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Human behavior and disease dynamics

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We tend to think of epidemics as beginning slowly, accelerating to a peak, and then falling off in intensity. This pattern is commonly observed for a wide range of diseases both human and animal and has even motivated an empirical formulation, known as Farr's law, for the shape of epidemic trajectories (1, 2). Loosely speaking, the law states that what goes up must come down—and will do so somewhat symmetrically. Such a pattern emerges directly from the susceptible–infected–removed (SIR) models often used to describe disease dynamics. While extremely simple, the SIR model does a good job capturing the shapes of epidemic curves for a range of different infectious agents (Fig. 1 *A*–*E*). But the COVID-19 pandemic followed a very different trajectory (Fig. 1*F*). In between multiple waves of infection, we saw lengthy plateaus. This cannot happen in conventional SIR models, in which cases are either increasing because enough people are susceptible, or decreasing because enough people are immune. Why was the COVID-19 pandemic different? In their paper, "Dynamics in a behavioral–epidemiological model for individual adherence to a nonpharmaceutical intervention," Saad-Roy and Traulsen explore one reason, which the classical SIR framework ignores: Humans change their behavior during epidemics to avoid infection (3).

In the SIR model, the only reason that case counts peak and decline is that the pathogen runs out of people to infect. In reality, there is almost always something else going on alongside immunity. Fig. 1*A* shows plague death data from Bombay that were used to illustrate the original development of the SIR model (4); in reality, seasonality likely played an important role (5). The H1N1 "swine flu" pandemic of 2009 was similar. As the days lengthened the known seasonal factors that impede influenza transmission, heat and humidity among them worked together with the accumulated immunity in the population (Fig. 1*B*). We see this clearly when comparing epidemic curves in the southern hemisphere, where most countries experienced a single winter peak around July 2009, with the northern hemisphere, where two separate peaks were observed in spring and fall (6). For West Nile virus, seasonality of the mosquito vector is critical in shaping the epidemic trajectory (Fig. 1*C*).

Interventions can also play an important role, and they need not be based on vaccination or other pharmaceuticals. During the 2001 foot-and-mouth disease outbreak among livestock in the United Kingdom (Fig. 1*D*), a controversial contiguous cull strategy was employed to reduce the number of onward transmission events to new farms before they could be infected. As another example, behavioral changes among affected populations, in concert with testing and alongside vaccination, brought the 2022 Mpox outbreak under control (Fig. 1*E*).

The math of the simple SIR model combines the rate of encounters among infected and infectable people and the probability of transmission between them into a single parameter. From this, we can see that there is more than one way to slow infections. Either reducing the numbers of contacts or probability of transmission will reduce the rate at which the outbreak spreads. We do this with "non-pharmaceutical interventions" or NPIs. For example, NPIs like school closures and event cancellations reduce contact rates, whereas masks and air filtration reduce transmission when contact does occur.

However, NPIs will only work so long as they are used and nothing else changes. Human behavior can vary depending on the prevalence of disease and the perception of risk. A frightened public might take extraordinary precautions, but over time, as fatigue sets in, people may lose enthusiasm for voluntary actions to limit disease exposure and become reluctant to comply with policies such as stay-at-home orders, mask mandates, or school closures.

Saad-Roy and Traulsen capture this process mathematically with what they describe as a socio-epidemiological model. They couple a simple model of human behavior—the replicator equation as a model of social learning—to the usual SIR-type differential equations. This explicitly captures an important feedback process: the prevalence of disease influences human behavior, which in turn feeds back to shape the prevalence of disease. This framework readily captures those long flat periods in between COVID surges.

Predicting epidemic trajectories is difficult (10), especially because people are harder to predict than viruses. We lack adequate models to describe how people behave during pandemics. We do not understand how behavior would change over time even in an idealized situation where people have accurate information about the real risk of disease, let alone what happens when people form their beliefs and opinions through interactions with the complex and politicized information environments that characterize contemporary society (11). To be clear, Saad-Roy and Traulsen are not aiming to provide such a model here. Instead they draw attention to the interactions between behavior and disease spread, using for illustration a simple

