Disease Control Through Use of Multilines: A Theoretical Contribution

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We thank the National Research Council of Canada for financial support.
Accepted for publication 30 January 1978.

ABSTRACT

FLEMING, R. A., and C. O. PERSON. 1978. Disease control through use of multilines: a theoretical contribution. Phytopathology 68: 1230-1233.

A mathematical model of a multiline is used to illustrate the relationships between the composition of the host crop, the readiness of the plant breeder to change crop composition, the yield losses, and the composition of the pathogen population. Under the assumptions of the model it is shown that an infrequently adjusted multiline is incapable of preventing one genotype from dominating the pathogen population. It is also shown that the relative merits of multiline and monoculture strategies of employing resistance genes depend on the circumstances. Important considerations are stabilizing selection and the readiness of the plant breeder to change crop composition. Practical aspects of multiline implementation are discussed.

Additional key words: plant breeding, disease resistance, gene strength, virulence, complex races, gene-for-gene relationship, epidemic.

When a multiline is grown widely, pathogens with multiple virulence (i.e., "complex races" capable of inciting disease on two or more components of the multiline) may be favored by natural selection (7). However, with each component of the multiline carrying only a single resistance gene (*R*-gene), actually only a single virulence gene (*v*-gene) is required in any particular interaction that results in disease; the one or more additional *v*-genes that are carried by complex races are, in fact, unneeded in particular interactions with individual plants of the multiline. Assuming that stabilizing selection (8) operates against unneeded *v*-genes, and that every addition of an unneeded *v*-gene is accompanied by a fitness loss, an endpoint should be reached at which the accumulation by the complex race of yet another *v*-gene is no longer advantageous (7). It has been shown mathematically (3) that this endpoint will be determined independently of the number of *R*-gene components in the multiline. Furthermore, by enlarging the multiline to include more components, the development of complex races will proceed at a slower rate and the proportion of the multiline that can be diseased by any complex race able to maintain itself at a significant quantity will be made smaller (3).

In this communication we hope to carry the mathematical development of multiline theory a step further by considering the relationships between the choice of crop composition, the readiness of the plant breeder to change crop composition, the yield losses, and the composition of the pathogen population. We assume: (i) that a breeder is able to produce n different genotypes of host resistance by using the available *R*-genes singly and in combination in a series of agronomically identical varieties; (ii) that all host:pathogen interactions are based on gene-for-gene relationships (1); (iii) that each of the n pathogen genotypes that could be differentiated by phenotype on the n host resistance genotypes is originally present in the pathogen population; (iv) that stabilizing selection is operating as expected by experiment (4, 5, 8) and theory (6, 7, 8); (v) that competitive or synergistic interactions between different units of inoculum on a common host plant are negligible; and (vi) that removals are negligible [as they would be during the early stages of an epidemic (8)].

Mathematical model of a multiline.—Now let m_j be the *proportion* of the multiline that is of component j, let Q_i (t) be the *amount* of pathogen genotype i present at time t, and let $R_{i,j}$ be the rate of increase of inoculum of pathogen genotype i on host genotype j.

Then, assuming the m_j's and $R_{i,j}$'s are constant

$$Q_i(t) = Q_i(t-1)\, e^{r_i}, \qquad \text{(Eq. I)}$$

where

$$r_i = \sum_{j=1}^{n} m_j R_{i,j}. \qquad \text{(Eq. II)}$$

The frequency of pathogen genotype i at time t is

$$f_i(t) = Q_i(t-1)\, e^{r_i} \Big/ \sum_{i=1}^{n} Q_i(t-1)\, e^{r_i}$$

$$= f_i(t-1)\, e^{r_i} \Big/ \sum_{i=1}^{n} f_i(t-1)\, e^{r_i}$$

Assuming that any *differential* selection acting on the parasite outside the disease season is negligible, the genotypic frequencies would not change between disease seasons although the population size may be drastically

