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The influence of social behaviour on competition between virulent pathogen strains

Joe Pharaon, Chris T. Bauch*

Dept. of Applied Mathematics, University of Waterloo, 200 University Ave West, Waterloo, ON N2L 3G1, Canada

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ABSTRACT

Infectious disease interventions like contact precautions and vaccination have proven effective in disease control and elimination. The priority given to interventions can depend strongly on how virulent the pathogen is, and interventions may also depend partly for their success on social processes that respond adaptively to disease dynamics. However, mathematical models of competition between pathogen strains with differing natural history profiles typically assume that human behaviour is fixed. Here, our objective is to model the influence of social behaviour on the competition between pathogen strains with differing virulence. We couple a compartmental Susceptible-Infectious-Recovered model for a resident pathogen strain and a mutant strain with higher virulence, with a differential equation of a population where individuals learn to adopt protective behaviour from others according to the prevalence of infection of the two strains and the perceived severity of the respective strains in the population. We perform invasion analysis, time series analysis and phase plane analysis to show that perceived severities of pathogen strains and the efficacy of infection control against them can greatly impact the invasion of more virulent strain. We demonstrate that adaptive social behaviour enables invasion of the mutant strain under plausible epidemiological scenarios, even when the mutant strain has a lower basic reproductive number than the resident strain. Surprisingly, in some situations, increasing the perceived severity of the resident strain can facilitate invasion of the more virulent mutant strain. Our results demonstrate that for certain applications, it may be necessary to include adaptive social behaviour in models of the emergence of virulent pathogens, so that the models can better assist public health efforts to control infectious diseases.

1. Introduction

Modern approaches to developing a theory of the spread of infectious diseases can be traced to 1927 when Kermack and McKendrick developed an integro-differential equation model now widely described as the SIR (Susceptible-Infected-Recovered) model (Kermack and McKendrick, 1932). The model tracks changes in the number of individuals susceptible to an infection $\boldsymbol{S(t)}$, the number of infected individuals $\boldsymbol{I(t)}$, and (implicitly) the number of recovered individuals $\boldsymbol{R(t)}$. Compartmental models such as the SIR model are useful for mechanistic modelling of infection transmission in populations. They have since been further developed to study the evolution and epidemiology of multiple species of pathogens in a population or different strains of the same species (Frank, 1996). Some models focus on between-host competition while some others on within-host competition (Mideo et al., 2008). Bull suggested in the 1990s that coupling inter-host and intra-host dynamics in models may be desirable (Bull, 1994). Models linking between-host transmission dynamics to within-host pathogen growth and immune response are now becoming commonplace (Alizon and Fraser, 2013; Feng et al., 2013; 2012; Handel and Rohani, 2015; Mideo et al., 2008). One such approach is to link host viral load (which is a necessary condition of virulence) to the between-host transmission rate.

Compartmental models have also been used to study the phenomenon of pathogen virulence–the rate at which a pathogen induces host mortality and/or reduces host fecundity (Anderson and May, 1982; Cressler et al., 2016; Murall et al., 2015; Thomas and Elkinton, 2004). It was initially believed that hosts and parasites co-evolved to a state of commensalism (whereby parasites benefit from their host without harming them) (Simon et al., 1960; Swellengrebel et al., 1940) but this hypothesis was later challenged (Alizon et al., 2009). In mathematical models, virulence is often treated as a fixed model parameter expressing the excess mortality rate caused by the pathogen. For instance, virulence has been assumed to depend on the intrinsic reproductive rate of the parasite (Bremermann and Pickering, 1983). Other research expresses

* Corresponding author.
E-mail address: cbauch@uwaterloo.ca (C.T. Bauch).

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the transmission rate β and the recovery rate μ in terms of a parameter ν that represents virulence (Day, 2001). When the impact of human behaviour is discussed in such models, it is discussed in terms of hypothesized effects of human behaviour on the value of the fixed parameter representing virulence. A Human Immunodeficiency Virus (HIV) virulence model by Massad (1996) shows that reducing the number of sexual partners could possibly drive HIV to be a more benign pathogen. However, the model assumes that the number of sexual partners can simply be moved up or down as a model parameter, whereas in reality the number of sexual partners in a population is the outcome of a dynamic socio-epidemiological process that merits its own mechanistic modelling, and itself responds to pathogen virulence. In general, these models do not treat human behaviour as a dynamic variable that can evolve in response to transmission dynamics and influence the evolution of virulence. (A few exceptions exist, including work that allows virulence to be a function of the number of infected hosts, thus capturing a situation where the magnitude of the epidemics affects the ability of health care services to host patients (Dieckmann, 2002).) However, as human responses to both endemic and emerging infectious diseases show, human behaviour can have a significant influence on how infections get transmitted. For instance, an early and well-documented example shows how the residents of the village of Eyam, England quarantined themselves to prevent the spread of plague to neighbouring villages (Scott and Duncan, 2001). Individuals moved to less populated areas during the Spanish Influenza pandemic in the early 20th century (Crosby, 2003). More recently, masks became widely used during the outbreak of the Severe Acute Respiratory Syndrome (SARS) at the beginning of the 21st century (Lau et al., 2005), and it has been shown pathogen virulence in Marek's disease can evolve in response to how vaccines are used (Read et al., 2015).

