

PATTERNS OF SELECTION ON PHYTOPHAGE RESISTANCE IN *IPOMOEA PURPUREA*

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Key words.—Herbivores, *Ipomoea purpurea*, parasites, pathogens, selection.

Received July 27, 1992. Accepted December 29, 1992.

Adaptive landscapes with multiple peaks have been invoked to explain a variety of evolutionary phenomena, including the divergence of populations in mean phenotype (Futuyma 1986), the evolution of coadapted gene complexes (Charlesworth and Charlesworth, 1976; Turner 1977) and the rapid spread of favorable gene combinations by group selection and genetic drift (Wright 1932, 1977, 1978). Nevertheless, definitive examples from natural populations of adaptive surfaces with multiple peaks are few (but see Lewontin and White 1960; Smith 1990; Wiggins 1991; Brodie 1992), particularly for suites of quantitative characters (Lande and Arnold 1983). It is thus unclear how commonly natural populations are characterized by multiple adaptive peaks; and hence, in general what the potential is for multiple peaks to influence evolutionary processes in natural populations. Addressing this issue will require estimating the form of the adaptive surfaces of a variety of organisms (Lande and Arnold 1983). In this report we describe the adaptive surface corresponding to a suite of resistance traits in the annual morning glory, *Ipomoea purpurea*.

Several investigators have suggested that the evolution of resistance against particular parasites (pathogens, herbivores) may be influenced by the presence of, and the pattern of selection imposed by, other types of parasites (Janzen 1980; Fox 1981; Simms and Rausher 1987; Gould 1988; Pilsen 1992). Because little empirical evidence bearing on this issue exists, however, it is far from clear whether, in general, resistances to different parasites evolve independently in natural populations. The presence of multiple adaptive peaks in the adaptive surface associated with resistance traits could generate nonindependence if different combinations of resistance were associated with different peaks (e.g., one favored

combination is resistance to one parasite but susceptibility to another, while another favored combination is the opposite). We examined this possibility for one system by quantifying the adaptive surface for resistance to two parasites in *Ipomoea purpurea*.

MATERIALS AND METHODS

Experimental Organisms and Study Site

The experimental system consisted of the self-compatible annual morning glory vine *Ipomoea purpurea* Roth (Convolvulaceae), one of its major pathogens, *Colletotrichum dematium* Pers. ex Fr. f. *ipomoeae* v. Arx (Fungi Imperfecti), and the suite of insect herbivores described previously (Rausher and Simms 1989; Simms and Rausher 1989). *Colletotrichum* is the asexual, or imperfect, stage of the ascomycete *Glomerella cingulata* (Stonem.) Spauld & v. Schrenck, and various members of the genus cause the disease anthracnose. *Colletotrichum dematium* infects *I. purpurea* after warm summer rains, causing small regular lesions on the leaves. The fungus overwinters on dried infected leaves in the soil (Agrios 1988, pp. 385–389). Significant levels of additive genetic variation for resistance to *C. dematium* have been documented in this population of *I. purpurea* (Simms in press).

The study site was an old agricultural field in Durham County, North Carolina, that had last been planted in soybeans in 1983, but has since only been disked every spring. The natural population of *I. purpurea* in this field provided all the plants involved in the experimental crosses described below.

Field Protocol

The experimental plants were the same as those analyzed previously by Simms and Rausher (1989) and Simms (1990). One-week-old seed-

lings obtained from a replicated partial diallel cross with 142 full-sib families distributed among 28 paternal and 30 maternal half-sib families were transplanted into the field in July 1985. Plants from each full-sib family were randomly allocated among four spatial blocks, and placed into a square grid of points 0.7 m apart. Plants were allowed to climb up 1-m-tall bamboo stakes to mimic growth in corn fields. Native vegetation in the field was not weeded during the experiment. The total number of plants was 2840.

We defined resistance operationally as the complement of the proportion of leaf area damaged by herbivores (see Rausher and Simms 1989). In late August we estimated damage as the proportion of the total leaf area occupied by anthracnose lesions. A clear plastic grid with 0.59 cm² 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. The areas damaged by each of three insect folivores (described by Simms and Rausher 1989) were also recorded. Damage by each type of plant enemy was summed over all leaves on a plant to yield total damage in each category, then divided by total leaf area to yield proportion damage, which was arcsine square-root transformed for statistical analyses to normalize the residuals. Because the leaf measurements were performed before any leaves senesced, we did not need to correct this measure for senescence. Damage and plant growth continued after August; therefore, we have estimated selection only on resistance expressed before the census.

