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A koinobiont parasitoid mediates competition and generates additive mortality in healthy host populations

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Insects are subject to attack from a range of natural enemies. Many natural enemies, such as parasitoids, do not immediately, or ever, kill their victims but they are nevertheless important in structuring biological communities. The lag that often occurs between attack and host death results in mixed populations of healthy and parasitised hosts. However, little is understood about how the effects of parasitism during this lag period affect the competitive ability of parasitised hosts and how this, in turn, affects the survival and dynamics of the surviving healthy host populations. Here we investigate the impact of the timing of introduction, and the strength of that introduction, of a parasitoid natural enemy *Venturia canescens* (Gravenhorst) on the outcome of intraspecific competition between larvae of the Indian meal moth, *Plodia interpunctella* (Hübner). In contrast to healthy hosts alone, we find reduced survival of healthy larvae with increasing periods of exposure to greater numbers of parasitised conspecifics. This represents indirect mortality of the host, which is in addition to that imposed by parasitism itself. Furthermore, longer periods of exposure to parasitised larvae resulted in an increase in development time of healthy individuals and they were larger when they emerged as adults. These results are relevant to both insect–parasitoid and insect–pathogen systems where there is a lag in host death following infection or attack.

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In many animal populations mortality events are stage-dependent, such as larval competition for limited resources or egg predation, and the timing of mortality in an animal's life cycle can be an important factor in terms of its impact on survival and reproductive rates, the vital rates by which a population compensates for losses. Previous empirical studies of compensation by resource populations in response to mortality have considered direct mortality or killing power of natural enemies through manipulating enemy densities (Huffaker and Matsumoto 1982, Graham and Lambin 2002, Lane and Mills 2003) or artificially imposed stage-dependent mortality (Cameron and Benton 2004). This approach omits the influence of the timing of a mortality event relative to other life history events such as resource consumption, dispersal and reproduction. Additionally, the above approaches often only consider natural enemies that kill their victim immediately. Many natural enemies do not immediately or ever kill their victims, but they are nevertheless important in structuring biological communities through differentially altering vital rates (Poulin 1999).

Koinobiont parasitoids, an example of such a natural enemy, inject their hosts with an egg (or a clutch of eggs), whereupon hatching the juvenile permits the host to live until some later stage (Haeselbarth 1979). The length of

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this delay, or lag, in host death is often determined by the age or size of the host when it is parasitised and by the cue from the host to the juvenile parasitoid that the host contains sufficient resources for the parasitoid to complete its development (Godfray 1994). Attack by parasitoids with this life history strategy will generate a mixed host population that comprises both healthy and parasitised hosts, thereby leading to competitive interactions between them. Any changes in behaviour, physiology or growth of the parasitised host before the onset of destructive feeding by the parasitoid larvae are akin to sublethal effects, which are better known in host–pathogen systems (Sait et al. 1994, Boots and Norman 2000, Boots et al. 2003). The period over which these effects can influence competitive dynamics in the host population would be determined by the time of introduction of the parasitoid natural enemy relative to the period of competition in the host life history, usually the duration of the larval stage. Recent work in numerous systems has begun to demonstrate the importance of infected competitors in enemy–victim dynamics and in the structuring of ecological communities (e.g. bacterial communities, Kusch et al. 2002; lepidopteran pest communities, Bernstein et al. 2002; natural lepidopteran populations, Sisterson and Averill 2003). However, few studies have focused on how competition for resources between parasitised and healthy individuals may influence the feedback between interacting parasitoid and host populations.

In this paper we investigate how the timing of introduction of a common natural enemy may alter the competitive dynamics within a population with limited resources. Such a question is timely given the progress made in understanding the role of predation and parasitism in mediating density-dependent competition within populations and between species (Chase et al. 2002). An emerging body of empirical and theoretical evidence has highlighted the importance of the timing of mortality events, including enemy introduction, to the outcome of dynamics within age-structured populations (parasitism, Münster-Swensden and Nachman 1978, Godfray et al. 1994, Briggs and Latto 1996, van Nouhuys and Lei 2004; commercial harvesting, Astrom et al. 1996, Jonzen and Lundberg 1999, Tang and Chen 2004; hunting, Kokko 2001).

