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Achievements and Prospects of Common bean (*Phaseolus vulgaris* L.) Breeding for Disease Resistance: A Review

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Abstract

Common bean (*Phaseolus vulgaris* L.) is an important legume crop that provides a significant source of protein and nutrients to a large part of the world's population. However the yield and qualities of common bean are often compromised by various diseases caused by pathogens such as fungi, bacteria and viruses. Breeding for disease resistance is a sustainable and cost effective approach to mitigate the impact of these diseases. This review paper aims to discuss the achievements and prospects of common bean breeding for disease resistance, including the identification of resistance source, and future prospects. The importance of incorporating diverse resistance genes and the utilization of modern breeding techniques to enhance disease resistance in common bean varieties highlighted.

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Common bean, gene pyramiding, genetic diversity, host-pathogen interaction.

Introduction

The common bean (*Phaseolus vulgaris* L.) is the most important legume directly used for human consumption globally. It is consumed worldwide for its edible seeds and pods. It is the most important source of proteins for nearly five hundred million people in Africa, Latin America and the Caribbean particularly for low income earning households.

Among the five domesticated and grown species of the genus Phaseolus, a genus comprised of some 70 species (Freytag and Debouck, 2002), *P. vulgaris* accounts for more than 90% of the cultivated crop in the world (Singh *et al.*, 2001). The immediate ancestors of the cultivated common bean (2n = 2x = 22) are wild populations distributed from northern Mexico to northern Argentina (Gepts, 1998; Koenig *et al.*, 1990). There are two types of common bean: those used for the dry and green-shelled seed and green, garden, snap, or string less bean in which pods are consumed (Myers and Baggett, 1999; Singh, 1992).

Production Constraints

Abiotic Constraints

Abiotic stress is the major constraint to bean productivity in most tropical countries. Abiotic factor such as extreme limited water stress (drought) cause yield loss in Mexico, Brazil, Central America, and Eastern and Southern Africa. Heat stress adversely affects the cultivation of beans in Central and Southern America and Africa (Beebe *et al.*, 2011). Nutrient deficiencies of phosphorous (P) and nitrogen (N) also reduces yield, while Aluminum and Manganese toxicity associated with

acid soil, as well as low Calcium availability, cause significant common bean yield loss (Table 1).

Biotic Constraints

Biotic stresses such as diseases and pests are universal constraints to common bean production, especially fungal pathogens. Several bacterial, fungal, and viral diseases attack aerial and underground parts of common bean (Schwartz *et al.,* 2005). Under favorable disease conditions, fungal pathogens cause significant yield losses. Yield losses also occur due to insect damage (Table 2).

Anthracnose, rust, and angular leaf spot are widely distributed, while rhizoctonia web blight and ascochyta blight can be locally intense in warm-moist environments, respectively. In recent years, root rots have emerged as a significant problem for common bean production, especially those caused by *Pythium* spp. and *Fusarium* spp. Insects are occasional problems. In Central America the bean pod weevil, *Apion godmani* and *A. aurichalceum*, is the most important pest, while in East Africa the bean stem maggot, aphids, and pod borers cause the most serious problems.

Fungal Diseases

Anthracnose

Anthracnose caused by the fungal pathogen *Colletotrichum lindemuthianum* is a serious seed-borne disease of common bean and can cause devastation to farmers' fields, resulting in yield losses as high as 100% in susceptible cultivars (Singh and Schwartz, 2010). This pathogen overwinters in seed and crop residues (primary source of infection) and infects all aerial parts of the bean plant. Recent literature shows presence of more than 100 races of pathogen worldwide (Gonzalez *et al.*, 2015), and 1590 isolates of *C. lindemuthianum* inoculated on 12 bean differential cultivars have resulted in the identification of 182 races worldwide.

