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Review Paper

A comprehensive review on angular leaf spot disease of beans (*Phaseolus vulgaris* L) caused by *Peudocercospora griseola* (Sacc.) Crous & Braun

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ABSTRACT

Cercosporoid fungi are one of the largest and most economically important groups of plant pathogenic fungi causing a variety of leaf spots across diverse crop species including beans and may cause significant yield loss as well as deterioration of pod and seed quality. The widespread occurrence and high pathogenic success of Cercosporoids has been attributed to a highly toxic and photoactivated metabolite, cercosporin, visible in mycelia as well as a high degree of pathogenic variability. Angular leaf spot of beans caused by *Pseudocercospora griseola*, has a broad host range including cultivated bean species, *Vigna*, *Pisum*, Desmodium, and Lablab. Currently, disease is managed by a combination of cultural and chemical management strategies, which is ecologically unsustainable. The resistance to *P. griseola* is governed by both dominant and recessive genes. Currently, six genes namely *Phg-1*, *Phg-2*, *Phg-3*, *Phg-4*, *Phg-5* and *Phg-6* are reported in common bean. The *Phg-1*, *Phg-4*, and *Phg-5* genes are from an Andean origin, and the *Phg2* and *Phg-3* genes are from a Mesoamerican origin. Phg-1 and Phg-2 are dominant resistance genes mapped on chromosomes Pv01 and Pv08 respectively. Various QTLs have also been associated with ALS resistance in beans. The known resistance sources as well as the availability of genetic and genomic resources, coupled with a better knowledge of biology, and pathogenicity have advanced our understanding of angular leaf spot and developed resistant cultivars for a sustainable bean production system, especially in low input small holder farming systems. This review presents updated information on various aspects of disease and research efforts towards effective management.

Key words: Common bean, Cercosporoids, Cercosporin, Genomics, Molecular markers, Gene pyramiding

INTRODUCTION

The current projections estimate the global population to stand at around 9.7 billion by 2050 (FAO 2012). This coupled with the looming threats of climate change implications presents a daunting challenge of ensuring food and livelihood security, especially in developing countries, where farmers have limited adaptive capacities. The crops like common bean (*Phaseolus vulgaris* L.: Phaseolaceae) which are cheaper yet have tremendous food and nutritional value, hold great promise (Broughton *et al.* 2003). The common bean gives better yields under low-input farming systems, can grow on less soil moisture, and can improve soil health by fixing atmospheric nitrogen (Sofi *et al.* 2018). It is one of the most important legume crops, that has originated in Andean and Mesoamerican regions through independent domestication events resulting in modern-day small to large-seeded, easy to cook and non-shattering beans from twining wild vines. Because of two independent domestication events two distinct major gene pools namely Andean and Meso-American types have evolved (Bitocchi *et al.* 2013, Bellucci *et al.* 2014). The common bean, which is used in various forms such as dry, green, and shelled, is one of the most important pulse crops especially in Sub-Saharan Africa and Latin America. On account of its being a cheap source of protein, it is called "Poor man's meat". Global exports of common bean stand at 4.23 million tons (31%), only next to peas among pulses (Rawal and Navarro 2019). Bean is also regarded as a "nearly perfect food" as it contains a balanced mixture of different nutrients that promote better health and

fight certain diseases. It has a rich nutritional profile with about 22.8% proteins, 307 Kcal of energy, 1.6% of fat, and adequate dietary fibers (21.7%). It also has a very low glycemic index (24%) as compared to cereals and is also rich in nutrients such as Iron (8.8 mg/100g). However, global consumption (21 g/person/day) has been stagnant for the last few decades (Rawal and Navarro 2019).

Among all *Phaseolus* species, the common bean (*Phaseolus vulgaris* L.), is the most widely grown and consumed throughout the world (Beebe *et al.* 2012) accounting for more than 85% of *Phaseolus* species. It is an important source of protein in many countries especially in Latin America, the Middle East, Europe, and Africa, where it is consumed in large quantities (Broughton *et al.* 2003). On account of its worldwide distribution, various pathogens including fungi have coevolved with this crop and have assumed potential pest status due to human selection pressure under commercial cultivation and are presently major production constraints of common bean worldwide (Schwartz *et al.* 1989). Among various fungi attacking common bean crop, *Pseudocercospora griseola* (Sacc.) Crous & Braun (previously referred to as *Phaeoisariopsis griseola* Sacc. Ferrari) causes Angular Leaf Spot (ALS) (Miklas *et al.* 2006) is the major fungal disease causing significant economic damage to bean crop with wide occurrence (Figure 1). The severity of ALS is due to the occurrence of the warm humid conditions in bean areas that favor pathogen attack triggered by increased sporulation (Correa-Victoria *et al.* 1989). In Latin America and Africa, the two major dry bean-producing regions of the world, angular leaf spot is currently one of the most common and destructive diseases that affect dry beans (Sartorato 2004, Crous *et al.* 2006). In Africa, yield losses of 50–60% have been documented (Elena *et al.* 2017). There have also been reports of occasional occurrences of angular leaf spots in temperate temperature nations, such as the US and Canada (Melzer *et al.* 2001) and in Spain ALS infestation has also been reported for the first time by Landeras *et al.* (2017). Yield losses by ALS reported in various bean growing areas are 61% in Tanzania (Mongi *et al.* 2018), 55% in Uganda (Pamela *et al.* 2014), 47% in Ethiopia (Lemessa *et al.* 2011), 45-80% in Brazil and Columbia (Guzman *et al.* 1995).

The main sources of inocula for infection caused by ALS diseases are infected seeds, plant waste, and aerial dispersion (Girma *et al.* 2022). Accordingly, management strategies to lessen disease intensities could include the use of healthy seeds, field sanitation, chemicals for seed treatment and spraying, soil amendment, appropriate crop density, and crop rotation (Fininsa and Yuen 2001, Amin *et al.* 2013). However, crop rotation may be difficult due to the lack of suitable crop land and the high cost of chemicals used for seed treatment and spraying, which smallholder farmers may not be able to afford or conveniently obtain. Accordingly, the most effective, economical, and ecologically safe way to manage disease in the production of common beans is to apply genetic resistance (Tumsa *et al.* 2020, Adila *et al.* 2021). Increased distribution of pathogen-free seeds, decreased use of insecticides, reduced production losses, and an integrated disease management plan could all be facilitated by high levels of host resistance (Singh and Schwartz 2010).

Due to the increasing dependence of integrated disease management systems on host plant resistance as well as the widespread cultivation of grain legumes, which supply essential protein, in developing nations where farmers have limited access to resources, multiple disease resistance has gained significance in recent times. To find elite genotypes or sources of resistance that can be employed for production or frequently incorporated into commercial cultivars, it is crucial to assess common bean genotypes under natural infection for diseases (Fininsa and Tefera 2006). This could strengthen resistance in the already-existing resistant cultivars and counteract the recently emerging plant pathogenic races.

DISTRIBUTION AND ECONOMIC IMPACT OF *PSEUDOCERCOSPORA GRISEOLA*

Cercosporoid fungi are one of the largest and most economically important groups of plant pathogenic fungi causing a variety of leaf spots across diverse crop species. They are highly diverse in tropical and subtropical areas of Africa, Asia, Australia, and Central to South America (Braun *et al.* 2016). Among the cercosporoid fungi, *Pseudocercospora griseola* causing angular leaf spot is considered one of the most devastating fungal diseases of common bean in the tropics and subtropics, with significant impact on common bean yield, leading to loss of as high as 80% and is reported in more than 70 countries (Figure 1) in the world (Stenglein *et al.* 2003, Miklas *et al.* 2006). The greater prevalence of ALS in the tropics is largely due to intermittent cool and warm, wet and dry weather (Celetti *et al.* 2006), which favors disease development. The extent of economic loss assessed

across 70 bean-growing countries could be as high as 80 percent (Stenglein *et al.* 2003, Miklas *et al.* 2006), with huge perpetual losses reported from major bean-growing areas such as Brazil (Vieira *et al.* 2006) and African countries such as Uganda (Namayanja *et al.* 2006. However, in temperate bean areas such as Spain, Canada, and the USA, ALS occurs sporadically, but in cases of severe infestation losses up to 80 percent have been reported (Landera *et al.* 2017).

