Letter to the Editor

Multilines and "Super Races": a Simple Model

J. V. Groth

Assistant Professor, Department of Plant Pathology, University of Minnesota, St. Paul 55108.
Paper No. 9424, Scientific Journal Series, Minnesota Agricultural Experiment Station, University of Minnesota, St. Paul 55108.

Multiline cultivars are being tested or developed in several places (3). Their epidemiological effectiveness has been established empirically (4) and theoretically (1, 9). The validity of this important aspect of multilines is not doubted, nor is it the subject of this discussion. Rather, this letter is concerned with our lack of theoretical understanding of the long-term fate of multilines. The questions are: Will multilines select complex races of a pathogen (those capable of infecting all or most of the components)? If so, how complex will such races become? The first question was examined most thoroughly by Leonard (5, 6) who outlined the problem well, but who chose to begin with complex empirical data. Because of this, his model was not succinct, and it is difficult to generalize from his example. It is clear to me from his discussion, however, that Leonard had a good understanding of the effects a multiline might have on the pathogen population. He found that carefully controlled mixtures of components, based primarily on level of stabilizing selection associated with each virulence gene of importance to the pathogen, would be necessary if multilines are to be effective in the long run. His work may not have received sufficient attention. To my knowledge, the second question has not been explored at all.

Though some unpublished evidence exists that multilines are stabilizing pathogen populations (2) and empirical evidence is beginning to be collected, nothing more has been done theoretically, aside from some enlightening but esoteric mathematical work by Mode (8). There is room in this area now for some further and simpler induction. Maximum progress in any field should occur with a balance between experimental and theoretical research. A thorough understanding of the multiline concept should precede any major move toward their large-scale use. To do otherwise is dangerous.

The proposed mathematical model with two variations is quite simple. It is replete with assumptions that are as realistic as possible while still allowing simplicity. Its purpose is to stimulate further thinking as well as to clarify some ideas which have been expressed in words, but never established in more rigorous ways. The model also might represent a simple beginning for understanding how equilibria are maintained in natural host-parasite systems.

Whereas the reality of some portions of such a model can be questioned, it should prove useful as a starting point to which more realistic but complicating factors can be added. The assumptions of the first variation of the model are:

(i) The multiline is composed of n components, each containing a single resistance gene. Each component occurs with frequency $1/n$.

(ii) Each resistance gene is overcome by a race with the matching virulence gene (v-gene). Races with all possible combinations of v-genes exist.

(iii) If a race does not possess the matching v-gene, it will not reproduce on the resistant host.

(iv) If v-genes reduce within-component fitness, they do so equally and additively; i.e., if by definition, a simple, one-gene race has a fitness of 1 on the component which it infects, more complex races have fitnesses of $1-(m-1)s$ on the components on which they are able to reproduce, where m = the number of susceptible components, and s = the within-component reduction in reproduction associated with a single v-gene (the selection coefficient).

Whereas a single-gene (simple) race will infect $1/n$ plants in the stand, a two-or-more-gene (complex) race will infect m/n. Overall reproduction of a complex race R_m can be expressed as a proportion of susceptible plants multiplied by within-component fitness:

$$R_m = (m/n)\,[(1-(m-1)s]$$

This is better expressed in terms of reproduction of the simple race, R_1, which, as has been defined, has susceptible proportion of $1/n$ and fitness of 1. Hence:

$$R = \frac{R_m}{R_1} = \frac{(m/n)\,[1-(m-1)\,s]}{1/n\,(1)}$$

or

$$R = m\,[1-(m-1)s]$$

Complex races which reproduce better ($R > 1$) than the original simple races will be selected for, and will eventually replace the simple races. To determine the degree of complexity which will maximize R, one needs only to differentiate with respect to m, and set the derivative equal to zero:

$$\frac{\delta R}{\delta m} = 1-2ms + s = 0$$

or, in another form, maximum reproduction occurs in the race with complexity of

$$m = \frac{1+s}{2s}$$

This simply means that until this equilibrial level of m is reached, selection should favor (stepwise) increasing complexity. Figure 1-A illustrates this for several levels of s. It shows increasing reproduction as the v-genes increase in number until the maximum value of R (where $\frac{\delta R}{\delta m} = 0$) is reached, after which stabilizing selection operates against further gains in virulence. An important point is that relative reproductivity of simple and complex races is a function of s, but *not* of n, the number of component lines in the multiline; the maximum value of R will be associated with a given m whether there are five or 50 component lines. Nevertheless, the number of component lines should be of ultimate importance epidemiologically. Not only will a greater number of lines slow progress toward increasing complexity by slowing the epidemic, it will also determine the impact that the moderately complex races have on the multiline's effectiveness. This can be illustrated. Figure 2-A presents the levels of s required to prevent complex races, able to infect more than half of the component lines, from becoming established. These are inversely proportional to n, meaning that greater levels of stabilizing selection will be necessary to curb this shift if fewer component lines are present. Also, note that the greatest changes in required values of s occur to the left, when the number of lines is small.

