



Biotic constraints to legume production under a changing climate: An integrated review of diseases and sustainable management strategies

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Abstract

Legumes are central to global food and nutritional security, sustainable cropping systems, and the livelihoods of smallholder farmers, yet diseases caused by fungi, bacteria, viruses, and nematodes persistently constrain their productivity. Climate change is now reshaping these pressures by accelerating pathogen life cycles, expanding pathogen ranges and weakening host defences. This review provides a consolidated account of (i) the economic and nutritional importance of legumes and the scale of disease-related losses (ii) the influence of a changing climate on pest and pathogen dynamics (iii) the biology, epidemiology and impact of the major fungal diseases (*Botrytis*, *Ascochyta*, *Fusarium*, *Sclerotium rolfsii*, rusts, root rots and *Sclerotinia sclerotiorum*), the re-emerging bacterial wilt of dry beans and the major viral diseases (iv) pathogen virulence factors and plant defence signaling and (v) the full spectrum of management options, from cultural practices, host resistance and genomics-assisted breeding to biological control, botanicals, essential oils, organic amendments, fungicides, nanotechnology and digital agriculture. It indicates that no single tactic is sufficient, and durable disease control depends on integrating clean seed, rotation and sanitation, resistant cultivars, biocontrol agents, botanicals and judicious fungicide use, supported by stronger seed systems, surveillance and inclusive policy.

Keywords: Bacterial wilt, biocontrol, climate change, *Fusarium* wilt, host resistance, legumes, plant disease management; viral diseases

Introduction

Legumes (family Fabaceae) rank among the most important plant families for human nutrition and agricultural sustainability. They are a primary source of plant-based protein, dietary fibre and micronutrients such as iron and zinc and they fix atmospheric nitrogen through symbiosis with rhizobia, improving soil fertility and reducing dependence on synthetic fertilizers (Meghwal., 2026; Sbai., 2024) [18, 21]. Pulses are cultivated on roughly 12-15% of the world's arable land and supply about 30% of the protein-derived nitrogen requirement of the human diet (Graham & Vance, 2003) [10]. The Food and Agriculture Organization recognizes 11 pulse crops, grown on approximately 77.5 million hectares with an annual production of about 70.41 million tonnes and an average yield of only 907 kg/ha (Sbai., 2024) [21]. Pulses account for around 33% of the world's protein requirements and support more than 700 million smallholders in developing nations (Sbai., 2024) [21]. Despite this importance, productivity remains low and unstable. Biotic stresses, particularly diseases, are among the most important constraints. In India, insects, diseases and other factors have been estimated to account for losses of 29%, 22% and 12%, respectively, while global estimates attribute 30%, 20% and 5% losses to insects, diseases and other pests (Yaduraju, 2006) [34].

Three developments make a fresh, integrated appraisal timely. First, climate change is altering the geography and severity of legume diseases, in some cases contributing to the re-emergence of pathogens once thought to be under control (Harveson., 2015; Meghwal., 2026) [11, 18]. Second, a

large and growing body of work describes non-chemical alternatives to synthetic fungicides, including biocontrol agents, botanicals and essential oils (Kaur., 2023; Sbai., 2024) [15, 21]. Third, advances in genomics, genome editing and digital tools are opening new routes to durable resistance (Jha., 2023; Meghwal., 2026) [13, 18]. This review synthesises these strands into a single framework, organised around disease burden, causal agents, host-pathogen interactions and management.

Disease Burden and Economic Importance of Legume Diseases

Legumes are attacked by a wide range of pathogens, including powdery and downy mildews, root rots, anthracnose, rusts, wilts, bacterial blights and mosaic diseases, in addition to pod borers, nematodes, parasitic weeds and sap-sucking insects (Kaur., 2023) [15]. Out of these, the fungal diseases predominate. Viral diseases alone have been estimated to account for about half of plant disease burdens globally, with economic losses of roughly US\$30 billion annually (Hilaire., 2022) [12].

Losses can be severe at the level of individual diseases and for smallholders, it can amount to a multi-season economic shock. Catastrophic yield losses of 50-100% from outbreaks such as *Mungbean yellow mosaic virus* have been described as long-term financial devastation that pushes households into cycles of debt, while yield losses of 20-40% due to pod borers or wilt in Sub-Saharan Africa and South Asia translate directly into reduced income and compromised protein security (Meghwal., 2026) [18].

