Calculation of Selection Coefficients Against Unnecessary Genes for Virulence from Field Data

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ABSTRACT

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A model of the decline in frequency of an unnecessary virulence gene in a pathogen population following the mass removal of cultivars with the corresponding resistance gene in an agricultural situation is presented. The model is based on both forward and backward mutation and on selection for virulence and for avirulence in the presence and absence, respectively, of the allele for resistance in the host population. The model is used to estimate the fitness difference between avirulent and virulent races from field data that show declining virulence gene frequencies with time. One set of data was the decline of virulence in *Puccinia graminis tritici* (the cause of stem rust) to the *Sr*6 resistance gene in wheat (*Triticum aestivum*) in Australia between 1948 and 1955. The second was the decline of virulence in *Erysiphe graminis hordei* (the cause of powdery mildew) to the *Mla*6 resistance gene in barley (*Hordeum vulgare*) in the United Kingdom between 1969 and 1975. In both cases the estimated fitness differences were of the order 4–6%, the avirulent races being fitter. These values are considerably smaller than has frequently been assumed and imply that the use of multiline cultivars is more likely to provide directional selection in favor of complex rather than simple races of pathogens.

Additional key words: stabilizing selection.

Considerable debate has taken place recently as to the likely efficacy of multiline cultivars in stabilizing complex races of plant pathogens (1–4,6–9,11,17,22). Little is known either practically or theoretically about the effect of multilines on pathogen race structure. The single most important question debated is whether complex races (possessing all or nearly all the virulence genes corresponding to the resistance genes in the multiline) will ultimately predominate in the pathogen or whether multilines will prevent the buildup of complex races and will result instead in the predominance of simple races (pathogen genotypes with one or a few virulence genes). Pathologists and breeders are divided on this issue, and with the lack of information, reaching an informed conclusion is difficult.

One of the keys in being able to predict whether complex races will predominate lies in determining the relative fitness of the various pathogen genotypes. If the average fitness of complex races is consistently greater than that of simple races, then they should ultimately predominate. Complex races possessing virulence genes to each resistance gene in the multiline have a great advantage over all simpler races in that they can grow on all isolines, whereas a simple race with one virulence gene can grow and sporulate fully only on one isoline, and at best may also sporulate sparsely on some isolines that possess a noncorresponding resistance gene. Barrett and Wolfe (3) have correctly pointed out that sporulation on noncorresponding host genotypes should not be ignored in models of multilines, but according to Leonard (13), such sporulation of an avirulent pathogen on a resistant host is usually very small, about 2% or less of that of the avirulent race growing on a susceptible host.

The advantage of complex races may be offset by directional selection for avirulence (14), synonymous with stabilizing selection (sensu Vanderplank [19]). If a complex race with M virulence genes infects any single isoline, then M-1 of these genes are unnecessary. Vanderplank (19) has indicated that certain unnecessary virulence genes may give the carrier a lowered fitness compared with that of a pathogen genotype lacking those unnecessary virulence genes. The difference in fitness would ultimately be expressed in terms of reproductive success and would lead to selection, in this case in favor of avirulence. Vanderplank's hypothesis of stabilizing selection is still highly contentious, but he does not believe that unnecessary virulence will automatically lead to strong or even detectable stabilizing selection; in fact, he has indicated (20) that in most cases, directional selection of this kind would not occur, because in the absence of most resistance genes, virulent races are not less fit or only slightly less fit than the avirulent races (ie, the resistance gene is "weak" sensu Vanderplank).

Central to the debate on the usefulness of multilines is the actual value of the fitness difference (s) between races possessing and lacking unnecessary virulence genes. If s values (which vary according to the unnecessary gene and environment, at least) are zero or insignificant, then complex races will possess large selective advantages over simple races in multilines; however if s values are significant, then simple races may on the average be fitter. Until accurate and realistic estimations of s are made, predictions of the effect of multilines in stabilizing complex races cannot be made (3). Most calculations of the values of s or similar terms have come from mixed race experiments in the glasshouse, in which the proportion of two races of a pathogen, one possessing and one lacking the allele under study, are monitored over successive generations (12,13,19). As Leonard (13) and others have pointed out, some studies of this type have limited value because the effects of the background genotype of the pathogen on fitness were disregarded. Elimination of such background genetic spuriae can be attempted either by using isogenic or near isogenic pathogens differing only in the single virulence allele or by bulking large numbers of different isolates of the races. Leonard (13) adopted this latter approach and used 50 isolates for each of two races of *Bipolaris maydis* in order to provide sufficient background genetic diversity so that genetic differences other than those for virulence would tend to be canceled or nullified. The value of s that Leonard calculated was 0.12. However, the highest s value he determined was 0.42, from data on races of *Puccinia graminis* carrying unnecessary virulence to the *Sr*6 resistance gene in wheat (13). We are unaware of any publication where s values have been determined from field data.

