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Effects of phenotypic plasticity on pathogen transmission in the field in a Lepidoptera-NPV system

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Abstract In models of insect–pathogen interactions, the transmission parameter (ν) is the term that describes the efficiency with which pathogens are transmitted between hosts. There are two components to the transmission parameter, namely the rate at which the host encounters pathogens (contact rate) and the rate at which contact between host and pathogen results in infection (host susceptibility). Here it is shown that in larvae of *Spodoptera exempta* (Lepidoptera: Noctuidae), in which rearing density triggers the expression of one of two alternative phenotypes, the high-density morph is associated with an increase in larval activity. This response is likely to result in an increase in the contact rate between hosts and pathogens. Rearing density is also known to affect susceptibility of *S. exempta* to pathogens, with the high-density morph showing increased resistance to a baculovirus. In order to determine whether density-dependent differences observed in the laboratory might affect transmission in the wild, a field trial was carried out to estimate the transmission parameter for *S. exempta* and its nuclear polyhedrosis virus (NPV). The transmission parameter was found to be significantly higher among larvae reared in isolation than among those reared in crowds. Models of insect–pathogen interactions, in which the transmission parameter is assumed to be constant, will therefore not fully describe the *S. exempta*-NPV system. The finding that crowding can influence transmission in this way has major implications for both the long-term population dynamics and the invasion dynamics of insect–pathogen systems.

Key words Transmission · Mass action assumption · Phase polyphenism · *Spodoptera exempta* · Baculoviruses

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Introduction

Pathogens can have a considerable impact on the population dynamics of their insect hosts, either directly or through sub-lethal infections (Begon et al. 1996; Rothman and Myers 1996). The interactions between insects and their pathogens have been described by mathematical models developed from those used to model the spread of human disease (Anderson and May 1979, 1981). In these early models, the rate of transmission of pathogens through insect populations depends on the density of both hosts and pathogens. In its simplest form, horizontal transmission is described as the *number of new infections per unit time* = νSP, where S is the density of susceptible hosts, P is the density of infectious pathogens and ν is the transmission parameter which describes the rate of acquisition of new infections. The transmission parameter embodies two components, the contact rate between susceptible hosts and infectious pathogens, and the probability of such a contact resulting in the host becoming infected (i.e. host susceptibility). Models of insect–pathogen interactions have often assumed that ν is constant, so the rate of acquisition of new infections will increase linearly with the density of both hosts and pathogens (e.g. Anderson and May 1981; Dwyer 1991). This is termed the mass action assumption, and implies that the efficiency of transmission does not vary with pathogen or host density.

However a number of studies have shown that transmission does not increase as a simple linear function of host and pathogen density. In the gypsy moth *Lymantria dispar* and its nuclear polyhedrosis virus (NPV) the transmission parameter has been found to decline with increasing host and pathogen density (D'Amico et al. 1996). The transmission parameter of both *Bacillus thuringiensis* (Bt) and a granulosis virus (GV) through populations of the stored-product pest *Plodia interpunctella* increases with the density of susceptible hosts and decreases with the density of infectious cadavers (Knell et al. 1996, 1998), and in the cabbage moth *Mamestra brassicae* and its NPV, the transmission parameter was found to increase with host density and decrease with pathogen density (Vasconcelos 1996).

In all four of these studies, the transmission parameter was found to decrease with increasing pathogen density. This is to be expected as a result of the law of diminishing returns; the risk of a host becoming infected cannot go on increasing indefinitely with pathogen density (Hochberg 1991). This will be particularly true for pathogens such as baculoviruses which are released into the environment following the death of an infected host, and so tend to be clumped around the site where the host died. An encounter with a single clump of pathogens is likely to be sufficient to cause a susceptible host to become infected; increasing the size of the clump (i.e. the yield of that cadaver) would not be expected to lead to a corresponding increase in new infections. Behavioural changes may also be responsible for causing ν to change with host density. The transmission of Bt and GV through *P. interpunctella* populations occurs mainly through cannibalism of infectious cadavers, so an increase in the rate of cannibalism at high host densities would lead to an increase in the rate of contact between host and pathogen (Knell et al. 1996, 1998). Density-dependent increases in host activity may also account for the rise in the transmission parameter with host density observed in *M. brassicae* and its NPV (S.D. Vasconcelos, unpublished data). The decrease in the transmission parameter at high host densities seen in the *L. dispar*-NPV system has proved more difficult to account for, although behavioural changes have been suggested as a possible explanation since larvae in low-density populations are more likely to visit daytime resting sites where they may come into contact with infected conspecifics (Dwyer and Elkinton 1993). Therefore there are a number of mechanisms that may operate to introduce non-linearities into the transmission process.

