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Population cycles of forest Lepidoptera: a maternal effect hypothesis

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Summary

1. Many species of forest Lepidoptera have cyclic population dynamics. Although there are numerous potential causes, including interactions with predators, parasitoids, pathogens, and food-plant quality, strongly density-dependent interactions are often difficult to demonstrate. Both autocorrelation analysis and attractor–reconstruction methods have recently been applied to a number of species' time series. Results suggest that complex dynamics, i.e. cycles or deterministic chaos, may be more prevalent than once thought, and that higher-dimensioned models are necessary.

2. We develop a two-dimensional difference equation model that relates the average quality of individuals to patterns of abundance. The delayed density dependence is caused by transmission of quality through generations via maternal effects. We show that the maternal effect model can produce patterns of population fluctuations similar to those displayed by one class of host–parasitoid models.

3. We review empirical evidence for maternal and quality effects in dynamics of forest Lepidoptera. We fit the maternal effect and delayed logistic models to six species of forest moths for which delayed density dependence and maternal or quality effects have been found. The maternal effect model was a good predictor of the period of the oscillations for the species that we examined. We discuss why models of this type give better fits to moth cycles than do first order models with added delays.

Key-words: delayed density dependence, population cycles, maternal effects, Lepidoptera, population quality.

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Introduction

Ecologists generally recognize two groups of animals as including species with cyclic population dynamics: small mammals (and their predators) and forest insects. Among the insects the most striking examples are from Lepidoptera of the temperate zone. A recent review (Myers 1988) lists 18 forest moth species whose population densities are known to oscillate. Unravelling the causes of such oscillations is not only of theoretical but also of practical interest, since many of these moths are economically important forest pests.

In one sense, of course, we *know* what causes population cycles: true oscillations can only arise via density-dependent regulation, and only if such regulation involves a time delay (Hutchinson 1948; Caswell 1972; May 1973). In other words, the system oscillates only if there are delayed negative feedbacks (Berryman 1987). In univoltine insects, like moths of the temperate zone, we expect that regulatory processes will involve time delays; this allows such systems to be modelled with difference equations (Royama 1981). However, the role of density dependence in population regulation remains controversial (e.g. Wolda 1991; Berryman 1991) despite the suggestion that the long-term persistence of populations by itself implies some form of regulation (Royama 1977).

Are forest Lepidoptera regulated by density-dependent processes? For animal populations in general, the answer to such a simple question seems to be nearly as elusive today as it was decades ago. There are several methods available with which one can test for density dependence, but all apparently have inherent problems when applied to available data. One can, for example, analyse life-table data (Dempster 1983) using methods originally formulated by Morris (1959) and modified by others. Alternatively one can look for density dependence in a time-series (Bulmer 1975; Pollard, Lakhani & Rothery 1987). Such tests have intuitively simple

logical bases but suffer from statistical difficulties (Solow 1990). When applied to forest moths, results of these methods are often mixed. For example, reviews of published life tables by Dempster (1983) and by Stiling (1987, 1988) were done specifically to assess the evidence for regulation in moths and other insects. Both authors reached similar conclusions, i.e. that the majority of species have no clearly demonstrable density-dependent regulatory factor. The earlier paper by Stiling was specifically concerned with the effects of parasitoids, an often-invoked explanation of moth cycles. Dempster's analysis (of 24 Lepidopteran species) suggested that the few cases of parasitoids acting as regulating agents were equivocal at best, and that all other forces shown to be density-dependent (reduced fecundity, starvation) involved intraspecific competition, which would only be important when the population approached the carrying capacity. In view of their results, both authors suggested that 'ceiling' models might be more appropriate for describing insect dynamics. Other factors besides natural enemies have been suggested as possible cases of moth cycles, for example changes in food-plant quality (Rhoades 1985), but again the evidence is most often equivocal (Myers 1988; Larsson 1989).

In contrast, recent statistical analysis of insect time-series suggests that many species are affected by some form of delayed regulation. Two studies have addressed the question of whether or not insect time-series are indicative of complex dynamics, i.e. cycles or deterministic chaos. Witteman, Readfern, & Pimm (1990) used autocorrelation analysis and attractor reconstruction methods to analyse a number of vertebrate and invertebrate time series. Turchin (1990) used partial autocorrelation functions and a general second-order nonlinear model to analyse time-series from 14 insect species, nine of which were forest moths included in the Dempster and Stiling papers. Turchin (1990) found that seven moth series had partial autocorrelation functions characteristic of a second-order autoregressive process. In other words, while it might be true that usual life-table regression methods did not detect any apparent regulation, such regulation was operating but with a lagged effect. Witteman *et al.* (1990) drew the same conclusion. They showed that several species' time-series indicated a low dimensional attractor, usually two or three dimensions being sufficient to embed the system. Further analysis by Turchin & Taylor (1992) supported this conclusion. The importance of delayed regulation has been discussed before, both theoretically and in individual cases (e.g. Varley 1949; Royama 1977; Hassell 1985; Berryman 1987; Murdoch & Reeve 1987), but Witteman *et al.* (1990) and Turchin (1990) were the first to broadly apply analytical methods of time-series analysis for the sole purpose of determining the proper dimension with which to begin modelling. Turchin & Taylor (1992) then took the next logical step in applying time-series modelling to populations with delayed regulation; they used response-surface fitting to reconstruct the dynamic patterns of these species. Other researchers working with moths subsequently applied Turchin's (1990) methods to test for delayed density dependence in insect time-series (Liebhold & Elkington 1991; Woiwod & Hanski 1992).

It must be mentioned here that the results discussed above must be interpreted with some caution. The reason for this lies in the nature of the data sets being analysed: ecological time-series are not only inherently 'noisy', but are also usually of such short duration that it is difficult to draw conclusions with much statistical significance. For example, Ellner (1991) demonstrated how a short, noisy time-series involving simple density dependence might resemble deterministic chaos when the attractor-reconstruction method is used with arbitrary time-lags. Methods exist for detecting a chaotic signal from a noisy time-series (Sugihara & May 1990), but again the available series are usually too short for such techniques to be reliable (Turchin & Taylor 1992). However, this limitation of the size of available data sets cuts both ways in the debate about density dependence: for example, the results of both Dempster (1983) and Stiling (1987, 1988) have been shown to be prone to Type II errors (Hassell, Latto & May 1989; Woiwod & Hanski 1992). One point seems, to us, to be both clear and of great importance: although constructing an actual attractor may be problematic, the *dimension* with which to begin is often greater than one, with two dimensions usually sufficient to embed the system. With univoltine species (which have natural time-lags in multiples of 1 year), it is not likely that truly first-order dynamics will resemble a second-order process unless the population is subject to external noise that is autocorrelated (Royama 1981).