Table 1: Selected major legume diseases, causal agents and reported yield losses

Crop	Disease	Causal agent	Reported losses	Source
Faba bean	Chocolate spot	<i>Botrytis fabae</i>	Up to 61% (susceptible genotypes); >60–80% under favourable conditions; 62% in Tigray, 2000 ^[35]	Sbai <i>et al.</i> (2024); Tadesse <i>et al.</i> (2008)
Faba bean	Rust	<i>Uromyces viciae-fabae</i>	2–15% (lower altitudes); 14–21% (intermediate altitudes)	Tadesse <i>et al.</i> (2008)
Chickpea	Ascochyta blight	<i>Ascochyta rabiei</i>	Up to 97% in epidemic years (Morocco)	Sbai <i>et al.</i> (2024)
Chickpea	Fusarium wilt / wilt-root rot complex	<i>Fusarium oxysporum</i> f. sp. <i>ciceris</i> and others	Up to 50% (Ethiopia); up to 60% nationally (India); complete loss possible	Tadesse <i>et al.</i> (2008); Kaur <i>et al.</i> (2023)
Lentil	Rust	<i>Uromyces viciae-fabae</i>	8.39% seed-yield loss per 1% increase in severity	Tadesse <i>et al.</i> (2008)
Lentil	Fusarium wilt	<i>F. oxysporum</i> f. sp. <i>lentis</i>	Loss approximates % plant mortality (e.g., 50% mortality ≈ 50% loss)	Kaur <i>et al.</i> (2023)
Various legumes	Collar rot	<i>Sclerotium rolfsii</i>	Up to 80% in heavily infested fields	Sbai <i>et al.</i> (2024)
Soybean	Sclerotinia stem rot	<i>Sclerotinia sclerotiorum</i>	>60% yield loss; >US\$200 million annually (USA)	Antwi-Boasiako <i>et al.</i> (2022)
Common bean	White mould	<i>S. sclerotiorum</i>	30–100%	Antwi-Boasiako <i>et al.</i> (2022)
Common bean	Anthraxnose	<i>Colletotrichum lindemuthianum</i>	Up to 100% with contaminated seed	Kaur <i>et al.</i> (2023)
French bean	Angular leaf spot	<i>Phaeoisariopsis griseola</i>	40–70% and can increase up to 80%	Kaur <i>et al.</i> (2023)
Dry bean	Bacterial wilt	<i>Curtobacterium flaccumfaciens</i> pv. <i>flaccumfaciens</i>	60–65% yield reduction in an affected drip-irrigated field	Harveson <i>et al.</i> (2015)
Soybean	Soybean mosaic virus (with BPMV)	Potyvirus + Comovirus	Up to 85%	Jha <i>et al.</i> (2023)
Common bean	BCMV/BCMNV	Potyviruses	6–98%	Jha <i>et al.</i> (2023)
Mungbean	Yellow mosaic disease	MYMV / MYMIV	Up to 100%	Jha <i>et al.</i> (2023)
Pigeonpea	Sterility mosaic	PPSMV	>US\$300 million annually in India	Jha <i>et al.</i> (2023)

Climate Change as a Driver of Altered Disease Dynamics

Climate change is now recognised as a contemporary catalyst that restructures the interaction between legume hosts and their biotic stressors (Meghwal., 2026)^[18]. Environmental changes such as rising temperature, elevated atmospheric CO₂ and erratic rainfall are projected to cut yields of critical legumes such as soybean, lentil and common bean by 10–49% by mid-century, before accounting for altered biotic pressure (Bishop., 2015; Daryanto., 2015)^[4, 7].

Temperature effects on pathogens and vectors

Rising temperatures shorten the incubation periods of fungal pathogens such as rusts (*Uromyces* spp.) and *Ascochyta* spp., allowing more polycyclic generations within a season and outpacing conventional chemical control (Meghwal., 2026)^[18]. Insect pests and viral vectors are poikilothermic, so, warmer conditions shorten development time, raise metabolic rates and allow additional generations per season. Warmer winters permit pests and vectors such as *Aphis craccivora* to overwinter at higher latitudes and altitudes, expanding their range (Meghwal., 2026)^[18]. Field observations on other aphid species point the same way: cabbage aphid (*Brevicoryne brassicae*) populations on kale correlated positively with both maximum and minimum temperature (Bhat., 2024)^[3]. Because many legume viruses depend on aphid, whitefly or thrips transmission (Jha., 2023; Kaur., 2023)^[13, 15], vector proliferation has direct implications for virus epidemiology.

Elevated CO₂ and host quality

Under elevated CO₂, legumes accumulate more carbon but exhibit a relative decline in nitrogen and protein. This “nutritional dilution” can trigger compensatory feeding by

herbivores, increasing defoliation and creating wounds that serve as entry points for necrotrophic pathogens. Also, denser canopies may also create humid microclimates favouring fungal growth (Meghwal, 2026)^[18]. The stimulatory effect of CO₂ on yield typically plateaus near 550 ppm and is often negated by compounding heat and drought stress (Dave., 2024)^[8].

Rainfall, humidity and drought

Altered precipitation creates divergent risks. Prolonged humidity and flooding favour foliar and soil-borne pathogens such as *Botrytis*, *Ascochyta*, *Phytophthora* and *Fusarium*, whereas water deficit weakens plant immunity and predisposes legumes to opportunists such as *Macrophomina phaseolina* (charcoal rot). Drought and root-infecting pathogens together cause greater damage than either stress alone and drought-induced stomatal closure can alter defence readiness (Meghwal., 2026)^[18]. Extreme rainfall variability also impairs rhizobial symbiosis, reducing nodulation and plant vigour.

Loss of phenological synchrony

Warmer winters and earlier springs may cause legumes to flower before pollinators or the natural enemies of pests emerge, creating a window in which pest populations increase unchecked and farmers turn to chemical control (Meghwal., 2026)^[18].

