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DISCRETE GENERATIONS IN HOST–PARASITOID MODELS WITH CONTRASTING LIFE CYCLES

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SUMMARY

(1) Previous theoretical studies have shown that parasitoids are capable of producing cycles in their host populations with periods of about one host generation. These studies have modelled host–parasitoid interactions where parasitoid development proceeds independently from that of its host.

(2) We present experimental results of a particular host–parasitoid association, *Cadra cautella*–*Venturia canescens*, and show that the development of the parasitoid is synchronized with that of its host.

(3) The empirical evidence is used to formulate an age-structured model of an idealized host–parasitoid system where the onset of parasitoid development is dependent on the state of the host. The properties of the model are described and contrasted with a model that assumes there is no developmental synchrony in host and parasitoid life cycles.

(4) Our results are similar to the findings of previous studies, showing that the differences in the behaviour of population models with and without life cycle synchrony are quantitative rather than qualitative. We show that when developmental synchrony occurs, ratios of parasitoid/host development durations must be interpreted with care, when predicting whether single generation cycles are to be expected in a particular host–parasitoid system.

INTRODUCTION

Some tropical insect populations, regulated in part by parasitoids, cycle with periods close to the host generation time in the apparent absence of environmental cues (Godfray & Hassell 1989). Similar behaviour has been observed in laboratory population studies of stored products Lepidoptera and their parasitoids (Flanders 1968; White & Huffaker 1969; Takahashi 1973; Podoler 1974). One intriguing aspect of both the laboratory and natural populations is that the ‘single generation cycles’ may be superimposed on background cycles with longer periods (Fig. 1.)

Population fluctuations such as those shown in Fig. 1 can only be described using age-structured models. Such models have the capacity to reveal both the internal

FIG. 1. Examples of generation cycles in field and laboratory insect populations. (a) The leaf-mining hispine beetle, *Coelaenomenodera minuta*, on oil palm in West Africa (after Mariau & Morin 1972). (b) Laboratory population of the stored products moth *Anagasta kuehniella* and the parasitoid *Venturia canescens* (after White & Huffaker 1969). (c). Laboratory population of the stored products moth *Plodia interpunctella* (—) and *V. canescens* (----) (after Podoler 1974).

mechanisms synchronizing generations, and the combined effects of such mechanisms with those that generate cycles with much longer periods. Theoretical studies have described mechanisms capable of producing generation cycles in single-species populations (Bellows 1982; Gurney, Nisbet & Lawton 1983; Diekmann *et al.* 1986; Gurney & Nisbet 1985); however, the literature on generation cycles in structured host-parasitoid models is sparse, and only two studies focus on mechanisms

capable of producing discrete generations: Auslander, Oster & Huffaker (1974), Godfray & Hassell (1987, 1989).

Motivated by the experiments described in Hassell & Huffaker (1969), Auslander *et al.* (1974) constructed an elaborate simulation model, and found that two modes of behaviour were possible: host and parasitoid populations could equilibrate with stable age distributions, or host reproduction could synchronize, resulting in discrete generations with the parasitoid establishing a bimodal pattern of reproduction, matching two generations to a host generation. The simulation model is too complicated for the cause of synchrony to be clear, but it is noteworthy that the model incorporates many of the mechanisms implicated in single-species models: cannibalism, larval competition, and density-dependent fecundity related to an individual's feeding history.

The Godfray and Hassell study is much more instructive. They developed a simple model where host fecundity and mortality (excepting parasitism) were constant, thereby excluding the single-species mechanisms that synchronize generations. Hosts were susceptible to parasitism for a (relatively short) part of their juvenile period. Density dependence was incorporated by assuming that the parasitoid's efficiency in locating hosts declined as parasitoid density increased. They produced a 'canonical' set of demographic parameters for the host, and studied the effect of the density dependence parameter and the ratio of host/parasitoid development durations on stability and the pattern of population fluctuations. Their main conclusions were that single generation cycles are only possible if host and parasitoid development times are unequal, and only likely with strong density dependence in the rate of parasitism.