Brazil is the largest consumer of beans in the world with an estimated production of 3.1 million tons (CONAB 2021), ALS there has assumed significant proportions since the 1980s and is now a major problem in common bean production in the country (Stenglein *et al.* 2003, Vieira *et al.* 2006). In the Great Lakes of Africa, it causes an estimated loss of 375000 MT annually (Mahuka *et al.* 2009, Stenglein *et al.* 2003). In Uganda, the yield loss is estimated up to 50% in released varieties due to ALS (Namayanja *et al.* 2006). In temperate climate zones, this disease is reported to occur sporadically (Melzer *et al.* 2001). In Northern Spain, the disease was recently reported for the first time (Landera *et al.* 2017). In Ontario, ALS has been reported on Snap beans and Roma beans resulting in 80% yield loss there. Similarly, Mongi *et al.* (2018) reported that ALS caused a yield loss of up to 61% in Tanzania under heavy rainfall and 37% under light rainfall.

The angular leaf was first identified in beans from various parts of India's Nilgiri Plateau by Srinivasan (1953). Subsequent reports from Uttarakhand (Bose and Sindhan 1972), Sikkim (Srivastava and Gupta 1994), and Himachal Pradesh (Sohi *et al.* 1963) were received. Now the disease frequently manifests in all of the nation's bean-growing regions, albeit to differing degrees (Gupta and Shiyam 2003). The disease typically strikes in late August in Punjab, resulting in early defoliation and negatively affecting seed yield (Sohi and Sharma 1969). In the Himalayan highlands, angular leaf spot is a serious issue that renders 40–70% of the green pods unmarketable (Singh and Saini 1980, Gupta *et al.* 1998). This disease is prevalent across the country, but it is especially devastating in Kashmir, where it destroys a large portion of the harvest each year (Kotwal 1994). In the Western Himalayan Kashmir valley, several authors have studied the incidence of ALS and reported disease incidence of 42.44-60.06% and disease intensity of 25.30-32.75% on pods (Bashir *et al.* 2005, Rashid *et al.* 2023).

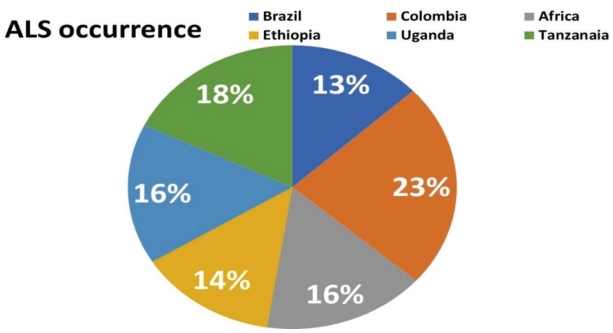

Fig. 1. Chart showing ALS distribution in different bean growing areas of world

VARIABILITY AND EVOLUTION IN *PSEUDOCERCOSPORA GRISEOLA*

Major disease-causing fungi including *Phaeoisariopsis griseola* causing ALS, *Colletotrichum lindemuthianum* causing anthracnose and *Uromyces appendiculatus* bean rust have undergone parallel micro-evolution with the host. Although there is considerable variation within gene pools, differences are more obvious in the reactions of isolates to known differential lines of Andean and Meso-American origin. As a general observation reported in the case of ALS, the isolates from the Andean highlands are specifically virulent on Andean large-seeded lines, whereas isolates from Meso-American ranges show virulence towards both groups (Araya *et al.* 2004). Based on isozyme polymorphism profiles, Correa-Victoria (1987) distinguished two groups in 55 *P. griseola* isolates from Africa, the U.S.A. and Latin America. All 26 isolates from Africa clustered in one group, whereas Latin American isolates clustered in both groups. However, recently the presence of both groups was reported from Africa (Liebenberg and Pretorius 1997, Wagara *et al.* 2004), which was also supported by data derived from isozyme analysis (Boshoff *et al.* 1996). Guzman *et al.* (1995) used RAPD analysis to divide 62 *P. griseola* isolates from Brazil, Wisconsin (U.S.A.) and Malawi into two broad groups. Isolates in the Andean group, collected predominantly from Andean bean host genotypes, were more pathogenic on Andean genotypes, whereas those from the second group, originating predominantly from Middle-American bean genotypes, were more pathogenic on Middle-American bean genotypes. The 11 Brazilian isolates fell in the second group, whereas 39 of the 42 Malawian isolates belonged to the Andean group. This grouping reflects the preference for small-seeded beans in Brazil and large-seeded beans in Malawi. A third, more virulent group reported in

Africa (CIAT 1996, Liebenberg and Pretorius 1997) has shown to be a variation of the Andean group (Mahuku *et al.* 2002). Thus, breeding strategies may use this knowledge to pyramid both Andean and Mesoamerican resistance genes to durable ALS resistance. Furthermore, Andean beans can be used as a source of resistance to the introgression of genes to Mesoamerican genotypes, as in the case of the carioca variety (Nay *et al.* 2019b).

There are obvious differences in the isolates between as well as within the isolate groups (Buruchara 1983) in terms of conidial size and amount of septation, in addition to the lesion size have been correlated with pathogenicity differences. However, Correa-Victoria (1987) observed no correlation between disease severity and lesion size, as well as no correlation between spore production and lesion size. Liebenberg *et al.* (1996) reported that the lesion size is more governed by the interaction between the pathogen and the host gene pool.

HISTORY AND TAXONOMIC POSITION OF *PSEUDOCERCOSPORA GRISEOLA*

The first report of *Pseudocercospora griseola* causing angular leaf spot on beans was reported by Saccardo (1878) who designated it as *Isariopsis griseola.* Later, Ellis (1881) named the fungus as *Graphium laxum* Ferraris (1909) reported that the genus *Isariopsis* is identical to *Phaeoisariopsis* and proposed a new name as *Phaeoisariopsis griseola*. The name Pseudocercospora was originally proposed by Spegazzini (1910) as a foliar pathogen of grapevine. In his bean pathogen monograph, Chupp (1954) described *Phaeoisariopsis griseola* as a synonym of *Cercospora columnaris* and favored the retention of *Phaeoisariopsis*. Fuckel (1863) established the brief taxonomical history of cercosporoids, including the genus Cercospora, from which the name of the group originates. Later, Deighton (1990) premised that synnematous arrangement of conidiophores could not be used as a sole criterion for generic differentiation, and confined *Phaeoisariopsis* to a few species similar to *P. griseola*, with non-geniculate conidiogenous cells with conspicuously flattened scars. He described species with geniculate conidiogenous cells and thickened and darkened scars as Passalora, and the taxa with quite inconspicuous conidiogenous loci in *Pseudocercospora*. Crous and Braun (2003) have revised the names of 5720 species in cercosporoids group and reduced the number to 3550 in *Cercospora* and *Passalora*, which was later further reduced to 940, of which 281 were placed in the morphologically indistinguishable *Cercospora apii* group. The current name of the ALS fungus *Pseudocercospora griseola* was proposed by Crous *et al.* (2006). Based on morphology, cultural characteristics, and DNA sequence analysis of the ITS region, calmodulin, and actin gene regions, they delineated *P. griseola* into two groups viz., *griseola* and *mesoamericana* The most predominant species is *Pseudcercopspora griseola* (74%), followed by *Phaseoropsis* (14.1%) and *Isaripsis griseola* (11.8%).

