Journal of Animal Ecology 1998, **67**, 592–599

Transmission of *Plodia interpunctella* granulosis virus does not conform to the mass action model

ROBERT. J. KNELL*, MICHAEL BEGON and DAVID J. THOMPSON

Population Biology Research Group, School of Biological Sciences, University of Liverpool, PO Box 147, Liverpool, L69 3BX, UK

Summary

1. Transmission of insect pathogens is traditionally described by a term which states that transmission is proportional to the densities of the susceptible hosts and the infectious units, multiplied by a constant, the transmission coefficient. Theoretical studies suggest that deviations from this can be important in host–pathogen population dynamics, but little is known of how commonly pathogen transmission conforms to the conventional model.

2. We describe a test of the traditional assumption for the Indian meal moth, *Plodia interpunctella* (Lepidoptera: Pyralidae) (Hübner) and its granulosis virus using a modification of the previous methods, which allows for unpredictable declines in the amount of infectious material present.

3. The estimated transmission coefficient increased with the density of susceptible hosts and showed a marked decline with density of infectious cadavers. This suggests that the usual assumption does not adequately describe transmission in this system.

4. The reasons for this deviation from the usual assumption are likely to be a combination of behavioural and physiological changes at high host density, and differential susceptibility to the pathogen leading to an effect analogous to pseudo-interference in parasitoids.

Key-words: density dependence, host–pathogen interactions, parasite–host models, transmission coefficient.

Journal of Animal Ecology (1998) **67,** 592–599

Introduction

The potential role of pathogens in the population dynamics of their hosts has increasingly been recognized. In attempts to understand this role various host–pathogen models have been analysed. A crucial part of all of these models is the term describing the way in which the pathogen is transmitted between hosts. All conventional invertebrate host–pathogen models make the assumption that the rate of pathogen transmission is equal to the product of the number of susceptible hosts, the number of infectious units (infectious hosts, infectious cadavers, free-living infectious spores or pathogen particles), and the transmission coefficient, a constant which represents the chance of transmission of the infection per contact between susceptible hosts and infectious units. This assumption has been a central part of the theoretical studies, for example, Anderson & May (1979a,b, 1981), Anderson (1982), Hochberg (1989), Bowers & Begon (1990), Hochberg & Waage (1991), Begon *et al.* (1992), Bowers, Begon & Hodgkinson (1993) and Dwyer (1994).

It is traditional to refer to this assumption about the way that transmission occurs as the 'mass action assumption', making reference to its similarity to mass action kinetics in chemical processes. De Jong, Diekmann & Hesterbeek (1995) and Bouma, De Jong & Kimman (1995) have, however, pointed out that there is some confusion regarding whether the simple transmission term described above refers to population densities or to absolute population numbers. They suggest that the traditional term, $transmission = \beta XY$ (β is the transmission coefficient, and X and Y are the sizes of the susceptible and infectious populations) should be referred to as 'pseudo mass-action', as it refers to the size of the population, and that a more suitable term to use when population densities are under consideration is $\beta XY/N$ (N refers to the total density of susceptible and infectious individuals) which they refer to as true mass-action.

* Current address: Department of Zoology, University of the Witwatersrand, Private Bag 3, Wits 2050, South Africa

The extent to which these describe the transmission of infectious disease is not known. The transmission coefficient is regarded as an exceptionally difficult parameter to measure (Anderson & May 1981) and experimental investigations of disease transmission dynamics are correspondingly rare. If transmission does deviate from these simple models, however, theoretical studies suggest that there can be important effects on the dynamics of the host–pathogen system. Hochberg (1991a), for example, used a transmission term with non-linearities in both the response to host density and the density of infected individuals, and found that this could lead to considerable changes in the dynamics and stability of the model depending on the degree of non-linearity. More recently, Briggs & Godfray (1995, 1996) have developed a suite of models of insect host–pathogen systems. The transmission term used incorporated a declining response to the density of infectious particles, which was highly stabilizing. In fact, the authors used the degree of non-linearity necessary to stabilize the system as an indicator of the overall stability of the model. A better understanding of how transmission changes with densities of susceptible hosts and infectious units is therefore of fundamental importance if we wish to understand the dynamics of real host–pathogen systems.

