J. C. Tu
Harrow Research Station, Agriculture Canada, Harrow, Ont.

Control of Bean Anthracnose Caused by the Delta and Lambda Races of *Colletotrichum lindemuthianum* in Canada

In Canada, dry beans (*Phaseolus vulgaris* L.) are produced in an area of southwestern Ontario centered on and within 60 km of Exeter. In 1985, the approximately 44,000 ha planted to dry beans produced 66,000 t, of which 75% was exported. At an average price of $650 U.S. per ton, the crop was worth about $43 million U.S. (1).

Bean anthracnose caused by *Colletotrichum lindemuthianum* (Sacc. & Magn.) Briosi & Cav. had not been of practical importance in Canada and the United States from 1955 to 1976 because most of the bean cultivars, with the exception of some colored beans, were bred for resistance to the prevalent races (alpha, beta, and gamma) of the fungus. An outbreak of anthracnose occurred in southwestern Ontario in 1976 (22). By 1977, 18% of 4,000 ha of select, foundation, and certified seed of the four recommended cultivars—Fleetwood, Kentwood, Seafarer, and Sanilac—had varying degrees of anthracnose infection (18). The disease affected yield, seed quality, and marketability of beans. The causal organism was identified as the delta race of *C. lindemuthianum*; identification was confirmed by N. Hubbeling of the Netherlands. Later, the lambda race was also found (23). Since none of the recommended cultivars at that time were resistant to the delta and lambda races, the sudden development of an epidemic was not surprising.

It is surprising, however, that the disease was not detected earlier, since a three-stage pedigreed seed program was in force at that time. All select, foundation, and certified seed fields were subject to inspection for common bacterial blight (*Xanthomonas campestris* pv. *phaseoli* (Smith) Dye). Judging from the extent of spread, the disease could have been present in Ontario for 3 years or more before the 1976 outbreak.

How and from where the new races were introduced into Canada and how they spread to the various seed growers' fields remain unresolved. The delta and lambda races might have been brought into Canada in diseased seeds of some novelty bean cultivar for home gardening and inadvertently spread from a family garden to the nearest select plot. The seeds from the diseased select field sent to seed dealers for cleaning and bagging could have contaminated the dealers' equipment and spread the pathogen to other seed growers' beans that required cleaning and bagging.

The disease reached epidemic proportions by 1978. In addition to the yield loss

Table 1. Disease reactions[a] of two groups of Ontario isolates of *Colletotrichum lindemuthianum* on a series of differential bean cultivars compared with reactions of known races of the pathogen

	Isolate groups		Races						
Cultivar	A	B	Epsilon	Alpha	Beta	Gamma	Delta	Kappa	Lambda
Dark Red Kidney	S	S	R	R	S	S	S	S	S
Widusa	S	S	R	S	R	R	S	S	S
Kaboon	R	S	R	R	R	S	R	R	S
Michelite	S	S	S	S	R	R	S	S	S
Sanilac	S	S	R	R	R	R	S	S	S
Prelude	S	S	S	S	R	R	S	S	S
Cornell 49-242	R	R	R	R	R	R	R	S	R

[a] Differential host reactions based on Hubbeling (10) and Krüger et al (12). S = susceptible, R = resistant.

caused by the disease, further problems resulted when the state of Michigan proclaimed a disease quarantine against beans from Canada. The ban was not lifted until 1982, after the disease had been brought under complete control. This article details how the epidemic was quickly and successfully managed.

Etiology

Bean anthracnose has been known to cause serious losses on susceptible cultivars and was once considered to be the most important disease of beans worldwide. The pathogen has several distinct races that exist in different regions of the world (24). In Canada and the United States, the prevalent races have been alpha, beta, and gamma and the majority of cultivars for commercial plantings have been bred for resistance to these races (24). Under these circumstances, it is reasonable to suspect that a new race (or new races) could incite an epidemic.

After the epidemic in 1977, many isolations were made from anthracnose lesions from diseased plants. All isolates were initially cultured on Mathur's agar (7) amended with 40 μg per milliliter of novobiocin to retard bacterial growth. After 4 days, colonies characteristic of *C. lindemuthianum* were transferred to new plates of Mathur's agar, and after 14–21 days, spore suspensions (10^7 spores ml^{-1}) were prepared from each isolate for inoculation (18). Race determination involved inoculating each isolate to a series of differential cultivars (Table 1) following the differential schemes of Hubbeling (10) and Krüger et al (12). The results showed that all the isolates were of two groups that corresponded to the delta and lambda races (Table 1).

