Offered Review

COLLETOTRICHUM LINDEMUTHIANUM, THE CAUSAL AGENT OF BEAN ANTHRACNOSE

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SUMMARY

Common bean (*Phaseolus vulgaris* L.) is an important constituent of people's diets especially in developing countries. Dry beans find a unique position in the culinary items because of their high nutritional value. For instance, rice and bean recipe (Rajmah Chawal) is famous in the northern part of India. Many fungal, viral and bacterial diseases affect the crop and cause heavy losses worldwide. Among the various fungal diseases, bean anthracnose caused by *Colletotrichum lindemuthianum* is a serious disease under cool and humid environments. Under favorable conditions, the yield losses may be up to 100 percent. The scientific community across the world has been studying the bean-anthracnose interaction for over 100 years and the information has helped to understand the pathosystem and devise better disease management strategies. Many excellent reviews on anthracnose resistance genes, marker aided breeding and R gene signatures highlight different tactics for disease management. Assembling the substantial literature available on the pathogen is necessary for better understanding of the pathogen biology. The present review consolidates this information and provides a comprehensive outline about the detection, pathogenicity genes, pathogenic variability and molecular diversity of *C. lindemuthianum*. The importance of the bean genome and availability of SNP markers to dissect the bean-anthracnose interface is also addressed.

Keywords: Detection; pathogenic variability; molecular diversity; pathogenicity genes

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INTRODUCTION

Common bean (*Phaseolus vulgaris* L.) belongs to the genus *Phaseolus*, subtribe *Phaseolinae*, tribe *Phaseoleae*, subfamily *Papilionoideae* in the family *Fabaceae* (Debouck, 1991; Singh *et al*., 1991, Singh, 2001; McClean *et al*., 2008). The genus includes many types of beans (like dry beans, green beans, shelling beans), and contains 50 different species (Delgado-Salinas *et al*., 1999), with most of them from Mesoamerica and South America. Among these, only five, i.e. common bean (*P. vulgaris*), runner bean (*P. coccineus*), year bean (*P. dumosus*), tepary bean (*P. acutifolius*) and lima bean (*P. lunatus*) are cultivated, bred and widely used for human consumption worldwide (Gepts *et al*., 2008; Aragão *et al*., 2011). *Phaseolus* species are diploid with 2n = 2x = 22 (Mercado-Ruaro and Delgado-Salinas, 1998).

Recent studies based on the molecular analysis of wild relatives of the crop suggest central Mexico as the centre of origin of *P. vulgaris* (Bitocchi *et al*., 2012). The crop was independently domesticated in both the Andean and Mesoamerican regions (Bitocchi *et al*., 2013; Bellucci *et al*., 2014) and was transported to Africa and Europe in the 1600s (Pathania *et al*., 2014; De Ron *et al*., 2016). Common beans grow at different altitudes ranging from 50 to 3000 meters above sea level and can withstand extreme environments too. The crop prefers temperatures ranging from 14 to 26°C, an annual precipitation between 400 and 1600 mm per year, a slightly acid soil pH (average 5-6), show a wide range for days to maturity (70-200) and seed yield potential (400-5000 kg ha^{-1}) (Wortmann *et al*.,, 1998; Debouck, 1999). Consumers eat edible dry seed and fresh (green) pods. The dry seeds are source of calories, minerals, fiber and are rich in dietary protein (18-40% of seed weight), which humans need on daily basis.

The low productivity of commercially cultivated bean varieties is due to many biotic and abiotic factors. Among the biotic factors such as anthracnose, angular leaf spot, *Bean common mosaic virus*, common bacterial blight and halo blight, bean anthracnose caused by *Colletotrichum lindemuthianum* (Sacc. & Magnus) Lams.- Scrib, is a serious seed borne pathogen throughout the world. Infection of susceptible cultivars in cool and humid environmental

