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Long-term population dynamics of the Indian meal moth *Plodia interpunctella* and its granulosis virus

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Summary

1. Theoretical studies suggest that disease may play an important role in the population dynamics of insects, but there have been no long-term empirical studies that have examined this interaction in the absence of other important biotic and abiotic factors.

2. In the laboratory, three virus-free (VF) populations of *Plodia interpunctella* were compared with three virus-infected (VI) populations that were continuously exposed to a granulosis virus. Data sets lasted for between 10 and 17 generations. Census data of dead adults were collected twice weekly and spectral analysis of the data was used to complement the observed population fluctuations.

3. The VF populations exhibited discrete cyclic fluctuations with a period slightly in excess of one generation's length. However, the cycle period decreased significantly in later cycles in all three cases.

4. The VI populations also exhibited discrete cycles, but with a period significantly longer and a population density significantly reduced compared with the VF populations. The VI populations exhibited no change in cycle period during the period of observation. The dynamics of the pathogen itself were highly unstable and there was little evidence for linked host–pathogen fluctuations.

5. The cyclic fluctuations in the VF and VI populations appeared to be caused predominantly by density-dependent larval competition for food, whilst the pathogen was thought to be responsible for the fundamental differences between the populations.

Key-words: baculovirus, cycles, granulosis virus, *Plodia interpunctella*, population dynamics.

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Introduction

In recent years, disease has been recognized as an important factor in the dynamics of plant and animal populations (e.g. Anderson & May 1986). Invertebrate host–pathogen interactions have generated a great deal of interest theoretically as an aid to understanding how diseases regulate populations (Anderson & May 1981; Onstad & Carruthers 1990; Anderson 1991) and in evaluating the potential of pathogens as pest control agents (Payne 1988). These theoretical studies suggest that pathogens may induce cycles in (and in that sense destabilize) otherwise non-cyclic host dynamics (Anderson & May 1981), in a manner analogous to conventional, 'Lotka-Volterra predator–prey cycles, and may even generate 'purely chaotic' dynamics (May 1985). Cycles are especially likely when the pathogen has free-living infective stages rather than being directly transmitted (Anderson & May 1981), though cycles are less likely to arise by this route when the host is subject to self-regulation that does not itself generate cycles (Bowers, Begon & Hodgkinson 1993). On the other hand, heterogeneities in pathogen dynamics, such as the existence of a long-lived reservoir of free-living infective stages (Hochberg 1989; Hochberg, Hassell & May 1990) can give rise to near-constant host abundance and, in this sense, pathogens may act as a stabilizing force in host dynamics. Generally, model predictions are based on components of the host–pathogen interaction that have been identified experimentally, such as pathogenicity, sublethal effects, disease trans-

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mission and so on, but these parameters have typically been determined from short-term studies (a single generation or cohort). In order to evaluate the true impact of pathogens on host population dynamics, however, and to provide data sets with which to explore model predictions, insect–pathogen relationships need to be studied continuously over many generations.

Unfortunately, there are few such long-term empirical studies. The majority have centred on forest insect pests, where in some cases a pathogen has been thought to be a contributory factor to both dramatic reductions and cyclic fluctuations in host abundance (e.g. Bird & Elgee 1957; Thompson & Scott 1979). Medium-term studies (generally two or three generations or seasons) of host–pathogen interactions have also been conducted in agricultural and pastoral environments (Hofmaster 1961; Hamilton 1979; Fleming *et al.* 1986). However, none of these field studies has been able to investigate the host–pathogen interaction alone, since many other factors play a role in host dynamics (Myers 1988). Furthermore, pathogen-free and pathogen-infected populations have not been directly compared, making it difficult to establish the true impact of the pathogen on the resulting dynamics.

Laboratory studies of insect populations are valuable in that environmental vagaries and other biotic factors can be controlled. Lepidopteran stored products pests in particular have been used for many years in long-term single species studies (e.g. Richards & Waloff 1946; Snyman 1949; White & Huffaker 1969a), as well as in long-term host–parasitoid studies (e.g. White & Huffaker 1969b; Benson 1974; Podoler 1974). Many stored-product pests are also readily susceptible to baculoviruses (essentially, nuclear polyhedrosis viruses, NPVs, and granulosis viruses, GVs; Payne 1988) which typically only infect the larval stages of the host when the pathogen has been ingested (Granados 1980). However, despite this, there do not appear to be any equivalent long-term host–virus studies of the kind cited above.

Here, therefore, in order to further understanding of insect–pathogen interactions, the long-term population dynamics of virus-free cultures of the cosmopolitan stored-products pest, the Indian meal moth, *Plodia interpunctella* (Hübner) (Lepidoptera; Pyralidae) are compared and contrasted with those that are continuously exposed to a granulosis virus.

Materials and methods

Each experimental population was maintained in a closed container with a base area of 22×22 cm, divided into six equal parts, and a depth of 9 cm. A nylon net sleeve facilitated removal of adults, replacement of food, etc. (see below), and a pierced lid aided ventilation. When each population was initiated, 84 g of food (Sait, Begon & Thompson 1994a) were added, covering the base to a depth of approximately 1 cm. Once the population was established, one section was replaced with fresh food weekly in sequence, so that each section was replaced after 6 weeks.

Three replicate virus-free (VF) populations were initiated using 15 female and 15 male late fifth instar *P. interpunctella* larvae. Three replicate virus-infected (VI) populations were initiated similarly, but with an additional 12 virus-killed fifth instar larvae (two per section).

Host densities were monitored by twice weekly counts of dead adults which were removed from each population. Each count gave a measure of the population size from the previous week, this being the approximate adult lifespan. This rearing and counting method for *Plodia* has been employed successfully by J. H. Lawton (unpublished, in Jones 1986; see also Gurney, Nisbet & Lawton 1983). From one randomly chosen VF and one VI population, dead adults were sexed and their forewing lengths measured using a binocular microscope (magnification $\times 10$) to monitor any possible long-term changes in sex ratio or size. Wing length was chosen as a measure of size because corpses were often incomplete, making any direct measurement of weight unreliable.

