Available online at www.sciencedirect.com
SCIENCE DIRECT®
Theoretical Population Biology 65 (2004) 193–203
Theoretical Population Biology
http://www.elsevier.com/locate/ytpbi

Prolonged diapause and the stability of host–parasitoid interactions

J.C. Corley,[a,*] A.F. Capurro,[b] and C. Bernstein[c]

[a] Laboratorio de Ecología de Insectos, INTA EEA Bariloche, CC 277, (8400) SC de Bariloche Río Negro Bariloche, Argentina
[b] Departamento de Investigaciones, Universidad de Belgrano, Zabala 1851, (1426) Buenos Aires, Argentina and Programa de Ecología Matemática, Universidad Nacional de Luján, Rutas 5 y 7, Luján, Argentina
[c] Biometrie et Ecologie Evolutive, UMR 5558, Université Claude Bernard Lyon 1, 43 Bd. du 11 Novembre 1918, 69622 Villeurbanne Cedex, France

Received 13 September 2003; received in revised form 22 September 2003; accepted 28 September 2003

Abstract

We investigated the effect on host–parasitoid dynamics of prolonged diapause, a feature of the life history of many animals living in unpredictable environments, by modifying the classical May (J. Anim. Ecol. 47 (1978) 833) host–parasitoid model. We considered three patterns of development of host and parasitoid: (a) prolonged parasitoid diapause controlled by host physiology, (b) parasitoid interference in host development, preventing parasitized hosts from prolonging diapause, and (c) host diapause independent of parasitoid attack. We found that single-year prolonged diapause shifted the boundaries of the May model towards a slight increase in stability. Longer periods of diapause prolongation had a stronger influence, but this influence remained modest if we considered realistic parameter values. In contrast to other recent studies, our results suggest that prolonged diapause does not necessarily compensate for the destabilizing effects of time lags on the influence of parasitoids on population dynamics.

Keywords: Prolonged diapause; Host–parasitoid models; Host–parasitoid dynamics; Population persistence; Stochastic environments

1. Introduction

Diapause, the break in morphogenesis not directly controlled by local environmental factors, is a life history feature of many insects which need overcome stressful periods (Tauber et al., 1986; Danks, 1987). For many univoltine species living in seasonal environments, diapause may simply involve the cessation of growth during a given predictable season (winter, for instance). Diapauses of this type are described as simple diapauses. For many other species, inhabiting more unpredictable environments, some of the population may extend diapause for 1 year or more, resulting in prolonged, extended or extra long diapause (Tauber et al., 1986; Danks, 1987; Turgeon et al., 1994).

Prolonged diapause makes it possible for the emergence of a given genotype to be spread out over time, making it possible for some progeny to grow and reproduce in potentially better conditions (bet-hedging, Philippi and Seger, 1989; Hanski and Stahls, 1990; Hopper, 1999; Menu et al., 2000). Unlike dormant seeds, which may remain in a quiescent state for many years until appropriate conditions occur, most insect populations displaying prolonged diapause emerge within the first 2 or 3 years (Danks, 1987; Hanski, 1988). This difference is probably due to the metabolic costs (Storey and Storey, 1986; Zhou et al., 1995; Ishihara and Shmida, 1995; Han and Bauce, 1998; Irwin and Lee, 2000; Ellers and van Alphen, 2002) and mortality risks (Irwin and Lee, 2000) that diapause entails.

The consequences of prolonged diapause for insect population dynamics have been studied (Menu, 1993; Menu et al., 2000). The key issue in these previous studies was the relationship between prolonged diapause and environmental stochasticity (Menu et al., 2000). Efforts were therefore focused on the study of single populations.

However, prolonged diapause may also have major consequences for the dynamics of interacting populations (Hanski, 1988; Hanski and Stahls, 1990; Ringel et al., 1998). Host–parasitoid systems are among the most frequently studied examples of interactions between populations, partly because parasitoids are important components of natural ecosystems, but also

*Corresponding author. Fax: 54-944-424875.
E-mail address: jcorley@bariloche.inta.gov.ar (J.C. Corley).

0040-5809/$ - see front matter © 2003 Elsevier Inc. All rights reserved.
doi:10.1016/j.tpb.2003.09.005

because they are frequently used in the biological control of insect pests (Godfray, 1994). Many insect pests, including those that eat the seeds and cones of coniferous trees, display prolonged diapause (Annila, 1982; Turgeon et al., 1994).

Only one study has dealt with prolonged diapause in a context of host–parasitoid interactions (Ringel et al., 1998). It suggested that prolonged diapause strongly increases the stability of the interaction. Ringel et al. (1998) assumed that the probability of an individual remaining in diapause is constant and therefore independent of the time already spent in diapause. Thus, total duration of diapause is geometrically distributed for the population as a whole. Although there are documented cases in which this assumption holds true, the duration of prolonged diapause is less than two years in most species (Danks, 1987; Ringel et al., 1998; Hanski, 1988; Menu et al., 2000). Ringel et al. (1998) made two further major assumptions: (1) The host population remaining in diapause is inaccessible to foraging parasitoids. Diapausing hosts may remain in a physical refuge or the period of susceptibility to parasitism and parasitoid activity may be temporally displaced. Although there is some evidence that prolonged diapause may be associated with a physical refuge for the host (Price and Tripp, 1972) in specific cases, this has not been shown to be common; (2) Parasitoid diapause was assumed to be independent of host physiology. Parasitoids undergo diapause like any other insect (Tauber et al., 1983, 1986), but host cues may play an important role in diapause induction and development (Askew, 1971; Beckage, 1985; Lawrence, 1986). In many cases, particularly for parasitoids of egg and early larval stages, host physiology plays a central role in determining parasitoid diapause (Tauber et al., 1986). However, in other cases, hosts are rapidly consumed, rendering host diapause irrelevant to the parasitoid. Indeed, the relationships between host development, parasitoid development and the timing of parasitoid attacks are probably as diverse as parasitoid life history (see for example Tauber et al., 1983; Beckage, 1985).

Parasitoid development generally follows one of two patterns (Lawrence, 1986). Some parasitoids remain in step with host development. They have no effect on host physiology but are instead affected by host hormones. Thus, when the host enters prolonged diapause, so does the parasitoid. Parasitoids of this type are known as 'conformers' (for examples, see Beckage, 1985). Others interrupt or interfere with host development, adapting it to their own requirements, and are known as 'regulators' (Lawrence, 1986). Regulators prevent the hosts they attack from entering prolonged diapause. Only hosts that successfully escape parasitoid attacks are able to extend their diapause.

In this work, we explore, from a theoretical point of view, the effect of prolonged diapause on the stability of host–parasitoid systems, using realistic host–parasitoid physiological relationships, diapause spans and parameter values. We considered three contrasting situations. Our analysis of these situations showed that prolonged diapause moderately increases population stability. We also found that some of the ecological details of the interaction, such as the duration, in years, of prolonged diapause and the proportion of animals in such state, affect the stability of host–parasitoid systems.

