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LETTER

Optimal time to patency in parasitic nematodes: host mortality matters

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Abstract

We develop an optimality model based on classical epidemiological models to investigate the optimal time to patency in parasitic nematodes in relation to host mortality and parasite mortality. We found that the optimal time to patency depends on both host longevity and prepatent mortality of nematodes. We tested our models using a comparative analysis of the relationships between nematode time to patency, nematode mortality and host mortality. Although we confirmed the importance of prepatent mortality, we also found a significant positive influence of host mortality. Host mortality rate affects parasite survivorship and life history strategies in the same way that habitat-specific mortality regimes drive the evolution of life histories in free-living organisms.

Keywords

Host mortality, life history, mammals, maturation time, nematodes.

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INTRODUCTION

Age-dependent mortality schedules are thought to be important driving forces in the evolution of life history strategies (Stearns 1992; Charnov, 1993), although specific models that make quantitative predictions are few. Gemmill *et al.* (1999) recently developed an optimality model that predicts the optimal age at maturity (i.e. time to patency) for parasitic nematodes. Time to patency, or the length of the time until maturation in the final host, is a determinant of body size and reproductive output in parasitic nematodes (Skørping *et al.* 1991; Read & Skørping 1995; Morand 1996; Poulin 1996; Morand & Sorci 1998) and also in parasitic platyhelminths (Trouvé *et al.* 1998; Trouvé & Morand 1998). The model of Gemmill *et al.* (1999) shows that the maturation time is inversely proportional to prematurational mortality rate (i.e. prepatent mortality). The model of Gemmill *et al.* (1999) does not consider host mortality and their finding suggests that host mortality has no significant influence on time to patency, which is in contrast to what Sorci *et al.* (1997) and Morand & Sorci (1998) suggested based on empirical evidence.

In this paper, we develop a different optimality model based on the macroparasite models of Anderson and May (May & Anderson 1978, 1979; Anderson & May 1985). We derive the basic transmission rate R_0, which can be used as a measure of parasite fitness (Lenski & May 1994; van Baalen & Sabelis 1995; Frank 1996), to highlight the importance of host mortality in determining the optimal time to patency. We also provide an empirical test of our model using a comparative analysis of the relationships between nematode time to patency, prepatent mortality and host mortality.

THE MODEL

We develop a model accounting for the dynamics of prepatent and adult populations of a nematode in its definitive host (Fig. 1).

The system can be modelled by two differential equations, which describe the larval population dynamics (L) and the adult parasite population dynamics (P):

$$\frac{dL}{dt} = \frac{\lambda\beta HP}{(\mu_w + \beta H)} - \frac{L}{\alpha} - (b + \mu_L)L \qquad (1)$$

Infection of the host is the result of egg production λ (per capita fecundity of adult worms), rate of infection β, host density H, and mortality of free-living larval stages μ w. Causes of mortality affecting prepatent stages are of intrinsic origin μ_L (mortality of prepatent stages) or due to host death (b). Pre-patent stages mature into adult parasites according to α, the time to patency.

The adult parasite population is affected by mortality due to host (b) and of intrinsic origin μ p (mortality rate of adult parasites)

