Dry Root Rot of Chickpea: A Disease Favored by Drought

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Abstract

Chickpea is an essential crop for protein nutrition and is grown around the world in rain-fed conditions. However, chickpea cultivation is under threat due to emerging diseases favored by drought stress. Dry root rot (DRR), an economically devastating disease, is an example. Chickpea-specific strains of a necrotic fungal phytopathogen, *Macrophomina phaseolina*, cause DRR. Microsclerotia of this fungus, which are capable of withstanding harsh environmental conditions, serve as primary inoculum. Initial symptoms are scattered necrotic spots in roots, progressing to rotting and withering lateral roots, accompanied by prematurely dried, straw-colored foliage. The recent rise in global temperature and worsening of drought spells have aggravated DRR outbreaks in chickpea. To date, DRR epidemiology has not been clarified in detail. Also, the literature lacks clarity on *M. phaseolina*

taxonomy, morphology, disease progression, and diagnosis. In this article, research progress on patterns of DRR occurrence in the field and belowground and aboveground symptoms are clarified. In addition, the current understanding of taxonomy and management practices is elaborated. We also summarize knowledge of the impact of drought and high temperature on DRR severity. Furthermore, we provide future perspectives on the importance of host resistance, quantitative trait loci identification, and genotype screening for the identification of resistant genotypes. The article proposes new research priorities and a corresponding plan for the mitigation of DRR.

Keywords: disease complex, drought stress, dry root rot, high temperature, *Macrophomina phaseolina*, microsclerotia

Chickpea (*Cicer arietinum* L.), also known as garbanzo, is an integral part of many Asian and sub-Saharan countries' food security programs, and this region accounts for more than 95% of the global chickpea production (Singh 1997; Wood and Grusak 2007) (Fig. 1). This versatile legume is grown in 57 countries around the world (Merga and Haji 2019). However, it is affected by at least 172 pathogen taxa, which cause both root and foliar diseases (Manjunatha et al. 2011; Nene and Sheila 1996; Pande et al. 2012). In addition to biotic stresses, abiotic stresses such as high temperature, drought, and salinity hamper chickpea production (Devasirvatham and Tan 2018; Devasirvatham et al. 2012; Flowers et al. 2010; Sinha et al. 2019). Yield losses due to damage to the root architecture are attributed to root diseases (Ahmad et al. 2005; Gaur et al. 2013). Among root diseases, in addition to Fusarium wilt (caused by *Fusarium oxysporum* f. sp. *cicero*), chickpea is affected by dry root rot (DRR), caused by a chickpea-specific strain of *Macrophomina phaseolina*. In earlier literature, the DRR causal agent was referred to as *Rhizoctonia bataticola*. Other root rots in chickpea include wet root rot (caused by *Thanatephorus cucumeris*, formerly known as *Rhizoctonia*

solani), collar rot (caused by *Athelia rolfsii*), and black root rot (caused by *F. solani*) (Ghosh et al. 2013; Kaur et al. 2012; Sharma et al. 2015).

DRR causes chickpea yield loss up to 100% in susceptible varieties under favorable conditions (Gupta and Sharma 2015). The DRR pathogen has a broad host range, which limits rotation options for disease management. Abiotic stresses such as drought and high-temperature stress aggravate DRR symptoms in the field (Sinha et al. 2019, 2021). DRR is widespread in chickpea-growing regions such as South Asia, Africa, Iran, Mexico, the United States, and Spain (Fig. 1) (Gupta and Sharma 2015). It is also noteworthy that most chickpea cultivation is in rainfed farmlands; in India, for example, chickpea is cultivated over 6.3 million hectares (mha), of which 85% is rainfed. Climate change is exacerbating erratic rainfall patterns and the severity and frequency of droughts (Fig. 1), and high-temperature stress, which elevates the risk of economic losses due to DRR. As a result, chickpea productivity was predicted to decrease substantially in India, Pakistan, and Ethiopia (Andrews and Hodge 2010; Foyer et al. 2016), where DRR occurs widely. Although the average worldwide yield of chickpea is about 1.8 tons/ha (FAO-STAT 2019; Merga and Haji 2019), the average yield in the west Asian and South Asian countries is only 1.46 tons/ha.

