



The evolution of host defence to parasitism in fluctuating environments.

Charlotte Ferris*, Alex Best

School of Mathematics and Statistics, University of Sheffield, Sheffield, S3 7RH, UK



ARTICLE INFO

Article history:

Received 17 July 2017

Revised 8 November 2017

Accepted 4 December 2017

Available online 6 December 2017

Keywords:

Adaptive dynamics

Host-parasite

Host evolution

Seasonality

ABSTRACT

Given rapidly changing environments, it is important for us to understand how the evolution of host defence responds to fluctuating environments. Here we present the first theoretical study of evolution of host resistance to parasitism in a classic epidemiological model where the host birth rate varies seasonally. We show that this form of seasonality has clear qualitative and quantitative impacts on the evolution of resistance. When the host can recover from infection, it evolves a lower level of defence when the amplitude is high. However, when recovery is absent, the host increases its defence for higher amplitudes. Between these different behaviours we find a region of parameter space that allows evolutionary bistability. When this occurs, the level of defence the host evolves depends on initial conditions, and in some cases a switch between attractors can lead to different periods in the population dynamics at each of the evolutionary stable strategies. Crucially, we find that evolutionary behaviour found in a constant environment for this model doesn't always hold for hosts with highly variable birth rates. Hence we argue that seasonality must be taken into account if we want to make predictions about evolutionary trends in real-world host-parasite systems.

© 2017 Elsevier Ltd. All rights reserved.

1. Introduction

Given the ubiquity of infectious diseases in natural systems there is strong selection pressure on host organisms to evolve costly defence mechanisms. A wide range of theoretical work has been developed to understand the evolution of host defence against parasitism, with much of this work focused on the ecological/epidemiological feedbacks that drive selection of quantitative host defence (van Baalen, 1998; Best et al., 2008; 2009; Boots and Bowers, 1999; 2004; Boots and Haraguchi, 1999; Carval and Ferriere, 2010; M.H., 2006; Miller et al., 2005; 2007; Restif and Koella, 2003). These studies have explored how long-term, stable investment in host defence varies with ecological/epidemiological parameters, as well as determining the conditions that can lead to coexistence of strains through evolutionary branching. However, the vast majority of these studies assume that the populations live in a temporally static environment. In reality, almost all natural systems are subject to some degree of temporal environmental heterogeneity, in particular fluctuations caused by seasonality. For example, many natural species exhibit seasonal reproductive strategies driven by regular environmental fluctuations (Furness, 2016; Ketterson et al., 2015; Rowan, 1938; Stawski et al., 2014). It

is therefore essential that we consider the impact of fluctuating environmental conditions on the evolution of host defences.

It is well established that variable climates affect ecological systems (Ewing et al., 2016), including the spread and impact of diseases (Altizer et al., 2006; Fine and Clarkson, 1982; Finkenstädt and Grenfell, 2000). Many theoretical studies have considered the effects of seasonality in purely epidemiological models (i.e., non-evolutionary), often through a periodic transmission rate (Aron and Schwartz, 1984; Olsen and Schaffer, 1990; Schwartz and Smith, 1983). Increasing the amplitude of the transmission rate can generate sub-harmonic oscillations or cause the population dynamics to move through a series of period-doubling bifurcations, eventually leading to chaotic dynamics (Childs and Boots, 2010; Grassly and Fraser, 2006; Greenman et al., 2004; Grossman, 1980; Schwartz and Smith, 1983). Small perturbations in these seasonal models can also trigger the system to switch between distinct attractors, often due to resonance, potentially leading to significant changes in the population dynamics and different patterns of outbreaks (Greenman et al., 2004; Kamo and Sasaki, 2002; Keeling et al., 2001; Schwartz, 1985; Smith, 1983). These complex dynamics have been found to exist less frequently when seasonality is assumed to occur in the host birth rate rather than transmission (Begon et al., 2009; Dorélien et al., 2013; Duke-Sylvester et al., 2011; Peel et al., 2014; White et al., 1996). Predictions about the impact of a disease are likely to be more accurate when either of these types of sea-

* Corresponding author.

E-mail address: ceferris1@sheffield.ac.uk (C. Ferris).

sonality are included in the model (Kamo and Sasaki, 2002; White et al., 1996).

There is an increasing appreciation of the importance of temporal heterogeneity in host–enemy interactions within the experimental evolution literature (Blanford et al., 2003; Friman and Laakso, 2011; Harrison et al., 2013; Hiltunen et al., 2012), for example showing that rapidly fluctuating environments constrain co-evolutionary arms races in a bacteria–phage system (Harrison et al., 2013). Theoretically, however, evolution and seasonality have rarely been studied together in a host–parasite context. The few studies that do exist have either investigated evolution of only the parasite (Donnelly et al., 2013; Koelle et al., 2005; Sorrell et al., 2009), or used a genetic-based, rather than ecology-driven, model for evolution of the host (Nuismer et al., 2003 and Mostowy and Engestädter, 2011, but see Poisot et al., 2012). Seasonality in the host's birth rate does not affect the evolution of the parasite's transmission/virulence strategy unless a density-dependence is applied to virulence (parasite-induced mortality) (Donnelly et al., 2013). This occurs because the average susceptible density, and therefore the parasite fitness, doesn't depend on the seasonal parameters unless this density-dependence is included. Elsewhere, step-wise environmental variation implemented through a dynamic resource was found to change the coevolutionary outcomes in a gene-for-gene based host–parasite system (Poisot et al., 2012). In particular, they found that both the host and parasite invest more in resistance and infectivity respectively for higher amplitudes in the seasonality. However, we currently have no theory specifically addressing the impact that seasonality has on the evolution of host defence to parasitism.

