Letter to the Editor

Rebuttal to "Multilines and Super-Races – a Reply"

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The model of Barrett and Wolfe (1) appears to be logically and mathematically sound. It also appears to represent an extension of my model, but one must suspect that the authors have been thinking about their model for some time, and have simply adapted portions of a more complete effort to fit my assumptions, symbols, and development. Their complete model should prove to be a worthy contribution. My further comments here are directed toward acknowledging, clarifying, or qualitatively extending their model and, briefly, toward defending the use of a discrete model, such as mine, both as a first effort and, in this instance, as realistically describing reproduction of the pathogen under certain circumstances. Barrett and Wolfe have presented three separate points to which I shall respond:

Point one, that of allowing for partial reproduction by the pathogen, when incorporated into their model, will be a major step toward realistically describing agricultural host-parasite systems and, perhaps even more so, natural systems.

Point two, the logical basis for multiplying fitnesses, rather than adding selection coefficients, was not made in my letter (3). A more complete comparison of the additive and multiplicative versions is seen when Fig. 1-A and 1-B are compared in my model (3). Here it is obvious that at high m or s values, the two become quite different. While point two amplifies the problem in using an additive model, it adds little to the argument.

Point three presents a dimension that should make the model of Barrett and Wolfe more flexible than mine. The remainder of this rebuttal will be confined to this point and will indicate a problem with it as well as positive aspects that are not brought out by them. The sequence of events in developing ecological and genetic population models generally begins with an assumption of discrete, nonoverlapping generations (2). In some cases these assumptions may prove to be accurate but often they are not, and such models are modified (and made more complex) to more closely fit the behavior of real populations. This is what Barrett and Wolfe have done. Briefly, they indicate, using the variable α, that overlapping generations will curtail the process of replacement of simple races by complex races, and this curtailment will be of greater significance if the proportion of parent lesions is high in all succeeding generations. Probably owing to the concise way they chose to present this variable, Barrett and Wolfe still elect to call their model simple. In one important sense it is far from simple: according to their model, pathogen population structure is a function of population dynamics (population growth). In view of the difficulty we have in accurately measuring pathogen increase, this is a sobering thought. Since their model may approximate reality with some pathogens this should not be taken as a criticism, but rather a warning: testing or proving such a model under a wide range of conditions will be quite difficult.

Under favorable conditions and in homogeneous host stands, rates of increase of those diseases (cereal rusts and mildews) for which multilines have been proposed or developed, are very high. Van der Plank [(5) page 23] cites an example for wheat stem rust in pure susceptible stands in which α can be estimated to be in the range of 5–10, depending on what portion of a generation is represented by 5 days. Table 1 of the Barrett and Wolfe model shows that an α of 10 gives a result that is quite similar to that seen in my multiplicative model [(3) Fig. 1-B]. Other estimates of r (5) for such diseases, usually expressed on a per-day basis, generally indicate that α is large enough under even only moderately favorable conditions to allow my model to approximate reality in this respect. For example, an α of 10 corresponds to a logarithmic infection rate of 2.30 per generation or .23 per day if generation length is 10 days. Much higher rates than this are common under favorable conditions for disease development (5). Opposing this is the fact that multilines slow disease increase (4) and hence reduce α. The Barrett and Wolfe model predicts that if we can slow an epidemic sufficiently we will gain an additional long-term victory in that we may prevent adverse changes in the pathogen population. This has the effect of causing the outcome to be a function of n (the number of components) which my discrete model did not predict. The use of chemicals as a supplemental measure is another approach, one that Barrett and Wolfe have advocated (Barrett, *personal communication*). Another interesting complication is that rates of disease increase over time are characterized by an S-shaped curve. During the early and late stage of epidemics, rates of increase may be low enough to disallow selection for complexity. This would mean that the time during which selection for complexity operates will be something less than the entire season. In short, anything that reduces α to a value at which selection for pathogen complexity will not occur should be considered. It seems likely that given the cost of producing multilines and the limited number of genes at our disposal, we will not, using multilines along, be able to reduce α sufficiently to prevent selection in the pathogen for some level of greater complexity. Admittedly the effect of using partial resistance genes [or as Leonard (4) advocated, a susceptible component] could change this forecast. As indicated in my letter (3), however, there is a price to be paid for taking such a course. Pathogen population stability can only be obtained if we are willing to allow some disease on the crop.

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LITERATURE CITED

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