Population biology of multispecies helminth infection: Competition and coexistence

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Abstract

The role that interspecific interactions play in shaping parasite communities is uncertain. To date, models of competition between helminth species have assumed that interaction occurs through parasite-induced host death. To our knowledge, there has been no theoretical exploration of other forms of competition. We examine models in which competition acts at the point of establishment within the host, and at the time of egg production by the adult worm. The models used are stochastic and we allow hosts to vary in their rate of exposure to infective larvae. We derive the Lotka–Volterra model of competition when exposure is homogenous and thus demonstrate that two helminth species cannot coexist on a single limiting resource. We show that coexistence of species is promoted by heterogeneity in host exposure provided that the rates of exposure to the two species are not perfectly correlated, and, if they are positively correlated, provided that the degree of heterogeneity in host exposure is similar for the two competing helminth species. These results are robust to the mechanism of competition.

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1. Introduction

The existence of interspecific interactions is becoming increasingly well documented in helminth parasites of mammalian hosts (Christensen et al., 1987; Behnke et al., 2001). Although the overall importance of interspecific interactions in shaping helminth communities has been questioned (Kennedy, 1975), it is likely that both exploitation competition (individuals interact negatively on one another indirectly through a limiting resource), and interference competition (individuals have a direct negative effect on other individuals) play a role in structuring some parasite communities (Roberts, 2000). Simberloff (1990) discusses four possible mechanisms for within-host competition: (1) competition for space (exploitation); (2) competition for nutrients (exploitation); (3) local inflammation of the gut (interference); (4) cross-reactivity to specific immune responses (interference). In addition to these within-host mechanisms, if parasite infection increases host mortality, then exploitation competition occurs as the host itself becomes the limiting resource.

Each of the above-mentioned mechanisms of within-host competition may affect one or more parameters in the life-cycle of the parasite. Parameters that might be impacted by competition are: (1) the rate at which adult worms establish in the host; (2) the death rate of adult worms; (3) the death rate of the host; (4) the rate of egg production by the parasite. To date, mathematical modelling of competition between helminth species has focused on parasite-induced host mortality (Dobson, 1985; Roberts and Dobson, 1995; Gatto and De Leo, 1998; Pugliese, 2000); the effects of competition on life-history parameters of the worms themselves have, to our knowledge, not been investigated.

In this paper we use models based on Markov processes to explore the population dynamics of two competing species, where competition occurs either at the point of parasite establishment or during egg production by the
adult worm. The models are individual-based, and we allow exposure to vary between hosts. It is therefore possible to examine the effect of heterogeneity in host exposure on the coexistence of helminth species.

Heterogeneity in host exposure is thought to be one of the main mechanisms for generating the aggregated distribution of worm burden amongst hosts that is typically observed (Anderson and May, 1985a), clumping of infective stages being another (Isham, 1995; Quinnell et al., 1998). To date, both single and multispecies models of the dynamics of helminth infection have tended to incorporate aggregation phenomenologically by assuming that the relationship between mean and variance is as for the negative binomial distribution with the overdispersion parameter kept constant over time. However, recently it has been shown that the dynamics may be strongly dependent on the mechanism used to generate aggregation (Rosà and Pugliese, 2002). Furthermore, by incorporating aggregation-generating mechanisms into multispecies models of helminth infection, Pugliese (2000) has shown that coexistence is not necessarily promoted by these mechanisms, as previous phenomenological models seemed to suggest (Dobson, 1985; Roberts and Dobson, 1995).

We introduce the modelling framework that will be used in this paper with a model for a single helminth species in a population of hosts, where all hosts have the same rate of exposure to infective larvae and density dependence occurs at the point of establishment in the host. Subsequently, the assumption of homogeneous exposure is relaxed. For the model of two competing species initially we assume that intra- and interspecific competition occurs during parasite establishment, and we explore heterogenous as well as homogenous exposure. In addition, we allow competition to take place during a different stage of the parasite life-cycle by incorporating density-dependent parasite fecundity. We discuss the implications of our results in relation to the competitive exclusion principle and the structure of helminth communities.

2. Single species with density-dependent establishment

We begin by introducing a fully stochastic model for a single, directly-transmitted, helminth parasite species in a population of hosts. In this model, the population of infective stages is of size \( L \), and there are \( X_i \) adult worms within host \( i \), where \( L \) and \( X_i \) are random variables. Infective larvae are produced by the population of adult worms at a rate, \( \varepsilon \sum_{i=1}^n X_i \). Thus the parameter \( \varepsilon \) represents the product of the rate of egg production and the probability that an egg develops into a larva with the potential to infect a host. Larvae are lost from the larval population either through larval death, at a per capita rate \( \mu_L \), or through host infection and subsequent maturation to the adult stage. Host \( i \) comes into contact with infective larvae at a rate, \( \phi_i L \), therefore larvae are lost through infection at rate \( \sum_{i=1}^n \phi_i L \). We assume that density dependence acts on the rate at which worms become established in a host, so that a larva infecting host \( i \) survives to become established as an adult worm with probability \( (1 - \gamma X_i)^z \), where the notation \( z^+ \) should be interpreted as \( z^+ = z \) if \( z > 0 \) and \( z^+ = 0 \). Thus the current worm burden, \( X_i \), of host \( i \) affects the establishment of incoming larvae. The number of adult worms in host \( i \) decreases by one when a worm dies and becomes zero when the host dies. The per capita rate at which worms and hosts die are, respectively, \( \mu_X \) and \( \alpha \). It is assumed that the rate of host death, \( \alpha \), is independent of worm burden and that each time a host dies it is immediately replaced by an uninfected host. One might imagine, for example, a host population limited by the number of available territories: as soon as a territory becomes available it is filled by a young, uninfected host. This assumption ensures that the host population size is maintained at size \( n \).

The model is defined in terms of the following \( n + 1 \) dimensional Markov process for larval population in the environment, and adult worm burdens within each of the hosts. Parameter definitions are given in Table 1:

1. \( L \to L + 1 \) at rate \( \varepsilon \sum_{i=1}^n X_i \),
2. \( L \to L - 1 \) at rate \( (\mu_L + \sum_{i=1}^n \phi_i) L \),
3. \( X_i \to X_i + 1 \) at rate \( L \phi_i (1 - \gamma X_i)^z \),
4. \( X_i \to X_i - 1 \) at rate \( \mu_X X_i \),
5. \( X_i \to 0 \) at rate \( \alpha \).

To begin with we assume that each host has the same per larva rate of contact, \( \phi \), with the population of infective larvae so that \( \Phi_i = \phi \) for all \( i \). To facilitate analysis, the random variable \( L \) is replaced by its mean, \( m_L \), in the third transition rate defined above. The model is no longer fully stochastic; such ‘hybrid models’ of helminth infection have been proposed by Nåsell and Hirsch (1972) and Nåsell (1985). As a result of this simplification, and assuming that \( X_i(0) \) are independent and identically distributed (i.i.d.), then \( X_i(t) \) are i.i.d. for all \( t \). It is now straightforward to derive the following differential equations for the mean larval population size \( m_L(t) \), mean host worm burden, \( m_X(t) \), and the variance in worm burden, \( \sigma_X^2(t) \) (see Appendix A for technical details):

\[
\frac{dm_L}{dt} = \varepsilon n m_X - (\mu_L + n \phi) m_L, \tag{1}
\]

\[
\frac{dm_X}{dt} = m_L \phi (1 - \gamma m_X) - (\mu_X + \alpha) m_X, \tag{2}
\]

\[
\frac{d\sigma_X^2}{dt} = m_L m_\phi \{1 - \gamma (2 \sigma_X^2 + m_X) \} + \mu_X m_X + \alpha (2 \sigma_X^2 + m_X) \sigma_X^2. \tag{3}
\]

Technically, \( m_X(t) \) and \( \sigma_X^2(t) \) represent limiting values of the mean and variance as host population size tends to infinity. In practice, they describe the mean and variance in finite populations well, provided that these are of a

\[1\]The notation \( z^+ \) is introduced here because \((1 - \gamma X_i)^z\) can become negative if \( 1/\gamma \) is not an integer.
reasonable size (e.g. $n = 100$ hosts as in Fig. 5). The quantity $m_L(t)$ is the larval population size at time $t$ when stochastic variation intrinsic to the random variable, $L(t)$, is ignored. Imagining infinitely many realizations of the same process, i.e. infinitely many identical populations, $m_L(t)$ is the average larval population over all these realizations. Alternatively, when the distribution of larval population size is at equilibrium, $m_L$ may be viewed as the long-term average larval population size.

