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# Host-pathogen population dynamics, basic reproductive rates and threshold densities

**Robert J. Knell, Michael Begon and David J. Thompson**

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Populations of the Indian meal moth, *Plodia interpunctella*, were maintained in the laboratory in the presence of 2 pathogens, *P. interpunctella* Granulosis Virus (PiGV) and *Bacillus thuringiensis*, for between 9 and 12 generations. Neither pathogen had any discernible effect on the population dynamics of the host, either individually or in combination.

The dynamics of the two pathogens were very different, with PiGV persisting within the host population and *B. thuringiensis* usually becoming extinct. This behaviour can be explained by the host threshold density and the relationship between host density and the basic reproductive rate ($R_0$) for each pathogen. The host threshold density was considerably less for PiGV than for *B. thuringiensis*, indicating that PiGV would be able to persist in much lower density populations than *B. thuringiensis*. None of the populations of *P. interpunctella* showed the generation-length cycles which have previously been described from similar experiments. This may be due to the quality, and possibly also the quantity of food available to the larvae.

*R. J. Knell, M. Begon and D. J. Thompson, Population Biology Research Group, School of Biological Sciences, Univ. of Liverpool, PO Box 147, Liverpool, UK L69 3BX (present address of RJK: Dept of Zoology, Univ. of Witwatersrand, Private Bag 3, Wits 2050, South Africa [robk@gecko.biol.wits.ac.za]).*

Following the realisation that the population dynamics of invertebrates can be significantly influenced by pathogens such as viruses, fungi and bacteria (Anderson and May 1981) there have been a number of studies published in which the interactions between an insect host and a pathogen have been described at a population level (e.g. Dwyer and Elkington 1993, Sait et al. 1994; see Briggs et al. 1995 for a review). These studies have all concentrated on the dynamics of a single pathogen. In this study we consider a laboratory system consisting of a host insect, *Plodia interpunctella* (Hübner), the Indian meal moth, and two pathogens: *P. interpunctella* Granulosis Virus (PiGV) and the bacterial pathogen *Bacillus thuringiensis*. This enables us to make comparisons between the population dynamics of two different pathogens infecting the same host population.

To account for the differences between the population dynamics of the two pathogens we present a calculation of the basic reproductive rates ($R_0$) and the threshold densities for transmission for the two pathogens. The basic reproductive rate is defined as the number of new infections produced in a completely susceptible host population by a single infectious individual (Anderson and May 1979, 1981), and the threshold density is the density of susceptible hosts at which $R_0$ is equal to one. If the density of susceptible hosts is less than this threshold density then each infectious individual will produce less than one new infection, and so the disease will not persist in the host population. If the density of susceptible hosts is greater than the threshold density, then each infectious individual will produce more than one new infection, and so the disease will sustain itself. An understanding of $R_0$

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and the threshold density, and especially of the overall relationship between $R_0$ and host density may be of considerable importance when seeking to understand the population dynamics of pathogens. In particular, the large-scale patterns observed in the population experiments seem to be explained well by differences between the $R_0$-density relationships for the two pathogens.

Many studies of laboratory ecosystems have used the group of Lepidoptera often referred to as "storage moths" (Flanders and Badgeley 1963, Flanders and Hall 1965, Flanders 1968, Hassell and Huffaker 1969, White and Huffaker 1969a, b, Benson 1972, Takahashi 1972, Podoler 1974). Recent work has included the use of data from populations of *P. interpunctella* in the construction of sophisticated population models (Gurney et al. 1983, Jones 1986), an investigation of the dynamics of a host-pathogen system, namely *P. interpunctella* and PiGV (Sait et al. 1994), studies of the population dynamics of *P. interpunctella* with the parasitoid *Venturia canescens* (Begon et al. 1995) and studies of the dynamics of *P. interpunctella* with both the parasitoid and the virus present (Begon et al. 1996).

*Plodia interpunctella* is therefore an ideal subject for this study because a considerable amount is known about how populations of the moth behave in laboratory ecosystems, both by itself and in the presence of natural enemies. PiGV is an obvious choice for one of the pathogens as it has been used in similar studies before. *B. thuringiensis* is the most widely used microbial control agent, and yet remarkably little is known of its ecology. One of the environments in which *B. thuringiensis* seems to function as a true entomopathogen is the stored product environment, in which it is known to appear in sudden devastating epizootics in pest insect populations, although it is not known to regulate any of these populations normally (Burges and Hurst 1977, Delucca et al. 1982, Meadows et al. 1992, Meadows 1993). As the environment in which *P. interpunctella* is maintained in the laboratory approximates to a stored product environment, it was hoped that this study would shed light on the ecology of this pathogen and lead to a greater understanding of its relationships with other natural enemies.