An important element of the Godfray–Hassell model is the assumption of a juvenile parasitoid stage of fixed duration, where juvenile development commences immediately following oviposition. However, for many parasitoids, juvenile development is delayed and does not start until the host reaches a particular stage in its life cycle. Delayed development is particularly prevalent among parasitoids with an endoparasitic life history; where juvenile development occurs within a viable and developing host (Askew & Shaw 1986). Our aim in this paper is to present a model that incorporates the phenomenon of host-dependent parasitoid development. We will present experimental results from a particular host–parasitoid system and use these results to justify our formulation of the model. The properties of this model will be described and contrasted with the Godfray–Hassell model.

HOST–PARASITOID DEMOGRAPHY

For an example of a host–parasitoid system in which parasitoid development is synchronized with that of its host, we undertook a series of experiments using the stored products moth, *Cadra cautella* (Walker) (Lepidoptera: Phycitidae) and the thelytokous, solitary, larval endoparasite, *Venturia canescens* (Gravenhorst) (Hymenoptera: Ichneumonidae). We will only briefly describe the experimental protocols, further details can be found in Gordon (1987), Gordon & Stewart (1988) and Trudeau & Gordon (1989).

The details of parasitoid juvenile development were studied by monitoring parasitoid development in cohorts of host larvae infected at either 8.5 or 18.5 days of age. Twenty wasps were added for 12 h to vials containing large numbers of larvae. Insects were removed daily and the development stage of the parasitoid determined.

The durations of the parasitoid egg stage, second to fifth instar period, and the pupal stage were essentially independent of host age at infection (Fig. 2a,b). First instar duration was longer when hosts were infected at the younger age (Fig. 2a,b).

A more general picture of parasitoid development duration as a function of host age at infection was obtained by adding a wasp to vials containing host larvae of known age for 24 h and recording when the parasitoids emerged. Parasitoid age at emergence (number of days from oviposition) declined in a linear manner as host age at infection increased until larval age at infection corresponded to hosts in their final instar (>16 days); from this point parasitoid development duration was independent of age at infection (Fig. 2c).

The dependence of the parasitoid's development duration on host age at infection is a result of the arrested development of first instar parasitoids. Following oviposition in a host, the parasitoid egg hatches and becomes a first instar. If the host is in its fifth and final instar, the parasitoid proceeds through its remaining developmental stages; however, if the host is not in its final instar, the parasitoid remains as a first instar until the host moults to its final instar. The increased juvenile period in young hosts is simply a reflection of the number of days remaining prior to the host attaining its final instar. The same phenomenon has been reported for *V. canescens* infecting the moth *Anagasta kuehniella* (Corbet 1968).

The arrested development of juvenile parasitoids has the effect that when the time of parasitoid emergence from the birth of a host cohort (host egg stage duration + larval age at infection + parasitoid development duration) is plotted as a function of larval age at infection, the time of parasitoid emergence is essentially independent of when the host cohort was infected (Fig. 2d). The departure from a constant time of parasitoid emergence seen in Fig. 2d for larvae infected at an advanced age is unlikely to be of importance for population dynamics, in view of the small number of host larvae that actually attain these ages. This is established in Fig. 2e for which host larval stage duration was determined by monitoring a cohort from egg hatching to pupation. No pupation occurred prior to 20 days and 90% of the larvae had pupated by 24 days, with an average stage duration of about 22 days.

The relationship between parasitoid searching efficiency and host larval age was determined by placing a wasp for 24 h in a container with larvae of known age. Young larvae, those in their first and second instar, are essentially invulnerable to parasitism. Parasitoid searching efficiency then increases as the larvae age and grow, until the hosts become late fifth instars when searching efficiency begins to decline (Fig. 2f).

These empirical results provide the basis for our population model.