We simplify the model by assuming that the free-living stage of the parasite is short-lived relative to that of the adult worm. This assumption will not hold for certain helminth species, but it is often reasonable, (e.g. Table 15.2(c) Anderson and May, 1991), and is therefore frequently used in mathematical models of helminth infection, (e.g. Anderson and May, 1978; Pugliese, 2000). By assuming life-expectancy of adult worms is much greater than that of the free-living stages we are able to set $dm_L/dt=0$ (for a formal justification of this procedure based on Korzuhin’s theorem, see Klonowski, 1983), so that the dynamics of the mean and variance become

$$\frac{dm_X}{dt} = \dot{e}m_X(1-\gamma m_X) - (\mu_X + \alpha)m_X,$$

$$\frac{d\sigma_X^2}{dt} = \dot{e}m_X[1-\gamma(2\sigma_X^2 + m_X)] + \mu_Xm_X + \alpha m_X^2 - (2\mu_X + \alpha)\sigma_X^2,$$

where $\dot{e} = \epsilon \phi n/(\mu_L + \phi n)$ is the product of the rate, $\epsilon$, at which an adult worm produces potentially infective larvae and the probability, $\phi n/(\mu_L + \phi n)$, with which a larva survives to infect a host.

The dynamics of the mean worm burden (Eq. (4)) can be understood in terms of the basic reproductive number, $R_0 = \dot{e}/(\mu_X + \alpha)$. When $R_0 < 1$ the helminth species is unable to establish itself in the host population since the fixed point $m_X = 0$ is stable. When $R_0 > 1$, $m_X = 0$ is unstable and mean worm burden increases approaching an equilibrium where $m_X = (1/\gamma)(1 - R_0^{-1})$.

Since the model is stochastic, there is significant chance of extinction soon after parasites are introduced into the host population. We therefore emphasize that when discussing establishment of helminth infection in a host population, we refer to the tendency of the mean worm burden to increase from a zero value. Taking this approach, even if extinction occurs after one particular introduction, future introductions will eventually lead to the establishment of a parasite population when $R_0 > 1$.

At the non-zero equilibrium, assuming that the rate of host mortality, $\alpha$, is much smaller than the other rates, it is straightforward to show that $\sigma_X^2/m_X$, the variance to mean ratio of worm burden, is given by $R_0^{-1}$. If $R_0 > 1$ then $(\sigma_X^2/m_X) < 1$ which implies that, for this simple model, worm burden is distributed more evenly across hosts than if worms were assigned to hosts at random (which would lead to a Poisson distribution). However, empirical data show that the distribution of worm burden is almost always aggregated (Anderson and May, 1985a) so that worm burden is less evenly distributed amongst hosts than in the Poisson distribution; realistic models must therefore incorporate mechanisms for generating aggregation (Anderson and Gordon, 1982). One such mechanism is heterogeneity in host exposure, and a model incorporating heterogeneity in host exposure is developed in the next section.

### 2.1. Heterogenous exposure

It is well established that heterogeneous exposure is significant to the population dynamics of helminth infection. In host–parasite systems in which there is parasite-induced host death, heterogeneity has either indirectly (Anderson and May, 1978; May and Anderson, 1978) or directly (Rosà and Pugliese, 2002) been shown to stabilize the equilibrium at which both host and parasite coexist. The stabilizing effect of heterogeneity contrasts with the destabilizing effects of parasite-induced reduction in host fertility, and time delays introduced as a consequence of the maturation process in larval stages (Anderson and May, 1978).

Heterogeneity is incorporated into deterministic models either through the level of aggregation in worm burden (as measured by the parameter $k$ in the negative binomial distribution) (Anderson and May, 1978), or explicitly by assuming that hosts fall into different categories (usually just two) of susceptibility to infection (Anderson and May, 1985b; Pugliese, 2000; Rosà and Pugliese, 2002). We now extend the stochastic model described in the previous section to allow for heterogeneity in host exposure. It is

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Definition</th>
<th>Units</th>
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<tbody>
<tr>
<td>$\Phi_i$</td>
<td>per larva rate at which host $i$ is exposed to infective larvae</td>
<td>month$^{-1}$</td>
</tr>
<tr>
<td>$\gamma$</td>
<td>per worm reduction in probability of establishment</td>
<td>dimensionless</td>
</tr>
<tr>
<td>$\mu_X$</td>
<td>per capita death rate of adult worms</td>
<td>month$^{-1}$</td>
</tr>
<tr>
<td>$\mu_L$</td>
<td>per capita death rate of larvae</td>
<td>month$^{-1}$</td>
</tr>
<tr>
<td>$\alpha$</td>
<td>per capita death rate of hosts</td>
<td>month$^{-1}$</td>
</tr>
<tr>
<td>$\epsilon$</td>
<td>per capita rate of infective larvae production by adult worms</td>
<td>month$^{-1}$</td>
</tr>
</tbody>
</table>
mathematically convenient to do this by allowing each host to have a random rate of contact with infective larvae, \( \Phi_i \), where the \( \Phi_i, i = 1, \ldots, n \) are independent and i.i.d. each with mean \( m_\Phi \) and variance \( \sigma_\Phi^2 \). Since the \( \Phi_i \) may be discrete or continuous, each host can have a different susceptibility to infection (the continuous case) or hosts may fall into different categories of susceptibility (the discrete case) as in previous models.

A closed set of differential equations for the first and second moments of the distribution of worm burden can be derived (Appendix A). To do this, we use a hybrid approximation (Näsell and Hirsch, 1972; Näsell, 1985) in which we replace \( \sum_{i=1}^n \Phi_i \) by \( m_\Phi n \) in the rate for the transition \( L \rightarrow L - 1 \), and we assume that third order moments can be written in terms of first and second order moments as for the bivariate normal distribution (Chan and Isham, 1998). Setting \( dm_L/dt = 0 \) as before, the mean and variance of worm burden, \( m_X \) and \( \sigma_X^2 \), respectively, and the covariance between exposure and worm burden, \( \sigma_{\Phi X} \), satisfy

\[
\frac{dm_X}{dt} = \varepsilon m_X\left(1 - \gamma \left( \frac{\sigma_{\Phi X}}{m_\Phi} + m_X \right) \right) - (\mu_X + \sigma m_X),
\]

\[
\frac{d\sigma_X^2}{dt} = \varepsilon m_X \left(1 + 2 \frac{\sigma_{\Phi X}}{m_\Phi} - \gamma \left( 2 \frac{\sigma_{\Phi X}}{m_\Phi} m_X + 2 \sigma_X^2 + \frac{\sigma_{\Phi X}}{m_\Phi} + m_X \right) \right)
+ \mu_X m_X - 2 \mu_X + (2 \mu_X + \sigma) \sigma_X^2,
\]

where \( \varepsilon = n m_\Phi / (\mu_X + n m_\Phi) \).

For the approximation to be effective, it is sufficient that the relationships between higher and lower order moments are similar to those of the bivariate normal distribution; it is not necessary for the joint distribution of \( (\Phi, X(t)) \) to be bivariate normal. Indeed, since \( \Phi \) and \( X(t) \) are non-negative, their exact distribution cannot be bivariate normal, although it may be a good approximation if their means are sufficiently large relative to their standard deviations.

A greater degree of accuracy may be obtained by basing approximations on other joint distributions (Chan and Isham, 1998); or using higher moments. However, at this stage we restrict ourselves to the first two moments and use the bivariate normal moment closure to demonstrate analytically tractable results.

By making the change of variables \( \tau = \varepsilon t \) and \( \tilde{\sigma}_{\Phi X} = m_\Phi^{-1} \sigma_{\Phi X} \), Eqs. (6) and (8) can be written as

\[
\frac{dm_X}{d\tau} = m_X(1 - \gamma(\tilde{\sigma}_{\Phi X} + m_X)) - R_0^{-1} m_X,
\]

\[
\frac{d\tilde{\sigma}_{\Phi X}}{d\tau} = m_X(\nu_\Phi - \gamma(\tilde{\sigma}_{\Phi X} + \nu_\Phi m_X)) - R_0^{-1} \tilde{\sigma}_{\Phi X},
\]

where \( \nu_\Phi = \sigma_{\Phi}/m_\Phi \) is the coefficient of variation for the rate of exposure.