2. Models for host–parasitoid relationships with prolonged diapause

For the most part, we consider here the simplest condition of extended diapause: diapausing insects delaying their development for 1 extra year. This pattern has been observed for almost half the insect species that undergo prolonged diapause (Hanski, 1988). However, later in the paper, we relax this assumption, and consider an additional year of extended host diapause (thus accounting for an additional 20% of known cases) and speculate on the possible effects of a longer diapause.

In our models, we consider three different cases, taking into account some of the most important biological aspects of host–parasitoid interactions in which prolonged diapause occurs. Firstly, we consider systems in which the parasitoids are physiological "conformers," this is that the populations display phenological synchronicity and all hosts are susceptible to parasitoid attack. Secondly, we model systems in which the parasitoids are physiological "regulators". In this case, the parasitoids can potentially attack all hosts, but do not undergo prolonged diapause themselves. Finally, we develop a third model, in which prolonged host diapause occurs at some stage before parasitoid attack. Diapause does not constitute a refuge from parasitoid attack in any of these models. Detailed descriptions of prolonged parasitoid diapause, the interaction between host diapause and parasitoid phenology and parasitoid attack are scarce (but see Beckage, 1985; Hanski, 1988).

We first assessed the stability of the models close to equilibrium densities. This was done by a combination of analytical and numerical techniques (see Appendix A). These results were complemented with simulations of the dynamics of the various systems.

2.1. Modeling framework

Our modeling framework was based on the classical Nicholson and Bailey (1935) model, as modified by May (1978) to take into account the possible aggregation of parasitoid attacks. The Nicholson-Bailey model

considers discrete generations and calculates the number of hosts and parasitoids at generation t (Nt and Pt, respectively) from their numbers in the previous generation ($t-1$), host reproduction rate (λ) and the fraction of hosts escaping parasitism ($f(Pt)$). Nt is best considered as the number of host larvae susceptible to parasitoid attack and Pt as the number of adult parasitoids searching for hosts. The model is

$$N_t = \lambda N_{t-1} f(P_{t-1}),$$
$$P_t = cN_{t-1}(1-f(P_{t-1})), \quad (1)$$

where c is the adult parasitoid yield from each parasitized host. As we modeled solitary parasitoids and as a means of simplifying calculations, we considered c to be equal to unity.

Nicholson and Bailey (1935) assumed that the occurrence of parasitoid attacks is random, following a Poisson distribution. This model is unstable, leading to expanding oscillations. May (1978) assumed instead that various processes may lead to the aggregation of parasitoid attacks, and adopted a negative binomial distribution to model this process. Specifically, $f(Pt)$ takes the form

$$f(P_t) = (1 + aP_t/k)^{-k}, \quad (2)$$

where a is the "searching efficiency" of parasitoids, also known as the "area of discovery" (Nicholson and Bailey, 1935) and k reflects the degree of parasitoid attack aggregation. Aggregation increases with decreasing k. For high values ($k \to \infty$), the distribution of attacks tends towards the original Poisson distribution assumed by Nicholson and Bailey (1935). May's (1978) model gives a locally stable equilibrium when $k < 1$.

We considered three models, defined according to the timing of parasitoid attacks—before or after the hosts enter prolonged diapause—and whether these attacks interrupt host development or conform to the extended diapause pattern.

2.2. "Conformer" parasitoids

In this model, we consider systems in which the prolonged host and parasitoid diapause are synchronic. For instance, host diapause occurs at an early stage (e.g. larvae) and parasitoid attack is limited to some stage before the host diapause occurs (e.g. eggs). Parasitoids (koinobionts) also enter prolonged diapause, conforming to host physiology. The conformer parasitoid model is:

$$N_t = (1-\alpha)\lambda N_{t-1} f(P_{t-1}) + \alpha\sigma\lambda N_{t-2} f(P_{t-2}),$$
$$P_t = (1-\alpha)N_{t-1}[1-f(P_{t-1})] + \alpha\sigma N_{t-2}[1-f(P_{t-2})]. \quad (3)$$

For both hosts and parasitoids, most of the individuals emerging from diapause at generation t originate from eggs laid at generation $t-1$. The rest originate from eggs laid in the previous generation ($t-2$), with prolonged diapause occurring at the larval stage. The first term on the right-hand side (r.h.s.) of both equations corresponds to individuals that did not undergo prolonged diapause (proportion $1-\alpha$). The second term corresponds to individuals that emerged after 2 years in diapause (proportion α), with a survival rate of σ (see also Ringel et al., 1998). As this model assumes that parasitoids physiologically adjust to host development, we considered both species to be affected by the same parameters concerning prolonged diapause (α and σ).

The frequency of parasitoid-host encounters is determined by $f(Pt)$, as in Eq. (2). As a first approximation, we considered only density-independent mortality factors that affect healthy and parasitized hosts similarly (but see Bernstein, 1986; Bernstein et al., 2002). As in previous models, we assume that survival through extended diapause is density-independent, for both hosts and parasitoids (Ringel et al., 1998). This framework does not imply that prolonged diapause provides the hosts with a refuge from parasitism and assumes that all adults will emerge 1 or 2 years after oviposition. The frequency of prolonged diapause is not simulated as following a particular distribution.

Fig. 1a (dotted line) shows the dynamics of this model as a function of the degree of parasitoid attack aggregation (k) and the fraction remaining in diapause (α) for a given set of parameter values. Intermediate proportions of individuals entering extended diapause result in a slight increase in stability and, as expected, very low and very high proportions tend to collapse the model to the stability conditions derived by May (1978). When $\alpha = 0$, all individuals emerge in the first year and the term corresponding to the fraction of individuals in prolonged diapause, disappears (see Eq. (3)). When $\alpha = 1$, all hosts and parasitoids undergo prolonged diapause and again, the system coincides with May (1978) model. The only difference is that each generation now takes 2 years.

2.3. "Regulator" parasitoids

In this model, we considered systems in which the parasitoids are physiological "regulators" (Lawrence, 1986). In such systems, host diapause may occur at a larval stage, for example, and parasitoid attack is limited to some stage before the initiation of host diapause (e.g. eggs). In contrast with the first model, the parasitoids do not undergo prolonged diapause and kill the attacked hosts before they are able to complete diapause (i.e., idiobionts).