Why is dimension so important? Consider simple one-dimensional models of the form

$$N_{t+1} = N_t F(N_t), \qquad \text{eqn 1}$$

such as the well-known logistic or Ricker equations. Ecologists are aware of the fact that such one-dimensional discrete models can display complex behavior, e.g. cycles with period 2, 4, 8, $\ldots = 2^k$ followed by deterministic chaos (May 1976; Lauwerier 1986a). In contrast, assume that the population is regulated by factors other than density N_t. In two dimensions:

$$N_{t+1} = N_t f(X_t) \qquad \text{eqn 2}$$
$$X_{t+1} = \varphi(N_t, X_t).$$

Eliminating X_t, equation 2 can be written in time-delay form:

$$N_{t+1} = N_t F\,(N_t, N_{t-1}). \qquad \text{eqn 3}$$

The delayed effect of N_{t-1} on N_{t+1} can be caused by a variety of mechanisms, such as interactions with predators and parasites, age distribution, or delayed effects of some other environmental variable, for example the quality of food plants.

The behaviour of equation 3 can be quite different from that of equation 1 (Levin & May 1976; Pounder & Rogers 1980; Royama 1981; Lauwerier 1986b). For example, equation 3 can often display cycles with any biologically realistic values of the parameters, and the cycle period may not progress through numbers in the sequence 2^k; in principle, all values (including irrational and fractional) are possible (Beddington, Free & Lawton 1975; Lauwerier 1986b). The important point for our purpose is that lower values of the parameters are necessary for cycles as the dimensionality of the model increases (Guckenheimer, Oster & Ipatkchi 1977).

With the proper dimension known, one can use a linear approximation (i.e. a *p*-order autoregressive model) for short-range prediction (Royama 1977), and methods such as Turchin & Taylor's (1992) response surface fitting seem to be fairly successful in predicting when one should find limit cycles or deterministic chaos. However, reconstructive methods do not help us determine the *biological* nature of the significant higher dimensions.

In our view, the results of Turchin (1990), Turchin & Taylor (1992) and Witteman *et al.* (1990), when coupled to those of Stiling (1987, 1988) and Dempster (1983) present something of a conundrum. On the one hand, there is good evidence that a biologically important second dimension exists in many univoltine insect species. On the other hand, strongly density-dependent trophic interactions are often difficult to demonstrate, whether that interaction is with natural enemies (Hassell 1985) or with food plants (Myers 1988; Larsson 1989).

In this paper we offer a two-dimensional model that does not involve a trophic level interaction, yet is based on a well-known aspect of the biology of forest moths. Our proposed second dimension is the average quality of individuals within a population. The model differs formally from other population quality models in that the transmission of quality between generations is via maternal inheritance. In our analysis of the model we show explicity that maternal effects can produce cycles that might be indistinguishable from those caused by a host–parasitoid interaction. We review evidence from empirical work suggesting that maternal quality effects are important regulatory forces in moth populations. Finally, we fit the maternal effect model to empirical data from six species of forest moths. The model is shown to be a very good predictor of the cycle period for all the species that we examined. We give a simple mathematical argument for why models of this type give better fits than first-order models with time delays.

The Model

The assumptions

1. Fecundity of females, and the survival of immatures to adulthood, are functions of individual quality. Normally this quality will relate to energy: the amount that a female moth has stored for reproduction, or the average size or weight of larval/pupal stages. However, other quality measures can be used; these might include the level of viral or protozoan parasites transmitted transovarially, or the level of disease resistance in immatures due to maternally influenced physiological vigour.

2. Offspring quality is a function of maternal quality. In other words, there are maternal effects.

3. Average quality is influenced by population density in the current generation. This can result from intraspecific competition for limited resources, or from other effects in the current generation, for example shifts in food quality due to high levels of herbivory.

The exact way in which the average individual quality is measured need not concern us here; some simple measures might be the dry weight of female pupae, or the caloric content of individuals exploded in a calorimeter. Our requirement is simply that mother–daughter relationships can be made, and that fecundity can be expressed as a function of the quality variable. Hereafter, for the sake of brevity, we will refer to average individual quality simply as 'quality'.

The equations reflecting the above assumptions have the form:

$$N_{t+1} = N_t f(x_t) \qquad \text{eqn 4}$$
$$x_{t+1} = \varphi(x_t, N_{t+1}),$$

where f is a monotonically increasing function of x_t and describes the net reproductive rate of an individual of quality x, and $\varphi(x_t, N_{t+1})$ is an increasing function of x (the maternal effect) and a decreasing function of N_{t+1} (as intraspecific competition for resources increases). Note that the argument N of the second equation is evaluated at the same generation as x on the left side of the equation. This is due to our assumption that quality is affected by density in the current generation. Mathematically, as will be seen, this is a crucial assumption of the model. In this respect equation 4 differs from usual discrete-time population models where the variables N and x would be interpreted as densities of interacting populations (Beddington, Free & Lawton 1976). Note that the model can be rewritten in the time-delay form in equation 3 by substituting the right side of the first equation for N_{t+1} in the second equation, then eliminating x_t.

In the absence of the maternal effect φ is independent of x, and equation 4 reduces to the form of equation 1, with

$F(N) = f(\varphi(N))$.

Thus, the presence of the maternal effect creates the delayed density dependence. Equilibrium density and quality N^*,x^* can be found as the roots of

$$f(x^*) = 1$$
$$\varphi(x^*,N^*) = x^*.$$

The model in equation 4 is analysed (see Appendix 1) by the method of local stability analysis. As shown, two parameters,

$$a = \varphi'_x > 0$$

and

$$b = -f'_x\varphi'_N N^* > 0$$

where subscripts refer to partial derivatives evaluated at equilibrium, control the dynamic behaviour through the roots of the characteristic equation

$$\lambda^2 - (1 + a - b)\lambda + a = 0.$$

Properties of the model

In analysing equation 4 it will be convenient to discuss a specific example. Consider the following parameterization:

$$N_{t+1} = N_t R\frac{x_t}{k + x_t} \qquad \text{eqn 5}$$

$$x_{t+1} = x_t M\frac{S/N_{t+1}}{p + S/N_{t+1}}$$

The parameter R represents the maximum reproductive rate given any quality x, and M is the maximum possible increase in average quality. Constants k and p control the rates of increase to the asymptotes R and M. The S term represents the total amount of resource available in the environment; we assume that this is constant each generation.

