The coevolution of host resistance and parasitoid virulence

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SUMMARY

Host–parasitoid interactions are abundant in nature and offer great scope for the study of coevolution. A particularly fertile area is the interaction between internal feeding parasitoids and their hosts. Hosts have evolved a variety of means of combating parasitoids, in particular cellular encapsulation, while parasitoids have evolved a wide range of counter-measures. Studies of the evolution of host resistance and parasitoid virulence are reviewed, with an emphasis on work involving *Drosophila* and its parasitoids. Genetic variation in both traits has been demonstrated using isofemale line and artificial selection techniques. Recent studies have investigated the fitness costs of maintaining the ability to resist parasitoids, the comparative fitness of flies that have successfully defended themselves against parasitoids, and the degree to which resistance and virulence act against one or more species of host or parasitoid. A number of studies have examined geographical patterns, and sought to look for local adaptation; or have compared the traits across a range of species. Finally, the physiological and genetic basis of change in resistance and virulence is being investigated. While concentrating on *Drosophila*, the limited amount of work on different systems is reviewed, and other possible areas of coevolution in host–parasitoid interactions are briefly discussed.

Keywords: Resistance, virulence, host, parasitoid, genetics, *Drosophila*.

INTRODUCTION

Parasitoids are insects, the vast majority wasps and flies (Hymenoptera and Diptera), that lay their eggs on or in the bodies of other insects, their hosts. The eggs hatch, and the parasitoid larvae develop, either singly or gregariously, their feeding eventually leads to the death of the host. Parasitoids thus occupy a somewhat intermediate position between predators and parasitoids; like predators they eventually kill their prey, like parasites they typically require just one host to complete development. Parasitoids are abundant members of nearly all terrestrial ecological communities, and there are probably somewhere between one half and two million species on earth (Godfray, 1994). Because they are capable of causing heavy host mortality, they have frequently been used as natural enemies of pests in biological control (Murdoch & Briggs, 1996; Mills & Getz, 1996). As a consequence, population biologists have intensively studied parasitoid population dynamics, and they have become a model system for investigating general questions of resource-consumer dynamics (Hassell, 1978, 1998; Godfray & Müller, 1998). The need to improve biological control was also responsible for intensive research on parasitoid behaviour, initially concentrating on studies of the mechanisms of host location and acceptance, but in more recent years on the evolution of reproductive strategies (e.g. Vinson & Iwantsch, 1980; Waage, 1986; King, 1987; van Alphen & Visser, 1990; Godfray, 1994, van Alphen & Jervis, 1995). Indeed, research on parasitoid wasps provides some of the most convincing evidence for the utility of the behavioural ecological approach to understanding behaviour, and here again parasitoids have become a model system for investigating questions of much broader relevance (Godfray, 1994).

A further area in which research on parasitoids has the potential for making important contributions is the study of coevolution or reciprocal adaptation. However, while many authors have acknowledged the importance of hosts and parasitoids as selective agents acting on each other, there has until recently been relatively little systematic investigation of this problem. Our aims here are to review ideas and experiments on host–parasitoid coevolution. We concentrate in particular on endoparasitoids which live internally in the host and typically spend some time in a quiescent stage as the host grows and increases in size. These parasitoids, like true parasites, have to resist the defence mechanisms of the living host and studies of the coevolution of resistance and virulence may be valuable in understanding related questions in parasites and pathogens. Much of this paper concerns *Drosophila* and its parasitoids, the system that has been most intensively investigated, and which we believe offers some of the most exciting future prospects.

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Resistance and virulence

Parasitoid biologists classify their study organisms as idiobiont or koinobiont (Askew & Shaw, 1986). Idiobiont parasitoids kill or permanently paralyse their hosts at oviposition so that their larvae develop on an inert unit of food. In contrast, koinobiont parasitoids allow their host to grow in size after parasitism, at the most only causing temporary paralysis. When the host is large enough, the parasitoid resumes development resulting in the host's death. The advantages of being a koinobiont are that the parasitoid can attack hosts that are too small at the time of oviposition to support development of the larvae, and that the host continues behaving normally, avoiding predators and other mortality factors. The disadvantages of this strategy are that the still-living host can mount a defensive response against the parasitoid.

Hosts can mount humoral or cellular responses against a parasitoid. Humoral responses have been little studied (but see Nappi *et al.* 1995), but may be important in groups such as aphids (Henter & Via, 1995). The cellular response is much better understood at physiological, and increasingly molecular, levels (Lackie, 1988; Ratcliffe, 1993; Strand & Pech, 1995; Gillespie, Kanost & Trenczek, 1997). Certain cells in the haemocoel recognize a parasitoid as foreign and adhere to the egg or larva and rupture. This triggers further cells to change morphology and aggregate to the parasitoid, forming a multi-layer capsule (encapsulation). The details differ amongst host taxa, but what normally happens is the capsule melanizes and hardens, killing the parasitoid by asphyxiation or through the release of necrotizing substances. Encapsulation is a general defensive response, displayed by all arthropods and related invertebrates (Brehélin, 1986). Acquired immunity of the vertebrate type is not found in insects.

Parasitoids can avoid host defences either passively or actively (Salt, 1970; Fleming, 1992; Beckage, Thompson & Federici, 1993; Rizki & Rizki, 1994; Strand & Pech, 1995; Carton & Nappi, 1997; Beckage, this volume). The former strategies include developing in tissues such as the brain where circulating haemocytes do not occur, or attacking host stages such as the egg or pupa which lack circulating haemocytes. Another strategy is molecular mimicry, the avoidance of being recognized as foreign. The surface of some parasitoid eggs is coated by molecules that mimic the insect basement membrane. But many parasitoids take more active steps to protect themselves. At oviposition, the parasitoid female may inject toxins and other substances that temporarily or permanently disable the host immune system. A number of parasitoids, particular wasps in the superfamily Ichneumonoidea, inject viruses into the host, whose DNA enters the cells of the immune system and is there expressed, causing a variety of effects including apoptosis. Viral DNA is stably integrated into the parasitoid genome, the virus particles themselves being only produced in the female reproductive system prior to oviposition; no viral replication occurs in the host. The parasitoid larva or its embryonic membranes may also actively secrete chemicals that attack the host immune system. In some species, cells of the embryonic membrane dissociate, enlarge and float free in the haemolymph (teratocytes), their dense endoplasmic reticulum confirming their secretory role. As described more fully by Beckage (this volume), toxins, viruses and teratocytes can be viewed as different delivery systems for immunosuppressant chemicals, and their relative roles typically change during the life of the immature parasitoid.

Questions about coevolution

Coevolution is used in evolutionary biology in at least two senses (Thompson 1982). The first sense reflects the fact that all organisms have trophic relationships with other organisms, and to a certain extent they must exert reciprocal evolutionary pressures. Such interactions are sometimes called diffuse coevolution and may be responsible for say, the fleet footedness of zebras, or the hard shells of many molluscs, which will have evolved as a result of predation risk from a number of different predator species. In contrast, coevolution may be tightly coupled and reflect the interaction between specific pairs of species. Examples of the latter are found particularly among mutualist pairs, and in specific host–parasite associations.

Coevolution can be inferred from the end product of evolutionary interaction or it can be demonstrated in action. As regards resistance and virulence, the evolution of parasitoid countermeasures has clearly been driven by host defences, but it is harder to argue that humoral and cellular defences are adaptations against parasitoids – they almost certainly evolved as a general defence against non-self challenges. These are several ways to demonstrate the coevolution of resistance and virulence in action. The first way is (a) to document additive genetic variation in host resistance and parasitoid virulence, and (b) to show that host resistance affects parasitoid fitness and parasitoid virulence affects host fitness. Genetic variation can be demonstrated using quantitative genetic techniques, or through artificial selection experiments. The second way to demonstrate coevolution in action is through controlled laboratory experiments. If genetic variation in virulence or resistance is demonstrated, a subsidiary question concerns how it is maintained. While increased resistance or virulence is clearly beneficial within the context of the host–parasitoid interaction, there may be trade-offs involving other components of fitness that might cause the optimum level of

either trait to be lower than the maximum achievable. Such arguments suggest yet further questions. Given that there are both positive and negative consequences of increased resistance or virulence, does the optimum level of each vary at different times or places, and can such patterns be detected in the field? What is the genetic architecture underlying the two traits? The evolution of virulence and resistance offers the prospects of insight into the genetics of adaptation. Finally, and a subject we shall not pursue here, do evolutionary changes in resistance and virulence influence host–parasitoid population dynamics (Godfray & Hassell, 1991).

We are still some way from complete answers to these questions for any system. We know most about host–parasitoid interactions involving *Drosophila* but we begin by considering the evidence from other insects.

INSECTS OTHER THAN *DROSOPHILA*

Examples from biological control

Given the number of times that new host–parasitoid associations have been created after biological control releases, one might think that these natural experiments would have provided valuable evidence on the evolution and coevolution of virulence and resistance. In fact, the evidence is meagre, and in some cases misleading, chiefly because biological control programmes seldom have the resources for detailed before-and-after biological studies.

A once frequently quoted example of the evolution of improved resistance concerns the larch sawfly, *Pristophora erichsonii*, a pest of Canadian forestry (Muldrew, 1953). An ichneumonid parasitoid, *Mesoleius tenthredinis*, from Europe was released in Manitoba in 1912–1913 and initially appeared to achieve good control. By the 1940s, however, the sawfly had increased again in economic importance and this was initially thought to be due to the evolution of improved defences against the parasitoid. Sawflies in Manitoba were able to avoid parasitism 96% of the time, while populations in British Columbia where the parasitoid had not spread escaped only 5% of the time. Careful reanalysis suggested a more prosaic explanation: the parasitoid was reared prior to release on European *P. erichsonii* stock which is a natural strong encapsulator. Releases were made by placing parasitized cocoons in the field and what seems to have occurred is that a European strain of the sawfly became established in Manitoba at the same time as the parasitoid, and that it was responsible for the subsequent pest outbreaks (Ives & Muldrew, 1981). Long-term coevolution may have been responsible for the differences between European and Canadian sawfly strains, but not the processes acting in the short term that had initially been suspected.

A second example from biological control concerns the evolution of parasitoid virulence. *Bathyplectes curculionis* is an ichneumonid parasitoid of the alfalfa weevil, *Hypera postica*, and was introduced from Europe into southern California to control this pest. After release, it was found also to attack the related weevil, *Hypera brunneipennis*, though survival was not very high with 40% encapsulation. 15 years after release, the same population was studied again and it was found that the parasitoid was much more efficient with only 5% encapsulation (van den Bosch, 1964; Salt & van den Bosch, 1967). Interestingly, this sharp increase in virulence against *H. brunneipennis* was accompanied by a slight decline in the parasitoids ability to avoid encapsulation on *H. postica*. Clearly, there are insufficient data to conclude definitely that parasitoid virulence has increased over time, but it is an intriguing possibility.