We manipulate the time over which host larvae parasitised by a parasitoid, *Venturia canescens* (Gravenhorst) (hereafter *Venturia*), compete within cohorts of healthy larvae of the Indian meal moth, *Plodia interpunctella* (Hübner) (hereafter *Plodia*). *Venturia* is a solitary koinobiont endoparasitoid of Pyralidae larvae such as *Plodia*. Wasps lay a single egg inside a larva that, as described above, hatches and delays development until the final instar of the larva (Harvey et al. 1994). The parasitoid takes about 20–25 days to develop depending on temperature, which instar it attacked and how large it is (Harvey et al. 1994) and it prefers to attack larger instar larvae (Sait et al. 1997), although all instars except the smallest 1st instar larvae can support parasitoid development.

Previous investigations with this natural enemy have shown that greater numbers of adult *Venturia* attacking cohorts of healthy larvae result in an increased total number of hosts attacked, but reduced per capita success for each parasitoid (Huffaker and Matsumoto 1982, Lane and Mills 2003). Additionally, changes in the competitive nature of the host environment from resource limitation to enemy free space has been demonstrated (i.e. competition for access to a physical refuge, Begon et al. 1995, Lane and Mills 2003). Uniquely we consider the additional indirect effects of parasitised competitors in an experimental community.

Methods

Plodia have been reared as an out-bred colony at Leeds for three years following transfer from the University of Liverpool where they were cultured under identical conditions for ten years. Eggs were collected daily from *Plodia* reared on bran diet (broad bran 800 g, honey 200 ml, glycerol 200 ml, yeast 160 g, preservatives 12 g) to start cultures of uniform age (Begon et al. 1995, Sait et al. 2000). *Venturia* were from a thelytokous laboratory population reared on *Plodia* for over a decade. The parasitoids were maintained in a constant environment of $28 \pm 2^{\circ}C$ during development and $22 \pm 2^{\circ}C$ post emergence. Only parasitoids between 2–4 days post emergence were used in the experiment. Host stock populations and the experiments were maintained at $27 \pm 2^{\circ}C$ and LD cycle of 16:8 h.

Forty early third instar larvae (13 days old) were selected randomly from the stock cultures and placed in a clear plastic box (dimensions $73 \times 73 \times 73$ mm, Azpak, Loughborough) containing 2.2 g of bran diet. This represented 50% of the diet required for all 40 larvae to pupate successfully (Reed 1998), but did not provide a physical refuge from parasitism (Begon et al. 1995). On day 1 of the experiment (one day after the larvae were put in the box) a single 2–4 day old parasitoid was added to the box and allowed to search for and parasitise hosts for a fixed period of 6 h before being removed. A pilot study demonstrated that a 6 h period of attack from a single parasitoid gave rise to populations of both healthy and parasitised host larvae (34 ± 28 sd successful attacks/100 hosts). This process was subsequently repeated in separate boxes on days 2, 4, 6, 8, 10, 12 and 14, which resulted in decreasing periods of competition between parasitised and unparasitised hosts. Our final treatment (day 14) was determined by a pilot study that showed that this was approximately when host pupation occurred. In this treatment parasitised and unparasitised

larvae compete for the shortest period of time. Each of the eight parasitism treatments plus a control, in which no parasitoid was added, was replicated nine times.

The boxes were monitored daily and the survival of moths and their development times were recorded. Adult moths were sexed and placed in labelled tubes and frozen for later leg measurements (right mid femur). Leg lengths are correlated with egg load in *Plodia* (Gage 1998, Reed 1998), representing a potential delayed effect of competition since growth and reproduction are influenced by resource acquisition during the larval stages in this and many insects (Benson 1973). Additionally, adult parasitoid numbers from each treatment were recorded.

Response variables and statistical analysis

Host survival

The effect of the timing of parasitoid introduction on host survival was determined by a general linear model (ANOVA) of the arcsine square root transformation of the survival data. Additionally, the effect of parasitised larvae density, which is an estimate of the strength of competition with parasitized hosts, on the survival of the healthy larvae was examined by plotting the total emergence of parasitoids from each cohort against proportional survival of healthy larvae to adult moths after each 6-hour bout of parasitism. This is calculated after first correcting for background mortality (Table 1). Data were arcsine transformed for analysis and the means and standard deviations were back transformed for graphical presentation.