## Methods

### Population experiments

The experiments followed the protocols described by Sait et al. (1994) and Begon et al. (1995). Each population was kept in a plastic box, $22 \times 22 \times 13$ cm, with a sleeve of nylon netting attached to the opening to allow access by the experimenter, and a lid, pierced for ventilation, securing the sleeve. The base of each box was divided into six equal sections. Twenty g of food, consisting of 10:1:1:1 parts wheat bran:yeast:glycerol:honey was placed into each section and spread evenly into a layer approximately 0.5 cm thick.

Populations of *P. interpunctella* were established by adding 15 female and 15 male late 5th instar larvae (5th instar larvae can be sexed, as the gonads of the males are visible) to each population cage. The pathogens were introduced by adding 2 5th instar larvae freshly killed by the appropriate pathogen to each section of food, for a total of 12 per population cage, this being a quantity of pathogen which had been found to successfully introduce PiGV into previous populations. The population cages were kept in an incubator at $28 \pm 2$°C (range) with a 16:8 L:D cycle.

Once the populations were initiated, one section of food was removed and replaced with fresh food once per week in sequence. The populations of the moth were estimated by counting the dead adults present once per week, giving a measure of the cumulative population present in the previous week. As larvae of the moth live within the food and early instar larvae are very small, it was not feasible to obtain estimates of the overall larval populations. Numbers of infected larvae and cadavers found in each of the sections of food removed were recorded, however, to give an approximate index of the pathogen population present. Once counted, infected larvae and cadavers were returned to the population cage. Larvae infected with or killed by PiGV (white) are easily distinguished from those infected with *B. thuringiensis* (black). Both are also different in appearance to healthy larvae or those which have died from other causes.

Three population cages were set up with both *B. thuringiensis* and PiGV present, and three with *B. thuringiensis* only present. As a considerable amount is already known about the dynamics of both *P. interpunctella* alone and *P. interpunctella* with PiGV in very similar systems (Sait et al. 1994, Begon et al. 1995, 1996) only one with PiGV alone and one with no pathogens were initiated.

When the populations had been running for 25 weeks, it was noted that *B. thuringiensis* was either extinct or nearly extinct in 5 out of the 6 cages into which it had been introduced, and it was reintroduced into these cages at a density of 8 freshly killed *B. thuringiensis* infected 5th instar larvae per food section, for an overall total of 48 infectious cadavers per cage. At the same time, due to a suspicion that infected cadavers were surviving the six weeks between each section of food being replaced, and therefore being counted twice, cadavers were marked with white or black paint before they were replaced in the cages. The ages of infected larvae and cadavers were also recorded from this point.

### Calculating the basic reproductive rate and the threshold density

For directly transmitted microparasites, the basic reproductive rate can be calculated by multiplying the rate at which new infections are produced by a single infectious individual by the period of time for which that individual remains infectious. The formula given for $R_0$ of a basic host-parasite model (Anderson and May 1981), for example, is as follows:

$$R_0 = \frac{\beta X}{(\alpha + b + \gamma)}. \quad (1)$$

In this expression $\beta$ is the transmission coefficient, representing the probability of the disease being transmitted per contact between an infectious individual and a susceptible host and $X$ is the density of susceptible hosts. $\beta X$ is therefore the rate at which new infections are produced, and $1/(\alpha + b + \gamma)$ is the average life span of an infectious individual: $\alpha$ is the increase in the death rate due to the disease, $b$ is the underlying death rate of healthy hosts and $\gamma$ is the rate of recovery of infectious hosts.

The basic reproductive rate for the two pathogens considered here differs from eq. 1, for two reasons. Firstly, rather than the direct transmission from one live but infected host to a healthy host assumed above, transmission of both *B. thuringiensis* and PiGV is largely by cannibalism of infectious cadavers (Knell 1996). Secondly, the rate of production of new infections in eq. 1 assumes that transmission is proportional to the density of susceptible hosts multiplied by the transmission coefficient: "mass action" transmission. This is a standard assumption about transmission of pathogens (Anderson and May 1979), but it has recently been demonstrated that transmission of neither *B. thuringiensis* (Knell et al. 1996) nor PiGV (Knell et al. 1998) conforms to this model. In both cases the transmission coefficient increased with the density of susceptible hosts. For these reasons a modified expression for $R_0$ is necessary:

$$R_0 = \frac{(\beta' + aX)X}{\mu + dX}. \quad (2)$$

In both cases the transmission coefficient increased in a linear way with the density of susceptible hosts. $\beta'$ is the intercept of this relationship, and $a$ is the slope. This gives a rate of production of new infecteds of $(\beta' + aX)X$. The time during which an infectious cadaver is able to produce new infections is given as $1/(\mu + dX)$, with $\mu$ being the decay rate of the infectious cadaver and $d$ the rate at which infectious cadavers are removed by cannibalism.

