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Review

Phytophthora blight of Pigeonpea [*Cajanus cajan* (L.) Millsp.]: An updating review of biology, pathogenicity and disease management

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ABSTRACT

Phytophthora blight (PB), caused by *Phytophthora drechsleri* f. sp. *cajani*, is reoccurring as an economically important disease of pigeonpea (*Cajanus cajan*), especially when excessive rains fall within a short span of time and hot and humid weather persists during the crop season. A few years after the initial reviews of Kannaiyan et al. (1984), the disease was coming to halt. Despite earlier investigations on pathological and physiological characteristics of *P. drechsleri* f. sp. *cajani*, the nature of infection process and genetic basis of pathogen variability have not been clearly established. Therefore, information on the biology and survival of the pathogen is needed to devise effective management strategies. Attempts have been made to develop green-house and field screening techniques since three decades ago for identification of host plant resistance. However, only few pigeonpea germplasm and breeding lines belonging to cultivated and wild *Cajanus* spp. were found tolerant to PB. The recent frequent recurrence of PB epidemics in the major pigeonpea growing areas prioritized the search for higher levels of disease resistance. There is a need to study the biology of the pathogen, epidemiology of the disease and refinement of the resistance screening techniques and develop integrated disease management technology for the disease. In this review, the symptomatology of the disease, biology of pathogen including its variability, epidemiology, sources of resistance, other management options and available information on biochemical and genetic basis of disease resistance have been updated and discussed with the identification of future research priorities.

1. Introduction

Pigeonpea [*Cajanus cajan* (L.) Millsp.], a often cross-pollinated, diploid perennial grain legume, is the fourth most important food legume in the world after dry bean (*Phaseolus vulgaris* L.), field pea (*Pisum sativum* L.) and chickpea (*Cicer arietinum* L.). It provides a high quality diet for human consumption as a main source of protein, especially for vegetarian population of the Indian subcontinent. It is also used in stock feed rations. Being a perennial crop, it is used for soil conservation and raising the Lac (*Laccifera lacca*) insect. Pigeonpea grown in rotation with cereals increases the yield of cereals by enhancing soil nitrogen and breaking the disease cycle of important cereal pathogens. Because of its tolerance to heat and drought, it is suitable for low-fertility soils. Globally, pigeonpea is cultivated on about 4.58 million ha, adding 3.27 million tonnes of grain to the global food basket. Asia is the sole contributor and India alone accounts for over 77% area and 81% production (FAO, 2005). Despite the large acreage under pigeonpea cultivation, the total production and productivity are quite low in most of the pigeonpea growing areas and a wide gap exists between the yield of pigeonpea achieved in experimental plots, frontline demonstrations and farmers' field. Susceptibility of pigeonpea to a number of pathogens from seedling stage till harvest is the primary cause for low yields.

Phytophthora blight (PB), caused by *Phytophthora drechsleri* Tucker f. sp. *cajani* (Pal et al.) Kannaiyan et al. (Pdc), is the third potentially important disease of pigeonpea after Fusarium wilt (*Fusarium udum* Butler) and pigeonpea sterility mosaic disease (pigeonpea sterility mosaic virus). History of pigeonpea PB has been previously reviewed (Williams et al., 1975; Kannaiyan et al., 1984). The first suspected occurrence of PB on pigeonpea in India was reported in 1966 by Williams et al. (1968). Since that time, the disease has spread to most pigeonpea growing areas in Asia (Pal et al., 1970; Williams et al., 1975), Africa, America (Kannaiyan et al., 1984), Australia (Wearing and Birch, 1988), Dominican Republic, Kenya, Panama and Puerto Rico (Nene et al., 1996). Recently, the recurrence of PB as a major threat to pigeonpea production and productivity in the Deccan Plateau of India was

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reported irrespective of cropping system, soil types and cultivars (Pande et al., 2006; Sharma et al., 2006). Information on worldwide losses caused by PB is not available, but there is no doubt that the disease is of growing importance and has the potential to cause devastation in a susceptible cultivar, particularly in the context of changing pattern in total rainfall in the Semi-Arid Tropics where pigeonpea is being cultivated as the primary rainy season pulse crop. The effect of PB on grain yield depends on the onset of the disease in relation to crop growth and disease incidence, both of which largely depend on weather conditions and inoculum levels of the pathogen.