In order to account for encapsulation, when the host immune system encloses and kills a developing parasitoid egg or larvae, and its potential effect on the surviving larva, the effect of the number of emerging parasitoids was also analysed after weighting by an approximate encapsulation rate of 5% (Reed et al. 1996, S. M. Sait unpubl.). However, as *Plodia* encapsulates *Venturia* eggs soon after parasitism (Salt 1970), there is likely to be a negligible effect on subsequent host development and we assume that in our experiment those hosts that encapsulate the parasitoid will develop and have the same competitive ability as healthy hosts. Larval mortality, resulting from injury during parasitoid attack, is negligible in 3rd to 5th instars (Table 1). In this study, superparasitism is unlikely to occur but it has to be assumed that if it does it has no effect on the results.

As parasitism events occur independently at different points in time, each parasitoid introduction treatment (i.e. days 1–14) is considered separately in the analyses. Differences between the replicates and their interactions explained little of the variation in survival and were non-significant (ANOVA block $F_{1,63}=0.12$, $p=0.72$, all interactions $p=0.39$).

Size and development time

Leg size data were analysed with a general linear model (ANOVA). Differences in the time to emergence of moths were analyzed with an accelerated failure time survival model (Fox 2001) using a Weibull distribution. This distribution best fit the censored and normalized deviance residuals from our model (Harrell 2003). We modelled emergence time as a function of the timing of parasitoid introduction, the strength of the parasitoid introduction (strength = the number of successful parasitoid attacks) and the sex of the adults. Initial model assessment indicated that males consistently mature earlier than females ($p<0.0001$) and that there was no interaction between treatment and sex ($p=0.585$), so we examined each sex separately. All the data from all the treatments were pooled for the failure time model such that each adult moth emergence was an individual event in the model and this took account of differences between replicates.

For all analyses, post-hoc multiple comparisons were conducted using the best.fast method that determines the smallest critical value by choosing from various methods and minimises type II error by simultaneously

Table 1. Estimates of background survival of different instars of parasitised and healthy larvae. The survival of both parasitised and healthy larvae when put in competitive environments is also given. Rates refer to either mean values or ranges. In these estimates, sample size refers to the number of replicates with the number of larvae in each replicate given in brackets.

Vital rate	Instar	Rate (%)	Sample size	Reference
Background mortality of healthy larvae	3	0	99	
	4	0	97	Sait et al. 1994a
	5	0	93	
Background mortality of parasitised larvae	3	6	148	Sait et al. 1995
	5	9	53	Harvey et al. 1995
	5	9	52	S. M. Sait, unpubl.
Background mortality of parasitised larvae attacked once	5	5	33	S. M. Sait, unpubl.
Survival of parasitised larvae under competition	2–5	60–85	7 (80)	Begon et al. 1995
	3–5	66–86	9 (10,20,40)	Cameron, T.C, unpubl.
Survival of healthy larvae under competition	3–5	70	9 (40)	Cameron, T.C, unpubl.

running the procedure for all treatments versus the control. All analyses and models were run in S-plus statistical software (Version 6.1.2 student ed. for MS Windows, © 2002 Insightful Corp. Seattle WA).

Results

Effects of parasitism and competition on survival of healthy host larvae

Overall, earlier parasitoid introduction, which leads to longer periods of competition between parasitised and healthy larvae, results in a significant reduction in survival of healthy moth larvae compared to the control where healthy larvae were reared alone (Fig. 1, ANOVA, $F_{8,18}=8.91$, $p<0.0001$). Parasitoid introduction on days 12 and 14, which results in shorter periods (1–3 days) of competition with parasitised larvae, were the only treatments where there was no significant reduction in the survival of healthy larvae.

Increased strength of exposure to competition with parasitised hosts also significantly reduces the survival of the healthy unparasitised larvae (Fig. 2, ANOVA, $F_{28,18}=6.06$, $p<0.001$). Additionally, there was a significant interaction between the time of parasitoid introduction and the strength of competition with parasitised hosts (ANOVA, $F_{26,18}=3.46$, $p<0.005$); earlier introduction coupled with higher numbers of parasitised larvae leads to a greater reduction in survival of remaining healthy larvae.

