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Viruses as costs to gregarious feeding behaviour in the Lepidoptera

Michael E. Hochberg

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I present evidence that gregariously feeding species of lepidoptera have evolved resistance to infection by their viral parasites, in terms of the larger amounts of virus needed to cause an infection as larvae age. First, I show a significant positive association between age-related resistance to infection by viruses and gregarious feeding behaviour in 13 case studies involving temperate lepidoptera. Since pronounced levels of resistance to viral infection are not found in solitary species it can be inferred that this resistance has evolved in gregarious species at a cost. Further, that gregariousness itself often breaks down with larval age, suggests that the host responds to the selection pressure of the virus through not only physiological adaptations, but also behavioural ones. Second, I present evidence from a laboratory experimental system *(Pieris brassicae* and a granulosis virus) showing that observed age-related resistance is sufficient to explain the insensitivity of the per capita risk of infection to the number of larvae in the brood. The findings presented here are relevant to the use of pathogens for the biological control of insect pests.

M. E. Hochberg, NERC Centre for Population Biology, Imperial College, Silwood Park, Ascot, Berk SL5 7PY, England (present address: Laboratoire d'Ecologie – URA 258, Ecole Normale Supérieure – CNRS, 46 Rue d'Ulm, F-75230 Paris, France).

Aggregations of individuals are a pervasive feature of animal populations. There are a number of benefits to being in the proximity of siblings, conspecifics or even individuals from other species (Alexander 1974, Pulliam and Caraco 1984). These include higher realised fecundity, and the abilities to secure resources, defend against predators, and compete with other species. Living in the proximity of other individuals, however, is not always without its costs, such as increased intraspecific competition (Charnov et al. 1976, Buss 1981, Hochberg 1991a), and vulnerability to natural enemies (Kareiva and Odell 1987).

Another cost to aggregations, widely recognised for vertebrates, is increased vulnerability to parasitism (for review, see Dobson 1988). For example, Freeland (1979) has argued that inter-group transmission may be substantially lower than intra-group transmission in primates and that, as such, may constitute a constraint to group size. Increased risk of infection between related individuals living in groups is especially pertinent for ectoparasites transmitted from parents to offspring (Hoogland 1979, Brown and Brown 1986). For example, Brown and Brown (1986) demonstrated that cimicid bugs *(Oeciacus vicarius)* could impose significant costs to the fitness of colonially nesting cliff swallows *(Hirundo pyrrhonota)*, both in terms of survival and fecundity.

Nevertheless, there is ample evidence that group living can be maintained in the presence of parasites. For example, 13 different types of virus have been identified in the honey bee, *Apis mellifera* (Bailey 1981); this, despite sophisticated nest cleaning behaviours of worker bees (Rothenbuhler 1964). And, for some parasites, aggregations of the host may be a necessary requirement for persistence (Dobson 1988, 1989). For example, transmission of the monogenean parasite, *Pseudodiplorchis americanus*, in the spadefoot toad, *Scaphiophus couchii*, is synchronized to congregations

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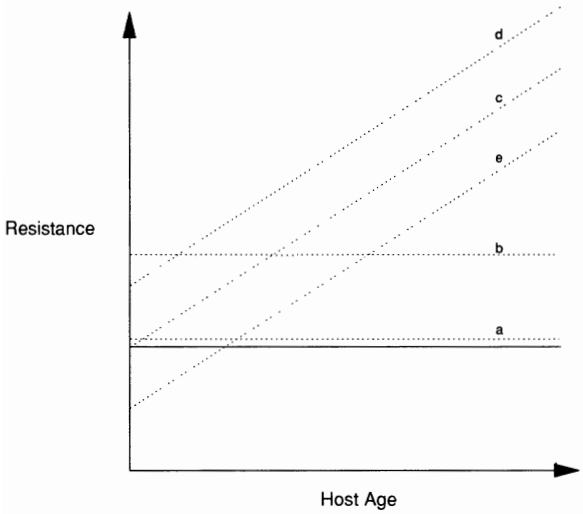

Fig. 1. Some hypothetical relationships between host age and resistance to parasitic infection for solitary and gregarious feeding hosts. Solid line refers to solitary hosts and dotted lines to gregarious hosts. See text for further details.

associated with host mate finding (Tinsley and Jackson 1986). The fact that these congregations occur very seldomly reflects the array of special adaptations for transmission on the part of the parasite (Combes 1968).

