

Two Diseases in *Stylosanthes* spp. Caused by *Colletotrichum gloeosporioides* in Australia, and Pathogenic Specialization within One of the Causal Organisms

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Abstract

Two anthracnose diseases caused by *Colletotrichum gloeosporioides* have been found in *Stylosanthes* spp. in northern Australia. The two diseases can be readily distinguished by their symptoms and their pure-culture characteristics. The disease classified as type A was widespread, but the other, type B, was found at only two localities. In spray-inoculation tests under controlled conditions, *S. guianensis* cv. Endeavour was highly susceptible to type B isolates, and some lines of *S. fruticosa*, *S. humilis*, *S. scabra* and *S. viscosa* were highly susceptible to the type A isolates. Two pathogenic races of type A were recognized from the reaction of *S. viscosa* 33941 towards them.

The importance of these diseases is discussed, together with aspects of disease dissemination and future control strategies.

Introduction

The symptoms of an anthracnose disease of *Stylosanthes* spp. and a description of the organism responsible (*Colletotrichum gloeosporioides* Penz.) were first reported by Sonoda *et al.* (1974) in Florida, U.S.A. *C. gloeosporioides* was also reported as being the cause of a leaf spot and stem canker disease of *Stylosanthes* spp. in Colombia, South America (Anon 1973; Baldion *et al.* 1975). These workers presented some evidence of the presence of pathogenic races of the causal organism, but Sonoda (1973) found no pathogenic specialization among Florida isolates of the fungus. The commercial cultivars of *S. guianensis*, Schofield, Cook and Endeavour, were all highly susceptible to the Colombian isolates (Baldion *et al.* 1975).

A *Colletotrichum* leaf and stem disease of *Stylosanthes* spp., having symptoms identical with those described by Sonoda *et al.* (1974), was first recorded in Australia at Kalinga, northern Queensland in 1973 (Pont and Irwin 1976). Field reports of serious losses to *Stylosanthes* spp. caused by anthracnose diseases have been made in Queensland in 1975, 1976 and 1977 (unpublished records, Queensland Department of Primary Industries and Division of Tropical Crops and Pastures, CSIRO).

This paper reports studies made to determine the incidence of anthracnose diseases of *Stylosanthes* spp. present in Australia. Detailed descriptions of the symptoms of the diseases, and the results of morphological and physiological studies made on the causal organisms are given. Evidence of pathogenic specialization within one of the causal organisms is presented.

Materials and Methods

Collection and Pathogenicity Testing of Anthracnose Isolates

Specimens of *Stylosanthes* spp. showing anthracnose symptoms were collected by the authors from 20 localities in southern and northern Queensland. Specimens were also submitted for pathological examination from other sites in Queensland, from Katherine, N.T., and from Derby, W.A. The symptoms on each specimen were recorded, and isolations made by sterilizing the surface of diseased stem and leaf tissue for 1 min in 0.1% mercuric chloride solution, rinsing the samples in three changes of sterile water, blotting dry and plating onto potato dextrose agar (PDA), four tissue pieces per plate.

To test the pathogenicity of the *Colletotrichum* isolates, plants were grown in a glasshouse in 15-cm diameter plastic pots, 10 plants per pot, in a peat/sand mix (1/1 by vol.) fertilized with the necessary nutrients. Each isolate was tested on two pots of each plant species when the plants were 4–6 weeks old. An inoculum was prepared by incubating spores streaked onto Ca^{2+} V-8 agar at 25°C under near ultraviolet light for 10 days. The leaves and stems of the plants were atomized with a spore suspension (c. 1×10^6 spores ml^{-1}) until run-off and the pots enclosed in plastic bags that had been moistened on the inside. Treated plants were transferred to naturally lit growth cabinets maintained at a temperature of $25 \pm 2^\circ\text{C}$ with a relative humidity of $>60\%$. The plastic bags were removed after 72 hr.

Similar methods were made use of to compare the disease reaction of seven of the isolates listed in Table 1 on 10 species and lines of *Stylosanthes* (Table 2). Inoculated plants were placed in two naturally lit, controlled-environment cabinets with one pot of each line/isolate combination and one control pot of each line in each cabinet. Within a cabinet the 10 lines inoculated with a common isolate were arranged in a separate block. Two separate runs were conducted, the first being planted on 1 April 1976 and the second on 27 September 1976. The same 10 lines of *Stylosanthes* were also inoculated, at 4 months of age, with each of three isolates, 21365, 21364 and 21423a. After inoculation the plants were transferred to a Polythene humidity chamber in a glasshouse with a relative humidity of $>80\%$.

The stems and leaves of each plant were rated for disease severity, 10 days after inoculation, with the following rating systems.

Stems: 0, no visible symptoms;

- 1, small spots, <1.0 mm in diameter;
- 2, lesions 1–2 mm long, narrow;
- 3, lesions >2.0 mm long, wide, but do not girdle stem;
- 4, long coalescing lesions that girdle the stem;
- 5, lethal to entire stem.