When the population of susceptible hosts is at the threshold density, all infectious cadavers will produce an average of one new infection throughout their life span. This condition is equivalent to setting $R_0$ equal to one, and this can be used to derive the threshold density from eq. 2:

$$1 = \frac{(\beta' + aX_T)X_T}{\mu + dX_T}, \quad (3)$$

which can be rearranged to give

$$aX_T^2 + (\beta' - d)X_T - \mu = 0. \quad (4)$$

Only one of the two roots of this quadratic equation is relevant here, the other being negative. Estimates for all of the parameters in this expression are available (Knell 1996, Knell et al. 1996, 1998), and so a value can be calculated for $R_0$ and for the host threshold density for both of the pathogens used in this study.

## Results

### Population experiments

Fig. 1 shows the population trajectories obtained for each of the long-term population experiments, and Table 1 gives a set of summary statistics. Most of the experiments ran for 51 or 52 weeks, 11–12 generations, the exception being population G, which ran for 42 weeks, or 9–10 generations.

### The host populations

All the host populations, with the possible exception of population G, underwent a few oscillations which were quickly damped. There was a suggestion of an upward trend in abundance in each case.

As regards the mean numbers of moths found, the data are striking in their similarity. None of the populations is different from any other (ANOVA, $p = 0.952$), and all but two of these means lie within the range 61.4–64.0. In these experiments, therefore, none of the combinations of pathogens had any discernible effect on the overall densities of the host populations.

In order to investigate whether there was any significant periodicity in the data, the trajectories for the *P. interpunctella* populations were analysed using autocorrelation functions (ACFs). The first 15 data points from each time series were removed because of the overriding effect of the large initial fluctuations, and the remaining data were detrended by differencing. The ACFs produced (not shown) were essentially a set of random fluctuations about zero. There were a few nearly significant peaks around the 6–8-week lag, but these are not enough, in themselves, to indicate cycles. The ACFs do not indicate any dynamic difference

