Genetic Variation for Pathogen Resistance in Tall Morningglory

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ABSTRACT

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An experimental population of tall morningglory (*Ipomoea purpurea*) was established from the progeny of a replicated diallel design to assess the extent of heritable genetic variation in damage by the naturally occurring fungal pathogens *Colletotrichum dematium* and *Coleosporium ipomoeae*. Resistance to both pathogens varied significantly among paternal half-sib families, indicating significant levels of heritable variation in these traits. In addition, resistance to *Coleosporium ipomoeae* appears to be controlled at least in part by a major gene with a dominant resistance allele. These results suggest that increased resistance to *Coleosporium ipomoeae* and *C. dematium* would evolve if either was used as a mycoherbicide to control tall morningglory. The importance of these results to the durability of mycoherbicides is discussed.

Like other types of parasites, plant pathogens are presumed to exert a major influence on the structure of populations and communities of their hosts (8,9). An understanding of how plant defenses against pathogens evolve is therefore a requisite for understanding many aspects of plant ecology. Moreover, with the advent of mycoherbicides to control weeds (46), understanding how natural plant populations evolve resistance to pathogens has become particularly important for agronomists. Despite the ecological and agronomic importance of pathogen resistance, much less is known about the genetics of such characters in natural plant populations than in crops (5,8,14).

Many pathogenic fungi are being investigated as potential mycoherbicides (4,6,10,13,21,24,33,43). In contrast to classical biocontrol techniques, mycoherbicide technology involves augmenting populations of indigenous fungal pathogens by culturing them on a large scale and applying them as inundative inoculum in the manner used for chemical herbicides (46). Mycoherbicides have several advantages over traditional chemical herbicides. They are less likely to be toxic to human consumers, environmental effects are likely to be reduced or absent, and particular fungal pathogens can be used to target specific weeds, leaving the desired crop undamaged.

One major disadvantage of all pesticides is that by reducing the fitness of susceptible individuals of the target pest population, treatment imposes strong selection favoring resistant individuals. If such individuals exist in the population, that is, if the population exhibits additive genetic variance for resistance to the pesticide, then it will quickly evolve resistance to the pesticide (7,20,35). Unfortunately, if mycoherbicides are effective weed control agents, they are unlikely to be exempt from this problem.

The objective of this study was to examine the level of genetic variation in resistance to infection by natural inoculation of two fungal pathogens with the potential as mycoherbicides in an experimental population of tall morningglory (*Ipomoea purpurea* (L.) Roth). If such variance in resistance to a pathogen were found, it would suggest that a mycoherbicide based on that pathogen would not be durable in the morningglory population from which the experimental population was sampled.

The tall morningglory is a noxious annual weed in corn, soybeans, and cotton in the southeastern United States (31). Resistance to two of several fungal pathogens that attack *I. purpurea* has been observed in North Carolina tall morningglory populations (22). *Coleosporium ipomoeae* (Schwein.) Burrill, a heteroecious rust fungus in the Melampsoraceae, heavily infects tall morningglory leaves late in the season. This fungus undergoes sexual reproduction on an alternate host in the genus *Pinus* (probably *P. echinata* Mill. and *P. taeda* L. in the piedmont region of North Carolina). Spores are aerially dispersed, and populations build throughout the summer to high levels when leaves are senescing.

Another important pathogen is *Colletotrichum dematium* (Pers.) Grove f. *ipomoeae* Arx nom. nud., an imperfect fungus that causes anthracnose. The fungus overwinters on dried infected leaves in the soil (1) and infects *I. purpurea* through rain splash, appearing after warm summer rains as small regular lesions on young leaves. Many species of *Colletotrichum* are being investigated as potential mycoherbicides to control weeds (11,12). One commercially developed mycoherbicide is Collego, which contains *C. gloeosporioides* (Penz.) Penz. & Sacc. in Penz. f. sp. *aeschynomene* and is used in the control of northern jointvetch (*Aeschynomene virginica* (L.) B.S.P.) (16,44,45). *C. capsici* (Syd.) E.J. Butler & Bisby has been considered as a potential mycoherbicide against *I. lacunosa* L. (11), and *C. dematium* may be effective against *I. purpurea* (T. Vision and E. L. Simms, *unpublished*).