Leaves: 0, no visible symptoms;

- 1, spots <0.5 mm in diameter;
- 2, spots 0.5–1.0 mm in diameter;
- 3, spots 1.0–2.0 mm in diameter, with small percentage of spots coalescing;
- 4, c. 50% of leaf area destroyed;
- 5, complete leaf blight and/or abscission.

The oldest unexpanded leaf at inoculation (L_0), the uppermost expanded leaf at inoculation (L_1), and the second uppermost expanded leaf at inoculation (L_2) were

rated and analyses of variance were performed on the mean leaf [$\frac{1}{3}(L_0 + L_1 + L_2)$] and stem ratings. The experimental design for both the seedling and older plant tests was a split/split plot with runs as main plots, isolates as subplots and lines as subsubplots.

Fungal Morphology and Growth-Temperature Relations

The size and shape of spores of six type A and two type B isolates were recorded on single-spore cultures prepared on Ca²⁺ V-8 agar as described above. The morphology of the fungus on the tissue of the host was examined by means of transverse sections (15–20 μ m) of diseased leaf tissue cut 12 days after inoculation of 6-week-old plants of *S. fruticosa* CPI41116* (type A, 21365) and *S. guianensis* cv. Endeavour (type B, 21423a).

The growth-temperature relations of five type A isolates and one type B isolate were investigated in single-replicate experiments conducted three times. Discs of the test fungus (6-mm diameter) were cut from the margin of 7-day-old PDA cultures and placed with the mycelium side downwards on PDA petri dishes. These were incubated under a range of temperatures from 3° to 36°C and the diameters of the colonies measured after 72 hours' incubation.

Results

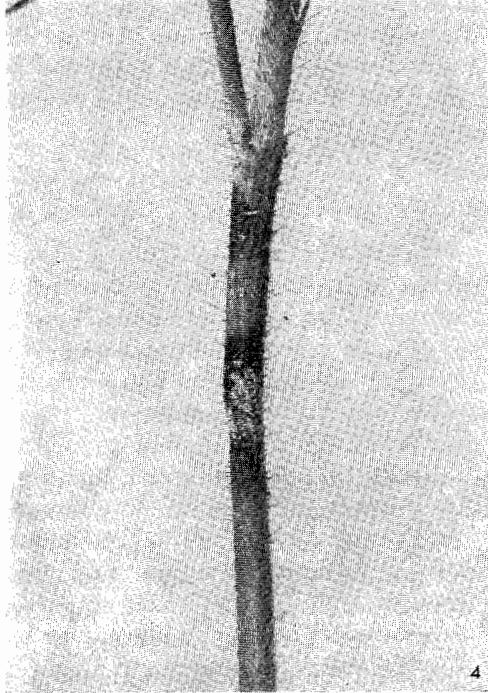
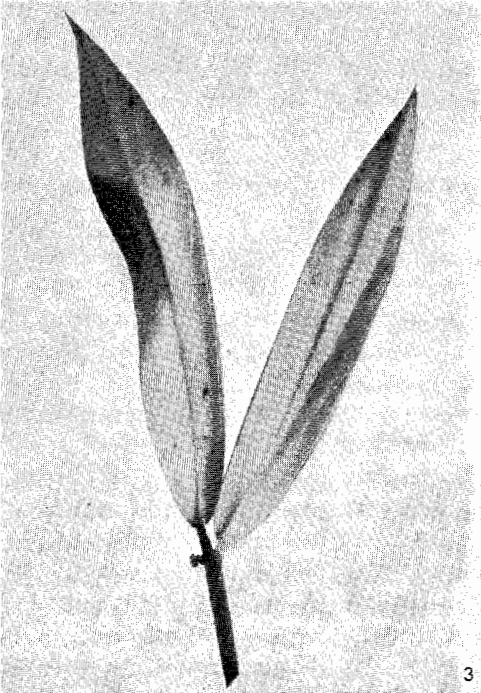
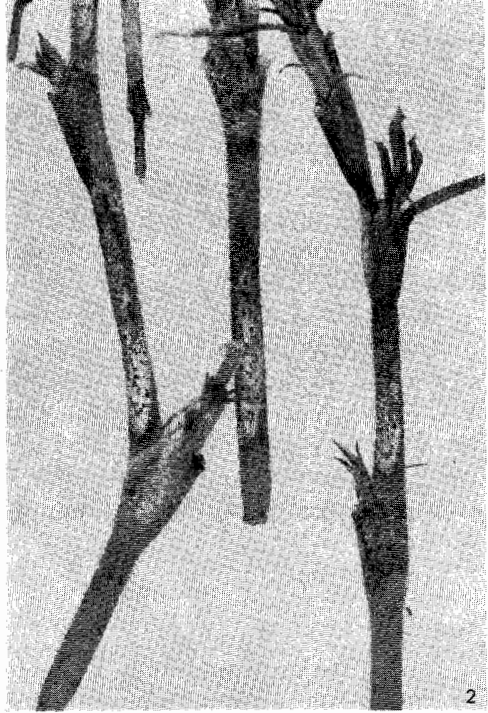
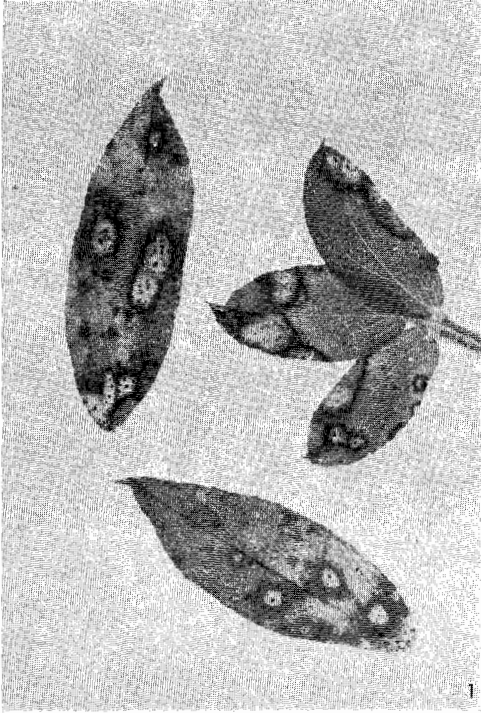
During the course of these studies, it became apparent that two anthracnose diseases of *Stylosanthes* spp., readily distinguishable by their symptoms, were present in Australia. One disease, described by Pont and Irwin (1976), was present in all *Stylosanthes* pastures sampled from Queensland, the Northern Territory, and Western Australia. This disease has been designated type A. Another disease, type B, was found at only two localities, Samford in southern Queensland and Tully in northern Queensland.

