Optimal timing of first reproduction in parasitic nematodes

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Abstract

The time between infection and the onset of reproduction (maturation time) is a key determinant of body size, fecundity and generation time in parasitic nematodes. An optimality model for maturation time is developed centred on prematurational growth, the duration of which has opposing consequences for fecundity and for survival to reproductive age. The maturation time favoured by natural selection is found to be inversely proportional to prematurational mortality rate. The product of maturation time and mortality rate is predicted to be an invariant number equal to the allometric slope linking daily fecundity to maturation time. Predictions are tested using comparative data on mammalian gastrointestinal nematode taxa. Half the cross-species variation in prepatent period (the time from infection until propagules are shed from the host) is accounted for by this adaptive trade-off hypothesis, even though many biological details are not explicitly modelled. These results are discussed in the light of previous studies and in the context of general models of life history evolution.

Introduction

Maturation time in the final host is a major determinant of generation time, body size and reproductive output in parasitic nematodes (Skorping *et al*., 1991; Read & Skorping, 1995; Morand & Sorci, 1998). These parameters not only represent some key components of parasitic nematode fitness, they also affect levels of infection and pathology experienced by hosts (Read & Skorping, 1995). An improved understanding of how selection acts on maturation time is of applied as well as theoretical interest since medical and veterinary intervention programmes are expected to alter selection on parasite life history schedules (Medley, 1994; Read & Skorping, 1995; Buckling *et al*., 1997; Poulin, 1998; Skorping & Read, 1998).

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As in many organisms (Peters, 1983), adult female body size is closely linked to reproductive success in parasitic nematodes (Skorping *et al*., 1991). Across species there is a positive correlation between fecundity and prepatent period (the time from infection until parasite propagules are shed from the host; Skorping *et al*., 1991; Morand, 1996). This almost certainly arises because bigger worms take longer to grow and are more fecund than smaller worms. Where somatic growth either ceases or slows at maturity, as seems to be the case in nematodes (Malakhov, 1994), age and size at maturity must, in general, be positively correlated (Stearns, 1992). Thus, *Trichinella spiralis* is a few millimetres long and commences reproduction less than a week after infection, while *Ascaris lumbricoides* is about 30 cm in length and can spend several months growing before beginning to produce eggs.

On a cross-taxa level, the relationship between daily fecundity and prepatent period in parasitic nematodes is well described by an allometric relationship (Skorping *et al*., 1991) $Y = cX^{\beta}$, where X is prepatent period, Y is daily fecundity, and the exponent β is the

allometric slope (the slope of the regression of log Y on log X). The allometric slope of daily fecundity with prepatent period is greater than +1 across gastrointestinal nematode taxa that parasitize mammals; Skorping *et al.* (1991) reported a slope of 2.66 across 19 genera. This has the immediate implication that daily fecundity increases disproportionately (i.e. more than linearly) for a given delay in the onset of reproduction. Since a female's future reproductive output can be substantially enhanced by postponing maturation, it is natural to ask what constrains the evolution of ever longer maturation times.

Here we develop an optimality model incorporating a trade-off between the size-mediated fecundity advantage of delaying reproduction and the potential disadvantage of doing so: longer growth phases entail increased exposure to the risk of prematurational mortality. Natural selection should act to optimize the fitness consequences (costs and benefits) of this trade-off, an intuitive expectation that has been supported in several theoretical studies (e.g. Bell, 1980; Kozlowski & Wiegert, 1987; see Stearns, 1992, for discussion). We ask how well the model explains quantitative variation in prepatent period across gastrointestinal nematode taxa and conclude by discussing results in relation to more general life history frameworks.

Model

Maturation time (α) is defined as the time (post infection) at which a female first reproduces. We wish to know how the optimal maturation time of a female parasitic nematode, α^*, varies as a function of mortality rate and potential fecundity.

We assume the following.

(i) Lifetime reproductive output (total number of offspring produced by a parasitic female in her lifetime) is an appropriate measure of fitness (ω). This assumption holds if R_0, the average number of parasitic females produced by a parasitic mother which then survive to reproduce, is, on average, 1. When $R_0 = 1$, the population is neither increasing nor decreasing. This is believed to be approximately true of helminth populations in general (Anderson & May, 1985).

(ii) Average per unit time fecundity (m) depends on maturation time (α) according to the relationship $m = c\alpha^{\beta}$, where c is a constant and the exponent β is the allometric slope (the slope of the regression of log m on log α).

(iii) Upon entering a host, parasites are first subject to a constant prematurational mortality rate, M_i, until maturation (at time α) followed by a constant postmaturational mortality rate, M_a.

