Effects of competition on resistance gene polymorphism in a plant/pathogen system

JOHANNE BRUNET* & CHRISTOPHER C. MUNDT

Department of Botany and Plant Pathology, Oregon State University, Cordley Hall 2082,
Corvallis, OR 97331-2902, U.S.A.

Puccinia striiformis, a fungal pathogen, has been shown to cause direct, negative frequency-dependent selection on its host, wheat (*Triticum aestivum*). This disease-induced frequency-dependent selection was not sufficient to maintain polymorphism for resistance genes in the host populations. The present study examines whether interactions between disease and competition could impact upon the maintenance of genetic polymorphism in a highly self-pollinated species such as wheat, where strong associations between traits are likely.

Four different two-way mixtures of wheat genotypes, susceptible to different races of *P. striiformis*, were planted at different frequencies in both the presence and absence of disease. In order to examine the influence of competition and disease on the maintenance of genetic polymorphism, relationships between host absolute fitness and host frequency were studied for each genotype in the mixtures of plants both in the presence and in the absence of disease. In the absence of disease, the absolute fitness of the stronger competitor was often negatively frequency-dependent, or else it did not vary with host frequency; the absolute fitness of the weaker competitor was often positively frequency-dependent. Disease typically rendered the slopes between absolute fitness and genotype frequency more negative for the stronger competitor. However, the influence of disease was not strong enough to reverse the sign of the slope between absolute fitness and genotype frequency for the genotype that was the weaker competitor in the absence of disease. Thus, disease was unable to reverse the relative ranking of the two genotypes caused by competition and create the negative frequency dependence on both genotypes in a mixture that is required for the maintenance of genetic polymorphism.

Keywords: absolute fitness, competition, genetic polymorphism, linkage disequilibrium, resistance genes.

While resistance genes bring a strong selective advantage to the host, phenotypic variation in disease resistance is commonly found in plant populations (Segal *et al.*, 1980; Alexander *et al.*, 1984; Parker, 1985; Harry & Clarke, 1986; Dinooor & Eshed, 1987; Burdon & Jarosz, 1988, 1991; Burdon & Thompson, 1995; Clarke, 1997). The discrete phenotypic variation in resistance is commonly caused by genes of major effect (Burdon, 1987, 1994; Harry & Clarke, 1987; Parker, 1988; Jarosz & Burdon, 1990; Alexander, 1992). Different hypotheses have been proposed to explain the maintenance of polymorphism for resistance genes, ranging from equilibrium (balancing selection), to nonequilibrium (transient polymorphism, past selection) conditions and involving direct or indirect selection (multilocus associations) (reviewed in Parker, 1992). While these hypotheses imply that forces within populations are

sufficient to maintain polymorphism, metapopulation explanations have also been proposed (Thompson & Burdon, 1992; Antonovics *et al.*, 1994; Burdon, 1997). Besides genetic models, ecological models have examined how differential pathogen impact could permit the coexistence of two or more plant species or genotypes with different competitive abilities in the absence of pathogens (Chilvers & Brittain, 1972; Burdon & Chilvers, 1977; Gates *et al.*, 1986).

We have demonstrated previously that host-specific races of *Puccinia striiformis*, the fungal pathogen that causes stripe rust, created direct negative frequency-dependent selection on its host, *Triticum aestivum* (wheat) (Brunet & Mundt, 2000). However, despite this pathogen-induced negative frequency-dependent selection, no genetic polymorphism was maintained in host populations (Brunet & Mundt, 2000). As our experiment used resistance genotypes of wheat planted in two-way mixtures, we hypothesized that nonrandom

*Correspondence. E-mail: brunetj@bcc.orst.edu

associations between resistance genes and genes for competitive ability may help explain the lack of genetic polymorphism despite the presence of disease-induced negative frequency-dependent selection (Brunet & Mundt, 2000). Wheat is a highly self-pollinated plant and nonrandom associations between traits (linkage disequilibrium) are commonly associated with this breeding system (Allard, 1988; Parker, 1992). In genotype mixtures competition is likely to influence the relationship between absolute fitness and host genotype frequency in the absence of disease, while both disease and competition will play a role in inoculated plots. When frequency-dependent selection maintains a genetic polymorphism, absolute fitness of a genotype is expected to decrease as its frequency increases and this is true for all genotypes in the mixture. In order to determine whether interactions between competition and disease influenced the maintenance of genetic polymorphism in our system, we examined the relationship between absolute fitness and host genotype frequency in both the absence and presence of disease for four different two-way mixtures of wheat resistance genotypes.