Disease symptoms

Type A disease, the most common, can be distinguished by the presence of lesions on the stems, leaflets and inflorescences, 1–4 mm in diameter, light in the centre and surrounded by a dark margin. On highly susceptible lines the leaf lesions coalesce, resulting in yellowing and abscission of the affected leaflet (Fig. 1). Lesions on the stems are usually elliptical and similar in colour to leaf lesions (Fig. 2). On highly susceptible lines the stem lesions may coalesce, resulting in the death of the stem. Species that have shown severe symptoms of this disease in the field include some lines of *S. fruticosa*, *S. humilis*, *S. viscosa*, *S. subsericea* and *S. scabra*, while lines of *S. guianensis* and *S. hamata* have shown only moderate susceptibility to the disease.

The other disease, type B, can be recognized by a general necrosis of the terminal shoots extending for several centimetres down the stem. The diseased tissue is always black in colour and the leaves are also affected, becoming completely blighted and showing the characteristic black discoloration. During periods of showery weather, pink spore masses of the fungus are visible on the affected leaf and stem tissue (Figs. 3, 4). In the field, disease symptoms have only been observed on lines of *S. guianensis*.

* Commonwealth Plant Introduction number.



Fungal morphology and growth-temperature relations**Cultural Characters**

The cultural characteristics of type A and type B isolates were compared on PDA.

Type A isolates. Growth was fast. The aerial mycelium was cottony or woolly, typically abundant and well elevated from the surface of the medium. The colour was white, turning dark grey with age. From below, the substrate is coloured cream, turning to various densities of grey with age. Conidia appear as pink masses formed

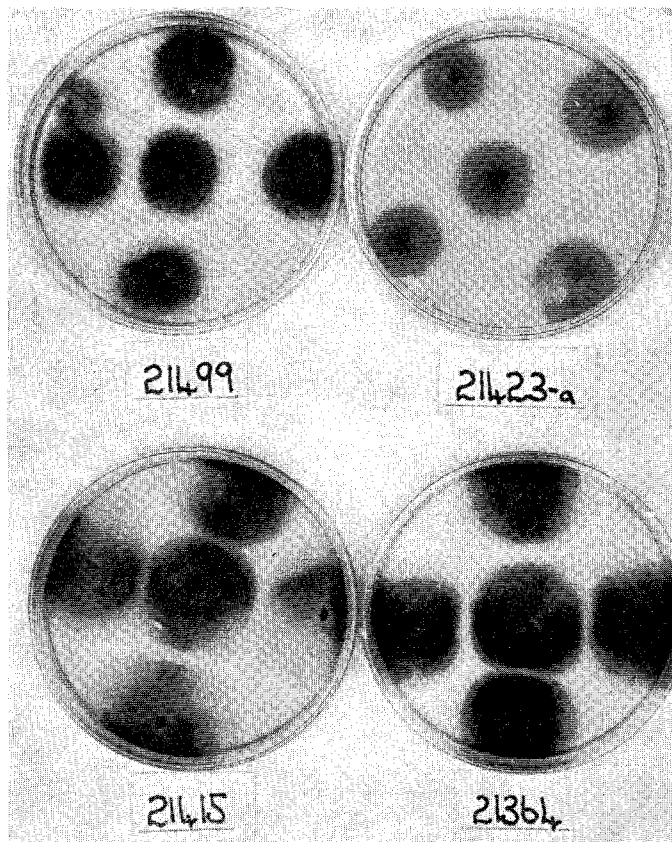


Fig. 5. Colonies on PDA of type B (top right-hand petri plate) and type A (remaining petri plates) *C. gloeosporioides* isolates. The photographs are of isolation plates made following glasshouse inoculations with each isolate. Note that the type B isolates are lighter in colour than the type A isolates.