$$\frac{dP}{dt} = \frac{L}{\alpha} - (b + \mu_p)P \qquad (2)$$

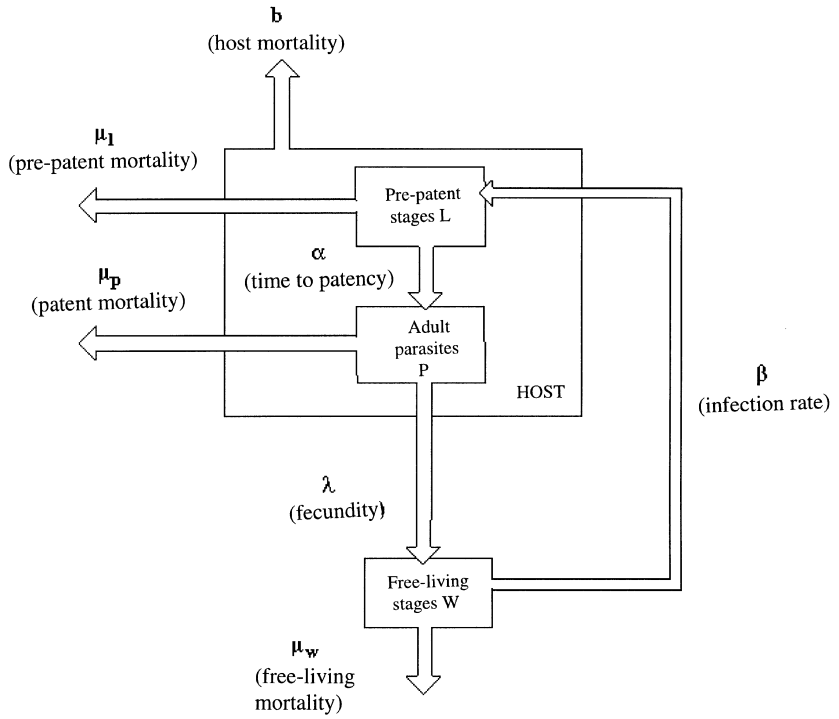

Figure 1 Flowchart of the model showing variables and parameters used.

The expression of the basic transmission rate is obtained by considering the increase of the parasite population when one parasite is introduced in a population of uninfected hosts (see Anderson & May 1985 for derivation).

$$R_0 = \frac{\lambda\beta H}{\alpha(\mu_w + \beta H)\left(\frac{1}{\alpha} + b + \mu_L\right)(b + \mu_P)} \qquad (3)$$

The basic transmission rate can be used as a fitness measure of a mutant parasite, assuming that multiple infections do not occur (van Baalen & Sabelis 1995; Frank 1996), which is obviously not often the case in natural conditions (but see the applications of epidemiological models in nematode parasites in Morand & Guégan, 2000).

The parasite fitness is maximal if its time to patency is optimal. The optimal time to patency is obtained by solving:

$$\frac{dR_0}{d\alpha} = 0$$

An empirical relationship between the time to patency and the length of the adult worm is

$$\text{Length} = \alpha^a$$

previously reported by Morand (1996).

We know that per capita fecundity is given by

$$\text{Length}^c$$

Hence the per capita fecundity is

$$\lambda = (\alpha^a)^c$$

It should be noted that the above relationships were obtained from interspecific studies and do not account for intraspecific variations, i.e. the negative relationship between nematode intensity and body size (and hence fecundity).

Incorporating this last expression into R_0 and deriving according to α gives

$$\frac{(\alpha^a)^c \mu_P \beta H(ca\mu_L\alpha + ca + cab\alpha - \mu_L\alpha - b\alpha)}{\alpha(\mu_w + \beta H)(\mu_L\alpha + 1 + b\alpha)^2(\mu_P + b)} = 0$$

The optimal time to patency is given by

$$\alpha^* = \frac{-ca}{ca\mu_L + cab - \mu_L - b} \qquad (4)$$

Using estimated values of c (1.89 ± 0.31) and a (0.39 ± 0.15) from Morand (1996), and independent contrasts on data compiled by Morand (1996), gives us the time to patency

$$\alpha^* = \frac{2.80}{\mu_L + b} \qquad (5)$$

with the following range,

$$\left[\frac{0.63}{\mu_L + b}, \frac{90}{\mu_L + b}\right]$$

The optimal time to patency depends on both the prepatent mortality of the parasite and the host mortality.

Note that the time to patency cannot be derived for $ac = 1$.

COMPARATIVE TEST

We used the data on life history traits of nematodes parasitic in mammals compiled by Morand (1996). Estimates of mortality rates are scarce. We assumed that mortality rates do not change before (μ_L) and after maturation (μ_p) and then assumed that prepatent and patent mortality are similar. Data on mortality of mammals are from Eisenberg (1981) (Table 1). We used maximum life span. The average instantaneous mortality rates of parasites and hosts were calculated following Purvis & Harvey (1995) as

$$-\ln(1-1/E)$$

where E is the maximum life expectancy at maturity in days.

To control for the effects of phylogeny, we used the method of independent contrasts (Felsenstein 1985). We used the CAIC program (Purvis & Rambaut 1995). We constructed a phylogeny of the nematodes based on the taxonomic information in Morand (1996) and the molecular phylogeny of De Blaxter *et al.* (1998). We used a gradual model of evolution but all branch lengths were assumed to equal 1 because of the lack of information on actual branch lengths. In order to verify that contrasts are properly standardized we performed a regression of the absolute values of standardized contrasts *versus* their standard deviations (Garland *et al.* 1992).