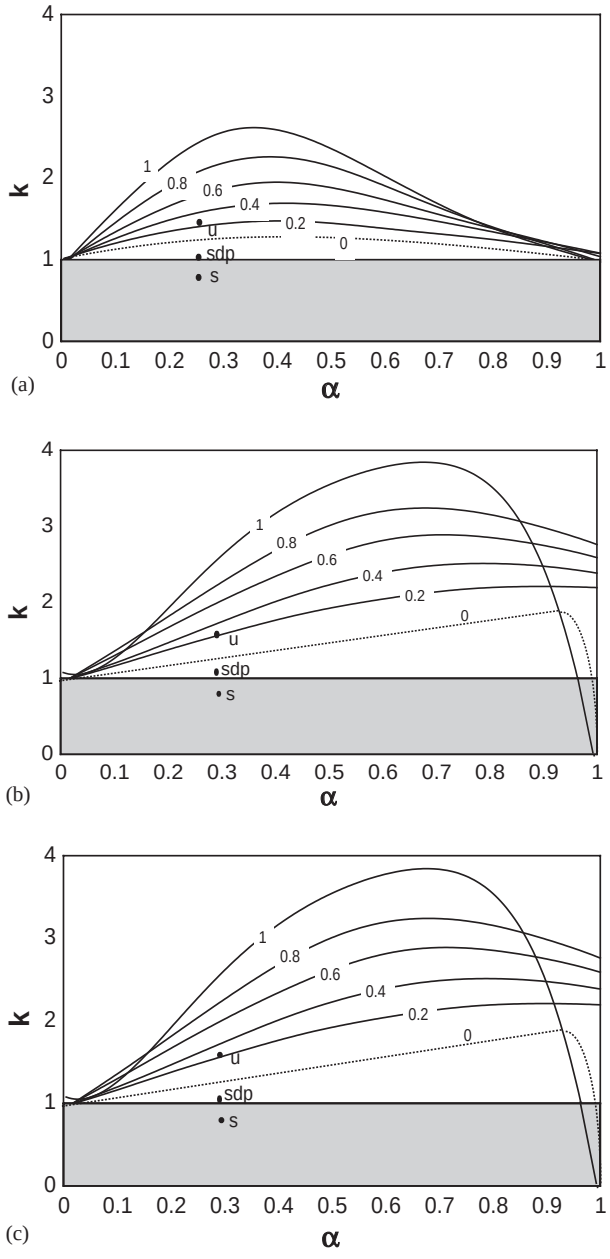

Fig. 1. Stability boundaries for three host–parasitoid models with prolonged diapause, in terms of α, the proportion of individuals in the population undergoing prolonged diapause and k, the coefficient defining the aggregation of parasitoid attacks. The life cycles considered are: (a) parasitoids conforming to host physiology, with both species undergoing diapause (Eqs. (4) and (B.1), see main text); (b) parasitoids regulating host physiology, with only hosts undergoing diapause (Eqs. (5) and (B.2)) and (c) host diapause independent of parasitoid attack (Eqs. (6) and (B.3)).The dotted line represents the case for a single-year prolonged diapause, whereas the various unbroken curves correspond to extension of the diapause for a second year, β (values are shown above the lines). The systems are stable below the curves. The area below the line at $k = 1$ corresponds to the stability boundaries for the May (1978) model, the equivalent model without prolonged diapause. Other values: $\sigma = 0.8$; $a = 0.008$ and $\lambda = 2$. Black squares with the legend *u* (for unstable), *s* (for stable) and *sdp* (stable through prolonged diapause) indicate the parameter values with which the simulations in a stochastic environment were performed (see Table 1).

The equations for the model are:

$$N_t = (1-\alpha)\lambda N_{t-1}(1 + aP_{t-1}/k)^{-k} + \alpha\sigma\lambda N_{t-2}(1 + aP_{t-2}/k)^{-k},$$
$$P_t = N_{t-1}\{1 - (1 + aP_{t-1}/k)^{-k}\}. \quad (4)$$

Note that the host equation for this model is the same as that in Eq. (3). The difference between the two models lies in the parasitoid equation, which takes into account only those hosts attacked during the previous generation.

Fig. 1b (dotted line) shows the stability boundaries for the regulator parasitoid model (Eq. (4)) for a given set of parameter values. Local stability is slightly higher than that for the May (1978) model, only for intermediate values of the fraction of the host population in prolonged diapause.

The asymmetry observed in Fig. 1b results from differences in survival between hosts that prolong diapause and those that do not. It should be borne in mind that hosts undergoing diapause for longer periods of time are subject to an additional risk of death not mediated by the parasitoid (represented by the survival rate, σ).

Fig. 1b shows the stability boundaries for a high host survival rate ($\sigma = 0.8$). The decrease in stability observed for extremely high fractions of hosts prolonging diapause (high α) was also observed in conditions in which the survival rate was low. In such cases, the host population suffers the burden of the additional mortality and the proportion of hosts prolonging diapause contributing to the overall population is small; the effects of prolonged diapause therefore become negligible. Conversely, if both survival rate and α are high, most hosts prolong their diapause (and survive diapause). In this case, most hosts will reproduce 2 years after hatching (at $\alpha = 1$ the host life cycle will always take two years). The parasitoid generation span is assumed to be 1 year. In these conditions, every 2 years, parasitoids will encounter too few hosts to reproduce. Consequently, in the next generation, hosts escape parasitoid attack and the host population grows exponentially. However, we did not consider the more complex situation in which two asynchronous cohorts of hosts are attacked by a univoltine parasitoid. This model gives results very different to those obtained with the conformer parasitoid model. In the latter case, the parasitoids closely follow their host population and are "carried over" by the attacked hosts undergoing prolonged diapause, as the two populations are tightly synchronized. Both populations are also equally affected by mortality during diapause.

2.4. Parasitoid phenology is independent of host diapause

In the third model, we consider systems in which parasitoid phenology is totally independent of prolonged

host diapause. An example would be a system in which the hosts enter and leave diapause at a stage (e.g. larvae) prior to that at which they are susceptible to parasitoid attack (e.g. pupae or adults). In this case, parasitoids do not enter prolonged diapause. This model therefore differs from Eqs. (3) and (4) in that the searching parasitoids of a given generation attack hosts of two different generations. In contrast to what is found with the regulator parasitoid model (Eq. (4), all surviving hosts can be attacked as the "window" for parasitoid attack occurs after host diapause.

The equations for this model are:

$$N_t = \{(1-\alpha)\lambda N_{t-1} + \sigma\alpha\lambda N_{t-2}\}\{(1+aP_{t-1}/k)^{-k}\},$$
$$P_t = \{(1-\alpha)N_{t-1} + \sigma\alpha N_{t-2}\}\{1-(1+aP_{t-1}/k)^{-k}\}. \quad (5)$$

The stability boundaries for this model are shown in Fig. 1c (dotted line). The results of this model are similar to those obtained with the regulator model: stability is higher than that for the May model, with an increase in α. Conversely, for extreme values ($\alpha > 0.96$) of the fraction of the population in prolonged diapause, the system becomes locally unstable, even for $k < 1$. Again, dynamic behavior should not be the same for the extreme values of α and $(1-\alpha)$, due to differences in survival.

2.5. Population persistence in stochastic environments

Previous studies have suggested that prolonged diapause may make a major contribution to population persistence in stochastic environments (Menu, 1993; Menu et al., 2000). We checked whether this holds true for host–parasitoid interactions modeled in terms of difference equations by performing a series of simulations in which we introduced continuous perturbations into our models. We assumed one year of prolonged diapause, intermediate α values, and a gamma distribution for the random variables net rate of increase (λ) and mortality rate of diapausing individuals ($\mu, \mu = 1-\sigma$). In our simulations, we fixed mean values of λ and μ and expressed the extent of disturbance as a coefficient of variation (CV), taking values from 0.1 to 0.5. Random values were generated as $x = x^*\beta(\alpha,1)/\alpha$ where x is a random deviate, x^* the chosen mean, and $\beta(\alpha,1)$ are deviates of a standard gamma distribution of integer order α with $\alpha = 1/CV^2$ (Press et al., 1992).

