ASSESSING LOSS DUE TO BACTERIAL BLIGHT

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Bacterial blight [*Xanthomonas campestris* pv. *malvacearum* (Smith 1901) Dye 1972], of cotton produces angular spots on leaves and squares, causes defoliation, lesions on stems and bolls and reduces yield. Downward spread of the disease from the principal veins of the leaf blade or by direct infection of lateral branches results in the black arm symptom. This is the most serious form of bacterial blight and occurs in seasons most favourable to the pathogen or when crops have been severely stressed. Each season bacterial blight is present in varying levels of severity in the majority of crops. The grower is most aware of this disease by the damage it causes to the boll. This consists of discolouration of the lint, destruction of one or more locules, bolls failing to open and increased boll drop.

Survey for bacterial blight

Bacterial blight of cotton was first recorded in Queensland in 1923 (Simmonds 1966). Since that time the importance of this disease to the industry has been debated but, until recently, work had not been initiated to quantify the effect of bacterial blight on yield.

Disease crop loss assessment comparing healthy and diseased plantings is largely dependent on effective disease control measures. In the absence of any chemical control, another approach used in crop loss assessment is the use of isogenic varieites (genetically similar in all respects other than disease resistance). Because bacterial disease control measures and suitable isogenic cotton varieties are not available, assessment of yield loss by bacterial blight requires a different approach. One option available when assessing crop loss by a bacterial disease is to inoculate plots with different inoculum concentrations to obtain varying levels of disease severity. Correlation of a disease severity index with yield loss gives a measure of yield reduction for a specific amount of disease. Establishing disease epidemics and controlling disease severity within specific limits is a major problem in such work. Research workers in the New South Wales Department of Agriculture are involved in such detailed disease assessment investigations.

Some estimate of yield loss can be obtained by surveying crops for boll infection. Arnold (1965) concluded from his work on the effect of bacterial blight infection on growth and yield, that at normal spacing, crop loss due to this disease can be attributed to damage to fruiting structures provided it occurs late in the season and for which the plant cannot compensate. Over the last few years cotton crops at early boll opening were surveyed measuring percentage boll infection. This gave an estimate of the level of bacterial blight in crops in the different districts (Table 1). Figures presented here do not take into account loss from boll drop nor do they show the upper and lower limits; for example 20-30% boll infection in a few plantings and 0% in others.

Table 1 Percentage bolls damaged by bacterial blight in cotton surveyed during seasons from 1981 to 1984

District	Mean % diseased bolls		
	1981/82	1982/83	1983/84
Biloela-Theodore	1	1.6	0.7
Emerald	4	4.6	1.0
Forest Hill	0	0	0.7
Cecil Plains-Brookstead	3	3.4	2.2
St. George	4	4.2	-
Namoi	4	-	12.1
Gwydir	13	-	13.8
Macquarie	8	-	11.2

Boll infection was higher in New South Wales than in Queensland in the two seasons surveyed. In the 1983/84 season, possibly due to the wet season, lesions were predominately basal resulting in total loss of the majority of infected bolls. Lesions, other than basal, caused varying degrees of damage from none to total boll loss. Queensland surveys revealed that despite the comparatively low district averages, the incidence and severity in a small number of crops was significant with yield losses in excess of 20%. Why such plantings should be more severely damaged by bacterial blight than others was not obvious. In order to understand the factors contributing to disease severity, experiments were carried out in controlled environment cabinets to examine environmental factors and the inoculum concentrations most likely to influence disease development.

Incubation time at high relative humidity

The effect of high relative humidity at inoculation on disease severity was examined using three inoculum concentrations. Cotton (Deltapine 61) was spray inoculated with 10^7, 10^5 and 10^4 cells of *X. campestris* pv. *malvacearum* cm^{-1} leaf disc and incubated at increasing times of incubation from 1 to 24 hours. Infection occurred within 1 hour of inoculation. The histogram (Fig. 1) shows that disease severity did not increase by prolonging exposure to 100% relative humidity after inocualtion, but that inoculum concentration had a much greater influence on disease severity.