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DRR Symptoms

DRR-affected chickpea plants in the field exhibit leaf yellowing and premature drying (Figs. 2 and 3). Unmanned aerial vehicle-based field imaging shows that DRR-affected plants occur in patches of irregular size and shape, distributed unevenly in the field (Figs. 2A to C and 3A to C). DRR-affected fields have patches of straw-colored, dried plants. This pattern may be due to the uneven distribution of the DRR pathogen in the field and plant resistance. Under mild infection by *M. phaseolina*, patches of diseased plants are relatively small, corresponding with yield losses of up to 5 to 10% per hectare (Figs. 2A and 3B). However, under moderate infection,

infected patches can expand and coalesce, corresponding to yield losses of 30 to 50% (Fig. 2A). Under severe infection, the entire chickpea field shows prematurely dried plants, accounting for 60 to 80% yield loss (Figs. 2B and 3C). At the seedling stage, in a particular disease patch, some seedlings are not infected after emergence and show a green canopy; these are assumed to be disease escapes (Fig. 2D). Although plants might be infected at the seedling stage, DRR foliar symptoms usually appear at the reproductive stage (Fig. 3A to C).

Belowground symptoms. In the field, symptoms start with the root, while aerial symptoms are exclusively attributed to root damage (Fig. 4). Typical root symptoms of DRR disease include brownish to black lesions in the taproots and lateral roots (Fig. 4E to H) (Hwang et al. 2003; Sharma et al. 2013). *M. phaseolina* causes necrosis in the roots, and gradual progression of necrosis results in the shedding of lateral roots (Fig. 4F to H). Root necrosis increases with pathogen exposure, and severe DRR appears as dark asymmetric lesions on the root epidermis, brittle and rotten primary roots, and a lack of lateral roots (Fig. 4G and H). The taproots remain blackish-brown but intact, whereas the bark and lateral and finer roots are destroyed and shredded (Fig. 4H). Many black microsclerotia of *M. phaseolina* are visible in the root bark, cortex, and pith regions (Fig. 4J and L). It is plausible that premature drying occurs due to the blockage of the stele by fungal microsclerotia and mycelia (Singh et al. 1990), compromising the transport of water and nutrients. Rotten primary roots, lack of lateral roots, and blockage of vascular bundles underlie sudden premature foliar drying, which is distinct from yellowing at physiological maturity.

Aboveground symptoms. Foliar yellowing usually occurs during the transition from vegetative to flowering stages (Fig. 4B to D). Foliar symptoms start with gradual yellowing from the base to the top leaves (Fig. 4B). DRR-affected plant shoots do not appear wilted,

unlike those affected by *Fusarium* wilt, but, instead, stand upright with straw-colored shoots and leaves (Fig. 4C and D). The shoot base develops a brownish color. Premature drying is brought about by the combined action of progressive infection in the roots, plant developmental stage, and low soil moisture. It is important to distinguish foliar yellowing due to DRR from yellowing caused by plant physiological maturity. Healthy plants are difficult to uproot because their intact root system anchors them firmly. In contrast, DRR-affected plants are easy to uproot because they have dry, rotten roots and are usually devoid of lateral roots. After uprooting, most of the primary root remains in the soil because the rotten primary root is broken, which provides a refugium for DRR inoculum. Healthy chickpea plants undergo drying with matured pods only at the physiological maturity stage (90 to 120 days after sowing [DAS]), whereas DRR-affected plants undergo sudden premature drying with underdeveloped pods at 60 to 80 DAS (Fig. 3A to C); plants stay green from the vegetative to the podding stage, and foliar yellowing starts only at the harvest stage. In contrast, DRR-affected plants turn yellow during the flowering stage (Fig. 3A to C). Noninfected chickpea fields display yellow foliage at maturity, whereas DRR-infected fields have scattered straw-colored plants in patches because DRR-infected plants dry prematurely (Figs. 2A and 3B and C).

The Pathogen and the Disease Cycle

M. phaseolina (Tassi) Goid. is a soilborne necrotrophic fungal phytopathogen. Based on the morphology and sequence data, it is classified under division Ascomycota. Mycelia and microsclerotia of the fungus are present in soil and chickpea plants. Neither pycnidia nor a sexual stage have been reported to date in chickpea. The fungus reproduces by undergoing fragmentation (Ghosh et al. 2013; Sharma et al. 2013).

Fig. 1. Prevalence of dry root rot (DRR) disease around the world. The total chickpea cultivation area in the world encompasses 13 million hectares (mha), including countries with cultivated areas ranging from 0.1 to 9.5 mha and total production ranges from 14 to 16.5 million tons per year. Chickpea yield is compromised by an emerging disease called DRR caused by chickpea-specific *Macrophomina phaseolina*. *M. phaseolina* is reported widely in the countries marked in light blue. Drought stress ranges from low (light yellow) to high (dark orange); geographical locations marked in red indicate DRR-affected countries. Countries marked in the dark blue show the distribution of chickpea-infecting *M. phaseolina* isolates and strains. Area harvested data were collected from FAOSTAT (2021) for the last 10 years (2009 to 2019), and the average value is presented next to each country's name. Data on disease incidence level (values provided in parentheses next to the area harvested) were collected from existing literature. Drought stress indices were taken from World Resources Institute's Aqueduct Water Risk Atlas.

