

Aphid population dynamics: does resistance to parasitism influence population size?

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Abstract. 1. Insect population size is regulated by both intrinsic traits of organisms and extrinsic factors. The impacts of natural enemies are typically considered to be extrinsic factors, however insects have traits that affect their vulnerability to attack by natural enemies, and thus intrinsic and extrinsic factors can interact in their effects on population size.

2. Pea aphids *Acyrtosiphon pisum* Harris (Hemiptera: Aphididae) in New York and Maryland that are specialised on alfalfa are approximately two times more physiologically resistant to parasitism by *Aphidius ervi* Haliday (Hymenoptera: Braconidae) than pea aphids specialised on clover. To assess the potential influence of this genetically based difference in resistance to parasitism on pea aphid population dynamics, pea aphids, *A. ervi*, and other natural enemies of aphids in clover and alfalfa fields were sampled.

3. Rates of successful parasitism by *A. ervi* were higher and pea aphid population sizes were lower in clover, where the aphids are less resistant to parasitism. In contrast, mortality due to a fungal pathogen of pea aphids was higher in alfalfa. Generalist aphid predators did not differ significantly in density between the crops.

4. To explore whether intrinsic resistance to parasitism influences field dynamics, the relationship between resistance and successful field parasitism in 12 populations was analysed. The average level of resistance of a population strongly predicts rates of successful parasitism in the field. The ability of the parasitoid to regulate the aphid may vary among pea aphid populations of different levels of resistance.

Key words. Aphid, biological control, parasitoid, population dynamics, resistance.

Introduction

The dynamics of insect populations have been studied for decades (reviewed by Price, 1997) but are still difficult to predict. Factors that influence population dynamics can be categorised as extrinsic or intrinsic (Price, 1997). Extrinsic regulators of population size are factors such as predation, and food and habitat availability. Intrinsic factors are genetically based attributes such as fundamental population growth rate, competitive ability, and vulnerability to attack by natural enemies. Sophisticated models have been developed from which predictions can be made and tested regarding the relative importance of different factors (reviewed by Royama, 1992; Cappuccino & Price, 1995; Price, 1997). These models

and classic empirical studies such as Morris's work on the fall web worm (e.g. Morris, 1967, 1971, 1972) and Price and colleagues' work on gall-forming sawflies (reviewed by Price, 1997) have provided insights that begin to explain the incredible variability in both time and space in the size of insect populations.

One area of research on population dynamics that has received less attention is the potential for interactions between intrinsic and extrinsic factors. It is recognised that extrinsic factors can shape intrinsic factors. For example, metapopulation ecologists perceive that environmental changes that lead to local expansions or extinction of subpopulations will affect the genetic structure of metapopulations (e.g. Hastings & Harrison, 1994), and extrinsic factors such as numbers of predators or competitors and availability of food resources influence the spatial distribution and organisation of individuals and genotypes within a population (Rhodes & Odum, 1996).

It is also possible for intrinsic factors to shape extrinsic regulators of population size. For example, the intrinsic ability

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of a species to evade or survive attack by natural enemies is likely to affect the extrinsic levels of predation and parasitism. By exploring interactions between intrinsic traits and extrinsic regulators of population size, the gap between genetics and population dynamics can begin to be closed. Several recent models have addressed this issue in host–parasitoid and predator–prey systems (Abrams & Matsuda, 1997; Doebeli, 1997; Holt & Hochberg, 1997; Sasaki & Godfray, 1999; Tuda & Bonsall, 1999; Fellowes & Travis, 2000) but there are few empirical data (but see Little & Ebert, 2000).

The work reported here explored whether the level of genetically based resistance to parasitism among populations of pea aphids *Acyrtosiphon pisum* Harris is correlated either with the rate of successful parasitism in the field or with aphid and parasitoid densities. The pea aphid is a species that comprises genetically differentiated races specialised on different crops (Via, 1991a,b, 1994, 1999; Sandstrom, 1994; Caillaud & Via, 2000; Via *et al.*, 2000; see Study system, below). These races differ in their susceptibility to parasitism by *Aphidius ervi* (Hymenoptera: Braconidae) (Hufbauer & Via, 1999; Hufbauer, 2001) but do not differ in average fecundity on their preferred host plant (Via, 1991a). This situation provides an opportunity to begin to examine the influence on population dynamics of genetically based differences among populations in resistance to a natural enemy. Survey data of pea aphid densities, rates of successful parasitism, and the densities of several natural enemies in fields of red clover and alfalfa are presented. Then it is explored whether differences in resistance to parasitism that have been documented in previous laboratory experiments (Hufbauer, 2001, in press) are correlated with differences in successful parasitism rates and population sizes in the field.

Materials and methods

Study system

Pea aphids are cyclically parthenogenetic: they reproduce asexually during the summer, passing through eight to 10 clonal generations in temperate areas. Sexual forms develop in the autumn and produce eggs that overwinter on the host plant (Lamb & Pointing, 1972); they do not alternate hosts. Pea aphids feed on herbaceous legumes and are pests of alfalfa *Medicago sativa* and red clover *Trifolium pratense*, which are grown for hay by dairy farmers. Pea aphids are native to Europe and Asia and were introduced to North America inadvertently in the 1800s (Angalet & Fuester, 1977).