Major Fungal Diseases of Legumes

1. Botrytis diseases: grey mould and chocolate spot

Botrytis spp. are among the most widespread plant pathogens, which able to infect over 1,400 plant species and to cause disease in the field, in storage, in transit and at market (Sbai., 2024)^[21]. *Botrytis cinerea*, the causal agent of grey mould, is transmitted by seed and stubble. It favours

moderate temperatures (15-25 °C) and prolonged surface wetness and can survive as conidia, mycelium or sclerotia in crop residues. It is difficult to control because of its high reproductive rate, frequent mutation and strong adaptability (Sbai., 2024) ^[21]. Infection is aided by mechanical damage, hail and dense planting. Virulence involves effector proteins, toxins, reactive oxygen species (ROS)-generating enzymes, oxalic acid, cell wall-degrading enzymes and even small RNAs that suppress host immunity (Sbai., 2024) ^[21]. Chocolate spot, caused chiefly by *Botrytis fabae*, is the most destructive disease of faba bean (Sbai., 2024) ^[21]. In Ethiopia, yield losses reached up to 61% in susceptible genotypes and 34% in tolerant ones, and complete crop failure occurs where favourable weather persists (Tadesse., 2008) ^[26]. Epidemics are favoured by humid ($\geq 70\%$ relative humidity), warm (10-23 °C) and rainy conditions, with apparent infection rates of 0.142-0.164 units per day, which means several disease cycles can occur within one season. Also, sclerotia can persist in debris on the soil surface for about two years but die within four months when buried at 20 cm (Tadesse., 2008) ^[26].

2. Ascochyta blight

Ascochyta spp. are seed-borne necrotrophs affecting cool-season legumes, including *A. rabiei* (chickpea), *A. lentis* (lentil) and *A. fabae* (faba bean). They overwinter in crop residue and soil and spread by rain-splashed conidia and airborne ascospores (Sbai., 2024) ^[21]. Ascochyta blight of chickpea thrives at 10-25 °C with wet periods of 12 hours or more and has caused losses of up to 97% in epidemic years in Morocco (Sbai., 2024) ^[21]. The pathogen produces non-selective phytotoxins including solanapyrones, cytochalasin D and proteinaceous toxins that act as virulence factors (Sbai., 2024) ^[21].

3. Fusarium wilts and root-rot complexes

Fusarium oxysporum and *F. solani* cause wilt and root rot of chickpea, lentil and other pulses, colonising the xylem and blocking water and nutrient transport. Under favourable conditions they may cause complete harvest failure (Sbai., 2024) ^[21]. Fusarium wilt of chickpea is reported to cause national-level losses of up to 60% in India, with complete grain loss possible when infection occurs at vegetative and reproductive stages and greater incidence at flowering and podding when temperature rises suddenly or water stress occurs (Chaudhry., 2007) ^[6]. Wilt of pigeonpea has been associated with losses of about 97,000 tonnes per year in India (Joshi., 2001) ^[14]. In lentil, wilt caused by *F. oxysporum* f. sp. *lentis* is the most widespread disease in India, with yield loss broadly proportional to plant mortality (Kaur., 2023) ^[15].

In Ethiopia, chickpea wilt/root-rot pathogens (*F. oxysporum* f. sp. *ciceris*, *Rhizoctonia bataticola*, *F. solani*, *R. solani* and *Sclerotium rolfsii*) often occur together and cause losses of up to 50% (Tadesse., 2008) ^[26]. Their development is strongly linked to soil temperature and moisture, inoculum density and crop age, which includes optimum temperatures of about 25 °C for *F. oxysporum*, *R. solani* and *F. solani* and 30 °C for *R. bataticola* and *S. rolfsii*. *R. bataticola* is favoured by low soil moisture, whereas *S. rolfsii* and *R. solani* prevail in wet soils and about 33 propagules of *F. oxysporum* f. sp. *ciceris* per gram of soil is a damaging level for susceptible cultivar JG-62 (Tadesse., 2008) ^[26].

4. Sclerotium rolfsii (collar rot)

Sclerotium rolfsii is a soil-borne necrotroph with a host range of about 500 plant species, including lentil, chickpea, soybean and common bean, and can cause up to 80% loss in heavily infested fields (Sbai., 2024) ^[21]. It thrives in warm, humid conditions (25-30 °C) and produces oxalic acid, cell wall-degrading enzymes and sclerotia. Undecomposed cereal residues such as wheat stubble favour rapid multiplication of the pathogen and destroying this inoculum pool at seedbed preparation effectively reduces collar rot (Tadesse., 2008) ^[26].

5. Rusts

Rust caused by *Uromyces viciae-fabae* affects faba bean, pea and lentil and is favoured by cool, humid and cloudy weather (about 20-25 °C) (Sbai., 2024) ^[21]. In faba bean it is endemic below about 2,300 m above sea level and it spreads mainly over short distances by wind and causes losses of 2-15% at lower and 14-21% at intermediate altitudes (Tadesse., 2008) ^[26]. In lentil, each 1% increase in rust severity reduced seed yield by 8.39%, and severity of 4.7% or more at early flowering substantially reduced yield (Tadesse., 2008) ^[26]. Rust is also a serious threat to field pea in North India (Mishra., 2009) ^[19].

