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Modelling contact spread of infection in host–parasitoid systems: Vertical transmission of pathogens can cause chaos

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ABSTRACT

All animals and plants are, to some extent, susceptible to disease caused by varying combinations of parasites, viruses and bacteria. In this paper, we develop a mathematical model of contact spread infection to investigate the effect of introducing a parasitoid-vectored infection into a one-host–two-parasitoid competition model. We use a system of ordinary differential equations to investigate the separate influences of horizontal and vertical pathogen transmission on a model system appropriate for a variety of competitive situations. Computational simulations and steady-state analysis show that the transient and long-term dynamics exhibited under contact spread infection are highly complex. Horizontal pathogen transmission has a stabilising effect on the system whilst vertical transmission can destabilise it to the point of chaotic fluctuations in population levels. This has implications when considering the introduction of host pathogens for the control of insect vectored diseases such as bovine tuberculosis or yellow fever.

1. Introduction

There is a rapidly accumulating body of evidence demonstrating that many taxa of insects harbour a considerably greater variety of micro-organisms than had previously been suspected, including relatively benign symbionts at one end of a continuum, through to manipulative intracellular parasites at the other (Douglas, 2008; Engelstadter et al., 2007; Kenyon and Hunter, 2007). Microbial pathogens vectored by insects have been mostly well described, as have those infections which kill insects. By contrast, primary endosymbionts have been much less well studied, and secondary endosymbionts remain extremely poorly characterised. As the study of ecological diversity within this last group of bacteria develops it becomes increasingly evident that it is likely that they exercise considerable influence over interspecific interactions, community structure and species biodiversity in insect multi-trophic assemblages (Blaustein and Kiesecker, 2002; Murray, 2002; Collins and Storfer, 2003; Hatcher et al., 2006), and that they may well play significant roles in the dynamics of both competitive and parasitic relations (Anderson, 1995; Tompkins and Begon, 1999; Bowers and Turner, 1997; Tompkins et al., 2003).

The study of host–pathogen interactions has received a good deal of recent attention (Briggs and Godfray, 1995, 1996; Grenfell and Dobson, 1995; Hudson et al., 2002; Bonsall, 2004; Elderd et al., 2008), but the theoretical investigation of disease dynamics in host–parasitoid systems has not kept pace with associated empirical research (Grenfell and Dobson, 1995; Gulland, 1995; Sait et al., 2000). It has been demonstrated that the inclusion of competitive disease dynamics into a model system can not only promote biodiversity of the system, but also can have sometimes unexpected effects on its dynamics (Holt and Roy, 2007). Preedy et al. (2006), for example, examined the consequences of introducing contact spread host infection into a simple model of a one-host–two-parasitoid system, and found that the presence of the infection not only promoted coexistence of the two parasitoid species, but also that it induced complex population dynamics in the system as a whole, including chaos, and when an explicitly spatial element was introduced into the model, complex spatio-temporal heterogeneity was observed. Clearly, however, infections may be transmitted not only through direct contact between susceptible and infected individuals, but also indirectly through a vector (Bonsall, 2004). In human systems, much has been done on

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the spread of malaria and yellow fever via mosquitoes (Aron and May, 1982; Anderson, 1982; Brauer and Castillo-Chavez, 2001), and host–parasitoid models have often been used as metaphors for the dynamics of such infections (see for example Barlow, 2000). However, the primary aim of these models has been to investigate the promulgation of the infection in the host population, and the consequences of the infection for the vectors themselves are often not considered. Indeed, vectored diseases are frequently modelled in host populations by a frequency-dependent process (McCallum et al., 2001) which takes no account of the effect of the pathogen on the vector population and which, when there are two vector (parasitoid) species involved, does not allow for indirect interaction between them.