Sections of food removed from the VF populations were discarded since they contained only a few pupae; the active larval stages showed a preference for the fresh medium. Those sections of food removed from the VI populations were examined for virus-infected larvae to give an index of the level of the infection. These were easily identified, whether recently infected (mottled white appearance) or heavily infected (opaque white). Again, healthy larvae and infecteds still capable of movement showed a preference for fresh medium, so only inactive or dead infecteds remained in these sections. Those infected larvae recovered from the discarded food were returned to their populations with the fresh food. This ensured that the host–pathogen interaction was maintained, especially initially when few infecteds were present. Infected first instar larvae were not monitored for reasons of practicality.

The VF populations were regularly examined for signs of virus contamination, but none was found. All populations were maintained at 28 ± 2 C, $65 \pm 5\%$ RH, (ranges) and a 12 : 12 light : dark cycle.

Cycles in host abundance occurred (see below) and dead adults were assigned to different phases of each cycle. Phase 1 adults were those collected prior to the 'peak' of a cycle. In phase 2, adults were collected from the peak (the maximum density in a cycle plus the two adjacent samples). Phase 3 adults were sampled when numbers were declining. The end of phase 3 and the start of phase 1, between cycles, was chosen when the number of adults was zero or, when occasionally necessary, after the lowest number in a cycle. Size differences between phases of emergence were tested for significance, as was the difference between VF and VI populations. However, since

phase 1 tended to be very short and the numbers of adults low, no analysis of data was performed involving wing lengths from this phase.

Results

VIRUS-FREE POPULATIONS

Each population exhibited fluctuations that corresponded to 15 discrete cycles (Fig. 1a–c). In each cycle, a period with none or few adults present was followed by a rapid increase in adult numbers until emergence peaked. The decline in numbers was generally extended, giving each cycle a skewed appearance. The maximum peaks of adult numbers were 296, 268 and 295 (A, B and C, respectively) and the minimum peaks 130, 108 and 101 adults (A, B and C). There was no significant difference between the mean population densities overall or the mean number per cycle between the three replicates (ANOVA) (Table 1).

The smoothed spectra of the three replicates (spectral analysis carried out using the SAS SPECTRA procedure; SAS Institute 1988) indicated that their fluctuations had mean cycle periods of 41·8, 40·0 and 41·0 days for A, B and C, respectively (Table 1 and Fig. 1d–f), and again, there was no significant difference between them (ANOVA). In each replicate,

