

# Diseases of Common Bean

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## Abstract

Common beans are among the most grown vegetables in the tropical and temperate countries. Early maturing varieties can be cultivated even in the short summer periods of temperate regions, making it one of the most used vegetables around the world. Common beans are an important source of proteins (>25% in dry weight), minerals (iron and zinc) and vitamins for many human populations. Immature pods are eaten fresh and can be easily preserved. Mature pods and seeds are dried. Beans are eaten boiled, baked, fried, or ground into flour. Crop residues and processed by-products are good fodder for animals. The wide variety of uses and the wide geographic cultivation has pre-disposed the crop to several pests and diseases. Diseases caused by fungi have been the most destructive and include anthracnose, rusts, damping off, root rots and molds among others. Important bacterial diseases include blights, wilts and leaf and pod spots. Several types of mosaics and root-knot diseases make up the viral and nematode diseases of common bean, respectively. Several other diseases that may not be of global importance are not mentioned in this review, but remain important locally, as they destroy the crop and farmers' livelihoods. Preventing the trans-boundary spread of these diseases and managing them locally is critical for the sustainable supply of common beans. Several management options are provided for specific diseases but growing resistant varieties and adapting integrated pest management strategies will remain the preferred method of disease control.

**Key words:** *Alternaria*, *Colletotrichum*, *Fusarium*, *Meloidogyne*, *Pseudomonas*, *Pythium*, *Rhizoctonia*, *Sclerotinia*, *Thielaviopsis*, and *Xanthomonas*.

## 1 Introduction

The word bean is a term loosely used to describe any legume whose seeds or pods are eaten. Most beans were classified in the genus *Phaseolus* with the common bean, *Phaseolus vulgaris* L. Common bean includes the following bean varieties: Black Turtle Bean, Cranberry bean, Flageolet, kidney bean, navy bean, Pinto bean, Pink bean, and Romano. It also includes immature green beans, also known as, string beans or snap beans. Common bean is believed to have originated from central Mexico and South America (Rodriguez et al. 2015). From here, common bean was distributed to Africa, Europe and Asia, and all around the world subsequently. According to the database of the Food and Agriculture Organization in 2019, global production as a dry grain was estimated at 30.2 million tons, while production as a vegetable or green bean was 48.7 million tons. Myanmar, India, Brazil, China, the USA, Mexico and Tanzania are some of the world's leading producers of common beans representing two-thirds of the world total production of dry beans (Heuzé et al. 2015). With minimum or no processing requirement, common bean is one of the most important sources

of proteins for humans and livestock particularly in the developing world. In addition to the caloric intake, it is a good source of minerals, fiber, thiamine, folate, and phytochemicals with analgesic and neuro protective properties (Blair et al. 2013; Jha et al. 2015). As such, besides development of high yielding varieties with different levels of resistance to major pests and diseases, recent trends in common bean breeding have also focused more on biofortification with the aim of addressing some nutritional disorders (Petry et al. 2015; Mulambu et al. 2017). As a legume, common bean is well known for its ability to fix nitrogen in the soil through an association with the bacteria, *Rhizobia* – a process commonly referred to as symbiotic nitrogen fixation. Although significant variation in symbiotic nitrogen fixation exists among bean cultivars with Mesoamerican indeterminate and climbing cultivars having better nodulation and symbiotic nitrogen fixation, than Andean determinate type (Wilker et al. 2019). This property can reduce the use of synthetic fertilizers, which is key for sustainable agriculture (Khan et al. 2020). Economic valuation of common bean at global level often includes other species making it difficult to isolate precise value for *P. vulgaris* alone. Nevertheless, with production of 18.9 million tons for all types, it is the most widely produced grain legume and ranked third after soybean and groundnut for oilseed and grain legumes combined (Myers and Kmiecik, 2017). Despite the nutritional and economic importance of beans, production of the crop is negatively affected by several biotic and abiotic stresses. Among the biotic stresses, pests and diseases pose a major threat in common bean production. This chapter provides detailed description of the major common bean diseases, their identification, geographic occurrence and management among others.

## 2 Fungal Diseases

### 2.1 Alternaria leaf and pod spot [ *Alternaria alternata* (Fries) Keissler]

**Geographic occurrence and impact** *A. alternata* has been reported in several countries from Africa, Asia, the Middle East USA, and Australia. Considering that they are normal constituents of soil mycota and the air worldwide (Patriarca and Pinto 2018), the fungus has the potential to spread as infected beans are transported as vegetable around the world. High relative humidity, rainfall, and cool temperatures (28°C to as low as -3°C) favour development of Alternaria leaf and pod spot. Besides common beans, the fungus has a wide host range in cereals, vegetable crops and fruit trees, infecting different plant tissues such as the fruits, seeds, stems, leaves and flowers. Yield and/or quality losses are induced either directly through decay of the attacked plant tissue or indirectly by impairing the physiological processes of the plant. Once the fungi sporulate, resting spores may develop making them resistant to post-harvest treatments in the harvested produce. This has significant implications on international trade because such spores may start infecting the produce in-transit and get transported to a new destination. Secondary metabolites that some isolates of *A. alternata* produce are phytotoxins and are harmful to humans and animals that consume them (Patriarca and Pinto 2018).

**Symptoms and Signs** Alternaria leaf and pod spot symptoms begin as water-soaked dots (about 1mm in diameter) that generate into circular to irregular spots with pale-brown centers and reddish-brown borders (**Fig. 1a and b**). Older lesions are characterized by light and dark concentric rings. As they enlarge, the brown spots coalesce resulting in reduction of the photosynthetic area, accelerated senescence, and premature leaf drop. Infected pods show dark brown to black flecks, maybe smaller, ripen earlier and often shatter before harvest. Alternaria leaf and pod spot symptoms usually appear in patches that are spread throughout the field. Undernourished plants (particularly those that are deficient in nitrogen and/or potassium) are more susceptible to Alternaria leaf and pod spot.



**Fig. 1.** Alternaria Leaf and Pod spot of common bean (a) foliar symptoms (b) pod symptoms. Courtesy S. Zhang IFAS Ext [PP-61/PP106: Alternaria Leaf and Pod Spot of Snap Bean in Florida \(ufl.edu\)](#) all rights reserved® 2024

**Biology and epidemiology** *A. alternata* belongs to the fungi imperfecti (meaning a sexual lifecycle is not known) belonging to the dictyosporic genus *Alternaria* of the family Dematiaceae, order Hyphomycetes. Typical colonies of *A. alternata* are lettuce-green to olive green in colour and measure over 70 mm in diameter after 7-10 days of growing on artificial media. *A. alternata* is distinguished by the formation of conidial chains six to 14 conidia in length and the development of numerous secondary, and occasionally tertiary, chains of two to eight conidia in length (Troncoso-Rojas and Tiznado-Hernández, 2014). Infection process begins with adhesion of the spore on the cuticle layer of the plant tissue followed by direct growth of the germ tube. Penetration into the plant is through natural openings, wounds and direct/physical breaching of the cuticle by penetration pegs (Mersha et al. 2012). During the infection process, *A. alternata* is known to secrete enzymes that disintegrate the cell structure of the host plant thereby interfering with the vital functions of the host cell. In addition to enzymes, a group of mycotoxins are produced which disrupt the host-defence system of the infected plant. Severe outbreaks and spread of Alternaria leaf and pod spot may be observed under high relative humidity and rainfall conditions with average temperatures of 25°C.

## Management

- **Cultural practices** that help in the management of Alternaria leaf and pod spot include avoiding high plant population densities and maintaining adequate nutrient status of the crop. Since infection is through wounds or natural opening, careful handling during harvest and subsequent processing is essential to prevent physiological injuries that may provide room for infection.
- **Chemicals and Biologicals** - Several fungicides are used before and after harvest as a treatment to prevent or control the development of *A. alternata*. They include

strobilurins (e.g. Compass O 50WDG), hexaconazole, and Quadris™/Amistar™ (manufactured and traded in different names by Syngenta, Bayer and Crop Life Sciences Ltd among others). It is, however, recommended that spraying be done when pods are still small to avoid visible damage on mature pods.

## 2.2 Anthracnose [*Colletotrichum lindemuthianum* (Saccardo & Magnus) Briosi & Cavara (= *Glomerella lindemuthianum*)]

**Geographic occurrence and impact** - The disease was first registered on bean in France in 1843 (Martínez-Pacheco et al, 2009). It is one of the major diseases of common bean and occurs in both temperate and tropical countries worldwide where beans are grown (Kiptoo et al. 2020). It is favored by wet and humid environments. The disease affects the quality of beans and their marketability. Yield losses are of economic significance. In Brazil, for instance, bean anthracnose is widespread with yield losses of up to 100% (Fioreze et al. 2018). In Africa, similar yield losses have been reported with 63% and up to 100% in Ethiopia and Kenya, respectively (Hussien et al. 2020; Kiptoo et al. 2020). In Tanzania alone, losses are in the range of 40-80% with an estimated loss of \$304 million per annum. Although the introduction of certified and approved bean seed schemes has resulted in reduced impact in many countries, the disease remains a problem in most developing countries particularly with emergence of new races of the fungus.

**Symptoms and Signs** Dark brown to black sunken lesions appear on seedlings that emerge from infected seed (primary infection) (**Fig 2.**). These symptoms are also expressed on the first leaves, the cotyledons. As the lesions on stems increase in size and number, they coalesce, and the seedling eventually collapses. If the stem survives, the sunken lesions contain pink-colored spores that spread to other leaves as they emerge. Brown to black streaks appear on the lower surface of the leaf, particularly on the veins. As the disease advances, this may result in wilting and flagging of chlorotic leaves similar to that of other foliar pathogens. Infection on petioles produces oval or circular spots with red-brown margins. If spots surround the petioles, they collapse. Dark brown irregular specks or circular sunken spots, 1-10 mm diameter, bordered by distinct brown to black margins also appear on bean pods - probably the most distinctive symptoms of anthracnose in common beans. Under wet conditions, these spots also produce spores that can be spread to other plant tissues. Severely infected pods abort prematurely and fall early while mature pods shrivel and the seed become infected with dark cankers.



**Fig 2.** Anthracnose of bean (Courtesy E. Bush, Va Tech, Bugwood.org, all rights reserved® 2024).

**Biology and epidemiology** - Based on the principles and rules of the International Code of Botanical Nomenclature, most authors agreed that *C. lindemuthianum* belongs to: Family, Melanconiaceae; Order, Melanconiales; Sub Class, Coelomycetidae; Class, Deuteromycetes; Subdivision, Deuteromycotina; Division Amastigomycota; Kindom Myceteae; Super Kingdom, Eucariota (Mohammed, 2013). *C. lindemuthianum* is considered as a hemibiotrophic fungus, i.e. it may live and grow on any carbon source as a saprophyte or derive nutrition from living cells of a host plant as a biotroph. As a saprophyte, the germination process begins with adhesion of the spore to the plant surface. Stimulated by adequate humidity and moisture in the mucilage (spore envelope), there is subsequent active growth followed by formation of a germ tube. Later, the infection hypha grows and elongates to colonize the substrate (Mohammed, 2013). Then aerial mycelia appear followed by reproductive structures where spores are formed and stored. On the surface of a living host plant, the germ tube develops an appressorium (pressing organ). As the germ tube grows, and with the help of the appressorium, an indentation occurs in the cell wall of the host plant. An infection peg then protrudes from the appressorium and penetrates through the cell wall. Once inside the host plant, an infection hypha grows and proliferates throughout host tissues, inside cells, through walls and in intercellular spaces. *C. lindemuthianum* is known to produce cell wall-degrading enzymes and phytotoxins that contribute to necrotrophic growth of the fungus. The spores are spread from one plant and area to another through wind, animals, rainwater, insects and long-distance transportation by humans among others. The

fungus may also spread and survive from one season to the next through seed and infected crop debris. This has a significant bearing on small scale farmers particularly in Africa who recycle their seed from one year to the next.

## Management

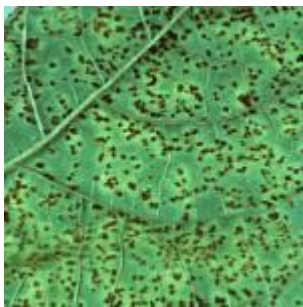
- **Cultural practices** include establishment of a clean seed system and use of certified seed free from infection; avoiding high plant population; mulching the field to prevent water and soil splashes and avoid undertaking field operations when the plants are wet. Intercropping beans with maize also minimize disease incidence. It is also important to follow crop rotation with cereals such as maize when signs are noticed.
- **Chemicals and Biologicals** - Seed dressing and treatment using Benlate and Mancozeb at a rate of 2-3g/kg seeds is one of the available chemical control options. During the vegetative growth stage, anthracnose severity and incidence may be reduced with foliar application of Difenconazole and carbendazim. Effectiveness of fungicides, however, will depend on several factors including time of spraying, weather conditions, *C. lindemuthianum* race, and also health status of the plants among others.
- **Genetic resistance** - Being a seed-borne pathogen, the first management opportunity is to eradicate or reduce the pathogen inoculum in the seed production field. Growing of resistant varieties presents one of the most effective means of managing the disease. Several cultivars with resistance genes have so far been identified which include G2333 and AB136 (Nkalubo et al. 2009, Kiryowa et al. 2017). Although most effective, this approach is challenged by the high degree of genetic and physiological variability of *C. lindemuthianum*.