Pupal parasitoid of houseflies

Nasonia vitripennis is a small chalcidoid wasp that attacks the pupae of houseflies, *Musca domestica*, and other of the larger cyclorraphous Diptera. In a long series of experiments, Pimentel and colleagues (e.g. Pimentel, 1968; Pimentel *et al.* 1963, 1978; Zareh, Westoby & Pimentel, 1980) investigated coevolutionary interactions in laboratory cultures of hosts and parasitoids. The aims of the experiment were to investigate whether more stable population dynamics might arise through a coevolutionary interaction between host and parasitoid: 'ecological homeostasis brought about by genetic feedback'. Today such processes are thought unlikely to exist by most biologists, but Pimentel's experiments provide interesting insights into possible evolutionary interactions. The main change that occurred in the laboratory experiments was that host pupae became heavier and stronger and the fly spent less time in the vulnerable pupal stage. The more robust pupa is a clear defensive adaptation against the wasp, while the shorter pupal period accords with the general life history principle that development should be accelerated through life history stages where the risks of mortality are high. Adult flies tended to be smaller in experimental lines, and this may be a cost of improved resistance. We thus have evolution, but do we have coevolution? Pimentel and colleagues thought so, but we are less convinced, at least from their published work. True, parasitoids in the long-term evolutionary culture had reduced fecundity, but this may have been because they developed in hosts that evolved partial resistance (see also Weiss, McCrea & Abrahamson, 1989). Unless we have misinterpreted their papers, the crucial experiment of testing the parasitoids on 'control' hosts was not performed.

Pea aphid and its parasitoid

Outside *Drosophila*, the most detailed study of the

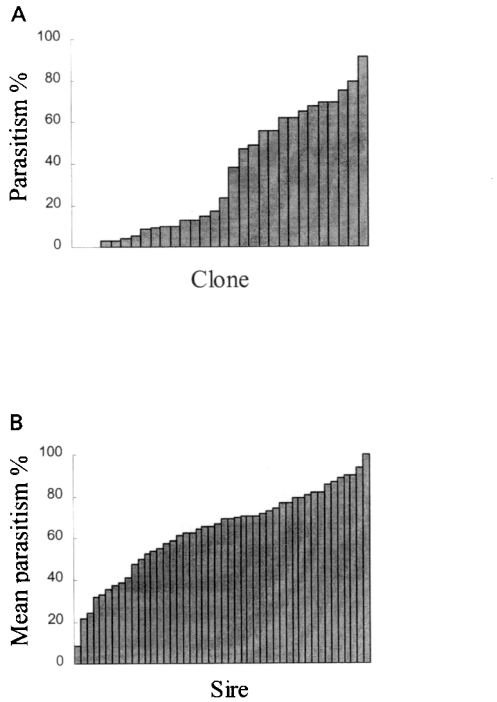

Fig. 1. An illustration of the genetic variation found (a) for resistance in the aphid *Acyrthosiphon pisum* and (b) for virulence in its parasitoid *Aphidius ervi* (Henter & Via, 1985; Henter, 1985). As described more fully in the text, the top figure shows the susceptibility to parasitism of different clones of aphids, while the bottom figure shows the genetic contribution of different males (sires) to parasitoid virulence against a particular clone of aphid.

genetic basis of resistance and virulence to date is Henter & Via's (1995) and Henter's (1995) work on the pea aphid, *Acyrthosiphon pisum*, and its braconid parasitoid, *Aphidius ervi*. Pea aphids are clonal throughout the summer which simplifies the assessment of the genetic variance in defensive ability. To measure resistance, Henter & Via (1995) allowed parasitoids from a laboratory culture to search and parasitize aphids in Petri dishes. Differences between clones may thus reflect behavioural or physiological differences, but supplementary studies suggested that differences in the level of humoral defences against parasitoid eggs was the most important aspect differentiating between clones. Fig. 1a illustrates the considerable amount of variation in parasitoid resistance observed among clones. The coefficient of clonal variance varied from 50 to 77% which suggests great potential to respond to selection pressures exerted by the wasp. Exactly how the clonal variance translates into additive genetic variance across the winter sexual generation is complicated because clonal variance includes in addition to additive genetic variance, dominance and epistatic components as well. Conceivably some of the differences may also be due to maternally inherited micro-organisms or other factors. However, the six or so clonal generations during the summer allow sufficient time for a significant response to parasitoid selection.

Knowing the clonal variation for resistance, and also the parasitism rates in the field, Henter & Via predicted that relatively resistant strains should increase in frequency over the season. However, this did not occur. Why, they were unable to say, but possibilities include trade-offs between resistance and other components of fitness, or variation in the response of aphid clones to different parasitoid genotypes (recall all parasitism assays were performed with a single strain of parasitoid). A possible trade-off involved parasitoid resistance and resistance to entomopathic fungi, a common source of aphid mortality in the field.

Henter & Via were unable to explore interactions between aphid and wasp genotypes but Henter (1995) was able to make an assessment of the genetic variability in virulence in natural populations of *A. ervi*. Assessing genetic variance is harder in a sexual organism and a half-sib analysis was performed, with parasitoids allowed to attack a single clone of aphids. Again, significant amounts of genetic variation were found (Fig. 1b), and the coefficient of additive genetic variation was 26%. As Henter points out, this is large for a trait that has clear survival value; the equivalent figure for fecundity in *Drosophila*, for example, is 12%. Dominance and environmental variance were also significant, the latter including maternal effects which might perhaps be influenced by maternally-inherited micro-organisms.

DROSOPHILA

Natural history

Before turning to work on *Drosophila*, we digress briefly to introduce the parasitoids of *Drosophila*. All the work we refer to involves fruit and sap flux feeding species of *Drosophila*, especially *D. melanogaster* which has been spread throughout the world by man, but also the related *D. simulans* and the common north European species *D. subobscura*. For a general view of the parasitoid fauna attacking *Drosophila*, including species living in decaying vegetation and fungi, see Vet & van Alphen (1985) and Carton *et al.* (1986).

Two guilds of parasitoids attack *Drosophila*; those attacking the larval and pupal stages. The larval parasitoids are koinobiont (see above), remaining as first instar larvae until the host pupates when they resume development, quickly destroy the host, and then pupate themselves within the host puparium. Coevolutionary studies on larval parasitoids have chiefly concentrated on two unrelated genera of wasps. *Asobara* (Ichneumonoidea: Braconidae) contains about ten described species worldwide with at least ten more awaiting description. The species most frequently studied in laboratories is *A. tabida* which is common in Europe where in the

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north it most frequently attacks *D. subobscura* while in the south *D. melanogaster* is the main host. Other species include *A. citri* from tropical Africa whose adults have the unusual behaviour of fighting to defend patches from conspecifics. Rather little is known about how *Asobara* counters host defences (Kraaijeveld & van Alphen, 1994; Monconduit & Prévost, 1994; Eslin *et al.* 1996). Toxins are injected at oviposition and by analogy with related species it is possible that further substances are secreted by the embryonic membranes, but injected viruses or virus-like particles are not thought to be involved (Eslin *et al.* 1996). As will be discussed below, the eggs of different strains of *A. tabida* 'stick' to different degrees to host tissue, and it is likely that they gain some protection from being hidden in fat bodies and other tissue away from circulating haemocytes (Kraaijeveld & van Alphen, 1994).

The second major genus of larval parasitoids is *Leptopilina* (Cynipoidea: Eucoilidae) (Nordlander, 1980), part of a superfamily that contains not only parasitoids but also the gall wasps. The two most important species in studies of host-parasitoid coevolution are *L. boulardi* and *L. heterotoma* (called *Pseudeucoila bochei* in the older literature). The first is a specialist in *D. melanogaster* and its close relatives, the second has been recorded from a much wider range of species. The physiological and biochemical interactions between *Leptopilina* and *Drosophila* have been quite intensively studied (reviewed by Carton & Nappi, 1997). In *Drosophila*, capsules are formed by a type of haemocyte called the lamellocyte and a substance injected by the wasp that caused the death of these cells was identified and called lamellolysin (Rizki & Rizki, 1984). More recently, lamellolysin has been recognized to be a virus or virus-like particle, though unrelated to the well-studied viruses found in the Ichneumonoidea (Rizki & Rizki, 1990; Dupas *et al.* 1996; Russo *et al.* 1996). It is not yet clear whether DNA is injected and expressed in the host, or whether the protein coat alone is responsible for lamellolysin's action. Virus-like particles, lacking DNA, have been shown to have biological activity in other host-parasitoid systems (Schmidt & Schuchmann-Feddersen, 1989; Asgari & Schmidt, 1994). What is safe to assume is that *Asobara* and *Leptopilina* have independently evolved means to overcome host defences. That the host or parasitoid wins is not the only possible outcome of parasitism. In some interactions, especially 'unnatural' combinations of hosts and parasitoids in the laboratory, parasitism results in the death of both partners (e.g. Carton, 1984; Boulétreau, 1986; Eslin & Prévost, 1998). Presumably parasitism, or the host's reaction to parasitism, fatally disrupts the host's physiology, but the mechanism is not understood.

The second guild of *Drosophila* parasitoids attack the pupal stage when the host is no longer protected by a cellular defence mechanism. The majority of pupal parasitoids belong to the family Diapriidae (Proctotrupoidea) or Pteromalidae (Chalcidoidea) with the pteromalid *Pachycrepoideus vindemiae* (also referred to as *vindemmiae* and *dubius*) being the species most frequently used in laboratory experiments. Pteromalid pupal parasitoids lay their eggs inside the puparium (the hardened skin of the final larval instar) but external to the developing pupae. They are thus ectoparasitoids and hence less susceptible to host resistance than endoparasitoids. In consequence, they have not evolved specific adaptations to individual host species and they tend to have a wide host range, sometimes encompassing many families of Diptera. Diapriid pupal parasitoids feed internally and tend to be more host specific, though as host pupae are less protected than larvae they have a wider host range than larval parasitoids.

Genetic variation: isofemale lines

A powerful way of demonstrating the presence of genetic variation for a trait is through isofemale lines in which a laboratory strain is bred from a single mated individual (Parsons, 1980). For hymenopteran parasitoids, which are haplodiploid, a strain can be derived from a single female if it is possible to keep her alive long enough to mate with a son. An array of lines are scored for a trait under identical laboratory conditions, and the presence of significant between-line variation is attributed to genetic differences. However, the problem with this technique is that the inevitable inbreeding required to establish the isofemale lines means that between-line differences are caused by a mixture of additive, dominance and epistatic components, and it is not possible to predict quantitatively how the population will respond to directional selection on the trait (as Henter & Via (1995) were able to do in the aphid study discussed above). Technically, isofemale lines can be used to establish an estimate of heritability that is intermediate between broad sense (h^2_B) and narrow sense (h^2_N) heritabilities (Hoffmann & Parsons, 1988).