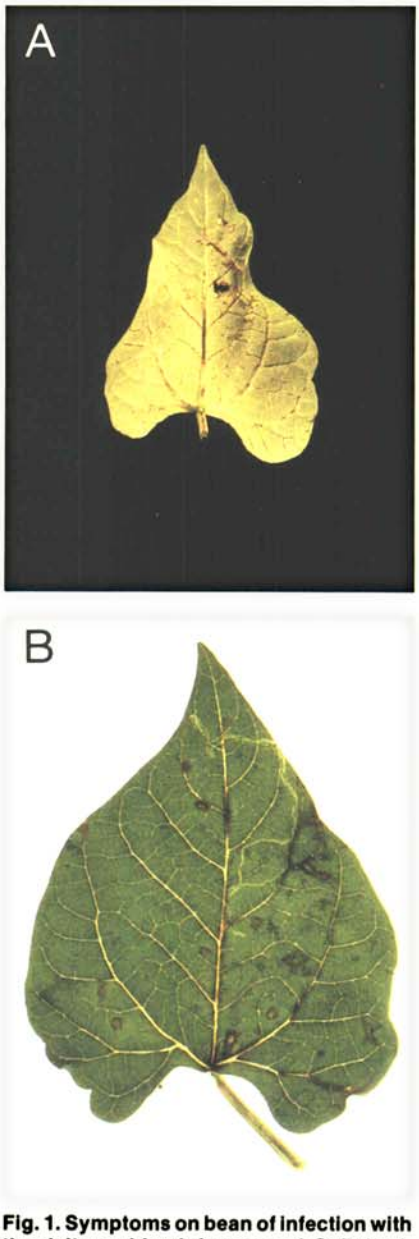

Fig. 1. Symptoms on bean of infection with the delta and lambda races of *Colletotrichum lindemuthianum*. Discoloration appears (A) early on the lower leaf surface and (B) later on the upper surface, accompanied by brown lesions.

Fig. 2. Rusty brown spots on pods of anthracnose-diseased white bean.

Fig. 3. Seeds from bean pods heavily infected with anthracnose.

Symptoms

The symptoms caused by the delta and lambda races are identical to those caused by other races of the pathogen. Although infection may occur on both sides of the leaf and on the petiole, early symptoms of infection usually appear on the lower leaf surface along the veins, which show brick red to purplish red discoloration (Fig. 1A). Later, such discoloration appears on the upper leaf surface and brown lesions of various sizes, with black, brown, or purplish red margins, develop around small veins (Fig. 1B).

Dark brown eyespots that develop longitudinally along the stems are an early symptom of stem infection. On young seedlings, the eyespots enlarge and the stem may break off. On older stems, the eye-shaped lesion is approximately 5–7 mm long and often has a sunken, cankerous center.

The most striking symptoms are the rusty brown spots with small brown specks that appear on the pods. As the lesions enlarge, their centers turn brown and many tiny black acervuli appear randomly on the brown area, replacing the brown specks. Each acervulus contains a mass of pinkish spores, often visible as a viscous droplet in humid conditions. The lesions on the pod usually reach a diameter of 5–8 mm, are slightly sunken at the center, and have a dark brown or purplish brown margin (Fig. 2).

The seeds obtained from heavily infected pods may show brown to light chocolate spots on the seed coats (Fig. 3). In badly infected seeds, the lesions may extend into the cotyledon.

Epidemiology

The initial infection comes from fungus propagules that are carried in the seed or, to a lesser extent, in dry bean straw, though not in wet or buried straw. The seedborne nature of the bean anthracnose pathogen was first demonstrated by Barrus (4). I (17) showed that longevity of the fungus varied greatly depending on environmental conditions. Moisture had a profound effect on its longevity. The fungus survived at least 5 years in infected pods and seeds of *P. vulgaris* that were air-dried and stored at 4 C. In southern Ontario, *C. lindemuthianum* could overwinter in the field in infected plant materials kept dry in sealed polyethylene envelopes, but if the infected materials were placed in nylon-mesh pouches and buried in the field in November, the fungus could not be isolated after mid-May (17). Beans planted on sites with heavily infected crops the previous year failed to develop anthracnose (17). Laboratory tests showed that an alternating wet-dry cycle was detrimental to survival of the fungus

(17). The fungus in the infected pod segments lost viability after three cycles of 72 hours wet, 72 hours dry.