DEVELOPMENT OF DIFFERENTIAL CULTIVAR SETS FOR *PSEUDOCERCOSPORA GRISEOLA*

To develop effective resistance to ALS, it is imperative to characterize the genetic resources of beans to diverse forms of the *Pseudocercospora griseola* isolates that differ in pathogenicity towards diverse bean accessions. Such diverse forms of pathogens are designated as races, strains, pathotypes or biotypes. To elucidate the resistance response (resistant vs susceptibility), a set of cultivars with known reactions (differential hosts) are used to get insights into pathogenic variations in the pathogen. In the case of ALS, a set of 12 differential cultivars (six Andean and six Mesoamerican) was developed by CIAT in 1995 (Table 1) for the identification of *P. griseola* isolates. The pathotype designation is based on a binary value system comparing the reaction of differential genotypes with the respective *P. griseola* isolates (Pastor- Corrales *et al.* 1998). In the case of field and greenhouse experiments, bean germplasm is evaluated for disease response on a scale of 1- 9 proposed by Librelon *et al.* (2015) with the following screening criteria. Genotypes are designated as resistant (rating score of 1-3) and susceptible (rating score > 3). These differentials have been used extensively across all ALS resistance breeding programs to elucidate the race diversity of *P. griseola*. Race 63-63, first isolated from Latin America, is the most aggressive isolate of *P. griseola* and is virulent on nearly all Andean and Mesoamerican differentials (Silva *et al.* 2008). Around the end of the 20th century, races that infected mainly Andean differentials but also a few Mesoamerican differentials were discovered and designated as Afro-Andean races (CIAT 1997, Wagara *et al.* 2011). However, later on, molecular studies established them as variants of Andean group (Mahuku *et al.* 2002). Among all the races of *P. griseola*, race 61:63 is the most virulent race (Kyomugisha *et al.* 2023).

Biology and epidemiology of *Pseudocercospora griseola*

Angular leaf spot primarily affects aerial parts of bean plants leading to reduction in photosynthetic area and consequent yield loss as well as deteriorating pod quality by lesions on pods especially in warm and humid areas (Saettler 1991). Development of bean cultivars with durable resistance to ALS resistance is hampered by the high pathogenic variability of *P. griseola* (Pastor-Corales *et al.* 1998). *P. gresiola* has been reported to have high levels of pathogenic and genetic variations (Mahuku *et al.* 2002, Wagara *et al.* 2004). ALS is a hemibiotrophic fungus comprising a biotrophic phase, where hyphal growth occurs intercellularly in leaf mesophyll, and a neutrophic phase, where hyphae penetrate host cells causing plasmolysis (Monda *et al.* 2001). However, current information about the infection process of *P. griseola* is limited to spore germination and hyphae penetration through epidermis or stomata. This disease outbreak mainly occurs through primary inoculum from infected seeds or infected plant debris (Dellard and Cobb 1993). In the case of seed, the fungus is usually present on the seed coat primarily at the hilum, or in some cases may be internal. The transmission of *P. griseola* is also a minor source of primary inoculum (Correa-Victoria *et al.* 1989). The ALS infection is a five-step (Figure 2) process viz. (a) lesion establishment, (b) lesion extension; (c) infected leaf defoliation, (d) sporulation on infectious sites, and (e) spore dispersal (liberation and deposition) (Allorent and Savary 2005).

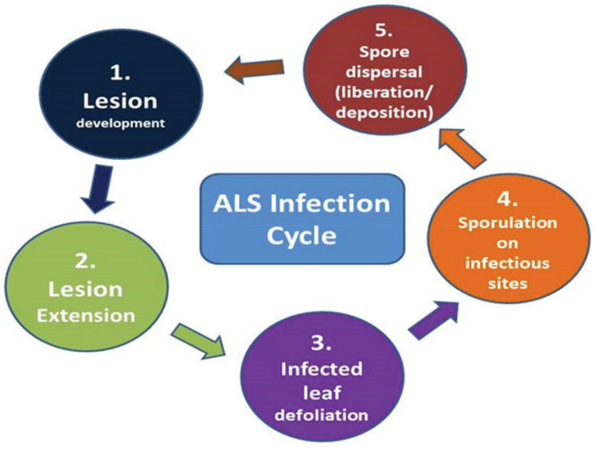

Fig. 2. Stages of infection process angular leaf spot

The infection starts when the pathogen lands on leaf tissue and then enters it through stomata and advances in the mesophyll intracellularly and palisade parenchyma. After nine days of infection, the fungus develops intracellularly throughout necrotic lesions. During the period of continuous moisture, stromata develop in the sub-stomatal cavity and then sporulation occurs. Under field conditions relatively dry and fluctuating conditions (temperature, relative humidity, and sunlight) favors the disease development by stromata formation and spore release and dissemination (Correa-Victoria *et al.* 1989). At the range of 16-28 ^{o}C the infection and disease development due to ALS can occur with an optimum temperature of 24^{o}C. As the plant approaches maturity after flowering the symptoms of ALS in the field do not become severe after that stage (Correa-Victoria *et al.* 1989).

The primary inoculum of ALS comes from pathogen-infected plant debris in fields as well as contaminated seed. In the case of seed, the fungus is usually associated with its seed coat primarily at the hilum or may also be internal. The transmission of *P. griseola* is also a minor source of primary inoculum (Correa-Victoria *et al.* 1989). The ALS fungus cannot survive for a longer period if the infested debris is buried and decomposed in the soil. But if not decomposed, later on, from these infested debris spores produced are transmitted to healthy tissues after planting via rain splashes or/and wind-blown. Then on landing healthy susceptible tissues the spores germinate and infect through natural pores (stomata). In Ontario, ALS tends to develop and spread quickly during late summer on late-seeded snap bean crops when day temperatures are warm and night temperatures become cool resulting in dew formation on plants. However, significant disease development and yield loss can also occur on earlier-seeded crops if moderate temperatures coincide with prolonged periods of wet weather. In *P. griseola* the sexual life cycle has not been known as it is difficult for many pathogens to carry out direct observations of the occurrence of different reproduction modes due to their small size and variable life forms (Samils *et al.* 2001). Fungi can reproduce both asexually (by mitosis) producing spores and sexually. However, regardless of the mode of reproduction, almost all fungi undergo some form of recombination (Taylor and Berbee 2006).

ALS infection caused by *P. griseola* is invariably detected in the form of necrotic spots that are more or less angular in their outer boundaries and can be visible on the leaf surface, or characterized by shriveled pods and shrunken seeds (Schwartz *et al.* 1982). The symptoms appear on all aerial plant parts, including leaves, petioles, stems and pods

but symptoms are most recognizable on leaves. On the leaves, the lesions usually appear as brown spots with tan or silvery centre that are initially confined to tissue between major veins, which gives it an angular appearance. The symptoms sometimes show a yellow halo occasionally surrounding lesions that eventually lead to yellowing of whole leaves before senescing in some varieties. The lesions on the underside of the leaf are also produced that are paler than those on the upper surface of the leaf. These underside leaf lesions revealed tiny dark tufts (synnemata) protruding from the lesions when observed with a magnifying lens. These dark tufts are the collection of stalks (conidiophores) that produce the spores that are later blown by wind or are rain splashed to the healthy tissues and spread the infection. On stems and petioles, the lesions appear as dark brown elongated spots, and on pods lesions are circular, black, and sunken and appear like anthracnose (*Colletotrichum lindemuthianum*), but lesions often penetrate deeper in the case of anthracnose. Based on severity score, mean, and variance genetic component it has been concluded that different genes control the reaction of the bean angular leaf spot on leaves and pods (Borel *et al.* 2011).