Materials and methods

The wheat/stripe rust system

Puccinia striiformis and wheat interact in a gene-for-gene system (Flor, 1971), where each avirulence gene in the pathogen interacts with one corresponding resistance gene in the host. Wheat is a predominantly self-pollinated plant and *P. striiformis* reproduces asexually. Stripe rust epidemics begin from small levels of infection in the autumn and increase through repeated cycles of asexual spores when temperatures increase during spring through to early summer (Shaner & Powelson, 1971).

Host genotypes

The four wheat genotypes used in the experiment are cultivated varieties (cultivars) of winter club wheat (*Triticum aestivum*) of similar height and maturity, except for the cultivar Jacmar, which is shorter than the other three. The wheat genotypes differ in their resistance to different races of *P. striiformis* and can be distinguished phenotypically with the use of chaff colour and phenol oxidation by seeds (Table 1). Commercial seed of the cultivar Faro in Oregon consists of a mixture of two morphologically indistinguishable lines with slightly different resistance spectra to *P. striiformis*, 'true Faro' and OR 7142 (a sibling of true Faro). We purified the line OR 7142, which is susceptible to *P. striiformis* race Cereal Diseases Laboratory (CDL) 29, and refer to this line as 'Faro'.

Table 1 Reactions to stripe rust (*Puccinia striiformis*) races and phenotypic markers for the wheat genotypes used in this study. The wheat genotypes in the mixtures were chosen such that each genotype could be identified with a genetic marker and each genotype in the mixture was susceptible to a different race of the pathogen

Wheat genotype	Reaction to <i>Puccinia striiformis</i> ^a			Phenotypic markers	
	Race 27	Race 29	Race 41	Chaff colour	Seed reaction to phenol
Faro	R	S	R	brown	light
Jacmar	R	S	S	brown	dark
Tres	R	R	S	white	light
Tyee	S	R	R	white	dark

^aS, susceptible; R, resistant.

Experimental location and design

Experimental plots were located at the Columbia Basin Agricultural Research Center field station at Pendleton, Oregon. The experiment entailed a randomized, split-plot design with four replications. The main plots were presence or absence of stripe rust, while subplots included four two-way mixtures (Faro–Tres, Faro–Tyee, Tres–Tyee, and Jacmar–Tyee), each planted at five frequencies of 10:90, 25:75, 50:50, 75:25, and 90:10 (Table 2). The mixtures were chosen based on reaction of the four wheat genotypes to available rust races, and availability of phenotypic markers to distinguish the wheat genotypes in the mixtures (Table 1). Both host genotypes in a mixture are susceptible, but each was affected by a different race of the pathogen. There were 20 subplots per mainplot, four mainplots inoculated with stripe rust, and four mainplots treated with fungicide to prevent infection, for a total of 160 subplots in the whole experiment.

Each subplot was 6.1 m-long and four rows wide, with 0.36 m between rows. To reduce travel of fungal spores among subplots and to facilitate harvest, each subplot was mowed to 4.9 m in length in the spring. Subplots were further separated in the narrow dimension by a subplot of the common soft white winter wheat cultivar Stephens, which is immune to the *P. striiformis* races used in this experiment. The planting density (253 seeds m⁻²), fertilization, and weed and insect control practices used were typical of commercial practice at that location.

The experiment was conducted over three years, with the first planting in the autumn of 1991 and the last harvest in the summer of 1994. For the autumn 1992 and 1993 plantings, seeds of each experimental host

Table 2 The field experiment had four replicates, with disease as the mainplot factor and wheat genotype and frequency as the subplot factors. All subplots within four of the mainplots were inoculated with stripe rust (I), all subplots in the remaining four mainplots were treated with fungicide to prevent disease (F). There were 20 treatments within each mainplot: four two-way mixtures, each planted at five different frequencies. For example, the frequency 10:90 for a mixture refers to 10% of genotype 1 and 90% of genotype 2

Mainplots

I	F	I	F
F	I	F	I
Rep1	Rep 2	Rep 3	Rep 4

Subplots within each mainplot

Genotype(s)	Genotype frequency (%)				
Faro–Tyee	10:90	25:75	50:50	75:25	90:10
Jacmar–Tyee	10:90	25:75	50:50	75:25	90:10
Tres–Tyee	10:90	25:75	50:50	75:25	90:10
Faro–Tres	10:90	25:75	50:50	75:25	90:10

population were harvested and carried over from the 1992 and 1993 summer harvests, respectively. Thus, frequencies of the host genotypes were allowed to change over years. Mixing the seeds from all four replications of each treatment, before they were packaged for replanting the next autumn, reduced the effect of genetic drift.