We divide the numerator and denominator of equation 5 by S; then by expressing N and x in the appropriate units we can eliminate the other two parameters k and p so that equation 5 becomes

$$N_{t+1} = N_t\frac{Rx_t}{1 + x_t} \qquad \text{eqn 6}$$

$$x_{t+1} = x_t\frac{M}{1 + N_{t+1}}$$

Stability analysis of equation 6 shows that non-damping oscillations occur whenever the parameter R is greater than unity, assuming that M is also greater than unity. The period of the oscillations in the linearized form of equation 6 is a function of

$$b = (1 - 1/R)(1 - 1/M).$$

Thus, if $M \gg 1$ the length of the period is determined only by the maximum rate of increase R. In this model low values of R lead to longer cycles.

Figures 1 & 2 show some typical behaviour of

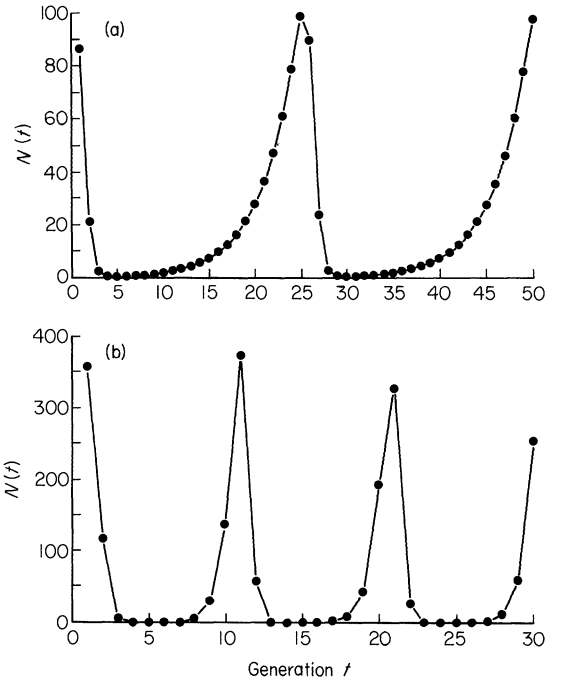

Fig. 1. Typical behaviour of the maternal effect model (equation 6) shown in time-series plots: (a) $R = 1{\cdot}3$, $M = 10$; (b) $R = 3{\cdot}0$, $M = 10$.

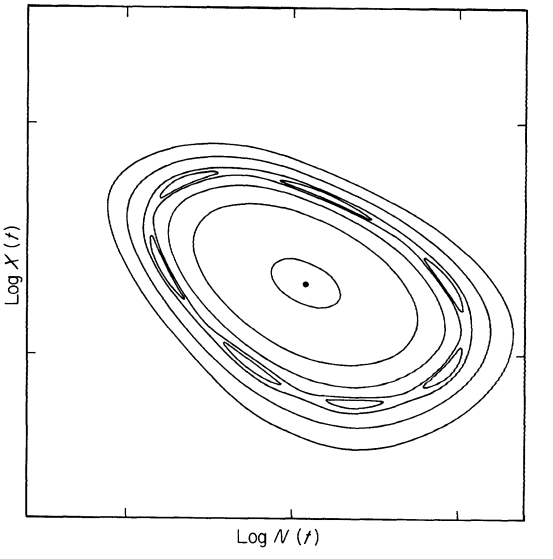

Fig. 2. Typical phase-plane portrait of the maternal effect model 6, shown in logarithmic scale. Each ellipse corresponds to a unique set of initial conditions. Note the formation of 'island chains' for some initial values.

model 6 displayed in both time series and phase-plane plots. As can be seen from the phase diagram, the cycles are neutrally stable, i.e. the amplitude and period of the cycles are dependent upon initial conditions. In this way, equation 6 is similar to the familiar Lotka-Volterra predator and prey equations, and also to several other two-dimensional models (Beddington *et al.* 1975; Anderson & May 1980; Lauwerier & Metz 1986). The cycles usually have fractional values for the period, and the system

is not precisely periodic, as in a continuous model. Note also in the phase diagram the formation of 'island chains' for some initial conditions, a feature common to this type of two-dimensional discrete map (Lauwerier 1986b; Lauwerier & Metz 1986).

Models that generate neutral cycles are of course pathological from a biological viewpoint (May 1976). Modification of equation 6 to produce true 'limit' cycles can be done in any number of ways by adding one or more parameters. Available data do not allow us to distinguish between different modifications, but our main conclusions do not depend on the exact form of the functions. A functional sensitivity analysis that begins with model 6 is given in Appendix 2.

In comparing the behaviour of the maternal effect model to that of other published two-dimensional discrete models, we found that similar dynamics are produced by the following class of host-parasitoid equations:

$$N_{t+1} = \lambda N_t f(P_t) \qquad \text{eqn 7}$$
$$P_{t+1} = N_t - 1/\lambda\, N_{t+1}$$

where N is host density, P is parasitoid density, λ is the maximum per capita increase per generation in the absence of parasitoids (analogous to R in our model) and $f(P)$ is a decreasing function of P. The above general form was developed in Hassell & May (1973); further analysis can be found in Lauwerier (1986b) and Lauwerier & Metz (1986).

Models 6 and 7 have in common several properties of biological interest. First, and perhaps most important, is that *cycle periods have an absolute minimum of six*. This result was stated as a theorem by Lauwerier & Metz (1986) for equation 7; we extend this result to models of the form of equation 6, given certain restrictions on functions f and φ (see Appendix 2). The minimum six-cycle is in perfect agreement with observation: to our knowledge no forest moth has a period less than six (Myers 1988), in contrast to mammalian cycles.

Second, in both models *cycle periods decrease as the growth parameters (R and λ) increase*. This pattern is opposite to that of some other delay models such as the discrete predator–prey (Beddington, Free & Lawton 1975) or delayed logistic (Levin & May 1976), for example (Taneyhill 1993).

