Archives of Phytopathology and Plant Protection, 2016
http://dx.doi.org/10.1080/03235408.2016.1140564

Taylor & Francis
Taylor & Francis Group

Dry root rot (*Rhizoctonia bataticola* (Taub.) Butler): an emerging disease of chickpea – where do we stand?

Mamta Sharma*, Raju Ghosh and Suresh Pande

Legumes Pathology Division, Research Program-Grain Legumes, International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Greater Hyderabad, India

(*Received 13 March 2015; accepted 4 July 2015*)

Dry root rot caused by *Rhizoctonia bataticola* (*Macrophomina phaseolina*) of chickpea (*Cicer arietinum* L.) is gaining importance in the changed scenario of climate when growing crop is predisposed to high temperature and moisture stress. Being mainly a soil-inhabiting pathogen, many environmental and soil factors are responsible for the development of disease. No systematic research related to the biology, ecology and epidemiology of dry root rot in chickpea has been conducted so far. Research is needed to improve the identification and characterisation of variability within its epidemiological and pathological niches. Limited literature available on host plant resistance for dry root rot indicated lack of resistant sources for this disease. The present article discusses current status of the disease in the context of climate change and possible management options to alleviate the problem.

Keywords: chickpea; climate change; *Macrophomina phaseolina*

1. Introduction

Chickpea (*Cicer arietinum* L.) is the world's second-most widely grown legume after beans (*Phaseolus vulgaris*). It is rich in dietary proteins and good for human consumption; moreover, its ability to form nitrogen-fixing nodules via interaction with rhizobia adds to its uniqueness (Ferguson et al. 2010). It is grown in over 50 countries of Asia, Africa, America, and Oceania in rain-fed environments (https://www.croptrust.org/crop/chickpea/). The annual production of chickpea is 11.30 million tonnes from 12.14 million hectare (FAOSTAT 2012) worldwide. South Asia is the largest producer of chickpea (68%) and India is the largest chickpea growing country with an annual production of 7.70 million tonnes from 8.32 million hectares (FAOSTAT 2012).

The production of chickpea is largely constrained by Fusarium wilt (*Fusarium oxysporum* f. sp. *ciceris*); however, recent reports indicated that dry root rot (DRR) is emerging as a potential threat to chickpea production (Pande et al. 2010; Sharma et al. 2010; Ghosh et al. 2013). The DRR is caused by *Rhizoctonia bataticola* (Taub.) Butler. (Synonym: *Macrophomina phaseolina* (Maubl.) Ashby.) and is an important component of the disease complex that causes root rots and seedling blight in many grain legumes when they are weakened by other stress factors (Hwang et al. 2003). *R. bataticola* is a soil-inhabiting organism capable of infecting chickpea at any crop stage, but most commonly infects chickpea at post-reproductive stage in dry and warm regions (Sharma & Pande 2013). The information on worldwide losses caused by DRR is not available, but

*Corresponding author. Email: mamta.sharma@cgiar.org

there is no doubt that the disease is of increasing importance and has the potential to cause devastation in susceptible cultivars, particularly in the conditions of high temperature and soil moisture stress. Savary et al. (2011) described DRR as an acute-emerging disease that occurs irregularly, both temporally and spatially, and may cause massive disruptions in system performances and whose range is expanding to new areas.

Our knowledge and understanding of plant–pathogen interaction are on the rise with the advancement of technology. Recent reports of complete genome sequence of chickpea (Varshney et al. 2013), for instance, have provided invaluable resources for conducting future research on disease resistance in this crop. The present review focuses on the current status of DRR disease of chickpea with special reference to its occurrence and distribution under changing climatic conditions. Additionally, a detailed account of infection, advances made in biology, epidemiology and management strategies for DRR are also included.

