

Is there local adaptation in *Drosophila*–parasitoid interactions?

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ABSTRACT

Local adaptation has received little attention in host–parasitoid associations. Here we combine data on the outcome of parasitism in 20 sympatric populations of *Drosophila melanogaster* and its parasitoid *Asobara tabida*. We present data on resistance and virulence when each host is tested against a single allopatric strain of parasitoid, and when each parasitoid is tested against a single allopatric strain of host. We argue that the extent to which these allopatric interactions can be used to predict sympatric interactions sets an upper bound to the importance of local adaptation. In a statistical model, we found that 56% of the variance in the outcome of sympatric interactions could be explained by parasitoid virulence and host resistance measured using the allopatric reference strains, with the former being the much more important of the two. The geographical distance between the provenances of the sympatric and reference parasitoid (but not host) populations was also statistically significant and increased the variance explained to 69%; but against expectation, parasitoid success was negatively correlated with distance. We also explore the factors determining the frequency with which neither host nor parasitoid survive. We conclude that, although the critical tests have yet to be performed, the available evidence points towards local adaptation not being of major importance in this system.

Keywords: encapsulation, local adaptation, parasitoid, resistance, virulence.

INTRODUCTION

If parasites or pathogens evolve to become more efficient at overcoming the defences of their particular host population compared to other host populations, then local adaptation is said to occur. Parasites and pathogens are expected to become locally adapted to their host rather than vice versa because of their typically shorter generation time (although see Lively, 1999, for a simulation model arguing against this), and because they must certainly avoid or evade host defences to survive while some hosts may never encounter natural enemies (Gandon *et al.*, 1996). Local adaptation has been found in parasite–host systems, including snails and trematodes (Lively, 1989, 1999), fish and trematodes (Ballabeni and Ward, 1993), *Daphnia* and micro-parasites (Ebert, 1994), plants and fungal pathogens (Parker, 1985; Kaltz *et al.*, 1999), insect herbivores on trees (Mopper *et al.*, 1995) and

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birds and brood-parasites (Soler and Møller, 1990), although it is far from a universal phenomenon (Imhoof and Schmid-Hempel, 1998; Mutikainen *et al.*, 2000; see review by Kaltz and Shykoff, 1998).

Surprisingly, perhaps, local adaptation in host–parasitoid systems has received little attention (no examples are quoted in Kaltz and Shykoff, 1998). Parasitoids are insects whose larvae develop by feeding externally or internally on the body of another insect, which they eventually kill (Godfray, 1994). They thus have features in common with parasites (i.e. an intimate association with a single host) and predators (i.e. they invariably kill their host). To test for local adaptation in a set of host–parasitoid interactions, one would ideally test all allopatric and sympatric combinations of hosts and parasitoids to determine whether parasitoids (or possibly hosts) did consistently better in sympatric rather than allopatric combinations (Kaltz *et al.*, 1999; Mutikainen *et al.*, 2000), something that has not been attempted for hosts and parasitoids (although see Carton, 1984, and the Discussion below).

Here we analyse a data set that allows us to investigate some aspects of local adaptation in host–parasitoid systems. The first part of the data set consists of the relative performance (i.e. the probability that the parasitoid or the host survives) of 20 sympatric host and parasitoid populations. The second part consists of the frequency of successful host defence when the host and parasitoid are tested in allopatric combination with a single parasitoid or host strain (which we shall refer to as the reference strains). We investigate, using statistical modelling, the extent to which sympatric performance can be explained by the success of the host and parasitoid against the allopatric reference strains. If host or parasitoid performance is a simple function of a population's investment in virulence and resistance, then knowing the relative success against the reference strain should allow us to predict the outcome of the sympatric association, something that would not occur in the face of complex local adaptation. We also ask whether the success of the parasitoid or the host against their respective reference strain is most informative in predicting the outcome of the sympatric association, and whether the geographical distance separating the sympatric populations from the provenance of the two reference strains is also significant in explaining sympatric performance. An alternative outcome to parasitoid or host survival is the death of both parties and we also investigate whether this can be predicted from information obtained with reference strains.