For each model and set of parameter values, we ran 1000 simulations, each covering 5000 generations, and estimated the extinction frequency (proportion of runs in which the system collapsed before the end of the simulation) and the mean time to extinction. Simulations were run for values of k giving stable ($k = 0.8$) or unstable ($k = 1.5$) models or that gave systems in which prolonged diapause was predicted to result in an increase in stability ($k = 1.08$). The parameter values are shown in Fig. 1, as *s*, *u* and *spd*, according to their effects on stability.

The consequence of random perturbations in both λ and μ are shown in Table 1. For the three prolonged diapause models, extinction probabilities and mean times to extinction were similar. In general terms, the three models behaved as predicted by their deterministic counterparts. With parameter values for which the deterministic models are stable ($k = 0.8$ and $k = 1.08$), and for moderated coefficients of variation ($CV < 0.4$), with rare exceptions, the three models persisted for at least 5000 generations. With $k = 1.08$, all models were more susceptible to perturbation and often led to extinction if CV was greater than 0.4. For $k = 1.5$, a value for which all deterministic models are unstable, none of the simulations persisted over 5000 generations. These results suggest that, in a stochastic environment, one year of prolonged diapause makes a limited contribution to the persistence of host–parasitoid systems.

2.6. The influence of 2 years of prolonged diapause

We modified our three models to incorporate a second year of extended diapause. This involved adding extra terms similar to those on the r.h.s. of Eqs. (3)–(5) and an extra parameter, β, corresponding to the fraction of diapausing individuals that remain in diapause for an additional year (see Appendix B, Eqs. (B.1)–(B.3)). Fig. 1 shows the stability boundaries for the three models for different β values. The curve with $\beta = 0$ corresponds the situation in which diapause is prolonged by only one year (i.e., Eqs. (3)–(5)).

For the three models and for intermediate α and β values, an extra year of prolonged diapause extends the stability boundaries. For instance, for the conformer parasitoid model (Eq. (B.1)), for intermediate and realistic α and β values ($\alpha = \beta = 0.4$, see below), stability boundaries are extended up to values of $k \approx 1.4$. For extreme α values, an additional year in prolonged diapause affects neither stability nor the qualitative behavior of the model.

The qualitative behavior of the regulator and independent phenology models (Eq. (B.2) and (B.3), respectively), for low and moderate α values, is similar to that of the corresponding models with a single year of prolonged diapause, with stability boundaries becoming extended as β increases. For $\alpha = \beta = 0.4$, the stability boundaries reach $k \approx 2$. However, due to the metabolic costs and the risk of mortality associated with diapause, these values are unlikely.

A key issue is the proportion of animals undergoing successive years of prolonged diapause. Table 2 presents the α and β values reported in previous studies for various insect species (regardless of whether their parasitoids have been identified). Although few data

Table 1
The influence of prolonged diapause in a stochastic environment

k value	C.V.	Conformer	Regulator	Independent phenology
0.8	0.1	0	0	0
(*s*)	0.2	0	0	0
	0.3	0	0	0
	0.4	0	0	0
	0.5	0.007(2703.83)	0.003 (1769.10)	0.017 (2947.44)
1.08	0.1	0	0	0
(*sdp*)	0.2	0	0	0
	0.3	0	0	0
	0.4	0.26 (2451.71)	0.004 (2515.33)	0.005 (2153.75)
	0.5	0.95 (1338.98)	0.25 (1783.11)	0.38 (2698.07)
1.5	0.1	1 (289.59)	0.98 (1781.24)	0.98 (1722.53)
(*u*)	0.2	1 (236.58)	1 (882.38)	1 (751.02)
	0.3	1 (203.96)	1 (593.08)	1 (524.11)
	0.4	1 (171.96)	1 (353.08)	1 (295.79)
	0.5	1 (136.44)	1 (251.23)	1 (228.79)

Frequency of population extinction for 3 models including a 1-year prolonged diapause (''conformer'' parasitoid, ''regulator'' parasitoid and independent phenology, see main text) in stochastic environments, for three degrees of parasitoid attack aggregation (k). The numbers shown are the proportions of replicates not surviving 5000 generations. The figures in brackets correspond to the mean time to extinction for those replicates in which the system collapsed before the end of the simulation. In these simulations the rate of increase, λ and the mortality rate μ were taken as random variables (with means $\bar{\lambda} = 2$ and $\bar{\mu} = 0.2$ respectively). The coefficients of variation are shown in the table (C.V.). Other parameter values: $\alpha = 0.25$ and $a = 0.008$. The values of k and α are also shown in Fig. 1 as u (for unstable), s (for stable) and sdp (for stable as a consequence of prolonged diapause). See main text for further details.

Table 2
Examples of the frequency of prolonged diapause (α and β values) in insects

Species	α	β	Source
Curculio elephas (Curculionidae)	0.41	0.09	Menu (1993)
Gilpinia hercyniae (Diprionidae)	0.18	0.02	Danks (1987)
Rhagoletis mendax (Tephritidae)	0.20	0.2	Danks (1987)
Rhagoletis completa (Tephritidae)	0.34	0.23	Danks (1987)
Leptinotarsa decemlineata (Chrysomelidae)	0.019	0.003	Tauber and Tauber (2002)
Diabrotica virgifera (Chrysomelidae)	0.315	0.16	Levine et al. (1992)
Laspeyresia strobilella (Tortricidae)	0.17	0.05	Annila (1981)
Pegomya zonata (Anthomyiidae)	0.20	N/A	Hanski and Stahls (1990)
Pegomya scapularis (Anthomyiidae)	0.16	N/A	Hanski and Stahls (1990)
Hylemyia anthracina (Anthomyiidae)	0.41	0.34	Annila (1981)
Megastigmus spermotrophus (Torymidae)	0.07	0	Annila (1982)
Megastymus spermophilus (Torymidae)	0.19	N/A	Roux et al. (1997)
Contarinia sorghicola (Cecidomiiydae)	0.26	0.1	Danks (1987)
Contarinia pisi (Cecidomyiidae)	0.9	0.025	Keller and Schweizer (1994)

Values were estimated from previously reported data on the proportions of the population emerging after prolonged diapause. In cases in which more than one data set was available, we took mean values. N/A indicates data not measured or not available.

are available, it is clear that α and β rarely take values higher than 0.4.

3. Discussion

Prolonged diapause, the condition in which a fraction of the population extends diapause for supplementary years, is common in many insect populations (see Waldauber, 1978; Annila, 1981; Danks, 1987; Hanski, 1988; Menu and Debouzie, 1993, for examples), including parasitoids (Annila, 1981; Danks, 1987; Hanski, 1988). For about half the insect species for which prolonged diapause has been described (Hanski, 1988) and in which diapause extends for a single year, the effect of this prolongation of diapause is thought to be limited: in our models, stability requires values of k close to unity (the stability criterion for a model without prolonged diapause). Including an additional year in prolonged diapause, corresponding to approximately

20% of known cases (Hanski, 1988), may extend the stability boundaries up to k values approaching $k = 2$, for realistic parameter values. Models with geometrically distributed diapause spans (Ringel et al., 1998 and Appendix C (Section C.2) suggest that diapause spans longer than 2 years would further extend the stability boundaries.