Here we investigate the impact of a continuous seasonal birth rate on the evolution of quantitative host avoidance through small mutation steps using an evolutionary invasion (adaptive dynamics) method. We use a classic SIS (Susceptible–Infected–Susceptible) model, and focus on how the amplitude and period of the implemented seasonality impacts the ecological/epidemiological dynamics, and therefore the evolution of the host.

2. Methods

The population is modelled using an SIS (susceptible–infected–susceptible) framework with the following set of ordinary differential equations:

$$\frac{dS}{dt} = a(1 - qN)S - bS - \beta SI + \gamma I, \quad (1)$$

$$\frac{dI}{dt} = \beta SI - (b + \alpha + \gamma)I, \quad (2)$$

where S and I are the susceptible and infected population sizes respectively, and $N = S + I$ is the total population size (Anderson and May, 1981). All offspring are born susceptible at rate a , and only susceptible hosts are able to reproduce, i.e. the parasite renders the host (temporarily) sterile. The births are limited by density with crowding coefficient q , so that birth rate is low when competition is high. All hosts die at baseline mortality rate b , with an additional infected death rate α . The parasite is transmitted to susceptible hosts at rate βI due to contact with infected individuals. Hosts recover from the parasite at rate γ and return to the susceptible class with no acquired immunity. Default parameter values are given in Table 1.

We assume that seasonality occurs on the ecological timescale, so to incorporate this we let the birth rate depend periodically on time t :

$$a = a(t) = a_0(1 + \delta \sin(2\pi t/\epsilon)), \quad (3)$$

where a_0 is the average birth rate, $\delta \in [0, 1]$ is the amplitude and $\epsilon > 0$ is the period of the forcing. Periodic birth rates have been ob-

Table 1
Parameter definitions and default values.

Parameter	Definition	Default value
\hat{a}_0	Trade-off coefficient in the average birth rate	108
p	Trade-off coefficient in the average birth rate	103.75
c	Trade-off coefficient in the average birth rate	1.5
β	Transmission coefficient	Varies
β_{\min}	Minimum transmission coefficient	0.5
β_{\max}	Maximum transmission coefficient	10
δ	Amplitude of the birth rate forcing	Varies
ϵ	Period of the birth rate forcing	1
q	Crowding coefficient acting on births	0.1
b	Baseline mortality rate	1
γ	Recovery Rate	Varies
α	Virulence/additional death rate due to parasite	1

served in a large number of species (Ketterson et al., 2015; Rowan, 1938), and this type of function has been used many times to model a time-varying birth rate (Donnelly et al., 2013; Dorélien et al., 2013; He and Earn, 2007) or transmission rate (Childs and Boots, 2010; Grassly and Fraser, 2006; Schwartz and Smith, 1983). For our default parameter values, the period ϵ is the same as the average lifespan b (1 year), but see Section 3.4 for varying ϵ or Appendix F for alternative b .

We assume that the host evolves defence through the transmission coefficient (avoidance) β . We let the average birth rate depend on this as a trade-off so that there is a cost to resisting the parasite, as there is experimental support for such a relation to exist (Boots and Begon, 1993). We use the following trade-off function based on that used by White et al. (2006):

$$a_0 = a_0(\beta) = \hat{a}_0 - p \frac{\left(1 + \frac{\beta - \beta_{\min}}{\beta_{\max} - \beta_{\min}}\right)}{\left(1 + c \frac{\beta - \beta_{\min}}{\beta_{\max} - \beta_{\min}}\right)}, \quad (4)$$

where $\hat{a}_0 > 0$, $0 < p < \hat{a}_0$, $c > 1$ and $\beta \in [\beta_{\min}, \beta_{\max}]$. $a_0(\beta)$ has minimum $\hat{a}_0 - p$, and parameters p , c determine the gradient and curvature of the trade-off, which needs to have positive gradient: as the host invests in defence against the parasite (β decreases), less can be invested in reproduction ($a_0(\beta)$ decreases) (Boots and Haraguchi, 1999; Geritz et al., 2007). The constraints on the trade-off parameters give accelerating costs of defence, so that it is more costly to invest in resistance when defence is already high ($\frac{d^2 a_0(\beta)}{d\beta^2} < 0$), see figure A.1 in Appendix A. Accelerating trade-offs generally lead to evolutionary attractors (Hoyle et al., 2008), which will be our focus here.

We use the adaptive dynamics method to study evolution of the host in the transmission coefficient β . The method involves adding a rare mutant with susceptible and infected population sizes S_m , I_m and transmission coefficient β_m very close to the resident transmission coefficient β . We assume that mutants occur infrequently so that the resident population reaches the dynamic attractor of the population dynamics (generally a limit cycle here) before the next mutant is introduced (Geritz et al., 1998). When a new mutant arises, it is rare compared to the current population, so we assume that the resident remains at its limit cycle as long as the mutant population is small (Geritz et al., 1998). To analyse how the host evolves, we consider the mutant's fitness, defined to be the long-term exponential growth rate of the mutant in the current environment (Metz et al., 1992).

In the case where $\gamma = 0$, the fitness is relatively simple to find. We no longer have infected mutants (they are absorbed into I), and we can read off the time-varying growth rate $r(t)$ of the mutant host from the linearisation of the equation for the susceptible mutant ($dS_m/dt = r(t)S_m$, see Appendix B). Following the method from Donnelly et al. (2013), we can then take the average of this

Download English Version:

<https://daneshyari.com/en/article/8876846>

Download Persian Version:

<https://daneshyari.com/article/8876846>

[Daneshyari.com](https://daneshyari.com)