

Modeling macroparasite infection dynamics

Modelización de la dinámica de infección de macroparásitos

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Abstract— In this work we present a general framework for the modeling of the transmission dynamics of macroparasites which do not reproduce within the host like *Ascaris lumbricoides*, *Trichuris trichiura*, *Necator americanus* y *Ancylostoma duodenale*. The basic models are derived from general probabilistic models for the parasite density-dependent mating probability. Here we considered the particular, and common case, of a negative binomial distribution for the number of parasites in hosts. We find the basic reproductive number and we show that the system exhibits a saddle-node bifurcation at some value of the basic reproduction number. We also found the equilibria and basic reproduction number of a model for the more general case of heterogeneous host populations.

Keywords—Basic reproductive number; Macroparasite; Mathematical modeling; Negative binomial distribution; Saddle-node bifurcation

Resumen— En este trabajo presentamos un marco general para la modelización de la dinámica de transmisión de macroparásitos que no se reproducen dentro del hospedador como *Ascaris lumbricoides*, *Trichuris trichiura*, *Necator americanus* y *Ancylostoma duodenale*. Los modelos básicos se derivan de modelos probabilísticos generales para la probabilidad de apareamiento denso-dependiente del parásito. Aquí consideramos el caso particular y común de una distribución binomial negativa para el número de parásitos en hospedadores. Encontramos el número reproductivo básico y mostramos que el sistema presenta una bifurcación nodo silla en algún valor del número reproductivo básico. También encontramos los equilibrios y el número básico de reproducción de un modelo para el caso más general de poblaciones heterogéneas de hospedadores.

Palabras clave—Bifurcación nodo silla; Distribución binomial negativa; Macroparásito; Modelo matemático; Número reproductivo básico

INTRODUCTION

Mathematical models play an important role in understanding the transmission and impact of macroparasite diseases control measures (Anderson and May, 1992; Anderson et al., 2014; Truscott et al., 2016).

The first works on the theory of helminth infection was published in the 1960's by Tallis and Leyton by developing stochastic models of nematode parasite transmission in sheep and cattle (Leyton, 1968; Tallis and Leyton, 1966, 1969).

Simultaneously Macdonald show that a consequence of sexual reproduction of distributed parasites within individual hosts was the inability to generate fertile infectious material when prevalence is low (Macdonald et al., 1965).

Anderson and May introduced a much more general descriptions of helminth population dynamics based on host age, distribution of parasite numbers per host, density dependen-

ce of egg production, and sexual mating functions that depend on parasite distribution and reproductive habits (Anderson and May, 1982, 1985, 1992).

In this article we develop an analytical framework to describe the transmission dynamics of most macroparasite infections. We show how the classical deterministic models are derived from probabilistic considerations about parasite distribution in hosts, egg production, and mating probability.

We first describe the dynamics of infection transmission by macroparasites. Then we present two deterministic models for the transmission dynamics. The first model the simpler case of a homogeneous host community while the second model the more complex case of a heterogeneous host community.

In both models, reproductive characteristics of the parasite are considered, such as egg production and mating probability, both modeled by the density-dependent fecundity of the

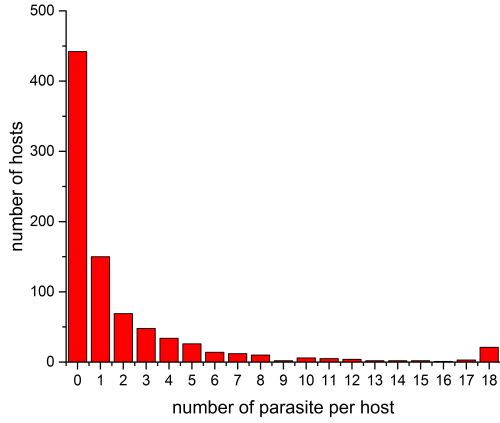


Figure 1: Distribution of *Ascaris lumbricoides* parasite numbers per host in a study in rural populations in Korea (Seo et al., 1979).

Most hosts are uninfected or infected with a low burden of parasites while few are infected by large numbers of parasites.

parasite and the distribution of parasites per host, which we assume to be negative binomial.

