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Population dynamics of the Indian meal moth: demographic stochasticity and delayed regulatory mechanisms

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Summary

1. Laboratory populations of the Indian meal moth [*Plodia interpunctella* (Hübner) (Lepidoptera: Pyralidae)], undergo sustained periodic fluctuations in abundance. The period is just longer than the generation time. The fluctuations are accentuated in the presence of the *P. interpunctella* granulosis virus (PiGV).

2. Time series spanning 8–10 generations from three replicate populations of the virus-free (VF) system and three from the virus-infected (VI) system are investigated using nonparametric autoregressive time series models.

3. The dynamics are concluded to correspond to a third order process consistent with interactions in a three-dimensional stage-structured model for both systems. The functionally different interactive stages are believed to be the egg stage (preyed upon by larvae), small larvae (competing for resources and cannibalized by large larvae) and large larvae (competing for resources).

4. The virus is seen as a modulator of the host vital rates more than an independent agent in a trophic host–pathogen interaction. The virus increases developmental time and decreases fecundity of the moths.

5. A significantly nonlinear additive autoregressive model of order 3 appears to give a parsimonious description of the series.

6. The demographic (birth and death) nature of the stochasticity inherent in the system is explicitly incorporated in the statistical model for the time series by assuming an overdispersed Poisson process. The variability around the skeleton is found to conform closely to this assumption. The demographic nature of the stochasticity cannot be fully understood on the basis of Gaussian (least-squares) models on transformed (variance-stabilized) data.

7. Significant density dependencies are found at a 1-week lag, a 2- to 3-week lag and at a 6- to 7-week lag. These are argued to be the signatures of within-stage competition, between-stage interactions and reproduction, respectively. Negative and statistically significant density dependence is apparent for the first two of these. No significant negative density dependence is apparent in the lag corresponding to reproduction.

8. The fluctuations in both the VF and VI system appear to represent limit cycles or weakly dampened cycles clothed by Poisson demographic stochasticity.

9. The enhanced cycles of the VI system are demonstrated to be consistent with a situation where the functional forms for the interactions are nearly the same as for the VF, but with delay structure shifted by just less than a week.

Key-words: additive Poisson autoregression, competition and cannibalism, ecological dimension, nonlinear dynamics, periodic fluctuations, stage-structured model, stochastic limit cycle, time series analysis.

Introduction

The central task for ecologists studying biological populations is to develop an understanding of the dynamics of those populations. Such understanding can only be forged by combining a consideration of the dynamics themselves with a consideration of the underlying responses of individuals and the interactions between individuals. In order to understand the dynamics, it is necessary to describe the patterns they display in a way which distinguishes signal from noise and thereby characterize the deterministic and stochastic components as accurately as possible. At its simplest level, 'description' involves mere visual inspection, and 'characterization' merely the suggestion of an explanation gleaned from natural history. Several methods have been employed which take description beyond visual inspection, and understanding beyond intuition. Fluctuations in insect populations, for example, are commonly quantified by autoregressive models (Readshaw & Cuff 1980; Tsay 1988; Turchin 1990, 1991; Royama 1977, 1981, 1992; Turchin & Taylor 1992; Cheke & Holt 1993; Berryman 1991, 1995). The number of statistical and numerical tools available to reconstruct the 'laws' of dynamics on the basis of time series has expanded steadily (Moran 1953; Bulmer 1974; Pollard, Lakhani & Rothery 1987; Dennis & Costantino 1988; Royama 1977, 1981, 1992; Turchin & Taylor 1992; Dennis & Taper 1994; Berryman 1991, 1995; Ellner & Turchin 1995). Alongside this, there has been an increasing effort to bridge the gap between theoretical models of fluctuating populations and statistical models for empirical time series (Gurney, Blythe & Nisbet 1980; Mueller & Huynh 1994; Dennis *et al*. 1995; Costantino *et al*. 1995, 1997). Here, we build on advances in nonparametric regression for time-series data in examining the dynamics of laboratory populations of the Indian meal moth, in isolation and in the presence of a viral disease. As part of the present study the current methodology is extended to accommodate demographic stochasticity explicitly in nonlinear autoregressive models. The structures identified are interpreted in terms of statistical density dependence at various delays, and the bridge to theoretical models is made through a mathematical model designed to capture the essence of the intra- and interstage interactions in the populations.

Within- and between-stage competition for resources and asymmetric interactions (such as cannibalism) between individuals of different larval stages (*instars*) are crucial determinants of the reproduction and survival of certain insects. Disregarding environmental variability, fluctuations may be caused by both demographic stochasticity (disruptive forces) or delays in regulatory mechanisms (controlling forces). The delays may arise because of developmental time and/or interactions between individuals of different stages (age-, stage- or size-structured dynamics; Gurney, Nisbet & Lawton 1983; Caswell 1989; MacDonald 1989; Royama 1992). From a mechanistic point of view, fluctuations caused by regulatory delays in structured populations may be divided into two broad categories: (i) those driven by symmetric interactions between similar individuals (belonging to the same stage); and (ii) those resulting from asymmetric interactions between individuals of different stages (Gurney & Nisbet 1985; Nisbet & Onyiah 1994). Sustained population cycles may result in either case if the interactions are highly nonlinear (see, for example, May 1987) or if weakly nonlinear or linear dynamics are disrupted by stochastic forces (see, for example, Stenseth, Bjørnstad & Saitoh 1996a). Cycles arising from within-stage competition that affect either reproduction or the vital rates of those same individuals at a later developmental stage commonly have a period of around two generations (Gurney *et al*. 1983; Gurney & Nisbet 1985; Nisbet & Onyiah 1994). Nicholson's (1957) blowflies are a classic example. The different stages of the flies were kept apart and thus prevented from interacting, with the resultant populations undergoing sustained bigenerational cycles (Gurney *et al*. 1980). Between-stage interactions, or within-stage interactions which are immediately expressed in the form of mortality, may give rise to fluctuations with a period approximately equal to the generation time (Gurney & Nisbet 1985). Laboratory populations of the Indian meal moth (*Plodia interpunctella* (Hübner), Lepidoptera: Pyralidae) provide an example (Gurney *et al*. 1983; Sait, Begon & Thompson 1994a–c; Begon, Sait & Thompson 1996).

This study investigates three replicate time series of the Indian meal moth. The *P. interpunctella* granulosis virus (PiGV) is known to affect the dynamics of this species (Sait *et al*. 1994a,b). Three replicate series of the moth–virus system are therefore also investigated to shed light on the effect of this infection at the population level. The layout of the paper is as follows: After a brief description of the natural history of the host and host–pathogen populations, the essence of the demography of the host is highlighted through a stage-structured model of the life cycle. The statistical modelling and analysis of the time series is then described, a process which itself comprises several steps to identify an appropriate description of the controlling as well as the disruptive forces. Once the most parsimonious statistical model has been identified and analysed, the results are interpreted within the context of the stage-structured demographic model. The paper ends with a discussion of more general biological and statistical issues resulting from the present study.

Materials and methods

LIFE HISTORY OF THE HOST AND THE VIRUS

Three populations of the moth (VF) were maintained for 2 years at the University of Liverpool at 28 ± 2°C, 65 ± 5% relative humidity, and a 13 : 11 light : dark cycle. The diet consisted of 800 g wheat middlings, 160 g dried brewer's yeast, 200 mL glycerol, 200 mL clear honey, 1 g sorbic acid and 1 g methyl paraben. Adults do not eat (and therefore do not compete – at least not directly), and produce an average of 42 eggs per day over a 5–6-day life span. The eggs hatch after 4–5 days, producing first instar larvae. The larvae undergo a further four instar stages before pupating. The pupal period lasts approximately 8 days. This, combined with a larval period of approximately 20 days gives a generation time of just under 40 days in optimal conditions (Begon *et al.* 1996). Both intra- and inter-instar competition for food occurs among the larvae. This is likely to cause decreased survival, but also an increased developmental time from those quoted (obtained from larvae with excess food). Cannibalism of smaller larval instars by larger ones is known to occur (Sait *et al.* 1994a). Cannibalism of eggs by larvae has been suggested but not described in detail (Richards & Thomson 1932). The life cycle is summarized in Fig. 1a, and the main interactions in the cycle in Fig. 1b (see Sait *et al.* 1994a for details).

The three replicate host–pathogen populations (VI) were kept under identical conditions to the three moth populations except for the adding of the granulosis virus at the onset of the study. The PiGV virus spreads by ingestion of infected material by larvae (Sait *et al.* 1994a–c). It causes death of younger larvae at appropriate doses, but can also, at sublethal doses, prolong the developmental time of instars 4 and 5 (by 4–5 days) and of pupae (by around 1 day) and reduce the reproductive potential of the adults by about 25% (from 42 to 32 eggs day^{-1}; Sait *et al.* 1994b). A possible complicating factor is the demonstration of evolution towards resistance of the moth to the virus in some virus-infected populations (Boots & Begon 1993).