The simplest way to test if this assumption describes transmission in a particular system is to estimate the transmission coefficient at a variety of densities of susceptible hosts and infectious units in order to see whether the estimate remains constant. Dwyer (1991) presented the first attempt to do this with an insect pathogen, the nuclear polyhedrosis virus (NPV) of *Orgyia pseudotsugata*, the Douglas-fir tussock moth. Larval age was found to have a strong influence on transmission which obscured any potential influence of density. D'Amico *et al.* (1996) demonstrated that the transmission coefficient of the NPV of gypsy moth, *Lymantria dispar*, was reduced at higher densities of both susceptible larvae and pathogen particles. This was predicted by Dwyer & Elkinton (1993) as a reason for some epizootics of the virus behaving in a way not predicted by a simple model. Knell, Begon & Thompson (1996) measured the transmission coefficient of the bacterial pathogen *Bacillus thuringiensis* infecting larvae of the Indian meal moth, *Plodia interpunctella*, at a variety of densities of two larval instars and under different conditions of food availability. Estimates increased with the density of susceptible hosts in both 4th and 5th instar larvae, and decreased with the density of infectious cadavers in 5th instar larvae. Food availability had a pronounced effect. Goulson *et al.* (1995) measured transmission coefficients in the field, although not for purposes of questioning the normal transmission assumption. Two other studies are notable, although the transmission coefficient was not measured directly. First, Hochberg (1991b) found that the age of susceptible hosts and the number of initial infected hosts were related to risk of infection in broods of *Pieris rapae* infected with a granulosis virus (GV), but that the density of susceptible hosts had no effect. Secondly, Ebert (1995) found that transmission of a microsporidian parasite of *Daphnia magna*, *Pleistophora intestinalis*, did not decrease as rapidly as would be expected on the basis of the normal assumption with increases in the volume of experimental containers. This was attributed to swarming behaviour by the host increasing the effective density of hosts and pathogen.

A final study of relevance is that by Bouma *et al.* (1995), in which pigs were housed in conditions of equal density, but different population size and transmission of pseudo-rabies virus estimated by calculating a reproductive ratio for the virus. It was found that transmission did not differ between treatments, suggesting that the 'true' mass action model is appropriate here rather than 'pseudo mass action'.

The studies described above which measured the transmission coefficient all used the same technique. This was first suggested by Dwyer (1991), and relies on measurement of the proportion of a group of susceptible hosts which become infected over a defined time period during which the density of infectious particles (Dwyer 1991) or infectious cadavers (Knell *et al.* 1996) is known to either remain constant or, as in the case of the study of D'Amico *et al.* (1996), to decrease at a known rate. We describe here a test of the assumption for the granulosis virus of *P. interpunctella* (PiGV), using a modification of this method which allows transmission coefficients to be estimated in cases where the amount of infectious material present decreases at a rate which varies between treatments.

The infectious unit

Theoretical studies of insect pathogen population dynamics usually express the population of infectious units in terms of the numbers of free-living infectious stages present in the environment (Anderson & May 1981), for example, the occlusion bodies of baculoviruses or the spores of fungi or microsporidia. Empirical studies of NPVs have tended to express pathogen populations in this way (Dwyer 1991; Dwyer & Elkinton 1993; Goulson *et al.* 1995; D'Amico *et al.* 1996). Studies of other types of pathogens, however, have used the infectious cadaver as a unit: Grosholz (1992) concentrated upon the individual infected cadaver in his study of an Isopod Iridescent Virus, Thomas, Wood & Lomer (1995) used the individual grasshopper cadaver in their study of the fungus *Metarhizium flavoviride*: and Knell *et al.* (1996) considered the individual cadaver of *P. interpunctella* when infected with *B. thuringiensis*. In the *P. interpunctella*–PiGV system transmission appears to be largely by means of cannibalism of infectious cadavers rather than by release of large numbers of free-living infectious particles. Hence, the infectious unit in which

PiGV density is measured throughout this study is also the individual infectious cadaver.

Estimating the transmission coefficient

Dwyer (1991) used a reduced, within generation model of transmission such that:

$$\frac{dX}{dt} = \beta' XY, \qquad \text{eqn 1}$$

in which Y is the number of infectious units and X is the number of susceptible hosts. β' is used to represent the transmission coefficient here instead of the more usual β (used in the case of close contact transmission) or v (in the case of free-living infective stages) (Anderson & May 1981) because the route of transmission in this system is by cannibalism of infectious cadavers, so neither of the normal terms is appropriate. Integration of this equation within limits gives

$$\beta' = \frac{\ln\left(\frac{X_0}{X_t}\right)}{Yt} \qquad \text{eqn 2}$$

in which X_0 is the initial number of susceptible individuals, X_t the number of uninfected individuals remaining at the end of the experiment and t the time period for which susceptible hosts were exposed to infection. For a given density of infectious units Y, the transmission coefficient can therefore be estimated by performing a short-term experiment in which the number of new infections arising within a single cycle of infection is measured.