2. Disease symptoms and causal organism

The symptoms of the PB on pigeonpea have been described as stem rot (Pal et al., 1970), stem blight (Williams et al., 1975; Amin et al., 1978; Kannaiyan et al., 1980), stem canker (Kaiser and Melendez, 1978) and root rot (Wearing and Birch, 1988). Generally, pigeonpea seedlings become infected with PB as soon as they emerge. Characteristic foliage blight symptoms are visualized on $\geq$1 month old seedlings. Blight symptoms first appear as water-soaked lesions on the primary and triplicate leaves which become necrotic within a week (Fig. 1A and B) when humidity $\geq$ 80% and temperature 20–30 °C persist. The leaflet lesions are circular to irregular in shape and can be as large as 1 cm in diameter. Stem symptoms appear as brown to dark brown lesions distinctly different from healthy green portions on main stem, branches and petioles (Fig. 2A). The lesions on stems and branches increases rapidly and extend up to 20 cm, girdle and cracks and dry the stem. It is also common to find stems swollen into cankerous structures (Fig. 2B). Infected stem and branches break easily in wind. Phloem vessels show smoky gray colored discoloration and xylem vessels remain healthy (Fig. 3).

The PB can be easily mistaken for Fusarium wilt because the general symptoms of these two diseases are similar. An attempt has been made by us to differentiate PB symptoms from Fusarium wilt, based on repeated observations on wilt and PB symptomatology in farmer's field and at research stations on several pigeonpea varieties and hybrids belonging to different genetic make up for the last one decade. Comparative symptomatology distinguishing PB from Fusarium wilt based on visible symptoms at seedling, foliage, stem and roots are given in Table 1, and Figs. 1–6.

Williams et al. (1968) first isolated a PB-causing pathogen from wilted pigeonpea plants with stem canker symptoms at New Delhi, India. Pal et al. (1970) identified the pathogen causing PB as *Phytophthora drechsleri* Tucker var. *cajani* Pal, Grewal and Sarbhoy; and

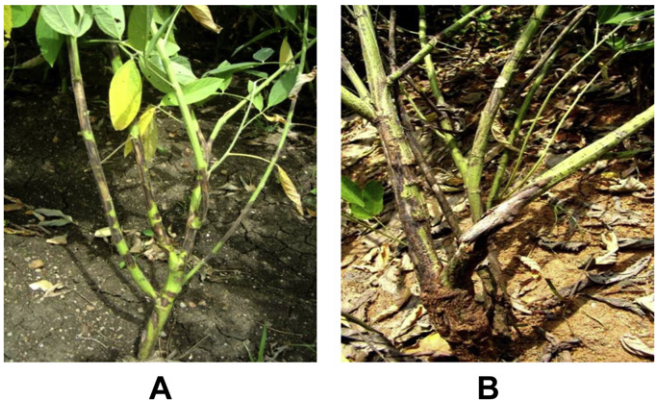

Fig. 2. A. PB symptoms – brown to dark brown lesions on the stem. B. PB symptoms – girdling of infected stem. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Amin et al. (1978) identified the PB pathogen as *P. cajani* Amin, Baldev and Williams. Kannaiyan et al. (1980) carried the detailed investigations on five isolates of the pathogen from India, [P2 (Hyderabad), P3 (New Delhi), P4 (Kanpur), P5 (Kalyanpur) and P6 (Deeg)], and named it *P. drechsleri* Tucker f. sp *cajani* (Pal et al.) Kannaiyan et al. based on the shape and size of sporangia, oogonium and oospore formation, temperature requirement and pathogenicity tests. The use of forma specialis was considered appropriate according to the International Rules of Botanical Nomenclature, Article – 4 (Stafleu et al., 1972).