The data set consists of the relative performance of European populations of *Drosophila melanogaster* and its larval parasitoid, the braconid wasp *Asobara tabida*. *Drosophila* larvae are attacked by a number of species of parasitoid which oviposit their eggs into the larval haemocoel. The host attempts to defend itself by mounting a cellular immune response (encapsulation) in which haemocytes aggregate around the egg forming a capsule that melanizes, leading to the parasitoid's death (Carton and Nappi, 1997). *A. tabida* seems to defend itself by its eggs becoming embedded in host tissue where they are protected from circulating haemocytes (Kraaijeveld and Van Alphen, 1994; Eslin *et al.*, 1996). Note that virulence in parasitoids is defined slightly different from that in true parasites: the probability that an egg is not killed by the host's immune system. The data were originally collected to study geographical variation in resistance and virulence: parasitoids from the Mediterranean tend to have higher virulence than those from the north, east or west of Europe (Kraaijeveld and Van Alphen, 1994). The picture for host resistance is more complex: it is strongest in central southern Europe, but weaker in the north, in the Iberian peninsula and in the south east (Kraaijeveld and Van Alphen, 1995).

METHODS

The 20 sympatric populations of host and parasitoids were collected from geographical locations throughout Europe (see Kraaijeveld and Van der Wel, 1994, for exact localities and collection procedures). The outcome of parasitoid attack on sympatric hosts was recorded in the laboratory under standardized conditions (for details, see Kraaijeveld and Van der Wel, 1994). The rate at which parasitoids from different populations avoided encapsulation (henceforth parasitoid virulence) was measured against a reference strain of host collected at Hamburg, northern Germany, while the rate at which hosts encapsulated parasitoids (henceforth host resistance) was measured against a parasitoid reference strain collected at Sospel, southern France (Kraaijeveld and Van Alphen, 1994, 1995). To obtain the most useful data pertaining to the relative virulence of different parasitoid populations, the host reference strain was chosen from preliminary studies to have a relatively low encapsulation ability (a high-resistance strain would have led to the death of most parasitoids). For a similar reason, the reference strain of parasitoid was chosen to have a relatively high virulence, as a weak strain would have been encapsulated by most hosts. In 18 of 20 cases, parasitoid survival on sympatric hosts was less frequent than the avoidance of encapsulation on the reference strain; this is not evidence of local maladaptation, but was due to our deliberate choice of a low-resistance reference host strain. Similarly, in 17 of 20 cases, host survival was more frequent when parasitized by sympatric parasitoids than the fraction encapsulating the reference strain of parasitoid, but again this is not local adaptation but was due to our choice of parasitoid reference strain.

There are three outcomes to parasitoid oviposition: the host survives, the parasitoid survives, or both host and parasitoid die. The latter may be due to factors that are intrinsic to the host–parasitoid interaction, or to extraneous factors that would have led to mortality in the absence of parasitism. We begin by ignoring mortality and analyse the fraction of hosts that produced parasitoids, the denominator being the total number of hosts that survived to produce either flies or parasitoids. We perform a multiple regression on arc-sine square-root transformed data, where the response variable is parasitoid success in the sympatric interaction, and the explanatory variables are parasitoid virulence (avoidance of encapsulation in the host reference strain), host resistance (encapsulation of the parasitoid reference strain) and the geographical distance between the provenance of the sympatric and the two reference strains. Parasitoid virulence and host resistance were both arc-sine square-root transformed.

We initially chose a best-fit model using the automated model selection procedure implemented in the *S-Plus 4* statistical package, which employs the Akaike information criterion (AIC; a log-likelihood-based method that balances changes in the explanatory power of a statistical model against the loss or gain of degrees of freedom). We then tried to explain the factors that lead to the mortality of both hosts and parasitoids. The risk of mortality may be due to intrinsic host factors that would have led to host death even in the absence of parasitism. We call this factor ‘control mortality’ and incorporated this into the analysis using data from Kraaijeveld and Van der Wel (1994). It is possible that a cost of host resistance is increased mortality; therefore, we included encapsulation ability against the reference parasitoid strain, mortality after attack by the parasitoid reference strain, and geographic distance between the sympatric and parasitoid reference strains in the list of possible explanatory factors. Finally, we explored explanatory factors associated with the parasitoid: virulence on the host reference strain, reference strain mortality when

attacked by the parasitoid, and geographic distance between the sympatric and host reference strains. We sifted these seven potential explanatory variables using AIC automated stepwise regression (see above).