The purpose of this paper is to present two examples in which s values have been calculated from suitable field data and to comment on their potential significance to the debate on multiline theory. It is well, first, to define clearly what we mean by the selection coefficient, s. It is the average difference in fitness between a race carrying a virulence allele (V) and one carrying the

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corresponding avirulence allele (A) when both races are growing on a host susceptible to both races, ie, a host that renders the virulence allele unnecessary. When the allele carried by the race is A, the average fitness is 1; when the allele is V, the average fitness is $1 - s$. The fitness of the virulent race is reduced by s to a certain fraction of that of the avirulent race, and the fraction is also a measure of the strength of selection that would act against the virulent race when growing in a pure stand of the susceptible host. Naturally, on the resistant host, the virulent race would be much fitter than the avirulent race.

MATERIALS AND METHODS

***Puccinia graminis tritici* in Australia from 1948 to 1955.** In the mid-1940s the wheat (*Triticum aestivum* L. em. Thell) cultivar Eureka was discarded by farmers in Australia following the failure of its former resistance to *Puccinia graminis tritici* (19,21). In 1945, the acreage of Eureka was about 18% of the total wheat acreage in northern New South Wales and Queensland, and about 70% of all collections of the fungus possessed virulence to *Sr*6 carried by Eureka. By 1948, the acreage of Eureka had declined to about 1.5% of the total, but the percentage of isolates that could attack Eureka had dropped only to about 56%. From 1948 on, the frequency of *Sr*6 virulence rapidly declined, and by the 1956 rust race survey no isolates had virulence to this gene. Thus it is advocated (19) that directional selection caused the very dramatic change in frequency of the Eureka-attacking races over a short period, ie, from 56% to zero in just 8 yr in the near absence of selection for virulence. The percentage of Eureka remained roughly constant at about 1.5% of the total wheat acreage during this period. The decline in virulence occurred because the pathogen population with virulence against *Sr*6 must have suffered a selective disadvantage compared to the population carrying the corresponding avirulence gene. One could argue that because the cultivar Eureka with *Sr*6 was rapidly replaced by cultivars with the *Sr*11 resistance gene (15,21), the decline in virulence to *Sr*6 was a result of the increase in a pathogen genotype possessing only virulence to *Sr*11 but not to *Sr*6. This would mean that the decline in virulence to the latter (and the increase in frequency of the corresponding avirulence allele by a "hitchhiking" effect [23]) would not be due to any inherent selective disadvantage of unnecessary virulence. This argument seems feasible because *P. graminis tritici* in Australia reproduces asexually; hence rapid sexual recombination of genes cannot occur. However, the asexual populations of *P. graminis tritici* in Australia are very large and the number of uredospores produced annually is enormous. Many of these will carry combined virulence to two resistance genes, due to recurrent mutations. Day (4) citing Slootmaker in a personal communication stated that *Erysiphe graminis hordei* at the 10% level of infection may produce 10^{13} spores per hectare per day. Assuming a similar rate of production for *P. graminis tritici* and a mutation rate of 1×10^{-7} to virulence to *Sr*11, then daily 1×10^{6} uredospores with this virulence gene are produced per hectare. The expected number carrying virulence to *Sr*6 in addition is $q \times 10^{6}$, where q is the frequency of virulence to *Sr*6 in the population. At the time of introduction of *Sr*11, q was greater than 0.5 (21). Thus, each hectare would have generated several hundred thousand uredospores carrying combined virulence daily, and the likelihood of a pathogen genotype carrying only virulence to *Sr*11 being selected is very small. Instead, it is much more likely that pathogen genotypes carrying virulence to *Sr*11, combined with either virulence or avirulence to *Sr*6 in proportion to the frequencies of the latter alleles were selected. This means the decline in virulence to *Sr*6 must have occurred by selection against the unnecessary virulence gene and not by a "hitchhiking" effect during selection for *Sr*11.