Plant fitness was estimated by collecting and counting all seeds produced by the experimental plants, including those that died before producing seeds. Seed production ended with the first killing frost on December 7, 1985. Inviolate seeds were not counted. Germination assays indicated that more than 95% of seeds counted as living were viable. Because this fitness measure incorporates the effects of mortality over most of the growing season, it is probably a reasonable estimate of fitness. However, this measure does not include mortality that occurs between seed maturation and the first week after germination, nor does it explicitly include any effects of differences among plants as pollen donors.

Statistical Analysis

To determine the type and magnitude of selection acting on resistance traits, we employed

the regression analysis described by Rausher (1992). This analysis modifies the standard Lande and Arnold (1983) phenotypic regression of fitness on traits by using estimates of breeding values rather than phenotype values. This approach eliminates biases caused by environmental covariances between traits and fitness. In addition, exclusion of genetically uncorrelated traits does not bias the remaining traits analyzed, thus simplifying portrayal of adaptive surfaces (Rausher 1992).

In this analysis, directional selection on trait i is detected as the linear coefficient β_i , from the regression of the breeding value of relative fitness (absolute fitness divided by mean fitness), w , on the breeding values of the individual traits, x_i , using the model

$$w = \alpha + \sum_{i=1}^P \beta_i x_i + \epsilon, \quad (1)$$

where α is a constant, ϵ is the usual error term, and P is the number of traits being examined. We obtained the nonlinear (stabilizing/disruptive) selection gradients from the second-order coefficients, γ_{ij} , of a quadratic regression on relative fitness using the model

$$w = \alpha + \sum_{i=1}^P \beta'_i x_i + \sum_{i=1}^P \sum_{j>1}^P (1 - \delta/2) \gamma_{ij} x_i x_j + \epsilon, \quad (2)$$

where $\delta = 1$ if $i = j$ and 0 otherwise. The significance of each parameter in equations (1) and (2) was determined after the effects of all other parameters had been removed.

We estimated the breeding value for each trait as twice the deviation of the paternal half-sib family mean from the population mean (Falconer 1981, p. 106). Family-mean regression coefficients, like family-mean correlations, converge to the true regression coefficients for breeding values as the number of individuals per family increases (Rausher 1992; see also Arnold 1981). With half-sib family sizes of 50 to 60 individuals, our family mean regression coefficients should be relatively unbiased.

RESULTS

We first used the pattern of genetic correlations between resistances to herbivores and pathogens to determine which traits should be included in a multivariate selection analysis. Anthracnose resistance exhibited a significant family-mean correlation with tortoise beetle resistance ($r =$

TABLE 1. Directional (β) and stabilizing/disruptive (γ) selection gradients from analyses using original traits (A) and from canonical analysis (B). Numbers in parentheses are standard errors.

| Trait | γ_{ij} | | | | |
|------------------------------|---------------------|---------------------|--------------------|------------------------|--|
| | β | β' | Anthracnose damage | Tortoise beetle damage | Leaf area |
| A. Original analysis | | | | | |
| Anthracnose damage | -3.21 (2.41) | -8.63** (2.49) | 263.07 (183.07) | -87.27** (22.75) | -2.15* (0.17) |
| Tortoise beetle damage | 0.31 (0.62) | 0.84 (0.57) | | -13.73 (18.06) | 0.06* (0.03) |
| Leaf area | 0.01**** (0.003) | 0.02**** (0.003) | | | -3.1 × 10 ⁻⁵ (2.4 × 10 ⁻⁴) |
| B. Canonical analysis | | | | | |
| Axis | β | β' | β' | γ_{ii} | |
| 1 | -0.18 (0.60) | | -0.18 (0.48) | | -20.45 (8.73)†† |
| 2 | -3.22 (2.42) | | -3.22 (2.35) | | 269.79 (66.31)** |
| 3 | 0.01 (0.002)*** | | 0.01 (0.003)*** | | -1.6 × 10 ⁻⁴ (8 × 10 ⁻⁵)† |

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$; **** $P < 0.0001$; † $P = 0.08$; †† $P = 0.06$.

