A Stochastic Model for Anthracnose Development in *Stylosanthes scabra*

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**ABSTRACT**


Spatial and temporal progress of anthracnose caused by *Colletotrichum gloeosporioides* in quantitatively resistant and susceptible tropical pasture legumes *Stylosanthes scabra* was studied in a field experiment at the Southedge Research Station, Queensland, Australia. An anthracnose epidemic was initiated by inoculating a group of susceptible seedlings planted at the center of each plot. The speed with which the disease spread from the infection focus to plants within a plot depended on their proximity to the focus and level of resistance of the accessions. A stochastic Markov chain model, in which the probability of a plant developing a given disease severity level depends on its current disease state and that of its neighbors, was used to describe disease progress. The probability of a disease-free plant with disease-free neighbors developing anthracnose within a 1-wk period was estimated to be 52% for the susceptible cultivar Fitzroy, 2.6% for the resistant accession 93116, and 6.5-23% for accessions with quantitative resistance. In all accessions, the probability of a plant becoming diseased or progressing to a higher state of severity increased with the severity level of its nearest neighbors. An accession effect parameter served as an estimate of the relative susceptibility of the accessions. Accession ranking based on this parameter was highly correlated with that based on the area under the disease progress curve. The model effectively described both spatial and temporal aspects of anthracnose progress.

Additional keywords: logistic regression, nearest-neighbor analysis, ordinal regression, probability model.

**Anthracnose**, caused by *Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc. in Penz., is the most destructive disease affecting species of the tropical pasture legume *Stylosanthes*. It was responsible for the devastation of 500,000 ha of *S. humilis* Kunth (Townsville stylo) pastures in Australia during the mid-1970s. Although this highly susceptible species has since been abandoned, anthracnose continues to cause severe loss in other species of commercial value. Extensive pathogenic specialization has been identified with strains of *C. gloeosporioides* that cause anthracnose of *Stylosanthes* spp., and in Australia, new strains of the pathogen have arisen following the introduction of host cultivars and accessions (13,19).

As a management approach, accessions of *S. hamata* (L.) Taub. and *S. scabra* Vogel are being evaluated for quantitative resistance for effective protection against all or most of the known pathogenic variants. Several accessions of *S. scabra* have shown little or no race specificity in greenhouse screenings (9). These accessions have maintained their resistance under different levels of inoculum and at different day-night temperatures (8).

To test the effectiveness of quantitative resistance under field conditions, selected accessions of *S. scabra* have been grown for two successive summer seasons. Preliminary analysis of anthracnose progress curves, using growth curves and piecewise linear models, demonstrated that accessions consistently expressed the same relative degree of resistance (10). The disease in this field experiment was initiated from an infection focus at the center of each plot, and this resulted in, for a splash-dispersed pathogen, steep disease gradients in many accessions. While useful for initial analysis, all the preliminary models considered were deterministic, in the sense that average disease progress was assumed to follow a curve of a specified shape. Although these models could have been extended to incorporate spatial as well as temporal distribution of disease, as in Reynolds and Madden (24), even the most elaborate deterministic models do not take into account the discreteness of the disease-rating class most plant pathologists use for disease assessment, or satisfactorily model the lack of homogeneity of variance and the nonstationarity of the response. An alternative approach is available through stochastic models, which explicitly recognize the above features and are, in general, more realistic (18) in their representation of natural variability, especially when dealing with small population sizes. In the related and considerably more developed field of medical epidemiology, emphasis has shifted from the early deterministic models to stochastic models (4). Relatively few stochastic models of plant disease have been published so far (7,27,28,31), although several authors have advocated their use (15,26).

Stochastic models, in which the current disease severity of a given plant is dependent only on its severity at the previous time and on other factors such as the disease severity of its neighbors, and not on disease severities in the more distant past or other history, can be viewed as Markov chains (14). The purpose of this paper is to investigate the usefulness of a discrete Markov chain model in characterizing the quantitative resistance to anthracnose in *S. scabra*.