Fig. 1. Leaf symptoms of type A disease on *S. viscosa* 33941.

Fig. 2. Stem symptoms of type A disease on *S. viscosa* 33941.

Fig. 3. Symptoms of type B disease on a leaflet of *S. guianensis* cv. Endeavour. The developing lesion does not have a light coloured centre as shown by type A disease in Fig. 1.

Fig. 4. A stem of *S. guianensis* cv. Cook showing a developing lesion typical of type B disease. Conidial masses of the causal fungus are present on the lesion.

in acervuli throughout the substrate. Numerous dark, sterile, perithecial elements were produced on tufts of mycelium above the agar surface.

Type B isolates. Growth was slow. The aerial mycelium was fine, white to light grey, and of moderate elevation. From below, the substrate was cream to light olive. The production of conidia occurred throughout the mycelium, giving a pinkish tinge to the culture. Perithecial initials were not observed in culture.

Typical colonies of type A and type B isolates are shown in Fig. 5.

Spore Morphology in Pure Culture

The origin of each isolate and the size and shape of conidia are listed in Table 1. The type B isolates have a much lower percentage of spores rounded at both ends and are more variable in length than the type A isolates (Table 1, Figs. 6 and 7).

Table 1. Origins of isolates studied and their conidial morphology

Isolates Type	Accession no. ^A	Host and locality of collection	Mean length (μm)	Mean breadth (μm)	Ratio of mean length to mean breadth	Range (μm)	Conidia rounded at both ends (%)
A	21499	<i>S. hamata</i> cv. Verano Katherine (N.T.)	15.05 \pm 0.135	4.53 \pm 0.058	3.32	12.1-24.2 \times 3.3-6.6	77
A	21461	<i>S. guianensis</i> cv. Endeavour Tully (Qld.)	17.48 \pm 0.157	4.49 \pm 0.033	3.89	13.2-22.0 \times 3.3-5.5	43
A	21415	<i>S. scabra</i> CPI40205 Mackay (Qld.)	16.10 \pm 0.157	4.32 \pm 0.039	3.73	12.6-19.2 \times 3.3-5.5	64
A	21364	<i>S. scabra</i> CPI49834 Townsville (Qld.)	16.42 \pm 0.147	4.37 \pm 0.050	3.76	12.1-22.0 \times 3.8-5.5	58
A	21365	<i>S. viscosa</i> CPI33941 Townsville (Qld.)	14.74 \pm 0.160	4.34 \pm 0.038	3.40	12.1-22.0 \times 3.3-5.5	43
A	21257	<i>S. fruticosa</i> CPI41118A Beerburum (Qld.)	14.43 \pm 0.180	4.50 \pm 0.051	3.21	11.0-22.0 \times 3.3-5.5	80
B	21423a	<i>S. guianensis</i> cv. Endeavour Samford (Qld.)	16.64 \pm 0.471	4.38 \pm 0.062	3.80	8.8-35.2 \times 3.3-5.5	24
B	21718	<i>S. guianensis</i> cv. Endeavour Tully (Qld.)	17.02 \pm 0.309	4.37 \pm 0.052	3.89	12.1-33.0 \times 3.3-5.5	21

^A Queensland Department of Primary Industries, Plant Pathology Branch accession numbers.

On the criteria of Von Ark (1957), type A and type B isolates key out to *C. gloeosporioides* Penz. Miss J. E. M. Mordue of the Commonwealth Mycological Institute has confirmed that conidia of the type B isolates (I.M.I.202550) are closer to *C. gloeosporioides* than to any other known species of *Colletotrichum*.

The perfect state of the type B isolates has not been observed either on the host or in pure culture. Sterile perithecia are commonly observed in pure culture with pathogenic type A isolates. *Glomerella cingulata* (Stonem.) Spauld. & V. Schreuk has been occasionally observed on field-infected plants with type A symptoms.

Morphology on the Host

The following fungal descriptions have been prepared from the examination of leaf sections cut 12 days after inoculation.

Type A isolate (21365). Acervuli on clearly defined lesions on leaves and stems, setose, erumpent, developing in a subcuticular or subepidermal position, rounded to elongated, range 81–162 μm , mean 123 μm (Fig. 8). Setae 3–4-celled, brown, swollen at base and tapered to a rounded or pointed apex, on which conidia are occasionally borne, range 64–88 by 3–6 μm , mean 73 by 4.5 μm . Conidia cylindrical, usually with both ends rounded, hyaline, aseptate, range 12–17 by 3.5–5.5 μm , mean 14.5 by 4.5 μm , formed on unicellular, hyaline, straight or curved, cylindrical, phialidic conidiophores, range 9–14 by 3–4 μm , mean 11 by 3.5 μm .

