Ecological Entomology

Ecological Entomology (2009), **34**, 763–771 DOI: 10.1111/j.1365-2311.2009.01131.x

Parasitism and constitutive defence costs to host life-history traits in a parasitoid–host interaction

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Abstract. 1. The level of an organism's investment in defences against natural enemies depends on the fitness costs of resisting parasitism and on the costs of maintaining defences in the absence of infection. Heritable variation in resistance suggests that costs exist, but very little is known about the nature or magnitude of these costs in natural populations of animals.

2. A powerful technique for identifying trade-offs between fitness components is the study of correlated responses to artificial selection. We selected for increased resistance in the Indian meal moth, *Plodia interpunctella*, following parasitism by the koinobiont parasitoid, *Venturia canescens*, and measured the cost of resistance to parasitism and the cost of maintaining resistance in the absence of immune challenge during the next generation.

3. Parasitism decreased larval host size, growth, and developmental time and was significantly negatively correlated with the size of surviving host adults. Larvae of the next generation also had a reduced developmental period, whilst the duration of the invulnerable pupal instar was increased. There was no effect on host adult size and related fecundity in the F_1 generation.

Key words. Developmental time, growth, parasitism, *Plodia interpunctella*, resistance, selected populations, *Venturia canescens.*

Introduction

Organisms continually exposed to parasites generally evolve some means of defence. Understanding how the magnitude of investment in host defences correlates with the costs of infection represents one of the exciting research areas in evolutionary ecology. Mostly found in the orders Hymenoptera and Diptera, parasitoids are widespread components of most terrestrial ecosystems, and are estimated to constitute more than 20% of all insect species (Strand & Pech, 1995). Parasitism involves the laying of parasitoid eggs on, in or near susceptible hosts (usually the juvenile stages of another insect). Koinobiont parasitoid larvae develop within their larval host, before eventually killing it by destructive feeding and emerging as adults (Godfray, 1994). Since only one of the two protagonists can survive parasitism, there is strong selection pressure on hosts to be resistant and on parasitoids to evolve counter-defence mechanisms. The costs of actual defence against parasitoids always have to be paid, as the only alternative for undefended hosts is death (Kraaijeveld *et al*., 2002).

The primary cellular defence mechanism used by Lepidoptera is haemocytic encapsulation of parasitoid eggs or larvae (Lackie, 1988). Encapsulation involves surface recognition of the parasitoid by host haemocytes and eventually results in a multicellular capsule that kills the parasitoid by asphyxiation (Strand & Pech, 1995). Inevitably, the fight against parasites is metabolically costly. So-called 'induced costs' may be associated with the direct negative effects of parasitism (e.g. the reallocation of host resources towards parasitoid growth). Alternatively, there may be an indirect 'constitutive cost' associated with the maintenance of host defence machinery, even in the absence of immune challenge. As the resources required for resistance may be the same as those required by non-immune related fitness components (such as growth and reproduction) (Zuk & Stoehr, 2002), the resistance to pathogens needs to

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be examined in cost–benefit terms within a life-history theory framework.

Most of the previous research on host resistance to parasitoids has been carried out on fly–parasitoid systems, e.g. with *Drosophila* (Kraaijeveld *et al*., 1997, 1998, 2001; Kraaijeveld & Godfray, 1997, 1999; Fellowes *et al*., 1998a,b,c, 1999a,b; Hoang, 2001) and aphid–parasitoid systems (Milner, 1982; Hufbaeur & Via, 1999; Müller *et al*., 1999; Ferrari *et al*., 2001; Hufbaeur, 2001; Ferrari and Godfray, 2006; Von Burg *et al*., 2008). Some of these studies have uncovered several trade-offs between immunity and life-history traits. For example, Pimentel *et al*. (1978) showed a shortening of the susceptible instar in *Musca domestica* parasitised by the pupal parasitoid *Nasonia vitripennis* (Hymenoptera: Pteromalidae), with emerged fly adults smaller when the pupa survived parasitism. Similarly, Carton and David (1983) showed that *D. melanogaster* individuals which successfully encapsulate parasitoids, are smaller and have reduced fertility. On the other hand, when they were interested by the costs of constitutive defences in *D. melanogaster* using selection experiments for resistance, Kraaijeveld and Godfray (1997) found that these costs were only visible when the fly larvae were subject to high food competition, but no trade-offs with life-history traits (size, developmental time, or asymmetry) were observed when flies were reared with excess larval food.