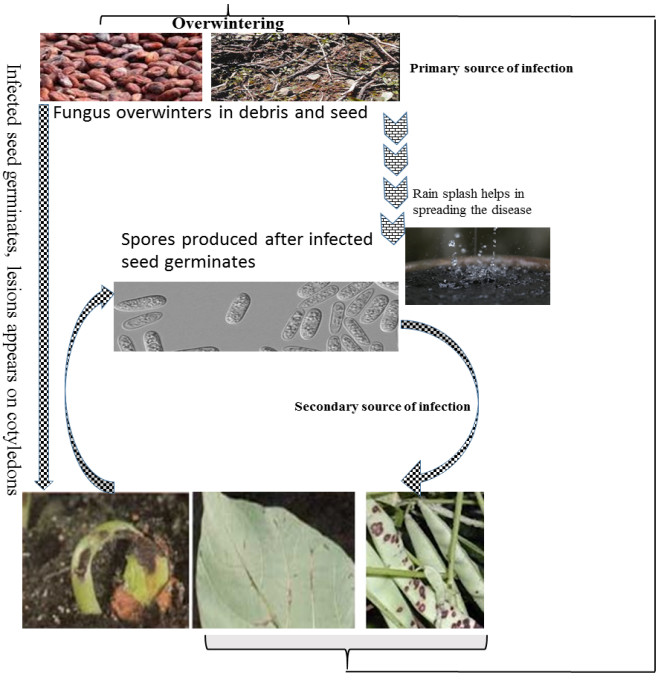

Fig. 1. Disease cycle of *Colletotrichum lindemuthianum.*

conditions can result in yield losses as high as 100% (Sharma *et al.*, 1994; Fernandez *et al.*, 2000; Padder *et al.*, 2007; Sharma *et al.*, 2008). Despite availability of management practices like seed and foliar treatment with fungicides, crop rotation, certified seed and genetic resistance, the crop is vulnerable to the disease because of prevalence of diverse pathogen races. Though the disease can be managed successfully through the use of resistant cultivars, the high pathogenic variability renders the majority of the cultivars susceptible (Pastor-Corrales *et al.*, 1995; Balardin *et al.*, 1997; Balardin *et al.*, 1999; Mahuku and Riascos, 2004; Pathania *et al.*, 2006; Sharma *et al.*, 2007). The international bean differential set and the corresponding binary codes (Drijfhout, 1978; Pastor-Corrales, 1991) for anthracnose race designation have allowed consistent comparison of virulence among different research groups. Anthracnose resistance genes present in different cultivars were comprehensively documented (Kelly and Vallejo, 2004; Ferreira *et al.*, 2013) and the pathogenic variability was first summarized by Melotto *et al.* (2000). Bean genome availability was recently used to dissect the anthracnose resistance genes in terms of their R gene signatures (Meziadi *et al.*, 2016). Similarly marker assisted backcross breeding has resulted in development of anthracnose resistant cultivars in different parts of the world (Alzate-Marin *et al.*, 2004; Faleiro *et al.*, 2004; Ferreira *et al.*, 2012; Madakbas *et al.*, 2013; Hegay *et al.*, 2014).

Comprehensive information on anastomosis, pathogenesis, pathogenic and molecular variability is available for *C. lindemuthianum*, but needs to be consolidated into a comprehensive review. The focus of the present review is to discuss the various features of pathogen biology so that the information may be helpful to research groups working on bean anthracnose around the world.

THE PATHOGEN

The fungus *Colletotrichum lindemuthiamum* taxonomically belongs to: Fungi, Ascomycota, Pezizomycotina, Sordariomycetes, Hypocreomycetidae, Glomerellales, Glomerellaceae; *Colletotrichum lindemuthianum* as reported by Mycobank (http://www.mycobank.org/). The fungus was discovered by Lindemuth in 1875 (Tiffany and Gilman, 1954) and first described by Saccardo (1878). Then the disease was reported not only on *P. vulgaris* but also on other *Phaseolus* species in different parts of the world (Sicard *et al.*, 1997b). The infectious nature of the pathogen was established by Frank in 1883 (Dey, 1919). Since then, anthracnose has occurred widely in Europe and North America. In 1891, the bean crop was completely destroyed in certain parts of Italy and in 1915 and 1916 serious epidemic occurred in Germany. The pathogen overwinters in seed and crop residues (primary source of infection) and infects all aerial parts of the bean plant. Typical symptoms are deep, shrunken lesions containing flesh-colored spores on bean pods that are the most distinctive symptoms of anthracnose. Lesions also commonly appear on stems, hypocotyls and leaf veins of seedling plants, with more advanced disease resulting in wilting and flagging of chlorotic leaves similar to that of other foliar pathogens. Further advancement of the disease leads to complete girdling and eventually death of the plant. Infection of the bean pods results in rust-colored lesions that develop into sunken cankers with black ring borders. Severely infected premature pods abort and fall early, while pods that mature produce infected seed with dark cankers that make the seed unmarketable to consumers (Pastor-Corrales and Tu, 1989). The anamorph stage produces acervuli that erupts through epidermis. It has short crowded conidiophores at the bottom with conidia and the spores escape through an opening at the top. Water splashes disperse spores and result in the secondary spread of disease (Fig. 1).

Sexual form (teleomorph) of the pathogen was reported more than 100 years ago by Shear and Wood (1913) in the laboratory grown cultures. These cultures produced perithecia and asci, a typical characteristic of ascomycetes and named the perfect stage as *Glomerella lindemuthiana*. Later, Kimati and Galli (1970) rediscovered the perfect stage of the fungus by mating two different isolates. Since the ascospores were pathogenic to beans only, they renamed it to *G. cingulata* f. sp. *phaseoli*. The teleomorph stage rarely occurs under field conditions, however since 1970 many attempts to develop perithecia under laboratory conditions have been successful in Brazil (Ishikawa *et al.*, 2010a; Barcelos *et al.*, 2011, 2014). Recently *G. cingulata* f. sp. *phaseoli* or *G. lindemuthiana* have been detected on bean lesions exhibiting typical anthracnose symptoms on pods, leaves or stems (Camargo *et al.*, 2007; Ishikawa *et al.*, 2010a; Souza *et al.*, 2010; Barcelos *et al.*, 2014). Sexual compatibility studies performed in the laboratory have revealed both homo- and heterothallism in different isolates

of *G. cingulata* f. sp *phaseoli* (Mendes Costa, 1996; Rodriguez-Guerra *et al*., 2005; Camargo *et al*., 2007; Souza *et al*., 2010). Heterothallism investigation in *C. lindemuthianum* lead to characterization of MAT1-2-1 (Garcia-Serrano *et al*., 2008). Both parent strains contained a single copy of this gene encoding high mobility group motif (HMG). Recently, Barcelos *et al.* (2014) conducted a comprehensive study of *Glomerella* species isolated from anthracnose infected bean lesions. The authors rejected the theory of *Glomerella* being the perfect stage of *C. lindemuthianum*. They suggested that *Glomerella* species present in the lesions are epiphytes that grow opportunistically like *C. gloeosporioides* species complex that are weak pathogens of beans and take the advantage of lesions produced by infection of aggressive *C. lindemuthianum*. A recent study by Mota *et al.* (2016) showed pathogenic nature of *Glomerella* species group II of Barcelos *et al.* (2014). The isolates caused mild symptoms on the host surface after 10 days post inoculation and at a later stage it is difficult to distinguish between the *C. lindemuthianum* and *Glomerella* spp. based on the symptoms. The study reflects the anthracnose/scab complex on *P. vulgaris* and warrants a detailed investigation because isolates belonging to *Glomerella* spp. group I (Mota *et al*., 2016) failed to cause disease.