Pathogenicity of *Pseudocercospora griseola*

Pseudocercospora griseola causing ALS, is the largest group of ascomycetous fungi and is also the most diverse in terms of host crops and symptoms, containing many important plant pathogens, endophytes, and saprophytes (Crous *et al.* 2006). Pathogenic success and global distribution in many fungi has been attributed to various toxic chemicals. In the case of Cercosporoids, the widespread distribution and high pathogenic success has been attributed to cercosporin, a red-colored and highly toxic chemical. Cercosporin is a photoactivated metabolite and is easily visible in mycelia (Figure 3). Cercosporin causes peroxidation of membrane lipids and consequent membrane. Cercosporin has a typical perylene-quinone structure, whose toxicity has been well demonstrated in mice and bacteria (Yamazaki *et al.* 1975). It is the first plant pathogenic toxin with photosensitive activity. Such photoactivated perylenequinones are produced by various other fungal plant pathogens such as *Alternaria, Cladosporium, Elsinoe*, and *Hypocrella.*

Cercosporin is the first plant pathogenic toxin reported to have photosensitive activity. Cercosporin produced by Pseudocercosporalacks toxicity in dark, and light in the wavelength range

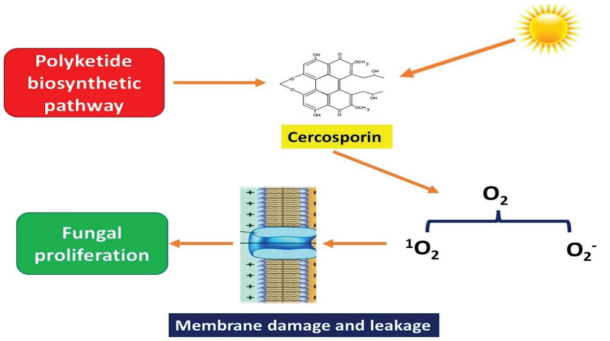

Fig. 3. Chemical structure of cercosporin and its mode of action

of 400-600 nm is the most important factor for its production and toxicity. However, various other parameters affect the production and toxicity of cercosporin, including pH, temperature, nutrient status, carbon and nitrogen source, and C: N ratio (Daub and Ehrenshat 2000). Once activated in light, cercosporin is converted into an energetically activated triplet state, which reacts with oxygen and results in the generation of toxic, activated oxygen species such as singlet oxygen ($^1O2$) and superoxide (O2-.) that causes peroxidation of membrane lipids and consequent leakage (Daub and Chung 2007). Cercosporin has been reported to be synthesized only in vegetative cultures and is repressed under nutrient conditions that induce conidiation. It is a potential driver of singlet oxygen species production and exposure of plant cells to cercosporin causes peroxidation of the membrane lipids, leading to membrane breakdown and death of the cells (Daub and Briggs 1983). Such leakage promotes fungal growth and proliferation as the nutrients are leaked into intercellular spaces.

Cercosporin is a non-host-specific polyketide toxin produced by several Cercosporoid plant pathogens. It is a light-activated red-pigmented, that acts as an important determinant of pathogenicity for Cercosporoid pathogens (Mitchell *et al.* 2002). As early as 1970's, the cercosporin biosynthetic pathway was reported by Okubo *et al.* (1975) using *Cercospora kikuchii* grown on with radioisotope-labelled acetate. They reported that cercosporin biosynthesis occurs via a polyketide pathway. The first step is the condensation of acetate and malonate molecules. No chemical intermediates or enzymes directly involved in the cercosporin biosynthetic pathway have been identified. However, significant progress has been made in understanding the regulation of the cercosporin pathway (Ehrenshaft and Upchurch 1991).

Upchurch *et al.* (2002) were the first to identify a gene CFP (Cercosporin Facilitator Protein) governing cercosporin production. The protein encoded by the CFP gene is similar to the group of membrane transporter proteins designated as the Major Facilitator Superfamily (MFS). Deletion mutants of the *CFP* gene in *C. kikuchii* were rendered partially defective in both cercosporin production and resistance. *CFP* gene product is primarily required for the export of cercosporin out of the mycelium. Chung *et al.* (2003) used restriction enzyme-mediated insertion (REMI) mutagenesis in *C. nicotianae* and isolated several fungal polyketide synthases (PKS's). One such gene namely Cercosporin Toxin Biosynthesis (*CTB1*) was found to be highly photoactivated and regulated by light. The CTB gene cluster, comprising eight genes (*CTB1*– *CTB8*) has now been characterized in detail (Upchurch *et al.* 2002). Out of the eight *CTB* genes, The *CTB1* gene encodes a protein consisting of 2196 amino acids with several conserved domains: a keto synthase (KS) domain, an acyltransferase (AT) domain, a thio-esterase (TE) domain and two acyl carrier protein (ACP) domains (Choquer *et al.* 2005). The functional domains in *CTB1* regulate three early steps in cercosporin biosynthesis viz., condensation of acetyl-CoA and malonyl-CoA units, chain elongation to form a pentaketide molecule, and ring closure, which are followed by oxidation and hydration reactions to form aromatic rings, catalyzed by a monooxygenase and methyltransferase domain encoded by *CTB3* (Dekkers *et al.* 2007) resulting in polyketomethylene units. The last step is a dimerization process that results in the bilaterally symmetrical structure of cercosporin. Once it is synthesized, cercosporin must be exported out of fungal cells by *CTB4* encoding a major facilitator superfamily (MFS) transporter (Figure 4).