Again the dynamics are determined by the value of \( R_0 \). When \( R_0 < 1 \) the helminth species cannot establish itself in the host population, while when \( R_0 > 1 \) it tends to a non-zero equilibrium distribution. Using Eq. (9) it can be shown that there are two fixed points where \( m_X > 0 \) whenever \( \nu_\Phi > 1 \); however, only one of these is stable (Fig. 1). At the stable fixed point,

\[
m_X = \begin{cases} 
\frac{1}{2\gamma(\nu_\Phi - 1)} \left( \nu_\Phi^2 - 1 + 2R_0^{-1} \right), & \nu_\Phi \neq 1, \\
\frac{1}{\sigma_\Phi^2} (1 - R_0^{-1}), & \nu_\Phi = 1.
\end{cases}
\]

Fig. 1. Phase portraits of mean worm burden, \( m_X \), and the covariance of exposure and worm burden, \( \sigma_{\Phi X} \), scaled by mean exposure, \( m_\Phi \). One of two scenarios is possible, depending on the level of heterogeneity in host exposure as measured by \( \nu_\Phi \). In (a) \( \nu_\Phi < 1 \) and there is a single fixed point at which \( m_X > 0 \). In (b) \( \nu_\Phi > 1 \) and \( m_X \) is positive at two fixed points, but only one of the fixed points is stable. The two scenarios are illustrated using parameter values \( R_0 = 5 \) and \( \gamma = 0.01 \).
Thus, we see that the equilibrium mean worm burden is dependent on the strength of density-dependence, $\gamma$, the degree of heterogeneity, $v_\phi$, and the basic reproductive number, $R_0$. This result is analogous to that of the deterministic models where mean worm burden is a function of $\gamma, R_0$ and the level of aggregation in the distribution of worm burden as measured by the over-dispersion parameter, $k$, of the negative binomial distribution (Anderson and May, 1985a; Churcher et al., 2005).

The equilibrium mean worm burden decreases as heterogeneity in exposure, $\sigma_\phi$, increases. A maximum mean worm burden of $m_X = (1/\gamma)(1 - R_0^{-1})$ is reached at $\sigma_\phi = 0$; mean worm burden approaches zero as $\sigma_\phi \to \infty$. Intuitively this can be explained as follows. With increasing heterogeneity there are more individuals with very high and very low rates of exposure. However, since no host can harbour more than $1/\gamma$ adult worms, those individuals with very high rates of exposure do not have correspondingly high worm burdens. On the other hand, hosts with rates of exposure close to zero will have very low worm burdens. Thus mean worm burden decreases as heterogeneity increases (Fig. 2).

As in other models (Pacala and Dobson, 1988; Isham, 1995), the variance:mean ratio, $\sigma_X^2/m_X$, can be shown to be an increasing function of the heterogeneity in host exposure. This suggests that a comparison between the distributions of helminths of different communities will reveal a negative relationship between variance:mean ratio and mean worm burden. Indeed, such a negative relationship has been observed for Onchocerca volvulus (Basáñez et al., 2002). However, this argument assumes that there is no relationship between $m_\phi$ and $\sigma_\phi$. If, for example, an increase in $m_\phi$ is accompanied by an increase in $\sigma_\phi$, then this will affect the nature of the relationship between $m_X$ and $\sigma_X^2/m_X$.

At equilibrium, $\tilde{\sigma}_X = (1/\gamma)(1 - R_0^{-1}) - m_X$. Thus the covariance (and therefore correlation) between worm burden and susceptibility is positive since the equilibrium mean worm burden cannot exceed $(1/\gamma)(1 - R_0^{-1})$. This is important in terms of anthelminthic control. At any given time, targeting individuals with the highest worm burdens for continued treatment will also ensure that those most susceptible and therefore responsible for greatest transmission in the future will be treated. The extent to which this is true depends on the magnitude of the correlation between susceptibility and worm burden which will vary for different parameter combinations; this will be the subject of further investigation.

3. Two competing species

The single species model can be extended to include a competitive interaction with a second species. In the following model, competition may be for limited resources (e.g. space, nutrients) or it may be indirectly-mediated through the host immune system. Competition occurs at the point of establishment within the host: the probability with which a larva of either species becomes established is dependent on the number of adult worms of the heterologous species already residing within the host (as well as the number of adult worms of its own species). Host $i$ now has $X_{1i}(t)$ adult worms of species 1 and $X_{2i}(t)$ worms of species 2 at time $t$. The numbers of species 1 and species 2 larvae are, respectively, $L_{1i}(t)$ and $L_{2i}(t)$. Transition rates for species 1 adult worm burdens in each host, and numbers of larvae as follows (transitions for species 2 are similar):

1. $L_{1i} \rightarrow L_{1i} + 1$ at rate $v_1 \sum_{j=1}^n X_{1j}$.
2. $L_{1i} \rightarrow L_{1i} - 1$ at rate $(\mu_{L1} + \sum_{j=1}^n \Phi_{1j})L_{1i}$.
3. $X_{1i} \rightarrow X_{1i} + 1$ at rate $L_i\Phi_i(1 - \gamma_{11}X_{1i} - \gamma_{21}X_{2i})^+.$
4. $X_{1i} \rightarrow X_{1i} - 1$ at rate $\mu_{X1}X_{1i}$.
5. $X_{1i} \rightarrow 0$ at rate $z$.

The parameter $\gamma_{jk}$, $j,k = 1,2$ represents the effect of species $j$ worms on the probability of establishment of species $k$. Other parameters are defined as in the single species case (Table 1). The notation $z^+$ (which implies $z^+ = z$ when $z > 0$ and 0 otherwise) is used as the quantity $(1 - \gamma_{11}X_{1i} - \gamma_{21}X_{2i})$ could become negative and so the transition rate for $X_{1i} \rightarrow X_{1i} + 1$ is set to zero whenever this occurs. As in the single species models, analysis is facilitated by replacing $L_{1i}$ with $m_{L_{1i}}$ in the third transition rate above, and when $\Phi_i$ is random, replacing $\sum_{j=1}^n \Phi_{ij}$ with $m_{\Phi_i}$ in the second transition rate. The pairs of random variables $(X_{1i}(t), X_{2i}(t))$ $i = 1, \ldots, n$ are then i.i.d. for all $t$ (provided they are i.i.d. at $t = 0$).

Taking larval exposure to each species to be the same for all hosts, so that $\Phi_{1i} = \Phi_1$ and $\Phi_{2i} = \Phi_2$ for $i = 1, \ldots, n$, and setting $dm_{L_{1i}}/dt = dm_{L_{2i}}/dt = 0$, the mean worm burdens can be approximated by quantities satisfying the pair of differential equations,

$$\frac{dm_{X1}}{dt} = m_{X1} \phi'_1(1 - \gamma_{11}m_{X1} - \gamma_{21}m_{X2}) - (\mu_{X1} + z)m_{X1},$$
$$\frac{dm_{X2}}{dt} = m_{X2} \phi'_2(1 - \gamma_{22}m_{X2} - \gamma_{12}m_{X1}) - (\mu_{X2} + z)m_{X2},$$

(11)
where \( e_i' = e_i \phi_i n_i (\mu_L + n_i \phi_i) \) \( i = 1, 2 \) is the product of the rate at which potentially infective larvae are produced by an adult worm and the probability a larva survives to infect a host.

The solution to these differential equations only approximates the mean worm burdens for the two species because \((1 - \gamma_1 X_1 - \gamma_2 X_2)\) (and the equivalent species 2 expression) is potentially negative. More specifically, the approximation given in Eq. (11) is good if the following two quantities are small in magnitude:

\[
\sum (1 - \gamma_j x_j - \gamma_k x_k) p_j(x_1, x_2) \quad (j, k = 1, 2; j \neq k),
\]

where \( p_j(x_1, x_2) = P(X_{1j} = x_1, X_{2j} = x_2) \) and the summations are over the sets \([x_1, x_2] : \gamma_j x_j + \gamma_k x_k > 1\).

The solution to the differential equations is compared with mean worm burden obtained by simulating the model. Parameters were chosen, for the comparison, to be consistent with estimates for helminth species (e.g. see Tables 15.2–15.4 Anderson and May, 1991), where such estimates are available. In Fig. 5a,b it can be seen that the approximation works very well for the parameters used.