There are a number of conditions set by the assumptions behind this model. First, there should be no secondary transmission. In other words, all new infections should arise from the initial primary infected specimens introduced into an experiment. Secondly, mortality due to causes other than disease should be negligible during the experiment, and thirdly, the amount of infectious material present should remain constant. The first condition can be met by running the experiments for a shorter period than the time taken for a newly infected larva to become infectious (in the present study infected larvae only became infectious after death, which takes in excess of 2 weeks (Sait *et al.* 1994), whereas no experiment took more than 10 h), and the second can be tested during the course of the experiment by measuring non-disease mortality.

This model also assumes that transmission follows the 'pseudo mass action' model (de Jong *et al.* 1995), in other words that transmission is dependent on overall population size rather than density. We use an experimental design in which the population is confined by a small container, so that density is equal to population size, meaning that if transmission does occur according to the conventional assumptions the 'pseudo mass action' term should be appropriate.

The studies of Dwyer (1991) and Goulson *et al.* (1995) both assumed that there were a constant number of virus particles present during their experiments. This assumption may be justified if the number of virus particles removed by the hosts from the environment is small compared to the numbers present, and if the decay rate of the virus particles is slow compared to the time scale over which the experiment is conducted. D'Amico *et al.* (1996), however, reported that the half-life of *L. dispar* NPV particles in the environment was less than the period over which the transmission coefficient was estimated. Their transmission coefficient was therefore estimated using a version of equation 2 which took this into account. Knell *et al.* (1996) demonstrated that the infectivity of the infectious cadavers used to estimate the transmission coefficient with *B. thuringiensis* did not change during their experiments. This was attributed to the antifeedant effect of *B. thuringiensis*, which led to only small amounts of the infectious cadavers being cannibalized. The transmission dynamics of the two pathogens might be expected to be similar. However, the virus does not have the anti-feedant effect of *B. thuringiensis* (Angus 1956) and infectious cadavers may be eaten entirely in a relatively short period (Knell 1996). This can lead to the amount of infectious material declining during an experiment (see Results). The rate of decay and quite probably the shape of the relationship will depend upon the densities of hosts and infectious cadavers, and so will vary with experimental treatment. A measure of the transmission coefficient can be obtained for this system, however, by obtaining estimates (using the method outlined above) at a variety of time intervals, and then extrapolating back to time zero. This is the only time when the density of infectious cadavers is reliably known, as it is the density with which the experiment was started.

Experimental methods

Experimental larvae were produced by adding ≈ 200 eggs of *P. interpunctella* to 200 g of culture medium consisting of 10 parts wheat bran to 1 part brewer's yeast to 1 part glycerol. These were then kept at 25°C with a 16:8 light:dark cycle and at roughly 60% humidity. Age of larvae was determined from the width of the head capsule (Lindfield 1990).

Virus-infected cadavers were produced by allowing ≈ 50 3rd instar larvae to feed for 24 h on 1 g of culture medium mixed with 10 freshly dead infected 5th instar larvae homogenized in 1 ml of distilled water. Excess food was then added, and after 7 days infected larvae (distinguishable by their white colouration) were removed to individual cells of a clean 10 × 10-cm Petri dish divided into 25 2 × 2-cm compartments with excess food. Death generally occurred 12–16 days later in the 4th instar.

Healthy 4th instar larvae were placed in 50 mL

screw-top glass jars with 50 mg of food. The uninfected larvae were then kept for 36 h, to eliminate any effects from handling. Any larvae that died during this period were replaced from equivalent jars randomly included in the experiment for this purpose. Three experiments were carried out.

EXPERIMENT 1: THE AMOUNT OF INFECTIOUS MATERIAL

To investigate the extent to which the amount of infectious material declined during the course of an experiment, 20 fourth instar larvae were placed in each jar. After 36 h, two infectious 4th instar cadavers were added for 2, 4, 6, 8 or 10 h, following which time all the susceptible hosts were removed, placed individually in cells of 5 × 5 divided Petri dishes and excess food added. The experiment was replicated 10 times. This allowed an estimate of the transmission coefficient to be obtained by the method of Dwyer (1991). A decrease in this estimate with the length of time during which larvae were exposed to the infectious cadavers would indicate that the amount of infectious material present was changing.