Although aggregations of the host need not be important for the persistence of some parasite populations (Hochberg and Lawton 1990), experimental studies show that they may be a feature of many insect host-parasitoid associations (Hassell 1978, Jones and Hassell 1988). Increased vulnerability to parasitism of host aggregations may have important implications for the evolution of habitat selection. For example, Valladares and Lawton (1991) have suggested that increases in the risk of parasitism of the holly leaf-miner, *Phytomyza ilicis*, may explain its lower than expected densities in intrinsically superior patches. Spatial density dependence in the per capita risk of infection of insect hosts by microparasites (i.e. viruses, fungi, bacteria, protozoa) has been little explored experimentally (Hochberg 1991b), but is implied by the theoretical requirement of sufficient densities of the host for parasite persistence (Anderson and May 1981).

This article investigates how group feeding behaviour in insects may put them at higher risk to infection by lethal microparasites than would be the case for solitary species. Specifically, I present evidence that gregarious feeding lepidoptera have evolved age-related resistance in the risk of infection to pathogenic viruses. The finding that solitary species have not evolved pronounced resistance suggests that there is a cost involved in its evolution.

An hypothesis

In general, age-related resistance to parasitism can be expected to evolve when (1) the per capita lifetime probability of infection increases with group size, (2) the period between infection and infectiousness is short compared to the generation time of the host, (3) the disease caused by the parasite reduces sufficiently host (inclusive) fitness, (4) the costs of evolving resistance are sufficiently low compared to the increases in fitness conveyed, and (5) there exists sufficient genetic variation in the host population for evolution of the resistance trait(s).

Many viruses of the Lepidoptera are transmitted when healthy hosts consume food contaminated by virus particles released from infected hosts after succumbing to the disease. Because the duration of the disease may be much shorter than the length of the larval period, feeding in the proximity of conspecifics or (as argued here) siblings can entail an elevated risk of contracting the disease with time, should the virus enter the group early in larval development. I would therefore expect gregarious feeding lepidoptera to have a more pronounced risk of infection as the larvae age than would species displaying solitary feeding habits. This increased risk is the basis for the hypothesis proposed here, that the maintenance of group feeding behaviour in the Lepidoptera has required the evolution of resistance to their viruses.

Fig. 1 presents several plausible hypothetical relationships between host age and resistance to parasitic infection. The null hypothesis of the present study is that resistance is not associated with host feeding behaviour (Fig. 1a). Alternative hypotheses to explain the generally higher levels of resistance observed in gregarious species as compared to solitary ones include: resistance irrespective of age (significantly greater intercept, but not slope, Fig. 1b), resistance solely as a function of age (significantly greater slope, but not intercept, Fig. 1c), resistance as a function of age and other age-independent components (significantly greater slope and intercept, Fig. 1d), or resistance as a function of age with a cost in terms of greater susceptibility to infection of young gregarious as compared to solitary hosts (significantly greater slope and smaller intercept, Fig. 1e).

Comparative analysis

The insect pathology literature is replete with examples of a positive relationship between the amount of pathogen required to kill a host and host age, such that regressions of the lethal dose required to kill 50% of a host sample, or LD_{50}, versus larval age (i.e. instar or weight) result in a significant positive association between age and resistance (Payne et al. 1981, Briese 1986). This may reflect a dilution effect, such that age-

Table 1. Age-related resistance to virus and associated larval feeding behaviour for the lepidoptera-virus systems employed in this study.