6. Other root and foliar diseases

Rhizoctonia solani causes root rot and web blight of French bean, with recorded green pod losses of 8.4-64.6% in India (Sharma & Sohi, 1980, 1981) ^[22]. *Macrophomina phaseolina* causes dry root rot of mungbean and charcoal rot of cowpea (Kaur., 2023) ^[15] and is particularly associated with drought-stressed tissues (Meghwal., 2026) ^[18]. Mungbean also suffers from powdery mildew (9-50% losses) and *Cercospora* leaf spot (50-70%) (Kaur., 2023) ^[15]. Pea is affected by *Pythium* seed and seedling rot, Fusarium root rot and rust and cowpea by anthracnose, which is seed-borne and favoured by warm, humid conditions (Kaur., 2023) ^[15]. Molecular sequencing aids identification of diseases (Rashid., 2025) ^[20].

7. Sclerotinia sclerotiorum (white mould and stem rot)

Sclerotinia sclerotiorum infects over 600 plant species and is the most economically damaging of the three *Sclerotinia* species (*S. sclerotiorum*, *S. minor* and *S. trifoliorum*) affecting legumes, causing white mould, crown rot and stem rot in soybean, common bean, chickpea, faba bean, groundnut, lupin, alfalfa and red clover (Antwi-Boasiako., 2022) ^[1].

Sclerotia and survival: The pathogen persists as melanised sclerotia that develop through initiation, development and maturation phases. Sclerotial survival is influenced by flooding, drought, burial depth and soil moisture and flooding can cause decay within 14-45 days, sclerotia at 5 cm survive less well than those at 10-30 cm and overall survival has been reported from 1 to 5 years and even 5 to 11 years, making the pathogen very difficult to eradicate (Antwi-Boasiako., 2022) ^[1].

Infection process: Under adequate moisture and light, sclerotia germinate to produce apothecia, releasing ascospores that infect flowers and senescing tissue, aided by canopy microclimate. Infection then spreads to leaves, petioles and stems, producing water-soaked lesions,

bleached stems with cottony mycelium and eventual sclerotia and causing wilting, premature ripening and lodging. Effects are greatest at flowering, pod development and grain filling (Antwi-Boasiako., 2022) ^[1]. Sclerotia can also infect roots, stems and leaves directly through myceliogenic germination.

Pathogenicity: Oxalic acid is a broad-spectrum pathogenicity factor and oxalate-deficient strains show sharply reduced virulence on soybean, common bean, tomato, canola, sunflower and *Arabidopsis*. Oxalic acid also ruptures chloroplast membranes, reducing chlorophyll and photosynthesis (Antwi-Boasiako., 2022; Sbai., 2024) ^[1, 21].

Losses: Sclerotinia diseases reduce yield components (seeds per pod, pods per plant, 100-seed weight) and seed quality, with a linear relationship between infection level and yield loss (Antwi-Boasiako., 2022) ^[1]. Soybean losses exceed US\$200 million a year in the United States and losses of more than 50% have been recorded across several legumes (Antwi-Boasiako., 2022) ^[1]. Interestingly, conservation rotation practices promoted *S. sclerotiorum* in common bean relative to conventional rotation, a reminder that soil-conserving practices can shift disease risk (Larney., 2015) ^[16].

Bacterial Diseases of Legumes

Bacterial diseases of legumes can be severe and are frequently seed-borne. Kaur. (2023) describe bacterial leaf spot and halo blight of mungbean caused by *Xanthomonas campestris* pv. *vignaeradiatae*, characterised by brown, circular to irregular, raised dry lesions on leaves and stems. Common bacterial blight (*Xanthomonas axonopodis* pv.

phaseoli), halo blight (*Pseudomonas syringae* pv. *phaseoli*) and bacterial brown spot are important in dry beans (Harveson., 2015) ^[11].

1. Bacterial wilt of dry beans

Bacterial wilt is caused by the pathogen, *Curtobacterium flaccumfaciens* pv. *flaccumfaciens*.

Symptoms and diagnosis: Wilting during warm, dry or water-stressed periods, plant recovery at night, seedling death and interveinal necrotic lesions with bright yellow borders (“firing”) are diagnostic. Unlike common bacterial blight, wilt often kills severely infected plants (Harveson., 2015) ^[11]. Colonies are gram-positive, slow-growing and yellow, orange, pink or purple pigmented and the pathogen can stain seed yellow, orange, purple or pink (Harveson., 2015) ^[11].

Epidemiology: The seed is the major source of inoculum and dissemination. The bacterium is favoured by temperatures above 30 °C (optimum in culture 27-28 °C), high leaf moisture, reduced tillage and wounding from hail, wind-driven rain or equipment, because it cannot penetrate intact plant surfaces (Harveson., 2015) ^[11]. It survives poorly in soil but persists on crop residue and on seed and has been reported viable for up to 24 years under laboratory conditions (Burkholder, 1945) ^[5]. Strikingly, the pathogen has also been recovered from soybean, corn, wheat, sunflower and alfalfa. Across a survey of 548 fields or sites, 26 isolates from five rotation crops were pathogenic to dry bean, suggesting that alternate hosts and residues may sustain inoculum between bean crops (Harveson., 2015) ^[11].