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reduced. Thus, if time is measured only during the disease season,

$$f_i(t) = f_i(0)\, e^{r_i t} \Big/ \sum_{i=1}^{n} f_i(0)\, e^{r_i t},$$

and

$$\frac{f_i(t)}{f_j(t)} = \left[\frac{f_i(0)}{f_j(0)}\right] e^{(r_i - r_j)\,t} \qquad \text{(Eq. III)}$$

Application of the model.—In the real world, pathogen reproductivity is likely to be greatly influenced by weather and other environmental factors. While these influences would introduce a certain amount of "noise" (i.e., unpredictable fluctuations in both the size and genotypic content of the parasite population), the processes represented by Eq. III would be expected to persist over the long term.

Equation III makes it clear that if pathogen genotype i is reproducing more quickly than genotype j (i.e., $r_i > r_j$), then the ratio of their frequencies increases exponentially with time. Therefore the frequency of that genotype which reproduces most rapidly will approach 1.0 as t becomes large. Hence, in analogy with Eq. I, the size of the pathogen population at time t, x (t), when t is large, can be written

$$x(t) \approx x(t-1)\, e^{r_k}, \qquad \text{Eq. IV}$$

where parasite genotype k is that one which reproduces more quickly than any other on the particular host population described by the set of m_j's. The relative rate of increase in size of the pathogen population $(1/x)\cdot(dx/dt)$ is plotted against time in Fig. 1.

Van der Plank (8) has indicated that yield losses in a season are roughly proportional to the total amount of disease, Z, present throughout the season.

$$Z = \int_{t=0}^{t_f} x(t)\, dt,$$

where t_f denotes the length of the disease season.

Using Eq. IV an upper bound can be placed on Z:

$$Z_{max} \leqslant \int_{t=0}^{t_f} x(0)\, e^{r_k t}\, dt,$$

and therefore

$$\frac{Z_{max}}{x(0)} \leqslant \frac{1}{r_k}\,(e^{r_k t_f} - 1).$$

This equation is plotted in Fig. 2.

An objective of the plant breeder is to minimize Z and thus to maximize yield and ultimately revenue while keeping costs low. He would work toward this objective by determining that set of m_j's for which Z is minimal.

The response time, T, the practical minimum to the length of time, in pathogen generations, between adjustments of crop composition, provides a measure of

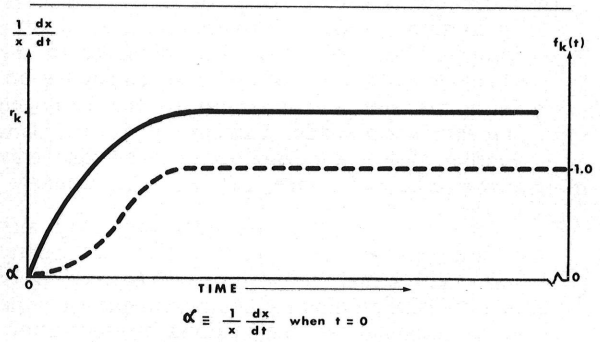

Fig. 1. Plots against time of the relative rate of increase in size of the pathogen population, $(1/x)\cdot(dx/dt)$ (solid line); and the proportion of the pathogen population composed of that genotype, call it k, which reproduces more quickly than any other, f_k (t) (broken line), on the particular host population described by the set of m_j's. The degree to which the pathogen population has adapted to the host population is indicated by f_k (t). Initially the rate of increase of f_k (t) is limited by the scarcity of genotype k. When f_k (t) approaches 1.0 its rate of increase again is limited, but now because the excess in fitness of pathogen genotype k over the population average is very small. The relative rate of increase in size of the pathogen population, $(1/x)\cdot(dx/dt)$, increases at a decelerating rate as the pathogen population becomes adapted to the host population.