**MATERIALS AND METHODS**

Host accessions and field plot establishment. Six accessions of *S. scabra*, 55803, 55800, 92873, 92918, 93055, and 93099, were selected from the Commonwealth Scientific and Industrial Research Organisation (CSIRO) collection because they showed low levels of anthracnose severity with three races of *C. gloeosporioides* in a previous greenhouse screening (9). *S. scabra* 'Fitzroy' and accession 93116 were used as susceptible and resistant controls, respectively. *S. hamata* 'Verano' was planted as an additional control. Seedlings, 6 wk old, raised in a 3:2:1 mixture of loam, sand, and peat in 4-×-4-cm "rite gro" pots (Cheetham Plastics Ltd., Brisbane, Australia) in a greenhouse, were transplanted into 4.5-×-4.5-m field plots on 16 December 1987 at the Southedge Research Station (17°0' S, 145°20' E) of the Queensland Department of Primary Industries. Each plot con-
tained 100 plants at between- and within-row spacings of 50 cm. There were three replicate plots for each accession/cultivar. Plots were separated from each other on all sides by a 5-m fallow to reduce interplot interference. Further details on experimental design and other aspects of methodology have been reported earlier (10).

Plot infestation and disease assessment. An anthracnose epidemic was initiated by inoculating three Fitzroy plants, raised in the center of each plot, with three different isolates of C. gloeosporioides representing all three S. scabra races as per methods described earlier (10). Inoculated plants were then covered with a reflective plastic bag for about 20 hours to provide the necessary leaf wetness (11).

Starting 4 wk after inoculation, all 20 plants along the two diagonals in each plot were assessed on eight occasions, and intervals between observations were 7, 7, 7, 7, 14, 21, and 19 days, respectively. The double diagonal configuration meant that the assessed plants were at approximate radial distances of 0.35, 1.06, 1.77, 2.47, and 3.18 m from the infection focus, and that four plants were available per plot for each distance. The percentage of leaf area diseased was estimated from the top 10- to 15-cm length of a randomly selected branch for each plant, using a 10-point rating scale (8) (0 = no visible symptoms, 1 = 1-3%, 2 = 4-6%, 3 = 7-12%, 4 = 13-25%, 5 = 26-50%, 6 = 51-75%, 7 = 76-87%, 8 = 88-94, and 9 = 95-100%). A total of 540 plants (20 plants in each of three plots for each of nine cultivars) were assessed, and each of those returned a series of eight assessments.

Model description. Two features of the data that need to be captured in a statistical model are the ordered categorical scale on which the disease assessments are recorded and the substantial interdependence of assessments over both time and space. To model responses on the ordered scale, the ordinal logistic regression model (22, 32) was used. Let \( \pi_j, j = 1, \ldots, k \) be the probabilities of a plant being in each of the \( k \) disease assessment categories (\( k = 10 \) for the 10-point scale). The ordinal logistic model, also called the cumulative odds or proportional odds model, assumes that

\[
\logit(\gamma_j) = \ln \frac{\pi_j}{1 - \pi_j} = \theta_j + \beta^T x, \quad j = 1, \ldots, k - 1
\]

where \( \gamma_j = \pi_j + \pi_{j+1} + \ldots + \pi_k \) is a vector of explanatory variables, and \( \theta_j \) and \( \beta \) are unknown parameters. The parameters \( \theta_j \) represent the baseline logits, and \( \beta \) represents the regression parameters through which the effect of the explanatory variables is mediated. The \( \gamma_j \) are the cumulative probabilities of exceeding a given disease level. In particular, \( \gamma_k \) is the probability of being diseased, and on the 10-point scale \( \gamma_9 \) is the probability of 95-100% disease.

The use of cumulative probabilities ensures that \( \beta \) is consistently defined, even if the ordered categories are regrouped—an important property, since the divisions between the assessment scale classes are essentially arbitrary. Ordinal logistic regression is a direct generalization of binary logistic regression. It can be viewed as a grouped-continuous model, the ordinal response being formed by taking contiguous intervals of an unobserved underlying continuous response variable, with the \( \theta_j \) as cut points. Armstrong and Sloan (2) provide an introduction to this model and a comparison with others.

The interdependence of assessments is modeled by including the disease assessments of the plant and its neighbors at the previous observation time among the explanatory variables. Conditional upon this information about the prior state is the assumption that the disease develops independently on each of the plants between the observation and the next. The resulting model is a conditional generalized linear model as defined elsewhere (20, 33). Since the probabilities depend only on observations at the previous time and not on events in the more distant past, the model may be also characterized as a first order Markov chain (14), and use is made of this characterization in the analysis.