For both models we computed the endemic equilibrium and the basic reproduction number R_0 , defined as the average number of new parasite offspring produced by a typical female parasite, from one generation to the next. Finally we show that the homogeneous model undergoes a saddle-node bifurcation.

GENERAL FRAMEWORK

Microparasite diseases are usually modeled using compartmental models. After infection, microparasite population may rapidly grow into the host. This intra-host parasite dynamics determines the level of infectiousness of the individual. In a simple compartmental model like the *SIR*-model all the susceptible individuals are grouped in one class of size S , all the infected and infectious individuals in a class of size I and all the recovered individuals in a class of size R . Many refinements are possible, but the evolution of the parasite population within the host it is not considered or very simplified (for models including intra-host population dynamics see for example Gandolfi et al. (2015)). The most common refinement consists in dividing infected individuals in two classes, exposed (those infected but yet not infectious) and infectious, which leads to the well known *SEIR* type models.

For most macroparasites, the situation is completely different as these types of parasites do not reproduce within the host. Most infected individuals have few macroparasites with a non-bell shaped distribution (see Figure 1) where few individuals concentrate most of the parasites in the host population (Seo et al., 1979; Lopez and Aparicio, 2023). Negative binomial distributions usually provide a good description of the data. On the other hand, there is no host-to-host transmission of macroparasites as the life cycle completes in the environment (from where the host gets infected).

Therefore the number of infected hosts is not a representative variable of the parasite burden. Simple models for macroparasites consider the evolution of the mean burden of parasite within the population as well as the environmental

parasite reservoir (which is composed of eggs and/or larvae). From the mean burden, the total parasite population is easily estimated.

A BASIC MODEL

Model structure

The model presented in this paper is based on a model developed by Anderson and May (Anderson and May, 1992). The conceptual framework of parasite transmission dynamics is conceptualized as a population of mature parasites within human hosts and a population of infective stages (eggs and/or larvae) found in the environment (reservoir). Hosts may become infected by contact with the infective stages of the parasites and can contaminate the environment by releasing parasite's eggs to the environment (see Figure 2).

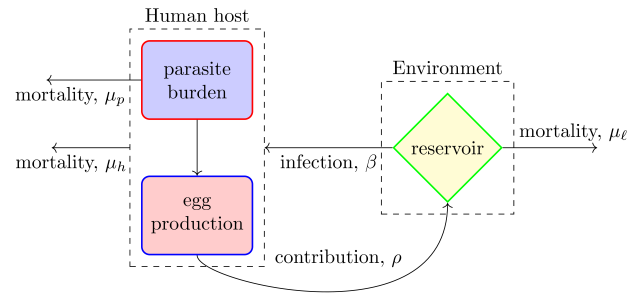


Figure 2: Conceptual framework of parasite transmission dynamics.

In a simple model for transmission dynamics of parasites, the dynamic variables are the mean parasite burden in the host population, m ; and the population of infective stages (reservoir) in the environment, formed by eggs and/or larvae, ℓ .

In the following we will sketch the procedure to find parasite-related parameters from a statistical-probabilistic model for the parasite population.

The environmental parasite reservoir, composed by eggs or larvae, increases due to the contribution of adult parasites within the hosts. As most hosts harbor only few parasites, only hosts with at least one female and one male parasites will contribute with fertilized eggs to the reservoir. We will consider that the random variable W , the number of parasites in a host, follows a negative binomial distribution. Therefore, the probability of observing n parasites in a host is

$$P(W = n) = \frac{\Gamma(k+n)}{\Gamma(n+1)\Gamma(k)} p^n (1-p)^k, \quad (1)$$

where Γ is the gamma function and $p = \frac{m}{m+k}$ with m the mean value (the mean population parasite burden) and k the shape parameter. The variance may be expressed in terms of m and k as $\sigma^2 = m + m^2/k$. The term p is the host's probability of acquiring a parasite and $1-p$ is the probability of not acquiring a parasite, in each of $k+n$ Bernoulli experiments.

Mean egg production depends on the number of parasites within the host, and it is a density-dependent process. A simple model for the mean female fecundity of a female parasite

in competition with $n - 1$ parasites is given by

$$\lambda(n) = \lambda_0 z^{n-1}, \quad (2)$$

where λ_0 is the rate of egg production per female independent of parasite density in host and $z = e^{-\gamma}$ with γ a parameter quantifying the intensity of the competition. A study of the *Ascaris lumbricoides* fecundity is presented in Hall and Holland (2000).

