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# Forecasting the risk of crown rot between successive wheat crops

*David Backhouse*

Centre for Sustainable Farming Systems; postal address School of Environmental Sciences and Natural Resources Management, University of New England, Armidale, NSW 2351, Australia; email: dbackhou@une.edu.au

Short title: Forecasting crown rot in wheat

*Abstract.* Published data from long-term trials at Moree, New South Wales and Billa Billa, Queensland, Australia were analysed to determine the factors that influence the incidence of crown rot, caused by *Fusarium pseudograminearum*, in successive wheat crops and to examine the feasibility of developing a forecasting system for the disease. Polyetic progress of the epidemics could be described by a form of the logistic growth model with a carrying capacity ($K$) approximately 5% higher than the maximum recorded incidence at each site. Infection rate between seasons was positively correlated with yield and in-crop rainfall in the previous season, which were indicators of biomass. Infection rate was negatively correlated with rainfall parameters during the summer fallows, which were indicators of conditions favouring residue decomposition. In-crop rainfall, stored soil moisture and temperature parameters were not significantly correlated with infection rates. Multiple regressions based on incidence in the previous season, summer rainfall and either yield or in-crop rainfall in the previous season accounted for 65-81% of the variation in disease incidence at Moree and 86% of the variation in incidence at Billa Billa. Simplified parameters for use in on-farm forecasting systems were explored. The most useful of these was the square root of the product of incidence and either yield or in-crop rainfall, which gave sufficiently accurate predictions at each site to estimate the qualitative risk of crown rot in the following crop.

*Additional keywords: Fusarium graminearum* Group 1, *Gibberella coronicola*

## Introduction

Crown rot, caused by *Fusarium pseudograminearum*, is a major disease of small-grain cereals in Australia. It infects the stem bases of wheat and other cereals, causing browning of the leaf sheaths and lower internodes. If plants are water stressed during grain filling, heads on infected tillers may ripen prematurely leading to 'whiteheads' that contain shrivelled or no grain. The fungus survives between seasons in infested stubble. Although conidia, and less frequently ascospores of the teleomorph (*Gibberella coronicola*), are produced, these are considered unimportant and most infection is believed to occur from hyphae growing out of pieces of infested residue (Burgess *et al*. 2001).

The disease is thought to be essentially monocyclic, with inoculum present at the start of the season being the main determinant of terminal incidence (Summerell *et al*. 1989; Burgess *et al*. 1993). However, this has never been tested by epidemiological analysis. When disease has been monitored over successive seasons, it increases from a low incidence over 3-4 years to a plateau level that is usually less than 100% (Burgess *et al*. 1993; Wildermuth *et al*. 1997). In subsequent years incidence can fluctuate greatly, or even appear to decline (Summerell *et al*.

1989; Burgess *et al*. 1993; Wildermuth *et al*. 1997). The factors that cause these fluctuations have not been identified, although it has been noted that incidence tends to be low after drought-affected crops, which leave less residue (Swan *et* al. 2000; Burgess *et al*. 2001).

There have been few attempts to define the relationship between incidence (proportion of plants infected) or severity (degree of symptom expression) of crown rot and yield. Klein *et al*. (1991) used a single-tiller technique to show that loss in potential yield of wheat was positively correlated with proportion of plants with whiteheads, proportion of plants with stem browning, and incidence of infection (measured by isolation on selective media) during grain fill. Kierkegaard *et al*. (2004) reported that yield of durum wheat was reduced by 1% for every 1% increase in the proportion of tillers showing stem browning, and that this was strongly correlated with the proportion of plants infected.

Because yield loss is related to disease incidence, it would be useful to be able to predict likely incidence of crown rot before decisions are made about crop options for a season. A predictive system for take-all, caused by *Gaeumannomyces graminis* var. *tritici*, that could be used as a template for a crown rot forecasting system has been described by Roget (2001). In this system, crops are placed into high, medium or low risk categories for take-all based on a soil DNA test, and environmental and management factor inputs are used to estimate likely yield loss and economic effects (Roget 2001). Although there is a lot known about various components of the crown rot pathosystem, it has not yet been brought together in a way that would allow development of a similar model, and there remain gaps in knowledge of quantitative epidemiology of crown rot.

Crown rot is an example of a polyetic disease (Zadoks 1999) in which the epidemic develops over successive years and the incidence of disease in one year is influenced by the incidence in the previous year. Some of the published work on crown rot has failed to take this into account, and comparisons between management treatments have been made in one year without considering the previous history of the disease (Felton *et al*. 1998). Developing a full-scale predictive system for crown rot will require an understanding of the mathematical nature of polyetic changes in disease incidence.