PATHOGEN DETECTION

Although bean anthracnose is caused by *C. lindemuthianum*, the occurrence of other *Colletotrichum* and *Glomerella* species (Mota *et al*., 2016) from infected host tissues warrants accurate detection of the pathogen. Identification of the fungus based on morphology, symptoms on host surface and pathogenicity is time-consuming and needs a specialist in taxonomy. The detection becomes strenuous in case of seed and donor plant certification, therefore an accurate, sensitive and effective diagnostic tool is necessary. Among the various methods for fungal identification, PCR or qPCR is a robust, quick and sensitive method. Pathogen specific primers exist for a particular pathogen and *C. lindemuthianum* is not an exception. A sensitive and specific PCR based anthracnose pathogen detection method was developed by Chen *et al.* (2007). They developed a forward primer within the vicinity of ITS region. This primer, when used with universal ITS4 primer (White *et al*., 1990), amplifies a 461 bp rDNA region specifically in *C. lindemuthianum* with a detection limit as low as 10 fg. Wang *et al.* (2008) used two primer pairs (CY1, CY2 and CD1, CD2) for accurate evidence of anthracnose infection in bean tissues and seeds. Detection using CY1/CY2 primer pair was not specific as the 442 bp amplicon was also amplified in *C. orbiculare* but amplification using CD1/CD2 primer pair distinguished between *C. lindemuthianum* and *C. orbiculare.* Unfortunately, the authors did not sequence the 638 bp DNA segment amplified using CD1/CD2 primer pair. However, recently Gutierrez *et al.* (2014) sequenced a *C. lindemuthianum* isolate (A83) through pyrosequencing and local BLASTX search against *de novo* assembly resulted in identification of a contig carrying CD1 and CD2 specific sequences. The sequence encodes for an iron permease (*Ftr1*) pseudogene flanked by a gene encoding for a polyhydroxyproline-rich protein in *Colletotrichum*. A primer pair specific to *C. lindemuthianum* HMG domain developed by Garcia-Serrano *et al.* (2008) was used by Pinto *et al.* (2012) and Mota *et al.* (2016) for a more accurate detection of anthracnose pathogen. Among the primers used for detection of anthracnose pathogen, HMG primer proved to be the best for PCR detection. Among the two mating idiomorphs (MAT1-1 and MAT1-2) present in the genome of ascomycetes, all *Colletotrichum* isolates carry the MAT1-2 idiomorph, while MAT1-1 idiomorph has never been reported from the genus *Glomerella* despite several attempts to amplify the alpha domain (Vaillancourt *et al*., 2000; Rodriguez-Guerra *et al*., 2005; Menat *et al*., 2012). Sequence analysis of MAT-1-2-1 suggests a huge difference among different *Colletotrichum* species (Garcia-Serrano *et al*., 2008). Because of sequence variation (above 45%) in the MAT1-2-1 locus, this region seems a better choice to develop species specific markers than the universal ITS region. Recently Chen *et al.* (2013) developed a qPCR based detection of *C. lindemuthianum* with a detection limit up to 5 fg of *C. lindemuthianum* genomic DNA. Primer pair for qPCR detection was developed within the ITS region of fungus.

ANASTOMOSIS

Heterokaryon formation between compatible strains of the same fungus (anastomosis) is an important and common component in the life cycle of many fungi. Strains that form a stable heterokaryon are referred to as vegetative compatible and the resulting groups are ascribed to a particular vegetative compatible group (VCG). Vegetative incompatibility is a genetic mechanism that restricts the heterokaryosis between individuals that differ in one or more *het* or *vic* loci (Glass *et al*., 2000, 2004; Xiang and Glass, 2004). Mating of genetically identical strains leads to death because of presence of different *het* loci (Hall *et al*., 2010) and similar cell death occurs in *C. lindemuthianum* (Carvalho and Mendes-Costa, 2011). Based on nitrate metabolism, Carvalho and Mendes-Costa (2011) identified 18 *nit* mutants in *C. lindemuthianum* and classified them into four groups (*nit1, nit2, nit3* and *nitM*) with high frequency of *nit2* group. Confrontation among different *nit* mutants resulted in hyphal anastomosis in 32 cases whereas 23 confrontations were incompatible. Castro-Prado *et al.* (2007) reported the occurrence of successful parasexuality between five races of *C. lindemuthianum* under laboratory, however, a few race confrontations were vegetative incompatible. Confrontations within 13 isolates of race 65 suggest that anastomosis also happens within race isolates

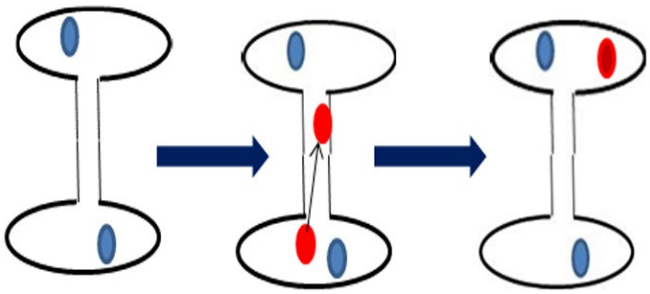

Fig 2. Process of CATs fusion in *Colletotrichum lindemuthianum*. After mitotic division the newly formed nucleus moves to other conidia through the opening and after 46 h after CATs fusion the movement of nucleus completes (Ishikawa *et al.,* 2010, 2013).