In accordance with equation 5, we found a significant positive relationship between the time to patency (α) and the inverse of the sum of parasite mortality and host mortality $1/(\mu_1 + b)$, although the slope of the observed relationship (0.10, range 0.07–0.15 using Major Axis Regression, 95% confidence interval) is less than the range of slopes expected by the optimality model (0.63–90) (Fig. 2A).

We also found a positive relationship between parasite mortality and host mortality (Fig. 2B). Finally, using a multiple linear regression we found that the time to patency is better explained by host mortality ($P = 0.0102$) than by parasite mortality ($P = 0.1486$).

Because of the positive relationship between parasite mortality and host mortality, we used adjusted values for nematode mortality given by the preceding relationship. Using these estimates, we then found a new significant relationship between the time to patency (α) and the inverse of the sum of adjusted parasite mortality and host mortality. The slope of the new relationship is 2.84 (range 1.33–32.59 using Major Axis Regression, 95% confidence interval), which is consistent with the slope expected from the optimality model (2.80; range 0.63–90, 95% confidence interval) (Fig. 2C).

Equation 5 allows the estimation of the invariant αM, which is equal to 2.8.

Table 1 Time to patency, parasite longevity and host mortality of nematodes of mammals (data from Eisenberg 1981; Stearns 1992; Morand 1996

Nematode species	Time to patency (days)	Parasite longevity (days)	Host longevity (months)
Acanthocheilonema vittae	50	450	36
Ancylostoma duodenale	39	1640	720
Ascaris lumbricoïdes	65	548	720
Callodium hepaticum	16	59	18
Enterobius vermicularis	25	60	720
Litosomoides carinii	74	360	36
Muellerius capillaris	7	2000	144
Necator americanus	42	2008	720
Nematospiroides dubius	10	84	18
Nippostrongylus brasiliensis	6	15	36
Ostertagia ostertagi	21	365	144
Parascaris equorum	93	270	600
Pseudoterranova decipiens	25	40	228
Strongyloides stercolaris	35	56	120
Syphacia obvelata	10	16	18
Toxocara canis	32	14	180
Toxocara cati	38	90	252
Trichuris muris	14	38	36
Trichuris trichiurus	49	720	720
Wuchereria bancrofti	175	1642	720