At the end of each week, all dead adults were removed from the six populations and counted (Fig. 2). The counts serve as the measure of the abundance for that week. Each population was thus sampled approximately six times per generation. The basic statistics for each time series are summarized in Table 1. All populations exhibit significant periodic cycles (Table 1, Fig. 2). The cycle is just longer than the generation length, τ estimated from individuals with excess food ($\approx 1{\cdot}1\tau$) (Sait *et al.* 1994a; Begon *et al.* 1996). In addition to the periodicity, Fig. 2 highlights that the populations require some 20 weeks to attain a pattern of stationary fluctuations (that is, time-independent mean, variance and covariance).

The weekly prevalence of the virus was quantified as the number of visibly infected larvae. A long period of convergence to the stationary situation or the occurrence of host evolution are possible causes of the apparent increase in prevalence of the virus over time (Fig. 2).

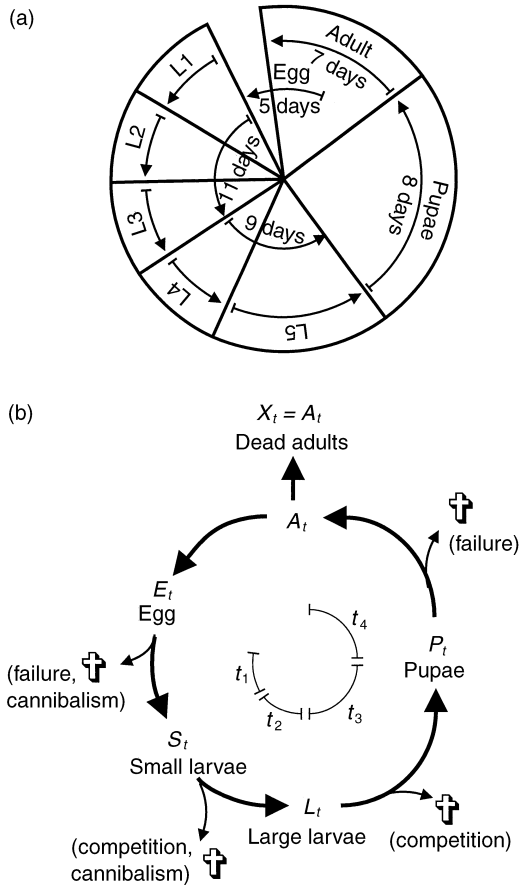

Fig. 1. A summary of the life history of the Indian meal moth. (a) All eight stages in the cycle as a pie chart. L1–L5 represent larval instars 1–5. (b) A plausible web of demographic processes between the functionally different stages. The five larval instars are divided in two broad categories: small larvae, S_t, and large larvae, L_t. E_t, P_t and A_t represents the abundance of eggs, pupae and adults, respectively. The number of dead adults, X_t, constitute the data for the analyses.

A STAGE-STRUCTURED DEMOGRAPHIC MODEL

The life cycle of *P. interpunctella* is composed of eight morphologically distinct stages (including five larval instars). If all of these were interactive and functionally (dynamically) different, the population dynamics would be of order $\geqslant 8$ (see, for example, Schaffer 1981; Royama 1981, 1992; Gilbert 1993; Stenseth, Bjørnstad & Falck 1996b; leaving aside whether, in fact, such a high dimension is possible to identify on the basis of the available time series data). Focusing on any one life stage (adults), this will manifest itself by requiring an autoregressive model (a model in delay co-ordinates) of order $\geqslant 8$ (Royama 1992; Chan, Tong & Stenseth unpublished; see also Broomhead & Jones 1989; Gilbert 1993). However,

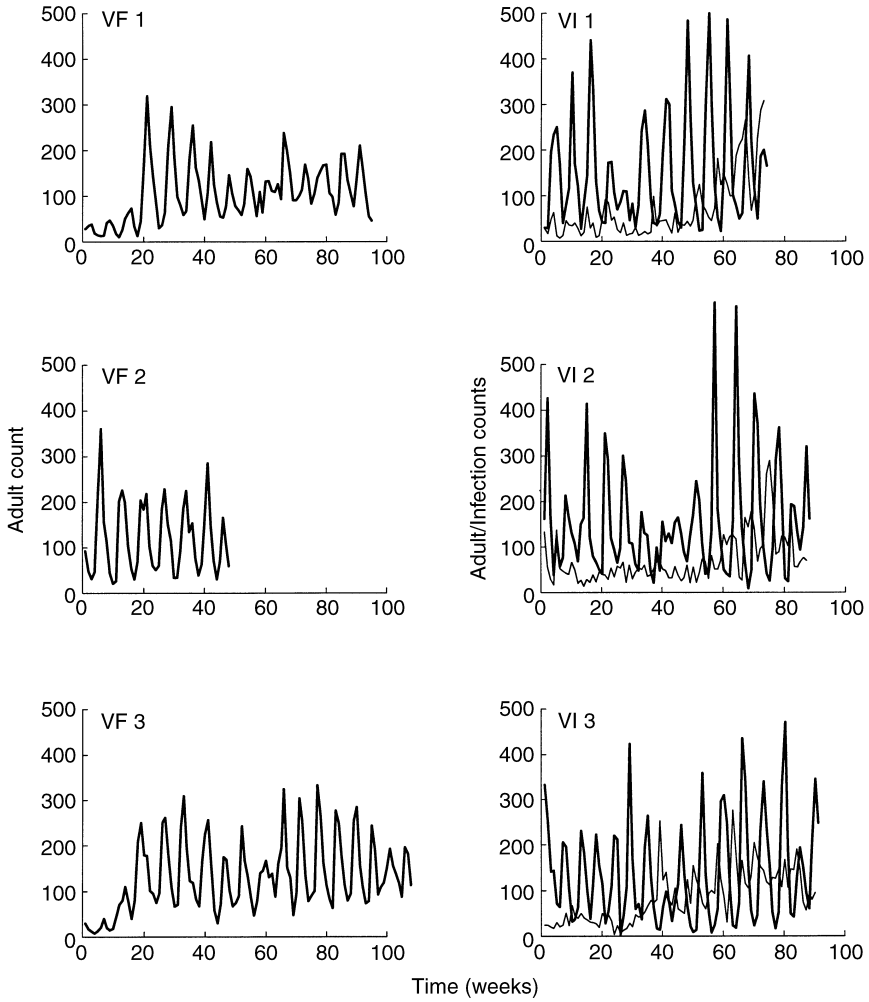

Fig. 2. The six time series analysed. There are three replicates of the virus-free (VF) populations and three of the virus-infected (VI) populations. The thick lines represent counts of the weekly number of dead adults of the experimental period. The thin lines (of the virus-infected populations) represent the number of visibly infected larvae each week.

Table 1. Summary of the basic statistics for each time series. VF represent virus-free populations (1–3), VI represent virus-infected populations (1–3). *n* is the length of the series (number of weekly counts). 'Coef Var' is the coefficient of variation. Period signifies dominant period in the estimated spectrum in days, calculated on square-root-transformed data, disregarding the first 20 observations of each series. P'gram is a nonparametric estimate based on the periodogram. AR is a parametric estimate based on the linear autoregressive model (order as selected by the Akaike information criteria). Approximate 95% confidence intervals (CI) are calculated by resampling (1000) the autoregressive coefficients according to the variance-covariance matrix of the maximum likelihood estimates.

Population	*n*	Mean	Median (range)	Coef Var	Period (days) P'gram	AR ($CI_{95\%}$)
VF1	95	105·6	98 (10, 318)	40·1	44·3	46·0 (39·1, 65·9)
VF2	48	108·2	92 (10, 349)	57·5	52·5	45·5 (40·3, 51·2)
VF3	108	132·4	114 (8, 333)	46·3	45·0	43·3 (41·6, 46·3)
VI1	74	155·5	114 (22, 500)	96·6	47·3	46·9 (23·8, 50·9)
VI2	88	151·9	124 (14, 618)	91·0	43·3	46·0 (41·8, 50·7)
VI3	91	137·0	108 (4, 472)	89·9	45·8	44·7 (41·0, 48·9)

the number of functional groups is likely to be fewer (see, for example, Caswell 1989, chapter 9) and not all groups (pupae) are likely to be interactive. The realized dynamics may, thus, be of a lower dimension than eight (for a related discussion see Stenseth *et al.* 1997).