Salient morphological features. The typical morphological features of *M. phaseolina* are microsclerotia, mycelia, myceliogenic germination, right-angle mycelial branching, multinucleate septate mycelia, cross-wall formation at the beginning of new branching mycelia, and partial hyphal fusion (Fig. 5) (Irulappan and Senthil-Kumar 2021; Sharma et al. 2015; Sinha et al. 2019). Microsclerotia are quiescent structures by which *M. phaseolina* maintains its viability under harsh conditions such as winter, drought, and flood. This quiescent state can often last for 10 months under dry storage. Scanning electron micrographs demonstrated that it produces a rindless type of microsclerotium (Fig. 5B), in which all of the cells have a thick cell wall. Because the microsclerotia are cemented together by a dark-pigmented gelatinous matrix, they appear rigid and dark in color (Fig. 5C). The pigmentation is most intense at the center of a microsclerotium and least at the periphery. The diameter of microsclerotia of *M. phaseolina* ranges from 50 to 150 μm (Fig. 5B, C). Moisture, temperature, aeration, soil organic matter content, and fungicides influence the survival of microsclerotia.

Fig. 2. Distribution patterns of dry root rot (DRR) disease in a chickpea field. Chickpea plants (genotype JG 11) with DRR exhibited symptoms during the reproductive stage. Pathogen-infected plants were scattered in the field and exhibited prematurely dried straw-colored foliage. Symptoms started with sudden yellowing of the foliage and ended with the premature withering of the plant. In a row of DRR pathogen-infected plants with foliar yellowing symptoms, asymptomatic green plants were also present. **A**, Aerial view of a chickpea field moderately infected with DRR at the reproductive stage (90 to 100 days after sowing) (location: 14.922662°N, 77.263522°E; the photo was taken in January 2020). The dotted line indicates scattered DRR symptomatic plants in patches. **B**, Aerial view of the severely infected chickpea field at crop maturity (location: 14.8887°N, 77.2075°E, the photo was taken in January 2021). **C**, Detailed view of panel A shows chickpea plants with typical symptoms. **D**, Closer view of the field in panel B shows a field patch where healthy plants escaped infection (yellow arrow) within a row of randomly distributed infected plants (white arrow). Photo credit: Drs. Chandra Obul Puli Reddy and Vadivelmurugan Irulappan.

Under in vitro conditions, the hyphae originating from germinating microsclerotia are hyaline, branched, and septate, with mycelia that are usually fluffy and gray. The mycelia turn dark colored at an advanced growth stage (Fig. 5A to C). The characteristic dark melanin pigments of microsclerotia are antioxidant substances that serve as free radical scavengers to reduce the concentration of intracellular reactive oxygen species (Butler and Day 1998; Henson et al. 1999). On potato dextrose agar (PDA), when a few microsclerotial bodies start to aggregate, microsclerotia formation is initiated 36 h postincubation at 28°C. Subsequently, microsclerotial formation and count increase until 48 h postincubation, and dark pigmentation appears at 60 h (Fig. 5C). Hyphae of myceliogenic origin anastomose frequently and generally branch at a 90° angle.

Disease cycle. Field conditions. The rate of progress of a DRR outbreak under field conditions is determined by host plant susceptibility, initial inoculum concentration, soil moisture, and temperature. Cultivation of DRR-susceptible chickpea under high pathogen inoculum levels facilitates the outbreak. Microsclerotia released from DRR-affected roots in the previous season act as primary inoculum (Fig. 6). Microsclerotia stay dormant but viable in the soil, free or attached to plant debris. Overwintering or oversummering microsclerotia are important to between-season survival (Fig. 6A). Microsclerotia survival is reduced under high soil moisture conditions (Lodha et al. 1990) but can stay quiescent but viable for up to 15 years in soil (Baird et al. 2003; Gupta et al. 2012). The disease cycle has an extended incubation period under field conditions, often because of relatively low inoculum concentrations in the field (Irulappan and Senthil-Kumar 2021). After attachment of microsclerotia to the plant root (Fig. 6B, 1 to 10 DAS), necrosis begins to appear in the root epidermis (Fig. 6B to C, 10 to 20 DAS), and the number of necrotic lesions in the root epidermis increases with incubation time. Until the reproductive stage, the infection level increases in the root with asymptomatic foliage (Fig. 6D, 20 to 40 DAS). At the reproductive stage, most of the plant roots are infected with the DRR pathogen, and lateral and fine root loss begins (Fig. 6E, 40 to 60 DAS). In addition to the loss of roots due to infection, drought stress periods on the rainfed crop in the “rabi” season (agricultural season in India from November to April) further weaken the root system. At flowering and pod filling stages, the development of DRR symptoms is quickened in the presence of moderate drought stress (Fig. 6F and G). The infection period after foliar symptom appearance is called the active infection period (Fig. 6E to G, 40 to 90 DAS).