The second model variation differs from the first only in the manner in which fitnesses due to virulence genes combine. They are now assumed to combine multiplicatively (so that individual fitnesses associated with the genes are multiplied) rather than additively. This is probably a more realistic assumption (7), but one which, as shall be shown, complicates the model somewhat. The within-component fitness now can be generally expressed as $(1-s)^{m-1}$, using the same symbols and other assumptions as in the first variant. Using parallel arguments as with the additive variant, the overall reproduction of complex in terms of that of simple races will be:

$$R = m\,(1-s)^{m-1}$$

This function results in a considerably more complex derivative,

$$\frac{\delta R}{\delta m} = [m \ln(1-s) + 1]\,(1-s)^{m-1}$$

Figure 1-B represents, as in Fig. 1-A, the reproductive capabilities of complex races relative to those of simple races for various values of s, when fitnesses combine multiplicatively. Note that an important difference is illustrated: if fitnesses are multiplicative, more complex races will occur for a given value of s than if selection coefficient are simply additive. Multiplicative fitness represents a case of "diminishing returns", whereby each

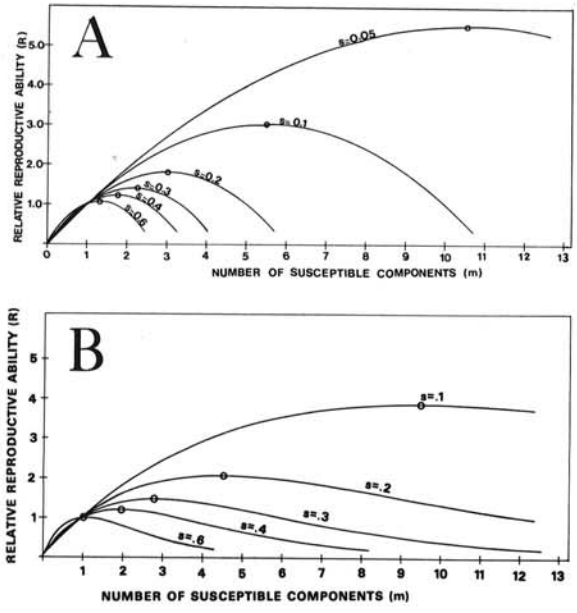

Fig. 1-(A, B). Relative reproductive ability on a multiline cultivar of complex pathogen races, having more than one effective virulence gene, expressed in terms of the reproduction of simple (one virulence gene) pathogen races for **A)** additive combinations of fitness and **B)** multiplicative combinations of fitness. Curves for several values of s, the within-component reduction in fitness which is a measure of stabilizing selection, are shown. Maxima in R are circled.

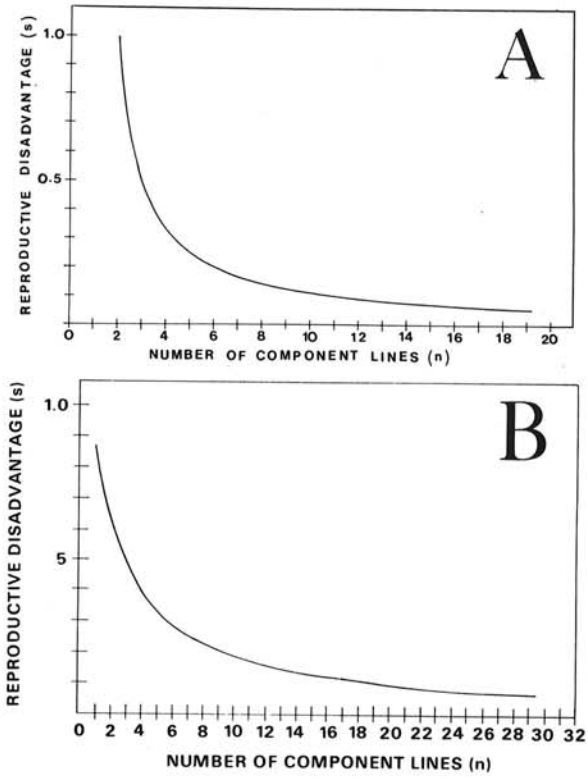

Fig. 2-(A, B). The amount of within-component reproductive (fitness) disadvantage or stabilizing selection (s), associated with each pathogen virulence gene that is necessary to offset the selective advantage of a race that can attack half of the n component isolines in multiline cultivars for **A)** additive combinations of fitness and **B)** multiplicative combinations of fitness.

added virulence gene detracts less than the one before it.