RESULTS

The data used in the analysis are shown in Table 1. The success of parasitoids in sympatric associations (expressed as a fraction of emerging hosts plus parasitoids) varied from 0.1 to 0.94 with a mean of 0.34. Among sympatric associations, both host and parasitoid died in 0.06 to 0.29 of cases (mean 0.16).

First, we analyse parasitoid success ignoring host and parasitoid mortality. The automated multiple regression procedure fitted a model that contained, in the following order, parasitoid virulence, host resistance and the geographical distance between the sympatric population and parasitoid reference strain. The geographical distance between the sympatric population and the host reference strain did not enter into the selected model. Overall, the model with the three explanatory variables was highly significant ($F_{3,16} = 11.95$, $P = 0.0002$) and explained 69% of the variance. Adding parasitoid virulence first produced a highly significant improvement in fit ($F_{1,18} = 14.19$, $P = 0.001$), accounting for 44% of the variance. The addition of host resistance second ($F_{1,17} = 4.61$, $P = 0.046$) increased the variance explained to 56%; the remaining variance of the full model was accounted for by the addition of parasitoid distance ($F_{1,16} = 6.81$, $P = 0.019$). Interestingly, if host resistance was forced into the model as the first term, it did not improve the fit significantly ($F_{1,18} = 0.54$, $P = 0.47$), again suggesting that virulence is the dominant factor affecting the outcome of parasitism. Virulence and resistance influenced the outcome of parasitism in the expected direction but parasitoid success was negatively related to parasitoid distance. If a correlation with distance does exist, the expected relation when local adaptation occurred would be positive rather than negative. The relationship between sympatric parasitoid survival and parasitoid virulence is shown in Fig. 1a. In Fig. 1b, we plot the residuals from this regression against host resistance and in Fig. 1c the residuals from the model containing both parasitoid virulence and host resistance against the geographical distance to the parasitoid reference strain. Finally, Fig. 1d shows a plot of the data against the fitted values from the model.

