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Commensal pathogens as a source of a coexistence mechanism

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HIGHLIGHTS

- Coexistence can emerge through spatial segregation in the presence of mutually lethal viruses.
- Coexistence can only emerge in discrete stochastic simulations.
- In two dimensions, one dimensional domain walls emerge to produce large uni-population domains.

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ABSTRACT

Most known organisms carry commensal viruses or bacteria. These parasites are often treated as an inevitable nuisance. We here show that they may be essential for the survival of the host species, and may actually be the force driving speciation.

Viruses that do not hurt their natural host but are deadly for other species have been argued to facilitate invasion. We show using a generic SIR model that the opposite may be the general case. Such viruses may be the force sustaining multiple distinct populations through spatial segregation, in the absence of physical barriers. This segregation protects the hosts against invasion by neighboring, possibly more fit, populations. The virus induced segregation can eventually lead to allopatric speciation, with no animal dispersal, geographical changes or human activities.

We further propose a speculative mechanism, where the introduction of a new virus to a population with a heterogeneous response (based for example on the MHC polymorphism) may lead to the segregation of distinct sub-populations reacting to different strains of the virus. The existence of such a mechanism will require further experimental validation.

1. Introduction

Pathogens have been proposed to play a critical role in invasions by plants and animals. In such events, pathogens can determine which of the populations survives (Prenter et al., 2004; Settle and Wilson, 1990). This effect has been studied in field studies, theoretical models (Torchin et al., 2002; Hoogendoorn and Heimpel, 2002) and in dynamical models based on field-derived parameters (Borer et al., 2007), at the single species level or at the ecological network level (Roy and Lawson Handley, 2012). A well known such example are red and gray squirrels. The parapoxvirus disease is not harmful to gray squirrels, but kills red squirrels (Tompkins et al., 2002). In the recent invasion of gray squirrels in the UK, this virus led to local extinctions of red squirrels, easing the gray squirrel invasion (Rushton et al., 2001). Such models and observations characterize the result of invasions, where species cross a natural boundary or are imported into a new ecological system.

The effect of pathogens on diversity has been studied in a few other contexts (e.g. Tompkins et al. (2001)), and in most cases the effect of pathogens was to decrease the host diversity (by allowing one host to take over the other or exclude the other) (De Castro and M Bolker, 2005), or in the best case leave it constant. An exemption to that would be models with one strain having an advantage in the absence of the pathogen, and the other having an advantage in its presence (Lanchier and Neuhauser, 2006, 2010).

A seemingly contradicting observation is that many organisms carry commensal viruses that are hostile to similar species, competing for a similar niche (Hudson and Greenman, 1998; Tompkins et al., 2002). For example, among SIV viruses, there is good evidence of non-pathogenicity for African green monkeys and sooty mangabeys (Laguette et al., 2010). The chimpanzee virus (SIVcpz) appears to cause AIDS in chimpanzees (Apetrei et al., 2004) (chimpanzees acquired this virus recently from other monkeys); and obviously does (as HIV-1) in humans (Sharp and Hahn, 2010; Huet et al., 1990), the sooty mangabey virus causes AIDS in humans (Apetrei et al.,

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2004) (as HIV-2), and in macaques (as SIVmac, SIVstm, SIVmne). In other organisms, the Hendra virus and the Nipah virus are bat viruses that kill other organisms (Nel and Weyer, 2007; Eaton et al., 2005). Phocine morbilliviruses are thought to have one seal species as a natural host, but cause lethal epidemics in other seal species, as observed in dead seals washed up on the shores of the North Sea. These are but a few of the commensal pathogens lethal in similar hosts. However, in contrast with the theories and examples of viral induced invasion, these hosts do not invade the domain of other hosts. Thus, in the more generic case, pathogens do not seem to facilitate invasion.

In the following sections, we show using a generic model that invasion and coexistence are just two sides of the same dynamical model. We propose that viruses can have the role of sustaining multiple distinct populations, each carrying a different virus, inducing spatial segregation, with no physical barrier. This segregation protects each population against invasion by possibly more fit populations. Events of invasion occur in the cases where symmetry is broken, and only one of the two populations has a protective virus. Thus, in contrast with current views, pathogens may actually be in most cases a source of diversity. The observed opposite cases of invasions may be the exception that proves the rule.

Specifically, we propose an extension of invasion models with hostile pathogens (Hudson and Greenman, 1998; MacNeil et al., 2003; Ricklefs, 2010) to two populations, each with a virus that is hostile to the opposite population (Hudson and Greenman, 1998; Tompkins et al., 2002), and show that the viruses can lead to a stable spatial segregation of highly similar species. The model results are not sensitive to the precise details of the host and virus dynamics, and are a generic feature of stochastic host–pathogen models.

Classical niche theory predicts that two sufficiently similar species cannot stably coexist following competitive exclusion (Gause, 2003; Hardin, 1960; MacArthur and Levins, 1967), and that stable coexistence requires significant ecological differences between species (Leibold, 1995; Chesson, 1991). However, there are numerous observations of apparently similar species coexisting (Bickford et al., 2007; Sáez and Lozano, 2005). A simple explanation for this coexistence emerging from our model is that the response to pathogens segregates similar populations to spatially distinct regions. The response to viruses that are lethal to all but a given sub-strain can lead to the emergence of domain walls, allowing the separation of each group of individuals not killed by a specific strain to survive irrespectively of their fitness in the absence of the virus.

The same model can be extrapolated to a putative mechanism of sub-speciation in the absence of physical barriers. The introduction of a virus in a population with a heterogeneous response to the virus can lead to the segregation of the population into regions with populations protected from specific strains of the virus, producing a virus induced barrier that no population can cross. Such a barrier can lead to allopatric speciation (Werth and Windham, 1991; Turelli et al., 2001), and may be proposed as a mechanism leading to speciation, when no physical barriers are observed.