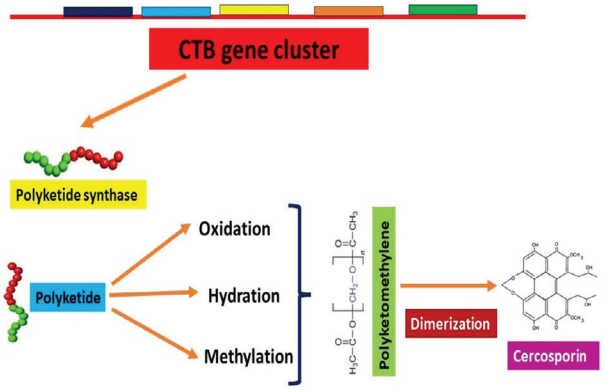

Fig. 4. Gene cluster for synthesis of cercosporin

Pathogenic variability of *Pseudocercospora griseola*

The high degree of pathogenic variability has been reported in *Pseudocercospora griseola* (Mahuku *et al.* 2009). Various approaches such as differential cultivars, biochemical approaches as well as molecular markers such as RAPD, ISSR, and ITS have been used to study the genetic diversity within *Pseudocercospora* populations (Sartorato 2002, Sebastian *et al.* 2006). Two major pathogenic groups viz., Andean (isolated from Andean gene pool) and Mesoamerican (isolated from Meso-American gene pool) have been identified, with Andean group reportedly more pathogenic to Andean gene pool, and Mesoamerican group pathogenic to both gene pools (Pastor-Corrales 1998). However, the virulence range is broader in the Mesoamerican group. Using isozyme analysis (Correa-Victoria 1987, Liebenberg and Pretorius 1997, Wagara *et al.* 2004) and RAPD analysis (Boshoff *et al.* 1996), both Andean and Mesoamerica isolates were identified in Africa, Brazil, USA, Malawi, Argentina, Columbia. However, in Africa, the third group was reported that was found to be more virulent and identified as a possible variant of Andean group (designated as Afro-Andean) (CIAT 1996, Nietsche *et al.* 2001, Mahuku *et al.* 2002). However, molecular studies could not distinguish it from Andean group (Wagara *et al.* 2004). In the initial studies, pathogenic variability was associated with lesion size (Verma and Sharma 1984), however, later it was found that the lesion size had no significant correlation with disease severity, and the observed variation in the lesion size is due to interaction between host gene pool and pathogen origin (Liebenberg and Pretorius 1997). Even though sexual reproduction is not reported in *P. griseola* (Stenglein *et al.* 2003), the high level of pathogenic variability could be attributable to various factors like mutations, migration, and para-sexuality (Anderson and Kohn 1995, Zeigler *et al.* 1995). To establish the system of differentials for characterizing isolates of ALS, the First International Angular Leaf Spot Workshop held at CIAT in 1995 developed a standardized method for the identification of *P. griseola* isolates using a set of 12 differential cultivars (Table 1). The set comprises six Andean and six Mesoamerican cultivars. The designation of pathotype was often defined using binary values of the differential genotypes that were compatible with the respective *P. griseola* isolate (Pastor- Corrales *et al.* 1998).

The first attempt to use RAPD markers for estimating the genetic diversity of *P. griseola*

was done by Pastor-Corrales and Jara (1995) which revealed significant genetic diversity in *P. griseola* isolates. Stenglein and Balatti (2006) used both RAPD and ISSR markers to assess the genetic diversity of 45 isolates of *P. griseola*, that delineated 18 haplotypes. ISSR markers have been highly effective on account of high polymorphism for studying genetic diversity, phylogeny, and evolutionary biology (Reddy *et al.* 2002). In another study, Abadio *et al.* (2012) used ISSR-PCR analysis of *P. griseola* isolates that identified 56 loci (300-2000 bp), out of which 40 were polymorphic. Serrato-Diaz *et al.* (2020) used four genes (ITS, rRNA, β-tubulin, and actin) to study the genetic diversity and phylogenic relationship among 171 isolates of ITS markers of *P. griseola* and could classify these isolates into different groups.

As compared to BCMV and anthracnose, the other notable bean diseases, angular leaf spot epidemic usually occurs in the later stages of the crop cycle (Allen *et al.* 1998) and is sequentially detected as the initial establishment of lesions, lesion extension, defoliation, sporulation and spore dispersal. Lesions are established on leaves upon the germination of *P. griseola* spores under moist conditions (Monda *et al.* 2001). Germination occurs under a wide range of temperatures (5–33 ^{o}C), with the optimal range of 18–280 C (Sindhan and Bose 1979). This is followed by a temperature-dependent incubation period (Bassanezi *et al.* 1998) that varies from 9 to 23 days, with the lowest incubation period recorded at 24^{o}C (Cardona-Alvarez and Walker 1956, Correa-Victoria 1987). The primary infection infects the healthy plant parts and latent sites are produced through the resulting infection. These latent sites may become infectious after a latency period, which may coincide with the rate of lesion appearance.

Lesion extension is influenced by the temperature and host genotype. The maximum lesion size has been reported to be at 15oC (Verma and Sharma, 1984). However, it has been observed in field conditions that larger lesions develop during cooler conditions (18–22 ^{o}C) compared to warmer (28–32^{o}C). The sporulation of *P. griseola* is implicated by several factors such as temperature (10-30^{o}C), relative humidity (>70%), and variety (Sindhan and Bose 1979). The formation of coremia in *P. griseola* is completed in 24 hours and then an additional 48 hours with humid conditions are necessary for spore production (Cardona-Alvarez and Walker 1956). There are about 5-800 spores mm2 of the lesion area and the sporulation density is largely affected by variety-isolate interactions (Correa-Victoria 1987). Very little information is available for spore liberation and dispersal processes. The available evidence points towards an apparent role for both rain and wind in spore liberation and dispersal. The dissemination of spores occurs as wind-blown particles from infested soil, wind-blown spores, and rain droplet-borne spores (Cardona-Alvarez and Walker 1956). *P. griseola* can survive under harsh conditions by encrusting itself to avoid desiccation and regenerating when conditions become conducive. The pathogen can also survive between seasons on infested seed, which is one pathway of introduction into the field in the proceeding season.

EXISTING MANAGEMENT STRATEGIES OF ANGULAR LEAF SPOT

Angular leaf spot disease is controlled by several management strategies including cultural practices, chemical, and biological control as well as using an integrated management module comprising a mix of various strategies. A straightforward strategy is to avoid growing beans with high humidity as high humidity promotes fungal growth and proliferation. Since the plant debris infected with the pathogen as well as the infected seeds are the primary sources of inoculum for initiating the disease cycle (Correa-Victoria *et al.* 1989), the infection chain could be effectively broken by crop rotation of two years at minimum, removal of bean debris through ploughing and using certified seeds (Celetti *et al.* 2005). Several classes of fungicides have been evaluated and found effective against this disease. Benzimidazoles like Benomyl and thiabendazole (Jesus Junior *et al.* 1999) and strobilurins, trifloxystrobin, and Azoxystrobin have also been found effective against common bean angular leaf spot (Ploper *et al.* 2003). Carbendazim 50 WP and Mancozeb 75 WP are reported as effective fungicides against the pathogen under both in vitro and in vivo conditions (Rashid *et al.* 2023). Boscalid, pyraclostrobin, pyramethanil, vinclozolin, and thiophanate-methyl can also manage the disease effectively in field (Celetti *et al.* 2005). Librelon *et al.* (2022) also proposed that the application of fungicides is an effective way of managing angular leaf spot in common bean. They reported that ALS fungus was sensitive (reduced sporulation capacity) to fungicides including pyraclostrobin, mancozeb, pyraclostrobin + metconazole, chlorothalonil, and tebuconazole. Fungicides were most effective when sprayed at 10-30% or 50-70% bloom. Various biological control modules have

also been optimized including several endophytic fungi, exhibited promising efficacy in both in-vitro and in-vivo control of the pathogen (Mota *et al.* 2021). Charimbu *et al.* (2009) found that aqueous and methanolic extracts of Allium sativum showed significant disease control against this disease in in vivo conditions. Sprays of thiophanate methyl have been reported to decrease ALS severity significantly and result in a consequent increase in seed yield by around 83-159% for fortnightly sprays and 95-184% for weekly sprays. The available results indicate that monitored sprays in line with disease progress are the optimum disease management strategy for ALS in beans (Fontem *et al.* 2007). Seed treatment with systemic fungicides such as carbendazim is an important protective measure, whereas mancozeb or copper-based fungicides can effectively control lesions or spots on leaves and pods. copper fungicides or mancozeb can be sprayed. Tricyclazole has also been proposed as a possible management option for ALS as it has been reported to reduce *P. griseola* f. *mesoamericana* growth by way of reduced melanin synthesis through Flaviolin intermediate, thereby affecting pigmentation and the cell wall structure of mycelium, reduced growth, decreased stratification and deposition of melanin in the hyphal wall (Barcena *et al.* 2015).