Our results also show that certain ecological details of the interaction—the number of years in diapause and the proportion of animals undergoing diapause for various lengths of time in particular—strongly influence the stability of host–parasitoid systems. Our findings also suggest that the timing of attacks, the time at which hosts are killed and the physiological relationship between the species involved may also affect the dynamics of the system. Many pest insects and their parasitoids undergo prolonged diapause (e.g. Annila, 1981; Brockerhoff and Kenis, 1996). The outcome of programs for the biological control of pest insects may therefore depend on this phenomenon.

We considered three main relationships between the host and parasitoid development. If hosts are not killed during early development, parasitoids commonly adopt the strategy of conforming to host physiology (Eqs. (3) and (B.1)). This leads to developmental synchrony of the two populations. Several examples in which such synchrony is observed have been described in previous studies by Annila (1981), Beckage (1985), Danks (1987), Hanski (1988) and Brockerhoff and Kenis (1996). Another example is the cynipid wasp *Ibalia leucospoides*, a parasitoid of the woodwasp *Sirex noctilio* (J. Corley, pers.obs.).

Alternatively, hosts may be killed by parasitoids at early stages, before or during diapause. In such cases, parasitoids may not display prolonged diapause. Persistence in a state of prolonged diapause for additional years is possible only for the fraction of hosts that have escaped parasitism and so only hosts emerge after prolonged diapause (Eqs. (4) and (B.2)). Such behavior has been reported for many ichneumons (Askew, 1971).

Finally, we considered situations in which parasitoids attack late larval instars, pupae or even adults, and in which prolonged host diapause occurs at some stage before parasitoid attack. In these cases, prolonged diapause and parasitoid attack are totally independent events (Eqs. (3) and (B.3)). Although we know of no specific cases of such systems, they are very likely to occur and are likely to be reported only rarely. The results for our three models suggest that prolonged diapause would have only a moderate influence on stability. We did obtain stronger predicted effects on stability, but only in what are probably unrealistic conditions.

The stabilizing mechanism in our models is probably similar to that found in metapopulation models. In these models, there is asynchrony across subpopulations linked by migration, caused by intrinsic patch dynamics. The number of immigrants is independent of local population density, whereas its *relative* contribution is negatively related to local density (Murdoch et al., 1992).

However, in the prolonged diapause models, the number of individuals originating in different years and contributing to the present population should be correlated. This might account for the weaker stabilizing effect of prolonged diapause than reported in studies of metapopulation dynamics. However, time delays may have complex effects on population dynamics (Crone, 1997).

Menu (Menu, 1993; Menu et al., 2000) showed that in stochastic environments, prolonged diapause makes a strong contribution to the persistence of single-population models. Our work suggests that parasitoids would strongly decrease this effect. This is an important issue because few, if any, insect populations are likely to be free from parasitoid attack and environmental perturbations. As shown above, in a stochastic environment, our models for one year of prolonged diapause are only slightly more persistent than the equivalent host–parasitoid model without prolonged diapause.

Ringel et al. (1998) recently incorporated prolonged host and parasitoid diapause into a Nicholson-Bailey framework and concluded that stability was greatly increased by prolonged host diapause (stability boundaries include values up to $k \to \infty$). Ringel et al. (1998) assumed that diapause always constitutes a refuge from parasitism. Their results are not surprising given the well-known effect of refuges on the stability of Nicholson-Bailey models (Hassell, 1978; Hochberg and Holt, 1999; Hassell, 2000; Bernstein, 2000). The stabilizing influence of refuges in host–parasitoid systems including diapause is further illustrated in Appendix C (Section C.1).

Diapause may be a physical refuge against parasitism, as has been suggested for *Neodiprion swainei*, the Swaine jack pine sawfly (Price and Tripp, 1972), but there is little evidence to suggest that this is a common feature of host–parasitoid systems including diapause.

One major assumption made by Ringel et al. (1998) is that the duration of prolonged diapause is geometrically distributed. This corresponds to the limiting case of the models presented here in which, at least potentially, insects can diapause for an indeterminate number of years. This assumption, which reflects the biology of some natural populations, implies that there is a chance (albeit a very small chance) that a given individual will remain in prolonged diapause for an unlimited number of generations. As shown in Appendix C (Section C.2), geometric distributions of diapause spans may strongly stabilize host–parasitoid systems. However, for many host–parasitoid systems such distributions are unrealistic. Insects rarely prolong diapause for more than 2

years (Hanski, 1988; Turgeon et al., 1994) and the maximum duration of extended diapause for any individual does not exceed a fixed number of generations. This is probably because the costs of prolonging diapause for long periods are rarely compensated by the advantages of emerging later (Kroon and Veenendaal, 1998; Menu et al., 2000).

Our models suggest that, in general terms, prolonged host diapause makes much less of a contribution to the stability of host–parasitoid interactions than suggested by Ringel et al. (1998). There are even conditions in which prolonged diapause would reduce stability. When interpreting these conflicting results, it should be borne in mind that host–parasitoid systems framed in terms of difference equations are intrinsically unstable. The instability of these systems stems from the one generation delay between changes in the host population and changes in parasitoid attacks. The Ringel et al. (1998) model includes two elements (refuges and the geometric distribution of prolonged diapause spans), each of which can stabilize host–parasitoid systems on their own (see Appendix C) but that are unrealistic for many host–parasitoid interactions.

In conclusion, this work shows that for most host–parasitoid systems, prolonged diapause is likely to make a modest (and much smaller than previously claimed) contribution to the stability and persistence of coupled host–parasitoid interactions. This is particularly true if biologically realistic parameters are considered. Finally, our results also suggest that some features of the life history of the populations involved may have a crucial influence on the dynamics of the systems (e.g. whether diapause creates refuges from parasitism), whereas others may have only a minor effect (similar predictions are generated by all three models presented here).

Acknowledgments

This work was carried out during an exchange program between France and Argentina supported by the SECyT-ECOS-SUD Program (A98B05). Angel Capurro/Jorge Rabinovich received financial support through CONICET Grant PIP# 529/98 and Juan Corley through SAGPyA PIA# 08/98 and 01/00. We thank Fréderic Menu, Emmanuel Desouhant, Thierry Spataro, Rafael Bravo de la Parra, Muriel Ney-Nifle and three anonymous reviewers for their very valuable comments on the text.