This paper describes an analysis of epidemics of crown rot from two series of long-term field trials. The approaches taken were to attempt to fit simple epidemiological models to disease progress over successive seasons, and to identify environmental or agronomic parameters that were strongly correlated with changes in crown rot incidence between seasons. The ability to predict crown rot incidence based on these parameters was tested, and a simplified subset of parameters suitable for use in forecasting systems was identified.

## Methods

### *Data sources*

Published data were obtained from field trials at Moree, New South Wales, and Billa Billa, Queensland. At each site data were selected where the incidence of infection or of disease symptoms had been measured in the previous crop, and where full stubble retention with zero tillage had been practised. Data used were treatment means, not individual plot data.

The Moree data came from a series of trials on two adjacent areas on a neutral self-mulching grey clay in Paddock U at Livingston Farm. The first of these was a long-term stubble management trial running from 1986-1996. Disease incidence and yield data from this trial for 1986-1991 have been presented as site B in Burgess *et al*. (1993), and for 1994-1996 in Swan *et al*. (2000). Disease incidence, but not yield, for 1992 and 1993 were reported by Burgess *et al.*

(1996) as the continuous wheat treatment in a sorghum rotation experiment. The second trial was established as a comparison of no-till and late stubble burning in 1991, and data for the years 1991-1994 have been reported in Burgess *et al.* (1996). In 1995 the plots were split to give a comparison of 3 wheat varieties, with all plots being converted to zero tillage with stubble retention. Plots that were previously no-tillage were considered to be a high inoculum treatment, and those plots previously burnt as a low inoculum treatment (Swan 1998). Data from these treatments were kept separate in this analysis. Because not all of the disease incidence and yield data from these trials have been published, they are presented in Table 1.

Bread wheat varieties used at Moree were Sunstar (sown 1986-1990), Janz (1991), Miskle (1992, 1994), Sunbri (1993) and Sunstate (1995, 1996). Incidence of infection was estimated by isolation of the fungus from stem bases onto selective media at around the soft dough stage (Zadoks GS 85). Isolations were done from 50 plants from each of 4 or 6 replicate plots. The only agronomic parameter collected consistently that could be used in analyses was grain yield. Rainfall was measured on site. Daily weather observations for Moree, taken approximately 5 km from the trial site, were purchased from the Bureau of Meteorology. Data were obtained for 17 crops at the Moree site for which incidence had been measured in the previous year (Table 1).

Data for Billa Billa came from a series of publications (Radford *et al*. 1992; Thomas *et al*. 1995; Wildermuth *et al*. 1997) describing a long-term tillage and stubble management trial on a red-brown earth. The variety sown in all years was Hartog, a bread wheat. Usable pathological data were obtained for the years 1986-1993, as the incidence of plants with symptoms of stem browning at anthesis (Wildermuth *et al*. 1997). This was assessed on 50 plants from each of 3 replicate plots. Agronomic parameters used in analyses were dry weight of plant tops at anthesis and grain yield. In-crop and fallow rainfall on site, and stored soil moisture at sowing, were obtained from the publications (Radford *et al*. 1992; Thomas *et al.* 1995). Daily weather data were obtained from the Bureau of Meteorology for the nearest official weather station at Goondiwindi, approximately 40 km distant. Data were obtained for 7 crops at Billa Billa. These were generally used to validate relationships developed from the larger data set from Moree. All data required for these validations can be found in the publications, except for rainfall in December-February which was estimated from the records for Goondiwindi (data not shown).

*Epidemiological models of disease progress*

In one of the trials at Moree (Site B of Burgess *et al*. 1993) and at Billa Billa incidence increased over a series of seasons (1986-1989 at Moree, 1986-1990 at Billa Billa) from a low level to the maximum recorded at each site. These years had adequate rainfall for crop growth, and grain yield at both sites was within normal range for each district. It was considered that there were no climatic or other constraints on disease in these years, and that the assumptions required for fitting growth curves to disease progress (Campbell 1998) were met. The usefulness of standard growth curve models for describing the increasing phase of crown rot epidemics was tested.

There are few precedents for fitting epidemiological models to epidemics of soil- or residue-borne diseases over successive years (Zadoks 1999). However, it can be assumed that disease progress during the increasing phase of a multi-year epidemic will be similar to a discontinuous form of epidemics of polycyclic disease within a season. Three commonly used disease progress models, the monomolecular, logistic and Gompertz (Campbell 1998), were fitted to the data.