## 2.3 Bean rust [*Uromyces appendiculatus* (Persoon) Unger]

**Geographic occurrence and impact** Bean rust is one of the most economically important diseases of common bean and is distributed practically in all production areas worldwide including; the Americas, Africa, Europe, Australia and Asia. The disease causes yield losses that range from 25 to 100% in susceptible cultivars. Total yield losses, for instance, have been reported in parts of the USA and Africa (Liebenberg and Pretorius, 2010). In the Colombian Cauca valley alone, losses due to common bean rust were estimated at US\$1 million (Graham in Liebenberg and Pretorius, 2010). Extent of yield loss depends on, among others, the degree of susceptibility of the bean variety, the prevailing weather conditions and time/period of infection. The losses are because of reduced photosynthetic area of the affected leaves that culminates into reduced numbers of pods, reduced pod fill, and therefore, fewer seeds.

**Symptoms and Signs** Symptoms appear on most aerial parts of the common bean plant including stem and pods but are more prominent on the abaxial side of leaves (**Fig. 3**). In the early stages, rust symptoms appear on leaf surfaces as small yellow or white spots. These dots enlarge and become raised to form rust-colored pustules (uredinia) containing microscopic urediniospores, also called uredospores. Unlike on leaves, pustules on stems and pods are elongated. The pustules are usually surrounded by a chlorotic or yellow halo. As the plant grows older with the season, the uredinia develops into dark telia (a form of dark or black pustules) containing brown to black teliospores - survival structures allowing overwintering in some production areas.

One key feature of common bean rust is that unlike other leaf spots, the spores rub off onto the fingers. Severe infection may result in premature chlorosis, senescence, leaf curling, and leaf drop due to high respiration rates (Duniway and Durbin 1971). The view of the field looks like it has been scorched.



**Fig 3.** Bean rust (Courtesy RL Wick, Univ of MA, all rights reserved® 2024

**Biology and epidemiology** - The common bean rust disease is caused by the basidiomycete fungus *U. appendiculatus*. It is an obligate parasite that completes its life on the common bean host (autoecious) and capable of producing different types of spores (macrocytic) during its life cycle (Harter and Zaumeyer, 1941). Asexually, the life cycle begins with germination of the urediniospores on the aerial part of the host plant, usually the leaf. A germ tube containing cytoplasm and two nuclei then develops and grows over the leaf epidermis. The cytoplasm and two nuclei are known to migrate with the growing tip of the germ tube (Heath and Heath 1976). Triggered by abrupt change in the topography of the leaf surface and other signals, an appressorium forms from the germ tube tip over the stomatal opening. At this point, the cytoplasm and nuclei move into the newly formed appressorium, and mitosis occurs. An infection peg then forms from the appressorium and penetrates the stomata. Once penetration occurs, an infection hypha grows and colonizes the cells through the intercellular spaces. The fungus then develops haustoria (the feeding structure) and acquires nutrients and energy from the host mesophyll cells (Voegelé et al. 2001). Thus, from the point of germination, the urediniospore contains all the necessary resources and energy to complete this chain of processes which normally occurs within hours (Mendgen et al. 2006). For resistant varieties, a hypersensitive reaction is triggered that results in the death of the host cells at the infection site. Consequently, this may also result in premature death of the fungus before spore production (Wingard 1935 and Mendgen 1978 in Liebenberg and Pretorius, 2010). For susceptible varieties, however, asexual reproductive structures referred to as mycelia grow from the parasitizing fungus and aggregate to form sorii where more

urediniospores are produced. Approximately 7 – 10 days after urediniospore germination, the sorus will rupture through the leaf epidermis, creating uredinia, also known as pustules and release the spores into the atmosphere. Rust damage, as such, is a combination of parasitism and damage to the structural components of the leaf. The name of the disease comes from the rusty color of uredospores sloughing off the leaf.

The life cycle of *U. appendiculatus* is greatly influenced by environmental factors that sometimes induce the changeover from uredinial to telial production (Waters in Liebenberg and Pretorius, 2010). Under field conditions, however, pustules containing urediniospores, are replaced by dark brown teliospores at the end of the growing season as the host plant grows older. The single celled teliospores undergo Karyogamy (fusion of haploid nuclei) and germinate giving rise to the basidium. The basidium undergoes meiosis to generate four single celled basidiospores capable of germinating on any aerial surface of the bean plant, forming a single germ tube (Liebenberg and Pretorius, 2010). An appressorium develops from the germ tube that helps the fungus to adhere to the plant surface and from which a penetration peg emerges. This penetration peg is responsible for rupturing the epidermal cell wall and giving rise to a network of intra and intercellular hyphae that colonizes the apoplastic space of the leaf. From this network, sexual reproductive structures, spermatogonia (pycnia), arise and reach about 3 to 5mm in diameter in 6 – 7 days. As these structures grow, they rupture the leaf epidermis causing yellow chlorosis and distortion of the leaf. Positive or negative pycniospores (spermatia) are produced and released from the pycnia of opposite mating types. The two opposite spermatia are cross-fertilized largely by insects (Liebenberg and Pretorius, 2010) to form white aecium where aeciospores are produced. These aeciospores are able to infect the host plant and produce a uredium with uredospores eight to ten days later completing the *U. appendiculatus* life cycle (Groth and Mogen 1978). Unlike the urediniospores, basidiospores, pycniospores, and the white aeciospores are rarely seen under field conditions.

Bean rust is, primarily, disseminated through wind which disperses spores from one plant and area to another. Seasonal crossover is possible largely with teliospores, and to a lesser extent also urediniospores through plant debris. Prolonged periods of free water on the leaf surface because of cool to moderate temperatures and moist conditions promote development of the disease (Schwartz et al. 2004).

## Management

No single control or disease management measure prevents rust fungus infection. As such, integrated approach is encouraged and may include the following:

- **Cultural practices** control measures include crop rotation (two to three years), removal of volunteer plants, deep ploughing and removal of bean debris, which is the potential source of rust spores, early planting and maintaining recommended plant density.
- **Chemicals and Biologicals** - Several fungicides are available for the management of bean rust and are summarised in Liebenberg & Pretorius, 2010. They include systemic fungicides such as carboxins and protectants such as chlorothalonil. Nevertheless, use of fungicides increases production costs and may be a threat to the environment and human health especially when applied without good expertise.

- **Genetic resistance** - Use of resistant varieties is the most effective bean rust management strategy. Several race specific resistance genes have been identified from bean cultivars and have since been stacked into preferred varieties. For instance, BelNeb-RR-1 released as “Kambidzi” in Malawi and “Mkuzi” in South Africa are reported to have high levels of resistance to rust in Africa (Liebenberg et al. 2005). Although effective and sustainable, the emergence of new and diverse races of *U. appendiculatus* pose a major threat to the development of resistant cultivars.

## 2.4 Black root rot [*Thielaviopsis basicola* (Berkeley & Broome) Ferraris (= *Berkeleyomyces basicola*)]

**Geographic occurrence and impact** Based on geographical data from Geldenhuis et al. (2006) and Nel et al. (2018), a putative distribution was plotted for both *Berkelyomyces* species as causative agents for black root rot (Nel et al. 2019). The distribution map showed that both species are present in the Netherlands and South Africa while *B. basicola* occurs in Australia, Belgium, Ecuador, Indonesia and Uganda and *B. rouxiae* in Canada, Chile, Ethiopia, Israel, Switzerland and New Zealand (Nel et al. 2019). Although the two studies include only a small number of global reports, the two species are arguably distributed worldwide and so is the associated disease. Black root rot is commonly found in cultivated and non-cultivated soils. The disease has a wide host range that includes ornamental; many other legumes such as groundnuts and peas; carrots, tobacco, cotton and citrus. While its impact on common bean production is not documented in the literature, huge economic losses have been reported on other crops such as cotton (Niu et al. 2008, Nehl et al. 2004). Niu and colleagues, for instance, estimated an annual loss of about \$897 million in revenue on cotton in the year 2005 in the USA alone.

Since the mid-1800s *T. basicola* has been well known to be the cause of black root rot. Recent studies based on DNA sequence analysis, however, have shown that the *Ceratocystidaceae* family constitutes two distinct pathogens that are responsible for black root rot disease. The two, *B. basicola* and *Berkeleyomyces rouxiae* have been assigned to a new genus *Berkeleyomyces* (previously *Ceratocystis*) [Holland et al. 2019; Nel et al. 2019]. As such, *B. basicola* and associated nomenclature have been adopted in this section to refer to *T. basicola* and previously associated nomenclature.

**Symptoms and Signs** While the disease primarily attacks the root system, above-ground symptoms may be observed and include yellowing of leaves, plant stunting and wilting (Fig. 4). Severely infected plants may collapse and eventually die. Above-ground symptoms of black root rot are typical of many other root rots or root problems and often confused with *Pythium* root rot. The infected roots have dark brown to black elongated lesions that contrast sharply to healthy roots. Initial lesions are observed in the middle of the roots and proliferate in both directions. As the disease progresses, much of the root area takes on a black discoloration. In some cases, black lesions may extend into the crown and on the plant stem near the soil line causing stems to be necrotic. All these events induce above-ground foliar symptoms such as wilting. It must be noted however that most of these symptoms were determined when it was considered that the disease is caused by a single pathogen *T. basicola*. Now that two distinct species are known to cause black root disease, variation in

symptom expression may exist among crops based on host specificities (Nel et al. 2019).



**Fig 4.** Black root rot of common bean, (Courtesy S. Chatterton, Agric Agri-Food Can, all rights reserved® 2024)

**Biology and epidemiology** *T. basicola* (now *B. basicola*) is a soilborne, filamentous hemibiotrophic fungus that belong to the division *Ascomycetes*, Class *Sordariomycetes*, Order *Microascales*, Family *Ceratocystidaceae*. Genus *Berkeleyomyces* (previously *Ceratocystis*) [Holland et al. 2019; Nel et al. 2019]. Within the *Ceratocystidaceae* family and based on DNA sequence analysis *T. basicola* was found to represent two distinct species: *B. basicola* and *B. rouxiae* within the newly established genus *Berkeleyomyces* (Nel et al. 2019). The two are hemibiotrophic known to colonize living root tissue during the short biotrophic phase of their life cycles (Nel et al. 2019).

While fungi belonging to *Ascomycota* are known to produce asexual and sexual spores, a sexual stage in *Berkeleyomyces* remains hypothetical and yet to be validated (Nel et al. 2019). Asexually, *Berkeleyomyces* spp. start their infection cycle with a biotrophic and then

necrotrophic phase (Pereg, 2013). Stimulated by host root exudates, thick walled chlamydospores in the soil germinate on the root surface of the host. Contact with the host is made through a germ tube that grows and penetrates through root hairs, the root epidermal cells via wounds and through stomatal openings. From the germ tube, a penetration peg or an appressorium-like structure emerges that is used to penetrate the root tissue cells (Hood & Shew, 1997; Mauk & Hine, 1988). For common beans, however, it is reported that direct penetration without appressoria is also possible (Christou 1962). Upon penetration, infection hyphae develop and colonize the root tissue through intra and inter-cellular spaces. This constitutes the biotrophic phase of the life cycle. As infection progresses, infected cells become necrotic which leads to characteristic black discoloration of the plant roots (Nehl et al. 2004). This constitutes the necrotrophic phase. Distinctive dark-colored and muriform chlamydospores are later formed within the necrotic areas. These spores are produced in chains and contained within a vesicle until maturation (Pereg in Nel et al 2019).

The chlamydospores are pigmented, have thick walls and contain melanin to protect them from adverse conditions. These features, among others, enable chlamydospores to survive in soil for several years (Hewajulige and Wijesundera, 2014). Spores can be disseminated via contaminated soil, plant and soil amendment or directly by humans, livestock, farm tools and machinery. Insect vectors such as shore flies can spread the disease within and between fields (El-Hamalawi, 2008). Run-off and irrigation water may also aid in the dissemination of *Berkeleyomyces*. Cool, humid and wet weather favor development and spread of black root rot disease. Lloyd and Lockwood, 1963 reported that soil temperatures below 24–26°C increase disease severity in a variety of hosts.

