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To delay once or twice: the effect of hypobiosis and free-living stages on the stability of host–parasite interactions

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The life cycle of many endoparasites can be delayed by free-living infective stages and a developmental arrestment in the host referred to as hypobiosis. We investigated the effects of hypobiosis and its interaction with delay in the free-living stages on host–parasite population dynamics by expanding a previous attempt by Dobson & Hudson. When the parasite life cycle does not include free-living stages, hypobiosis destabilizes the host–parasite interactions, irrespective of the assumptions about the regulation of the host population dynamics. Interestingly, the destabilizing effect varies in a nonlinear way with the duration of hypobiosis, the maximal effect being expected for three to five months delay. When the parasite life cycle involves free-living stages, hypobiosis of short or intermediate duration increases the destabilizing effect of the first time delay. However, hypobiosis of a duration of five months or more can stabilize interactions, irrespective of the regulation of the host population dynamics. Overall, we confirmed that hypobiosis is an unusual time delay as it can stabilize a two-way interaction. Contrary to the previous conclusions, such an atypical effect does not require self-regulation of the host population, but instead depends on the existence of free-living stages.

Keywords: host–parasite interaction; hypobiosis; free-living stages; stability; time delay

1. INTRODUCTION

Host–parasite interactions are of central importance to population dynamics with many implications for evolution (Poulin *et al.* 2000; Buckling & Rainey 2002; Moore & Wilson 2002; Forde *et al.* 2004; Nunn 2004) and public health (Anderson & May 1991; Dieckmann *et al.* 2002; Koella & Boete 2003), as well as biological control and conservation biology (Hawkins & Cornell 2000). Various mathematical models have been used to investigate the key question of the stability of these interactions. Most of the theoretical contributions are related to the seminal works of Anderson & May (1978) and May & Anderson (1978). These authors developed very influential models for host–parasite interactions. The success of this framework partly lies in the neutral stability of the initial model (model A in Anderson & May 1978). Accordingly, it has been used as a reference model into which more realistic features of host–parasite populations have been added to evaluate their stabilizing or destabilizing effects on the interaction (see reviews by Scott & Dobson 1989; Tompkins *et al.* 2002). Among those factors, the importance of time delay induced by free-living stages of the parasite has been previously addressed, since the time between egg production and larval stage transmission in the host can vary widely between parasite species. May & Anderson (1978) clearly demonstrated that such time delay has a destabilizing influence on the host–parasite interaction and, since then, this effect has been repeatedly described in other interactions between host and parasite (e.g. McGlade 1999; Kot 2001).

The life cycle of several endoparasites is characterized by another time delay, which is caused by developmental arrestment at the larval stage inside the host. This form of diapause, referred to as hypobiosis, may last up to six months (Michel 1974; Gibbs 1986) and has been documented in many nematode species (e.g. *Ostertagia* sp. parasites in reindeer, Irvine *et al.* (2000), and in cattle Chiejina *et al.* (1988); *Trichostrongylus* sp. in ruminants, Chiejina *et al.* (1988), and in

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red grouse, Hudson *et al.* (1985)). Surprisingly, little attention has been given to the consequences of such a time delay on the population dynamics of host and parasite. The only attempt was made by Dobson & Hudson (1992) for a specific host–parasite interaction between the nematode *Trichostrongylus tenuis* and its host the red grouse, *Lagopus lagopus scoticus*. They extended the basic framework of Anderson & May (1978) by including free-living stages and hypobiosis. The authors set up three models. The first, which includes free-living larvae but no hypobiosis, is a restatement of model F of Anderson & May (May & Anderson 1978). In the second model, all individuals enter hypobiosis and have free-living stages. The third model includes free-living stages and only a proportion of the parasites in the host enter hypobiosis. These models were considered with and without density-dependent regulation due to grouse territorial behaviour. From their analysis of the first two models, the authors concluded that hypobiosis had a destabilizing effect in the absence of self-regulation of the host, 'while it acts to stabilize the system, giving rise to cycles of longer period that tend to fade out more rapidly' when density dependence is included (p. 493). This last result apparently surprised the authors themselves who claimed that 'larval arrestment would seem to provide an interesting example of a time delay stabilizing a two-species interaction!', which indeed strongly contrasts with the usual effect of time delays on population dynamics. From the last model investigated, they finally concluded that 'as the proportion of larvae that enter a period of arrested development increases, the host and parasite populations exhibit more heavily damped cycles of longer frequency' (p. 495).

The main goal of Dobson & Hudson's (1992) study was to determine which features of the host–parasite interaction caused the long-term cycles observed between red grouse and their nematode parasite. Many other aspects of this specific interaction were thus investigated, but no systematic analysis of the importance of hypobiosis on the stability of the host–parasite relationship was provided with respect to the proportion of individuals entering such an arrested stage, the duration of this arrestment and its interaction with other stabilizing or destabilizing factors. Accordingly, the importance of developmental arrest inside the host is still claimed to be one of the major gaps in our understanding of host–parasite interactions from both a theoretical and an empirical point of view (Wilson *et al.* 2004).