Case	Lepidoptera species	Virus[1]	Slope[2,4]	Behaviour[3,4]	Source
1	*Anticarsia gemmetalis*	NPV	NS	S	Boucias et al. 1980
2	*Anticarsia gemmetalis*	I	NS	S	Sieburth and Carner 1987
3	*Heliothis armiger*	NPV	NS	S	Teakle et al. 1985
4	*Heliothis punctiger*	NPV	–	S	Teakle et al. 1986
5	*Heliothis zea*	NPV	NS	S	Allen and Ignoffo 1969
6	*Hyphantria cunea*	GV	+	G	Boucias and Nordin 1977
7	*Hyphantria cunea*	NPV	+	G	Boucias and Nordin 1977
8	*Lymantria dispar*	NPV	+	S[5]	Burgerjon et al. 1981
9	*Mamestra brassicae*	NPV	+	G	Evans 1981
10	*Mamestra configurata*	NPV	NS	S[5]	Bucher and Turnock 1983
11	*Operophthera brumata*	NPV	NS	S	Wigley 1976
12	*Pieris brassicae*	GV	+	G	Payne et al. 1981
13	*Pieris rapae*	GV	NS	S	Payne et al. 1981

[1]Virus type: NPV = nuclear polyhedrosis virus, GV = granulosis virus, I = iridescent virus.
[2]Significance of slope of regression of log (LD_{50}/host weight) versus log (host weight). + = significantly positive, NS = not significantly different from zero, – = significantly negative (each + or – at least $p<0.05$).
[3]Feeding behaviour: S = solitary, G = gregarious.
[4]Spearman rank test of association between slope (coding: negative = 0, not significant = 1, positive = 2) and behaviour (coding: solitary = 0, gregarious = 2) is statistically significant ($r_s = 0.801$, $p<0.002$ for all 13 studies, and $r_s = 0.694$, $p<0.05$ for the nine studies involving NPVs).
[5]Eggs are laid in batches, but larvae disperse soon after hatching.

related increases in virus acquisition rate (via greater feeding capacity) are offset by the smaller surface to volume ratio of the ageing host's gut (the normal site of baculovirus infection).

In order to standardize and discriminate between simple dilution effects and more specific age-related mechanisms of resistance, it is necessary to express resistance as a function of rate at which virus is consumed along with food. One way of accomplishing this is to assume that the encounter rate between the feeding host and the parasite is proportional to the weight of the feeding larva. (In fact, there is plentiful evidence that metabolic rate scales as the ca. 0.75 power of body weight (Peters 1983), but employment of such a relationship does not affect the rank order of the regression slopes in the comparative analysis presented here). If there is no significant relationship between LD_{50}/unit host weight and host weight, then it can be concluded that a dilution effect may explain the increase in resistance with age. If resistance increases faster than body weight, then other mechanisms are possibly involved.

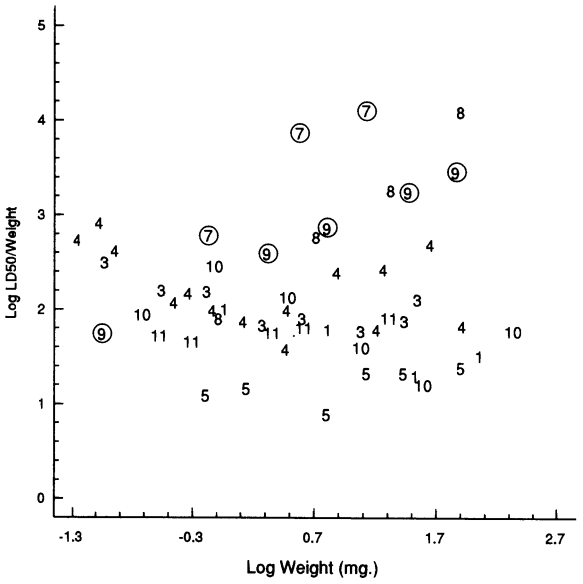

Fig. 2. Relationship between larval weight and viral dose/unit weight for nine case studies involving nuclear polyhedrosis virus. Number corresponds to the study (given in Table 1). Numbers without markers refer to solitary feeding species and numbers within circles refer to gregarious feeding species. Weights for study 9 are based on fitted regression.