After the initial infection, the disease is spread by spores carried in splashing raindrops and by people or machines that come in contact with the diseased plants. In southern Ontario, rain-splash dispersal of the delta race from an initial infection focus in field plots of a susceptible white bean cultivar was rapid. Long-distance spread, 3–4.6 m per rainstorm, was caused by splashing raindrops blown by gusting winds. Spread of disease from the infection focus toward the northeastern quarter of the plot followed the direction of prevailing winds. Disease severity was highest at or near the initial infection focus and decreased gradually toward the periphery of the disease area. The fungus required about 10 mm of rain to establish initial infections (15).

Prevailing field temperatures did not limit infection and spread of bean anthracnose in the area. With sufficient precipitation, the disease spread readily despite high daytime temperatures (25–35 C) between mid-July and mid-August. The low night temperatures appeared to play a role in mitigating the effect of high daytime temperatures by providing environmental conditions conducive for infection and disease development. Infected plants kept under constant high temperatures (28–32 C) in growth chambers showed only restricted disease development (16).

Control Measures

A two-tier disease management program of short-term and long-term measures was developed. The short-term program included intensified pedigreed seed inspection, chemical seed treatment, and crop rotation. For the long term, a backcross breeding program was initiated to incorporate genetic resistance into the recommended cultivars in use at that time.

Pedigreed seed inspection. Beginning in 1977, intensified pedigreed seed inspections were conducted each year in the second and third weeks of August by qualified plant inspectors. All pedigreed seed fields that included select, foundation, and certified bean seed were inspected. Inspection included visual observation for any symptoms of anthracnose in the field. Inspectors walked through the field at four-row intervals. Plants with definite or suspected symptoms were collected and examined in the laboratory. In some cases, isolations were made to confirm field observations. A zero tolerance was set for all seed fields, and seed fields with any anthracnose were downgraded and designated for consumption only, as either food or animal feed. This action created a pedigreed seed shortage for the 1978 growing season, but all the available pedigreed seed was anthracnose-free. To make up for the shortfall of pedigreed seed for the 1978 crop and to discourage the use of infected commercial seed, seed from areas where anthracnose did not occur was identified, inspected, treated, and offered for sale as pedigreed seed.

The seed inspection appeared to be an effective means of reducing anthracnose incidence. The number of seed fields that developed anthracnose was reduced from 18% in 1977 to 0% in 1981 (Table 2). Undoubtedly, seed inspection played an important role in disease control and was a major factor contributing to the lifting of Michigan's ban on Canadian beans in 1982.

Seed treatment. As an additional precaution, seed treatment was required for all seed to be used in commercial plantings. Seed was treated with one of the two products given a temporary registration in 1978. These were IF Plus (captan 25%, diazinon 12.5%, benomyl 17.5%) and DCT (diazinon 6%, captan 18%, thiophanate-methyl 14%) (8). In 1981, DCT (Ciba-Geigy Canada Ltd.) was permanently registered. The diazinon was for root maggots, the captan was for seedling rot and seedling blight, and the thiophanate-methyl was for anthracnose.

The effectiveness of seed treatment was about 95% (9), indicating that some deep-seated infection might escape and survive the treatment. Edgington and French (8) showed that the fungus isolated from diseased plants was not resistant to benzimidazole fungicides, although the fungus was known to easily develop tolerance to benomyl (19,20). Growth room tests with beans having slight, moderate, or severe seed lesions proved that good control of anthracnose could be achieved only with slight and moderate lesions. Apparently, the benzimidazoles are not sufficiently systemic in necrotic tissue. Thus, seed with severe lesions should not be used even though treated with benzimidazole fungicides.

Crop rotation. Fields that had anthracnose were removed from bean cultivation for 3 years. A 3-year rotation was recommended on the basis of published reports that suggested the fungus could survive 2 years in dry debris and in seed (14,24). The 3-year rotation was later found to be unnecessary, however, because the fungus could not survive under natural conditions in the field in Ontario. In addition, disease spread was unlikely provided infected dry debris was removed and contaminated machines stored under dry conditions were thoroughly decontaminated before being introduced into the field.

Breeding program. A backcross breeding program was initiated at Harrow to incorporate genetic resistance to the delta and lambda races into the recommended cultivars. The program involved the transfer of the "ARE" gene, a single dominant gene reported by Mastenbrock (13) that confers resistance to the alpha, beta, and gamma races as well as the delta and lambda races; the gene is from PI 326.418 (Cornell 49-242), a cultivar derived from Corbett Refugee (21). This was incorporated into the recommended cultivars Fleetwood, Kentwood, Seafarer, and ExRico 23 as well as into many breeding lines. PI 326.418 also carries the "I" gene, which confers resistance to bean common mosaic virus (BCMV) strains 1 and 15, both of which are prevalent in Ontario.