We have simplified the life cycle depicted in Fig. 1a by assuming that the five larval instars can be classified into two functional groups (Fig. 1b): small larvae (consisting of instars L1–L3), *S*, and large larvae (consisting of instars L4–L5), *L*. Because adults do not require any resources, we assume the number of eggs

produced at time t, E_t, to be directly proportional to the number of adults, A_t:

$$E_t = rA_t, \qquad \text{eqn 1}$$

where, r is the per capita reproduction. Specifically, this study makes the simplifying assumption that any delayed effects of larval competition on reproductive output of adults are unimportant. Throughout the duration of the egg-stage, t_1 (4–5 days under optimal conditions) some of these eggs, $f_{1a}(S_{t-t_1})$, may be eaten by the smaller larvae, and some, $f_1(L_{t-t_1})$, may be eaten by the larger larvae. Assuming a constant hatching rate, b, for the eggs, there are three possible formulations:

$$S_t = bE_{t-t_1} - f_{1a}(S_{t-t_1}) - f_{1b}(L_{t-t_1}), \qquad \text{eqn 2i}$$

$$S_t = bE_{t-t_1} - f_{1b}(L_{t-t_1}), \qquad \text{eqn 2ii}$$

$$S_t = bE_{t-t_1}, \qquad \text{eqn 2iii}$$

depending on whether both larval classes cannibalize eggs (equation 2i), the effect of small larvae is negligible (equation 2ii), or that all larval egg-cannibalism is unimportant, that is, the egg stage is non-interactive (equation 2iii).

During the duration of the small larval stage, t_2 (around 11 days under optimal conditions), a number of individuals, $f'_{2a}(S_{t-t_2})$, will die as a result of within-stage (density-dependent) competition for resources. An additional number, $f_{2b}(L_{t-t_2})$, will die as a result of asymmetric interactions (competition and cannibalism) with the larger larvae. As a result,

$$\begin{aligned} L_t &= S_{t-t_2} - f'_{2a}(S_{t-t_2}) - f_{2b}(L_{t-t_2}) \\ &= f_{2a}(S_{t-t_2}) - f_{2b}(L_{t-t_2}), \end{aligned} \qquad \text{eqn 3}$$

large larvae will emerge at the end of this developmental period.

Assuming competitive dominance of the large larvae, the number of individuals dying in this developmental stage will be a function of the abundance of large larvae (Cushing 1994): $f'_3(L_{t-t_3})$. In this way, the number pupating at the end of the developmental time, t_3 (around 9 days under optimal conditions) is

$$P_t = L_{t-t_3} - f'_3(L_{t-t_3}) = f'_3(L_{t-t_3}). \qquad \text{eqn 4}$$

Finally, assuming density-independent adult emergence from the pupae at the end of the pupal period, t_4 (*c*. 8 days under optimal conditions), the adult abundance will be:

$$A_t = cP_{t-t_4}. \qquad \text{eqn 5}$$

A virus may have two (not necessarily mutually exclusive) effects on the dynamics of its host. First, it may enter as an interacting component to produce a host–specialist consumer coupling (similar to a parasitoid–host or predator–prey interaction). In such a case, the order of the dynamics of the VI populations (both when examining the time series of the host and that of the virus) should be higher than that of the VF populations (Schaffer 1981; Royama 1981, 1992; Bjørnstad, Falck & Stenseth 1995; Stenseth *et al.* 1996b, 1997). Alternatively, the pathogen may act to modulate the vital rates of the host without altering the structure of the interactions. This will lead to altered dynamics of the host, but not to altered dimensionality of the dynamics.

STATISTICAL MODELLING OF THE POPULATION DYNAMICS

Identifying an appropriate statistical model to describe and interpret a population time series is a process involving several steps. The sequence, for the present data, is (i) choice of an appropriate error structure: what is the stochastic nature of the data and the underlying process? (ii) choice of the order or dimension of the model, where the model is framed in time-delay co-ordinates (essentially investigating how complex the model should be); (iii) determination of the degree of nonlinearity of the dynamics: nonlinear with interacting lags, nonlinear additive or linear (and by definition additive); (iv) given the order, d, the identification of which exact time delays (or lags) are involved.

Given the availability of replicate data for both VF and VI populations, we seek to achieve model consistency between replicates whenever possible. An ultimate aim is to find a final model synthesizing the available information across the replicates.

Error structure

In these enclosed laboratory populations of *P. interpunctella* neither immigration nor emigration is possible. Furthermore, because adults do not require food, *per capita* birth rate may be expected to vary relatively little with density. Hence, demographic influences on mortality are likely to be the forces most strongly shaping the dynamics. The temporal scaling of reproduction and mortality may add to this. Reproduction takes place at the time scale of the generation length, whereas mortality will at least partly be determined at the scale of the stage durations. The sampling of the populations at more frequent intervals than the generation length thus emphasizes the importance of mortality.

Within any time period an individual can either die or survive. Density-dependent mortality means that the binomial probability, π, of surviving from time t to time $t + \Delta t$ decreases with density. In a stage-structured population, this may be a function of the abundance of several of the stages (above). For an ensemble of individuals, the total number of deaths within a stage, a, will be the sum of such binomial events:

$$\sum_{i=1}^{N_{a,t}} pr\{\textit{individual } i \textit{ dying}|\underline{N}_{\bullet,t-\Delta t}\} = \sum \pi(\underline{N}_{\bullet,t-\Delta t})$$

$$\sim Po(N_{a,t-\Delta t}\pi(\underline{N}_{\bullet,t-\Delta t})) \equiv Po(F(\underline{N}_{\bullet,t-\Delta t})),$$

where $\underline{N}_{\bullet,t}$ signifies that the covariate may be vectorial (if more than one stage is involved in the interaction), and $\sim Po(\ )$ signifies that the sum of such binomial events will be approximately Poisson distributed (see, for example, Bartlett 1960; Bickel & Doksum 1977).

In the absence of other sources of variability (which is approximately true for the present laboratory study, with negligible sampling error and environmental variability), the dynamics should be governed by the deterministic density-dependent component, $F(\underline{N}_{\bullet,t-\Delta t})$, but clothed by the Poisson demographic stochasticity. If the population is governed by only one interactive stage and influenced by no temporally autocorrelated variables (either due to latent variables or to misspecification of the statistical model; see, for example, Zeger 1988), we would expect the abundance of each stage to follow exactly a (conditional) Poisson distribution. However, with several interactive stages, the stochasticity will be compounded to produce more variability than expected from a pure process. Extra-Poisson variation will also result from temporal autocorrelation in the residuals (Zeger 1988). For *P. interpunctella*, therefore, we anticipate extra-Poisson variation. However, the *variance function* (McCullagh & Nelder 1989) will be similar to that of a Poisson variable in that the variance will be proportional (but not equal) to the mean under demographic stochasticity (Engen & Lande 1996a, b). This type of data may be modelled by a quasi-Poisson distribution (Zeger 1988; McCullagh & Nelder 1989; Dean 1992). The variance should be stabilized by the square-root transformation (see, for example, Sen & Srivastava 1990, chapter 6) and, in the case of an autoregressive (Markov) process, the marginal distribution of the square-root-transformed data should be approximately symmetrical (due to ergodic convergence of the marginal distribution on the conditional distribution; see, for example, Gilks, Richardson & Spiegelhalter 1996).

Figure 3 indicates that the quasi-Poisson argument appears valid for the present data, in that the square-root transformation makes the distribution of the counts symmetrical. This is in contrast to counts of open populations affected by stochastic forcing from the environment, for which abundances appear conditionally log-normal or Gamma distributed. For such systems the variance will be approximately proportional to the square of the mean (Engen & Lande 1996a, b), so that the variance is stabilized by a log-transformation (Sen & Srivastava 1990, chapter 6; see also Cushing *et al*. 1996).

It is worth stressing that under the above-specified demographic stochasticity, the total demographic variability increases with abundance. Hence, the dynamics will not converge on the underlying attractor (Bartlett 1960). The full model for the dynamics of *P. interpunctella* should therefore incorporate this important stochastic effect.

Dimensionality

The stationary dynamics of a population may, under the density-dependent paradigm, be approximated by an autoregressive process (see, for example, Turchin 1995). Focusing on any one life stage, a, the expected number of individuals is $E[N_{a,t}|N_{\bullet,t-\Delta t}] = F(\underline{N}_{\bullet,t-\Delta t})$, where the expectation is taken with respect to the stochastic forces outlined above. This may (according to Takens theorem; Broomhead & Jones 1989) be written in delay co-ordinates, according to the general autoregressive model:

$$E[N_{a,t}|N_{a,t-\tau_1}, N_{a,t-\tau_2}, \ldots, N_{a,t-\tau_d}]$$
$$= G(N_{a,t-\tau_1}, N_{a,t-\tau_2}, \ldots, N_{a,t-\tau_d}), \quad \text{eqn 6}$$

where d is the order of the model. Note that eqn 6 involves only the sequence of the one stage, a. To simplify notation, below, the stage subscript a, is suppressed in such delay-coordinate models. In the case of Poisson (or quasi-Poisson) processes, the variance of the square-root-transformed data will be constant, so that Gaussian theory for time series analysis may be applied (above).