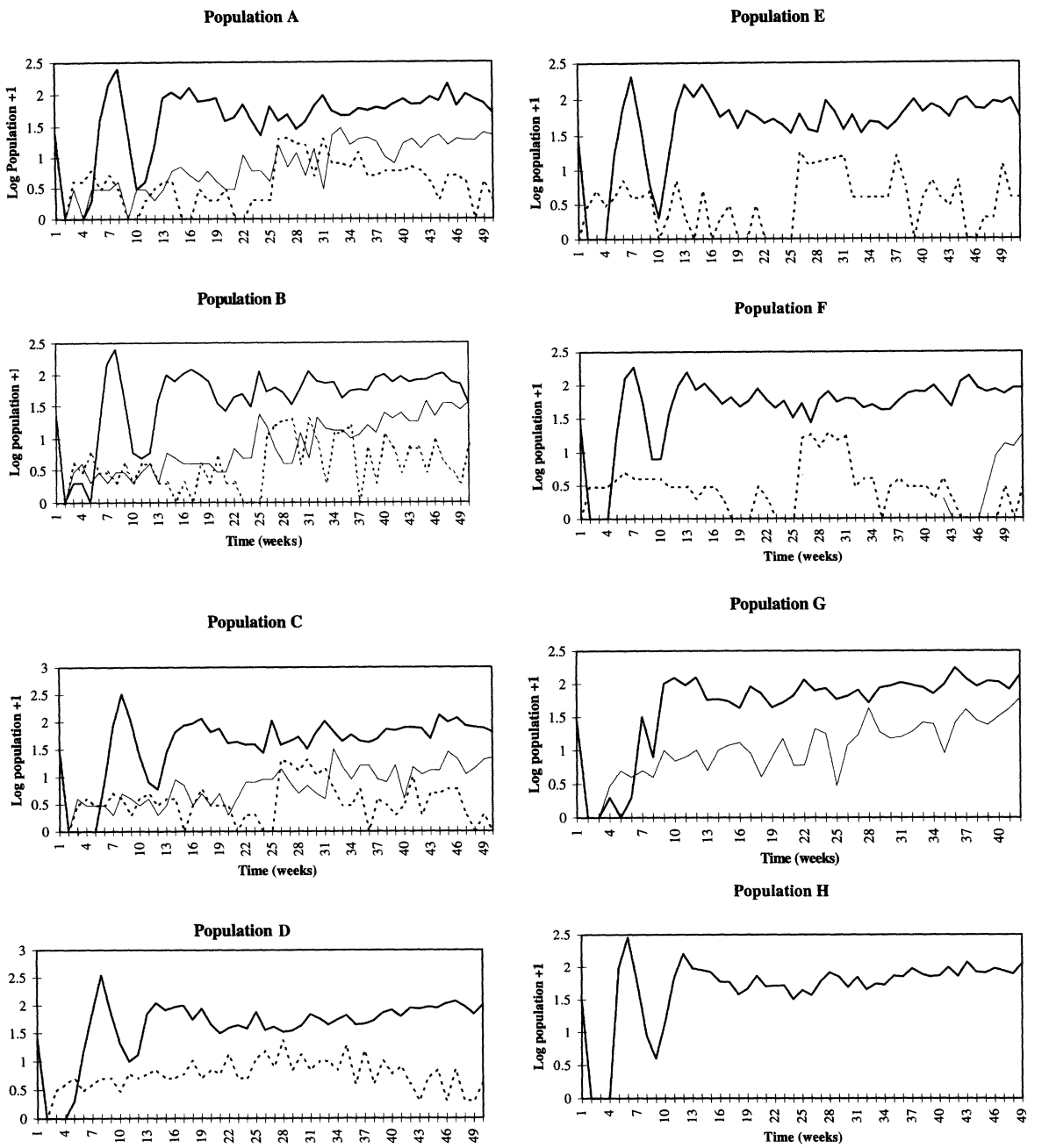


Fig. 1. Time series for each population experiment. Heavy lines are counts of dead adult moths, fine lines are numbers of larvae found infected with PiGV and dotted lines are numbers of larvae infected with *B. thuringiensis*. All population counts are shown as logs to the base 10 with 1 added.

between populations with different pathogen treatments.

To conclude, the host populations oscillated once or twice before settling down to a non-cyclic near-equilibrium with a slight rising trend. Any short-term fluctuations appear to be due to random variability. In these experiments, neither pathogen appears to have had any effect on either the mean densities or on the population dynamics of the host, either separately or together.

## The pathogen populations

*B. thuringiensis* failed to establish and was reintroduced as described above in populations A, B, C, E and F, and only persisted throughout the course of the experiments in population D. *B. thuringiensis* was not completely lost from most of the populations following reintroduction, but the numbers of larvae and cadavers found infected with *B. thuringiensis* show a distinct decline from the time of reintroduction to the point where the experiments were terminated, a decline also seen in population D over this period. PiGV persisted in all the populations into which it was introduced, and the numbers of infected larvae and cadavers found increased throughout the course of the experiments. There is therefore a difference in the behaviour of the two pathogens, with the virus persisting and increasing in numbers throughout the course of the experiments, and the bacterium failing to persist even when reintroduced at high densities.

Although both the host and the pathogen populations fluctuate, there seems to be no relationship between the two. The PiGV population trajectories were differenced to remove the rising trend and ACFs plot-ted (not shown). These showed no periodicity or other pattern in the data; nor did an ACF for the *B. thuringiensis* population from cage D – the only one which gave a time series long enough to be analysed in this way.

The population trajectories for both pathogens appear very similar in both the single-pathogen populations and in the two-pathogen systems, and there is no significant difference between any of the populations in terms of the mean numbers of *B. thuringiensis* infected cadavers found, either in the whole data set (ANOVA, 5 df. $p = 0.143$) or when the data before 25 weeks, (after which *B. thuringiensis* was reintroduced to 5 of the 6 cages and infected cadavers started to be marked so that only new infections were counted) are removed from the analysis (ANOVA, $p = 0.533$). Mixed infections are easily identifiable only in those larvae which are heavily infected with PiGV before receiving a dose of *B. thuringiensis*, and no definite cases of this were found.

Table 1. Summary statistics for the population experiments

| Population | Species | Mean population (dead adult moths) | Standard error |
|---|---|---|---|
| A | *P. interpunctella* | 61.5 | 6.24 |
| | *B. thuringiensis* | 4.3 | 0.72 |
| | PiGV | 9.3 | 1.15 |
| B | *P. interpunctella* | 62.6 | 6.34 |
| | *B. thuringiensis* | 4.5 | 0.72 |
| | PiGV | 10.1 | 1.44 |
| C | *P. interpunctella* | 61.4 | 7.13 |
| | *B. thuringiensis* | 3.8 | 0.66 |
| | PiGV | 7.9 | 0.99 |
| D | *P. interpunctella* | 62.1 | 7.34 |
| | *B. thuringiensis* | 5.7 | 0.62 |
| E | *P. interpunctella* | 61.5 | 5.64 |
| | *B. thuringiensis* | 3.9 | 0.64 |
| F | *P. interpunctella* | 64.0 | 8.51 |
| | *B. thuringiensis* | 3.1 | 0.66 |
| G | *P. interpunctella* | 71.0 | 6.36 |
| | PiGV | 14.3 | 2.05 |
| H | *P. interpunctella* | 66.7 | 6.50 |