Controlled conditions. Investigating disease progression under controlled conditions is valuable for conducting advanced studies for understanding the plant–pathogen interaction and for screening genotypes for disease tolerance. In this regard, the standard techniques commonly used are the sick pot and blotting paper techniques (Irulappan and Senthil-Kumar 2021). The sick pot technique involves adding artificially propagated microsclerotia to potting mix for infecting plants during germination and subsequent plant growth phases. The blotting paper technique involves inoculating the fungal propagules on the plants and assessing the infection in vitro on blotting paper (Irulappan and Senthil-Kumar 2021; Pande et al. 2012). In a sick pot, in the presence of host plant roots, microsclerotia give rise to vegetative mycelium, as confirmed with chitin-specific lectin stain (wheat germ agglutinin labeled with fluorescein isothiocyanate) (Fig. 5D). The fungus attaches to epidermal root tissues with germ tubes 1 to 3 days after seed sowing (Fig. 5E). All of the cells of microsclerotia can produce germ tubes (Coley-Smith and Cooke 1971; Smith et al. 2015). Germ tube structures contain plant cell-wall-degrading enzymes (PCWDEs), lipases, oxidases, and cutinases to aid in the degradation of plant wax and cuticle (Mengiste 2012). The invasion starts with microsclerotia differentiation into hyphae, which grow along the longitudinal axis of the root epidermis and penetrate between root epidermal cells (Fig. 5F and G). During root colonization, after extensive hyphal branching, the DRR pathogen grows intercellularly (4 to 6 DAS) (Fig. 5G). At this stage, hyphae become melanized and invade intercellular spaces of root epidermal cells, forming a network around the root (Fig. 5H). The DRR pathogen forms mycelium or mycelial aggregates with 50 to 200 cells

containing melanin pigments with little or no carbon supplementation (Fig. 5G and H) (Coley-Smith and Cooke 1971; Smith et al. 2015). Formation of daughter microsclerotia is observed at the surface of the root epidermal cells after colonization (Fig. 5I), suggesting that the DRR pathogen invades the taproot system and damages epidermal root cells by secreting PCWDEs (Amadioha 1998). Extensive colonization occurs after intra- and intercellular penetration through cortex and xylem vessels, resulting in thick, dark-colored, depressed necrotic lesions. In the advanced stages of DRR development, naked-eye examination clearly shows dark-brown, localized discoloration and daughter microsclerotia at the surface of chickpea roots (Fig. 4J and L). In controlled conditions, *M. phaseolina* appears to establish preferentially on mature roots.

Rhizosphere Microenvironment and Edaphic Factors Impact Pathogenicity of the DRR Pathogen

Lodha et al. (1990) demonstrated that topsoil with decomposed infected roots had more microsclerotia of *M. phaseolina* than underlying soil layers. Similarly, the microsclerotium count was more at 0 to 5 cm of soil depth than at deeper levels (Singh et al. 1990).

Plant root exudates trigger pathogen germination, acting as nutrients. Studies on the effect of root exudates on the DRR pathogen are sparse. However, we assume that low soil moisture and high soil temperature aggravate disease incidence and severity. Drought stress and soil temperature increase plant root exudates, which increases colonization in plants under drought stress (De Coninck et al. 2015).