Appendix A. Stability analysis for the three host–parasitoid models with a 1-year prolonged diapause

Analysis of the stability of the various models close to equilibrium densities requires that the systems are fully specified in terms of densities at times t and $t-1$. For this, we introduce two variables, Zt and Yt, corresponding to the densities at time t of hosts and parasitoids, respectively, that hatched at time $t-1$. We define $Zt = Nt-1$ and $Yt = Pt-1$. The models take the following forms:

Model 1

$$N_t = (1-\alpha)\lambda N_{t-1}(1+aP_{t-1}/k)^{-k} + \sigma\alpha\lambda Z_{t-1}(1+aY_{t-1}/k)^{-k},$$

$$Z_t = N_{t-1},$$

$$P_t = (1-\alpha)N_{t-1}\{1-(1+aP_{t-1}/k)^{-k}\} + \sigma\alpha Z_{t-1}\{1-(1+aY_{t-1}/k)^{-k}\},$$

$$Y_t = P_{t-1},$$

Model 2

$$N_t = (1-\alpha)\lambda N_{t-1}(1+aP_{t-1}/k)^{-k} + \sigma\alpha\lambda Z_{t-2}(1+aY_{t-1}/k)^{-k},$$

$$Z_t = N_{t-1},$$

$$P_t = N_{t-1}\{1-(1+aP_{t-1}/k)^{-k}\},$$

$$Y_t = P_{t-1},$$

Model 3

$$N_t = \{(1-\alpha)\lambda N_{t-1} + \sigma\alpha\lambda Z_{t-1}\}\{(1+aP_{t-1}/k)^{-k}\},$$

$$Z_t = N_{t-1},$$

$$P_t = \{(1-\alpha)N_{t-1} + \sigma\alpha Z_{t-1}\}\{1-(1+aP_{t-1}/k)^{-k}\}.$$

The equilibrium for each model is found by setting

$$N^* = N_{t-1} = N_{t-2} \text{ and } P^* = P_{t-1} = P_{t-2},$$

where

$$H = [(1-\alpha)+\sigma\alpha].$$

Thus, for Models 1 and 3,

$$P^* = \left[(rH)^{\frac{1}{k}} - 1\right]\frac{k}{(1-\alpha)},$$

$$N^* = P^*\frac{1}{H}\frac{1}{\left(1-\left(1+\frac{aP^*}{k}\right)^{-k}\right)},$$

whereas, for Model 2

$$P^* = \left[(rH)^{\frac{1}{k}} - 1\right]\frac{k}{(1-\alpha)},$$

$$N^* = P^*\frac{1}{\left(1-\left(1+\frac{aP^*}{k}\right)^{-k}\right)}.$$

The corresponding Jacobian matrices (J_i) for each model i are,

$$J_1 = \begin{pmatrix} 1+\left(\frac{ap_{t-1}}{k}\right)^{-k}(r(1-\alpha) & \left(1+\frac{ap_{t-2}}{k}\right)^{-k}r\sigma\alpha & -aN_{t-1}\left(1+\frac{ap_{t-1}}{k}\right)^{-1-k}(r(1-\alpha) & -aN_{t-2}\left(1+\frac{ap_{t-2}}{k}\right)^{-1-k}r\sigma\alpha \\ 1 & 0 & 0 & 0 \\ \left(1-\left(1+\frac{ap_{t-1}}{k}\right)^{-k}\right)(1-\alpha) & \left(1-\left(1+\frac{ap_{t-2}}{k}\right)^{-k}\right)\sigma\alpha & aN_{t-1}\left(1+\frac{ap_{t-1}}{k}\right)^{-1-k}(1-\alpha) & aN_{t-2}\left(1+\frac{ap_{t-2}}{k}\right)^{-1-k}\sigma\alpha \\ 0 & 0 & 1 & 0 \end{pmatrix},$$

$$J_2 = \begin{pmatrix} \left(1+\frac{ap_{t-1}}{k}\right)^{-k}r(1-\alpha) & \left(1+\frac{ap_{t-2}}{k}\right)^{-k}r\sigma\alpha & -aN_{t-1}\left(1+\frac{ap_{t-1}}{k}\right)^{-1-k}r(1-\alpha) & -aN_{t-2}\left(1+\frac{ap_{t-2}}{k}\right)^{-1-k}r\sigma\alpha \\ 1 & 0 & 0 & 0 \\ 1-\left(1+\frac{ap_{t-1}}{k}\right)^{-k} & 0 & aN_{t-1}\left(1+\frac{ap_{t-1}}{k}\right)^{-1-k} & 0 \\ 0 & 0 & 1 & 0 \end{pmatrix},$$

$$J_3 = \begin{pmatrix} \left(1+\frac{ap_{t-1}}{k}\right)^{-k}r(1-\alpha) & \left(1+\frac{ap_{t-1}}{k}\right)^{-k}r\sigma\alpha & -aN_{t-2}\left(1+\frac{ap_{t-1}}{k}\right)^{-1-k} & r\sigma\alpha - aN_{t-1}\left(1+\frac{ap_{t-1}}{k}\right)^{-1-k}r(1-\alpha) \\ 1 & 0 & & 0 \\ 1-\left(1+\frac{ap_{t-1}}{k}\right)^{-k}(1-\alpha) & 1-\left(1+\frac{ap_{t-1}}{k}\right)^{-k}\sigma\alpha & aN_{t-2}\left(1+\frac{ap_{t-1}}{k}\right)^{-1-k} & \sigma\alpha - aN_{t-1}\left(1+\frac{ap_{t-1}}{k}\right)^{-1-k}(1-\alpha) \end{pmatrix}.$$

Local stability is ensured only if all the *eigenvalues* of the Jacobian matrix are less than 1 in absolute value. For each model, as a function of the relevant parameters, this condition was found numerically using the symbolic-numerical package *Mathematica*©. Simulations were also performed with a program written in C.

Appendix B. The three models in which diapause extended over 3 years

The models for the three different situations, with 2 years of prolonged diapause. Parameter β is the fraction of the population staying in prolonged diapause for an additional year. For the other parameters, see the main text.

(a) "Conformer" parasitoids

$$\begin{aligned} N_t &= (1-a)\lambda N_{t-1}(1+aP_{t-1}/k)^{-k} \\ &\quad + \sigma(1-\beta)a\lambda N_{t-2}(1+aP_{t-2}/k)^{-k} \\ &\quad + \sigma\beta\alpha\lambda N(1+aP_{t-3}/k)^{-k}, \\ P_t &= (1-\alpha)N_{t-1}\{1-(1+aP_{t-1}/k)^{-k}\} \\ &\quad + \sigma(1-\beta)aN_{t-2}\{1-(1+aP_{t-2}/k)^{-k}\} \\ &\quad + \sigma\beta\alpha N_{t-3}\{1-(1+aP_{t-3}/k)^{-k}\}. \end{aligned} \tag{B.1}$$

(b) "Regulator" parasitoids

$$\begin{aligned} N_t &= (1-a)\lambda N_{t-1}(1+aP_{t-1}/k)^{-k} \\ &\quad + \sigma(1-\beta)\alpha\lambda N_{t-2}(1+aP_{t-2}/k)^{-k} \\ &\quad + \sigma\beta\alpha\lambda N_{t-2}(1+aP_{t-3}/k)^{-k}, \\ P_t &= N_{t-1}\{1-(1+aP_{t-1}/k)^{-k}\}. \end{aligned} \tag{B.2}$$

(c) "Independent" parasitoid phenology

$$\begin{aligned} N_t &= \{(1-\alpha)\lambda N_{t-1} + \sigma(1-\beta)\alpha\lambda N_{t-2} \\ &\quad + \sigma\beta\alpha\lambda N_{t-3}\}\{(1+aP_{t-1}/k)^{-k}\}, \\ P_t &= \{\alpha N_{t-1} + \sigma(1-)\beta\alpha N_{t-2} \\ &\quad + \sigma\beta\alpha N_{t-3}\}\{1-(1+aP_{t-1}/k)^{-k}\}. \end{aligned} \tag{B.3}$$

Appendix C. The stabilizing influence of refuges and the geometric distribution of prolonged diapause spans

The aim of this appendix is to describe the influence of refuges and a geometric distribution of prolonged diapause spans on the stability of host–parasitoid systems including prolonged diapause. For this, two models, each including one of these processes, were analyzed for local stability (as in Appendix A).