In North America, populations of pea aphids found in alfalfa and clover in Iowa and New York are extremely specialised on one or the other crop (Via, 1991a, 1994; Caillaud & Via, 2000; Via *et al.*, 2000); many of the aphids collected from one crop will not feed or reproduce successfully on the other crop even after several generations of acclimatisation (Via, 1991b). Similar host-plant specialisation has been observed in Sweden (Sandstrom, 1994). Movement of these specialised races of pea aphids between alfalfa and clover is restricted due to habitat choice of alate migrants even when fields are adjacent to each

other (Via, 1999). This habitat choice contributes to assortative mating among the two races so that there is little gene flow across crops (Via, 1999).

Aphidius ervi is a solitary endoparasitoid of the pea aphid; the female wasp lays a single egg inside an aphid, the egg hatches, and the larva feeds on the aphid's internal tissues, eventually killing it. The parasitoid pupates inside the dried, hardened exoskeleton of the dead aphid, or mummy. In temperate areas, *A. ervi* has about 10 generations per year. This parasitoid was introduced into the north-eastern United States by the USDA in 1959 to control the pea aphid. It was collected from alfalfa fields in France for this release (Halfhill *et al.*, 1972; Mackauer & Campbell, 1972; Angalet & Fuester, 1977). Since *A. ervi* established, it has spread rapidly to become the dominant parasitoid of pea aphids in North America (Angalet & Fuester, 1977).

Pea aphids are genetically variable in their ability to resist parasitism by *A. ervi* (Henter & Via, 1995). This resistance is physiological and genetically based. Wasps attack resistant aphids, but their eggs do not hatch successfully (Henter, 1995; Henter & Via, 1995). Laboratory assays have demonstrated that pea aphid populations specialised on alfalfa are twice as resistant on average as aphids specialised on clover (Hufbauer & Via, 1999; Hufbauer, 2001, in press). The difference in resistance between the races of aphids on alfalfa and clover is due to differences in the physiological, genetically based resistance trait; it is not a function of aphid or wasp behaviour on the two plants, and is not induced by the aphids feeding on one host plant or the other (Hufbauer & Via, 1999).

Sampling regime

To place previous laboratory assays (Hufbauer & Via, 1999; Hufbauer, 2001, in press) on resistance to parasitism into an ecological context, aphids, parasitoids, and other natural enemies of aphids from three clover and three alfalfa fields in Tompkins County, New York (42.4°N, 76.5°W) were sampled. Each field was sampled approximately every 2 weeks (eight times) from late May to early September 1996. Rates of successful parasitism by *A. ervi*, *A. ervi* density, pea aphid density, rates of death due to the fungal pathogen *Erynia (Pandora) neoaphidis*, rates of successful parasitism by the less common parasitoids in the genus *Praon*, and the densities of five other groups of generalist aphid predators: spiders (Araneae), syrphid flies (Syrphidae), ladybird beetles (Coccinellidae), damsel bugs (Nabidae), and minute pirate bugs (Anthocoridae) were measured.

In addition to the field sampling regime in 1996, supplemental data on rates of successful parasitism in clover and alfalfa fields were collected in May 1997 in two clover and two alfalfa fields in Maryland, and June 1997 in two clover and two alfalfa fields in New York (different fields from those sampled in 1996). The sampling protocols were the same as in 1996 (detailed below) but the fields were sampled only once.

Rates of successful parasitism by *A. ervi* and *Praon* spp. and the proportion killed by the fungal pathogen were determined by rearing out aphids collected from the field. Third- and

fourth-instar aphid nymphs were collected from eight to 10 separate areas in each field using a beat tray. Sample locations were placed every 30–40 paces along three haphazardly chosen transects. When aphids were abundant, 20 or more nymphs were taken in each sample. When aphids were rare, an area of $\approx 2 \text{ m}^2$ was searched thoroughly for 10–12 min in each sample location before moving to the next location. The aphids in these collections were brought back to the laboratory and placed in cages on their appropriate host plant to mature. The proportion of the aphids sampled that became *A. ervi* mummies, *Praon* spp. mummies, or fungal cadavers was counted when the aphids had matured. The mummies of *Aphidius* and *Praon* parasitoids are easy to distinguish from each other.

Aphid densities were determined by beating aphids from the plants growing in a 20×10 -cm area onto a white tray. The number of aphids in the tray was recorded and the height of the vegetation was measured. Density was calculated as the number of aphids per beat, with plant height included in the analysis as a covariate. To account for variation in aphid density within a field, samples were taken from six to eight locations in each field (chosen as described for the aphid density samples above) on each sample date. The growth forms of clover and alfalfa differ and those differences could bias these estimates of aphid density, however initial sampling efforts in which the vegetation was cut off at ground level and aphids were subsequently counted in the laboratory showed relative abundance similar to beat sample counts (unpublished data). This method was abandoned because aphids were crushed in the process and could not be used for estimates of parasitism rates.

The abundance of adult *A. ervi* and the five groups of generalist aphid predators was determined from sweep net samples. Six to eight samples of 30 sweeps each were taken per field, and insects were counted on site. If there was any doubt as to the identity of an insect, it was brought into the laboratory for identification.

