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of an entrance door and a small window (appendix p 3). In the best ventilated room, after 30 s the number of droplets had halved, whereas with no ventilation this took about 5 min, in agreement with the air drag calculation that shows that 5 μ m drops from the average cough or speech height take 9 min to reach the ground. In a poorly ventilated room, the number of droplets was halved in 1–4 min.

Although we only studied healthy volunteers and did not study patients with COVID-19 or virus-laden aerosol droplets directly, our data on droplet size distribution and persistence does have implications on requirements to use face masks to prevent virus transmission. Transmission by aerosols of the small droplets studied here can only be prevented by use of high-performance face masks; a conventional surgical mask only stops 30% of the small aerosol droplets studied here for inhaled breath;⁹ for exhaled breath the efficacy is much better.¹⁰

Additionally, the long airborne time of aerosols we found here affects the reliability of temporal and spatial contact data between individuals as monitored by proximity tracing via smartphone apps. These findings need to be considered in the development and implementation of these apps.

This study shows that better ventilation of spaces substantially reduces the airborne time of respiratory droplets. This finding is relevant because typically poorly ventilated and populated spaces, like public transport and nursing homes, have been reported as sites of viral transmission despite preventive physical distancing. The persistence of small respiratory droplets in such poorly ventilated spaces could contribute to the spread of SARS-CoV-2. Our findings confirm that improving ventilation of public spaces will dilute and clear out

potentially infectious aerosols. To suppress the spread of SARS-CoV-2 we believe health-care authorities should consider the recommendation to avoid poorly ventilated public spaces as much as possible. The implications are also important for hospital settings where aerosolisation by coughing and medical treatments and close contact with COVID-19 patients is very common.

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COVID-19 and the impact of social determinants of health

The novel coronavirus disease 2019 (COVID-19), caused by the pathogen severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), originated in Wuhan, China, and has now spread internationally with over 4·3 million individuals infected and over 297 000 deaths as of May 14, 2020, according to the Johns Hopkins Coronavirus Resource Center. While COVID-19 has been termed a great equaliser, necessitating physical

distancing measures across the globe, it is increasingly demonstrable that social inequalities in health are profoundly, and unevenly, impacting COVID-19 morbidity and mortality.

Many social determinants of health—including poverty, physical environment (eg, smoke exposure, homelessness), and race or ethnicity—can have a considerable effect on COVID-19 outcomes. Homeless



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For more on how ethnic minorities are affected by COVID-19 see [News](#)
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families are at higher risk of viral transmission because of crowded living spaces and scarce access to COVID-19 screening and testing facilities.¹ In a Boston study of 408 individuals residing in a shelter, 147 (36%) had a positive SARS-CoV-2 PCR test.² Smoke exposure and smoking has been linked to adverse outcomes in COVID-19.³ A systematic review found that current or former smokers were more likely to have severe COVID-19 symptoms than non-smokers (relative risk [RR] 1.4 [95% CI 0.98–2.00]) as well as an increased risk of intensive care unit (ICU) admission, mechanical ventilation, or COVID-19-related mortality (RR 2.4, 1.43–4.04).³ In the USA, the COVID-19 infection rate is three times higher in predominantly black counties than in predominantly white counties, and the mortality rate is six times higher.⁴ In Chicago alone, over 50% of COVID-19 cases and almost 70% of COVID-19 fatalities are disproportionately within the black population, who make up only 30% of the overall Chicago population.⁴

It is also poignant that physical distancing measures, which are necessary to prevent the spread of COVID-19, are substantially more difficult for those with adverse social determinants and might contribute to both short-term and long-term morbidity. School closures increase food insecurity for children living in poverty who participate in school lunch programmes. Malnutrition causes substantial risk to both the physical and mental health of these children, including lowering immune response, which has the potential to increase the risk of infectious disease transmission.⁵ People or families who are homeless are at higher risk of infection during physical lockdowns especially if public spaces are closed, resulting in physical crowding that is thought to increase viral transmission and reduce access to care.¹ Being able to physically distance has been dubbed an issue of privilege that is simply not accessible in some communities.⁴