Empirical evidence

QUALITY AND MATERNAL EFFECTS IN MOTHS

Population quality as a factor in the dynamics of moth populations is generally known from the work of Wellington (1957), who studied the western tent caterpillar *Malacosoma pluviale*. Larval tent caterpillars could be classified into various types, depending on the amount of yolk provided by the female moth. These larval types displayed large differences in their survival, behaviour, disease resistance and dispersal ability (Wellington 1960). The survival and eventual fecundity of the larval forms was influenced by maternal quality. The highest quality larvae (Type I in Wellington's terminology) laid more eggs, and more of the high-quality type, when all larval types were fed to repletion (Wellington 1965).

The biology of the tent caterpillar is in broad outline typical of many forest Lepidoptera. Females generally lay clutches that number in the several hundreds. The first eggs laid often receive a disproportionate amount of yolk, and quality of the larvae often depends on the amount of maternal provisioning. Larval quality influences factors such as weight at pupation, which affects subsequent fecundity (Szujecki 1987).

Researchers working with other forest moths and insects found Wellingtonian effects in their species as well. Greenblatt & Witter (1976) found the same kind of larval quality types in the tent caterpillar *Malacosoma neusstria*; Campbell (1962), working on the spruce budworm *Choristoneura fumiferana*, found larval quality effects associated with various forms of the maternal X-chromosome. Campbell's work recalled some earlier study by European researchers who noticed different larval or adult moth forms associated with forest insect outbreaks; for example, Franz (1941, 1942, 1965) with the budworm *Choristoneura* (= *Cacoecia*) *murinana*, and Engel (1942) with the pine looper *Bupalus piniarius*. As did Greenblatt & Witter (1976), Laux (1962) found different larval types associated with outbreak and non-outbreak populations of *Malacosoma neusstria*.

Differences in larval quality forms within outbreak populations have also been seen in another forest insect, the sawfly *Acantholyda nemoralis*. Novikova (1969) described two forms of sawfly in this species: an early form with larvae less susceptible to natural enemies, and a late form that had more rapid reproduction and a higher proportion of females. Proportions of the two forms in the population changed as numbers approached outbreak levels. Sawflies, it should be noted, are the major forest insects besides lepidopterans that display cyclic or outbreak dynamics (Larsson & Tenow 1984).

Recently there has been renewed interest in the Wellingtonian ideas. Myers (1990), working with *Malacosoma*, has shown that transplanted tent caterpillars tend to oscillate in phase with the parent population. Rossiter 1990, 1991a,b) has made quantitative genetic studies of the gypsy moth *Lymantria dispar* in regard to maternal effects and population regulation, and has suggested that maternal effects are an important factor in the cause of outbreaks (Rossiter 1991c).

It is worthwhile here to mention the gypsy moth in greater detail, since it is this species that has perhaps been most extensively researched with regard to the

type of effects that we discuss in the model. We have found in the literature evidence for all of the relationships postulated by the maternal effect equations. For example, Rossiter (1990) found that larval quality and various life-history traits were influenced by the amount of maternal provisioning (function $\varphi(x)$), and that the rate of larval development was influenced by density in the current generation (function $\varphi(N_{t+1})$). The last result was also found by Campbell (1978). Capinera & Barbosa (1976), in laboratory studies, found that larvae from small eggs go through more instars and lay more, smaller eggs (function $f(x)$); they discuss the Wellington hypothesis. Finally, both Lance, Elkington & Schwalbe (1986) and Leonard (1970) proposed population quality hypotheses for the gypsy moth; for example, from Leonard: 'Evidence is presented to suggest that the gypsy moth . . . is numerically self-regulating through a shift in quality of individuals induced by changes in nutrition.'

More discussion of empirical research on maternal and quality effects may be found in the next section, which considers moth species individually.

MODEL FITTING

In this section we fit the maternal effect model to the moth species listed in Table 1. We chose species on the basis of the following criteria: (i) only univoltine forest moths were considered; (ii) at least one reference for each moth has suggested that the species is regulated by delayed density dependence; (iii) quality and/or maternal effects have been suggested or demonstrated for each species; (iv) a time-series exists for that species that is adequate for use in estimation of model parameters.

(Some readers may wonder why we did not choose to include the larch moth *Zeiraphera diniana* in our analysis. The reason was that we had *a priori* evidence that *Z. diniana* should be described by a higher order model (Royama 1977; Turchin & Taylor 1992), with N_{t+1} a function of N_t, N_{t-1}, and N_{t-2}. We will comment further in the Discussion section).

As a contrasting two-dimensional non trophic-level model, we also fit the data to the well-known delayed logistic equation:

$$N_{t+1} = N_t \exp(r_0(1 - N_{t-1}/K) \qquad \text{eqn 8}$$

This model has been recommended as an all-purpose model for univoltine species affected by delayed density dependence (Berryman, Millstein & Mason 1990).

Estimation of Parameters

Before fitting the models to empirical data we must discuss several potential pitfalls. To our knowledge there are no experimental results that would allow us to make independent parameter estimates for the f and φ functions. The alternative is to take advantage of the embedding theorem of Takens (1981) for generic dynamic systems and use lagged densities as the predictor variables, i.e. rewrite the model so that N_{t+1} is a function of N_t and N_{t-1}. This method of parameter estimation is very convenient but contains its own problems (Morris 1990). In fitting a non-linear model to the data the difficulty of convergence of an iterative regresion procedure increases with the dimension (i.e. the number of parameters). For the maternal effect model this is more of a problem than for the host–parasitoid model in which the growth parameter λ does double-duty. In addition the predictor variables, being lagged densities, will contain errors of the same magnitude as those of the response variable, violating one of the major assumptions of regression analysis.

With these caveats in mind, we proceeded as follows. We fit model 6 in its time-lag form (Appendix 1) via a non-linear iterative regression (Procedure NLIN of SAS, SAS Institute 1989). Since the dynamics of the model are only weakly dependent on the parameter M when $M \gg 1$, we concentrated on estimating the parameter R. For some series the iterative procedures used by NLIN did not easily converge. In these cases we used the one-parameter model

$$N_{t+1} = RN_t(1 + N_{t-1} - 1/RN_t)^{-1} \qquad \text{eqn 9}$$

Table 1. Predicted dynamics of six species of forest moths, using model 11. Superscripts refer to references from which the parameter R was estimated by the non-linear regression method. Parameters β, x_{min}, and M estimated by computer simulation

Species	Ref.*	R	M	x_{min}	β	T (predicted)	T (actual)
Choristoneura fumiferana	6	1·34	2·0	0·02	1·005	34	30–38
Hyphantria cunea	4	1·64	2·75	0·5	1·0025	12	11·8
Lymantria dispar	2	4·05	12·0	0·03	1·0025	7–8	7–8
Epirrita autumnata	1	4·68	3·0	0·04	1·0025	9	9
Bupalus piniarius	5	4·8	2·5	0·0375	1·0025	8	8
Acleris variana	3	5·12	5·0	0·02	1·005	7–8	7

* References: 1, Andersson & Jonasson 1980; 2, Bess 1961; 3, Morris 1959; 4, Morris 1964; 5, Schwerdtfeger 1941; 6, Royama 1984.