MATERIALS AND METHODS

Study site. This experiment was performed in an old agricultural field of sandy loam in Durham County, North Carolina, that had last been planted in soybeans in 1983 but had since only been disked every spring. The natural populations of *I. purpurea, Coleosporium ipomoeae*, and *C. dematium* provided all the material involved in the experiments described below.

Field protocol. The experimental plants were the same as those analyzed previously by Simms and Rausher (40, 41). Seeds were obtained from a replicated partial-diallel cross involving 30 parental plants. There were three diallels, each involving a different set of 10 plants. Each plant was crossed as a female with five of the nine other plants in the same diallel and also crossed as a male with five of the plants in the diallel. Because two plants in one of the diallels failed to produce seed in time for planting, there were ultimately 5,600 seeds from 140 full-sib families distributed among 30 male half-sib and 28 female half-sib families. The design also produced one reciprocal full-sib family for each parent plant.

Forty seeds from each full-sib family in this mating design were sandpaper-scarified and planted during 7–10 July 1985 into Roottrainers (Spencer-Lemair Industries, Ltd.) filled with potting mix. On 24–30 July, when seedlings had reached the two-leaf stage, they were transplanted into the freshly disked field. Plants from each full-sib family were randomly allocated among four spatial blocks and placed in a square grid of points 0.7 m apart. Plants were allowed to climb up 1-m tall bamboo stakes to mimic natural growth in cornfields. Natural vegetation in the field was not weeded during the experiment. Warm rains during late July and early August permitted infection of plants from the indigenous population of *C. dematium*

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in decomposing plant material in the soil.

In late August a census was performed to estimate resistance to *C. dematium* and *Coleosporium ipomoeae* on a subset of 2,800 plants in the field (20 plants from each full-sib family). Resistance to *C. dematium* was estimated as the complement of the proportion of total leaf area occupied by anthracnose lesions. A clear plastic grid with 0.6 cm^2 squares was placed over each leaf and the number of squares, to the nearest quarter-square, that covered damaged and undamaged portions of a leaf were counted. Leaf area damaged was summed over all leaves on a plant to yield total damage, then divided by total leaf area to yield proportion damage. The complement of this value was arcsine-square root-transformed for statistical analysis to normalize the residuals. Because the leaf measurements were performed before any leaves senesced, we did not need to correct this measure for senescence. Resistance to early infection by *Coleosporium ipomoeae* was assessed simply as presence or absence of rust pustules on each plant. Because rust infection often increases with plant maturity, this measure may not be indicative of resistance later in the season. In each case, the operational definition of resistance encompassed both active and passive mechanisms of disease resistance (8), including escape from disease.

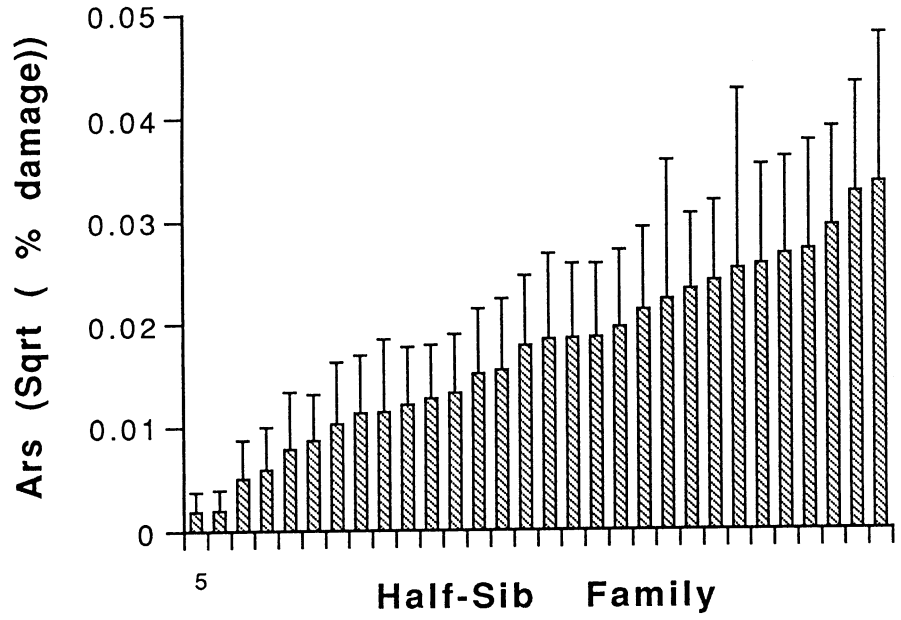

Fig. 1. Average level of damage by anthracnose exhibited by each sire half-sib family. Values were arcsine-square root-transformed as in the analysis of variance used for estimating heritability.