The monomolecular model takes the form,

$$y = 1\text{-}(1\text{-}y_0)\exp(-r_M t)$$

where $y$ is incidence at time $t$, $y0$ is initial incidence, and $r_M$ is a rate parameter specific to this model. It can be linearised by using the transformation $\ln[1/(1\text{-}y)]$ in place of $y$. This model has most commonly been used for monocyclic diseases.

The logistic model is of the form,

$$y = 1/\{1+[(1\text{-}y_0)/y_0]\exp(-r_L t)$$

where $r_L$ is the rate parameter specific to the logistic model. It can be linearised with the transformation $\ln[y/(1\text{-}y)]$. This is the most common model fitted to polycyclic diseases.

The Gompertz model takes the form

$$y = \exp\{[\ln(y_0)]\exp(-r_G t)\}$$

where $r_G$ is the appropriate rate parameter. It can be linearised using the $–\ln[-\ln(y)]$ transformation (Nutter and Parker 1997).

The models were fitted in their linear forms. As expressed above, the models assume a carrying capacity $K$, or maximum possible disease incidence, equal to 1 (100%). Because disease peaked at less than 100% at both sites, values of $K$ which gave the best linear fit for each model at each site were determined. Models were compared by regressing back-transformed predicted values against actual incidence for each site (Campbell 1998). The models were also compared with an assumption of linear increase in disease or infection incidence.

*Regression analysis*

Incidence of crown rot in each crop was transformed with the $\ln[y/(K\text{-}y)]$ transformation according to the logistic model, using the optimum values of $K$ calculated for each site. Infection rates ($r_L$) were calculated as the difference in transformed incidence between successive crops. Simple and multiple regressions of infection rate on factors that were considered to influence epidemiology were done using data from Moree. These included incidence of the disease in the previous crop, and yield of the previous crop because it had been noted that disease incidence was low in years following low-yielding crops (Burgess *et al*. 2001). Swan *et al*. (2000) reported that rate of disease progress within seasons was dependent on soil moisture, with increases in incidence following major rainfall events. Therefore in-crop rainfall and the number of in-crop rainfall events greater than 10 mm were used. Mean monthly temperatures were tested because of the reported association between temperature and infection (Wildermuth and McNamara 1994).

Felton *et al*. (1998) reported that soil moisture at sowing was related to incidence of disease at the end of the season. Soil moisture was not measured in most crops at Moree, so the soil moisture model in the DYMEX software package (Maywald *et al.* 1999) was used to estimate stored moisture. This is based on the water-balance model of Fitzpatrick and Nix (1969) and also uses an estimate of evaporation based on temperature, humidity and daylength. Parameters used in the soil moisture model were a storage capacity of 180 mm assuming a rooting depth of 1.2 m and storage of 150 mm $m^{-1}$ (Crofts *et al*. 1988), evaporation from the soil surface during fallows of 0.4 of pan evaporation (Fitzpatrick and Nix 1969) and no loss by drainage. Fallow rainfall was also tested as an indicator of water available to the crop.

Survival of the fungus in residues is reduced by warm, moist conditions (Burgess *et al*. 2001), and Roget (2001) has reported that survival of *G. graminis* var. *tritici* was affected by summer rainfall events greater than 25 mm. Mean temperature, total rainfall and number of rainfall events >25 mm during the period December-February preceding each crop were therefore tested. The data set used for the regression analysis at Moree is given in Table 2.

Relationships between variables suggested by analysis at Moree were tested on 7 crops at Billa Billa for which comparable data were available.

*Predicting future incidence at critical decision points*

The ability to predict incidence of crown rot in the next wheat crop using data available at harvest or prior to sowing was tested. All available data from Moree were used in simple and multiple regressions. The factors or combinations of factors that gave the best regressions at Moree were used in equivalent regressions on data from Billa Billa. Parameters that were considered to be most useful in simple forecasting systems were identified, and predictions of crown rot incidence based on these parameters were compared with actual incidence in a long-term trial at Moree and at Billa Billa.

**Results**

*Epidemiological models of disease increase*

All models tested gave highly significant fits ($r^2 > 0.94$, $p < 0.01$) when back-transformed predicted values were compared with actual data (Table 3). The best fits for the monomolecular model were obtained with $K$ = 100%, but this model gave the least satisfactory fits at both sites. The best fits for the logistic model were obtained with values of $K$ approximately 5% above the highest incidence recorded. This was the best-fitting model at Billa Billa, and was only very slightly poorer than the linear or Gompertz models at Moree. Best fits with the Gompertz models were obtained with $K$ approximately 25% above the maximum recorded incidence.