(Ishikawa *et al*., 2008). In addition to hyphal anastomosis, *C. lindemuthianum* shows conidial anastomosis (Roca *et al*., 2003) and later this phenomenon was reported to occur in various fungal species (Roca *et al*., 2004). Conidial anastomosis in the fungus occurs on the host surface (Ishikawa *et al*., 2010a) and within the acervulus either on the host or in the culture (Roca *et al*., 2003). Conidial anastomosis occurs between a specialised hypha called as conidial anastomosis tube (CAT) that allows fusion between conidia and conidial germlings (Read *et al*., 2009, 2010). This fusion between germlings acts as a single coordinating unit for the exchange of nutrients, water, signal molecules, nuclei and other organelles (Read *et al*., 2009, 2010). In asexually reproducing fungi, CATs may contribute to high level of variability through parasexual recombination that occurs within and between *C. lindemuthianum* and *C. gossypii* (Roca *et al*., 2004; Read and Roca, 2006). Detailed studies on CATs using live cell imaging over a time course showed that it depends on culture age, media and the strain used (Ishikawa *et al*., 2010b). High numbers of CATs take place in water after 72 h of incubation whereas nutrient media inhibited its formation. The blending takes about 60 h to complete and at 56 h, cytoplasm moves between the conidia (Ishikawa *et al*., 2010b, 2013). Labelling nuclei with green fluorescent protein (GFP) and tracking with confocal microscopy revealed that the nuclei move after 40 to 41 h after CATs formation (Fig. 2). Labelling nuclei of two strains of *C. lindemuthianum* with green and red fluorescent proteins, respectively, revealed that vegetative incompatibility was suppressed by the CATs formation (Ishikawa *et al*., 2012).

PATHOGENESIS AND PATHOGENICITY GENES

Based on the feeding habit, *C. lindemuthianum* is a hemibiotroph (Ferreira *et al*., 2013) that relies on the common bean for nutrients before causing cell death. A number of reviews on how the pathogen infects the host has been published (O'Connell *et al*., 1985; Pastor-Corrales and Tu, 1989; Bailey and Jeger, 1992; Ishikawa *et al*., 2010a). Conidia germinate on the host surface and the germ tube differentiates into a specialized penetration structure known as appressorium. Melanized appressorium produces high turgor pressure to penetrate the host surface directly. An infection peg emerges from the appressorium and afterwards the fungus forms infection vesicles and primary hyphae (biotrophy). Biotrophic hyphae spread to a few adjacent cells and then the fungus switches to necrotrophy by producing secondary hyphae.

The expression of genes during biotrophic and necrotrophic phases is indispensible for successful infection in order to prevent pathogen triggered and effector triggered immunity defences (O'Connell *et al*., 2012). Forward fungal genetics, and particularly insertion mutation studies, have provided valuable insights on the molecular mechanism of *Colletotrichum* infection process. Traditionally, mutants in fungi were developed using transposons, polyethylene glycol (PEG) and restriction enzyme–mediated integration (REMI). However, recently *Agrobacterium tumefaciens* mediated transformation (ATMT) has gained popularity because of its simplicity. Several *Colletotrichum* mutants deficient in the pathogenicity at a particular stage of infection have revealed key pathogenicity genes. Expression of these genes is necessary for successful establishment and colonization of host tissue (Munch *et al*., 2011; Nakamura *et al*., 2012; Liu *et al*., 2013a; Korn *et al*., 2015). For instance, studies on *Colletotrichum* species have shown that melanization of appressorium is indispensable for virulence (Rasmussen and Hanau, 1989; Lin *et al*., 2012; Takahara *et al*., 2012; Liu *et al*., 2013b). Unlike other *Colletotrichum* species, there is a lack of literature on pathogenicity genes in *C. lindemuthianum* (Table 1). Most of these studies have shown key genes and transcriptional factors necessary for causing infection of bean. Random insertional mutagenesis first carried out by Dufresne *et al.* (1998) in *C. lindemuthianum* identified a mutant that showed inability to penetrate bean leaves. Sequence analysis showed that the gene named *clk1* is a member of serine/threonine protein kinases. In another study, Dufresne *et al.* (2000) identified a transcriptional factor (*CLTA1*) belonging to the zinc cluster ($Zn[II]_2Cys_6$) family essential for regulating the switch between biotrophic and necrotrophic phases. Another gene (*CLNR1*) responsible for causing necrotrophy in bean tissue induces nitrogen starvation *in planta* (Pellier *et al*., 2003). Nitrogen starvation genes are expressed during the early phases of infection in many fungi (Talbot *et al*., 1993; Stephenson *et al*., 1997; Stephenson *et al*., 2000). Formation of a penetration peg in *C. lindemuthianum* is necessary for successful infection of bean tissues. This specialized structure is under the control of several genes (Xu *et al*., 1997, 1998; Balhadere and Talbot, 2001; Clergeot *et al*., 2001; Kim *et al*., 2002; Park *et al*., 2002). Among these, *PLS1* (punchless) gene is crucial for the peg formation in *Magnaporthe grisea*, the rice blast fungus (Clergeot *et al*., 2001). The gene encodes for a protein tetraspanin that plays an important role in animals

Table 1. Genes responsible for successful infection in *Colletotrichum lindemuthianum* with their putative function and mutants impaired in the infection process.