Isofemale lines have been used extensively by French workers to investigate within and between population differences in resistance and virulence. Boulétreau & Fouillet (1982), Carton & Boulétreau (1985), Wajnberg, Prévost & Boulétreau (1985), Carton, Cary & Nappi (1989) and Carton & Nappi (1991, 1992) found that populations of *D. melanogaster* and *D. simulans* from France, Tunisia and the Congo all contained within-population genetic variation for resistance against *L. boulardi*. Broad sense heritability could be as high as 0·43 (Carton & Nappi, 1991). In one of the few studies concerning *Drosophila* pupal parasitoids, Delpuech, Frey & Carton (1994) found significant genetic variation amongst isofemale lines of *D. melanogaster* in survival against the pupal parasitoid *P. vindemiae*.

Fewer studies have looked for genetic variation in

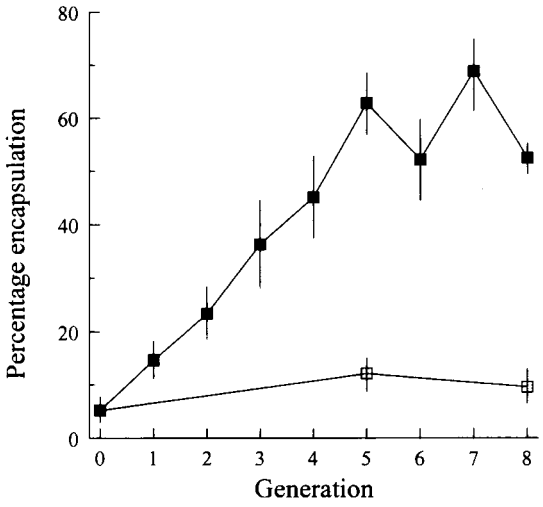

Fig. 2. Artificial selection for increased resistance in *Drosophila melanogaster*. The percentage of encapsulation in selected (black) and control (white) lines. Means and standard errors of four lines. For logistic reasons control encapsulation ability was assayed less frequently than that of the selected lines (from Kraaijeveld & Godfray, 1997).

parasitoid virulence but Carton *et al.* (1989) and Carton & Nappi (1991) found substantial differences in the ability of north African *L. boulardi* to avoid encapsulation in *D. simulans*, with estimated minimum broad sense heritabilities of the order of 0·3–0·5.

Genetic variation : selection experiments

Artificial selection experiments are valuable means of demonstrating the presence of genetic variation in natural populations. The first attempts to select for increased resistance to parasitoids in *Drosophila* were by Schlegel-Oprecht (1953), Hadorn & Walker (1960) and Walker (1961) working with *D. melanogaster* and *L. heterotoma* (then called *Pseudeucoila bochei*). They found a marked increase in the concentration of dispersed, melanised haemocytes, which they equated with encapsulation ability. However, Carton & Kitano (1981) have pointed out difficulties with this measure, and have suggested that the actual increase in survival of parasitism might be as low as 2%. The lack of a clear increase in encapsulation frequency may also have been affected by low effective population size and weak selection (only a fraction of flies each generation were parasitised). Walker (1962) attempted to select for increased virulence in field strains of *L. heterotoma* but failed to get a response. For both hosts and parasitoids, possible responses to selection were found when the initial base stock was composed of a mixture of strains from different geographical regions which differed in resistance or virulence, but in the absence of statistical analysis the results are hard to interpret. Kraaijeveld (1994) selected for higher resistance in a single line of *D. melanogaster* derived from wild Dutch flies. The fraction of hosts encapsulating parasitoid eggs rose from 6% in the base stock to 49% after five generations and 66% after ten.

Most of the selection experiments discussed so far are unreplicated and/or have used laboratory stocks of flies that may have lost some of their ability to defend themselves against parasitoids. Two studies have tried to avoid these problems. Hughes & Sokolowski (1996) set up six replicate lines of *D. melanogaster* that were composed of flies largely with a wildtype genetic background, except that they were a mixture of individuals homozygous for the rover and sitter behavioural polymorphism (see below). Three of the lines were protected from parasitism while the other lines were subject to a modest risk of parasitism (c. 6–9% in each generation). The parasitoid used was *A. tabida*. Wasps emerging from the experiments were used in future rounds of selection, but supplemented with insects from stock cultures. There is thus some possibility of co-evolution. After 19 generations, flies in the lines exposed to parasitoids had evolved a greater ability to encapsulate the wasp. In a test assay, wasps successfully survived in 43% of control hosts but only 31% of treatment hosts.

Kraaijeveld & Godfray (1997) initiated a large outbred culture of *D. melanogaster* from wild Dutch flies. The aim was to study selection for increased resistance against parasitoids that themselves were prevented from evolving (and also to study trade-offs, see next section). Eight separate lines were initiated from the base stock, four of which were subject to selection by parasitoids, the other four acting as controls. The parasitoids used in the experiment were *A. tabida* from a laboratory culture maintained on *D. subobscura*. *A. tabida* invariably destroys *D. subobscura*, and by keeping the parasitoid on a different host species, adaptation to *D. melanogaster* was prevented. In the selection lines, larval flies were exposed to parasitoids and allowed to develop until pupation. At this stage the capsule in flies that had successfully defended themselves is visible through the puparial wall, allowing them to be separated and used to initiate the next generation. Populations in each line were kept above 100 to minimize the effects of inbreeding.

In the base stock, only about 5% of flies were able to encapsulate parasitoid eggs. Over about five generations, this percentage rose rapidly and consistently across the selection lines to a value of about 60%, where it appeared to asymptote (Fig. 2). The wild population from which the experimental lines were established thus appears to have a considerable potential for increased resistance, though is seemingly not able to become totally resistant (at least against this relatively virulent strain of parasitoid), even when the parasitoid itself does not evolve.

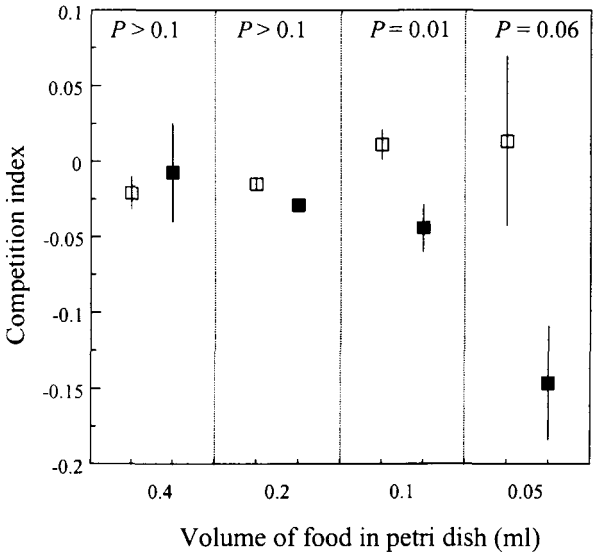

Fig. 3. The costs of increased resistance in *Drosophila melanogaster*. Relative larval competitive ability of selected (black) and control (white) lines at different levels of resources (from Kraaijeveld & Godfray, 1997).

Using Falconer's (1996) formula for calculating the heritability of a threshold character we obtained a value of 0·26, though this assumes a polygenic trait which we think is probably unlikely. We (the Imperial College group) are currently selecting for increased virulence in *A. tabida*, and for increased resistance in *D. melanogaster* against a different parasitoid, *L. boulardi* (M. Fellowes unpublished).

The selection experiments described so far have largely concentrated on one member of the host–parasitoid pair, normally the host. A clear next step is to compare what happens when resistance or virulence are individually selected, with what happens when the host and parasitoid are allowed to coevolve. This has yet to be done, though an interesting experiment by Boulétreau (1986) hints at what might occur. He maintained two population cages of *D. melanogaster* (a Tunisian strain) and *L. boulardi* for 70 generations and then tested for resistance and virulence in a laboratory assay where single parasitoids were allowed to attack groups of 100 larvae. There are only two lines and just preliminary analyses are given, but it appears that flies evolved the ability to avoid parasitoid attack (perhaps through selection on the type of behavioural polymorphism discussed below), and that both higher virulence and higher resistance evolved in the experimental lines (though definitive conclusions are not possible from the data in the paper).

Trade-offs

Why should there be so much genetic variance in resistance? A major possible reason is trade-offs between improved resistance and other components of fitness (including susceptibility to attack by other natural enemies which we discuss in a separate section below). The critical issue is not whether encapsulation itself is costly, if the alternative is death then costs are of little importance, but whether maintaining the machinery of defence just in case parasitism occurs has negative effects on other components of fitness. Trade-offs are central to many issues in life history theory and artificial selection experiments are an important means of their identification and quantification.

As described in the last section, Kraaijeveld & Godfray (1997) selected four lines of *D. melanogaster* for increased resistance, maintaining a further four lines as controls. They then measured a variety of demographic parameters for flies in each line, first under conditions of abundant resources, and then when the flies were subject to increasing levels of larval competition for food. When food was abundant, no differences in early fecundity, immature mortality, development time, adult size, fluctuating asymmetry or starvation resistance were found amongst selected and control lines. Note, in these types of experiments replicates are lines and hence the statistical power of tests is not very high (because $n = 4$ in each treatment). However, no strong but insignificant trends were observed. In the second set of experiments, larvae from each of the eight lines were placed in Petri dishes, with equal numbers of genetically marked 'tester' larvae, and supplied with a certain amount of larval food. The survival (competitive ability) and adult size of flies were then measured. The results for competitive ability are shown in Fig. 3 for four levels of larval food. When the larvae were supplied with 0·4 or 0·2 ml of food, no differences in larval survival were observed. However, when only 0·1 ml of food were available, flies from selected lines performed significantly ($p = 0{\cdot}011$) worse than control lines. At the most extreme level of resource restriction (0·05 ml), a similar but less strong effect was found ($p = 0{\cdot}061$), the reduced statistical significance probably being the result of much higher variability in survival under these very stringent conditions. There was a trend for selected flies to be smaller than control flies under strong competition, but the differences were not statistically significant. When selection was removed, the previously selected lines decreased in encapsulation ability to about 40%. Although flies under culture conditions on average experience lower intensities of competition than in the experiments where the trade-off was revealed, we suspect that the costs of increased resistance are responsible for the relaxation of the trait after the selection pressure was removed.