The negative relationship is consistent across all treatments (Fig. 2), but not always statistically significant (parasitoid introduction on days 4 and 6 results in a regression of $p>0.05$). In these particular treatments there was little variation in the strength of attack between the replicates, which resulted in a clumping of the data. Weighting the survival data for known encapsulation rates makes no difference to the outcome of competition (results not shown), which supports our prediction that hosts that have successfully encapsulated the parasitoid compete at the same level as healthy hosts.

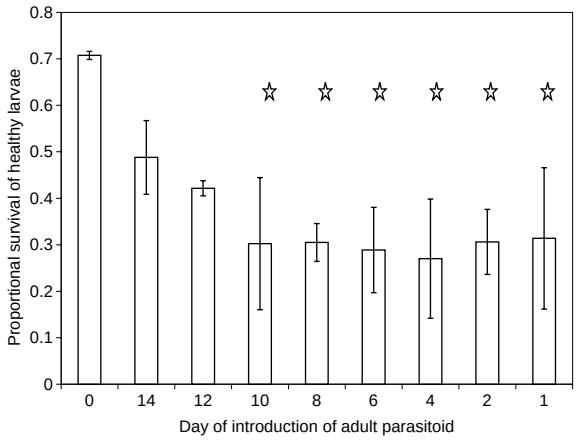

Fig. 1. Proportional survival (±sd) of healthy larvae with increasing periods of exposure to competition with parasitised larvae. A greater period of exposure relates to an earlier adult parasitoid introduction (day of attack). A period of 14 days exposure is when the day of parasitism was day 1. Values significantly different ($p<0.05$, Dunnet method) from the control, no exposure to parasitoids, are denoted by ☆.

Effects of parasitism and competition on pattern of adult emergence

Since the qualitative effects of the treatments on both sexes were similar we have only provided detailed analyses of the effects of timing and strength of introduction on female development. A breakdown of these results for both sexes is provided online.

Increased periods of exposure to parasitised conspecifics following earlier introduction (e.g. comparing introduction on day 1 against controls, $p<0.001$) results in an increase in development time (1–4 days delay, Fig. 3 a–c) whereas parasitoid introduction on days 12 and 14, at the end of the period of competition, results in a significant decrease (2 days, $p<0.01$ and $p<0.01$ respectively, Fig. 3 a–c) compared to the controls. Across all introduction treatments, increased strength of exposure (parasitised larvae density) results in a reduction in time to emergence (approximately 3 days, $p<0.01$, Fig. 3 d–f).

Effects of parasitism and competition on healthy adult size

There were differences in leg size measurements between the sexes; males were smaller at eclosion (males 1.72 ± 0.13 mm; females 1.89 ± 0.15 mm, students t-test: $t_{1,\ 387}=-7.881$, $p<0.0001$), but there were also no significant treatment-sex interactions (ANOVA, $F_{1,834}=0.264$, $p=0.60$) and the data for each sex was analysed separately.

In general, larger adult moths of either sex emerged from boxes invaded by a parasitoid (Fig. 4a, only females shown as graphically similar to males, ANOVA: females $F_{8,271}=5.54$, $p<0.0001$, males $F_{8,38}=4.68$, $p<0.0001$). The effect was only significant in later periods of introduction (treatments on days 12 and 14, post hoc mca, Sidak method), when competition between healthy and parasitised larvae is most limited, but also when competition between healthy hosts alone has been most prolonged.

Increased strength of exposure results in significant changes in the average size of healthy larvae in the cohorts relative to the control treatments (Fig. 4b only females shown as males graphically similar, ANOVA