Table 1. Analysis of variance table for damage in *Ipomoea purpurea* due to natural infection by *Colletotrichum dematium* using the entire diallel

Source	df	Type III SS	*F*	*P*
Block	3	0.81584	64.59	0.0001
Diallel	2	0.02379	2.82	0.0596
Sire (diallel)	27	0.19507	1.72	0.013
Dam (diallel)	25	0.13402	1.27	0.2
Sire × dam (diallel)	85	0.37389	1.04	0.4
Error	1,452	6.11311		
Sire (diallel)[a]	27	0.19507	1.35	0.23
Error	25	0.13402		

[a] Test of hypothesis that sire within diallel and dam within diallel do not differ (using sire [diallel] type III mean square as an error component).

Table 2. Analysis of variance table for damage in *Ipomoea purpurea* due to natural infection by *Colletotrichum dematium* using only reciprocal families[a]

Source	df	Type III SS	*F*	*P*
Block	3	0.14886	10.25	0.0001
Diallel	2	0.03262	3.42	0.034
Family (diallel)	10	0.07330	1.54	0.126
Recip (diallel × family)	13	0.06621	1.07	0.39
Error	270	1.28753		

[a] A significant reciprocal within diallel × family interaction would indicate that offspring within a single half-sib family differ because of the identity of their seed parent and that maternal effects are significant.

Statistical analysis. Analysis of resistance to *C. dematium* followed standard procedures from analysis of variance for factorial designs (38). A significant sire main effect indicates additive genetic variance for resistance (18). The expected mean square associated with the dam effect includes both additive genetic variance and maternal effects. The expected mean square for the sire × dam interaction includes mainly nonadditive genetic variation (epistasis and dominance).

Reciprocal effects were examined in two ways. First, the ratio of dam mean square over sire mean square was tested; a ratio significantly greater than one indicates that the dam mean square exceeds that of the sire because of maternal-effect variance. Second, a separate paired analysis of variance of reciprocal full-sib families was performed to determine whether offspring from the same full-sib family differ depending upon the identity of their seed parent. Environmental variation appears in the block main effect and error sums of squares. All analyses of variance were performed with the general linear model (GLM) procedure of the SAS statistical packages using type III sums of squares (37).

The VARCOMP procedure of SAS (36) was used to estimate the variance components for calculating heritability. The narrow-sense heritability is defined as $h^2 = V_A / V_P$, in which V_A is the additive genetic variance component and V_P is the total phenotypic variance (18). The 95% confidence interval (CI) around the heritability estimate was produced by a remove-one-paternal-half-sib-family jackknife procedure (15,17).

Resistance to *Coleosporium ipomoeae* was assessed simply as presence or absence, which is a categorical, bivariate variable. Consequently, standard analysis of variance could not be used to estimate heritability in resistance to this fungus. Instead, a logistic analysis of variance was performed using the CATMOD procedure of SAS (36). All null hypotheses that the probability of infection was independent of a particular class variable were tested with maximum likelihood statistics. The analysis of variance (ANOVA) table resulting from a logistic model can be interpreted in essentially the same way as a standard ANOVA table. A chi-square goodness-of-fit test was performed on the progeny from reciprocal full-sib families to determine whether maternal effects were important.

RESULTS

Damage by *C. dematium* was continuously distributed among individuals

in this experimental population of *I. purpurea* (Fig. 1), suggesting that the trait is controlled by the small effects of a large number of genes (18). The plant population exhibited additive genetic variation for damage by *C. dematium* (sire within diallel effect in Table 1). There was no evidence of any maternal effects on damage by this pathogen (Tables 1 and 2). An insignificant interaction term also suggests that dominance and epistatic components of variance were minimal and that essentially all of the genetic variance was additive. Heritability was low ($h^2 = 0.06$) but significantly different from zero (95% CI = 0.05). Low heritability was likely due in part to environmental variation at the spatial scale of the experimental block, as indicated by the significant block main effect.