In order to identify the skeleton model underlying an ecological time series, the function in delay co-ordinates for the conditional expectation (mean), $E(N_t|N_{t-\tau_1},\ldots)$, must be reconstructed. Because the class of nonlinear processes is very large (see, for example, Tsay 1988; Tong 1990; Tjøstheim 1994), one ought, as a first step, to consider a flexible class of models. A possible choice is the nonparametric kernel regression model for

$$E(N_t|N_{t-\tau_1},\ldots) = \hat{G}(N_{t-\tau_1}, N_{t-\tau_2}, \ldots, N_{t-\tau_d}),$$

that is flexible in that it may accommodate a variety of nonlinearities, including interactions between the different lags (Härdle 1990; Cheng & Tong 1992, 1994; Tjøstheim & Auestad 1994; Hjellvik & Tjøstheim 1995). The disadvantage is that the model suffers from *the curse of dimensionality* (see, for example, Scott 1992; Tjøstheim & Auestad 1994). That is, the sample size required for reliable estimation is exponential in d. However, Cheng & Tong (1992, 1994; see also Yao & Tong 1994) demonstrated that a leave-one-out cross-validation will produce a consistent estimator of d itself, $\hat{d}$, with minimal assumptions, and the sample size requirement for this increases much more slowly with d (only quadratic). Its estimation is therefore quite feasible with the data at hand (Cheng & Tong 1992).

Thus, we estimate the order of each of the six square-root-transformed time series using the method of Cheng & Tong (1992, 1994) as modified by Yao & Tong (1994; see also Fan 1992; Fan, Yao & Tong

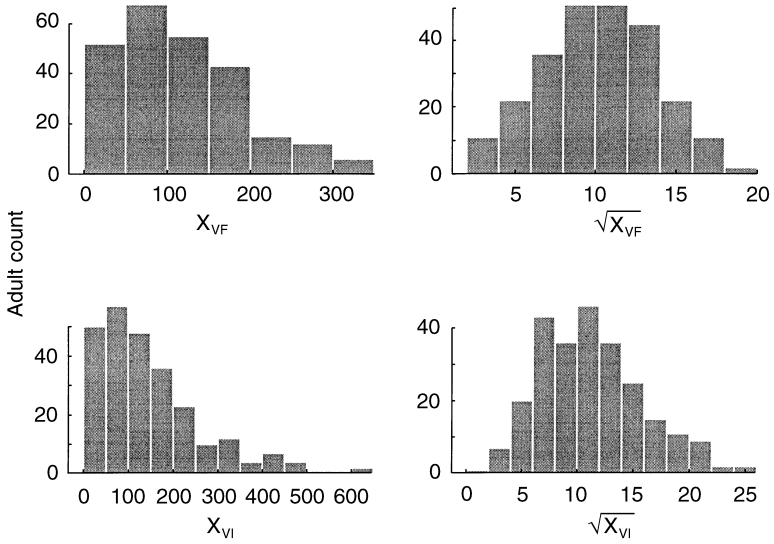

Fig. 3. Histograms of the raw data and the square-root-transformed data for the weekly counts for the three virus-free (VF) and virus-infected (VI) populations. The counts are aggregated across the three replicates of VF and VI. The marginal distribution will asymptotically converge on the conditional distribution for ergodic autoregressive processes (see text).

1996). In each case, the bandwidth of the Gaussian kernel was optimized using cross-validation. In addition, we estimate the order through cross-validation of the linear autoregressive model, using a Kalman filter applied to the state space representation of the likelihood (Kohn & Ansley 1986; *S-plus version 3·3*, Statistical Sciences 1995). The latter method is very powerful for linear or near-linear processes, and does not suffer from the curse of dimensionality. However, it will produce a biased (upwards) estimate in the case of strong nonlinearity (Tong 1990; Royama 1992).

Nonlinearity

One way to circumvent the curse of dimensionality is to assume an additive model (Chen, Liu & Tsay 1995; Eubank *et al*. 1995):

$$E(N_t|d=\hat{d}, N_{t-\tau_1}, \ldots) = g_1(N_{t-\tau_1}) + g_2(N_{t-\tau_2}) + \cdots + g_d(N_{t-\tau_d}). \quad \text{eqn 7}$$

Chen *et al*. (1995) have developed tests for the statistical permissibility of using equation 7 instead of 6. The test assumes constant conditional variance, and is thus applied to the square-root-transformed data. We employ the Lagrange multiplier test of Chen *et al*. (1995: test II). A biological motivation of an additive structure for the present data may be drawn, for example, from equation 3 (see also Chan *et al*. 1997).

The most extreme simplification of equation 6 is to assume a linear autoregressive model:

$$E(N_t|d=\hat{d}, N_{t-\tau_1}, \ldots) = \alpha_0 + \alpha_1 N_{t-\tau_1} + \alpha_2 N_{t-\tau_2} + \cdots + \alpha_d N_{t-\tau_d}, \quad \text{eqn 8}$$

where α_0 is the mean and $\alpha_1, \ldots, \alpha_d$ are the autoregressive coefficients. [For log-transformed annual data from free-ranging vertebrate populations, this model has proved valuable as a population dynamic model capturing patterns of direct and delayed density dependence (Reddingius 1990; Royama 1992; Myers & Cadigan 1993; Bjørnstad *et al*. 1995; Stenseth *et al*. 1996a,b, 1997)]. Several tests for nonlinearity in time series (and hence for the permissibility of equation 8) have been developed (reviewed in Tong 1990; Tjøstheim 1994; Chan 1997). Using square-root-transformed data we, first, apply the nonparametric test of Hjellvik & Tjøstheim (1995) with order as determined above, and kernel band width optimized by cross-validation (see also Stenseth *et al*. 1996a), and second, we apply the Tukey one-degree-of-freedom test of Tsay (1986).

In order to construct a complete dynamic model for the populations, the stochastic nature of the series should ideally be modelled at the scale of the observations (the counts). We have argued above that the stochastic components of this system are not noise to be considered a nuisance, but rather an integral part of the birth and death process, and that the conditional variance of the data is an overdispersed Poisson process. This type of error-structure can be modelled directly for both linear and additive processes using the method of quasi-likelihood (Breslow 1984; Zeger 1988; McCullagh & Nelder 1989; Dean 1992). The quasi-likelihood model under the assumption of overdispersed Poisson variance is (McCullagh & Nelder 1989):

$$\mu = E(N_t) = \sum_{lags} g_i(N_{t-i}) \quad \text{eqn 9i}$$

$$Var(\mu) = \sigma^2 \mu, \quad \text{eqn 9ii}$$

where σ^2 is the scale-parameter quantifying the degree of overdispersion. This model on the untransformed scale represents a GENERALIZED ADDITIVE MODEL (GAM;

Hastie & Tibshirani 1990), and has the advantage of being easily interpretable: the functions will be reflections of the patterns of reproduction, intrastage competition and interstage interactions (see below).

Using the cross-validation estimate of the order of the process, d, quasi-likelihood theory for equation 9 can be used to identify which lags should be included in the final model through an analysis of deviances. This study considers a priori all lags up to t-G (the generation time). In addition, sums of any two adjacent lags (e.g. $N_{t-2}+N_{t-3}$, denoted $N_{t-2/3}$) are considered to allow for (i) time-delays that fall between the sampling interval (1 week; see Sait *et al.* 1994a), (ii) distributed delays (Blythe, Nisbet & Gurney 1984; MacDonald 1989), and/or (iii) a restricted form of non-additivity. In the cases of approximate additivity (possibly including the restricted form of non-additivity), the d lags ($\tau_1, \tau_2, \ldots, \tau_d$) that maximise the quasi-likelihood (assuming 4 d.f. for each lag) were selected from the set $\{1, 2, \ldots, 7, {}_{1/2}, {}_{2/3}, \ldots, {}^{6/7}\}$.

Manifestation of density dependence

Whenever the population is sampled more often than the generation length, density-independent mortality, in the additive setting, implies: (i) all g_i-functions in equation 7 have a positive slope; and (ii) the slope is constant. This is because density-independent mortality implies that the proportion surviving is independent of density (no curvature in recruitment function; cf. equation 4). A test for negative density dependence is therefore to test first for a significantly negative slope, and if not, to test for significant convex curvature. Testing may be carried out according to Hastie & Tibshirani (1990).

The fitting and inference regarding linear and additive models were carried out using *S-plus for Windows version 3·3* (Statistical Sciences 1995). Additive models were fitted using cubic B-spline smoothers (Hastie & Tibshirani 1990). Nonparametric estimation of the order and test for linearity were carried out using a kernel conditional expectation estimator coded in *Borland Pascal version 7·0* (Borland 1992) interfaced with S-plus.