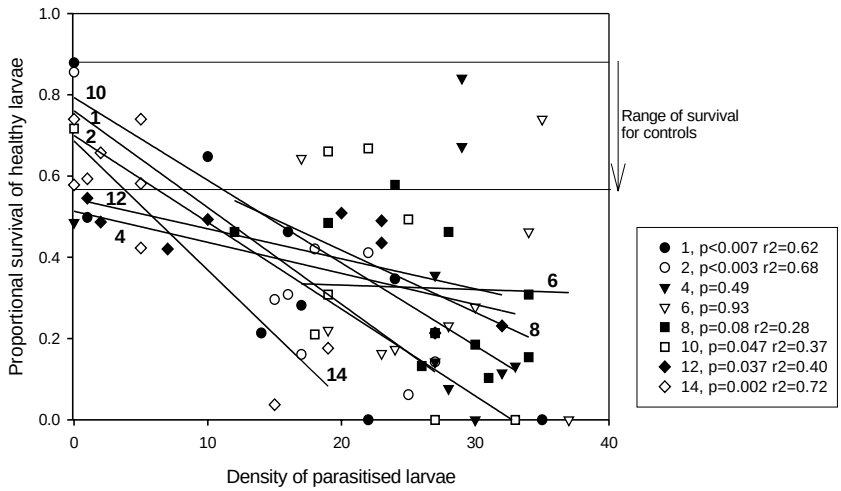

Fig. 2. The proportional survival of healthy larvae against the density of parasitised larvae (strength of introduction). Each regression line, denoted by a number, corresponds to the day of parasitoid introduction. The figure caption describes the summary statistics for each regression.

females $F_{24,364}=3.81$, $P<0.001$, males $F_{25,376}=3.33$, $P<0.001$). It would appear, however, that the direction of the effects on size changes with increasing parasitized larval density (second order polynomial $y=1.8853+0.0089\times x+-0.0004\times x^2$, $F_{2,409}=8.63$, $p=0.002$). Fig. 4b suggests that there may be a threshold (para-