MODEL FORMULATION

We partition the host life cycle into four intervals: the time from oviposition to when the host is susceptible to attack, a susceptible period, followed by a stage where the host is neither susceptible nor reproducing, and the reproductive adult. For convenience we designate these stages as: egg, larval, pupa, and adult, respectively. The parasitoid life cycle consists of two parts, the juvenile stage and the reproductive adult. Our representation of a parasitoid life cycle where the onset of juvenile development is delayed is depicted in Fig. 3a. The life cycle described by the Godfray–Hassell model, where juvenile development commences immediately following oviposition, is shown in Fig. 3b.

FIG. 2. Demographic characteristics of the parasitoid *Venturia canescens* and its host, the stored products moth *Cadra cautella*. (a & b) Development of the parasitoid in the larvae of its host: (a) host larval age at infection 8.5 days; (b) age at infection 18.5 days. E corresponds to the egg stage; L1, first instar; L2-L5, instars 2-5; Pupa, pupal stage; Imago, adult wasp. Numbers are the average stage duration. (c) Parasitoid age at imago emergence (number of days from oviposition) as a function of host age at infection. Symbols depict mean and its 95% confidence interval. (d) Time from the birth of the host cohort (egg stage duration + larval age at infection + parasitoid development duration) at which parasitoids emerge as a function of larval age at infection. (e) Proportion of hosts in the larval stage with respect to larval age. (f) Parasitoid attack rate as a function of host larval age.

The key difference in the two models results from our idealization of the parasitoid life cycles. For the delayed parasitoid development model we assume that following oviposition in a susceptible host the parasitoid egg does not hatch but remains as an egg until the host matures into the next stage. At this time the egg hatches and development commences. This idealization produces the pattern of constant time of parasitoid emergence from the birth of the host cohort that is observed in Fig. 2d, and creates two classes of 'susceptible' hosts in the delayed development model: uninfected hosts susceptible to infection, and infected hosts which are no longer

FIG. 3. Idealized host and parasitoid life cycles. (a) Life cycle for the parasitoid delayed development model. Infected individuals in the susceptible stage do not become juvenile parasitoids until the time at which susceptible individuals would normally mature into the next stage. (b) Life cycle with no delay in parasitoid development. Susceptible hosts that are infected immediately become developing parasitoids. Stages in the host life cycle are: E, pre-susceptible (egg) stage; S, susceptible (larval), S_s uninfected, S_i , infected; HP, post-susceptible (pupal); H, reproductive adults. Parasitoid stages: JP, juveniles; P, reproductive adults.

susceptible. Upon entering the next stage infected hosts die and a developing parasitoid remains.

In the model where we assume parasitoid development begins immediately following oviposition, the host dies leaving a developing parasitoid. Therefore, in this model there is no infected class in the 'susceptible' host stage. This model is very similar to that of Godfray & Hassell (1989).

Table 1 summarizes the variables and parameters of the models. Common to both models is the assumption of an asexual host and parasitoid. Additionally, all host and parasitoid stages are subject to a constant density-independent per capita death rate. This simplifies the Godfray–Hassell description: these authors included a maximum adult life span.

The mathematical equations representing the models can be derived using a formalism developed for single-species stage-structured populations (Nisbet & Gurney 1983; Gurney *et al.* 1983) and more recently extended to cover host–parasitoid systems (Murdoch *et al.* 1987). The population balance equations, rates of recruitment, maturation, and parasitism are summarized in Table 2. Derivation of the various rates is sufficiently mundane that we will not describe them in detail. (Readers wishing more detail may refer to Murdoch *et al.* (1987) and Godfray & Hassell (1989) for examples of methods for formulating stage-structured host–parasitoid models.