The present analysis uses the results of 10 studies on virus-lepidoptera interactions compiled by Briese (1986), with the addition of 3 more recent investigations (Table 1). The criteria for employing a given study is that both larval weights and LD_{50}'s are estimated. Unfortunately, statictical tests involving differences in the regression intercepts (level of resistance at larval birth) could not be made since information on the weights of larvae at birth were not tabulated in the case studies. Therefore, only the age-dependent component of resistance is tested in the present study (i.e. lines c, d, and e of Fig. 1 cannot be statistically distinguished from one another).

Further, host feeding behaviour is coded as either solitary or gregarious, and age-dependent responses to the virus are coded as positive (i.e. increased resistance with age), none, or negative, depending on the signif-

icance of the slope in the regression between log (larval weight) versus log (LD_{50}/larval weight) (Table 1). Data points in which obvious 'maturation resistance' occurs (i.e. striking increases in resistance just prior to pupation; sensu Whitlock 1977), are not included in the analysis (see Discussion). All statistical testing is two-tailed.

The data set has some noticeable weaknesses, including different instars employed, congeneric hosts, different viruses applied to the same host species, and different host species exposed to closely related viruses. The arguments developed here should therefore be interpreted cautiously.

Results

There is a significant association between gregarious feeding behaviour and age-dependent response to the virus ($p < 0.002$, $n = 13$, Table 1; for those studies involving nuclear polyhedrosis viruses, $p < 0.05$, $n = 9$, Table 1, Fig. 2). With just one exception *(Lymantria dispar)* all of the solitary feeding species exhibit either no resistance or increased susceptibility to their viruses as larvae age (Table 1). All four cases in which a gregarious species is involved show a positive association between host age and resistance. Finally, host weight is not significantly associated with host feeding behaviour (youngest and oldest larvae tested for each study, $p > 0.05$, $n = 11$, not including studies 12 and 13 which did not explicitly tabulate host weight).

Furthermore, inspection of a number of additional studies which could not be standardized based on host weight (and therefore are not included in the statistical analysis), support the finding presented here that gregarious feeding hosts exhibit higher age-related levels of resistance than do solitary feeding species (see Briese 1986: Table 1).

Other experimental evidence

A separate study (Hochberg 1991b), investigating the spread of a granulosis virus through larval broods of *Pieris brassicae*, demonstrated that (1) the number of larvae initially infected by the virus and (2) the age at which the virus is first introduced into the brood, have significant impacts on the per capita risk of infection of the host – but, (3) the initial number of healthy larvae in the brood (over the tested range of 10 to 80) had no effect. This final point suggests that the age-related resistance identified by Payne et al. (1981) for *P. brassicae* (Table 1) may have evolved to offset the increased probability of contact between susceptible larvae and their virus-infected siblings. If this were not the case then we would expect that increasing the number of sibling larvae per brood should increase their per capita probability of contracting the infection (since the local per capita environmental load of the virus increases faster than per capita growth rate of the larvae; see Becker 1989 for discussion of chain binomial processes). Because the larvae were confined to a much smaller area than would normally be the case in the field (fourth and fifth instars usually disperse), it can be reasonably inferred that the physiological resistance of older larvae is sufficient to explain the insensitivity of risk of infection to brood size. It could not be assessed to what extent the loss of gregariousness in older larvae contributes to overall resistance.

Discussion

Two different lines of evidence support the hypothesis that gregariously feeding species of lepidoptera exhibit age-dependent resistance to infection by their viruses as compared to solitary feeding species. That resistance to infection remains roughly constant as solitary lepidoptera age, implies that there is a cost associated with the evolution of resistance in gregarious species. If this were not the case, then we would expect both solitary and gregarious species to show similar increases in resistance to infection with advancing age.