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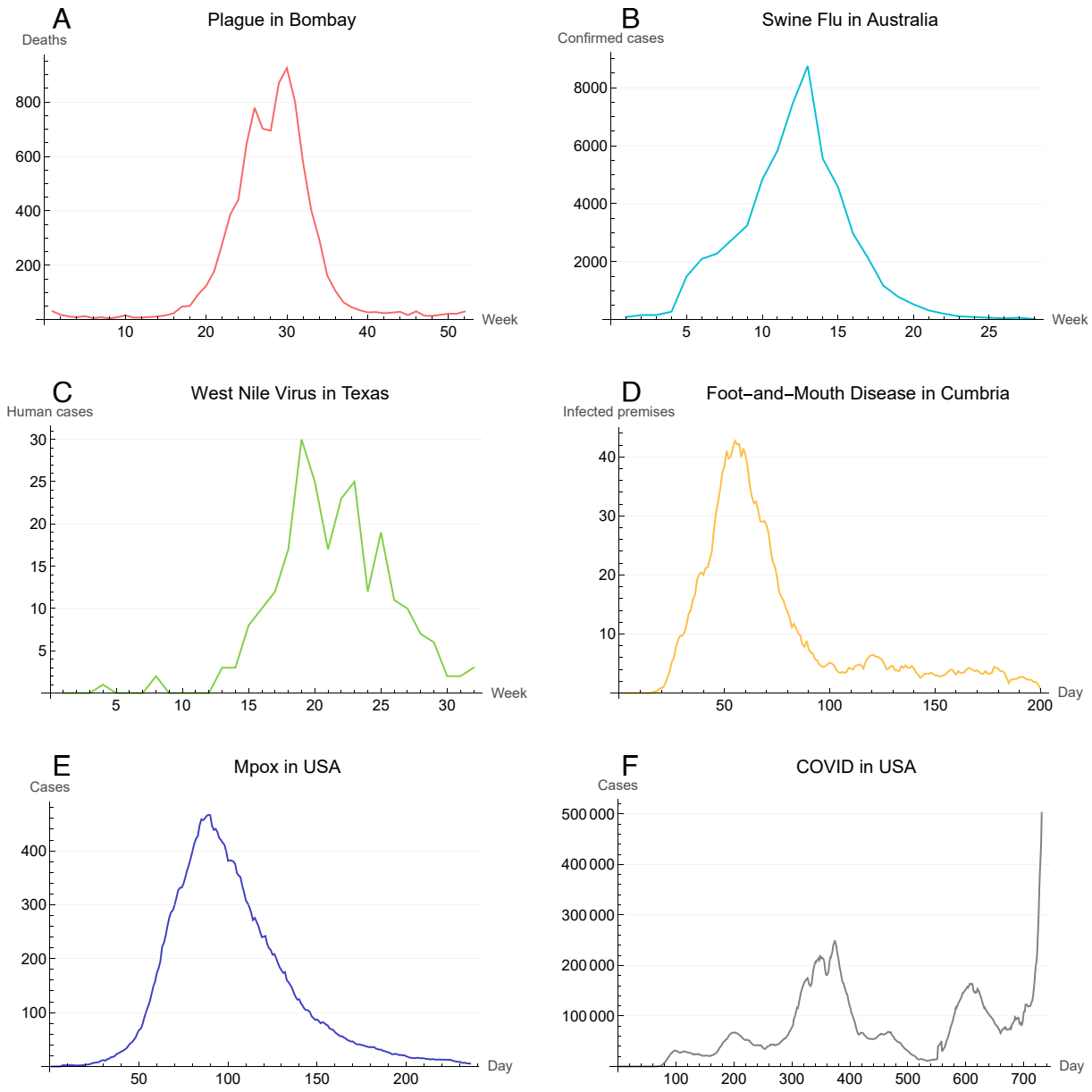

Fig. 1. Epidemic curves for six disease outbreaks. Even when assessed at different levels of granularity, most (*A*–*E*) share a common form. The COVID-19 pandemic (*F*) had a very different trajectory. (*A*) Plague deaths in Bombay 1905 to 1906, from ref. 7, as used in the 1927 paper that introduced SIR modeling (4). (*B*) Swine Flu cases in Australia, 2009. Data extracted from figure 4 in ref. 8. (*C*) Premises with livestock infected with foot and mouth disease in Cumbria, 2001, rolling 7-d average from ref. 9. (*D*) Human cases of West Nile Virus in Texas May to December 2015, data from Texas Department of State Health Services. (*E*) Mpox cases in the USA, May 10 to December 31, 2022, data from CDC. (*F*) COVID-19 in the USA 2020 to 2021, rolling 7-d average with data from the World Health Organization.

model of reasonable ways in which people might adhere to NPIs under various circumstances. Their sometimes counterintuitive results illustrate the urgent need for better models of behavioral responses during pandemics.