In Fig. 2-B, with multiplicative fitness, the amount of stabilizing selection necessary to offset the reproductive advantage gained by a race capable of attacking half the components of a multiline is plotted as a function of n, the number of components. The plot is similar in shape to that of the additive variant, and except for very low n values, shows higher levels of required s than with additive combinations.

The model says nothing about the expected rate of establishment of complex races. This will depend on many factors relating to the nature of the disease. For some polycyclic pathogens, such as rusts, it seems likely that the population should shift rather rapidly.

Perhaps the most unrealistic facet of the model is the simplistic treatment of the parameter s. Stabilizing selection, if it even exists for all v-genes (9) probably will not be the same for different v-genes (4, 9). Nothing is known about how the stabilizing selection effects of genes will combine. Additivity of fitness is a useful starting assumption for two reasons. First, it is easy to handle mathematically and conceptually. It has served as a useful preliminary model. This is poor justification if the assumption is quite unrealistic, which clearly it is at high levels of m or s (where fitnesses of less than 0 are possible). But at more likely small m or s levels, the additive variant is not so unrealistic, and provides an upper limit; viz., nondiminishing decrements of fitness are probably the best (in terms of being economically most favorable) we can expect. In view of our lack of knowledge about how fitnesses combine, the additive case should not be dismissed. Other possibilities involve diminishing fitness reductions, of which the multiplicative case is but one. More rapidly diminishing decrements are also likely in which case even more complex races will be possible for each level of s than shown by the multiplicative variant. Modification of this or any other aspect of the model, though readily done, will quickly increase its complexity. In such a case, the importance of stochastic methods becomes obvious. The intent here has been to set forth a basic mathematical treatment which is clear enough to illustrate some possibilities. The model identifies the importance of (i) being able to precisely measure pathogen fitness and (ii) understanding how fitnesses combine (including understanding the genetic basis of stabilizing selection).

As most people who have discussed potential selection for race complexity in multilines have indicated, resistance genes will not be useful in multilines if the races capable of overcoming them are not subject to stabilizing selection. It is obvious that such races would enjoy increased reproduction, since they pay no penalty for possessing extra virulence. In the model, m of the best-reproducing race is without limit if such is the case for all R genes employed; i.e., the most complex races will reproduce best.

That stabilizing selection will be necessary for the success of multilines is fairly obvious. Assuming that it occurs, both variants of the model show two things which are not so obvious: that if the level of stabilizing selection is similar to that reported by Leonard (5) or van der Plank (9), then there will be an upper limit to complexity, and that *relative* reproductive advantage of simple and complex races will be independent of n, the number of multiline components.

A modification of the model to allow the addition of susceptible components shows, as Leonard (5) also concluded, that for a given value of s, complexity of the best-reproducing race will be less than if susceptible plants are absent. For example in the additive case, if $1/n$ of the multiline has no resistance genes, then one less v-gene will be present in the best-reproducing race. There will also be more direct competition between simple and complex races on the susceptible plants, so that removals might play a more important role in such a situation. Though it would have a stabilizing effect on the pathogen population, addition of a susceptible component would have an adverse epidemiological effect. I believe that only experimental evidence will tell us the optimum composition of a multiline. Models such as this however, might narrow the range of possibilities to be explored experimentally.

LITERATURE CITED

1. BROWNING, J. A., and K. J. FREY. 1969. Multiline cultivars as a means of disease control. Annu. Rev. Phytopathol. 7:355-382.
2. COYNE, D. P., and M. L. SCHUSTER. 1975. Genetic and breeding strategy for resistance to rust [Uromyces phaseoli (Reben) Wint.] in beans (Phaseolus vulgaris L.). Euphytica 24:795-803.
3. FREY, K. J., J. A. BROWNING, and M. D. SIMONS. 1973. Management of host resistance genes to control diseases. Z. Pflanzenkrankh. 80:160-180.
4. LEONARD, K. J. 1969. Factors affecting races of stem rust increase in mixed plantings of susceptible and resistant oat varieties. Phytopathology 59:1845-1850.
5. LEONARD, K. J. 1969. Selection in heterogeneous populations of Puccinia graminis f. sp. avenae. Phytopathology 59:1851-1857.
6. LEONARD, K. J. 1969. Genetic equilibria in host-pathogen systems. Phytopathology 59:1858-1863.
7. MAYNARD SMITH, J. 1968. Evolution in sexual and asexual populations. Am. Nat. 102:469-473.
8. MODE, C. J. 1960. A model of host-pathogen system with particular reference to the rust of cereals. Pages 84-96 *in* O. Kempthorne, ed. Biometrical genetics. Pergamon Press, New York. 234 p.
9. VAN DER PLANK, J. E. 1968. Disease resistance in plants. Academic Press, New York and London. 206 p.