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Disease progression in common root rot of spring wheat and barley

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Field plots of spring wheat and barley infected with common root rot caused primarily by *Helminthosporium sativum* were examined periodically throughout the growing season. Disease progress curves did not fit the simple interest model of van der Plank. Neither ontogenic changes in susceptibility nor environmental effects on rates could be shown. Most of the disease progress curves fitted the compound interest model when tested either by Van der Plank's transformation or by fitting the Weibull probability distribution function. The implication of this analysis is that secondary infection is involved in common root rot disease progression throughout the season.
Additional index words: epidemiology, soil-borne disease, *Cochliobolus sativus*

Les parcelles de blé de printemps et d'orge infectés de piétin commun causé principalement par *Helminthosporium sativum*, furent examinées périodiquement tout au long de la saison de végétation. Les courbes de progression de la maladie ne convenaient pas au modèle à intérêt simple de Van der Plank. Ni les changements ontogéniques de sensibilité, ni les effets de l'environnement sur les taux de changement n'ont pu être démontrés. La plupart des courbes d'évolution de la maladie étaient adaptées au modèle à intérêt composé, lorsque testées soit par la transformation de Van der Plank, soit par adaptation à la fonction de distribution des probabilités de Weibull. L'implication de cette analyse vient du fait qu'une infection secondaire prend place en même temps que progresse le piétin commun au cours de la saison.

Common root rot of spring wheat and barley, caused principally by *Helminthosporium sativum* P.K. & B. (= *Drechslera sorokiniana* (Sacc. ex Sorokin) Ito; *Cochliobolus sativus* (Ito and Kurib) Drechsl. ex Dastur) is a widespread, important disease on the northern great plains (6,8,11). The pathogen can infect any of the below-ground parts of the plant, roots, crown, or stem. Infections occur as discrete lesions, although these may enlarge or coalesce (3,12).

There is a correlation between root rot lesions on different underground plant parts (17). Lesions on the subcrown internode have often been used as an index of the disease (6,20) as this part is quite central and is easily evaluated. A standard system for rating it has been published with photographs showing the disease severity classes (6).

In their epidemiological studies on Manitou wheat, Verma et al. (20) assumed that common root rot was a simple interest disease. They took samples only three to five times during the season. In preliminary studies, where 10 to 12 sampling dates were used, we found that the rates of disease development were different early and late in the season and that the disease probably was not of the pure simple interest type (13).

In foliar disease the processes of infection, colonization, sporulation, etc. can be observed directly on the same plant at several times, but observation of root rot under natural conditions requires destructive sampling. To follow disease over time, therefore, requires indirect techniques and subsequent inference about the biological processes actually occurring. We must make use of some differential property of the types of disease development that can be determined by destructive sampling methods. We may then draw biological inferences from these observable effects in much the same way as chemists use product rate curves to infer reaction mechanisms (5).

Simple interest transformed disease progress curves may be nonlinear for several reasons. Nonlinearity may be due to some change in the environment such as moisture or temperature (10). It may also result from changes in host susceptibility associated with ontogeny or stage of development (14). Nonlinearity may also be due to the disease being not of the simple interest type at all (4).

One important factor in disease development is whether secondary infection occurs, which by definition is whether disease progression is of the simple interest (SI) or compound interest (CI) type (19). A simple interest disease is one in which there is no secondary inoculum and no secondary cycle of infection. The disease progress curve of a simple interest disease is hyperbolic and gives a straight line when transformed by the function $\text{Log}_e\ 1/1\text{-}x$ (19). A compound interest disease is one having secondary inoculum and secondary infections. Its disease progress curve is a logistic and is transformed to a straight line by the function $\text{Log}_e\ x/1\text{-}x$ (19).

It has been observed by Jowett et al. that many disease progress curves involving secondary spread fail to exactly fit the logistic growth model and they have proposed other growth models which may fit observed epidemics better (4). However, even when secondary-spread disease curves fit a growth model other than the logistic, there is still a much better fit to the logistic than to a simple interest model. If one

is interested only in addressing the question of whether secondary infections are involved in disease development, then one can usefully limit comparisons to the two models of Van der Plank (19). An interesting property of these transformations is that if both are applied to the same disease progress data and then plotted, one obtains two lines - one straight and the other curved. Which transformation function gives the straight line and which the curve depends on whether the disease is simple interest or compound interest. The "correct" transformation for that data type will be closest to the straight line. This leads logically to an analysis wherein one performs both transformations and then determines which one is straight and which curved. This type of analysis was used by Rowe and Powelson in studies of cercosporella footrot (10).