One surprising outcome of the Saad-Roy and Traulsen model is that under some circumstances, mandating a new NPI has no effect on disease prevalence. The basic logic is that when a mandate is instituted, disease prevalence drops, and people compensate by decreasing their use of other non-mandated NPIs. This result provides an important cautionary lesson: public health interventions, even effective ones, can influence human behavior in ways that modulate and reduce their overall impact. As a familiar example, consider how public awareness of polio, measles, and other childhood diseases has fallen as vaccination has successfully mitigated their threat.

The value of this model derives from how it quantitatively captures that intuition, not in any capacity for predicting the consequences of real-world interventions. Saad-Roy and Traulsen find that in their model this buffering effect exactly cancels out the effect of an NPI mandate, but that result should not be seen as a general principle of infectious disease epidemiology, but a result of the model structure. While the model assumes that individual behavior converges to an optimal response, the dramatic regional, racial, and socioeconomic variation in individual attitudes and responses to the recent COVID-19 pandemic (12) remind us that the situation is far more complicated.

While the model assumes simple learning dynamics for behavior, in practice there will be a gulf between what people might wish they could do and what they will have to do to get by. Exhortations and mandates are of little use if people lack the means to comply. Access to resources such as masks and rapid tests, and paid sick leave to prevent infections in the work place for those occupations deemed essential—such are means to improve the uptake and effectiveness of NPIs.

Moreover, this model assumes a homogeneous population with respect to risks from disease and the cost of interventions. In the case of COVID-19, older people are far more likely to be seriously ill (13) and so may behave differently. Imagine an intervention that reduces, but does not eliminate, the chance that a person will transmit infection or acquire it if they are exposed. The impact on infection rates will accumulate in a non-linear fashion as more people use the NPI (see e.g. ref. 14). Yet if those at lower risk of severe illness drop the intervention, the force of infection in the community will increase, and vulnerable individuals will be more likely to be exposed.

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The impacts of NPIs do not evaporate with vaccination. And Saad-Roy and Traulsen show NPIs remain particularly valuable in concert with vaccination when transmission rates are high. As usual, these results depend on our assumptions about how effective or otherwise the vaccines will be, and how enthusiastically people adhere to NPIs. Moreover, vaccination is added to the model at birth and at random—neither of which are how vaccines are likely to be used in any actual pandemic.

Returning the shapes of epidemic curves with which we began this perspective, there is a reason epidemiologists have been taught about Farr's law for more than a century. Yet we need more that empirical generalizations about the shape of epidemic trajectories, especially during a pandemic in which vast numbers of people are at risk. Most of us recognize that attempts to predict the course of the recent pandemic by fitting early trajectories in one location to empirically observed disease trajectories elsewhere had to be updated, revised, and retrofit time and again to account for the changing epidemiological reality—or simply met with dismal failure, according to taste.

An SIR model is only one way of producing a bell-shaped curve, and as shown in Fig. 1 the processes driving SIR dynamics rarely operate in isolation. Saad-Roy and Traulsen's framework moves us toward a reckoning with the crooked timber of humanity. They persuasively demonstrate that human behavior will influence the equilibrium numbers of people infected in a pandemic. While few epidemiologists would disagree, it is imperative that we develop quantitative methods to account for such effects and how humans actually do behave, which is rarely optimal. At the same time, we need to think beyond equilibrium dynamics. Pandemics are by definition out-of-equilibrium phenomena, as are shifting human beliefs. A key challenge for predictive modeling in infectious disease epidemiology is to help us navigate outbreaks better. Even if an endemic equilibrium is where we are destined to arrive, it very much matters how we get there because some trajectories take a far greater toll along the way (15). Saad-Roy and Traulsen provide an important step in the right direction.

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