Initial observation of the proportions of progeny with rust infection for each full-sib family revealed that two parent plants produced almost no infected offspring (Fig. 2). This observation suggests that these plants each possessed a single dominant allele for resistance that was apparently absent in other parents. This observation alone indicates the presence of genetic variation in the probability of infection by rust in this population. Unfortunately, further analysis of the data including these families is hampered because the logistic analysis of variance necessary for analyzing this categorical variable treats cells (full-sib families) with zero frequencies as structural zeros (36) and thus cannot be used to test whether the probability of infection by rust is heritable. To deal with this impediment, offspring of the two resistant parents were removed from the dataset and logistic analysis of variance was used to determine whether the remaining families exhibited heritable variation for resistance. The maximum likelihood method for testing hypotheses is computer-intensive, and limitations on computer resources necessitated analyzing the data in several steps. The first step involved determining whether the spatial block in which a plant occurred or the diallel in which its parents were crossed were significant factors determining its level of resistance. In this analysis, both block and diallel were significant factors explaining whether plants were infected by *Coleosporium ipomoeae* (Table 3). Differences among dialleles indicate the presence of genetic differences among the three random samples of parent plants used to produce the dialleles. Inadequate replication within blocks precluded retaining the block term in subsequent models. Consequently, tests for significant sire or dam effects were conservative because environmental variance had not been removed from the residual term. Furthermore, because of the large number of structural zeros present in the partial diallel mating design, a main-effects-only model was fitted (Table 4).

A nonsignificant goodness-of-fit statistic (the residual chi-square) indicates that the additive model provides an adequate fit to the data. Sire and dam each explain a significant component of the phenotypic variance among progeny of the remaining parents in probability of infection by rust, indicating that there is heritable variation in this trait. To determine whether maternal effects influence the proportion of offspring with rust, the chi-squared goodness-of-fit statistic was used to test whether progeny are likely to differ in probability of infection from their reciprocal sibs (sibs with the same parents but in which seed and pollen parents are switched) by more than chance alone. In the eleven reciprocal full-sib families, the probability of an offspring having rust did not differ significantly between mothers ($\chi^2_{10} = 10.1$, $0.995 < P < 0.990$). Instead, the very low value of the statistic suggests that progeny within a full-sib family are more likely to resemble their reciprocal sibs than expected by chance alone (two-tailed test, $0.01 < P < 0.02$).

DISCUSSION

Clearly, this weed population contains statistically significant amounts of heritable genetic variance in resistance to anthracnose caused by *C. dematium* and rust caused by *Coleosporium ipomoea*. Furthermore, variation in resistance to rust appears to involve a major gene with a dominant resistance allele, as well as quantitative variation due to the additive effects of a large number of loci. In contrast, resistance to anthracnose was continuously distributed, suggesting polygenic control. It seems that resistance to neither fungus is influenced by maternal effects.

The evidence for both quantitative heritable variance for resistance to rust and a dominant resistance allele at a single major locus suggests that any program to develop *Coleosporium ipomoea* as a mycoherbicide would probably be doomed to early failure. Fortunately, other problems have diminished interest in using this fungus for weed control, including difficulty in culturing it and the fact that it has little apparent influence on host survival.

In contrast, heritability of damage by anthracnose is low. However, because all damage was caused by natural infection, it is likely that much of the environmental variance in damage was due to variance in escape. If so, heritability for resistance to an artificially inoculated (sprayed) pathogen and its disease would probably be higher than that measured here. Furthermore, E. L. Simms and J. K. Triplett (*unpublished*) have documented substantial levels of genetic variation among inbred lines of *I. purpurea* for resistance to anthracnose when artificially inoculated in the laboratory with