Various media, such as potato dextrose agar (PDA), oatmeal dextrose agar (OMDA), lima bean agar (LBA), cornmeal agar (CMA), oat meal agar (OMA), V-8 Juice Agar (V8JA) and pigeonpea seed meal agar (PSMA), have been proposed for growing, multiplying and selective recovery and assay of the genus *Phytophthora* from infected plant parts, infected crop debris and soil (Amin et al., 1978; Ribeiro, 1978; Kannaiyan et al., 1980; Erwin et al., 1981; Sheila et al., 1983). Sporangia of *P. drechsleri* Tucker f. sp *cajani* (*Pdc*) are of proliferating type with sizes ranging from 42–83 × 28–48 μm (average 61.8 × 37.3 μm). The sporangial stalks within the same culture are either narrowly tapered or widened some what at the base of the sporangium. Oogonium and oospore sizes show little variation (19–29 to 34–44 μm). Terminal and outer calary hyphal swellings with finger like projection are only observed at low temperatures (9–18 °C). Chlamydospores were not formed on any media at any temperature (Kannaiyan et al., 1980). Singh and Chauhan (1988) reported the oospore formation in *Pdc*.

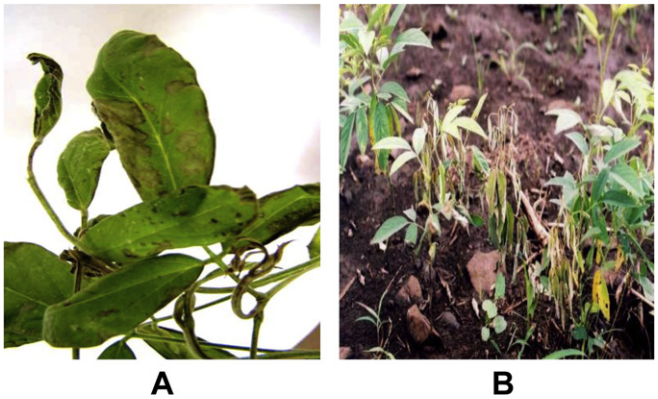

Fig. 1. A. PB symptoms – water-soaked lesions on leaves. B. PB symptoms – foliage blight giving desiccated appearance.

Fig. 3. PB symptoms – smokey, gray colored phloem vessels.

Table 1
Differentiation of symptomatology of Phytophthora blight and Fusarium wilt in pigeonpea.

Plant part/ growth stage	Phytophthora blight	Fusarium wilt
Seedling (up to 30 days)	Young seedlings are killed with in 3–10 days.	Young seedlings gradually wilt and die within 10–30 days retaining dull green color.
Foliage (30–45days)	Water-soaked lesions (Fig. 1A) seen on the leaves and whole foliage gives desiccated appearance (Fig. 1B).	Initial visible symptoms are loss of turgidity, slight chlorosis and drooping of leaves (Fig. 4).
Stem (45–>75 days)	Brown to dark brown lesions, distinctly different from the healthy green portions (Fig. 2A). These lesions increase rapidly and usually girdle the stem (Fig. 2B); infected stems break easily at lesion site. Phloem is smoky gray colored and the xylem remains clear (Fig. 3).	Dark brown to purple streak band extending upward from soil level and usually seen only on one side of the stem (Fig. 5). Characteristic internal symptom of wilt is the browning of the xylem vessels (Fig. 6).
	It is also common to find stem and branches swollen at base or else turning into a cankerous hypertropical structures.	In humid weather a pinkish mycelial growth is often observed on the part of the wilted plants.
Root	The roots of the PB infected plants are healthy and can not be uprooted easily.	The roots of the Fusarium infected plants are dried and can be pulled easily.