In order to calculate the value of s, we have modeled the decline in the virulence gene frequency as follows, with the variables and constants shown below:

p_k = frequency within the population of the avirulence allele (A) to *Sr*6 after k generations of selection.

q_k = frequency within the population of the virulence allele (V) to *Sr*6 after k generations of selection; $p_k + q_k = 1$.

μ_a = forward mutation rate from avirulence to virulence.

μ_v = backward mutation rate from virulence to avirulence.

m = proportion of susceptible (non-Eureka) hosts.

n = proportion of resistant (Eureka) hosts; $m + n = 1$.

t = the fitness difference of an avirulent race and a virulent race growing on a resistant host; ie, if the fitness of the latter is 1, then that of the former is $1 - t$. If there is no sporulation of the avirulent race on the resistant host, then $t = 1$. As Leonard (13) had indicated, values of t are usually very close to 1 (0.98 or greater).

s = the selection coefficient as defined above.

Generally the fitness of the avirulent race (W_a) and the virulent race (W_v) are given by the equations:

$$W_a = m + n(1 - t) = 1 - nt \tag{1}$$

$$W_v = m(1 - s) + n = 1 - sm \tag{2}$$

We can model the decline in the frequency of the virulence gene as follows: the race with allele A has a frequency ($k = 0$) of p_0 and a fitness (W) of $(1 - nt)$; that with allele V has a frequency of q_0 and a fitness of $(1 - sm)$.

In the next generation:

$$p_1 = \frac{p_0(1 - nt) - p_0\mu_a + q_0\mu_v}{1 - p_0nt - q_0sm} \tag{3}$$

and

$$q_1 = \frac{q_0(1 - sm) + p_0\mu_a - q_0\mu_v}{1 - p_0nt - q_0sm} \tag{4}$$

where the denominator term is a necessary division factor ensuring that p_1 and q_1 sum to 1 and is obtained by summing the two numerator terms:

$$\begin{aligned} &p_0(1 - nt) - p_0\mu_a + q_0\mu_v + q_0(1 - sm) + p_0\mu_a - q_0\mu_v \\ &= p_0(1 - nt) + q_0(1 - sm) = p_0 - p_0nt + q_0 - q_0sm \\ &= 1 - p_0nt - q_0sm \end{aligned}$$

The values of p_2 and q_2 and generally $p_{(k+1)}$ and $q_{(k+1)}$ are easily found from p_1, q_1 and p_k, q_k, respectively. A computer program of the model was written and used to calculate successive values of p and q if p_0, n, μ_a, μ_v, s, and t are given suitable numerical values. If this is carried out for a large number of generations (500–1,000) with $q_0 = 1$ initially, the value of q first declines slowly, then rapidly over a relatively small number of generations, and finally slowly again as it approaches zero. Many generations may then occur without any detectable change in q if the latter is monitored using standard race survey sampling techniques. Eventually, a balance value of q approximately equal to μ_a/s is reached. In small overwintering or oversummering populations, the virulence allele may be temporarily lost by drift effects, but it will always be generated again by mutation as soon as reproduction occurs in the next season.

If μ_a is varied from 1×10^{-6} to 1×10^{-5} or μ_v is varied from 1×10^{-9} to 1×10^{-7}, no significant change in the rate of decline of q occurs for the first few hundred generations. Generally the μ_a/μ_v ratio would be expected to lie between 10 and 1,000, for fungi (5,13,17).

The model is much more sensitive to the value of s. As s is increased, the flat tails of the asymptotic curve—where q is close to zero or one—are reduced in length and the gradient of the curve over the region of fastest decline ($0.30 < q < 0.70$) becomes steeper. The same occurs as n is reduced for any given value of s. This is expected because as n is reduced, selection for the virulence gene is less.

The model was used to estimate s for the *P. graminis tritici* data by using various values of s until the simulated rate of decline of q equaled the rate observed in the field. The number of pathogen generations was taken as 10–15 annually (K. J. Leonard and R. A. McIntosh, *personal communications*); thus from 1948 to 1955,

when the observed value of q fell from 0.560 to 0.085 (19,21), between 70 and 105 generations of selection had possibly occurred. A 7-yr period was chosen rather than the 8-yr period ending in 1956 when q was found to be zero, because of the asymptotic nature of the decline in q with time. When q is estimated as zero from a race survey, a small error in its estimation can cause a large error in the calculated number of generations of selection.