In this paper, we outline laboratory experiments to address questions concerning the magnitude of direct and indirect defence costs, and the potential trade-offs that may impact on other life-history components of fitness in parasitised and unparasitised hosts. The underlying mechanisms of host defence strategies and their consequent trade-offs are vital to the understanding of the evolution of adaptations to escape parasitism in the short term (resistance to pathogens). An understanding of these trade-offs becomes particularly important when a pathogen is identified as a potential biological control agent (Boots & Begon, 1993).

The host is the Indian meal moth, *Plodia interpunctella* (Hübner) (Lepidoptera, Pyralidae), a common and widespread pest that feeds on stored food products. *Plodia* caterpillars are considered to be one of the most important stored product pests in Europe and the U.S.A. The solitary endoparasitoid, *Venturia canescens* (Gravenhorst) (Hymenoptera, Ichneumonidae), is a parasitoid which attacks hosts and continues to feed and grow during the course of parasitism (koinobiont). Consequently it develops in *Plodia* larvae until the host reaches the pupal instar. Despite the agricultural importance of stored grain pests, few studies have empirically investigated the evolution of resistance using selection experiments in these systems. Our primary research objective in the present paper is to address this knowledge gap by investigating the evolutionary mechanisms of a stored grain pest, *Plodia interpunctella*, and its parasitoid, *Venturia canescens*, and by quantifying the cost of resistance in the offspring of the generation following an immune challenge in the previous generation.

Materials and methods

Insect cultures were maintained in a sterile laboratory in the University of Georgia's Odum School of Ecology. Two populations of the Indian meal moth, *P. interpunctella* (Gainesville – Alachua, Florida, U.S.A. and Leeds, U.K.), were maintained in the laboratory. The two populations of *P. interpunctella* were reared in cylindrical, transparent Nalgene containers ($h = 130$ mm, $Ø = 115$ mm) in temperature and light controlled incubators [Percival Intellus® environmental controller; 28 °C, optimal temperature for *P. interpunctella* growth and reproduction (Fields, 1992), 60 ± 5% RH; LD 13:11 h] (Sait *et al*., 1995). Insect cultures were prepared weekly by adding ~100 third-instar larvae (L_3) obtained from week-old population stocks to 40 g of fresh food (mixture 800 g finely milled bran, 60 g dried powdered brewers' yeast, 200 ml glycerol, 200 ml clear honey, 6 g sorbic acid, 6 g methyl paraben).

Cultures of an asexual strain of *V. canescens* (USDA, San Diego, California), which reproduces by thelytokous parthenogenesis, were reared in the same temperature and light controlled incubators in plastic, transparent containers (150 × 90 × 60 mm). New cultures were prepared weekly by adding 15 wasps to stock boxes containing 50–70 third-instar *P. interpunctella* larvae. Wasps were fed a solution of honey and distilled water.

Wasp stock populations were reared on Floridian caterpillars, while wasps used in selection experiments were reared with British caterpillars. This precaution was taken to prevent potential adaptation of wasp virulence to British stock populations. Thus, the observed difference will be due to adaptation for host resistance and not for parasitoid virulence.

Parasitism:

Ten third-instar larvae of the same age were placed in a Petri dish (55 × 15 mm) and allowed to settle for 1 h before being presented to 24-h old parasitoids. Parasitoid oviposition was confirmed by the insertion of the ovipositor into the host larva followed by a characteristic 'cocking' movement (Roger, 1972). Parasitoid wasps were changed after five parasitisations ($n = 6$ wasps used). Parasitised larvae were removed after oviposition and placed individually in Petri dishes (55 × 15 mm) and were fed *ad libitum*. To compare the life-history traits of parasitised *Plodia* larvae with healthy larvae, control lines were reared and manipulated under the same conditions as those parasitised by *Venturia*, without encountering a parasitoid wasp.