Cultivating resistant varieties is the most economical and effective approach for controlling common bean diseases and is an important goal for common bean breeders. Currently, more than 20 resistance loci are identified against anthracnose disease with a gene symbol *Co* (Coimbra-Gonçalves *et al.,* 2016). Incorporating genetic resistance to anthracnose is the area of research and development that holds the most promise for reducing the effects of the pathogen in common bean. Careful selection of genes providing resistance to races producing anthracnose disease is required. The varieties carrying resistance genes provide a short-lasting control over the disease lasting only until new strains of fungus emerge. The major difficulty with the process of compiling the resistance genes is that detection of the resistant plants takes time since it requires systematic inoculation with different strains of the fungus. A literature review indicates that in other countries where cultivation is common, strains of the common fungi have been identified, and the varieties resistant to the identified strains have been developed.

MAS have been used successfully to breed for enhanced resistance to anthracnose in the cultivar Perola in Brazil (Ragagnin *et al.,* 2003) and in pinto beans in the United States (Miklas *et al.,* 2003b). Genchev *et al.,* (2010) developed anthracnose resistance source using several physiological races of anthracnose identified in Bulgaria which was performed from F1 to F5 generation. The line DG 2-36-58-3 was identified as the most promising by quality complex of growth habit type, vegetation period, type of seeds, yield and presence of *Co-1* and *Co-4* genes that confer resistance to 74 out of 78 worldwide recognized anthracnose races. Achieving durable anthracnose resistance poses a challenge to bean breeders.

Due to the high degree of pathogen variability and the continual emergence of new races, single-gene deployment is not an effective strategy to control bean anthracnose. The pyramiding of resistance genes which have complementary spectra of resistance has been suggested as a strategy to circumvent the problem of pathogen variability.

Angular Leaf Spot (ALS)

Angular leaf spot (ALS) disease caused by *Pseudocercospora griseola* (Sacc.) is a major disease of common beans in the tropics and subtropics (Stenglein *et al.,* 2003). Studies on the variability of *P. griseola* isolates revealed the existence of two major groups of the pathogen, Andean and Mesoamerican, which correspond to and have co-evolved with the Andean and Mesoamerican gene pools of common bean (Crous *et al.*, 2006). Mesoamerican strains of this pathogen are considered more virulent as compared to Andean strains, and they tend to affect both Mesoamerican and Andean beans, while Andean strains are less virulent, affecting mostly Andean genotypes. The disease is of great economic importance in Eastern and Central African

countries of Uganda, Kenya, Tanzania, Ethiopia, Rwanda, Burundi and Kivu Province of the Democratic Republic of Congo (Pastor-Corrales *et al.,* 1998). According to Stenglein *et al.,* (2003), every 10% increase in ALS severity results in 7.9% yield loss. ALS disease is spread within and among fields by wind-blown particles of infested soil and wind-blown and rain-splashed spores.

However, the primary source of infection is considered to be infested seed. Resistance to ALS in common bean is controlled by single dominant (Correa *et al.,* 2001; Namayanja *et al.,* 2006) as well as recessive genes (Correa *et al.,* 2001). As the best form of disease control includes using resistant cultivars, the genetic characterization of resistance sources is very important for the genetic improvement of the crop. In the case of ALS, two dominant resistance genes have been described so far.

The first, called *Phg-1*, was identified in the AND 277 variety. While, the second, called *Phg-2*, was identified in the Mexico 54 varieties (Sartorato *et al.,* 2000) linked to SCAR OPN02 and RAPDOPE04 markers. Apart from these two genes, dominant monogenic inheritance for resistance to ALS has also been described in the Ouro Negro (Correa *et al.,* 2001) and G10474 varieties. But the relationship of these genes with *Phg-1* and *Phg-2* remains unknown. In addition to qualitative resistance genes, there are also reports of QTLs controlling resistance to ALS. Five QTLs were mapped on linkage group B04, one on B08, one on B09 and three on linkage group B10 (Lopez *et al.,* 2003; Mahuku *et al.,* 2009; Mahuku *et al.,* 2011). The Mesoamerican ALS resistance locus, *Phg-3*, mapped in accession Ouro Negro, is linked to marker G2303 at a distance of 0 cM (Gonçalves-Vidigal *et al.,* 2013).