**Management.** Pereg et al. (2013) summarized several available options for the management of black root rot and include the following:

- **Cultural practices** - Since *B. basicola* is a hemibiotrophic requiring a live host to complete its life cycle and produce new spores, rotation with non-hosts for up to 3 years reduces its inoculum density in soil and the incidence of black root rot. While monocots in general are preferred in such rotation (Holtz and Weinhold, 1994), some cereals which were considered non-hosts may increase spore load in the soil (Pereg et al. 2013). Care must therefore be taken in choosing the appropriate rotation crop.

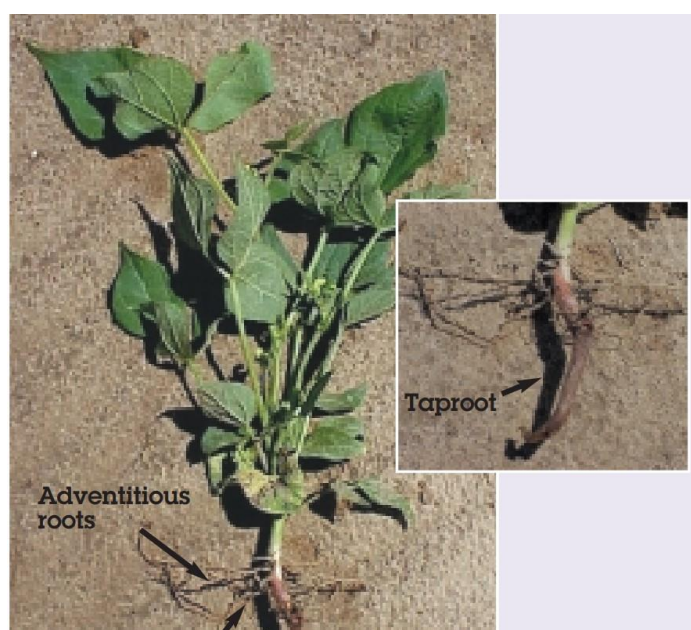
Addition of organic matter reduces the incidence of several soil-borne plant pathogens including *B. basicola* (Hewajulige and Wijesundera, 2014). High levels of ammonium are known to suppress growth of *B. basicola*, probably since plants exposed to high levels of ammonium produce substances such as putrescine that are toxic to the fungus (Pereg et al. 2013). Managing crop residues and general good farm hygiene is key to managing black root rot in common beans. Farm hygiene extends to cleaning protective wear, farm tools and machinery before leaving the field.

- **Chemicals and Biologicals** Application of acibenzolar-S-methyl reportedly induces systemic acquired resistance against black root rot and other pathogens. Based on studies with cotton, Mondal et al. (2005) indicated that with appropriate rates and application methods, the chemical has potential as a component in an integrated disease management strategy for black root rot.

## 2.5 Fusarium root rot [*Fusarium solani* (von Martius) Saccardo]

**Geographic occurrence and impact** *F. solani* is a soil borne persistent and destructive fungus, well adapted to a wide range of environmental conditions. It is generally considered ubiquitous in soils from any part of the world and more importantly where common bean is grown. As species complexes, however, there are suggestions that while some members of the species complex may occur in all soils, some have narrower distributions (Summerell et al. 2010). Potential yield losses due to *F. solani* are estimated to be up to 100%.

**Symptoms and Signs** - When planted on soil carrying the pathogen, symptoms will appear two or three weeks after planting (Román-Avilés, 2003) (**Fig. 5**). Narrow, long, red to brown streaks appear on hypocotyls and taproot surfaces. As the disease progresses, these symptoms may extend to the stems and longitudinal cracks often develop on the infected plant parts. The taproot eventually shrivels resulting in a reduced growth rate of the plant and general stuntedness. The infected plants turn light green to yellow due to disruption in the nutrient uptake and supply and may lead to plant death. In some cases, infected plants may compensate for the dying taproot by developing clusters of lateral or adventitious roots just above the shriveled main root (Román-Avilés 2003). Depending on the variety, and prevailing weather conditions, the reaction may mask above-ground symptom expression.



**Fig. 5** Fusarium root rot of common beans (Courtesy HM Young-Kelly, Univ Knox TN, all rights reserved® 2024)

**Biology and epidemiology** - *F. solani* belongs to the Kingdom Fungi; Phylum Ascomycota; Class Sordariomycetes; Order Hypocreales; Family Nectriaceae and Genus *Fusarium*. It was originally described as one species but recently been recognized as the *F. solani* species complex (FSSC) with up to 60 phylogenetically distinct species split into 10 *formae speciales* based on host specificity (Zitnick-Anderson et al. 2020, Matuo and Snyder 1973). The infection process begins with attachment of plant-pathogenic fungal spores to a susceptible

host. Different mechanisms of spore attachment have been reported by a number of authors and include virulence factor for some *Fusarium* species, production of spore tip mucilage and associated 90-kDa glycoprotein (reviewed by Coleman 2016). After spore attachment, entry into the host plant is primarily through stomates. Some isolates, however, can penetrate the hypocotyl directly through the epidermis aided by several cell wall degrading enzymes such as cutinase and pectin lyases. After invasion of the hypocotyl, hyphae spread to the upper portion of the tap root and the epicotyl, eventually colonizing the xylem and causing wilting of the above-ground portions of the host plant (Stahl et al. 1994).

**Persistence and transmission.** The chlamydospore can survive in the soil environment for many years and may be spread from one field to the other through water, wind, farm implements and crop residues. Although the host range of *F. solani* is relatively narrow, other alternative hosts such as soybeans may provide refuge for the pathogen and facilitate its spread across seasons and fields. In the absence of control/management practices, *F. solani* population increase with each successive cropping season.

## Management

- **Cultural practices** that promote vigorous plant growth are recommended to minimize disease damage. These cultural practices include long crop rotation (more than 3 years) with non-host crops e.g. wheat and corn; plant beans on well-drained and fertilized soil; avoid a dense plant population that may trigger competition for growth resources and favor infection due to stress; maintain adequate soil moisture levels and avoid soil compaction; ensure that field operations are done properly to avoid root injuries.
- **Chemicals and Biologicals** - So far, there are no cost-effective chemical or biological control measures for *Fusarium* root rot.

**Genetic resistance** - Although the most sustainable and durable approach for controlling the disease is through genetic resistance, currently available commercial cultivars are known to have limited *Fusarium* root rot resistance. Through recent studies, however, new sources of resistance to *Fusarium* root have been identified within common bean germplasm collections (Zitnick-Anderson et al. 2020). Use by farmers of bean genotypes with a high level of *Fusarium* root rot resistance can also provide significant contribution towards management and control of the disease. So far, there are no cost-effective chemical control measures for *Fusarium* root rot.

## 2.6 White mold (*Sclerotinia* timber rot) [*Sclerotinia sclerotiorum* (Lib.) de Bary]

**Geographic occurrence and impact** White mold caused by *S. sclerotiorum* de Bary affects the common bean (*Phaseolus vulgaris*. L) crop worldwide. Its global presence is guaranteed by the fact that it is capable of colonizing over 400 plant species, the majority being dicotyledonous species, although some monocotyledonous plants are also hosts (Bolton 2006). Favored by moderate temperatures (18–23 °C) and moist conditions, epidemics have been reported in a number of bean producing regions of Australia, Africa and Asia (Jones et al. 2011, Allen 1983). The disease results in both yield and quality losses. In Argentina, yield and quality losses up to 100% reported on susceptible common bean cultivars under favorable weather conditions. In parts of Europe, e.g. the UK, green beans have zero tolerance for infection and the entire crop may be rejected if contamination with sclerotia

(resting structures of the fungus) is detected. Annual losses from *S. sclerotiorum* in the United States, have exceeded \$200 million (Bolton et al. 2006).

**Symptoms and Signs** Under a conducive environment, the fungus infects all aerial parts of the host plants (leaves, stems, branches, flowers and pods) both in vegetative and reproductive growth stages. On the leaves of an infected plant, water-soaked lesions appear. Initially small, circular and dark green, these lesions expand rapidly and move down the petiole into the stem. Infected stems also develop water soaked or dark lesions which usually develop into necrotic tissues as the disease progresses. Subsequently, patches of fluffy white mycelium appear in internal and external tissues of the affected plant parts causing plant wilt and death (**Fig. 6**). During early stages of infection, there may be no noticeable changes in plant growth. However, as the disease progresses into the main stems, the plants start to wither and later appear bleached. Black and irregularly shaped sclerotia are typically formed on and within infected tissue which also tend to become shredded. Plants can be infected individually or in small patches. Once the disease has been initiated, however, infection can spread to adjacent plants through plant-to-plant contact (Bolton et al. 2006).



**Fig 6.** White mold of bean caused by *Sclerotinia sclerotiorum* (Courtesy of R. Harveson, Univ of NE, all rights reserved® 2024)

**Biology and epidemiology** - *S. sclerotiorum*: belongs to the kingdom Fungi, phylum Ascomycota, class Discomycetes, order Helotiales, family Sclerotiniaceae, and genus *Sclerotinia*. The development of a sclerotial stroma (sclerotia) – a hyphal aggregate containing melanin, is the common characteristic of all members of the Sclerotiniaceae. The structure is believed to aid in protection from adverse conditions and microbial degradation in many fungi. The fungus is a necrotrophic homothallic pathogen, which reproduces sexually by self-fertilization, resulting in the formation of apothecia that produces clonal ascospores, and asexually by mycelial germination of sclerotia. Infection is by ascospores which are discharged forcibly from apothecia into the air. These spores primarily land on senescent blossoms but also colonize other dead or dying tissues (leaves, cotyledons) of the host plant (Miklas et al. 2013). The dead tissues or senescent blossoms are used as a nutrient source for germination. After germination, an appressorium then develops which is used for mechanical penetration through the epidermis of the host plant. Penetration into the host tissue is also possible by use of cell wall degrading enzymes or passively through the stomata. Mycelial

infection normally arises from neighbouring infected plants and from germinated sclerotia in the soil. So far, only a few plants e.g. sunflower is known to be infected by mycelial invasion of the roots (Miklas et al. 2013). In both cases, there is rapid mycelial growth in the invaded tissues that may last several days resulting in the development of small, compact bodies (sclerotia) either on the surface or within cavities of an infected region. At first, they look fluffy white but turn black when mature. These aggregates of mycelium are the young resting vegetative structures of the pathogen and can remain viable in the soil for up to 9 years (Adams and Ayer 1979). When conditions are favorable, sclerotia may germinate into infective hyphae that can, in the later stages of development directly invade the host plant. This constitutes myceliogenic germination. Alternatively, sclerotia may germinate and produce apothecia from which ascospores, the sexual spores of the fungus, are developed in large numbers. After maturity, ascospores are released and some land on susceptible plants. Dictated by environmental conditions, these spores may germinate, and a new infection cycle begins. The latter constitutes carpogenic germination (Willems and Wong 1980). High humidity and wet plant canopies provide a conducive environment for the spread of the disease such that development of the disease is often towards the end of the season. A key factor that contributes to the epidemiology of *S. sclerotiorum* diseases is the massive reproductive potential and the long-term survival ability of their reproductive structures in the soil. Besides being able to remain viable for up to 9 years, Cosic et al. (2012), demonstrated that after three years, the percentage of viable sclerotia can be up to 100%. In addition, *S. sclerotiorum* has a very wide host range – over 400 species making it more persistent in cultivated areas. Ascospores are airborne and can be dispersed in the air over long distances. Along with mycelia, they may also be moved from one point to another through soil, running water, rain, farm implements, humans, etc.

## Management

- **Cultural** management options for *S. sclerotiorum* include planting of bean varieties with less dense canopies to promote aeration and reduce humidity; increasing soil organic matter that improves soil properties, plant health and yield; crop rotation with about 4 years between susceptible crop species; and avoiding over-irrigation that may create a favorable environment for disease outbreaks. Although environmentally friendly, cultural practices are inadequate, especially for high value crops or highly specialized farms.
- **Chemicals and Biologicals** When applied before germination, during growth or after harvest, there are chemical alternatives for the control of white mold disease. Switch (cyprodinil + fludioxinil) and Filan (boscalid) for instance, are effective for the control of white mold in green beans. Application timing, however, is critical for the best results. Switch and Filan applied at flowering have been shown to reduce Sclerotinia by more than 80%.

Many microorganisms, particularly *Coniothyrium minitans* have shown mycoparasitic activities towards *S. sclerotiorum*. Contans®WG, a commercial formulation of *C. minitans* (strain CON/M/91-08), is reportedly effective in minimizing damage caused by *S. sclerotiorum* to several crops including beans (EFSA 2016). The mycoparasitic activity of this formulation is, however, affected by factors such as temperature and pH with optimum temperature being 15-20°C and pH 4.5-5.6 (Zeng et al. 2012). While fungi of the genus *Trichoderma* have also demonstrated parasitism of *S. sclerotiorum* sclerotia by the significant reduction of apothecia density (Geraldine et al. 2013), their mycoparasitic activity under field

conditions remains sketchy. Meanwhile, common bean commercial cultivars with white mold resistance are not available, but resistance genes have been identified in some accessions under controlled environment and field trials in wild beans.

