Application of Two Epidemiological Models for the Identification of Slow Stem Rusting in Wheat

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ABSTRACT

Two stem rust (caused by *Puccinia graminis* f. sp. *tritici*)-susceptible spring wheat cultivars (Pitic 62 and Penjamo 62) were compared to the slow rusting cultivar Bonza 55 for those gross epidemiological attributes (i.e. reduced spread, rate of increase) which might characterize slow rusting. Analyses of physically isolated stem rust epidemics indicated that reduced spread and lessened rates of increase on Bonza 55 relative to the susceptible varieties, characterized slow rusting. Application of these relationships to the identification of slow rusting is proposed. This method would combine the slow rusting components of rate of disease increase and spread into a single slow rusting index.

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Additional key words: Puccinia graminis f. sp. *tritici*, disease resistance, disease spread.

It is generally acknowledged that some wheat cultivars invariably become rusted at a slower rate during an epidemic than do other cultivars. Such "slow rusters" are valuable as they retard the epidemic in field plantings. This approach to plant disease control stresses an acceptable level of disease which should have little or no effect on yield or quality. Slow rusting is thought to be more lasting than race specific resistance because it is considered not subject to sudden race changes of the pathogen (6, 7). Cryptic error (6) (i.e. excessive inoculum loads from adjacent plots) in small plot tests could, however, mask slow rusting cultivars and make them appear susceptible.

"Slow rusting" implies a lessened rate of epidemic acceleration. This effect could be the result of reduced pathogen multiplication and/or spread. Gregory (2) pointed out the usefulness of plant pathogen propagule dispersal gradients for a better understanding of disease epidemics. An application of this concept would be the determination of the plant disease spread gradient from a point source of inoculum to characterize the movement of a pathogen within populations of cultivars thought to differ in their rate of rusting.

Van der Plank (6, 7, 8) stressed the concept of disease increase as a function of time. By his method the rate (r) of disease (x) increase in time (t), corrected for decreasing amounts of healthy tissue $(1 - x)$, is given by the formula:

$$r = \frac{1}{t_2 - t_1} \left(\log_e \frac{x_2}{1 - x_2} - \log_e \frac{x_1}{1 - x_1} \right)$$

where the subscripts denote the beginning and end points of the time interval for which (r) is calculated.

Van der Plank (6) implies that varieties with only hypersensitive resistance have disease increase rates (r) similar or equal to susceptible cultivars when attacked by races which overcome this form of resistance. Conversely, "one determines the effect of horizontal (race nonspecific) resistance on an epidemic by its effect on the infection rate" (6). Slow rusters should therefore be characterized by a lower (r) value when compared to susceptible varieties subjected to the same pathogen population under the same environmental conditions.

The objective of this study was to test the application of the epidemiological models of Gregory and van der Plank for the characterization of slow stem rusting in wheat.

MATERIALS AND METHODS.—Square plots 48 rows wide by 14.4 m long of cultivars Pitic 62 (3 plots), Bonza 55 (2 plots) and Penjamo 62 (1 plot) were planted 23 December 1970 at the CIANO Experiment Station, Ciudad Obregon, State of Sonora, Mexico. The Colombian cultivar Bonza 55 was selected to represent the attribute of slow rusting. This cultivar has been in commercial production for over 15 years in Colombia, and has not succumbed to stem rust under the explosive stem rust race situation in that country (1). However, light and scattered infections in some plantings suggest some nonspecific mechanism of resistance to this pathogen. Two semi-dwarf Mexican cultivars (Pitic 62 and Penjamo 62) were selected for their susceptibility to many races of *P. graminis* f. sp. *tritici*. Each plot was intentionally isolated by eight surrounding 14.4-m square plots of