(iv) Per unit time fecundity, m, is determined by body size at maturation (time α) and is independent of the time since maturation and of any postmaturational growth.

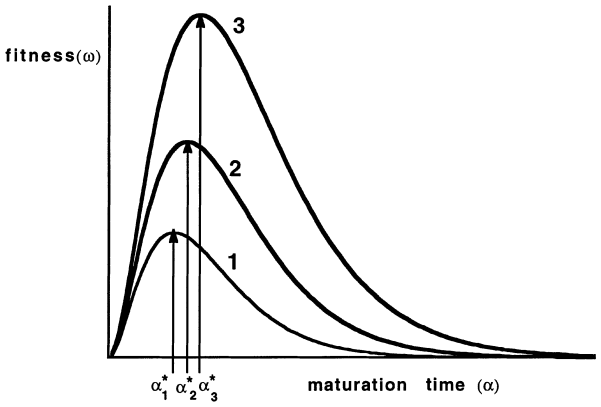

Fig. 1 Fitness (ω = lifetime reproductive output) as a function of maturation time (α) under three different prematurational mortality rates (M_i). The curve labelled 1 was generated with the highest value of M_i and the curve labelled 3 with the lowest. The peak of each fitness curve corresponds to the optimal maturation time (α^*) as indicated by arrows. Scale and units arbitrary.

Under assumptions (i) to (iv), we can write

$$\omega = c\alpha^{\beta} e^{-M_i\alpha} \frac{1}{M_a}, \qquad (1)$$

since $\exp(-M_i\alpha)$ is the proportion of worms surviving at maturity (time α), daily fecundity is $c\alpha^{\beta}$ and life expectancy after time α equals $1/M_a$. This function has a single maximum at α^*, the maturation time that maximizes fitness (ω). Figure 1 shows fitness (ω) plotted against maturation time (α) for a range of values of prematurational mortality rate (M_i).

Differentiating eqn 1 with respect to α gives

$$\frac{d\omega}{d\alpha} = \beta \frac{1}{M_a} c\alpha^{(\beta-1)} e^{-M_i\alpha} - M_i \frac{1}{M_a} c\alpha^{\beta} e^{-M_i\alpha}.$$

To find α^* we set $d\omega/d\alpha = 0$, which, after some rearrangement, gives

$$\alpha^* = \frac{\beta}{M_i}. \qquad (2)$$

Hence the model predicts optimal maturation time (α^*) to be proportional to the inverse of prematurational mortality rate (M_i). The constant of proportionality is β, the allometric slope of per unit time fecundity with maturation time. As shown, α^* becomes progressively smaller as prematurational mortality rate increases. Note that the fitness costs of longer maturation time arise solely because delaying reproduction results in fewer worms surviving to reproductive age. Post-maturational mortality rate (M_a), which is not a function of α in the model, affects the height of the fitness function but not the position of the optimal maturation time (α^*).

Under the assumptions listed above, eqn 2 is also given by expansion of either $\sum_{x=0}^{\infty} l(x)m(x)$ or $\int_{x=0}^{\infty} l(x)m(x)$ (where $l(x)$ is the probability of survival to time x and

$m(x)$ is fecundity if alive at time x) using geometric series (see Roff, 1992; Bulmer, 1994).

Testing the model

We now test how well the model explains observed maturation time in a range of mammalian gastrointestinal nematodes. To do this, we use life history data compiled from the literature for 66 species (Skorping *et al*., 1991). First we estimate β, the allometric slope linking per unit time fecundity to maturation time. As a measure of maturation time we use data on prepatent period, the time from infection until parasites' propagules are shed from the host. Per unit time fecundity is represented by data on daily fecundity (offspring per female parasite per 24 h). The resulting estimates of β are then used to generate predicted values. Next, mortality rates are estimated from data on patent period to allow a test of the model's predictions. Observed and predicted values are compared by regression.

Estimating β

Maturation time and fecundity

Determining the relationship between fecundity and maturation time among individual parasitic nematodes is, for obvious practical reasons, not easy and we know of no relevant estimates based on intraspecific data. We therefore used cross-taxa data to estimate an average value of β. Arguably, the relationship between maturation time and fecundity across taxa more closely reflects the relationship that natural selection acts on, spanning, as it does, greater variance in both variables and being less influenced by environmental variation faced by individual worms.