Using the parasite host distribution (1) we may compute the mean egg production per host as (Lopez and Aparicio, 2022) $\lambda_0 \alpha m \psi(m, k, z)$ where α is the fraction of female parasites in a host and ψ , the average effective contribution per female parasite to the parasite reservoir (see Churcher et al. (2006); Lopez and Aparicio (2022)), is given by

$$\psi(m, k, z) = \left[1 + (1 - z) \frac{m}{k} \right]^{-(k+1)}, \quad (3)$$

However, only hosts with at least one female parasite and one male parasite will effectively contribute to the parasite reservoir by the production of fertilized (or infective) eggs. Therefore, the mean fertilized egg production per host is

$$\lambda_0 \alpha m \psi(m, k, z) \phi(m, k, z), \quad (4)$$

where $\phi(m, k, z)$ is the mating probability for the negative binomial distribution Lopez and Aparicio (2022)

$$\phi(m, k, z) = 1 - \left[\frac{1 + (1 - \alpha z) \frac{m}{k}}{1 + (1 - z) \frac{m}{k}} \right]^{-(k+1)}. \quad (5)$$

Therefore, the mean fertilized egg contribution to the environmental reservoir per host and per unit of time is $\rho \lambda_0 \alpha m \psi(m, k, z) \phi(m, k, z)$ where ρ is the host's own contribution rate and the total contribution of eggs to the reservoir per unit of time of a host population of size N is $\rho \lambda_0 \alpha m \psi(m, k, z) \phi(m, k, z) N$.

The population of eggs and/or larvae in the environment (ℓ) also decreases due to egg/larval mortality (at the rate μ_ℓ) and due to host infection at the rate $\beta \ell$ per host, however, we consider this last term is negligible relative to the size of ℓ . Therefore, the dynamics of the reservoir is given by

$$\frac{d\ell}{dt} = \rho \lambda_0 \alpha m \psi(m, k, z) \phi(m, k, z) N - \mu_\ell \ell. \quad (6)$$

Finally, the dynamics for the mean parasite burden m is obtained as follows. Parasites are taken from the environment at a rate $\beta N \ell$ and therefore, the mean parasite burden increases at a rate $\beta N \ell / N = \beta \ell$. Parasites within the host die at a rate μ_p and hosts at a rate μ_h (killing all their parasites). Thus, the dynamics of m is given by

$$\frac{dm}{dt} = \beta \ell - (\mu_h + \mu_p) m. \quad (7)$$

Because an average host has contact with a small part of the reservoir ℓ (by infection and contribution), we rename the variable, relative reservoir to a host, ℓ/N to ℓ . Then, the dynamic of the new variable ℓ is given by

$$\frac{d\ell}{dt} = \rho \lambda_0 \alpha m \psi(m, k, z) \phi(m, k, z) - \mu_\ell \ell. \quad (8)$$

Therefore, the conceptual framework of parasite transmission dynamics is conceptualized as shown in Figure 2. A basic model of the transmission dynamics of macroparasite infection in a homogenous host population is given by the following the system of nonlinear ordinary differential equations

$$\begin{aligned} \frac{dm}{dt} &= \beta \ell - (\mu_h + \mu_p) m, \\ \frac{d\ell}{dt} &= \rho \lambda_0 \alpha m \psi(m, k, z) \phi(m, k, z) - \mu_\ell \ell. \end{aligned} \quad (9)$$

Equilibria and basic reproduction number

In this section we find some useful expressions involving the equilibrium values of the dynamical variables and the basic reproduction number R_0 defined as the average number of female offspring produced per female adult worm, that survive to reproductive maturity in the absence of density-dependent constraints on parasite population growth (Anderson and May, 1992). Assuming the mating probability (ϕ) and mean fertilized egg production (ψ) equal to one (this is an usual, but somewhat strong assumption, which will be discussed elsewhere), an average female parasite would release $\lambda_0 \rho$ per unit of time to the environment. As the mean life of a parasite is approximately $1/(\mu_h + \mu_p)$ the total contribution of fertilized eggs become $\frac{\lambda_0 \rho}{(\mu_h + \mu_p)}$. On the other hand, female parasites in hosts increase as the rate $\alpha \beta$ during an average time $1/\mu_\ell$. Therefore, the basic reproduction number is (Anderson and May, 1992)

$$R_0 = \frac{\lambda_0 \alpha \rho \beta}{\mu_\ell (\mu_h + \mu_p)}, \quad (10)$$