We conclude that increased resistance is not an unalloyed blessing for *D. melanogaster*. When resources are scarce, resistant larvae are at a competitive disadvantage. This difference appears when competition is severe, but no more severe than that likely to be experienced by *D. melanogaster* in fruit in the field. Carton & Kitano (1981) noted that there

was a negative correlation between resistance and competitive ability of three fly species in population cages (*D. melanogaster*, *D. simulans* and *D. yakuba*). To test such a hypothesis, more species are required, and phylogenetic confounding effects must be controlled, but it is consistent with the within-species data from *D. melanogaster*.

Costs of successful resistance

Higher levels of resistance evolve if the costs outweigh the benefits, and an important factor influencing the magnitude of the benefits is the fitness of flies that have successfully encapsulated parasitoids. If the benefits are greater than the costs, then the speed with which a resistance gene spreads will also be influenced by the relative fitness of flies with capsules.

Carton & David (1983) studied the fitness of *D. melanogaster* that had successfully encapsulated *L. boulardi*. Female flies with capsules tended to be smaller, and to produce fewer eggs. Fellowes, Kraaijeveld & Godfray (1999) also looked at *D. melanogaster*, but this time after attack by *A. tabida*. Again female flies containing capsules were smaller and produced significantly fewer eggs than controls. Male flies had smaller thoraxes, though not smaller wing lengths. Capsule bearing males allowed repeated copulations with females do not show a reduction in their number of offspring, but those allowed to copulate only once did. Finally, there is also a reduction in the thickness of the puparial wall (Fellowes *et al.* 1998).

What causes the reduced fitness of flies with capsules? We do not yet know but it is likely to be either a direct negative effect of parasitism, or a consequence of the redirection of resources away from growth towards capsule formation. In the second case, the limiting resource might be energy, or it might be the chemicals required for encapsulation. Successful capsule formation requires the deposition of melanin, and it is possible that as a consequence less melanin is laid down in the puparial wall. Interestingly, in isofemale line studies, parasitoid survival increases under conditions of larval crowding (Wajnberg *et al.* 1985), perhaps because the host has fewer resources to devote to defence.

Because pupae with capsules produce smaller adult flies, Fellowes *et al.* (1998) predicted that they might be relatively less preferred by the pupal parasitoid *P. vindemiae* in comparison with unparasitised pupae. This species is known to be able to discriminate between hosts of different quality (van Alphen & Thunnissen, 1983). In fact, the parasitoid did not distinguish between old pupae with and without capsules, while actually showed a preference for young pupae with a capsule. The reason for this preference is not yet understood, though one possibility is that *P. vindemiae* favour parasitized pupae as it takes less time to drill through their thinner puparial walls.

A final and counter-intuitive consequence of encapsulation was suggested by Carton (1987; referring to unpublished experimental work by Gouyon *et al.*). They had preliminary data that females preferred to mate with males bearing a capsule, visible through the abdominal wall. Perhaps females choose males that were able to demonstrate their ability to survive parasitism and which could thus pass on these good genes to their offspring (though they also demonstrate the fact that they were discovered by searching parasitoids which may be due to 'bad genes'). This intriguing notion chimed with modern ideas about the role of parasites in sexual selection (Hamilton & Zuk, 1982). Unfortunately, more recent attempts to demonstrate a preference for males carrying capsules have failed to show this effect (Kraaijeveld, Emmett & Godfray, 1997).

Cross resistance and cross virulence

Is improved resistance against one species of parasitoid associated with better defence against other species? Similarly, is parasitoid virulence against one host species correlated with success against others?

The little evidence available to date largely addresses the first question. Boulétreau & Wajnberg (1986) and Delpuech *et al.* (1994) found a correlation among isofemale lines in their ability to defend themselves against *L. boulardi* and *L. heterotoma*. However, there was no correlation (positive or negative) between resistance against the larval parasitoid *Leptopilina* species, and the pupal parasitoid *P. vindemiae*. Below, we discuss the isolation of a gene that confers resistance to *D. melanogaster* against *L. boulardi*. Susceptible flies have normal resistance against *Asobara tabida* (Vass, Nappi & Carton, 1993). A final piece of evidence is that in a Europe-wide geographical survey (discussed further below), there was no correlation in the resistance of *D. melanogaster* against *A. tabida* and *L. boulardi* (Kraaijeveld & van Alphen, 1995). Thus the very limited data available suggest cross-resistance within, but not between, parasitoid genera.

Parasitoids and Drosophila *behavioural polymorphisms*

An interesting behavioural polymorphism is known in *Drosophila* that may influence susceptibility to parasitism (Sokolowski, 1980; Sokolowski *et al.* 1986; de Belle & Sokolowski, 1987; de Belle *et al.* 1989, 1993). 'Rover' larvae move actively through the medium while 'sitter' larvae are significantly less mobile. The polymorphism is due to a single gene on

chromosome 2 with rover fully dominant. Very recently, Osborne *et al.* (1997) have found, using molecular mapping techniques, that the gene involved codes for a cGMP-dependent protein kinase, and this is thus one of the very few behavioural polymorphisms to be characterized molecularly. Sokolowski suggested that the two morphs might be differentially susceptible to parasitoids that locate their hosts by vibrotaxis. This idea was supported by Sokolowski & Turlings (1987) who compared the susceptibility of rover larvae with a temperature sensitive paralytic mutant that behaves like a rover below 29 °C but is motionless above this temperature. *A. tabida*, which uses vibrotaxis to locate its hosts (Vet & van Alphen, 1985), did not distinguish between the two categories of larvae below 29 °C but parasitized significantly more rovers at higher temperatures. *L. heterotoma*, which locates hosts by probing the medium, did not distinguish between the two types of larvae at any temperature. Carton & Sokolowski (1992) studied the relative susceptibility of rovers and sitters to *L. boulardi* which also probes the medium and a further species of eucoilid *Ganaspis xanthopoda* (rarely used in the laboratory) which employs vibrotaxis. *L. boulardi* parasitized significantly more sitters, and *G. xanthopoda* more rovers. They also presented evidence that in population cages, the presence of *L. boulardi* led to a decrease in sitter frequencies, and suggested that rover/sitter frequencies in the wild may be influenced by the searching strategies of the local parasitoid community.

Though more easily located by *A. tabida*, rover larvae are more resistant to this parasitoid than sitters. We described above Hughes & Sokolowski's (1996) selection experiment in which they found an increase in encapsulation frequency in *D. melanogaster* lines subject to *A. tabida* parasitism. However, the frequency of the rover morph did not differ between control and treatment lines. The authors suggest that the advantages rovers enjoy through higher resistance are counterbalanced by their more frequent discovery by the parasitoid. Alternatively, as the rover/sitter gene and known genes for parasitoid resistance (see below) are linked hitchhiking may be involved.

Several other behavioural polymorphisms may affect susceptibility to parasitism. Carton & David (1985) studied 'digger' and 'non-digger' *D. melanogaster* larvae which tend to feed at different depths in the medium. They found greater parasitism (by *L. boulardi*) of non-digger morphs near the surface. Wong, Sokolowski & Kent (1985) found that some *D. melanogaster* larvae dig a tunnel prior to pupation, exposing only a small portion of the pupae (embedded) while others pupate on the surface with whole pupal cases exposed (non-embedded). The difference in pupation behaviour was genetically based, probably due to one or a few major genes. Carton & Sokolowski (1994) studied whether embedded or non-embedded flies were attacked more often by the pupal parasitoid *P. vindemiae*. The answer depended on the frequency of the two morphs. When embedded flies were rare, there was no difference in the rates of attack, but when embedded pupae were common they were more frequently parasitised. Possibly the wasp obtains protection from desiccation when developing in embedded pupae, though alternatively the wasp may just be responding to the relative abundance of pupae embedded in the medium and on the walls of the container.

Geographical patterns and local adaptation

The earliest studies of encapsulation in *Drosophila* established that populations from different geographic locations varied in their ability to survive parasitism (Oprecht & Hadorn, 1952; Schlegel-Oprecht, 1953; Walker, 1959; Hadorn & Walker, 1960; Hadorn & Grassmann, 1962). Boulétreau and colleagues (Boulétreau & Fouillet, 1982; Carton & Boulétreau, 1985; Wajnberg *et al.* 1985; Boulétreau, 1986) found that *D. melanogaster* from the Congo performed better against *L. boulardi* than flies from France which in turn did better than flies from Tunisia. Curiously, *L. boulardi* from Tunisia were particularly good at avoiding encapsulation. Carton & Nappi (1991) studied five *D. melanogaster* populations from three continents and found low encapsulation rates against sympatric *L. boulardi*, with the exception of a population from the Congo (Brazzaville). Only at this site does *L. boulardi* attack other hosts in addition to *D. melanogaster*, and hence it may have evolved to be less closely adapted to this species of fly.

The most important study of local adaptation is that of Carton (1984, see also Carton & Nappi, 1991) who investigated strains of *D. melanogaster* and *L. boulardi* from Gaudeloupe, southern France, Tunisia, Brazil & Italy. In comparing parasitoid performance, it is important to take into account host (and parasitoid) mortality that occurs at the time of oviposition or shortly after, and which can be quite substantial. Carton (1984) made allowances for this using a weighting scheme and statistical techniques about which we have some reservations. We have reanalysed the data, and our conclusions differ slightly from Carton (1984). The most striking result concerns mortality around parasitism. Strains of parasitoid from Gaudeloupe and Brazil caused substantially higher mortality in four allopatric host strains than in their sympatric host strain (two-tailed exact probability test: $p = 0{\cdot}0078$). However, given the host survived parasitism, the probability of the parasitoid successfully emerging did not differ significantly in sympatric and allopatric hosts. Reducing mortality at parasitism clearly benefits the