C.1. Prolonged diapause as a refuge from parasitism

In this model we consider a host–parasitoid system in which a fraction of the host population undergoes 1 year of prolonged diapause, and in which, as described in the study by Ringel et al. (1998), individuals undergoing prolonged diapause are fully protected from parasitism.

The equations for the model are:

$$N_t = (1-\alpha)\lambda N_{t-1}(1+aP_{t-1}/k)^{-k} + \sigma\alpha\lambda N_{t-2},$$
$$P_t = (1-\alpha)N_{t-1}\{1-(1+aP_{t-1}/k)^{-k}\}, \qquad \text{(C.1)}$$

where parameters are defined as in the main text.

The stability boundaries of this model are presented in Fig. 2 for two values of $k(k=2, k=\infty)$, for which the models including prolonged diapause (Eqs. (3)–(5), (B.1), (B.2) and (B.3)) would be unstable. The stability boundaries for $k=2$ are somewhat more extended than those of the classical Hassell and May (1973) (model D) refuge model, which does not include prolonged diapause (for comparison, Fig. 2 presents the stability boundaries of Eq. (C.1) as a function of the same parameters as the Hassell and May (1973) (model). Note that Hassell and May (1973) implicitly assumed that $\sigma = 1$.

C.2. Geometrical distribution of diapause spans

In this section, we considered a "conformer" parasitoid model (see Eq. (3)) in which prolonged diapause spans are geometrically distributed (as assumed by Ringel et al., 1998) and in which diapause does not constitute a refuge from parasitism. The equations for the model are:

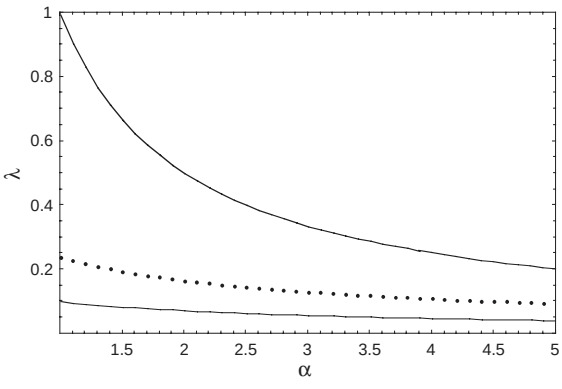

Fig. 2. Stability boundaries, for a model in which prolonged diapause extends over a single year and provides a refuge from parasitism for diapausing hosts. The stability boundaries for two values of k, the coefficient controlling the aggregation of parasitoid attacks ($k=2$, unbroken lower line and $k=\infty$, dotted line), are shown in terms of α, the proportion of individuals in the population undergoing prolonged diapause and λ the host reproduction rate. The model is stable for combinations of parameter values between the upper curve and either of the two lower curves, depending on the value of k.

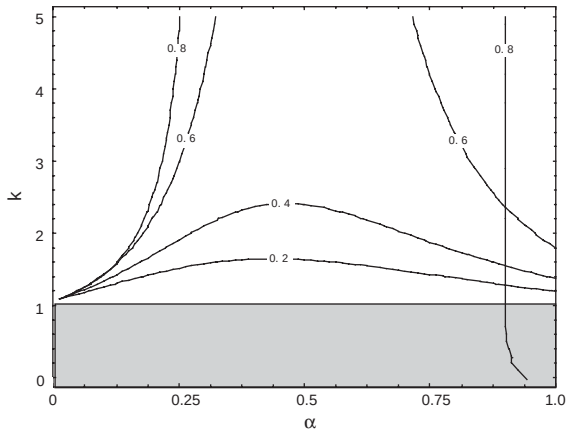

Fig. 3. Stability boundaries for a "conformer" model with a geometric distribution of diapause spans. Stability boundaries are drawn as a function of α, the fraction of newly emerged individuals undergoing prolonged diapause and k, the aggregation coefficient, for different values of β, the fraction of individuals remaining in prolonged diapause, in a given year. The system is stable for the values of k and α below the curves corresponding to each β value. Other parameter values are as in Fig. 1. The area below the line at $k=1$ corresponds to the stability boundaries for the May (1978) model, the equivalent model without prolonged diapause. The nearly vertical line for $\beta = 0.8$ at high α values results from the combination of a large proportion of the population undergoing prolonged diapause and low survival rates. For slightly higher survival rates, this line disappears, and so, for $\beta = 0.8$ the model becomes stable for any high value of α.

$$N_t = (1-\alpha)\lambda N_{t-1}(1+aP_{t-1}/k)^{-k} + \sigma(1-\beta)\lambda ND_{t-1},$$
$$ND_t = \alpha N_{t-1}(1+aP_{t-1}/k)^{-k} + \sigma\beta ND_{t-1},$$
$$P_t = (1-\alpha)N_{t-1}\{1-(1+aP_{t-1}/k)^{-k}\} + \sigma(1-\beta)PD_{t-1},$$
$$PD_t = \alpha N_{t-1}\{1-(1+aP_{t-1}/k)^{-k}\} + \sigma\beta PD_{t-1}, \qquad \text{(C.2)}$$

where β is the fraction of individuals remaining in prolonged diapause for an additional year, and ND_t and PD_t are the numbers of hosts and parasitoids, respectively, in prolonged diapause at year t. Other parameters are as in the main text.

Fig. 3 shows the stability boundaries of this model for two values of k ($k=2, k=\infty$). These results indicate that a model in which diapause spans are geometrically distributed is stable for a range of parameter values over which models including prolonged diapauses of only 1 or 2 years (Eqs. (3) and (B.1), Fig 1a) would be unstable.

References

Annila, E., 1981. Fluctuations in cone and seed insect populations in Norway spruce. Commun. Inst. For. Fenn. 101, 1–32.

Annila, E., 1982. Diapause and population fluctuations in *Megastimus specularis* Walley and *Megastimus spermotrophus* Wachtl (Hymenptera: Torymidae). Ann. Entmol. Fennici 48, 33–36.

Askew, R.R., 1971. Parasitic Insects. Elsevier, New York.

Beckage, N.E., 1985. Endocrine interactions between endoparasitic insects and their hosts. Ann. Rev. Entomol. 30, 371–413.

Bernstein, C., 1986. Density dependence and the stability of host–parasitoid systems. Oikos 47, 176–180.

Bernstein, C., 2000. Host–parasitoid models: the story of a successful failure. In: Hochberg, M.E., Ives, A.R. (Eds.), Parasitoid Population Biology. Princeton University Press, Princeton, pp. 41–57.