## **2.7 Damping-off or Rhizoctonia root rot [*Rhizoctonia solani* Kühn]**

**Geographic occurrence and impact** *R. solani* causes economically important root and hypocotyl diseases in common bean and is present all over the world. Yield losses due to the disease are as high as 100% (Singh and Schwartz 2010). In southern Australia, *Rhizoctonia* alone causes an estimated loss of \$77 million each year in production (Roget, 2006). Besides members of the Fabaceae family that includes common beans, *R. solani* inflicts damage on a wide range of other economically important crop species belonging to the Poaceae, Solanaceae, Amaranthaceae, Malvaceae and Brassicaceae families among others (Ajayi-Oyetunde and Bradley 2017).

**Symptoms and Signs** - While different *R. solani* isolates cause either aerial or below-ground symptoms, Valentin-Torres (2016) reported that some *R. solani* isolates cause both web blight of the above ground structures and root rot symptoms of the bean host plant. Below-ground symptoms induced by *R. solani* isolates include lesions on hypocotyls and roots that begin as small, sharp-edged oval to elliptical, reddish-brown sunken areas (**Fig. 7**). As the disease progresses, the lesions enlarge and may girdle the stem causing damping-off of seedlings. On older plants, the pith is attacked turning it brick-red, leading to stunting and consequent death of the plant (Hua 2014).

Above-ground symptoms constituting web blight of beans caused by some *R. solani* isolates include lesions on the primary leaves that appear as small necrotic spots with brown centers and olive-green margins. These lesions are typically induced by sclerotia or fungal hyphae and they normally coalesce thereby affecting the entire leaf under normal environmental conditions and in the absence of any control measures. Light brown or brick red, small, necrotic, circular lesions may appear when induced by basidiospores. These lesions, however, do not enlarge much nor coalesce. Later stage symptoms include yellowing of the lowermost leaves followed by wilting of plants (Chaudhary et al. 2021, Hua 2014).



**Fig. 7.** Rhizoctonia root rot of Common bean (Courtesy H. Schwartz, CO State Univ, Bugwood.org, all rights reserved® 2024)

**Biology and epidemiology** - *R. solani* belongs to kingdom Fungi, subkingdom Dikarya, phylum Basidiomycota, subphylum Agaricomycotina, class Agaricomycetes, order Ceratobasidiaceae, family Cantharellales, genus *Rhizoctonia*. Propagules of this fungus include the thick-walled, melanized hyphae (sclerotia), mycelium and basidiospores. In the absence of a host plant, *R. solani* can persist for many years in the soil via sclerotia and also mycelium on decomposing plant debris. Basidiospores are airborne, very fragile and they only survive on host plant parts (Naito 1996). The role of basidiospores as an inoculum source for the seedling diseases they incite on some crops such as soybean remains unknown (Ajayi-Oyetunde and Bradley 2017).

Most *Rhizoctonia* diseases result from mycelium and/or sclerotia infections. Under favorable field conditions and triggered by secretions of the host plant roots, sclerotia germinate to form mycelia that grow towards the host plant. Mycelial growth is succeeded by hyphal attachment to the host's external surface. After attachment, *Rhizoctonia* hyphae proliferate along walls of the host's epidermal cells and start to form special T-shaped branches with appressoria-like infection structures. Later, infection pegs formed from swollen hyphal tips on the infection cushions penetrate the cuticle and epidermal cell walls leading to the collapse of cell structures while the pathogen derives nutrients from the host. *R. solani* is a necrotrophic pathogen that kills its host in advance of colonization promoted by the presence of extracellular enzymes synthesized by the fungi and exudates released by actively growing cells of plant roots (Ajayi-Oyetunde and Bradley 2017, Hua 2014). Following the establishment of the pathogen in the host tissues, there is rapid hyphal growth and colonization of the dying host tissues along with formation of sclerotia leading to the host's expression of symptoms, such as soil rot, stem rot, damping-off, etc. (Chaudhary et al. 2021).

Several factors affect *R. solani* epidemiology. For instance, the pathogen is said to be more aggressive at temperatures between 15 and 18°C and in moist soils (Mayo 2015). *R. solani* spreads rapidly through water (irrigation or rain), soil movement (by farm implements or human beings), and infected crop residues during land preparation and by using infected planting materials. Although spread via airborne basidiospores is possible when the teleomorph (sexual) stage is present, this mode of spread rarely occurs in the field. Vegetative mycelia and sclerotia of the asexual stage play a critical role in disease epidemiology as they can colonize the soil environment and remain viable for several years. Spread from plant to plant is through the formation of mycelial bridges between plant roots and infested soil debris (Oladzad et al. 2019).

**Management** For a necrotrophic pathogen like *R. solani* has a wide host range, integrated disease management strategies are considered essential for reducing disease pressure (Ajayi-Oyetunde and Bradley 2017).

- **Cultural practices** - These measures involve agronomic practices such as crop rotation (3 to 5 years between two susceptible crop species); deep ploughing for good soil drainage; avoiding crop residues that may harbour the disease; good plant nutrition to increase the plant's chances of withstanding the disease; minimise soil contamination through farm implements and protective clothing and control of irrigation water to minimise excess run off and water logging.
- **Chemicals and Biologicals** - While the use of fungicides is restricted in some regions, they may be used in integrated disease management. Fungicides like pyrimidine derivatives for instance are known to be effective against *R. solani*. Under field conditions, soil treatment with benomyl also reduces the rate of damage by *R. solani* in beans (Hussein et al. 2018). Azoxystrobin has been used in some parts of the United States where *Rhizoctonia* root rot is problematic.

Many microorganisms have been evaluated for the control of *R. solani*. Among others, many *Trichoderma* species are well known for their ability to antagonize and suppress *R. solani* activities in soil (Mayo et al. 2015). Effectiveness of bio-control measures, however, is subject to several factors around the environment, pathogen species and crop variety.

## 2.8 *Pythium* root rot [*Pythium* spp.]

**Geographic occurrence and impact** *Pythium* species are spread worldwide.

Damping-off and root rot disease caused by *Pythium* spp. have a devastating impact on quantity and quality of common bean and a range of other different crops of economic importance (Li et al. 2014). The disease is considered one of the major constraints to common bean production in many parts of the world including the east and central African countries of Burundi, the Democratic Republic of Congo, Kenya, Rwanda and Uganda, among others. Yield losses of up to 70% have been reported in Rwanda and Kenya (Nzungize et al. 2012). Between 1991 and 1993, severe outbreak of root rot diseases partly caused by *Pythium* spp stopped farmers from growing the crop which led to serious shortage of beans and price increment (Nekesa 1998 in Nzungize et al. 2012). Li et al. (2014) reported a widespread occurrence of eight *Pythium* species causing common bean root rot disease in Western Australia with

some species exhibiting high degree of virulence and severe symptoms on the crop. Over 20 different *Pythium* species have been reported and vary widely with host, geography and optimum temperature for infection and pathogenicity.

**Symptoms and Signs** Plants may be infected by *Pythium* species at different growth stages - including planted seeds prior to germination causing seed decay and death; germinating seedlings causing poor seedling establishment and consequent seedling death; young or older plants during blossoming and pod formation causing a variety of growth disorders (Schwartz et al. 2007). Initial root rot symptoms appear typically two to three weeks after planting and encompass elongated water-soaked areas on the hypocotyl and root surfaces (**Fig. 8**). These water-soaked lesions extend upwards above the soil surface and start to dry, becoming sunken and turning brown in the process. Meanwhile, extensive premature defoliation may occur accompanied by reduced growth rate and devastation of the hypocotyl and main root system. Eventually, this leads to wilting and death of the plant. Depending on the species and bean variety involved, disease development may occur later during pod formation when pods contact infected soil. In this case, watery soft rot or mass of white fungal growth appear on developing pods.



**Fig. 8.** *Pythium* seed/root rot of bean (Courtesy H Schwartz, CO State Univ, Bugwood.org, all rights reserved® 2024)

**Biology and epidemiology** *Pythium* is a complex genus containing over 200 described species with a broad host range. Several species are responsible for root rot in beans occupying a variety of terrestrial and aquatic ecological habitats. For instance, eleven pathogenic species were identified in Tanzania, all causing crown necrosis of common bean plants with *P. aphanidermatum* and *P. splendens* being the

most widely distributed (Binagwa et al. 2016). In a similar study, eight different *Pythium* species were isolated from common beans in Western Australia (Li et al. 2014). All the isolated species, except *P. perplexum*, were pathogenic to the hypocotyl and root of common beans. Among the species, *P. irregulare* was the most widespread. *Pythium* spp. belong to the kingdom Chromista, Phylum Oomycota, class Oomycetes, order Pythiales, family Pythiaceae, and genus *Pythium* (Kirk et al. 2008). Members of the genus *Pythium* are no longer considered to be true fungi although they share some similarities with true fungi by having vegetative (mycelium), asexual (sporangia and zoospores), and sexual (oospores) stages in their life cycle (Ho 2018). In the presence of exudates released from the germinating seeds, the propagules of *Pythium* spp. germinate and penetrate the host plant through the formation of a germ tube (Arora 2021, Nzungize et al. 2012, Martin and Loper 1999). Once penetration takes place, *Pythium* grows and colonizes a plant by producing hyphae, threadlike, filamentous cells that extract nutrients from the host plant. Sexual reproduction involves the female organs, the oogonia and the male organs, the antheridia. When two mating types meet, fertilization takes place to produce thick-walled oospores. Oospores are the primary survival structures and infecting unit of many *Pythium* species. Under favorable conditions oospores germinate to produce more hyphae or develop a zoosporangium in which motile zoospores are produced. The latter constitutes asexual reproduction. The zoospores are not formed directly in the zoosporangium itself but in a vesicle that is connected to the sporangium through a tube (Stanghellini et al. 1971). Under wet conditions, the zoospores are released and swim away in divergent directions (Nzungize, et al. 2012). Upon reaching plant root surface and in the presence of favorable conditions, zoospores form cysts that then germinate and penetrate plant root. In the absence of conditions suitable for infection, zoospores will still form cyst but remain viable in soil until soil moisture content and temperature are favorable (Martin and Loper 1999).

*Pythium* spp. are naturally attracted to root exudates from germinating plants which stimulate their growth (Arora 2021). This alone perpetuates their prevalence in the soil environment. Their level of damage, however, depends on other factors that include soil moisture, temperature, and organic matter content (since they are also able to survive saprophytically) and more importantly the pathogen species under consideration. While generalizations about the influence of temperature on disease severity caused by *Pythium* sp. should be approached with caution, Pieczarka and Abawi, (1978), Sippell and Hall (1982) reported that *P. ultimum* caused less disease to bean at 27 to 28°C than at temperatures below 21°C. On the other hand, root rots caused by *Pythium* spp. are more prevalent in fields with wet soils than in fields with low soil moisture (Martin and Loper 1999). The pathogen can be transported within and between fields by contaminated irrigation water, crop residues and weeds, wind, farm implements, and humans. Poor crop rotation may also aid in the build-up of the pathogen.

## Management

- **Cultural practices** - bean rotation with maize reduces *Pythium* root rot disease incidences. Since the disease thrives well in soils with high soil moisture content, improving water drainage is vital. This can be achieved by deep ploughing and growing beans on raised ridges. Buruchara (1991, 1993) reported that incorporating

leaves and twigs of *Leucaena* spp., *Calliandra magrantha* and *Sesbania* as green manure two weeks before planting reduced plant mortality and increased bean grain yield.

- **Chemicals and Biologicals** - Several chemical pesticides used for soil drenching, fumigation, and seed coating have proven efficient for the control of *Pythium* root rot diseases on beans (Arora 2021). These include etridiazole, carboxin, propamocarb hydrochloride, thiram, captan and captafol. Nevertheless, it must be considered that some chemicals are only effective during the vegetative growth stage (mycelium) of the pathogen and not during the resting phase (Nzungize 2012). Unless all safety measures are employed, chemical treatments may have other negative effects to human health and the environment. Use of a single active ingredient has also resulted in the development of resistant strains, such as the metalaxyl-resistant *Pythium* spp. Seed coating with chemical treatments is reportedly one of the safest and most economical means of controlling *Pythium* pathogens resulting in effective protection of seeds and young seedlings for about 2 to 3 weeks after sowing (Abawi et al. 2006; Schwartz et al. 2007). While these measures are effective, they may not be affordable and sustainable for poor farmers in most under-developed countries.