The simulation was run with the following numerical values: $\mu_a = 1 \times 10^{-6}$, $\mu_v = 1 \times 10^{-8}$ (these were arbitrarily chosen because precise values are not known nor required. This is because the decline in q as given by the model is very insensitive to changes in μ_a and μ_v. Esser and Kuenen [5] cite estimated rates of mutation for fungi as $1 \times 10^{-7} - 1 \times 10^{-6}$ for forward mutation and $1 \times 10^{-9} - 1 \times 10^{-7}$ for backward mutation), $m = 0.985$, $n = 0.015$, $p_0 = 0.44$, and $q_0 = 0.56$. The common avirulent race 126-6 at the time of introduction of Eureka failed to sporulate on Eureka (21); thus $t = 1.0$. The value of s was varied until one value caused q to fall from 0.560 to 0.085 in exactly 70 or 105 generations.

***Erysiphe graminis hordei* in the United Kingdom from 1969 to 1975.** In the early 1960s, cultivars of barley (*Hordeum vulgare* L.) carrying the *Mla*6 gene for resistance to powdery mildew caused by *Erysiphe graminis hordei* became popular in Europe. As a consequence, *Va*6 (virulence gene to *Mla*6) genotypes of the pathogen increased in frequency. At this time, barley cultivars with the *Mlg* resistance gene were also common; hence the corresponding virulence gene *Vg* was widespread within the pathogen population. Thus Wolfe and Schwarzbach (24) reported that in the U.K. physiologic race survey in 1968 all samples tested from *Mlg* or *Mla*6 hosts possessed both virulence genes *Vg* and *Va*6. The pathogen genotype with both these genes was genetically flexible in being able to infect cultivars with genes *Mlg* and *Mla*6 together or separately, conferring a large selective advantage over single virulence genotypes.

In the late 1960s, *Mla*6 cultivars were rapidly withdrawn from the United Kingdom because they became unpopular due, amongst other reasons, to the ineffectiveness of the resistance. This had the effect of reducing the selective advantage of the combined virulence, and the frequency of *Vg* + *Va*6 from *Mlg* hosts fell progressively and significantly from 100% in 1968 to 35% in 1975 (24). Thus during this time, the unnecessary virulence gene *Va*6 was gradually lost from *Mlg*-attacking races. It was replaced by its avirulence allele, which we may call *Aa*6. Such a decline in the virulence gene frequency is consistent with the causative force being directional selection, *Va*6 having a fitness of $1 - s$ compared with a fitness of 1 for *Aa*6.

The value of s can be calculated in a way similar to that described above for *P. graminis tritici*. The value of q_0 was taken as 0.87 (the value of q for 1969 [24]); p_0 thus = 0.13, $m = 0.957$, and $n = 0.043$ (an average for these years calculated from data from M. S. Wolfe, *personal communication,* and the cereal seed sales surveys of the U.K. Ministry of Agriculture, Fisheries and Food, Cambridge); t, μ_a and μ_v were as above. The number of *E. graminis hordei* generations was calculated using meterological data (Meterological Office, Head Office, Bracknell, England) for the years 1969–1975 and data on the incubation period of barley mildew in the United Kingdom (10). For the 6 years in question, between 158 and 268 generations were estimated to have occurred. Wolfe (*personal communication*) stated that 28–43 asexual generations could be expected annually, giving 168–258 in total. The two independent estimates are similar.

The simulation was run with various values of s until a drop in q from 0.87 to 0.35 was obtained in exactly 158 and 268 generations.

RESULTS AND DISCUSSION

Calculation of selection coefficients from field data. For *P. graminis tritici* virulent on *Sr*6 hosts, the required drop in q from 0.560 to 0.085 occurred in 70 generations when s was 0.0524 and in 105 generations when s was 0.0400. Thus the virulent races of *P. graminis tritici* were between 4.00 and 5.24% less fit than their avirulent counterparts in northern New South Wales and Queensland between 1948 and 1955.

For *E. graminis hordei* virulent on *Mla*6 hosts, the required drop in q from 0.87 to 0.35 occurred in 158 generations when s was 0.0608 and in 268 generations when s was 0.0543. Thus the virulent races of *E. graminis hordei* were between 5.43 and 6.08% less fit than their avirulent counterparts.