In addition to differences caused by host behaviour, variation in host susceptibility can also affect the transmission parameter. It is often assumed that hosts in high-density populations will be 'stressed' and therefore more susceptible to infection (Steinhaus 1958). However, among the Lepidoptera there is growing evidence that the opposite may be true, and that individuals in high-density populations may actually develop increased resistance to disease (Kunimi and Yamada 1990; Goulson and Cory 1995; Wilson and Reeson 1998; Reeson et al. 1998). This greater resistance at high population densities could potentially lead to a decrease in the efficiency of pathogen transmission as host population densities increase. This may provide another possible explanation for the higher than expected transmission parameters among low-density *L. dispar* populations.

The aim of this study was to determine how density-dependent changes in the host population influence transmission. The system used was the African armyworm *Spodoptera exempta* (Lepidoptera: Noctuidae) and its NPV. This species is a major migratory pest over wide areas of East and central Africa, and causes considerable economic losses during its outbreak season. Nuclear polyhedrosis virus is found in wild populations of *S. exempta*, often causing high levels of mortality (Odindo 1983). *S. exempta* larvae show a marked response to population density, with individuals developing at low density showing green/brown cryptic coloration, while larvae reared in crowds are heavily melanised (Gunn 1998). This is known as density-dependent phase polyphenism. The high-density form is also associated with shorter larval duration and increased migratory ability in the adults (Simmonds and Blaney 1986; Gunn and Gatehouse 1987; Woodrow et al. 1987). The susceptibility of *S. exempta* to its NPV varies with rearing density, with the melanised, high-density form having an LD_{50} ten times higher than that of the typical solitary form (Reeson et al. 1998). There is also evidence that rearing density can affect the behaviour of some species of phase polyphenic Lepidoptera, with crowd-reared larvae showing increased activity levels (Iwao 1963; Tojo 1991). These density-dependent changes in host populations have potentially opposing effects on the transmission of pathogens. Greater activity among crowded larvae is likely to increase their contact rate with a pathogen and so increase the efficiency of transmission; however the increased levels of resistance seen in crowded *S. exempta* larvae mean that such an encounter would be less likely to result in the host becoming infected.

The first part of this study set out to determine the effect of rearing density on locomotory behaviour in *S. exempta* using a computer-automated video tracking system, which allows accurate quantitative analysis of locomotion in a laboratory setting (Baatrup and Bayley 1993; Sorensen et al. 1997). A field trial was then carried out in which the transmission parameter of an NPV among populations of *S. exempta* larvae reared in isolated and crowded conditions was measured, so combining any density-dependent changes in behaviour with the known differences in susceptibility. The effects of both rearing density and local density in the field on the transmission parameter were examined.

Materials and methods

Larval behaviour

S. exempta larvae from a laboratory stock were reared at densities of either one (solitary) or six (crowded) per 12 ml plastic pot from within 24 h of hatching. The crowded treatment represented the

maximum number of larvae that could reasonably fit into the pots by the time they reached the fourth instar, and has been shown in previous studies to induce the high-density morph observed in the field under outbreak conditions (Reeson et al.. 1998). During development they were maintained at 27°C, 12:12 L:D and fed on freshly cut wheat seedlings (*Triticum aestivum*, Axona). Approximately 15 h prior to each experiment larvae were placed in isolation in fresh 12 ml pots and deprived of food. Larvae were weighed at the start of each experiment; each larva was used once only. Locomotory behaviour was measured by placing one early fifth instar larva into each of six 20 cm diameter circular arenas. Prior to each trial the bases of the arenas were moistened with distilled water to prevent the larvae desiccating; fresh plaster of Paris bases were prepared for each replicate.