Gene name	Annotation	Infection process	Strategy	Reference
clk1	serine/threonine protein kinases	appressorium functionality	REMI*	Dufresne *et al.,* 1998
CLTA1	Transcriptional activator belonging to the fungal zinc cluster ($Zn[II]_2Cys_6$) family	Switch between the biotrophy and the necrotrophy	REMI	Dufresne *et al.,* 2000
CIH1	Proline-rich glycoprotein	Biotrophy	Immunofluorescence	Perfect *et al.*, 2000
clap1	Copper-transporting ATPase	Fewer appressoria with less melanin	REMI	Parisot *et al.,* 2002
CLNR1	AREA and NIT2 global fungal nitrogen regulator	Few anthracnose lesions seldom occur and the mutant is impaired in causing necrotrophy	REMI	Pellier *et al.,* 2003
ClPLS1	Tetraspanin super family	Infection vesicles and primary hyphae	REMI	Veneault-Fourrey *et al.,* 2005
PacCl	pH-responsive transcriptional regulator	Maceration on the infected plant tissue	DNA hybridization to single plaques	Soares *et al.,* 2014

* Restriction Enzyme Mediated Insertion (REMI)

and fungi (Boucheix and Rubinstein, 2001; Gourgues *et al.*, 2002; Hemler, 2003; Stipp *et al.*, 2003; Gourgues *et al.*, 2004). Insertional mutagenesis in *C. lindemuthianum* led to the discovery of *ClPLS1* pathogenicity gene that is similar in function to *M. grisea PLS1* gene (Veneault-Fourrey *et al.*, 2005).

PATHOGENIC VARIABILITY

Variability in *C. lindemuthianum* was first described by Barrus (1911) when he noticed differences between virulence of two races of anthracnose against 139 bean cultivars. These first two races were identified as α and β, and laid the foundation for the discovery of greater pathogenic variability. Prior to 1988, fourteen bean anthracnose races were reported using different sets of differential cultivars (Burkholder, 1923; Frandsen, 1953; Oliari *et al.*, 1973; Wallen, 1979). However, many researchers in various countries used local codes instead of the Greek letters to identify anthracnose races (Melotto *et al.*, 2000). No standardized system limited the knowledge of the global variability of *C. lindemuthianum*. Recently an international differential set comprising of 12 bean cultivars (Pastor-Corrales, 1991) was established to determine isolate race(s) (Table 2). Each differential has a binary number and the sum of the cultivars with susceptible reactions gives the binary number of a specific race. For example, anthracnose race 73 is virulent on Mexico 222 [64], Cornell 49242 [8], and Michelite [1]. Such naming has allowed for a consistent comparison of data among different research groups. Literature shows presence of more than 100 races of pathogen worldwide (Gonzalez *et al.*, 2015). To date about 1590 isolates of *C. lindemuthianum* inoculated on 12 bean differential cultivars have resulted in the identification of 182 races worldwide (0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 15, 16, 17, 18, 19, 22, 23, 31, 32, 36, 38, 39, 47, 52, 54, 55, 64, 65, 66, 67, 69, 73, 81, 83, 87, 89, 95, 96, 101, 102, 103, 105, 115, 119, 121, 127, 128, 129, 130, 131, 132, 133, 134, 135, 136, 137, 139, 141, 183, 192, 193, 195, 201, 209, 231, 233, 256, 257, 261, 264, 272, 288, 290, 292, 295, 298, 304, 320, 321, 328, 336, 337, 384, 385, 357, 385, 388, 392, 393, 401, 407, 448, 449, 453, 457, 465, 467, 469, 485, 513, 515, 517, 520, 521, 523, 525, 529, 535, 581, 585, 593, 591, 598, 613, 615, 631, 639, 641, 643, 645, 647, 651, 653, 707, 775, 833, 903, 905, 931, 935, 989, 1025, 1032, 1033, 1049, 1088, 1089, 1093, 1097, 1153, 1161, 1165, 1217, 1344, 1417, 1431, 1433, 1435, 1472, 1473, 1481, 1489, 1497, 1545, 1549, 1561, 1600, 1601, 1609, 1645, 1673, 1677, 1741, 1929, 1945, 1985, 1993, 2001, 2009, 2047, 2560, 2690, 2816, 3195, 3632, 3481, 3545, 3977, 3993) depicting the high pathogenic variability in the pathogen population (Table 3). Race 0 does not cause infection on any of the 12 differential cultivars but is present in Mexico, France and India (González-Chavira *et al.*, 2004; Sharma *et al.*, 2007). However, race 0 infects other bean cultivars, so there is a need to include a susceptible cultivar in the differential set that does not possess any major anthracnose resistance

Table 2. Anthracnose differential series, host gene pool, resistance genes, and the binary number of each cultivar used to characterize races of anthracnose in common bean.