When considering the age classes of the infected larvae, it should be noted that as total numbers of hosts are not available, the proportions of infected individuals found in each age class for each pathogen do not represent the overall prevalence of pathogen in each age class. These data nevertheless give relative prevalences and allow comparisons between the pathogen populations. Fig. 2 shows the mean proportions of larvae found infected in each age class for *B. thuringiensis* both in the presence of PiGV and alone, and for PiGV in the presence of *B. thuringiensis*. There is a clear difference between the two pathogens, a greater proportion of the virus-infected larvae being in the younger age classes whereas most of those infected by the bacterium were found in the older age classes.

The age structures of the *B. thuringiensis* populations with PiGV present or absent can be compared with a repeated measures MANOVA. A repeated measures analysis is necessary because multiple measurements have been made on the same population cage, and there are multiple response variables (proportions of infected larvae in each age class). Table 2 gives the results from this analysis.

There is no significant effect of time within the *B. thuringiensis* infected populations, nor is there any significant interaction between time and the presence or absence of PiGV. There is a significant effect of the presence of PiGV on the relative proportions of 5th and 2nd instar larvae, with proportionally more *B. thuringiensis* infected cadavers being in the 5th instar and less in the 2nd instar when PiGV was also present. A significant interaction was found between time and the presence of *B. thuringiensis* in first instar larvae only.

The PiGV populations with and without *B. thuringiensis* present cannot be compared statistically because there was no replication of the population with only PiGV present.

## $R_0$ and the host threshold density

Table 3 gives values for the parameters in eqs 2 and 5 for both of the pathogens, with all values being given for 4th instar *P. interpunctella*. In the experiments described in Knell et al. (1996, 1998) the relationship

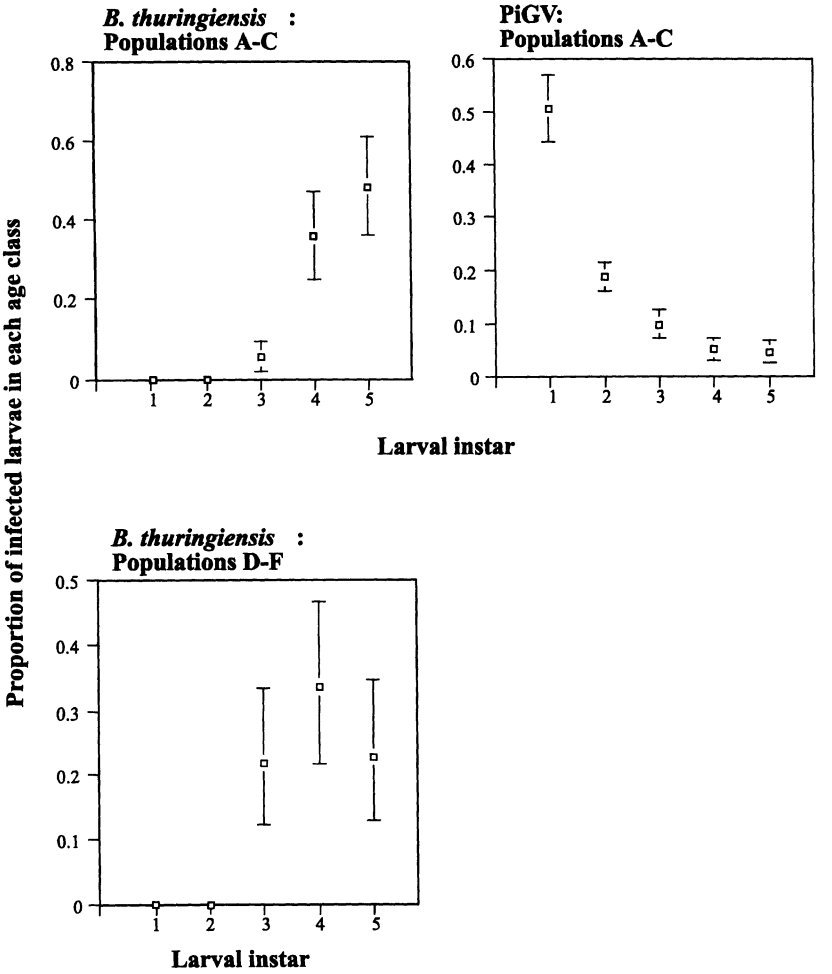


Fig. 2. Mean proportions of larvae becoming infected with each pathogen in populations A–C and D–F. Error bars are 95% confidence limits.