Images of the arenas taken by video camera were passed via a frame grabber into a personal computer. GIPSTRA software (Image House, Copenhagen) was trained to distinguish the caterpillar from the background of the arena at the start of each trial on the basis of different light reflectance. This was done by assigning blue, green and red search criteria to each arena. Once per second a centroid was calculated from the pixels which matched the search criteria, and this was taken as the position of the larva. The trials were run over a 2-h period, and this resulted in a series of time and (x,y) co-ordinates for each larva. The data were analysed using MOTIO software (Institute of Biological Sciences, University of Aarhus, Denmark), which calculates, among other locomotory parameters, the total distance moved by each larva, the length of time for which it was active and the average speed of movement.

Virus transmission in the field

The field trial was carried out inside mesh cages with removable lids enclosing an area of 1 m^2, at a field site in Oxfordshire, UK. The trial was carried out in late summer (September 1997); the weather conditions at this time of year in Britain would not be atypical of the wet season in East Africa, when *S. exempta* outbreaks occur. Within each plot there were nine maize plants (*Zea mays* L. var Banker), each approximately 40 cm in height, arranged in a 3×3 grid. NPV was introduced to the plots by releasing infected larvae shortly before they died. To this end a group of 1200 *S. exempta* larvae were reared at 27°C, 12:12 L:D in 1000-ml boxes containing artificial diet (a modified form of Hoffman's tobacco hornworm diet as detailed by Hunter et al. 1984). Early in the third instar these larvae were fed a lethal dose (ca.3×10^7PIBs) of *S. exempta* NPV on diet plugs and then transferred back to tubs containing artificial diet. Once some individuals started showing signs of disease the larvae were taken to the field site and released into the plots at a density of two larvae per plant. The larvae had time to establish themselves on the plant and to die in a 'natural' position. After death the cadavers would lyse, releasing virus over the plant. This method ensured that virus would be distributed patchily, as in the natural environment. In order to judge when the infected larvae had died, 30 were kept in individual pots containing artificial diet which were placed outdoors and monitored daily until death. These 30 larvae were also used to estimate the yield of virus. Shortly after death each cadaver was transferred to an individual Eppendorf tube, homogenised with 1 ml of water, and the virus was then counted using a haemocytometer.

Once all the infected larvae were believed to have died from NPV the uninfected test larvae were introduced into the plots. These test larvae had been reared in the laboratory in either solitary or crowded conditions as described above. All the larvae had hatched on the same day and were carefully staged to minimise any variation in resistance due to differing development rates. Larvae were introduced into the field as fourth instars at either 1, 3 or 9 larvae per plant. The experiment was carried out using a factorial design, with rearing density and the number of larvae per plant as factors. There were two timepoints; three replicates of each treatment combination were sampled 3 days after the introduction of the uninfected larva, with a further four replicates sampled 2 days later. The number of replicates was limited by the availability of larvae, so the design was non-orthogonal. The plots were destructively sampled, and the larvae collected and placed individually in pots containing artificial diet. In addition there were four control plots (two with crowd-reared larvae and two with solitary-reared larvae) into which no infected larvae had been placed. Healthy larvae were introduced at a density of four per plant, and one of each treatment was destructively sampled at each timepoint. Once the larvae had been collected they were reared in the laboratory at 23°C until death or pupation. The transmission parameter (ν) was calculated using a protocol developed by Dwyer (1991) which is derived from the basic Anderson and May (1981) model.

$$\nu = \frac{-1}{P_0 t}\ln\left[1-\left(\frac{I_t}{S_0}\right)\right] \quad (1)$$

where: P_0 = initial pathogen density; t = number of days of exposure to inoculum; I_t = density of infected hosts at time t; S_0 = density of susceptible hosts at time 0

This assumes that the change in the density of infectious pathogens over the course of the experiment was negligible, all infections were lethal and that non-viral deaths were negligible compared to viral deaths.

Nine additional plots were set up to test the assumption that virus degradation over the course of the trial was negligible. This was done by releasing larvae of equivalent age into plots at various intervals after the introduction of infected larvae. Again there were nine maize plants per plot and two infected larvae on each plant. Uninfected third instar larvae that had been reared on artificial diet in the laboratory were introduced into these plots 24 h prior to the virus degradation sampling points. Samples were taken at 1, 4 and 6 days after the introduction of uninfected larvae into the plots in the main experiment. Three replicate degradation plots were sampled at each of these timepoints. Larvae were collected, placed individually in pots with artificial diet and reared in the laboratory at 23°C until death or pupation.