Differential cultivar	Gene pool	Host genes	Binary number
michelite	Middle American	Co-11	1
MDRK	Andean	Co-1	2
Perry Marrow	Andean	$Co\text{-}1^3$	4
Cornell 49242	Middle American	Co-2	8
Widusa	Andean	$Co\text{-}1^5$	16
Kaboon	Andean	$Co\text{-}1^2$	32
Mexico 222	Middle American	Co-3/Co-9	64
PI 207262	Middle American	$Co\text{-}3^3$, $Co\text{-}4^3$	128
TO	Middle American	Co-4	256
TU	Middle American	Co-5	512
AB 136	Middle American	Co-6, co-8	1024
G 2333	Middle American	$Co\text{-}4^2$, $Co\text{-}5^2$, Co-7	2048

Binary number 2n, where n is equivalent to the place of the cultivar within the series. The sum of the cultivars with susceptible reactions gives the binary number of a specific race (Pastor-Corrales, 1991). For example, race 73 is virulent on Mexico 222 [64], Cornell 49242 [8], and Michelite [1].

Table 3. *Colletotrichum lindemuthianum* races present in various countries across the world.

Country	Number of isolates used	Races	Total number of races	References
Brazil	474	0,1, 4, 5, 7, 8, 17, 21, 23, 31, 38, 52, 55, 64, 65, 66, 67, 69, 71, 72, 73, 75, 77, 79, 81, 83, 85, 86, 87, 89, 93, 95, 96, 97, 101, 102, 103, 105, 109, 111, 117, 119, 121, 123, 125, 127, 131, 193, 217, 249, 320, 321, 337, 339, 343, 351, 453, 581, 585, 2047	60	Pinto *et al.,* 2012; Silva *et al.,* 2007; Goncalves-Vidigal *et al.,* 2008; Thomazella, 2002; Somavilla and Prestes, 1999; Ribeiro *et al.*, 2016
Turkey	51	1, 2, 6, 8, 17, 32, 47, 54, 55, 64, 96, 102, 130, 141, 231, 233, 515, 520, 585, 641, 833, 1032, 1153, 1165, 1344, 1472, 1601, 1929, 1993, 2047, 2560, 2690, 2816, 3195, 3632, Alfa, beta, Gamma, Delta	39	Madakbas *et al.,* 2013; Alam and Rudolph, 1993
USA	491	0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 12, 16, 17, 18, 19, 23, 38, 39, 65, 73, 87, 89, 129, 130, 131, 132, 133, 134, 136, 137, 183, 192, 201, 290, 295, 393, 407, 513, 989, 1032, 1153, 1161, 1481, 1993, 65-Epsilon	117	Goswami *et al.,* 2011; Awale *et al.,* 2008; Ansari *et al.,* 2004; Balardin and Kelly, 1997; Kelly *et al.,* 1994; Sicard *et al.,* 1997b; Pastor-Corrales *et al.,* 1993
Greece	35	2, 6, 22	3	Bardas *et al.,* 2007
Colombia	234	0, 1, 2, 3, 4, 5, 6, 7, 9, 11, 15, 17, 23, 31, 36, 38, 39, 47, 64, 65, 73, 81, 87, 89, 121, 128, 129, 131, 132, 133, 135, 133, 137, 139, 192, 256, 257, 261, 320, 385, 388, 393, 448, 449, 453, 457, 513, 515, 517, 521, 523, 525, 529, 535, 593, 641, 643, 645, 647, 651, 653, 905, 1025, 1033, 1049, 1088, 1089, 1093, 1097, 1153, 1161, 1217, 1417, 1433, 1435, 1473, 1481, 1489, 1497, 1545, 1549, 1561, 1609, 1645, 1929, 1945, 1985, 1993, 2001, 2009, 2047, 3481, 3545, 3977, 3993	95	Mahaku and Riascos, 2004; Santana and Mahaku, 2002; Tamayo-Molano *et al.,* 1995
Bulgaria	34	2, 6, 22, 54, 81	3	Kiryakov, 2000; Kiryakov and Genchev, 2004
Spain	115	3, 6, 7, 19, 38, 102	6	Ferreira *et al.,* 2008; Fernandez *et al.,* 2000; Ferreira *et al.,* 1998
India	175	0, 1, 2, 3, 17, 39, 73, 83, 101, 103, 115, 119, 130, 131, 195, 513, 515, 521, 529, 537, 547, 551, 581, 585, 591, 598, 613, 615, 631, 639, 643, 647, 707, 775, 903, 931, 935, Alfa Brazil, Beta, Gamma, IndI, IndII, IndIII, IndIV, IndV, IndVI (Alfa-Brazil), IndVII, IndVIII, IndIX	49	Sharma *et al.,* 2007; Sharma *et al.,* 1999
Mexico	284	0, 1, 2, 7, 8, 9, 17, 19, 55, 64, 65, 73, 81, 89, 96, 128, 193, 201, 209, 256, 257, 264, 272, 288, 292, 298, 304, 320, 321, 328, 336, 337, 357, 384, 392, 448, 449, 453, 457, 465, 467, 469, 521, 833, 1033, 1088, 1165, 1344, 1431, 1472, 1545, 1600, 1601, 1673, 1677, 1741, 1929, 1993	57	González-Chavira *et al.,* 2004; Gonzalez *et al.,* 1998; Balardin *et al.,* 1997; Sicard *et al.,* 1997a; Rodríguez-Guerra *et al.,* 2003
Kenya	4	485	1	Ombiri *et al.,* 2002
Africa	12	9, 69, 87, 384, 385, 401, 448, 449, 485	9	Bigirimana *et al.,* 1999, 2000
Canada	44	Alfa Brazil	1	Tu, 1994
	1590[$]			