*Regression analysis*

At Moree there were significant positive correlations between infection rate and yield in the previous season, and in-crop rainfall in the previous season, and significant negative correlations with disease incidence in the previous season, rainfall in December-February and number of rainfall events greater than 25 mm in December-February (Table 4). Most of these factors also showed strong correlations with infection rate at Billa Billa, although only the correlation with yield in the previous season was significant because of the smaller number of data points.

The negative correlation between infection rate and disease in the previous season probably reflects the positive infection rates while incidence was increasing from a low level, and negative infection rates following some of the most heavily infected crops (Figs 1, 2). The other factors that showed significant correlations fell into two groups. Yield and in-crop rainfall in the previous season, which were positively correlated with infection rate, were most likely indicators of vegetative growth of the crops and hence of the quantity of residue produced. The summer rainfall parameters, which were negatively correlated with infection rate, indicated the conditions that favoured residue decomposition and decline in inoculum between crops.

Stepwise multiple regression was used on the Moree data to find combinations of factors that were correlated with infection rate. When in-crop rainfall in the previous season was included in the regressions, no other factors made significant improvements to the model, although the addition of fallow rainfall made a slight increase in $R^2$, from 0.59 to 0.65. No other regression models gave better fits to the data than those which included rainfall in the previous season.

*Predicting future incidence at critical decision points*

The ability to predict incidence of crown rot in the next season using simple models based on data available at harvest or sowing was tested. Data available at harvest were yield, in-crop rainfall and incidence of infection (Moree) or of symptoms (Billa Billa). Dry matter at anthesis was also available at Billa Billa. Incidence of infection at Moree was significantly positively correlated with incidence in the previous season ($r^2 = 0.33$, $p < 0.05$) but not with the other factors. This correlation was also significant at Billa Billa ($r^2 = 0.54$, $p < 0.05$).

Multiple regressions of incidence of infection on incidence in the previous season and either yield of the previous crop or in-crop rainfall in the previous season gave much better fits to the data than simple regressions at Moree (Table 5). These factors were likely to be estimating two components of quantity of inoculum, that is the quantity of residues produced and the proportion of residue infested with the fungus. Various combinations and transformations of these factors were tested, but the best fit was obtained with the square root of the product of incidence in the previous season and either yield or in-crop rainfall in the previous season (Table 5).

Additional data available for forecasting at sowing were summer (December-February) rainfall and rainfall events > 25 mm, mean temperature in December-February, fallow rainfall and stored soil moisture. Of these, only December-February rainfall made a significant ($p < 0.05$) contribution when it was added to regressions on the square root of the product of incidence in the previous season and either yield or in-crop rainfall in the previous season (Table 5). December-February rainfall had a small, negative effect on incidence consistent with its effect on infection rate.

At Billa Billa, the same combinations of factors gave similar results in multiple regressions as at Moree, although the proportion of variability in the observations accounted for by the regressions was higher (Table 5). Substituting dry matter at anthesis for yield in the regressions resulted in poorer fits which were generally not significant.

A farm-based forecasting system should use calculations that are as simple as possible. The ability of simple regressions through the origin to forecast disease incidence in the longest run of data at Moree, which was for a long-term stubble management trial, and at Billa Billa was tested. The regressions were

$$\text{Incidence} = 5.96\sqrt{(\text{previous incidence x previous yield})}$$

and

$$\text{Incidence} = 0.49\sqrt{(\text{previous incidence x previous in-crop rainfall})}$$

at Moree and

$$\text{Incidence} = 4.45\sqrt{(\text{previous incidence x previous yield})}$$

and

$$\text{Incidence} = 0.66\sqrt{(\text{previous incidence x previous in-crop rainfall})}$$

at Billa Billa.

Predictions based on these models were generally within 5-10% of actual disease incidence at both Billa Billa and Moree over a wide range of incidences (Figs 1, 2). However, the model using yield underestimated the incidence of infection at Moree in 1996. Addition of December-February rainfall made only a slight improvement to the predictions (not shown).

**Discussion**

Of the growth curve models fitted to the expanding phase of crown rot epidemics at Moree and Billa Billa, the logistic model appears to be the most appropriate. Although there was little difference in goodness of fit between this and the Gompertz model, the optimum carrying capacity ($K$) calculated for the logistic models made most sense, being slightly above the maximum recorded incidence at each site. Both the logistic and Gompertz growth curves are sigmoidal, but the Gompertz model has a lower point of inflection, $K$/e, compared with $K$/2 for the logistic model (Nutter and Parker 1997). A higher value of $K$ would give a Gompertz curve very similar to a logistic curve. However, since the values of $K$ needed to give the best fit with Gompertz models can not be readily explained in terms of any observed features of the epidemic, the logistic model is to be preferred.