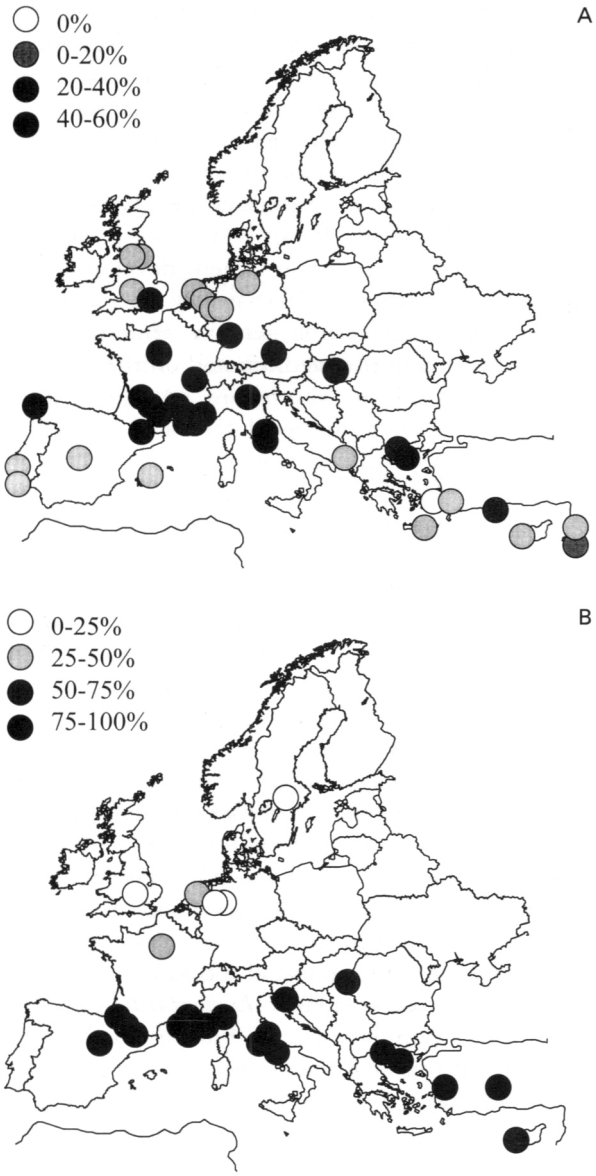

Fig. 4. (a) The percentage of *Drosophila melanogaster* larvae, from different geographical sites, that encapsulate a reference strain of *Asobara tabida*. (b) the percentage of *A. tabida*, from different geographical sites, that avoid encapsulation in a reference strain of *D. melanogaster* (from Kraaijeveld & van Alphen, 1994, 1995).

parasitoid, but also benefits the host which may survive to mount a successful immune response (we think it unlikely that *Drosophila* gain any inclusive fitness benefit from destroying a parasitoid through adaptive suicide). Thus a locally adaptive response may have evolved when it benefits both partners, but not when the beneficiary is just the host or the parasitoid. Carton (1984) also found reduced mortality at the time of oviposition when parasitoids (the Gaudeloupe strain) were allowed to attack hosts from areas where *L. boulardi* was absent. The exact significance of this is hard to assess because these hosts tend to come from more northerly latitudes and hence are probably subject to a variety of different selection pressures.

Kraaijeveld & van Alphen (1994) surveyed the virulence of *Asobara tabida* populations across Europe against a standard strain of *D. melanogaster* collected in Germany (Fig. 4a). A clear pattern results with less virulent strains in the north and more virulent strains in the south. The most likely explanation for this pattern is that in the north *A. tabida* is primarily a parasitoid of *D. subobscura* which appears to be completely unable to defend itself against this wasp, or indeed any other (Kraaijeveld & van der Wel, 1994). In the south, *D. subobscura* is rare or absent and *D. melanogaster* is the main host. *A tabida* may thus have evolved to be more virulent against *D. melanogaster* where this species is the primary host. Differences in wasp populations from the north and south of Europe appeared to be associated with the 'stickiness' of their eggs: the degree to which they embed themselves in host tissue away from circulating haemocytes.

Kraaijeveld & van Alphen (1995) also found a geographic pattern, though more complicated, in *D. melanogaster* resistance against *A. tabida* (Fig. 4b). In this case, fly populations were tested against a strain of wasp from southern France. Fly populations from central southern Europe had the highest resistance, with resistance being lower in north-western Europe, but also in the south-west and south-east. While the increased use of *D. melanogaster* by *A. tabida* in the south might explain selection for increased resistance in central southern Europe, the low resistance in the south-east and south-west lacks an explanation. Kraaijeveld & van Alphen (1995) also surveyed *D. melanogaster* populations for resistance against *L. boulardi*. Differences amongst populations were found, but they lacked any clear clinal patterns, and there was also no correlation between the ability of different populations to defend themselves against *A. tabida* or *L. boulardi*.

In interpreting these results, Kraaijeveld & van Alphen assumed there was no local adaptation between host and parasitoid. This assumption was based on preliminary observations that the *relative* performance of different parasitoid isolates appeared the same across host strains that differed in resistance, and similarly for the relative performance of different host isolates. Further evidence is provided by the replicates lines of *D. melanogaster* selected for improved resistance against *A. tabida* (Kraaijeveld & Godfray, 1997). Their performance on more and less virulent strains of *A. tabida* is exactly what would be predicted if virulence is a linear trait with no specific adaptation to the strain used for selection (unpublished results).

To conclude, most studies have found geographic variation in host resistance and parasitoid virulence to be common. The single most important determinant of this variation appears to be alternative

hosts and parasitoids, though a definitive test of this hypothesis is lacking. Most evidence suggests resistance and virulence to be linear traits, without local adaptation, though there is a possibility that local adaptation leads to a reduction in mortality associated with parasitoid oviposition. However, a systematic study of the performance of a geographical range of parasitoid strains, on an equivalent range of host strains, has yet to be done.

Cross-species comparisons

Many authors have compared the performance of one parasitoid species on a variety of different hosts, or of a number of different species of parasitoid on a single host. The mere fact that differences exist is not very surprising, as all hosts have the ability to encapsulate foreign bodies, and the extent to which a particular parasitoid species have evolved to overcome host resistance will be influenced by a wide array of genetic, ecological, phylogenetic and historical factors. However, patterns in cross-species comparisons of resistance and virulence are more interesting, and several have been detected. One cautionary note, the statistical comparison of cross-species patterns is technically difficult as species cannot be treated as independent data points. Modern comparative techniques to overcome these problems have yet to be applied to studies of parasitoid virulence and resistance.

The first observation is that there are some host species that are poorly defended against nearly all parasitoids. The best example of this is *Drosophila subobscura*, a north-European species that has never been observed to encapsulate the parasitoids that attack it naturally in the field, and also has no resistance against a number of exotic parasitoids (Kraaijeveld & van der Wel, 1994). If resistance is costly, then possibly some species have evolved to make a minimal investment, in effect gambling that they will not be discovered by parasitoids.

Carton & Kitano (1981) surveyed the ability of seven species of the *D. melanogaster* group to survive parasitism by *L. boulardi*. Species differed in their suitability as hosts for *L. boulardi*, but also in the extent to which they were killed by the act of parasitism. Using these two criteria, the authors divided the seven species into three classes, and noted that with one exception, the classification reflected the established phlyogenetic relationships of this group. The exception was *D. simulans*, which was considerably more resistant than its sibling species *D. melanogaster*. The authors note that in interspecific competition *D. melanogaster* excludes *D. simulans* and speculate that the interaction might be stabilised by a parasitoid. As will be discussed in the next section, Eslin & Prévost (1998) have suggested a physiological mechanism underlying different levels of resistance in the *D. melanogaster* group.

In the face of host species that vary in resistance, parasitoids should evolve behavioural adaptations so as not to waste time or eggs on poor quality hosts. The behavioural ecology of host choice in *Drosophila* parasitoids has been extensively investigated, but is beyond the scope of our review (see van Alphen & Janssen, 1982; van Alphen & Vet, 1986; Driessen, Hemerik & Boonstra, 1991) except to note one interesting finding. Janssen (1989) followed *A. tabida* and *L. heterotoma* as they searched for hosts in fruit and sap-fluxes in the field. Nearly all hosts were accepted, even though parasitoid survival would have been zero or very low in many of the hosts encountered. However, encounter rates with hosts were so low that Janssen calculated from optimal foraging models that oviposition in a host would be selected as long as the probability of larval survival was greater than a threshold of around 0·002 to 0·03 (depending on species and microhabitat). What this work indicates is that parasitoid eggs or larvae will often find themselves in unsuitable hosts, and hence where appropriate genetic variation exists there will be great scope for selection for increased virulence.

Physiological basis of differences in resistance and virulence

The size of *Drosophila* larvae make them a poor model organism for physiologists investigating the basis of defence and resistance, though clearly they have enormous advantages genetically. Isofemale lines resistant to *L. boulardi*, obtained as part of the experiments discussed above, have been used extensively by Nappi and colleagues (e.g. Nappi, 1981; Nappi, Carton & Frey, 1991; Cousteau *et al.* 1996) to study physiological and biochemical aspects and a recent review of this work is provided by Carton & Nappi (1997).

Eslin & Prévost (1996) found that the concentration of circulating haemocytes was higher in *D. simulans* than in *D. melanogaster* and suggested that this might account for the former species' higher resistance to *A. tabida*. More recently Eslin & Prévost (1998) extended their survey to six species in the *melanogaster* group and found a significant correlation between circulating haemocyte concentration and resistance (though note that possibly confounding phylogenetic effects were not controlled in the analysis). Differences in resistance to *A. tabida* are associated with differences in the degree to which the parasitoid egg embeds itself in host tissue (Kraaijeveld & van Alphen, 1994; Monconduit & Prévost, 1994; Eslin *et al.* 1996). In Eslin & Prévost's view (see also Strand & Pech, 1995), the outcome of parasitization depends on whether the host can produce sufficient haemocytes to prevent embedding occurring. Higher concentrations of haemocytes prior to parasitization gives the host an advantage.

Genetic basis of difference in resistance and virulence

Schlegel-Oprecht (1953), Walker (1959, 1961, 1962) and Hadorn & Walker (1960) were the first workers to study the genetics of *Drosophila* resistance. They crossed two field strains of *D. melanogaster* that differed in their resistance to *L. heterotoma* and concluded that resistance was a quantitative trait determined by genes at many loci. Rather different results have been obtained by Carton and colleagues (Carton, Frey & Nappi, 1992; Carton & Nappi, 1997) who have studied the genetic differences between isofemale lines of *D. melanogaster* derived from Congolese flies (see above) that are resistant and susceptible to a sympatric strain of *L. boulardi*. In a series of crosses, they showed resistance was due to an autosomal, dominant gene in the interval (55D-55F) of chromosome 2. The susceptible strain is not immune incompetent as it can defend itself against *A. tabida* (Vass *et al.* 1993).

Orr & Irving (1997) have studied the genetic basis of the differences in *A. tabida* resistance amongst flies from different parts of Europe (see above). Crosses between flies from the north and south of Europe showed intermediate levels of resistance. Using classical *Drosophila* genetic crossing techniques, except employing only markers that are visible in the larval stages, they were able to localize the genetic difference to the centromere region of chromosome 2. These results strongly indicate a simple, possibly monogenic, basis for the geographical variation in resistance to *A. tabida*. Benassi, Frey & Carton (1998) studied an isofemale line of *D. melanogaster* that was highly susceptible to *A. tabida*. The susceptibility is due to a single recessive autosomal gene, though whether it is the same gene as that found by Orr & Irving is not yet known.

The genetic basis of the increased resistance to *A. tabida* in the *D. melanogaster* lines studied by Kraaijeveld & Godfray (1997) has yet to be established. Crosses between selected and control lines produced flies with intermediate levels of resistance, suggesting either a quantitative trait or major genes without dominance. The response of F1 flies is independent of whether their mothers or fathers were from selected lines, hence the gene or genes responsible are not sex-linked.