From the equation (8) we obtain that at equilibrium

$$\ell^* = \frac{\lambda_0 \alpha}{\mu_\ell} \rho m \psi(m) \phi(m), \quad (11)$$

and substituting (11) in equation (7) we obtain the following equation for the dynamics of the mean parasite burden m

$$\frac{dm}{dt} = (\mu_h + \mu_p) [R_0 \psi(m) \phi(m) - 1] m, \quad (12)$$

Therefore from the equation (12), the mean parasite burden (m^*) satisfy

$$\psi(m^*, k, z) \phi(m^*, k, z) = 1/R_0. \quad (13)$$

This equation presents two equilibrium solutions for the mean parasite burden.

As shown in the next section, the dynamic system (9) presents a saddle-node bifurcation. The bifurcation occurs at the point (m^b, R_0^b) where

$$\begin{aligned} m^b &= \frac{k \left(\frac{1-\alpha z}{1-z} \right)^{\frac{1}{k+2}} - k}{(z-1) \left(\frac{1-\alpha z}{1-z} \right)^{\frac{1}{k+2}} + (1-\alpha z)}, \\ R_0^b &= \left[\psi(m^b; k, z) \phi(m^b; k, z) \right]^{-1}. \end{aligned} \quad (14)$$

Therefore, for $R_0 > R_0^b$ there are three equilibria in the dynamic system (9) (see Figure 3),

- An equilibrium is the **disease-free equilibrium** present at $m^* = 0$, which is the trivial solution of equation (12). This equilibrium is an attractor for all values of R_0 .
- The other equilibrium is the **endemic equilibrium**, which is one solution of equation (13). This equilibrium is an attractor for a range of values of $R_0 > R_0^b$.
- The last equilibrium is an **unstable equilibrium** and corresponds to the other solution of equation (13). This equilibrium is a repulsor in the phase plane, that is, a barrier where values of $m(t)$ above the unstable equilibrium are attracted towards the endemic equilibrium and values of $m(t)$ below the unstable equilibrium are attracted to the disease-free equilibrium.

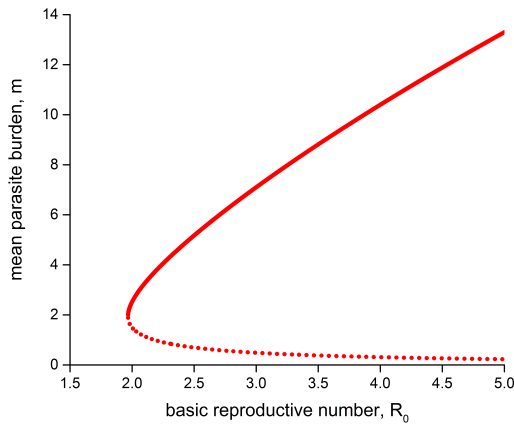


Figure 3: Saddle-node bifurcation generated by eq. (13), parameter values $\alpha = 0.57$, $k = 0.7$ and $z = 0.93$. The solid line and dotted line correspond to the stable and unstable branch, respectively.

Dynamics and bifurcation analysis

Dynamics of the reservoir is much faster than the parasite-host dynamics. This fact allows for a simplification by adiabatic elimination, that is we may assume that the dynamical variable ℓ is at a equilibrium at all times and therefore the two dimensional system (9) reduces to the one-dimensional system

$$\frac{dm}{dt} = (\mu_h + \mu_p) [R_0 \psi(m) \phi(m) - 1] m,$$

which we compactly denote by $\frac{dm}{dt} = f(m, R_0)$. This system undergoes a saddle-node bifurcation. A necessary condition for the existence of a such bifurcation at (m^b, R_0^b) is