EXPERIMENT 2: THE INFLUENCE OF HOST DENSITY ON THE TRANSMISSION COEFFICIENT

To investigate whether the density of susceptible hosts influenced the transmission coefficient, 5, 10, 15 or 20 4th instar larvae were placed in jars. Two infectious cadavers were added 36 h later. All susceptible hosts were removed 1, 2, 3 or 4 h later and placed in individual cells of 5 × 5 divided Petri dishes with excess food. This protocol was used because the results of experiment 1 indicated a rapid decline in the amount of infectious material (see Results). The transmission coefficient was estimated from extrapolating the estimates of β' against time, and using the y-intercept where time = 0. The experiment was replicated eight times.

The range of host densities used was chosen because it spanned a range from excess food being available to each larva to all the food being rapidly eaten. Higher densities of susceptible hosts would have led to considerable amounts of non-disease mortality from aggressive interactions and cannibalism between larvae, thereby violating the assumption that non-disease mortality is negligible as discussed above.

EXPERIMENT 3: THE INFLUENCE OF THE DENSITY OF INFECTIOUS CADAVERS ON THE TRANSMISSION COEFFICIENT

To investigate the influence of the density of infectious cadavers, 10 fourth instar larvae were placed in each jar, and 1, 2, 3 or 4 infectious 4th instar cadavers added. This range of densities of infectious cadavers was chosen because preliminary experiments suggested that higher densities would lead to all the susceptible hosts present becoming infected in some replicates, making it impossible to calculate a meaningful value for the transmission coefficient. All susceptible hosts were removed 1, 2, 3 or 4 h later and placed in individual cells of 5 × 5 divided Petri dishes with excess food. An estimate of the transmission coefficient was again obtained by back extrapolation. The experiment was replicated eight times.

All larvae were monitored for signs of PiGV infection 7 days later. Those that were infected were easily distinguished by their white colour. Any non-disease mortality was also recorded.

Results

EXPERIMENT 1: THE AMOUNT OF INFECTIOUS MATERIAL

Results are shown in Fig. 1. There was a significant effect of time on the estimate of the transmission coefficient (ANOVA, $P < 0{\cdot}001$). This pattern could arise from a decline in the amount of infectious material present or from strong heterogeneity in the susceptibility of the hosts to the pathogen. After about 6 h there was very little of the infectious cadavers remaining in the experimental containers, however, and substantial reductions in the amount of infectious material could be observed after 4 h and even after 2 h in many cases. This suggests that removal of infectious material is the more important cause of the reduction in the transmission coefficient. Non-disease mortality was 12·8%, but the results of the analysis were unchanged, even when those replicates with 10% or more mortality from factors other than PiGV were excluded.

EXPERIMENT 2: THE INFLUENCE OF HOST DENSITY ON THE TRANSMISSION COEFFICIENT

All of the treatments showed a significant linear relationship between mortality and time, as sum-

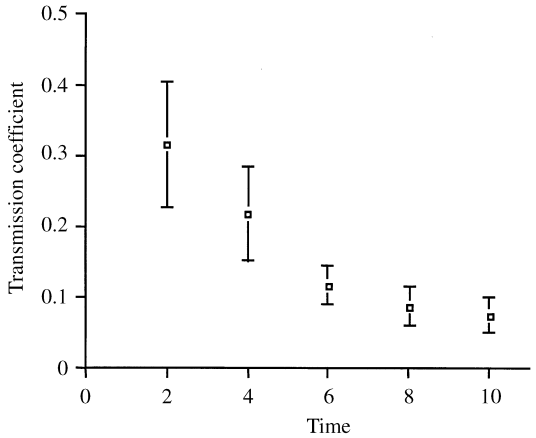

Fig. 1. Transmission coefficient of PiGV plotted against the length of experiment. Error bars are 95% confidence limits.

Table 1. Results of regressions of transmission coefficient (y) against time (x) for experiments to measure the transmission coefficient of PiGV for different densities of susceptible hosts

Host density	Equation	r^2	t	P
5	$y = 0{\cdot}278 - 0{\cdot}057x$	0·18	2·82	0·008
10	$y = 0{\cdot}324 - 0{\cdot}048x$	0·12	2·34	0·026
15	$y = 0{\cdot}399 - 0{\cdot}071x$	0·34	4·15	<0·001
20	$y = 0{\cdot}376 - 0{\cdot}075x$	0·34	4·15	<0·001

Degrees of freedom = 30 for each regression.