RESISTANCE SOURCES FOR ALS

Genetic resistance is the most appropriate, safe, and cost-effective disease control strategy available to small-scale-bean farmers (Busogoro *et al.* 1999). However there is a huge racial diversity of ALS fungus (Busogoro *et al.* 1999b, Guzman *et al.* 1995, Sartorato 2000) due to which cultivar resistance response is not stable across locations and years. To identify patterns of such resistance responses as well as identify new sources of resistance that could provide buffering against evolving races of pathogen, continuous germplasm evaluation, including monitoring and characterization of pathogenic variability, and eventual introgression of genes in cultivated varieties and breeding lines. The ALS resistance in common bean has been reported to be pathotype-specific with large differences in effectiveness in different locations and continents (Pastor-Corrales and Jara 1995, Pastor- Corrales *et al.* 1998, Mahuku *et al.* 2002, Silva *et al.* 2008). Thus, new sources of resistant germplasm are continually needed. Different strategies have been adopted to identify resistant genotypes. One of the ways is to expose the sources of resistance to existing pathogenic variation over different production areas (Beebe and Pastor-Corrales 1991, Milgroom and Fry

Table 1. Resistance sources of common bean for various races of ALS

S.No	Resistance source	Races against which resistant	Reference
1	Kentucky Wonder	Unknown	Gardner and Mains (1929)
2	Alabama No. 1, California Small White, and Epicure, Case Knife-Erecta	Unknown	Brock (1951)
3	Acc-156, Acc-50	Andean: IGI-77, IG5-78, IG6-79, IG7-79, IG8-79, IG9-79, IGlO-79.	Schwartz *et al.* (1982)
4	G10613, G10474,	Andean: PG	Pastor-Corrales *et al.* (1998)
5	MAR 1, MAR 2, MAR 3, Mexico 54 and BAT 332, G5686	Andean: PG03, PG243, PG267, PG270, PG289-1 Mesoamerican: PG01, PG08, PG61, PG81, PG242, PG244, PG261	Mahuku *et al.* (2003)
6	G23478, G23434C, G23477 and G23479	Andean race	Mahuku *et al.* (2003)
7	AND 277, AND 279, CAL 143, DRK 57, SUG 92, SUG 118, and AFR 150	12 races (six Andean and six Mesoamerican)	Aggarwal *et al.* (2004)
8	AND 277	Andean 63-69	Gonçalves-Vidigal *et al.* (2011)
9	BAT 332, Cornell 49-242, MAR-2, Mexico 54 and Ouro Negro	31-17, 63-19, 63-23, and 63-39	Sanglard *et al.* (2013)
10	U00297, AND277, G5686	Andean: 61:63 Mesoamerican: 1:6, 21:39, 17:39	Ddamurila *et al.* (2015)
11	ARA 4, COD MLV 059, MLV 224/94B, LSA 144 and Mexico 54	Andean and Mesoamerican isolates	Kijana *et al.* (2017)
12	Mexico 54, MAR 2 , BAT 332 G5686, G11796, Bolon Bayo	MesoAmerican Andean	Nay *et al.* (2019b)
13	Jalo Vermalo, Jalo ListrasPretas, Corinthiano, JlaoPintado-2, AmendoimCavalo, Jalo EEP558	Andean: 63-69	Gonçalves-Vidigal *et al.* (2020)
14	DAB-388, DAB-478, DRKDDRB-70, DRKDDRB-81, NUA-225, NUA-517, NUA-536 and NUA-577	Ethiopian race	Girma *et al.* 2022
15	WB-371, WB-1677	Andean: Pg01 and Pg02	Gani *et al.* (2023)

1997). Another way is to identify the diversity that exists within the pathogen and then sample this diversity adequately, to use representative races to screen sources of resistance (Milgroom and Fry 1997). The most virulent isolates from Andean and Mesoamerican groups can be used to identify durable ALS resistance.

GENETICS OF RESISTANCE TO ANGULAR LEAF SPOT

The speed and success of breeding programs are largely determined by our knowledge about the genetic basis of a trait viz., number of genes and their alleles, and inheritance pattern including the dominance relationships. The resistance to *P. griseola* has been reported to be controlled by both dominant and recessive genes (Mahuku *et al.* 2009). Currently, six genes namely *Phg-1, Phg-2, Phg-3, Phg-4, Phg-5* and *Phg-6* are reported in common bean (Oblessuc *et al.* 2012, Keller *et al.* 2015). The *Phg-1, Phg-4,* and *Phg-5* genes are from an Andean origin, and the *Phg-2* and *Phg-3* genes are from a Mesoamerican origin. *Phg-1* and *Phg-2* are dominant resistance genes mapped on chromosome Pv01 and Pv08 respectively (Goncalves-Vidigal *et al.* 2011). *Phg-1* is of Andean origin from cultivar AND277 derived from a cross of G 21720 × BAT 1386. *Phg-1* and *Co-l4* loci are tightly linked (0.0 cM) on Pv01 (Goncalves-Vidigal *et al.* 2011). *Phg-2* is of Mesoamerican origin from cultivar Mexico-54 mapped on Pv08 (Sartorato *et al.* 2000). *Phg-2* has also mapped on Pv08 in Mesoamerican cultivars such as Cornell 49-242, MAR 2, G10474, BAT 332, and G10909 (Souza *et al.,* 2016). The *Phg-2* gene and its potential alleles reported in various Mesoamerican cultivars are to date reported to confer the broadest known resistance to ALS. *Phg-3* gene has been found in Mesoamerican lines Ouro Negro, a highly productive black-seeded bean variety and mapped to Pv04 and reported to co-segregate with Co-03 conferring resistance to anthracnose (Corrêa *et al.* 2001). Phg-4 has been discovered in CIAT line G5686 and mapped on Pv04. The line G5686 has shown resistance to more than 500 isolates from 27 countries (Mahuku *et al.* 2009). The gene *Pg-5* has been mapped on chromosome Pv10. It was mapped from two bean lines CAL-143 and G5686. The *Phg-5* locus was first reported in a CAL 143 x IAC-UNA RIL population screened under natural infection and inoculated with *P. griseola* race 0-39 (Oblessuc *et al.* 2012). Four resistance gene analogs were reported on chromosome Pv10 and linked to ALS resistance in a RIL population from the G19833 x DOR364 cross (López *et al.* 2003). Another minor QTL from AND 277 on chromosome Pv10, conferring resistance to race 1-21 was reported by Bassi *et al.* (2017). The first report of recessive ALS inheritance was published by Singh and Saini (1979) in an interspecific cross of resistant scarlet bean accession PLB-257 and susceptible bean cultivar Contender revealing that PLB 257 carries a recessive gene. Fortunately for plant breeders, *Phg-4* has been co-localized with genes *Co-03* and *Co-10* governing resistance to anthracnose.