All backcrossing and advancing of generations were conducted in the greenhouse during the winter and spring. Each of the cultivars—Fleetwood, Kentwood, Seafarer, and ExRico 23—was crossed with PI 326.418 during the winter of 1976–1977. F_1 seeds were increased to yield F_2 seeds. F_2 plants were screened for resistance to the delta race and to BCMV strains 1 and 15. Resistant F_2 plants were used in making the first backcross. Subsequent backcrosses were made with F_1 plants.

An accurate and rapid assay method was needed to screen the backcross progenies. Two were available at that time—a dip method and a spray method (11,12). The dip method appeared to be too severe for the young germinating seedlings, and a precise differential response between a susceptible and a resistant reaction was sometimes difficult to obtain. The spray method tended to produce variable results in which many susceptible plants escaped infection. A new method of screening was therefore needed.

An effective brushing method was developed (18). An aqueous spore

Table 2. Summary of field inspections for bean anthracnose (1977–1981)

Year	Number of fields		Hectarage inspected (no.)	Hectarage diseased (%)
	Inspected	Diseased		
1977	323	64	2,598	18.00
1978	189	1	2,188	0.91
1979	210	5	2,407	2.33
1980	181	1	2,399	0.34
1981	227	0	2,837	0

suspension (10^7 spores per milliliter) harvested from a 3-week-old culture on Mathur's agar was gently applied to the upper and lower surfaces of the primary leaves with a camel's-hair brush. The inoculated seedlings were covered with transparent plastic bags and incubated for 2 days at 20 C with 14 hours of light per day, at 280 $\mu Em^{-2}sec^{-1}$. After removal of the plastic bags, the inoculated plants were kept at the same conditions for 4 days for symptom development.

Although inoculation by brushing a spore suspension onto the leaves was tedious, the consistency and uniformity of the results far outweighed the tedium. Accuracy and promptness in differentiating susceptible from resistant progeny is most desirable to reduce the amount of work, facilities, and time required in a breeding program.

The results of backcrossing and screening have enabled the selection of homozygous "ARE" plants in the progenies of the sixth backcross (BC_6). The bulked Fleetwood-BC_6, Kentwood-BC_6, Seafarer-BC_6, and ExRico 23-BC_6 were designated as Harofleet, Harokent, OAC Seaforth, and OAC Rico, respectively. They were entered in Ontario field bean variety trials in seven locations in 1981 and 1982, new cultivars were licensed (2,3,5,6), and breeder seed was released to seed growers in 1983.

J. C. Tu

Dr. Tu is a research plant pathologist with Agriculture Canada at the Harrow Research Station in Ontario. He obtained his B.Sc. and M.S. degrees from National Taiwan University and his Ph.D. degree in 1966 from Washington State University at Pullman. He investigates diseases of white beans, soybeans, and peas, and his current interests center on the epidemiology, etiology, and integrated pest management of these crops.

It must be noted that the "ARE" gene confers resistance to the alpha, beta, gamma, delta, lambda, and epsilon races but not to the kappa (Ebnet) race (10). The "ARE" gene has been considered to be of a "uniform" or "horizontal" type because it is active against many races of the fungus. However, the vulnerability of this resistance to the kappa race suggests that this gene may give only a "vertical" race-specific resistance (12). Fortunately, the kappa race is not found in North America. Nevertheless, a gene that confers resistance to the kappa race is present in the cultivar Kaboon (10). Thus, if the kappa race were inadvertently introduced into Canada in the future, a breeding program could be instituted easily because many of the technical problems associated with backcross breeding, including backcrossing and advancing of generations and screening for anthracnose and virus resistance, have been worked out.

Conclusions

Control of bean anthracnose by breeding for disease resistance is a costly and time-consuming proposition. For the Ontario dry bean industry, however, a breeding program is easily justified because only four or five cultivars are in use and the large hectarage of dry bean is concentrated in a relatively small area. As for snap beans and colored dry beans, both of which have numerous and limited hectarage, anthracnose can be kept to a manageable level by using disease-free seeds produced in semiarid areas or from inspected pedigreed seed fields. Seed treatment with effective fungicide formulation should reduce the disease to a minimum, and a breeding program therefore may not be necessary.

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