Analysis of sampling data

The proportion of aphids parasitised by *A. ervi*, the densities of pea aphids and *A. ervi*, and the proportion of pea aphids killed by the fungal pathogen in 1996 in New York were analysed using repeated measures mixed linear models (the Mixed Procedure; SAS Institute, Inc., 1997a). All of these models included the crop sampled (crop), and the sample period (week), which were treated as fixed effects, and the interaction between crop and week. The particular field on which the repeated measures were taken was nested within crop and treated as a random effect. The model for the aphid density data also included the height of the crop (crop height) as a covariate. Interactions between crop height and other terms in the model for aphid density were tested, and removed from the model if they were not significant. All of these models employed the first-order autoregressive [AR(1)] covariance structure, which treats measurements from adjacent sample dates as more strongly correlated than measurements from not-

adjacent sample dates (SAS Institute, Inc., 1997a). This covariance structure fitted the data better than other available covariance structures (i.e. it optimized the values of Akaike's Information Criterion and Schwarz's Bayesian Criterion; Littell *et al.*, 1996; SAS Institute, Inc., 1997a). To improve the normality of the residuals, aphid density (number of aphids per beat sample) and parasitoid density (number of *A. ervi* per sweep sample) were natural log transformed, and the proportion parasitised by *A. ervi* and the proportion killed by the fungal pathogen were arcsin-square-root transformed.

Because the proportion of aphids parasitised by *Praon* spp. and the densities of the five generalist predators in the sweep samples were low, these data were summed within each field through the season and ANOVAs to test for differences between alfalfa and clover were performed (JMP Version 3.2; SAS Institute, Inc., 1997b). A natural log transformation improved equality of variance of the summarised data, and a Bonferroni correction was used to adjust for multiple response variables (Neter *et al.*, 1990).

Can laboratory resistance explain successful parasitism in the field?

To examine more closely the relationship between resistance measured in the laboratory and successful parasitism rate in the field, the proportion of variation in successful field parasitism explained by the average resistance level of a population was explored. Data on laboratory resistance level from 12 populations of aphids for which there were also data on rates of successful parasitism in the field were used: two of the three alfalfa fields and two of the three clover fields sampled in 1996 in New York (Hufbauer, 2001), the two alfalfa and two clover fields sampled in May 1997 in Maryland (Hufbauer, 2001, in press), and the two alfalfa and two clover fields sampled in June 1997 in New York (Hufbauer, 2001, unpublished). See Hufbauer and Via (1999) and Hufbauer (2001) for methodological details on measurements of aphid resistance to parasitism. Briefly, genetically variable test groups representing field populations were constructed in the laboratory and exposed to attack by *A. ervi* in standard assays. Approximately 15 aphid clonal lineages from an individual field were combined to form the genetically variable test group representing that field. Aphids in the assays were reared and presented to wasps on the host plant from which they had been collected. The host plant upon which the aphids fed did not affect successful parasitism rate (Hufbauer & Via, 1999). Relative resistance is defined as the proportion of aphids parasitised successfully in these assays. Low rates of successful parasitism indicate that many of the aphids in a test group were able to prevent the parasitoid egg from developing and that the population is relatively resistant; high rates of successful parasitism indicate more susceptible aphids. The relationship between successful parasitism in the field and laboratory resistance was analysed in a model that included year and state. Because resistance was not a treatment imposed experimentally, but rather a variable measured with error, it was treated as a random effect (Sokal & Rohlf, 1995). The

other predictor variables (year and state) were also treated as random effects in order to allow generalisation across years and states. This analysis was performed using the Mixed Procedure (SAS Institute, Inc., 1997a), in which continuous variables such as laboratory resistance can be classified as random. A disadvantage of the Mixed Procedure is that it is not possible to calculate coefficients of partial determination (R^2) to examine the proportion of the variance in successful field parasitism explained by each significant predictor. To obtain estimates of these coefficients, an additional analysis of these data treating resistance, year, and state as fixed factors (JMP Version 3.2; SAS Institute, Inc., 1997b) was performed. The data from the aphids from alfalfa and clover were analysed both together and separately.