We have chosen to ignore our observations concerning the dependence of the parasitoid attack rate on host larval age and assume that the attack rate is independent

TABLE 1. Variable definitions and parameter definitions and default values for the population models

Variables		
E		Pre-susceptible stage 'egg' numbers
S		Susceptible stage 'larvae' numbers
HP		Post-susceptible stage 'pupae' numbers
H		Host adults numbers
JP		Juvenile parasitoid stage numbers
P		Parasitoid adults numbers
Parameters		
T_e	11 days	Pre-susceptibles
T_ℓ	14 days	Susceptibles
T_{hp}	8 days	Post-susceptibles
T_{jp}	varied	Juvenile parasitoids
Through stage survivals		
s_e	1.0	Pre-susceptibles
s_{hp}	1.0	Post-susceptibles
s_{jp}	1.0	Juvenile parasitoids
Per capita death rates		
μ_1	0.0 day ⁻¹	Susceptibles
μ_h	0.1 day ⁻¹	Host adults
μ_p	0.5 day ⁻¹	Parasitoid adults
r	84	Host lifetime fecundity
a	0.1 day ⁻¹ P^{-1}	Area of discovery
k	varied	Interference parameter

TABLE 2. Equations for the delayed parasitoid development model and the model with no host-dependent parasitoid development. The models are distinguished only by their assumptions concerning parasitoid development

Population balance equations	
$\dot{S}(t) = R_s(t) - M_s(t) - \Pi(t) - \mu_1 S(t)$	Susceptibles
$\dot{H}(t) = R_h(t) - \mu_h H(t)$	Host adults
$\dot{P}(t) = R_p(t) - \mu_p P(t)$	Parasitoid adults
Rates of recruitment, maturation and parasitism	
$R_s(t) = r s_e H(t - T_e)$	Susceptible recruitment
$\Pi(t) = f(t) S(t) P(t)$	Instantaneous rate of parasitism
$\theta(t) = \exp\left\{-\int_{t-T_e}^t f(t') P(t') dt'\right\}$	Susceptible survival from parasitism
$M_s(t) = R_s(t - T_e) \exp\{-\mu_1 T_e\} O(t)$	Host pupal recruitment
$R_h(t) = s_{hp} M_s(t - T_{hp})$	Host adult recruitment
Adult parasitoid recruitment	
$R_p(t) = \begin{cases} R_s(t - T_1 - T_{jp}) [1 - \theta(t - T_{jp})] s_{jp} \\ \Pi(t - T_{jp}) s_{jp} \end{cases}$	Delayed development No delayed development
Parasitoid pseudo-interference	
$f(t) = k [P(t) T_1]^{-1} \ln[1 + a T_1 P(t) / k]$	Godfray-Hassell form

of the age of the susceptible stage. Following the example of Godfray & Hassell we incorporate density dependence in our models by assuming the parasitoid attack rate depends on adult parasitoid density in the following manner:

$$k/T_1 P \ln(1 + a T_1 P k^{-1}) \quad (1)$$

Godfray and Hassell derived eqn (1) from a controversial model of non-random parasitoid search (May 1978). We prefer to view this function as simply describing a

density-dependent parasitism rate, irrespective of its cause, rather than a measure of aggregation (Chesson & Murdoch 1986; Perry & Taylor 1986).

As it is impractical to explore the behaviour of the two models over a full range of all possible parameter combinations, we have chosen to define a default set of parameter values (Table 1). This 'canonical' parameter set closely approximates the *Cadra cautella*–*Venturia canescens* system. Host stage durations, adult fecundity and life span were taken from Gordon & Stewart (1988). Parameter values concerning the parasitoid are from Gordon (1987). As the data presented in Fig. 2f suggests that young host larvae are invulnerable to parasitism we have included this period (7.5 days) in the 'egg' or pre-susceptible stage. In the results section we explore and compare the properties of these models concentrating on the effect of the density dependence parameter k , the ratio of parasitoid/host development durations, and adult life spans.