Inoculum concentration

Earlier workers (Innes and Last 1961), using cotton lines with different levels of bacterial blight resistance, found that by increasing inoculum concentration, size and severity of individual lesions increased. Because the previous experiment also demonstrated that inoculum concentration was a factor influencing disease severity, the effect of inoculum concentration on pathogen multiplication within the host was examined. Cotton was inoculated with 10^8, 10^5 and 10^4 cells cm^{-1} leaf disc and the growth of the pathogen within the leaf tissue was followed over a period of 14 days. The multiplication pattern of the bacterium at the two highest concentrations appeared similar (Fig. 2) and symptoms developed within 9 days. Symptom expression was related to population numbers of 2 - 5 x 10^6 cells cm^{-1} leaf disc (Fig. 2). Where initial population was 10^4 cells cm^{-1} leaf disc, the rate of multiplication was similar to that of the two highest concentrations but an extra six days was needed for the population to reach numbers necessary for symptom expression. The longer time taken for lesion development may have important implications for inoculum production within the crop.

Relative humidity and temperature

Stoughton (1933) indicated that relative humidity had little direct effect on the pathogen after penetration into the plant. This could explain why prolonging time of exposure to relative humidity after inoculation did not increase disease severity. Examining the effect of low and high relative humidities on pathogen multiplication in leaf tissue would reveal any effect relative humidity had on growth rate of the pathogen after inoculation. Cotton was inoculated with 10^6 cells cm^{-1} leaf tissue and grown at 50 or 90%

relative humidities and 28°C. The growth rate of the pathogen was determined over 16 days. The growth patterns (Fig. 3) was similar at both relative humidities. Although symptoms appeared about 1 day later at 50% relative humidity, the delay could not be considered significant. This work confirms that relative humidity is not important in the disease development process.

Stoughton (1933) concluded that high air temperatures favoured disease development and that fluctuating temperatures had the same effect on disease progress as a constant temperature near to the mean of the variation. Temperature effect on disease development was examined by following the multiplication patterns of the pathogen at constant 35, 28 and 20°C. In this experiment cotton plants were inoculated with 10^5 cells cm^{-1} leaf disc. The growth patterns of the pathogen were similar at 35 and 28 but the rate of multiplication appeared to be slower at 20°C delaying symptom expression (Fig. 4).

Conclusion

The results of these experiments have not yet been analysed but the figures indicate certain trends. Increasing inoculum concentration appears to accelerate disease development and disease severity. Innes and Last (1961) suggested that increased inoculum concentration may lower the hosts resistance by the more numerous infections or that lesion size may be directly related to the number of bacteria at each infection site. Our experiments and observations indicate that size and number of lesions are related to the number of infection sites and these are related to inoculum concentration. Adjacent lesions by coalescing increase the pathogen number cm^{-1} leaf area as well as increasing lesion size. The more rapid lesion development with high inoculum concentration has important implications for inoculum availability for subsequent infections and build up of disease in the crop.

High temperatures by increasing multiplication rate of the pathogen contributes to rapid build up of disease. This is not the situation with relative humidity which had no obvious effect on pathogen growth after infection. The role of humidity in the infection process is probably related to prolonging the time infection droplets persist on the host (Stoughton 1933) giving greater opportunity for infection.

References

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Stoughton, R.H. (1933). The influence of environmental conditions on the development of the angular leaf-spot disease of cotton V. The influence of alternating and varying conditions on infection. Annals of Applied Biology 20, 590-611.