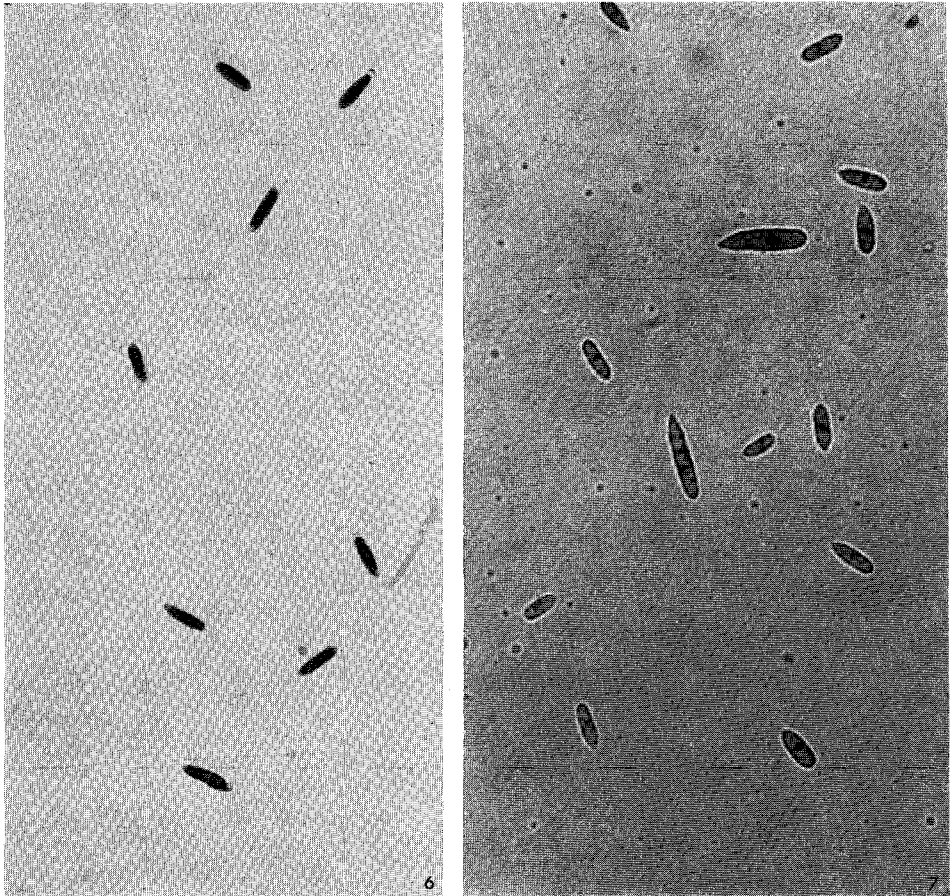


Fig. 6. Conidia of type A *C. gloeosporioides* ($\times 300$). These conidia have predominantly rounded ends and are relatively uniform in size.

Fig. 7. Conidia of type B *C. gloeosporioides* ($\times 300$). Note the high proportion of spores with one end acutely pointed and the variability in spore size.

Type B isolate (21423a). Acervuli on blighted, dark, necrotic areas of leaves and stems, setose, erumpent, developing in a subcuticular position, rounded to elongated, range 38–189 μm , mean 79 μm . Setae 2–3-celled, brown, swollen at base and slightly tapered to a rounded or pointed apex, on which conidia are usually borne, range 27.5–77 by 3.3–5.5 μm , mean 48 by 4.5 μm (Fig. 9). Conidia cylindrical, mostly with one end pointed, occasionally both ends rounded, hyaline, aseptate, range