[$] Total number of isolates.

gene. Other studies suggest high pathogenic variability within the isolates of a race, for instance, high variability exists within isolates of race 65 in Brazil (Ishikawa *et al.*, 2008; Davide and Souza, 2009; Ishikawa *et al.*, 2011). A few races are present in a particular region, whereas some are prevalent in many countries. For instance, race 73 is prevalent in the USA, Canada and Mexico (Kelly *et al.*, 1994), race 65 in Brazil (Ishikawa *et al.*, 2011; Pinto *et al.*, 2012) whereas races 903, 931, 935 are only present in India (Sharma *et al.*, 2007). Races present in the USA have coevolved with the bean gene pools (races present in the Andean region only infect Andean bean cultivars and the races present in Mesoamerica infect Mesoamerican bean cultivars). However, coevolution between *C. lindemuthianum* and *P. vulgaris* does not exist outside host gene pools, as the majority of races present in India infect beans in both the gene pools (Sharma *et al.*, 1999, 2007). Evolutionary models based on the combination of phenotypic and molecular data have revealed the existence of many evolutionary routes in *C. lindemuthianum* populations (Alzate Marin *et al.*, 1999; Padder *et al.*, 2009). Both these models predicted the presence of additional virulence factors in their respective regions.

Among the 12 differential cultivars, eight are from the Mesoamerican gene pool, while four belong to the Andean gene pool. Andean differential cultivars contain *Co-1* locus whereas Mesoamerican lines contain *Co-2* to *Co-11* genes (Table 2), with majority of them dominant except *Co-8*. Comprehensive information is available on resistance genes present in the differential set (Kelly and Vallejo, 2004; Ferreira *et al.*, 2013). Besides the genes present in the differential set, there are major resistance genes such as *Co-12*, *Co-13*, *Co-14*, *Co-15*, *Co-16* and *Co-17* present in different Andean and Mesoamerican genotypes (Vidigal Filho *et al.*, 2007; Goncalves-Vidigal *et al.*, 2008, 2009, 2012; Coelho *et al.*, 2013; Sousa *et al.*, 2015). In addition to numbered Co genes, there are also *Co-w*, *Co-x*, *Co-y* and *Co-z* genes present in different cultivars (Geffroy *et al.*, 1999, 2008). Since not all the anthracnose resistance genes are present in the differential set, there is compelling evidence to revise the current differential set and include all major anthracnose resistance genes. An effort to develop

near isogenic lines (NIL) carrying all resistance genes in a susceptible background would be helpful in devising management strategies for different countries.

MOLECULAR DIVERSITY AND POPULATION GENETICS

Disease management is proportional to population dynamics and understanding different causes of pathogen variability and population genetics is essential. Phenotypic markers do not infer how pathogen population is structured in a particular region because of their low abundance and many other disadvantages. To overcome these problems, molecular markers offer better insights about the population genetics and various evolutionary forces that are shaping a pathogen population in a particular region. DNA-based markers have many advantages over phenotypic markers and prove valuable to understand the population biology of almost all phytopathogenic fungi. Many excellent reviews on the role of molecular markers in deducing diversity in phytopathogens exist and are useful for further readings (Michelmore and Hulbert, 1987; Majer *et al.*, 1996; McDonald, 1997). RAPDs, ISSR, RFLP, PCR-RFLP and ITS sequencing were used among various markers for deducing variability in *C. lindemuthianum* (Balardin *et al.*, 1997; Balardin and Kelly, 1998; Mahuku and Riascos, 2004; Bardas *et al.*, 2007; Padder *et al.*, 2007; Sharma *et al.*, 2007; Silva *et al.*, 2007; Bardas *et al.*, 2009; Kachapulula *et al.*, 2010). The first molecular analysis of *C. lindemuthianum* was carried out by Fabre *et al.* (1995). They used three different markers (RAPD, RFLP and PCR-RFLP) to infer the genetic variability among different pathogen isolates collected from diverse regions. Most of the *C. lindemuthianum* diversity studies have shown high variability in the pathogen without congruence of phenogram with pathogenicity of the region, suggesting the pathogen has not evolved in a specific gene pool. However, a few studies have shown positive correlation of dendrogram with pathogenicity traits. Comparing regional studies to build up an international perspective of *C. lindemuthianum* population dynamics would be worthwhile, but unfortunately has not proved possible. Population genetic study of *C. lindemuthianum* suggested Mesoamerica as the origin of pathogen (Sicard *et al.*, 1997a, 1997b; Ansari *et al.*, 2004).

All the diversity and population genetic studies in *C. lindemuthianum* are based on RAPD as dominant marker. Numerous disadvantages with RAPD technique have made it obsolete and new markers, especially SSRs, are being preferred as the marker of choice. Unfortunately, to date no SSR marker system is available for *C. lindemuthianum*. However, SSR markers developed for many *Colletotrichum* species (Ranathunge *et al.*, 2009; Ciampi *et al.*, 2011; Moges *et al.*, 2016) have proved valuable in understanding their population genetics. With the advancement of new sequencing tools, particularly next gene sequencing, at low costs, it is easy to develop SSR markers for *C. lindemuthianum* that may be used to infer population structure at regional and global level. These SSR markers will certainly help in deducing the population genetics of pathogen.

