Phytophthora blight of pigeonpea: present status and future priorities†

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Abstract. Phytophthora blight caused by *Phytophthora drechsleri* f. sp. *cajani* is a major production constraint to pigeonpea in India. The disease has also been reported from other countries. It was first reported in 1966 from India and is currently considered to be more important in short-duration pigeonpeas than in traditional medium- and long-duration types. Little work has been done on disease epidemiology and management. Screening methods have been standardized and work on host—pathogen interaction indicates that the blight pathogen is variable and that high and stable sources of resistance have not so far been located in the cultivated germplasm. We present a critical analysis of research conducted on this disease and strategies for disease management and future research priorities are suggested.

1. Introduction

Pigeonpea [Cajanus cajani (L.) Millsp.] is an important grain legume crop of rain-fed agriculture in south Asia, east Africa, and central America (Nene and Sheila, 1990). It provides protein to the diet and also contributes to the sustainability of cropping systems and rotations. The stems can be used as fuel, and the dried leaves and husk are used as animal feed. Williams et al. (1968) first recognized phytophthora blight (PB) caused by Phytophthora drechsleri f. sp. cajani (Pal et al.) Kannaiyan et al. as an important disease of pigeonpea. Information on the pathogen, its epidemiology and disease management is comparatively limited. Though the disease has been reported from several countries (Nene et al., 1989), precise information on its distribution and severity is lacking. The disease is relatively more serious in short-duration (3-4) months) pigeonpeas than in traditionally cultivated medium- and long-duration types (5-10 months). Shortduration pigeonpeas have great potential to increase grain yield and to extend the adaptation of the crop to non-traditional areas but management of blight is essential for the realization of their potential yields. The purpose of this paper is to assess the work done on this disease so far, identify the gaps in knowledge, and indicate further areas for future research.

2. Disease surveys and crop losses

Surveys carried out in India between 1975 and 1980 indicated that PB is prevalent in the states of Andhra Pradesh, Bihar, Orissa, Uttar Pradesh and West Bengal, but not in the states of Gujarat, Karnataka, Madhya

Pradesh, Maharasthra, Rajasthan and Tamil Nadu (Kannaiyan *et al.*, 1984). This information was based on observations on disease incidence in the traditionally grown medium- and long-duration pigeonpeas in which blight was not considered to be a serious problem. The disease gained in importance from the early 1980s with the introduction of short-duration pigeonpeas. Total yield loss has been observed in some short-duration pigeonpea crops in southern India. There is a need for a further comprehensive survey of pigeonpeas, especially in the short duration pigeonpea-growing areas in north-western India, to assess the distribution and severity of PB and to estimate crop losses. There is also a need to assess the importance of PB, especially in countries where short-duration pigeonpeas have potential.

3. Causal fungus

Based on morphological studies carried out at the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) Center, Patancheru, India and the University of California, Riverside, USA, and on host specificity, the fungal pathogen was designated as *P. drechsleri* f. sp. *cajani* (Pdc) (Kannaiyan *et al.*, 1980). Ho and Jong (1991) studied the taxonomy of various *Phytophthora* species and redefined *P. drechsleri* to accommodate only those isolates that grow well at 35°C, with the pigeonpea blight pathogen treated as conspecific.

The pathogen so far has been reported from the Dominican Republic, India, Kenya, Panama and Puerto Rico (Nene *et al.*, 1989). In India, the fungus affects the collar region and all above-ground parts of the plant (Figures 1–3). In Australia, *P. drechsleri* has been reported to cause a serious root rot in addition to chlorosis and lesions on stems of pigeonpea (Wearing and Birch, 1988); however, the host specificity has not been reported. The identity of the pathogen from other pigeonpea-growing countries needs to be confirmed.

The morphology and disease cycle of the blight pathogen have been described in detail (Singh and Chauhan, 1992). Although the disease can be identified by the symptoms produced, several workers have encountered difficulties in isolating Pdc. The fungus can be isolated on dehydrated potato dextrose agar medium after surface sterilization of the infected tissues with 0·1% mercuric chloride for 30 s. Bisht and Nene (1988) formulated a selective medium to isolate the fungus based on a mixture

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Figure 1. Pigeonpea plants killed by phytophthora blight in a field.