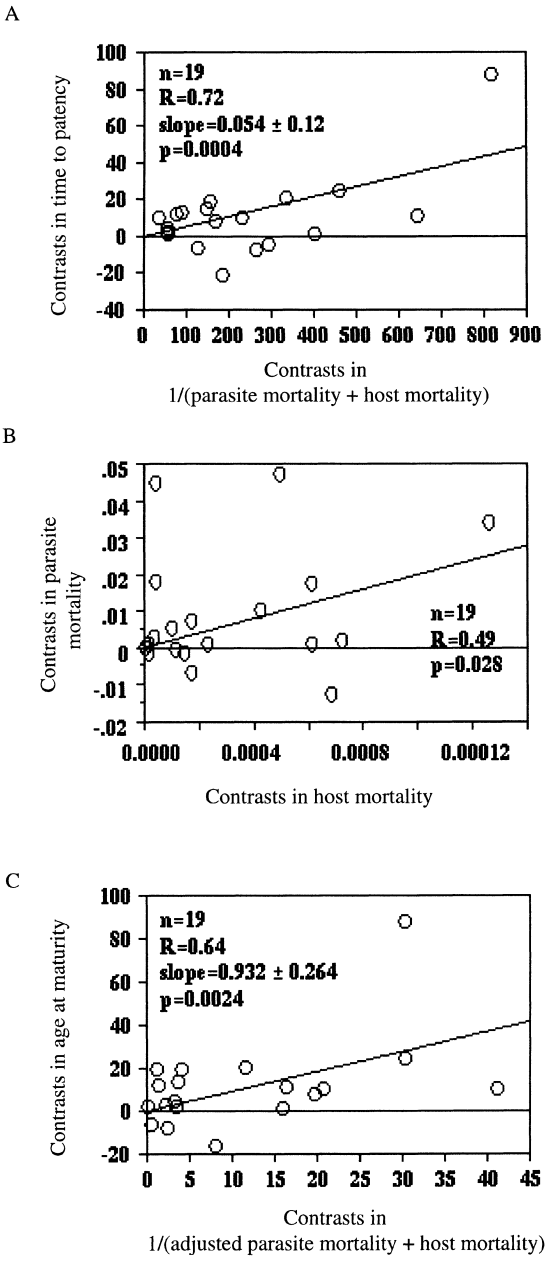

Figure 2 (A) Relationship between time to patency of parasitic nematodes of mammals and the inverse of the sum of parasite mortality and host mortality using independent contrasts (observed Ordinary Least Squares slope in solid line). The value of the RMA slope is 0.10 (95% confidence interval: 0.07–0.15). (B) Relationship between parasite mortality and host mortality (b), using independent contrasts. (C) Relationship between time to patency of parasitic nematodes of mammals and the inverse of the sum of adjusted parasite mortality and host mortality (adjusted μ_1 + b), using independent contrasts (observed OLS slope in solid line). The value of the RMA slope is 2.84 (95% confidence interval: 1.33–32.59).

DISCUSSION

The results of our model contrast with some of the results of Gemmill *et al.*'s (1999) optimality model, although we confirmed the importance of the prepatent mortality of the parasite for the time to patency. It must be pointed out that we assumed that prepatent mortality is equal to adult parasite mortality. Indeed, if prepatent mortality is higher (or lower) than adult mortality, this would change the value of the observed slope between time to patency and the sum of parasite mortality and host mortality. As it is, our model does not capture all of the variation in time to patency. This may be explained by its over-simplification, i.e. no influences of multiple infection, and by the difficulty in obtaining good estimates of life history parameters, in particular estimates of prepatent mortality. However, we adjusted values for parasite mortality given by the relationship between host mortality and parasite mortality. Using this estimate, we found a new significant relationship between the time to patency (α) and the inverse of the sum of adjusted parasite mortality and host mortality. The slope of the new relationship (2.84) is the slope expected by the optimality model (2.80).

The relationship between host longevity and time to patency in worms is intriguing since host longevity is considerably longer than the worm longevity (by one order or two orders of magnitude). However, it is well known that host longevity correlates with other host life history traits such as age at reproduction. A positive correlation between parasite and host life history traits was previously shown for oxyuroid–primate associations (Harvey & Keymer 1991; Sorci *et al.* 1997). The results of Harvey & Keymer (1991), Sorci *et al.* (1997) and the present study underline the coevolutionary process that may shape life history traits in host-parasite systems.

The model of Gemmill *et al.* (1999) explained better a greater proportion of the variance in the time to patency in nematodes than did our model using nonadjusted values of nematode mortality. They also gave a better estimate of the invariant αM (1.45–2.5), i.e. the product of the time to patency and the mortality rate, than previously estimated by Morand (1996) (0.23). Our model gives an estimate of αM of 2.8, which is closer to Gemmill *et al.*'s estimate. The poor predictive value of our model may be due either to the epidemiological model not capturing all the biological information (presence of multiple infections rather than single infection, and competition processes) or to the poor estimates of prepatent mortality (assumed to equal to the adult parasite mortality rate). This was confirmed by the use of adjusted values of nematode mortality. The correlation of parasite mortality with host mortality supports our hypothesis.

Nematode maturation presumably occurs at the time that maximizes reproductive success. Hence, high levels of larval parasite mortality should select for a reduction of time to patency; whereas greater host longevity should favour delayed parasite maturity. Interspecific studies seem to support this view but the more critical intraspecific studies are lacking. Host mortality rate may affect parasite survivorship and life history strategies in various ways: in a way similar to habitat-specific mortality regimes driving the evolution of life histories in free-living organisms (Southwood 1977; Stearns 1992), or because host mortality rate is linked to other relevant life traits that our model does not incorporate. For instance, the investment in immune response might differ between long-lived and short-lived hosts. The latter possibility will require testing when data become available.

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BIOSKETCH

Serge Morand is interested in the ecology and evolution of host–parasite interactions, with a main emphasis on life history evolution and macroecology in a comparative perspective.

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