2. *Rhizoctonia bataticola*: general description

Rhizoctonia is a genus of anamorphic fungi in the order Cantharellales and family Ceratobasidiaceae. *Rhizoctonia* species do not produce spores, but are composed of hyphae and sclerotia (hyphal propagules), asexual stage of fungi. *Rhizoctonia* species are saprophytic, but some act as facultative plant pathogens causing commercially important crop diseases. The genus *Rhizoctonia* (means "root killer") was described in 1815 by the French mycologist Augustin Pyramus de Candolle for plant pathogenic fungi that produce both hyphae and sclerotia. Subsequent authors added over 100 additional names to the genus. A comprehensive survey and redisposition of species name "*Rhizoctonia*" was published in 1994 by Anderson and Stalpus. According to this published information, *Rhizoctonia bataticola* is used as a synonym to *Macrophomina phaseolina* (Tassi) Goid 1947. Currently, *M. phaseolina* is officially recognised as the correct taxonomic name (CMI description of pathogenic fungi and bacteria No.275) with the sclerotial phase known as *Rhizoctonia bataticola* (Holliday & Punithalingam 1970). Due to the presence of only sclerotial phase in chickpea, the pathogen is referred as *Rhizoctonia bataticola* (Taub.) Butler in this article.

3. Dry root rot: an emerging disease in chickpea

The DRR in chickpea was first reported from India by Mitra (1931); later, the disease has been reported from most chickpea-growing areas in India and other countries like Iran (Kaiser et al. 1968), the USA (Westerlund et al. 1974) and several countries in Asia and Africa (Nene et al. 1996). The disease was earlier known as "Rhizoctonia wilt" in chickpea; however, later it was named as "dry root rot". The DRR was not of much significance in chickpea earlier; however, it has become a major threat to chickpea production in recent years due to altered weather conditions, particularly on the account of longer drought spells. Higher temperature and soil moisture depletion during crop growth period particularly at post-harvesting stage are predisposing chickpea to DRR (Sharma & Pande 2013).

Recent surveys conducted during 2010–2013 indicated widespread and increased incidence of DRR in the central and southern states of India (Ghosh et al. 2013). Disease was found irrespective of soil types, cropping system and cultivars used and incidence ranged from 5 to 50% or more in badly infected soils. This noticeable widespread geographic distribution of DRR probably makes it a significant disease in

chickpea for in-depth studies aimed at understanding the fungal behaviour under future climate change projections. Based on the available reports, the following sections linked the role of climatic factors to disease development, defence mechanisms and strategies for its management.

4. External manifestation of dry root rot: phenotypic changes

The DRR symptoms are most commonly observed in chickpea during post-flowering stage which include drooping and chlorosis of petioles and leaflets, initially confined to top leaves of the plant (Figure 1(a) and (b)). Leaves and stems of affected plants are usually straw coloured and in some cases, the lower leaves and stems are brown. The tap root turns black with signs of rotting and is devoid of most of the lateral and finer roots (Figure 1(c)). The dead roots are quite brittle and show shredding of the bark. The

Figure 1. Symptoms of dry root rot disease of chickpea (a) field symptoms, (b) dry root rot infected plant, (c) rotting of root system, (d) and (e) minute sclerotia inside the exposed root.

Table 1. Comparative symptomology of dry root rot and Fusarium wilt diseases of chickpea.

Plant stage/part	Dry root rot	Fusarium wilt
Seedling stage	• Symptoms generally not seen at this stage, unless favourable conditions occurs	• Young seedlings are killed within 3 weeks of sowing. Whole seedling collapse and retain their dull-green (slightly yellow) colour
Flowering to podding stage	• Symptoms most commonly observed at flowering to pod formation stage • Drooping of petioles and leaflets confined only to the top of infected plant • Stems of affected plants are usually straw coloured, sometime brown	• Most commonly observed at vegetative to flowering stage • Affected plant show typical wilting i.e. drooping of petioles, rachis and leaflets. Lower leaves are chlorotic but most of the other leaves droop while green • Stem retain green colour
Root	• Tap root is generally dark and rotten and most of the lateral and finer roots are dried • Micro-sclerotia can be seen underneath bark • Entire root system is rotten • Affected plant when uprooted breaks easily	• Roots generally healthy and shows no sign of rotting • No microsclerotia formed • Xylem vessels show brown to black discoloration when split vertically • Difficult to uproot easily

tip of the root is easily broken leaving the lower portion of the tap root in the soil when plants are uprooted. Dark minute sclerotial bodies can be seen on the roots exposed and inner side of the bark or when split open at the collar region vertically (Figure 1(d) and (e)).