$$\begin{aligned} f(m^b, R_0^b) &= 0, \\ \frac{\partial f}{\partial m}(m^b, R_0^b) &= 0, \end{aligned} \quad (15)$$

where the first of these conditions is the equilibrium condition (13) of the dynamic system

$$\psi(m^b; k, z) \phi(m^b; k, z) = 1/R_0^b,$$

and using the second condition of (15) we obtain the following equation for m^b

$$\frac{\partial}{\partial m} \psi(m^b; k, z) \phi(m^b; k, z) = 0, \quad (16)$$

The value of m^b corresponding to this last condition is

$$m^b = \frac{k \left(\frac{1-\alpha z}{1-z} \right)^{\frac{1}{k+2}} - k}{-(1-z) \left(\frac{1-\alpha z}{1-z} \right)^{\frac{1}{k+2}} + (1-\alpha z)}, \quad (17)$$

and its corresponding basic reproductive number is

$$R_0^b = \left[\psi(m^b; z, k) \phi(m^b; z, k) \right]^{-1}, \quad (18)$$

A sufficient condition for the existence of a saddle-node bifurcation at (m^b, R_0^b) is

$$\begin{aligned} \frac{\partial f}{\partial R_0}(m^b, R_0^b) &\neq 0 \\ \frac{\partial^2 f}{\partial m^2}(m^b, R_0^b) &\neq 0 \end{aligned} \quad (19)$$

By a Taylor series expansion of the function f in a neighborhood of (m^b, R_0^b) , the equation (12) is given by

$$\begin{aligned} \frac{dm}{dt} &= f(m^b, R_0^b) + (m - m^b) \frac{\partial f}{\partial m} \Big|_{(m^b, R_0^b)} \\ &\quad + (R_0 - R_0^b) \frac{\partial f}{\partial R_0} \Big|_{(m^b, R_0^b)} \\ &\quad + \frac{1}{2} (m - m^b)^2 \frac{\partial^2 f}{\partial m^2} \Big|_{(m^b, R_0^b)} + \dots \end{aligned} \quad (20)$$

Therefore locally at the point (m^b, R_0^b) the equation is of the form

$$\frac{dm}{dt} = A(R_0 - R_0^b) + B(m - m^b)^2, \quad (21)$$

where the values A and B are

$$A = (\mu_h + \mu_p) \frac{m^b}{R_0^b}, \quad B = (\mu_h + \mu_p) R_0^b m^b \frac{\partial^2 F}{\partial m^2}(m^b), \quad (22)$$

with $F(m) = \psi(m, z, k) \phi(m, z, k)$, which is the normal form of a saddle-node bifurcation.

As a result of the above, we can obtain the following result

Theorem 1 *The model (9) exhibits a saddle node bifurcation at the point (m^b, R_0^b) , if $R_0 \geq R_0^b$.*

Sensitivity analysis

The transmission of macroparasitic diseases is related to the value of R_0 . To predict which parameters have a higher impact on R_0 , we may perform a sensitivity analysis on R_0 .

The elasticity index or normalized sensitivity index measures the relative change of R_0 with respect to a parameter x , denoted by $\Gamma_x^{R_0}$, and defined as (see Van den Driessche (2017))

$$\Gamma_x^{R_0} = \frac{\partial R_0}{\partial x} \frac{x}{R_0}, \quad (23)$$

The sign of $\Gamma_x^{R_0}$ tells us whether R_0 correlates positively or negatively with the parameter x ; whereas its magnitude determines the relative importance of the parameter.

For this model, the calculation of the elasticity indices are given by

$$\begin{aligned}\Gamma_{\lambda_0}^{R_0} &= \Gamma_{\alpha}^{R_0} = \Gamma_{\rho}^{R_0} = \Gamma_{\beta}^{R_0} = 1, \\ \Gamma_{\mu_\ell}^{R_0} &= -1, \\ \Gamma_{\mu_h}^{R_0} &= -\frac{\mu_h}{\mu_h + \mu_p}, \\ \Gamma_{\mu_p}^{R_0} &= -\frac{\mu_p}{\mu_h + \mu_p},\end{aligned}\quad (24)$$

if $\frac{1}{\mu_h} \gg \frac{1}{\mu_p}$, then $\Gamma_{\mu_p}^{R_0} \approx -1$ and $\Gamma_{\mu_h}^{R_0} \approx 0$. In Figure 4 illustrated the sensitivity indices of R_0 which were obtained and evaluated using parameter values $\mu_h = \frac{1}{70}$ and $\mu_p = 1$.

