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Dynamics of SI models with both horizontal and vertical transmissions as well as Allee effects ☆

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ABSTRACT

A general *SI* (Susceptible-Infected) epidemic system of host–parasite interactions operating under Allee effects, horizontal and/or vertical transmission, and where infected individuals experience pathogen-induced reductions in reproductive ability, is introduced. The initial focus of this study is on the analyses of the dynamics of density-dependent and frequency-dependent effects on *SI* models (*SI*-DD and *SI*-FD). The analyses identify conditions involving *horizontal and vertical transmitted reproductive numbers*, namely those used to characterize and contrast *SI*-FD and *SI*-DD dynamics. Conditions that lead to disease-driven extinction, or disease-free dynamics, or susceptible-free dynamics, or endemic disease patterns are identified. The *SI*-DD system supports richer dynamics including limit cycles while the *SI*-FD model only supports equilibrium dynamics. *SI* models under "small" horizontal transmission rates may result in disease-free dynamics. *SI* models under with and inefficient reproductive infectious class may lead to disease-driven extinction scenarios. The *SI*-DD model supports stable periodic solutions that emerge from an unstable equilibrium provided that either the Allee threshold and/or the disease transmission rate is large; or when the disease has limited influence on the infectives growth rate; and/or when disease-induced mortality is low. Host-parasite systems where diffusion or migration of local populations manage to destabilize them are examples of what is known as *diffusive instability*. The exploration of *SI*-dynamics in the presence of dispersal brings up the question of whether or not diffusive instability is a possible outcome. Here, we briefly look at such possibility within two-patch coupled *SI*-DD and *SI*-FD systems. It is shown that relative high levels of asymmetry, two modes of transmission, frequency dependence, and Allee effects are capable of supporting *diffusive instability*.

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1. Introduction

Parasitism contributes to the selection of future generations of hosts through their impact on factors that lead to reductions in fitness [31] and as a result, wildlife managers must account for emerging and/or re-emerging diseases. Competition for space and resources (finding mates or food) also impact the reproductive ability and likelihood of survival of individuals, particularly those housing pathogens or parasites. Hosts' dynamics (survival in particular) often depends on the ability of a population to maintain a critical mass [36]. The impact of heterogenous transmission factors including multiple transmission modes by altering a population's dynamics may lessen the plausibility of conservation goals or the economic viability of selected management policies [44]. Hence, it is not surprising that the pressure which parasites or pathogens place on their hosts and the relation of such interactions to community and/or ecosystem structure has been the subject of continuous empirical and theoretical studies. Some of the theoretical consequences associated to host-pathogen dynamics with factors like: (i) multiple modes of disease transmission; (ii) host population density; and (iii) the presence or absence of critical host population thresholds, are addressed in this manuscript.

Modes of disease transmission, like horizontal and vertical, differentially facilitate the colonization of host populations by

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bacteria, fungi, or viruses. Colonization (horizontal transmission) is sometimes seen as the result of close interactions (contacts) between disease-free host and infected individuals. A contact process that implicitly assumes the sharing of a common, local habitat. The passage of a disease-causing agent from a mother to offspring during the " birth" process is also sometimes possible (vertical transmission). Feline leukemia (FeLV) and feline immunodeficiency (FIV) viruses are transmitted horizontally and vertically. Leishmaniasis, a disease caused by the protozoan parasite *Leishmania infantum*, is transmitted horizontally and vertically. Domesticated dog populations are presumed to be a reservoir for *Leishmania infantum*; a reservoir maintained by the differential contributions of multiple modes of transmission [46]. The deadly *septicaemia,* which manages to kill 80% of *septicaemia*-infected birds, gets lodged in the ovary of surviving birds; passed later to the birds eggs (vertical transmission); spreading horizontally within the hatcher and brooder.

Traditionally, density-dependent transmission (DDT) and frequency-dependent transmission (FDT) are two extreme forms of disease transmission. DDT refers to parasite transmission in which the rate of contact between susceptible hosts and the source of new infections increases with host density while FDT refers to the rate of contact between susceptible hosts and the source of new infections is independent of host density. Teasing out the roles of density- and frequency-dependent transmission (DDT versus FDT) on the dynamics of host-parasite systems is carried out for theoretical and policy reasons. FDT is the result of density-independent contact rates between susceptible and infected individuals. DDT assumes that *infection risks increase* with host density. Density-dependent transmission (DDT) may require a minimal number of available susceptible hosts, that is, a threshold density, for transmission to occur. Density-dependent parasitic disease transmission plays a role in regulating host population size [3] while frequency-dependent parasitic transmission does not require host density thresholds or regulatory host population constraints on the birth or death rates to "work".

