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LETTER TO THE EDITOR

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Where have all the viruses gone? Disappearance of seasonal respiratory viruses during the COVID-19 pandemic

Many viruses cause seasonal respiratory infections in temperate climates that peak in the winter months. They cause varying degrees of respiratory illness across all age groups and include respiratory syncytial virus (RSV), parainfluenza viruses (types 1–4), human metapneumovirus, rhinoviruses, seasonal coronaviruses (OC43, 229E, NL63, HKU1), enteroviruses, adenoviruses, and influenza viruses. This results in a significant healthcare burden across all populations with now well-established seasonality patterns.[1]

We now have good evidence that the spread of most seasonal respiratory viruses has been severely curtailed in Australia,[2] New Zealand,[3] and other Southern Hemisphere populations including Chile and South Africa,[4] believed to be attributable to the COVID-19 restrictions.

A similar pattern is now being observed in the United Kingdom[5] and Europe[6] with minimal influenza and RSV activity during the 2020–2021 Northern Hemisphere winter season, and most influenza-like illness consultations being COVID-19 related. During this period, rhinoviruses have become predominant and appeared to be relatively unaffected by the COVID-19 restrictions. Across the three regions NSW (New South Wales, Australia), WA (Western Australia), CB (Canterbury, New Zealand), regardless of differing patterns of COVID-19 restrictions, the rhinovirus positive cases showed large biphasic peaks on the background of little or no detectable activity of the other seasonal respiratory viruses (adenovirus, human metapneumovirus, parainfluenza viruses, RSV, Influenza)—until a late resurgence of RSV in NSW and WA (Figure 1).

A recent study comparing the relative effectiveness of various nonpharmaceutical government-mandated COVID-19 interventions across multiple countries during the first wave indicated that the most effective measures included the cancellation of social gatherings (i.e., bars, restaurants, spectator events) to reduce contact rates, border closures, and movement restrictions, and the increased provision of personal protective equipment.[7] After the relaxation of measures following the first wave, the virus began to spread throughout Europe again, exceeding the size of the first wave.[6] So why are these control measures not working as well for SARS-CoV-2 in some countries as well as they have for other seasonal respiratory viruses?

One possible answer is that we have achieved some degree of herd immunity to these pre-existing seasonal respiratory viruses, to which we now require a higher exposure dose before developing successful infection and disease. Such pre-existing immunity may increase the exposure dose required for successful reinfection and/or disease.[8]

Although we do not yet know the infectious dose for SARS-CoV-2, one estimate, based on older animal studies with SARS-CoV-1 suggests that just 200–300 viruses are needed to cause successful infection in 50% of an exposed population.[9] In human patients, one study demonstrated that thousands of SARS-CoV-2 RNA copies may be exhaled each minute by a patient in the acute phase of COVID-19,[10] potentially expelling tens of infectious doses into the surrounding air that could infect susceptible people nearby. In the absence of specific, pre-existing immunity, the infectious dose for SARS-CoV-2 is likely to be lower than for the seasonal respiratory viruses, against which most of us will have some degree of immunity.

More recently, with the easing of COVID-19 restrictions, some parts of Australia have been experiencing high levels of RSV activity (Figure 1). These RSV peaks are occurring later than any previous seasonal RSV activity, which suggests that the COVID-19 prevention measures may have simply delayed the epidemics of some of these viruses, with subsequent reappearance when measures are relaxed beyond a certain level.

Clearly, we do not yet fully understand the drivers for the very unusual patterns of activity of our normally relatively predictable winter respiratory viruses. Nevertheless, the massive reduction in the incidence of influenza and other seasonal respiratory viruses during this COVID-19 pandemic has been a welcome respite. These viruses usually cause considerable morbidity and mortality,[11,12] which could be exacerbated if cocirculating with COVID-19. For example, data from the 2019–2020 winter in the United Kingdom suggested that SARS-CoV-2/influenza coinfections resulted in more severe disease outcomes (https://www.medrxiv.org/content/10.1101/2020.09.18.20189647v2#disqus_thread).

This will allow resources to be reallocated to coping with the COVID-19 cases being admitted to the hospital throughout the remainder of this winter season, and demonstrates an incidental benefit (a "silver lining") of such restrictions, despite their general unpopularity.

Yet, we do need to be prepared for a possible resurgence of the other seasonal respiratory viruses such as RSV (and potentially influenza), as COVID-19 restrictions are eased going into the UK Spring/Summer seasons, particularly as the new COVID-19 vaccines are rolled-out more widely, and that this resurgence may not follow the usual seasonal patterns.