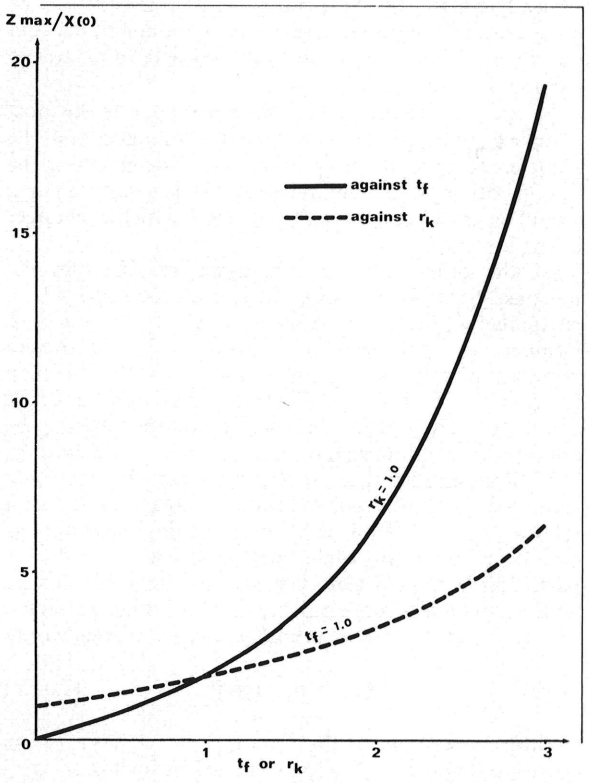

Fig. 2. Plot of the ratio of the upper bound on disease quantity present throughout the season, Z_{max}, to the initial amount, x (0), against the duration of the disease season in pathogen generations, t_f, when the maximum rate of increase in disease quantity per pathogen generation, r_k, is 1.0 (solid line); and r_k, for t_f = 1.0 (broken line). Z_{max}/x (0) increases more quickly with t_f than with r_k.

the readiness of the plant breeder to alter the m_j's. Clearly T depends upon a number of factors such as the cost/benefit ratio, the accessibility of sufficient quantities of the various host R-genotypes in agronomically similar varieties, and the ease with which adjustments can be made to the m_j's. These factors, in turn, are influenced by crop acreage and value, by availability of resistance sources and genetic information, by the nature of the crop (e.g., whether inbreeding or outcrossing, annual or perennial), and by other factors that may determine how quickly the results of plant breeding are realized.

We may imagine that at one extreme T is very short; at the other it is enormous. Where T is short the plant breeder will have the option of reacting to even slight shifts in the pathogen population; he will attempt to minimize α of Fig. 1 by readusting the m_j's as often as is practicable. This would amount to the repeated imposition of intensely disruptive selection on the pathogen population. It is obvious that a strategy such as this would depend on accurate genetic data of the kind provided by a sensitive monitoring program. With continual adjustment of m_j's it is unlikely that any particular genotype ever will dominate the pathogen population. Thus, at this extreme [where we assume response times are negligible, and where α depends critically on the Q_i (0)'s], no general statement can be made concerning the relative merits of monocultures and multilines. Either could be preferred according to the circumstances.

Where stabilizing selection does not operate the most complex genotype i would quickly dominate and the plant breeding strategy would be one of determining the host genotype j which minimizes $R_{i,j}$. Host genotype j then would be grown in monoculture, pending further changes in the system.

At the other extreme, where we imagine that the response time is very long, an introduced crop with a particular set of m_j's must be left unchanged for a large number of pathogen generations. The pathogen population will have ample time to adapt to crop composition and will become dominated by a single genotype. The plant breeder therefore should be attempting to minimize r_k of Fig. 1 when he alters the m_j's.