Viral Diseases of Legumes

Viral diseases are the major biotic stresses in grain legumes.

Table 2: Major legume viruses, vectors and reported impacts.

Crop	Virus (genus)	Vector / transmission	Reported impact	Source
Soybean	<i>Soybean mosaic virus</i> (Potyvirus)	Aphids; seed-borne	Severe yield and quality losses; up to 85% with BPMV	Jha et al. (2023)
Soybean	<i>Tobacco ringspot virus</i>	–	25–100% yield loss	Jha et al. (2023)
Common bean	BCMV / BCMNV (Potyvirus)	Aphids; seed-borne	6–98%	Jha et al. (2023)
Common bean	BGYMV (Begomovirus)	Whitefly	Up to 100% in epidemics	Jha et al. (2023)
Mungbean / urdbean	MYMV / MYMIV (Begomovirus)	Whitefly (<i>Bemisia tabaci</i>)	Up to 100%	Jha et al. (2023)
Mungbean	Urdbean leaf crinkle virus	Aphids, beetles, whitefly; sap; seed (up to 15%)	Major constraint in India	Kaur et al. (2023)
Pigeonpea	Pigeonpea sterility mosaic virus (Emaravirus)	Eriophyid mite	>US\$300 million annually in India	Jha et al. (2023); Kaur et al. (2023)
Chickpea	<i>Cucumber mosaic virus</i>	Aphids	45% at 75% incidence	Jha et al. (2023)
Chickpea	Beet western yellows virus (chickpea stunt)	Aphids	Most prevalent virus in Ethiopia; losses not quantified	Tadesse et al. (2008)
Groundnut	Peanut bud necrosis virus (Tospovirus)	<i>Thrips palmi</i>	30–90%	Jha et al. (2023)

Viral disease management is especially challenging because chemical therapies do not exist and because control of vectors is often insufficient. For example, control of pigeonpea sterility mosaic depends largely on resistant cultivars (Kaur, 2023) ^[15].

Virulence Factors and Mycotoxins of Legume Pathogens

Necrotrophic fungal pathogens secrete a wide array of secondary metabolites and enzymes that determine symptom expression. Sbai. (2024) catalogue phytotoxins by pathogen: anthraquinones (lentiquinones, lentisone,

pachybasin) and other compounds from *Ascochyta lentis*; solanapyrones and cytochalasin D from *A. rabiei*; naphthalenones such as botrytone and scytalone from *Botrytis fabae*; botrydial and related lactones from *B. cinerea*; oxalic acid from *Sclerotinia sclerotiorum* and fusaric acid, moniliformin, fusarins, enniatins and beauvericin from *Fusarium* species. These compounds target host cellular machinery, interfere with immunity and cause necrosis, chlorosis, wilting and ultimately, results in plant death. They are classified as host-selective or non-host-selective toxins and non-selective toxins occur in

Fusarium and *Ascochyta* (Sbai., 2024) ^[21]. Some are also harmful to humans and animals, linking crop disease to food safety (Sbai., 2024) ^[21]. Pest-induced stress can further predispose crops to *Aspergillus* infection and aflatoxin contamination, undermining trade and public health (Waliyar., 2015; Williams., 2004) ^[27, 33].

Plant Defence Responses and Molecular Basis of Resistance

1. Immune signalling

Plants perceive pathogens through pattern-triggered immunity (PTI) and effector-triggered immunity (ETI) (Jha., 2023) ^[13]. Both biotic and abiotic stresses are sensed by receptors that initiate signal transduction via ROS and calcium ions, activating mitogen-activated protein kinase (MAPK) cascades and phytohormone pathways that modulate transcription factors and defence genes, callose deposition and secondary metabolite production (Meghwal., 2026) ^[18].

2. Hormonal crosstalk

Salicylic acid (SA) is predominantly induced by biotrophs and mediates systemic acquired resistance, whereas jasmonic acid (JA) and ethylene (ET) are induced by necrotrophs and herbivorous insects and often act synergistically. SA and JA/ET pathways can be antagonistic (Meghwal., 2026) ^[18]. Because plants in the field face combined stresses, interactions between abiotic and biotic responses are frequently synergistic in damage rather than additive (Meghwal., 2026) ^[18]. Melatonin, an eco-friendly tryptophan-derived compound, enhances resistance to *Botrytis cinerea* in tomato by upregulating defence genes and activating jasmonic acid (JA) signaling (Wani *et al.*, 2024) ^[3].

3. Defence against Sclerotinia

In legumes infected with *S. sclerotiorum*, defence-related genes are up-regulated and metabolic genes down-regulated. Phenolics, phytoalexins and signalling compounds accumulate and ROS-scavenging enzymes (peroxidase, catalase, superoxide dismutase, ascorbate peroxidase and others) regulate cell wall formation and pathogen establishment in common bean and soybean (Antwi-Boasiako., 2022) ^[1].