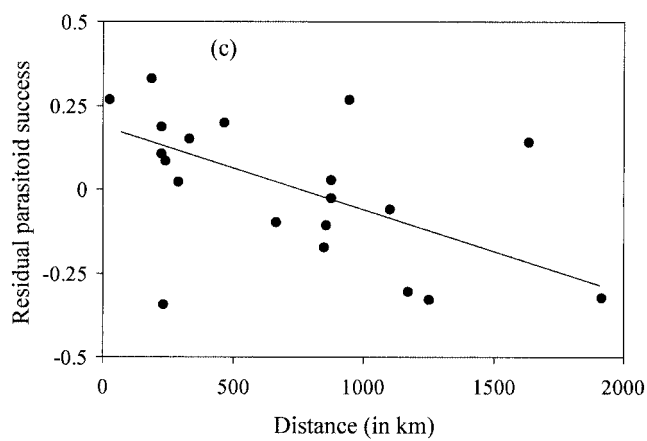
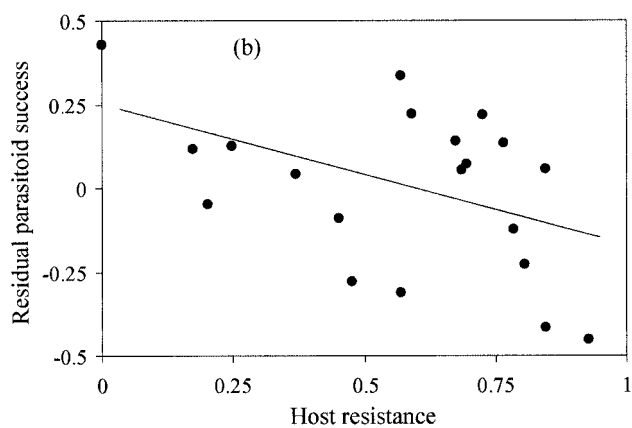
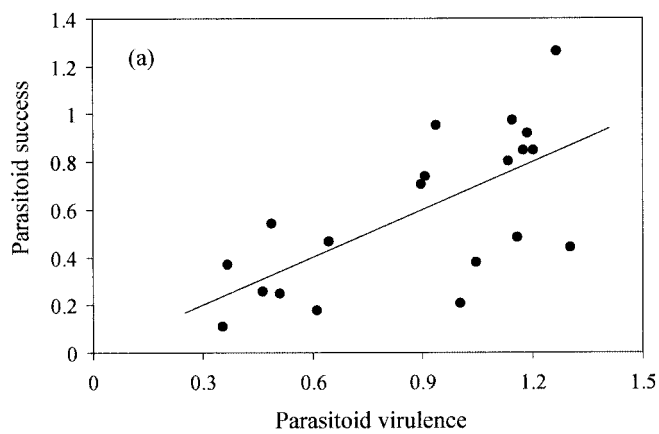
Using standard regression diagnostics, we checked for individual data points that were heavily influential. One observation that had a major effect was an interaction where the host showed no encapsulation against the reference strain of parasitoid (the host population came from the Greek island of Kos). We repeated the analysis omitting this point but found essentially the same result: the selected model, which contained the same three variables, was highly significant ($F_{3,15} = 10.10$, $P = 0.0007$) and explained 67% of the variance.

Turning to the analysis of the fraction of cases where both hosts and parasitoids died, the automated stepwise regression chose a model including just two of the seven potential explanatory variables: control host mortality (i.e. mortality in the absence of parasitism) and the mortality of the host population when parasitized by the reference strain of parasitoid. The addition of no other variable came close to improving significantly the explanatory power of the model ($P > 0.4$). The selected model was highly significant ($F_{2,17} = 8.93$, $P = 0.002$) and explained 51% of the variance in the data. The contributions of the two explanatory variables were remarkably similar: simple regressions of the response

Table 1. The data set used in the analyses

	A. Sympatric parasitoid success	B. Virulence	C. Resistance	D. Control mortality	E. Sympatric mortality	F. Mortality with reference host	G. Mortality with reference parasitoid	H. Distance to host reference (km)	I. Distance to parasitoid reference (km)
1	0.27	0.22	0.31	0.10	0.11	0.03	0.08	799	945
2	0.13	0.13	0.06	0.21	0.23	0.17	0.11	423	878
3	0.06	0.24	0.19	0.04	0.12	0.30	0.10	407	858
4	0.06	0.20	0.04	0.05	0.09	0.04	0.01	383	852
5	0.03	0.33	0.52	0.18	0.26	0.19	0.21	767	665
6	0.01	0.12	0.50	0.05	0.12	0.23	0.05	919	293
7	0.04	0.71	0.64	0.04	0.06	0.29	0.05	1134	1171
8	0.66	0.65	0.29	0.13	0.16	0.09	0.05	1246	186
9	0.56	0.87	0.56	0.11	0.20	0.10	0.07	1294	333
10	0.56	0.85	0.41	0.11	0.12	0.07	0.08	1262	226
11	0.51	0.82	0.40	0.13	0.22	0.10	0.09	1214	240
12	0.22	0.84	0.21	0.14	0.21	0.09	0.12	1230	233
13	0.45	0.62	0.39	0.22	0.29	0.08	0.09	1238	226
14	0.68	0.83	0.44	0.07	0.09	0.21	0.05	1230	27
15	0.63	0.86	0.48	0.24	0.16	0.04	0.09	1350	466
16	0.18	0.93	0.56	0.04	0.15	0.09	0.05	1917	1251
17	0.91	0.91	0.00	0.03	0.08	0.13	0.11	2444	1637
18	0.14	0.75	0.29	0.11	0.19	0.18	0.12	2668	1917
19	0.20	0.36	0.13	0.11	0.13	0.12	0.04	447	878
20	0.42	0.61	0.03	0.02	0.24	0.14	0.11	807	1104