The two ODE’s in Eq. (11) are identical in form (though the interpretation of the model parameters is different) to the Lotka–Volterra model used to explore the effects of competition on the coexistence of free-living species (e.g. Maynard-Smith, 1974). The behaviour of the system is determined by the basic reproductive numbers for the two species and the inter- and intra-specific interaction parameters. For \( m_{Xk}(t) \) \( k = 1, 2 \) to increase, it is necessary that \( R_{0k} > 1 \)

\[
R_{0k} = \frac{e_k'}{\mu X_k + z}
\]

where \( R_{0k} = e_k'/(\mu X_k + z) \) is the reproductive number for species \( k \).

When both reproductive numbers exceed unity, and worms of both species are present initially, it is straightforward to show, by phase-plane analysis, that the system approaches either a single species equilibrium where one species has excluded the other, or a mixed equilibrium where both species coexist (see below and also Maynard-Smith, 1974).

### 3.1. Competitive exclusion

It is useful for conceptual purposes to distinguish two different types of interspecific competition, namely exploitative competition and interference competition (Schoener, 1983). Exploitative competition occurs when both species utilize the same limiting resource. Once a unit of resource has been consumed by one species, it is no longer available for consumption by the other species. In cestode species, for example, it appears that the carbohydrate intake of the host is frequently a limiting resource (Roberts, 2000).

The alternative, interference competition, encompasses a variety of mechanisms, that are not resource-mediated, where one species has a negative impact on the other, e.g. territoriality, excretion of toxins, overgrowth in plants. Among helminth species, interference competition may occur between species as a consequence of parasite-induced inflammation within the gut (Behnke et al., 2001).

As defined, the model does not distinguish between the two classes of interaction. However, whilst there are no restrictions on the intra- and interspecific terms for interference competition, when competition is exploitative and the probability of establishment is determined by the availability of a single resource, then \( \gamma_{11} = \gamma_{12} \) and \( \gamma_{22} = \gamma_{21} \). To see this, recall that the term \((1 - \gamma_{11} X_1 - \gamma_{22} X_2)^+\), (and the equivalent term for species 2) represents the probability of establishment in a host where \( X_1 \) worms of species 1 and \( X_2 \) worms of species 2 are present. If the probability of establishment in both species is determined by the availability of a single resource, then the probability of establishment, for both species, is reduced by the same amount for each worm of species 1 present, and therefore we must have \( \gamma_{11} = \gamma_{12} \). Similarly each worm of species 2 has the same effect on the probability of establishment of species 1 and species 2, and therefore \( \gamma_{22} = \gamma_{21} \).

Under this restriction, phase plane analysis reveals that the two non-zero isoclines are parallel lines; the mixed equilibrium can therefore not exist. The species with the higher \( R_0 \) ‘wins’, excluding the species with the lower \( R_0 \) (Fig. 3a). This result is equivalent to that obtained for free-living species by Volterra (Armstrong and McGehee, 1980). As with the result for free-living species, it implies the competitive exclusion principle which says that two species cannot occupy the same ecological niche (Hardin, 1960).

Coexistence becomes possible if each adult worm affects the establishment of its own species to a greater extent than that of the other species (\( \gamma_{11} > \gamma_{12} \) and \( \gamma_{22} > \gamma_{21} \)). This situation might arise if, for example, there is site segregation between the species within the host. In Fig. 3b it is shown by phase plane analysis that the species will coexist if \( \gamma_{11}(1 - R_{02}^{-1}) > \gamma_{12}(1 - R_{01}^{-1}) \) and \( \gamma_{22}(1 - R_{01}^{-1}) > \gamma_{21}(1 - R_{02}^{-1}) \). Clearly, for \( R_{01} \gg 1 \) and \( R_{02} \gg 1 \), coexistence is guaranteed when interspecific effects are weaker than intraspecific effects. However, a species may still out-compete another species if the \( R_0 \) of the competitively inferior species is sufficiently close to unity. For a collection of helminth species with low \( R_0 \) values, and correspondingly low densities, this suggests that competitive advantage will vary as \( R_0 \) values of species change between localities.

### 3.2. Heterogeneous exposure

Heterogeneity in exposure can be modelled by reverting to the case of a correlated pair of random variables, \((\Phi_1, \Phi_2)\), as in the original description of the model in the previous section. The same simplifying assumptions are made as in the model with homogeneous exposure. In addition, it is assumed that the expectation of products of three random variables can be expressed in terms of lower order moments as would be the case if they followed a
multivariate normal distribution, i.e.

This moment closure assumption is similar to that used for the single species model with heterogeneous exposure.

Setting \( dm_{x_1}/dt = 0 \), the following set of differential equations can be derived (the notation \( E(Z) = m_Z \), \( \text{var}(Z) = \sigma^2_Z \), \( \text{cov}(Y,Z) = \sigmaYZ \) is used as for the single species model)

\[
\begin{align*}
dm_{x_1} &= \gamma' m_{x_1} \left( 1 - \gamma_{11} \left( \frac{\sigma_{\phi_1} x_1}{m_{\phi_1}} + m_{x_1} \right) \right) - \gamma_1 \left( \frac{\sigma_{\phi_1} x_2}{m_{\phi_1}} + m_{x_1} \right) - \mu'_{x_1} m_{x_1}, \\
\end{align*}
\]

\[
\begin{align*}
d\sigma_{\phi_1} &= \gamma' m_{x_1} \sigma_{\phi_1} \left( m_{\phi_1} - \gamma_{11} \left( \frac{\sigma_{\phi_1} x_1}{m_{\phi_1}} + m_{x_1} \right) \right) - \gamma_1 \left( \frac{\sigma_{\phi_1} x_2}{m_{\phi_1}} + m_{x_1} \right) - \mu'_{x_1} \sigma_{\phi_1} x_1, \\
\end{align*}
\]

\[
\begin{align*}
d\sigma_{x_2} &= \gamma' m_{x_2} \sigma_{\phi_1} \left( m_{\phi_1} - \gamma_{22} \left( \frac{\sigma_{\phi_1} x_1}{m_{\phi_1}} + m_{x_2} \right) \right) - \gamma_1 \left( \frac{\sigma_{\phi_1} x_2}{m_{\phi_1}} + m_{x_2} \right) - \mu'_{x_2} \sigma_{\phi_1} x_2, \\
\end{align*}
\]

where \( \mu'_{x_k} = \mu_{x_k} + \alpha \) and \( \gamma' = (m_{\phi_1} n/(\mu_{Lk} + m_{\phi_1} n)) \gamma_k \) (\( k = 1, 2 \)).

Similarly, differential equations may be derived for \( m_{x_2}, \sigma_{\phi_2} x_2 \) and \( \sigma_{x_2} \), to give a closed set of differential equations.

**Coexistence**: The criterion that will be adopted for the coexistence of two species is that of mutual invadability: two species coexist when each species can invade an equilibrium where only the other species is present (Hutson and Schmitt, 1992). Introduction of a small quantity of worms of the invading species will perturb the means and covariances by a small amount. To determine whether invasion is likely to be successful we therefore perform stability analyses for the equilibria where only species 1 or only species 2 is present; we denote these equilibria by \( e_1 \) and \( e_2 \), respectively. Our criterion for coexistence is that both \( e_1 \) and \( e_2 \) must be unstable, i.e. each species is susceptible to invasion by the other species. The full stability analysis is given in Appendix B.

The criteria for coexistence, formally derived in Appendix B via stability analysis can also be derived intuitively as follows. Consider perturbing \( e_2 \) by introducing a small number of worms of species 1. Species 1 will invade if, after a small time increment, there is an increase in \( m_{x_1} \), i.e. if \( dm_{x_1}/dt > 0 \). Since the number of worms of species 1 introduced is small, the quantities \( m_{x_1} \) and \( \sigma_{x_1} x_1 \) are small relative to \( m_{x_2} \). From Eq. (12) the rate, \( r_1 \), of increase in \( m_{x_1} \) is therefore given by

\[ r_1 = \gamma' \left( 1 - \gamma_{21} \left( \frac{\sigma_{\phi_1} x_2}{m_{\phi_1}} + m_{x_2} \right) \right) - \mu'_{x_1} \gamma_2. \]

We have used the notation \( |e_2| \) here to emphasize that the time-dependent variables \( m_{x_1} \) and \( \sigma_{x_1} x_1 \) are being evaluated at the equilibrium \( e_2 \).

