognised as a contributing factor in ageing and in the development of neurodegenerative diseases, including Alzheimer's disease.⁸ However, such lifestyle factors could also mediate direct associations between depression, loneliness, and cognitive decline—for example, depression and social isolation could lead to an increase in alcohol consumption and sedentary behaviour, consequently influencing the rate of cognitive decline through systemic inflammation⁹ or reduced mental and social engagement. Longer periods of follow-up—in addition to the availability of key data from before, during, and after the pandemic—are necessary. The wealth of longitudinal cohort studies of ageing in the UK could help to provide some of the necessary answers.

Looking back, COVID-19 has revealed the astonishing vulnerability of our societies, but also the lack of strategy and organisation from so many governments worldwide, and our shared fragility when confronted with infections. Older people (aged ≥ 50 years) with cardiovascular disease and complex comorbidities have almost double the risk of being infected with SARS-CoV-2 compared with those without chronic conditions.¹⁰ Let us remind ourselves that COVID-19 is not a thing of the past, but a reality for which we must continue to adapt and prepare.

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