The values of s obtained, which range from 4.0 to 6.1%, are very small in comparison with the fitness differences between avirulent and virulent races on resistant hosts, where the former may be 98–100% less fit. They are also small in comparison with the values obtained by other authors for rust and mildew fungi using other techniques (3,12,13). For example, using Australian data for *P. graminis tritici*, Leonard (13) obtained a value for s of 0.42 for races virulent on *Sr*6 cultivars. He calculated this value from inoculation experiments of mixed avirulent and virulent races of the pathogen under glasshouse conditions. Leonard (*personal communication*) has suggested that a possible reason for the large difference between his and our values of s is that selection in the field may be less intense than it is in glasshouse experiments, in which pathogen population densities may be much higher than in the field. Another difference is that our s values take into account selection during the survival phase between seasons, unlike that of the glasshouse experiments.

The selection coefficients calculated in this paper lie below the values generally assumed to be typical and hence utilized in models on the usefulness of multiline cultivars. For instance, Barrett (1,2) used values of s as high as 0.1 and 0.5 and concluded (2) that "super races" may not necessarily evolve in such circumstances.

Significance of low s values for disease control in multiline cultivars. If our values of s are typical for virulent races of pathogens with unnecessary virulence genes growing on susceptible hosts, then complex races in multilines should have large selective advantages over simpler races, according to models of race growth in multilines. This can be seen if values of $\hat{m}$, the number of virulence genes carried by the pathogen genotype that shows optimal fitness for a multiline, are calculated following the method of Barrett and Wolfe (3). These authors showed that:

$$\hat{m} = \frac{-\alpha - n \cdot \log_e (1 - s)}{\alpha \cdot \log_e (1 - s)} \qquad (5)$$

where n is the number of equally represented isolines in the multiline and α is the realized reproductive rate. The multiplication rate is $(1 + \alpha)$, however (1). Thus, if X pathogen individuals exist at genèration 0, then $X + \alpha X = X (1 + \alpha)$ individuals exist at generation 1. Hence if $\alpha = 1.0$, the population doubles each generation.

Values of $\hat{m}$ were calculated for various values of α and n, assuming a constant value of s of 0.061 (the highest figure obtained above). The higher the value of α and the lower the value of n, the more likely it is that the multiline will select for the most complex races, if s is about 6% for each unnecessary virulence gene (Table 1).

TABLE 1. Values of $\hat{m}$ required to maximize pathogen fitness on a multiline cultivar for ranges of n and α with s set at 0.061[a,b]

	α			
n	0.1	0.5	1.0	10.0
2	−4.102[c]	11.898	13.898	15.698
5	−34.102[c]	5.898	10.898	15.398
10	−84.102[c]	−4.102[c]	5.898	14.898
15	−134.102[c]	−14.102[c]	0.898[c]	14.398

[a] $\hat{m}$ = the number of virulence genes that maximize the fitness of the pathogen, n = the number of equally represented isolines in the multiline, s = the selection coefficient acting against each virulence allele in the absence of a corresponding host resistance gene, α = the realized rate of reproduction. Thus if X pathogen individuals exist at generation 0, $X + \alpha X$ individuals exist at generation 1.

[b] Where values of $\hat{m}$ fall outside the range 1 to n, take true $\hat{m}$ as 1 when the table value is negative or less than 1 and n when the table value exceeds n.

[c] Simple races are fitter than complex races. In all other cases, the most fit races are complex.

We believe that for airborne diseases such as rusts and mildews, which multilines have been designed to control, values of α will be 5–10 or more. This can be seen if α is converted into a more familiar term, the logarithmic infection rate, r_l, given by Vanderplank (18) in the equation:

$$X_t = X_0 e^{r_l t} \quad (6)$$

where X_t and X_0 are amounts of disease at t and 0 days, respectively. The term r_l is related to α as shown:

$$X_0 e^{r_l t} = X_0 (1 + \alpha) \quad (7)$$

Hence

$$r_l = \frac{1}{t} \log_e (1 + \alpha) \quad (8)$$

If $\alpha = 5.0$, $r_l = 0.223$, and if $\alpha = 10.0$, $r_l = 0.299$, assuming a mean generation time, t, of 8 days.