Another type of analysis which also allows resolution of patterns of disease progression has been proposed by Pennypacker et al. (7). This analysis makes use of the Weibull probability distribution. This distribution can assume a wide variety of shapes and has the simple interest model and the compound interest model as special cases. When the Weibull shape parameter 'c' equals 1.0, the distribution fits the simple interest curve; when 'c' equals 3.6, it fits the compound interest curve (7). They propose fitting observed disease progress data to the Weibull by readily available computer methods and using the estimated value of the shape parameter 'c' as an indicator of the relative importance of secondary spread. One advantage of this analysis is quantification of intermediate forms which may have some secondary development but less (or more) than a true logistic or compound interest disease (7).

Some characteristics of common root rot related to disease development have been reported. Chinn (1) showed that *H. sativum* sporulates freely on infected residues under field conditions. Tinline (16) showed that multiple infections of individuals were common and that new infections occurred throughout the growing period. Verma et al. (20) reported similar findings. There is a poor correlation between seedling disease and adult plant disease with common root rot (2).

Materials and methods

Field preparation and planting. All the experiments reported herein were done in large field plots at Fargo, ND. The soil is a Fargo silty clay loam. Each year the fields were fall plowed and left rough over winter. Spring land preparation was limited to smoothing with a drag harrow just prior to planting. All plots were planted with a commercial-type seed drill of 1.83 m width. Drill row spacing was 15 cm and depth of seeding was ca.5 cm.

Experimental design. There were three experiments, two in 1978 and one in 1979. One of the 1978 experiments (1978A) was done in a plot 30 x 30 m which had been planted to wheat for many years. This plot was planted with Marquis hard red spring wheat on May 16, 1978. Disease evaluations were made on each of 12 sampling dates from 23 to 97 days after planting and stages of plant development from the 3-leaf to maturity. On each sampling date, 24 plants were removed from each of eight locations in this plot and severity of common root rot evaluated on individual plants using the method of Ledingham and Sallans (6), described below.

The other two experiments, one in 1978 and the other in 1979 were of a different design. In each of these, drill strips 1.83 x 100 m were planted of each of several wheat or barley cultivars as listed in Table 1. In the 1978 experiment the strips were planted on May 16 and the first disease evaluation was made when plants were at the 3-5 leaf stage, 24 days after planting. Disease ratings were made at 11 times (10 times for the one barley cultivar) during the season at approximately weekly intervals. The last sampling date was at plant maturity, 97 days after planting. In 1979, strips were planted on May 24. There were seven sampling dates at about 10-day intervals. The first samples were taken when plants were in the tillering stage, 37 days after planting, the final when plants were mature, 98 days after planting. In both of these experiments, the 100-m-long drill strip of each cultivar was divided into four blocks. In each block on each date, 24 to 36 plants were individually rated for disease (15).

Table 1. Relation of simple interest inflection points to time after planting and plant stage

Crop	Inflection point* of simple interest transformed progression (Days)	(Stage)
Marquis 78A	62	late anthesis
Conquest 78	55	milk
Manitou	63	late anthesis
S680 78	80	mid dough
Thatcher 78	56	anthesis
Marquis 78	63	late anthesis
Marquis 79	65	heading
Chris 79	84	mid dough
Waldron 79	56	early anthesis
Thatcher 79	75	soft dough
Conquest 79	80	hard dough-mature
Dickson 79	64	soft dough
Beacon 79	66	soft dough

*Inflection point determined as intersection of straight lines fitted to early and late parts of simple interest transformed disease progression.

Disease evaluation. Disease was measured as the proportion of the subcrown internode tissue covered by lesions. Every plant was individually rated on the four point scale of Ledingham et al. (6). This system was chosen because it has been widely used and found reliable in the spring grain areas of the northern great plains (6,8). To assist in obtaining uniform field disease ratings, the colored pictures of the rating categories illustrated in Ledingham et al. (6), mounted on cardstock and laminated in clear plastic, were used as field guides. Since more than one person was needed to make the disease ratings, a person rated a uniform number of blocks of all cultivars. Differences in rating between persons, therefore, was confounded with block variation rather than with differences among cultivars or over time.