cultivated oats. One central plot of Pitic 62 was uninoculated and served as a check for interplot spread of the pathogen. Twenty tillers in the windward corner of each of the remaining experimental plots were needle
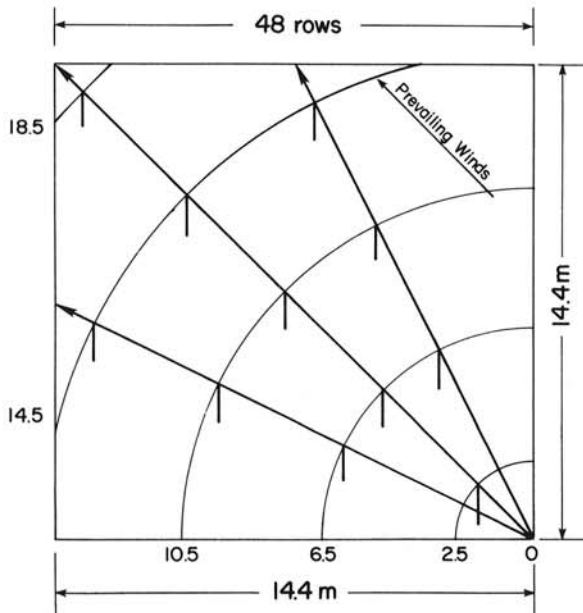

Fig. 1. General intra-plot experimental design used in studies of wheat stem rust epidemiology. The point source of inoculum is indicated as (0), five arcs of increasing distance from (0) and three vectors which served as replications for the sampling of disease movement from (0) to the sample areas of one linear meter.

TABLE 1. Components of linear regression model ($\log_{10} y = \log_{10} a + b \log_{10} X$ where $\log_{10} y$ is the predicted quantity of disease at distance $\log_{10} X$, a is the scaling factor and b is the slope of the best fitting line) for the $\log_{10}$ x-axis and $\log_{10}$ y-axis transformed Pitic 62 (2 plots), Penjamo 62 and Bonza 55 (2 plots) data. Sample periods 1 and 2 were 32 and 39 days, respectively, after inoculation of point sources with a racial composite of *Puccinia graminis* f. sp. *tritici*. The degree of goodness of fit to the linear model is indicated by the coefficients of determination (r^2_{cd}) expressed as a percentage

	$\log_{10} a$	b	r^2_{cd} (%)
Sample Period 1			
Pitic 62 Plot 1	4.63 a[2]	−2.70 a[2]	99.7
Pitic 62 Plot 2	4.62 a	−2.86 a	88.7
Penjamo 62	4.98 a	−3.82 b	99.9
Sample Period 2			
Pitic 62 Plot 1	5.62 b	−2.65 a	95.5
Pitic 62 Plot 2	5.79 b	−2.83 a	94.1
Penjamo 62	6.55 b	−4.54 b	97.9
Bonza 55 Plot 1	3.48 c	−2.33 a	89.8
Bonza 55 Plot 2	3.74 c	−3.96 b	97.6

[1]No disease intensity gradients were observed at the first sampling period for either of the two Bonza 55 plots.

[2]Values followed by the same letter are not considered significantly different, $P = 0.05$.

injected in the boot (77 days after planting) with an aqueous suspension of *Puccinia graminis* Pers. f. sp. *tritici* Erikss. and Henn. uredospores. This mixture was a composite of unidentified isolates used in the CIMMYT wheat breeding program for general rust inoculum supplemented with standard races 12 and 151. The intention of the heterogeneous rust spore mixture was to include, by chance, races of *P. graminis* f. sp. *tritici* that would be fit (sensu Darwin) and pathogenic rather than employing a single "super race" that might lack other characters for epidemiological fitness.

The intra-plot experimental design (Fig. 1) allowed replicated measurements from one linear m of row area for estimates of rates of disease increase (r) and disease spread within the experimental plots.