Figure 2. Discontinuous lesions caused by Phytophthora drechsleri f. sp. cajani on stems of pigeonpea.

of antimicrobial agents used with potato dextrose agar. Pscheidt *et al.* (1992) evaluated the sensitivity of a *Phytophthora*-specific monoclonal antibody-based immunoassay kit on 17 species of *Phytophthora*, including *P. drechsleri*. All the *Phytophthora* species tested produced a positive reaction. A similar kit could be developed specifically for the pigeonpea blight pathogen. This would



Figure 3. Blight lesions caused by Phytophthora drechsleri f. sp. cajani on a pigeonpea leaf.

be a useful diagnostic tool, especially when the diseased plants are old or dried or have rotted and are thus in a poor condition for isolation of *Phytophthora*.

4. Biology and ecology

4.1. Variability in the pathogen

The blight pathogen appears to be highly variable, as experienced from research for the past 15 years at ICRISAT Center. When research on phytophthora blight was initiated at ICRISAT in 1976, evaluation for resistance was carried out with the P2 isolate prevalent at that time in the ICRISAT fields. Several pigeonpea lines such as ICP 2376 and ICP 7065 were found to be resistant to the P2 isolate (Kannaiyan et al., 1981). In subsequent tests they were found to be susceptible to the disease in the same field. The pathogen isolated from such diseased plants was found to be more virulent than P2 and was designated as P3 isolate. At present there is no pigeonpea genotype with a high level of resistance to P3 isolate. Studies with other isolates collected from different locations in India confirmed that the fungus is pathogenically highly variable (Nene et al., 1991). Precise studies to identify the different races using host-plant differentials are needed. Host/ disease reaction in the form of lesion type and size may be a better criterion than disease incidence for the differentiation of races. Based on disease incidence, the studies carried out so far indicate that P2 and P3 isolates can be distinguished on the basis that ICP 7119 was killed by both the isolates (100% blight incidence and mortality) whereas ICP 2376 showed resistance to P2 (0% blight incidence) but not to P3 (100% blight incidence and mortality). Morphological variation also exists among the Indian isolates of Pdc (Kannaiyan *et al.*, 1980; Nene *et al.*, 1992).

4.2. Host range

In experimental plots at ICRISAT Center, natural infection was observed not only on pigeonpea but also on wild

Cajanus spp. The fungus (P3 isolate) attacked 13 wild Cajanus spp. when a few accessions were tested in potscreening tests (Sarkar et al., 1991). However, none of 55 plant species other than Cajanus spp. tested were either hosts or symptomless carriers of Pdc (Sarkar et al., 1991). The wild Cajanus spp., especially C. scarabaeoides var. scarabaeoides which is a common perennial weed, may serve as alternative hosts of the fungus. Further research is needed to confirm this.

4.3. Survival and dispersal

The fungus survives in soil, even in the absence of a living host, and also in infected crop debris for at least 1 year (Bisht and Nene, 1990). Sarkar (1988) reported that Pdc survives in the form of chlamydospores in field soil and diseased stubble. Singh and Chauhan (1992) observed that a living host is not essential for oospore formation, but that temperature is critical and oospores are produced only at 25°C. The role of chlamydospores and oospores in survival and perpetuation of the fungus needs to be elucidated. This is important because the disease suddenly appears, in a severe form, in fields where pigeonpea has not been cultivated for several years. The fungus is soil- and water-borne, but not seed-borne; splashing rain and wind also contribute to short distance dispersal of the zoospore inoculum (Bisht and Nene, 1990).