Values of r, (apparent infection rate, where $r_l > r$) for rusts may be as high as 0.4 under very favorable conditions (18). Fried et al (7) estimated values of r for powdery mildew of wheat in multilines to be in the region of 0.20–0.23. Since values of r_l are greater than values of r, values of α should exceed 5.0 and may even be greater than 10.0.

In commercial multilines, n varies from 6 to 15 (16). However if Vanderplank (20) is correct and significant selection against most unnecessary virulence genes does not occur, then a value of $s = 0.061$ would only be realistic for a few of the 6–15 corresponding unnecessary virulence genes. For the rest, s values may be much lower. Table 1 was calculated on the basis that all n corresponding unnecessary virulence genes carry the same fitness loss, $s = 0.061$. Realistic values of n where $s = 0.061$ are therefore lower than 6–15. From Table 1, one can predict that with low values of n, 2 or 5, or with high values of α, 5 or 10, complex races will dominate the race structure in multilines, if these s values are typical for the relatively few unnecessary virulence genes that are subject to strong selection. Other workers have concluded the opposite, that simple races should be selected in multilines more often than not (3), but the values of s required, 0.10–0.75 (1–3), we believe to be unrealistically high.

Barrett (1,2) has recently introduced into his models the distinction between infections produced by inoculum originating from the same plant (autoinfection) and infections produced by inoculum originating largely from different plants (alloinfection). He has shown that if autoinfection rates are high, then the fitnesses of simple races on multilines will be much higher than predicted by models that assume unrealistically that all spores produced enter an aerial pool and infect randomly. However, the incorporation of alloinfection and autoinfection rates, ϕ and $1 - \phi$, respectively, into multiline models will not alter the conclusions drawn here, provided that s values are about 0.06 and ϕ is large. Using Barrett's (2) three-host component model, we have shown that when $\phi > 0.21$, the complex race eventually predominates with frequencies greater than 0.5; this occurs by 48 generations if $\phi > 0.25$. If ϕ is very small, ie, $\phi < 0.10$, then the most simple races quickly predominate in the multiline. Values of ϕ are not easy to determine, but for windborne pathogens of cereals, alloinfection rates on the average are likely to be greater than autoinfection rates. Leonard and Czochor (14) reported ϕ values of 0.90–0.95 for *P. graminis avenae*, from Leonard (11), who concluded that the nonrandom component of dissemination (autoinfection) had a negligible effect on rates of increase of simple races in mixtures of susceptible and resistant plants. Leonard and Czochor (14) also cited evidence for this conclusion from work on *Pyricularia oryzae*, the cause of rice blast.

The model we used to estimate values of s requires knowledge of a number of other parameters, some of which are known fairly precisely, namely q_0 and q_k (virulence gene frequencies), m and n (host gene frequencies), and t (the selective coefficient against avirulence). Others are known with less precision, namely μ_a and μ_v (the mutation rates) and k (the number of pathogen generations). As discussed above, the model is very insensitive to changes in μ_a and μ_v. The model is also relatively insensitive to error in estimates of k. For instance, for the *Puccinia graminis tritici* data, if instead of a minimum-maximum generation range of 70–105, s values are calculated assuming only half these generation numbers, ie, 35–52, then the range in s is increased only to 0.0652–0.0893 instead of 0.0400–0.0524. Similarly for the *Erysiphe graminis hordei* data, an s range of 0.0543–0.0608 assuming 158–268 generations is increased only to 0.0674–0.0766 if the generation numbers are halved.

Despite our prediction that complex races will dominate the pathogen race structure based on these models (2,3), the conclusion reached by Barrett (1,2)—that there will be less disease in multilines compared with that in pure stands—is not challenged. Regardless of which races predominate, the models predict there should always be less disease in multilines than in pure stands, other things being equal. These predictions have been based upon mathematical models that include directional selection for avirulence as the only factor opposing complex race buildup. We accept that this may prove to be an oversimplification in practice, but our basic conclusion concerning the likely dominance of complex races we consider to be a valid and timely warning.

Barrett and Wolfe (3) have indicated that values of s may not be constant. It is reasonable to expect s to vary according to the host resistance gene involved, the pathogen background genotype, and environmental factors. The values of s reported here have been obtained from long-term field data and represent the mean selection pressures averaged over the various stages of several seasons for pathogens with population sizes that show marked seasonal fluctuations. We consider that the use of field data, provided that they are derived by reliable sampling, is likely to provide the most realistic estimates of the magnitude of directional selection for avirulence.

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