The rate \( r_1 \) determines the stability of \( e_2 \): the equilibrium is stable when \( r_1 < 0 \) and unstable when \( r_1 > 0 \). An equivalent rate, \( r_2 \), determines the stability of \( e_1 \) therefore, based on these two rates, the condition for coexistence can be written as

\[ R_1 > 1 \quad \text{and} \quad R_2 > 1, \]

where

\[
\begin{align*}
R_1 &= R_{01} \left( 1 - \gamma_{21} \left( \frac{\sigma_{\phi_1} x_2}{m_{\phi_1}} + m_{x_2} \right) \right) |e_2|, \\
R_2 &= R_{02} \left( 1 - \gamma_{12} \left( \frac{\sigma_{\phi_1} x_1}{m_{\phi_1}} + m_{x_2} \right) \right) |e_1|, \\
\end{align*}
\]

and \( R_{0k} = (\gamma'_{k}/\mu'_{x_k}) \) (\( k = 1, 2 \)).

\( R_1 \) and \( R_2 \) are, respectively, the effective reproductive numbers of species 1 at \( e_2 \) and species 2 at \( e_1 \). That is to say,
\( R_1(R_2) \) is the number of adult worms produced by an invading parasite of species 1(2) during its lifetime, at the equilibrium where only species 2(1) is present. Each effective reproductive number is the product of the basic reproductive number and a term representing the probability of establishment. It can be seen that this probability depends not only on the average worm burden of the resident species, but also on the covariance between the worm burden of the resident species and the host’s susceptibility to the invading species. This is intuitively reasonable since the probability of establishment for the invading species will be small if hosts that are more susceptible than average to the invading species have large worm burdens of the resident species, conversely the probability will be large if these hosts have few worms of the resident species.

At \( \epsilon_2 \), \( \sigma_{\phi_1}x_{22} \) can be written in terms of \( m_{x_{22}} \) since

\[
\frac{\sigma_{\phi_1}x_{22}}{\sigma^2_{\phi_2}} = \frac{\rho \sigma_{\phi_1}(\tilde{m}_{x_2} - m_{x_{22}})}{\sigma_{\phi_2}},
\]

where \( \tilde{m}_{x_2} = (1/\gamma_{22})(1 - R_{02}^{-1}) \) is the maximum possible value of the equilibrium mean worm burden for species 2, attained when \( v_{\phi_2} = 0 \), and \( \rho = \sigma_{\phi_1} / \sigma_{\phi_2} \) is the correlation between susceptibility to species 1 and susceptibility to species 2. Similarly, \( \sigma_{\phi_1}x_{11} = (\rho \sigma_{\phi_1} / \sigma_{\phi_2})(\tilde{m}_{x_1} - m_{x_{11}}) \), where \( \tilde{m}_{x_1} = (1/\gamma_{11})(1 - R_{01}^{-1}) \). Thus the effective reproductive numbers can be written as

\[
R_1 = R_{01} \left( 1 - \gamma_{21} \left( \frac{\epsilon_{\phi_1}}{v_{\phi_2}} (\tilde{m}_{x_2} - m_{x_{22}}) + m_{x_{22}} \right) \right), \tag{17}
\]

\[
R_2 = R_{02} \left( 1 - \gamma_{12} \left( \frac{\epsilon_{\phi_2}}{v_{\phi_1}} (\tilde{m}_{x_1} - m_{x_{11}}) + m_{x_{11}} \right) \right). \tag{18}
\]

At \( \epsilon_2 \), the equilibrium where species 2 alone is present, the species 2 mean worm burden, \( m_{x_{22}} \), is independent of the correlation, \( \rho \), between host susceptibility to species 1 and host susceptibility to species 2. From Eq. (17), the effective reproductive number for species 1 at \( \epsilon_2 \) is therefore a decreasing function of \( \rho \). Similarly, \( R_2 \) is also a decreasing function of \( \rho \). Thus, as one might expect intuitively, coexistence is promoted by decreasing the correlation between exposure rates.

When heterogeneity, as measured by the coefficient of variation for host exposure, is the same for both species, i.e. \( \epsilon_{\phi_1} = \epsilon_{\phi_2} = \epsilon_{\phi} \), then the effective reproductive numbers \( R_1 \) and \( R_2 \) are increasing functions of \( \epsilon_{\phi} \) for \( \rho < 1 \). This result follows from the results of the single species model which imply that at \( \epsilon_1 \), \( m_{x_1} \) is a decreasing function of \( \epsilon_{\phi} \), as is \( m_{x_2} \) at \( \epsilon_2 \). Consequently, coexistence is facilitated by increasing the heterogeneity in host exposure to both parasite species, \( \epsilon_{\phi} \), provided that \( \rho \neq 1 \) (Fig. 4).

Heterogeneity in host exposure is most likely to facilitate coexistence when hosts have similar degrees of heterogeneity in their exposure to both species. When \( \rho > 0 \), it can be seen from Eqs. (17) and (18), that it is hard for a species for which host heterogeneity in exposure is relatively high to invade a population where host heterogeneity to the already established parasite species is relatively low. For example, if species 1 is invading a host population where species 2 is already established, and \( \epsilon_{\phi_1} \gg \epsilon_{\phi_2} \), then the effective reproductive number for species 1 (Eq. (17)) will be small because \( \epsilon_{\phi_1} / \epsilon_{\phi_2} \gg 1 \). Coexistence therefore becomes increasingly hard the more the degree of heterogeneity in host exposure differs between the parasite species.

These results are illustrated in Fig. 5 through the results of model simulations as well as by numerical solution of the set of differential equations that approximate the moments (Eqs. (12)–(14)). For the simulations (Fig. 5c,d), 100 realizations were obtained from each of two independent gamma distributions representing rates of host exposure to the two species. Dependence was introduced (Fig. 5d) by splitting the 100 realizations for each species into quartiles and pairing values at random from each quartile of species 1 with the corresponding quartile of species 2. Results from the simulation were compared with the approximation using the means, variances and correlation of the two sets of realizations as the values for \( m_{\phi_1} \), \( \sigma_{\phi_1}^2 \) \( (k = 1, 2) \) and \( \rho \), respectively. The results of the simulation suggest that the approximation is reasonable for the parameters used, although it tends to underestimate mean worm burden.

4. Density-dependent fecundity

In the model presented it is assumed that competitive interactions affect parasite establishment. Alternatively, competition may impact rates of egg production. Parasite stunting and low rates of egg production in hosts with high worm burdens were originally observed in cestodes
(Read, 1951). This ‘crowding effect’ has also been observed in non-cestode species (e.g. Fleming, 1988; Stear and Bishop, 1999) and has been shown to be attributable to inter- as well as intraspecific competition (Holmes, 1961, 1962).

Density dependent fecundity can be modelled using the framework previously described by making the rates of increase for the species 1 and species 2 larval populations functions of the within-host worm burdens. Thus, for species 1, larval $L_1 \rightarrow L_1 + 1$ transitions occur at rate $\gamma_1 \sum_{i=1}^{m_1} X_{1i}(1 - \gamma_1(X_{1i} - 1) - \gamma_2 X_{2j})^+$. The rate of $L_2 \rightarrow L_2 + 1$ transitions is defined similarly. Observe that $\gamma_i$ now represents the effect that a species $j$ worm has on the per capita rate of egg production of adult worms of species $i$. For simplicity, density-dependent establishment is neglected so that the rate of $X_{1i} \rightarrow X_{1i} + 1$ transitions is $L_1 \Phi_{1i}$ (and similarly for species 2). Otherwise transition rates are as in the model for density-dependent establishment.