Table 2. Results of regressions of transmission coefficient (y) against time (x) for experiments to measure the transmission coefficient of PiGV for different densities of infectious cadavers

Cadaver density	Equation	r^2	t	P
1	$y = 0{\cdot}703 - 0{\cdot}155x$	0·49	5·40	<0·001
2	$y = 0{\cdot}325 - 0{\cdot}048x$	0·12	2·34	0·026
3	$y = 0{\cdot}265 - 0{\cdot}049x$	0·31	3·86	<0·001
4	$y = 0{\cdot}203 - 0{\cdot}037x$	0·14	2·51	0·017

Degrees of freedom = 28 in the case of one infectious cadaver and 30 in all other cases.

marized in Table 1. The relationship between time and the estimated transmission coefficient appeared non-linear in the case of 20 susceptible hosts, but when linearized by taking logs of the transmission coefficient, r^2 actually decreased and so the original regression was retained. Figure 2 plots the estimates of the transmission coefficient, as calculated from the regression intercepts, against the density of susceptible hosts. There was a significant difference between the estimates (ANCOVA, $P = 0{\cdot}034$, test for heterogeneity of slopes non-significant $P = 0{\cdot}579$). Non-disease mortality was 3·3% and the results of the analysis were unchanged when those data points with 10% or more non-disease mortality were removed.

The relationship between the transmission coefficient and susceptible host density may be somewhat non-linear. However, the errors associated with the data are large (a consequence of the extrapolation used to obtain the estimates), making it difficult to draw conclusions about the exact shape of the relationship. A linear regression of the transmission coefficient against susceptible host density weighted by $1/\sigma^2$ ($y = 0{\cdot}253 + 0{\cdot}007x$) was highly significant ($P < 0{\cdot}001$).

EXPERIMENT 3: THE INFLUENCE OF THE DENSITY OF INFECTIOUS CADAVERS ON THE TRANSMISSION COEFFICIENT

All regressions of the transmission coefficient against time were significant, as summarized in Table 2. Two extreme outlying data points were removed from the data set for one infectious cadaver before analysis. Figure 3 shows the estimates of the transmission coefficient as calculated from the regression intercepts plotted against the densities of infectious cadavers.

A test for heterogeneity of slopes was significant ($P < 0{\cdot}001$), but if the values for the transmission coefficient were $\log_{10}$ transformed then the differences in the slopes became non-significant ($P = 0{\cdot}459$). There was a very highly significant effect of density of infectious cadavers on the transmission coefficient when the $\log_{10}$ transformed transmission coefficients were analysed (ANCOVA, $P < 0{\cdot}001$). Non-disease mortality was 3·0% overall, and if all the data points

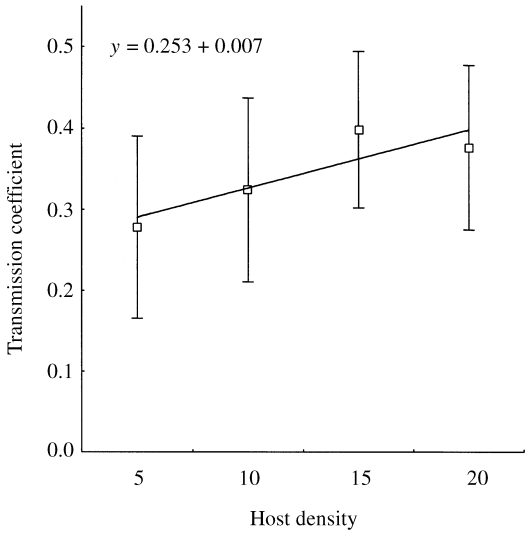

Fig. 2. Transmission coefficients plotted against densities of susceptible hosts for PiGV. Error bars are 95% confidence limits.

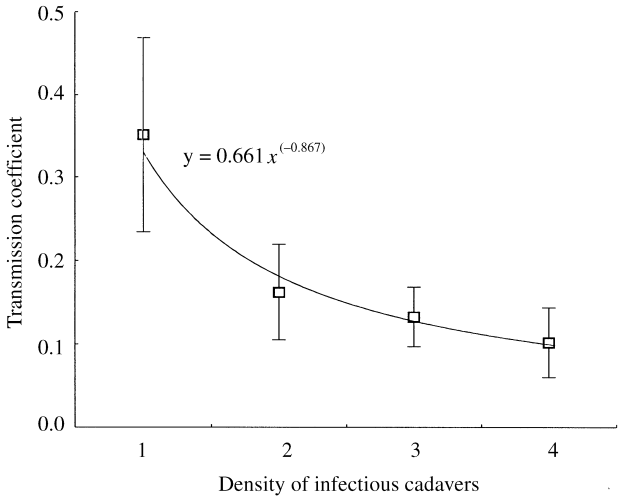

Fig. 3. Transmission coefficients for PiGV plotted against densities of infected cadavers with a power relationship fitted to the same data. Error bars are 95% confidence limits.

with 10% or more non-disease mortality were removed then the results of the analysis were unchanged.