In chickpea, DRR is easily mistaken for Fusarium wilt as the general symptoms of these two diseases are similar. Hence, we have made an attempt to differentiate DRR from Fusarium wilt based on visible symptoms at seedling, foliage and roots. Comparative symptomology of DRR and Fusarium wilt is provided in Table 1.

5. Variability in pathogen population

The necrotrophic fungus *R. bataticola* exists in an anamorph (sclerotial) stage in soil and on crop residues. Under *in vitro* conditions, the fungus grows rapidly on potato dextrose agar (PDA) and produces brown to grey coloured mycelium that become darker with age. The young hyphae are thin, hyaline, aseptate and dichotomously branched and later produce typical black sclerotia. The fungus may produce abundant aerial mycelium, as high as to touch the cover of the culture plates, or mycelium is found to be completely or partially suppressed (Sharma et al. 2012a). The characteristic features of *R. bataticola* are right angle branching of the mycelium and constriction of the branch near the point of origin. The sclerotia formed are black, smooth, varying from spherical through oblong to irregular shapes (Sharma et al. 2012a).

Much work has been done to elucidate the variability in morphology, physiology, pathogenicity and genotypes of *M. phaseolina* in different hosts [(Dhingra & Sinclair 1973 (castor); Anilkumar & Sastry 1982 (sunflower); Sobti & Sharma 1992 (groundnut); Ratnoo et al. 1997 (cowpea); Atiq et al. 2001 (sunflower); Okwulehie 2001 (groundnut); Suriachandraselvan & Seetharaman 2003 (sunflower); Sharma et al. 2004 (pearl millet, sesame, horsegram and mothbean); Fernandez et al. (2006) (bean); Ndiaye 2007 (cowpea)]. The pathogenic variability in *R. bataticola* is assumed to be due to mutation, hyphal fusion and mitotic recombination. However, not much has been reported on *R. bataticola* of chickpea. Recently, we reported cultural, morphological and molecular variations in 94 isolates of *R. bataticola* collected from various chickpea grown in various agro-ecological zones of India (Sharma et al. 2012a). The isolates varied in cultural and morphological characters like colony colour, growth pattern, growth rate and sclerotial initiation and production. Significant relationships between sclerotial initiations, sclerotial intensity and disease severity between isolates were found. Positive correlation between pathogenic isolates and sclerotial production (Hooda & Grover 1988) was found in mung bean; however, Manici et al. (1992) found no such correlation in sunflower. Than et al. (1991) studied the relationship among *R. bataticola* isolates based on colony fusion type with 37 isolates in chickpea and found existence of more than one isolate at different places. Recently, on the basis of virulence study of 40 isolates of *R. bataticola* on chickpea-susceptible cultivar BG 212, two pathotypes related to the central region in India were reported (Gupta et al. 2012b).

High levels of pathogenic variability and genetic diversity have been observed between *R. bataticola* isolates collected from different geographical origins after characterization with different markers [Random Amplified Polymorphic DNA (RAPD), Internal Transcribed Spacer Restriction Fragment Length Polymorphism (ITS-RFLP), ITS sequencing and Amplified Fragment Length Polymorphism (AFLP)]. The phytogenetic tree based on rDNA-ITS analysis showed that diversity in *R. bataticola* isolates was independent on their geographic origin (Sharma et al. 2012b). There have been recent efforts to describe the population structure of *R. bataticola* with in India (Sharma et al. 2012b). They reported the overall estimated genetic fixation index (G_{st}), of 0.132, when the isolates from different geographical locations, different years of collection and different fields were compared. This high fixation index suggests genetic differentiation amongst all the locations, year of collection and different fields groups. The overall effective migration rate (Nm) across the groups was >1, indicating that gene flow between these populations is very extensive. Jana et al. (2003) developed a single RAPD primer OPA-13 that can be used to differentiate numerous isolates of *M. phaseolina* from soybean, sesame, groundnut, chickpea, cotton, common bean, okra and 13 other hosts.