We tested whether there is a cost of 'induced' and 'constitutive' defence (resistance ability without immune challenge) by comparing a number of life-history traits in a parasitised line (F_0), the first generation of resistant hosts: offspring from the surviving parasitised (F_1), and control lines of *P. interpunctella*. Here we use the term 'resistant' for offspring from mated adults that survived previous parasitism. Life-history traits measured were larval growth and development time, and adult size. Moth adults that emerge from parasitised populations were considered to

have successfully encapsulated the parasitoid egg or larva (Harvey *et al*., 1994). Adults of *P. interpunctella* that emerged on the same day were randomly paired and mated in a mating arena for both parasitised and unparasitised lines. Moth adult sizes were measured post-mortem. This study concerned the F_0 (parasitised and unparasitised populations) and F_1 (F_1 larvae were obtained from adults surviving parasitism) generations. Indeed, after these two previous generations, it was impossible to obtain enough males and females of F_1 that emerged the same day to obtain F_2. We followed the growth and developmental time of 87, 29, 60, 16 individuals for 'control F_0', 'parasitised F_0', 'control F_1', and 'resistant F_1' respectively.

Biological and morphometric measurements:

All morphometric measurements (mean ± SD) were digitised using a stereoscopic microscope (SMZ645 Nikon®) interfaced with a 1000 Motic® digital camera and Motic® Image Plus (version 2.0ML) image analysis software. The following measurements were made: *Growth*: The width of the larval pronotum (μm) was used as an index of the larval growth for both *P. interpunctella* treatments, as previously used by Nakamura (2002) and Lopez-Rodriguez and Tierno de Figueroa (2006) in other Lepidoptera. *Adult size*: The length of the rear femur (μm) of the dead adults taken from the experimental samples was used as an index of adult size. In *Plodia*, it is correlated with female ovary weight and male testis size – a proxy for reproductive fitness (McVean *et al*., 2002). Adult size is positively correlated with fecundity in *P. interpunctella* (Mbata, 1985). *Developmental time*: The width of the larval cephalic capsule is an indication of the larval instar in *P. interpunctella* (Harvey *et al*., 1994). As described in Table 1, daily measures of cephalic width were used to track changes in instar.

Statistical methods:

One way repeated measures analysis of variance (ANOVAr) followed by a HSD Tukey post-hoc comparison method were performed to evaluate whether the size and developmental time (dependent variables) changed over larval instars (repeated factor) between treatments (Statistica® 6.0 package). Due to the low rate of parasitoid encapsulation, the number of survival pupae and adults did not allow us to use ANOVAr analysis to compare those host instars. Generalised Linear Models (GLMs) were used to compare the pupal and adult developmental time, and adult size (R® software). χ^2 were performed to compare the number of adults that successfully encapsulated the parasitoid according to their sexes (Statistica® 6.0 package). $P < 0.05$ was considered significant.

Table 1. Cephalic measures to determine larval stage.

Larval stage	Cephalic width (μm)
First	0–175.5
Second	214–303
Third	349–460
Fourth	482–782
Fifth	794–1017

Results

Effect of parasitism on host developmental time (F_0)

Host larvae parasitised at the third larval instar significantly delayed their developmental time during the third and fourth larval instars ($P < 0.001$) compared with unparasitised populations (control). No difference was recorded for the fifth larval instar (L_5), pupa and adult emergence ($P > 0.05$) (Fig. 1).

Effect of parasitism on host larval size and growth (F_0)

Parasitised larvae were significantly bigger in L_3 and L_4 after parasitism occurred ($P < 0.01$ and $P < 0.001$ respectively) and significantly smaller in L_5 ($P < 0.001$) (Table 2). This difference in L_3 may be due to the parasitoid choice for the biggest L_3 host larvae, increasing the parasitoid success rate (see Sait *et al*., 1995).

Effect of parasitism on host adult size (F_0)

There was no difference in adult size between both sexes in control (1398.50 ± 64.42 and 1366.32 ± 187.85 μm, for female and male respectively; $P > 0.05$) and parasitised populations (1248.35 ± 264.81 and 1136.12 ± 104.69 μm, for female and male respectively; $P > 0.05$). Adults that emerged after a successful encapsulation of the parasitoid were significantly smaller than control adults (1198.47 ± 211.97 and 1381.17 ± 142.97 respectively; $P < 0.01$) (Fig. 2). Parasitism triggers the same cost for both sexes in terms of size reduction.