Biologically, *Pythium* pathogens can also be controlled by microorganisms that compete with the pathogen for growth resources in the soil, produce antifungal metabolites, parasitize the pathogen or trigger plant defence mechanisms (Arora 2021). Such microorganisms have been identified and include isolates of *Trichoderma* spp. and *Gliocladium* spp. that are antagonists of *Pythium*-induced soil-borne diseases. Success of the biological approach in the management of *Pythium* spp. is, however, complex because the pathogens occur in a dynamic environment at the rhizosphere interface and exist in soil in different forms (Nzungize 2012).

- **Genetic resistance** -Although no commercially available root rot resistant bean variety has been reported so far, resistance genes have been identified in some Central American and Andean gene pools including RWR 719 and AND1062 that are extensively used as parents to improve RRR in commercial cultivars (Dramadri 2020). Colored common bean genotypes have a higher level of resistance to *Pythium* than the white seeded bean genotypes. Meanwhile, the Central American beans are considered less susceptible to *Pythium* root rot disease than the Andean bean genotypes.

## 2.9 Angular leaf spot [*Pseudocercospora griseola* (Sacc.) Crous and Braun]

**Geographic occurrence and impact** Angular leaf spot has been reported in about 80 countries in the tropics and sub-tropics. Where common bean is extensively grown, Angular leaf spot is one of the most devastating and recurrent diseases with yield losses of up to 70% of the production (Canpolat and Maden, 2021). Variation in yield and economic loss due to Angular leaf spot is attributed to the environmental conditions, the level of resistance/susceptibility of the bean variety and virulence of the isolate. The disease affects the quality and marketability of seed across bean-producing areas of the world (Pastor-Corrales et al. 1998)

**Symptoms and signs** Angular leaf spot symptoms appear on the leaves, petioles, pods, seeds and stems of the host plant. On the leaves lesions start as small, grey spots on the lower surfaces which later extend to the upper leaf surfaces (**Fig. 9**). As the disease progresses, the lesions turn brown often round shaped on primary leaves and angular on trifoliate leaves. Delimited by leaf veins, the lesions become necrotic and may be covered with grey-black conidia. Later, the lesions coalesce causing chlorosis that leads to premature defoliation (Correa-Victoria *et al.* 1989, Saettler 1991). Pod, seed, petiole and stem symptoms consist of circular to elliptical red-brown lesions. The symptoms can be confused with those produced by other pathogens, such as *C. lindemuthianum* that cause the anthracnose, or those caused by *P. syringae* pv. *phaseolicola* that produces halo blight.



Fig. 9. *Cercospora* Leaf spot of bean (Courtesy H Maryam Shahzady et al. 2017 <http://dx.doi.org/10.22576/ijfsf/sf-2017-84>, all rights reserved ©2024)

**Biology and epidemiology** *P. griseola* belongs to kingdom Fungi, phylum Ascomycota, subphylum Pezizomycotina, class Dothideomycetes, subclass Dothideomycetidae, order Mycosphaerellales, family Mycosphaerellaceae and genus *Pseudocercospora*. Its life cycle is hemibiotrophic with two distinct phases – biotrophic and necrotrophic. Under moist conditions, conidia of *P. griseola* germinate on the leaf surface of a host plant by forming a germ tube that elongates along the epidermal cells. Once over the stomata, penetration takes place through formation of an appressorium-like swelling and in other cases without the formation of the penetration structure (Monda et al. 2001). After penetration, hyphae emerge from the swellings or the germ tube and colonize the air spaces of the mesophyll and palisade tissue. This early intercellular growth of hyphae constitutes the biotrophic phase. Subsequent hyphal growth results in damage of the cell structures such as the disintegration of the chloroplast envelop and the plasma membrane. Since there is no evidence of direct penetration of the host cells and intracellular growth of hyphae, Monda and colleagues suggested that damage of the plasma and chloroplast membranes could be a function of toxins produced by the pathogen. Nevertheless, proliferation of the hyphae that causes plasmolysis of the host cells constitutes the necrotrophic phase of the life cycle of *P. griseola*. Later, a stroma (mass of fungal tissue that has spore-bearing structures) is formed. The stroma permits the fungus to remain dormant until favorable conditions for sporulation arise (Monda et al. 2001). As these structures grow, they protrude to the leaf surface either through the stomata or by rupturing the leaf epidermal wall. From the stroma, conidiophores develop and elongate to form synnema (joined conidiophores that are up to 500 µm in length). The cycle is completed by the formation of conidia at the tip of the conidiophore. The spores are spread over long distances by wind. Spread over short distances is also possible through rain splashes. The disease is favored by rain or humid conditions with a temperature range of 16-

28°C. The pathogen survives from one season to another through infected crop debris, volunteer plants and seeds.

### Management

- **Cultural practices** - Angular leaf spot management options include use of certified disease-free seed; removal of infected crop debris after harvest; crop rotation of at least 2 years with crops such as cassava and sweetpotato. Using these cultural practices, Brazil and Argentina reportedly minimised yield losses associated with the pathogen (Sartorato, 2002; Stenglein et al. 2003). Several fungicides such as Mancozeb, Captafol, Benomyl, Ferbam-Sulfur, Maneb, Metiram, Copper Oxichloride, Thiophanate methyl, Zineb, Ziram, and Bordeaux Broth, have proved to be effective in controlling Angular leaf spot (Stenglein et al. 2003). Nevertheless, use of chemicals is an expensive measure and has some potential threats to human health and the environment when poorly handled especially by small producers.
- **Genetic resistance** - Use of resistant varieties is the most effective way to control the disease. Already, sources of resistance have been identified that include AND 277 cultivar that is widely used in breeding programs (Nay et al. 2019).

### 2.10 Botrytis blight [*Botrytis cinerea* Pers. Fr. (teleomorph *Botryotinia fuckeliana* (de Bary) Whetzel)]

**Geographic occurrence and impact** With more than a thousand known hosts widely spread across the plant kingdom, *B. cinerea* is a cosmopolitan pathogen. The typical economic losses occur when the pods, flowers or leaves are damaged by the fast-growing necrotic lesions or by girdling stems. In severe epidemics 100% yield loss can occur (Davidson et al. 2007; Williamson et al. 2007). Worldwide, economic annual losses range from US\$10 to \$100 million (Boddy, 2016). The disease has a significant bearing on marketability of the harvestable parts since infection may not be detected at harvest but could develop rapidly under the moist conditions encountered during storage and transport, even at a temperature as low as 0 °C (Romanazzi et al. 2016). As such, countries globally have invested considerably in protecting the agricultural produce against *Botrytis* before and after harvest with the market size for anti-*Botrytis* products estimated at US\$ 15-25 million in recent years (Elad et al. 2004).

**Symptoms and Signs** The disease usually initiates on senescent flowers or cotyledons as dark, water-soaked, slimy lesions, with concentric zonation and sometimes a yellowish margin and spreads as a watery soft rot to the adjacent parts including the developing fruit/pods. The soft rots are often associated with the rapid appearance of grey masses of conidia (Williamson et al. 2007) (**Fig. 10**). Under humid conditions, *B. cinerea* can sporulate profusely on inflorescences and stems of the infected plant. Symptoms are more common on plants that have been damaged by wind, farm implements, frost or hail (Davidson et al., 2007).



**Fig 10.** Botrytis blight of bean on a seedling (Courtesy H Schwartz, CO State Univ, Bugwood.org, all rights reserved® 2024)

**Biology and epidemiology** *B. cinerea* is a necrotrophic pathogen belonging to the kingdom Fungi, phylum Ascomycota, subphylum Pezizomycotina, class Leotiomycetes, order Helotiales, family Sclerotiniaceae, and genus Botryotinia. The fungus has at its disposal, different survival structures that enable its existence under different habitats. They include mycelia, micro- and macro-conidia, chlamydospores, sclerotia, apothecia and ascospores (Holz et al. 2007). Inoculum sources for these propagules are within the crop, debris from a previous crop or from a different crop or weed host. While survival through mycelium is poorly understood under natural conditions, sclerotia are generally considered to be the most important structures involved in the survival of *B. cinerea*. From the primary source to a susceptible tissue surface, these propagules are dispersed by wind, rain and insects. By far, dry and predominantly wind-dispersed conidia are generally regarded as the most important dispersal propagules of *Botrytis* species and are found in highest concentration in the atmosphere during the daytime (Holz et al. 2007). This feature makes each part of the host plant a potential target for adhesion, germination and penetration. Under favorable conditions, conidia begin to germinate on the plant surface evidenced by the protrusion of the germ tube. When the germ tubes reach a length of about 10-15  $\mu\text{m}$  penetration structures, the protoappressoria develop which later give rise to infection pegs. Multiple routes used by the pathogen to enter the host have been reported and include undamaged host tissue (cuticle, trichome, guard cells); natural openings (stomata, micro-fissures) and wounds, caused by insects, farm tools etc. (Holz et al. 2007). Physical penetration of the undamaged host tissue such as the cuticle is mostly aided by enzymes that include cutinases and lipases and also hydrogen peroxide produced at the tip of the penetration peg (Boddy, 2016). During and after penetration, *B. cinerea* produces a wide range of chemicals that trigger hypersensitive cell death in the host tissue. Although this acts as a plant defense strategy, it is highly beneficial for necrotrophs such as *B. cinerea* as they can survive and sporulate as saprophytes on the necrotic tissue, or produce long-term survival structures, such as sclerotia. Conidia can be produced from sclerotia within the living plants or from plant debris lying on or buried in soil and serve as sources of inoculum for the next disease cycle.

## Management

Controlling *B. cinerea* is difficult because of its broad host range, various attack modes, and its multiple survival structures.

- **Cultural practices** - Removal of crop debris and rotation with non-host crops such as cereals will significantly reduce carry-over inoculum. Maintaining good crop density and judicious watering also help in reducing the onset of the disease (Davidson et al. 2007).

- **Chemicals and Biologicals** - For a long time, the principal means to control grey mold has been mixed sprays of synthetic fungicides such as thiram, mancozeb, captan, dichlofluanid, tolylfluanid and fenhexamid. Nevertheless, continuous use of these fungicides remains undesirable on environmental and economic grounds and may lead to rapid development of fungicide resistance.

To date, several products have been approved constituting biological control agents such as the filamentous fungi *Trichoderma harzianum*, *Clonostachys rosea* (*Gliocladium roseum*) and *Ulocladium oudemansii*, the yeast *Candida oleophila*, or bacteria *Streptomyces griseoviridis*, *Bacillus subtilis* and *Pseudomonas syringae* (Williamson et al. 2007). Unlike fungicides, biological control agents require specific temperature and humidity regimes for them to be effective. Compared with fungicides, biological control agents often have restricted ranges of temperature or humidity for maximum microbial action, and they may be influenced by a wide range of prevailing crop management practices.

### 3.0 Bacterial and Phytoplasma Diseases

#### 3.1 Common bacterial blight [*Xanthomonas campestris* (Pammel) Dowson syn. *Xanthomonas axonopodis* (Doidge)]

**Geographic occurrence and impact** - Common bacterial blight is widely distributed in over 104 countries across all six continents (Chen et al. 2021, Bultreys and Gheysen 2020). It is most common in regions where common bean is cultivated except in dry tropical areas. In Europe, the latest disease outbreak was reported in Belgium in 2019 (Bultreys, and Gheysen 2020). In Uganda, common bacterial blight is the most serious bacterial disease of common bean causing severe yield losses of up to 62% (Alladas et al. 2017). In Ethiopia, the disease affects seed quality and can reduce yield by up to 45%, or more in susceptible cultivars (Belete et al. 2017). The disease affects marketability of produce, especially infected seeds as they may not be sold in disease-free areas, even in the absence of symptoms (Chen et al. 2021).

**Symptoms and Signs** In the early stages of disease expression, symptoms appear as small, water-soaked spots on the leaves, usually more prominent on the underside. The spots enlarge and coalesce becoming dry, brown and brittle with distinct, rather narrow, lemon-yellow halos bordering the dried necrotic lesions (**Fig. 11**). As the disease progresses, the yellowing of leaves becomes more general while attached to the infected plants. Nevertheless, under severe conditions defoliation and plant death may occur. In systemic infection, the veins have a reddish-brown discoloration with a water-soaking of adjoining interveinal areas. Reddish longitudinal streaks may also appear in stems of an infected plant (Chen et al. 2021). Symptoms on affected pods appear as circular water-soaked areas that are slightly sunken. Later, these pod lesions turn brick-red or reddish-brown in color and are more deeply pitted. Under very humid conditions, yellowish bacterial ooze that later dries into yellow crusty masses appears to cover pod lesions. (Harverson, 2009). Under severe infection, the pods may shrink extensively and result in death. Symptoms on the seed include butter yellow spots that later turn brown. These symptoms appear either at the micropyle, hilum area or the entire seed surface depending on the infection pathway (Chen et al. 2021). In severe cases, infected seeds may shrink thereby affecting the germination and vigor of the seedlings.