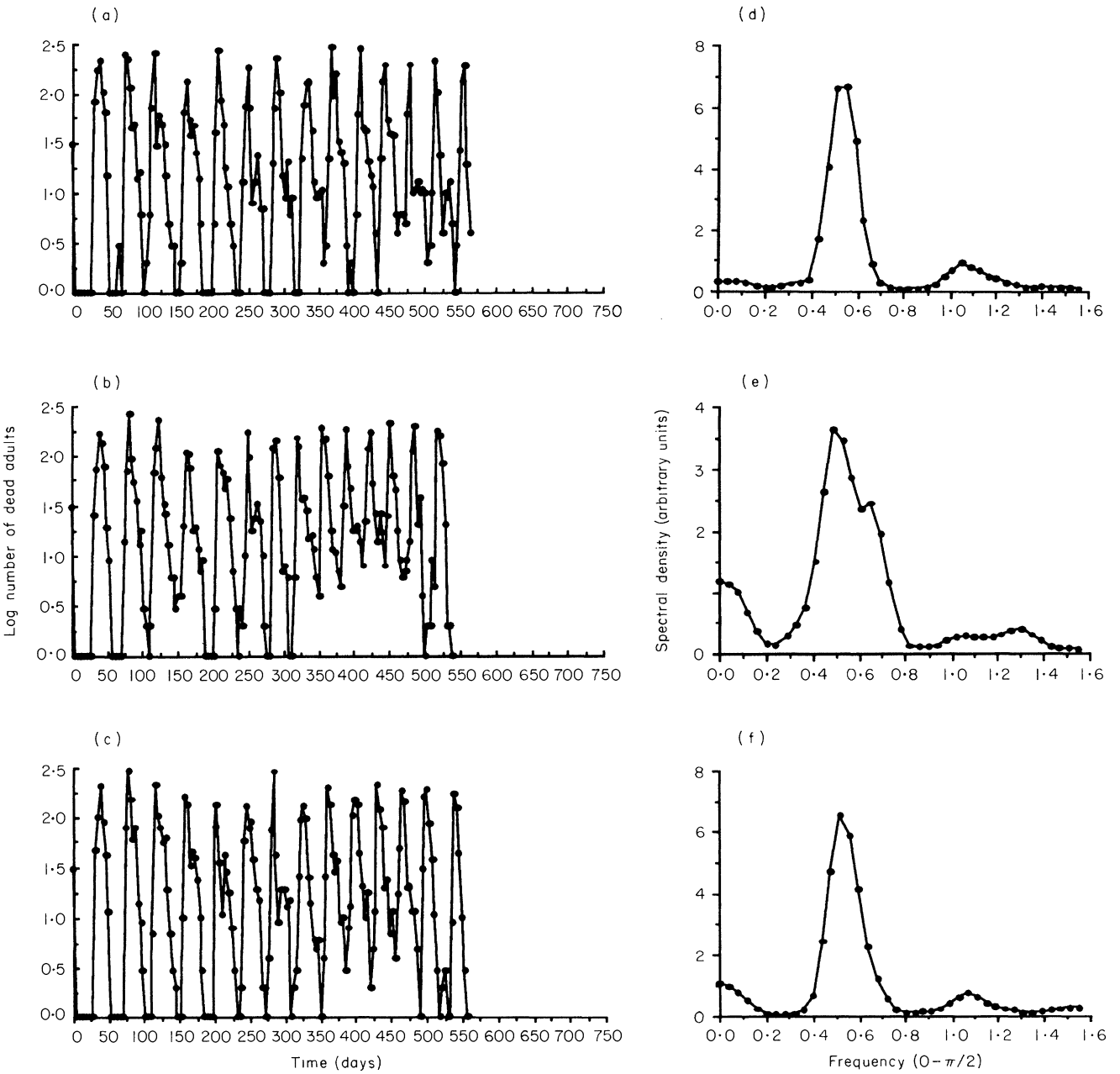

Fig. 1. (a–c) Population fluctuations from the three virus-free (VF) replicates recorded as twice-weekly counts of dead adults. (d–f) Power spectra of each replicate illustrating that the cycles could be described by possibly two underlying periodicities. The cycle length in all the analyses could be calculated using the transformation 7π/frequency.

Table 1. Summary of observed fluctuations and spectral analyses from the virus-free and the virus-infected populations.

Observed fluctuations in host populations

Treatment*	Population replicate	Number of days monitored	Number of cycles	Population† density (mean ± SD)	Cycle‡ density (mean ± SD)
VF	A	560	15	42·4 ± 66·9	454·9 ± 162·0
	B	536	15	41·4 ± 57·5	425·0 ± 130·9
	C	550	15	42·1 ± 60·7	443·5 ± 140·0
VI	A	714	17	33·7 ± 54·5	406·1 ± 131·8
	B	420	10	32·0 ± 51·4	385·4 ± 162·5
	C	536	13	33·5 ± 53·4	396·8 ± 163·1

Observed fluctuations in the pathogen populations

Treatment	Replicate	Number of days monitored	Number of cycles	Density of infecteds per food section (mean ± SD)
VI	A	714	—	11·6 ± 7·9
	B	420	—	5·1 ± 3·3
	C	536	—	13·0 ± 9·1

Spectral analyses of the host populations

Treatment	Replicate	Overall cycle period (days)	1st data section§ cycle period (days)	2nd data section¶ cycle period (days)
VF	A	41·8	42·4	37·9
	B	40·0	42·5	32·8
	C	41·0	41·1	37·9
VI	A	44·8	—	—
	B	47·1	—	—
	C	44·9	—	—

*VF = virus-free populations. VI = virus-infected populations.
†Mean density of adults overall per 484 cm^2.
‡Density of adults in each cycle.
§Length of sections were 336, 294 and 326 days for A, B and C, respectively.
¶ Length of sections were 224, 242 and 224 days for A, B and C, respectively.

however, after approximately nine cycles, a clear decrease in the period between adult emergence became evident. For each population, therefore, the data were divided after nine cycles and the two parts re-analysed. For all three replicates, the second part of the data exhibited a significantly shorter underlying periodicity than the first (32·8–37·9 days compared to 41·1–42·5 days; ANOVA, $F_{1,13} = 6{\cdot}14$, 33·17 and 17·45 for A, B and C, respectively, $P < 0{\cdot}05$) (Table 1). As well as a reduced cycle period, the fluctuations in the latter half of each replicate suggest that the integrity of their discrete cycles was being lost (Fig. 1a–c).

VIRUS-INFECTED POPULATIONS

The VI replicates exhibited fluctuations that corresponded to 17, 10 and 13 discrete cycles (A, B and C, respectively, Fig. 2a–c), and the pattern of adult emergence was the same as in the VF populations. The maximum adult peaks were 260, 283 and 277 (A, B and C), and the minimum peaks were 101, 136 and 106 adults (A, B and C). As above, there was no significant difference between the mean population densities overall or the mean densities per cycle between the replicates (ANOVA) (Table 1).

Unlike the virus-free populations, the VI replicates exhibited a stable pattern of fluctuations, in which the cycles remained discrete throughout. The smoothed spectra described the entire data sets in terms of one underlying periodicity, corresponding to cycle periods of 44·8, 47·1 and 44·9 days (A, B and C, respectively, Table 1 and Fig. 2d–f), and there was no significant difference between them (ANOVA).

There was an increase in the level of infection in all three populations. The mean density of infected larvae per section was 11·6, 5·1 and 13·0 in A, B and C, respectively, compared with an initial density of two per section (Table 1). In replicates A and C an overall rising trend was evident in the level of infection, but not in B (Figs 3–5). The fluctuations in the number of virus-infected larvae was highly variable, and, after detrending the data where necessary (Chatfield 1989), the smoothed spectra suggested that they exhibited essentially non-cyclical dynamics. (The host-density

spectral analyses in Figs 1 and 2, by contrast, show unequivocal evidence of cycles.) However, replicates B and C did exhibit some slight degree of periodicity corresponding to that of their host populations (Figs 3–5).

COMPARISON BETWEEN VF AND VI POPULATIONS

Adult numbers

The combined mean cycle periods from each complete replicate were compared to test for the overall effect of continuous presence of virus on the host population. The increase in mean ($\pm$SD) cycle period of the VI population compared with the VF population was significant ($45{\cdot}6 \pm 1{\cdot}3$ and $40{\cdot}9 \pm 0{\cdot}90$ days, respectively; $t_4 = 5{\cdot}11$, $P < 0{\cdot}01$). There was also a significant reduction in the mean ($\pm$SD) densities of adults between the VF and the VI populations ($42{\cdot}0 \pm 61{\cdot}8$ and $33{\cdot}2 \pm 53{\cdot}3$ adults, respectively; $D = 2{\cdot}345$, $P < 0{\cdot}01$).