Results

Locomotory behaviour

Data were analysed using the Generalised Linear Interactive Modelling program (GLIM) (McCullagh and Nelder 1989). The total distance moved by each larva in the video tracking trials, the length of time for which it was active and the average speed of movement were $\log_e$ transformed and analysed using ANOVA; any effects of variation in larval weight were accounted for by including it in the model as a covariate. *S. exempta* larvae reared in crowded conditions moved significantly further than those reared in isolation (Fig. 1), covering an average of 8.8 m in a 2-h period, compared to 3.3 m for solitary-reared larvae ($F_{1,30}$=7.13; P=0.012). Individuals from crowded populations were active for an average of 69% of the time over which they were monitored, compared to 26% for the solitaries ($F_{1,30}$=16.2; P<0.001). There were no significant differences in the average speed of movement, with both crowd- and solitary-reared larvae covering approximately 3.1 mm^{-1} during periods of activity ($F_{1,37}$=2.1; $P>0.05$).

Virus transmission in the field

Mortality in the virus degradation plots was analysed in GLIM assuming binomial errors, an assumption which was

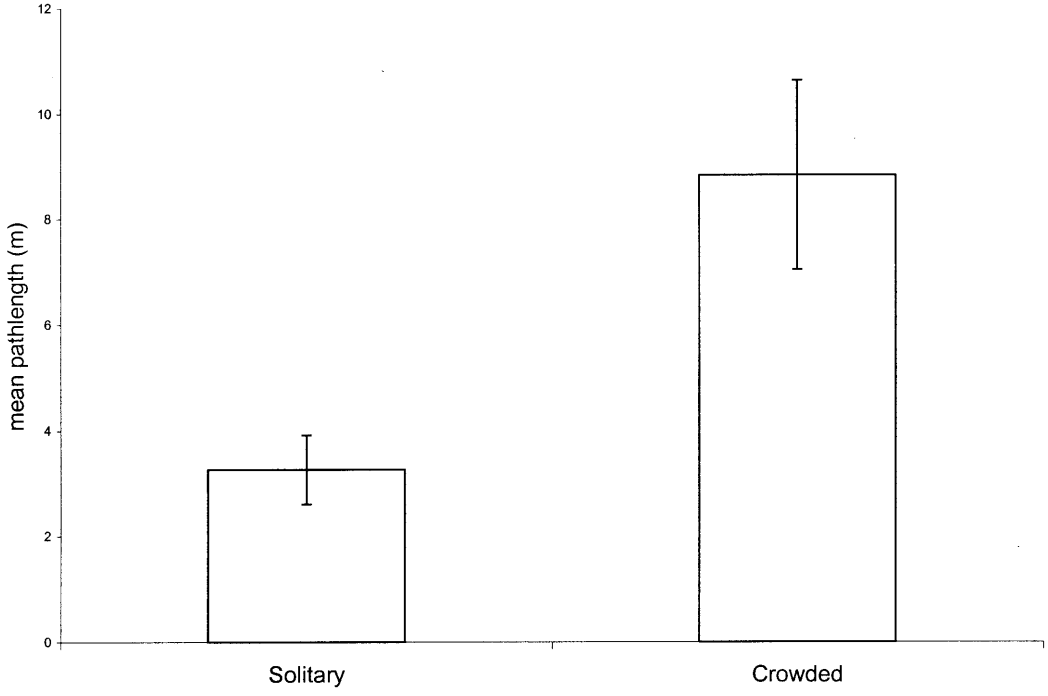

Fig. 1 Mean distances moved (± SE) by fifth instar *Spodoptera exempta* larvae over a 2-h period in the video tracking experiment. Larvae reared in crowded conditions up until the start of the experiment moved significantly further than those reared in isolation ($F_{1,30}$=7.13; P=0.012)

