Phytophthora drechsleri and *Eucalyptus viminalis*

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There has been discussion by Pratt and Heather (2) and Marks (personal communication) as to whether *Phytophthora drechsleri* Tucker is pathogenic to native forest species. The following investigation is relevant.

In 1974 I inspected a grassed valley bordering a creek in the Dandenongs, about 40 km east of Melbourne. There were mature manna gums, *Eucalyptus viminalis* Labill., about 30 m high scattered on the grassy flat. Some of these appeared healthy, 50% were dead, and 25% had severe dieback symptoms. Examination failed to disclose specific symptoms on aerial parts of the trees. Isolations were made from surface-sterilized roots on various media and samples containing soil and fine roots were baited with New Zealand blue lupin seedlings (1). From all tests, *P. drechsleri* was the only pathogen isolated, and it was isolated consistently from all samples both by baiting and from surface-sterilized roots. Identification was checked by Dr. Stamps of the C.M.I. (I.M.I. 187472).

Six-month old seedlings of *E. viminalis* were inoculated with the same isolate. Ten plants were grown, one in each 15 cm pot of non-sterile soil which was tested for a number of pathogens with different media and found pathogen-free. Five of these were inoculated by the addition of washed mycelium to small holes in the soil, and five were maintained as controls. All plants were grown in the glasshouse at 25° ± 1°C, were saturated with water for 3 days, allowed to dry out and watered as required. This treatment was repeated at weekly intervals for 3 weeks. No plants died and after 4 months increase in height was measured and roots were surface-sterilized and plated on corn-meal agar. *P. drechsleri* was isolated from all inoculated plants, but not from the controls. Roots of all plants appeared intact. No lesions, stains or decays were observed. However, some decay of the finest roots may have occurred in the soil. Control plants showed a greater height increment compared with inoculated plants but the differences were not significant.

In this experiment *P. drechsleri* grew in the roots of *E. viminalis* but did not kill the seedlings, although it apparently reduced height increment. No other symptoms were present. It seems likely that other factors must have operated, perhaps in conjunction with *P. drechsleri* and waterlogging, to destroy the mature trees in the Dandenongs.

REFERENCES

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NEW DISEASES

Cercospora sorghi on Sorghum in Australia

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The grey leaf spot fungus *Cercospora sorghi* Ell. & Ev. was identified on *Sorghum bicolor* (L.) Moench collected in November, 1974 at Berrimah (near Darwin) N.T. and at Kununurra, W.A. The identifications were confirmed by Dr. J.L. Mulder of the Commonwealth Mycological Institute (IMI 190212, 190210). These appear to be the first records of *C. sorghi* for Australia. The known geographical range of the pathogen in 1974 did not include Australia (1). *C. sorghi* was previously unknown in Western Australia (R.F. Doepel, pers. comm.) and only unidentified species of *Cercospora* have been recorded on sorghum in Queensland (3) and in New South Wales (J. Walker, pers. comm.).

Lesions caused by *C. sorghi* are bacilliform to oblong (Fig. 1) and usually 5-15mm by 2-3mm in size. The colour of the lesions is a homogeneous reddish-brown except where heavy sporulation occurs in which case a diffuse grey colour appears on both upper and lower lesion surfaces.

Since the two original cases reported above, *C. sorghi* has been seen on sorghum at Milingimbi and at several locations in the Katherine area in the Northern Territory. A similar fungus has been found on *Sorghum almum* Parodi near Darwin and Humpty Doo.

A severe grey leaf spot infection on sorghum cultivar 300F near Katherine in May, 1975 was rated 4 on a 0-5 lesion density scale (2). The disease in other Northern Territory localities was less severe but still significant.

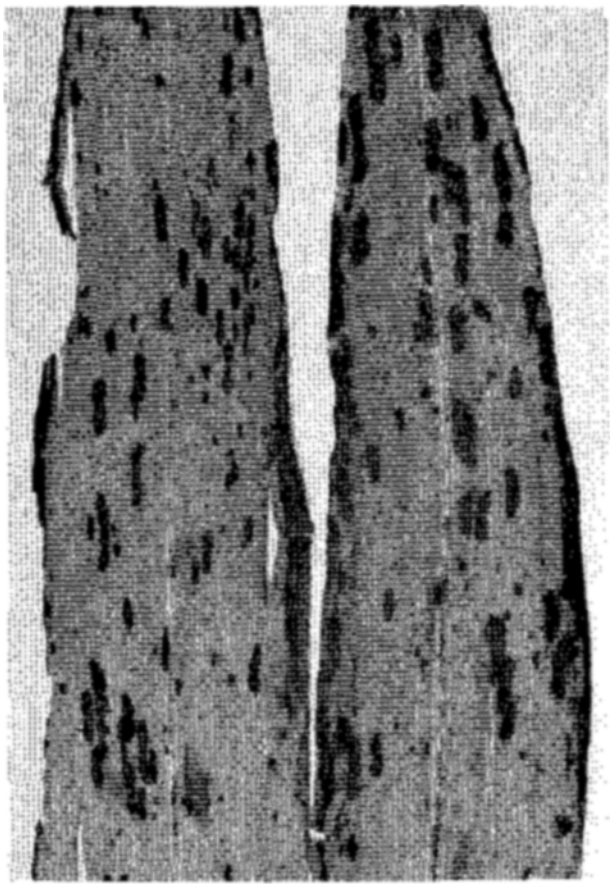

Figure 1 – Grey leaf spot (*Cercospora sorghi*) lesions on unidentified cultivar of *Sorghum bicolor* (approximately natural size).