between the transmission coefficient and the density of susceptible hosts was determined at a density of 2 infectious cadavers. As the transmission coefficient of PiGV also changed with the density of infectious cadavers in the 4th instar the value for $\beta'$ (the intercept) for PiGV was calculated for 1 infectious cadaver assuming that the slope ($a$) does not change with the density of infectious cadavers.

The rate of removal of infectious cadavers due to cannibalism ($d$) was set at zero for *B. thuringiensis*; there was no observable decline in the amount of infectious material present over 16 h when cadavers were exposed to cannibalism by 20 susceptible hosts (Knell et al. 1996). As the time period for which cadavers remain infectious in the absence of susceptible hosts is of the order of days, the removal of infectious material by cannibalism over this time is likely to be negligible. The rate for PiGV was calculated by performing a regression of the slopes of relationships between transmission coefficient and time against the density of susceptible hosts present anchored through the origin, with the slopes weighted by the inverse of their variances. The rate is the negative of the slope of this regression. This rate was then recalculated to a rate per day, rather than per hour, in order to give conformity with the decay rate in the absence of susceptible hosts ($\mu$).

Using these values we can now produce an expression for the respective $R_0$ values for the two pathogens using eq. 2.

$$\text{For } B.\ thuringiensis\ R_0 = \frac{(-0.008 + 0004X)X}{0.059}, \quad \text{and}$$

$$\text{for PiGV } R_0 = \frac{(0.633 + 0.007X)X}{0.117 + 0.203X}.$$

Fig. 3 shows how $R_0$ varies for host densities between 0 and 15 for both pathogens. In the case of PiGV, $R_0$ increases rapidly at low host densities, but the rate of

Table 2. Results of repeated measures MANOVA on *B. thuringiensis* age structure. * indicates a significant result.

| Effect of time<br>Instar | Mean sqr effect | Mean sqr error | *F* (df 21, 84) | *p* |
|---|---|---|---|---|
| 1 | 0.0081 | 0.0084 | 0.9710 | 0.5060 |
| 2 | 0.0149 | 0.0173 | 0.8158 | 0.6934 |
| 3 | 0.1528 | 0.1488 | 1.0268 | 0.4420 |
| 4 | 0.2063 | 0.2238 | 0.9218 | 0.5649 |
| 5 | 0.3572 | 0.2262 | 1.5786 | 0.0744 |
| Effect of presence/absence of PiGV<br>Instar | Mean sqr error | Mean sqr error | *F* (df 1, 4) | *p* |
| 1 | 0.0051 | 0.0052 | 0.9810 | 0.3780 |
| 2 | 0.0806 | 0.0078 | 10.3728 | 0.0323* |
| 3 | 1.1909 | 0.6826 | 1.7447 | 0.2570 |
| 4 | 0.1893 | 0.7357 | 0.2572 | 0.6387 |
| 5 | 2.8379 | 0.1766 | 16.0709 | 0.0160* |
| Interaction<br>Instar | Mean sqr effect | Mean sqr error | *F* (df 21, 84) | *p* |
| 1 | 0.0073 | 0.0084 | 0.8635 | 0.6359 |
| 2 | 0.0114 | 0.0174 | 0.6574 | 0.8618 |
| 3 | 0.0958 | 0.1488 | 0.6440 | 0.8734 |
| 4 | 0.2345 | 0.2238 | 1.0480 | 0.4188 |
| 5 | 0.2386 | 0.2263 | 1.0542 | 0.4121 |

increase with host density shows a declining response until at host densities greater than about 10 there is little increase with host density. The $R_0$ values for *B. thuringiensis*, conversely, are very low when host density is low but increase exponentially with increasing host density.

The values for the host threshold density, calculated using eq. 4, are 0.271 for PiGV and 4.963 for *B. thuringiensis*. This is what would be expected from the results of the population experiments: the observation that PiGV persisted in all experiments into which it was introduced, and *B. thuringiensis* became extinct in all but one experiment would lead to the prediction that PiGV should have a substantially lower host threshold density than *B. thuringiensis*.