6. Disease cycle and histopathology

The DRR infection is initiated generally by soil-borne inoculum present in the form of hyphae and sclerotia. The pathogen causes destruction of epidermal cells and penetrates through the roots. The mechanical plugging of the xylem vessels by microsclerotia, toxin production, enzymatic action and mechanical pressure during penetration leads to disease development (Sharma et al. 2004) in addition to direct secretion of macerating enzymes (Bhatt & Vadhera 1997). However, infection of *R. bataticola* on chickpea may also occur through cotyledons during emergence, through small rootlets or through

small wounds on the root surface. The fungus grows inter- and intra-cellularly and invades the cortical cells. It primarily grows inter-cellularly forming thick, short and dark coloured cells that result in the formation of large depressed necrotic lesions. The invaded cortical cells result in disintegrated or severe rotting of the roots (Singh & Mehrotra 1982). Hyphae colonize the vascular system and sclerotial bodies of *R. bataticola* plug the xylem vessels as observed by Singh et al. (1990a). The extent of root necrosis gradually increases with time without any apparent symptoms on the parts of the above ground till flowering and podding growth stages. The schematic representation of DRR disease cycle on chickpea has been illustrated in Figure 2.

7. Climate and other factors influencing dry root rot infection, symptoms expression and severity

Chickpea is largely grown in rain-fed environments and climate variability such as high temperature within the rain-fed ecologies leads to varying intensities of biotic and abiotic stresses. With increasing temperature and more frequent moisture stress, DRR is becoming more intense in typically tropical humid areas. Increasing incidence of DRR at various locations over years suggests a strong influence of rising temperatures on *R. bataticola* Savary et al. (2011). Data collected in India from 2000 to 2010 showed higher incidence of DRR in chickpea varieties that are resistant to Fusarium wilt in years when temperatures exceed 33°C at flowering to podding stages (Pande et al. 2010; Sharma et al. 2010). This is consistent with greenhouse experiments where

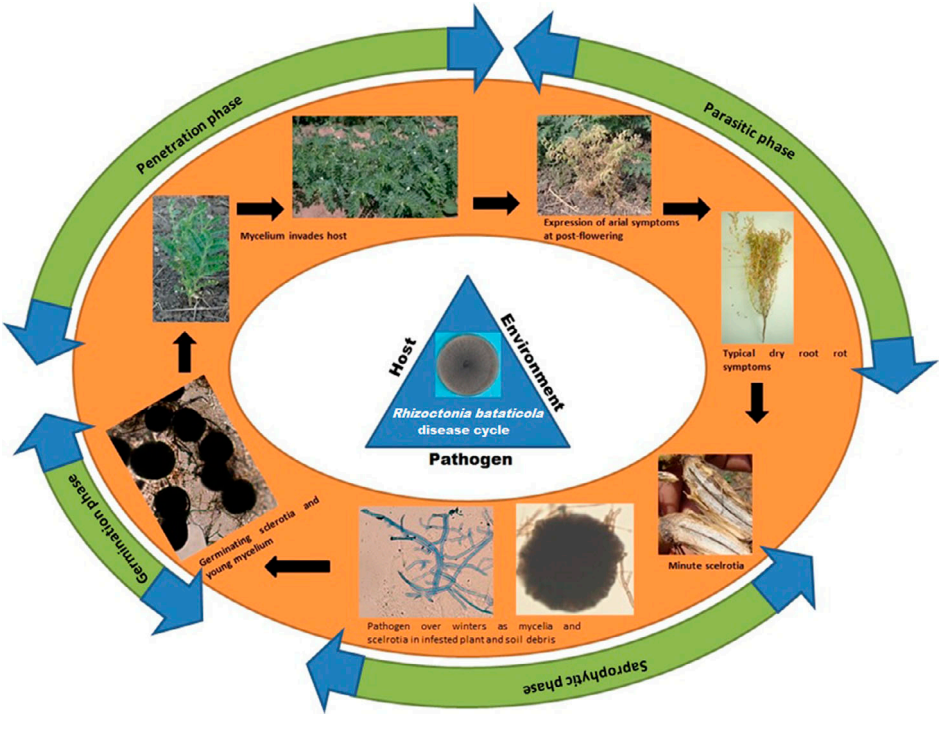

Figure 2. The disease cycle of dry root rot pathogen, *Rhizoctonia bataticola*.

different soil moisture levels and temperatures were manipulated, showing that *R. bataticola*-infected chickpea plants had caused DRR faster at 35°C with soil moisture levels less than or equal to 60% (Figure 3; Sharma & Pande 2013). Further, *R. bataticola* propagules were negatively correlated with high soil moisture and positively with less soil moisture content.