Size and sex ratio

The data collected for size and sex ratio from each of one VF and VI population are summarized in Table 2. The sex ratios were arcsine transformed for analysis. For both populations, the mean wing length of females and males increased significantly from phase

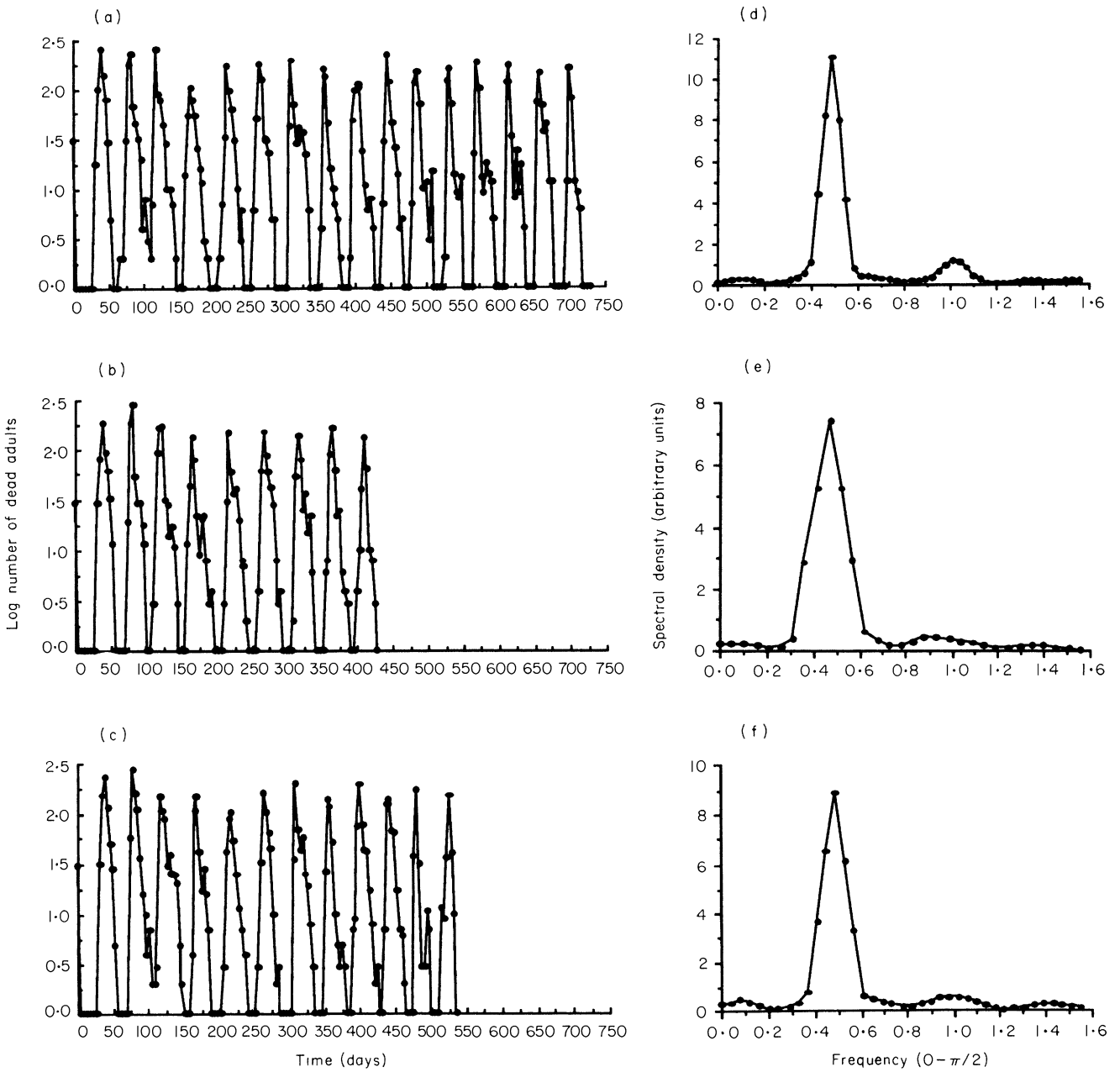

Fig. 2. (a–c) Population fluctuations from the three virus-infected (VI) replicates recorded as twice-weekly counts of dead adults. (d–f) Power spectra of each replicate illustrating that the cycles could be described by one underlying periodicity.

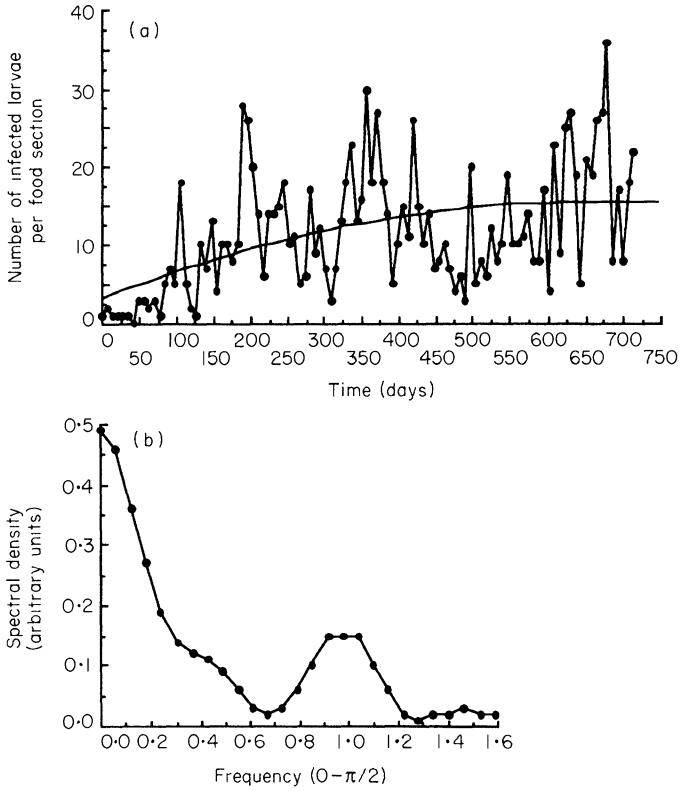

Fig. 3. (a) Fluctuation in numbers of infected larvae found in food sections removed from VI replicate A. Increasing trend shown by regression ($y = 3{\cdot}17 + 3{\cdot}65 \times 10^{-2}x + 2{\cdot}68 \times 10^{-5}x^2$, $r^2 = 0{\cdot}23$; $P < 0{\cdot}001$). (b) Power spectrum of detrended data showing noisy characteristics (compare with spectra in Figs 1 & 2).