Results

1996 field samples

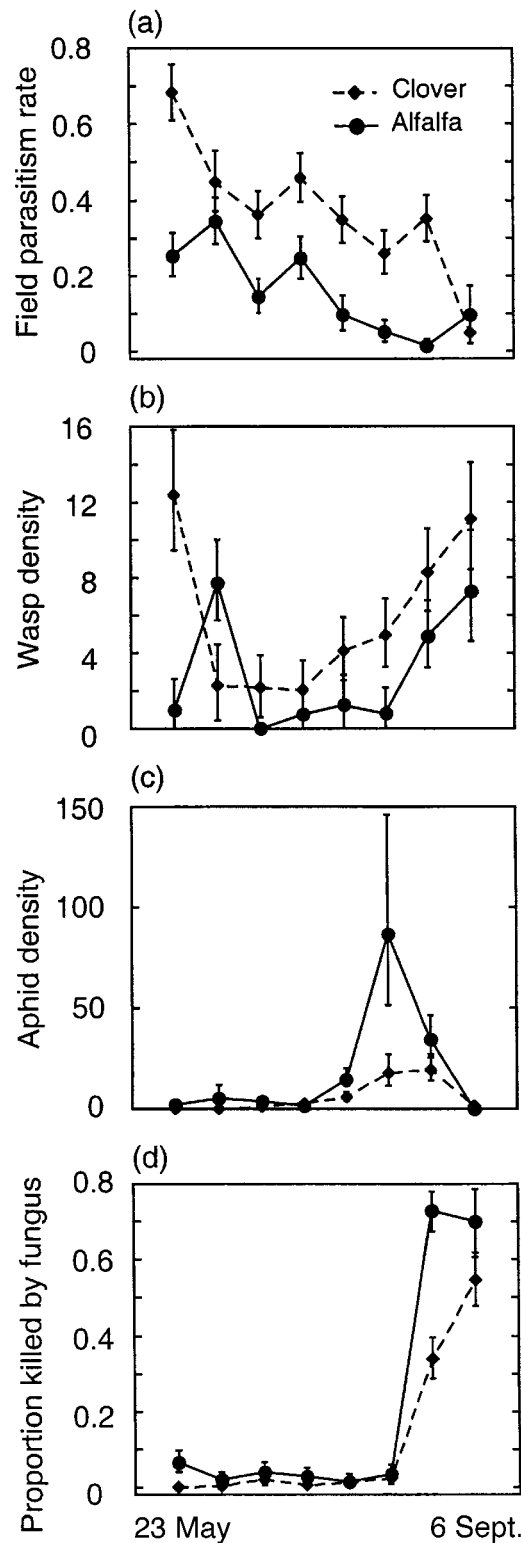
The rate of successful parasitism by *A. ervi* was affected significantly by crop, week, and their interaction. Successful parasitism rates were significantly higher in clover than in alfalfa throughout most of the season, and rates of successful parasitism in both crops decreased through the season (Table 1, Fig. 1a). The significant interaction between crop and week was due to the slightly higher rates of successful parasitism in clover than in alfalfa on the last sample date, and the faster decline in successful parasitism in clover (Table 1, Fig. 1a). Additional data on rates of successful parasitism from May 1997 in Maryland (mean \pm SE in alfalfa = 0.34 ± 0.04 ; clover = 0.56 ± 0.04) and June 1997 in New York

Table 1. Rate of parasitism in alfalfa and clover in Tompkins County, New York from May to September 1996. Data were arcsin-square-root transformed to improve the normality of the residuals.

Source	Test statistic		
Fixed effects	Type III <i>F</i>	d.f. ₁ , d.f. ₂	<i>P</i>
Crop	54.50	1,4	0.002
Week	10.40	1,22	0.0001
Crop \times week	2.84	1,22	0.029
Random effects	Likelihood ratio	d.f.	<i>P</i>
Field (crop) [AR(1)]	0.05	1	0.828

Fig. 1. Samples from three alfalfa fields and three clover fields from May to September 1996. Points are least squares means \pm 1 SE. See text for details. (a) Rate of parasitism of third-instar pea aphids by *Aphidius ervi*. (b) *Aphidius ervi* density (number per 10 sweep samples). (c) Pea aphid density (number per beat sample). (d) Proportion of third-instar pea aphids succumbing to the fungal pathogen *Erynia neoaphidis*.

(mean \pm SE in alfalfa = 0.27 ± 0.05 ; clover = 0.48 ± 0.05) showed the same basic pattern; rates of successful parasitism were higher in clover than in alfalfa.



The density of *A. ervi* was affected significantly by both crop and week but over the season parasitoid density was lower on average in alfalfa than in clover. Parasitoid density fluctuated throughout the season but these changes were roughly parallel in the two crops and there was no interaction between week and crop (Table 2, Fig. 1b).

The density of aphids was affected significantly by both crop and week, and by their interaction. On average, aphid density was higher in alfalfa than in clover (Table 3, Fig. 1c). There was a small but significant, positive relationship between aphid density and crop height (parameter estimate for crop height = 0.023 cm; Table 3).

Mortality of aphids in the samples due to the fungal pathogen *Erynia neoaphidis* was affected significantly by both crop and week, and by their interaction. On average, the fungal pathogen was more abundant and more prevalent at the end of the season in alfalfa than in clover (Table 4, Fig. 1d). There were no significant differences between alfalfa and clover in the densities of the five groups of generalist predators, or in the rate of successful parasitism by *Praon* spp. (Table 5).

Table 2. Parasitoid density (number per sweep sample) in alfalfa and clover in Tompkins County, New York from May to September 1996. Data were natural log transformed to improve the normality of the residuals.

Source	Test statistic		
Fixed effects	Type III <i>F</i>	d.f. ₁ , d.f. ₂	<i>P</i>
Crop	32.01	1,4	0.005
Week	12.52	1,18	0.0001
Crop × week	1.92	1,18	0.126
Random effects	Likelihood ratio	d.f.	<i>P</i>
Field (crop) [AR(1)]	5.11	1	0.024

Table 3. Aphid density (number per beat sample) in alfalfa and clover in Tompkins County, New York from May to September 1996. Data were natural log transformed to improve the normality of the residuals.