Field inoculations

All 20 treatments within each of four mainplots were inoculated by transplanting into the field plots wheat seedlings of the cultivar Nugaines infected with races 27, 29, and 41 of *P. striiformis* (Brunet & Mundt, 2000). The infected spreader plants were of the cultivar Nugaines because it is winter-hardy, susceptible to all three rust races, and supports profuse sporulation when temperatures are low. Two to three weeks after greenhouse inoculation, just prior to eruption of pustules through the plant epidermis, the spreader plants were transplanted into field plots at the rate of two pots per subplot. Spreader plants were placed in the centre of each plot. In the 1991–92 field season, spreader plants were transplanted on 19 March; in the 1992–93 field season, spreaders were transplanted on 10 November and 12 March; in the 1993–94 field season, a single inoculation occurred on 4 March. Although two inoculations (autumn and spring) increase the probability of establishing epidemics in the plots, adequate quantities

of rust were not always available for the autumn inoculation. The spreader plants served to establish disease in the plots, while subsequent epidemic development was primarily the result of spread among other plants in the plots. Epidemic progression of rust diseases is very insensitive to the amount of initial infection (Vanderplank, 1963). Hence, the presence of the spreader plants and possible initial differences in frequency among races on the spreader plants are expected to have a small impact on subsequent epidemic progression.

All subplots that were not inoculated received three applications of the fungicide triadimefon (Bayleton) to prevent the development of rust. This procedure prevents significant rust development and has not influenced the yield of these wheat genotypes in the absence of disease at Pendleton in previous seasons (Mundt *et al.*, 1995). We detected no interference from other pathogens or from outside sources of *P. striiformis*.

Field sampling

For a subset of the experimental plots, we calculated the area needed to contain 120 tillers (on average) per subplot from the two middle rows. Disease readings and host genotype frequency were estimated using all tillers within each miniplot. Miniplots of the desired size were then set up in the two middle rows of each subplot, midway between the centre and the downwind end of each subplot. Miniplot sizes were 48 cm long in both 1992 and 1993, and 41 cm long in 1994.

Genotype frequencies

All mature tillers within each miniplot (all disease and no-disease treatments) were hand-harvested with a sickle, bagged, and transported to the laboratory where they were separated by chaff colour. The total number of white and brown tillers in each miniplot was recorded. White and brown tillers were threshed separately with a stationary plot thresher, and the weight of seeds from the bulk of white or brown tillers was recorded separately. To determine the number of seeds in each of these samples, two sets of 100 seeds were counted and weighed. The mean seed weight was calculated and used to determine the total number of seeds per sample. For the genotypes that could be distinguished by chaff colour, in any given year, genotype frequency at planting was calculated using the total number of seeds harvested per genotype per treatment the previous summer (four replicates). A phenol oxidation assay was used to distinguish between the white-chaffed genotypes Tres and Tyee (Brunet & Mundt, 2000).

In a given year, the absolute fitness of a genotype was calculated using the number of seeds collected in a miniplot for that genotype in July of that year, divided by the number of seeds planted in the miniplot for that genotype the previous Autumn. Seeds were planted at a constant density each year (253 seeds m^{-2}). The number of seeds planted per genotype was calculated as: miniplot size (m^2) $\times$ planting density (seeds m^{-2}) $\times$ planted genotype frequency. There were four replicates, hence four measures of absolute fitness per genotype per treatment each year. Because wheat is an annual this measure of absolute fitness represents fitness over the lifetime of a genotype.