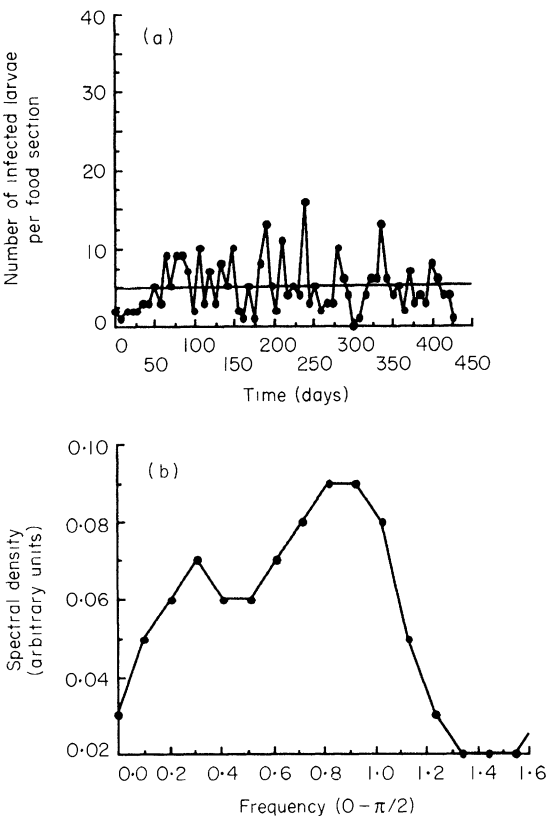

Fig. 4. (a) Fluctuation in numbers of infected larvae found in food sections removed from VI replicate B. No increasing trend in level of infection. (b) Power spectrum of data.

2 to phase 3 as density decreased, but there was no difference in the proportion of males or females between the 3 phases. There were no significant differences in size or proportion of each sex either within or between the VF and VI populations.

Discussion

VIRUS-FREE POPULATIONS

The present results are directly comparable with and remarkably similar to those recorded in another long-term study of *P. interpunctella* populations (Jones 1986, based on unpublished data of J. H. Lawton; see also Gurney *et al.* 1983). Also, Gordon & Stewart (1988) described the dynamics (unpublished) of long-term populations of another pyralid stored products pest, closely related to *P. interpunctella*, *Ephestia cautella*. Like the present study, populations fluctuated with a period slightly in excess of the egg to adult emergence time.

As with other insects, a number of authors have found that larval mortality and development rates were increased, whilst adult fecundity decreased in *P. interpunctella* when food was limited (Snyman 1949; Williams 1964; Benson 1973; Podoler 1974), and that cannibalism occurred at high population densities (Benson 1973; Podoler 1974; Jones 1986). In the case of the Lawton study, the age-structured model of Gur-

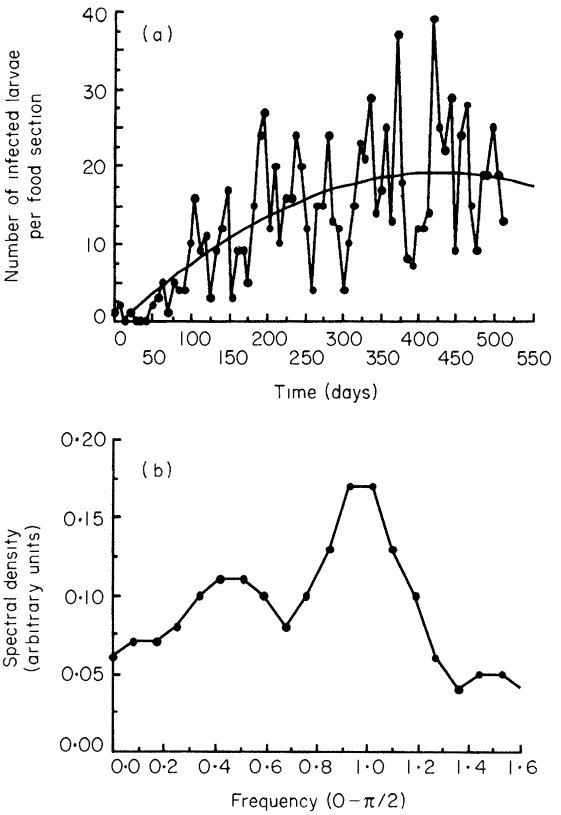

Fig. 5. (a) Fluctuation in numbers of infected larvae found in food sections removed from VI replicate C. Increasing trend shown by regression ($y = -1{\cdot}11 + 9{\cdot}51 \times 10^{-2}x - 1{\cdot}11 \times 10^{-4}x^2$, $r^2 = 0{\cdot}46$; $P < 0{\cdot}001$). (b) Power spectrum of detrended data.

ney *et al.* (1983) suggested that the form of competition for food was crucial in determining the nature and cycle period of observed fluctuations. If all larvae competed against one another for resources, cycles with a period close to the generation time were generated. If only larvae of a given age were competing, the resulting cycles had a period of more than two generations (overlapping). This work was extended by Jones (1986) who suggested that larval competition for food and cannibalism were almost entirely responsible for the long-term dynamics of the Lawton population. Similarly, Gordon & Stewart (1988) ascribed the approximate one-generation periodic fluctuations observed in *E. cautella* to the direct effect of larval competition and other inter-stage interactions. However, the model of Gordon *et al.* (1988), which did not include cannibalism, tended to overestimate the effects of food exhaustion.