Frequent disease measurements would have best quantified the course of each of the intra-plot epidemics, but the physical disturbance to the infected plants could significantly alter the dispersal of uredospores. Thus, the
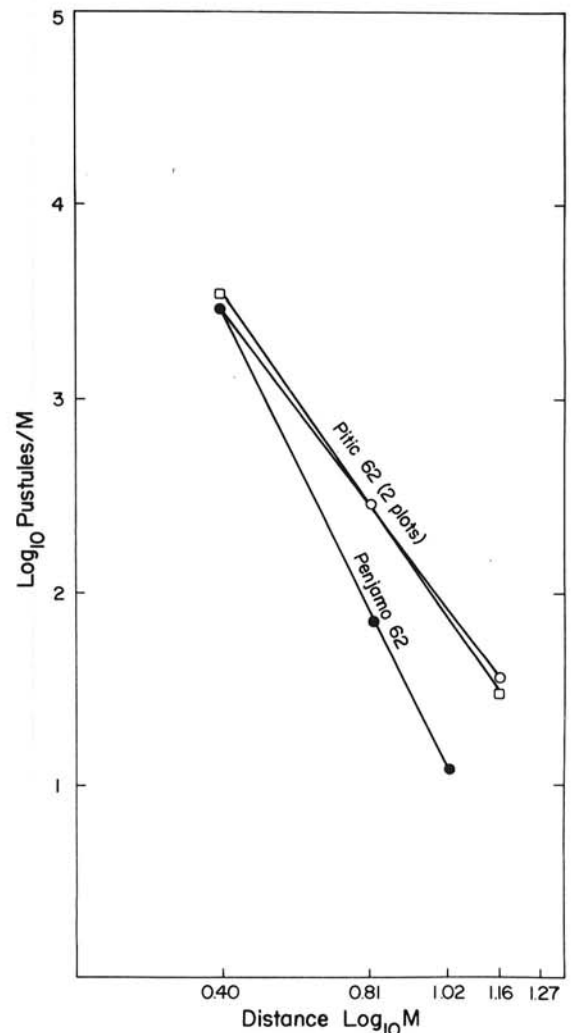

Fig. 2. Stem rust intensity gradients from point sources 32 days after inoculation of the wheat cultivars Pitic 62 (2 plots) and Penjamo 62.
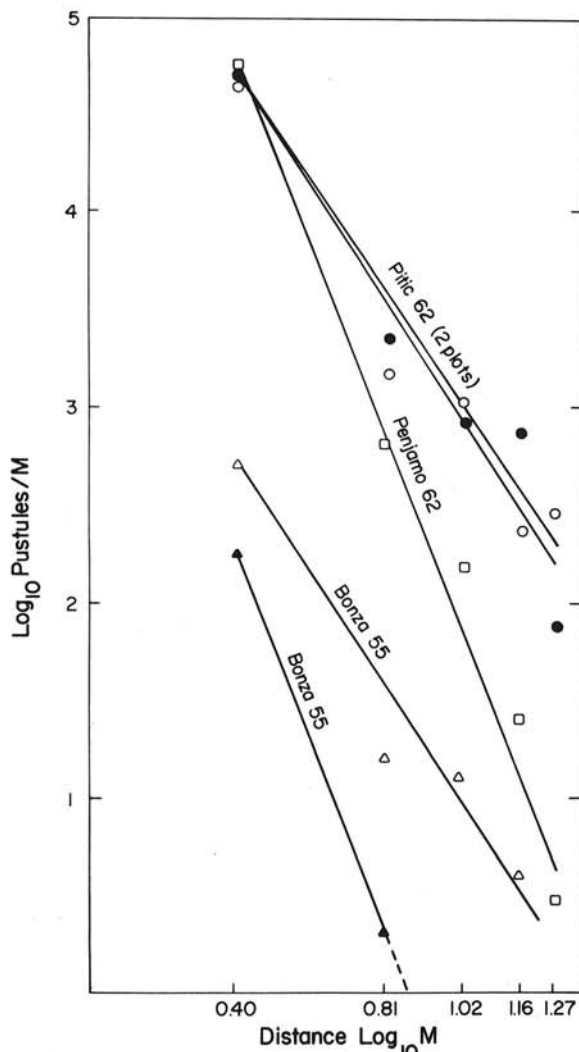
Fig. 3. Stem rust intensity gradients from point sources 39 days after inoculation of the wheat cultivars Pitic 62 (2 plots), Penjamo 62 and Bonza 55 (2 plots).
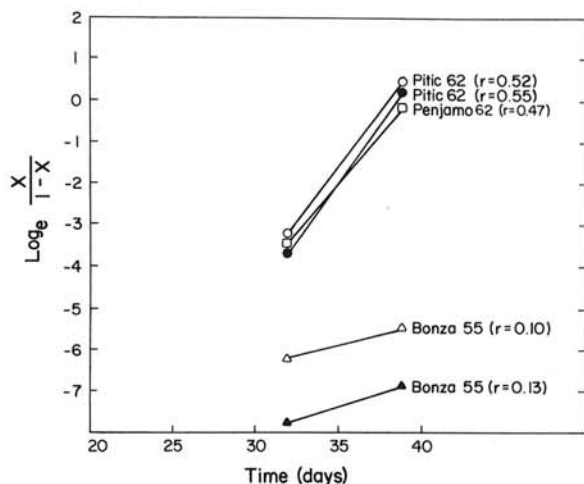