Early experiments that involved crossing lines of *L. heterotoma* that differed in virulence from different geographical localities, suggested polygenic inheritance with maternal effects (Walker, 1962). Recently Dupas, Frey & Carton (1998) have performed crossing experiments with isogenic lines of *L. boulardi*, derived originally from African populations. Crosses between lines of high and low virulence indicated that the differences were due to a single, semi-dominant gene.

Thus while we are still only at the beginning of the genetic analysis of resistance and virulence, especially of the latter, recent research suggests that the underlying genetic basis is more likely to involve major genes than to be polygenic.

CONCLUSIONS AND FURTHER AREAS OF STUDY

Although in this paper we have concentrated on the coevolution of the physiological aspects of resistance and virulence, there are a number of other arenas in which hosts and parasitoids can act as selective agents on each other, and we finish by briefly discussing two of them. There has been an enormous amount of research into the means by which parasitoids locate hosts (for reviews see Vinson & Iwantsch, 1980; Nordlund, Jones & Lewis, 1981, Vinson, 1985; Godfray, 1994; van Alphen & Jervis, 1995). The most common means of host location is through chemical signals and numerous studies have identified examples of parasitoids employing cues emanating from the host habitat, from substances associated with the host (for example frass), and from the host itself. Parasitoids also display a sophisticated ability to learn stimuli associated with the presence of their host (e.g. Lewis & Tumlinson, 1988). If parasitoids employ chemical stimuli to locate hosts, they will exert selection pressure on their host to avoid producing these chemicals, the entomological equivalent of a communications blackout. However, in some cases the host may require to produce chemical signals for other reasons, for example sex pheromones or aggregation pheromones (examples are known of where both these types of signals have been hijacked by parasitoids). Coevolution of 'infochemical' (Dicke & Sabelis, 1988; Dicke, 1998) use and production may be more complicated and involve further partners. There is strong evidence that some plants respond to herbivore attack by producing chemical signals that attract the herbivore's parasitoids (Turling & Tumlinson, 1992; Dicke, 1997). More speculatively, some hyperparasitoids use chemical cues emitted by the host to locate possible primary parasitoids (Read, Feeny & Root, 1970; Micha, Stammel & Höller, 1993) and the host may be selected actively to attract its enemy's enemy.

In addition to chemical cues, some parasitoids use auditory, visual or vibrational signals in host location. How parasitoids influence the fitness of different *Drosophila* behavioural polymorphisms has already been discussed. Some dipterous parasitoids use the stridulation of male orthopterans in host location, and this may influence the fitness of caller and satellite strategies in mate location (Zuk, Simmons & Rotenberry, 1995). After a host patch has been discovered, parasitoids adopt sophisticated behavioural rules to determine when to leave the present patch and search for another. This behaviour has been closely studied as a model system with which to investigate the classical patch use model of

behavioural ecology (van Alphen & Vet, 1986, Godfray, 1994). Thompson (1986) has suggested that the outcome of host-parasitoid coevolution over patch use is that hosts distribute their eggs across patches to provide the minimum amount of information to searching parasitoids (though see possible objections in Godfray, 1994). Currently, most research has focused on identifying the form of host location, rather than quantifying selection pressures and reciprocal responses, or formal comparative analyses; but this is an exciting area of future investigation of coevolution.

The second area in which coevolutionary effects are important is the study of the host niche. In particular, parasitoids may influence both the temporal and spatial distribution of the host, as well as the manner in which the host utilises its resources. Again there have been few formal studies of coevolutionary patterns or processes, though a wealth of evidence that suggests its potential importance. For example, many studies have quantified differential parasitoid pressures on hosts active at different times of years (e.g. Myers, 1981), in different microhabits (e.g. van Alphen & Vet, 1986), or on different host plants (e.g. Lawton, 1986). In some cases, selection pressures acting in the opposite direction to parasitism have been found, and frequency-dependent effects identified. It has often been suggested that insect galls have evolved as a means of protection against parasitoids (though gall formers are often severely attacked by highly adapted parasitoids, this does not diminish their potential importance in the initial evolution of galling). Weis & colleagues, (Weis & Abrahamson, 1985; Weis *et al.* 1985, 1992; Weis & Kapelinski, 1994; Weis, 1996) found that the tephritid *Eurosta solidaginis* produced galls of variable size (on *Solidago canadensis*) and that large galls were more likely to avoid parasitoid attack. Moreover, they were able to show additive genetic variation in gall size in the host. But while size was beneficial with respect to parasitism, large galls attracted heavier bird predation.

Our main aims in this paper have been to review the coevolutionary interactions between parasitoid virulence and host resistance. We believe this subject is fascinating it its own right, and it may have applied importance in the design of biological control programmes using insect parasitoids. However, we also believe that host–parasitoid systems, especially the *Drosophila*-parasitoid interaction, have a valuable role in exploring general issues of the coevolution of virulence and resistance that are much large tractable to study using parasites and pathogens. Also, although the genetic analysis of *Drosophila* resistance is still in its infancy, we concur with Orr & Irving (1998) who argue that this is good model with which to study the genetic basis of adaptation. Beyond, virulence and resistance, many other aspects of host–parasitoid biology are likely to have been shaped by reciprocal evolution, and are amenable to study using ecological genetic and comparative biological techniques.

REFERENCES

ASGARI, S. & SCHMIDT, O. (1994). Passive protection of eggs from the parasitoid, *Cotesia rubecula*, in the host, *Pieris rapae*. *Journal of Insect Physiology* **40**, 789–795.

ASKEW, R. R. & SHAW, M. R. (1986). Parasitoid communities: their size, structure and development. In *Insect Parasitoids* (ed. Waage, J. K. & Greathead, D.), London, Academic Press.

BECKAGE, N. E., THOMPSON, S. N. & FEDERICI, B. A. (1993). *Parasites and Pathogens of Insects. Volume 1 : Parasites*. San Diego, Academic Press.

BENASSI, V., FREY, F. & CARTON, Y. (1998). A new specific gene for wasp cellular immune resistance in *Drosophila Heredity* (in press).

BOULÉTREAU, M. (1986). The genetic and coevolutionary interactions between parasitoids and their hosts. In *Insect Parasitoids* (ed. Waage, J. K. & Greathead, D.), pp. 169–200. London, Academic Press.

BOULÉTREAU, M. & FOUILLET, P. (1982). Variablitié génétique intrapopulation de l'adéquation de *Drosophila melanogaster* à un de ses parasites hyménoptères. *Comptes rendu de l'Académie des Sciences* **295**, 775–778.

BOULÉTREAU, M. & WAJNBERG, E. (1986). Comparative responses of two sympatric parasitoid cynipids to the genetic and epigenetic variations of the larvae of their host, *Drosophila melanogaster*. *Entomologia Experimentalis et Applicata* **41**, 107–114.

BREHÉLIN, M. (1986). *Immunity in Invertebrates*. Berlin, Springer Verlag.

CARTON, Y. (1984). Analyse expérimentale de trois niveaux d'interactions entre *Drosophila melanogaster* et le parasite *Leptopilina boulardi* (sympatrie, allopatrie, xénopatrie). *Genetics, Selection & Evolution* **16**, 417–430.

CARTON, Y. (1987). Ecological and genetic significance of the encapsulation process in *Drosophila* against a parasitic wasp. *Les Colloques de l'INRA* **58**, 99–100.

CARTON, Y. & BOULÉTREAU, M. (1985). Encapsulation ability of *Drosophila melanogaster*: a genetic analysis. *Developmental & Comparative Immunology* **9**, 211–219.

CARTON, Y., BOULÉTREAU, M., VAN ALPHEN, J. J. M. & VAN LENTEREN, J. C. (1986). The *Drosophila* parasitic wasps. In *The Genetics and Biology of Drosophila, Volume 3e* (ed. Ashburner, M., Carson, L. & Thompson, J. N.), pp. 347–394. London, Academic Press.

CARTON, Y., CARY, P. & NAPPI, A. (1989). Genetic variability of host-parasite relationship traits: utilization of isofemale lines in a *Drosophils simulans* parasitic wasp. *Genetics, Selection & Evolution* **21**, 437–446.

CARTON, Y. & DAVID, J. R. (1983). Reduction of fitness in *Drosophila* adults surviving parasitization by a cynipid wasp. *Experientia* **39**, 231–233.

CARTON, Y. & DAVID, J. R. (1985). Relation between the genetic variability of digging behaviour of *Drosophila* larvae and their susceptibility to a parasitic wasp. *Behavioural Genetics* **15**, 403–408.

CARTON, Y., FREY, F. & NAPPI, A. (1992). Genetic determinism of the cellular immune reaction in *Drosophila melanogaster*. *Heredity* **69**, 393–399.

CARTON, Y. & KITANO, H. (1981). Evolutionary relationships to parasitism by seven species of the *Drosophila melanogaster* subgroup. *Biological Journal of the Linnean Society* **16**, 227–241.

CARTON, Y. & NAPPI, A. (1992). Methods for genetic investigation of cellular immune reaction in insects, with the parasitic (*Drosophila*) system as a model. In *Insect Immunity* (ed. Pathak, J. P. N.), pp. 91–101. Oxford, Oxford & IBH Publishing Company.

CARTON, Y. & NAPPI, A. (1997). *Drosophila* cellular immunity against parasitoids. *Parasitology Today* **13**, 217–227.

CARTON, Y. & NAPPI, A. J. (1991). The *Drosophila* immune reaction and the parasitoid capacity to evade it: genetic and coevolutionary aspects. *Acta Oecologica* **12**, 89–104.

CARTON, Y. & SOKOLOWSKI, M. B. (1992). Interactions between searching strategies of *Drosophila* parasitoids and the polymorphic behaviour of their hosts. *Journal of Insect Behaviour* **5**, 161–175.

CARTON, Y. & SOKOLOWSKI, M. B. (1994). Parasitization of embedded and non-embedded *Drosophila melanogaster* (Diptera: Drosophilidae) pupae by the parasitoid *Pachycrepoideus vindemniae* (Hymenoptera: Pteromalidae). *Journal of Insect Behaviour* **7**, 129–131.

COUSTEAU, C., ROCHELEAU, T., CARTON, Y., NAPPI, A. J. & FFRENCH CONSTANT, R. H. (1996). Induction of a putative serine protease transcript in immune challenged *Drosophila*. *Developmental & Comparative Immunology* **20**, 265–272.

DE BELLE, J. S., HILLIKER, A. J. & SOKOLOWSKI, M. B. (1989). Genetic localization of foraging (*for*), a major gene for larval behaviour in *Drosophila melanogaster*. *Genetics* **123**, 157–163.

DE BELLE, J. S. & SOKOLOWSKI, M. B. (1987). Heredity of rover/sitter: alternative foraging strategies of *Drosophila melanogaster* larvae. *Journal of Heredity* **75**, 131–134.