This paper focuses on the impact of hypobiosis on host–parasite interaction in a more general and methodical way. In particular, we wish to (i) determine the importance of the proportion of larvae that enter hypobiosis and the duration of the arrestment and (ii) compare the effect of this time delay with the effect of the time delay due to free-living stages. We address these two points for parasite populations with different levels of aggregation and for host populations with and without (density-dependent) self-regulation. We account for the aggregation of parasites because it is the most widely recognized stabilizing factor in host–parasite relationships. We also investigate the influence of density dependence as Dobson & Hudson (1992) stated that hypobiosis is destabilizing in the absence of host self-regulation, while it is stabilizing when such regulation is included. The influence of other parameters of the model is also investigated through partial sensitivity analysis to provide a good understanding of the effect of the two time delays on the stability of host–parasite interactions in a broader set of ecological conditions.

2. MATERIAL AND METHODS

The basic framework of the Anderson & May (1978) model was modified to include hypobiosis. Free-living stages and self-regulation of the host population were then included independently and in combination leading to four models. In all models, hypobiosis is included and can be tuned by changing the proportion of larvae in hypobiosis. In the first two models (referred as models A and B), host population is regulated only by the parasites, whereas it is also self-regulated by a density-dependent process in the two other models (referred to as C and D). Models B and D differ from models A and C by the presence of free-living larval stages. Therefore, models A and C consist of three coupled differential equations describing changes in the number of hosts (H), the number of larvae in hypobiosis (A) and the number of adult parasites (P). An additional equation is included in models B and D to account for the variations in the number of free-living larvae (W). The notations used for the parameters included in these equations are reported in table 1. They correspond to the notations previously established by Anderson & May (1978), May & Anderson (1978) and Dobson & Hudson (1992).

Model A is described by the following set of equations, where (i) no time delay occurs between the production of free-living stages and their capacity to infect a host, (ii) the parasites are aggregated among hosts with a degree of aggregation $1/k$ and (iii) a proportion of larvae σ enter arrestment for a period of time equal to $1/\theta$. Model A with $\sigma=1$ is equivalent to model II in Dobson & Hudson (1992).

$$\frac{\mathrm{d}H}{\mathrm{d}t} = (a-b)H-(\alpha+\delta)P, \tag{2.1}$$

$$\frac{\mathrm{d}A}{\mathrm{d}t} = \sigma\frac{\lambda PH}{(H+H_0)}-(\mu_{\mathrm{A}}+b+\theta)A-\alpha\frac{PA}{H}, \tag{2.2}$$

$$\frac{\mathrm{d}P}{\mathrm{d}t} = \theta A+(1-\sigma)\frac{\lambda PH}{(H+H_0)}-(\mu_{\mathrm{P}}+b+\alpha)P -\alpha\frac{P^2}{H}\left(\frac{k+1}{k}\right). \tag{2.3}$$

When free-living stages are taken into account, a time delay is introduced between the production of free-living stages and the ability to infect a host. This leads to the definition of an additional equation that describes variation in the number of free-living stages. Model B then consists of four coupled differential equations. The equation for host population size is the same as in our first model (2.1). The three other

Table 1. Description and standard values of the parameters used in the models.

parameters	description	standard values	reference
a	instantaneous birth rate of the host (yr^{-1})	3	Anderson & May (1978)
b	instantaneous death rate of the host due to all causes except the parasite (yr^{-1})	1	Anderson & May (1978)
α	instantaneous death rate of the host due to the parasite ($worm^{-1}\,yr^{-1}$)	0.5	Anderson & May (1978)
δ	instantaneous reduction in the host fecundity due to the parasite ($worm^{-1}\,yr^{-1}$)	0	
Δ	density-dependent reduction in grouse fecundity and survival ($host^{-1}\,yr^{-1}$)	0.0025	Dobson & Hudson (1992)
λ	instantaneous birth rate of the parasite (yr^{-1})	20	Anderson & May (1978)
μ_i	instantaneous death rate of adult μ_P and of arrested larvae μ_A (yr^{-1})	$\mu_A=0.05$, $\mu_P=0.1$	Anderson & May (1978) and Dobson & Hudson (1992)
γ	instantaneous death rate of free-living stages (yr^{-1})	15	
β	instantaneous transmission rate of parasite infective stages ($host^{-1}\,yr^{-1}$)	1.5	
H_0	transmission efficiency constant ($=\gamma/\beta$)	10	Anderson & May (1978)
k	parameter of the negative binomial distribution that is inversely proportional to the degree of aggregation	2	Anderson & May (1978)
σ	proportion of larvae that undergo hypobiosis		
θ	rate at which arrested larvae develop into adult worms ($month^{-1}$)		Dobson & Hudson (1992)
d'	$\mu_P+\alpha+b+((k+1)/k)\times((\alpha(a-b))/(\alpha+\delta))$		May & Anderson (1978)
d''	$\mu_A+b+\theta+((\alpha(a-b))/(\alpha+\delta))$		Dobson & Hudson (1992)

equations are

$$\frac{dW}{dt}=\lambda P-\gamma W-\beta WH, \tag{2.4}$$

$$\frac{dA}{dt}=\sigma\beta WH-(\mu_A+b+\theta)A-\alpha\frac{AP}{H}, \tag{2.5}$$

$$\frac{dP}{dt}=\theta A+(1-\sigma)\beta WH-(\mu_P+b+\alpha)P -\alpha\frac{P^2}{H}\left(\frac{k+1}{k}\right). \tag{2.6}$$

The mortality rate of free-living stages is governed by the parameter γ and the transmission rate per host β. A proportion σ of transmitted free-living parasites enter hypobiosis while the rest of transmitted free-living parasites directly develops into adults. Model B corresponds to Dobson & Hudson's (1992) model III.