Analysis. Let \( \gamma_{it} \) be an integer between 1 and \( k \) representing the disease assessment of the \( i \)th plant at the \( t \)th observation time, and let \( \gamma_{itn} \) be the cumulative probability of exceeding level \( c \). Let \( b = \gamma_{it+1} \) be the disease level of the plant at the previous time. The regression model used in this study had the form

\[
\logit(\gamma_{itn}) = \theta + \mu_b + \alpha_i + \beta f_i + \beta_2 f_2 + \beta_3 f_3 + \beta_4 f_4 + \beta_5 f_5 + \beta_6 f_6
\]

where \( f_1 \) is a measure of the average disease level of the plant’s nearest neighbors at the previous time, \( f_2 \) is a similar measure for the more distant neighbors, and \( d \) is the number of days between observation times \( t - 1 \) and \( t \). In this model, the \( \mu_b \) represent effects for the previous disease level and, like the \( \theta \), are not of primary interest. Of central interest are the \( \alpha_i \), which represent the relative susceptibility of each cultivar/accession. The remaining covariates are continuous. The influence of neighboring plants is approximated by two quadratics, and \( \beta \) is included to allow for the fact that larger changes can be expected over longer periods.

The average disease level of the neighboring plants is calculated on a logit scale, using the midpoints of percent diseased for the assessment categories. For example, suppose that the two nearest neighbors (0.71 m distant) were in assessment categories 3 and 4 at time 1. The midpoint of percent diseased is 9.5% for assessment class 3 and 19% for class 4, so \( f_1 = \logit(0.095) + \logit(0.19) \). Similarly, \( f_2 \) is calculated from the more distant (1.42 m distant) neighbors. The logit transforms used offset 0.01, as \( \logit(p) = \ln[p/(p + 0.01)] \), to avoid taking the logarithm of 0; Cox and Snell (12) provide a justification of this type of offset. The use of logits here, rather than raw percentages or category labels, improved the model fit and had the intuitive appeal of putting the covariate disease levels on the same scale as the continuous response variable underlying the grouped-continuous interpretation of the ordinal regression model.

To ensure that a sufficient number of observations were available in all assessment scale categories, the 10-point scale was collapsed into four broad classes when categorizing the response \( \gamma_{itn} \). The categories were disease-free, moderate (ratings 1–3), severe (ratings 4–6), and very severe (ratings 7–9). The covariates \( f_1 \) and \( f_2 \), however, were calculated from the 10-point scale.

Some plants died during the experiment of causes other than anthracnose and therefore had no further observations recorded. If neighboring plants were missing, the closest available neighbors were used. All plants were supposed to be disease-free at time 0. Plants at the outer edge of each plot with no physical outer neighbors were assumed to have disease-free outer neighbors for the purpose of the analysis. The inoculated plants at the center of the plot were not assessed; they were assumed to have a constant effective disease level throughout the experiment, and this level was estimated from the data.

Most published discussion of ordinal regression (1, 22), an exception being Ashby, Pocock, and Shaper (3), has been in the context of data sets that are small or can be summarized in contingency tables with a small number of levels. The data considered here contains several continuous covariates and 4,061 observations, and it is essential that the calculations be programmed in regression rather than contingency table form. The SAS program PROC CATMOD (29), PLUM (21), and GLIM (23), for example, are unsuitable for this application. Maximum likelihood estimation of the ordinal logistic model is available in the SAS supplemental library program PROC LOGIST (16), or in the S (5) program LOGIST available from the Statlib database (17). For this study a Matlab (Mathworks) program was developed on a Sun 3/60 workstation. The program is available from the Netlib database (30).