To determine conditions for coexistence, it is again helpful to derive effective reproductive numbers at the single species equilibria $e_1'$ and $e_2'$. This will be done intuitively here, while a formal analysis of the dynamics is given in Appendix C. Consider the introduction of a single species 1 worm into a host population where worm burden is at the equilibrium $e_2'$, i.e. only species 2 is present. If exposure to the two species is the same for all hosts, then the invading species 1 worm has a probability $1/n$ of being present in any given host. Given the host has a species 2 worm burden $x_2$, then the rate of larval production by the species 1 adult worm is $e_1(1 - \gamma_2 x_2)$. The expected larval production rate of the species 1 worm is therefore $(1/n) \sum_{i=1}^{m_1} e_1(1 - \gamma_2 x_2) = e_1(1 - \gamma_2 m_{X_21} e_2')$. A proportion $\phi_{1n}/(\mu_{X_1} + \phi_{1n})$ of larvae survive to infect hosts, and the life-expectancy of adult worms is $1/(\mu_{X_1} + \mu_{X_1} +\mu_{X_1})$. Thus the effective reproductive number for species 1 at $e_2'$ is

$$R_1 = \frac{e_1(1 - \gamma_2 m_{X_21} e_2')}{\mu_{X_1} + \mu_{X_1} + \mu_{X_1} + \mu_{X_1} + \mu_{X_1}},$$

where, as before, $e_1' = e_1(\phi_{1n}/(\mu_{X_1} + \phi_{1n}))$.

An effective reproductive number for species 2 at $e_2'$ can be derived similarly. The effective reproductive numbers $R_1$ and $R_2$, as derived here, are equivalent to those for the
model with density dependent establishment when all hosts have the same rate of exposure. Thus the conditions for coexistence ($R_1 > 1$ and $R_2 > 1$) are equivalent for the two models, when host exposure is homogeneous. In fact, when the rate of host death is sufficiently small and exposure is constant, the dynamics of the differential equations that approximate the moments of the model are equivalent to those that approximate the model for density-dependent establishment (Appendix C). The approximation to the moments is reasonable, although less good than it is for the model of density-dependent establishment (Fig. 6a,b).

An intuitive derivation of the effective reproductive rates cannot be undertaken when host exposures are random because the probability that the invading species 1 worm is in host $i$ will depend on the species 2 worm burden of host $i$. However, from simulation (Fig. 6), it appears that coexistence is promoted when the degree of heterogeneity in host exposure is similar for the two species and the correlation between rates of exposure is imperfect. Thus the conclusions are similar to those of the model of density-dependent establishment.

5. Discussion

Helminth communities are hierarchical in nature. Within a single host, members of different helminth species may co-occur forming an infra-community. At the next level up, helminths within a single host population form the component community. Finally, the compound community is the sum of all component communities in an ecosystem (Holmes and Price, 1986). Competition between helminth species will shape the structure of each of these communities, be it in terms of a functional or numerical response (Poulin, 1998) at the level of the individual host, the distribution of worm burdens in a component community, or the distribution of helminth species in a compound community. By exploring how competition affects the coexistence of species in host populations we focus on the structuring of the component and compound communities by interspecific competition.

The models we have developed here for homogeneous exposure of hosts to parasites suggest that competitive exclusion will occur if the probability of establishment or fecundity for both species is determined by a single, limiting resource. This is because under such circumstances, the effect that each species has on the establishment (or fecundity) of members of its own species is the same as the effect it has on the establishment of the heterologous species. In the situation where interspecific effects between two species are weaker than intraspecific effects, the species with the higher basic reproductive value ($R_0$) may still drive its competitor to extinction if the difference between the reproductive values of the two species is sufficiently great. In particular, competitive exclusion is most likely to occur in communities where
reproductive values are close to unity, and parasite densities are correspondingly low. This result is of significance because component communities are often classified according to the interactive-isolationist continuum based on parasite population densities (Poulin and Luque, 2003). At the interactive end of the continuum are component communities where parasite population densities are high. Because the potential for interaction is high in such communities, it is argued that competition maybe a significant structuring force. Conversely, competition is thought to play a minimal role in isolationist component communities where parasite densities are low. The concept was originally proposed by Holmes and Price (1986) at the level of the infra-community. At this level it is intuitively reasonable that competitive interactions will only become important in determining the spatial distribution of parasite species within the host, e.g. in the intestine, if parasite densities are sufficiently high. However, as we have shown, competitive exclusion may be more likely in component communities where parasites occur at low density. One can imagine that the component communities of different localities may vary in the composition of their parasite species as a result of small shifts in competitive advantage. Thus it may be that the variability in the combinations of species that parasitize eels in different localities (Kennedy, 2001) is, in part, the result of shifting competitive advantage, since parasite densities are small and competitive interspecific effects have been demonstrated (Bates and Kennedy, 1990, 1991).

A potentially significant factor in determining when species will coexist is the distribution of worm burdens amongst hosts, or for non-parasitic species, the distribution of individuals amongst patches. It has been argued that independent aggregation of species promotes their coexistence (Shorrocks, 1979; Atkinson and Shorrocks, 1981). The hypothesis considers a species utilizing a temporary resource that is patchily distributed, such as Drosophila species laying eggs on fruit. Atkinson and Shorrocks (1981) simulate a model where each species lays eggs in the patches independently of the other according to a negative binomial distribution. Competition is then assumed to take place between the fly larval stages. In this model, coexistence is promoted by increasing aggregation in the distribution of eggs amongst patches.

Similar conclusions have been drawn from models of competition between helminths species, where the mechanism of competition is parasite-induced host mortality (Dobson, 1985; Roberts and Dobson, 1995; Gatto and De Leo, 1998). Again helminths occur in patches (hosts) and typically follow an overdispersed distribution among hosts. In these models, it is assumed that the relationship between the mean and variance of worm burden is the same as the relationship between mean variance in the negative binomial distribution with a constant degree of overdispersion. The covariance between worm burdens of different species is assumed to be zero (Gatto and De Leo, 1998) or a function of mean worm burden (Dobson, 1985; Roberts and Dobson, 1995).

However, in the case of competition between helminth species, such results are harder to interpret since the level of aggregation is itself dependent on the degree of parasite-induced host mortality (Anderson and Gordon, 1982; Herbert and Isham, 2000). Similarly, it is likely that the dependence (and therefore covariance) between worm burdens of the two species is determined by the severity of parasite-induced host mortality.

Pugliese (2000) has incorporated causes of aggregation, namely host heterogeneity in exposure and clumping of infective stages, into a multispecies model where competition is through parasite-induced host mortality. He finds that including these causes does not in itself promote coexistence. However, for heterogeneity in host exposure, Pugliese considers only the case where each host is equally susceptible to both species. We have relaxed this assumption so that the correlation between host exposures to the two species, $\rho$, is not necessarily +1. By doing so we have found that coexistence can be promoted by heterogeneity in host exposure when $\rho < 1$ and the extent of the heterogeneity is similar for both species. However, heterogeneity in host exposure may reduce the likelihood of coexistence if $\rho > 0$ and there is a substantial difference in the level of heterogeneity for the two species. These results hold when competition occurs through either density-dependent establishment or fecundity. We have not examined the case where competition acts through parasite-induced host mortality, but it maybe possible to extend these results to cover this case.

For clarity, we give two examples of scenarios that will promote the coexistence of competing species. First, consider the case where heterogeneity among hosts is a result of differences in immune status between hosts. This may be due to host genetics as well as environmental determinants such as nutrition and exposure to other parasite species. The many determinants of immune status are likely to affect susceptibility to the two species differently, thus while host susceptibility to one parasite species may be positively correlated with susceptibility to a different species, the correlation is unlikely to be close to unity. For the second example, consider two species, each of which preferentially parasitizes a different host type, e.g. young vs old hosts. In this case, hosts will differ in their exposure to each species depending on the variable used by the parasite to identify preference, e.g. host age. Furthermore, in this example exposures to the two species will be negatively correlated: those hosts that have greater exposure to one species are less likely to be exposed to the other species. This negative correlation will greatly enhance the likelihood of coexistence.

These ideas can be extended to competing strains within a single species. The second scenario, favouring coexistence, seems particularly relevant. One can imagine
different strains becoming adapted to different host types. Evidence for the existence of genotype-dependent life-history traits is limited. Nonetheless the hypothesis has been tested in *Strongyloides ratti* (Paterson and Viney, 2003) using genetically homogenous lines. Differences were observed in the number of worms present in rats 23 days post-infection for different genetic lines, reflecting differences in establishment and/or survivorship. Clearly these differences require explanation in terms of other differences that might exist between the lines. Otherwise one would predict that in a natural setting the genotype with the greatest rate of establishment (and therefore greatest predict that in a natural setting the genotype with the greatest rate of establishment (and therefore greatest 

(33x596) should out-compete the other genotypes. Paterson and Viney (2003) explained this finding in terms of a trade-off between survivorship and/or survivorship. An alternative explanation is that the different genotypes parasitize different host types so that the heterogeneity allows coexistence even if the $R_0$ values differ.