RESULTS

Equilibrium densities for adult parasitoids and hosts can be derived analytically. For both models the equilibrium density of adult parasitoids reflects the requirement that host larval survival is at a level that ensures that host adult recruitment and death rates are equal. The result is:

$$P^* = k/aT_\ell [(rs_{\text{shp}})^{1/k} \exp(-\mu_1 T^{1/k}) - 1]$$

In contrast, the host equilibrium is set by the requirement that parasitoid recruitment and loss rates balance. For the delayed parasitoid development model the adult host equilibrium density is

$$H^* = \mu_p s_{\text{hp}} P^* / [\mu_h s_{\text{jp}} (rs_{\text{shp}} - 1)],$$

while for our version of the Godfray–Hassell model, the equilibrium density of adults hosts is:

$$H^* = \mu_p s_{\text{hp}} P^* (\mu_1 + g) / [\mu_h s_{\text{hp}} rs_{\text{shp}} - 1]g,$$

with

$$g = [\ln(rs_{\text{shp}})/k] - \mu_1$$

In the absence of any larval mortality the models have identical host equilibria. However, in the delayed development model death of 'infected' larvae represents reduced parasitoid recruitment; as a result the host equilibria in the two models differ when $\mu_1 \neq 0$.

For both models we identify three major regions of population behaviour: (i) locally stable equilibria and therefore continuous overlapping generations; (ii) unstable equilibria that show stable cycles with a period of about one host generation; and (iii) unstable equilibria where population cycles have much longer periods (see Appendix).

Population behaviour as a function of the parameter k and the ratio of parasitoid/host development durations, for two adult life spans, is shown in Fig. 4. Figure 5 presents the angular frequency, ω (period = $2\pi/\omega$) of the population cycles along the stability boundaries depicted in Fig. 4a, b.

The behaviour of the two models is qualitatively similar. In the delayed development model, the regions where discrete generation cycles occur are shifted to the left as

FIG. 4. Population behaviours for the two models as a function of the density dependence parameter k , and the ratio of parasitoid/host development durations. (a & b) Host adult lifespan is 2 days: (a) delayed development model; (b) no delay in the start of parasitoid development. (c & d) Host adult life span is 10 days: (c) delayed development; (d) no delay in the start of development. The lines denote the parameter values at which the real parts of a root equal zero and define the boundaries for the different population behaviours: S, stable equilibria (overlapping generations); G, unstable equilibria with stable cycles having a period of about a host generation (generation cycles); H, other unstable equilibria where cycles have much longer periods (host-parasitoid cycles). All other parameters as in Table 1.

compared to the model where there is no delay in parasitoid development. The shift results from the additional time delay imposed by the dependence of parasitoid development on host development, and represents the difference between the duration of the susceptible stage and the average age at which susceptible hosts become infected.

For the delayed development model, locally stable equilibria are found in a smaller region of parameter space, illustrating that this model is more unstable. This greater instability explains the observation that generation cycles are more likely, as host adult life span increases, when there is a delay in parasitoid development (Fig. 4).

FIG. 5. Angular frequencies of the population cycles along the stability boundaries shown in Fig. 4a, b: (a) the delayed development model; (b) the model with no delay in the start of parasitoid development. Dotted and solid lines correspond to the dotted and solid lines in Fig. 4a, b.

The critical importance of the host adult life span is illustrated in Fig. 6 and illustrates that the appearance of generation cycles is only possible when the reproductive period of the host is short. As shown previously, the delayed development model produces generation cycles over a slightly greater region of parameter space.

Although the likelihood of generation cycles is very dependent on host adult life span, the models are relatively insensitive to increases in adult parasitoid life span. We illustrate this using the delayed development model with a host life span of 2 days and a parasitoid life span of 10 days (Fig. 7). Contrasting Fig. 7 with Fig. 4a, c reveals that although a five-fold increase in host adult life span dramatically decreases the region of generation cycles, the same increase in parasitoid life span has little effect.

DISCUSSION

The dependence of parasitoid development on the development of its host, exemplified by the *Cadra cautella*–*Venturia canescens* association, is a phenomenon that serves to synchronize parasitoid and host life cycles. Our model demonstrates that this developmental synchrony has little impact on the population dynamics of such host–parasitoid associations when compared to the behaviour of a host–parasitoid model where no such developmental synchrony occurs.