Results

The results from both the nonparametric and the linear cross-validation estimates of the order are summarized in Table 2. The data are scaled such that the CV-values are comparable. The order that minimizes the out-of-sample prediction error is 3 for four of the series (two of the VF series, and two of the VI series). The remaining two series have order estimates greater than 3. However, for one (VI3) the difference in prediction error between the third- and the fourth-order model is only 3% units. Such differences may well arise by chance alone and may be considered insignificant. This interpretation is strengthened by the third-order model being selected using the linear model. The last time series (VF3) stands out as having a markedly higher order of dynamics (the order 5 model does better than the order 3 model by 7% units). The reason for this is unclear. Nonetheless, overall, the parsimonious choice of order across the replicates is 3 for both the VF and VI populations: there is no evidence for the dynamics of the VI populations having higher order than the VF.

The two tests reveal strong evidence for nonlinearity (Table 2). The null hypothesis of linearity is rejected at a nominal 5% level for four of the series (two VF and two VI). Of the remaining two, one (VF2) is far shorter than all other series (VF2; $n = 28$, once the 20 initial points have been removed). The lack of rejection of the null hypothesis, is likely to be an effect of low power. The null hypothesis is rejected at a nominal 10% level for the remaining series (VI3). Thus, the dynamics of both the VF and VI populations show clear evidence of nonlinearity.

The test for nonadditivity does not reject the null hypothesis of additivity for any of the series (Table 2). A nonlinear but additive autoregressive model of order 3 appears therefore to be a parsimonious choice for modelling the population dynamics of both VI and VF populations of *P. interpunctella*.

A third-order additive model with a quasi-Poisson error structure and the full range of lags was fitted to the data (using 4 d.f. for the spline smoother of each lag). For each series the models were ranked with respect to goodness-of-fit according to residual deviance. A word of caution is in order: because the lag 1 autocorrelation is high for all these series (Spearman rank correlation VF: $\rho = 0{\cdot}63$, VI: $\rho = 0{\cdot}47$), the effect of any lag can partially be substituted by adjacent lags. Statistical uncertainty is therefore likely to be associated with the inference; consistency across different replicates is therefore crucial. The most appropriate model structures for each time series are summarized in Table 3. Based on the ranking of the models, the most parsimonious model for the VF populations was:

$$E(N_t) = g_1(N_{t-1}) + g_2(N_{t-2/3}) + g_3(N_{t-6}) \qquad \text{eqn 10i}$$

with the second choice identical to equation 10i but with $g_2(N_{t-2})$. The model with $g_3(N_{t-6/7})$ carries rank 5 (out of 41). The most parsimonious model for the VI populations was:

$$E(N_t) = g_1(N_{t-1}) + g_2(N_{t-2/3}) + g_3(N_{t-6/7}). \qquad \text{eqn 10ii}$$

The second choice is identical to (10i), that is with $g_3(N_{t-6})$.

In summary, statistical analyses indicate that the lag-structures of the VF and VI populations are very similar. However, the VI populations tend to have slightly longer delays (shifted by approximately half a week). The fitted functions for the parsimonious (and

Table 2. Results from both nonparametric and linear cross-validation calculated on square-root-transformed data disregarding the first 20 observations of each series. 'LL_{cv}' give the order estimates for the nonparametric order estimator of Cheng & Tong (1992, 1994) modified to use the local linear estimator of Yao & Tong (1994; see also Fan 1992; Fan *et al.* 1996). The leave-one-out cross-validation method was applied to normalized, square-root-transformed data. The 'CV' values can be interpreted as the percentage of unpredictable variation. The optimal order is indicated in 'D_{opt}'. When D_{opt} is different from three, the difference in explanatory power of the optimal and the order three model is given ('Δcv'). 'Lin_{cv}' give the order estimates based on cross-validation of the linear (MLE) autoregressive model. The maximum likelihood estimates for the linear model with one data point removed are estimated using a Kalman filter applied to the state space representation of the likelihood (Kohn & Ansley 1986; *S-plus version 3·3*, Statistical Sciences 1995; see also Stenseth 1995; Stenseth *et al.* 1996b,c). 'H-T' give the *P*-value for the nonparametric test of Hjellvik & Tjøstheim (1995) for nonlinearity (see also Stenseth *et al.* 1996a). 'Tsay' give the *P*-value for the Tukey one-degree-of-freedom test for nonlinearity of Tsay (1986). 'Additive' give *P*-value for the Lagrange multiplier test for nonadditivity of Chen *et al.* (1995). The asterisks indicate results supporting the 3^{rd} order nonlinear but additive model.

Population	LL_{cv} D_{opt}	CV	Δcv	Lin_{cv} D_{opt}	CV	Δcv	Lin. Test H-T	Tsay	Additive
VF1	5	0·50	0·01	3·00*	**0·45**		0·05*	0·18	0·31*
VF2	3	0·39		3·00*	**0·36**		0·27	0·20	0·47*
VF3	5	**0·40**	0·07	5·00	0·49	0·01	0·00*	0·21	0·52*
VI1	3*	**0·29**		4·00	0·33	0·03	0·01*	0·01*	0·61*
VI2	3*	**0·52**		4·00	0·53	0·02	0·11	0·03*	0·83*
VI3	4	**0·37**	0·03*	3·00	0·42		0·22	0·10	0·61*

Table 3. The best five additive models of order 3 for each time series (ranked according to the residual deviance; given below each model). The first line indicate the lag structure: 1, indicates lag *t*-1, etc., 23 indicates lag *t*-2/3, etc. (see text). The longest lag considered was 7 — just longer than the generation length.

Population	1st	2nd	3rd	4th	5th
VF1	1–23–6	1–3–6	1–56–6	1–34–6	1–2–6
	549·8	589·4	608·4	616·1	617·0
VF2	1–2–56	1–23–6	1–2–67	1–2–7	1–2–6
	75·8	99·4	100·0	105·3	112·1
VF3	1–23–6	1–34–6	1–23–4	1–23–7	1–23–67
	798·2	830·1	848·3	850·8	851·4
VI1	1–23–6	1–23–4	1–2–6	1–23–67	1–2–67
	802·9	811·3	890·8	899·3	927·8
VI2	1–5–7	1–23–7	1–2–67	1–23–67	1–23–5
	1699·7	1763·3	1789·7	1816·1	1863·7
VI3*	1–3–6	1–23–6	1–23–67	1–3–56	1–3–67
	939·0	961·4	999·4	1000·4	1043·0

* Due to problems of singularities in the model for VI3 only the subset of the data from week 25–77 was used (disregarding 19 data points).

the second choice) models as applied to each replicate are depicted in Fig. 4. The remarkable consistency across replicates may be indicative of successful model selection. Note that the second choice function for VF gives, if anything, a better consistency across the replicates than the first choice. This is not the case for the VI models. The close correspondence between the functions of the different replicates encourages the aggregation of the information from the different replicates to produce two final models (Fig. 5).

The predicted dynamics of models 10i and 10ii (Fig. 5) in the absence of stochasticity, are depicted in Fig. 6. The observed fluctuations have the correct qualitative behaviour (cycles). The amplitudes are, however, too small and the rate of dampening too fast. The prediction is particularly unsatisfactory for the VI populations. As the period of transience may be longer than 20 observations for the VI populations (Fig. 2), the model was re-estimated assuming a transient period of 50 weeks (which is likely to be conservative). The resultant model gave rise to a limit cycle in the absence of stochasticity (Fig. 6). All skeleton behaviours represented dampened or limit cycles that were less wildly fluctuating than the data and that had a slightly too long period (*c.* 50 days for VF and *c.* 54 days for VI). The lower amplitude is, at least partly, a trivial effect of filtering away the inherent demographic stochasticity.