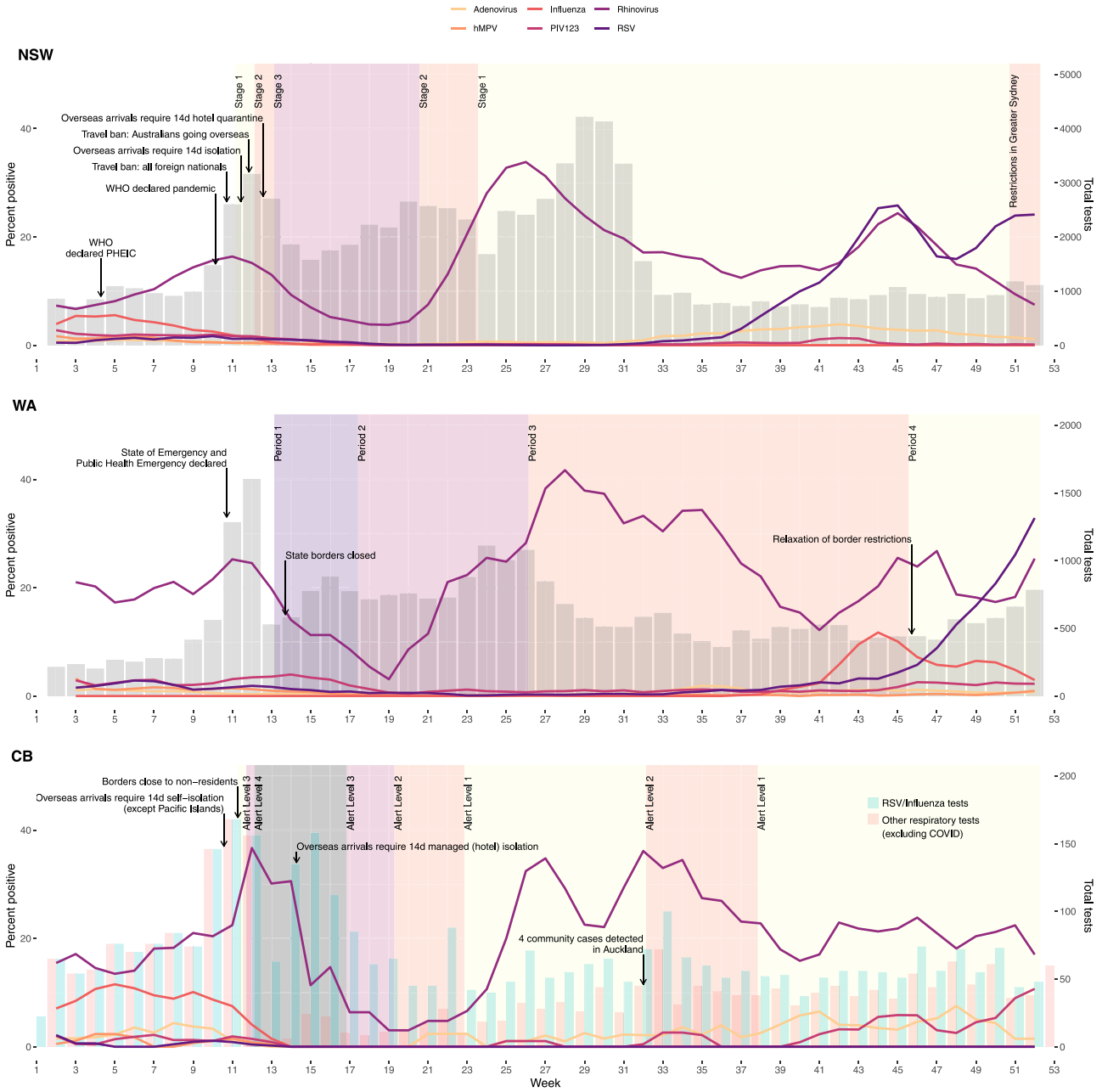

FIGURE 1 Weekly seasonal respiratory virus cases in Southern Hemisphere locations: Perth, Western Australia (Source: Co-authors Avram Levy, David Smith) (A), Sydney, New South Wales, Australia (Source: Co-authors Jen Kok, Janette Taylor) (B), Canterbury, New Zealand (Source: Co-authors Meik Dilcher, Harry Hua, Lance Jennings) (C). Note the predominance of rhinovirus and the reduced incidence of the other noninfluenza seasonal respiratory viruses.

DATA AVAILABILITY STATEMENT

Raw data are available upon request.

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