Thus, the population dynamics observed in the virus-free *P. interpunctella* populations appear be driven predominantly by intense larval competition for food, resulting in starvation and cannibalism of eggs and early instars. The smallest instars would more readily succumb to starvation by being outcompeted for the remaining food, and to cannibalism from the larger instars. Only those at the 'head' of each cohort, that managed to obtain enough nutrients and avoid being eaten, will have developed into the next generation of adults. Those at the 'tail', failing to survive, would destroy any tendency for the population to move towards a complete overlap of stages, resulting in the observed population cycles.

The reduction in cycle period evident in the later generations has not been observed in other studies. One obvious explanation is selection for more rapid development. Clearly, those larvae that develop more quickly will be able to pass more quickly through the most vulnerable stages. The decrease in cycle period appeared to occur quite suddenly, however, and it is not clear why this should have been so.

VIRUS-INFECTED POPULATIONS

As with the virus-free cultures, the virus-infected populations exhibited remarkably similar discrete cyclic fluctuations. However, their periods were both significantly longer than those of the VF populations and showed no evidence of a changing cycle period. The mean abundances of adults in the VI populations were also very similar, and were also significantly lower than those in the VF populations.

It would appear, then, that explanations for the cycles themselves in the VI populations do not emerge readily from the theoretical literature on host–pathogen dynamics, since, as explained, this tends to envisage cycles being generated by a host–pathogen interaction in an otherwise non-cyclic host population. Rather, the dynamics observed in these populations are most likely, as in the VF populations, to have been largely determined by density-dependent larval interactions. However, as we now discuss, the increased period of their fluctuations, perhaps even the increased constancy in their cycles, and the reduced host abundance, could be explained by age-dependent virus-induced mortality (Sait *et al.* 1994a), by the effects of sublethal virus infections (Sait *et al.* 1994b), by evolutionary change in the host population (Boots & Begon 1993), but also by other processes that have been highlighted in theoretical studies.

Any factor that changed the intensity of density-dependent competition might alter the pattern of fluctuations. Virus-induced mortality may have supplemented the effects of larval competition since early instars would die from starvation when food was depleted, would be cannibalized by more resistant larger instars, but would also die from disease. However, if intense enough, virus-induced mortality might reduce the host population so that the level of competition for resources was decreased, allowing a much more mixed-age population to develop. The difference between adult size recorded from the VF and VI populations was not significant, but it was interesting to note that for any particular phase of emergence, male and female adults from the VI populations were larger. This may have been evidence of

Table 2. Summary of changes in size and sex ratio from a randomly chosen virus-free (VF) and virus-infected (VI) population

Size			
Population	Phase	n*	Wing length (mm)† (mean ± SD)
Female			
VF	1	—	—
	2	15	6·40 ± 0·31a
	3	11	6·71 ± 0·17b
VI	1	—	—
	2	10	6·54 ± 0·30a
	3	7	6·82 ± 0·33b
Male			
VF	1	—	—
	2	15	5·65 ± 0·24c
	3	11	5·77 ± 0·16d
VI	1	—	—
	2	10	5·75 ± 0·23c
	3	7	5·92 ± 0·32d

Sex ratio			
Population	Phase	n*	Sex ratio† (Proportion of females) (mean − SD, mean, mean + SD)
VF	1	6	0·47 − 0·56 + 0·65a
	2	15	0·51 − 0·56 + 0·62a
	3	13	0·48 − 0·57 + 0·65a
VI	1	7	0·40 − 0·51 + 0·62a
	2	10	0·51 − 0·56 + 0·60a
	3	7	0·50 − 0·58 + 0·67a

*Number of cycles from which adults were measured for wing length and sex ratio recorded.

†Means followed by different letters are significantly different ($P < 0·05$).

reduced larval competition as a result of disease-induced mortality.

On the other hand, virus pathogenicity in the populations could have been enhanced, since it has been suggested that poor food quality and high levels of crowding in other species leads to increases in susceptibility to baculovirus disease (David & Gardiner 1965; David & Taylor 1977; Biever & Wilkinson 1978). Moreover, in periods of depleted food and high rates of cannibalism, it is likely that healthy larvae would eat more dead or dying infecteds, increasing the transmission of virus throughout the population and the prevalence of infection (Evans & Allaway 1983; Young & Yearian 1988; Woods *et al.* 1991). Thus, direct lethal effects of the GV on the host could be expected to contribute to the lower average abundance in the host population, though the process is likely to have been complex.

Sait *et al.* (1994b) showed that there was evidence of an increased development time in *P. interpunctella* larvae sublethally infected with the GV and that the reproductive capacity of sublethally infected individuals is significantly reduced. Sublethal effects may therefore have contributed to the greater cycle period observed in the VI populations and, through reduced recruitment, to the lower abundance.

In addition, however, these same VI populations have been shown to have evolved increased resistance to the pathogen (Boots & Begon 1993), associated with which were significantly and substantially increased rates of development and marginally (though significantly) decreased egg viability (as trade-offs), and significant increases in size (as a positive correlate). Vail & Tebbets (1990) also found, in a comparative study, that more resistant *P. interpunctella* strains took longer to develop and tended to be larger. Furthermore, the cycle period of VI replicate B was 47 days compared with 45 days in the other two populations, and this was associated with a lower and non-increasing level of infection in comparison with the other two replicates. This could have been due to greater resistance in replicate B associated with an increased development time as a trade-off.