**Fig. 11.** Bacterial blight of common bean (Courtesy of H. Schwartz, Colorado State University, Bugwood.org, all rights reserved® 2024)

**Biology and epidemiology** - Common bacterial blight pathogens belong to the Kingdom: Bacteria; phylum: *Proteobacteria*; class: *Gammaproteobacteria*; order: *Lysobacterales* (also known as *Xanthomonadales*); family: *Lysobacteraceae* (also known as *Xanthomonadaceae*); genus: *Xanthomonas*; species: *X. citri* pv. *fuscans* and *X. phaseoli* pv. *phaseoli*. *Xanthomonas* species are gram-negative aerobic, rod-shaped and move using a tail-like structure called flagellum. The two species responsible for common bacterial blight represent four genetic lineages (GL) namely: GL1 corresponding to *X. phaseoli* pv. *phaseoli* (Xpp, previously *X. axonopodis* pv. *phaseoli*); GL2, GL3 and GL fuscans corresponding to *X. citri* pv. *fuscans* (Xcf) [Constantin et al. 2016, Chen et al. 2021]. Sources of inoculum for common bacterial blight include seed contamination (both internal and external), bean crop debris, volunteer bean crops and perennial alternate host plants (including weeds). By far, contaminated seed is probably the main source of introducing the bacteria into new bean fields and areas (Akhavan et al. 2013). Subject to storage conditions, common bacterial blight agents may survive on infected seed for up to 30 years (Chen et al. 2021). Two phases are involved in *Xanthomonas* infection cycle: epiphytic and endophytic. Infection usually starts with epiphytic growth of the pathogen on the host-plant surface usually the leaf. With the help of biofilms, bacterial cells connect through an extracellular polymeric substance matrix composed of exopolysaccharides, extracellular DNA, proteins and lipids (An et al. 2020) The bacterial population grows and stabilizes on the host tissue. Penetration into the host tissue is through openings such as stomata in leaves and other plant organs, hydathodes in the leaf margins and wounds that may be created by various agents (Akhavan et al. 2013, Chen et al. 2021). Primary infection is through an infected seed that germinates, and the bacterium is transmitted from the seed to the seedling. Once penetration takes place, the bacteria enter the endophytic phase which is characterised by rapid multiplication of the bacteria until a threshold of about  $10^6$  bacterial cells per  $\text{cm}^2$  is reached (Weller and Saettler 1980). With this population threshold colonization of the vascular tissue begins. Progressive colonization of the internal host tissue structures leads to wilting of the plant. Upon reaching high population densities, the bacteria re-emerge onto the outer leaf surface and are transmitted mostly through wind or rain to a new host where the infection cycle starts again. When plants are wet, bacterial cells are also readily transmitted mechanically through humans, farm

implements or animals. Likewise, arthropods may transmit the bacterium from plant to plant. The disease is aggravated under optimal temperatures of 28 to 32 °C and a relative humidity above 80% (Chen et al. 2021).

## Management

- **Cultural practices** - As a seed borne pathogen, the use of pathogen-free seeds is certainly the most efficient management strategy. Disease free seeds may be produced in specified areas that are either free of common bacterial blight agents or whose climatic conditions are nonconductive for the disease. This, however, demands a vibrant seed system that would regulate the seed production process. Other cultural practices involve field sanitation through cleaning of farm tools/machines, seed containers, weeding, removal and disposal of crop debris, and long-term crop rotations of 3 years or more to limit the risk of contamination of common bean crops by pathogens surviving on alternate hosts.
- **Chemicals and Biologicals** - For decades, chemical control has been restricted to the application of copper and copper-based antimicrobial compounds in bean fields. Being vascular pathogens, however, the effectiveness of foliar copper application is limited. Moreover, in the past few decades there have been concerns on the environmental consequences of continuous application of these copper-based compounds with resistance to the pesticide becoming more prevalent (An et al. 2020). Other chemical alternatives include antibiotics such as kasugamycin and manganese-based foliar fertilizer (Viecelli & Moerschbacher, 2013) although for the latter high amounts may potentially have a negative environmental effect. The potential application of antimicrobial peptides as a biocontrol agent for *Xanthomonas* treatments is currently under investigation (An et al. 2020).

Naturally occurring antagonistic bacteria such as *Pseudomonas* spp., *Rahnella aquatilis*, *Rhodococcus fascians*, or *Bacillus* spp. can also reduce common bacterial blight disease either by suppressing the pathogen or by induction of host defense system. Several commercially available formulations derived from *Bacilli* and *Streptomyces* strains are available for foliar application (Van Hop et al. 2014)

### 3.2 Fuscos blight *Xanthomonas fuscans* subsp. *fuscans* [syn. *X. campestris* subsp. *phaseoli* (Smith) Dye]

**Note:** Based on recent classification (Constantin et al. 2016, Chen et al. 2021), *Xanthomonas fuscans* subsp. *fuscans* now is classified as *X. citri* pv. *fuscans* and is responsible for common bacterial blight described above.

### 3.3 Bacterial brown spot [*Pseudomonas syringae* van Hall]

**Geographic occurrence and impact** - Bacterial brown spot has been reported world-wide and causes significant yield losses in agricultural industries in the US, Africa, Australia, Asia, Brazil and Canada. In 1992 on the Transvaal Highveld, South Africa, an epidemic was recorded with 100% disease incidence and 55% yield losses on dry beans (Serfontein 1996). *Pseudomonas syringae* pv. *syringae* that causes bacterial brown spot has a wide host range, infecting more than 180 host plant species including woody plants and weeds (Muedi et al. 2015).

**Symptoms and Signs** Symptoms can be seen on leaves, pods and seeds of an infected plant.

However, the most distinctive symptoms on diseased crops occur on the leaves. Leaf lesions start as small (up to 1 mm) brown circular, water-soaked spots mostly visible on the underside of young foliage (**Fig. 12**). A thin pale-yellow ring develops around the circular spots. These small spots begin to coalesce and enlarge (up to 7mm) with the necrotic tissue in the center often turning pale brown or greyish and dark brown around the margin (Watson 1980). As the disease progresses, the necrotic area may dry out, become brittle and give the leaves a tattered or shot-hole appearance. Under severe cases, the disease can cause a lot of leaf defoliation. Depressed, red-brown, elliptical lesions may also appear on the stems or petioles of infected plants (Watson 1980). On pods, dark-brown lesions are seen but they are generally smaller, and necrotic and may cause pod malformation and consequently seed abortion. While common bacterial blight, halo blight and bacterial brown spot display similar symptoms characterized by brown necrotic centers, the ring of chlorosis [yellowing] is typically smaller with bacterial brown spot as compared to halo blight and common bacterial blight. Similarly, bacterial brown spot lesions are slightly smaller on the pods, than those induced by common bacterial blight. Probably the tattered appearance of leaves infected by bacterial brown spot is the key distinguishing feature among the three diseases.



**Fig 12.** Brown spot of bean (Courtesy of H Schwartz, Colorado State University, Bugwood.org, all rights reserved® 2024)

**‘Biology and epidemiology-***Pseudomonas syringae* pv. *syringae* belongs to the Kingdom: Bacteria; phylum: *Proteobacteria*, class *Gammaproteobacteria*; order: *Pseudomonadales*; family: *Pseudomonadaceae*; genus: *Pseudomonas*; species *Pseudomonas syringae*; pathogenic variety *syringae*. *P. syringae* is easily identified as a gram-negative strict aerobe, rod shaped with polar flagella. The life cycle of *P. syringae* constitute two interconnected phases: the epiphytic phase, when the bacteria live on the surface of plant tissues (leaves, stems and fruits, collectively referred to as the phyllosphere), and the endophytic phase, when bacteria enter the host tissue and colonize the intercellular space called the apoplast. While many *P. syringae* strains, such as those of *Pseudomonas syringae* pv. *syringae* are strong epiphytes, they must make a transition from the epiphytic phase to endophytic phase to cause

disease. They achieve this by entering the plant tissue and multiplying exponentially within the apoplast. Entry into the host tissue is through stomata and other openings resulting from plant injuries including frost injuries induced by the ice-nucleation gene of the pathogen (Xin et al. 2018). Upon successful entry into the host tissue, the endophytic phase characterized by colonization of the apoplast begins. Several bacterial virulence strategies are employed by the pathogen to suppress plant immunity and ensure living in the apoplast. In the leaf apoplast, *Pseudomonas syringae* pv. *syringae* initially propagates biotrophically, keeping the host cells alive, and later causes necrotic lesions. Although *Pseudomonas syringae* pv. *syringae* is a common leaf epiphyte, disease outbreaks are often seasonal and conditional. Disease outbreaks under such conditions and seasons are due to abundant inoculum accumulated epiphytically before the predisposing conditions arise. bacterial brown spot development is favored by high humidity, wet conditions, and temperatures from 12 to 20°C. *Pseudomonas syringae* pv. *syringae* has a very low seed transmission rate. Sources of primary infection are usually weed hosts on which the bacterium survives as an epiphyte on the leaf surface. It can survive on plant debris for one year. Spread of the pathogen is by wind-blown rain or overhead sprinkler irrigation.

**Management.** Since bacterial pathogens cannot be eliminated completely, management options demand integrated crop production strategies that reduce or delay disease severity during the critical periods of vegetative and reproductive plant growth.

- **Cultural practices** - A three to four-year rotation may effectively reduce overwintering populations. Disease outbreaks may be minimized through proper sanitation of bean crop debris. This involves complete incorporation of straw into the soil and the elimination of volunteer beans early the following year. Proper sanitation is also required on farm equipment, seed-cleaning equipment, storage containers and protective wear.
- **Chemicals and Biologicals** - Sprays of copper compounds have been used for decades as standard bactericides to combat many bacterial diseases, but their use is subject to a number of constraints including lack of systemic activity, difficulty in timing, emergence of resistant strains, phytotoxicity and the increasing demand for organic crops. Kennelly et al. (2007) suggested, therefore, that applications of copper must be timed to coincide with periods when the host is susceptible, when the pathogen is accessible, and when conditions are favorable to the disease. Antibiotics such as streptomycin and oxytetracycline, which are registered in some countries, have also been proved effective for the control of bacterial diseases such as bacterial spot of peach. Furthermore, their long-time use may result in development of resistance in *Pseudomonas syringae* pv. *syringae* as has been observed elsewhere (McManus et al. 2002). Systemic acquired resistance of the host plants can be induced by application of Acibenzolar-S-methyl (ASM, trade name Actigard in the United States and Bion in Europe) (Kennelly et al. 2007).

**Genetic resistance** to bacterial brown spot has been identified within some bean lines. These traits are yet to be introgressed into preferred bean cultivars around the globe. Meanwhile, the large-seeded bean varieties are a little more tolerant to brown spot than the small-seeded.

**Halo blight [*Pseudomonas savastanoi* pv. *phaseolicola* (Burkholder) (syn. *P. syringae* subsp. *phaseolicola* (Burkholder))]**

**Geographic occurrence and impact** - Halo blight caused by *P. savastanoi* pv. *phaseolicola* has a worldwide distribution, occurring in more than 50 countries, over five continents (Marques and Samson 2016). It is particularly damaging in the Eastern and Midwestern United States (e.g., Colorado, Michigan, Minnesota, Nebraska, North Dakota, New York, Wisconsin, and Wyoming); the humid highland production areas of Guatemala and Mexico; Argentina; central and southern Brazil; Turkey; Spain; and various countries in Africa, including Malawi, Rwanda, Tanzania, and South Africa (Duncan et al. 2014). The first report of the disease in the US was 1925 with an estimated yield loss of 45%. In China bean halo blight was first reported in 1987 with an associated yield loss of 50%. It is considered a quarantine organism in China and the US among other countries (Sun et al. 2017; Arnold et al. 2011). Under severe conditions, however, yield losses may be as high as 80 to 100% (Schoonhoven and Voysest 1989).

**Symptoms and Signs** - Halo blight symptoms appear on leaves, pods, and seeds. On leaves, symptoms initially appear as small, greasy water-soaked spots generally most visible on the underside of young leaves about one week after infection (Schwartz 2011) (**Fig. 13**). Later the spots enlarge into angular necrotic lesions that are surrounded by a light green to yellow halo that is caused by a bacterial toxin, phaseolotoxin. Development of the halo is said to be a diagnostic symptom of the disease (Duncan et al. 2014). Development of the halo, however, is dictated by the prevailing environmental conditions and may not be produced under high temperatures (Schwartz 2011). With time and under favorable conditions, foliar lesions enlarge and coalesce into necrotic areas that may result in defoliation. The disease may become systemic under severe infection causing stunting, yellowing and subsequent death of newly emerging leaves. Symptoms on pods also begin as small, circular, water-soaked spots or streaks that expand into large circular spots surrounded by a necrotic brown border. Infected pods are associated with a light cream or silver-colored bacterial ooze (Schwartz 2011). There is often discoloration and pods shrivel. As the lesions extend into the pods, the seed may be contaminated resulting in poor seed development, and abnormal shape and size of the seed and pods. Under less severe conditions or with tolerant varieties, however, seeds may show no visible symptoms. Seedlings that develop from infected seed are systemically infected and usually have the characteristic halo blight symptoms on stems, cotyledons, and first true leaves.