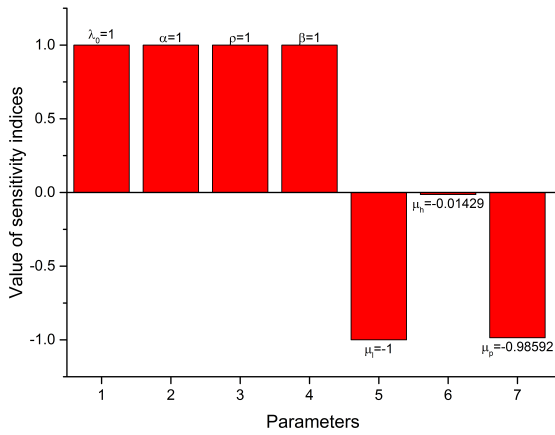


Figure 4: Sensitivity analysis for R_0 with respect to each model parameter.

Clearly the most sensitive parameters for R_0 are λ_0 , α , ρ , β , μ_ℓ and μ_p . However, λ_0 , α and μ_ℓ correspond to parameters related to the life-cycle of the parasite which are quite difficult to modify, so a control measure for macroparasitic diseases should target the reduction of ρ and β , and the increase of μ_p .

Therefore, we can conclude from this analysis that the reduction of R_0 is possible by reducing the egg contribution from the hosts to the reservoir, for example, by building latrines in the host community or by reducing the infection from the reservoir to the hosts, for example, by washing hands and personal hygiene, or by increasing parasite mortality, for example, through the application of periodic and specific antiparasitic treatments.

A HETEROGENEOUS MODEL

In this section, we consider the most general and realistic case of a heterogenous host population.

For this model, we assume that the host population, H , is divided into subpopulations, or groups, H_i , which present different characteristics, and therefore different risks of infection (for example, by age differential susceptibility, environmental conditions, access to sanitation and hygiene, etc.) Anderson and May (1992); Anderson et al. (2014); Brooker et al. (2006); Freeman et al. (2015); Truscott et al. (2014)).

The dynamics of parasitic infection for the case of a hete-

rogenous population is described as follows:

$$\begin{aligned}\frac{dm_i}{dt} &= \beta_i \ell - (\mu_h + \mu_p) m_i, \\ \frac{d\ell}{dt} &= \lambda_0 \alpha \sum_i \pi_i \rho_i m_i F(m_i) - \mu_\ell \ell,\end{aligned}\quad (25)$$

where $i = 1, \dots, n$ with n the number of groups in H . The other parameters corresponding to each group H_i are detailed in the following

- m_i is the mean parasite burden,
- β_i and ρ_i are the rate of contact (or exposure) and the rate of contribution of a host to the reservoir ℓ , respectively,
- N_i is the number of hosts in the group i and $\pi_i = N_i/N$,
- F is a product of two functions: the mean effective contribution per female parasite to the reservoir, ψ (see eq (3)), and the mating probability, ϕ (see eq (5)),

the rest of the parameters are defined as in the previous section.

Equilibria and basic reproduction number

In this section we find some useful expressions involving the equilibrium values of the dynamic variables and the basic reproduction number R_0 defined as in the previous section.

Assuming the reservoir is at equilibrium

$$\ell^* = \frac{\lambda_0 \alpha}{\mu_\ell} \sum_i \rho_i \pi_i m_i F(m_i), \quad (26)$$

and substituting this expression in the rest of the equations of the system (25), we obtain the following equation for the dynamics of the mean parasite burden, m_i , of the host group H_i ,

$$\frac{dm_i}{dt} = \beta_i \frac{\lambda_0 \alpha}{\mu_\ell} \sum_j \rho_j \pi_j m_j F(m_j) - (\mu_h + \mu_p) m_i, \quad (27)$$

where $j = 1, \dots, n$.