In practice a linear model of disease increase gave a fit to the observed data that was very little different from the logistic model, especially at Moree. This suggests that calculations in predictive systems for the increasing phase of the disease could be simplified by assuming linear increase, with very little loss of precision.

The method chosen to explore environmental effects on disease progress was to examine correlations with infection rates based on the logistic growth model. This automatically corrects for differences in behaviour of the epidemic at low and high incidences under uniform conditions. The factors that appeared to have the greatest influence on infection rate were those related to biomass production, and those related to stubble decomposition rates. While in-crop rainfall and yield should have simple relationships with dry matter production (Passioura 1977) they also have possible interactions with crown rot. Severity of disease is inversely related to rainfall (Burgess *et al*. 2001), while crown rot also reduces yield (Klein *et al*. 1991). However the very strong correlations between infection rate and either yield or in-crop rainfall in the previous season suggest that these interactions have only a small effect on inoculum production. In particular, the differences in disease severity that might have been expected in the seasons in the data set, which included two severe droughts (1991 and 1994) as well as years with above-average rainfall, did not appear to be reflected in infection rate.

The negative correlation between summer rainfall and infection rate is consistent with observations that stubble decomposition is fastest under warm, moist conditions (Summerell and Burgess 1989) and that mortality of the fungus is linked to decomposition rate (Summerell and Burgess 1988). Summer temperatures were not strongly correlated with infection rate, indicating that moisture rather than temperature is the key factor controlling mortality. These findings confirm suggestions that crown rot incidence may be higher than expected following dry periods in which stubble breakdown is reduced (Burgess *et al*. 2001).

Other factors explored, such as soil moisture at sowing, in-crop rainfall in the current season, and temperature were not correlated with infection rate, indicating that these factors did not have a significant effect on crown rot incidence in these trials. This is somewhat surprising, especially given the apparent link between rainfall events and infection during the 1994 drought in one of the trials included in this analysis (Swan *et al*. 2000). However, they could be expected to have a greater effect on the severity of symptom expression, and hence on yield. These effects could not be tested using the data available, but are worth exploring further.

The product of incidence and either yield or in-crop rainfall has intuitive appeal as a measure of inoculum. It combines an index of the quantity of residue produced by a crop, which will be related to yield or rainfall, with an index of the proportion of residue pieces infested with the fungus, which will be related to incidence. Because there has been very little previous work on estimating inoculum of residue-borne diseases with similar epidemiology, there are no guides as to the expected relationship between quantity of inoculum and incidence of disease. However, a square root relationship was found to give the best predictions among a range of linear, logarithmic, exponential, polynomial and other functions tested.

The predictive system for take-all described by Roget (2001) divided crops into high, medium and low risk categories based on inoculum. By analogy a predictive system for crown rot would probably need to be able to estimate future incidence to within about 10% in order to give reliable segregation into three similar risk categories. A simple relationship based on the square root of the product of incidence and either yield or in-crop rainfall was able to do this for most of 17 crops tested at Moree and Billa Billa. A forecasting system should not place a crop into a lower risk category than actually observed. The only observation where this was likely to have been a problem was for the Moree crop in 1996 using a prediction based on yield, when the observed incidence was 17% higher than predicted. One possible explanation for this is the presence of grassy weeds, which had become a problem after the long period of continuous wheat (LW Burgess, pers. comm.). High levels of susceptible grassy weeds (mainly wild oats) in the 1995 crop would have meant that crop yield would underestimate the total biomass of infested residue. The prediction based on in-crop rainfall was very close to the observed incidence for this crop, indicating that it was giving a better estimate of total crop plus weed biomass.

Adding other epidemiological factors did little to improve the accuracy of predicted incidence. Only summer (December-February) rainfall made a significant contribution to regressions. Calculating risk category based on a multiple regression equation is unnecessarily complex for an on-farm forecasting system. In practice, it may be best to place the next crop into a risk category using an index calculated from disease incidence and either yield or in-crop rainfall, and modify this with a set of rules for other factors. For example, if the index was close to the boundary between categories, the crop could be placed in the higher category if summer rainfall was below average, and in the lower category if summer rainfall was above average. Given that the disease incidence at Billa Billa was determined by visual assessment of stem browning, it should be possible to gather all the data required for forecasting from on-farm observations.