Singh *et al.,* (2003) developed ALS resistant dry bean breeding lines including A 339, MAR 1, MAR 2 and MAR 3 from interracial populations between the Middle American common bean races. Mahuku *et al.,* (2003) identified 78 interspecific dry bean lines with resistance putatively transferred from the secondary gene pool, which represents important germplasm for future utilization. Traditional breeding at CIAT involving hybridization among resistance sources in single or multiple interracial crosses followed by selection under disease pressure in field nurseries and greenhouse screening trials has resulted in development of germplasm lines MAR1, MAR 2, MAR 3, AND 277 and CAL 143 with improved broad-based resistance to angular leaf spot (Singh *et al.,* 2003; Aggarwal *et al.,* 2004). Pyramiding resistance genes into a single genotype is one of the practical approaches through which durable resistance can be achieved. There are several *Phg* genes that have been identified as sources of resistance to *P. griseola* from landraces, secondary and tertiary genes pools. However, utilization of these genes in a breeding programme will depend on the mode of inheritance and the background of the cultivar carrying them.

Landrace varieties might be excellent sources for resistance breeding against ALS. Landraces are readily available, adapted to the environments and have been kept by farmers because of their desired traits. In this regard, breeding against multi-races of ALS disease is an overriding consideration which requires gene pyramiding that involves several parents. Therefore, appropriate mating design and genetic analysis that will provide information of the best parent in a combination and best selection methods to identifying superior progenies is important.

Powdery Mildew

Powdery mildew is a serious disease for many crops worldwide including common bean. Common bean powdery mildew causal agent has been frequently ascribed to *Erysiphe poligony* DC. But recent studies suggest that it is closer to *Erysiphe diffusa* (Cooke and Peck) U. Braun and S. Takam, formerly *Microsphaera diffusa* Cke. and Pk. (Almeida *et al.,* 2008). Limited information about sources of resistance to the fungus and the nature and inheritance of resistance are available to bean breeders and plant pathologist.

A few sources of resistance to powdery mildew have been described (Schwartz *et al.,* 1981), and a qualitative nature of resistance has been suggested (Ferreira *et al.,* 2001). The response of common bean to powdery mildew was previously reported to be governed by a single dominant gene (Dundas, 1936), by one dominant and other recessive resistance genes (Bett and Michaels, 1995) or by two complementary dominant genes. Sources of resistance to PWM have also been described (Schwartz *et al.,* 1981) including "Cornel 49, 242, Porrillo Sintetico, Negro San Luis and ESAL 686" cultivars (Rezende *et al.,* 1999; Trabanco *et al.,* 2012; Perez-Vega *et al.,* 2013). Much of these sources are characterized by possessing a few genes involved in the trait with different patterns of action. The genetic positions of resistance genes were first investigated in

Xana/Cornell 49,242 recombinant inbred line population. Results showed that the resistance in the genotype Cornell 49,242 was conferred by two independent and epistatic genes: *Pm1*, located in linkage group (LG) Pv11 conferring total resistance in which there is no visible disease symptoms, and *Pm2*, located in LG Pv04, conferring intermediate resistance with a very limited disease development (Perez-Vega *et al.*, 2013). From a breeding perspective, molecular-genetic maps and QTL mapping are tools that allow the localization of some genomic regions that control both single and complex inheritance. This information could be used in breeding programmes for producing new cultivars for resistance to powdery mildew.