**RESULTS**

**Graphical data summary.** The central Fitzroy plants in all plots developed symptoms within a week of inoculation with the three races. Because of very dry weather conditions, there was no further spread or development of anthracnose until 4 wk after inoculation, when plants adjacent to the central Fitzroy showed symptoms. Disease assessment was started at that time. In one Fitzroy plot,
a few plants became infected with inoculum coming from a nearby field before the central plants were inoculated. Anthracnose in all other plots developed from the central infection focus. The speed with which plants became infected depended on their proximity to the infection focus and, more importantly, on the resistance level of the accessions. Disease severity on Fitzroy plants increased rapidly from an early part of the season, irrespective of their radial distance from the source (Fig. 1). Anthracnose was detected on plants at all distances from the focus within 5 wk of inoculation. In other accessions, spread of anthracnose to the outside plants was delayed by several weeks, and in 93116 and 93099 the disease was never detected on the perimeter plants in some plots.

In the susceptible Fitzroy and 55803, considerable disease developed on plants located more than 3 m from the focus. In 92873, 92918, and Verano, some disease developed at this distance, whereas in the resistant 93116 very little disease was detected, even on plants adjacent to the focus. In accessions 55860, 93055, and 93099, increases in anthracnose severity were mainly restricted to the plants closest to the focus. By using the area under the disease progress curves (AUDPC) averaged over all positions, the accessions can be sorted in an increasing order of resistance as Fitzroy, 55803, 92873, Verano, 92918, 55860, 93055, 93099, and 93116.

**Ordinal regression analysis.** Parameter estimates and standard errors for the ordinal logistic regression model are given in Table 1. Positive parameters can be interpreted as increasing the probability of exceeding any given disease level. The parameter $\mu_b$, which measures the effect of the prior disease level, increases monotonically, with $b$ reflecting the intuitive property of higher prior disease levels being associated with higher current disease levels.

The accession effect parameter $\alpha_6$, which is given relative to Fitzroy, orders the cultivars and accessions in increasing order of disease resistance as Fitzroy, 55803, 92873, 92918, Verano, 93099, 55860, 93055, and 93116. The Spearman's rank correlation coefficient ($r_s = 0.93$) is highly significant ($P < 0.001$) for this and the overall AUDPC-based ranking of accessions, reinforcing the usefulness of this parameter as a measure of relative susceptibility of the accessions. Compared with AUDPC, the accession parameter has the advantage of taking into account more features of the disease epidemic and in having standard errors attached.

The effect of neighboring plants on disease level is difficult to read directly from the parameter estimates but can be depicted graphically, as in Figure 2. The influence of the neighbors is substantial but reaches a limit as their disease level increases. The influence of the more distant neighbors is between one fourth.
and one third of that of the nearest neighbors. This is consistent with the hypothesis that influence is proportional to squared
distance, since the more distant neighbors are exactly twice the
distance from the plant influenced as are the nearest neighbors.
The number of days \( d \) since last observation was also positively
associated with disease development. Although not given in Table
1, the effective assessment scale category of the inoculated plants
in the middle of each plot was estimated to be 5 on the 10-
point scale.

**Fitted probabilities.** The model gives probabilities of being in
each disease category for any plant as

\[
\pi_j = \frac{1 - \gamma_j}{\sum_{j=2}^{k} \gamma_j}, \quad j = 2, \ldots, k
\]

where \( \gamma_j \) are the cumulative probabilities. Estimated probabilities
for a set of four selected accessions of \( S. scabra \) and the susceptible
and resistant controls at four different severity levels of the
neighbors are given in Table 2. For simplicity in presentation, the
period since last observation is assumed to be 1 wk, and all four
neighboring plants are assumed to have been equally diseased.
The probability of a plant developing anthracnose, given that it
and its neighbors were disease-free at the previous time, was
52\% for the susceptible Fitzroy, 28\% for the resistant accession
93116, and from 6.5 to 23\% for accessions 55860, 93055, and
93099 with quantitative resistance. This means that plants were
being infected by inoculum coming from sources beyond nearest
neighbors, although sampling along the two diagonals meant that
only two of the four nearest neighbors were assessed. In all access-
ions, the probability of a plant remaining disease-free decreased
with increasing severity levels of its neighbors. However, the prob-
ability of further increase in the disease level was reduced for
plants with an already high level of disease. This is mainly because
at high severities, both the number of disease-free leaves remaining
to be infected and, with leaf loss, the amount of secondary inocu-
lation available for fresh infections are reduced.