Effect of constitutive defence on host developmental time (F_1)

The resistant population was derived from eggs laid by moths that successfully encapsulated the parasitoid in F_0. Larvae from the resistant population had a reduced development time of the second and third larval instar compared with the control ($P < 0.01$ and $P < 0.05$ for L_2 and L_3 respectively). The difference in developmental time between control and resistant larvae disappeared at the two last larval instars (fourth and fifth) ($P > 0.05$). The resistant population significantly increased pupal development compared with the control population (32.00 ± 3.96 days and 28.75 ± 3.48 days respectively; $P < 0.001$), but this difference disappeared in adult stage ($P > 0.05$) (Fig. 3).

Effect of constitutive defence on host growth (F_1)

There was no difference in size between control and resistant larvae from L_2 to L_5 ($P < 0.05$) (Table 2).

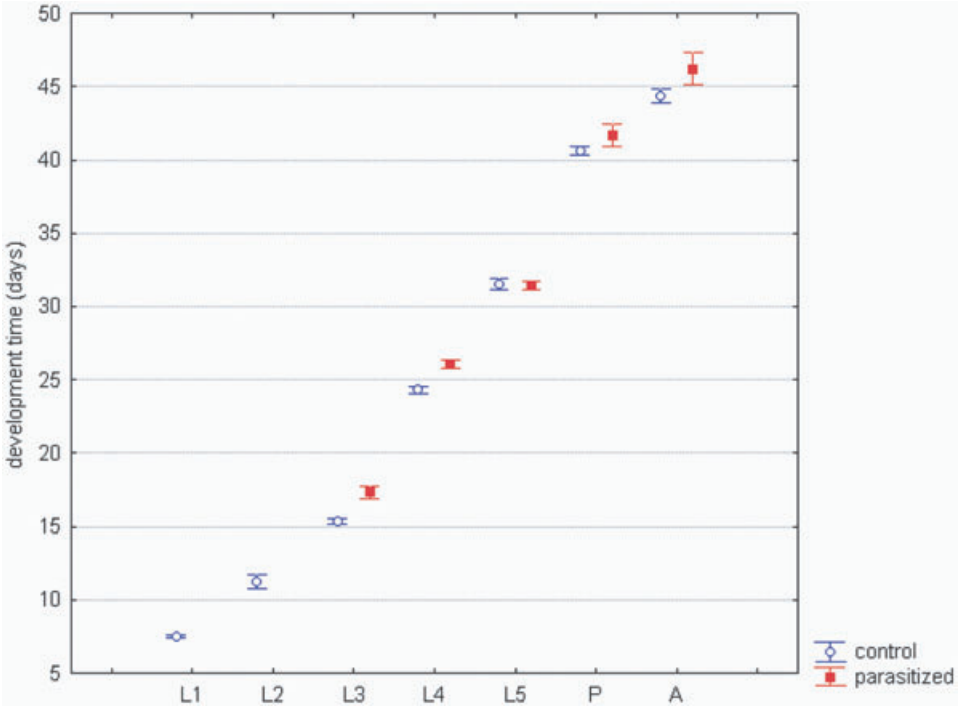

Fig. 1. Evolution of the developmental time (days) of *Plodia interpunctella* generation F_0 according to developmental instars and treatments (open symbols, control population; filled symbols, parasitised population). L_1–L_5, First to fifth larval instar; P, pupa; A, adult emergence. For the population F_0, all parasitised individuals were represented in the larval instars (L_1–L_5); only individuals surviving parasitism were represented in pupal (P) and adult (A) instars. Error bars represent standard errors (SE).

Table 2. Growth given by the pronotum width measurement (in micrometers) per stage of unparasitised and parasitised larvae.