Understanding how competing species are able to coexist has been a long-standing question in ecology. Here we have shown that the coexistence of helminth species is promoted by heterogeneity under a range of circumstances. In particular, heterogeneity promotes coexistence when the correlation between exposure rates is imperfect and the degree of heterogeneity in host exposure to the competing species is not too dissimilar.

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**Appendix A. Single species with heterogeneous exposure**

Let host $i$ ($i = 1, \ldots, n$) have a worm burden $X_i$ at time $t$, and a rate $\Phi_i$ of contact with infective larvae. Let $L_i$ be the size of the infective larval population at time $t$. Then given $(L_i, X_i, \Phi_i)$, after a small interval of time, $\delta$, the expected value of $L_{i+\delta}$ is

$$E[L_{i+\delta}|L_i, X_i, \Phi_i] = e^{X_i \delta(L_i + 1)} + \left( \mu_L + \sum_i \Phi_i \right) L_i \delta(L_i - 1) + \left( 1 - e^{X_i \delta} - \left( \mu_L + \sum_i \Phi_i \right) L_i \delta \right) L_i,$$

(A.1)

to first order in $\delta$.

We begin with the case where $\Phi_i = \phi$ for all $i$, i.e. the rate of exposure, $\phi$, is the same for all hosts. Taking the expectation with respect to $(L_i, X_i)$ (we have dropped the $i$ since $X_{it}$ are i.i.d. random variables), subtracting $E[L_i]$ from both sides, dividing through by $\delta$ and taking the limit as $\delta \to 0$ gives

$$\frac{dm_L}{dt} = nm_X - (\mu_L + \phi n)m_L.$$  

(A.2)

Substituting $m_L$ for $L$ in the rate for the $X \to X + 1$ transition, and applying the above procedure to $X_i$ gives the following expression:

$$\frac{dm_X}{dt} = m_L \phi (1 - \gamma m_X) - (\mu_X + \alpha)m_X.$$  

(A.3)

Strictly speaking, $1/\gamma$ must be an integer for Eq. (A.3) to hold. This is because $(1 - \gamma m_X)$ might otherwise be negative. However, Eq. (A.3) appears to work well as an approximation, even when $1/\gamma$ is not integer-valued.

When $\Phi$ is random, there is dependence between $\Phi$ and $L$, and between $\Phi$ and $X$. To deal with the dependence between $\Phi$ and $L$ we approximate $\sum_i \Phi_i$ by $m_\phi n$. The equation for $m_L$ then becomes

$$\frac{dm_L}{dt} = nm_X - (\mu_L + m_\phi n)m_L.$$  

(A.4)

With random $\Phi$, the equation for $m_X$ is

$$\frac{dm_X}{dt} = m_L (m_\phi - \gamma E[\Phi X]) - (\mu_X + \alpha)m_X.$$  

(A.5)

Thus $E[\Phi X]$ must be determined for random $\Phi$. The differential equation for $E[\Phi^2 X]$ is a function of $E[\Phi^2]$:

$$\frac{dE[\Phi^2 X]}{dt} = m_L (E[\Phi^2] - \gamma E[\Phi^2 X]) - (\mu_X + \alpha)E[\Phi X].$$  

(A.6)

In general, to determine $E[\Phi^p X]$, $E[\Phi^{p+1} X]$ must be known leading to an infinite set of differential equations. However, the system of differential equations can be closed by expressing $E[\Phi^2 X]$ in terms of lower moments. One way to do this is based on the bivariate normal distribution, in which case,


In terms of the covariance, $\sigma_{\Phi X}$, Eqs. (A.5) and (A.6) become

$$\frac{dm_X}{dt} = m_L (m_\phi - \gamma (\sigma_{\Phi X} + m_\phi m_X)) - (\mu_X + \alpha)m_X,$$

(A.7)

$$\frac{d\sigma_{\Phi X}}{dt} = m_L (\sigma_{\Phi}^2 - \gamma (\sigma_{\Phi} m_\phi + \sigma_{\Phi}^2 m_X)) - (\mu_X + \alpha)\sigma_{\Phi X}.$$  

(A.8)

An expression for the variance in worm burden can be derived along the same lines. The second moment of worm burden satisfies the following differential equation

$$\frac{dE[X^2]}{dt} = m_L \left\{ 2E[\Phi X] - 2\gamma E[\Phi X^2] + m_\phi - \gamma E[\Phi X] \right\} + \mu_X m_X - (2\mu_X + \alpha)E[X^2].$$

Using the bivariate normal moment closure approximation

and writing in terms of variances and covariances gives

$$\frac{d\sigma_X^2}{dt} = m_L(2\sigma_X + m_\phi - \gamma(2\sigma_X + 2\sigma_X^2 + m_\phi + \sigma_X^2 \mu_X + 2\sigma_X^2 \mu_X + \sigma_X^2)) + \mu_X m_\phi \mu_X^2 + 2m_X^2 - (2\mu_X + x)\sigma_X^2. \quad (A.9)$$

For the special case where $\phi$ is a constant, $\Phi$, so that $\sigma_X = 0$, Eq. (A.9) simplifies to

$$\frac{d\sigma_X^2}{dt} = m_Lm_\phi \mu_X(1 - \gamma(2\sigma_X^2 + m_X)) \mu_X m_X + 2m_X^2 - (2\mu_X + x)\sigma_X^2. \quad (A.10)$$

(Note that Eq. (A.10) can be derived directly from the model with constant $\Phi$ without making the moment closure assumption and is exact in that case).

**Appendix B. Stability analysis of single species equilibrium**

The Jacobian, $J$, corresponding to the system of differential equations for two competing species with random exposure (Eqs. (12)–(14) and the corresponding differential equations for species 2) is

$$\begin{pmatrix}
A_1 - \mu'_X & -\frac{\sigma_\phi X}{m_\phi} \gamma_1 X & -\frac{\sigma_\phi X}{m_\phi} \gamma_2 X \\
C_1 & -\frac{\sigma_\phi X}{m_\phi} \gamma_1 X & -\frac{\sigma_\phi X}{m_\phi} \gamma_2 X \\
-\frac{\sigma_\phi X}{m_\phi} \gamma_1 X & -\frac{\sigma_\phi X}{m_\phi} \gamma_2 X & -\frac{\sigma_\phi X}{m_\phi} \gamma_1 X \\
B_2 & 0 & 0
\end{pmatrix} =
\begin{pmatrix}
0 & 0 & 0 \\
-\frac{\sigma_\phi X}{m_\phi} \gamma_1 X & 0 & 0 \\
0 & 0 & 0 \\
0 & 0 & 0
\end{pmatrix}$$

where

$$A_1 = \frac{\gamma_1 X}{m_\phi} \left(1 - \gamma_1 \frac{(\sigma_\phi X)}{m_\phi} X + 2X\right),$$

$$B_1 = \frac{\gamma_2 X}{m_\phi} \left(\sigma_\phi X + 2\frac{(\sigma_\phi X)}{m_\phi} X\right),$$

$$C_1 = \frac{\sigma_\phi X}{m_\phi} \left(1 - \gamma_1 \frac{(\sigma_\phi X)}{m_\phi} X + 2\frac{(\sigma_\phi X)}{m_\phi} X\right),$$

$$\mu'_X = \mu_X + \alpha, \quad k = 1, 2,$$

and $A_2, B_2, C_2$ are the species 2 counterparts to $A_1, B_1, C_1$. At $e_2$ (the equilibrium where species 2 is present and species 1 is absent),

$$\text{Det}(J|e_2 - \lambda I) = \left((\mu'_X - \lambda)^3 - A_1 e_2 + \lambda(\sigma_\phi X X + \mu'_X + \lambda)\right) + (\mu'_X - A_2 e_2 + \lambda(\sigma_\phi X X + \mu'_X + \lambda)$$

$$+ C_2 e_2 \frac{\gamma X}{m_\phi} \gamma_1 X), \quad (B.1)$$

where

$$A_1 e_2 = \frac{\gamma_1 X}{m_\phi} \left(\sigma_\phi X + 2\frac{(\sigma_\phi X)}{m_\phi} X\right),$$

$$A_2 e_2 = \frac{\gamma_2 X}{m_\phi} \left(\sigma_\phi X + 2\frac{(\sigma_\phi X)}{m_\phi} X\right),$$

$$C_2 e_2 = \frac{\sigma_\phi X}{m_\phi} \left(\sigma_\phi X + 2\frac{(\sigma_\phi X)}{m_\phi} X\right),$$

and $m_X, \sigma_X X, \sigma_\phi X X$ take on the values that they have at the equilibrium, $e_2$.