0.41, $P < 0.025$, $N = 30$) and a marginally significant correlation with leaf area ($r = 0.33$, $P = 0.07$, $N = 30$). By contrast, correlations between anthracnose resistance and resistances to other herbivores were low and nonsignificant ($r = 0.07$, 0.07, and 0.25, respectively, for resistance to corn earworms, flea beetles, and generalist folivores). Similarly, correlations between tortoise beetle resistance and resistance to the other herbivores were uniformly low and nonsignificant (maximum $r = 0.20$; Rausher and Simms 1989; see table 1). These results indicate that the pattern of selection on anthracnose and tortoise beetle resistances and on leaf area must be examined simultaneously using a multivariate approach because these three traits were genetically correlated. However, resistance to the other herbivores examined by Rausher and Simms (1989) may be omitted because they were weakly, if at all, genetically correlated with these traits. Their omission from the analysis will, therefore, not lead to inaccurate estimates of selection on anthracnose and tortoise beetle resistance (Rausher 1992).

For neither anthracnose resistance nor tortoise beetle resistance did we detect significant directional or stabilizing/disruptive selection (β , and γ_{ii} , table 1A). However, the correlational selection gradient (γ_{ij}) for these two resistances was large and significant, as were the correlational selection gradients for each of these traits and leaf area. This result suggests that there was a complex pattern of selection acting on the two resistance traits that is not readily interpretable using the standard multivariate regression approach.

One method for visualizing and interpreting correlational selection of the type displayed in table 1A is the canonical analysis described by Phillips and Arnold (1989) and Simms (1990). Essentially, this analysis is a method of rewriting equation (2) in a simplified form. This simplification is achieved by transforming the trait values in a manner that rotates the axes spanning the multidimensional breeding-value space so that cross-product terms are removed from the equation. In this new coordinate system, each orthogonal axis explains the maximum amount of residual variation in the selection surface. The axes, which are linear combinations of the original traits, are aligned with the principle axes of the estimated selection surface. Peaks, saddles, ridges, and valleys in the selective surface that are located away from axes corresponding to the

TABLE 2. Loadings of original traits on new axes from canonical analysis. Values in parentheses indicate correlation of original trait with new axis.

| | New axis | | |
|------------------------|------------------------------|---------------------------------|------------------------------|
| | 1 | 2 | 3 |
| Anthracnose damage | 0.15 (0.44)* | 0.99 (0.53)** | 7.0×10^{-4} (0.33) |
| Tortoise beetle damage | 0.99 (1.0)**** | -0.15 (-0.36)* | -5.1×10^{-5} (0.31) |
| Leaf area | -1.6×10^{-4} (0.28) | -6.9×10^{-4} (-0.46)** | 1.00 (1.0)**** |

* $P < 0.05$; ** $P < 0.01$; **** $P < 0.0001$.

original trait values are rendered more detectable by this type of analysis.

The interpretation of the new axes produced by the canonical analysis is straightforward. The third axis was dominated almost exclusively by, and thus was parallel to, the original axis defined by leaf area (table 2). By contrast, leaf area contributed minimally to the first two new axes, as indicated by its very low coefficient of loading on these axes. Both anthracnose resistance and tortoise beetle resistance contributed positively to and were significantly correlated with axis 1. This axis may thus be interpreted as representing a measure of total resistance to these two enemies. Both resistances also contributed substantially to axis 2, but the signs of their loading coefficients were opposite. This axis may thus be interpreted as representing a measure of proportional allocation to resistance; negative values represent relatively high resistance to anthracnose and low resistance to tortoise beetles, while positive values represent the opposite.

Directional selection was not detected on either new axes 1 or 2 (table 1B). By contrast, directional selection was highly significant for axis 3, indicating selection for increased leaf area. Adding the quadratic terms to the regression of fitness on the traits significantly improved the fit over the linear model ($F_{6,20} = 3.75$, $P < 0.05$). Axis 2 exhibited significant upward concavity (positive γ_{ii} in table 1B), whereas axes 1 and 3 each exhibited marginally significant downward concavity (negative γ_{ii}).

Because significant values of the γ_{ii} technically indicate only curvilinearity in the selective surface, it cannot be assumed that selection was truly stabilizing or disruptive unless there exists an intermediate fitness maximum or minimum within the population's range of breeding values for a given axis. In the canonical analysis of this surface, the value on each axis corresponding to

the single such stationary point did lie within the range spanned by the variability in the population (minimum, stationary point, and maximum values respectively were: for axis 1, -0.13, 0.009, 0.17; for axis 2, -0.04, 0.012, 0.05; and for axis 3, -28.2, 64.2, 68.0).

We used the constrained-regression method (Mitchell-Olds and Shaw 1987; Simms 1990) to determine whether the estimated fitness function fits the data significantly better than other curves with fitness maxima or minima outside the observed range of breeding values. This technique involves constraining the fitness function along each axis to have its stationary point at $z_{H_0}^*$, equal to a breeding value just outside the range observed in the experimental population. Because the range of breeding values has two extremes (a maximum and a minimum), we ran two separate regressions, one to constrain the stationary point to lie just above the range of breeding values, the other placing it below the range.