Theory, directly related to host–pathogen interactions (Hassell & Anderson 1989; Hochberg 1989; Hochberg *et al.* 1990) and more generally (Taylor 1993), suggests that the failure of the pathogen to destabilize host dynamics and its possible tendency even to enhance their stability, may have been due to the stabilizing effects of heterogeneities present even within this simple laboratory system. More specifically, the conscious retention of infected cadavers

within the present system is reminiscent of the long-lived reservoir effect envisaged by Hochberg (1989) and shown by him to be capable of enhancing stability. It is noteworthy, too, that while in the present case interstage interactions were sufficiently intense to generate 'generational' cycles in the absence of the pathogen, the enhancement of such effects by the pathogen, in the ways previously described, raises the interesting possibility that pathogens may be capable themselves of generating cycles in host populations through their influences on the population's stage structure.

The heterogeneity arising out of stage structure is itself related, in our system, to another type of heterogeneity, namely the interconnected spatial variations in food quality, host density and disease prevalence. It is possible, for example, that if the one-sixth of the food changed each week had been distributed amongst a large number of small patches, then the encounter rates between individuals in different stages and between susceptible and infected individuals would have been greater. The effects described above may then have assumed even greater importance, and the population dynamics may have moved into even more pronounced cycles or perhaps into some qualitatively different pattern altogether.

Any pattern in the level of infection was much less definite than the cycles in host abundance, although there was some hint of linked host–pathogen dynamics. This link between host and pathogen dynamics is further supported by the previously mentioned association between host and infection patterns in VI replicate B. The index of infection in the population was conservative, as a result of the sampling method used. First instars were not sampled because of the time and effort involved, but their greater susceptibility (Sait *et al.* 1994a) would suggest that many more succumbed to the disease than the other instars. It is likely, however, that the number of infected larvae sampled reflected any overall trends in the infected population. Fleming *et al.* (1986) investigated possible density-dependent virus mortality in the soil-dwelling pasture pest *Wiseana* sp. (Lepidoptera: Hepialidae), and showed that this relationship only existed in young pastures. In old pastures, the lack of density dependence was attributed to the accumulation of virus from previous generations, since contamination of the environment with virus in this manner has been shown to be one of the most effective means of transmission to other generations (Kellen & Hoffmann 1987; Young 1990; Woods *et al.* 1991). A similar situation probably existed in the VI population cages, where high levels of virus would rapidly accumulate and persist for long periods, and may explain the lack of density-dependent levels of infection.

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References

Anderson, R.M. (1991) Populations and infectious diseases: ecology or epidemiology. *Journal of Animal Ecology*, **60**, 1–50.

Anderson, R.M. & May, R.M. (1981) The population dynamics of microparasites and their invertebrate hosts. *Philosophical Transactions of the Royal Society, London B*, **291**, 451–524.

Anderson, R.M. & May, R.M. (1986) The invasion, persistence and spread of infectious diseases within animal and plant communities. *Philosophical Transactions of the Royal Society, London B*, **314**, 533–570.

Benson, J.F. (1973) The biology of Lepidoptera infesting stored products, with special reference to population dynamics. *Biological Reviews*, **48**, 1–26.

Benson, J.F. (1974) Population dynamics of *Bracon hebetor* Say (Hymenoptera: Braconidae) and *Ephestia cautella* (Walker) (Lepidoptera: Phycitidae) in a laboratory ecosystem. *Journal of Animal Ecology*, **43**, 71–86.

Biever, K.D. & Wilkinson, J.D. (1978) A stress-induced granulosis virus of *Pieris rapae*. *Environmental Entomology*, **7**, 572–573.

Bird, F.T. & Elgee, D.E. (1957) A virus disease and introduced factors controlling the European spruce sawfly, *Diprion hercyniae* (Hartig) Central New Brunswick. *Canadian Entomologist*, **139**, 371–372.

Boots, M.R.J. & Begon, M. (1993) Trade-offs with resistence to a granulosis virus in the Indian meal moth, examined by a laboratory evolution experiment. *Functional Ecology*, **7**, 528–534.

Bowers, R.G., Begon, M. & Hodgkinson, D.E. (1993) Host-pathogen population cycles in forest insects? Lessons from simple models reconsidered. *Oikos*, **67**, 529–538.

Chatfield, C. (1989) *The Analysis of Time Series: An Introduction*, 4th edn. Chapman & Hall, London.

David, W.A.L. & Gardiner, B.O.C. (1965) The incidence of granulosis deaths in susceptible and resistant *Pieris brassicae* (Linnaeus) larvae following changes of population density, food and temperature. *Journal of Invertebrate Pathology*, **7**, 347–355.

David, W.A.L. & Taylor, C.E. (1977) The effect of sucrose content of diets on the susceptibility to granulosis virus in *Pieris brassicae*. *Journal of Invertebrate Pathology*, **30**, 117–118.

Evans, H.F. & Allaway, G.P. (1983) Dynamics of baculovirus growth and dispersal in *Mamestra bassicae* L. (Lepidoptera: Noctuidae) larval populations introduced into small cabbage plots. *Applied Environmental Microbiology*, **45**, 493–501.

Fleming, S.B., Kalmakoff, J., Archibold, R.D. & Stewart, K.M. (1986) Density-dependent virus mortality in populations of *Wiseana* (Lepidoptera: Hepialidae). *Journal of Invertebrate Pathology*, **48**, 193–198.

Gordon, D.M., Gurney, W.S.C., Nisbet, R.M. & Stewart, R.K. (1988) A model of *Cadra cautella* larval growth and development. *Journal of Animal Ecology*, **57**, 645–658.

Gordon, D.M. & Stewart, R.K. (1988) Demographic characteristics of the stored-products moth *Cadra cautella*. *Journal of Animal Ecology*, **57**, 627–644.

Granados, R.R. (1980) Infectivity and mode of action of baculoviruses. *Biotechnology and Bioengineering*, **22**, 1377–1405.

Gurney, W.S.C., Nisbet, R.M. & Lawton, J.H. (1983) The systematic formulation of tractable single-species population models incorporating age structure. *Journal of Animal Ecology*, **52**, 479–495.