3. Host range and pathogen variability

The pathogenicity tests with *Pdc* on 15 weed and 17 cultivated plant species revealed that lucerne, safflower and skeleton weed showed marked response to the pathogen (Cother, 1975). Kannaiyan et al. (1980) based on pathogenicity tests on several hosts reported that *Pdc* isolates from pigeonpea are host specific. However, Kannaiyan and Nene (1985) reported *Atylosia scarabaeoides* and *A. platycarpa* as an alternative host for the pathogen to survive and perpetuate from season to season. It is important to note that pathogenicity testing for host range determination should be a three-phase process employing laboratory, glasshouse and field conditions. Techniques chosen should include the natural situation in terms of environment, plant choice, use of inoculums, flooding times and damage/injury to plant organs. Therefore, research is needed to determine the range of hosts on which *Pdc* can survive and or reproduce in order to develop an effective management plan in cultivated pigeonpea.

The pathogen *Pdc* is reported to have variability in pathogenicity, cultural and morphological characteristics. *P. drechsleri* isolates have great variation in the size of sporangia, presence or absence of chlamydospores and hyphal swellings. Some isolates of *Pdc* have been reported as being homothallic (Tucker, 1931; Tompkins et al., 1936) or heterothallic (Waterhouse, 1963, 1970; Savage et al., 1968; Shepherd, 1978; Kannaiyan et al., 1980). The information so far available indicates that *Pdc* in India is pathogenically variable as isolates differ in their virulence. Multilocation evaluation of pigeonpea lines also indicated possible variation in *Pdc*. Variable reaction on thirteen pigeonpea genotypes to eight isolates was also reported in pot culture studies by Sarkar (1988a) (Table 2). Zoospores, chlamydospores and oospores constitute effective inoculum units and are the forms of *Pdc* most likely to be found in soil.

Recently, Singh and Dubey (2005) characterized the *Pdc* isolates from the northwestern plains of India into two groups based on the rate of mycelial growth. Based on morphology, radial growth, colony color and mycelial characters, 39 isolates of *Pdc* from different locations of Uttar Pradesh were categorized into three groups: fast growing, moderate growing, slow growing. Singh et al.

Fig. 4. Wilt symptoms – chlorosis and drooping of leaves.

Fig. 5. Wilt symptoms – dark brown to purple band on the stem. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Fig. 6. Wilt symptoms-internal browning or blackening of xylem vessels.

(2008) also reported radial growth on PDA after 96 h of inoculation at 28 ± 2 °C as fast growing (85.0–90.0 mm), moderate growing (50.0–84.9 mm) and slow growing (<50 mm).

No specific work has been reported based on molecular variability of *Pdc* so far. However, with the help of molecular sequence data, we can now determine the genetic relationships among several species that are morphologically similar to *P. drechsleri* and *P. cryptogea* (Mostowfizadeh et al., 2010). Phylogenetic analysis of ITS rDNA sequences showed *P. cajani* as a distinct species that is distantly related to *P. drechsleri* (Cooke and Duncan, 1997). In the literature, there are some conflicts in the taxonomic and phylogenetic position of *Phytophthora* spp. and a comprehensive investigation of isolates from different parts of the world and various hosts is necessary.

4. Epidemiology and disease development

4.1. Pathogen survival

The survival and perpetuation of *Pdc* from one season to another is not clear. In addition to cultivated pigeonpea (*C. cajani*), *C. scarabaeoide* var. *scarabaeoides*, a wild relative of pigeonpea, was found to be naturally infected with PB (Kannaiyan and Nene, 1985; Sharma et al., 2006). Recently, Pande and Sharma (2010, unpublished) observed the typical foliar and stem symptoms of PB on wild perennial pigeonpea plants grown at the ICRISAT farm in Patancheru. This observation suggests that *Pdc* is capable of perpetuating from one season to another season on its wild alternative hosts. Agarwal et al. (2009) observed oospore production on several cultivated pigeonpea lines particularly on infected stem tissues but not on leaves after the cessation of rains. Kannaiyan et al. (1983) observed the survival of *Pdc* in the infected stem bits only up to three months, suggesting that stubbles from diseased pigeonpea plants may not support the survival of *Pdc* from one crop season to another. However, Agarwal and Khare (1988) and Bisht (1985) found *Pdc* could survive on infected pigeonpea stems until next crop season.