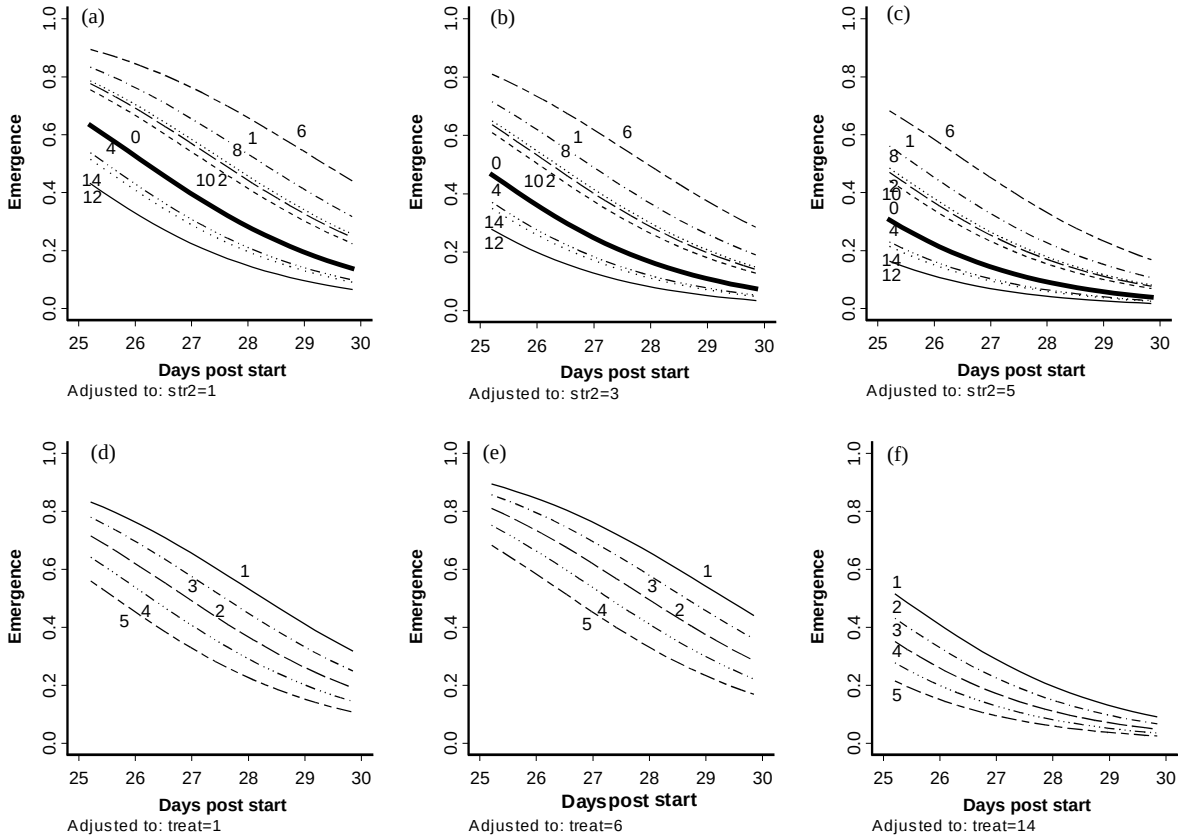

Fig. 3. Chart (a–c) show the survival model outputs for the effects of the timing of introduction of the adult parasitoid on the proportion of *Plodia* not emerged (Y-axis) over time (X-axis). The analysis is adjusted for increasing density of parasitised larvae from (a) low (str = 1) (b) medium (str = 3) (c) high (str = 5). The solid dark line represents the control treatment where no parasitoid invaded. Charts (d–f) shows the survival model outputs for the effects of exposure to increasing density of parasitised larvae (1 = 0–7, 2 = 8–14, 3 = 15–21, 4 = 22–29, 5 = 30+) independent of the period of exposure. The analysis is adjusted for (d) short (treat = 1) (e) medium (treat = 6) (f) long (treat = 14) periods of exposure. Compare plots at 0.5 on Y-axis where 50% of moths have emerged.

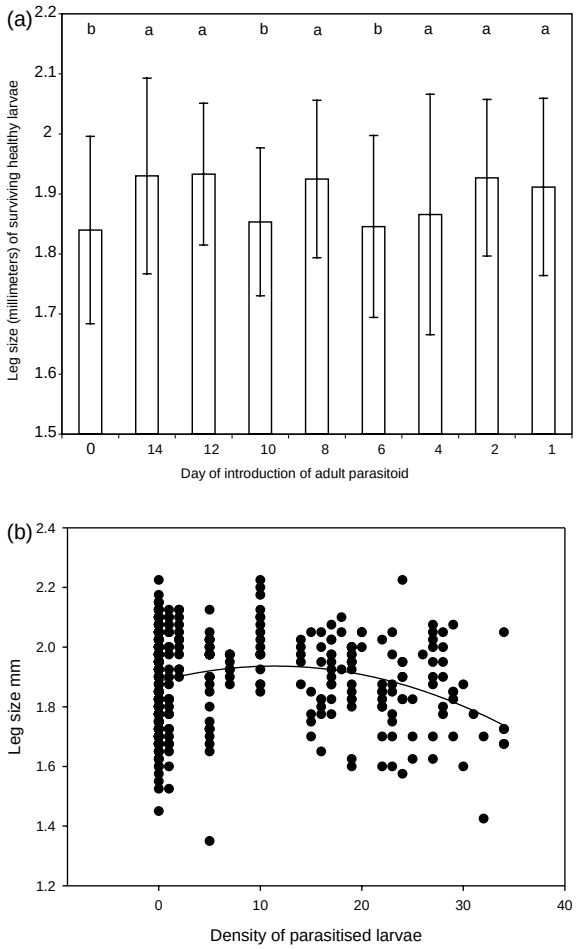

Fig. 4. (a) Mean leg size in millimeters ($\pm$ sd) as a function of the day of parasitoid introduction. A greater period of exposure relates to an earlier adult parasitoid introduction (day of attack). A period of 14 days exposure is when the day of parasitism was day 1. The same letter indicates treatments that were not significantly different from each other. (b) The relationship between leg size of surviving healthy moths and the density of parasitised larvae. All data presented are for female moths only as males show similar results. See text for equation.

sitised larvae at densities of 10–12) where the effects of increasing numbers of parasitised conspecifics at low densities leads to larger adults, but at high densities leads to smaller adults.

Discussion

Intuitively we might expect that parasitised hosts would exert a weaker competitive effect on healthy, unparasitised hosts. In this study we have found the opposite effect. An increase in either the period or strength of competitions with parasitised conspecific larvae can decrease the survival of the remaining healthy larvae relative to the survivorship that results from competition between healthy larvae alone. Furthermore, these effects are additive, so that mortality of healthy larvae is higher under longer periods of competition with greater densities of parasitised conspecifics.

There are a number of possible mechanisms driving the results we obtained for the interaction between healthy *Plodia* larvae and *Venturia*-parasitised larvae: (i) parasitised larvae inflict more fatal injuries on competitors; (ii) parasitised larvae behave differently from healthy larvae; (iii) parasitism results in decreased resource requirements of the host larvae. We will discuss each of these possibilities in turn.

(i) Are parasitised *Plodia* more aggressive?