The disease categories illustrated by Ledingham et al. (6) are presented by the authors as 0, 20, 50, and 100% disease, respectively. Our observation were that few plants were completely free of tiny lesions when examined critically. We therefore assigned values of 1.0% disease and 99.% disease to the first and fourth categories respectively.

Data analysis. Disease progression data were first transformed by the simple interest transformation of Van der Plank (19). Changes in environment (10) or ontogenic host susceptibility (14) could produce nonlinear simple interest curves, often with sharp or simultaneous changes in slope (10). For convenience we defined the inflection point of the disease progress curve as the point where straight lines fitted to the early and late points of the curve intersected. The inflection points of the nonlinear, simple-interest-transformed progressions were then compared to time after planting which would indicate involvement of some environmental phenomenon, and to stage of plant development which would suggest ontogenic change in susceptibility.

Two other analyses were performed on the disease progression data to see if they better fit the compound interest model than the simple interest one. The inference from a best fit to the former model would be that secondary spread was occurring, even though it had never been observed directly.

One of these analyses involved calculating both simple interest (SI) and compound interest (CI) transformations on the data and then determining which was a better straight line fit. Calculations were done on the computer using the SAS statistical package (SAS Users Guide, 1979 edition, SAS Institute, Raleigh, NC, USA). It was also useful to compare the effect of the 'wrong' transformation, that is, use of the simple interest transformation on compound interest data and vice versa. These 'wrong transformation' lines are curved in the form of $Y = t^e$ in the case of simple interest transformation of compound interest data and in the form of $Y = t^{-e}$ in the case of compound interest transformation of simple interest data. We compared the SI and CI transformed data set to curves of these forms.

Multiple regression was used to determine which of the two possible shapes, straight line or curve, better fit the transformed data. The values of R^2 for goodness of fit of these regression functions to the transformed data give an indicaion of the better choice between the two models. The statistical test used for comparing these R^2 values is to compare the residual mean squares by the standard F-test for homogeneity of variance. Moreover, the analyses of the two transformations on a single progression reinforce each other, since a straight line best fit to one transformation should give a curved best fit to the other transformation if the response is really due to the difference of simple vs. compound interest form.

The Weibull distribution was fitted to the observed disease levels using two different computer methods. One program, provided by L.V. Madden (7), was written in FORTRAN specifically for fitting the Weibull distribution. The other method used a nonlinear regression procedure from the SAS statistical package.

Results

The actual disease data for one cultivar (Marquis 1978) is shown in Figure 1A. This is typical of all the disease progressions analyzed herein. To see if some environmental factor could have caused nonlinearity in the disease progress curve, linear regressions were fitted to the early and to the late parts of the simple interest (SI) transformed progressions (Figure 1B) The intersection of the two linear regression lines was taken as representing the point of inflection of the progression. For each SI transformed disease progression, the intersection point was compared to time after planting (Table 1). Inflection occurred anywhere from 56 to 84 days after planting. This indicated that inflection does not occur simultaneously in all cultivars as would be the case if changing environment were the cause. The inflection point of each cultivar's disease progression was compared to its stage of growth. The inflection of the SI transformed curves occurred as early as anthesis and as late as near-maturity, depending on cultivar.

Figure 2A shows both the SI and CI transformations applied to the data set of Figure 1A. To simplify visual comparison of the shapes, the two trans-

