CANADIAN JOURNAL OF PLANT PATHOLOGY 3: 182-185, 1981

Letters to the editor/Lettres à la rédaction

Disease progress curves, linear transformations, and common root rot of cereals

In a recent paper R.W. Stack (5) concludes that common root rot disease "is of the compound interest type, although perhaps not of the exact logistic model." This constrasts with an earlier report from Saskatchewan (11) that common root rot is a simple interest disease. In our opinion Stack has made two errors that leave substantial doubt about his conclusion. First, he has applied Van der Plank's (8) transformations to measurements of disease in an invalid manner. Second, he has tried to draw a biological conclusion from mathematical relationships without sound biological support for the conclusion.

Stack (5) measured disease by estimating the proportion of subcrown internode tissue covered by lesions. Plants were assigned to four severity categories: — 0.01, 0.20, 0.50 and 0.99 of the subcrown internode diseased (discolored). The mean proportion of subcrown internode diseased in the plant population was called the disease proportion (5, Fig. 1) and was subjected to Van der Plank's simple interest and compound interest transformations. Although slightly different weightings have been given to the four severity categories, Stack's "disease proportion" is a very similar measure to the "disease rating" referred to in the earlier paper from Saskatchewan (11) and used by many other Canadian workers (3). However both measurements of disease combine the proportion of infected plants with the disease severity (i.e. size of lesions) on each infected plant. Thus changes of disease proportion in relation to time in a plant population can be greatly affected by the spread of lesions on individual plants. Lesion spread is extensive during the growing season in common root rot (10, 11). It is theoretically possible that the disease proportion or disease rating could change from 0.01 to 0.99 without a change in the proportion of infected plants, i.e. with no change in the population of diseased individuals. All that would be required is spread of existing lesions. While such extremes are unlikely, substantial changes in disease rating, due mainly to lesion spread, have been reported (11, 12). On Manitou wheat at Matador in 1970 and 1971, when infection rates were high, in some treatments disease ratings increased between Day 40 and Day 100, even though about 0.90 of the plant population was infected by Day 40 (12, Tables 2-4).

Van der Plank (8) specifically excludes lesion spread after infection from his analogies of disease progress with simple and compound interest increase. He states (Chapter 4, p. 30) "We shall often use the term compound interest disease when the pathogen multiplies through successive generations in the course of an epidemic. Note that the multiplication of disease . . . implies that the pathogen moves from lesion to lesion, or from plant to plant if the disease is systemic. The multiplication of the pathogen within a lesion . . . (or) plant is not relevant here." The fundamental point is that the logistic growth characteristic of compound interest diseases (13) applies to populations of "disease units" such as infected plants or lesions on plants, not to the size of the units themselves. It is true, of course, that epidemiologists studying foliar diseases such as rusts, mildews, and leaf spots frequently measure disease in terms of percentage of leaf area infected. Changes in this disease unit reflect increases in both lesion size and population size, but the logistic transformation can be effectively applied to straighten progress curves and derive infection rates (13) because the size range of lesions is small relative to the size of the organ being studied (the leaf or the whole plant). Changes in lesion size are almost insignificant. In root pathology, however, the logistic transformation should not be applied to a variable like Stack's disease proportion (5) which is known to be greatly affected by individual size (10) as well as population size. In common root rot, lesions vary from being almost microscopic in size to covering the entire subcrown internode (3, 5, 9). Thus using the logistic transformation on disease proportion values (5) is comparable to claiming that the population of trees in a forest increased because the weight of lumber harvested exceeded that of the seedling trees 100 years previously.

A more philosophical objection to Stack's conclusions centres around his statement in the Discussion, "The implication of disease progression fitting the compound interest model is that secondary inoculum is playing a role in the disease." It is incorrect to try and draw biological conclusions merely from mathematical relationships. In botanical epidemiology Van der Plank's simple interest and compound interest transformations are devices to enable the calculation and comparison of mean infection rates of epidemics, not devices to make decisions about the nature of the disease cycle. In previous work in Saskatchewan (9, 11) a variety of different curve types were fitted to disease progress data for common root rot. High R^2 values were obtained not just for the hyperbolic curve type, which is characteristic of simple interest diseases,

but for several others, including sigmoid curves. This was partly due to a low number of degrees of freedom, but also because of variability within treatments, a frequent characteristic of root disease data. However the choice of a transformation to straighten a disease progress curve and enable the calculation of an infection rate cannot be based on which type of fitted curve gives the highest R^2 value. A biological basis must exist. Thus to select the logistic or compound interest transformation one must normally have evidence of plant to plant spread in root diseases, or lesion to lesion spread in foliar diseases. Even then the transformation may not produce a perfectly straight line, especially when infection rates change.

Stack (5) recognizes that propagule spread from plant to plant is unlikely in common root rot disease. However he raises the interesting possibility of secondary autoinfection (4) causing multiple infections on the subcrown internode (6). If this were the case, it would provide the opportunity for logistic growth of a population of subcrown internode lesions (disease units) during the season. However Stack did not measure this population; his "disease proportion" confounds increasing lesion population with increasing lesion size. Secondary autoinfection will, of course, have no effect on disease progress curves based on the proportion of infected plants. In our opinion, occasional secondary alloinfection will also occur when the subcrown internodes of two plants are very close to each other in the soil. This will result in slight deviations from a simple interest disease pattern, but it is unlikely to produce the logistic progress curves that result from the general secondary plant to plant spread characteristic of compound interest diseases. The possibility of occasional secondary alloinfection between adjacent plants in the field might be investigated by using Van der Plank's "doublet method" (1, 7).

Our criticisms above should not detract from an important concept brought forth in Stack's paper, namely that an upward inflection occurred in the progress curves for disease proportion with each cultivar tested. This appeared to relate to plant phenology, not to environmental conditions; cultivars grown side-by-side varied in their stage of development when the inflection occurred. Stack suggests that this characteristic might be manipulated by plant breeders to produce cultivars with field resistance based on (dare we say it) "late inflection". Such information would never have been revealed without the frequent sequential sampling Stack carried out. It is also noteworthy that the inflection is evident without transformation of the disease progress curves; this is an excellent illustration of Kranz's (2) argument that linearization of entire progress curves may not facilitate the comprehension of epidemics because of information loss.

Finally, this letter provides a good opportunity to point out that in the earlier work from this laboratory (11), Van der Plank's (8) simple interest transformation was slightly misapplied. Instead of plotting and calculating

$$\log_e \frac{1}{1-x} \text{ in reaction to time, } \log_{10} \frac{1}{1-x}$$

was used. Consequently the infection rates cited are 2.3 times smaller than they should be. The true daily simple interest infection rates (13) for Manitou wheat on summerfallow land at Matador should have been 0.023 (1969), 0.030 (1970), 0.045 (1971), and 0.030 (mean of all 3 years).

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R.A.A. Morrall and P.R. Verma

Department of Biology, University of Saskatchewan, Saskatoon, Saskatchewan S7N 0W0 and Research Station, Agriculture Canada, 107 Science Crescent, Saskatoon, Saskatchewan S7N 0X2

Received 1981 08 05