Our results add generality to the conclusions of Godfray & Hassell (1989). Strong levels of density dependence in the rate of parasitism can dramatically effect the age structure of the host population and cause the appearance of generation cycles. However, the results of our delayed development model show that generation cycles can occur when the ratio of parasitoid/development durations are other than 2:1 or 3:2.

In host–parasitoid systems, where parasitoid development is in some way dependent on host development, care is needed when predicting from experimental results whether a parasitoid is capable of producing generation cycles in its host population. In the *Cadra*–*Venturia* system the parasitoid's pre-adult period can vary

FIG. 6. Population behaviours for the two models as a function of host adult life span and the ratio of parasitoid/host development durations. The parameter k was equal to 1.0, all other parameters as in Table 1. (a) Delayed parasitoid development model. (b) No delayed development. S, overlapping generations; G, generation cycles; H, host-parasitoid cycles.

FIG. 7. The effect of increasing parasitoid adult life span on the behaviour of the delayed development model. Host adult life span was 2 days and parasitoid adult life span was 10 days. S, overlapping generations; G, generation cycles; H, host-parasitoid cycles.

from 19 to 35 days, depending on host larval age at infection (Fig. 2c), results that suggest the ratio of parasitoid/host durations would range from 0.6 to 1.1. However, 14 days is the estimate of juvenile duration (T_{jp}) appropriate to the delayed development model formulation (time of parasitoid emergence from birth of host cohort (39 days, Fig. 2d) minus the durations of the pre-susceptible and susceptible stages (25 days, Table 2). The resulting parasitoid/host development duration ratio is 0.4 and reference to Fig. 4a, b suggests the parasitoid has the potential to produce generation cycles, though these would most likely be swamped by the predator-prey cycles unless the density dependence in the rate of parasitism is strong (low k).

The results in this paper and those of Godfray & Hassell (1989) are critically dependent on the assumption of a parasitoid density-dependent rate of parasitism. There is relatively little experimental evidence to support such an assumption (Free, Beddington & Lawton 1977; Hassell 1978). In a subsequent paper we will show that a similar range of population behaviour can be obtained in a host–parasitoid model that does not depend on these assumptions.