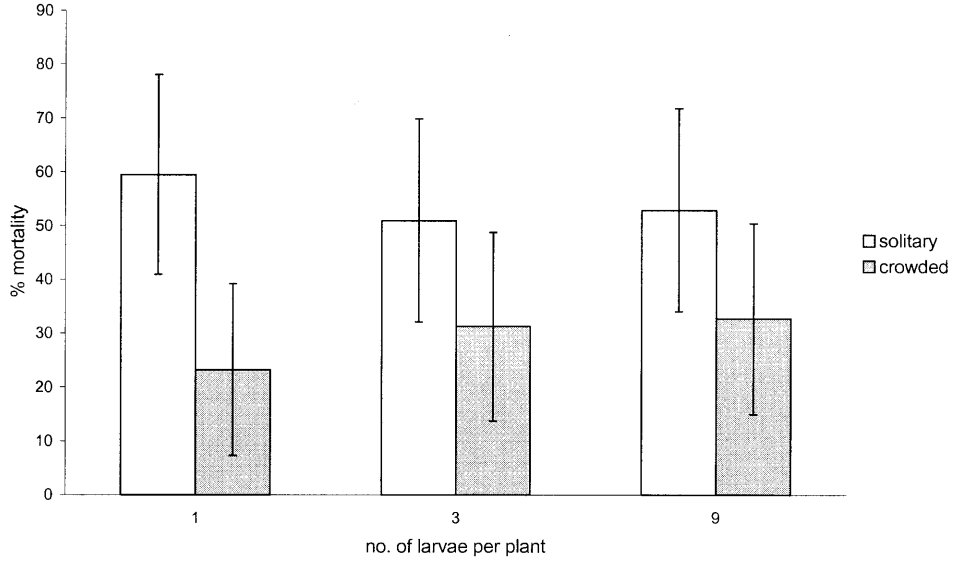

Fig. 2 Overall percentage mortality by rearing density and experimental density (± SE from the binomial distribution). Mortality was significantly higher among solitary-reared larvae ($F_{1,41}$=21.9; P<0.001). The number of larvae per plant had no significance ($F_{1,41}$=0.43; P>0.05)

substantiated during the analysis (by inspection of residuals and the scale parameter). The length of time between the introduction of virus into the plots and the release of uninfected larvae proved to have no significant effect on viral mortality (χ^2_1=2.63; P>0.05). This upholds the assumption that virus levels did not vary over the course of the trial, which is implicit in the equation used to calculate the transmission parameter. There were no viral deaths among the larvae recovered from the control plots.

In the main part of the field trial, virus-induced mortality was found to vary significantly with the density at which the larvae had been reared, with 52% of the solitary-reared larvae dying from NPV compared to 32% of the crowd-reared individuals ($F_{1,41}$=21.9; P<0.001) (Fig. 2). The number of larvae released onto the plants had no significant effect; average mortality was 36%, 41% and 44% for 1, 3 and 9 larvae per plant respectively ($F_{1,41}$=0.43; P>0.05) (Fig. 2). These mortality data were used to calculate transmission parameters for each plot according to Eq. 1; the data were then $\log_e$ transformed and analysed in GLIM using ANOVA. The number of larvae per plant was included as a continuous variable, while rearing density

