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Understanding why superspreading drives the COVID-19 pandemic but not the H1N1 pandemic

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Two epidemiological parameters often characterise the transmissibility of infectious diseases: the basic reproductive number (R_0) and the dispersion parameter (k). R_0 describes, on average, how many individuals in a susceptible population will be infected by someone with that disease, and k details the variation in individual infectiousness. The smaller the k value, the greater the variation. That is, fewer cases cause the majority of infections, and a greater proportion of infections tend to be linked to large clusters via superspreading events. This phenomenon, called overdispersion in transmissibility, has been found in many infectious diseases, yet the factors that mediate it remain poorly understood.[1]

During the COVID-19 pandemic, transmission of SARS-CoV-2 has been highly overdispersed, as 60–75% of cases infect no one and, propelled by superspreading events, 10–20% of cases cause 80% of secondary infections.[2–4] By contrast, the most recent pandemic, H1N1 in 2009, had more uniform transmission of influenza A/H1N1pdm09 and few instances of superspreading.[5] Both SARS-CoV-2 and A/H1N1pdm09 distributed globally over long periods of time, and they have similar modes of transmission (aerosols, contact, and droplets) and asymptomatic spread,[6–8] which prompts the question: why is there broad overdispersion in SARS-CoV-2 transmissibility during the COVID-19 pandemic but not in A/H1N1pdm09 transmissibility during the 2009 H1N1 pandemic?

Generally, k remains similar across distinct outbreaks of a virus.[1] Studies have documented this for SARS-CoV-2, including across disparate geographical locations with varying population demographics and behavioural norms, over different seasons, and under various public health interventions, such as during and outside lockdown.[2–4] The emerging betacoronaviruses, SARS-CoV-2, SARS-CoV (the aetiological agent of SARS), and Middle East respiratory syndrome coronavirus (MERS-CoV), show overdispersion, whereas common cold betacoronaviruses appear to have more homogeneous profiles.[1,9] Conversely, both seasonal and pandemic strains of influenza have transmitted with relatively uniform patterns over the years.[5] Children and adolescents contribute more to the transmission of common cold betacoronaviruses and influenza than they do to the emerging betacoronaviruses. However, despite the association of young age with high contact patterns and susceptibility to influenza and common cold betacoronaviruses,[9,10] these viruses are infrequently involved in superspreading events.

Cumulatively, these observations suggest that behavioural, interventional, general demographic, seasonal, and other environmental factors might affect transmissibility but are not the key determinants of overdispersion for directly transmitted viruses. They suggest instead that k, at least in part, is an intrinsic characteristic of these viruses and, as obligate intracellular parasites, their host interactions. That is, poorly ventilated, crowded spaces with susceptible individuals facilitate superspreading, but whether a virus tends to transmit via large clusters in the first place seems to be intrinsic to that viral infection. Thus, the previous question can be reframed: which virological factors mediate k?

This is a long-standing question in infectious disease epidemiology, and recent work has begun to delve into the topic. For SARS-CoV-2, SARS-CoV, and A/H1N1pdm09, greater case variability in respiratory viral load during their infectious periods is associated with overdispersion.[11] Moreover, models of expelling viable virus via respiratory droplets and aerosols concur with the observed transmission patterns: inherently, most COVID-19 cases are minimally infectious, but highly infectious individuals are estimated to expel hundreds to thousands of virions per minute while talking, singing, or coughing. Meanwhile, a greater proportion of people infected with A/H1N1pdm09 are inherently infectious but expel virions at low rates.[11]

The mechanistic basis of these determinants of overdispersion is a promising area of study. For example, intrinsic case heterogeneity in shedding could arise from individual differences in susceptibility, previous exposures, immunity, or viral host factors,[10,12–14] or could be related to phenomena associated with recent zoonotic spillover. In the event that common molecular mechanisms are indicated with k,[14] they could

guide the rational design of host-directed therapies that reduce the incidence of superspreading for those virus classes. As these drugs act on host pathways, they can be evaluated beforehand for safety and then tested for efficacy in the early stages of a novel outbreak. Furthermore, many factors relevant to individual infectiousness, including how case characteristics affect the viability of the shed virus and the distributions of expelled respiratory particles, remain unclear. How behaviour, environment, host, and virus interplay to affect transmission modes or infection risk is also unclear. As seen over the past year,[6,11] cross-disciplinary interaction among researchers is needed to best understand these myriad matters.

Research into this nascent topic uncovers epidemiologically relevant biological insight and might provide key considerations for public health. When *k* is small, few cases transmit but are more likely to be superspreaders, meaning epidemics are infrequent but explosive. Overdispersion increases the likelihood of disease extinction when case numbers are low, and control measures targeting high-risk settings or individuals disproportionally curb transmission.[1,15] These measures can be particularly effective when implemented early in an overdispersed outbreak, as reflected in areas that have eliminated COVID-19, but have diminished effects on outbreaks with more uniform transmission. Currently, however, there is no way to predict the transmission patterns of novel viruses. Contact-tracing studies empirically characterise *k*,[2–4] meaning considerable spread must have already occurred before its estimation. Broadly understanding the factors that mediate overdispersion, from virological to clinical and environmental, might provide early, predictive correlates for transmission patterns—including superspreading—before widespread infection by novel viruses. In this case, a playbook of control strategies, each specified by transmission patterns, can be developed to then specifically address outbreaks.

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Lessons about COVID-19 vaccine hesitancy among minority ethnic people in the UK

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According to data collected by Public Health England, in the UK, minority ethnic groups were between two and four times more likely to die due to COVID-19 compared with those from a White ethnic background.[1] These outcomes are independent of age, sex, or socioeconomic factors. Moreover, at the start of the