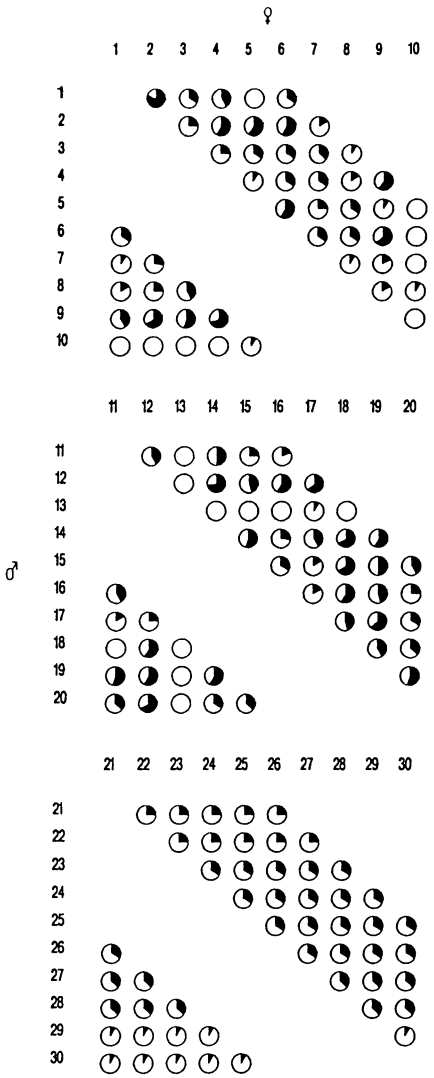

Fig. 2. Distribution of rust-infected progeny among full-sib families. Each pie diagram represents all progeny from a single full-sib family growing in all four spatial blocks. The solid section indicates the proportion of offspring with rust infection. Sample sizes varied from $n = 5$ to $n = 15$, with most being $n = 12$.

Table 3. Logistic analysis of variance for effects of block and diallel on likelihood of infection by *Coleosporium ipomoeae*

Source	df	χ^2	P
Intercept	1	48.18	0.0000
Block	3	21.89	0.0001
Diallel	2	10.59	0.005
Likelihood ratio	6	7.17	0.3

Table 4. Logistic analysis of partial dataset (without offspring of plants whose progeny are almost all resistant) for effects of sire and dam on likelihood of infection by *Coleosporium ipomoea*

Source	df	χ^2	P
Intercept	1	7.24	0.0071
Sire (diallel)	27	65.10	0.0001
Dam (diallel)	23	63.00	0.0000
Likelihood ratio	69	71.48	0.4

specific isolates of *C. dematium* at conidial concentrations used for screening potential mycoherbicides.

The evolutionary change in a character in response to selection is determined by the product of the heritability of the character in the population and the selection differential (18,23), $R = h^2s$, in which R is the population response to selection (change in mean resistance) and s (the selection differential) represents the intensity with which selection acts on the character. Because pesticides are designed to kill the target organism in large numbers (i.e., be effective), they can impose very strong selection intensities (34,35). Even if h^2 is low, a high selection differential might cause rapid, large changes in mean levels of pesticide resistance in a pest population.

With the goal of replacing conventional chemical herbicides with mycoherbicides, developers have focused on especially pathogenic organisms applied at lethal densities (11,39,46). Thus, mycoherbicides are also likely to be strong selection agents. Were *C. dematium* to be used as a mycoherbicide against *I. purpurea*, the strong selection imposed by such a measure would likely cause evolution to a higher mean level of resistance in the plant population studied here. Furthermore, patterns of natural selection on this resistance trait reported by Simms and Rausher (42) suggest that other populations of tall morningglory are also likely to possess genetic variation for resistance to this fungus.

In the historical context of the evolution of pesticide resistance, observation of heritable genetic variation for damage by, and therefore resistance to, anthracnose in this morningglory population should provide a cautionary note to developers of mycoherbicides. Although this is the first study to measure genetic variation in resistance to a member of the genus *Colletotrichum* in a weed species that is a potential candidate for mycoherbicide control by anthracnose, reviews of the literature suggest that genetic variation in pathogen resistance in natural plant populations is the rule rather than the exception (2,3,8,14,32). Furthermore, coevolutionary theory predicts that extensive genetic variation in disease resistance could be maintained in natural plant populations by a variety of mechanisms (19,25–29). These theoretical and empirical results suggest that developers of mycoherbicides should heed the precautionary measures suggested by applied evolutionary biologists to delay evolution of pesticide resistance (30,35).

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