In both models A and B, the growth of the host population is thought to be exponential when not controlled by the parasite. In models C and D, host population is self-regulated by a density-dependent process. For the purpose of comparison, we accounted for exactly the same kind of self-regulation as Dobson & Hudson (1992), who proposed a mechanistic model of density-dependent reduction in the survival of the host. This model leads to a carrying capacity equal to $(a-b)/\Delta$, where Δ specifies the effect of resource limitation on host survival. Host population dynamics can be described by

$$\frac{dH}{dt}=(a-b-\Delta H)H-(\alpha+\delta)P. \tag{2.7}$$

The dynamical properties of these models have been investigated by finding their steady states and evaluating their local stability properties using the Routh–Hurwitz criterion (Edelstein-Keshet 1988; for more details refer to the stability analysis in the electronic supplementary material). Predicted time series shown in figure 1 were established using the package 'odesolve' of R (R Development Core Team 2005). While checking the stability criterion, we considered the values used by Anderson & May (1978) as 'standard values' of the parameters listed in table 1, in order to allow consistent comparisons and extension of their seminal contribution. We then varied in a systematic way the level of aggregation (k), the proportion of individuals entering hypobiosis (σ) and the duration of hypobiosis ($1/\theta$). The level of aggregation varied in the range of k values observed in host–parasite systems. According to a meta-review provided in the electronic supplementary material, estimates of k range from 0.5 to 24.58 with only one value over 20. We therefore focused on the range 0–20. The proportion of individuals entering hypobiosis was smoothly varied from 0 to 1, although only results for 0, 0.25, 0.5 and 1 are shown in figure 2. The duration of hypobiosis was finely tuned from zero to seven months, according to the few available data (Michel 1974; Cabaret 1977). A sensitivity analysis was also performed with respect to the other parameters of the models. The parameters (except k, σ and θ) were all varied over the range $\pm 50\%$. Conclusions about the influence of hypobiosis on the host–parasite population dynamics were deeply sensitive to three of these parameters: decrease in free-living stage mortality (γ); in host fecundity (a); and in parasite fecundity (λ). We provide a restrictive (although sufficient) description of these effects in figure 2 by displaying results while varying each of these parameters independently.