Edaphic factors can negatively affect microsclerotia survival and germination efficiency or promote antagonistic microbes. Although microsclerotia disintegrate under moist soil conditions, saturated soil causes much less damage. The microsclerotia load was higher in dry soil than in saturated soil (Lodha et al. 1990). Olaya and Abawi (1996) demonstrated that high (−8.0 MPa) and low (−0.6 MPa) osmotic potential in PDA caused by sucrose harbored fewer microsclerotia than at intermediate osmotic potential (−2 MPa). Lodha et al. (1990) demonstrated that low soil temperature reduces *M. phaseolina* microsclerotia germination efficiency and pathogen populations. Low soil temperature promotes the growth of antagonistic microbes, including actinomycetes. Given that chickpea is a rabi (or winter-sown) crop in India, the plant and pathogen are exposed to saturated soil at the beginning of the season, which reduces the amount of microsclerotia. This phenomenon explains reduced disease incidence

Fig. 3. Dry root rot symptoms at different developmental stages. Symptoms are less frequently observed at the vegetative stage in field-grown chickpea (variety JG11) but are prominent at the reproductive stage, especially in the presence of drought. **A**, Chickpea fields at the vegetative stage (30 to 40 days after sowing [DAS]). **A** healthy chickpea field (left) and a field showing the onset of dry root rot (DRR) symptoms (white arrows) (right). **B**, Chickpea fields at the reproductive stage (50 to 80 DAS). No symptoms (left) and representative DRR symptoms such as straw-colored foliage. **C**, Fully mature chickpea field (90 to 120 DAS) with healthy pods (left) and moderately infected field with lower yield (right). Location: 14.537813°N, 78.560633°E; 14.4673°N, 78.8242°E. Photo credit: Drs. Chandra Obul Puli Reddy and Vadivel-murugan Irulappan.

Fig. 4. Symptoms in the aerial parts and roots of chickpea. **A**, Control plants (JG 11) without any foliar symptoms; **B**, dry root rot (DRR)-affected plant with the onset of foliar yellowing; **C**, complete foliar yellowing; **D**, premature dried and straw-colored foliage; **E**, control plant root with complete lateral and primary root with root nodules; **F**, DRR-affected plant with a lack of nodules; **G**, decreased growth of the primary root and lateral roots; **H**, brittle primary root lacking lateral roots; **I**, control root with no infection; **J**, microsclerotia (fungal propagules) in the root cortex and stele; **K**, transverse view of a control plant root; and **L**, transverse view of microsclerotia in the epidermis, cortex, vascular, and pith regions. Images were captured during the reproductive stage in control (**A**, **E**, **I**, and **K**) and DRR-affected (**B** to **D**, **F** to **H**, **J**, and **L**) fields. Photo credit: Drs. Chandra Obul Puli Reddy and Vadivelmurugan Irulappan. Scale bars = 2 cm (**E**, **F**, **G**, and **H**), 1 mm (**I**), and 500 µm (**K** and **L**).

Fig. 5. Morphological characteristics of the dry root rot (DRR) pathogen and stages of infection on chickpea root. **A**, *Macrophomina phaseolina* colony (5 days old) on potato dextrose agar (PDA). **B**, Microsclerotia at 250 $\times$ magnification under scanning electron microscope from a 10-day-old fungal plate (scale bar, 100 μ m). Individual microsclerotia at 2,000 $\times$ (right) (scale bar, 20 μ m). **C**, Growth of *M. phaseolina* on PDA from new mycelia (12 h) to melanized and pigmented mature daughter microsclerotia (60 h). Roots (genotype JG 62) were infected with the pathogen in the sick pot, collected, and processed, and mycelium was labeled with the wheat germ agglutinin labeled with fluorescein isothiocyanate fluorescent stain. **D**, Septate mycelium (arrow indicates septum). **E**, Infected roots containing germinated microsclerotia at 3 days after sowing (DAS). **F**, Mycelium penetrating the plant root epidermis (arrow) at 3 DAS. **G**, Mycelium extension (arrow) over the surface of the root epidermis at 3 DAS. **H**, Initial stages of daughter microsclerotia formation (arrow) on the infected root at 6 DAS. **I**, Mature daughter microsclerotia (arrow) on the infected root at 6 DAS.

in furrow-irrigated chickpea fields and in well-irrigated pot experiments (Sharma and Pande 2013). An artificial neural-network-based prediction showed that edaphic factors such as clay content and micronutrients such as potassium and organic carbon increase DRR severity (Sinha et al. 2021). The aeration inherent with certain soil types affects microsclerotial germination efficiency, because the accumulation of CO₂ compromises germination efficiency. For instance, sandy loam soil can hold adequate air in its abundant macropores, thereby supporting microsclerotial germination, resulting in higher DRR incidence than clay loam soil (Jordan et al. 1984). It is reported that the high iron content, acidic pH, exposure to sunlight, and high oxygen levels induce intracellular oxidative stress, regulating microsclerotium formation. Among these, pH is the primary determinant regulating microsclerotium formation and mycelium emergence. In synthetic culture medium, sclerotia developed poorly at neutral or alkaline pH (Rollins and Dickman 2001). Lowering the soil pH from 8.0 to 6.0 removed inhibitory effects on root rot diseases by affecting interactions among pathogens and their antagonists (Cruz et al. 2019). Optimum growth of the DRR pathogen occurs at pH 5.5 (Dhingra and Sinclair 1973).