Rust

Among the fungal diseases, one of the most a widespread and important disease of common bean is rust, caused by a highly variable basidiomycete fungus *Uromyces appendiculatus* (Pers.Pers.) unger which has a narrow host range -attacks only common bean. This disease is distributed throughout the world, but it effectively causes major production problems in humid tropical and subtropical areas and periodic severe epidemics in humid temperate regions (Pastor-Corrales, 2003). Severe bean rust epidemics have been reported in Australia, China, the United States and some areas of Europe. Major losses have occurred in Burundi, Ethiopia, Kenya, Malawi, Rwanda, South Africa, Tanzania, Uganda and Zimbabwe. In Latin America, the bean rust is also a serious problem; major losses occurred in Argentina, Bolivia, Brazil, Colombia, Costa Rica, Cuba, Dominican Republic, Ecuador, El Salvador, Guatemala, Haiti, Honduras, Jamaica, Mexico, Nicaragua and Peru. Disease losses worldwide measured in greenhouse and field conditions can vary from 18 to 100%. The *U. appendiculatus* is a highly variable and is among the most pathogenically variable of all plant pathogens.

It has been identified and reported in all bean production areas of the world (Stavely and Pastor-Corrales, 1989) and is characterized by highly diverse virulence phenotypes (Souza *et al.*, 2005). According to Lindgren *et al.*, (1995), 1% increase in bean rust severity leads to a yield loss of approximately 19 kg/ha. Resistance to bean rust is mainly controlled by major single dominant genes (Souza *et al.*, 2007). However, it can be also controlled by single recessive genes (Zaiter *et al.*, 1989), two genes (Finke *et al.*, 1986), two complementary dominant genes (Grafton *et al.*, 1985) or by many genes with minor effect (Edington *et al.*, 1994). According to Souza *et al.*, (2011), 13 dominant rust resistance (RR) genes (*Ur-1* to *Ur-13*) have been identified. In addition to these genes, other important unnamed genes have been identified, such as those present in the common bean cultivars as BAC6, CNC, CSW 643, Dorado, Ouro Negro and PI 260418.

Bacterial diseases

Common Bacterial Blight (CBB)

Common bacterial blight (CBB) is a significant foliar disease of dry bean caused by the pathogen *Xanthomonas axonopodis* pv. *phaseoli*, a gram-negative bacillus with a genome of approximately 3.9 Mb. The bacteria are seed-borne, and under field conditions, dissemination can occur through wind-driven rain or mechanical transfer by insect vectors. In addition to natural methods, the bacteria can be spread by overhead sprinkler systems and the use of infected seeds (Singh and Schwartz, 2010). Studies of the genetic diversity of *X. axonopodis* pv. *phaseoli* have found that the pathogen can be grouped into four genetic lineages. Three of these groups are composed exclusively of *X. axonopodis* pv. *phaseoli*, while the last group contains the *X. fuscans* subsp. *fuscans* strains (Alavi *et al.*, 2008).

Table.1 A schematic comparison of different bean production limitation, classified for their frequency, likely intensity, and risk to farmers.

Limitation	Frequency	Intensity	Risk
Pest and diseases	high	High	Very high
Drought	low	Very high	
Low soil fertility	Very high	High	Very low
High temperature	Very high	High	Very low

Source: Adapted from Beebe *et al.*, 2006.

Table.2 Summary of major diseases attacking common bean, yield loss potential, and seed transmissibility and survival

Common Name	Organism	Yield loss potential	Transmission and survival
Angular leaf spot	Phaeoisariopsis griseola	80%	Seed, wind, plant debris
Anthracnose	Colletotrichum lindemuthianum	100%	Seed, wind, plant debris
Bacterial brown spot	Pseudomonas syringae pv. Syringae	25%	Seed, wind, water, plant debris
BCMV	Bean common mosaic virus	100%	Seed, plant debris, aphid
BCMNV	Bean common mosaic necrosis virus	100%	Seed, plant debris, aphid
BCTV	Beet curly top virus	100%	Leafhopper (Circulifer tenellus)
BGMV	Bean golden mosaic virus	100%	Whitefl y (Bemisia spp.)
BGYMV	Bean golden yellow mosaic virus	100%	Whitefl y (Bemisia spp.)
Common bacterial blight	Xanthomonas campestris (axonopodis) pv. Phaseoli	45%	Seed, wind, water, plant debris
Halo blight	Pseudomonas syringae pv. Phaseolicola	45%	Seed, wind, water, plant debris
Root rots	Aphanomyces, Fusarium, Pythium, Rhizoctonia, Thielaviopsis species	100%	Seed, wind, water, plant debris
Rust	Uromyces appendiculatus	50%	Wind, plant debris
Web bligh	Thanatephorus cucumeris	100%	Seed, wind, water, plant debris
White mold	Sclerotinia sclerotiorum	90%	Seed, wind, water, soil, plant debris