The estimated functions (Fig. 5) can be interpreted in terms of statistical density dependencies exhibited by the populations (Royama 1992; Bjørnstad *et al.* 1995):

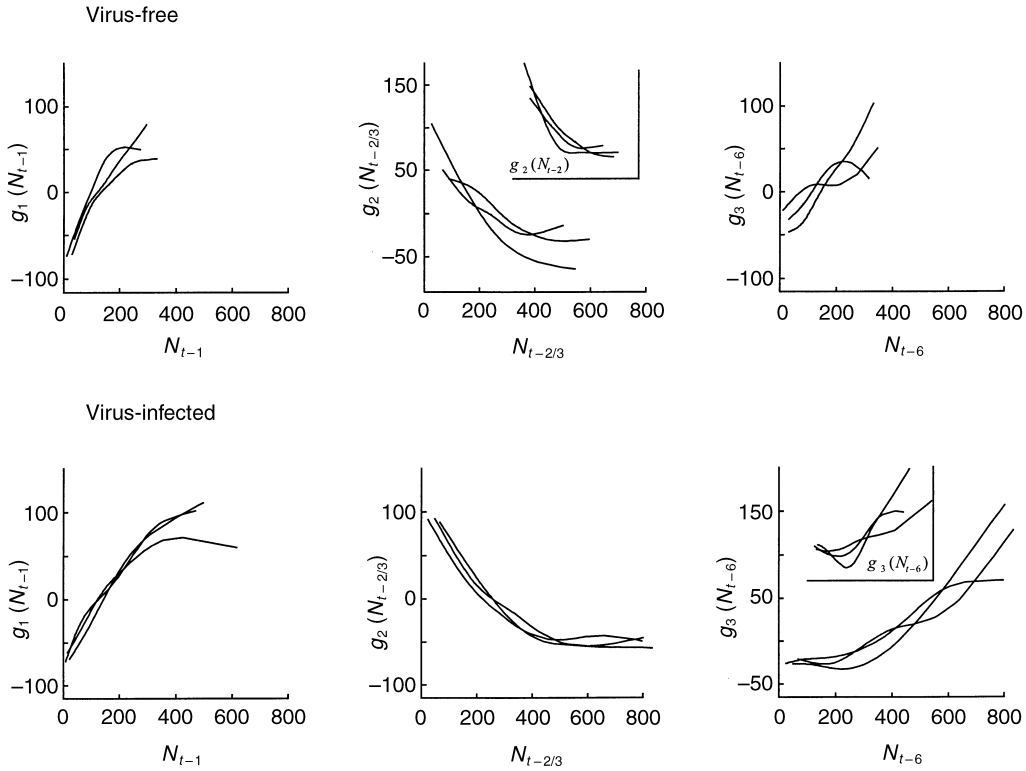

Fig. 4. The estimated nonparametric regression lines for the six most parsimonious generalized additive autoregressive models for the time series (using quasi-Poisson error, identity link and cubic B-splines with 3 d.f. for each lag). Residuals are suppressed (see Fig. 5) to simplify the figure. The functions for the second choice models are superimposed wherever different in lag structure from equations 10i and 10ii. Due to the shortness of VF2, all data (including the initial 20 points) were used to estimate the model for this series and the total number of degrees of freedom for the model was decreased from 10 to 9. For VI3, 4 observations had to be excluded to obtain a stable model.

1. The populations exhibit severe negative density dependence at the 2–3 week lag (see also Fig. 4)—the slope is significantly negative for both the VF and VI populations.

2. The tests for curvature in the positive slope at lag 1 identify significant density dependence for the VF populations ($F_{1\cdot9,163\cdot2} = 6\cdot03$, $P = 0\cdot003$). The nonlinearity at lag 1 for the VI population is not statistically significant ($F_{2\cdot1,159\cdot7} = 1\cdot67$, $P = 0\cdot19$). For both the VF and the VI convexities are apparent at high densities.

3. There is no significant statistical density dependence at the 6 week lag for the VF ($F_{1\cdot9,163\cdot2} = 1\cdot28$, $P = 0\cdot27$). The curvature at the 6–7 week lag for the VI is not statistically significant at a nominal 5% level either ($F_{2\cdot1,159\cdot7} = 2\cdot74$, $P = 0\cdot066$). The probability under the null hypothesis of linearity is, however, low. The curvature at lag 6–7 in the VI represents a concavity at low abundances (contrary to the lag 1 function). There is, thus, some evidence of depensation in the VI.

Superimposing the models for VI and VF reveals great similarity (Fig. 7). The most apparent difference is the possible lag shift (Table 3) with a slightly longer delay in the VI populations. The pattern of statistical density dependence at the longest lag (6 vs. 6–7) is also divergent: the slope for the VI populations is significantly shallower than that of the VI (Fig. 7).

Discussion

Laboratory populations of the Indian meal moth undergo marked cycles in abundance with a period just over the generation length (Sait *et al.* 1994a; Begon *et al.* 1996). The dynamics appear from the present analysis to be the result of nonlinear interactions in a three-dimensional stage-structured system. The most dramatic negative density-dependent force engraved in the time series has a delay in around 2–3 weeks and is apparently governed by asymmetric interactions between different stages of the life cycle. Specifically, the crux of the dynamic pattern appears to be the cannibalistic and competitive effects exerted by large larvae on small larvae and/or eggs (see below for a formal treatment). This parallels the conclusion drawn by Gurney *et al.* (1983) and Gurney & Nisbet (1985), from purely theoretical work: generation cycles in stage-structured insect populations result from interactions between stages that are manifested directly through forces of mortality (not indirectly through reproduction). Asymmetric competition between large and small larvae has been suggested previously as a crucial element in *Plodia* dynamics, although without any supporting analysis (Sait *et al.* 1994a). Cannibalism of eggs, although described (Richards & Thomson 1932), has previously been overlooked. The three-dimensional

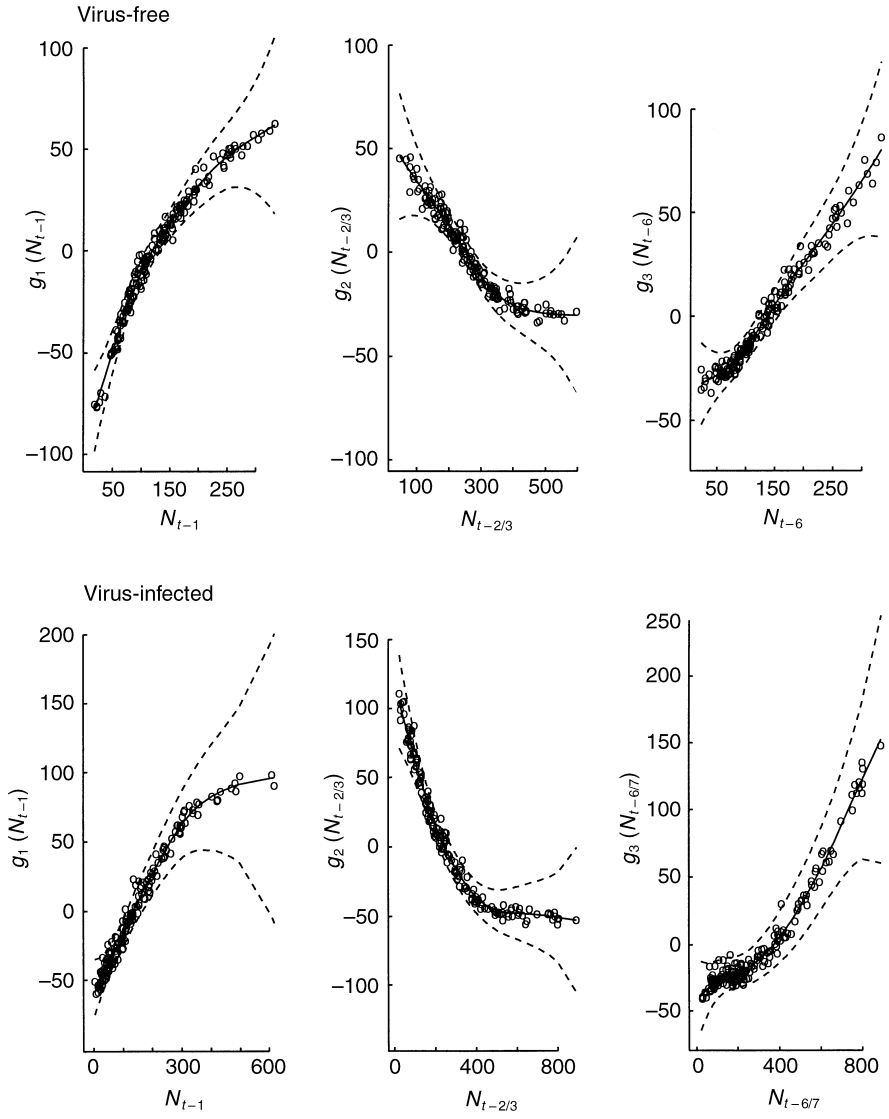

Fig. 5. The estimated nonparametric regression lines with standard errors and Pearson residuals (residuals that are scaled by their variance (McCullagh & Nelder 1989) for the two generalized additive autoregressive models for all virus-free (top) and all virus-infected (bottom) populations (using quasi-Poisson error, identity link and cubic B-splines with 3 d.f. for each lag).

structure of the dynamics indicates that the life cycle involves three functionally different and interactive stages (see below).

A second negative density-dependent force, less important than the above (significant only in the VF populations and apparent only at high densities), operates with a lag of just 1 week. This is likely to reflect competitive interactions between larvae of the same stage. The apparently lowered importance of this in the VI populations may reflect an interplay between competition and infection, where infection lowers the density of developing larvae sufficiently to reduce competitive pressures.