	Growth (μm)			
F_0 instars	Unparasitised		Parasitised	
	Mean ± SD	*n*	Mean ± SD	*n*
L_3	433.25 ± 39.83	87	456.17 ± 36.79**	29
L_4	692.38 ± 102.92	120	698.71 ± 111.51***	190
L_5	1098.78 ± 100.7	90	1037 ± 79.20***	139
F_1 instars	Unparasitised		'Resistant'	
	Mean ± SD	*n*	Mean ± SD	*n*
L_2	287.6 ± 44.41	61	278.33 ± 31.71^{NS}	60
L_3	429.76 ± 57.20	55	437.6 ± 50.2^{NS}	56
L_4	718.31 ± 80.27	88	686.1 ± 88.5^{NS}	57
L_5	1070.75 ± 60.96	112	1067.8 ± 74.48^{NS}	69

Measurements were taken from the wasp oviposition occurring at the beginning of the third instar (L_3) in F_0. Results were analysed using repeated measures ANOVA followed by HSD Tukey *post hoc*. $^{*}P < 0.05$; $^{**}P < 0.01$; $^{***}P < 0.001$; NS: $P > 0.05$.

Effect of constitutive defence on host adult size (F_1)

There was no difference between both sexes in the size of adults emerged from control (1410.30 ± 187.36 and 1344.54 ± 210.08 μm for female and male respectively; $P > 0.05$) and resistant populations (1315.90 ± 85.42 and 1311.42 ± 204.83 μm for female and male respectively; $P > 0.05$), and there was no difference between both treatments ($P > 0.05$) (Fig. 2).

Discussion

Since the host represents the whole nutritional and physiological environment during immature development of the parasitoid (Colinet *et al.*, 2005), the parasitoid itself may need to alter or modify host physiology and/or development to optimise its own fitness. In such a control of the host physiology by the parasitoid, the host has to adapt its defence and must

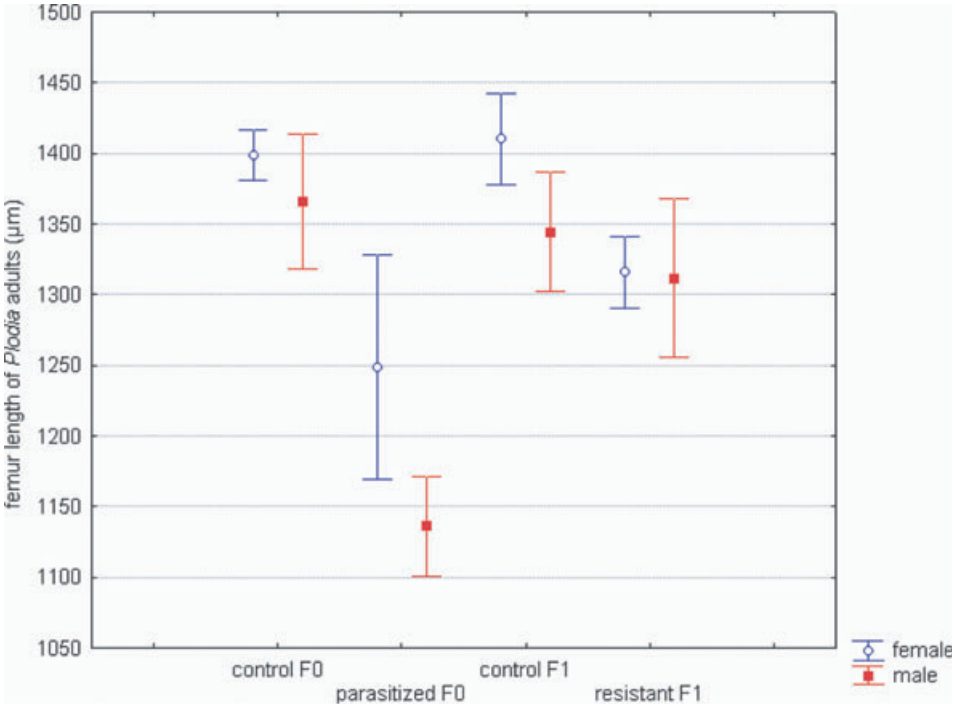

Fig. 2. Femur length of *Plodia interpunctella* adults (mean ± SE in micrometers) according to the generations (F_0 and F_1) and treatments (control, parasitised, resistant populations). Open symbols, females; filled symbols, males.