Table 2
Reactions of pigeonpea genotypes to *Phytophthora drechsleri* f. sp. *cajani* (*Pdc*) isolates from India in pot culture studies at ICRISAT Center.

Genotype	Disease reaction of isolates[a]							
	P4	P3	BHU	KPR	P2	HIS	IARI	P2BF
ICP 6997	HS	S	S	S	S	M	M	M
ICP 7119	HS	S	S	S	S	S	S	M
ICP7910	HS	S	S	M	S	M	M	R
ICP113	HS	S	S	M	S	R	R	R
ICP1788	HS	S	S	R	S	R	R	M
ICP4882	HS	S	S	R	R	R	R	M
ICP7657	HS	M	S	M	R	R	R	M
ICP752	HS	M	S	M	M	R	R	R
ICP2376	HS	S	S	R	M	R	R	R
KPBR80-1-4	HS	M	M	R	R	R	R	R
ICP7065	HS	S	S	R	M	R	R	M
ICP7269	HS	S	S	M	R	M	R	R
ICP7795	HS	M	M	R	R	R	R	M

P2, P3, P4 = *Pdc* isolates.
BHU = *Pdc* isolate from Banaras, India.
KPR = *Pdc* isolate from Kanpur, India.
HIS = *Pdc* isolate form Hisar, India.
IARI = *Pdc* isolate form IARI, New Delhi, India.
P2BF = unknown.
[a] The data source: Sarkar (1988a). R = resistant, 0–20% blight; M = moderately susceptible, 21–50% blight; S = susceptible, 51–80% blight; HS = highly susceptible, 81–100% blight.

4.2. Disease development and spread

Phytophthora drechsleri can exists symptomless in the rhizospheres of many plant species, and that infection was only evident when the balance was upset by environmental extremes (Stanier et al., 1971; Lewis, 1973). The development of PB in the field is correlated with its soil inoculum potential (Sarkar, 1988b). Bisht (1985) found that zoospores are the primary source of inoculum and that wind contributes to inoculum dispersal over short distances during rains. Williams et al. (1975) related high disease incidence to poor soil surface drainage, while Singh and Chauhan (1985) found PB developing to an epidemic level in well drained fields. Similarly, Sharma et al. (2006) reported an outbreak of PB in well drained, partially drained and temporarily water logged fields irrespective of cropping systems, soil types and crop cultivars, soon after the heavy down pore in Deccan Plateau, India.

Singh and Chauhan (1985) reported more rapid development of PB at night in the field, and confirmed this under conditions of artificial darkness in greenhouse. Reddy et al. (1991) found that PB outbreak usually occurs when there was a decrease in day temperatures of the previous week and difference between maximum and minimum temperatures are the least. The weather data and PB incidence collected in India from 2000–2010 show higher incidence of PB when maximum temperature (28–40 °C), minimum temperature (12–24 °C); relative humidity 75–96% is coupled with ≥300 mm rain fall within a week (Sharma et al., 2006; Pande and Sharma, 2010). Few studies on relationships between PB incidence and soil nutrition indicated that in the absence of potassium (K) and high doses of nitrogen (N), PB incidence increased (Pal and Grewal, 1975). However, the addition of K decreased disease incidence regardless of the presence of N or phosphorus (P) in the soil.

4.3. Plant age and susceptibility

Mishra and Shukla (1986) found high incidence (100%) of the disease in the 15-day-old seedlings and least incidence (25%) on four-month-old plants. In a pot experiment using three isolates (P2, P3 and P4) of *Pdc*, Sarkar et al. (1992) reported that older plants of all pigeonpea cultivars tested showed less plant mortality than the younger plants irrespective of *Pdc* isolate used for inoculation. They also observed significant variation in plant mortality, particularly in the 30-day-old plants of the test genotypes. The reasons for such reduced susceptibility with increased age in pot experiments are not understood. Pande and Sharma (2010, unpublished) observed the susceptibility of pigeonpea to PB irrespective of growth stage both in the field and greenhouse. These observations need detailed investigations, before any conclusion can be drawn on the relationship of plant age and susceptibility to PB. Further this

experiment does not establish any correlation between the susceptibility of pigeonpea in the pot experiment and field environment. Under field conditions, it is not uncommon to observe plants dying even after 60 days and disease symptoms continue expressing from seedling stage to flowering and beyond. In such cases it is possible that infection might have occurred at an early age, and that the disease progresses slowly, killing the plants later.