The order estimate and the lag structure can be further interpreted in the light of the life cycle graph (Fig. 1b). The basic transitions are governed by equations 1–5, although the structure will depend on which of the three egg survival functions (equations 2i–2iii) is the more appropriate. A model in delay co-ordinates can be developed for adults (through some tedious back-substitutions, and assuming that all functions have inverses—denoted by $^{-1}$) under the different assumptions:

$(2i) \Rightarrow$

$$A_t = cf_3(f_{2a}[rbA_{t-G} - f_{1a}(f_{2a}^{-1}(f_3^{-1}(A_{t-t_1})/c) - f_{2b}(f_3^{-1}(A_{t-t_1-t_2})/c))) - f_{1b}(f_3^{-1}(A_{t-t_1-t_2})/c))] - f_{2b}(f_3^{-1}(A_{t-t_2})/c))) \quad \text{eqn 11i}$$

which is a function of the form $N_t = G(N_{t-1/2}, N_{t-2/3}, N_{t-G})$ (apparent from the duration of stages detailed in *Materials and methods*). Clearly, the cycle including assumption 2i (both smaller and larger larvae interfere with the egg stage) predicts three-dimen-

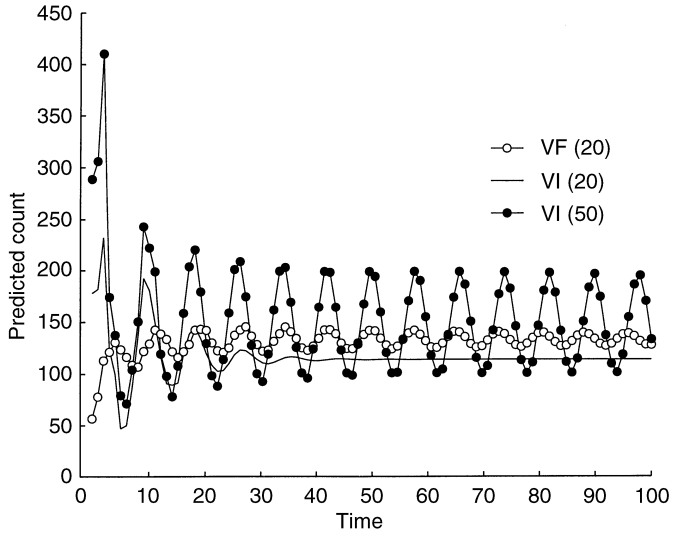

Fig. 6. Predicted dynamics of the set of skeleton functions (the deterministic component of Fig. 5). VF (20) represent the skeleton of the virus-free population models using a transient period of 20 weeks. VI (20) represent skeleton of the virus-infected model based on a transient period of 20 weeks. VI (50) is the same, but assuming a transient period of 50 weeks (see text for detail).

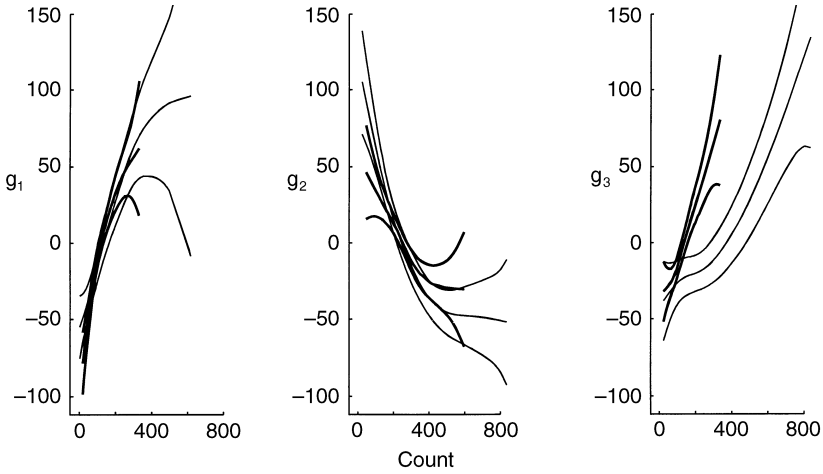

Fig. 7. The nonparametric autoregressive models for the VF and VI systems (Fig. 5) overlaid and with approximate 95% confidence intervals superimposed. Thick lines represent VF model and thin lines represent the VI model. Residuals are suppressed.

sional dynamics, and a delay structure similar to that of the parsimonious statistical model (approximately lag 1, lag $G/2$ and lag G). Note, though, that the predicted model has interactions between all lags. A similar order and delay structure is predicted by assumption 2ii (only larger larvae significantly interfere with the egg stage), a third-order model with the correct lag structure:

$$(2ii) \Rightarrow A_t = cf_3(f_{2a}[rbA_{t-G} - f_{1b}(f_3^{-1}(A_{t-t_1-t_2}/c))] - f_{2b}(f_3^{-1}(A_{t-t_2}/c))). \quad \text{eqn 11ii}$$

In contrast, the most simplifying assumption 2iii (no effect of larvae on egg stage) implies:

$$(2iii) \Rightarrow A_t = cf_3(f_{2a}(rbA_{t-G}) - f_{2b}(f_3^{-1}(A_{t-t_2}/c))), \quad \text{eqn 11iii}$$

which is a function of the form $N_t = G(N_{t-1/2}, N_{t-G})$. Thus, assumption 2iii fails as a predictor for the pattern in the time series of *P. interpunctella* by predicting too simplistic a delay structure. Thus, equations 11i–iii detail how the order 3 structure may imply three functionally different and interactive stages.

Inspection of equations 11i and 11ii reveals (through consideration of the derivatives of the functions and the inverse functions 1–5) that the negative slope of g_2(lag 2/3) is tightly linked with either f_{1b} (the effect of large larvae on eggs), f_{2b} (the effect of large

larvae on small larvae), or both. That is, it is governed by asymmetric interactions between life stages as conjectured above. Thus, the simplified life cycle model involving tight interactions between eggs and two larval stages may account for the delay structure engraved in these time series. A possible alternative may be that the five larval instars are divided into three (rather than two) functionally different and interactive stages and the egg-stage is non-interactive. It is also conceivable that competition may manifest itself indirectly through reduced reproductive capacity of the adults. This would serve to make adults an interactive stage. Any firm conclusion must await further investigation.

Studies of the possible demographic consequences of PiGV on *Plodia* dynamics have revealed both direct lethal effects and indirect effects: the former acts as a force of mortality, and the latter acts by modulating the host's demographic parameters through delaying development and reducing reproduction (Sait *et al.* 1994b, c). The present analyses of time series data indicate that, of these, the latter appear to be dominant. First, the VI populations are governed by processes operating at slightly longer delays than the VF populations (apparently reflecting a delay in development). Second, the reproductive output of the VI populations, as reflected by the generation-lag dependence ($\hat{g}^3$; Figs 4, 5 and 6), is lower than that of the VF populations (apparently reflecting a sublethal effect on reproduction). And last, there is no evidence of higher dimensional dynamics in the VI system—the virus modulates demographic parameters rather than enter into a coupled predator–prey interaction). The detailed analyses of time series has thus helped to identify which potential ecological forces are of sufficient importance to be expressed at the population level.

In view of the conspicuous prolongation of the delay structure in the time series analyses of the VI populations, we may speculate that their enhanced cyclicity (larger amplitude and more regular periodicity) is chiefly due to changes in delay structure, rather than to changes in the functions themselves. A way to investigate this is to shift the delay, but not the functional relations, of the empirical functions (Figs 5–7; equations 10i and 10ii). The resultant skeleton behaviour is depicted in Fig. 8. As conjectured, the subtle *decrease* in the delay (applying $\hat{g}_3$ of the VI model to N_{t-6} and not $N_{t-6/7}$) stabilizes the model for the VI populations. Corroborating this, a subtle *increase* in the delay in the model for the VF population (applying $\hat{g}_3$ of the VF model to $N_{t-6/7}$ and not N_{t-6}) produces divergent dynamics. Alterations in the functional forms (such as that indicated by $\hat{g}_3$; Figs 4, 5 and 6) may certainly add to this. We can only reiterate the conclusion of Sait *et al.* (1994b) and Begon *et al.* (1996)—subtle and/or sublethal effects of a pathogen may lead to important alterations of population dynamics.

Subtle differences in the environment are also likely to influence the effects of the pathogen. The time series analysed here (see also Begon *et al.* 1996) are different from those described in the study of Sait *et al.* (1994a). The latter study maintained a richer food medium and VI populations exhibited reduced densities and clearly extended cycle periods compared to VF populations. Such effects were not apparent in the present data. How these differences in the dynamic patterns result from the effects of food on the host's demographic parameters is worthy of future enquiry.