Larval Pyralidae fight and inflict injury on competitors with their mandibles and injuries may result in infection and/or death (Corbet 1971). It has been shown that parasitised *Plodia* larvae are more likely to be injured or cannibalised than their healthy counterparts if larvae are kept with no food (Reed et al. 1996), though any amount of food provision negated this observation. Parasitised larvae of the cranberry fruitworm *Acrobasis vaccinii* Riley (Lepidoptera: Pyralidae) were also less able to defend a territory than healthy larvae (Sisterson and Averill 2003). So, it is unlikely that parasitised larvae are more aggressive.

Considering that *Venturia* selectively parasitise the largest larvae (Sait et al. 1995, 1997), it is well documented that larger *Plodia* larvae are stronger, more aggressive competitors and asymmetric competition is important in driving the population dynamics observed in closed experimental populations (Sait et al. 1994, Bjørnstad et al. 1998, Briggs et al. 2000, Wearing et al. 2004a, 2004b). Parasitised larvae may then be competitively dominant in fighting bouts over healthy larvae due to their larger size, rather than their infective state. However, as the greatest reduction in healthy larval survival followed early parasitoid introduction, when hosts are small and there is little variation in host size, and parasitism reduces host body size compared to healthy larvae (Harvey et al. 1994), we do not feel that selection of larger larvae by the searching adult parasitoid is an important mechanism in these experiments.

(ii) Do parasitised larvae behave differently?

Contestants often display a signal to deter or acknowledge a competitor in order to prevent an actual bout taking place (Parker 1974). In *Plodia*, larvae regurgitate fluid from their mandibular glands on meeting a conspecific, after which the two competitors withdraw (Corbet 1971). Overcrowding stimulates the excess production of mandibular fluids which is thought to

induce dispersal behaviour (Corbet 1971, Mudd and Corbet 1973, Mossadegh 1980). This behaviour could be considered as interference competition as they disrupt the time available to the larvae for feeding.

It is unknown whether parasitism of *Plodia* by *Venturia* results in a different response of parasitised hosts to mandibular fluids, but if parasitised larvae were behaving according to different density-dependent functions and requirements, they may have a competitive advantage over healthy hosts by avoiding competitors and suffering less interference competition.

Parasitised hosts often appear to behave differently from their healthy counterparts (Fritz 1982). Whether this is due to manipulation of the host by the juvenile parasitoid is still open to debate (Godfray 1994). However, it is clear that some parasitised hosts, although likely to perish due to infection, suffer reduced rates of other mortality factors in the interim period between infection and death. There are documented cases of reduced mortality, or reduced probability of further attack of insect larvae following initial attack by a parasitoid (Stamp 1981, Brodeur and McNeil 1992). Competition for resources is not cited as a possible selection pressure for the evolution of host behaviour modification. However, other selective pressures, such as reduced activity or dispersal or conspecific patch avoidance, may have the same effect of increasing access to resources. While it is intuitive that larvae may behave differently following parasitism, it is not clear that it could generate the survival patterns observed here.

(iii) Do parasitised hosts require fewer resources?

Harvey et al. (1994, 1995) observed that hosts that were parasitised when small and subject to long periods of parasitism, were significantly smaller and had reduced growth rates than their healthy unparasitised conspecifics. This result has been confirmed in other pyralid moths (Harvey and Vet 1997). Thus, *Venturia* successfully develops and emerges from hosts that are often smaller than healthy *Plodia* larvae. This suggests that, although the parasitoid does not commence destructive feeding until the host has reached a threshold size, *Venturia's* minimum requirements for development are less than that of its host (approximately 50–70%). Such a difference in requirements may render parasitised larvae less susceptible to competition-derived mortality when resources are scarce. This was suggested by Bernstein et al. (2002) who compared the outcome of competition on host survivorship between either healthy *Ephestia kuehniella* (Zeller) alone or exclusively between those parasitised by *Venturia*. There were a greater number of insects emerging from high densities of entirely parasitised larvae than from entirely healthy larval populations.