The relationship appears to be non-linear. A non-linear regression weighted by $1/\sigma^2$ gave an r^2 value of 0·956 for a power relationship of the form $y = 0.661x^{(-0.867)}$, a substantially better fit than for a linear model ($r^2 = 0.701$).

Discussion

The transmission coefficient has been measured in the case of PiGV–*P. interpunctella* system, using a technique that assumes that transmission follows the equation $transmission = \beta' XY$. This is the standard transmission term in models of insect–pathogen systems. There was a significant increase with increases in the density of susceptible hosts and a significant decrease in response to increases in the density of infectious cadavers. This indicates that the usual assumption, namely that transmission is linearly related to the numbers of susceptible hosts and of infectious units multiplied by a transmission coefficient, is not adequate to describe this system.

A comparison may be drawn with the transmission dynamics of *B. thuringiensis* infecting the same host under similar conditions (Knell *et al.* 1996). In common with PiGV, changes were demonstrated in the transmission of *B. thuringiensis* with both the density of susceptible hosts and the density of infectious cadavers, although the latter was significant only in 5th instar larvae. While the overall trends of the relationships between transmission and density of infectious cadavers and susceptible hosts were the same for both pathogens, the forms of the relationships observed were different. All of the relationships shown with *B. thuringiensis* that were significant were adequately described by straight lines. The relationship between the transmission coefficient and the density of infectious cadavers, however, was distinctly non-linear for PiGV and, although the transmission coefficient increased approximately linearly with susceptible host density, the intercept for PiGV was 0·253 compared to −0·008 for B. thuringiensis (and the slopes were of the same order: 0·007 for PiGV vs. 0·004 for *B. thuringiensis*).

The transmission of a pathogen has two separate components. The first is the chance of a susceptible host coming into contact with an infectious unit of the pathogen. This depends on many factors, including the movement rate of both susceptible hosts and infectious units, the patterns of movement employed by both, and also the spatial structure of their populations. The standard bi-linear transmission term assumes both to be moving randomly and to be randomly distributed spatially. The second element of transmission is the probability of transmission of the pathogen once the susceptible host and infectious unit have come into contact. Once again this is complex, being affected by the nature of the pathogen and of the transmission route, and by the behavioural and physiological status of the susceptible host, and possibly of the infectious unit, particularly if the infected host itself is the infectious unit. It is usually assumed that the probability of transmission per contact is constant at all densities of both the host and the infectious units.

With regard to the changes with host density, the reason may lie in the amount of food available to the susceptible hosts. This was very important in determining the transmission coefficient of *B. thuringiensis* and is likely again to be the driving force. Less food will be available per larva at higher densities. This could influence the movement rates of susceptible hosts, increasing the probability of contacts with infectious units. Less food would also lead to susceptible hosts becoming hungry and, therefore, being more likely to cannibalize infectious cadavers when contact is made and the chance of transmission per contact could also be increased by increased susceptibility to disease arising from lack of food (Steinhaus 1958). The difference in the intercepts of transmission rate against the density of susceptible hosts for PiGV and *B. thuringiensis* may be due to the lack of the anti-feedant effect of *B. thuringiensis* (Angus 1956) in PiGV. This should lead to susceptible larvae being more likely to cannibalize PiGV infected cadavers at lower densities when food was not particularly scarce.

There was a marked decline in the estimate of the transmission coefficient with increasing density of infectious cadavers. This may arise from a process analogous to the phenomenon of pseudo-interference in predators and parasitoids (Free, Beddington & Lawton 1977), in which patches with a high prey density are depleted by density-dependent predator aggregation, leading to reduced intake rates which appear to result from mutual interference between the predators. In the case of pathogens, if susceptibility to infection is not uniform throughout the population of susceptible hosts then as the density of infectious units increases so the proportion of the population which is more susceptible will decrease as these individuals become infected. This will lead to the average susceptibility of the remaining population of healthy hosts available for infection decreasing (Knell *et al.* 1996). Each additional infective unit will therefore infect fewer susceptible hosts, simply due to the differential depletion of the population of susceptible hosts.