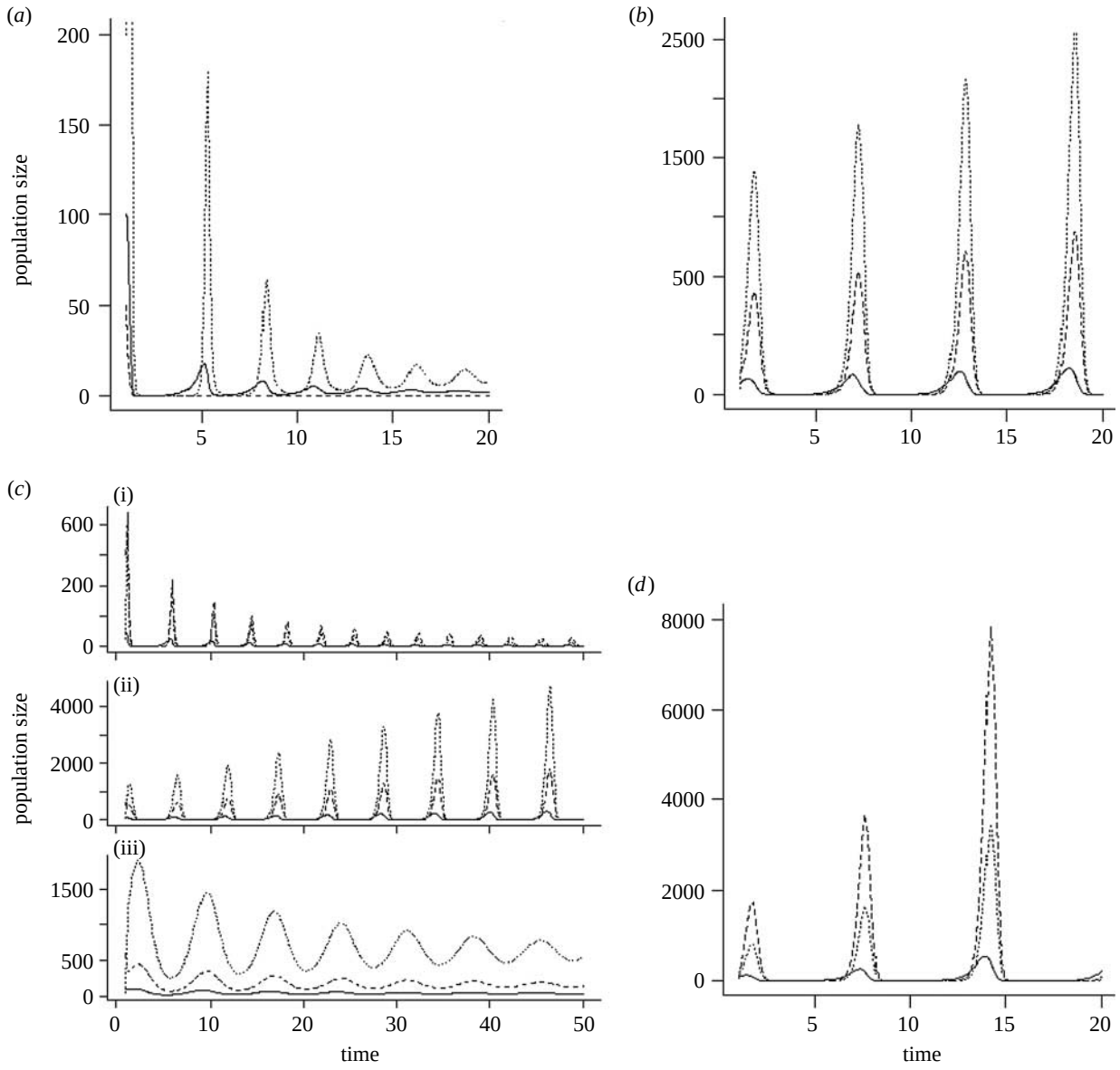

Figure 1. Dynamics of the host and the parasite populations with (*a*) no hypobiosis ($\sigma=0$) without free-living stages, (*b*) obligatory hypobiosis ($\sigma=1$) without free-living stages, (*c*) obligatory hypobiosis ($\sigma=1$) without free-living stages for different durations of hypobiosis ((i) 1 month, (ii) 5 months and (iii) 12 months) and (*d*) obligatory hypobiosis ($\sigma=1$) and free-living stages. The solid, dashed and dotted lines represent the changes in host abundance, in arrested larvae and in adult parasites, respectively. The parameter values are equivalent to those defined in table 1 except that $k=6$. In (*a*), host–parasite populations exhibit damped cycles whereas they exhibit diverging cycles in both (*b*,*d*); (*c*) illustrates the nonlinear effect of hypobiosis duration.

3. RESULTS

3.1. Effect of hypobiosis alone

The effect of hypobiosis alone was investigated by analysing model A in which there is no other time delay. The numbers of both adult parasites and arrested larvae at equilibrium directly depend on the host density at equilibrium

$$H^* = \frac{d'd''H_0}{\lambda\sigma\theta - d'd'' + \lambda d''(1-\sigma)}, \tag{3.1a}$$

$$P^* = \frac{(a-b)}{(\alpha+\delta)}H^*, \tag{3.1b}$$

$$A^* = \frac{\lambda\sigma}{d''}\frac{(a-b)}{(\alpha+\delta)}\frac{H^*}{H^*+H_0}H^*, \tag{3.1c}$$

where d' and d'' are compounded parameters defined in table 1. Both of them are positive when $a>b$. Then, for the three equilibria to be positive, it is required that

$$\text{(i)} \quad a>b \tag{3.2a}$$

and

$$\text{(ii)} \quad \lambda\sigma\theta + (1-\sigma)\lambda d'' > d'd''. \tag{3.2b}$$

The effect of hypobiosis on the stability of this equilibrium level can be seen in figure 2*a* by comparing the situation 'no hypobiosis' ($\sigma=0$) with others where the proportion of hypobiosis (σ) and its duration ($1/\theta$) varies.

In standard conditions (figure 2*a*(i)), hypobiosis has either no effect or a destabilizing effect, depending on the level of aggregation of the parasite. For high levels of aggregation ($k\leq0.5$, left part of figure 2*a*(i)), the