The slopes of the relationships between predictive parameters and incidence differed between the sites. This could reflect environmental differences, such as the heavier soils at Moree being more conducive to the disease. However, disease was estimated in different ways at the two sites. At Moree the disease data were incidence of infection, while at Billa Billa they were incidence of symptoms of stem browning Typically, 5-10% of infected plants do not show stem browning (Klein *et al*. 1989; Summerell *et al*. 1989). Disease data were also collected later in the season at Moree. Together these could account for much of the difference in maximum observed incidence and slopes of regression lines between the two sites.

The regressions tested accounted for a greater proportion of variability at Billa Billa than at Moree. One possible explanation for this is the use of single variety at Billa Billa and several varieties at Moree. There is still very little known about differences between wheat varieties in contributions to inoculum or in susceptibility to infection. Also, the Moree site covered a much larger area and may have been subject to greater spatial heterogeneity. Despite the variability at Moree it was still possible to predict incidence of infection reasonably accurately using a small number of parameters.

This analysis has demonstrated the feasibility of a forecasting system for crown rot. There are a number of areas of further work required before it could be adopted. The relationship between incidence and potential yield loss needs to be determined over a range of environments so that appropriate risk categories can be defined, and the values of constants need to be determined for different regions. The analysis also needs to be extended to durum wheat, which shows a higher incidence of crown rot than bread wheat at equivalent inoculum potentials (Kirkegaard *et al*. 2004).

The epidemiological analysis and forecasting system described here apply to continuous cropping of wheat with zero tillage and stubble retention. The effects of management practices such as fallowing and rotation, and environmental factors linked to locality and soil type, could possibly be determined by using the estimators of inoculum found here as covariates in meta-analyses of survey and trial data from a broader range of sites and seasons. This would allow the development of more comprehensive forecasting and decision support systems for crown rot in the northern grains region.

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Table 1. Incidence of infection with *Fusarium pseudograminearum*, logistic infection rate between seasons, and yield of wheat in three series of trials on adjacent sites at Moree.

| Year | Incidence (%) | Infection rate ($r_L$)[A] | Yield (t/ha) |
|---|---|---|---|
| *Long-term management trial* | | | |
| 1986 | 4 | | n.d.[B] |
| 1987 | 22 | 2.02 | 1.31 |
| 1988 | 43 | 1.23 | 2.68 |
| 1989 | 65 | 1.95 | 1.61 |
| 1990 | 60 | -0.69 | 1.07 |
| 1991 | 55 | -0.46 | 0.63 |
| 1992 | 35 | -1.26 | 2.50 |
| 1993 | 42 | 0.40 | 1.70 |
| 1994 | 53 | 0.71 | 0.28 |
| 1995 | 28 | -1.51 | 1.03 |
| 1996 | 53 | 1.51 | 1.00 |
| *Late stubble burning trial* | | | |
| 1991 | 23 | | 0.92 |
| 1992 | 15 | -0.58 | 2.69 |
| 1993 | 38 | 1.46 | 2.71 |
| 1994 | 34 | -0.23 | 0.42 |
| 1995 | 31 | -0.17 | 1.16 |
| 1996 | 55 | 1.49 | 2.01 |
| *Variety trial – low inoculum* | | | |
| 1994 | 8 | | 0.38 |
| 1995 | 7 | -0.15 | 1.38 |
| 1996 | 31 | 1.96 | 2.32 |

[A] Using $K$ = 71%; [B] not determined

Table 2. Environmental data at Moree used in analyses.