4. Genetic architecture of resistance

Resistance to viruses has been characterised through Mendelian genetics, biparental QTL mapping, genome-wide association studies (GWAS), whole-genome resequencing and pangenomes (Jha., 2023) ^[13]. Examples include the *Rsv* loci for soybean mosaic virus and the recessive *bc* genes (*bc-1*, *bc-3*, *bc-u*) for BCMV/BCMNV in common bean (Jha., 2023) ^[13]. For *Sclerotinia*, partial resistance has been mapped in common bean, soybean, groundnut and chickpea. Candidate genes such as *GmGST* (glutathione transferase) and *GmCH1* (chitinase) enhance soybean resistance and common bean regions WM2.2a and WM2.2b are associated with physiological resistance and avoidance, respectively (Antwi-Boasiako., 2022) ^[1].

Management Strategies

1. Cultural, sanitary and agronomic practices

Cultural practices remain the foundation of disease management and are usually the most economical.

Clean seed and sanitation: Many legume pathogens are seed-borne (bacterial wilt, *Ascochyta*, anthracnose, several viruses), certified disease-free seed together with sanitation and rotation virtually eliminated bacterial wilt in the United States by the early 1980s, and lapses in these practices are considered a likely contributor to its return (Harveson., 2015) ^[11].

Residue and tillage management: Destroying undecomposed cereal residue at seedbed preparation reduces collar rot (Tadesse., 2008) ^[26] and deep ploughing after harvest reduces chocolate spot risk (Tadesse., 2008) ^[26]. However, reduced tillage may favour pathogen survival (Harveson., 2015) ^[11] and burial depth influences sclerotial survival (Antwi-Boasiako., 2022) ^[1].

Sowing date and cropping system: Adjusting sowing date can desynchronise vulnerable stages from peak pest pressure (Meghwal., 2026) ^[18]. Late sowing shortens chocolate spot epidemics but reduces yield (Tadesse., 2008) ^[26]. Intercropping with non-hosts (e.g., mustard) and push-pull systems disrupt pest and pathogen cycles (Meghwal., 2026) ^[18]. Faba bean-field pea mixtures reduced chocolate spot and rust severity in Ethiopian trials, although the benefit is contested and may be impractical at scale (Tadesse., 2008) ^[26].

Rotation and soil health: Diverse rotations can build “rhizosphere legacy” effects, in which root exudates shape a suppressive microbiome; such effects have been reported to reduce *Fusarium* wilt incidence by up to 40% in subsequent seasons and conservation agriculture supports microbial diversity that outcompetes *Fusarium* and *Macrophomina* (Meghwal., 2026) ^[18].

2. Host resistance and conventional breeding

Resistant cultivars are the most sustainable option but are limited by narrow genetic bases. In *Sclerotinia*, only 285 of 8,596 soybean lines and 12 of 519 common bean accessions were resistant, no commercial resistant cultivars exist for soybean, alfalfa, red clover or faba bean, and groundnut is a notable exception, where a resistant cultivar has saved an estimated US\$5 million per year in the United States (Antwi-Boasiako., 2022) ^[1]. For chickpea, most nationally distributed cultivars (e.g., Arerti, Chefe, Akaki and Worku) carry resistance mainly to *Fusarium* wilt (Tadesse., 2008) ^[26].

Wild relatives and landraces are important reservoirs of resistance and stress-tolerance genes; for example, wild chickpea (*Cicer reticulatum*) provides natural resistance to heat-tolerant pests and drought-induced pathogens through secondary metabolites and physical barriers such as trichomes (Meghwal., 2026) ^[18]. Screening germplasm for viral resistance has identified sources in wild *Vigna* species (Jha., 2023) ^[13].

3. Genomics-assisted breeding and biotechnology

Molecular tools accelerate resistance breeding.

Marker-assisted selection (MAS) tracks major resistance genes and genomic selection (GS) uses genome-wide markers to predict polygenic traits and has been reported to shorten breeding cycles by up to 30% while maintaining protein content under combined stress (Meghwal., 2026) ^[18].

GWAS, pangenomes and transcriptomics identify candidate resistance genes and structural variation (Jha., 2023)^[13].

RNA interference (RNAi), virus-induced gene silencing (VIGS) and host-induced gene silencing (HIGS) offer targeted, non-toxic protection, for example, silencing three pathogenicity genes of *S. sclerotiorum* in *Brassica napus* reduced disease by 40% (Antwi-Boasiako., 2022; Jha., 2023; Meghwal., 2026)^[1, 13, 18].

Genome editing (CRISPR/Cas9) can knock out susceptibility (S) genes and enable *de novo* domestication of resistant wild relatives, and synthetic biology (synthetic promoters and transcription factors) and rapid generation advance further extend the toolbox (Jha., 2023; Meghwal., 2026)^[13, 18].

4. Biological control

Biological control agents (BCAs) are an environmentally friendly alternative to synthetic fungicides and align with sustainable agriculture (Antwi-Boasiako., 2022; Sbai., 2024)^[1, 21].