Phylogeny

Statistical nonindependence of species values due to shared ancestry needs to be controlled for when estimating slopes of allometric relations from comparative data (Harvey & Pagel, 1991). Several methods (and associated computer programs) are available which use phylogenetic information to calculate standardized differences (independent contrasts, here abbreviated to ICs) between pairs of sister taxa as first advocated by Felsenstein (1985). While the values of sister taxa themselves are not independent, ICs are and can therefore be used to test for relationships between variables by standard statistical methods.

Daily fecundity and prepatent period data were first $\log_{10}$ transformed. Sets of ICs were generated with the CAIC computer package (Purvis & Rambaut, 1995) using a phylogeny based on that given in Read & Skorping (1995) with additional resolution provided by data in Blaxter *et al*. (1998) and Newton *et al*. (1998). Data on the majority of branch lengths in this phylogeny (Fig. 2) are unavailable and in this situation several approaches are possible. The assumption that branch lengths are equal (punctuated evolution model) has proved the most robust option in simulation studies (Purvis *et al*., 1994) and was used here. All regressions involving ICs must be forced through the origin (see Garland *et al*., 1992).

Regression

To estimate the value of a cross-taxa regression coefficient, a choice of regression model has to be made. Ideally, where the error rates in both variables (or their ratio, $Y_{\sigma^2_e}/X_{\sigma^2_e}$) are known, the structural relation (SR) should be used (Rayner, 1985; Harvey & Pagel, 1991; Sokal & Rohlf, 1995). So-called Model I (ordinary least squares or OLS) and Model II (major axis and reduced major axis) regression are particular cases of the SR (Rayner, 1985). The OLS slope estimate converges on the SR as $(Y_{\sigma^2_e}/X_{\sigma^2_e})^{-1}$ approaches zero (Kuhry & Marcus, 1977). In Model II methods, major axis (MA) assumes equal X and Y error variances ($Y_{\sigma^2_e}/X_{\sigma^2_e} = 1$) and reduced major axis (RMA) assumes the ratio of error variances to be equalled by the ratio of the variances ($Y_{\sigma^2_e}/X_{\sigma^2_e} = Y_{\sigma^2}/X_{\sigma^2}$).

Since the error rates in estimates of our Y-variable, daily fecundity (offspring per female parasite per day), are almost certainly far more substantial than the error rate in estimates of prepatent period, the equal error variance assumption of MA seems least appropriate. OLS may be an adequate model if $(Y_{\sigma^2_e}/X_{\sigma^2_e})^{-1}$ is sufficiently small (Rayner, 1985). Since we have no reason to expect OLS or RMA to be more or less successful estimators of the functional relationship, results are reported for analyses employing both regression models.

Daily fecundity and prepatent period data are available for 28 species from six nematode orders. The OLS and RMA slopes of the cross-species regression of fecundity on prepatent period (±95% CI) are 2.42 (±0.21) and 2.48 (±0.22), respectively (Fig. 3a). The CAIC analysis yields 22 pairs of ICs. Five of the Y-values are negative and therefore some care is required before proceeding to a slope estimate. Because of the way in which ICs are calculated, all X-values are positive (Garland *et al*., 1992). Negative Y-values therefore indicate that an increase in X (prepatent period) is associated with a decrease in Y (daily fecundity). If negative values of Y are as likely as positive values, there is no evidence of an association of X with Y and little point in fitting a regression line. Consequently, a binomial (or similar) test should first be applied to determine whether the frequencies of positive and negative Y-values are significantly different from the 50:50 ratio expected under the null hypothesis of no association of X with Y (Harvey & Pagel, 1991). This is indeed the case (one-tailed binomial probability < 0.01) indicating that a positive relationship of daily fecundity with prepatent period exists across gastrointestinal nematode taxa once phylogenetic effects are accounted for. OLS and RMA regressions through the origin give slopes (±95% CI) of 1.45 (±0.95) and 2.5 (±1.6), respectively (Fig. 3b). As expected, the OLS slope is lower than the