Source: Singh and Schwartz, 2010

Table.3 Summary of bean fungal, bacterial and viral disease distribution, transmission and resistance genes (Teshale *et al.*, 2019)

Disease	Distribution	Seed	Transmission	Resistance
Fungi				
Anthracnose (*Colletotrichum*)	Worldwide (cool = 14–18 °C, humid, and >1000 m in tropics	Yes	Wind and rain; animal and insect	QTL: Pv01, Pv02, Pv04, Pv10, Co-1, Co-2
Angular leaf spot (*Phaeoisariopsis*)	Worldwide (moderate temp.18–25 °C, high moisture)	Yes	Wind	QTL: Pv04, Pv10 (ALS10.1) Phg1, Phg2
Rust (*Uromyces*)	Worldwide	No	Wind, crop and residue	KASP SS68 marker associated with Pv11
Leaf spot (*Ascochyta*)	Tropics (high altitude: >1500 m)	Yes	Splash, contact, crop residue	Quantitative (*P. polyanthus*)
Pythium root rot	Worldwide	Yes	Crop residue, infected soil	QTL: ER3XC (LG3), SV6XC(LG6), Py-1 (LG7)
Fusarium	*Worldwide*	*No*	*Crop residue, infected soil*	*QTL (FRR3.1 km (Pv03), LGs B2 and B3*
Web blight	Worldwide (hot-humid)	Yes	Infected soil, seed	Quantitative
Bacteria				
Common blight (*Xanthomonas*)	Worldwide (cool to moderate temp.)	Yes	Rain, moisture	QTL: BC420 (Pv06), SU91 (Pv08), Xa11.4OV1(Pv11)
Halo blight (*Pseudomonas*)	Worldwide (cool to moderate temp.)	Yes	Rain, moisture	Quantitative: Pse-1, Pse-2, Pse-3, Pse-4, Pse-6, HB5.1 98BB, 98LFA, 96LFA, 96BBS
Bacterial brown spot (*Pseudomonas*)	Worldwide (warm-humid weather)	Yes	Seed, wind	LG02
Viruses				
Bean common mosaic virus and mosaic necrosis virus	Worldwide	Yes	Aphid	Qualitative: bc-1, bc-2, bc-3
Bean golden yellow mosaic virus	Central America, Caribbean	No	Whitefly	Qualitative, quantitative, Bgm-1, RNAi

Several inheritance studies have been conducted on CBB resistance, and different results were reported depending on various factors such as the pathogenic variability and the genetic background of the parental lines (Fourie *et al.,* 2011). CBB resistance is inherited quantitatively, with largely additive effects, low to moderately high heritability and transgressive segregation (Aggour and Coyne, 1989; Arnaud-Santana *et al.,* 1994). Similarly, Miklas *et al.,* (2003a) reported that the inheritance of CBB resistance in Montana No. 5 was polygenic with at least one major gene effect. Likewise, Zapata *et al.,* (2011) reported that CBB resistance was governed by a single dominant gene in resistant lines Wilk-2 and VAX6 and VAX4 and PR 0313-58, respectively. The identification of major quantitative trait loci (QTL) controlling resistance to CBB (Singh and Miklas, 2015) has facilitated marker-assisted breeding for higher levels of CBB resistance into better-adapted and higher-yielding dry bean lines (Miklas *et al.,* 2000). CBB-tolerant/resistant dry bean varieties have been developed from germplasm derived from interspecific crosses by various breeding programs in North America. Singh and Muoz (1999) and developed CBB-resistant interspecific breeding lines VAX1 and VAX 2 from a multiple-parent interspecific cross between common and topiary bean G 40001.