**Fig. 13.** Halo Bight of bean (Courtesy RL Wick, Univ MA, all rights reserved® 2024)

**Biology and epidemiology-** *P. savastanoi* pv. *phaseolicola* belongs to the Kingdom: Bacteria; phylum: *Proteobacteria*, class *Gammaproteobacteria*; order: *Pseudomonadales*; family: *Pseudomonadaceae*; genus: *Pseudomonas*; species *Pseudomonas savastanoi*; pathogenic variety *phaseolicola*. However, a pathovar of *P. syringae* pv. *phaseolicola* has been re-classified as *P. savastanoi* pv. *phaseolicola* following DNA studies by Gardan et al. (1992). The synonym *P. syringae* pv. *phaseolicola* is, nevertheless still in common usage. The disease cycle begins with an already existing or introduced *P. savastanoi* pv. *phaseolicola* population on the leaf surface of the host plant. Prior to enabling the environment for their germination, the bacteria multiply epiphytically and may disperse through neighboring plants without causing disease symptoms (Marques and Samson, 2016). The population density of *P. savastanoi* pv. *phaseolicola* on the host's leaf surface during the epiphytic phase does not determine the disease severity level once infection occurs. Stomata and wounds on the leaf surface will warrant entry of the pathogen when conditions are conducive. Apparently, under field conditions foliar spots generally appear right after heavy rain, inducing infection. Like most endophytes, *P. savastanoi* pv. *phaseolicola* colonizes the intercellular spaces in the epidermal and cortical regions and in some cases. At lower cell densities, the pathogen can also invade the vascular tissue therefore allowing its spread into different host tissues. Transmission from plant to plant and field to field is aided by wet weather, hail, violent rain, and windstorms. The bacteria can overwinter in infested debris or seed with the latter being the most important inoculum source for movement over longer distances.

### **Management.**

*P. savastanoi* pv. *phaseolicola* is a quarantine pest in most developed countries such as the US, China and Israel. It is on the EPPO A1 list in Brazil, Paraguay, Uruguay, and Bahrain and on the A2 list in Chile and Jordan. It is on USDA's Harmful Organism list for Argentina, China, Guatemala, Honduras, Israel, Jordan, Panama, Paraguay, Taiwan, and Uruguay. These national regulations safeguard local agricultural industries from the potential negative impact of the disease.

- **Cultural practices** - As a seed borne disease, planting of pathogen-free seed, e.g., produced in arid environments and tested free of *P. savastanoi* pv. *phaseolicola* provides the most effective and practical management strategy. Other management practices include crop rotation (a minimum rotation of 2 yr), and deep plowing or removal of crop debris.
- **Genetic resistance** - Although planting of disease-resistant cultivars is among the desirable management strategy, its durability is limited by the considerable pathogenic variation that exists among strains of the bacterium (Duncan et al. 2014). Nevertheless, remarkable progress has been made in the development of race-specific and non-race specific resistant varieties. More importantly, high levels of resistance to *P. savastanoi* pv. *phaseolicola* race 6, one of the most virulent and economically important races, was identified and could also be used to generate germplasm lines and cultivars with resistance to all known races of *P. savastanoi* pv. *phaseolicola* (Duncan et al. 2014).

### **3.5 Bacterial wilt [*Curtobacterium flaccumfaciens* pv. *flaccumfaciens* (Hedges) Collins & Jones [syn. *Corynebacterium flaccumfaciens* subsp. *flaccumfaciens* (Hedges)**

**Geographic occurrence and impact** - Bacterial wilt *C. flaccumfaciens* pv. *flaccumfaciens* has been reported in countries across all the six continents. First described in South Dakota in 1922 (Hedges, 1922), the disease has continued to spread into South American countries including Brazil; then Spain in Europe, Iran in Asia; and Kenya, Mauritius, South Africa, and Tunisia in Africa. Other than common beans occurrence of bacterial wilt caused by *C. flaccumfaciens* pv. *flaccumfaciens* has been reported on a number of crops within the Fabaceae family including soybean in Zambia and Germany; mungbean and cowpea in Australia and Iran (Osdaghi et al. 2020). Following the first report of its occurrence in 1922, *C. flaccumfaciens* pv. *flaccumfaciens* became one of the most important bacterial diseases of beans in the USA, causing up to almost total losses in some years (EPPO 2011). Harveson et al. (2015) later reported disease incidences as high as 90% in areas where the disease is endemic with yield reduction of 60 to 65% and another 10 to 15% of discolored seed. After about 20 years of being regarded as a disease of minor importance, the recent *C. flaccumfaciens* pv. *flaccumfaciens* epidemics in North America, associated with novel pigmented variants, present “a new threat from an old enemy” (Osdaghi et al. 2020) which may result in significant economic implications.

**Symptoms and Signs** Early symptoms of bacterial wilt consist of general wilting of the plant foliage during warm, dry weather or periods of moisture stress. This is attributed to blockage by the pathogen of the vascular tissue which limits water movement from roots to foliage (Harveson et al. 2015). At lower temperatures, typically in the evening/night or during cloudy or wet weather, the plants tend to recover. Symptoms on leaves appear as foliar wilting and chlorosis leading to necrotic areas that are surrounded by a yellow halo (**Fig. 14**). Young plants or seedlings with primary infection (through seed), are worst hit by the disease. Once attacked, seedlings of about 5 to 7.5 cm tall rarely recover and will die shortly after emergence (Harveson et al. 2015). Those that survive remain stunted with severe yellowing and wilting symptoms and do not produce fertile pods (Osdaghi et al. 2020). In contrast, symptom development on older plants is slow and less prominent. However, when these infected plants reach podding stage, seed set may be severely reduced, and seeds are usually discolored; a common symptom in bacterial wilt disease. Seed discoloration is more common in white-seeded cultivars. *C. flaccumfaciens* pv. *flaccumfaciens* may cause yellow, orange, pink, or purple discoloration of the seed coat while the external surface of the pod appears symptomless (Harveson, 2013). Unlike the common bacterial blight pathogen *Xanthomonas axonopodis* pv. *phaseoli*, lesions caused by *C. flaccumfaciens* pv. *flaccumfaciens* occur between veins (interveinal necrosis with yellow haloes) often accompanied by wilting and firing symptoms that result in death of severely affected plants. Wilting and plant death are not common with common bacterial blight, halo blight and brown spot caused by *P. savastanoi* pv. *phaseolicola* and *P. syringae* pv. *syringae*, respectively (Harveson and Schwartz, 2007; Schwartz et al. 2005). Furthermore, water-soaked lesions are not a common feature of *C. flaccumfaciens* pv. *flaccumfaciens* induced symptoms in bacterial wilt as opposed to common bacterial blight, halo blight and brown spot.



**Fig. 14.** Bacterial wilt of common bean (Courtesy of H Schwartz, Colorado State University, Bugwood.org, all rights reserved® 2024)

**Biology and epidemiology** - *C. flaccumfaciens* pv. *flaccumfaciens* belongs to a group of non-spore-forming coryneform bacteria in the Domain: Bacteria; phylum: *Actinobacteria*; class: *Actinobacteria*; order: *Actinomycetales*; family *Microbacteriaceae*; genus: *Curtobacterium* and species: *Curtobacterium flaccumfaciens* pv. *flaccumfaciens*. (Osdaghi 2020). *C. flaccumfaciens* pv. *flaccumfaciens* is a seed borne pathogen that can survive and remain viable on seed for up to 24 years under laboratory conditions (Silva Junior et al. 2012). It can also cross seasons epiphytically through crop residues of host or non-host plants. On infected bean residues *C. flaccumfaciens* pv. *flaccumfaciens* may survive under field conditions for about eight months subject to the surrounding environmental factors such as soil type, moisture content, and climatic conditions (Silva Junior et al. 2012). Initial sources of infection for the new crop, therefore, includes infected seed and crop debris, the former by far being the most important means of introducing the pathogen into a new crop and production area (Zaumeyer and Thomas, 1957; Saettler and Perry, 1972). Infection occurs when the pathogen enters the vascular system of a new plant either through the infected seed or wounds and physical openings on leaves, stems or roots. In the soil, the root-knot nematode, *Meloidogyne incognita*, was found to provide a mechanism for introducing the pathogen into host plants (Schuster, 1959). Inside the host, the pathogen multiplies and colonizes vascular tissue causing associated symptoms on the host. The pathogen may be distributed through the vascular system to different host tissues including developing pods and seed embryos. Upon harvest, the infected seed and plant tissues provide an effective media for survival, both externally and internally until the occurrence of environmental conditions that favor re-infection. Bacterial wilt is favored by high temperature (>30°C). Storms, hail and strong winds provide good opportunities for pathogen infection and establishment within the plant as they can cause wounding of the host foliage (Harveson et al. 2015).

### Management

*C. flaccumfaciens* pv. *flaccumfaciens* is under the A2 (high-risk) list of quarantine pathogens by the European and Mediterranean Plant Protection Organization (EPPO), hence it is under strict quarantine control. The disease has zero tolerance in the bean industry in many countries (EPPO, 2011).

- **Cultural practices** - As a seed borne pathogen, use of certified seed which is disease free is highly encouraged. A minimum 2-year rotation with non-host crops such as small-grain cereals is yet another management option for reducing the build-up and spread of bacterial wilt. Meanwhile, proper field sanitation in general, and careful management of crop debris may significantly reduce inoculum from overwintering pathogens. For instance, incorporation of infected crop debris 20 cm deep in the soil results in significant reduction in survival rate of *C. flaccumfaciens* pv. *flaccumfaciens* unlike that of infested dry straw left on the soil surface (Silva Júnior *et al.* 2012).
- **Chemicals and Biologicals** - Surface contamination of seeds can be minimized through seed treatment with antibiotics such as Streptomycin Agri-Strep 500 (Schwartz, 2007). Complete elimination of the bacterium from naturally infected seeds is also possible by immersing the seeds in Agrimycin 500 solution, containing 500 g copper sulphate and 30 g oxytetracyclin per kilogram of the product in concentrations of 10 g/L of water for 30–60 min (Estefani *et al.* 2007). While use of copper-based pesticides has been effective and a traditional chemical option in the management of bacterial wilt, recent reports of resistance by some *C. flaccumfaciens* pv. *flaccumfaciens* strains have made the option less preferred (Osdaghi *et al.* 2020).  
Seed inoculation with an antagonistic bacterial suspension consisting of *Bacillus cereus* and *Pseudomonas fluorescens* strains was reported to reduce the severity of bacterial wilt and other bacterial diseases of common beans (Corrêa *et al.* 2014; Martins *et al.* 2013). Although several studies have investigated the efficiency of biological control in the management of bacterial wilt under controlled environment, none of them has been commercialized on a large scale while some remain under research (Osdaghi *et al.* 2020).
- **Genetic resistance** - Different bean varieties react differently to the disease with some being tolerant or resistant and others very susceptible. Where possible, use of resistant or tolerant varieties must be prioritized. Although use of tolerant varieties is recommended, some argue that these may also end up being carriers and help in the build-up of the pathogen. So far, resistant varieties have been bred such as Emerson in the central high plains of the USA. Access and use of this variety, however, is limited as it is a specialized cultivar with restricted target markets in Europe (Osdaghi *et al.* 2020).

## 4. Viral Diseases

### 4.1 Bean common mosaic virus (BCMV)

**Geographic occurrence and impact** - BCMV originated from Latin America, the center of diversity of *Phaseolus vulgaris* (Mukeshimana et al. 2003). Due to a wide host range within the legume family, BCMV is distributed worldwide wherever common beans and other legumes such as pea, cowpea and soybean are grown (Worrall et al. 2015). The disease thrives well in areas with medium rainfall and warm temperatures which favour the population build-up of aphids – the virus transmission agents. Yield losses due to BCMV have been reported as high as 100 percent depending on varieties, the growing environment, time of infection and mode of infection (primary as a seed-borne infection or secondary as aphid-mediated infection).

**Symptoms and Signs** BCMV mainly causes “common mosaic” symptoms characterized by light green or yellow and dark green mosaic patterns on leaves; green vein banding; leaf curling, distortion, blistering, and rolling (**Fig. 15**). Yellow dots or necrotic local lesions may result in plant stunting. Depending on the variety, some BCMV-infected plants may not express typical symptoms. Nevertheless, even where such symptoms are not expressed, yield reduction as a result of the BCMV may be as high as 50% (Morales 2006). Unlike with secondary infections, plants grown from infected seed are almost always stunted and less productive.