ROLE OF GENOME WIDE ASSOCIATION STUDIES (GWAS) AND BEAN SNP CHIP IN DISSECTING BEAN-ANTHRACNOSE PATHOSYSTEM

Plant disease resistance is either simple (major gene) or complex (quantitative) in inheritance. The former provides complete or near complete resistance through direct or indirect interaction between the pathogen effector and R gene encoding proteins, but the loss of the pathogen recognition target makes the resistance ineffective. Conversely, quantitative resistance is more durable, as a pathogen strain that overcomes a single allele with minor effect does not leave the host completely susceptible. Various bi-parental populations used for inheritance and allelism tests against anthracnose have shown presence of more than 20 major resistance genes in different bean genotypes. The major anthracnose resistance loci have been mapped to Pv01, Pv02, Pv03, Pv04, Pv07, Pv08, Pv09 and Pv11 bean chromosomes (Geffroy *et al.*, 2008; Ferreira *et al.*, 2013; Meziadi *et al.*, 2016; Zuiderveen *et al.*, 2016). In addition to these major genes, nine anthracnose QTLs were identified in nuna bean PHA1037 against races 23 and 1545 of the pathogen (Oblessuc *et al.*, 2014). The identification of additional resistance specificities in anthracnose resistant genotypes through classical bi-parental population analysis and afterwards allelism tests is time-consuming. GWAS are complementary to bi-parental analysis and can identify traits of economic importance in the crops. GWAS take advantage of natural variation in the population accumulated during historic recombination. GWAS provided comprehensive insights to identify complex traits in both model and non-model plants. The availability of bean genome sequence (Schmutz *et al.*, 2014) and SNP markers in BARCBean6K_3 BeadChip (Hyten *et al.*, 2010; Song *et al.*, 2015) has given fresh impetus to the bean scientific community to map R genes. The SNP chip has resulted in the fine mapping of many resistance sources including: *Co-x* (Richard *et al.*, 2014), *Co-1* (Zuiderveen *et al.*, 2016) *Co-1*^{2} (Vazin, 2015) and the *Co-4*^{2} (Oblessuc *et al.*, 2015) and the discovery of new genomic regions and candidate genes associated with anthracnose resistance (Gonzalez *et al.*, 2015). The *Co-x* gene was fine mapped to Pv01, independent of the *Co-1* locus, and to a syntenic region, located at one end of soybean (*Glycine max*) chromosome 18 that carries *Rhg1*, a major gene conditioning resistance to soybean cyst nematode (Richard *et al.*, 2014). Fine mapping of the *Co-4* (COK-4) locus to Pv08 revealed 18 copies of the COK-4 gene in a 325 kbp segment of that chromosome (Oblessuc *et al.*, 2015). Andean bean diversity panel (Cichy *et al.*, 2015) was recently used to identify extra anthracnose

resistance specificities and new genomic regions involved in bean anthracnose resistance (Zuiderveen *et al*., 2016). The SNP chip currently available to the bean consortium has resulted in its use in the US, but the recent publication of a second bean genome by an international consortium (Vlasova *et al*., 2016) should hasten additional research on the *P. vulgaris*-anthracnose pathosystem. In addition to the SNP chip, RNAseq based transcriptome analysis has been used to study the interaction between common bean NIL pair that differs at *Co-1* locus following infection with race 73 of *C. lindemuthianum* (Padder *et al*., 2016). Differentially expressed transcripts adjacent to the *Co-1* locus suggest the global reprogramming in the host.

CONCLUSION AND FUTURE THRUST

Studies on pathogen biology and variability at morphological and molecular level have certainly improved our understanding about the fungus structure and its population genetics. Similarly, host-pathogen dialogue between bean and *Colletotrichum* advanced our understanding about defence mechanism, resistance genes that are expressed during the interaction with the host. The identification of 182 bean anthracnose races following inoculation of 12-member differential set with more than 1500 isolates illustrates the high pathogenic variability of *C. lindemuthianum*. Many differential genotypes carry more than one anthracnose resistance gene. There is a need to monitor the prevalence of new isolates for virulence and improve the differential set by developing NIL lines carrying a single anthracnose resistance gene similar to the rice blast pathogen (Telebanco-Yanoria *et al*., 2010; Telebanco-Yanoria *et al*., 2011)

We know fungi secrete a plethora of effector proteins for successful colonization of the host tissue. Studies showed that *Colletotrichum* species also produce effectors, but how many of them are secreted by bean anthracnose fungus is not known (Tang *et al*., 2006; O'Connell *et al*., 2012; Alkan *et al*., 2013; Schliebner *et al*., 2014). Effectors produced by *Colletotrichum* species are cysteine-rich but many of the effectors reported do not share homology with known effectors. The use of next generation sequencing platforms has resulted in genome sequencing at affordable costs and has increased the genomic resources available to study *Colletotrichum* (Gan *et al*., 2013; Baroncelli *et al*., 2014, 2016; Han *et al*., 2016). In Brazil, genome sequencing of races 83 and 89 is in progress (http://www.colletotrichum.org/genomics/) and access to this information should boost bean/anthracnose research in the years to come. Availability of these resources to the scientific community (http://www.colletotrichum.org/genomics/) needs to be mined for secretome and development of better marker systems such as SSRs and SNPs for discerning the population structure and for identification of evolutionary forces that shape the trajectory of the fungus at both the international and regional level.

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