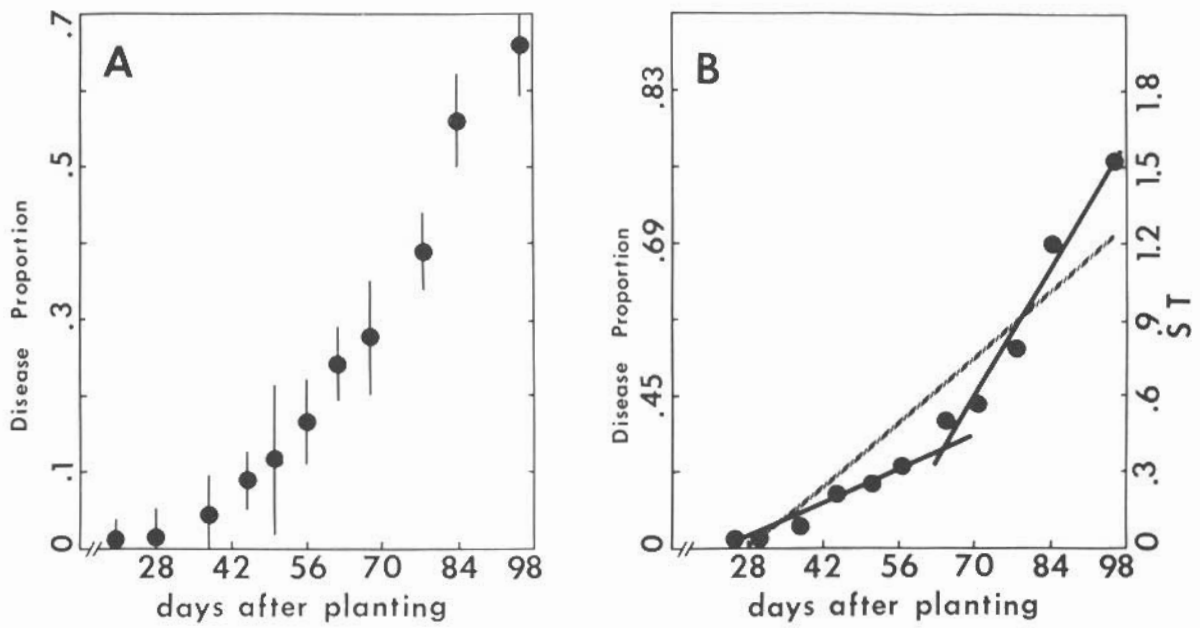

Figure 1. Common root rot disease progression in Marquis wheat in field experiment 1978.
A) Disease proportion. Proportion of subcrown internode tissue lesioned by *H. sativum*. Vertical bars denote standard deviation of means.
B) Simple interest transformation (ST) ($\log_e$ 1/1-x) of A. Solid lines represent linear regressions fitted to early and late portions of progression. Slashed line represents a linear regression to the entire progression.

formed lines are plotted on the same figure. The indicated regression lines are of the linear form $y = at + b$ for the CI points and of exponential form $y = ct^e + d$ for SI points. Transformed data for three of the other progressions are shown in Figure 2B-D. In each case the linear regression line is fitted to the CI points and the exponential regression to the SI points.

For each of the 13 disease progressions, both CI and SI transformations were made. Then each transformed progression was fitted to a linear and a curved regression function. The decision as to whether the data points were better fit by the linear or curved line was made by inspection of the R^2 (goodness of fit) statistic for each regression, as listed in Table 2. A higher R^2 for the linear regression to the CI transformed data and/or a higher R^2 for the curved regression to the SI transformed data indicate that the CI is the appropriate model for disease progression. All of the 1978 and about half of the 1979 progressions fit the CI model. None fit the SI model, although several in 1979 lacked sufficient precision to discriminate between models.

The two computer programs for fitting the Weibull distribution gave approximately the same results, except in two cases where the Fortran program failed to give a result for unknown reasons. The two were S-680, 1978 and Chris 1979. The Weibull 'c' values for each progression are given in Table 2. While the cultivars differed in the 'c' values, the mean 'c' value for 1978 was 5.33 and was 3.51 for 1979, well within the range indicated for compound interest type disease involving secondary spread (7).

Discussion

The results of this work support a compound interest type of progression for common root rot. The comparison of inflection points of the disease progressions (Table 1) failed to show any simultaneous change. Since the cultivars were grown side by side, it is unlikely that soil environmental factors could be responsible for the changes in shape of the SI progressions. Since the amount of tissue (subcrown internode) being evaluated remains relatively constant for the period studied, this disease progress pattern cannot be attributed to growth, as might be the case if roots were measured. The most likely remaining explanation is that the disease is of the compound interest type, although perhaps not of the exact logistic model.

This finding of compound interest disease development in *Helminthosporium* root rot is a departure from the way in which this disease has been considered in the past (20). The implication of disease progression fitting the compound interest

model is that secondary inoculum is playing a role in the disease.