Sorghum crops have been grown in the Northern Territory on a fairly extensive scale for the past 8 years and grey leaf spot symptoms have not been observed before. The sudden conspicuous appearance of this disease during the 1974-75 wet season suggests either that *C. sorghi* was not present before or that it existed in a relatively mild form.

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"The Rhizosquare Disease"

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It is axiomatic that, before disease of any kind can occur, a potential host and pathogen must come together in space. In air-borne diseases it is necessary for inoculum of the pathogen to be deposited on a host surface before the infection process can be initiated. In many soil-borne diseases, on the other hand, it is often sufficient that inoculum should be located within the rhizosphere region, where exudates, diffusing outwards from the host root, can stimulate spore germination and induce chemotropically orientated growth or movement towards the root surface prior to penetration (1). Baker (1) has analysed real data and constructed 2 and 3 dimensional models to examine how the frequency of disease foci is related to variations in the density of inoculum when host and pathogen interact through such a rhizosphere effect.

"The Rhizosquare Disease" is a simple 2-dimensional model of such a disease situation, invented like "Popper-drop" (3) to aid in teaching elementary epidemiology. It employs a square "experimental plot" divided into 2500 locations in the form of 50 x 50 small squares. Down the left-hand side of the plot, the 50 rows of small squares are designated by the numbers 01-50, and, along the top, the 50 columns of small squares by the numbers 51-100 (written 00). Thus any one of the 2500 small squares within the plot can be specified by two two-figure numbers. In the exercise, doses of inoculum are distributed about the plot at random by using two random number tables (01-50 and 51-00 respectively) to assign locations to individual doses. The hosts, which for convenience, are imagined to have single tap roots growing vertically down through the soil producing a rhizosphere region of square cross sectional area (equal to 36 small squares), are "planted" in a regular array on a transparent or perforated overlay of the same size as the plot. The effect of varying inoculum density can then be simulated by adding successive batches of inoculum to the plot. Disease incidence can be assessed after each batch, using the overlay to see which plants become diseased as a result of their "rhizosquare" intersecting with one or more doses of inoculum.

Figure 1 shows the mean results obtained from duplicate runs of the model in which inoculum was added, ten doses at a time, to an array of 25 host plants. The progressive total of infection events increased linearly in proportion to the amount of inoculum added to the plot, but, as a result of an increasing number of multiple infections, the graph relating number of infected plants to applied inoculum has the familiar curvi-linear shape. For example, 10 plants became infected as a result of adding 30 doses of inoculum to the plot, but it required an additional 60 inoculum doses to infect a further 10 plants. As well as bringing this point home to students, results obtained from the model can be used to demonstrate how Gregory's (4) multiple infection transformation can be used to calculate back from observed disease incidence to produce estimates of actual numbers of infections.

The advantage of using an overlay for the host array is that, by the use of several different overlays, the model can also be used to assess how disease incidence is affected by

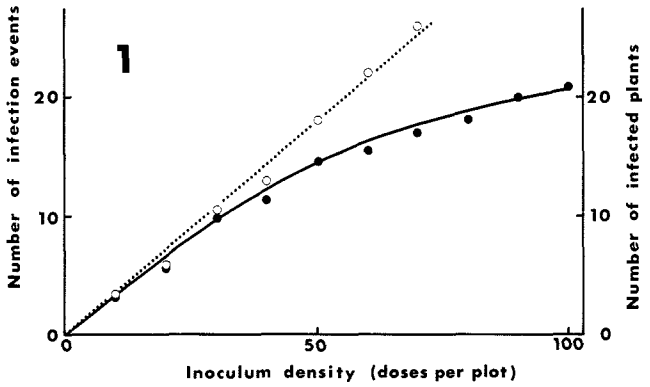

Figure 1 — Number of infections (dotted line) and number of infected plants (solid line) plotted against density of inoculum applied to a stand of 25 plants.

changing host density instead of inoculum density. Figure 2 shows the mean result obtained from duplicate runs of the model in which different density arrays of host plants were exposed to 50 randomly placed doses of inoculum. The graph lines for number of infections and disease incidence again diverge, due to the occurrence of multiple infections on the one plant, but the graph relating disease incidence to host density is linear because the proportion of plants present that receive multiple infections remains a constant in this simple system. This second exercise draws attention to host density as an important component of the disease triangle. This has received insufficient attention in the past, and only recently have experiments with damping-off (2) provided a clear demonstration that varying host density can have just as potent an effect on disease incidence as varying inoculum density.

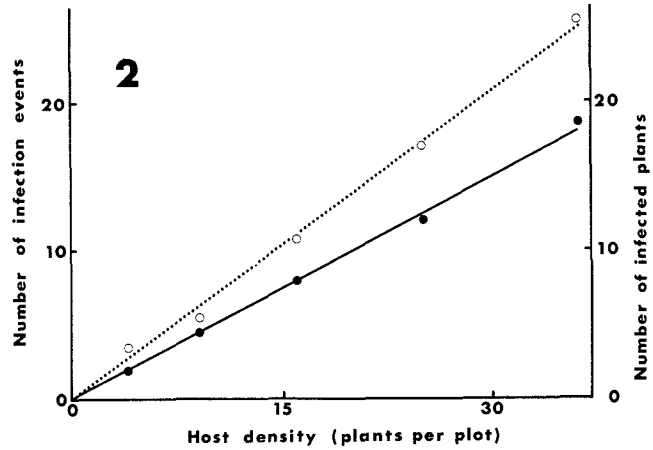

Figure 2 — Number of infections (dotted line) and number of infected plants (solid line) plotted against density of plants exposed to a standard 50 doses of inoculum.

Teachers of Plant Pathology who would like to evaluate "The Rhizosquare Disease" for possible use in their own courses are invited to send to one of the authors (G.A.C.) for a full copy of the student exercise.