Contrary to this are the findings of a similar series of experiments with *Drosophila melanogaster* and the parasitoid *Asobara tabida* (Hymenoptera: Braconidae) (Kraaijeveld and Godfray 1997, Tien et al. 2001). Parasitised larvae were found to have reduced background survival rates compared to healthy larvae and their competitive abilities were compromised (Tien et al. 2001). This was due to the costs involved in the hosts immune response to a parasitoid egg. In our system *Plodia* is not a strong encapsulator of *Venturia* eggs (approximately 0–10% depending on instar attacked) and *Venturia* will passively avoid the immune response of its host (Kinuthia et al. 1999, Beck et al. 2000). Similarly, parasitised cranberry fruitworm *Acrobasis vaccinii* Riley (Lepidoptera: Pyralidae) were found to give up their resource, a single cranberry, more readily under strong direct contest competition (Sisterson and Averill 2003). In this situation the resource that is challenged is both a territory and a food source and it is therefore difficult to compare to our system.

If reduced resource consumption due to reduced requirements is the mechanism driving reduced susceptibility to competition of parasitised larvae, we must ask why it is not releasing healthy larvae from competition? This is because a greater number of both adult moths and parasitoids develop compared with the number of developing moths in the parasitoid-free populations, which reflects the number of individuals that survive competition. The overall resource use is greater and the competitive environment is increased when parasitoids are present (Takahashi 1973, Bernstein et al. 2002, Lane and Mills 2003, T. C. Cameron unpubl.). This hypothesis is not unlike that of the R* theorem (Tilman 1982); in our case parasitism reduces the minimum requirement of parasitised hosts, allowing them to survive periods of strong competition at the expense of healthy host survival. We predict that this pattern of host–parasitoid interaction will be observed more broadly when the level of host competition, host survival and development is dependent on meeting stage thresholds that are governed by food intake, when larval parasitoid survival is high and when parasitoid resource requirements for successful development are less than that of its host.

Patterns of size and date at emergence

The introduction of parasitised competitors resulted in an increase in size of surviving adult moths. Survivors, then, gain from enemy introduction as larger body size can confer benefits to the individual (e.g. mate access or fecundity, Gage 1998). However, increases in moth size are reduced when the exposure to parasitised conspecifics is for a longer period, emphasising that the potential

benefits following survival from parasitoid attack can be countered by prolonged periods of competition with parasitised competitors.

Late parasitoid introduction, which results in significantly larger moths that develop faster than controls, demonstrates an important point. Despite much of the resource depletion having already occurred, large larvae that escape parasitism during late parasitoid introduction benefit over larvae in the control experiments. The decrease in development time is likely to occur due to a relaxation of interference competition for safe pupation sites i.e. cannibalism free space for vulnerable pupae. A possible explanation for this is that large final instar larvae parasitised by *Venturia* are entering the pre-pupal stage more quickly than a similar sized healthy larva, a potential mechanism of risk avoidance from cannibalising larvae (J. Whittingham and S. M. Sait, unpubl.).

The effects of parasitised larvae competitor density on healthy larval development are non-linear as was found in *E. keuhniella* (Lane and Mills 2003). Overall, surviving healthy larvae that took longer to develop were smaller as adults, contrary to what might be expected from traditional life history theory. When faced with a competitive environment at earlier stages of development, an individual can avoid the lethal effects of competition through delaying growth (Day and Rowe 2002). There are costs involved in this tradeoff and they arise during later stages of competition, where resource quality and quantity has been reduced. Delaying growth to increase survival results in smaller sized adults, which in turn results in a reduced reproductive potential, as in female *Plodia*, size is correlated with egg load (Gage 1998). This is evidence of a delayed density-dependent response.

The results presented here are relevant to the understanding of parasitoid–host systems where competition between hosts is important, and perhaps of particular interest to infestations of larvae in stored products as the outbreaks are often patchily distributed, competition within a patch is strong (Arbogast and Mullen 1978), and the aim of biocontrol release is for parasitism rates to be high. Koinobiont parasitoids may be useful biological control agents not only because of direct mortality caused by killing the host, but if the larvae they parasitise are also less susceptible to competition they can also suppress pest populations through density-dependent additive mortality. Indeed, pest management and general ecological models predict that reduced susceptibility to competition of parasitised hosts can benefit control through introducing stability in the enemy–victim dynamics (Spataro and Bernstein 2004). There has been little exploration of how influential or widespread these effects may be in nature.

Additional information can be found at:
http://www.fbs.leeds.ac.uk/People/staffprofile.php?staff = bgytcc

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