When a character is uncorrelated with other characters, as is true for the axes of the canonical function, then the quadratic estimate of the fitness function is described by

$$w = \beta_0 + \beta_1 z + \beta_2 z^2 + \epsilon, \quad (3)$$

with a minimum at

$$z^* = \beta_1 / 2\beta_2. \quad (4)$$

Therefore, the partial regression coefficients for the fitness function constrained to have a stationary point at $z_{H_0}^*$ are defined as $\beta_1^* = -2z_{H_0}^* \beta_2^*$ (Mitchell-Olds and Shaw 1987). Letting, for each individual observation, $y_i = z_i^2 + 2z_{H_0}^* z_i$, then (3) can be rewritten as

$$w = \beta_0 + \beta_2 y + \beta_3 z + \epsilon. \quad (5)$$

With the $\beta_3 z$ term, (5) equals (3), but without it, (5) equals the constrained model. Therefore, the null hypothesis of no disruptive or stabilizing

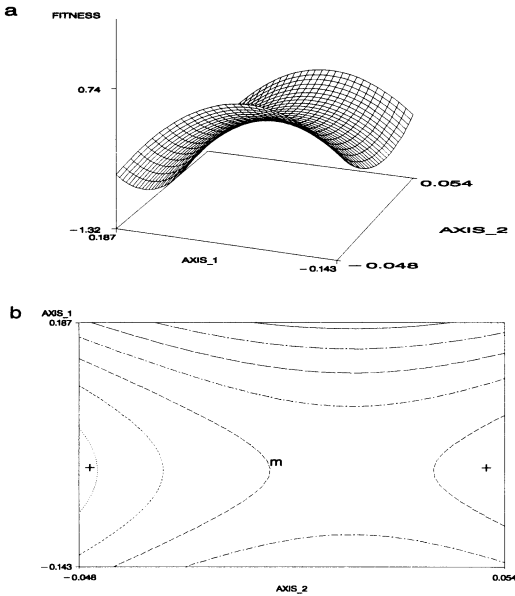


FIG. 1. Estimated selection gradients on the orthogonal index traits one and two. The axis for each trait is determined by the actual range of breeding values for that trait within the population. A three-dimensional representation of the surface is illustrated above (a) and the contour plot of that surface is drawn below (b). The population mean is indicated on the contour plot as "m."

selection, which is equivalent to the null hypothesis that an extreme breeding value possesses the maximum or minimum fitness, can be rejected only if β_3 in (5) is found to differ significantly from zero (Mitchell-Olds and Shaw 1987).

A regression in which minimum fitness was associated with an intermediate breeding value along axis 2 fits significantly better than one in which the minimum fitness was associated with either an extremely high breeding value ($\beta_3 = 14.1$, $P = 0.02$) or an extremely low breeding value ($\beta_3 = -233.3$, $P = 0.004$). Thus, the second index trait is likely to have experienced disruptive selection. In contrast, intermediate values along axes 1 and 3 did not provide a significantly better fit than a stationary value below the minimum along axis 1 and above the minimum along axis 3 ($\beta_3 = 3.83$, $P = 0.12$ and $\beta_3 = -0.01$, $P = 0.25$, respectively).

Biologically, the pattern of selection revealed by the canonical analysis can be interpreted as follows: Absence of detectable selection along axis 1, total resistance, suggests that total resistance may have been a neutral trait. However, the marginally significant downward concavity of the fit-

ness function along this axis suggests that this axis may have been associated with a broad, gently sloping fitness ridge or peak. By contrast, disruptive selection on axis 2, which represents proportional allocation to the two resistance traits, suggests that for a given total allocation to resistance, resistance to either parasite alone was favored, but intermediate levels of resistance to both were disfavored. Directional selection on axis 3 indicates directional selection that favored increased leaf area. These interpretations should be viewed as tentative pending more detailed experimental examination of the ecological and genetic factors responsible for the form of the adaptive surface.