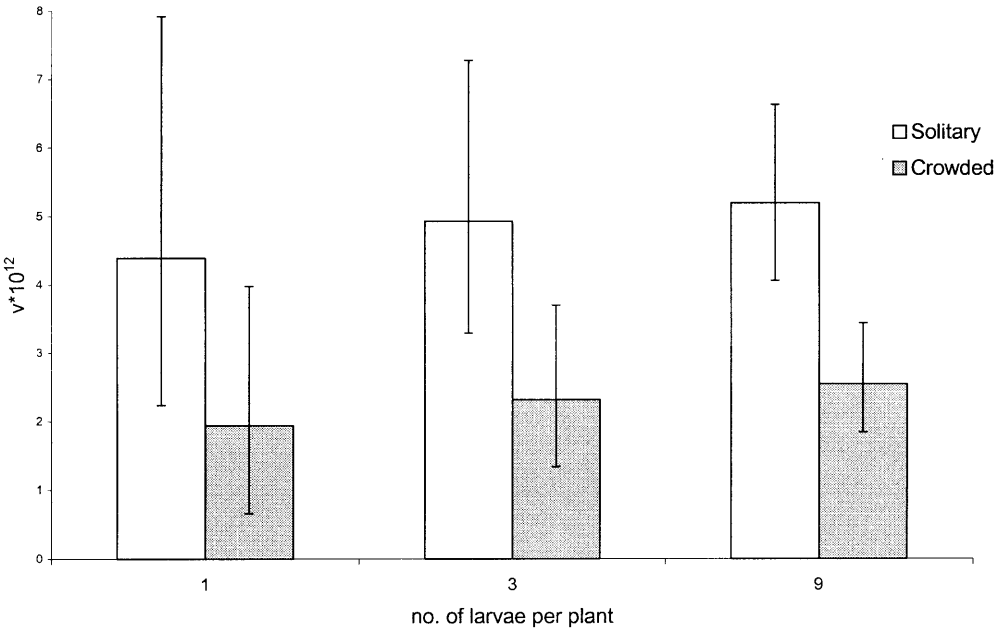

Fig. 3 Transmission parameter for the second timepoint, showing bootstrapped means and 95% confidence intervals obtained from a standard random sampling with replacement procedure, repeated 9000 times. The transmission parameter was significantly higher among larvae reared in isolation than among those reared in crowds ($F_{1,41}$=19.5; P<0.001). The number of larvae per plant had no effect on transmission ($F_{1,41}$=1.84; P>0.05)

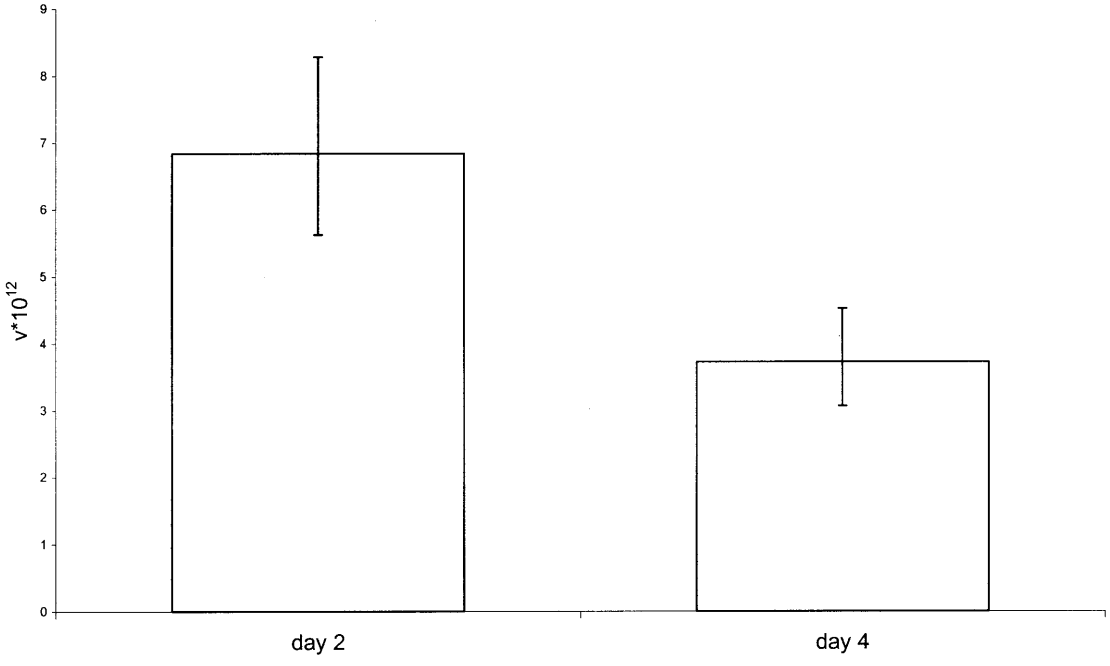

Fig. 4 Transmission parameter by timepoint, showing bootstrapped means and 95% confidence intervals obtained from a standard random sampling with replacement procedure, repeated 9000 times. The transmission parameter was significantly higher at the first timepoint ($F_{1,41}$=13.3; P<0.001)

and timepoint were considered as fixed variables. The model was weighted for the number of larvae recovered from each plot. The usual model checking procedures were employed for each analysis. The transmission parameter was found to be significantly higher in the plots containing larvae reared in solitary conditions, at 6.65×10^{-12}, compared to 3.38×10^{-12} for those containing crowd-reared larvae ($F_{1,41}$=19.5; P<0.001) (Fig. 3). The number of larvae per plant had no significant effect on the transmission parameter ($F_{1,41}$=1.84; P>0.05) (Fig. 3). There was a significant difference between the two timepoints, the transmission parameter being higher in the plots sampled after 3 days than in those sampled 5 days after the release of the uninfected larvae ($F_{1,41}$=13.3; P<0.001) (Fig. 4). All non-additive interactions between the variables were non-significant.