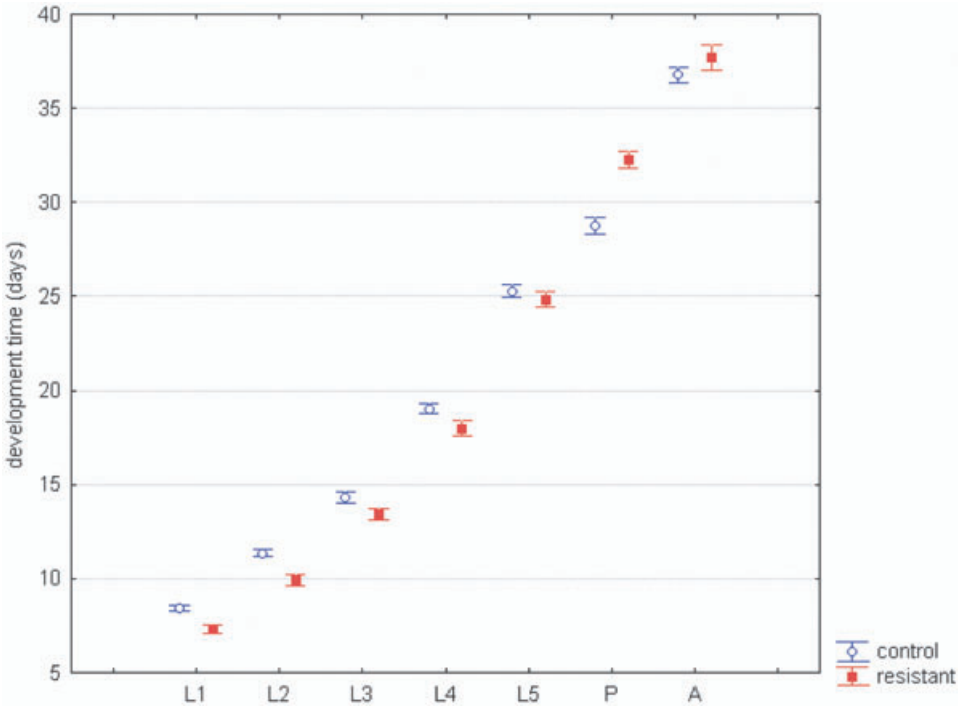

Fig. 3. Evolution of the developmental time (days) of *Plodia interpunctella* generation F_1 according to developmental instars and treatments (open symbols, control population; filled symbols, resistant population). L_1–L_5, First to fifth larval instar; P, pupa; A, adult emergence.

involve avoidance of parasitism either by escaping detection or through preventing oviposition (Kraaijeveld & Godfray, 2003). This present study examined the impact of *V. canescens* attacks on *P. interpunctella*'s fitness and following the host response to prevent further parasitism.

Impact of parasitism on the host life-history traits

Our results showed a large impact of parasitism by *V. canescens* on life-history traits in *P. interpunctella*. Larval size of the parasitised population increased after the wasp's oviposition of the third and fourth larval instars, and then the growth rate decreased when parasitised individuals significantly reached a smaller size in the last larval instar (L_5).

The decrease of the growth rate observed between L_3 and L_5 parasitised instars in *P. interpunctella*, is commonly found in other host–parasitoid systems (Thompson, 1982; Slansky, 1986) where growth rates are more severely reduced in later host instars.

Although many larval endoparasitoids adjust their development to certain host endocrine events (Beckage, 1985) following a 'conformer' strategy (synchronisation of the parasitoid larval moults, destructive feeding, and emergence on the host larval development) (Lawrence, 1986; Harvey, 1996), the degree of host growth reduction between different instars of *P. interpunctella* is evidence that parasitism is also disrupting or directing selected host biochemical processes (Vinson & Barras, 1970; Iwantsch & Smilowitz, 1975; Vinson & Iwantsch, 1980; Harvey *et al*., 1994).