Data analyses

The relationship between host absolute fitness and host genotype frequency was examined by calculating regression coefficients for each genotype, in each mixture, for both the disease and no-disease treatments. Both linear and second-order polynomial regressions were tested and the model best fitting the data is presented in each case. Regressions were performed using all replicates and block effects were removed from the regression sums of squares using analysis of variance (Proc GLM: SAS, 1985). As disease levels were very low in 1992 and did not influence absolute fitness (Brunet & Mundt, 2000), only data from 1993 and 1994 are presented here.

Results

For the stronger competitor in the absence of disease, the slope between absolute fitness and genotype frequency is negative and statistically significant, or not significantly different from zero (Figs 1 and 2, stronger competitor). For the weaker competitor in the absence of disease, the slope is positive and statistically significant, or not statistically different from zero (Figs 1 and 2, weaker competitor). When disease was present, the slope between absolute fitness and genotype frequency often becomes more negative for the genotype that was the stronger competitor in the absence of disease, and less positive for the genotype that was the weaker competitor in the absence of disease (Figs 1 and 2). When both genotypes were predicted to be maintained in the population at equilibrium, slopes between absolute fitness and genotype frequency are negative and statistically significant or not significantly different from zero; the slopes are never positive and statistically significant (Figs 1 and 2). Competitive outcomes are based on relative fitness analyses of Brunet & Mundt (2000).

Discussion

The presence of disease-induced, negative frequency-dependent selection has been demonstrated previously in this system (Brunet & Mundt, 2000). The relative fitness of a host genotype was shown to decrease as its frequency increased, and this occurred only in the presence of disease (not in its absence) (Brunet & Mundt, 2000). The presence of negative frequency-dependent selection does not necessarily imply the maintenance of a genetic polymorphism in the population (Antonovics & Kareiva, 1988). The relative fitness of a genotype can decrease as its frequency increases, but a genetic polymorphism will be maintained only when the relative fitness of the genotype is greater than 1.0 when the genotype is at low frequency, and smaller than 1.0 when the genotype is at high frequency. The genotype frequency at which the relative fitness regression line crosses the line of 1.0 relative fitness indicates the genotype frequency expected at equilibrium in the population (Brunet & Mundt, 2000). When a genetic polymorphism is maintained by negative frequency-dependent selection one expects the absolute fitness of a genotype to decrease as its frequency increases and this will be true for each genotype in the mixture.

In our study, in the presence of disease a negative relationship was often observed between absolute fitness and host genotype frequency for the host genotype that was the stronger competitor. For the genotype that was the weaker competitor, however, the relationship between absolute fitness and host genotype frequency was either positive or not statistically significant. To help understand why the presence of disease did not create a negative relationship for the genotype that was the weaker competitor in the absence of disease, we compared the relationships between absolute fitness and host genotype frequency between the no disease and disease treatments.

If resistance genes were the only genetic traits influencing genotype fitness in this system, one would expect no relationship between genotype frequency and absolute fitness in the absence of disease. In our study, in the absence of disease the slope between absolute fitness and genotype frequency was often negative for the stronger competitor and positive for the weaker competitor. These results suggest that the stronger competitor was better at intergenotypic than intragenotypic competition, while the reverse was true for the weaker competitor. When a genotype is at low frequency, it interacts more with individuals of different genotypes, and intergenotypic competition is greater than intragenotypic competition. The opposite trends are expected when a genotype is at high frequency. The change in relationship between absolute fitness and genotype

Fig. 1 Absolute fitness (number of seeds harvested per seed planted) vs. planted frequency of each genotype in the Faro–Tres and Faro–Tyee mixtures in (A) No disease plots (treated with fungicide to prevent disease) and (B) disease plots (inoculated with stripe rust), in 1994 and 1993. Titles indicating competitive outcomes are based on relative fitness analyses of Brunet & Mundt (2000). Regressions (solid lines) were performed on four replicates and block effects were removed from the regression sums of squares using analysis of variance. The diamonds represent the means of four replicates and the error bars show the associated standard deviations. The slope of each regression (b), its significance level (P), and the proportion of the variance in absolute fitness explained by the model (r^2) are presented on each graph. Both linear and second-order polynomial regressions were fitted to the data and the best model is presented in each case. When the polynomial regression model was significant ($P < 0.05$), an asterisk associated with the linear (first-order) or quadratic (second-order) coefficients indicates statistical significance of that coefficient.