Table 2. Predicted dynamics of forest moths, using the time-delayed logistic model $N_{t+1} = N_t \exp(r_0(1 - N_{t-1}/K))$. Parameters estimated by a linear regression using the same time series as in Table 1

Species	r_0	K	Predicted dynamics
Choristoneura fumiferana	0·325	65	Stable
Hyphantria cunea	0·415	5·53	Stable
Lymantria dispar	1·32	2620	7-cycle
Epirrita autumnata	1·54	178	8-cycle
Bupalus piniarius	0·308	154	Stable
Acleris variana	1·13	141	6-cycle

which, we found converges to values quite close to those estimated from equation 6. Equation 9 corresponds to equation 7 with the function $f(P_t)$ equal to $(1 + P_t)$; it would describe a neutrally stable host–parasitoid model. For the delayed logistic, we used a linear regression of the log rate of change $r_t \equiv \log_e(N_{t+1}/N_t)$ as a function of N_{t-1} to estimate the parameters r_0 and K.

Model 6 is, of course, neutrally stable; thus the amplitude, and to a lesser extent the period of the resulting cycles will be dependent on initial conditions. Since we do not have data to suggest the proper functional form for $\varphi(x,N)$ we adopted alternative procedures. First, the estimates of R from the above method were used in simulations of equation 6. The initial conditions were varied until the amplitude of the resulting oscillations matched the observed amplitude for each species' cycle. The reported period of the cycle was then read from the simulation. This method sacrifices possible comparisons of amplitude in order to test predicted period of the cycles. True limit cycles can be generated by the model

$$N_{t+1} = N_t \frac{Rx_t}{1 + x_t} \qquad \text{eqn 10}$$

$$x_{t+1} = x_t^{\beta} \frac{M}{1 + N_{t+1}}$$

$$x_t \geq x_{\min}$$

which adds the assumptions that the maternal effect is not linear and that there is a lower limit to the average individual quality. This model is biologically more realistic but is not everywhere differentiable; thus, we used simulations to estimate the new parameters $x_{\min}$ and β.

We compared published values for the period of each species to those predicted by both the maternal effect and delayed logistic models. Results of the analysis are shown in Table 1. Below we briefly discuss each moth individually.

Choristoneura fumiferana. The spruce budworm, with its long-period oscillation (Royama 1984) is one of the most interesting cases of all the known cyclic moths. Population quality effects for this moth have been noted by Campbell (1962), who found variations in fecundity, egg size and egg weights correlated with several forms of maternal X-chromosome; Neilson (1963) studied disease resistance and physiological vigour in this species; Thomson (1958) found that microsporidian parasites could be transmitted transovarially.

Royama (1984) has concluded that the survival of late instar larvae is the factor driving oscillations in *C. fumiferana*. Suggested causes of the oscillating mortality in old larvae were parasitism and an unknown 'fifth agent'.

The long cycle period of *C. fumiferana* makes estimation of the parameter R somewhat difficult, since we only have census data for slightly less than one cycle (Morris 1963; Royama 1984), and this time-series actually consists of two from different localities. The regression converged to values of 1·05 and 2·54 for the two parts of the data set. The true value is probably somewhat between these two extremes. Turchin (1990) used the following general model for the purpose of extracting the proper dimension from a time-series:

$$N_{t+1} = N_t \exp(r_0 + \alpha_1 N_t + \alpha_2 N_{t-1}) \qquad \text{eqn 11}$$

Using log-transformed data, equation 10 estimated r_0 as 0·29 for *C. fumiferana*, or $R = 1{\cdot}34$. This is almost identical to the estimate from the logistic model and, in addition, to the weighted mean of the estimates from the two parts of the time-series by the maternal effect model (1·38 and 1·3, respectively; see also Fig. 3b). The logistic equation predicts stability for the budworm.

The estimate of R for this species is quite interesting in itself, being quite low for the intrinsic rate of reproduction for an insect. One possible explanation is that the budworm is simply tracking changes in the quality of its food plant, which happens on a slow time-scale (Ludwig, Jones & Holling 1978), making the time-series look like a long-period linear oscillator (Royama 1977).

Hyphantria cunea. Historical records indicate that the fall webworm has been cycling for more than a century with an average period of about 12 years (Morris 1964). This is exactly what is predicted by the maternal effect model. Quality effects on population dynamics of the webworm have been documented by Morris (1971, 1976). An important part of this work was the demonstration that the web-

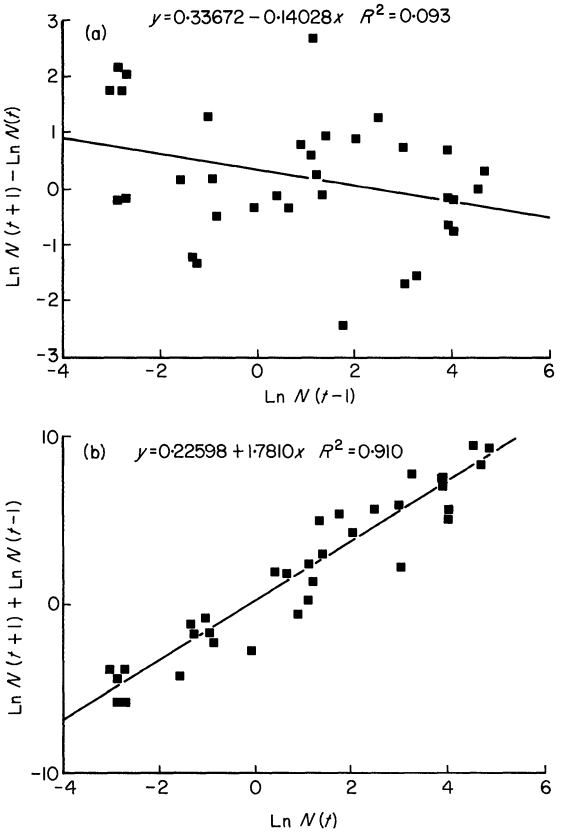

Fig. 3. Two models of delayed density dependence for the spruce budworm data (Royama 1984). (a) Logistic-type model with delay in which the log rate of change is a function of log density in the previous generation. (b) Oscillator type model in which the change in log rate of change (second difference of the log) is a function of log density. The slope estimates $(2-k)\ln N(t)$; i.e. the ordinate has $2\times \ln N(t)$ added so as not to confound errors by also having $\ln N(t)$ in the dependent variable.

worm larvae's ability to encapsulate parasites was a function of larval quality (Morris 1976). The delayed logistic equation predicts stability for the webworm also.