In population biology we often lack absolutes. And so, vector- and sexually-transmitted diseases have been seen to thrive in frequency-dependent transmission settings while density-dependent infections that lead to pathogens being shed by infected hosts into common environments may sometimes need a critical mass of susceptible individuals to thrive [4,5]. Pathogens can be spread via "direct" contacts (kissing can spread herpes viruses), aerosol (sneezing can spread influenza viruses), or via indirect contacts (ingesting water contaminated with fecal material can cause result in cholera infections), or through vectors (ticks and mosquitoes often spread viruses and bacteria to their hosts), or via some combination of direct and indirect modes, sometimes mediated by a vector. Empirical work on mice, voles, lady bird beetles, frogs, and plants has shown that pathogen transmission often involve DD and FD transmission modes, with one predominant mode [31]. The negative impact of deliberate releases of pathogens via aerosol or in water systems tends to increase with host density. On the other hand, sexually transmitted pathogens seem to thrive equally well or bad in small or large population settings while some vector-borne diseases have been shown to support frequency-dependent transmission patterns [4,5,21]. Antonovics and Alexander [5] manipulated the density and frequency of infected hosts *Silene latifolia* and in the process they found out that deposition of the anther smut fungus *Microbotryum violaceum* by pollinating insects managed to increase with the frequency of infection.

A pathogen may or may not be deleterious enough to regulate the dynamics of host populations and so it is not surprising that the impact of pathogens on hosts is tied in to virulence. Pathogen's levels of virulence differentially impacts host's fitness. Often, increases in virulence result in a reduced probability survival or a diminished ability of a host to reproduce successfully, or both [3,31,27]. Pathogens whose transmission successes increases with host density seem to have managed to select for variants capable of regulating a host population. [19] studied a host-pathogen system where a detailed account of virus titer on infected hosts could be estimated. Their study focused on studying the ability of the Myxoma virus to control an exploding rabbit populations over a long window in time. Empirical evidence from systems involving conjunctivitis in house finches or parasitic nematodes in red grouse and feral Soay sheep provide an example of a system where disease regulates population size [24,30,29]. Pathogen infections are contributors to the decline or the extinction of some species [19,14,49,52]. The deleterious role of *chytridiomycosis* in amphibians, chestnut blight in American chestnuts, avian malaria in Hawaiian birds, devil facial tumour disease in Tasmanian devils, or sudden oak death in Californian trees provide classical examples of the role of disease in regulating a population. Theory suggests that density-dependent specialist pathogens (i.e., those infecting a single host) alone rarely drive their hosts' extinction but can lead to extinction of the pathogen while frequency-dependent transmission may be capable of supporting significant decreases, including the potential extinction of host and parasite populations in the presence of moderately lethal pathogens [10,21,39].

The impact of disease outbreaks can be devastating and their dynamics must be particularly monitored within populations near extinction; that is, those with population levels near established Allee effects thresholds [1,12,35,50,51]. The relevance of threshold effects has been identified within a wide array of taxa [12,40]. Populations under Allee effects or facing extinction or both must be effectively managed [16,27]. The fragility of these populations means that limiting the transmission of highly deleterious diseases is critical [11,27]. Recurrent infectious disease outbreaks tend to enhance the deleterious role of Allee effects within diseases capable of inducing reductions in host fitness [25,11,54,27,52,28,22,37,38]. The results of this manuscript seem to be in sync with the overall conclusions reached the study of predator–prey systems (e.g., [13,20,18,33,8,34,36]).

Parasites and hosts co-evolve in response to environmental clues and/or selective pressures [39]. Mammals, birds, fish, and insects generate mobility patterns as they track resources and as it is well known movement and/or dispersal can impact disease dynamics [2]. In short, mobility has been a key player in the evolution of host-parasite systems. Studies that in addition to disease and mobility (dispersal) also include the impact of Allee effects are not well understood [45,26,37,38]. Hilker et al. [26] used a reaction–diffusion *SI* model within a frequency-dependent transmission framework in their explorations of the impact of disease and mobility on the spatiotemporal patterns of disease transmission. *SI* models that incorporate disease-reduced fertility have been explored by a number of researchers (see [17,7]). In [38] a two-patch SI model with density-dependent transmission is used to show that the differential movement of susceptible and infected individuals can enhance or suppress the spread of a disease. A *SI* model that incorporates a horizontally and vertically transmitted disease; infectives giving birth to infectives; susceptibles giving birth to susceptibles; Allee effects within the net reproduction term; disease-induced death rate; and disease reduced reproductive ability, is used in this manuscript to begin to address questions that include: What is the role of multiple modes of transmission? Will density-dependent and frequency-dependent vertical transmission affect host-parasite dynamics differentially? Under what conditions would Allee effects alter disease-free dynamics or facilitate disease-driven extinction? Would Allee thresholds on reproductive fitness become altered (reduced) by disease? What is the role of DDT or FDT in support of diffusive instability?