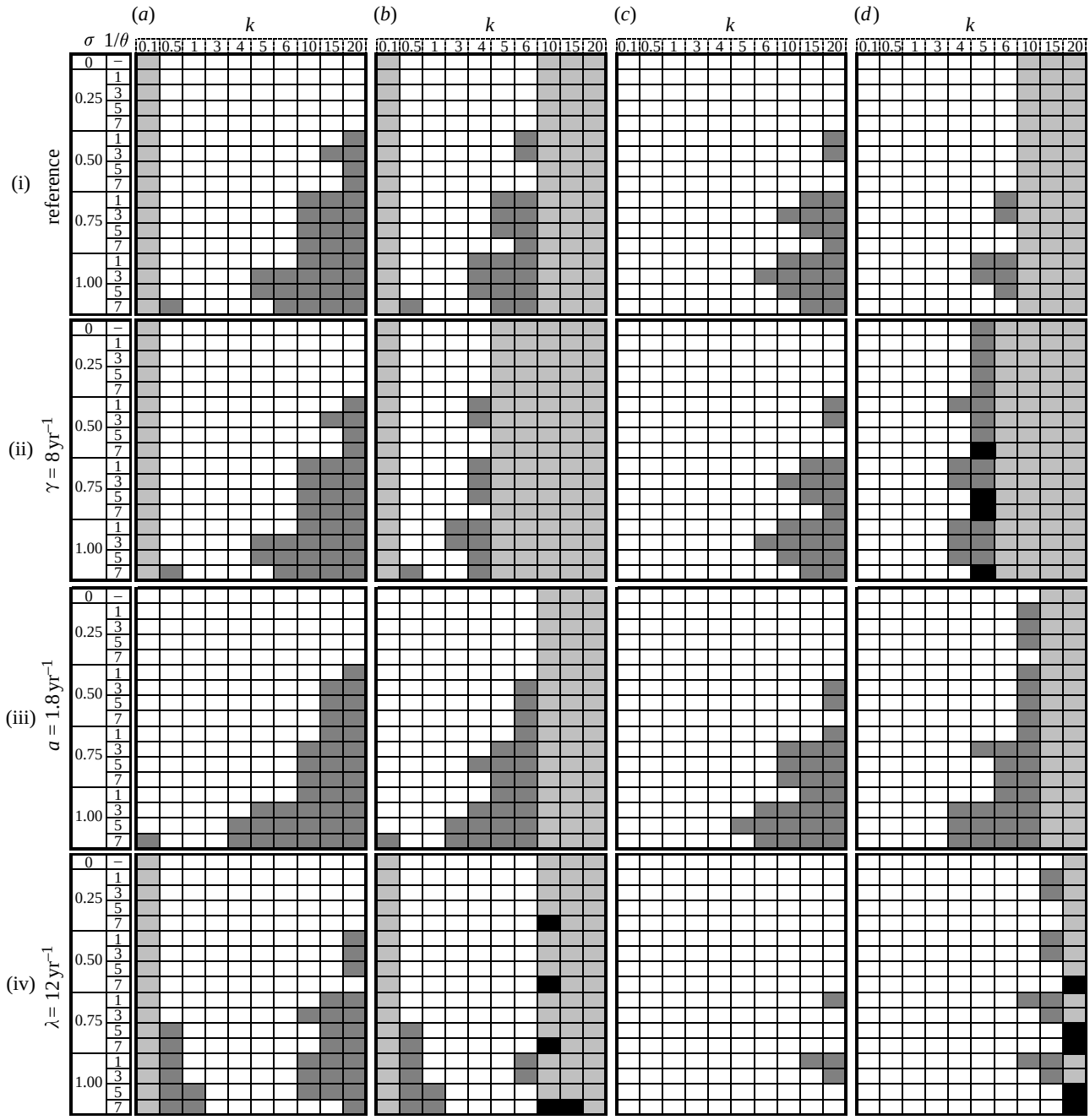

Figure 2. Stability properties of the four models according to the level of aggregation of the parasite ($1/k$), the proportion of larvae undergoing hypobiosis (σ) and the duration of hypobiosis ($1/\theta$). From left to right, we display the results for (a) model A (with hypobiosis without free-living stages), (b) model B (with hypobiosis and long-lived free-living stages), (c) model C (with hypobiosis, without free-living stages and density-dependent regulation of host population) and (d) model D (with hypobiosis, long-lived free-living stages and density-dependent regulation of host population). ((a,b) No self-regulation of host population; (c,d) with self-regulation of host population.) For (i) reference case, the parameter values are given in table 1 and the results of the sensitivity analysis are displayed for (ii) $\gamma = 8\ \mathrm{yr}^{-1}$, (iii) $a = 1.8\ \mathrm{yr}^{-1}$ and (iv) $\lambda = 12\ \mathrm{yr}^{-1}$. White and shaded areas denote the set of parameter values where the interaction is stable and unstable, respectively. Light grey areas indicate the instability induced by hypobiosis and dark grey areas indicate the instability non-induced by hypobiosis, since the interaction is unstable in the absence of this time delay. Black areas indicate the regions where host–parasite interaction is unstable in the absence of hypobiosis but stable with hypobiosis.

stability of host–parasite interaction does not vary (or very weakly varies) with either the proportion of larvae entering hypobiosis or the duration of hypobiosis. Only if all the individuals ($\sigma = 1$) enter a long period of arrestment ($1/\theta = 7$), the range of k values for which the dynamics are stable is slightly reduced. On the contrary, for low levels of aggregation ($k \geq 5$, right part of figure 2a(i)), hypobiosis has a destabilizing effect as soon as a proportion $\sigma \geq 0.5$ of larvae enter hypobiosis (lower right corner of figure 2a(i)). As shown in figure 1, diverging cycles then appear (figure 1b) instead of damped oscillations (figure 1a).

Interestingly, the duration of hypobiosis ($1/\theta$) has a nonlinear effect on the stability of the host–parasite interaction (figure 1c and figure 2a(i); for $k > 5$ and a fixed σ). From short to long arrestment duration, the

range of k values where the interaction is unstable can increase and then decrease (e.g. for $\sigma=0.5$ and 0.75). Accordingly, an increase in the duration of hypobiosis ($1/\theta$) can be either destabilizing (e.g. figure 2*a*(i), $\sigma=1$, $k=6$) or stabilizing (e.g. figure 2*a*(i), $\sigma=1$, $k=5$, when $1/\theta$ varies from three to seven months), although hypobiosis always has a destabilizing effect (comparing with the situation with no hypobiosis). The effect of the duration of hypobiosis is shown in figure 1*c* where an increase in the duration first turns a stable interaction ($1/\theta=1$ month) into diverging cycles ($1/\theta=5$ months), but then again into a stable interaction for longer delays ($1/\theta=12$ months).

Variations in free-living stage mortality (figure 2*a*(ii)) do not change the above results, since model A does not explicitly consider free-living stages. At high levels of aggregation, a decrease in host fecundity (figure 2*a*(iii)) does not change the weak destabilizing effect of hypobiosis (left part of figure 2*a*(iii) versus left part of figure 2*a*(i)), but increases it at low levels of aggregation (right part of figure 2*a*(iii) versus right part of figure 2*a*(i)). Conversely, a decrease in parasite fecundity strengthens the destabilizing effect of hypobiosis when aggregation is high (left part of figure 2*a*(iv) versus left part of figure 2*a*(i)), but lowers it at low aggregation levels (right part of figure 2*a*(iv) versus right part of figure 2*a*(i)). Overall, hypobiosis can destabilize host–parasite interaction when more than 50% ($\sigma\geq0.5$) of larvae enters hypobiosis and when k is either ≤1 or ≥4. This effect is modulated by the demography of the host and the parasite populations. The relationships between the duration of hypobiosis ($1/\theta$) and the range of k values where the interaction is unstable are also affected by demographic parameters leading to either an increase in the instability area (e.g. right part of figure 2*a*(iii) with $\sigma=1$) or a decrease (e.g. right part of figure 2*a*(iv) with $\sigma=1$).