Epirrita autumnata. Both quality and maternal effects have been demonstrated in the autumnal moth (Haukioja & Neuvonen 1985, 1987). Althugh available time-series are quite short (Andersson & Jonasson 1980), there is an extensive record of outbreaks (Tenow 1972), and analysis of the outbreak pattern confirms the 9–10 year period (Haukioja *et al.* 1988). While the maternal effect model does predict the correct cycle period, the value from the logistic equation is only one less (see Haukioja *et al.* 1988).

Bupalus piniarius. The pine looper is a well-studied moth in Europe, with a number of available time-series (Schwerdtfeger 1941; Klomp 1966; Barbour 1988). Analysis of numerical records of *B. piniarius* suggests that the pine looper is regulated by strong delayed density dependence (Barbour 1988). The fecundity of female moths is a function of pupal weight, and the density of larvae influences the average weight at pupation (Klomp & Gruys 1965). The density of larvae in one generation has been shown to affect the survival of eggs and larvae in the next generation (Klomp 1966). Weights of female pupae oscillate of eggs and larvae in the next generation (Klomp 1966). Weights of female pupae oscillate slightly out of phase with the oscillations in numbers, with peak average weight preceding a population peak (Barbour 1988).

For our analysis we used the data of Schwerdtfeger (1941), which is one of the longest moth time-series available. The data are those reported by Varley (1949), the original data unfortunately being apparently lost. It is generally agreed (Den Boer 1990) that any inaccuracies in the data as reconstructed by Varley (1949) are random with respect to the precise values of the original data set. Turchin's (1990) partial autocorrelation analysis clearly indicated a second-order process apparent in the Schwerdtfeger data.

Analysis of this data set for *B. piniarius* illustrates the relevence of the maternal effect and other second-order models to the resolution of the current debate on the detection of density dependence (Berryman 1991; Wolda 1991; Reddingius & Den Boer 1989). The same time-series analysed by us and by Turchin (1990) has been thoroughly studied by Den Boer (1990), using the best available statistical methods for detection of density dependence in a series of population censuses. This analysis suggested that inferring density-dependent regulation from Schwerdtfeger's data was at best problematical. However, all of the tests used by Den Boer (1990) assume that the data are from a first-order Markov process; i.e. the regulation is direct, not delayed. But as Turchin's (1990) analysis has shown, that data strongly suggest a second-order process.

Acleris variana. The black-headed budworm is one of the first organisms for which delayed density dependence was demonstrated (Morris 1959); more recent analysis confirms the original conclusion (Berryman 1986; Turchin 1990). Morris' (1959) analysis suggested that the delayed regulation was caused by parasitoids. Varley, Gradwell & Hassell (1973), however, have shown that delayed regulation is actually stronger for another unknown cause; thus, we include *A. variana* in the analysis. Even if the regulation is entirely due to the action of parasitoids, the maternal model should fit the dynamics if the interaction with the parasitoid can be described by equation 7.

Lymantria dispar. The gypsy moth has perhaps been the most well-researched moth with regard to quality effects (Leonard 1970; Capinera & Barbosa 1976; Capinera, Barbosa & Hagedorn 1977; Lance *et al.*

1986; Rossiter 1990, 1991a) and maternal effects (Rossiter 1991b). Maternal effects on quality have been demonstrated via mother–daughter plots of a quality variable (Rossiter 1991b). Discussion in Lance *et al.* (1986) and Rossiter (1991c) could be considered as verbal descriptions of the maternal effect model. Note that the logistic model also predicts the correct period.

Summary

The maternal effect model predicts cycle periods that are close to the observed value for all six species. The logistic model predicts stability for three species. Of the remaining three, two (*A. variana* and *E. autumnata*) are predicted to cycle with periods 1 year less than the observed value. Both models accurately predict the period for *Lymantria dispar*.

The computer simulations yielded two new predictions. First, only a very small bit of non-linearity in the maternal effect is necessary to induce limit cycles. Second, the parameter M often seems biologically realistic, being usually around 2–3, i.e. one order of magnitude in the natural logarithm.

The results strongly suggest that a maternal effect model would be a better predictor of the dynamics than would a logistic-type model with a time delay. As we have stated above, a host–parasitoid model of the form of equation 7 should be able to do as well. Clearly, there is something in the mathematical form of the two models that more realistically describes the dynamics. We offer the following argument for why this is so.

Throughout this paper we have used the terms 'cycle' and 'oscillation' interchangeably. Most population biologists might do the same. Let us return, however, to a statement made above concerning models 6 and 7: the fact that the period of the cycles increases as the value of the growth parameter decreases. In logistic models the opposite is true: increasing r increases the period of the cycles. An approximation to models 6 and 7 can be written as

$$\frac{N_{t+1}}{N_t}\Big/\frac{N_t}{N_{t-1}} = f(N_t)$$

which in words means that the population density N_t affects the change in growth *rate* (not density) (Ginzburg 1993). The above equation with population densities expressed in logarithmic scale can be written

$$N_{t+1} - 2N_t + N_{t-1} = -kN_t$$

which is a discrete analogue of the familiar differential equation for an undamped linear oscillator (e.g. a mass on a spring with no friction). The parameter k represents the magnitude of the effect that the population density has on change in growth rate; it is analogous to a restoring force in our physical analogy. This is how the growth parameters affect cycles in the maternal and host–parasitoid models. But since in logistic models with delay the opposite is true, these models are not true 'oscillators' in the above sense. As Fig. 3 suggests, oscillator models should yield better statistical fits for the species we discuss.

Discussion

The possibility of natural populations undergoing complex dynamic changes has been discussed ever since the discovery that even the simplest models can display chaotic behaviour (May 1976). Finding cases of complex dynamics in the real world, however, has proven to be a more difficult task. Why should this be? In our opinion two issues — dimensionality and mechanism — are crucial to our understanding of populations as dynamic systems.