Fig. 4. Mean infection rates (r) between two sampling dates for stem rust epidemics in the sub-plots adjacent to point sources of inoculum of the wheat cultivars Pitic 62 (2 plots), Penjamo 62 and Bonza 55 (2 plots). [See text for explanation on the calculation of (r) values].

first disease assessments were delayed until the epidemics were well established (109 days from planting/32 days after inoculation).

Disease assessment was made directly by pustule count converted to per cent infection by defining 1,000 pustules per culm as 100 per cent infection (3). This conversion was found to be nearly identical to values obtained using the modified Cobb scale (4) for disease assessment.

One week following the first disease measurements, and just prior to crop maturity, a second lesion count was made. Any disturbance caused by the first pustule count was not reflected in the final count, since the latent period (i.e., time from spore deposition until new fruiting lesions would have been formed) was greater than the disease sampling interval.

RESULTS.—A negligible number of stem rust pustules was observed in the uninoculated Pitic 62 plot at the conclusion of the experiments. This suggests that little cross-interference between plots had occurred.

The disease intensity gradients from the point source of infection from the first disease assessments corrected for curvilinear relationship by $\log_{10}$ x-axis and $\log_{10}$ y-axis are given in Fig. 2. The best mathematical approximation for the relationship of disease intensity (y) to the distance from the point source (X in meters) is given by the exponential relationship $y = ax^b$. This relationship is equivalent to that of Roelfs (5) given as $\log_{10} y = \log_{10} a + b \log_{10} X$ for the dispersal of stem rust uredospores. This transformed expression was therefore used to calculate by regression techniques the best fitting lines for the $\log_{10}$ x-axis and $\log_{10}$ y-axis transformed data. In addition the calculated values for $\log_{10} a$ (scaling factor) and b (slope) coefficents of determination (r^2_{cd}) are presented in Table 1. No disease intensity gradient was observed in either plot of Bonza 55 at this time.

Identical transformations and analyses were performed on the data from the second disease measurement. The disease gradients from all five experimental plots (transformed to $\log_{10}$ x-axis and $\log_{10}$ y-axis) are presented in Fig. 3. Corresponding regressional information is given in Table 1. Significant differences in the regression slopes for the duplicate plots of Bonza 55 are considered to be the result of random sampling errors at remote distances from the point source where disease quantities were extremely small.

A graphic presentation of disease increase within the sub-plots closest to the point source of inoculum from the first to second sampling for all experimental plots is given in Fig. 4. Computation of the disease increase rates (r) by the van der Plank method (6) show the cultivars Pitic 62 and Penjamo 62 to be highly susceptible of stem rust and indistinguishable by this method. Rates of disease increase (r) in the Bonza 55 sub-plots were only one-fifth those of the other cultivars. This would be interpreted by the van der Plank model as disease rate limiting slow rusting for Bonza 55.

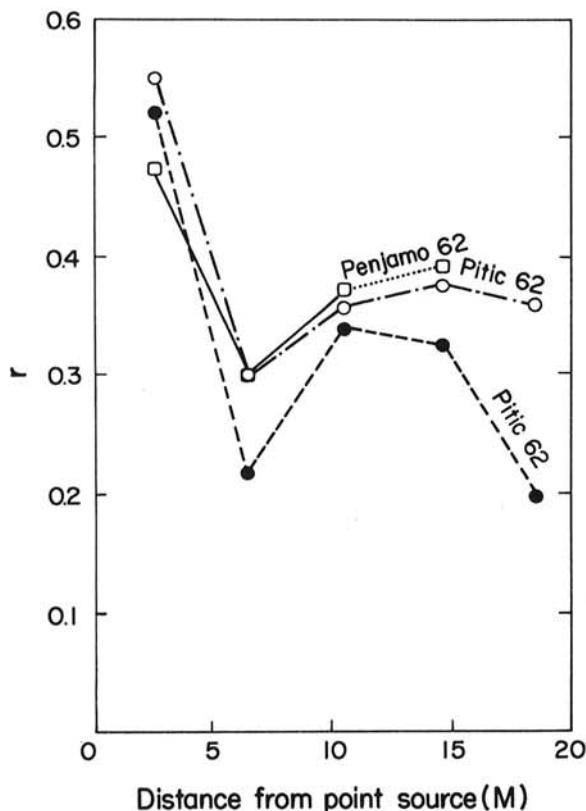The disease increase (r) values for cultivars Pitic 62