Impact of Abiotic Stresses

Drought stress. As a rainfed crop, chickpea often experiences moderate to severe drought stress during its life cycle (Beniwal et al. 1992; Manjunatha et al. 2011; Sharma and Pande 2013; Yimer et al. 2018). In India, the chickpea cultivation season spans from November to April. In this season, high soil moisture content occurs from November to January, and the soil moisture recedes to drought conditions from February to April (Manjunatha et al. 2011; Sharma and Pande 2013). In Ethiopia, chickpea is cultivated during the postrainy season (late September) (Beniwal et al. 1992; Yimer et al. 2018). Likewise, in Spain, chickpea is cultivated in winter (December) and as a spring crop (March), in which soil moisture is relatively less

(Navas-Cortés et al. 1998). Soil moisture status does not affect the initiation of infection by the DRR pathogen in chickpea roots (Fitt et al. 1989; Papavizas 1977; Sharma and Pande 2013). Even so, low soil moisture status influences further infection level, disease severity, and symptom development. The onset of drought stress begins during the transition from the vegetative to the reproductive stage (Sharma and Pande 2013; Sinha et al. 2019, 2021). In a field experiment (Sinha et al. 2019), DRR incidence increased with the level of drought stress; higher incidence (40 to 60%) occurred in severely drought-stressed plots, whereas pathogen treatment under sufficient irrigation had lower disease incidence (0 to 20%). Similarly, the increase in DRR incidence and severity level under drought stress was demonstrated under controlled conditions (Sharma and Pande 2013; Sharath Chandran et al. 2021; Sinha et al. 2019). Reduction in root water potential increased lytic enzyme activities of the DRR pathogen, which stimulated disease progression (Amadioha 1998). Under drought, microsclerotia and first-order lateral roots in the top-soil experience more intense drought stress than roots in the subsoil. This explains the shedding of lateral roots with DRR and, thereby, the ease with which plants can be uprooted.

High-temperature stress. Some soilborne necrotrophic pathogens tolerate high-temperature stress better than the host and have the ability for maximum reproduction and dissemination. This competitive advantage of the pathogen was demonstrated by studies showing increased necrosis and colonization in plants at high temperatures (Desaint et al. 2021; Elad and Pertot 2014). Soil temperature also affected the interaction of soilborne pathogens with plants (Delgado-Baquerizo et al. 2020; Gaur et al. 2013).

In addition, relatively warm temperatures can increase the accumulation of certain secondary metabolites and enzymes (Ahanger et al. 2013; Suzuki et al. 2014). The ability of DRR pathogens to degrade plant cell walls is dependent on the production of cellulolytic enzymes. In the carboxymethyl cellulose broth medium, soybean-specific *M. phaseolina* produced the maximum number of cellulolytic

Fig. 6. Disease cycle of dry root rot. **A**, Microsclerotia of *Macrophomina phaseolina* in soil. **B**, When microsclerotia encounter root exudates, they attach to the epidermis and germinate (infection stage 1). **C**, Mycelia give rise to appressoria and haustoria, which infect plant cells and cause initial local necrosis (infection stage 2). **D**, Once the infection is established, fungal mycelia grow intercellularly and infect more cells (infection stage 3). **E**, Production of secondary inoculum results in more infection and shedding of lateral roots, as well as the onset of foliar yellowing (infection stage 4). **F**, Extensive necrosis in lateral and primary roots and complete yellowing of foliage (infection stage 5). **G**, Premature drying and typical straw-colored foliage and brittle, rotted primary root with daughter microsclerotia (infection stage 6). Plants on the right side of the images are healthy. The dotted line indicates the transition from vegetative to the reproductive stage and the start of soil drying.

enzymes at temperatures ranging from 15 to 35°C and less at temperatures below 15 and above 35°C. In the chickpea–DRR pathosystem, disease incidence and severity were maximum at 35°C. Thus, the temperature increased infection and colonization (Gawade et al. 2018; Sharath Chandran et al. 2021; Sharma and Pande 2013).