The differential susceptibility that could lead to such an effect could arise from either physiological differences between susceptible hosts in a randomly mixing population or from non-random mixing. In the latter case some susceptible individuals would be more likely to acquire infection through proximity to infectious cadavers and would also be likely to acquire

Table 3. Parameter estimates for transmission of PiGV obtained by fitting the two models shown to the transmission rates obtained experimentally

Model	Parameters	Author
$transmission = \beta(X^p Y^q)XY$	$\beta = 0{\cdot}489$ $p = 0{\cdot}119$ $q = 0{\cdot}856$	Hochberg (1991)
$transmission = \left[k \ln\left(1 + \frac{\beta Y}{k}\right)\right]X$	$\beta = 214$ $k = 0{\cdot}087$	Briggs & Godfray (1995, 1996)

multiple infections that would appear to be a single infection in these experiments.

This decline in the transmission coefficient with increasing density of infectious units was also described for *B. thuringiensis* infecting *P. interpunctella* (Knell *et al*. 1996), and for *L. dispar* NPV by D'Amico *et al*. (1996).

The question remains as to what transmission term would be suitable for modelling this host–pathogen system. There are two possible published expressions. First, Hochberg (1991a) used a transmission term of the form:

$$transmission = \beta(X^p Y^q)XY.$$

This allows a variety of non-linear responses depending on the values of p and q. A value of p of greater than 0 would give higher transmission with increasing density of susceptible hosts, and a value of q of less than 0 decreasing transmission with increasing density of infectious units.

Secondly, Briggs & Godfray (1995, 1996) used an expression for transmission of the form:

$$transmission = \left[k \ln\left(1 + \frac{\beta Y}{k}\right)\right]X,$$

incorporating a negative binomial term for transmission with the density of infectious units. Transmission is proportional to the density of susceptible hosts. If the parameter k is large transmission tends towards the standard bilinear model, but as k declines the risk of infection increases at a decreasing rate with the density of infectious units. This may be an acceptable simplification for modelling the transmission dynamics of PiGV, as the effect of host density on transmission is not as pronounced as the effect of the density of infectious cadavers.

Both of these models have been fitted to the data presented here. The transmission coefficients were transformed back to instantaneous rates of transmission by multiplying by host density and the density of infectious cadavers, and a non-linear least-squares regression then performed with the two models. Both of the models gave a very good fit to the data, with r^2 being 0·97 for the Hochberg model and 0·96 for the Briggs and Godfray model. The comparable figure was 0·49 for 'pseudo mass action', while it proved impossible even to make the regression converge for 'true' mass action. The parameter estimates thus obtained are shown in Table 3. In both cases they put the relevant model in a very stable region of parameter space. The Hochberg model was most stable when transmission efficiency increased with host density ($p < 0$) and decreased with the density of infectious units ($q < 0$), as is the case here. Briggs and Godfray found that models generally became more stable with decreasing values of k (i.e. increasing density dependence), and the value of 0·087 found here would be very stabilizing. PiGV is known to have very stable interactions with its host when maintained in laboratory population cages (Sait, Begon & Thompson 1994b; Begon, Sait & Thompson 1996). The present results thus combine with previous published models to suggest that the explanation for this stability may lie in the transmission dynamics of the system.

Acknowledgements

We thank Tom Heyes and Rob Swain for technical assistance, and Roger Bowers and Liam Lynch for helpful discussion. We are also grateful to three anonymous referees for helpful comments on a previous version. RJK was supported by an NERC postgraduate studentship.

References

Anderson, R.M. (1982) Theoretical basis for the use of pathogens as biological control agents of pest species. *Parasitology*, **84,** 3–33.

Anderson, R.M. & May, R.M. (1979a) Population biology of infectious diseases: Part I. *Nature*, **280,** 361–7.

Anderson, R.M. & May, R.M. (1979b) Population biology of infectious diseases: Part II. *Nature*, **280,** 455–61.

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

Angus, T.A. (1956) General characteristics of certain insect pathogens related to Bacillus cereus. *Canadian Journal of Microbiology*, **2,** 111–21.

Begon, M., Bowers, R.G., Kadianakis, N. & Hodgkinson, D.E. (1992) Disease and community structure: the import-

ance of host self-regulation in a host-host–pathogen model. *American Naturalist*, **139,** 1131–50.

Begon, M., Sait, S.M. & Thompson, D.J. (1996) Predator-prey cycles with period shifts between two and three species systems. *Nature*, **381,** 311–5.