Note: The columns are as follows: (A) The dependent variable: the fraction of parasitoids surviving in sympatric host–parasitoid associations. (B) The virulence of parasitoids when tested against a reference host strain (proportion escaping encapsulation). (C) The resistance of hosts when tested against a reference parasitoid strain (proportion encapsulating parasitoid). (D) Fraction mortality in the absence of parasitism. (E) Joint mortality of host and parasitoid in sympatric association. (F) Joint mortality of parasitoid with reference host strain. (G) Joint mortality of host with reference parasitoid strain. (H) Distance between the provenance of the sympatric and reference host populations. (I) The equivalent distance for the parasitoid populations.



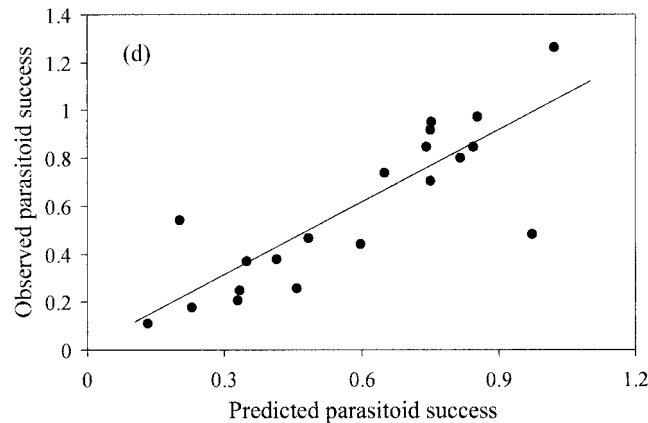


Fig. 1. The factors determining parasitoid success in sympatric host–parasitoid associations. (a) Parasitoid success plotted against parasitoid virulence measured against the reference host strain (both variables with angular transformation). (b) The residuals from (a) plotted against host resistance measured against the reference parasitoid strain (the latter with angular transformation). (c) The residuals from (b) plotted against the distance separating the sympatric and reference parasitoid strains. (d) The fit of the model containing all three explanatory variables.

variable against control host mortality were significant ($F_{1,18} = 9.66$, $P = 0.006$) and explained 35% of the variance in the data, while the equivalent values for a simple regression against mortality after parasitism by the reference strain were virtually the same ($F_{1,18} = 9.66$, $P = 0.006$, 35%).

DISCUSSION

There has been remarkably little study of local adaptation in host–parasitoid systems, despite its potential significance in biological control and pest management, and its importance in deciding how to model host–parasitoid co-evolution [for example, Doebeli (1997) and Sasaki and Godfray (1999) make different assumptions about how resistance and virulence traits determine parasitoid success]. Studies are required in which sets of host and parasitoid populations are tested in all allopatric and sympatric combinations (Kaltz *et al.*, 1999; Mutikainen *et al.*, 2000). We have been unable to do this; instead, we used data collected for different purposes to investigate the extent to which performance in sympatric associations can be predicted from measures of host resistance and parasitoid virulence obtained using standardized reference strains of hosts and parasitoids. If host resistance and parasitoid virulence are graded traits, analogous to anti-predator strategies such as thick shells and fast running speed, then information obtained using reference strains should be very efficient at predicting the outcome of sympatric associations. However, if there are complex co-adaptations among sympatric host and parasitoid populations, perhaps involving matching rather than graded traits, then performance against reference strains will be far less informative in predicting the outcome of local interactions. Our study is thus able to put bounds on the extent of local adaptation.