Discussion

The transmission process is central to all epidemiological models, so it is important to have a full understanding of the effects of host population density on the transmission parameter. The results of this study indicate that the population density of the host can affect both elements of the transmission parameter, namely the contact rate between host and pathogen and host susceptibility. In the first experiment population density did indeed affect larval behaviour in *S. exempta*, with larvae reared in crowded conditions moving significantly further than those reared in isolation. If these differences were translated into movement in the field, then larvae from high-density populations would be expected to cover greater distances. However, since larvae were tested in arenas from which food was absent, it is possible that individuals from crowded populations are more active in search of food. If food was present, the differences in behaviour may become less marked. With the video tracking system it was not possible to carry out experiments with food in the arenas as the software would be unlikely to distinguish the larvae from the food. The direct applicability of these results to a field situation is uncertain, but in the absence of any comparative data on activity levels from the field, it does provide some evidence of density-dependent differences in locomotion. In other species of Lepidoptera, increased dispersal activity has been observed among crowded larvae even in the presence of food (Poirier and Borden 1992; Rhainds et al. 1997). High levels of larval activity have been observed at *S. exempta* outbreak sites in the wild even when high quality food was apparently plentiful (K. Wilson, unpublished data), although little is known about activity in solitary larvae. Therefore it may not be unreasonable to believe that crowded *S. exempta* will be more active in the field as well as in the laboratory.

One consequence of increased locomotory activity will be a rise in the contact rate between crowded larvae and their pathogens, especially those pathogens that have clumped distributions (unless that movement results in dispersal out of virus-infected areas). Baculoviruses are released into the environment following the death of an infected host, and so tend to be clumped around the site where a host died. The importance of larval mobility and pathogen distribution has been demonstrated by Dwyer (1991) using *Orgyia pseudotsugata* and its NPV. Among third instar larvae Dwyer showed that the transmission parameter is lower when pathogens are clumped than when they are uniformly distributed. However, these differences were not apparent among late instar larvae, suggesting that the increased mobility of older larvae reduces the effects of clumping (Dwyer 1991). All other things being equal, if the increase in locomotory activity seen among crowd-reared *S. exempta* larvae in the laboratory translates to greater movement in the field, then this would be expected to lead to a greater efficiency of pathogen transmission through high-density populations.

However, all other things are not equal. In addition to these behavioural differences, crowd-reared *S. exempta* larvae are known to have increased resistance to pathogens (Reeson et al. 1998). They also have shorter larval durations (Simmonds and Blaney 1986) which might make them less susceptible to becoming infected in the field (although differences in development rate are not normally seen until the final instar). The results of this field trial show that any density-dependent behavioural differences are not sufficient to counter differences in susceptibility. The transmission parameter was significantly higher among solitary-reared larvae than among those reared in crowds. The mass action assumption, which states that transmission increases linearly with the densities of susceptible hosts and infectious pathogens, is not upheld for *S. exempta* and its NPV. Local density during the experiment (i.e. the number of larvae per plant) had no effect on transmission. Other studies on the transmission of lepidopteran pathogens have found that the transmission parameter increases with host density (Knell et al. 1996, 1998; S.D. Vasconcelos, unpublished data). However these studies were not designed to measure the effects of rearing density; instead they were looking at how immediate host density affects the transmission process through differences in contact rates between hosts and pathogens. Population density was only varied for a relatively short period during the trials, so even if these species are capable of developing increased resistance in response to crowding, it is unlikely that these experiments will have given them the chance to do so. In the real world immediate density is likely to reflect rearing density, so their effects will be difficult to distinguish. However experimental studies that examine only one type of crowding will not necessarily be applicable to the field. This study distinguishes between short-term (experimental density) and longer-term (rearing density) effects, indicating that the longer-term effects are more important (though, with secondary cycling of pathogen, experimental density would become more important over time, due to density-dependent pathogen transmission). As density-dependent increases in resistance may be widespread among the Lepidoptera (Wilson and Reeson 1998), it will be necessary to consider the effects of rearing density as well as experimental density in studies of pathogen transmission.