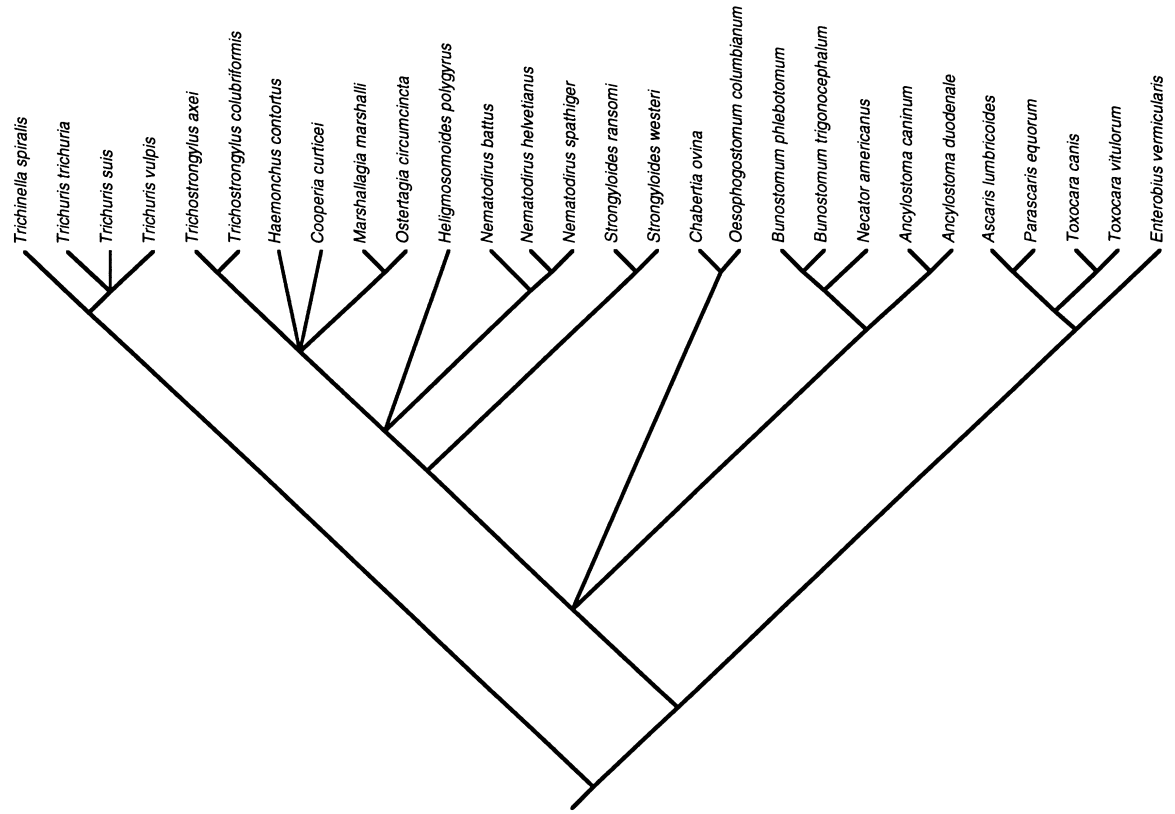

Fig. 2 Phylogenetic relationships of species used in the analysis of prepatent period and daily fecundity data. Depicted branch lengths are arbitrary.

RMA, but neither estimate is significantly different from the other or from the cross-species slope estimates of 2.42 and 2.48.

Estimating mortality rates

Direct estimates of mortality rates for parasitic nematodes are extremely rare. However, if we make the additional assumption (assumption v) that mortality rates do not change significantly after maturity (i.e. $M_i \approx M_a$) an estimate can be made using data on patent period (P), the duration of egg or larval shedding from an infected host.

We can write $M_i = M_a = M = (1/L)$, where M is the average mortality rate and its reciprocal, L, is average life expectancy. Cast in these terms the model's prediction is

$$\alpha^* = \beta L \tag{3a}$$

or

$$\alpha^* M = \beta. \tag{3b}$$

Our life history data ultimately derive from parasitological studies in which large numbers of parasites are inoculated into hosts. Thus, maximum lifespan (prepatent period + patent period, here called L_{max}) records the age of the longest lived parasite in a cohort. As discussed by Beverton and others (Beverton, 1992, and references therein) L_{max} overestimates L (the average life expectancy) by a factor g such that $L_{max} = gL$ (and hence $g = ML_{max}$). In studies involving other taxa, indirect estimates of g have been obtained and applied with some success (e.g. Hoenig, 1983; Beverton, 1992; Charnov, 1993). These methods rely on obtaining independent estimates of L_{max} and M in other closely related species or populations and, consequently, are not of use here. However, given assumption v, we can estimate g from the starting number in a cohort (N) as follows. Writing the standard equation for a survival curve under constant M we have

$$S(t) = \mathrm{e}^{-Mt},$$

where $S(t)$ = proportion surviving at time t. In the case of $t = L_{max}$, a single individual achieves age L_{max}, so that