Hamilton, J.T. (1979) Seasonal abundance of *Pieris rapae* (L.), *Plutella xylostella* (L.) and their diseases and parasites. *General Applied Entomology*, **11**, 59–66.

Hassell, M.P. & Anderson, R.M. (1989) Predator–prey and host–pathogen interactions. *Ecological Concepts* (ed. J.M. Cherrett), Symposium 29 of the British Ecological Society. Blackwell Scientific Publications, Oxford.

Hochberg, M.E. (1989) The potential role of pathogens in biological control. *Nature*, **337**, 262–265.

Hochberg, M.E., Hassell, M.P. & May, R.M. (1990) The dynamics of host–pathogen–parasitoid interactions. *American Naturalist*, **135**, 74–94.

Hofmaster, R.N. (1961) Seasonal abundance of the cabbage looper as related to light trap collections, precipitation, temperature and the incidence of a nuclear polyhedrosis virus. *Journal of Economic Entomology*, **54**, 796–798.

Jones, A. (1986) *Stage-structured insect population models of larval competition*. PhD thesis, University of Strathclyde, UK.

Kellen, W.R. & Hoffmann, D.F. (1987) Laboratory studies on the dissemination of a granulosis virus by healthy adults of the Indian meal moth, *Plodia interpunctella*, (Lepidoptera: Pyralidae). *Environmental Entomology*, **16**, 1231–1234.

May, R.M. (1985) Regulation of populations with non-overlapping generations by microparasites: a purely chaotic system. *American Naturalist*, **125**, 573–584.

Myers, J.H. (1988) Can a general hypothesis explain population cycles of forest Lepidoptera? *Advances in Ecological Research*, **18**, 179–232.

Onstad, D.W. & Carruthers, R.I. (1990) Epizootiological models of insect diseases. *Annual Review of Entomology*, **35**, 399–419.

Payne, C.C. (1988) Pathogens for the control of insects: Where next? *Philosophical Transactions of the Royal Society, London, B*, **318**, 225–248.

Podoler, H. (1974) Effects of intraspecific competition in the Indian meal moth (*Plodia interpunctella* (Hübner) (Lepidoptera: Phycitidae) on populations of the moth and its parasite *Nemeritis canescens* (Gravenhorst) (Hymenoptera: Ichnemonidae). *Journal of Animal Ecology*, **43**, 641–651.

Richards, O.W. & Waloff, N. (1946) The study of a population of *Ephestia elutella* Hübner (Lep.: Phycitidae) living on bulk grain. *Transactions of the Royal Entomological Society of London*, **97**, 253–298.

Sait, S.M., Begon, M. & Thompson, D.J. (1994a) The influence of larval age on the response of *Plodia interpunctella* to a granulosis virus. *Journal of Invertebrate Pathology*, **63**, 107–110.

Sait, S.M., Begon, M. & Thompson, D.J. (1994b) The effects of a sublethal baculovirus infection in the Indian meal moth, *Plodia interpunctella*. *Journal of Animal Ecology*, **63**, 541–550.

SAS Institute Inc. (1988) *SAS/ETS User's Guide, Version* 6, 1st edn. SAS Institute Inc., Cary, NC, USA.

Snyman, A. (1949) The influence of population densities on the development and oviposition of *Plodia interpunctella* Hübn. (Lepidoptera). *Journal of the Entomological Society of South Africa*, **12**, 137–171.

Taylor, A.D. (1993) Heterogeneity in host-parasitoid interactions: the CV^2 rule. *Trends in Ecology and Evolution*, **8**, 400–405.

Thompson, C.G. & Scott, D.W. (1979) Production and persistence of the nuclear polyhedrosis virus of the douglas-fir tussock moth, *Orygia pseudotsugata* (Lepidoptera: Lymantriidae), in the forest ecosystem. *Journal of Invertebrate Pathology*, **33**, 57–65.

Vail, PV. & Tebbets, J.S. (1990) Comparative biology and susceptibility of *Plodia interpunctella* (Lepidoptera: Pyralidae) populations to a granulosis virus. *Environmental Entomology*, **19**, 791–794.

White, E.G. & Huffaker, C.B. (1969a) Regulatory processes and population cyclicity in laboratory populations of *Anagasta kühniella* (Zeller) (Lepidoptera: Phycitidae). I. Competition for food and predation. *Research in Population Ecology*, **11**, 57–83.

White, E.G. & Huffaker, C.B. (1969b) Regulatory processes and population cyclicity in laboratory populations of *Anagasta kühniella* (Zeller) (Lepidoptera: Phycitidae). II. Parasitism, predation, competition and protective cover. *Research in Population Ecology*, **11**, 150–185.

Williams, G.C. (1964) The life-history of the Indian meal moth, *Plodia interpunctella* (Hübner) (Lepidoptera: Phycitidae), in a warehouse in Britain on different food. *Annals of Applied Biology*, **53**, 459–475.

Woods, S.A., Elkington, J.S., Murray, K.D., Liebhold, A.M., Gould, J.R. & Podgwaite, J.D. (1991) Transmission dynamics of a nuclear polyhedrosis virus and predicting mortality in gypsy moth (Lepidoptera: Lymantriidae) populations. *Journal of Economic Entomology*, **84**, 423–430.

Young, S.Y. (1990) Effect of nuclear polyhedrosis virus infection in *Spodoptera ornithogalli* larvae on post larval stages and dissemination by adults. *Journal of Invertebrate Pathology*, **55**, 69–75.

Young, S.Y. & Yearian, W.C. (1988) Secondary transmission of nuclear polyhedrosis virus by *Pseudoplusia includens* and *Anticarsia gemmatalis* larvae on semisynthetic diet. *Journal of Invertebrate Pathology*, **51**, 133–138.

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