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REFERENCES

- Askew, R.R. & Shaw, M.R. (1986). Parasitoid communities: their size, structure and development. *Insect Parasitoids* (Ed. by J.K. Waage & D.J. Greathead), pp. 225–264. Academic Press, London.
- Auslander, D.M., Oster, G.F. & Huffaker, C.B. (1974). Dynamics of interacting populations. *Journal of the Franklin Institute*, **297**, 345–376.
- Bellows, T.S. (1982). Simulation models for laboratory populations of *Callosobruchus chinensis* and *C. maculatus*. *Journal of Animal Ecology*, **51**, 597–624.
- Chesson, P.L. & Murdoch, W.W. (1986). Aggregation of risk: relationships among host–parasitoid models. *American Naturalist*, **127**, 696–715.
- Corbet, S.A. (1968). The influence of *Ephesia kuehniella* on the development of its parasite *Nemeritis canescens*. *Journal of Experimental Biology*, **48**, 291–304.
- Diekmann, O., Nisbet, R.M., Gurney, W.S.C. & van den Bosch, F. (1986). Simple mathematical models for cannibalism: a critique and a new approach. *Mathematics & Bioscience*, **78**, 21–46.
- Flanders, S.E. (1968). Mechanisms of population homeostasis in *Anagasta* ecosystems. *Hilgardia*, **39**, 367–404.
- Free, C.A., Beddington, J.R. & Lawton, J.H. (1977). On the inadequacy of simple models of mutual interference for parasitism and predation. *Journal of Animal Ecology*, **46**, 543–554.
- Godfray, H.C.J. & Hassell, M.P. (1987). Natural enemies can cause discrete generations in tropical insects. *Nature*, **327**, 144–147.
- Godfray, H.C.J. & Hassell, M.P. (1989). Discrete and continuous insect populations in tropical environments. *Journal of Animal Ecology*, **58**, 153–174.
- Gordon, D.M. (1987). *Population dynamics of a host–parasitoid system with particular reference to age-structured effects*. Ph.D. thesis, McGill University, Montreal, Canada.
- Gordon, D.M. & Stewart, R.K. (1988). Demographic characteristics of the stored-products moth *Cadra cautella*. *Journal of Animal Ecology*, **57**, 627–644.
- Gurney, W.S.C. & Nisbet, R.M. (1985). Fluctuation periodicity, generation separation, and the expression of larval competition. *Theoretical Population Biology*, **28**, 886–923.
- Gurney, W.S.C., Nisbet, R.M. & Lawton, J.H. (1983). The systematic formulation of tractable single species models incorporating age-structure. *Journal of Animal Ecology*, **52**, 479–495.
- Hassell, M.P. (1978). The dynamics of arthropod predator–prey systems. *Monographs in Population Biology*, **13**, 1–237.
- Hassell, M.P. & Huffaker, C.B. (1969). Regulatory processes and population cyclicity in laboratory populations of *Anagasta kuehniella* (Zeller) (Lepidoptera: Phycitidae). III. The development of population models. *Researches in Population Ecology*, **11**, 186–210.
- McDonald, N. (1989). *Biological Delay Systems: Linear Stability Theory*. Cambridge University Press, Cambridge, U.K.
- Mariau, D. & Morin, J.P. (1972). La biologie de *Coelaenomedera elaeidis*. IV. La dynamique des populations du ravageur et de ses parasites. *Oleagineux*, **27**, 469–474.
- May, R.M. (1978). Host–parasitoid systems in patchy environments: a phenomenological model. *Journal of Animal Ecology*, **47**, 833–843.
- Murdoch, W.W., Nisbet, R.M., Blythe, S.P., Gurney, W.S.C. & Reeve, J.D. (1987). An invulnerable age-class and stability in delay-differential parasitoid–host models. *American Naturalist*, **129**, 263–282.
- Nisbet, R.M. & Gurney, W.S.C. (1983). The systematic formulation of population models for insects with dynamically varying instar duration. *Theoretical Population Biology*, **23**, 114–135.
- Perry, J.N. & Taylor, L.R. (1986). Stability of real interacting populations in space and time: implications, alternatives and the negative binomial k_c . *Journal of Animal Ecology*, **55**, 1053–1068.

- Podoler, H. (1974).** Analysis of life tables for a host and parasite (*Plodia-Nemeritis*) ecosystem. *Journal of Animal Ecology*, **43**, 653–670.
- Takahashi, F. (1973).** An experimental study on the suppression and regulation of the population of *Cadra cautella* (Walker) (Lepidoptera: Pyralidae) by the action of a parasitic wasp, *Nemeritis canescens* Gravenhorst (Hymenoptera: Ichneumonidae). *Memoirs of the College of Agriculture, Kyoto University*, **104**, 1–12.
- Trudeau, D. & Gordon, D.M. (1989).** Factors determining the functional response of the parasitoid *Venturia canescens*. *Entomologia Experimentalis et Applicata*, **50**, 3–6.
- White, E.G. & Huffaker, C.B. (1969).** Regulatory processes and population cyclicity in laboratory populations of *Anagasta kuehniella* (Zeller) (Lepidoptera: Phycitidae). II. Parasitism, predation, competition and protective cover. *Researches in Population Ecology*, **11**, 150–185.