| Year | In-crop rain | | Fallow rain (mm) | Soil water at sowing (mm)[A] | December-February | | | Mean monthly temperature (°C) | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Total (mm) | Events > 10 mm | | | Rain (mm) | Rain events > 25mm | Mean temperature (°C) | Jun | Jul | Aug | Sep | Oct | Nov |
| 1986 | 293 | | | | | | | | | | | | |
| 1987 | 204 | 8 | 285 | 86 | 122 | 1 | 26.4 | 13.7 | 10.0 | 13.0 | 15.4 | 19.0 | 22.6 |
| 1988 | 289 | 8 | 483 | 149 | 211 | 4 | 23.5 | 11.5 | 12.2 | 13.0 | 16.5 | 22.4 | 22.9 |
| 1989 | 213 | 5 | 424 | 162 | 130 | 1 | 25.9 | 11.5 | 10.2 | 10.0 | 14.8 | 19.8 | 22.2 |
| 1990 | 152 | 5 | 337 | 171 | 104 | 2 | 26.4 | 11.2 | 10.8 | 10.3 | 15.2 | 19.9 | 24.3 |
| 1991 | 110 | 2 | 500 | 166 | 211 | 3 | 27.4 | 14.0 | 9.9 | 11.9 | 15.9 | 22.0 | 23.9 |
| 1992 | 275 | 8 | 404 | 113 | 381 | 5 | 25.2 | 11.2 | 11.5 | 12.4 | 14.7 | 19.1 | 21.4 |
| 1993 | 319 | 11 | 226 | 76 | 175 | 2 | 26.9 | 11.7 | 13.8 | 13.0 | 14.6 | 19.2 | 24.1 |
| 1994 | 110 | 4 | 271 | 81 | 186 | 3 | 26.4 | 12.2 | 11.3 | 12.4 | 15.0 | 20.6 | 24.0 |
| 1995 | 369 | 6 | 485 | 101 | 411 | 5 | 25.5 | 12.3 | 10.5 | 14.5 | 16.9 | 20.4 | 24.1 |
| 1996 | 184 | 8 | 294 | 97 | 209 | 3 | 24.4 | 13.8 | 10.7 | 12.5 | 16.0 | 20.5 | 22.5 |

[A] Modelled using the Evaporation and Soil Moisture modules in DYMEX (Maywald *et al*. 1999).

Table 3. Fits of population growth models to the increasing phase of epidemics of crown rot at Moree and Billa Billa. Fits of all models are significant at $p < 0.01$.

| Model | *K* of best fit (%) | | Goodness of fit ($r^2$) | |
|---|---|---|---|---|
| | Moree | Billa Billa | Moree | Billa Billa |
| Linear | - | - | 0.998 | 0.971 |
| Monomolecular | 100 | 100 | 0.966 | 0.942 |
| Logistic | 71 | 59 | 0.991 | 0.997 |
| Gompertz | 92 | 78 | 0.998 | 0.990 |

Table 4. Environmental and agronomic parameters significantly correlated with logistic infection rate ($r_L$) at Moree, and their correlations with infection rate at Billa Billa. Infection rates assume $K$ = 71% at Moree and $K$ = 59% at Billa Billa.

| Parameter | Correlation coefficient ($r$) | |
|---|---|---|
| | Moree | Billa Billa |
| Disease in the previous season | -0.622** | -0.689 |
| Yield in the previous season | 0.478* | 0.797* |
| In-crop rainfall in the previous season | 0.776** | 0.613 |
| Rainfall Dec-Feb | -0.621** | -0.457 |
| Rain events > 25 mm Dec-Feb | -0.651** | -0.506 |

* Significant at $p < 0.05$; ** significant at $p < 0.01$; 16 d.f. at Moree and 6 d.f. at Billa Billa.

Table 5. Regressions of crown rot incidence on parameters available for use in forecasting at harvest or prior to sowing at Moree and Billa Billa

| Regression equation | Significance of regression | Fit of prediction ($r^2$) |
|---|---|---|
| *Moree* | | |
| y = 10.83 + 0. 49(previous incidence) + 8.73(previous yield) | * | 0.48 |
| y = 13.28 + 4.20√(previous incidence x previous yield) | ** | 0.52 |
| y = 49.76 + 1.75√(previous incidence x previous yield) – 0.08(Dec-Feb rain) | ** | 0.65 |
| y = -8.55 + 0.67(previous incidence) + 0.11(previous in-crop rain) | ** | 0.75 |
| y = 0.53 + 0.48√(previous incidence x previous in-crop rain) | ** | 0.74 |
| y = 17.74 + 0.40√(previous incidence x previous in-crop rain) – 0.04(Dec-Feb rain) | ** | 0.81 |
| *Billa Billa* | | |
| y = -38.53 + 0.926(previous incidence) + 18.26(previous yield) | * | 0.87 |
| y = -3.5 + 4.85√(previous incidence x previous yield) | ** | 0.86 |
| y = -1.38 + 4.89√(previous incidence x previous yield) – 0.01(Dec-Feb rain) | * | 0.87 |
| y = -6.98 + 0.80(previous incidence) + 0.14(previous in-crop rain) | ns | 0.77 |
| y = -2.84 + 0.71√(previous incidence x previous in-crop rain) | ** | 0.86 |
| y = -0.50 + 0.72√(previous incidence x previous in-crop rain) – 0.01(Dec-Feb rain) | * | 0.86 |

* Significant at $p < 0.05$; ** significant at $p < 0.01$; ns not significant. 16 d.f. at Moree; 6 d.f. at Billa Billa.