Thus, a model which explicitly examines the dynamics of parasitoid populations may have very different outcomes from one which considers transmission via parasitoid attack to be a frequency-dependent process. The biology of host infections vectored by parasitoids is highly diverse. In the case of infection of Drosophila spp. by the bacterium Wolbachia, the vector for transmission of the bacterium may be a parasitoid which is itself infected by Wolbachia, from the same or a different strain. Indeed, the parasitoid may act as an agent for inter- as well as intra-species transmission (Haine et al., 2005). Ascoviruses infecting a host population of lepidoptera can be vectored by parasitoids with whom they may have a mutualistic, commensal or pathogenic relationship (Stasiak et al., 2005). A parasitoid may vector an infection between hosts without being affected itself, as in the case of Cotesia melanoscelus which has been shown to vector nuclear polyhedrosis virus (NPV) in the gypsy moth Lymantria dispar (Dwyer and Elkinton, 1995; Raimo et al., 1977), and there are various other NPVs recorded from host–parasitoid systems (Nguyen et al., 2005; Briggs et al., 1995). In this paper, we concentrate on the latter case, where the host is susceptible to a nonlethal infection which is carried by the parasitoid. The transmission of disease through parasitoid attack occurs when a parasitoid attacks an infected host, and its ovipositor becomes contaminated and the infection may be passed on when the contaminated ovipositor enters a susceptible host—the "dirty needle effect". We first describe a model of contact spread infection, then develop it to consider parasitoid vectored infection where only one parasitoid is present, and where there is no vertical transmission of the disease. We then consider a model in which there is perfect vertical transmission, and undertake an analysis of the steady-states of the model and their stability, as well as investigating transient dynamics using computational simulations. Such models are applicable not only to host–parasitoid systems, but also to an understanding of the role of infection in the structure of natural communities. The analytical and computational simulation results in this paper have implications for the effective application of biological control in agro-ecosystems and public health situations, and the general dynamics of disease processes.

2. Models and methods

Preedy et al. (2006) developed a mathematical model of the disease dynamics of host–parasitoid systems, modelling the (spatio-temporal) interactions between populations of uninfected hosts, infected hosts and two parasitoid species. They assumed infection was spread between hosts as a contact process, when an infected host encountered a susceptible, uninfected, host. The host population increased via density-dependent logistic growth and was depleted by parasitism by both parasitoid species that was modelled using an Ivlev functional response, an alternative to the Holling type II response (Sherratt et al., 1995; Pearce et al., 2006; Edelstein-Keshet, 1988; Petrovskii and Malchow, 1999, 2001). Infected hosts and both parasitoid species were subject to a linear death term, and infection was bilinear. It was found that in the absence of infection the weaker parasitoid competitor inevitably went extinct. When infection was included coexistence occurred for a wide parameter range. In the absence of vertical infection either extinction or stability was observed. However, when vertical infection was included, a much richer set of dynamics was observed—depending on the model parameters, the populations tended towards (i) a unique coexistence fixed point (a stable spiral), with the approach to the fixed point being able to be delayed depending on the initial conditions; (ii) a stable limit cycle, or (iii) a chaotic attractor.

In this paper we adapt this approach to a three species system—one host and two parasitoids—where infection is vectored by the parasitoids as a "dirty needle" process, rather than spread as a contact process between infected and susceptible hosts. We assume logistic growth of host populations and parasitoid search is modelled using an Ivlev functional response. However, in this paper we assume that infection is vectored from infected host to uninfected host by parasitoids that have previously parasitised an infected host and have acquired the infection which they carry on their ovipositors. Therefore, we assume the parasitoid may be in one of two infection states—"clean" or "carrier". We assume that successful parasitism by a carrier parasitoid always infects the host. However, it is possible for a host to be infected but resist parasitism through encapsulation of the parasitoid egg. We assume that there is vertical transmission of infection from infected host to its offspring at some rate that can vary between 0% and 100%. In this paper we present results for the two cases when there is no vertical transmission and when vertical transmission is 100% efficient (intermediate cases of vertical transmission would be straightforward to implement, and given the nature of the results presented in the later sections, we expect these would not introduce any new types of dynamics). We assume a simple linear death rate for parasitoids and an increased death rate of infected hosts over uninfected hosts.

The model therefore consists of six ordinary differential equations, representing the dynamics of H_u and H_i the uninfected and infected host populations and P_{1u}, P_{1i}, P_{2u}, P_{2i}, the noncarrier and carrier populations of the two parasitoid species P_1 and P_2.

2.1. Host dynamics

We assume the host population has logistic density-dependent growth with an intrinsic rate of growth ρ_u for uninfected hosts and ρ_i for infected hosts, with a carrying capacity H^*. We assume vertical transmission is at a rate ϕ, and correspondingly $(1-\phi)$ is the fraction of the growth rate of infected hosts that results in susceptible offspring. We model the parasitoid searching efficiency using the Ivlev functional response $(1-e^{-\mu H})$, where $H=H_u+H_i$ and μ is a parameter representing the parasitoid's ability to detect hosts. We assume that parasitoid species P_k attacks healthy hosts at a maximal rate of α_{ku} with probability of success β_{ku} and infected hosts at rates α_{ki} with probability of success β_{ki}. We assume successful parasitism of a host by a carrier parasitoid always leads to infection of the host and that unsuccessful attack of an uninfected host by an infected parasitoid leads to infection of the host at rates of ν_k. We also assume the infection induces an additional mortality at a rate ξ_i in infected hosts. These assumptions lead to the following equations for host