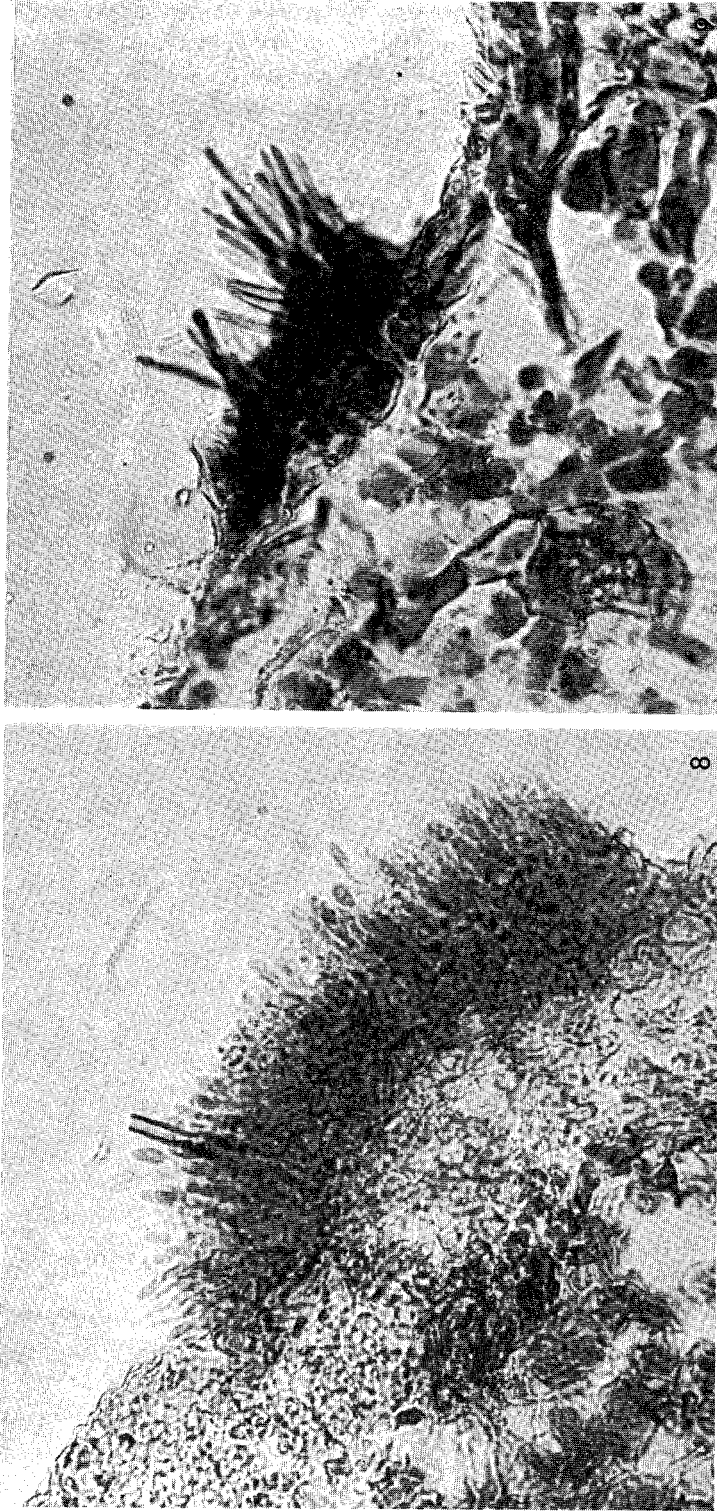


Fig. 8. Transverse section of an acervulus ($\times 360$) of the type A organism on *S. fruticosa* 41116, 12 days after inoculation.

Fig. 9. Transverse section of an acervulus ($\times 360$) of the type B organism on *S. guianensis* cv. Endeavour, 12 days after inoculation. Note a conidium produced on a seta and the smaller diameter of the type B acervulus compared with the type A acervulus shown in Fig. 7.

13–28 by $3.5\text{--}5.5\ \mu\text{m}$, mean 16 by $4.5\ \mu\text{m}$, formed on unicellular, hyaline, straight or curved, cylindrical, phialidic conidiophores range $10\text{--}17.5$ by $3\text{--}4\ \mu\text{m}$, mean 13 by $3.5\ \mu\text{m}$.

Growth–Temperature Relations

The five type A isolates (21364, 21365, 21415, 21461, 21499) showed a similar response over the entire experimental temperature range. For clarity, the results for only two type A isolates, 21415 and 21365 (representing the growth range for the five type A isolates), and the type B isolate 21423a are presented in Fig. 10. The cardinal temperatures for growth of all isolates were similar, but the type B isolate grew much less than any of the type A isolates over the temperature range of $18^{\circ}\text{--}33^{\circ}\text{C}$. Further comparison of 21365 and 21423a showed that the greater colony diameter of 21365 after 72 hr was due to the higher relative radial growth rate of 21365. In subsequent testing, the recently collected type B isolate 21718 also exhibited a similar growth–temperature response to that of 21423a.

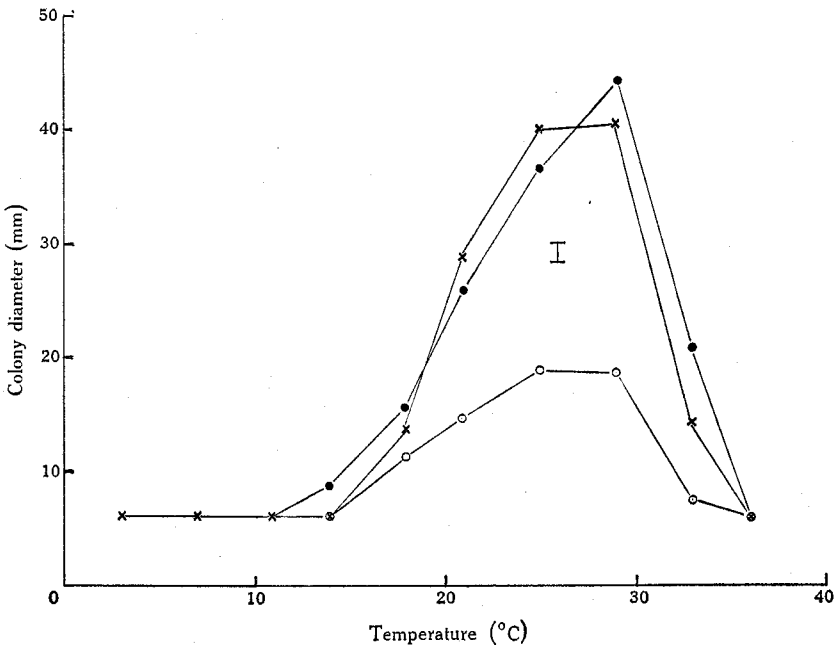


Fig. 10. Colony diameters of isolates of *C. gloeosporioides* 72 hr after incubating 6-mm discs at a range of temperatures. ●, 21415; ×, 21365; ○, 21423a. The bar represents LSD (5%).