2. Methods

2.1. Model

In order to understand the possible protective effect of viruses, we study a model, with two host populations consuming the same resource. Each population can be infected by two strains of the same virus/pathogen. Each population is killed by a single strain, and the other strain is harmless to it. In other words, each population carries a commensal pathogen/virus that kills only the other population.

The model in its most complex form can contain eleven elements: resources (denoted by C), two viruses (v_1 and v_2) and two populations (S_1 and S_2). Each population can carry both viruses but the virus is lethal only to the opposite population. Namely, v_1 kills population S_2, and v_2 kills population S_1. Each population can be infected by one or the other viruses or both, yielding 6 possible infected populations ($I^j_{k,l}$), where the superscript is the population type, and the subscript is the infecting virus type. For example, I^1_2 is population 1 infected by virus 2, and $I^2_{1,2}$ is population 2 infected by the two viruses. We present here results using the simplest possible SIR model. More complex model yields similar results. The model is solved by the following set of ODEs.

$$\begin{cases}
(a)\frac{dC}{dt}=\alpha-\delta_0 C-kC(a_1(S_1+I^1_1+I^1_2+I^1_{1,2})+a_2(S_2+I^2_1+I^2_2+I^2_{1,2}))\\
(b)\frac{dS_1}{dt}=a_1S_1C-\delta_1S_1-\eta_1(v_2+v_1)S_1\\
(c)\frac{dS_2}{dt}=a_2S_2C-\delta_2S_2-\eta_2(v_2+v_1)S_2\\
(d)\frac{dI^1_1}{dt}=a_1I^1_1C-\delta_1I^1_1+\eta_1v_1S_1-\eta_1v_2I^1_1\\
(e)\frac{dI^1_2}{dt}=a_1I^1_2C-\delta_1I^1_2+\eta_1v_2S_1-\eta_1v_1I^1_2-\theta I^1_2\\
(f)\frac{dI^1_{1,2}}{dt}=a_1I^1_{1,2}C-\delta_1I^1_{1,2}+\eta_1v_2I^1_1+\eta_1v_1I^1_2-\theta I^1_{1,2}\\
(g)\frac{dI^2_1}{dt}=a_2I^2_1C-\delta_2I^2_1+\eta_2v_1S_2-\eta_2v_2I^2_1-\theta I^2_1\\
(h)\frac{dI^2_2}{dt}=a_2I^2_2C-\delta_2I^2_2+\eta_2v_2S_2-\eta_2v_2I^2_2\\
(i)\frac{dI^2_{1,2}}{dt}=a_2I^2_{1,2}C-\delta_2I^2_{1,2}+\eta_2v_2I^2_1+\eta_2v_1I^2_2-\theta I^2_{1,2}\\
(j)\frac{dv_1}{dt}=\beta_1(I^1_1+I^1_{1,2}+I^2_1+I^2_{1,2})-\delta_3v_1\\
(k)\frac{dv_2}{dt}=\beta_2(I^1_2+I^1_{1,2}+I^2_2+I^2_{1,2})-\delta_4v_2
\end{cases}\tag{1}$$

This model contains birth based on the consumption of a resource (C) of both susceptible (S) and infected (I), with a rate a_i. In parallel, there is a common natural death term with a rate of δ_i for the populations $-i=1,2$. There is an infection term by each of the viruses, and a viral production rate with a rate of β_i for the two viruses $i=1,2$. Finally, there is an asymmetric viral induced death term, where each virus only kills the opposite population with a rate of θ.

A first simplification of the original model [System (1)] is to assume a quasi steady state (QSS) on the viruses v_1, v_2 (i.e., to assume that the dynamics of the viruses are so rapid that we can assume that the viruses have reached a local equilibrium much faster than the other variables). The QSS system (v_1 and v_2) includes nine variables ($C, S_i, I^i_1, I^i_2, I^1_{1,2} i=1,2$), as described in Supplementary material A.

A second simplification of the system can be performed by assuming that the killing rate of the virus is very high ($\theta\sim\infty$ in equations (A15)–(A23) in Supplementary material A). More precisely, we assume it is much larger than other parameters. In such a case, we can ignore the equations containing θ, and the appropriate variables, since their concentration will be close to 0, leading to a drastic simplification of the system to five equations

$$\begin{cases}
(a)\quad \frac{dC}{dt}=\alpha-\delta_0C-kC(a_1(S_1+I_1)+a_2(S_1+I_2))\\
(b)\quad \frac{dS_1}{dt}=a_1S_1C-\delta_1S_1-\eta_1S_1I_2-\beta_1S_1I_1\\
(c)\quad \frac{dS_2}{dt}=a_2S_2C-\delta_2S_2-\eta_2S_2I_1-\beta_2S_2I_2\\
(d)\quad \frac{dI_1}{dt}=a_1I_1C-\delta_1I_1-\eta_1I_1I_2+\beta_1I_1S_1\\
(e)\quad \frac{dI_2}{dt}=a_2I_2C-\delta_2I_2-\eta_2I_2I_1+\beta_2I_2S_2
\end{cases}\tag{2}$$

Finally, if one assumes that the viruses are endemic, we can assume that $S<<I$, and ignore S in the equation, and solve the dynamics with only three variables, I_1, I_2 and C

$$\begin{cases}
(a)\quad \frac{dC}{dt}=\alpha-\delta_0C-kC(a_1I_1+a_2I_2)\\
(b)\quad \frac{dI_1}{dt}=a_1I_1C-\delta_1I_1-\eta_1I_1I_2\\
(c)\quad \frac{dI_2}{dt}=a_2I_2C-\delta_2I_2-\eta_2I_1I_2
\end{cases}\tag{3}$$