Although probabilities as given in Table 2 give a complete
description of the distribution of disease, a less detailed and more
compact summary of disease prevalence is also desirable. In prob-
ability theory, time-homogeneous Markov chains have the prop-
erty of the distribution of states approaching a stationary distribu-
tion with time, irrespective of the initial values. The standard
method of summarizing a Markov chain is to calculate this sta-
nary distribution. Let \( \pi_j \) be the probability that a plant will
be in disease category \( j \), given that it was in category \( i \) at
the previous time. In general, this probability depends on the neigh-
bor-
of disease progress curves of widely different shapes. Although additional parameters may be included to accommodate this, often such parameters can not be easily interpreted as having a biological meaning, and if more than one model is used, parameters are not directly comparable between models. The Markov chain model makes no prior assumptions about the shape of the disease progress curves and is therefore applicable to data where a range of host cultivars and lines with varying levels of resistance have been included. This makes the model potentially useful to plant breeders and plant pathologists evaluating a range of host genotypes.

The model has several useful attributes. It provides an alternative to the existing methods (24) to simultaneously model temporal and spatial progress of disease. It gives interpretable results and is satisfying from a statistical point of view, since it explicitly handles the discreteness of the responses and the observation times. The estimated effect of first- and second-order neighbors is in agreement with the observation that infection gradients of splash-dispersed pathogens is typically steep and is consistent with the theory that the influence of neighbors for such pathogens should be inversely proportional to their squared distance (24). The estimated regression coefficients for the accessions can be used as a measure of relative susceptibility of the accessions. Usefulness of this accession effect parameter is apparent from the significant rank correlation with AUDPC-based ranking of accessions. The value of this parameter as a predictor of anthracnose development on the accessions needs to be established from long-term field studies.

For accessions other than Fitzroy, plants adjacent to the central focus are subjected to alioinfection (infection on a genotype resulting from propagules produced on a different genotype), whereas plants further removed from the focus are mainly subjected to autoinfection (infection on a genotype resulting from propagules produced on that same genotype) (25). The only defense against autoinfection is horizontal resistance (effective equally against all variants of a pathogen) (25). The position-wise plots of disease progress in accessions 55860, 93055 and 93099 clearly demonstrate the effectiveness of anthracnose resistance in these accessions.

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</table>

* Diseace ratings: 0 (disease-free); 1-3 (moderate); 4-6 (severe); 7-9 (very severe).
TABLE 3. Theoretical limiting distributions of anthracnose levels calculated from simplified Markov chain transition probabilities

<table>
<thead>
<tr>
<th>Accession or cultivar</th>
<th>Disease-free</th>
<th>Moderate</th>
<th>Severe</th>
<th>Very severe</th>
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<td>55803</td>
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<td>Verano</td>
<td>0.586</td>
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</table>

Whether this resistance is truly horizontal is yet to be determined.

The stochastic model used in our study has obvious limitations. For example, it does not consider the influence of weather on disease development. The overestimation of initial disease levels during the first 4 wk after inoculation, when extremely dry weather conditions arrested disease spread in the field, may be rectified by including weather as a covariate. This may also improve the predictive values of the long-run probabilities given in Table 3. Allowing the severity of Fitzroy plants used as the central inoculum source to increase with time is another possible refinement. The additional complexity, however, is not necessarily warranted for our purpose of judging the suitability of a Markov chain model to analyze the level of resistance in the accessions.

Often observations made on biological systems, such as the development of a plant disease, are characterized by unexplained variability. As opposed to a deterministic model, which implies that factors significantly influencing an event are known and accounted for, a stochastic model allows the inclusion of unexplained variability in the model (28). Our results have demonstrated the ability of a stochastic model to describe both spatial and temporal aspects of the anthracnose epidemic. Markov chains may also be useful in analyzing aspects such as the influence of weather on disease development and the role of nearest neighbors in a genotype mixture. This and other stochastic models (27, 28) offer an alternative approach in the analysis of plant disease epidemics, and they deserve more attention from plant pathologists.

LITERATURE CITED

30. Smyth, G. K. 1992. LOGIST. A Matlab function in Netlib (netlib@research.att.com), a network accessible database. AT&T Bell Laboratory, Murray Hill, NJ.