Pathogenicity of *Colletotrichum* isolates and variation in susceptibility of *Stylosanthes* spp.

Pathogenicity of Isolates

The pathogenicity of 20 *Colletotrichum* isolates obtained at different sites from plants showing type A symptoms (type A isolates) and of two isolates obtained from plants at Samford and Tully showing type B symptoms (type B isolates) were tested on two *Stylosanthes* lines, *S. guianensis* cv. Endeavour and *S. fruticosa* CPI41116. The 20 type A isolates produced typical type A symptoms on all plants of 41116 and c. 20% of the plants of cv. Endeavour. Strain 41116 was severely affected, with

50% of the plants dead 12 days after inoculation. All the surviving plants showed coalescing stem and leaf lesions. The 20% of the Endeavour plants showing visible symptoms after type A inoculations were only slightly affected, and leaf and stem lesions remained as discrete spots no greater than 2 mm in diameter.

The two type B isolates killed all Endeavour plants within 10 days of inoculation. The symptoms were typical of those described for the type B disease. Strain 41116 showed no visible symptoms after inoculation with the type B isolates.

Differential Reaction of Type A and Type B Isolates to a Range of Stylosanthes spp.

For the seedling inoculations the means (over the two runs) of the leaf ratings for each line/isolate combination and of the stem ratings for each line are presented in Table 2. There was close agreement in the results from the two separate runs and no disease symptoms appeared on any of the control plants.

Table 2. Leaf disease ratings of 10 *Stylosanthes* accessions following inoculation of 4-6-week-old seedlings with seven isolates of *Colletotrichum gloeosporioides*

Values are the means of two runs

Type	Isolates Accession no.	<i>S. guianensis</i>					Lines				
		Cook	Endeavour	11497	<i>S. hamata</i> Verano	<i>S. humilis</i> Paterson	<i>S. viscosa</i> 33941	<i>S. viscosa</i> 34904	<i>S. scabra</i> 40205	<i>S. scabra</i> Seca	<i>S. fruticosa</i> 41116
A	21257	0.4	1.0	0.1	0.6	2.2	0.4	0.1	1.2	0	3.9
A	21364	0.2	0.6	0.1	1.2	3.3	0.1	0	1.8	0	4.1
A	21365	0.9	1.0	0.1	1.0	2.3	3.5	0	0.7	0	3.3
A	21415	0.3	1.0	0.1	0.9	3.0	0.2	0	2.2	0	3.7
A	21461	0.3	0.9	0.2	0.8	2.4	0.2	0	1.4	0	4.1
A	21499	0.1	0.9	0.1	1.0	4.2	0.1	0	1.7	0	4.3
B	21423a	1.3	4.9	0.2	0	0	0	0	0	0	0
Mean leaf rating		0.5	1.5	0.1	0.8	2.5	0.6	0	1.3	0	3.3
Mean stem rating		0.1	0.9	0.1	0.4	1.2	0.2	0	0.8	0	1.3

For leaf ratings, LSD ($P = 0.05$) (between line means) = 0.22; LSD ($P = 0.05$) (for interaction table) = 0.05.

For stem ratings, LSD ($P = 0.05$) (between line means) = 0.16.

The type B isolate, 21423a, produced disease symptoms on the three *S. guianensis* lines only, killing almost all the leaf tissue on Endeavour and damaging a few leaves on Cook and 11497 (Table 2). By contrast, the six type A isolates produced relatively minor symptoms on the three *S. guianensis* lines but disease symptoms on the other seven lines varied from nil to very severe and, with the exception of 21365 on *S. viscosa* 33941, were generally consistent for the six isolates. There was severe damage on *S. fruticosa* 41116, moderate to severe damage on *S. humilis* cv. Paterson, and moderate damage on *S. scabra* 40205. *S. scabra* cv. Seca and *S. viscosa* 34904 showed almost no visible symptoms. Disease symptoms on the remaining lines were generally restricted to a few small spots on leaves and stipules except for *S. viscosa* 33941 which was severely damaged by isolate 21365 only. This divergent response to the 21365 isolate suggested pathogenic race specialization within the type A organism.

Severe stem lesions were observed on those isolate/line combinations where leaf damage was very severe. However, only minor stem symptoms were observed on all other combinations, so that mean ratings on stems for all 10 lines were much

lower than the corresponding leaf ratings. Nevertheless, the overall ranking of lines for stem ratings was identical with that for leaf ratings (Table 2). In these small seedlings most of the stem tissue is enclosed by the sheaths of the leaf stipules, which may protect the stem from direct fungal attack.

Plants inoculated at 16 weeks of age had a relatively large surface area of exposed stem tissue and stem ratings were much higher than for the 4–6-week-old plants and of similar magnitude to the leaf ratings on both age groups. However, the ranking of lines and isolates was essentially the same for both age groups. Pathogenic race specialization of the type A organism was again apparent from the severe leaf and stem lesions produced by 21365 on 16-week-old plants of *S. viscosa* 33941.