5. Disease management

5.1. Host plant resistance

The preliminary step for exploiting host plant resistance (HPR) is the development of reliable and repeatable techniques for large scale screening of germplasm and breeding lines. Several techniques suitable for PB resistance screening under field and green house conditions have been reported (Pal et al., 1970; Kannaiyan et al., 1981; Nene et al., 1981; Reddy et al., 1990).

5.1.1. Greenhouse screening

Pal et al. (1970) used a "leaf scar" method to inoculate 1–2 month old plants grown in pots. This method consisted of inoculating plants at the point of attachment of leaf after its removal with mycelial mats of the fungus multiplied on PDA. Kannaiyan et al. (1981) standardized the pot-culture "drench" inoculation and foliage inoculation techniques. In drench inoculation, 5–10 day old seedlings raised in pots filled with field soil are drench-inoculated with macerated mycelial suspension of the fungus multiplied on V-8 juice medium (one mycelial mat in 200 ml of water). Inoculum (100 ml) was poured around seedlings. Pots were liberally watered three times a day to assure adequate development of the disease. In this technique, disease developed after 7–10 days of inoculation. In foliage inoculation technique, inoculum is sprayed on 15–30 days old plants grown in a pot, the plants covered with polythene bags for 48 h, kept on glass house benches, and later sprayed with water for 10 days. Typical blight symptoms appeared with in 10 days after the inoculations.

5.1.2. Field screening

The components and procedures of the "field screening" of pigeonpea genotypes for PB resistance was standardized at ICRISAT by Nene et al. (1981), including planting of test material with a 30-cm row space and inter planting a susceptible cultivar (e.g. ICP 2376 and/or ICP 7119) to serve as an indicator line after every 2–4 rows. The collar region (base) of 1-month-old plants is inoculated with mycelial mats of the fungus after mixing with carborandum. Reddy et al. (1990) developed a diseased debris field inoculation technique. In this technique, a well leveled Alfisol is selected and PB susceptible cultivars (ICP 2376 and ICP 7119) are sown as closely as possible (30×10 cm) on flat beds preferably before the monsoon rain arrives. When the plants are about one month old, approximately 250 kg of diseased plant debris (pigeonpea stems with PB lesions collected during the previous season and stored dry in the field shelter) are scattered over the field. During rain-free days, sprinkle irrigation is liberally provided. This technique can produce near 100% PB incidence in susceptible controls at ICRISAT, Patancheru. However, in the greenhouse and field inoculation techniques, the production of disease-causing propagules such as sporangia and eventually zoospores and their role in infection and disease development have not been determined.

5.1.3. Resistance sources

In previous studies conducted in different pigeonpea growing areas particularly in India, several sources of resistance to PB were identified (Pal et al., 1970; Kannaiyan et al., 1980; Bhargava and Gupta, 1983; Singh and Chauhan, 1985; Mishra and Shukla, 1986; Sharma et al., 1995; Reddy et al., 1990). However, the most of these lines identified by various researchers were later found susceptible to *Pdc* under natural epidemic conditions in Deccan Plateau (Sharma et al., 2006). Pande et al. (2006) found varying levels of resistance among the improved lines and wild *Cajanus* species. They reported *C. sericeus* highly resistant (≤10% plant mortality), while *C. scarabaeoides* moderately (≤20%) and *C. cajanifolius* susceptible (≥40%) under natural PB epiphytic conditions at ICRISAT during the 2005 rainy season. Recently, Pande and Sharma (2009) evaluated a large number of wild *Cajanus* spp. and only few lines were identified to be resistant (Table 3). Pande and Sharma (2009) also evaluated newly developed pigeonpea lines and hybrids, vegetable type pigeonpea germplasm and minicore under natural infection conditions and found that most of them succumb to *Pdc* isolate with ≥40% plant mortality. Resistance identified so far in improved pigeonpea germplasm and breeding lines as well as wild accessions of *Cajanus* spp. and its relatives under field and greenhouse conditions needs to be reconfirmed under epidemiologically sound disease development environment and with the emergence of new pathotypes of *Pdc*.