In susceptible bean varieties, BCMV and bean common mosaic necrosis virus (BCMNV) usually induce very similar symptoms. However, in bean cultivars that contain the dominant *I* resistance gene, all BCMV strains except for strains NL-2 and NL-6 do not trigger a hypersensitive necrotic reaction (black root disease) that results in the death of the plant (Worrall et al. 2015, 2019). At temperatures above 30 °C, strains NL-2 and NL-6 of BCMV can trigger this reaction in bean cultivars with the resistance gene resulting in what is referred to as “temperature-dependent” black root (Kelly et al. 1995, Silbernagel et al. 2001).



**Fig. 15.** Common Bean Mosaic virus (Courtesy Nick Volesky and Mair Murray, Utah State Univ. all rights reserved® 2024)

**Biology and epidemiology** - BCMV is a single-stranded positive-sense RNA virus that forms rod-shaped virions (Ivanov et al. 2014). The genome is about 10 kb long and has an open read frame (ORF) that translates into a self-cleaving polyprotein. A total of 10 mature and functional proteins resulting for the self-cleaving polyprotein have been described in order from the 5' end of the genome: P1 proteinase; helper component-proteinase (HC-Pro); P3; a 6 kDa protein; cylindrical inclusion (CI); a second 6 kDa protein; the genome-linked viral protein (VPg); nuclear inclusion 'a' (Nia); nuclear inclusion 'b' (NIb); and the CP (Revers and Garcia 2015; Worrall et al. 2015). BCMV and BCMNV along with 13 other 13 species the BCMV subgroup belonging to the genus *Potyvirus* within the *Potyviridae* family. Based on differential symptoms induced on common bean cultivars, there are currently not less than 26 viruses that can infect common bean. Some of them include: adzuki bean mosaic virus (AzMV), bean common mosaic virus serotype B (BCMV), blackeye cowpea mosaic virus (BlCMV), dendrobium mosaic virus (DeMV), guar green sterile virus (GGSV), peanut chlorotic ring mottle virus (PCRMV), peanut mild mottle virus (PMMV) and peanut stripe virus (PStV) [Jordan and Hammond 2008; Worrall et al. 2015].

BCMV transmission is through aphid vectors that transmit the virus in a non-persistent manner. In bean cultivars that lack dominant *I* resistance gene, BCMV is also transmitted

through seeds and pollen (Morales 2003). The primary spread between production areas and seasons is through seed-borne inoculum while aphid vectors contribute to secondary spread of the disease.

## Management

- **Cultural practices** - Control of vectors provides other best forms of virus control. For instance, use of virus-free seeds may significantly reduce BCMV incidence. This calls for an establishment of clean seed system and strong/effective regulatory measures involving movement of seed.
- **Chemicals and Biologicals** Control of vectors provides other best forms of virus control.
- **Genetic resistance** - The most effective control measure against BCMV is the cultivation of common bean varieties with dominant *I* resistance gene. It must be considered, however, that a few BCMV strains will cause black root disease and that almost all cultivars with this dominant gene are susceptible to BCMNV. As such, this may only work in regions/areas where the latter is not present.

## 4.2 Bean common mosaic necrosis virus (BCMNV)

**Geographic occurrence and impact** Comparatively, BCMNV distribution is not as extensive as that of BCMV and seems to be more prevalent in parts of central and eastern Africa with reported occurrence on a wide variety of wild and forage legumes as well as beans grown for human consumption (Jordan and Hammond, 2008). Due to its prevalence and high strain diversity in central and eastern Africa, BCMNV is presumed to have originated from Africa (Spence & Walkey, 1995). Many bean varieties grown by farmers in eastern and southern Africa are, reportedly, susceptible to BCMNV (Jordan and Hammond, 2008) posing a serious threat to sustainable common bean production. Meanwhile, BCMNV has spread to other parts of the world such as the United States, the Dominican Republic and Haiti through germplasm introduction (Beaver et al. 2003, Hema et al. 2014). In India, infections of up to 100% have been reported in common beans and other legumes such as cowpea and mungbean (Jordan and Hammond, 2008). Associated with black root, BCMNV can cause yield loss of up to 100 % (Mukeshimana et al. 2003).

**Symptoms and Signs** Early symptoms of BCMNV include dwarfing, mosaic, leaf curling, and chlorosis (Flores-Estévez et al. 2003), very similar to BCMV. Unlike BCMV strains, however, all BCMNV strains trigger black root disease in susceptible bean cultivars (including those containing the dominant *I* resistance gene) regardless of temperature regime (Silbernagel et al. 2001). Leaves of an infected plant begin to display small, red-brown spots that enlarge with time into a dead area, leading to the necrosis and death of the whole plant (Mukeshimana et al. 2003) (**Fig. 16**). This distinct feature of all BCMNV strains is, nevertheless, shared with NL-2 and NL-6 strains of BCMV especially where the latter two infect bean cultivars carrying dominant *I* resistance gene rendering it difficult to distinguish the two based on symptom expression.



**Fig. 16.** Bean common mosaic necrosis virus (Courtesy Phil Miklas and Richard Larsen, USDA-ARS, Prosser, WA, all rights reserved® 2024)

**Biology and epidemiology** - BCMNV belongs to the genus *Potyvirus* in the *Potyviridae* family. A former BCMV serotype A, BCMNV has similar genome and structure to that of BCMV. It is a non-enveloped monopartite single-stranded positive-sense RNA virus that forms rod-shaped virions of 9600 nt (Hull, 2014, Jordan and Hammond, 2008). Similar to BCMV, the ORF of BCMNV is translated into a single polyprotein that self-cleaves into 10 mature proteins. Except for the HC-Pro/P3, however, most of the polyprotein cleavage sites differ between BCMNV and BCMV (Jordan and Hammond, 2008). Only five strains of BCMNV have so far been identified and include TN-1, NL-3, NL-3K, NL-5, and NL-8 (McKern et al. 1994; McKern et al. 1992; Mink & Silbernagel, 1992).

BCMNV transmission is aphid-mediated in a non-persistent manner (Worrall et al., 2015). While seed transmission is also possible, the death of plants because of the hypersensitive reaction breaks disease persistence by preventing either the plants or their seeds from serving as a source of inoculum for the next crop (Mukeshimana et al. 2003).

## Management

- **Cultural practices** - When bean cultivars resistant to BCMNV are not available, use of virus-free common bean seed can significantly minimize the risk of outbreaks. Control of aphid vectors through early planting, use of optimum plant densities and intercropping with maize may also help minimize crop losses.
- **Genetic resistance** - While use of resistant varieties remains the most sustainable means of controlling common bean viral diseases. BCMNV markers were identified

and are currently being used to introgress the dominant and *bc-3* recessive resistance genes into farmer preferred varieties in Africa (Singh et al. 2010, Mukeshimana et al. 2005). Meanwhile, based on study findings by Vallejos et al. (2006) and Worrall et al. (2015) suggested that once isolated, properties of the dominant *I* gene, present in some bean cultivars, may be altered to expand its resistance to include BCMNV.

## 5.0 Nematode Diseases

### 5.1 Root-knot nematode disease [*Meloidogyne* spp., *Pratylenchus* spp.]

**Geographic occurrence and impact** -Root-knot nematodes have a worldwide distribution due to their ability to feed on over 3000 host plants but they are known to cause major problems in Southeast Asia, South America, Africa and the USA. In the western USA alone, at least seven nematode species have been reported to attack about 874 crop species. Although problematic in common beans, root-knot nematodes feed on some legumes without causing galling. Infestations at high densities can cause significant yield losses varying 50% to 90%. They also predispose bean plants to soil-borne fungi that induce root rot and wilt diseases (Cuervo et al. 2021; Frison et al. 1990).

**Symptoms and Signs** Severely infected plants show leaf yellowing, reduction of root mass, a distortion of root structure due to enlargement of some infected areas, wilting and loss of vigour due to damaged roots (**Fig. 17**). Wilting is more pronounced during the hottest part of the day, even with adequate soil moisture. Plants attacked by nematodes often produce fewer and smaller leaves and fruits. Heavy infestation of plants early in the season may result in death of plants. Distinctive swellings (galls) are seen on the roots of affected plants (Cuervo et al. 2021). Although galls are diagnostic for root knot nematode, some bean types such as the blackeye CB-3 do not gall much. Severely galled roots can be short and thick. Nematode-induced galls may split open providing an opportunity for other plant pathogens such as soil-borne, disease-causing fungi to invade the root and thus further weakening the plant. This may potentially bring about a mixture of symptoms expressed by the host plant. Galls caused by root knot nematodes may easily be confused with those of nitrogen-fixing *Rhizobium* bacteria. Unlike *Rhizobium* nodules, root knot nematode galls cannot be rubbed off easily from the roots. Furthermore, *Rhizobium* nodules are often pink inside.



**Fig. 17** Root knot nematodes on common bean (Courtesy A Alkhateb, Faculty of Agric, Egypt, all rights reserved® 2024)

**Biology and epidemiology** Root-knot nematodes belong to the Kingdom: Animalia, Phylum: Nematoda, Class: Chromadorea, Order: Rhabditida, Family: Meloidogynidae, and Genus: Meloidogyne. Beans are attacked by at least half a dozen root knot nematodes in the genus *Meloidogyne* and *Pratylenchus*. The life cycle of a plant-parasitic nematode has six stages: egg, four juvenile stages and adult. Length of survival in soil varies considerably depending on nematode species; soil characteristics (texture, moisture, aeration, etc); temperature of the habitat; host plant and stage of development. Nematodes feed on living plant tissues with the help of an oral stylet that is used to puncture host cells. Most plant nematodes inject enzymes in the host plant tissue as they feed to partially digest the cell contents before being sucked into the gut. They are capable of “tricking” the plant cells to enlarge and grow into galls, thereby producing one or more nutrient-rich feeding cells. Depending on nematode species, feeding cells may vary from one to a half dozen. The feeding process damages the plant's root system and reduces the plant's ability to absorb water and nutrients thereby inducing the associated disease symptoms. Nematodes spread among fields and between growing regions mainly through irrigation water, crop debris and soil contaminated with eggs or larvae which adhere to farm implements, animals, or humans. Subject to species and environmental conditions, a complete life cycle of root knot nematode takes 17-57 days (Cuervo et al. 2021; Frison et al. 1990).

### Management

- **Cultural Practices** - Root knot nematodes are difficult to control as they reside in soil. Crops such as *Tagetes minuta* (marigolds), *Crotalaria spectabilis* or *Indigofera hirsute* are known to possess an antagonistic effect on root knot nematodes. Growth of

these crop species can significantly reduce nematode populations or even cause eradication of the pathogen in infested soils/fields. Nevertheless, nematode populations may increase again once susceptible crops species are introduced back. A two to three-year crop rotation with non-hosts such as maize may interrupt nematode build up in the soil. Long fallow periods subject the nematodes to starving thereby reducing their population. However, for fallowing to be effective, the field must be tilled regularly to suppress weed growth which may act as secondary hosts. Regular cultivation or deep ploughing of the field also helps to expose the soil to the effects of heating and drying. Flooding for one or two weeks may sometimes be used to help reduce nematode population. This method is practical where water level and flow can be controlled as it may also contribute to spread of other soil-borne pests/pathogens through contaminated water.

- **Chemicals and Biologicals** - While chemical control of plant parasitic nematodes with nematicides is very effective and used widely on annual agronomic crops, it is considered expensive. Application of nematicides requires good timing, care, and special equipment such as protective wear and applicators (Cuervo et al. 2021; Frison et al. 1990).
- **Genetic Resistance** - The most practical, inexpensive, and recommended form of nematode control is the use of nematode-resistant varieties.

## Conclusions

Due to its broad adaptation, diverse utilization, and ability to address some of the key human nutritional requirements, common bean will likely remain one of the most important grain legume and vegetable crops in the world. Its production, however, will continue to be impacted upon by well-known and emerging pest and diseases especially associated with climate change. While this chapter may not have covered all the common bean diseases occurring virtually in all production areas worldwide, it nevertheless, captures major biotic constraints of economic importance that are currently affecting common bean production globally. In general, most common bean diseases are widespread in bean producing areas of the world. Their epidemiology is influenced by several factors ranging from the nature/biology of the disease/pest itself, existing crop production systems and movement of seed and/or propagules within and across bean production areas. Although management options for each disease have been provided in this chapter, they are not universal across the regions. Generally, effective, and sustainable disease management strategies will require an integrated approach that combines effective and environmentally friendly options. Because pests and diseases of common bean are dynamic in space and time, there is proliferation of research work in several scientific disciplines including genomics and bioinformatics that seek to address these constraints. Findings from such global research initiatives are expected to shape the current understanding of common bean diseases and their management strategies.

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