Source	Test statistic		
Fixed effects	Type III <i>F</i>	d.f. ₁ , d.f. ₂	<i>P</i>
Crop	21.89	1,4	0.010
Week	9.74	7,14	0.0002
Crop height	6.22	1,14	0.026
Crop × week	7.23	7,14	0.001
Crop height × week	8.42	7,14	0.0004
Random effects	Likelihood ratio	d.f.	<i>P</i>
Field (crop) [AR(1)]	9.64	1	0.002

Can laboratory resistance explain successful parasitism in the field?

The relationship between rate of successful parasitism in the 12 fields (alfalfa and clover combined) and average laboratory resistance of those populations was significant and strong (Table 6). More resistant populations had lower rates of successful parasitism; less resistant populations had higher rates of successful parasitism (Fig. 2). The fixed-effect model gave similar results (not shown). In this model, 60% of the variation in successful parasitism rates was explained by resistance to parasitism (Table 6). Analysing the data from the host-plant specialist populations of aphids separately showed no relationship between successful parasitism in the field and laboratory resistance among alfalfa specialists (Table 6, Fig. 2), while there was a very strong relationship between successful parasitism in the field and laboratory resistance among clover specialists (Table 6, Fig. 2). Among the clover populations, the

Table 4. Rate of death due to the fungal pathogen *Erynia neoaphidis* in alfalfa and clover in Tompkins County, New York from May to September 1996. Data were arcsin-square-root transformed to improve the normality of the residuals.

Source	Test statistic		
Fixed effects	Type III <i>F</i>	d.f. ₁ , d.f. ₂	<i>P</i>
Crop	23.58	1,4	0.008
Week	58.20	1,22	0.0001
Crop × week	2.5	1,22	0.047
Random effects	Likelihood ratio	d.f.	<i>P</i>
Field (crop) [AR(1)]	0.99	1	0.319

Table 5. Densities of five generalist predators and parasitism rates of *Praon* spp. in alfalfa and clover in Tompkins County, New York from May to September 1996. Data were analysed with simple one-way ANOVAs. To control for multiple comparisons, a significance level of $\alpha=0.05$ corresponds to a *P*-value of 0.004 using a Bonferroni correction.

Predator	Mean number per 30 sweeps		Type III <i>F</i> _{1,4}	<i>P</i>
	Alfalfa	Clover		
Spiders	0.26	0.38	1.10	0.353
Syrphid flies	0.27	1.58	2.25	0.208
Ladybird beetles	0.19	0.49	1.54	0.282
Damsel bugs	0.44	0.28	0.80	0.421
Minute pirate bugs	0.30	0.32	0.02	0.907
Parasitoid	Parasitism rate			
<i>Praon</i> species	0.06	0.05	0.12	0.751

Table 6. Random-effects model exploring the relationship between resistance measured in the laboratory and rates of parasitism in the field. R^2 values provided for significant effects were generated from a fixed-effects model and should be considered approximate. See text for details.

Source	Likelihood ratio	d.f.	<i>P</i>	R^2
Aphids from alfalfa and clover combined				
Laboratory resistance	19.89	1	<0.0001	0.60
Year	8.39	1	0.002	0.09
State	7.62	1	0.003	0.10
Aphids from alfalfa only				
Laboratory resistance	0	1	0.5	–
Year	1.52	1	0.109	–
State	1.14	1	0.143	–
Aphids from clover only				
Laboratory resistance	6.40	1	0.006	0.86
Year	0	1	0.5	–
State	1.12	1	0.146	–

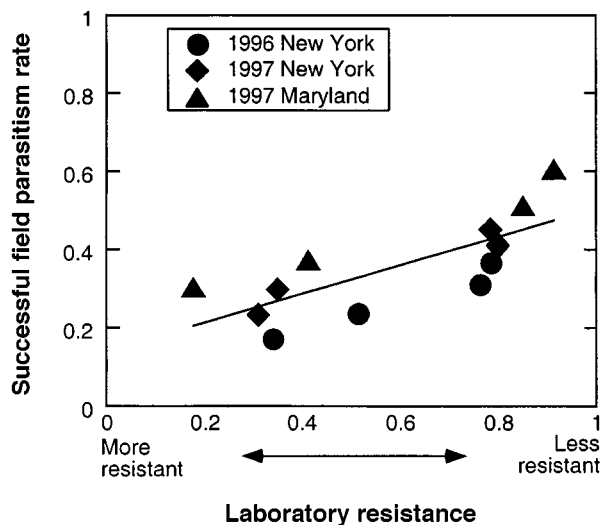


Fig. 2. Relationship between resistance to parasitism measured in the laboratory and rates of parasitism in the field for 12 populations, four from New York in 1996, four from New York in 1997, and four from Maryland in 1997 (successful field parasitism = $0.37 \times$ laboratory resistance + 0.14). The populations with resistance levels <0.6 are all from alfalfa, while populations with resistance levels >0.6 are all from clover. See text for details.

fixed-effect model showed that 86% of the variation in successful parasitism rates was explained by laboratory resistance to parasitism (Table 6).