To summarize, in the absence of density-dependent regulation of the host population and free-living stages in the parasite life cycle, hypobiosis always tends to destabilize the host–parasite dynamics at both low and high levels of aggregation. Interestingly, the duration of the arrestment either amplifies or lowers the destabilizing effect of hypobiosis depending on the biology of the host and the parasite populations.

3.2. Interaction between the two time delays

To analyse the interaction between the two time delays in the absence of regulation of the host population, we used our model B. The numbers of free-living stages, adult parasites and arrested larvae at equilibrium depend on the host density at equilibrium

$$H^* = \frac{d'd''\gamma}{\beta(\lambda\sigma\theta - d'd'' + \lambda d''(1-\sigma))}, \tag{3.3a}$$

$$P^* = \frac{(a-b)}{(\alpha+\delta)}H^*, \tag{3.3b}$$

$$W^* = \frac{\lambda}{(\beta H^*+\gamma)}\frac{(a-b)}{(\alpha+\delta)}H^*, \tag{3.3c}$$

$$A^* = \frac{\lambda\sigma}{d''}\frac{(a-b)}{(\alpha+\delta)}\frac{\beta H^*}{(\beta H^*+\gamma)}H^*. \tag{3.3d}$$

It is straightforward to show that conditions for all these equilibrium to be positive ($a>b$ and $H^*>0$) are the same as that for the similar equilibrium to be positive in model A.

We investigated the interaction between the two time delays by first considering only the effect of free-living stages (as previously done for hypobiosis) and then the effect of both time delays.

As previously established (May & Anderson 1978; Dobson & Hudson 1992), free-living stages have a destabilizing effect on the host–parasite interaction. This can be seen in figure 2 for each demographic condition (figure 2(i)–(iv)), by comparing the range of k values, where the interaction is unstable ($\sigma=0$), obtained for model A (figure 2*a*; without free-living stages) and model B (figure 2*b*; with free-living stages). The area of instability slightly changes at high levels of aggregation and is systematically increased at low levels. The effect of hypobiosis can be seen by comparing (figure 2*b*) the range of k values where the interaction is unstable ($\sigma=0$) with others in the same column where σ varies. Introduction of hypobiosis, while the parasite life cycle is already delayed by free-living stages, has either no effect or broadens the area of instability due to the first time delay. In standard conditions, the area is either unchanged ($\sigma=0.25$) or broadened ($\sigma\geq0.5$) for higher levels of aggregation (eventually up to $k\geq3$ instead of $k\geq10$ with no hypobiosis). These patterns remain unchanged when mortality of the free-living stages (figure 2*b*(ii)) or host fecundity (figure 2*b*(iii)) change. When parasite fecundity (figure 2*b*(iv)) decreases, the same conclusion holds as long as the duration of hypobiosis is shorter than seven months. Hypobiosis lasting for long periods ($1/\theta=7$ months) can stabilize the host–parasite population dynamics at low levels of aggregation ($k\geq10$; figure 2*b*(iv)). This is truly unexpected (although previously reported by Dobson & Hudson 1992), as time delays are broadly acknowledged as destabilizing factors of population dynamics and interactions (Renshaw 1991; Kot 2001). Such an unusual effect is observed for any proportion of larvae entering hypobiosis for lower parasite fecundity (figure 2*b*(iv)).

Thus, in the absence of density dependence and when the development is already delayed by free-living stages, hypobiosis almost always has a destabilizing effect. But in some demographic conditions, hypobiosis of long duration can potentially stabilize the interaction.

3.3. Effect of self-regulation of the host population

The effect of self-regulation of the host population was investigated by comparing results of models A and C, and results of models B and D. As expected, for both models C and D, in the absence of the parasite, the host population persists at a stable equilibrium level ($H^*=(a-b)/\Delta$). When the parasite is introduced, the equilibrium levels A^*, P^* and W^* are similar to

those of models A and B, except for H^* which is now a root of a third-degree polynomial (see electronic supplementary material).

As expected, self-regulation of the host population stabilizes host–parasite interaction, so that the white areas are overall much larger than those in models A and B. Without free-living stages, the conclusions remain globally unchanged, except for higher levels of aggregation, where hypobiosis has no more effect. In standard conditions, hypobiosis still has either no effect ($\sigma=0.25$) or a destabilizing one ($\sigma\geq0.5$) at low levels of aggregation ($k\geq6$ and usually $k\geq10$). Again, varying free-living stage mortality does not change the pattern (figure 2*c*(ii)) since model C does not explicitly include parasite free-living stages. As previously observed, a decrease in host fecundity (figure 2*a*(iii)) increases the destabilizing effect of hypobiosis at low levels of aggregation (right part of figure 2*c*(iii) versus right part of figure 2*c*(i)) whereas a decrease in parasite fecundity lowers it at similar levels of aggregation (right part of figure 2*c*(iv) versus right part of figure 2*c*(i)). Overall, hypobiosis can destabilize the interaction when a proportion $\sigma\geq0.5$ of larvae enter hypobiosis and when $k\geq5$ (usually $k\geq10$), depending on the demography of the host and the parasite populations. The relationship between the duration of hypobiosis ($1/\theta$) and the range of k values where the interaction is unstable is still affected by demographic parameters.