The mean parasite burden m of the host population is given by

$$m = \sum_i \pi_i m_i, \quad (28)$$

where $\pi_i = N_i/N$. Then, the dynamic of the mean parasite burden is described by

$$\begin{aligned}\frac{dm}{dt} &= \left(\sum_i \pi_i \beta_i \right) \frac{\lambda_0 \alpha}{\mu_\ell} \\ &\quad \times \sum_j \rho_j \pi_j m_j F(m_j) - (\mu_h + \mu_p) m.\end{aligned}\quad (29)$$

From this equation, the equilibrium mean parasite burden, m^* , is given by

$$\sum_i \pi_i \frac{\lambda_0 \alpha \rho_i}{\mu_\ell (\mu_h + \mu_p)} \left(\sum_j \pi_j \beta_j \right) F(m_i^*) m_i^* - m^* = 0, \quad (30)$$

where m_i^* is the equilibrium mean parasite burden correspond to each group H_i . An equilibrium condition for m_i^* is given by

$$F(m_i^*) = \frac{1}{R_0^i}, \quad (31)$$

where we define the basic reproductive number of each group H_i by

$$R_0^i = \frac{\lambda_0 \alpha \rho_i}{\mu_\ell(\mu_h + \mu_p)} \sum_j \pi_j \beta_j, \quad (32)$$

which is the number of adult females that are born from an adult female in the subpopulation H_i in the absence of the effects of density-dependence and mating probability.

Finally, from equation (27), the equilibrium mean parasite burden of each group H_i is given by

$$m_i^* = \frac{\beta_i \sum_j R_0^j \pi_j m_j^* F(m_j^*)}{\sum_j \pi_j \beta_j}. \quad (33)$$

Note that this is not an explicit expression for the equilibrium m_i^* . Therefore, the equilibrium value can only be solved numerically.

The general basic reproductive number R_0 for the total population is given by

$$R_0 = \frac{\lambda_0 \alpha}{\mu_\ell(\mu_h + \mu_p)} \sum_j \pi_j \rho_j \beta_j, \quad (34)$$

where we assume the absence of the effects of density-dependence and the mating probability (Anderson and May, 1992), that is, we assume in the system (25) the function F equal to unity. A relationship between R_0 and R_0^i is given by

$$R_0 = \frac{\sum_i \pi_i \beta_i R_0^i}{\sum_i \pi_i \beta_i}, \quad (35)$$

therefore, we obtained that $\min R_0^i \leq R_0 \leq \max R_0^i$, then we can interpret R_0 as an average value of the R_0^i .

In the heterogeneous model (25), bifurcation analysis is more complicated, however, numerical tests by considering different values of R_0^i can be considered, in order to better understand the dynamics of the model. A similar analysis can be found in (Bürger *et al.*, 2016).

DISCUSSION AND CONCLUSIONS

In this work, we developed deterministic mathematical models for the transmission dynamics of macroparasite infections.

We show how fundamental parameters related to production of fertilized parasites eggs are estimated from statistical models for the distribution of parasites within hosts.

We considered both homogeneous and heterogeneous host communities. The analyzed models show that the basic reproduction number R_0 strongly depends on the host egg contributions to the reservoir (which depend of the parameters ρ , α , and the parasite fecundity at low densities λ_0), on the host contact (or exposure) to the reservoir (which depend of the parameter β), and on the reservoir and parasite mortality (μ_ℓ and μ_p , respectively). Therefore, to achieve a reduction in R_0 we must, for example, provide access to hygiene and

build latrines in the host community, or implement regular and specific antiparasitic treatments.

For the homogeneous model we present a bifurcation analysis and show that this model undergoes a saddle-node bifurcation. The bifurcation parameter depends on the functions ψ and ϕ which in turn depend on the assumed distribution of parasites (see Lopez and Aparicio (2022)).

A puzzling result is that the disease-free equilibrium is locally asymptotically stable for all values of the basic reproduction number R_0 . Moreover, stable endemic equilibrium only exists for $R_0 > 1$ (see Figure 3). As the unstable equilibrium goes to zero as R_0 increases eventually any small perturbation will drive the system solutions to the stable endemic equilibrium which is not close to zero. Further reductions in R_0 will have little impact in the level of the infection in the population.

More refined models may be developed from the simple models presented here which may be useful in the design and evaluation of different control strategies.

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