Regarding dimension, almost all ecologists are aware of the study of Hassell, Lawton & May (1976), the first attempt to fit a number of species to a non-linear model and determine whether that species should be stable, oscillatory or chaotic. The well-known result, now reproduced in several textbooks, was that virtually all of the real-world populations had parameter values placing them within the exponentially stable or oscillatory stable regions of the parameter space. In a later work, Bellows (1981) reached the same conclusion. Yet is must be emphasized that the results were based on the *assumption* of a one-dimensional model, i.e. $N_{t+1} = f(N_t)$. The authors of these studies knew as well as anyone that their results would not be valid if the true dimension of the system were higher; for example Hassell *et al.* (1976) mentioned this caveat for the case of the larch budmoth *Zeiraphera diniana*, perhaps the most clear-cut case in the world of a truly cyclic moth. Yet this warning about choosing the proper embedding dimension was not fully appreciated until much later. In the words of Pimm (1991): 'Ecologists in general (including me) stopped looking for complex dynamics in real populations.'

It is our guess that the reason for not considering higher-dimension models at the outset was the prevailing opinion that delayed density dependence must arise from a trophic interaction, and that the evidence for such was and is difficult to demonstrate (Hassell 1985). Consider the arguments proposed by Dempster (1983): in most lepidopteran studies, density-dependent interactions with predators or parasitoids are unclear, while all other forces shown to be (direct or delayed) density-dependent, such as reduced fecundity, would only be important when the population size approached the carrying capacity. However, if the maternal effect model is correct, the above conclusion does not follow; rather an individual's fecundity at any population size is a function of its quality, the quality of the last generation, and

the amount of resource available in the environment.

It has already been pointed out (Royama 1981) that one cannot infer a causal density-dependent relationship from correlations with mortality. If population quality and maternal effects are the driving force behind moth cycles, then the usual mortality agents (pathogens, parasitoids) may be the *proximal* causes of an individual's death; yet that individual might be destined to die anyway because it is of low quality. Of course, the converse is probably true: if cycles are really caused by interactions with natural enemies, then one might find correlations of mortality with quality.

Besides interactions with natural enemies, the other quality mechanism considered important in insect population dynamics is the change in host-plant food quality due to levels of herbivory. Such changes have been noted for many forest moth systems (Lesniak 1973; Schultz & Baldwin 1982; Rhoades 1985). The two types of quality change thought to be important are changes in nitrogen content (White 1974; Tuomi *et al.* 1984) and in levels of secondary compounds (Rhoades 1985; Haukioja *et al.* 1988). The food-quality hypothesis remains speculative due to the difficulty of obtaining conclusive evidence (Myers 1988; Larsson 1989). As we noted in the model assumptions, the maternal effect equations themselves do not necessarily imply that the average quality be wholly within the body of the organism; thus, the change in food-plant quality can be reflected in the model by the argument N_{t+1} of function φ, the population in the current generation affecting average quality. There is experimental support for this (e.g. Valentine, Wallner & Wargo 1983). However, the change in quality must then be passed to the next generation (argument x of function φ to induce sustained cycles. This can occur either within the food plants, in the herbivore via maternal effects, or both, in which case the species might show evidence of being embedded within a third-order attractor. Changes in plant quality have been demonstrated to be capable of generating of complex dynamics when coupled to trophic interactions (Foster, Hunter & Schultz 1992). For most species, however, two dimensions seem to be sufficient to describe the system. A significant exception is the larch moth *Zeiraphera diniana* (Royama 1981; Turchin & Taylor 1992; Baltensweiler & Fisclin 1988); the third dimension found for this moth may indeed be indicative of a strong food-quality effect.

The linking of maternal and trophic causes can also be done with viral or protozoan parasites that are transmitted transovarially. We mentioned this in our short discussion of the spruce budworm (Thomson 1958). The maternal effect model thus provides an alternative framework for describing interactions with these natural enemies as well.

Since we have shown that population quality and maternal effects can generate cycles indistinguishable from those caused by interactions with natural enemies, only empirical work can tell us what the true mechanisms are. We have no illusions about this being an easy task: one only need look at the controversy over endogenous vs. exogenous explanations for cycles in the red grouse in Great Britain (Cherfas 1990). In our view, the only way to distinguish mechanisms is to perform manipulative experiments. In testing population quality hypotheses, individuals should be removed from the influence of their normal extrinsic mortality agents. As we noted above, we are aware of only one such experiment, that of Myers (1990) with *Malacosoma pluviale*. Since the results of that study supported an intrinsic hypothesis, and since the type of maternal effects first described by Wellington (1957) seem to be ubiquitous in temperate-zone forest Lepidoptera, the maternal effect model must be considered as a strong contender for explaining cycles in forest moths.

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Appendix 1

LOCAL STABILITY ANALYSIS

Beginning with the general model (equation 4), we first change the variables to logarithmic scale:

$$u = \ln N \qquad \text{eqn 12}$$
$$v = \ln x$$

and define new functions

$$f(v) = \ln f(e^v) \qquad \text{eqn 13}$$
$$\varphi(v,u) = \ln\varphi(e^v,e^u)$$

We thus have in the new variables

$$u_{t+1} = u_t + f(v_t) \qquad \text{eqn 14}$$
$$v_{t+1} = \varphi(v_t,u_{t+1})$$

The matrix for the linearized equations around the equilibrium point $u^* = \ln(N^*), v^* = \ln(x^*)$ is

$$\begin{matrix} 1 & f'_v \\ \varphi'_u & \varphi'_v + \varphi'_u f'_v \end{matrix}$$

where $'$ denotes the partial derivative. Note that the argument of the second equation is u_{t+1} and it should be substituted from the first when computing derivatives. Stability of the system is determined by the roots of the characteristic equation

$$\lambda^2 - (1 + \varphi'_v + \varphi'_u f'_v)\lambda + \varphi'_v = 0. \qquad \text{eqn 15}$$

Local stability is governed by, in the original variables,

$$a = \varphi'_x > 0 \qquad \text{eqn 16}$$

and

$$b = -f'_x \varphi'_N N^* > 0 \qquad \text{eqn 17}$$

These parameters are discussed in the text. In the special case of equation 6, $a = 1$ and $b = (1 - 1/R)(1 - 1/M)$, and the characteristic equation has the form

$$\lambda^2 - (2 - b)\lambda + 1 = 0. \qquad \text{eqn 18}$$

Since we assume that $R > 1$, $M > 1$, and $0 < b < 1$ we have two complex eigenvalues with product equal to 1. The period of the cycle for the linearized form is given by

$$T = \frac{2\pi}{\arctan \dfrac{\sqrt{4b - b^2}}{2 - b}},$$

which is readily seen from the complex plane representation of the eigenvalue. The minimum 6-cycle is guaranteed by noting that as $R \to \infty$ the value of $b \to 1$, and the value of the denominator in the above expression approaches $\arctan \sqrt{3}$, i.e. an angle of 60°.