$$S(L_{max}) = 1/N,$$

and we can write

$$\frac{1}{N} = \mathrm{e}^{-ML_{max}},$$

and so

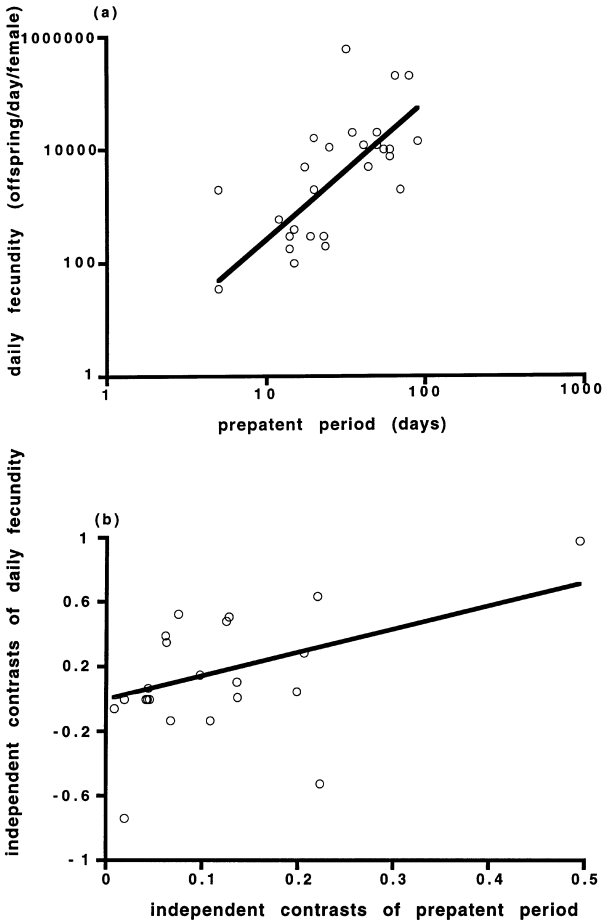

Fig. 3 Regressions through the origin of $\log_{10}$ daily fecundity on $\log_{10}$ prepatent period across gastrointestinal nematodes. In both cases plotted line is the OLS regression. In (a), data are average species values. In (b), data are independent contrasts controlling for phylogeny (see text). In (a), OLS slope = 2.42 (95% CI ± 0.21), $r = 0.97$, $P < 0.0001$; RMA slope = 2.48 (95% CI ± 0.22). In (b), OLS slope = 1.45 (95% CI ± 0.95), $r = 0.58$, $P < 0.005$; RMA slope = 2.5 (95% CI ± 1.6).

$$\ln(1/N) = -ML_{\max}.$$

Beverton's relation states that $g = ML_{\max}$ and so

$$g = -\ln(1/N) = \ln N. \qquad (4)$$

Thus for parasitic nematodes in experimental infections, g approximates to ln N. Patent period (P) can be written as $L_{\max} - \alpha$. Under assumption v, just as $(L_{\max}/L) = ML_{\max}$, so also $(L_{\max} - \alpha)/L = M(L_{\max} - \alpha)$. The factor by which patent period (P) overestimates L is therefore $ML_{\max} - \alpha M$ which (from eqns 3b and 4) we can write as $\ln N - \beta$. We will refer to $P/(\ln N - \beta)$ as L_{adj}, the adjusted estimate of life expectancy. Data on the dose of parasites administered (equivalent to the starting number, N) are not available for every species in the dataset. Most of the life history data are from monographs which cite numerous original studies when discussing general life history attributes of particular species. To estimate the dose in a typical infection, as many original references as could be matched with a specific estimate of patent period were compiled (Table 1). The average value of g in this compilation is 10 (95% CI ± 0.75). Accordingly, estimated life expectancy (L_{adj}) for a given species was calculated as $P/(10 - \beta)$. However, our conclusions are unchanged for a range of values of ln$N$ between 8 and 14.

Table 1 Doses of parasite infective stages and source references.

Reference	Doses administered (×1000)
Herlich (1954)	0.37; 0.62; 16; 28; 100; 140; 1000
Mayhew (1962)	6.16; 9.18; 9.88; 13.02
Bizzel & Ciordia (1965)	20; 22; 25; 25; 25; 35; 35; 38; 38; 40; 62