Non-linear transmission has been incorporated into models of insect–pathogen interactions in a variety of ways. Hochberg (1991) did this by making the transmission parameter a function of host and pathogen density. According to this model, stability is most likely if the transmission efficiency increases with host density; in such circumstances, hosts in low-density populations would effectively have a refuge from the pathogen. Where the transmission parameter decreases with host density, as is observed in the *S. exempta*-NPV system, regulation of the host by the pathogen would be less likely, and oscillatory dynamics may occur (Hochberg 1991). Briggs and Godfray (1995) found that where the transmission efficiency decreases with pathogen density

the system is stabilised. In such a system the density-dependent decrease in pathogen transmission would give the host a refuge at high pathogen densities. Dwyer et al. (1997) described the transmission parameter ν with a gamma distribution to incorporate heterogeneity in host susceptibility. This leads to a non-linear relationship between pathogen density and transmission. White and Wilson (1999) found that the inclusion of a resistant class of hosts tends to stabilise otherwise unstable interactions, and that density-dependent resistance is also stabilising, so long as the density-dependence is sufficiently small. Otherwise, the interaction is destabilised and bistable dynamics may result, in which the outcome (either extinction of the pathogen or unbounded growth of the resistant class of hosts) is dictated by the initial conditions of the model system.

The transmission parameter also varied between the two timepoints sampled in this field trial, being significantly higher at the first timepoint. There are a number of possible explanations for this. The equation used to calculate the transmission parameter assumes that virus degradation over the course of the trial is negligible. If the virus became less infective between the initial release and the second timepoint then the transmission parameter would decline. However, no significant decline in pathogen levels between the timepoints was found. Similar studies have also found virus degradation during short field trials to be negligible when the virus is released from infectious cadavers (Dwyer and Elkinton 1993; Goulson et al. 1995). Loss of pathogens from the environment due to consumption by hosts is also assumed to be negligible. This assumption was not tested directly, and since the fourth instar larvae used in this experiment are capable of consuming large amounts of foliage it is possible that much of the available pathogen was consumed early on in the experiment. If this was the case then the timepoint effect would be expected to be greater in the treatments with nine larvae per plant than in those with one larva per plant; however there was no significant interaction between timepoint and the number of larvae per plant. Increases in resistance with age might also have rendered larvae less susceptible to infection over the course of the trial. Another possibility is heterogeneities in susceptibility among the larvae used in each treatment. Larvae that are susceptible may pick up the virus early on, while those that do not become infected at this stage are then less likely to become infected subsequently. In a similar field trial using *M. brassicae* and *Trichoplusia ni* larvae, it has been shown that much infection occurs within the first 24 h, so later timepoints tend to underestimate the transmission parameter (Hails et al., unpublished data).

The results of this study indicate that density-dependent changes in *S. exempta* larvae do have an effect on the efficiency with which pathogens are transmitted through host populations. Conventional models of insect–pathogen interactions which incorporate the mass action assumption do not adequately describe the dynamics of *S. exempta* and its NPV. The type of non-linearity observed in this study would destabilise the dynamics of any *S. exempta*-NPV interaction, and at high densities the host may be able to escape regulation by the pathogen altogether. Instead high-density *S. exempta* populations may be regulated by other factors such as food availability or predation. High-density populations often cause widespread defoliation and so the food supply may become a limiting factor, and these populations also tend to attract large numbers of some predators such as storks (DLCO-EA/NRI 1987). At this stage, we do not know what regulates *S. exempta* numbers, but the fact that NPV is a major mortality factor at some outbreaks sites, suggests that NPV infections may be important, either alone or in combination with parasitoids or host plant availability. Moreover, the observation that both the number of moths and the number of outbreaks in East Africa is negatively correlated with early season rainfall (Tucker 1984; Harvey and Mallya 1995) suggests that climate also plays a part in limiting the extent of armyworm outbreaks. Thus, it is possible that as in some other systems (e.g. Grenfell et al.. 1998), there are important interactions between density-dependent and density-independent factors, with NPV transmission being enhanced during extended periods of rainfall. Another aspect of many existing models, a threshold host density below which a pathogen is unable to invade a host population, can also not be directly applied to the *S. exempta*-NPV system. Increased transmission efficiency among low-density hosts may allow pathogens to persist in low-density host populations, while high-density populations may be more resistant than expected to invasion by a pathogen. These results will have implications for the use of viruses and other pathogens as biopesticides to control *S. exempta* outbreaks. Further research is needed to determine whether density-dependent changes in the transmission efficiency of pathogens is a widespread phenomenon affecting a range of insect–pathogen systems. It is important that such research considers the effects of both long- and short-term variation in host density on pathogen transmission.

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