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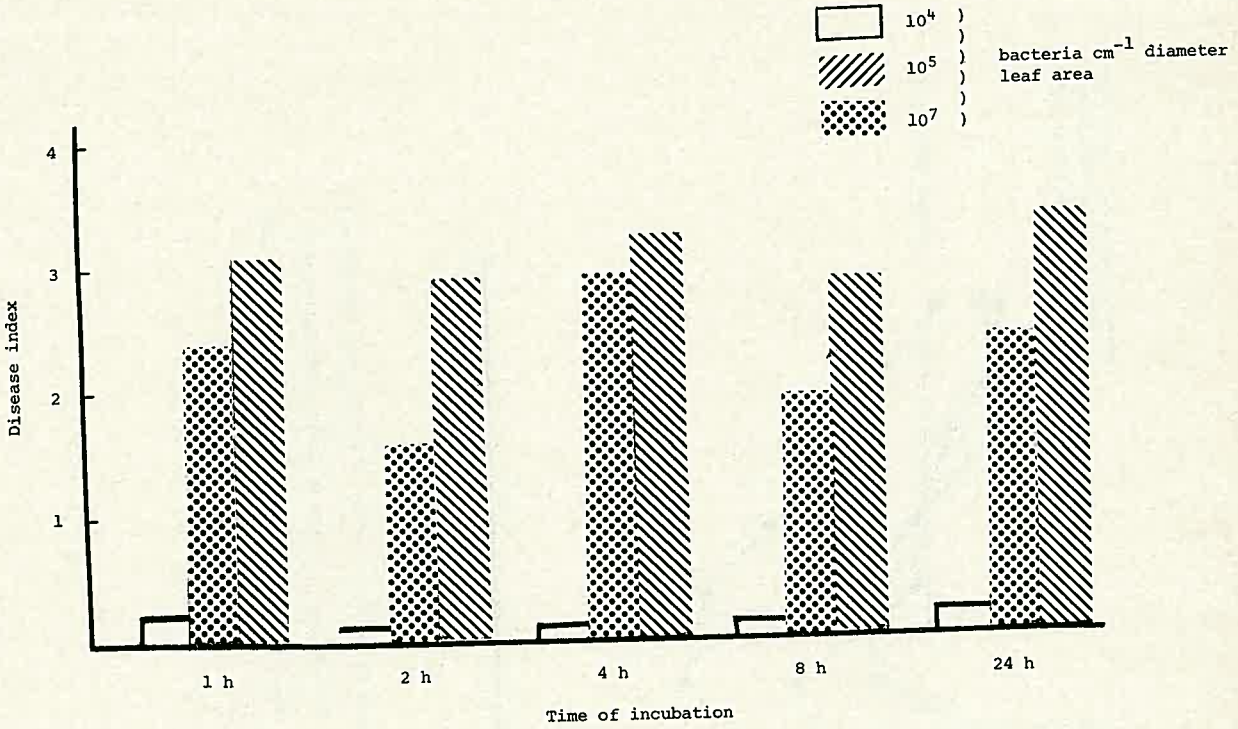

Figure 1. Disease severity expressed as a disease index following inoculation of cotton leaves with three concentrations of *Xanthomonas campestris* pv. *malvacearum* (10^4, 10^5, 10^7 bacteria cm^{-1} diameter leaf area) and incubation at 100% relative humidity for increasing time.

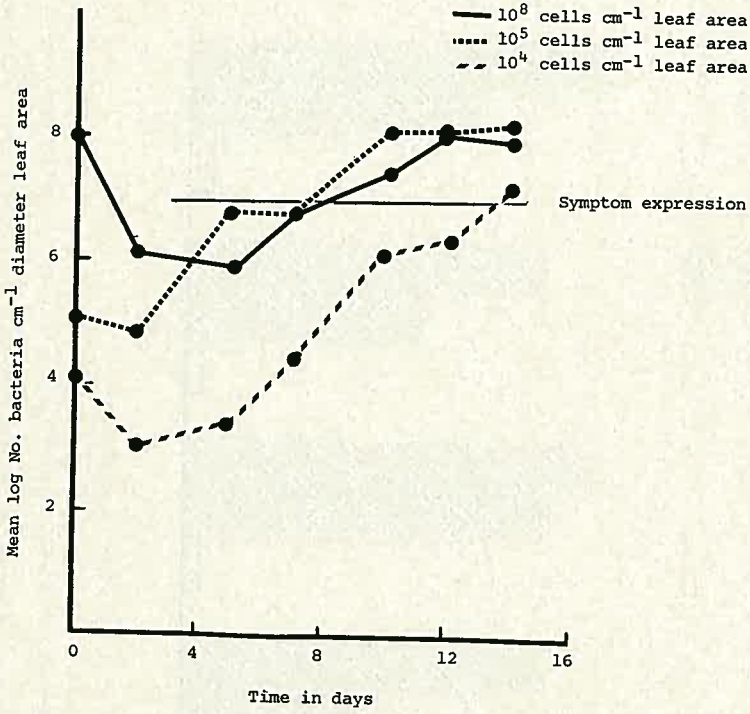

Figure 2. Multiplication of *Xanthomonas campestris* pv. *malvacearum* in cotton following inoculation with three concentrations 10^8, 10^5, 10^4 cells cm^{-1} leaf area