Discussion

Successful parasitism of pea aphids by *A. ervi* was significantly greater in clover fields than in alfalfa fields. In 1996 in New York, parasitism rates decreased throughout the season but the

difference in parasitism between clover and alfalfa remained fairly consistent. A similar decrease in successful parasitism rate through the season was found by Henter (1995), and may be due to increased hyperparasitism by *Dendrocerus* spp. as the season progresses or to increased rates of fungal disease. Parasitoids may be less likely to oviposit in hosts with fungal infections (e.g. Brobyn *et al.*, 1988), and fungal infections of hosts can kill larval parasitoids (e.g. Powell *et al.*, 1986; Askary & Brodeur, 1999). Other natural enemies of pea aphids did not differ significantly in abundance between alfalfa and clover, except for the fungal pathogen, which was more prevalent in alfalfa than in clover.

The laboratory measurements of relative resistance to parasitism (Hufbauer, 2001, in press) explain much of the variation in the successful parasitism rate across fields reported here. This relationship holds across host-plant specialists and among clover specialists alone, but not among alfalfa specialists alone. The lack of a significant relationship between laboratory resistance and field parasitism in alfalfa may be a result of the greater variability in resistance level among alfalfa specialists than among clover specialists (Hufbauer & Via, 1999). In alfalfa, aphid clonal lineages range from absolutely resistant (0% successful parasitism) to 90% susceptible to parasitism (Henter & Via, 1995), while clover clonal lineages range from 57% to 91% susceptible. Thus, it may be that the assays of average level of resistance of a population are more prone to sampling effects in alfalfa where there is greater variability than in clover. The 15 clonal lineages representing each field population may not have been an accurate reflection of the average resistance of alfalfa populations. Nonetheless, the overall strong relationship between laboratory resistance and successful parasitism across the 12 fields suggests that the genetically based difference in resistance of these populations does influence rates of parasitism in the field.

Several factors are likely to interact with aphid resistance levels and contribute to the higher rates of successful parasitism of aphids in clover. For example, the higher density of *A. ervi* found in clover fields in 1996 could enhance parasitism rates there simply because there are more parasitoids searching for and attacking aphids. Also, parasitoid densities might be higher in clover than in alfalfa in part because they survive better on the susceptible aphids there. *Aphidius ervi* does not appear to discriminate against resistant aphid genotypes, and oviposits in them readily (Henter, 1995; Henter & Via, 1995; Hufbauer & Via, 1999); however the difference in resistance could conceivably select for parasitoids that discriminate between the crops, because those that prefer clover may have higher fitness. If this is the case, it would help to account for higher parasitoid densities and rates of successful parasitism in clover. The lower density of aphids in clover fields in 1996 could also contribute to the difference in successful parasitism rates; not only are there more parasitoids in clover but there are also more parasitoids per aphid. Thus, the genetically based difference in resistance between aphids from clover and alfalfa may interact in a feedback loop with other factors such as aphid and parasitoid density to determine rates of successful parasitism in field populations.

Can resistance to parasitism account for the lower densities of aphids in clover than in alfalfa? This question is of particular interest because of the pest status of pea aphids. The other natural enemies of pea aphids either did not differ significantly between crops or, in the case of the fungus, were more prevalent in alfalfa than in clover, and so do not help to explain the lower densities of aphids in clover. Furthermore, fecundity of alfalfa- and clover-specialist pea aphids is roughly equivalent when reared on their preferred host plants (Via, 1991a). Thus, the most obvious difference between the two crops that can help to explain lower aphid density in clover is the difference in successful parasitism rate between the two crops.

Data on rates of successful parasitism, however, can give misleading impressions of parasitoid impact (Van Drieshe, 1983; Van Drieshe *et al.*, 1991). In particular, other factors not measured in this study may also play a role in generating the observed differences in aphid density. In order to evaluate more conclusively whether there is a causal relationship between aphid resistance to parasitism in the laboratory, rates of successful parasitism in the field, and aphid population sizes, controlled field experiments are necessary. Because of the host-plant specificity of the aphids, the obvious experiment in which aphids from one host plant would be transferred to the other host plant to separate out the genetics of the aphids from other differences between the crops, is not feasible; however both host races of pea aphid feed and reproduce successfully on fava bean *Vicia fabae*, so replicate experimental patches of fava could be seeded with aphids of known resistance level to explore the influence of resistance on rates of successful parasitism, without the potentially confounding influences of other differences between clover and alfalfa. This would allow an experimental assessment of the role of physiological resistance to parasitism on pea aphid and *A. ervi* population dynamics.

The difference in mortality due to the fungal pathogen across crops is particularly intriguing. Pea aphids from alfalfa are known to have significant genetic variation in resistance to *Erynia (Pandora) neoaphidis* (Hural, 1998), and preliminary data suggest that there may be a negative genetic correlation between resistance to *A. ervi* and resistance to the fungal pathogen within alfalfa populations (Henter & Via, 1995). If this negative genetic correlation is present across host plants, an interesting possibility is that the low mortality due to the fungal pathogen observed in clover relative to alfalfa might reflect higher levels of resistance to fungal attack in the clover populations. This is not, however, the only explanation for differences in fungal mortality across crops: the higher density of aphids in alfalfa could contribute to fungal epidemics, and the denser growth of alfalfa relative to clover may provide an environment particularly suitable to the fungus.