Trichoderma spp. act through competition, antibiosis, mycoparasitism, induced resistance and cell wall-degrading enzymes and are effective against *Fusarium*, *Botrytis*, *Sclerotium*, *Rhizoctonia*, *Sclerotinia*, *Ascochyta* and rusts (Antwi-Boasiako., 2022; Kaur., 2023; Sbai., 2024)^[1, 15, 21]. Against *S. sclerotiorum*, *T. harzianum* T-22 lowered disease severity index by 38.5% in soybean and *T. asperellum* reduced apothecia and severity in common bean, although inconsistent field performance limits commercialisation (Antwi-Boasiako., 2022)^[1]. Seed treatment with *T. harzianum* at 10 g/kg seed has been effective against chickpea wilt (Wani., 2024)^[3].

Coniothyrium minitans (commercialised as Contans WG) reduced apothecia by 81% and sclerotia by 50% in soybean, decreased white mould incidence by 90% in common bean and cut diseased alfalfa pods from 42-72% to 29-38% (Antwi-Boasiako., 2022)^[1].

Bacillus spp. produce lipopeptides (fengycin, iturin, surfactin) and antibiotics, form stress-tolerant spores and induce systemic resistance; *B. subtilis* limited apothecia by 91% and sclerotia by 30% under growth-chamber conditions (Antwi-Boasiako., 2022; Sbai., 2024)^[1, 21].

Pseudomonas and *Streptomyces* spp. provide additional antagonists through diffusible antibiotics, hydrogen cyanide, siderophores and chitinases (Sbai., 2024)^[21]. *Streptomyces lydicus* decreased apothecia by 100% in a growth-chamber soybean trial (Antwi-Boasiako., 2022)^[1].

Plant growth-promoting rhizobacteria (PGPR) can prime immune responses analogously to a “plant vaccine” and thermal priming of *Trichoderma* improves its tolerance to heatwaves, an adaptation to warming climates (Meghwal., 2026)^[18]. Synthetic microbial communities (SynComs) offer functional redundancy in variable environments (Meghwal., 2026)^[18].

5. Botanicals, plant extracts and essential oils

Plant extracts: Neem, garlic, ginger, *Lantana camara*, *Eucalyptus*, marigold and many other species have shown antifungal, antibacterial or antiviral effects (Kaur., 2023)^[15]. Kaur. (2023) compile examples such as ginger, garlic and neem extracts against lentil wilt, *Artemisia vulgaris* and *Lantana camara* extracts against *Rhizoctonia solani* in French bean, neem and *Lantana* against chickpea wilt and *Boerhaavia diffusa* and neem against mungbean yellow

mosaic virus. Neem products have insecticidal, fungicidal, nematocidal and antiviral properties and a neem oil formulation (“Trilogy”) is approved by the U.S. EPA for food crops (Kaur., 2023)^[15]. Other reports indicate neem aqueous extracts inhibited *Fusarium solani* and *S. sclerotiorum* mycelial growth and that garlic extracts controlled *A. rabiei* and *S. sclerotiorum* at 10% (Sbai., 2024)^[21]. Propolis extract and oregano essential oil reduced *S. sclerotiorum* severity by 40% and 60%, respectively (Antwi-Boasiako., 2022)^[1]. Macrofungal extracts, rich in polyphenols and flavonoids, also show antioxidant activity and merit evaluation alongside botanicals (Wani *et al.*, 2025)^[20].

Essential oils (EOs): EOs are volatile, biodegradable secondary metabolites generally recognised as safe that disrupt fungal membranes, cause electrolyte leakage and inhibit enzymes and respiration (Sbai., 2024)^[21]. Activity is usually attributed to major terpenoid and phenylpropanoid components such as thymol, carvacrol, eugenol, linalool, geraniol, citral, terpinen-4-ol and trans-cinnamaldehyde (Sbai., 2024)^[21]. Sbai. (2024) list oils achieving complete *in vitro* inhibition of *B. cinerea* (for instance thyme, oregano, clove and lemongrass oils), *F. oxysporum*, *A. rabiei*, *S. sclerotiorum* and *S. rolsii*, such as *Cinnamomum cassia* oil giving 100% inhibition of *S. sclerotiorum* at 256 µg/mL by direct contact and 64 µg/mL in vapour form. Combinations often show synergy (e.g., thymol with carvacrol, and oregano with thyme), and EOs combined with synthetic fungicides or biocontrol agents such as *Bacillus amyloliquefaciens* can enhance efficacy while lowering fungicide doses (Sbai., 2024)^[21].

6. Organic amendments

Decomposition of farmyard manure, compost, oil cakes and similar materials improves soil structure and stimulates antagonistic microbial populations, which compete with, parasitise or produce antibiotics against pathogens such as *Pythium* and *Phytophthora*; consistency depends on compost stability and quality

7. Chemical control

Fungicides remain the most effective short-term tool in outbreaks (Antwi-Boasiako., 2022; Sbai., 2024)^[1, 21]. Commonly used active ingredients include azoxystrobin, boscalid, difenoconazole, fluazinam, iprodione, procymidone, thiophanate-methyl, prothioconazole, carbendazim, mancozeb and thiram, with fluazinam identified as particularly promising and carbendazim showing strong inhibition of several pathogens (Sbai., 2024)^[21]. *In vitro* poisoned-food assays against *Colletotrichum lindemuthianum*, the cause of common bean anthracnose, showed that mancozeb 75 WP was the best non-systemic fungicide (75.63% mean mycelial inhibition) and carbendazim 50 WP the best systemic one (85.13%). The combination product mancozeb (63%) + carbendazim (12%) gave the highest inhibition overall (89.05%) (Wani., 2024)^[3]. Guidance from Ethiopia includes mancozeb at 2.5 kg a.i./ha at weekly intervals starting at about 5% infection for faba bean rust, and chlorothalonil at 2.5 kg a.i./ha once or twice after infection reaches 30% for chocolate spot (Tadesse., 2008)^[26].