5.1.4. Biochemical and histopathological basis of host plant resistance

Unlike other major plant pathogen systems of crop plants, detailed investigations have not been undertaken on the infection process of *Pdc* on the pigeonpea plant and the biochemical basis of PB resistance in pigeonpea. Preliminary investigations suggest that phenolic constituents of leaves and stems increased after inoculation in resistant varieties while they decreased in the PB susceptible variety of pigeonpea (Pal and Grewal, 1975). It appears that there may be stimulation of host defense reaction due to infection in the resistant variety while such mechanism may be absent in the susceptible one. Determination of PB resistance of pigeonpea by a diverse set of anatomical, biochemical, physiological and genetic characters is not known.

5.1.5. Genetic basis of host pathogen interaction

The lack of pigeonpea cultivars resistant to *Pdc* is due to the difficulty in working with this host-specific *Phytophthora* in breeding programs because of frequent evolution of new pathotypes and coexistence of more than one pathotypes at one location. Furthermore, sources of durable genetic resistance to *Pdc* in pigeonpea are not available. It appears that the identification of resistance to *Pdc* is a challenging task because of its cross pollinating ability. The limited reports available on genetics of PB resistance in pigeonpea suggest that a few major genes may control resistance in the host to PB. Kannaiyan et al. (1981) and Sharma et al. (1982) studied the mode of inheritance and allelic relationships of genes for resistance to P2 isolate of *Pdc* and combined resistance to the Kanpur isolate. They found a high degree of

Table 3
Phytophthora blight (PB) disease incidence in wild species of Pigeonpea during 2009–10 season, ICRISAT, Patancheru, India.

Wild species	PB incidence (%)
ICPW 40 (*Cajanus lineatus*)	10
ICPW 207 (*Paracalyx scariosa*)	10
ICPW 192 (*Flemingia bracteata*)	8
ICPW 41 (*Cajanus lineatus*)	10
ICPW 202 (*Flemingia stricta*)	10
Cajanus sericeius	≤10
Cajanus scarabaeiodes	15
Cajanus cajanifolius (Syn. *Atylosia* spp.)	100

specificity of reaction to isolate P2 by resistant lines from diverse resources is of limited use in breeding program. A systematic search for new genes for resistance to different races of *Pdc* is essential for developing cultivars with adaptability.

5.2. Cultural control

Cultural practices that reduce the main source of inoculum are most important in effective disease management. Planting pathogen free seed, crop rotation with non-host crops such as cereals, destruction of PB infected pigeonpea stubble and destroying alternative host species such as *Atylosia* spp. and wild *Cajanus* spp. are all important measures to reduce the amount of inoculum and the likelihood of PB epidemic. In addition to this, proper drainage of fields and sowing of crop on raised beds are the suggestive agronomical practices to reduce PB incidence, since water logging is believed to predispose pigeonpea crop to PB under favourable environmental conditions (Singh and Chauhan, 1985).

Phosphorus acid is known to effectively control various oomycetes diseases as phosphoric acid moves upward and downward through xylem and phloem in plants. Hae et al. (2007) developed a new system for phosphorus acid formulation using a carrier coated with polysaccharides. The direct application of this product around basal stem of pepper plants resulted in excellent control of PB caused by *P. infestans*. This study suggests further research on the management of PB of pigeonpea using phosphorus based fertilizers.