Slansky (1986) and Vinson (1988) argue that parasitoid-mediated changes in host development and physiology increase host quality by reducing constraints, and are therefore beneficial to parasitoid fitness (see Harvey *et al*., 1999). The amount of resources for parasitoid growth and development are not fixed and parasitoid development depends largely upon feeding rate and capacity for growth during the interaction (Godfray, 1994; Harvey & Thompson, 1995). Therefore, a redirection of host metabolic pathways to shuttle nutrients preferentially to the parasitoids may thus be involved (Alleyne & Beckage, 1997). Indeed, in several koinobiont species, parasitism (mainly Hymenoptera Braconidae, Eulophidae, and Ichneumonidae) induces biochemical alterations in the host's haemolymph by using active agents, including polydnaviruses (PDVs) (Pennacchio & Strand, 2006), venom (Nakamatsu & Tanaka, 2003), and teratocytes (Nakamatsu *et al*., 2002), which are injected with the parasitoid's egg into the host. During oviposition, *V. canescens* does not inject such PDVs, but injects other virus-like particles (VLPs) with the parasitoid's egg into its hosts (Schmidt *et al*., 2005). This suppresses the host's immunity by inducing host haemocyte apoptosis, mainly of the granulocytes (Suzuki & Tanaka, 2006), preventing encapsulation. The effects of such particles on host growth and development are not fully understood, but Harvey (1996) suggests that such VLPs may regulate host physiology more subtly, in addition to the host immunity suppression.

Parasitoid larvae may also release secretions that facilitate host regulation/alteration (Harvey, 1996). In Braconid and Eulophid parasitoid species, these alterations affect host proteogenesis, glucogenesis, and lipogenesis (Bischof & Ortel, 1996; Thompson, 2001; Jervis *et al*., 2008). Such metabolic changes may also occur in *P. interpunctella*, which would explain the reduced growth rate in parasitised larvae, but some of these alterations will also be the result of selective tissue feeding by the parasitoid larva.

Simultaneously to the reduced growth rate, our results clearly showed a delay in the developmental time of the third and fourth larval instars in parasitised populations of *P. interpunctella*, whereas no difference with control population was recorded in the fifth larval instar. Other host–parasitoid studies showed that koinobiont parasitoids may directly or indirectly affect development time of their hosts (Harvey, 1996; Harvey *et al*., 1996; Berstein *et al*., 2002). Additionally, Alleyne and Beckage (1997) showed that the L_4–L_5 moult in parasitised tobacco hornworm (*Manduca sexta*) larvae that were parasitised at the fourth instar by *Cotesia congregata*, was delayed by about 24 h. In our study, the significant average delay was about 48 h in both third and fourth larval instars, but this delay disappears in the final larval instar (L_5). Therefore, the point at which the parasitoid larva begins destructive feeding (host haemolymph and host tissue subsequently) strongly influences the final size of the adult parasitoid (which is highly correlated with its fertility–Harvey *et al*., 1994; Harvey & Thompson, 1995). When developing in small or suboptimal host instars, such as L_3 and L_4 larval instar in *P. interpunctella*, where there are insufficient resources to optimise parasitoid fitness, early destructive feeding may reduce the developmental time of the parasitoid, but also lead to a reduction in parasitoid adult size (Hemerik & Harvey, 1999). Alternatively, allowing the host to increase the larval development to compensate for the early parasitoid's feeding (haemolymph feeder) may reduce the host size losses and increase parasitoid size and fitness. Host developmental delay in *Plodia–Venturia* association seems to be a parasitoid-influenced impact on this host life-history trait to improve its own fitness. Such a pattern of development in *Venturia* suggests that the parasitoid also adopts a 'regulator' strategy (Harvey, 1996) by altering host development in order to increase its suitability for parasitoid development. Therefore, our results support the viewpoint of Vinson (1988) and Harvey (1996) that *V. canescens* may exhibit characteristics of both 'conformer' and 'regulator' strategies.

At emergence, *P. interpunctella* adults that successfully encapsulate the parasitoid larvae are smaller, and thus have a reduced fecundity (Mbata, 1985) compared to unparasitised individuals. These reductions in size and related fecundity showed the real cost to mounting of a successful immune response. This major encapsulation cost triggers a trade-off between host survival and fertility. It is interesting to note that Boots and Begon (1993, 1995) found similar results in *P. interpunctella* infected by its granulosis virus with a significant correlation between resistance to the virus and egg viability. For both cases, there is obviously a trade-off between immunity and other major components of the fitness such as fecundity, due to a resource re-allocation between these biological components.