Timing is critical for *Sclerotinia*: application at bloom limits ascospore infection and for soybean, a second application

two weeks after an R1 or R2 application may be needed (Antwi-Boasiako., 2022) ^[1]. Limitations include cost, ecological and health risks from residues, resistance development (for example in dicarboximide and benzimidazole fungicides) and regulatory withdrawal, such as bans on thiophanate-methyl and prothioconazole in Europe (Antwi-Boasiako., 2022; Sbai., 2024) ^[1, 21]. Rotating fungicides with different modes of action is advised (Antwi-Boasiako., 2022) ^[1].

8. Integrated management: combining biological and chemical tools

Combining biocontrol agents with reduced fungicide doses often outperforms either alone because many antagonists tolerate certain fungicides or effects can be synergistic (Sbai., 2024) ^[21]. Examples include seed treatment with *B. subtilis* plus foliar carbendazim + mancozeb, which gave the lowest incidence (7.8%) of *Ascochyta* blight in chickpea (Bashir., 2022) ^[2]; *T. harzianum* with carbendazim, which reduced chickpea wilt by up to 93.75% (Mahmood., 2015) ^[17]; and tebuconazole with *T. viride*, which gave up to 100%

inhibition of *S. rolfsii* (Sbai., 2024) ^[21]. Contans combined with reduced fungicide provided integrated control of white rot in beans (Elsheshtawi., 2017) ^[9]. The authors argue that reliance on chemical fungicides disrupts ecological balance and that bio-control agents and plant extracts in seed treatment should be developed as alternatives (Wani.,2025) ^[20].

9. Nanotechnology and digital agriculture

Nano-encapsulation protects active ingredients from heat and UV and allows stimulus-responsive release, while soil nano-sensors may detect stress volatiles early (Meghwal., 2026) ^[18]. AI-based forecasting that integrates weather, satellite imagery and remote sensing can improve the timing of biopesticide applications (reported efficacy gains of up to 25%) and UAV hyperspectral imaging can pinpoint hotspots, enabling chemical use reductions of as much as 80% (Meghwal., 2026) ^[18]. Machine-learning models also show promise for estimating *Sclerotinia* risk thresholds to guide spraying (Antwi-Boasiako., 2022) ^[1].

Table 3: Summary of management options and their principal strengths and limitations.

Approach	Main targets	Strengths	Limitations	Sources
Clean seed, sanitation, rotation	Seed-borne bacteria, fungi, viruses	Low cost; proven for bacterial wilt	Requires seed-system discipline; alternate hosts may sustain inoculum	Harveson <i>et al.</i> (2015)
Host resistance	All classes	Durable, low input	Narrow germplasm base; race variability; long breeding cycles	Antwi-Boasiako <i>et al.</i> (2022); Jha <i>et al.</i> (2023)
Genomics/gene editing/RNAi	Viruses, <i>Sclerotinia</i> , others	Precision; faster cycles	Cost, regulation, access	Jha <i>et al.</i> (2023); Meghwal <i>et al.</i> (2026)
Biocontrol agents	Soil-borne and foliar fungi	Eco-friendly; residue-free	Inconsistent field performance	Antwi-Boasiako <i>et al.</i> (2022); Sbai <i>et al.</i> (2024)
Botanicals/essential oils	Fungi, some viruses	Biodegradable; low toxicity	Phytotoxicity; limited field data; standardisation	Kaur <i>et al.</i> (2023); Sbai <i>et al.</i> (2024)
Fungicides	Fungi	Rapid, effective in outbreaks	Cost, resistance, environmental and health risks	Antwi-Boasiako <i>et al.</i> (2022); Sbai <i>et al.</i> (2024)
Digital/nano tools	Pest and disease forecasting; delivery	Precision, reduced chemical load	Access, cost, data governance	Meghwal <i>et al.</i> (2026)

Conclusion

Legume diseases are diverse, dynamic and increasingly shaped by climate change and changes in cropping practice. Seed-borne and residue-borne pathogens such as *Curtobacterium flaccumfaciens*, *Ascochyta*, *Fusarium*, *Botrytis* and *Sclerotinia* can re-emerge quickly when sanitation, rotation and clean seed are neglected and warming, drought, humidity swings and vector expansion tend to favour pathogens over hosts. No single measure is adequate. Durable protection rests on an integrated strategy: certified clean seed, sanitation and diversified rotations; resistant cultivars developed with modern genomic tools and wild germplasm; biological agents, botanicals and essential oils where field evidence supports them; judicious, well-timed fungicide use with attention to resistance management; and emerging nano- and digital technologies for targeted delivery and early warning. Realising climate-resilient, disease-resistant legume production will require sustained collaboration among pathologists, breeders, agronomists, extension services and policymakers.

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