frequency between the stronger and weaker competitor can be observed for the Faro–Tres mixture. In 1994, Tres was the weaker competitor and, in the absence of

disease, the relationship between absolute fitness and genotype frequency was positive for Tres. In 1993, for the same genotype mixture, Tres was the stronger

Fig. 2 Absolute fitness (number of seeds harvested per seed planted) vs. planted frequency of each genotype in the Jacmar–Tyee and Tres–Tyee mixtures in (A) No disease plots (treated with fungicide to prevent disease) and (B) disease plots (inoculated with stripe rust), in 1994 and 1993. See Fig. 1 for details.

competitor and the relationship between absolute fitness and genotype frequency was negative (Fig. 1). In the absence of disease, competition influenced the relationship between absolute fitness and host genotype frequency.

When disease was present, the slopes of the regression between absolute fitness and host genotype frequency often became more negative than when disease was absent, for the genotype that was the *stronger* competitor in the absence of disease. For the genotype that was the *weaker* competitor in the absence of disease, disease often rendered the slope between absolute fitness and genotype frequency less positive, but the influence of disease was not strong enough to reverse the sign of the slope (from positive to negative). Only for Jacmar (weaker competitor in the absence of disease) in the Jacmar–Tyee mixture in 1994 did we observe a tendency towards a negative slope in the presence of disease. Jacmar was susceptible to two races of stripe rust in our experiment and disease severity was often greater on that cultivar (Brunet & Mundt, 2000). However, greater disease severity on Jacmar has been observed previously in studies where Jacmar was inoculated with a single virulent race (Finckh & Mundt, 1992). Whether or not greater susceptibility to disease implies a greater rare genotype advantage requires further testing.

Interactions between competition and disease may help explain why, in the presence of disease, host absolute fitness did not decrease as host genotype frequency increased for the genotype that was the weaker competitor in the absence of disease. When a genotype is a stronger competitor its absolute fitness is expected to be higher than the absolute fitness of the weaker competitor at all frequencies, as the stronger competitor is predicted to dominate in the population. For a genetic polymorphism to be maintained in the population, the absolute fitness of the weaker competitor must become greater than the absolute fitness of the stronger competitor when the weaker competitor is at low frequency and the stronger competitor is at high frequency. As a consequence of the rare genotype advantage created by disease, a genotype is expected to have less disease and hence greater absolute fitness when at low than at high frequency. Because the stronger competitor is at high frequency when the weaker competitor is at low frequency in the mixture, disease severity is expected to be higher on the stronger competitor; this will decrease the absolute fitness of the stronger competitor. If the impact of disease is such that the absolute fitness of the weaker competitor when rare becomes greater than the absolute fitness of the

stronger competitor when common, then a genetic polymorphism is expected to be maintained in the population. Before such a polymorphism can be maintained in the presence of disease, however, the rare genotype advantage for the weaker competitor and the negative fitness impact of disease on the common stronger competitor must reverse the relative fitness ranking of the two genotypes between the disease-absent and disease-present treatments. The stronger the differences in competitive abilities among genotypes, the stronger the differential influence of disease must be before a genetic polymorphism can be maintained in the host population (Gates *et al.*, 1986). In our study, the impact of disease on host fitness was not strong enough to reverse the relative fitness ranking of the two genotypes that was created by competition (in the absence of disease). Hence, genetic polymorphisms were not maintained in the host populations despite the presence of disease-induced frequency-dependent selection.

In our experiment we tested resistance genotypes rather than compare the effects of resistance genes in a common genetic background. Hence, any trait correlated strongly with the resistance genes could influence how disease influenced host fitness. Such nonrandom associations between traits (linkage disequilibrium) are common in highly self-pollinated species such as wheat and in asexual species (Allard, 1988; Parker, 1992). Our results in the absence of disease suggest strongly that competition influenced genotype fitness and the relationship between genotype frequency and absolute fitness. In natural populations disease may not act alone in its effect on host genotype fitness and factors such as competition can influence the potential for maintenance of genetic polymorphism in the presence of disease. As nonrandom associations between resistance genes and other traits (linkage disequilibrium) will be more common in asexual and highly self-pollinated species, disease-induced frequency-dependent selection may be more likely to maintain genetic polymorphism in outcrossing species. However, even in highly outcrossed species, disease is not expected to maintain genome-wide variability; the effect of disease on genetic variability will tend to be limited to the portion of the genome involved in gene-for-gene interactions.

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