Theoretical models of the interactions between human behaviour and the spread of infectious diseases are increasingly studied (Bauch and Bhattacharyya, 2012; Bauch et al., 2003; 2005; Fast et al., 2015; Funk et al., 2010; Pandey et al., 2014; Shaw and Schwartz, 2008). For instance, Bagnoli et al. (2007) found that under certain conditions, a disease can be driven extinct by reducing the fraction of the infected neighbours of an individual. Zanette and Risau-Gusmán (2008) showed that if susceptible individuals decide to break their links with infected agents and reconnect at a later time, then the infection is suppressed. Gross et al. (2006) also shows that rewiring of edges in a network (and thus social interaction) can greatly influence the spread of infectious diseases. Of the compartmental models, we focus on those that have used concepts from evolutionary game theory such as imitation dynamics (Bauch and Earn, 2004) to describe the evolution of behaviour and its interplay with the epidemics. An example of imitation dynamics concerns, as described in detail in Bauch (2005), the effect of vaccination on the spread of infectious diseases. Each individual in the population picks one strategy and adopts it: "to vaccinate" or "not to vaccinate". The proportion of vaccinators is modelled using an ordinary differential equation and is coupled with a standard SIR model. An important aspect of behavioural models is to couple them with epidemiological processes such as transmission. This coupling creates a feedback loop between behaviour and spread of the disease.

Given that adaptive social behaviour is important in many aspects of infection transmission, we hypothesize that adaptive social behaviour can also influence selection between pathogen strains with differing virulence in ways that cannot be captured by assuming it to be represented by a fixed parameter. Our objective in this paper was to explore how behaviour and virulence influence one another, in a coupled behaviour-disease differential equation model. The model allows individuals who perceive an increase in the prevalence of infection to increase their usage of practices that reduce transmission rates (such as social distancing and handwashing) and thereby boost population-level immunity. This approach can help us understand the effects specific social dimensions, such as level of concern for a strain or the rate of social learning, on the coupled dynamics of pathogen strain emergence and human behaviour in a situation where virulence imposes evolutionary trade-offs and is strain-specific. Instead of considering long-term evolutionary processes with repeated rounds of mutation and selection, we focus on the case of invasion of a single mutant strain with a large phenotypical difference compared to the resident strain. In the next section, we describe a model without adaptive social behaviour as well as a model that includes it, and in the following Results section we will compare their dynamics.

2. Model

We compare dynamics of a two-strain compartmental epidemic model in the presence and absence of adaptive social behaviour. Individuals are born susceptible (S). They may be infected either by a resident strain (I_1) or a mutant strain (I_2). For simplicity, we assume that co-infection and super-infection are not possible. Infected individuals can either recover (R) or die from infection. We furthermore assume that recovery from either strain offers permanent immunity to both strains. The system of differential equations representing the SI_1I_2R model in the absence of adaptive social behaviour (we will refer to this as the "uncoupled model" throughout) is given by

$$\begin{aligned}
\frac{dS}{dt} &= \mu - \delta S - \beta_1 S I_1 - \beta_2 S I_2, \\
\frac{dI_1}{dt} &= \beta_1 S I_1 - (\gamma_1 + \delta + \nu_1) I_1, \\
\frac{dI_2}{dt} &= \beta_2 S I_2 - (\gamma_2 + \delta + \nu_2) I_2, \\
\frac{dR}{dt} &= \gamma_1 I_1 + \gamma_2 I_2 - \delta R,
\end{aligned} \tag{1}$$

where β_1 (β_2) represents the transmission rate of the resident (mutant) strain; γ_1 (γ_2) represents the recovery rate from the resident (mutant) strain; ν_1 (ν_2) represents the death rate from the resident (mutant) strain due to infection (virulence); μ is a birth rate and δ is the background death rate. All variables represent the number of individuals with the given infection status (for instance, S is the number of susceptible individuals). Since R does not appear in the other equations, we can omit R from the analysis.

The system of differential equations in the presence of adaptive human behaviour couples the SI_1I_2R epidemic spread with a differential equation for human behaviour ("coupled model"). Each individual in the population can choose to accept or reject behaviours that reduce infection risk (e.g. washing hands, wearing a mask, social distancing), and individuals imitate successful strategies observed in others. Let x represent the proportion of individuals accepting preventive behaviour (we will call these "protectors"). Individuals sample others in the population at rate κ, representing social learning. The choice is based on the perceived severity ω_1 (resp. ω_2) from the resident (resp. mutant) strain, where ω_1 (resp. ω_2) can be quantified as the probability that an infection by the resident (resp. mutant) strain results in a severe case of disease. The more severe cases the population observes, the more attractive preventive behaviour becomes: we assume that individuals respond to the total number of severe cases $\omega_1 I_1 + \omega_2 I_2$ they observe at a given time. Preventive behaviour is not always completely effective. We introduce efficacy of infection control ϵ_1 (ϵ_2) against the resident (mutant) strain. The efficacy of infection control influences the transmission process. The more effective infection control is against a strain, the less likely it will be transmitted.