The most suitable delay co-ordinate models (equations 11i and 11ii) exhibit interactions between the lags. This is at odds with the near additive structure of the square-root-transformed data. Based on the full set of analyses we can revisit the patterns of additivity, employing the most parsimonious lag structure as well as aggregating information across the replicates. As predicted, the null hypothesis of additivity is rejected for both the VI and weakened for the VF populations ($P = 0{\cdot}03$ and $P = 0{\cdot}10$, respectively). Thus, there is evidence of some degree of interaction between the lags but this is not sufficiently strong to penetrate to the individual series, even when employing the optimal lag structure (VF1: $P = 0{\cdot}42$, VF2: $P = 0{\cdot}51$, VF3: $P = 0{\cdot}21$, VI1: $P = 0{\cdot}16$, VI2: $P = 0{\cdot}46$, VI3: $P = 0{\cdot}16$). A methodological challenge in this regard is that no test for nonadditivity has been developed for non-Gaussian data, such as the present population counts. We have, therefore, been forced, in the absence of an alternative, to test for an additive structure on the variance-stabilized scale (the square-root). This is clearly suboptimal, so a healthy scepticism should be exercised with respect to the additivity. The ecological analysis reported in this paper could be improved if an appropriate statistical methodology was developed to fill this gap. One possibility is, perhaps, to proceed along the lines of the test applied above for nonlinearity in the GAM models, by comparing the fit of the additive quasi-Poisson model with a non-additive analogue (a SMOOTHING SPLINE ANOVA model; Wahba *et al.* 1995). We feel, nevertheless, that the accuracy and resolution of the inference based on the additive model is sufficiently impressive to warrant its use as a first approximation.

A gain from employing the assumption of approximate additivity is that it allows explicit modelling of the stochastic forces inherent in the birth- and death-processes governing the demography of *P. interpunctella*. The most usual way of dealing with time series of counts has been to build models and draw inferences from Gaussian models applied to the square-root-transformed data (as in the initial parts of our inference) or log-transformed data (Royama 1992; Myers & Cadigan 1993; Broekhuizen & McKenzie 1995; Stenseth *et al.* 1996a,b, 1997). In Fig. 9 we depict, with residuals, the parsimonious additive Gaussian models for the square-root-transformed data of the populations. Superimposed on this is the

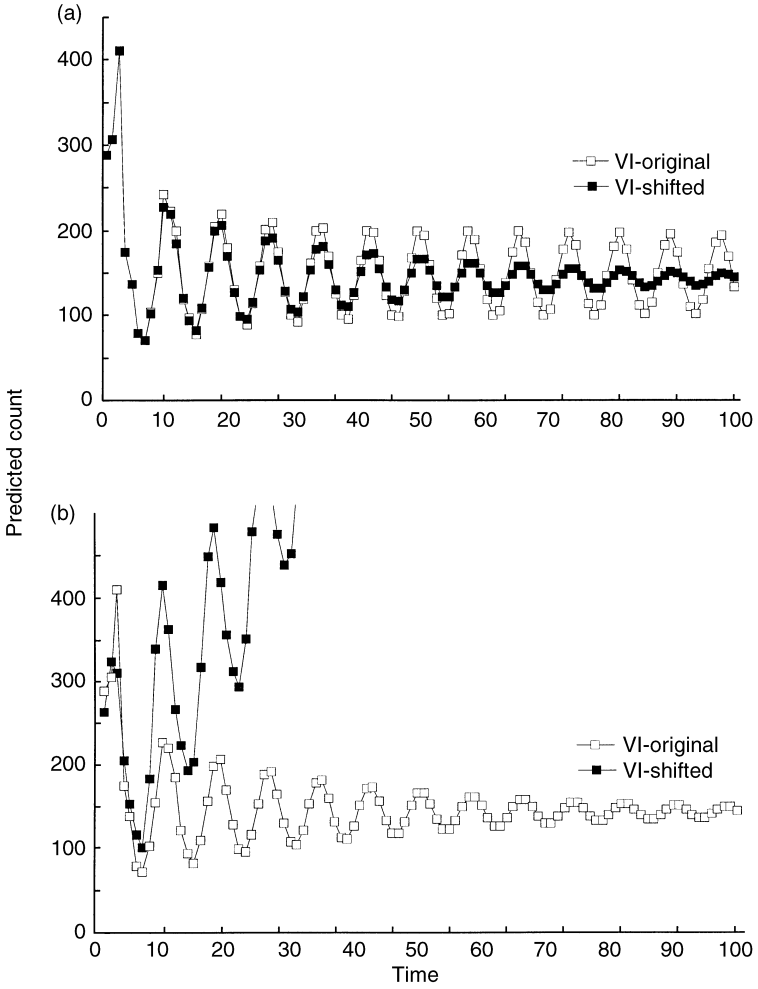

Fig. 8. The effect of shifting the lag on predicted skeleton behaviour. (a) The lag structure is shifted to apply $\hat{g}_3$ to N_{t-6} instead of $N_{t-6/7}$ in the model for the VI population (assuming a transient period of 50 weeks, and 4 d.f. for each lag). The original skeleton is given for comparison. (b) The lag structure is shifted so as to apply $\hat{g}_3$ to $N_{t-6/7}$ instead of N_{t-6} for the model for the VF population (assuming a transient period of 20 weeks). Stability is enhanced in the former case, whilst the dynamics explode in the latter.

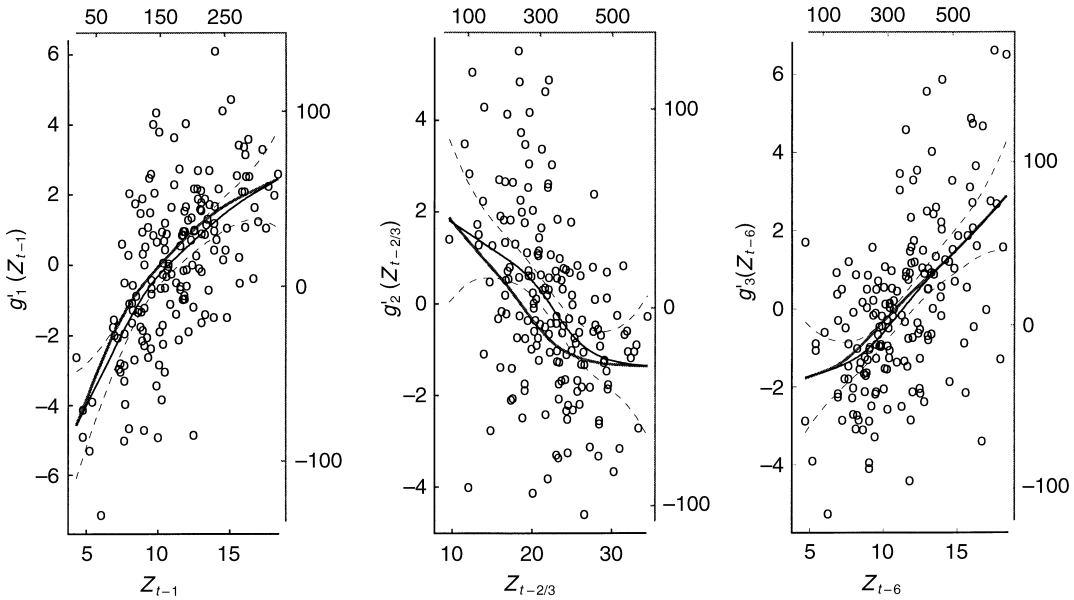

Fig. 9. Comparing the quasi-Poisson model for the raw data with the model one would estimate by applying a Gaussian model to the square-root-transformed data (denoted by Z) for the virus-free population. The grey, thick lines represent the regression lines for the former (cf. Fig. 5). The corresponding axes for this quasi-Poisson fit are drawn on the right and on the top. The thin lines, error lines, residuals and left-bottom axes depict the Gaussian model fit.

skeleton of the additive quasi-Poisson model (rescaled for comparison; the scaled residuals of which are depicted in Fig. 5). Clearly, the deterministic component of the dynamics is captured by the Gaussian model for the square-root-transformed data (the lines representing the functions are near identical in shape). However, the origin of the stochastic component is obscure; the Gaussian model suggests that there is much variability yet to explain (Fig. 9). In contrast, the model incorporating demographic stochasticity as an explicit force (Fig. 5) indicates that, while there may be room for improvement, there is not very much to gain. The Pearson residuals cluster tightly around the model, so that the variability around the predicted value is only marginally larger than that expected from the stochasticity inherent in demographic processes.

In conclusion, the analysis of abundance data from replicated populations in a controlled laboratory setting in which environmental variability is close to zero and sampling variability is negligible (the sampling effects inherent in the demography are unaltered, but the counting of dead adults is affected by little error), have led to equal emphasis on the inherent demographic stochastic forces and the inherent deterministic process. A distinct density-dependent signature (of order 3) is clearly imprinted in the time series, and this pattern of dependence appears to account for 40% of the variability in the data (range 29–52%). Of the remaining variability, most is accounted for by demographic stochasticity in the form of overdispersed Poisson variation. Intriguingly, it is highly accurate laboratory data, similar to those obtained by Nicholson (1957) that have led us to re-emphasize the importance of stochastic forces, as stressed by Andrewartha & Birch (1954), in the seminal debate over the relative importance of regulatory and stochastic forces in shaping population dynamics.

Acknowledgements

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