The association of social inequalities and COVID-19 morbidity is further compounded in the context of underlying chronic respiratory conditions, such as asthma, where there is a possible additive, or even multiplicative, effect on COVID-19 morbidity. Several adverse social determinants that impact the risk of COVID-19 morbidity also increase asthma morbidity, including poverty, smoke exposure, and race or ethnicity.⁶ Consistent associations have been noted between poverty, smoke exposure, and non-Hispanic

black race and measures of asthma morbidity, including poorer asthma control and increased emergency department visits for asthma.⁶ The interplay of social determinants, asthma, and COVID-19 might help explain the risk of COVID-19 morbidity imposed by asthma, such as the disproportionate hospitalisations for COVID-19 among adults with asthma living in the USA.⁷ The CDC note asthma to be a risk factor for COVID-19 morbidity.⁸ Data released from the CDC on hospitalisations in the USA in the month of March, 2020, notes that 12 (27%) of 44 patients aged 18–49 years who were hospitalised with COVID-19 had a history of asthma,⁸ in those aged 50–64 years, asthma was present in 7 (13%) of 53 cases, and in those 65 years or older asthma was present in 8 (13%) of 62 cases.⁸

The effect of social determinants of health and COVID-19 morbidity is perhaps underappreciated.⁶ Yet, the great public health lesson is that for centuries pandemics disproportionately affect the poor and disadvantaged.⁹ Additionally, mitigating social determinants—such as improved housing, reduced overcrowding, and improved nutrition—reduces the effect of infectious diseases, such as tuberculosis, even before the advent of effective medications.¹⁰ It is projected that recurrent wintertime outbreaks of SARS-CoV-2 will likely occur after this initial wave, necessitating ongoing planning over the next few years. Studies are required to measure the effect of COVID-19 on individuals with adverse social determinants and innovative approaches to management are required, and might be different from those of the broader population. The effect of physical distancing measures, particularly among individuals with chronic conditions facing adverse social circumstances, needs to be studied because adverse determinants and physical distancing measures could compound issues, such as asthma medication access and broader access to care. The long-term effect of school closures, among those facing adverse social circumstances, is also in need of study.

Moving forward, as the lessons of COVID-19 are considered, social determinants of health must be included as part of pandemic research priorities, public health goals, and policy implementation. While the relationships between these variables needs elucidating, measures that affect adverse determinants, such as reducing smoke exposure,

regular income support to low-income households, access to testing and shelter among the homeless, and improving health-care access in low-income neighbourhoods have the potential to dramatically reduce future pandemic morbidity and mortality, perhaps even more so among individuals with respiratory conditions such as asthma.⁷ More broadly, the effects of COVID-19 have shed light on the broad disparities within our society and provides an opportunity to address those disparities moving forward.⁶

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Novel viruses, old data, and basic principles: how to save lives and avoid harm amid the unknown



The 2013–16 west African Ebola epidemic had a staggering case fatality rate of 30–70%, yet surprisingly few of the dozens of Americans and Europeans medically evacuated from the region died, with the case fatality rate in Europe and the USA estimated at a mere 10%.¹ Every American received experimental antiviral medications or convalescent plasma, and the efficacy and ethics of these therapies occupied both our national headlines and headspace. However, randomised clinical trials for both therapies have since failed to show benefit.^{2,3} Why, then, did so many more Americans survive if not due to preferential access to experimental therapies? The likely answer is the Americans survived not due to preferential access to unproven therapies but to proven ones.

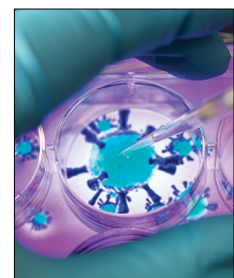
5 years later, the world is facing another, much larger, pandemic, and we worry the medical community has not learned from this recent experience.

To be clear, searching for effective new therapies against COVID-19 is highly important. At the same

time, we must remain cognisant that the odds are stacked against the candidates. Medications that decrease mortality are difficult to come by, leaving numerous diseases without direct remedies. Influenza provides an important perspective. Scientists have been searching for a cure since before the 1918 influenza pandemic, and more than 100 years later our best medicines for influenza merely shorten the duration of symptoms by a day at best.⁴ None has been shown to reduce mortality.

Influenza is not unique; sepsis has been subjected to decades of research resulting in a much advanced understanding of the syndrome's pathobiology. However, hundreds of therapeutic candidates with biological plausibility, from stem cells to vitamin C, have not consistently improved patient outcomes.

Of course, a lack of targeted therapies does not mean a patient with the 1918 influenza would not fare better today, or that someone with sepsis is not better off in 2020 than before the Surviving Sepsis



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