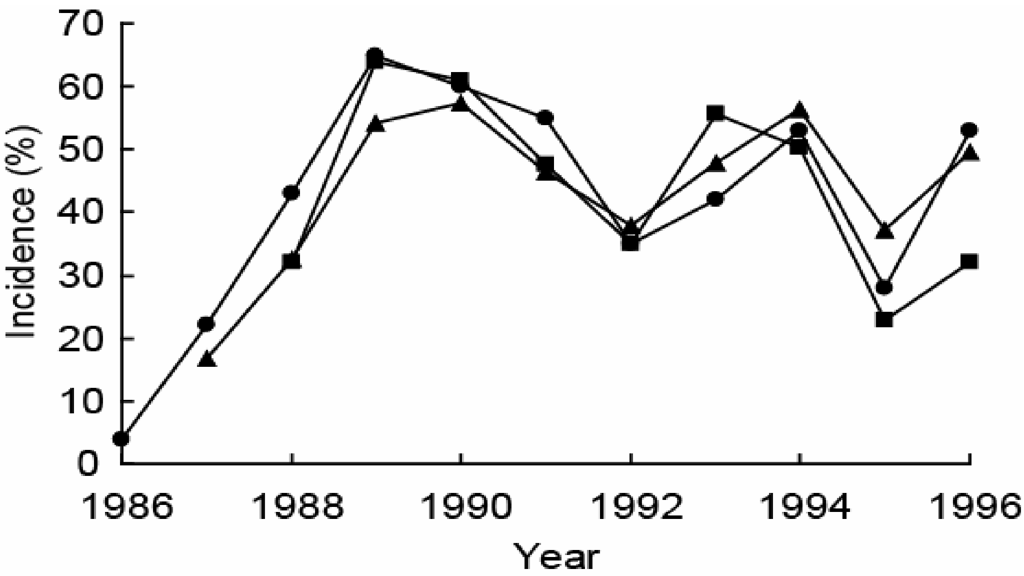


**Fig. 1.** Observed (●) and predicted incidence of crown rot in a long-term trial at Moree based on the formulae y = 5.96√(previous incidence x previous yield) (■) and y = 0.49√(previous incidence x previous in-crop rainfall) (▲). Yield in the previous season was not available for the 1987 crop.

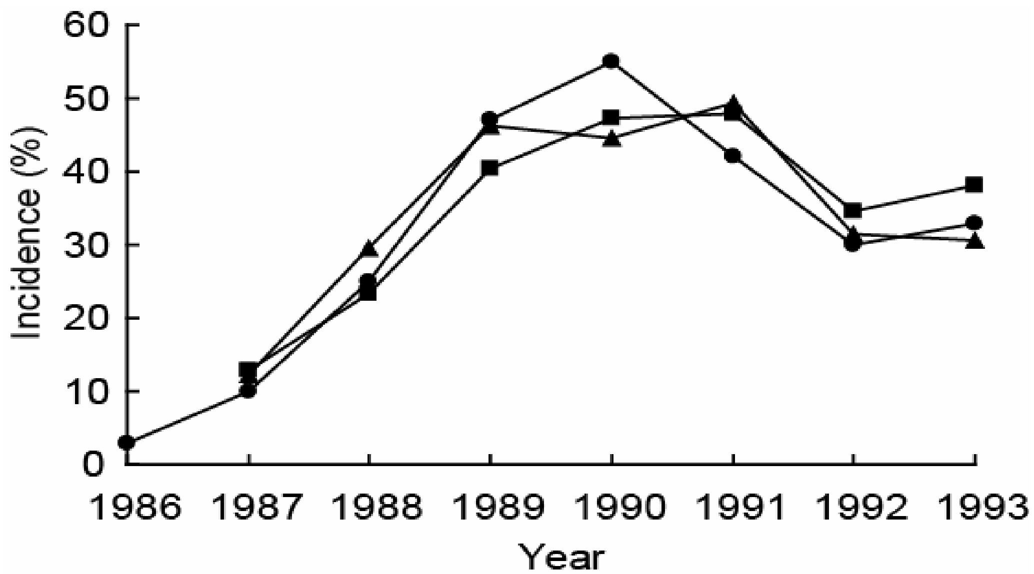


**Fig. 2.** Observed (●) and predicted incidence of crown rot in a trial at Billa Billa based on the formulae y = 4.45√(previous incidence x previous yield) (■) and y = 0.66√(previous incidence x previous in-crop rainfall) (▲).

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