Bernstein, C., Heizmann, A., Desouhant, E., 2002. Intraspecific competition between healthy and parasitized hosts in a host parasitoid system: consequences for life-history traits. Ecol. Entomol. 27, 415–423.

Brockerhoff, E.G., Kenis, M., 1996. Parasitoids associated with *Cydia strobiella* (L) (Lepidoptera: Tortricidae) in Europe, and considerations for their use for biological control in North America. Biol. Control 6, 202–214.

Crone, E.E., 1997. Delayed density dependence and the stability of interacting populations and subpopulations. Theor. Popul. Biol. 51, 67–76.

Danks, H.V., 1987. Insect Dormancy: an Ecological Perspective. Biological Survey of Canada No.1, Ottawa.

Ellers, J., van Alphen, J.J.M., 2002. A trade-off between diapause duration and fitness in female parasitoids. Ecol. Entomol. 27, 279–284.

Godfray, H.C.J., 1994. Parasitoids. Behavioural and Evolutionary Biology. Princeton University Press, Princeton.

Han, E.N., Bauce, E., 1998. Timing of diapause initiation, metabolic changes and overwintering survival on the spruce budworm, *Choristoneura fumiferana*. Ecol. Entomol. 23, 160–167.

Hanski, I., 1988. Four kinds of extra long diapause in insects: a review of theory and observations. Ann. Zool. Fennici 25, 37–53.

Hanski, I., Stahls, G., 1990. Prolonged diapause in fungivorous *Pegomya* flies. Ecol. Entomol. 15, 241–244.

Hassell, M.P., 1978. The Dynamics of Arthropod Predator–Prey Systems. Princeton University Press, Princeton.

Hassell, M.P., 2000. The Spatial and Temporal Dynamics of Arthropod Host–Parasitoid Interactions. Oxford University Press, Oxford, 200pp.

Hassell, M.P., May, R.M., 1973. Stability in insect-host parasite models. J. Anim. Ecol. 42, 693–726.

Hochberg, M.E., Holt, R., 1999. The uniformity and density of pest exploitation as guides to success in biological control. In: Hawkins, B.A., Cornell, H. (Eds.), Theoretical Approaches to Biological Control. Cambridge University Press, Cambridge, pp. 71–88.

Hopper, K.R., 1999. Risk-spreading and bet-hedging in insect population biology. Ann. Rev. Entomol. 44, 535–560.

Irwin, J.T., Lee, R.E., 2000. Mild winter temperatures reduce survival and potential fecundity of the goldenrod gall fly, *Eurosta solidaginis*. J. Insect Physiol. 46, 655–661.

Ishihara, M., Shmida, M., 1995. Trade-off in allocation of metabolic reserves: effects of diapause on egg production and adult longevity in an multivoltine bruchid, *Kytorhinus Sharpianus*. Funct. Ecol. 9, 618–624.

Keller, S., Schweizer, C., 1994. Populationdynamysche untersuchungen an der erbsengallmucke Contarinia pisi Winn. (dipt., Cecidomyiidae) und ihrer parasitoide. J. Appl. Entomol. 188, 281–299.

Kroon, A., Veenendaal, R.L., 1998. Trade-off between diapause and other life-history traits in the spider mite *Tetranychus urticae*. Ecol. Entomol. 23, 298–304.

Lawrence, P.O., 1986. Host–parasite hormonal interactions: an overview. J. Insect Physiol. 32, 295–298.

Levine, E., Oloumi-Sadeghi, H., Fisher, J.R., 1992. Discovery of multiyear diapause in Illinois and South Dakota Northern Corn rootworm (Coleoptera: Chrysomelidae) eggs and incidence of prolonged diapause trait in Illinois. J. Econ. Entomol. 85, 262–267.

May, R.M., 1978. Host–parasitoid systems in patchy environments: a phenomenological model. J. Anim. Ecol. 47, 833–843.

Menu, F., 1993. Strategies of emergence in the chestnut weevil *Curculio elephas* (Coleoptera: Curculionidae). Oecologia 96, 383–390.

Menu, F., Debouzie, D., 1993. Coin flipping plasticity and prolonged diapause in insects: example of the chestnut weevil *Curculio elephas* (Coleoptera: Curculionidae). Oecologia 93, 367–373.

Menu, F., Roebuck, J., Viala, M., 2000. Bet hedging diapause strategies in stochastic environment. Am. Nat. 155, 724–734.

Murdoch, W.W., Briggs, C.J., Nisbet, R.M., Gurney, W.S., Stewart-Oaten, A., 1992. Aggregation and stability in metapopulation models. Am. Nat. 140, 41–58.

Nicholson, A.J., Bailey, V.A., 1935. The balance of animal populations. Part I. Proc. Zool. Soc. London 3, 551–598.

Philippi, T., Seger, J., 1989. Hedging one's evolutionary bets, revisited. T.R.E.E. 4, 41–44.

Press, W.H., Teukolsky, S.A., Vetterling, W.T., Flannery, B.P., 1992. Numerical Recipes in C. Cambridge University Press, Cambridge.

Price, P.W., Tripp, H.A., 1972. Activity patterns of parasitoids on the Swaine Jack Pine sawfly *Neodiprion swainei* (Hymenoptera: Diprionidae) and parasitoid impact on the host. Can Entomol. 104, 1003–1016.

Ringel, M.S., Rees, M., Godfray, H.C.J., 1998. The evolution of diapause in a coupled host–parasitoid system. J. Theor. Biol. 194, 195–204.

Roux, G., Roques, A., Menu, F., 1997. Effect of temperature and photoperiod on diapause development in Douglas fir seed chalcid, *Megastimus spermotrophus*. Oecologia 111, 172–177.

Storey, J.M., Storey, K.B., 1986. Winter survival of the gall fly larva, *Eurosta solidaginis*: profiles of fuel reserves and cryprotectants in a natural population. J. Insect. Physiol. 32, 549–556.

Tauber, M.J., Tauber, C.A., 2002. Prolonged dormancy in *Leptinotarsa decemlineata* (Coleptera: Chrysomelidae): a ten-year field study with implications for crop rotation. Environ. Entomol. 31, 499–504.

Tauber, M.J., Tauber, C.A., Nechols, J.R., Obrycki, J.J., 1983. Seasonal activity of parasitoids: control by external, internal and genetic factors. In: Brown, V.K., Hodek, I. (Eds.), Diapause and Life Cycle Strategies. Junk Publishers, The Hague, pp. 87–108.

Tauber, M.J., Tauber, C.A., Masaki, I., 1986. Seasonal Adaptations of Insects. Oxford University Press, Oxford.

Turgeon, J.J., Roques, A., Groot, P., 1994. Insect fauna of coniferous seed cones: diversity, host plant interactions and management. Ann. Rev. Entomol. 39, 179–212.

Waldauber, G.P., 1978. Phenological adaptations and polymodal emergence patterns of insects. In: Dingle, H. (Ed.), Evolution of Insect Migration and Diapause. Springer, Berlin, pp. 127–144.

Zhou, X., Honek, A., Powell, W., Carter, N., 1995. Variations in body length, weight, fat content and survival in *Coccinella septempunctata* at different hibernation sites. Entomol. Exp. Appl. 75, 99–107.