Distinguishing Root Diseases and Root Rot Complex

In the field, chickpea plants encounter multiple root diseases. The foliar symptoms of root diseases appear similar at the reproductive stages of the plants. Nevertheless, the etiology and epidemiology of each root disease differ at early infection stages, and correct identification of disease is indispensable for disease management. Root rot often occurs as a disease complex, including DRR and the Fusarium wilt complex, in India, Ethiopia, Spain, and the United States (Beniwal et al. 1992; Pandey et al. 2017; Trapero-Casas and Jiménez-Díaz 1985;

Westerlund et al. 1974). The wilt–DRR complex has not yet been well studied.

Root diseases. Chickpea plants are exposed to infection as soon as the seed germinates. The diseases that develop include Fusarium wilt, black root rot, wet root rot, and DRR (Fig. 7A to E). Although microsclerotia are the primary inoculum of the DRR pathogen, conidia, chlamydospores, or macrosclerotia (0.5 to 2.5 mm in diameter and including a rind) are primary inocula of *F. oxysporum* and *A. rolfii*. The DRR pathogen causes necrosis in chickpea roots (Singh et al. 1990; Sinha et al. 2019), whereas *F. oxysporum* and *A. rolfii* do not (Pande et al. 2012). Another distinction lies in the plant stage at which the symptoms appear. For instance, DRR-infected, field-grown plants manifest symptoms mainly at the reproductive stage, whereas collar-rot-affected plants exhibit symptoms at the seedling stage and Fusarium wilt at both the seedling and vegetative stages (Pande et al. 2012) (Fig. 7F to H). Symptoms also differ by disease.

Fig. 7. Differences among the root diseases. **A**, Control (healthy) plants. **B**, Dry root rot (DRR)-infected plants with straw-colored foliage and brittle, rotted primary roots devoid of lateral roots. **C**, Wilted green foliage caused by *Fusarium oxysporum* f. sp. *ciceri* at the seedling stage. **D**, Collar-rot-infected plant at the seedling stage; the pathogen is *Athelia rolfii* Sacc. **E**, Wet root rot and black root rot caused by *Rhizoctonia solani* and *F. solani*, respectively. **F to H**, Influence of soil moisture and temperature on disease severity. High soil moisture favors collar rot and early wilt disease, whereas low soil moisture and high temperature favor DRR.

For instance, DRR-affected chickpea plants have dried, straw-colored foliage standing upright, whose rotted primary roots are dotted with minute microsclerotia. In contrast, collar-rot-affected plants have rotted collar regions, resulting in foliar wilting and yellowing (Irulappan and Senthil-Kumar 2021; Sharma et al. 2015; Sinha et al. 2019). Fusarium wilt-affected plants appear wilted, and the vascular region of the plant root exhibits brown discoloration due to toxins. *F. oxysporum* causes late wilt as well, appearing at the reproductive stage (Fig. 7A to H).

Root rot complex. In chickpea, *M. phaseolina*, *F. oxysporum*, *F. solani*, and other root-rot-causing pathogens participate in a disease complex. However, the interactions among the pathogens are poorly understood. Nevertheless, chickpea variety and date of sowing were hinted to play a role in the appearance of the disease complex. In addition to these factors, pathogen populations, temperature, and moisture conditions may be associated. DRR pathogen and black root rot pathogen coinfection has been reported in chickpea plants. In addition, late wilt-affected plants are more prone to infection by the DRR pathogen, increasing the severity of symptom development (Fig. 8) (Yimer et al. 2018).

Resolving the etiology of DRR. Completing Koch's postulates, along with DNA-sequence-based methods, are needed for confirming identity. PCR-based identification techniques use primers that amplify the internal transcribed spacer (ITS) region and other genes of the target fungus (Basandrai et al. 2021; Santos et al. 2020). The primary inoculum for this fungus is present in the soil, and the identification can be done by using DNA isolated from the soil. Furthermore, fungal DNA isolated from pathogen-infected plant roots was also successfully used for resolving the etiology (Babu et al. 2007; Schoch et al. 2012).