DISCUSSION

Although it has been argued that the evolution of resistance to one parasite may be constrained by selection imposed by other parasites (Janzen 1980; Fox 1981; Simms and Rausher 1987; Gould 1988; Simms 1992), convincing demonstration of such "diffuse" selection on resistance has been elusive. One reason for this may be that it has seldom been recognized that there are many possible causes of nonindependent evolution of resistances. These include, but are not limited to (1) the existence of genetic correlations among traits conferring resistance to different parasites (e.g., Kinsman 1982; Rausher and Simms 1989; Maddox and Root 1990), which may under some circumstances constrain the evolution of resistance even if there is directional selection acting directly on each resistance trait (Rausher 1992); (2) effects of one parasite on the abundance of, or susceptibility of a host to, another (e.g., Carroll and Hoffman 1980; Kuć 1982, 1987; Fritz et al. 1986; Karban 1986; Karban et al. 1987; Apriyanto and Potter 1990; Fritz 1992; Rausher et al. 1993); and (3) nonadditivity of the impact of two parasites on host fitness (e.g., Dickinson 1979; Powell 1979; Strauss 1991). [A general model indicating how (2) and (3) lead to nonindependent evolution of resistances is provided by Hougen-Eitzman 1991.]

Our results suggest that at least two of these mechanisms contribute to nonindependent evolution of resistance to anthracnose and to tortoise beetles in *Ipomoea purpurea*. On the one hand, resistance to these two parasites was positively genetically correlated. Selection for increased resistance to each parasite should thus be mutually reinforcing. Nevertheless, the observed genetic correlation of 0.41 is probably not sufficiently

close to 1.0 to act as a severe constraint on the evolution of the two traits (Via and Lande 1985).

Additionally, the existence of two adaptive ridges (or peaks) (fig. 1) indicates that genotypes that are resistant either to anthracnose or to tortoise beetles, but not to both, were favored, but that genotypes exhibiting intermediate levels of resistance to both parasites occupied an adaptive valley. This adaptive valley is most easily explained by assuming that the detrimental effect of anthracnose and tortoise beetles together was greater than the sum of their individual effects [cause (3) above].

It is easy to imagine how the adaptive surface portrayed in figure 1 could produce diffuse selection on resistance. Consider a situation in which anthracnose is newly introduced into a population of *Ipomoea purpurea* that had not previously been exposed to this pathogen. Then whether anthracnose resistance will evolve would depend on whether tortoise beetles have been and are present. In the absence of tortoise beetles, the adaptive ridge associated with complete allocation of resources to tortoise beetle resistance presumably does not exist, because there would be no advantage associated with being resistant to tortoise beetles. Upon introduction of anthracnose, the only ridge in the adaptive landscape would be that associated with complete allocation to anthracnose resistance. Thus, resistance to anthracnose would be expected to evolve as the population climbed that ridge.

By contrast, if tortoise beetles have historically been associated with this population, then there will historically have been an adaptive ridge associated with complete allocation to tortoise beetle resistance, and the population should have evolved to lie near the top of this ridge. Introduction of anthracnose to this population will then convert the adaptive landscape into one with two ridges, but anthracnose resistance will not evolve because the population will remain perched atop the adaptive ridge associated with complete allocation to tortoise beetle resistance. Thus, whether anthracnose resistance evolves would depend on whether tortoise beetles had been present recently, that is, selection on resistance would be diffuse.

Three caveats should be added to these interpretations. First, because we estimated several half-sib family mean correlations, there was a possibility of observing one or two correlations significantly different from zero by chance. Such chance correlations could produce an inaccurate

estimate of the selection surface. Second, we do not know the extent to which the pattern of selection detected in this study varies over years or from site to site (e.g., Kalisz 1986; Jordan 1991). Finally, because the evolution of resistance will often affect the abundances of a plant's pathogens and herbivores (Fritz 1992), the adaptive landscape is likely to change as evolution proceeds (i.e., fitnesses may be frequency dependent). It is conceivable that as an equilibrium is approached, the peaks in figure 1 may tend to coalesce, producing a single global equilibrium.

In spite of these caveats, our results suggest that at least at some point in the evolution of resistance, the adaptive landscape in morning glories has two ridges (or peaks). To the extent that these results are generalizable to plant-herbivore and plant-pathogen interactions in general, they suggest that diffuse selection may commonly be important in guiding the evolution of plant resistance. Examination of the adaptive topographics for resistance in other systems is needed to assess the validity of this generalization.

ACKNOWLEDGMENTS

We are grateful to M. Bucher, M. Evans, G. Grabowsky, and D. Pilson for their invaluable assistance in the field and in counting seeds. We thank D. Burdick for statistical advice. C. Pickett kindly provided and plowed the site used for this work. The manuscript was improved by two anonymous reviewers. This project was supported by National Science Foundation grant BSR 8507359.

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