The frequent observation that gregarious feeding behaviour breaks down as larvae age suggests that the selection pressure of the virus may involve behavioural forms too. Whether behavioural resistance is responsible for the rapid break-down in gregariousness for hosts such as *Lymantria dispar* and *Mamestra configurata* could not be determined from the present analysis. Nevertheless, physiological and behavioural mechanisms of resistance, taken together, lead to the conclusion that by feeding solitarily (either at birth or later in larval life) a lepidopteran host reduces its lifetime risk of infection by its viral parasite.

The one obvious exception to the hypothesis proposed here is the gypsy moth, *Lymantria dispar*. It is interesting to note that although young larvae of *L. dispar* usually disperse from the tree upon which they hatch, once settled they tend to feed on the same tree for the first few larval instars (Weseloh 1987). Thus, although there may be little feeding association between siblings per se, the diel intra-tree movements of young larvae could put them at an elevated risk of contact with recently liberated virus from other larvae on the same tree.

There are other reasons than that proposed here for why lepidoptera larvae may tend to feed more solitarily with age. These include increased intra-brood competition for food and conspicuousness to natural enemies, both which are often associated with aggregated host distributions. Even if mechanisms other than infectious disease drive the evolution of age-related solitariness,

there should be an incidental benefit in terms of reduced risk of infection by endemic parasites. I know of no temperate lepidopteran species whose larvae do not associate when young, but preferentially associate as they age.

In several of the studies listed in Table 1 (3, 4, 9, 10, 11), the sources include data points in which a striking increase occurs in the resistance to infection just prior to the pupation of the host. This is often termed 'maturation resistance', and is manifested by the slowing or stopping of virus replication as a result of changes in cell replication and hormone levels in the host both between instar moults (Teakle et al. 1986) and prior to pupation (Keeley and Vinson 1975, Whitlock 1977). Such resistance is probably not a mechanism evolved specifically in response to parasitism.

The results of this study are consistent with the hypothesis that the observed correlation between gregarious feeding and age-dependent resistance has arisen independently in several phylogenetic lineages, strengthening the hypothesis that there is a causal relationship between the two. For example, cases 12 and 13 (Table 1) involve congeneric species which exhibit different feeding behaviours and show the predicted age-dependent response to the virus. This also appears to apply to host-microparasite associations involving other orders of insect (case of *Gilpinia hercynae* and its nuclear polyhedrosis virus described in Cunningham and Entwistle 1981), and different types of virus (compare cases 1 and 2, or cases 6 and 7, Table 1). A rigourous test of the influence of host and parasite phylogenies on resistance to parasitism will require more case studies than are currently available.

Some of the host species listed in Table 1 are susceptible to closely related virus strains. For example, the granulosis viruses extracted from wild *Pieris brassicae* and *Pieris rapae* can be distinguished from one another both biochemically and in terms of their infectivity to congenerics and other conspecific strains (Crook 1986). Crook (1986) showed that the two virus isolates derived from *P. brassicae* were both highly infectious to *P. rapae*, whereas 13 isolates taken from various populations of *P. rapae* were much less virulent to *P. brassicae* than were the two isolates derived from the latter. It would be interesting to know to what extent the genetic diversity of the virus has arisen from sharing hosts with very different feeding behaviours.

That age-related resistance may be predicted from larval feeding behaviour has important implications for the biological control of insect pests using pathogens. Under natural conditions we might expect that, on average, there should be little difference in the risk of infection between solitary and gregarious feeding hosts. This is because the increased risk of parasitism to gregarious feeders is offset, to some extent, by their elevated age-related physiological and behavioural resistance as compared to solitarily feeding species. Under conditions in which the parasite population is artificially augmented, there should be increased selection pressure for the evolution of resistance to the parasite, irrespective of feeding behaviour. The consequence of augmenting parasite populations for gregarious feeding species is that specific genetic mechanisms for conferring physiological or behavioural resistance, which this study suggests are already in place, may be prone to amplification. In contrast, the evolution of resistance in solitary species would require *both* the appearance of genes conferring resistance *and* their subsequent amplification. This line of reasoning suggests that the long-term control of gregarious pests using augmented or inundated pathogens is apt to be more difficult than for solitary species.

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