The units in which the threshold density is expressed are problematical. In eqs 1 and 2, the host population is assumed to be randomly mixing so that all susceptible hosts are equally likely to encounter the infectious host or cadaver. This is unlikely to be the case in reality, and the threshold density as calculated here is therefore in units of susceptible hosts with which an infectious cadaver will come into contact. This makes it difficult to relate the values for the threshold density directly to the numbers of insects present in the population cages.

Table 3. Parameter estimates for calculating $R_0$ and the host threshold density. Superscripts indicate the origins of the data: 1 = Knell et al. 1996, 2 = Knell et al. 1998 and 3 = Knell 1996.

| Parameter | *B. thuringiensis* | PiGV |
|---|---|---|
| $\alpha$ | $0.0041^1$ | $0.007^2$ |
| $\beta'$ | $-0.008^1$ | $0.633^2$ |
| $\mu$ | $0.059^3$ | $0.117^3$ |
| $d$ | 0 | $0.203^2$ |

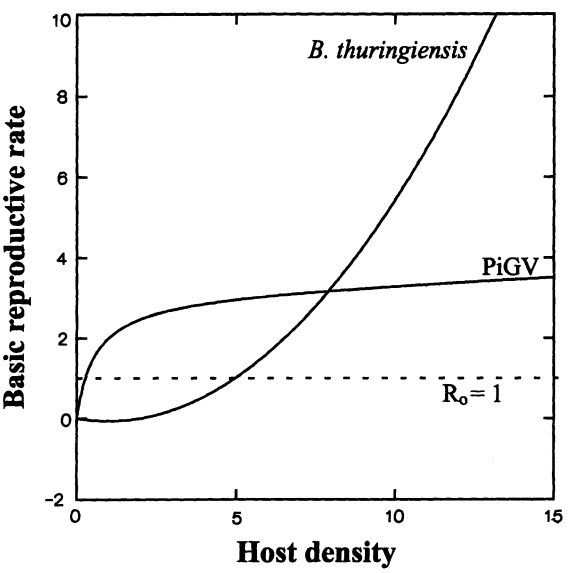


Fig. 3. Basic reproductive rate ($R_0$) calculated for *B. thuringiensis* and PiGV using eq. 2. The dotted line indicates an $R_0$ of 1, the host threshold density below which the pathogen will not persist.

## Discussion

### Host populations and pathogen interactions

There have been a number of other studies in which *P. interpunctella* has been maintained in long-term populations. In all of these studies which describe the long-term dynamics of the moth in sufficient detail to allow interpretation (Gurney et al. 1983, Jones 1986, Sait et al. 1994, Begon et al. 1995, 1996) the populations of adult moths underwent cycles of a period of approximately one generation: so-called "generation cycles".

A closer examination of the previous studies suggests that the nature of the food provided may determine whether or not the population cycles. The food provided in the experiments of Sait et al. (1994) was an extremely rich and high-quality food compared with that used here. Lawton (Gurney et al. 1983, Jones 1986) used two different food mixtures; one of which was roughly comparable to the low-quality food used in this study, while the second was of intermediate quality. The cycles found by Sait et al. were considerably clearer and less "noisy" than those found by Lawton, where power spectra comparing the two food regimes showed considerably clearer peaks for the higher quality food than the lower quality (Jones 1986). The amount, or possibly simply the depth of food available may also be of consequence. Begon et al. (1995) used identical food to that used in the present study, but at 2 different depths, 0.5 cm and 1 cm. The populations given 0.5 cm depth of food (as here) showed less regular cycling than those given 1 cm depth.

The mechanism bringing about cycles in *P. interpunctella* populations is thought to be density-dependent mortality of younger larvae and eggs, leading to the population becoming more or less synchronised in terms of age structure. This gives discrete "bursts" of population (Gurney et al. 1983, Jones 1986, Sait et al. 1994). Food quality is likely to be closely linked to the intrinsic rate of increase of a population and especially to individual fecundity, which can be important in determining whether a population shows generation cycles (Gurney and Nisbet 1985).

There may be effects of microclimate arising from the different depths of food which could explain the apparent influence of the quantity of food. Younger larvae are likely to be more vulnerable to such effects, which would increase the degree of asymmetry between younger and older larvae. Alternatively, the lower population sizes arising from a reduced quantity of food could lead to demographic stochasticity becoming important. This could cause the population dynamics to become increasingly unclear as chance events played a more important role.

There was no discernible effect of either pathogen on the behaviour of the host populations. The result for *B. thuringiensis* is thus similar to those described by Flanders and Hall (1965), White and Huffaker (1969a) and Benson (1972), all of whom found that similar bacterial pathogens had little effect on host dynamics in laboratory ecosystems. In contrast, Sait et al. (1994) found a significant decrease in the mean population size in the presence of the virus and also more subtle changes in the cycles undergone by the host population. The experiments of Sait et al. were conducted for roughly twice the period of the present experiments, and it may be that the virus would have become more important as a limiting factor were these experiments continued for a longer time, particularly given that the numbers of virus infected larvae found increased during the experiment, suggesting that the virus did not reach equilibrium with its host.