Observation and prediction

Data on patent period and prepatent period in natural hosts are available for 37 species. Applying eqn 3a, with $\beta = 1.45$ or $\beta = 2.5$ (the phylogenetically controlled allometric slope estimates of daily fecundity with prepatent period), the optimal maturation times predicted by our model are calculated as βL_{adj}. An obvious test of the predicted values' fit with observation is by regression. This will show a significant 1:1 relationship if observation and prediction agree. The distributions of observed and predicted values are right-skewed (data not shown) so that prior to testing their fit, an appropriate transformation is required for both variables (Roff, 1992). Box-Cox transformation (Sokal & Rohlf, 1995) with $\lambda = 0.2$ results in maximal normality of both variables and Fig. 4 shows the regression plots of the transformed data for $\beta = 1.45$ and $\beta = 2.5$, respectively. As shown in Fig. 4, the slopes and intercepts of these plots are not significantly different from +1 and zero, respectively. A value of $\beta = 1.45$ produces a tighter visual fit than $\beta = 2.5$. Nevertheless, for either value of β, prediction and observation are highly correlated ($r = 0.71$, $P < 0.0001$ in both cases). In fact, so long as β lies between 0.5 and 3.1 the data are a reasonable fit to either set of predicted values (slopes and intercepts not significantly different from 1:1 expectations).

There is another way to test the model's capacity for describing the data. Since $\alpha^* M = \beta$ (eqn 3b), the model yields a dimensionless number, αM, and predicts that independent of separate values of α and M, their product must always equal β. The number αM (the product of age at maturity and mortality rate) is a known life history invariant in a wide range of animal taxa (Charnov, 1993). Where αM is truly invariant (proportionality), the slope of a log–log regression of L on α is equal to unity (Charnov, 1993). Our model predicts this relationship. Taking logs of both sides of eqn 3b:

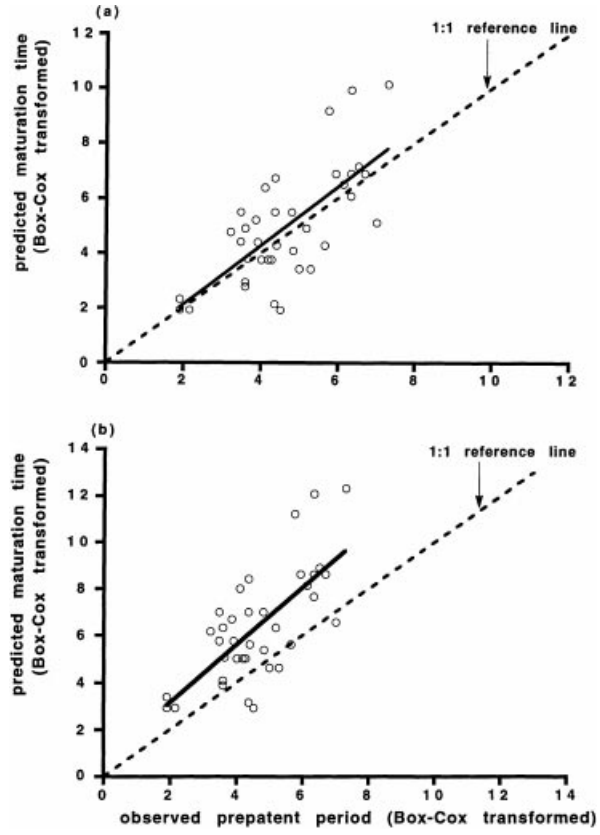

Fig. 4 Plots of predicted vs. observed values of maturation time ($n = 37$ species). In both plots, broken line is the 1:1 reference line. All data are Box-Cox transformed with $\lambda = 0.2$ (see text). In (a), $\beta = 1.45$; slope between observed and predicted values = 1.07 (95% CI ± 0.37); intercept = –0.02 (95% CI ± 1.79). In (b), $\beta = 2.5$; slope between observed and predicted values = 1.23 (95% CI ± 0.42), intercept = 0.70 (95% CI ± 1.65). In both regressions (OLS), $r = 0.71$, $P < 0.0001$.