Without stabilizing selection, only a static monoculture is needed (as above), and the response time is no longer a relevant factor. However, if stabilizing selection is operating, then for each host genotype j there is a particular pathogen genotype, call it j for convenience, which reproduces more quickly than any other pathogen genotype on that particular host genotype. In other words

$$R_{j,j} > R_{i,j},\ i \neq j. \qquad \text{(Eq. V)}$$

Pathogen genotype j is that genotype which has a *v*-gene corresponding to each *R*-gene carried by host genotype j and no unneeded *v*-genes which reduce fitness (8). Thus, if pathogen genotype i carries all the *v*-genes in genotype j plus one extra, then a measure of the strength (8) of the *R*-gene corresponding to this extra *v*-gene in terms of stabilizing selection is $R_{j,j} - R_{i,j}$. The larger this quantity, the stronger the stabilizing selection operating against the *v*-gene in the absence of its corresponding *R*-gene, and the stronger the *R*-gene *in this particular genetic background.*

Similarly, pathogen genotype i reproduces more quickly on host genotype i than on any other host genotype; i.e.,

$$R_{i,i} > R_{i,j},\ i \neq j. \qquad \text{(Eq. VI)}$$

In this case the plant breeder can determine which pathogen genotype will eventually prevail but he is still unable to prevent domination of the pathogen population by one genotype. His objective is essentially to find that set of m_j's and that pathogen genotype k, for which r_k is minimal (Fig. 1).

Based on II and V, the best choice of host for a monoculture is that genotype h for which $R_{j,j}$ is minimized over all host genotypes j; i.e.,

$$R_{h,h} = \min_j R_{j,j}.$$

The right hand side of this equation represents the minimum, for all possible values of j, of $R_{j,j}$.

Now consider a multiline which has $m_h = 1 - S$ and $m_i = S$ where S is small enough that genotype h continues to dominate the pathogen population. Then, by II and VI

$$r_h = (1 - S)\, R_{h,h} + SR_{h,i} < R_{h,h}.$$

Hence, given stabilizing selection, a multiline can always be formed for which its asymptotic value of $(1/x) \cdot (dx/dt)$ is less than that of the best monoculture.

Predictions of the model.—The following conclusions can be drawn from the above:

(i) In the presence of stabilizing selection the readiness with which the composition of the host crop can be altered is a key factor in determining the best strategy of employing resistance genes.

(ii) If the response time is relatively short the crop composition (whether monoculture or multiline) frequently can be adjusted in response to the changing constitution of the pathogen population. Both the size and make-up of the pathogen population are under the control of the plant breeder. Multiline and monoculture techniques may prove to be useful under different circumstances.

(iii) If the response time is sufficiently long and adjustments to crop composition sufficiently infrequent to enable the pathogen to become highly adapted, the multiline cannot prevent one genotype from dominating the pathogen population. However, in these circumstances, use of multilines can minimize the size of the pathogen population and, accordingly, will be preferable to monoculture.

(iv) The greater the frequency of rearrangement (made to the crop in response to changes in the pathogen population) the lower the yield losses will be. It should be emphasized, however, that in reality the costs of monitoring, and the time and expense required in making adjustments, will generally limit the frequency with which adjustments can be made. Thus, even under the best of conditions it is unlikely that the plant breeder will be able to operate in the vicinity of α in Fig. 1.

Multiline implementation.—In nature, it will be

impossible to predict accurately the effect of a multiline on the genotypic content of a pathogen population until the multiline has been in use for some time (2).

Initial decisions concerning the choice of *R*-gene components and proportions of each in the multiline must of necessity be based on the behavior of those parasites that happen to be present and available for study when the multiline is being assembled.

When a multiline is used, and a new selective regime is imposed on the parasite, it will be important to continually monitor the changes that take place in the parasite population. Data gathered in this way dictate adjustments needed in the multiline for improving its effectiveness. Because of the theoretical expectation that parasite reproductivity becomes maximal when all parasites are of one genotype (Fig. 1), the changes made to the multiline will be aimed at those components that carry the greatest load of the most common pathogen. These multiline components need not be removed altogether; it may be sufficient to reduce the proportions occupied by them in the multiline (a procedure that would probably be assisted, to some extent, by natural selection). It will be evident, of course, that the incorporation of new *R*-genes (as they become available) also will improve the performance of the multiline.

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