5.3. Chemical control

Although several fungicides have proved effective in control of PB, systematic studies on the control of disease using fungicides are limited. In a pot experiment, Pal and Grewal (1983) reported Brestan-60® effective in controlling PB in one-month-old plants when applied before inoculating with *Pdc*. Significant control of blight (>90%) was achieved with metalaxyl (1.75 g a.i kg^{-1}seed) in a greenhouse experiment (Chaube et al., 1984; Kannaiyan and Nene, 1984; Agarwal, 1987; Bisht and Nene, 1988). However, Chaube et al. (1987) reported the poor efficacy of metalaxyl applied as seed dressing in protecting older pigeonpea plants against PB. Sheila and Nene (1987) reported reduced PB incidence with the spray or soil drench with two phytoalexins Phytoalexin-84® and Induce®. Direct application of slow releasing of phosphorous acid formulations (curdlan or pestan) using a carrier coated with polysaccharides resulted an excellent control of PB disease of pepper (Park et al., 2007). They further suggested that application of formulation product once or twice during crop season can control Phytophthora diseases on various crops. However, there is a need to explore the possibility of using this product or similar product for the management of PB in pigeonpea.

5.4. Integrated disease management

Adoption of the integrated disease management (IDM) technology is essential for economical and effective control of PB. Moderate levels of HPR can be combined with other cultural practices and/or application of minimum dosage of fungicide for control of PB. The location specific recommended IDM practices include: (a) use of pathogen-free seed, (b) seed treatment with fungicide, (c) crop rotation, (d) raised bed planting, (e) adequate field drainage, (f) use of disease resistant/tolerant genotypes and (g) strategic application of foliar fungicides. The PB incidence can be reduced substantially with foliar sprays of metalaxyl more so in the field resistant genotypes than in susceptible genotypes (Bisht and Nene, 1988). The results obtained in pot experiment (Sarkar et al., 1992) conjectured that planting pigeonpea genotypes at least one month early so that when extended periods of rain occur after the beginning of the monsoon, plants will be partially resistant to PB. However, in the absence of knowledge on host resistance, effective fungicides and survival mechanism of *Pdc*, early planting of field resistant genotypes and judicious application of metalaxyl spray may provide effective IDM of PB.

6. Research needs

Shift in the variables of climate change, their unpredictable occurrence, and change in rainy day frequency is creating optimal conditions for frequent outbreaks of PB epidemics. Despite the extensive investigations on *Phytophthora* spp. on other hosts, the infection process of *Pdc* on pigeonpea has not been studied. Also, very little is known about resistance mechanism of pigeonpea against *Pdc*. Knowledge of the ecology of *Pdc* and the disease epidemiology in relation to changing rainfall patterns, infection process and host defense mechanism will help in devising effective management strategies for PB.

The management of PB is essential to provide increased and stable pigeonpea yields through out the pigeonpea growing regions. The PB management should not be completely rely on the use of fungicides, as the development of fungicide resistance in *Phytophthora* spp. has been commonly observed. Hence, IDM programs suitable for adoption by resource poor farmers should be emphasized. It is advised that the PB management in pigeonpea should be based on the location specific disease predictive models. HPR should be emphasized over other control measures as the most environmental-friendly and economic disease control strategy. Selection of resistant sources for genetic improvement programs should be based on resistance to PB at seedling, vegetative, flowering and podding stages since many lines resistant in seedling/vegetative stage can be susceptible/or show disease symptoms at later growth stages. However, there is a need of developing inoculation and screening procedures for exploiting HPR. Concerted efforts are needed to refine the components of screening procedures and understanding the epidemiology of the disease with focused research on the host × pathogen × environment interactions. Studies should be conducted to determine the genetics and allelic relationships of resistance to PB in different genotypes as an essential precursor to pyramid resistance genes.

The knowledge of the variability of *Pdc* is also a prerequisite for breeding programs. There is a need for further research on pathogenicity and study the variability of pathogen under varying climate conditions. In this regard, development of marker-assisted selection methods will enable rapid screening of different genotypes and breeding populations for disease resistance. Moreover, pyramiding of different sources and/or mechanisms of resistance sharing a similar phenotype will only be possible through the application of molecular breeding tools. There is a lack of published scientific research work particularly on outbreak of the disease in different regions in Asia and on management approaches such as biocontrol, particularly with field results and fungicide resistance.

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