Various QTLs have also been associated with ALS resistance in beans. ALS10.1 is a major QTL mapped to linkage group Pv10 using the IAC-UNA × CAL 143 RIL population along with two adjacent QTLs, *ALS4.1GS* and *ALS4.2GS* (Oblessuc *et al.* 2012). Five other QTLs have also been mapped on linkage group Pv04, one on Pv08, another on Pv09, and three on Pv10 (López *et al.* 2003, Mahuku *et al.* 2009, 2011). The line CAL 143 derived from G12229 x AND 277 has shown resistance to a large number of *P. griseola* isolates in the field (Aggarwal *et al.* 2004), with an additional feature of resistance to bean rust, alternaria leaf spot, and anthracnose (Vieira *et al.* 2002). This line has been widely used as a source of multiple disease resistance. Thus, the resistance locus ALS10.1 from CAL 143 was selected for the discovery of disease-responsive genes. From the same line, a QTL ALS11AS, linked to the SNP BAR-5054, has been mapped on chromosome Pv11 and has also been found closely linked to Powdery mildew QTLS indicating its pleiotropic nature (Bassi *et al.* 2017). In Andean germplasm, seven more ALS resistance QTLs namely *ALS2.1, ALS3.1, ALS4.1, ALS4.2, ALS5.1, ALS5.2,* and *ALS10.1* have been mapped on five chromosomes (Oblessuc *et al.* 2015a). In the Andean line G5686, three major R genes namely *PhgG5686A, PhgG5686B,* and *PhgG5686C* have been reported (Mahuku *et al.* 2009), which were later designated as QTLs by Keller *et al.* (2015) and mapped to the same loci as *ALS4.1, ALS4.2,* and *ALS9.1* (Oblessuc *et al.* 2012, Keller *et al.* 2015). Co-inheritance of ALS and anthracnose genes especially *Phg-04* with *Co-03* and *C0-10* have been confirmed by a single dominant locus namely, CoPv01CDRK/PhgPv01CDRK using RILs from CDRK × Yolano cross (CY). The fine mapping of the loci also identified a smaller genomic region of 33kb containing five predicted genes (Goncalves-Vidigal *et al.* 2020). A minor QTL was reported from AND 277 x SEA 5 RIL population and mapped to Pv06 (Bassi *et al.* 2017).

BREEDING EFFORTS FOR ALS RESISTANCE

Resistant varieties are the most economical and ecologically sustainable option for disease control/ management (Oblessuc *et al.* 2012). For diseases like ALS, the use of fungicides could be an option but the approach is ecologically compromising as well as economically unsustainable, especially for small bean producers in tropical countries, that account for the bulk of bean production (Nay *et al.* 2019b). Phenotyping for the severity of angular leaf spot (ALS) in the common bean is important to identify new sources of resistance and select progenies in conventional and molecular marker-assisted breeding programs. Despite being a powerful tool that complements conventional breeding for efficient screening and rapid outturn in case of complex traits such as disease resistance, marker-assisted breeding (MAS) does not provide a replacement for phenotypic selection. MAS has been used to introduce and combine genes of resistance to various fungal, viral, and bacterial diseases in common bean (Kelly *et al.* 2003, Miklas *et al.* 2006, Duncan *et al.* 2012, Tryphone *et al.* 2013, Souza *et al.* 2014, Meziadi *et al.* 2016). It has been used in the development of improved common bean germplasm in the U.S. (Singh and Miklas 2015), Canada (Durham *et al.* 2013), Brazil (Souza *et al.* 2014) and Tanzania (Tryphone *et al.* 2013).

Resistant cultivars are by far the most effective, safe, and inexpensive strategy for management compared to other approaches to disease management. However, the development of resistant varieties for ALS is impeded by the great pathogenic variability of *P. griseola* (Sartorato 2000, Mahuku *et al.* 2002). The problem could be overcome by the use of molecular approaches, especially if the molecular markers are closely linked to resistance genes that would greatly facilitate and accelerate marker-assisted selection (MAS), particularly at the initial and intermediate stages of the breeding process (Kelly 1995). MAS can also help reduce linkage drag and recovery of recurrent parent genomes in a sizeable number of generations (Openshaw *et al.* 1994). Also, MAS is very useful especially when the breeding program is aimed at the introgression of one or more favorable alleles or genes from the donor parent to the elite varieties. In common bean, MAS has also been the most practical and realistic approach to provide long-term control of various diseases such as Anthracnose (*Colletotrichum lindemuthianum*), ashy stem blight (*Macrophomina phaseolina*), bean common mosaic virus, bean common mosaic necrosis virus, bean golden mosaic virus (Miklas 2002a), bean rust (*Uromyces appendiculatus*) (Stavely 2000) and common bacterial Blight (*Xanthomonas campestris*) (Miklas *et al.* 2000, Yu *et al.* 2000). The ALS resistance in the common bean is controlled by single dominant (Ferreira *et al.* 2000, Mahuku *et al.* 2004, Namayanja *et al.* 2006) as well as recessive genes (Corrêa *et al.* 2001). Various markers associated with these genes have been identified (Corrêa *et al.* 2001, Ferreira *et al.* 2000; Namayanja *et al.* 2006, Nietsche *et al.* 2000; Caixeta *et al.* 2003, Ferreira da Silva *et al.* 2003, Mahuku *et al.* 2004, López *et al.* 2003). By the use of molecular markers homozygous common bean lines carrying angular leaf spot resistance genes derived from the cultivars 'Mexico 54', 'MAR 2', and 'BAT 332' have been developed through marker-assisted selection and this was the first report of lines resistant to angular leaf spot carrying genes of these cultivars developed with the aid of molecular markers (De Oliveira *et al.* 2005). The ALS-resistant dry bean breeding lines (A 339, MAR 1, MAR 2, and MAR 3) have also been developed from inter-racial populations between Mesoamerican races (Singh *et al.* 2003). MAS has also been applied for the transfer of the resistance from Mexico 54, MAR 2, and BAT 332 into a carioca bean 'Ruda' in Brazil (de Oliveira *et al.* 2005). The Phg-2 ALS resistance gene, which is found in highly resistant common bean genotypes has been investigated in crosses between Mesoamerican pre-breeding lines and elite Andean breeding lines. Some molecular markers are closely linked to this gene and also highly specific to the donor parent and were thus used in MAS to introgress this Phg-2 gene into other Andean breeding germplasm using MAB lines (Gil *et al.* 2019).

As the inheritance of resistance to ALS in the common bean is predominantly quantitative (Borel *et al.* 2011, Oblessuc *et al.* 2012, Pereira *et al.* 2015), the use of recurrent selection is a suitable breeding method to get the durable resistance. This breeding method has been used at Universidad Federal de Lavras (UFLA), in the state of Minas Gerais, Brazil and led to the development of several lines resistant to *P. griseola*. In the case of ALS, the genomic regions responsible for qualitative and quantitative resistance have been established (Arantes 2010, Rezende *et al.* 2014, Pereira *et al.* 2015). The common bean breeding program for resistance to ALS has been conducted in Brazil using recurrent selection since 1998 (Nay *et al.* 2019b) and to date, almost 17 cycles have been conducted and the effectiveness of this program in obtaining resistant progenies and lines has been reported (Amaro *et*

al. 2007, Nay *et al.* 2019b, Rezende *et al.* 2014). In this program, at the end of each selection cycle, the progenies are evaluated for ALS severity in the field using the diagrammatic scale proposed by the International Center for Tropical Agriculture (CIAT); van Schoonhoven, and Pastor-Corrales 1987). However, the assessment of ALS severity in the field can be hampered by the simultaneous occurrence of other diseases in a common bean crop causing difficulty for the evaluator assessing the symptoms. Furthermore, only one selective cycle is carried out per year in the dry season (sowing in February–March) presents favorable environmental conditions for the development of disease and depends on the natural occurrence of pathogen (Rezende *et al.* 2014).