DE BELLE, J. S., SOKOLOWSKI, M. B. & HILLIKER, A. J. (1993). Genetic analysis of the *foraging* microregion of *Drosophila melanogaster*. *Genome* **36**, 94–101.

DELPUECH, J. M., FREY, E. & CARTON, Y. (1994). Genetic and epigenetic variation in suitability of a *Drosophila* host to three parasitoid species. *Canadian Journal of Zoology* **72**, 1940–1944.

DICKE, M. (1998). Evolution of induced indirect defense of plants. In *Evolution of Induced Defenses* (ed. Harvell, C. D. & Tollrien, R.). Princeton, Princeton University Press (in press).

DICKE, M. & SABELIS, M. W. (1988). Infochemical terminology: based on cost-benefit analysis rather than origin of compounds. *Functional Ecology* **2**, 131–139.

DRIESSEN, G., HEMERIK, L. & BOONSTRA, B. (1991). Host selection behaviour in relation to survival in hosts of *Leptopilina clavipes*, a parasitoid of larval *Drosophila*. *Netherlands Journal of Zoology* **41**, 99–111.

DUPAS, S., BREHÉLIN, M., FREY, D. F. & CARTON, Y. (1996). Immune suppressive virus-like particles in a *Drosophila* parasitoid: significance of their intraspecific morphological variations. *Parasitology* **113**, 207–212.

DUPAS, S., FREY, D. F. & CARTON, Y. (1998). A single parasitoid segregating factor controls immune suppression in *Drosophila*. *Journal of Heredity* (in press).

ESLIN, P., GIORDANENGO, P., FOURDRAIN, Y. & PRÉVOST, G. (1996). Avoidance of encapsulation in the absence of VLP by a braconid parasitoid of *Drosophila* larvae: an ultrastructural study. *Canadian Journal of Zoology* **74**, 2193–2198.

ESLIN, P. & PRÉVOST, G. (1996). Variation in *Drosophila* concentration of haemocytes associated with different ability to encapsulate *Asobara tabida* larval parasitoid. *Journal of Insect Physiology* **42**, 549–555.

ESLIN, P. & PRÉVOST, G. (1998). Hemocyte load and immune resistance to *Asobara tabida* are correlated in species of the *Drosophila melanogaster* subgroup. *Journal of Insect Physiology* (in press).

FALCONER, D. S. (1996). *Introduction to Quantitative Genetics* (*third edition*). London, Longman Group UK Ltd.

FELLOWES, M. D. E., KRAAIJEVELD, A. R. & GODFRAY, H. C. J. (1999). The relative fitness of *Drosophila melanogaster* (Diptera, Drosophilidae) after successful defence against the parasitoid *Asobara tabida* (Hymenoptera, Braconidae). *Journal of Evolutionary Biology* (in press).

FELLOWES, M. D., MASNATTA, E. P., KRAAIJEVELD, A. R. & GODFRAY H. C. J. (1998). Pupal parasitoid attack influences the relative fitness of *Drosophila* that have encapsulated larval parasitoids. *Ecological Entomology* (in press).

FLEMING, J.-A. G. W. (1992). Polydnaviruses: mutualists and pathogens. *Annual Review of Entomology* **37**, 401–425.

GILLESPIE, J., KANOST, M. R. & TRENCZEK, T. (1997). Biological mediators of insect immunity. *Annual Review of Entomology* **42**, 611–643.

GODFRAY, H. C. J. (1994). *Parasitoids, Behavioral and Evolutionary Ecology*. Princeton, New Jersey, Princeton University Press.

GODFRAY, H. C. J. & HASSELL, M. P. (1991). Encapsulation and host–parasitoid population dynamics. In *Parasite–Host Association: Coexistence or Conflict* (ed. Toft, C. A., Aeschlimann, A. & Bolis, L.), pp. 131–147. Oxford, Oxford University Press.

GODFRAY, H. C. J. & MÜLLER, C. B. (1998). Host-parasitoid dynamics. In *Insect Population Dynamics* (ed. Dempster, J. P. & Maclean, I.). London, Academic Press (in press).

HADORN, E. & GRASSMAN, A. (1962). *Drosophila* und *Pseudeucolia* IV. Artspezifsche Unterschiede in der Abwehrreaktion auf verschieden resistente Wespenstämme. *Schweizerische Gesellschaft für Verberbungs-forschung Jahresbericht*. **37**, 21–27.

HADORN, E. & WALKER, I. (1960). *Drosophila* und *Pseudeucolia*. I Selektionsversuche zur Steigerung der Abwehrreaktion des Wirtes gegen den Parasiten. *Revue Suisse de Zoologie* **67**, 216–225.

HAMILTON, W. D. & ZUK, M. (1982). Heritable true fitness and bright birds: a role for parasites? *Science* **218**, 384–387.

HASSELL, M. P. (1978). *The Dynamics of Arthropod Predator–Prey Systems*. Princeton, Princeton University Press.

HASSELL, M. P. (1998). *The Dynamics of Insect–Parasitoid Interactions*. Oxford, Oxford University Press.

HENTER, H. J. (1995). The potential for coevolution in a host-parasitoid system. 2. Genetic variation within a population of wasps in the ability to parasitize an aphid host. *Evolution* **49**, 439–445.

HENTER, H. J. & VIA, S. (1995). The potential for coevolution in a host-parasitoid system. 1. Genetic variation within an aphid population in susceptibility to a parasitic wasp. *Evolution* **49**, 427–438.

HOFFMANN, A. A. & PARSONS, P. A. (1988). The analysis of quantitative variation in natural populations with isofemale strains. *Genetics, Selection and Evolution* **20**, 87–98.

HUGHES, K. & SOKOLOWSKI, M. B. (1996). Natural selection in the laboratory for a change in resistance by *Drosophila melanogaster* to the parasitoid wasp *Asobara tabida*. *Journal of Insect Behaviour* **9**, 477–491.

IVES, W. G. H. & MULDREW, J. A. (1981). *Pristiphora erichsonii* (Hartig), Larch Sawfly (Hymenoptera: Tenthredinidae). In *Biological Control Programmes Against Insects and Weeds in Canada 1960–1980* (ed. Kelleher, J. S. & Hulme, M. A.), pp. 369–380. Slough, UK, Commonwealth Agricultural Bureau.

JANSSEN, A. (1989). Optimal host selection by *Drosophila* parasitoids in the field. *Functional Ecology* **3**, 469–479.

KING, B. H. (1987). Offspring sex ratios in parasitoid wasps. *Quarterly Review of Biology* **62**, 367–396.

KRAAIJEVELD, A. R. (1994). *Local Adaptations in a Parasitoid-host System*. PhD thesis: University of Leiden.

KRAAIJEVELD, A. R., EMMETT, D. A. & GODFRAY, H. C. J. (1997). Absence of direct sexual selection for parasitoid encapsulation in *Drosophila melanogaster*. *Journal of Evolutionary Biology* **10**, 337–342.

KRAAIJEVELD, A. R. & GODFRAY, H. C. J. (1997). Trade-off between parasitoid resistance and larval competitive ability in *Drosophila melanogaster*. *Nature* **389**, 278–280.

KRAAIJEVELD, A. R. & VAN ALPHEN, J. J. M. (1994). Geographical variation in resistance of the parasitoid *Asobara tabida* against encapsulation by *Drosophila melanogaster* larvae: the mechanism explored. *Physiological Entomology* **19**, 9–14.

KRAAIJEVELD, A. R. & VAN ALPHEN, J. J. M. (1995). Geographical variation in encapsulation ability of *Drosophila melanogaster* larvae and evidence for parasitoid-specific components. *Evolutionary Ecology* **9**, 10–17.

KRAAIJEVELD, A. R. & VAN DER WEL, N. N. (1994). Geographic variation in reproductive success of the parasitoid *Asobara tabida* in larvae of several *Drosophila* species. *Ecological Entomology* **19**, 221–229.

LACKIE, A. M. (1988). Immune mechanisms in insects. *Parasitology Today* **4**, 98–105.

LAWTON, J. H. (1986). The effects of parasitoids on phytophagous insect communities. In *Insect Parasitoids* (ed. Waage, J. K. & Greathead, D.), pp. 265–287. London, Academic Press.

LEWIS, W. J. & TUMLINSON, J. H. (1988). Host detection by chemically mediated associative learning in a parasitic wasp. *Nature* **331**, 257–259.

MICHA, S. G., STAMMEL, J. & HÖLLER, C. (1993). 6-Methyl-5-heptene-2-one, a putative sex and spacing pheromone of the aphid hyperparasitoid, *Alloxysta victrix* (Hymenoptera: Alloxystidae). *European Journal of Entomology* **90**, 439–442.

MILLS, N. J. & GETZ, W. M. (1996). Modelling the biological control of insect pests: a review of host–parasitoid models. *Ecological Modelling* **92**, 121–143.

MONCONDUIT, H. & PRÉVOST, G. (1994). Avoidance of encapsulation by *Asobara tabida*, a larval parasitoid of *Drosophila* species. *Norwegian Journal of Agricultural Sciences* **Supplement 16**, 301–310.

MULDREW, J. A. (1953). The natural immunity of the larch sawfly (*Pristophora erichsonii* Htg.) to the introduced parasite *Mesoleius tenthredinis* Morley in Manitoba and Saskatchewan. *Canadian Journal of Zoology* **31**, 313–332.

MURDOCH, W. W. & BRIGGS, C. J. (1996). Theory for biological control: recent developments. *Ecology* **77**, 2001–2013.

MYERS, J. H. (1981). Interactions between western tent caterpillars and wild rose – a test of some general plant herbivore hypotheses. *Journal of Animal Ecology* **50**, 11–25.

NAPPI, A. (1981). Cellular immune response of *Drosophila melanogaster* against *Asobara tabida*. *Parasitology* **83**, 319–324.

NAPPI, A., CARTON, Y. & FREY, F. (1991). Parasite induced enhancement of haemolymph tyrosinase activity in a selected immune reactive strain of *Drosophila melanogaster*. *Archives of Insect Biochemistry and Physiology* **18**, 159–168.

NAPPI, A., VASS, E., FREY, F. & CARTON, Y. (1995). Superoxide anion generation in *Drosophila* during melanotic encapsulation of parasites. *European Journal of Cell Biology* **68**, 450–458.

NORDLANDER, G. (1980). A revision of the genus *Leptopilina* Förster, with notes on the status of some other genera (Hymenoptera, Cynipidae, Eucoilidae). *Entomologica Scandinavica* **11**, 428–453.

NORDLUND, D. A., JONES, R. L. & LEWIS, W. J. (1981). *Semiochemicals, their Role in Pest Control*. New York, John Wiley.