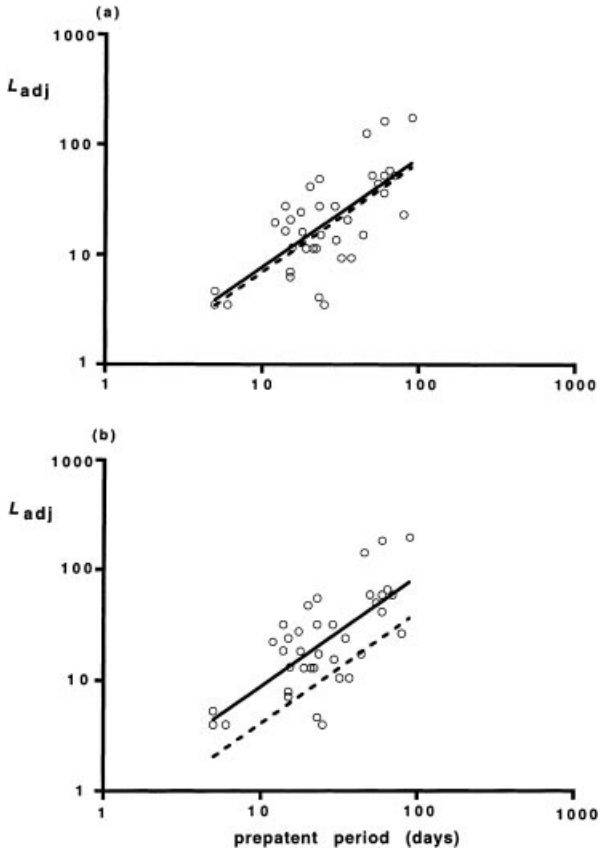

Fig. 5 Log_{10}–log_{10} plots of life expectancy (L_{adj}) and prepatent period with $g = 10$ ($n = 37$ species). The OLS regressions for $\beta = 1.45$ and $\beta = 2.5$ (solid lines) are plotted alongside the lines predicted by the model (broken lines). In (a), $\beta = 1.45$, OLS slope = 0.99 (95% CI ± 0.34), intercept = –0.10 (95% CI ± 0.49). In (b), $\beta = 2.5$, OLS slope = 0.99 (95% CI ± 0.34), intercept = –0.05 (95% CI ± 0.49). For both values of β, $r = 0.71$, $P < 0.0001$; RMA slopes = 1.41 (±0.48).

$$\log L = \log \alpha^* - \log \beta \tag{5a}$$

or

$$\log \alpha^* = \log L + \log \beta. \tag{5b}$$

Equations 5a and 5b define straight lines with slopes of +1 (proportionality) and intercepts at log β and –log β, respectively. We can ask whether parasitic nematode life histories conform to these lines by testing their goodness of fit with the observed log–log regressions. As with the data on fecundity and prepatent period, OLS and RMA regression can be used to estimate the functional relationship between prepatent period and L_{adj}. Since L_{adj} is likely measured with far greater error than prepatent period, when using OLS the regression of life expectancy on prepatent period (eqn 5a) is the most appropriate (regression of prepatent period on L_{adj} involves a major violation of the error variance assumption of OLS and may severely underestimate the slope). If eqn 5a successfully describes parasitic nematode life histories, the regression will have an intercept at –log β and a slope of +1. Figure 5 shows the OLS regressions of log L_{adj} on log prepatent period for $\beta = 1.45$ and $\beta = 2.5$. The slopes are not different from +1 (invariance). The regression lines fall close to the predicted lines and the intercepts are not statistically different from either predicted value (–0.16 and –0.39). RMA regressions give slopes of 1.41 (±0.48), again not different from the predicted value of +1. As in the comparison of observed and predicted values (Fig. 4), the regression lines account for around 50% of the variance ($r^2 = 0.497$).

Discussion

This study attempts to explain variation in a central life history trait in parasitic nematodes, in-host maturation time, in terms of a trade-off mediated by the opposing

effects of development time on fecundity and mortality. Worms that grow for longer periods before reproducing are less likely to survive until reproductive age but have potentially higher per day fecundity. The optimality model is simple and makes clear predictions about relationships between life history variables, explaining around 50% of the variation in prepatent period (Figs. 4 and 5).

None of our model's assumptions apply exclusively to interspecific variation. If individual parasitic nematodes were able to assess the prevailing in-host parasite mortality rate – and it may be a relatively straightforward matter to assess rate of immune attack for instance – natural selection should favour those individuals that modify their maturation time accordingly. In some other helminth parasites, host immune substances are known to cue parasite reproduction (e.g. Amiri *et al*., 1992). It would be extremely interesting to know to what extent, if any, facultative modulation contributes to the observed variation in prepatent period seen within species. In any case, the framework used here should prove useful in investigating the potential effects of medical and veterinary interventions on the evolution of size, fecundity and other life history traits in these parasites. Many of these latter traits are highly correlated with prepatent period and with each other across taxa (Skorping *et al*., 1991; Morand & Sorci, 1998).

The explanatory power of the model is perhaps surprising given the somewhat crude methods used to estimate parameters. For example, in testing our predictions we assumed that all gastrointestinal nematode species share approximately the same growth curve and estimated the allometric slope β from data showing considerable scatter (Fig. 3). Indeed, some taxa in our sample show patterns of life history traits which contradict the assumption that development time is positively associated with fecundity (i.e. the negative values in Fig. 3b). Similarly, the assumption (assumption v) of an unchanging mortality rate throughout infection is probably an oversimplification for at least some of the species in the analysis.