The roots $\lambda = -\left(\sigma_\phi X X + \mu'_X\right)$ and $\lambda = -\mu'_X$ are both negative. The stability of the equilibrium, $e_2$, is therefore determined by a third order polynomial in $\lambda$. Using the Routh–Hurwitz criterion for a third order polynomial (May, 1973), $e_2$ is stable if the following conditions are met

$$\mu_X - A_1 e_2 > 0,$$  \hspace{1cm} (B.2)

$$\mu_X - A_2 e_2 + \sigma_\phi X X + \mu'_X > 0,$$  \hspace{1cm} (B.3)

$$\mu_X - A_2 e_2 + \sigma_\phi X X + \mu'_X + C_2 e_2 \frac{\gamma X}{m_\phi} \gamma_1 X > 0.$$  \hspace{1cm} (B.4)

At $e_2$, from the species 2 equivalent of Eq. (12), $\sigma_\phi X X$ can be written in terms of $m_X$ as

$$\frac{\sigma_\phi X}{m_\phi} = \frac{1}{\gamma_1 X}(1 - R^{-1}) - m_X.$$

Using this relationship, $\mu'_X - A_2 e_2 = \sigma_\phi X X > 0$ and so condition (B.3) is satisfied.
Rewriting (B.4),
\[ \mu'_{X_2}(\mu_2' - A_2|c_2) + \nu_2' m_{X_2} \frac{C_2|c_2}{m_{X_2}} > 0. \]

For condition (B.4) to hold, it is sufficient to show that \((C_2|c_2/\mu_2) + \mu_2' - A_2|c_2 > 0\). Rewriting this condition,
\[ R_{c_0}^{-1} + (\nu_2^2 - 1)(1 - 2\nu_2^2 m_{X_2}) > 0, \tag{B.5} \]
where \( \nu_2^2 = \sigma_{\phi_2}/m_{\phi_2}. \)

Recall from the single species model that \( m_{X_2} \) is a decreasing function of \( \nu_2 \) and that at \( \nu_2 = 1 \), \( m_{X_2} = (1/2\nu_2^2)(1 - \alpha_{c_0}) \). Condition (B.5) holds as a result of this (and hence so does (B.4)) because when \( \nu_2 > 1 \)
\[ m_{X_2} < \frac{1}{2\nu_2^2}(1 - \alpha_{c_0}) \Rightarrow (1 - 2\nu_2^2 m_{X_2}) > \alpha_{c_0}^{-1} \]
and when \( \nu_2^2 < 1 \)
\[ m_{X_2} > \frac{1}{2\nu_2^2}(1 - \alpha_{c_0}) \Rightarrow (1 - 2\nu_2^2 m_{X_2}) < \alpha_{c_0}^{-1}. \]

The stability of \( e_2 \) is therefore determined by (B.2) which is equivalent to the condition given in the text (Eq. (15)): \( e_2 \) is stable if \( R_1 < 1 \) and unstable if \( R_1 > 1 \).

Appendix C. Density dependent fecundity

The transition rates for species 1 in the model of density-dependent fecundity are as follows (species 2 rates are equivalently defined):
1. \( L_1 \rightarrow L_1 + 1 \) at rate \( \delta_i \sum_{i=1}^n X_{1i}(1 - \gamma_{1i})X_{1i} + \gamma_{2i}X_{2i}) \),
2. \( L_1 \rightarrow L_1 - 1 \) at rate \( \mu_{L_1} + \sum_{i=1}^n (\Phi_{1i})L_{1i} \),
3. \( X_{1i} \rightarrow X_{1i} + 1 \) at rate \( \delta_i \Phi_{1i} \),
4. \( X_{1i} \rightarrow X_{1i} - 1 \) at rate \( \mu_{X_{1i}} \),
5. \( X_{1i} \rightarrow 0 \) at rate \( \alpha \),

where \((\Phi_{1i}, \Phi_{2i})\) are i.i.d. pairs of random variables. As with the model for density-dependent establishment, to facilitate analysis \( L_1 \) is replaced by \( m_{L_1} \) in the third transition rate, and \( m_{X_{1i}}/n \) replaces \( \sum_{i=1}^n \Phi_{1i} \). A closed set of differential equations for the first and second moments may be derived by assuming that
\[ \sum_{(x_1, x_2)} |x_1(1 - \gamma_{1i}X_{1i} + \gamma_{2i}X_{2i})| < 1, \]
where \( p_i(x_1, x_2) = P(X_{1i} = x_1, X_{2i} = x_2) \) and the summation is over the set \( (x_1, x_2) : 1 - \gamma_{1i}X_{1i} + \gamma_{2i}X_{2i} < 0 \).

\[
\frac{dm_{L_1}}{dt} = n\delta_i((1 + \gamma_{11})m_{X_1} - \gamma_{11}(\sigma^2_{X_1} + m_{X_1}^2) - \gamma_{21}(\sigma^2_{X_1} + m_{X_1}m_{X_2}) - (\mu_{L_1} + m_{\Phi_i}n)m_{L_1},
\]
\[
\frac{dm_{X_1}}{dt} = m_{L_1}m_{\Phi_i} - (\mu_{X_1} + \alpha)m_{X_1},
\]
\[
\frac{d\sigma^2_{X_1}}{dt} = m_{L_1}m_{\phi_i} + \mu_{X_1}m_{X_1} + 2m_{L_1}\sigma_{\phi_i}X_1
\]
\[ - (2\mu_{X_1} + \alpha)\sigma^2_{X_1} + 2m_{X_1}^2, \]

The accuracy of the approximation is only good when host death, \( \alpha \), is small relative to other rates. Intuitively, this is because net egg output per host is maximized at intermediate levels of worm burden, and intermediate levels will be attained with higher probability when old, heavily parasitized hosts are frequently replaced by uninfected hosts, i.e. when the rate of host death is high. Subsequently, it will be assumed that \( \alpha = 0 \).

When exposure is constant \((\Phi_i = \phi_i \ i = 1,2)\) the covariance terms are zero. Furthermore if \( dm_{L_1}/dt = 0 \) then Eq. (C.1) becomes
\[
\frac{dm_{X_1}}{dt} = n\delta_i((1 + \gamma_{11})m_{X_1} - \gamma_{11}(\sigma^2_{X_1} + m_{X_1}^2) - \gamma_{21}m_{X_1}m_{X_2})
\]
\[ - \mu_{X_1}m_{X_1}, \]
\[ d\sigma^2_{X_1}/dt = n\delta_i((1 + \gamma_{11})m_{X_1} - \gamma_{11}(\sigma^2_{X_1} + m_{X_1}^2) - \gamma_{21}m_{X_1}m_{X_2})
\]
\[ + \mu_{X_1}m_{X_1} - 2\mu_{X_1}\sigma^2_{X_1}. \tag{C.2} \]

If \( m_{X_1}(t) = 0 \) then \( m_{X_1}(t) = \sigma^2_{X_1}(t) \) for all \( t \). In fact it is apparent from the transition rates, having replaced \( L_1 \) by \( m_{L_1} \) in the third rate, that the distribution of adult worms of species 1 is given by a non-homogeneous immigration-death process with immigration rate \( m_{L_1}\phi_i \) and death rate \( \mu_{X_1}X_1 \), and is therefore Poisson provided that the initial distribution is also Poisson (Näsell, 1985). Therefore
\[
\frac{dm_{X_1}}{dt} = m_{X_1}\delta_i((1 + \gamma_{11})m_{X_1} - \gamma_{21}m_{X_1}) - \mu_{X_1}m_{X_1}. \tag{C.3} \]

This is identical to the expression for \( d\sigma^2_{X_1}/dt \) in the establishment model (Eq. (11)) with \( \alpha = 0 \). The criteria for coexistence are therefore as for the establishment model. Alternatively this can be demonstrated by performing stability analyses for the single species equilibria \( \delta_i \) and \( \delta_2 \).

References