HARNESSING GENOMICS FOR NEW SOURCES OF ALS RESISTANCE

Common bean has N=11 chromosomes with a genome size of about 587Mbp (Schmutz *et al.* 2014). The first draft of the common bean genome was published in 2014 (GenBank Accession No. ANNZ01000000) using an Andean gene pool line G19833 (race Peru) covering about 473 Mbp of the genome (Schmutz *et al.* 2014). Currently, there are two whole genome sequences available for the common bean, one corresponding to the Andean cultivar G19833 (Schmutz *et al.* 2014) and another to the Mesoamerican, cultivar BAT93 (Schmutz *et al.* 2014, Vlasova *et al.* 2016). However, the marker database of Phaseolus Genes (http://phaseolusgenes.bioinformatics.ucdavis.edu) is largely based on the Andean whole genome sequence. Using genomic resources and genetic maps, resistance (R) genes with nucleotide-binding—leucine-rich repeat (NB-LRR) or kinase domains, associated with anthracnose (Creusot *et al.* 1999, Melotto *et al.* 2004), BCMV (Vallejos *et al.* 2006) and common blight (Shi *et al.* 2011), have been identified. The reference genomes for both Andean and Mesoamerican gene pools have enabled the fine-mapping of many resistance gene loci, including *Co-AC, Co-1HY, Co-x,* and *Co-173-X* on Pv01 (Richard *et al.* 2014, Chen *et al.* 2017, Gilio *et al.* 2020, Murube *et al.* 2019) and *Co-42* on Pv08 (Oblessuc *et al.* 2015b). The first report of an R-gene cluster located on chromosome Pv04 were published by David *et al.* 2009 and Geffroy *et al.* 2009). Pv04 was later found to harbor several resistance genes against *Pseudocercospora griseola* (López *et al.* 2003, Oblessuc *et al.* 2012, 2014, Ferreira *et al.* 2013, Gonçalves-Vidigal *et al.* 2013).

GWAS as well as linkage mapping has been quite helpful in the case of ALS resistance, utilizing of complementary power of both approaches to identify conservative QTLs (Wu *et al.* 2020), as it provides high resolution and uses diverse genetic resources that provide insights into genomic signatures conditioning plant disease response (Perseguini *et al.* 2016, Nay *et al.* 2019b). A large number of mapping studies on ALS resistance involving different genetic materials, diverse environments, and isolates have already been reported (Faleiro *et al.* 2003, Gonçalves-Vidigal *et al.* 2013, Oblessuc *et al.* 2012, 2013, Keller *et al.* 2015). GWAS for ALS had revealed two ALS genes (*Phg-2* and *Phg-4*) of the five previously described resistance, that are significantly associated with ALS resistance. Moreover, it has also revealed the diversity of functional haplotypes for the resistant loci in common beans. Nay *et al.* (2019a) reported a haplotype group conferring broad-spectrum resistance to ALS, with six showing pathotype-specific effects, while seven did not show clear resistance patterns. In addition to the discovery and validation of *ALS* resistance genes and QTLs, GWAS can also help estimate the linkage disequilibrium levels and identify quantitative resistance loci (QRL) for ALS as well as identify co-localization patterns of ALS and anthracnose genes and identify new sources of resistance to both disease in Andean and Mesoamerican germplasm (Perseguini *et al.* 2016). Using such an approach, several markers associated with anthracnose were mapped on Pv04 (race 9 and 73), Pv01, Pv04, and Pv08 (race 65) and for ALS resistance markers were positioned on Pv02 and Pv04 (race31.23) and Pv03, Pv06 and Pv08 (race 63-39) (Vidigal Filho *et al.* 2020).

Recently, Almeida *et al.* (2021) studied association and linkage mapping for analyzing ALS resistance at the V2, V3, and R8 stages. Association mapping was done using the Cariocao diversity panel using 5,398 SNPs from Bead Chip assay technology, while as linkage mapping was done in the BC_2F_3 population of AND-277 × IAC-Milenio population with 1091 SNP's using GBS. Disease reaction was assayed in both populations at V2, V3, and R8 stages under controlled conditions. The study identified seven QTLs through interval mapping and 14 QTLs through association mapping, as well as validated *Phg-1, Phg-2, Phg-4,* and *Phg-5*. A novel QTL *ALS11.1AM* was mapped on chromosome Pv11. Several putative resistance genes at the three growth stages were identified, for which markers can be developed for use in MAS.

IMPROVING ALS RESISTANCE BY GENE PYRAMIDING

As a result of relatively higher pathogenic variability in *P. griseola,* new races are continuously emerging leading to frequent breakdowns of resistance that severely implicate the durability of the resistance (Young *et al.* 1998, Ramalho and Abreu, 1998). The observation that Mesoamerican races infect both Mesoamerican and Andean bean genotypes, while Andean races preferentially infect Andean genotypes (Guzman *et al.* 1995, Pastor-Corrales and Jara, 1995, Crous *et al.* 2006) has encouraged breeders to use these sources for pyramiding both Andean and Mesoamerican resistance genes to impart durable resistance to ALS. On such premise, Andean beans can be used as a source of resistance to introgression of genes to Mesoamerican genotypes (Nay *et al.* 2019b). However, this strategy would require continuous screening of bean germplasm with the occasional introgression of new resistance genes into commercial cultivars (Young and Kelly 1996). New virulence races of these pathogens have resulted in reduced or complete loss of yield in previously resistant commercial cultivars (Pastor-Corrales *et al.* 1998, Busogoro *et al.* 1999, Mahuku *et al.* 2002, Geffroy *et al.* 2009). The gene pyramiding approach for durable ALS resistance is greatly facilitated by marker-assisted selection, and the development and validation of highly accurate molecular markers that are tightly linked to important disease-resistance genes enables the pyramiding of these genes into single cultivars with broad-spectrum resistance.

The use of marker-assisted selection for *ALS* resistance genes is again impeded by the high pathogenic variability of *P. griseola* (Jeronimo *et al.* 2011) and gene pyramiding could be used to achieve durable and broad-spectrum resistance by converging several resistance genes in elite genetic backgrounds (Kelly *et al.* 2004) and improve the durability of resistance by delaying resistance break down and reducing the evolution of resistance against *P. griseola* (Ferre and Van Rie, 2002). There are published records of the advantage of combining multiple disease resistance in beans for developing bean varieties having resistant genes for ALS (*Phg-1*), anthracnose (*Co-o* and *Co-4*) and rust (*Ur-ON*) through a marker-assisted pyramiding. Costa *et al.* (2006) pyramided genes for anthracnose and angular leaf spot resistance in a black bean variety (Diamante Negro) and a red bean landrace (Vermelhinho) using molecular markers for improving resistance to these diseases in these popular bean varieties. Recently Ddamulira *et al.* (2015) pyramided three different angular leaf spot-resistant genes (*Phg-1, Phg-2* and *Phg-4*) for improved resistance to highly virulent isolate 61:63 in two common bean cultivars viz., K132 and Kanyebwa. Rezene *et al.* (2019) pyramided one ALS resistance gene (Phg-2 from Mexico-54) and two common blight resistance genes (from Vax-6) into the popular bean variety Red Wolatia using molecular markers.

CONCLUSION

ALS is a devastating fungal disease in common beans with a global impact especially in warm tropical areas. Since its first report, a large number of studies have elucidated its biology, pathogenicity, and race diversity which have helped us better understanding fungal survival and its infection process. This coupled with national and global efforts for identifying resistance sources and the underlying genetic and molecular basis of resistance have helped in creating huge genetic and genomic resources for improving resistance to ALS in cultivated beans. The genetic basis of ALS is quite understood and the focus is now on identifying more genes and QTLs for resistance to broaden the gene kitty and pyramid them for building durable resistance. Also, the focus is to monitor race development in ALS to deploy appropriate genes for preventing crop damage due to resistance breakdown. Newer frontiers of science such as gene editing using CRISPR-Cas are opening up new vistas for developing resilient varieties, an important responsibility of crop breeders, especially in view of the fact that existing and predicted climate change scenarios may severely increase the disease severity and impacts.

DECLARATION ON CONFLICT OF INTEREST

The authors declare no conflict of interest

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DATA AVAILABILITY STATEMENT

The data that support this study will be shared upon reasonable request to the corresponding author.

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