Bouma, A., De Jong, M.C.M. & Kimman, T.G. (1995) Transmission of pseudorabies virus within pig populations is independent of the size of the population. *Preventative Veterinary Medicine*, **23,** 163–72.

Bowers, R.G. & Begon, M. (1990) A Host-Host-Pathogen Model with Free-Living Infective Stages, Applicable to Microbial Pest Control. *Journal of Theoretical Biology*, **148,** 305–29.

Bowers, R.G., Begon, M. & Hodgkinson, D.E. (1993) Host-Pathogen population cycles in forest insects? Lessons from simple models reconsidered. *Oikos*, **67,** 529–38.

Briggs, C.J. & Godfray, H.C.J. (1995) The dynamics of insect–pathogen interactions in stage-structured environments. *American Naturalist*, **145,** 855–87.

Briggs, C.J. & Godfray, H.C.J. (1996) The dynamics of insect–pathogen interactions in seasonal environments. Theoretical Population Biology, in press.

D'Amico, V., Elkinton, J.S., Dwyer, G. & Burand, J.P. (1996) Virus transmission in gypsy moths is not a simple mass action process. *Ecology*, **77,** 201–6.

De Jong, M.C.M., Diekmann, O. & Heesterbeek, H. (1995) How does transmission of infection depend on population size? In: D. Mollison (ed.), Epidemic models: their Structure and Relation to data. Cambridge University Press, Cambridge, UK, pp. 84–94.

Dwyer, G. (1991) The roles of density, stage and patchiness in the transmission of an insect virus. *Ecology*, **72,** 559–74.

Dwyer, G. (1994) Density-dependence and spatial structure in the dynamics of insect pathogens. *American Naturalist*, **143,** 533–62.

Dwyer, G. & Elkington, J.S. (1993) Using simple models to predict virus epizootics in gypsy moth populations. *Journal of Animal Ecology*, **62,** 1–11.

Ebert, D. (1995) The ecological interactions between a microsporidian parasite and its host Daphnia magna. *Journal of Animal Ecology*, **64,** 361–9.

Free, C.A., Beddington, J.R. & Lawton, J.H. (1977) On the inadequacy of simple models of mutual interference for parasitism and predation. *Journal of Animal Ecology*, **46,** 543–54.

Goulson, D., Hails, R.S., Williams, T., Hirst, M.L., Vasconcelos, S.D., Green, B.M., Carty, T.M. & Cory, J.S. (1995) Transmission dynamics of a virus in a stage-structured insect population. *Ecology*, **76,** 392–401.

Grosholz, E.D. (1992) Interactions of intraspecific, interspecific and apparent competition with host–pathogen population dynamics. *Ecology*, **73,** 507–514.

Hochberg, M.E. (1989) The potential role of pathogens in pest control. *Nature*, **337,** 262–5.

Hochberg, M.E. (1991a) Non-linear transmission rates and the dynamics of infectious disease. *Journal of Theoretical Biology*, **153,** 301–21.

Hochberg, M.E. (1991b) Extra–host interactions between a braconid endoparasitoid, Apanteles glomeratus, and a baculovirus for larvae of Pieris brassicae. *Journal of Animal Ecology*, **60,** 65–77.

Hochberg, M.E. & Waage, J.K. (1991) A model for the biological control of Oryctes rhinoceros (Coleoptera: Scarabaeidae) by means of pathogens. *Journal of Applied Ecology*, **28,** 514–31.

Knell, R.J. (1996) The population ecology of two insect pathogens. PhD thesis, University of Liverpool.

Knell, R.J., Begon, M. & Thompson, D.J. (1996) Transmission dynamics of Bacillus thuringiensis infecting Plodia interpunctella: a test of the mass action assumption with an insect pathogen. *Proceedings of the Royal Society of London, B*, **263,** 75–81.

Lindfield, S. (1990) Microevolution in an insect–virus interaction. PhD thesis, University of Liverpool.

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,** 107–10.

Sait, S.M., Begon, M. & Thompson, D.J. (1994b) Long term population dynamics of the Indian meal moth Plodia interpunctella and its granulosis virus. *Journal of Animal Ecology*, **63,** 861–70.

Steinhaus, E.A. (1958) Stress as a factor in insect disease. Proceedings of the 10th International Congress of Entomology, Montreal, 1956. 725–730.

Thomas, M.B., Wood, S.N. & Lomer, C.J. (1995) Biological control of locusts and grasshoppers using a fungal pathogen: the importance of secondary cycling. *Proceedings of the Royal Society of London, B*, **259,** 265–70.

Received 2 October 1996; revision received 20 October 1997