Disease Management: Challenges and Solutions

In the future, climate change is expected to make rainfall patterns increasingly erratic which, in turn, is likely to aggravate drought and heat stress (Mittler and Blumwald 2010). These changes are expected to increase the incidence of DRR in chickpea (Pandey and Basandrai 2021). Furthermore, areas prone to DRR might expand to regions where it was not a problem before. Such a scenario calls for the implementation of integrated disease management (IDM) to reduce chickpea yield losses due to combined DRR, drought, and heat stresses. IDM includes application of fungicides (Khaliq et al. 2020; Sharma and Kumari 2017), seed treatment with biocontrol agents such as *Rhizobium* and *Trichoderma* spp. (Manjunatha et al. 2013), early disease diagnosis, use of DRR-resistant varieties, rotation to nonhost crops for a few successive crop seasons (Hornby 1983), and regulation of the irrigation schedule. In India, soil solarization and fumigation are currently parts of IDM to minimize initial pathogen inoculum. In this practice, the fields are covered with polythene sheets, which increases the soil temperature by 35 to 60°C, killing pathogens in the field soil. The disadvantage with this practice is that it also eradicates beneficial microbes (Chauhan et al. 1988). Similarly, soil fumigation employs volatile gases such as methyl bromide. The disadvantages of this method are the high cost and eradication of beneficial microbes. Another IDM practice is the application of broad-spectrum fungicides. Disadvantages of this approach are high cost, a limited spectrum of active ingredients, the challenge of establishing sufficient contact between the active ingredients and fungal pathogen propagules, and ingredients not being eco-friendly. Because DRR incidence and severity increase with drought stress, chickpea plants also need irrigation at branching and

Fig. 8. Late wilt and dry root rot. Symptoms of wilt caused by *Fusarium oxysporum* f. sp. *ciceri* and *Macrophomina phaseolina* in the foliage and root. Conidia, the primary inoculum for wilt, cause the infection in the root, and infected plants wilt progressively. Microsclerotia, the primary inoculum of the DRR pathogen, cause initial necrosis and colonize the root. Followed by the complete necrosis of lateral and primary roots, premature plant drying occurs. The DRR pathogen can infect wilt-affected plant roots and eventually lead to the root rot disease complex under field conditions.

pod-filling stages. Providing irrigation at these growth stages reduces DRR incidence and severity.

Early diagnosis of *M. phaseolina* using molecular tools helps timely initiation of IDM practices. For example, the loop-mediated isothermal amplification assay, which is based on amplification of the ITS region, has been used for early pathogen diagnosis (Ghosh et al. 2017). Variability in genetic architecture and virulence has been observed among DRR pathogen isolates collected from various geographic locations (Aghakhani and Dubey 2009; Devi et al. 2016; Mahdizadeh et al. 2011; Manjunatha 2014; Sundravandana et al. 2011). This could lead to infection spread to a larger number of cultivated varieties. An effective approach is the use of chickpea genotypes that show resistance to a wide range of pathogen isolates. The chickpea germplasm that has a genetic variation for DRR resistance is a useful resource for further breeding to select additional resistant genotypes (Upadhyaya et al. 2013). Various screening techniques employed under greenhouse and field conditions can be used for germplasm screening (Infantino et al. 2006; Irulappan and Senthil-Kumar 2021; Irulappan et al. 2021; Nagamma et al. 2015). Using such methods, several DRR-resistant genotypes have been identified over the years (M. Senthil-Kumar, unpublished data). Unfortunately, the identified genotypes have not proved promising in field conditions, owing to additional effects of drought and heat stress, which influence plant disease resistance.

In addition to genotype screening, identifying quantitative trait loci that confer DRR resistance can enrich breeding programs (Karadi et al. 2021; Talekar et al. 2021). Genome sequences for various genotypes are available; among these, Desi (ICC 4958) and Kabuli (CDC Frontier) genotypes are routinely used for analysis (Parween et al. 2015; Varshney et al. 2013, 2019). Breeding programs to identify DRR resistance by utilizing genetic variation in root exudate composition in different chickpea genotypes were shown to be partly successful (Singh et al. 1993). New techniques such as precision breeding using CRISPR technology may accelerate the development of DRR-resistant varieties. The heritability of DRR resistance traits in chickpea can be improved by considering the importance of tripartite interactions involving chickpea genotype–drought–soil microbiome. Genes associated with nonhost resistance can be identified and further used in chickpea crop improvement.

Conclusions

M. phaseolina has recently emerged as a devastating chickpea pathogen. Histopathological and physiological studies on DRR suggest that drought, high temperature, and edaphic factors influence DRR pathogen interactions with chickpea and exacerbate DRR severity. This paints a worrying picture for chickpea cultivation in the years to come. The elucidation of specific interactions between DRR pathogen and chickpeas such as the attachment of microsclerotia at infection sites and inter- or intracellular movement of the pathogen for extensive colonization in hosts deepens our understanding of this pathogen and its disease-causing ability. A more detailed understanding of the molecular basis of genetic resistance and DRR pathogen pathogenicity will translate into effective control strategies. For instance, decoding the genome of the DRR pathogen strains of chickpea will help identify virulence genes for understanding effector biology and provide new avenues for generating broad-spectrum resistance against the root rot disease complex in chickpea.

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