Chinn (1) showed that *H. sativum* spore populations in soil increased dramatically near the end of the cropping period. If such sporulation occurred over a larger part of the season, even if at lower rates, there would be a ready source of inoculum for secondary infections. Using morphologically distinguishable isolates, Tinline showed that lesions on the subcrown internode may be the result of multiple infections on the same plant (16). It seems unlikely that propagules could spread from plant to plant to any extent during the growing season under field conditions and, therefore, secondary infection would be mostly auto-infection (9). If this is so, there are important consequences for the usefulness of different types of resistance in breeding (9).

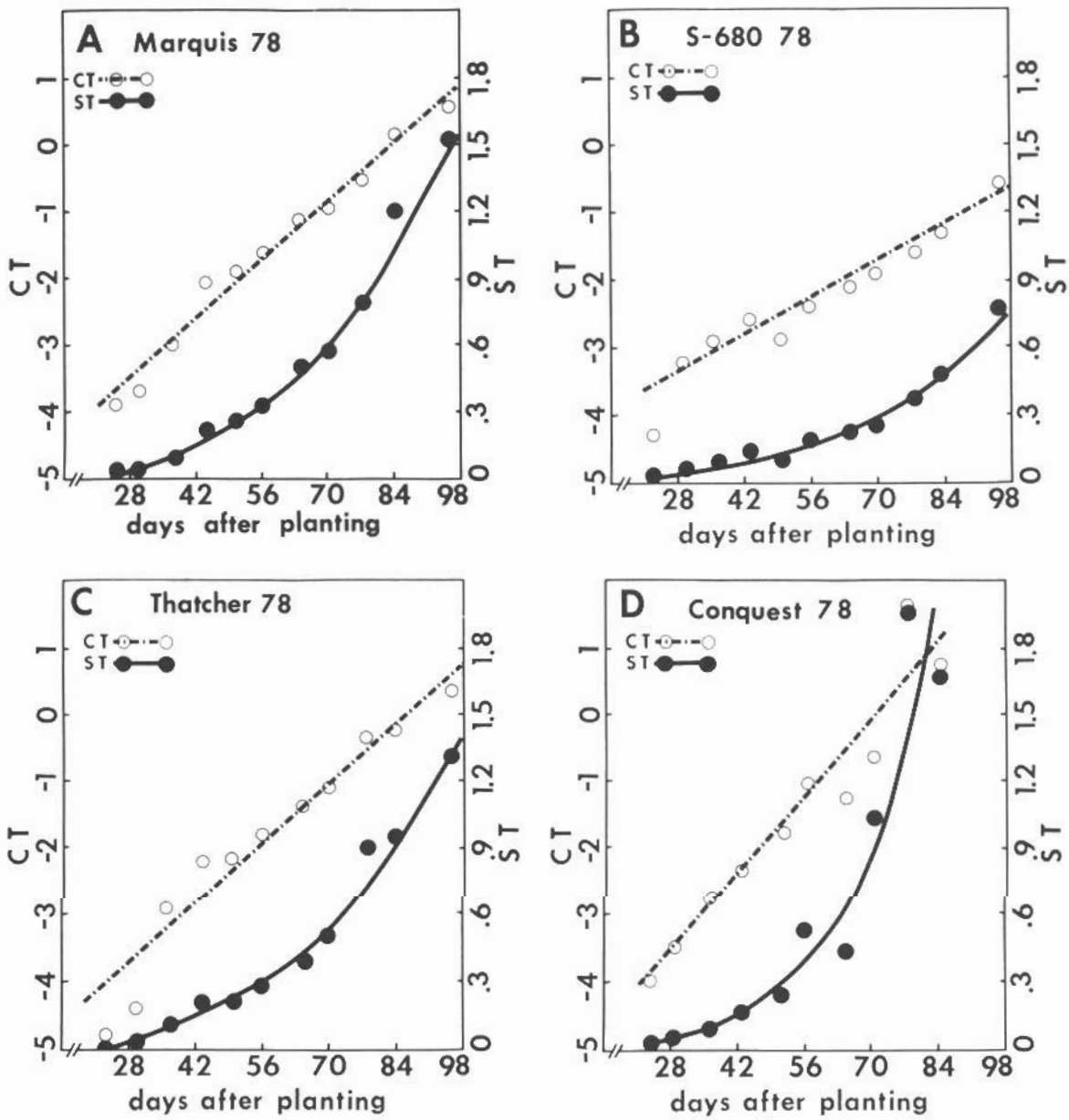

Figure 2. Transformed disease progression for common root rot of wheat and barley from 1978 field experiment.
A) Marquis wheat, **B)** S-680 wheat, **C)** Thatcher wheat, **D)** Conquest barley. ST = simple interest transformation, $y = \ln(1/1-x)$; CT = compound interest transformation, $y = \ln(x/1-x)$. Marquis wheat here (A) is same data as in Figure 1. A & B. S-680 is an experimental line supplied by Dr. R.D. Tinline, Saskatoon, Sask.

As Kranz points out, within a given disease only a limited amount of variation in the disease progression can occur, but within this allowable range the form of the disease progress curve may vary in response to environment, among cultivars, or by cultural practices (5). Examination of the Weibull 'c' values for the different cultivars and years (Table 2) shows that considerable variation in the form of the disease progress curves is occurring. There is some indication that the cultivars may respond differentially to environment. The cultivars Conquest, Marquis, and Thatcher were present in both 1978 and 1979; the 'c' values for Marquis and Conquest were higher in 1978 than in 1979, while it was lower in 1978 for Thatcher. Environmental interactions affecting the shape of the disease progress curve might account for the difference between the results of Verma (20) in Saskatchewan and those in North Dakota. Differences in form of disease progress curves might also go far to explain the variable relationship between disease level and yield loss reported by Tinline (18).