OPRECHT, E. & HADORN, E. (1952). Stock-specific defence reactions against a parasite. *Drosophila Information Service* **26**, 116.

ORR, H. A. & IRVING, S. (1997). The genetics of adaption: the genetic basis of resistance to wasp parasitism in *Drosophila melanogster*. *Evolution* **51**, 1877–1885.

OSBORNE, K. A., ROBICHON, A., BURGESS, E., BUTLAND, S., SHAW, R. A., COULTHARD, A., PEREIRA, H. S., GREENSPAN, R. J. & SOKOLOWSKI, M. B. (1997). Natural behavior polymorphism due to cGMP-dependent protein kinase of *Drosophila*. *Science* **277**, 834–836.

PARSONS, P. A. (1980). Isofemale strains and evolutionary strategies in natural populations. In *Evolutionary Biology* (ed. Hecht, M., Steere, W. & Wallace, B.), New York, Plentum Publishing Corporation.

PIMENTEL, D. (1968). Population regulation and genetic feedback. *Science* **159**, 1432–1437.

PIMENTEL, D., LEVIN, S. A. & OLSON, D. A. (1978). Coevolution and the stability of exploiter-victim systems. *American Naturalist* **112**, 119–125.

PIMENTEL, D., NAGEL, M. P. & MADDEN, J. L. (1963). Space-time structure of the environment and the survival of parasite–host systems. *American Naturalist* **97**, 141–167.

RATCLIFFE, N. (1993). Cellular defence responses of insects: unresolved problems. In *Parasities and Pathogens of Insects, Volume I. Parasites*. (ed. Beckage, N., Thompson, S. N. & Federici, B. A.), pp. 267–304. San Diego, Academic Press.

READ, D. P., FEENY, P. P. & ROOT, R. B. (1970). Habitat selection by the aphid parasite *Diaeretiella rapae* and its hyperparasitoid, *Charips brassicae*. *Canadian Entomologist* **102**, 1567–1578.

RIZKI, R. M. & RIZKI, T. M. (1990). Parasitoid virus-like particles destroy *Drosophila* cellular immunity. *Proceedings of the National Academy of Sciences, USA* **87**, 8388–8392.

RIZKI, T. M. & RIZKI, R. M. (1984). The cellular defence system of *Drosophila melanogaster*. In *Insect Ultrastructure, Volume 2* (ed. King, P. C. & Akai, H.), pp. 579–604. New York, Plenum Press.

RISKI, T. M. & RISKI, R. M. (1994). Parasitoid-induced cellular immune deficiency in *Drosophila*. *Annals of the New York Academy of Science* **712**, 178–194.

RUSSO, J., DUPAS, S., FREY, F., CARTON, Y. & BREHÉLIN, M. (1996). Insect immunity: early events in the encapsulation process of parasitoid (*Leptopilina boulardi*) eggs in resistant and susceptible strains of *Drosophila*. *Parasitology* **112**, 135–142.

SALT, G. (1970). *The Cellular Defence Reactions of Insects*. Cambridge, Cambridge University Press.

SALT, G. & VAN DEN BOSCH, R. (1967). The defence reactions of three species of *Hypera* (Coleoptera: Curculionidae) to an ichneumon wasp. *Journal of Invertebrate Pathology* **9**, 164–177.

SCHLEGEL-OPRECHT, E. (1953). Versuche zur Auslösung von Mutationen bei der zoophaen Cynipide *Pseudeucoila bochei* Weld und Befunde über die stammspezifische Abwekrreaktion des Wirtes *Drosophila melanogaster*. *Zeitschrift für Induktive Abstammungs-und Verberbungslehre* **85**, 245–281.

SCHMIDT, O. & SCHUCHMAN-FEDDERSEN, I. (1989). Role of virus-like particles in parasitoid-host interactions of insects. *Subcellular Biochemistry* **15**, 91–119.

SOKOLOWSKI, M. B. (1980). Foraging strategies of *Drosophila melanogaster*: a chromosomal analysis. *Behavioural Genetics* **10**, 291–302.

SOKOLOWSKI, M. B., BAUER, S. J., WAI PING, V., RODRIGUEZ, L., WONG, J. L. & KENT, C. (1986). Ecological genetics and behaviour of *Drosophila melanogaster* larvae in nature. *Animal Behaviour* **34**, 403–408.

SOKOLOWSKI, M. B. & TURLINGS, T. C. J. (1987). *Drosophila* parasitoid–host interactions: vibrotaxis and ovipositor searching from the host's perspectives. *Canadian Journal of Zoology* **65**, 461–464.

STRAND, M. R. & PECH, L. L. (1995). Immunological basis for compatibility in parasitoid–host relationships. *Annual Review of Entomology* **40**, 31–56.

THOMPSON, J. N. (1982). *Interaction and Coevolution*. New York, John Wiley.

THOMPSON, J. N. (1986). Oviposition behaviour and searching efficiency in a natural population of a braconid parasitoid. *Journal of Animal Ecology* **55**, 351–360.

TURLINGS, T. C. J. & TUMLINSON, J. H. (1992). Systemic release of chemical signals by herbivore-injured corn. *Proceedings of the National Academy of Sciences, USA* **89**, 8399–8402.

VAN ALPHEN, J. J. M. & JANSSEN, A. R. M. (1982). Host selection by *Asobara tabida* Nees (Braconidae: Alysiinae), a larval parasitoid of fruit inhabiting *Drosophila* species. II. Host species selection. *Netherlands Journal of Zoology* **32**, 215–231.

VAN ALPHEN, J. J. M. & JERVIS, M. (1995). Foraging behaviour. In *The Study of Natural Enemies*, (ed. Jervis, M. & Kidd, N. K.), pp. 1–62. London, Chapman & Hall.

VAN ALPHEN, J. J. M. & THUNNISSEN, I. (1983). Host selection and sex allocation by *Pachcrepoides vindemiae* Rondani (Pteromalidae) as a facultative hyperparasitoid of *Asobara tabida* Nees (Braconidae: Alysiinae) and *Leptopilina heterotoma* (Cynipoidea: Eucoilidae). *Netherlands Journal of Zoology* **32**, 232–260.

VAN ALPHEN, J. J. M. & VET, L. E. M. (1986). An evolutionary approach to host finding and selection. In *Insect Parasitoids* (ed. Waage, J. K. & Greathead, D.), pp. 23–61. London, Academic Press.

VAN ALPHEN, J. J. M. & VISSER, M. E. (1990). Superparasitism as an adaptive strategy for insect parasitoids. *Annual Review of Entomology* **35**, 59–79.

VAN DEN BOSCH, R. (1964). Encapsulation of the eggs of *Bathyplectes curculiionis* (Thompson) (Hymenoptera: Ichneumonidae) in larvae of *Hypera brunneipennis* (Boheman) and *Hypera postica* (Gyllenhal) (Coleoptera: Curculionidae). *Journal of Invertebrate Pathology* **6**, 343–367.

VASS, E., NAPPI, A. & CARTON, Y. (1993). Comparative study of immune competence and host susceptibility in *Drosophila melanogaster* parasitised by *Leptopilina boulardi* and *Asobara tabida*. *Journal of Parasitology* **79**, 106–112.

VET, L. E. M. & VAN ALPHEN, J. J. M. (1985). A comparative function approach to the host detection behaviour of parasitic wasps. I. A qualitative study on Eucoilidae and Alysiinae. *Oikos* **44**, 478–486.

VINSON, S. B. (1997). The behaviour of parasitoids. In *Comprehensive Insect Physiology, Biochemistry & Pharmacology* (ed. Kerkut, G. A. & Gilbert, L. I.), pp. 417–469. New York, Pergamon Press.

VINSON, S. B. & IWANTSCH, G. F. (1980). Host suitability for insect parasitoids. *Annual Review of Entomology* **25**, 397–419.

WAAGE, J. K. (1986). Family planning in parasitoids. In *Insect Parasitoids* (ed. Waage, J. K. & Greathead, D.), pp. 63–95. London, Academic Press.

WAJNBERG, E., PRÉVOST, G. & BOULÉTREAU, M. (1985). Genetic and epigenetic variation in *Drosophila* larvae suitability to a hymenopterous endoparasitoid. *Entomophaga* **30**, 187–192.

WALKER, I. (1959). Die Abwehrreaktion des Wirtes *Drosophila melanogaster* gegen die zoophage Cynipidae *Pseudeucolia bochei* Weld. *Revue Suisse de Zoologie* **66**, 569–632.

WALKER, I. (1961). *Drosophila* und *Pseudeucolia*. II. Schwierigkeiten beim Nachweis eines Selektionserfolges. *Revue Suisse de Zoologie* **68**, 252–263.

WALKER, I. (1962). *Drosophila* und *Pseudeucolia*. III. Selektionsversucke zur Steigerung der Resistenz des Parasiten gegen die Abwehrreaktion des Wirtes. *Revue Suisse de Zoologie* **69**, 209–227.

WEIS, A. E. (1996). Variable selection on *Eurosta*'s gall size. 3. Can an evolutionary response to selection be detected? *Journal of Evolutionary Biology* **9**, 623–640.

WEIS, A. E. & ABRAHAMSON, W. G. (1985). Potential selective pressures by parasitoids on a plant–herbivore interaction. *Ecology* **66**, 1261–1269.

WEIS, A. E., ABRAHAMSON, W. G. & ANDERSEN, M. C. (1992). Variable selection on *Eurosta*'s gall size. I. The extent and nature of variation in phenotypic selection. *Evolution* **46**, 1674–1697.

WEIS, A. E., ABRAHAMSON, W. G. & MCCREA, K. D. (1985). Host gall size and oviposition success by the parasitoid *Eurytoma gigantea*. *Ecological Entomology* **10**, 341–348.

WEIS, A. E. & KAPELINSKI, A. (1994). Variable selection on *Eurosta*'s gall size. 2. A path analysis of the ecological factors behind selection *Evolution* **48**, 734–745.

WEIS, A. E., MCCREA, K. D. & ABRAHAMSON, W. G. (1989). Can there be an escalating arms race without cooevolution? Implications from a host–parasitoid simulation. *Evolutionary Ecology* **3**, 361–370.

WONG, J. L., SOKOLOWSKI, M. B. & KENT, C. (1985). Prepupation behaviour in *Drosophila*: embedding. *Behavior Genetics* **15**, 155–165.

ZAREH, N., WESTOBY, M. & PIMENTEL, D. (1980). Evolution in a laboratory host-parasitoid system and its effect on population kinetics. *Canadian Entomologist* **112**, 1049–1060.

ZUK, M., SIMMONS, L. W. & ROTENBERRY, J. T. (1995). Acoustically-orienting parasitoids in calling and silent males of the field cricket *Teleogryllus oceanicus*. *Ecological Entomology* **20**, 380–383.