5. Epidemiology

Disease development is influenced by inoculum density and by several environmental factors. Agrawal and Khare (1987) reported that the maximum infection index (49·16%) was observed at 28·12 mm day⁻¹ rainfall, 100% rainy days, 27.45°C maximum temperature, 21.41°C minimum temperature, and 92.4% relative humidity; 6.52% infection index was observed at 9.44 mm day⁻¹ rainfall, 30% rainy days, 31.70°C maximum temperature, 22.71°C minimum temperature, and 84·1% relative humidity. They inferred that infection index was positively correlated with rainfall (mm day 1), rainy days (%) and relative humidity (%), and that number of rainy days (%) was more important than the amount of rain. Chauhan and Singh (1991a) observed that light inhibited zoospore germination; they obtained maximum germination in darkness. Singh and Chauhan (1992) further reported that light and darkness affected disease development; lesion size increased more rapidly in darkness than with continuous light. Rainfall, maximum temperature, and sunshine influenced blight infection and disease development; outbreaks occurred when day temperatures were less than 28°C and were accompanied by rainy and cloudy weather (Reddy et al., 1992). The increase in inoculum level and blight incidence was associated with a decrease in day temperature, and with high rainfall and cloudy weather (Reddy et al., 1992). Recent work carried out under controlled conditions in the glasshouse and laboratory at ICRISAT Center showed that duration of leaf wetness is more critical for pathogen

infection than temperature and inoculum load. A leaf wetness period of 12 h was necessary for infection. Infection occurred between 10 and 35°C.

6. Strategies for blight control

Pigeonpea is mainly cultivated by resource-poor farmers on marginal lands with minimal inputs. At present yield levels (700 kg ha⁻¹), management of the disease through the use of expensive fungicides such as foliar spray applications of metalaxyl would not be economical. The ideal way to manage the disease would be to grow diseaseresistant cultivars (Figure 4). However, the cultivated germplasm does not have high levels of resistance to the two widely prevailing isolates (P2 and P3) of the fungus, especially when the disease occurs in the first 2-3 weeks after sowing. Some accessions of the wild species C. platycarpus have high levels of resistance to the disease, but they are not yet able to be crossed with pigeonpea. Until higher levels of resistance to blight in cultivated pigeonpeas are developed, the best option for management of the disease will be through the combined use of adult plant resistance, fungicides, and land management. As the pigeonpea plant is susceptible to blight only up to 45 days, seed dressing with fungicides such as metalaxyl can be considered to protect the crop during the early stages. Providing better drainage in the field is useful in reducing disease severity. Future research can be intensified in enhancing resistance to blight in pigeonpea by making use of the resistance in C. platycarpus through introgression into the cultivated germplasm.



Figure 4. A pigeonpea line showing moderate resistance to phytophthora blight (right), and a susceptible line (left).

6.1. Host plant-resistance

6.1.1. Screening techniques. Although considerable work has been done on standardization of glasshouse and field inoculation techniques for evaluation of pigeonpeas for blight resistance (Reddy et al., 1990), it is still difficult to obtain uniform disease incidence in the field, and under greenhouse conditions. Establishment of uniform humidity after inoculation seems to be critical, but is difficult to simulate under field conditions. Using a well-levelled field with bunds erected at close intervals to cause temporary water inundation within the first 2–3 weeks after sowing may help in obtaining high and uniform blight infection.

Shohet and Strange (1989) detected a toxic component in culture filtrates of P3 and suggested that it could be of potential use as a selection tool *in vitro*; the toxin could be used in screening for insensitive lines, from which disease-resistant plants could be regenerated.

6.1.2. Crop duration, plant age, and blight susceptibility. Evaluation of the world collection of pigeonpea germplasm of more than 15 000 accessions at ICRISAT Center against the prevailing isolates of the fungus, failed to identify any source of high-level resistance to blight. All the short-duration accessions were highly susceptible. The reasons for this are not clear. It is possible that the short-duration types are genetically more susceptible to the disease than the medium- and long-duration types. Another reason could be that the high plant populations used for short-duration types results in fast ground cover providing a more favourable environment for blight development. Young plants are more susceptible to the disease than older plants (Sarkar et al., 1992) and young tissues are more susceptible than older tissues. The chances of inoculum (zoospores) from soil being disseminated by splashing onto younger tissues are greater in the short-duration types, because of their low plant height, than in the medium- and long-duration types. Thus the high susceptibility of the short-duration types to blight could be a result of their short plant stature.