Adaptive avoidance of parasitism by the host at the next generation

Our study also showed the adaptive response of *P. interpunctella* population (F_1) after parasitisation by *V. canescens*. We focused on the life-history trait adaptations of this new generation that leads to an avoidance of *V. canescens* parasitism.

Our results showed that the growth rate of the F_1 generation is not affected by the previous parasitism in F_0. On the contrary, our results showed a significant reduced developmental time for the 'resistant' population in L_2 and L_3 (reduction about 34 and 22 h, respectively; reduction about 36 h in L_4 without significance). *Venturia* wasps do not parasitise L_1 *P. interpunctella* instars. L_2 instars could be parasitised but represent a considerable cost to both host and parasitoid due to the host death rate after parasitism, while L_3 are very attractive for *Venturia* and provide a better chance for parasitism success (Sait *et al*., 1997).

These shortening larval periods of susceptible instars reduced exposure to parasitism. No significant difference was recorded between 'resistant' and control populations in L_5, the less successful instar for parasitism. Indeed, Harvey *et al*. (1994) showed that parasitoid survivorship was highest in L_3 and L_4 *P. interpunctella* hosts, with >90% successful emergence from these instars (85% in our study), but, on the other hand, these authors showed an increased parasitoid mortality to 16% in L_5 hosts. In *Drosophila*–parasitoid pairings, it has been shown that the total number of host haemocytes present is correlated with the ability to encapsulate parasitic invaders (Eslin & Prevost, 1996) and is known to increase during larval development, when haemolymph volume increases (Alleyne & Wiedenmann, 2001). Thus, the number of haemocytes used in the immune response against parasitism is correlated with the global quantity of haemolymph. These could explain why the most successful encapsulation rate occurs when the host is parasitised in the largest instar L_5, with enough haemocyte numbers per morphotype (morphotypes have different function in the encapsulation process) (Strand & Johnson, 1996). Consequently, later and bigger L_4 and L_5 represent the least susceptible larval instar, and the benefit of reducing parasitism in such a less-vulnerable stage may be reduced compared with the cost of such a reduced developmental time.

'Resistant' populations also showed a longer developmental time in the pupal instar; they spent on average 4 days more in the pupal instar than the control individuals. In *Plodia*–*Venturia* interaction, the koinobiont cannot parasitise the pupal stage of *Plodia*. An increase of the pupal duration may allow metabolic changes necessary to complete development without taking more risk of being parasitised. 'Resistant' adults emerged from these pupae had the same size, and then the same fertility as adults from control populations. This fact confirmed that reduced size and related fertility observed in survival of parasitised adults (F_0: encapsulation) was due to the cost of mounting a successful immune response, but also that the host adaptation in F_1 on their life-history traits to reduce and/or avoid parasitism risks (shortening of the susceptible instar and lengthening of the invulnerable host instars) has no visible cost in term of host fecundity.

Although there is an obvious impact of parasitism on *Plodia* populations due to the low encapsulation rate (due to the wasp-injected VLPs), surviving parasitism also shows important sublethal effects (growth rate, adult size related to fecundity) for host population due to the cost of encapsulation. This leads to important consequences for the dynamics of parasitised host populations. The most direct is that fecundity is reduced for survival individuals, which would reduce the host's innate capacity for population increase. However, the fact that 'resistant' offspring of the next generation reduced the developmental time of susceptible instars, is likely to decrease the probability that individuals will come into contact with parasitoids.

Natural enemies' interactions, as in the host–parasitoid system, affect the evolutionary trajectory of host populations. When the only alternative is the death of the host, the cost of mounting an immune response has to be paid. Such a cost is clearly visible on the host fecundity in *Plodia*–*Venturia* interaction. Adaptive answers have to be found in order to resist parasitism with a lower cost. We found that adaptations may occur on the development time to avoid the highest risk of parasitism without reducing the fecundity.

Acknowledgements

The authors would like to thank Dr Roche Benjamin (University of Georgia) for help with statistical analysis and the two anonymous reviewers for their useful comments and advice.

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Accepted 18 August 2009
First published online 20 October 2009