The substantial progress made in molecular marker technology for the common bean holds considerable promise for breeding genetic resistance to CBB. Molecular markers for disease resistance are powerful tools for analyzing the genome and are comprehensively applied in mapping genes and MAS (Boyle *et al.,* 2007). To date, 24 QTL conferring resistance to CBB have been identified, distributed across all 11 chromosomes of common bean (Singh and Schwartz, 2010; Shi *et al.,* 2011).

Halo Blight (HB)

It is an important seed-borne disease of dry beans caused by *Pseudomonas syringae* pv. *phaseolicola* (Burkh.) Dows. (*Psp*). The disease is a major constraint of dry bean production in moderately cool and wet regions of Africa, Europe, North America and South America (Taylor *et al.,* 1996; Rico *et al.,* 2003). The gene-for-gene interaction between common bean and HB pathogen races was demonstrated using molecular techniques and resistant genotypes (c). Nine races of the pathogen and five race-specific resistance genes have been previously described. However, a quantitative response to this pathogen has also been described (Yaish *et al.,* 2006). Several researchers studied genetic inheritance of HB resistance. As per the earlier findings, resistance to HB is inherited by single dominant or recessive genes (Taylor *et al.,* 1996). Taylor *et al.,* (1996) screened over 1000 accessions of *Phaseolus* spp. and identified both race-specific and non-race-specific resistance to halo blight. For example, CAL 143, great northern Nebraska #1 Sel. 27,'Jules', PI 150414 and Wis HBR 72 among others carry non-race-specific resistance. Molecular and genetic studies established that the relationship between the race specific genes and *Psp* races conformed to a gene-for-gene interaction (Jenner *et al.,* 1991). Furthermore, genetic studies have established that inheritance of halo blight resistance depends on the nature of the resistance as well as the genetic background. Chataika *et al.,* (2011) also reported a single dominant resistance gene in the large seeded (40 g per 100 seeds) Andean breeding line CAL 143. On the other hand, the quantitative race non-specific resistance in PI 150414 is due to a recessive gene (Taylor *et al.,* 1996). Miklas *et al.,* also mapped the dominant *Pse-2* gene, which is derived from the differential genotype ZAA 12 and confers resistance to seven *Psp* races (excluding *Psp* races 1 and 6), to linkage group Pv10. Miklas *et al.,* (2009) identified six random amplification of polymorphic DNA (RAPD) markers tightly linked (0–3.3 cM) with *Pse-1* in a 'CanadianWonder'/UI 3 dry bean population of which three completely linked markers were converted into SCAR markers SH11.800, SR13.1150 and ST8.1350. However, usefulness of these SCAR markers for marker-assisted selection would be limited to populations of large-seeded Nueva.

Significant progress has been made in developing cultivars with resistance to various diseases using conventional breeding. Some important resistance-mapping studies are summarized in Table 3. Markers associated with established resistance loci can be used for more efficient breeding to develop resistant cultivars.

Conclusions and Prospects

Breeding common bean for disease resistance is critical for enhancing its productivity and sustainability in the face of various pathogenic threats. Significant advances have been made in breeding common bean for resistance to specific diseases. Molecular marker-assisted selection is being increasingly used for bean improvement. Researchers should therefore strive to combine the best of conventional and modern molecular approaches to improve resistance to multiple diseases and other desirable traits in the shortest time possible in otherwise

high yielding, high quality, and broadly adapted cultivars of different market classes of common bean. Collaboration among breeders, pathologists, and molecular biologists is essential to achieve long term success in common bean breeding for disease resistance.

Modern biotechnological tools, such as genomics selection, gene editing, and transgenic approaches, hold great potential to expedite the development of disease resistant common bean varieties. The application of next-generation sequencing technologies and high-throughput phenotyping can facilitate the identification and characterization of novel resistance genes and QTLs. Integration of multi-omics approaches, such as transcriptomics and metabolomics, can provide a comprehensive understanding of the plant pathogen interactions and aid in the identification of key genes involved in disease resistance.

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