Model 6 can be expressed in time-lag form as

$$N_{t+1} = \frac{R_z}{1 + z} N_t$$

where

$$z = \frac{N_t/N_{t-1}}{R - N_t/N_{t-1}} \frac{M}{1 + N_t}$$

Appendix 2

SENSITIVITY ANALYSIS: FUNCTIONAL FORMS AND PARAMETER DEPENDENCE FOR DYNAMICAL BEHAVIOUR

Limit cycle models

The neutral stability of equation 6 results from the linear expression of x in the second equation, which causes the partial derivative $\varphi_x{}^*$ to equal 1 (see above). Since this partial derivative is the determinant of the stability matrix, the two eigenvalues lie on the unit circle in the complex plane for all values of the parameters. The first assumption to be modified then is the linearity of the maternal effect. We have:

$$\varphi(x_t,N_{t+1}) = x_t^{\beta} \frac{M}{(1 + N_{t+1})} \qquad \text{eqn 19}$$

With this simple modification the equilibrium is stable for all values of β less than 1, and unstable for all values of β greater than 1. We can produce true limit cycles realistically but mathematically crudely by imposing upper or lower limits on quality x:

$$\varphi(x_t,N_{t+1}) = x_t^{\beta} \frac{M}{1 + N_{t+1}} \qquad \text{eqn 20}$$

$$x_{\min} < x < x_{\max}.$$

Simulations using the above for $\varphi(x,N)$ show that cycles very similar to those shown in the figures are generated, with the exception that the cycles 'limit' cycles, in the sense that varied initial

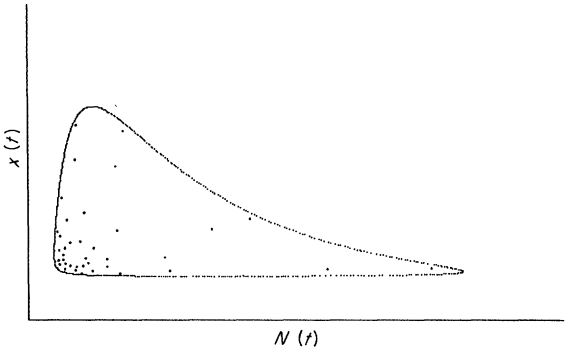

Fig. A1. Phase-plane portrait of the stable limit cycle model (equation 22). N_t scale from −1 to 35; x_t scale from −1 to 10. Parameter values $R = 3{\cdot}0$, $\beta = 1{\cdot}4$, $M = 2{\cdot}0$, $k = 0{\cdot}1$.

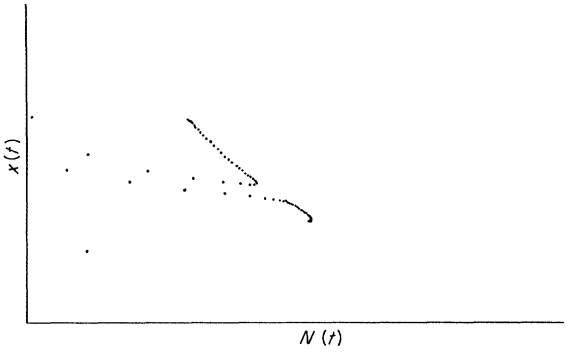

Fig. A2. Phase-plane portrait of the maternal effect model (equation 25) showing a flip bifurcation, with phase trajectories converging to the two phase points $(N_t, x_t) = (6{\cdot}814, 0{\cdot}232)$ and $(4{\cdot}037, 1{\cdot}154)$. N_t scale from 2·0 to 10·0; x_t scale from −0·5 to 2·0.

ditions lead to the same cycle. This model is biologically more realistic but cannot be fitted from a time-series alone.

Continuing with the above idea, we can add the assumption that a minimum quality x exists, and incorporate this explicitly into a differentiable form of $\varphi(x,N)$. An example is

$$\varphi(x,N) = k + \frac{(Mx_t)^{\beta}}{1 + N_{t+1}} \qquad \text{eqn 21}$$

where k is a small number representing the minimum quality. We can avoid adding k as an extra parameter if we make the additional assumption that k is equal to the equilibrium quality divided by the maximum rate of numerical increase R, meaning that species with higher possible increase rates have a smaller minimum quality. For the model this replaces k with the expression $1/R(R-1)$. This, it turns out, is a convenient form mathematically concerning the bifurcation behaviour of the model. The determinant of the matrix for the local linearization becomes

$$\lambda_1\lambda_2 = \beta(1 - 1/R). \qquad \text{eqn 22}$$

When this equation passes the value one we have a Hopf-type bifurcation as the eigenvalues cross the unit circle in the complex plane, with the creation of stable cycles in the form of an invariant Hopf curve. Thus, both the non-linearity and growth parameters can control the bifurcation behaviour of the model. A typical limit cycle generated by the model is shown in Fig. A1.

Hopf bifurcations in this model are possible when $\beta > 1$; but if function f is a linear function then stable limit cycles can be created for powers of β less than one, i.e. if

$$N_{t+1} = N_t R\, x_t. \qquad \text{eqn 23}$$

The above two models generate limit cycles similar to those of models 6 and 7, but due to the way N^* is changed the period of the cycles increases with increasing R, as in the logistic model. The attractors generated are similar to those from the discrete predator-prey model of Beddington *et al.* (1975).

If φ is a differentiable non-convex function of x, then qualitatively different bifurcation behaviour is possible. The model with

$$x_{t+1} = kx_t + \frac{M}{1 - \exp^{-x_t}}(1 + N_{t+1})^{-1} \qquad \text{eqn 24}$$

undergoes *flip bifurcation* with the creation of a two-cycle generated by eigenvalues of opposite sign (see Fig. A2). The two-cycle is stable for all realistic values of R. This example is perhaps of more mathematical than biological interest.