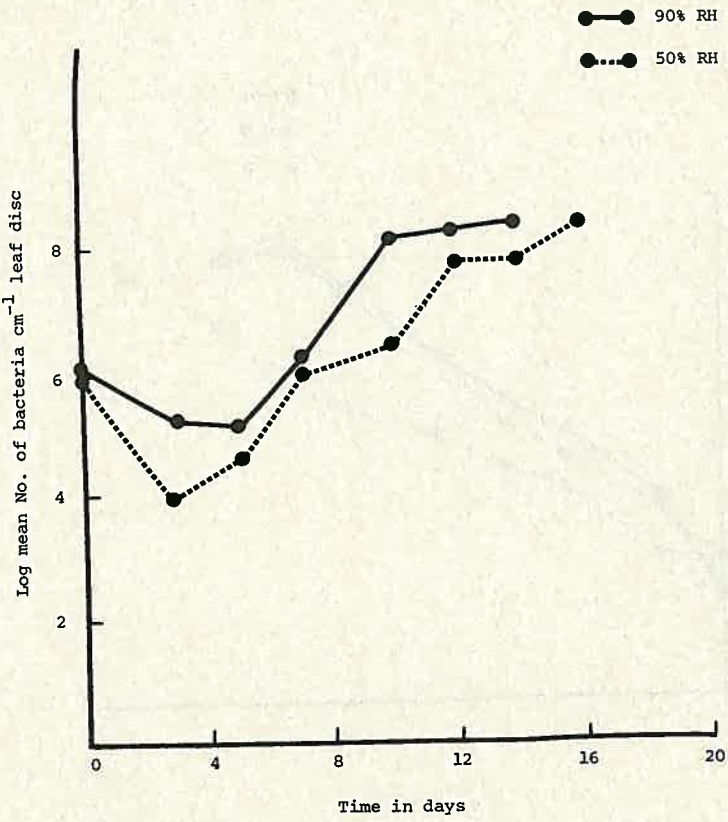

Figure 3. Multiplication of *Xanthomonas campestris* pv. *malvacearum* in cotton at 90 and 50% relative humidity and 28°C

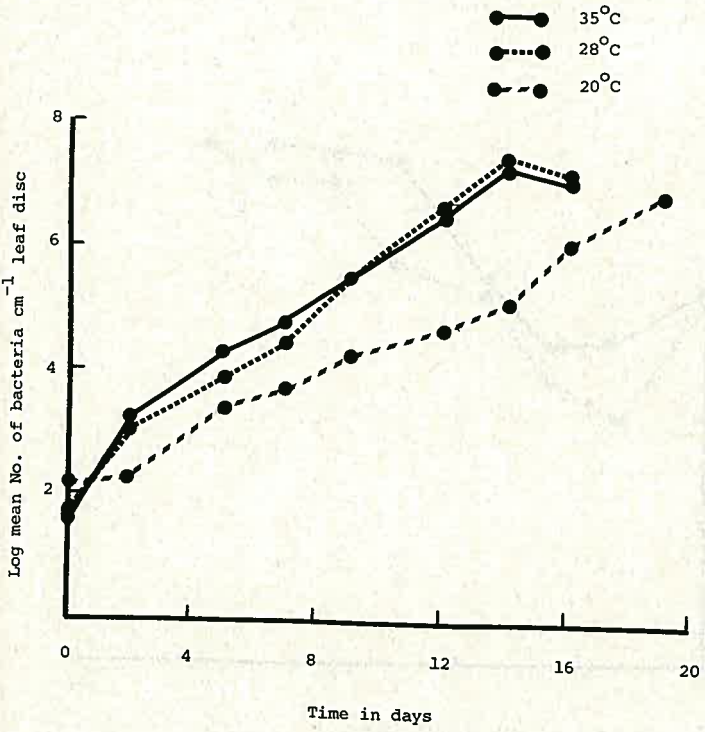

Figure 4. Multiplication of *Xanthomonas campestris* pv. *malvacearum* in cotton at 35, 28 and 20°C at 70% relative humidity