We found that over 56% of the variance in the outcome of sympatric associations can be explained by resistance and virulence measured against reference strains, and that the lion's

share of this variation can be attributed to differences in parasitoid virulence. A further 13% of the variance can be explained by the geographical distance between the provenance of the sympatric population being tested and the reference strain. We thought that this variable might act as a surrogate for local adaptation: if local adaptation is an important aspect of this system, the mismatch between host and parasitoid genotypes, and thus the difference between observed and predicted parasitoid success, should increase with geographical distance. However, the effect of this variable was in the opposite direction of that predicted and we suspect that this may reflect the previously identified geographical cline in parasitoid virulence across Europe (Kraaijeveld and Van Alphen, 1994). We conclude that the major determinant of the outcome of parasitoid attack in sympatric association is the absolute levels of virulence and resistance, with the former being of much greater importance, but stress that we cannot exclude a more minor role for local adaptations in the sense of Kaltz and Shykoff (1998).

Just over half the variation in host–parasitoid mortality (cases in which both host and parasitoid die) can be explained by two explanatory variables: mortality in the absence of parasitism and mortality when the host is attacked by the reference parasitoid strain. No other variable, including any of the parasitoid-associated factors, entered into the statistical model. This suggests that host–parasitoid mortality is largely a property of the host and its response to parasitism, and is not associated with the virulence of parasitoid attack.

One caveat about our analysis is that we treated the data from each sympatric host–parasitoid association as statistically independent data points. However, the data come from different spatial locations and it is possible that there may be spatial covariance in the error terms. We have insufficient data to examine this possibility at the present time, but we consider that it is unlikely to influence the above conclusions.

Although critical tests are still required, the available evidence points to *D. melanogaster* resistance against *A. tabida* being a graded trait without local adaptation. In addition to the data analysed here, Kraaijeveld and Godfray (1999) found that replicate lines selected for higher resistance against one strain of parasitoid (in fact, the reference strain used here) also performed better against parasitoids of the same species from Greece, the Netherlands and Canada. Green *et al.* (in press) maintained three outbred populations of *D. melanogaster* with different isofemale lines of *A. tabida* and found an increase in resistance over time but no adaptation to specific parasitoid genotypes. Our current knowledge of resistance and virulence mechanisms in these two species is also consistent with these being graded traits. Species of *Drosophila* with greater resistance against *A. tabida* have higher densities of circulating haemocytes (Eslin and Prévost, 1998), while an increase in haemocyte density occurs in lines selected for resistance to this wasp (Kraaijeveld *et al.*, in press). More virulent *A. tabida* strains have greater densities of proteinaceous filaments on the egg chorion, which cause the egg to stick to and become embedded in host tissue, away from the circulating haemocytes (Kraaijeveld and Van Alphen, 1994; Eslin *et al.*, 1996). We hypothesize that the density of circulating haemocytes, and the extent to which the parasitoid egg becomes embedded in host tissue, are graded traits that combine in a simple manner to determine the outcome of parasitoid attack.

The only other study to have examined local adaptation in parasitoids also concerned *Drosophila melanogaster* but a different parasitoid, the eucoilid *Leptopilina boulardi*. *L. boulardi* does not have adhesive eggs but counters the host immune system by injecting

virus-like particles that invade and disable host haemocytes. Carton (1984; see also Carton and Nappi, 1991) investigated strains of *D. melanogaster* and *L. bouleari* from Guadeloupe, southern France, Tunisia, Brazil and Italy in both allopatric and sympatric combinations. We (Kraaijeveld *et al.*, 1998) interpret the results to show that Guadeloupe and Brazil parasitoid strains cause substantially higher mortality in four allopatric host strains than in their sympatric host strain. However, given that the host survived parasitism, the probability of the parasitoid successfully emerging did not differ significantly in sympatric and allopatric hosts. This is a possible case of a locally adaptive response that has evolved to benefit both partners – both the fly and wasp survive this joint mortality to compete as normal for eventual dominion. Considering the very different virulence mechanisms in *A. tabida* and *L. bouleari*, and the recent demonstrations of differences in the genetic basis to resistance against these two wasps (Benassi *et al.*, 1998; Fellowes *et al.*, 1999), a comparison of the outcomes of sympatric and allopatric parasitism by these two parasitoids would be particularly interesting.

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