## Disease progression in common root rot

Morrall and Verma in their letter (4) make two objections to the results that I presented in "Disease progression in common root rot of spring wheat and barley", Can. J. Plant Pathol. 2: 187-193, 1980.

Their first objection involves a misrepresentation of their own paper, since data in the reference cited (11) are inadequate to distinguish between the two disease models mentioned, and their "conclusion" that the disease is of the simple interest (SI) type is not a conclusion but an assumption. They presented no data in either their 1974 publication or in their other papers from which one can conclude that disease fits the SI model.

The basic difficulty with this root rot disease is that one cannot directly study lesion expansion or look for secondary infection in the natural system. One must resort to some kind of destructive sampling and rely on seeking trends in large populations. Direct study is possible only under artificial conditions.

As Morrall and Verma admit, nearly all measurements of lesion-type diseases are composites of number and size of lesions. The effect of the individual components on the pattern of disease development has not been examined and would be an interesting study for computer simulation modeling. Since the relative importance of the two components has not been defined, just where the interaction becomes important, and how much so, rests with the judgement of the investigator. Morrall and Verma cite one extreme example where lesion expansion was more important and then generalize from that. They ignored other reports (8) that new infections can occur throughout the season. My own observations agree with those of Tinline (8).

If one accepts Morrall and Verma's argument that lesion expansion is responsible for most of the disease progression (I do not), then one must postulate a very peculiar pattern of lesion growth to account for the observed shapes of the disease progress curves. A lesion expansion rate, or a changing host susceptibility which exactly mimics logistic disease progression, while not impossible, seems much less likely than the possibility of secondary autoinfection. My proposed compound interest disease (CI) model for common root rot precludes the necessity for such a cumbersome and unnecessarily complex lesion-growth model. Morrall and Verma imply that no one has ever observed secondary infection in this disease. They wish us to conclude that secondary infection is not present, when actually no one has ever looked for it directly.

Their second objection, that it is incorrect to draw biological conclusions from mathematical relationships, indicates their failure to comprehend the whole idea of quantitative epidemiology. In its infancy, quantitative epidemiology has been purely descriptive, but as it matures it must become both analytical and predictive. My paper is an attempt to do this. Morrall and Verma object to such an application of Van der Plank's transformations. They choose to ignore the analysis of nontransformed data using the Weibull probability distribution function which leads to the same conclusion. In summary:

1) The data of Verma et al. (11) are inadequate to distinguish between the models of Van der Plank (10). Morrall and Verma's "conclusion" that the simple interest (SI) model is correct is no conclusion but an assumption based on conventional thinking about this particular disease.

2) Stack's data (6) are adequate for such analysis and indicate that something is causing a disease progression to stray from the SI pattern. Changes in host susceptibility associated with ontogeny and environmental changes did not provide an answer (6, Table 1).

3) Stack employed two methods of analysis, one using transformations of Van der Plank, the second using the Weibull distribution fitted to nontransformed data; both gave results consistent with the disease progression being of the CI type (6).

4) Stack's (6) proposal that secondary autoinfection is responsible for this observed disease progression is consistent with his data and with the reports of continuing new infection during the season (8) and of increase in population of spores during the season (2). It also explains why evaluations of disease on seedlings or young plants are not correlated to adult plant disease levels (3,7) and offers a plausible explanation for the highly variable correlation between adult plant disease and yield losses (9).

5) Finally, Stack's proposal is not an isolated case of re-thinking an old and supposedly well-studied disease. Another root-crown lesion type disease has been shown to exhibit disease progression not fitting the SI model (1); and a pattern of virus spread, formerly assumed to result only from primary infections, has been shown to exhibit an anomalous growth pattern satisfying the CI model (5). It seems likely that as quantitative epidemiological methods are applied more widely, other examples of changing concepts of disease will appear.

1. **Campbell, C.L., L.V. Madden,** and **S.P. Pennypacker.** 1980. Structural characterization of bean root rot epidemics. Phytopathology 70:152-155.

2. **Chinn, S.H.G.** 1965. Changes in spore populations of *Coch-*