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Partnership dynamics and strain competition

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Abstract

Models of epidemic spread that include partnership dynamics within the host population have demonstrated that finite length partnerships can limit the spread of pathogens. Here the influence of partnerships on strain competition is investigated. A simple epidemic and partnership formation model is used to demonstrate that, in contrast to standard epidemiological models, the constraint introduced by partnerships can influence the success of pathogen strains. When partnership turnover is slow, strains must have a long infectious period in order to persist, a requirement of much less importance when partnership turnover is rapid. By introducing a trade-off between transmission rate and infectious period it is shown that populations with different behaviours can favour different strains. Implications for control measures based on behavioural modifications are discussed, with such measures perhaps leading to the emergence of new strains.

Keywords: Epidemiology; Strain competition; Partnership model; Basic reproductive ratio

1. Introduction

Understanding the spread of infectious diseases is a complicated business, with the characteristics of both host and pathogen being variable and uncertain. A wide range of mathematical models has been developed to investigate key parameters and make usable predictions about epidemic spread and control (Anderson and May, 1991; Ferguson et al., 2003, 2005; Keeling et al., 1997, 2001; Longini et al., 2005); by their nature such models simplify reality, by ignoring aspects of differences between individuals, variation between pathogens, and so on. One common assumption is that host individuals interact at random—this greatly simplifies the model and is often, given a paucity of data, the only reasonable approach. However, it has been recognized that the common assumption of instantaneous interactions between members of a population is inappropriate in many situations (Dietz and Hadeler, 1988; Keeling et al., 1997, Kretzschmar and Morris, 1996). It is often the case that contacts between individuals exist for some non-negligible duration, and this has particularly been considered in the context of sexually transmitted diseases (STDs), for which the relevant sexual partnerships may be of considerable length (Johnson et al., 2001; Kretzschmar et al., 1996; Wellings et al., 1994). A number of models have been developed that include the partnership status of individuals (Dietz and Hadeler, 1988; Kretzschmar et al., 1996; Kretzschmar and Dietz, 1998; Kretzschmar and Morris, 1996). In such models, it is only between individuals within a sexual partnership that infection can be transmitted.

The inclusion of serially monogamous partnerships within models of epidemic spread has a number of effects on the dynamics of infection. Disease spreads through the population much less quickly, since each individual is only capable of passing infection to one contact at a time; for infection to travel further the current partnership has to break up and a new partnership form between an infectious and a susceptible individual. The speed at which infection moves through the population depends to a large extent on the partnership behaviour observed; if partnerships are too

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short then individuals spend most of their time single and infection will not spread—partnerships must persist for long enough to allow infection to be transmitted. However, if partnerships are too long then infection cannot emerge into the wider population. It has been shown that an intermediate partnership break-up rate maximizes the speed of spread of an epidemic (Kretzschmar and Dietz, 1998).

This paper presents a partnership model of simple STDs and investigates how competition between pathogen strains is affected by finite partnerships. The additional time-scale introduced to the system by the consideration of partnerships means that there are additional pressures on any pathogen. For an infection to persist it must be sufficiently infectious to be transmitted during a partnership and sufficiently long-lived to endure the periods between partnerships. Therefore, effect of partnership considerations on the evolutionary pressures experienced by pathogens is considered. In particular, it is shown that the behaviour of the population determines the success of a pathogen and the characteristics of the optimal pathogen strain. This effect is not seen in models that do not consider partnerships. By introducing a trade-off function constraining the relationship between transmission rate and infectious period it is shown that changes in population behaviour can result in changes in pathogen properties and that interventions based on modifying population behaviour may be less successful than might be hoped.

2. The model

A model for the spread of a relatively simple infection, based on that developed in Dietz and Hadeler (1988) is presented. Individuals can be in one of two infection states: susceptible or infected. On recovery, individuals become susceptible once more, leading to susceptible–infected–susceptible (SIS) dynamics (Hethcote and Yorke, 1984). Furthermore, we assume that individuals can only become infected when in a partnership, and that individuals are serially monogamous: that is, they have at most one partner at any time. Existing partnerships can break up and partnerships can form between any two single individuals. We model these processes (infection, recovery, and partnership dynamics) with a series of differential equations. We define the variables as follows: S and I are the numbers of unpartnered (single) susceptible and infected individuals. The numbers of partnerships of different types are denoted P_{SS}, P_{SI} and P_{II}, with pairs counted once in each direction (this means that a partnership between two susceptible individuals contributes two to P_{SS} and that $P_{SI} = P_{IS}$). The variables satisfy

$$S + I + P_{SS} + 2P_{SI} + P_{II} = N, \quad \text{the fixed population size.} \tag{1}$$

Formulation of a model requires some assumptions about the process of pair formation: following Kretzschmar and Dietz (1998) we assume that partnerships break up at rate ρ and that the rate of formation of new partnerships is proportional to the number of single individuals. This allows partnership formation to take place at a rate determined by the preferences of the individual rather than through chance interactions within the population; thus the rate with which a single individual forms a new partnership does not depend on the number of single individuals in the population; partnership behaviour is independent of the population size.

We can now write down the following system of equations:

$$\dot{S} = gI - \alpha S + \rho(P_{SI} + P_{SS}), \tag{2}$$

$$\dot{I} = -gI - \alpha I + \rho(P_{SI} + P_{II}), \tag{3}$$

$$\dot{P_{SS}} = 2gP_{SI} - \rho P_{SS} + \alpha\frac{S^2}{S+I}, \tag{4}$$

$$\dot{P_{SI}} = g(P_{II} - P_{SI}) - \tau P_{SI} - \rho P_{SI} + \alpha\frac{SI}{S+I}, \tag{5}$$

$$\dot{P_{II}} = -2gP_{II} + 2\tau P_{SI} - \rho P_{II} + \alpha\frac{I^2}{S+I}, \tag{6}$$

where τ is the transmission rate within a partnership, g the recovery rate, ρ the rate of partnership break-up and α the rate of partnership formation.

3. Analysis

Considering a disease-free population, the equations are reduced to

$$\dot{S} = -\alpha S + \rho P_{SS}, \tag{7}$$

$$\dot{P_{SS}} = -\rho P_{SS} + \alpha S, \tag{8}$$

with $S + P_{SS} = N$. At equilibrium, this means that a proportion $\alpha/(\alpha+\rho)$ of the population is in a partnership. Since infection can only be transmitted by individuals within the context of a partnership one might expect that by maximizing this proportion prevalence of infection might also be maximized. This is not so, however; although increasing α does indeed increase prevalence, the effect of the partnership break-up rate ρ is somewhat different (Fig. 1(a)); prevalence is maximized at some intermediate value. When partnerships break up too rapidly too few individuals are paired up to allow infection to be transmitted; conversely, when partnership turnover is too slow infection is trapped within a few pairs and cannot spread further. In the limit $\rho \to \infty$ or $\rho \to 0$ the population reaches a disease-free equilibrium.

3.1. Strain competition

The equations above can easily be adapted to allow for multiple strains of infection. If an individual can only be infected with one strain at any one time then within a homogeneous population only a single strain will persist. In this section we examine how the identity of the