There is a growing body of literature on variation among populations of potentially co-evolving species in intrinsic traits such as resistance to parasitism (e.g. Thompson, 1994, 1999; Dybdahl & Lively, 1996; Travis, 1996; Kraaijeveld *et al.*, 1998; Kraaijeveld & Godfray, 1999; Carton & Nappi, 2001). As that research, and research on the genetic structure of insect populations (e.g. Mopper & Strauss,

1998), is incorporated into work on population dynamics, a better understanding will emerge of how intrinsic, genetically based traits interact with extrinsic regulators of population size.

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References

- Abrams, P.A. & Matsuda, H. (1997) Prey adaptation as a cause of predator–prey cycles. *Evolution*, **51**, 1742–1750.
- Angalet, G.W. & Fuester, R. (1977) The *Aphidius* parasites of the pea aphid *Acyrtosiphon pisum* in the eastern half of the United States. *Annals of the Entomological Society of America*, **70**, 87–96.
- Askary, H. & Brodeur, J. (1999) Susceptibility of larval stages of the aphid parasitoid *Aphidius nigripes* to the entomopathogenic fungus *Verticillium lecanii*. *Journal of Invertebrate Pathology*, **73**, 129–132.
- Brobyn, P.J., Clark, S.J. & Wilding, N. (1988) The effect of fungus infection of *Metopolophium dirhodum* (Homoptera: Aphididae) on the oviposition behavior of the aphid parasitoid *Aphidius rhopalosiphii* (Hymenoptera: Aphidiidae). *Entomophaga*, **33**, 333–338.
- Caillaud, M.C. & Via, S. (2000) Specialized feeding behavior influences both ecological specialization and assortative mating in sympatric host races of pea aphids. *American Naturalist*, **156**, 606–621.
- Cappuccino, N. & Price, P.W. (eds) (1995) *Population Dynamics: New Approaches and Synthesis*. Academic Press, Inc., San Diego, California.
- Carton, Y.C. & Nappi, A.J. (2001) Immunogenetic aspects of the cellular immune response of *Drosophila* against parasitoids. *Immunogenetics*, **52**, 157–164.
- Doebeli, M. (1997) Genetic variation and the persistence of predator–prey interactions in the Nicholson–Bailey model. *Journal of Theoretical Biology*, **188**, 109–120.
- Dybdahl, M.F. & Lively, C.M. (1996) The geography of coevolution: comparative population structure for a snail and its trematode parasite. *Evolution*, **50**, 2264–2275.
- Fellowes, M.D.E. & Travis, J.M.J. (2000) Linking the coevolutionary and population dynamics of host–parasitoid interactions. *Population Ecology*, **42**, 195–203.
- Halfhill, J.E., Feathersen, P.E. & Dicke, A.G. (1972) History of *Praon*