The effect of hypobiosis when free-living stages are taken into account is quite similar to that observed in the absence of host self-regulation. The range of k values where hypobiosis can have a destabilizing effect on the interaction (depending on the demographic parameters used) is smaller ($k\geq4$). As previously observed in the absence of density dependence, long durations of hypobiosis ($1/\theta=5$ or 7 months) have a stabilizing effect on the interaction in the presence of free-living stages. This now seems to be favoured not only by a decrease in parasite fecundity (figure 2*d*(iv)) but also by an increase in free-living stage mortality (figure 2*d*(ii)).

To summarize, when the host population is self-regulated, the effects of hypobiosis when free-living stages are included or not are similar to those observed in the absence of host self-regulation, although the (unusual) stabilizing effect of hypobiosis appears in a wider range of demographic parameters.

4. DISCUSSION

We investigated the effects of hypobiosis and its interaction with a developmental characteristic (absence or presence) of free-living stages on the host–parasite population dynamics, by expanding previous attempts by Anderson & May (1978) and Dobson & Hudson (1992). We will now discuss our results with respect to the previous conclusions (Dobson & Hudson 1992) concerning the effect of hypobiosis on host–parasite population dynamics. Finally, we will identify parasite species that are good candidates for the study of the effect of hypobiosis in the field.

4.1. When does hypobiosis have a stabilizing and a destabilizing effect?

First, we showed that hypobiosis has a destabilizing effect in most of the host and parasite demographic conditions considered here. As soon as $\sigma>0.5$, the destabilizing effect is observed both at high ($k\leq1$) and intermediate or low ($k\geq3$) levels of aggregation in the absence of self-regulation of the host population and at low ($k\geq5$) levels of aggregation with such regulation. This is consistent with the common wisdom that time delays have destabilizing effects on population dynamics (e.g. May 1981). Furthermore, according to the available estimates of σ (Michel 1974; Cabaret 1977) and to our review of estimated values of k (see electronic supplementary material), the theoretical prediction concerns a realistic range of parameters and so deserves to be tested on specific host–parasite systems (see §4.3).

The second important finding is that, as previously reported by Dobson & Hudson (1992), hypobiosis of long duration ($1/\theta\geq5$ months) can also have a stabilizing effect at low levels of aggregation ($k\geq10$ with no host self-regulation and $k\geq5$ with host self-regulation). However, we do not confirm that hypobiosis has a destabilizing effect in the absence of self-regulation of the host, 'while it acts to stabilize the system, giving rise to cycles of longer period that tend to fade out more rapidly' when density dependence is included (p. 493). Hypobiosis can also stabilize the interaction in the absence of self-regulation in host population dynamics, even in a more restricted set of demographic conditions. Instead, we conclude that hypobiosis can stabilize the interaction induced by the presence of free-living stages and provide an explanation for this result by explaining the effect of the duration of hypobiosis (see §4.2).

The discrepancy between our conclusions and those of Dobson & Hudson (1992) probably arises from a confounding effect. As far as we understand, they actually compared the dynamics of a model with no hypobiosis (but with free-living stages, our model B or D with $\sigma=0$) with the dynamics of a model where all parasites undergo hypobiosis (but without free-living stages, our model A or C with $\sigma=1$). Unfortunately, free-living stages have a destabilizing effect. Thus, comparisons between the results coming from these two models do not allow us to disentangle whether the overall stabilizing effect is due to hypobiosis or the removal of a destabilizing factor. Figure 2 can show this misunderstanding: hypobiosis appears to be destabilizing in the absence of self-regulation (figure 2*b*(i) with $\sigma=0$ versus figure 2*a*(i) with $\sigma=1$) and stabilizing with host self-regulation (figure 2*d*(i) with $\sigma=0$ versus figure 2*c*(i) with $\sigma=1$).

4.2. Effect of the duration of hypobiosis

Dobson & Hudson (1992, 1994) concluded that an increase in the duration of hypobiosis results in host and parasite cycles with longer period. These theoretical predictions are backed up by the field studies of Haydon *et al.* (2002), who showed that red grouse

exhibit cycles with periods ranging from 4 years in southern parts of Britain to 12 years in northern parts when the duration of parasite arrestment is longer. Our own results indicate that an increase in the duration of hypobiosis not only changes the length of the host–parasite population cycles, but can also modify the stability properties of those cycles (by transforming damped cycles into diverging ones or diverging cycles into damped ones). Interestingly, the effect of hypobiosis on the stability of a host–parasite interaction can vary in a nonlinear way with the duration of hypobiosis and a maximal destabilizing effect can be observed at intermediate durations of arrestment. Accordingly, to increase the duration of hypobiosis, when this delay is already long, tends to stabilize the interaction. Without free-living stages, the stabilizing effect is not strong enough to observe a more stable dynamic in populations with hypobiosis than in those without hypobiosis. Hypobiosis is then always destabilizing. On the contrary, with free-living stages, hypobiosis amplifies the first delay up to a point where it eventually becomes stabilizing. The stabilizing effect of an increase in the duration of hypobiosis can be understood as follows.