Further, as a measure of maturation time in gastrointestinal nematodes, prepatent period is not perfect: the first eggs or larvae to appear will be those of the earliest maturing individuals, not those of the average individual whose behaviour the model predicts. The time lag between the production of eggs by parasites and their eventual exit from the host is also ignored. Other than making some modest across-the-board adjustment to the observed values of prepatent period, there is currently little we can do to rectify these problems. It would of course be of interest to know how the model performs when more direct estimates of average maturation time become available.

Several of the modelling assumptions are likely to be violated in nature. For example, we assume that once reproduction begins, per unit time fecundity remains constant until death (assumption iv) which is valid if growth ceases at time α and there is no senescence. But while growth slows at maturity it continues in many nematodes (Anderson, 1992), and a fall in reproductive output with time is common among gastrointestinal species (Wakelin, 1996). It may be that any gains in fecundity accruing through post-maturational growth are approximately balanced by a declining fecundity in later life.

Despite the above limitations, the model makes quantitatively successful predictions. Such predictions have been noticeably absent from the literature on parasite life histories (Skorping *et al*., 1991; Poulin, 1995; Read & Skorping, 1995; Morand & Sorci, 1998). Roff (1984) used a similar modelling approach to that employed here to predict age at maturity in teleost fishes. Despite containing some equally simplified assumptions (such as determinate growth), his model successfully described the pattern of maturation events across a large number of teleost species. Like nematodes, most teleosts are indeterminate growers whose fecundity is closely linked to their body size. It may be that a model similar to Roff's (it incorporates a Von Bertalanffy growth curve and assumes fecundity is proportional to length^3) would also be successful in describing nematode life histories. However, we are unaware of any of the parameter estimates needed to incorporate such growth curves in the model. It would be of interest to know how well our model could describe teleost life histories if relevant data on per unit time fecundity were applied.

In the present study, the product of mortality rate (M) and maturation time (α) is predicted to be invariant and equal to β, the allometric slope of per unit time fecundity with α. Morand (1996) first estimated αM (his aM) in nematode parasites of vertebrates as 0.23. This implies that a parasitic nematode maturing at 1 month post-infection has, on average, about 4 months left to live. We find a very different value of αM (1.45–2.5) suggesting that worms devote a substantially larger portion of total lifespan to maturation than previously suspected. However, Morand's estimate is based on a comparison of prepatent period with patent period (the maximum duration of egg or larval production). As recognized elsewhere, the use of maximum reproductive lifespan as a measure of life expectancy in these organisms tends to overestimation (Anderson & May, 1985). The use here of the correction factor g may be an improvement in this regard.

Our estimate of αM in nematodes is similar to that of other poikilothermic indeterminate growers, such as fish and shrimp ($\alpha M \approx 2$ in both cases) at the other end of a life history spectrum from birds and mammals ($\alpha M \approx 0.4$ and 0.71, respectively; Charnov, 1993, his figure 1.9). Charnov (1993) developed ESS models for the αM number in determinate growers, the most general formulation of which led to the prediction $\alpha M = 3(1 - \delta^{0.25})$, where δ is relative size at independence (offspring size/maternal size). When we apply this equation, calculating

δ as egg volume/female volume ($n = 38$ species), we find that for mammalian gastrointestinal nematodes $3(1 - \delta^{0.25}) = 2.52$ (95% CI ± 0.11). This is not statistically different from the cross-species estimates of β or from the phylogenetically controlled estimate from RMA regression (Fig. 3). It may be that Charnov's equation is so fundamental that the complications of indeterminate growth prove to be of relatively minor importance. In fact, there is recognition that the distinction between determinate and indeterminate growth is somewhat artificial, a more useful distinction being the one between organisms which approach their asymptotic size slowly and those which approach it abruptly (Beverton, 1992).

Charnov (1993) postulated that $V(\alpha) \propto \alpha^d$, where $V(\alpha)$ is the reproductive value of a female who attains maturity (at age α), and assuming that mortality is unchanged after maturity derived the result $\alpha M = d$. Thus αM was found to be equal to the exponent linking fitness to age at maturity, similar to our own finding for nematodes that $\alpha M = \beta$ (eqn 3b). That so general a relation should so closely predict the life histories of parasitic nematodes may point to some relatively simple facts governing nematode reproductive biology and to some very broad generalities underpinning the evolution of major life history traits.

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