- and *Aphidius* parasites of the pea aphid in the Pacific Northwest. *Environmental Entomology*, **1**, 402–405.
- Hastings, A. & Harrison, S. (1994) Metapopulation dynamics and genetics. *Annual Review of Ecology and Systematics*, **25**, 167–188.
- Henter, H.J. (1995) The potential for coevolution in a host–parasitoid system. II. 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. I. Genetic variation within an aphid population in susceptibility to a parasitic wasp. *Evolution*, **49**, 427–438.
- Holt, R.D. & Hochberg, M.E. (1997) When is biological control evolutionarily stable? *Ecology*, **78**, 1673–1683.
- Hufbauer, R.A. (2001) Pea aphid–parasitoid interactions: have parasitoids adapted to differential resistance? *Ecology*, **82**, 717–725.
- Hufbauer, R.A. Aphid resistance and parasitoid virulence among host races of the pea aphid: evidence for evolution following a biological control introduction. *Ecological Applications*. In press.
- Hufbauer, R.A. & Via, S. (1999) Evolution of an aphid–parasitoid interaction: variation in resistance to parasitism among aphid populations specialized on different plants. *Evolution*, **53**, 1435–1445.
- Hural, K. (1998) *Ecological genetics of the interaction between the pea aphid (Acyrtosiphon pisum) and its fungal pathogen, Pandora neoaphidis*. Doctoral dissertation, Cornell University, U.S.A.
- Kraaijeveld, A.R., van Alphen, J.J.M. & Godfray, H.C.J. (1998) The coevolution of host resistance and parasitoid virulence. *Parasitology*, **116**, S29–S45.
- Kraaijeveld, A.R. & Godfray, H.C.J. (1999) Geographic patterns in the evolution of resistance and virulence in *Drosophila* and its parasitoids. *American Naturalist*, **153**, S61–S74.
- Lamb, R.J. & Pointing, P.J. (1972) Sexual morph determination in the aphid *Acyrtosiphon pisum*. *Journal of Insect Physiology*, **18**, 2029–2042.
- Littell, R.C., Milliken, G.A., Stroup, W.W. & Wolfinger, R.D. (1996) *SAS System for Mixed Models*. SAS Institute, Inc., Cary, North Carolina.
- Little, T.J. & Ebert, D. (2000) The cause of parasitic infection in natural populations of *Daphnia* (Crustacea: Cladocera): the role of host genetics. *Proceedings of the Royal Society of London, Series B*, **267**, 2037–2042.
- Mackauer, M. & Campbell, A. (1972) The establishment of three exotic parasites (Hymenoptera: Aphidiidae) in British Columbia. *Journal of the Entomological Society of British Columbia*, **69**, 54–58.
- Mopper, S. & Strauss, S.Y. (eds) (1998) *Genetic Structure and Local Adaptation in Natural Insect Populations*. Chapman & Hall, New York.
- Morris, R.F. (1967) Influence of parental food quality on the survival of *Hyphantria cunea*. *Canadian Entomologist*, **99**, 24–33.
- Morris, R.F. (1971) The influence of land use and vegetation on the population density of *Hyphantria cunea*. *Canadian Entomologist*, **103**, 1525–1536.
- Morris, R.F. (1972) Predation by wasps, birds, and mammals on *Hyphantria cunea*. *Canadian Entomologist*, **104**, 1581–1591.
- Neter, J., Wasserman, W. & Kutner, M.H. (1990) *Applied Linear Statistical Models*. Richard D. Irwin, Inc., Boston, Massachusetts.
- Powell, W., Wilding, N., Brobyn, P.J. & Clark, S.J. (1986) Interference between parasitoids (Hymenoptera: Aphidiidae) and fungi (Entomophthorales) attacking cereal aphids. *Entomophaga*, **31**, 293–302.
- Price, P.W. (1997) *Insect Ecology*, 3rd edn. John Wiley and Sons, New York.
- Price, P.W., Bouton, C.E., Gross, P., McPherson, B.A., Thompson, J.N. & Weis, A.E. (1980) Interactions among three trophic levels: influence of plants on interactions between insect herbivores and natural enemies. *Annual Review of Ecology and Systematics*, **11**, 41–65.
- Rhodes, O.E., Jr & Odum, E.P. (1996) Spatiotemporal approaches in ecology and genetics: the road less traveled. *Population Dynamics in Ecological Space and Time* (ed. by O. E. Rhodes Jr, R. K. Chesser and M. H. Smith), pp. 1–7. University of Chicago Press, Chicago, Illinois.
- Royama, T. (1992) *Analytical Population Dynamics*. Chapman & Hall, London.
- Sandstrom, J. (1994) High variation in host adaptation among clones of the pea aphid, *Acyrtosiphon pisum*, on peas, *Pisum sativum*. *Entomologia experimentalis et applicata*, **71**, 245–256.
- SAS Institute, Inc. (1997a) *SAS/STAT Software: Changes and Enhancements through Release 6.12*. SAS Institute, Inc., Cary, North Carolina.
- SAS Institute, Inc. (1997b) *JMP Version 3.2*. SAS Institute, Inc., Cary, North Carolina.
- Sasaki, A. & Godfray, H.C.J. (1999) A model for the coevolution of resistance and virulence in coupled host–parasitoid interactions. *Proceedings of the Royal Society of London, Series B*, **266**, 455–463.
- Sokal, R.R. & Rohlf, F.J. (1995) *Biometry: the Principles and Practice of Statistics in Biological Research*, 3rd edn. W. H. Freeman, New York.
- Thompson, J.N. (1994) *The Coevolutionary Process*. University of Chicago Press, Chicago, Illinois.
- Thompson, J.N. (1999) The evolution of species interactions. *Science*, **284**, 2116–2118.
- Travis, J. (1996) The significance of geographic variation in species interactions. *American Naturalist*, **148**, S1–S8.
- Tuda, M. & Bonsall, M.B. (1999) Evolutionary and population dynamics of host–parasitoid interactions. *Researches on Population Ecology*, **41**, 81–91.
- Van Drieshe, R.G. (1983) The meaning of ‘percent parasitism’ in studies of insect parasitoids. *Environmental Entomology*, **12**, 1611–1622.
- Van Drieshe, R.G., Bellows, T.S., Jr, Elkinton, J.S., Gould, J.R. & Ferro, D.N. (1991) The meaning of percentage parasitism revisited: solutions to the problem of accurately estimating total losses from parasitism. *Environmental Entomology*, **20**, 1–7.
- Via, S. (1991a) The genetic structure of host plant adaptations in a spatial patchwork: demographic variability among reciprocally transplanted pea aphid clones. *Evolution*, **45**, 827–852.
- Via, S. (1991b) Specialized host plant performance of pea aphids clones is not altered by experience. *Ecology*, **72**, 1420–1427.
- Via, S. (1994) Population structure and local adaptation in a clonal herbivore. *Ecological Genetics* (ed. by L. Real), pp. 58–85. Princeton University Press, Princeton, New Jersey.
- Via, S. (1999) Reproductive isolation between symmetric races of pea aphids: gene flow restriction and habitat choice. *Evolution*, **53**, 1446–1457.
- Via, S., Bouck, A.C. & Skillman, S. (2000) Reproductive isolation between divergent races of pea aphids on two hosts. II. Selection against migrants and hybrids in the parental environments. *Evolution*, **54**, 1626–1637.

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