Comparisons of cultivars for root rot susceptibility have often been based on disease measurements taken at a single time, frequently after anthesis, in field studies. Verma et al. suggested that comparison of disease progression between cultivars might yield more useful findings (20); however comparisons of disease which have different forms of progress curves could lead to erroneous conclusions (5). The degree of variation in form of disease progressions of the different cultivars indicated by the 'c' values raises this exact question for comparisons among wheat or barley cultivars.

A major factor affecting the shape of the disease progress curve may be the delay before onset of the exponential phase of disease development (5,9). If such a characteristic were a heritable response to infection by *H. sativum*, it might be extremely useful in producing varieties with field resistance. Such a character would not be detected either by seedling screening or by field ratings at maturity.

These results carry importance for breeding for root rot resistance, and for the methodology of loss determination.

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Table 2. Regression analysis of disease progressions

	Goodness of fit (R^2) of regressions for disease progress transformations				
	CI Transformation $y = \ln(x/1-x)$		SI Transformation $Y = \ln(1/1-x)$		Weibull
Crop	linear $y = at$	curved $y = at^{-c}$	Linear $y = at$	curved $y = at^{c}$	'c' value§
Marquis 78A	0.96**	0.70	0.89	0.96*	2.96
Conquest 78	0.96**	0.68	0.75	0.87	9.36
Manitou 78	0.93*	0.76	0.87	0.95+	2.51
S-680 78	0.93*	0.72	0.79	0.95*	9.37
Thatcher 78	0.93+	0.82	0.88	0.98**	3.00
Marquis 78	0.97**	0.72	0.86	0.97*	4.77
Marquis 79	0.89	0.87	0.93	0.96	4.88
Chris 79	0.87	0.63	0.77	0.89	3.91
Waldron 79	0.96*	0.79	0.94	0.94	1.85
Thatcher 79	0.98*	0.84	0.90	0.98	6.69
Conquest 79	0.94	0.89	0.91	0.99	4.79
Dickson 79	0.94	0.91	0.95	0.98	2.00
Beacon 79	0.97+	0.89	0.93	0.98+	2.49

†Conquest, Beacon, Dickson are 6-row spring barleys. All other cultivars are hard red spring (hexaploid) wheats. S-680 is an experimental line supplied by Dr. R.D. Tinline, Saskatoon, Sask.

*Significant differences between linear and curved regressions indicated by: + = $p < 0.15$, * = $p < 0.05$, ** = $p < 0.01$.

§Weibull shape parameter 'c' fitted to non-transformed disease proportion progression. For a pure, simple interest disease c = 1.0; for a pure, compound interest c = 3.6.

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