A few medium- and long-duration lines such as KPBR 80–2 with field resistance to P2 and P3 isolates have been identified (Reddy *et al.*, 1991). However, even these lines were susceptible in the field when the disease occurred within 2 weeks after sowing. The adult plant resistance of these lines could be caused by development of resistance with increase in plant age. The reasons for higher susceptibility of the younger tissues of pigeonpea to PB than the older tissues remain to be understood. It is common to find mortality of plants in the field caused by PB at later stages of crop growth (>60 days old) when conditions for disease development remain favourable. This mortality may be a result of disease progress from lesions produced during early infections and not to fresh infections.

6.1.3. Genetics of resistance. Information on the genetics of resistance to PB is limited. Resistance in ICP 7065 and ICP 2376 against the P2 isolate was found to be controlled by a single dominant gene (Sharma *et al.*, 1982). Information on the genetics of resistance to the P2 isolate in other lines is not available. Furthermore, there is

no information available on the genetics of adult plant resistance to the P3 isolate. This information is essential to the formulation of future disease-resistance breeding strategies, including introgression, to enhance blight resistance in pigeonpea.

6.1.4. Mechanisms of host resistance. Kaur and Mehrotra (1990) analysed leaf extracts of PB-resistant (ICP 28) and PB-susceptible (ICP 7119) cultivars and observed quantitative and qualitative differences in amino acids, organic acids, sugars and phenols. Resistant lines had higher levels of organic acids, rhamnose and phenols than the susceptible lines. Glucose, fructose and raffinose were more abundant in leaves of susceptible plants than in leaves of resistant plants; salicylic acid was absent from leaves of the susceptible cultivar (Kaur and Mehrotra, 1990).

Histopathology should provide an insight into determining the sequence of infection, and understanding the host-pathogen interaction and mechanisms of field resistance.

6.2. Cultural control

Chauhan and Singh (1991b) reported that a weed canopy interfered with splash dispersal of Pdc from soil to aerial parts of pigeonpea plants and thus reduced PB intensity. They suggested that pigeonpea yields might be increased by mulching or by interplanting with dwarf leguminous crops such as mung bean [Vigna radiata (L.) Wilczek] and urd bean [Vigna mungo (L.) Hepper] to check PB incidence. However, at ICRISAT Center no significant differences in PB incidence were observed between weeded and weed-infested pigeonpea plots.

Soil solarization is effective in controlling *Phytophthora* spp. of some crops and may be useful in reducing PB in experimental plots of pigeonpea.

6.3. Chemical control

Kannaiyan and Nene (1984) reported that seed dressing with metalaxyl controlled PB in greenhouse trials but not in field tests. However, Bisht *et al.* (1988) found metalaxyl to be effective when used as a foliar spray alone, or in combination with seed dressing. The antithrombotic compound ajeone, isolated from garlic (*Allium sativum* L.), has inhibitory effects on Pdc growth and reproduction (Singh *et al.*, 1992). It has also been suggested that ajeone may be an effective chemical for control of the disease under field conditions if applied at low concentrations before zoospore formation (Singh *et al.*, 1992). However, this requires further experimentation and an evaluation of the economies and safety of application.

7. Future priorities

The most apparent gaps in knowledge of PB of pigeonpea are in the areas of disease epidemiology and hostplant resistance. The mode and duration of survival of the fungus in soil need to be understood further. Reasons for occurrence of the disease in epiphytotic form in fields where pigeonpea has not been grown for 5–10 years need to be found. More work is needed on the symptomatology of the disease in different countries, and on the pathogenicity and taxonomy of the fungus. Research on further understanding of variability of the blight fungus is required. To develop pigeonpea lines with a high level of resistance to PB, resistance identified in *C. platycarpus* needs exploitation. Also, further work on integrated disease management through land and water management to provide better drainage, development of seed dressing fungicides of greater efficacy than Ridomil® (metalaxyl), cheap foliar fungicides and elucidation of cropping patterns/rotations that inhibit PB pathogen population need to be carried out.

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