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APPENDIX

Local stability analysis

The equations describing the ‘delayed parasitoid development’ model (Table 2) can be recast so as to express the population dynamics in terms of three equations. These describe changes in the adult host (A_h) and parasitoid (A_p) populations in terms of an auxiliary variable $\theta(t)$, related to the probability of survival against death or parasitism:

$$\dot{H}(t) = r s_e \mu_h H(t - A_h) s_{hp} e^{-\theta(t-T_{hp})} - \mu_h H(t) \quad [A1]$$

$$\dot{P}(t) = r s_e \mu_h H(t - A_p) \{1 - e^{-\theta(t-T_{jp})}\} s_{jp} - \mu_p P(t) \quad [A2]$$

$$\theta(t) = \int_{t-T_e}^t [\mu_e + \frac{k}{T_e} \ln(1 + a T_e P(t')/k)] dt' \quad [A3]$$

$$\text{where } A_h = T_e + T_\ell + T_{hp}; A_p = T_e + T_\ell + T_{jp}. \quad [A4]$$

At equilibrium:

$$\theta^* = \ell n(s_e s_{hp} r) \quad [A5]$$

$$P^* = (k/a T_e) [(r s_e s_{hp})^{1/k} \exp(-\mu_e T_e/k) - 1] \quad [A6]$$

$$H^* = \mu_p s_{hp} P^* / [\mu_h s_{jp} (r s_e s_{hp} - 1)] \quad [A7]$$

Linearized equations describing the fate of the system following small perturbations from equilibrium are derived by the method used by Murdoch *et al.* (1987) (see also McDonald (1989) for methods for this type of equation). Following Laplace transformation, these equations yield a characteristic equation whose roots must have negative real parts for local stability:

$$\det \begin{pmatrix} 1 & - \left\{ \frac{ak}{k + aT_e P^*} \right\} \left\{ \frac{1 - e^{-\lambda T_\ell}}{\lambda} \right\} & 0 \\ \mu_h H^* e^{-\lambda T} & 0 & \lambda + \mu_h [1 - e^{-\lambda A_h}] \\ -\mu_h H^* \frac{s_{jp}}{s_{hp}} e^{-\lambda T_{jp}} & \lambda + \mu_p & -e^{-\lambda A_p} \mu_L s_e s_{jp} \left[1 - \frac{1}{r s_{hp} s} \right] \end{pmatrix} = 0 \quad [A8]$$

APPENDIX FIG. 1. Stability diagram for the delayed development model (as in Fig. 4a), with indication of the points referred to in the Appendix.

‘Boundaries’ in parameter space at which a root crosses the imaginary axis of the complex λ -plane are obtained by setting $\lambda = i\omega$, and taking real and imaginary parts of [A8]. The resulting pair of equations can be solved numerically for ω and any one of the model parameters, given the values of the other model parameters. The period of oscillations on the stability boundary is $2\pi/\omega$.

The interpretation of these boundaries requires some care. Suppose we know (for example from a numerical solution of the differential equations) that, for one set of parameter values, the equilibrium is stable, e.g. the point a in Appendix Fig. 1. Then we know that moving across a boundary (for example increasing k by moving to point b in Fig. 1) corresponds to a change from local stability to instability. The angular frequency ω on the boundary yields a good approximation to the frequency of cycles near the boundary. However, in our example further increase in k leads to a second boundary crossing. To determine the stability at the point d involves determining whether, at this boundary, a new root crosses the imaginary axis moving right, or the original root returns to the negative half plane. It is in principle possible, but in practice normally laborious to decide analytically which of these possibilities occurs (MacDonald 1989 and references therein), but again a few well-chosen numerical experiments can help. In the case of the point d, we find that a new root has moved into the right-half plane, this time with a much lower frequency which bears no apparent relationship to the host development time. We interpret cycles with approximately this frequency as ‘host–parasitoid’ cycles.

Thus, the assertion in the text that there are three important types of dynamics — stable equilibrium, single generation cycles, and host–parasitoid cycles — rests on both local stability analysis and a number of numerical solutions to the full non-linear equations describing the system. It is not a strict mathematical result; for example, there is a very small part of the region for single generation cycles within which there are approximately two cycles per generation.

The analysis of the model without delayed development proceeds along similar lines: eqns [A1] and [A3] are unchanged but [A2] is replaced by a more complicated set of three equations. However, no new principles are involved in the analysis or in the interpretation of the stability boundaries.