As stated by Dobson & Hudson (1994), an increase in the duration of hypobiosis increases the time lag on virulence (changes in host mortality and fecundity due to the parasite). Accordingly, parasitized hosts live longer when the infective larvae enter a period of non-pathogenic arrestment before developing into pathogenic adults. This leads to a larger increase in the host population size and has been shown to result in host–parasite cycles of longer amplitude. However, increasing the duration of hypobiosis also enlarges the time period when parasites are released into the environment leading to a more regular output of parasites. This induces cycles of smaller amplitude and acts as a buffer effect to stabilize the host–parasite interaction. When the duration of arrestment exceeds a threshold value, the buffer effect tends to lower the destabilizing effect of hypobiosis. On the contrary, free-living stages can contribute to buffering the effect of hypobiosis which then may stabilize host–parasite interaction.

In this paper, we also bring new insights into the relationship between the duration of hypobiosis and the stability of the host–parasite interaction, which will be investigated in the field only with difficulty. Depending on the host and parasite demography, all or only a part of the nonlinear relationship is revealed in a feasible range of hypobiosis durations. Results shown in figure 2 clearly show that only small changes in parasite aggregation can turn the non-monotonic relationship into a monotonic (stabilizing or a destabilizing) one. Even if enough genetic variability exists to sample over the entire probable range of hypobiosis duration, one then also needs a good understanding of both host and parasite demography to be able to specify the theoretical expectation to be tested. Likewise, in the absence of a good knowledge of demography, one may find contrasting and puzzling results even for biologically similar systems.

4.3. Hypobiosis and the dynamics of natural populations

Regulation of host populations by parasites has been supported by empirical studies in red grouse *L. l. scoticus* (Hudson *et al.* 1998), Soay sheep *Ovis ovis* (Gulland 1992), Svalbard reindeer *Rangifer tarandus plathyrynchus* (Albon *et al.* 2002; Stien *et al.* 2002) and recently in mountain hares *Lepus timidus* (Newey *et al.* 2005). Although all the parasites infecting these host species undergo hypobiosis, the ecological and evolutionary consequences of hypobiosis have never been investigated either in laboratory experiments or in the field. Clearly, this lack of experimental and field data can partly be explained by the difficulties in determining the effect of parasites in complex ecological systems with many potentially confounding factors (Albon *et al.* 2002). In such conditions, one expects theory to help decipher whether different biological systems are potentially suitable for the investigation of these difficult issues.

Therefore, testing our theoretical predictions required a deep understanding of the biological system. Leaving apart the effect of the duration of hypobiosis, only a few specific host–parasite systems have the required properties to test our theoretical predictions of the effect of hypobiosis on the stability of host–parasite interactions.

Dobson & Hudson (1992) and Hudson *et al.* (1998) showed that the influence of the nematode parasite *T. tenuis* was necessary and sufficient to produce the observed cycles in the red grouse populations. Decreases in host fecundity mediated by the parasite are thought to be the main cause of these cycles. Accordingly, the potential effect of hypobiosis, related in the first paper, was not even evoked in the later one (Hudson *et al.* 1998). Our own results confirm that hypobiosis should not have an important impact on the *T. tenuis*–red grouse interaction as we observed that for a level of aggregation as strong as that of *T. tenuis* ($k=1$, Dobson & Hudson 1992) hypobiosis is expected to have no effect on the host population dynamics, irrespective of the proportion of arrested larvae (σ) or the duration of arrestment ($1/\theta$).

Like the red grouse populations, Soay sheep on the island of Hirta undergo population crashes. The parasitic nematode *Teladorsagia circumcincta* is thought to play a role in these crashes (Tompkins & Begon 1999), even if it does not induce any reduction in host fecundity (Wilson *et al.* 2004). Estimated values of k for *T. circumcincta* vary widely from less than 1 to more than 12 (Gaba *et al.* 2005). In those conditions (especially when $k>6$), we have shown that hypobiosis has a strong destabilizing effect turning damped cycles into diverging ones (if the percentage of individuals entering hypobiosis exceeds 60%) or increasing the amplitude of damped cycles (if the percentage of individuals is lower than 60%). Depending on the environmental conditions, i.e. temperature, experienced by infected larvae, 10–70% of these parasites arrest their development (Cabaret 1977). Moreover, estimates of the fecundity of *T. circumcincta* are close to the lower value of parasite fecundity explored in this

paper (Cabaret & Ouhelli 1984). Therefore, empirical evidence for the influence of hypobiosis in natural populations could possibly be found in the Soay sheep populations, since qualitative changes are expected in host–parasite population dynamics within the range of observed values of parasite aggregation and percentage of individuals entering hypobiosis.

Estimates of k for *Ostertagia gruehneri* (parasites of reindeer) are also variable, ranging from 3.5 to 10.4 (Irvine *et al.* 2000), which suggests that hypobiosis could also play a role in determining host–parasite population dynamics in this system. However, no estimates of the proportion of individuals entering hypobiosis are currently available, which does not allow us to anticipate how strong the destabilizing effect could be.

To conclude, we have shown that hypobiosis is an original time delay which can have either a destabilizing or a stabilizing effect on host–parasite interactions within realistic parameter ranges. Given this specificity and the current scarcity of data on this process, it would be worthwhile to devote more field studies on acquiring information to test these theoretical predictions.

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