There is evidence of a competitive interaction between the two pathogens in the age-structure data. Proportionally more *B. thuringiensis* infected larvae were 5th instar and proportionally less were 2nd instar when PiGV is present. The virus mostly infected younger larvae, so this effect may be due to fewer early instar larvae being available for infection by *B. thuringiensis*, skewing the age distribution towards the later instar larvae. This interaction is not reflected in the overall numbers of larvae that were infected with each pathogen, however, and competition seems unimportant at the overall population level despite having clear effects when age-structure is incorporated into the analysis.

### Basic reproductive rates and transmission thresholds

The most obvious question raised by the population experiments described here is why the two pathogens used in this study exhibit such different long-term dynamics. The two pathogens are similar in many ways, particularly regarding their routes of transmission, yet one persists stably in populations of *P. interpunctella* and one does not. The differences in their respective host threshold densities offer an attractive explanation for this pattern: the host threshold density of PiGV (0.27) is far lower than that calculated for *B. thuringiensis* (4.96).

*B. thuringiensis* has been reported as occurring in sudden widespread epizootics in infested stored foodstuffs (Burges and Hurst 1977, Delucca et al. 1982, Meadows et al. 1990, Meadows 1993). This appears to be at odds with the patterns described here, but the changes in $R_0$ with host density may provide an explanation (Fig. 3). Once the density of hosts is greater than the threshold density for *B. thuringiensis* $R_0$ increases exponentially with host density. Thus if the density of hosts becomes much greater than the threshold density any individuals which become infected with *B. thuringiensis* will produce large numbers of new infect-

eds. This would lead to extensive epizootics in high density populations whereas low density populations would experience little or no mortality from the bacterium.

In contrast with *B. thuringiensis*, PiGV has frequently been shown to exist in relatively stable associations with its host, (this study, Sait et al. 1994, Begon et al. 1996), and seems to have little effect on the population dynamics of the host. The form of the relationship between $R_0$ and host density for PiGV is very different from that of *B. thuringiensis* (Fig. 3): $R_0$ for PiGV increases very rapidly at low densities, leading to a very low host threshold density, but the increase declines rapidly as host density increases, until above a host density of about 5 there is very little increase in $R_0$ with increases in host density. This would lead to PiGV being able to establish easily in low density host populations, but increases in host density would lead to less-than-proportional increases in transmission of the pathogen.

A biological explanation for the difference between the two pathogens may lie in the anti-feeding effect caused by *B. thuringiensis* (Krieg 1987). In these experiments both *B. thuringiensis* and PiGV were largely transmitted by cannibalism. While larvae will nibble *B. thuringiensis*-infected cadavers, they will not eat much of them. Transmission of the bacterium will be limited by this anti-feeding effect, as some larvae will not have consumed sufficient toxin to kill them before the anti-feeding effects begin. Virus-infected cadavers, on the other hand, were rapidly consumed, and it is likely that the high densities of virus particles found in freshly killed victims will be sufficient to infect most individuals that eat even a small amount of the cadaver. This appears to explain the difference between the two pathogens at low host densities.

The anti-feedant effect of *B. thuringiensis* can also provide an explanation for the differences between the $R_0$ values for the two pathogens at higher host densities. The rate of removal of infectious cadavers by cannibalism ($d$) places an upper limit on $R_0$ for PiGV: at high host densities the whole cadaver is consumed rapidly by a small number of susceptible hosts, and this number is the maximum value for $R_0$. *B. thuringiensis* on the other hand produces cadavers which susceptible hosts will only nibble, and this led to $d$ not being measurable in our experiments. No upper limit is placed on $R_0$ for *B. thuringiensis* therefore, leading to the exponential increase with host density. This must be unrealistic at the highest host densities, when even the nibbling of *B. thuringiensis* containing cadavers would result in the removal of a significant amount of the infectious material. Our measurement of the rate of removal was conducted at a high host density, however, and it is doubtful that natural populations ever reach the very high densities at which this would become important.

It is worth noting here that the age-structure effects that appear to be important in this system (Fig. 2) are not directly accounted for by this calculation of $R_0$. Despite this, and whatever the biological explanation for the difference between the two pathogens, the present results demonstrate, apparently for the first time, the potential power of $R_0$-against-density relationships as a way of explaining dynamic patterns in host-pathogen relationships.

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