Discussion

These studies have shown that two anthracnose diseases of *Stylosanthes* spp., readily distinguishable by their symptoms, are present in Queensland. The most prevalent disease, type A, has symptoms that correspond very closely with those reported by Sonoda *et al.* (1974) in Florida. In Queensland this disease has caused serious losses in commercial seed production crops of *S. humilis* and *S. hamata* cv. Verano. Although present in all commercial pastures of the *S. guianensis* cvv. Cook, Schofield and Endeavour, the type A disease causes only minor reductions in forage yields.

The type B disease has been found at only two sites in Queensland. However, the very severe symptoms produced by the type B fungus on Endeavour indicate that this disease could well pose a serious threat to the continued use of Endeavour in Queensland. Type B disease was first recorded on breeding lines of *S. guianensis*, including Endeavour, at Samford in March 1976, but was not found during an exhaustive survey of *S. guianensis* pastures in northern Queensland during April of that year. However, in May 1977 the disease was found in epiphytotic proportions in commercial seed-production crops of Schofield and Endeavour at Tully. These same crops had been free of the disease in April 1976.

The significance of both diseases to the future use of *Stylosanthes* spp. as the major pasture legumes in northern Queensland can not be underestimated. Already one introduction, *S. scabra* 40205, considered for commercial release in Australia, has been discarded because of its high susceptibility to anthracnose at a number of field sites in Queensland. Many other introductions, including lines of *S. fruticosa*, *S. viscosa*, *S. scabra*, *S. humilis* and *S. subsericea* have shown high susceptibility to the type A disease while undergoing field testing for agronomic characters at many sites in Queensland. The disease is not restricted to the wet coastal regions. Severe levels of the type A disease have been observed at inland sites with an annual rainfall of less than 700 mm. Glasshouse studies (Irwin, unpubl. data) have shown that both type A and type B isolates have a spore-generation period of 4 days on susceptible lines under conditions of high humidity and at temperatures of $25 \pm 2^\circ\text{C}$. This could explain the presence of high levels of the disease in relatively dry inland regions where the summer wet season would provide favourable conditions for infection and disease development.

The rapid spread of the type A disease throughout Queensland after 1973, when it was first recorded, could be due to the causal fungus being seed-borne. In Queensland (Irwin, unpubl. data), we have been able to isolate pathogenic type A *C. gloeosporioides*

from seed of *S. hamata* cv. Verano, and to observe seed transmission of the disease under uncontaminated glasshouse conditions. Ellis *et al.* (1976) in Colombia have also reported that pathogenic *C. gloeosporioides* isolates could be obtained from seed of *S. scabra*. Severe outbreaks of type A disease have occurred in seed-increase plots of *Stylosanthes* spp. at Beerburrum (southern Queensland) and Walkamin (Atherton Tableland). Seed harvested from these plots has subsequently been distributed throughout Queensland for agronomic testing. This could well explain the rapid spread of the disease since 1973.

Comparative morphological and physiological studies made on type A and type B isolates have shown that the two types can be distinguished by the characters of spore shape and length, colony colour, and radial growth rate in pure culture within the temperature range 20–30°C. However, there is considerable variability in spore morphology so that the colour and radial growth rate of cultures are the best criteria to separate the two types of *C. gloeosporioides* isolates.

The existence of two pathogenic races within the six type A isolates tested has been demonstrated by their disease reaction on *S. viscosa* 33941. Only isolate 21365 produced severe disease symptoms on this line. This isolate was obtained from severely diseased plants of 33941 at Townsville which did not show susceptibility until 18 months after sowing, even though lines planted concurrently in the same trial, such as *S. fruticosa* and *S. subsericea*, showed severe symptoms within 6 months of sowing (C. J. Gardner, personal communication). The situation at Townsville was ideal for the establishment of new pathogenic races as described by Van der Plank (1968): a large, susceptible population producing large quantities of inoculum, with an extensive screen of 33941 alongside to filter out the components of the pathogen population to which the resistance was not effective.

The glasshouse tests have shown the availability in some lines of high levels of resistance to type A and type B forms of *C. gloeosporioides*. Whether this resistance will remain stable must be questionable, in view of the breakdown of resistance in 33941. Further work is warranted to determine the number of genes that confer resistance in lines such as *S. scabra* cv. Seca, which is immune to all known types and races of *C. gloeosporioides*. If immunity is conferred by a single or small number of genes, then this form of resistance may prove to be transient in nature (Van der Plank 1968).

Future work should also be directed towards screening all available accessions of *Stylosanthes* spp. with a diverse range of types and isolates of *C. gloeosporioides* under controlled environmental conditions. When the resistance status of the accessions has been determined in this manner, divergent field reactions produced by new races of the fungus should be readily detected by careful monitoring of agronomic strain trials. With a clearer understanding of the pathogenic variability that is present in *C. gloeosporioides* types and of the modes of inheritance of resistance, a breeding program can then be put on a firmer basis than at present.

Acknowledgments

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