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Fig. 5. Mean stem rust infection rates of the wheat cultivars Pitic 62 (2 plots) and Penjamo 62 at varying distances from a point source of primary inoculum for the period 32 to 39 days after inoculation of plants at the point source. Calculation of (r) for Penjamo 62 at 18.5 m from the point source was not possible because no disease was observed at the first sampling.

(two plots) and Penjamo 62 were calculated for increasing distance from the point source and are given as Fig. 5. Insufficient disease increase in the Bonza 55 plots did not allow comparable calculations.

DISCUSSION.—The concept of slow rusting suggests reduced disease either in time, in space or both. Van der Plank (6) states that his analytical techniques "stress the role of multiplication rather than the spread" of disease. All of his analytical techniques assume (r) to be independent of the amount of disease. Disease intensity gradients cannot be analyzed by these techniques and are taken as an average over the entire field.

In a more recent treatment of this topic, van der Plank (8) develops the concepts of "historic factors that increase the variance of (r)." Such affects are wave like variations in (r) that can result from the arrival of a single spore shower. A similar explanation can be given for the wavelike variation in (r) measured over distance in these experiments (Fig. 5). Based on these results it is suggested that, from a point source of infection, the value of (r) may be a function of distance. This relationship must not be dismissed when interpreting slow rusting differences between cultivars even though "the mathematical difficulties of relating the infection rate and the rate of spread have remained insuperable" (8).

By the criterion of rate of disease increase, both Pitic 62 and Penjamo 62 would be considered equally susceptible to stem rust (Fig. 4). However, as indicated by the regression slopes for disease spread (b in Table 1) they differ significantly in their rates of spread. Slow rusting Bonza 55 had b-values similar to the susceptible check cultivars. This suggests that disease intensity gradients as measured by b do not indicate reduced rates of spread when considered apart from the scaling factor (a). The application of this regression technique does not overcome the previously mentioned difficulties in merging Gregorian and van der Plankian models. It does offer an alternate look at the relative rates of disease spread independent of the rate of disease increase.

It has been previously stated (2, 6, 8) that as a pathogen spreads from a point source, the disease gradient will become flattened [i.e., (b) would tend toward zero]. There was no statistical indication of this trend in these data although the disease levels in the subplot adjacent to the point source exceeded 50 per cent for the susceptible varieties. Logically, one would expect such a flattening of the disease spread gradient. It is concluded that this effect must be expressed at disease levels greater than those measured in these experiments.

The insuperable mathematical problems presented by the two models under discussion need not concern the application of this approach to the identification of slow rusting. By using a simple variation of the techniques described herein, qualitative information could be obtained on the relative slow rusting of wheat (or other small grain) cultivars. Wheat plants, heavily infected with race composites, could be placed in the windward corner of relatively small (approximately 10×10 m) isolated plots that would be expected to be otherwise free from infection. The diagonal (leeward) corner could then be checked periodically for rust occurrence. The number of days required for the leeward corner to reach a prespecified level of infection could be taken as a slow rusting index. Although far from quantitative, this index could be very informative when related to other known cultivars. Such a method would integrate all of the factors and forces of an epidemic [including (r) and rate of disease spread] into one easily obtainable index of slow rusting. More detailed information would not be necessary since, as demonstrated by the results reported herein, the epidemic will in time, magnify cultivar differences.

This method would not be applicable to early generation segregating material, nor for the large numbers of advanced lines common with active breeding programs.

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