High day temperature above 30°C and dry soil conditions at flowering and podding stages rapidly increase the severity of the DRR disease on chickpea (Gurha et al. 2003). Singh and Sharma (2002) observed that deficit soil moisture favours the severe disease development on pulse crops. Under hot and dry environmental conditions, many economically important crops are predisposed to the infection and colonisation of *R. bataticola* and caused drastic yield losses on chickpea (Thripathi & Sharma 1983), soybean (Pearson et al. 1984) and sunflower (Nawaz 2007). The linear regression used for analysis of the incidence of DRR on commercially grown cultivars of chickpea like JG 74, Annigeri and WR 315 and corresponding crop seasons' weather data clearly showed that rising temperature and corresponding evaporation are critical climate variables for increased DRR frequency and can be considered a good predictor weather variable for the disease (personal observation, data not shown).

Raabe (1985) found that the *R. bataticola* causes severe damage to chickpea in the warmer Salinas Valley in California as the pathogen is becoming more virulent under high temperatures (32°C). *R. bataticola* can grow and produce large amounts of microsclerotia under relatively low water potentials allowing the disease on bean to be recognised as favouring drought (Olaya & Abawi 1996). The survival of sclerotia of *R. bataticola* in black soil was affected by high soil moisture (80%), while the saprophytic activity of pathogen was maximum at low moisture levels (20, 30 and 40%) (Maheswari & Ramakrishnan 1999). The adverse effect of high soil moisture on survival of sclerotia of *R. bataticola* was also reported by Olaya and Abawi (1996) indicating that *R. bataticola* cannot survive for a longer period under anaerobic conditions. Similar to chickpea, in soybean and cowpea, *M. phaseolina* population in the soil was negatively correlated with soil moisture and positively with maximum soil temperature

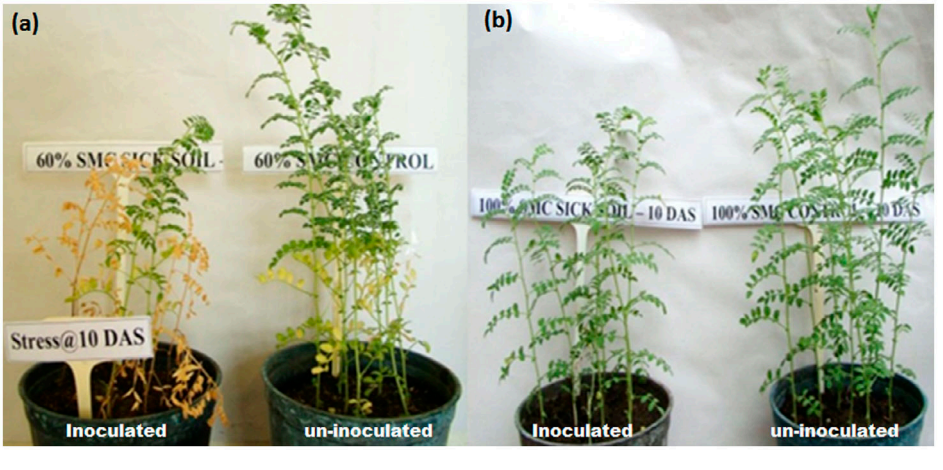

Figure 3. Effect of soil moisture on dry root rot of chickpea in greenhouse (a) inoculated and un-inoculated plants at 60% soil moisture content (SMC) and (b) inoculated and un-inoculated plants at 100% soil moisture content.

(Gupta & Gupta 1986). The DRR incidence of chickpea was maximum (86.6%) in sandy soils as compared with clay soils (61.6%) as observed by Taya et al. (1988) in sick pots at greenhouse. The deficit soil moisture was generally detrimental to both chickpea plant growth and pathogen activity (Bhatti & Kraft 1992).

7.1. Inoculum age and level

The younger culture of *R. bataticola* was found more aggressive causing more infection than the older culture. The mycelial form of inoculum was more effective in increasing the percentage of infected chickpea plants as compared to sclerotial inoculum which showed less effect (Singh, Yadav, et al. 1982). The younger the pathogenic culture, the more aggressive it is in infecting the cowpea seedlings, while the susceptibility of the crop increased with increasing plant age. Similar results were obtained in sunflower by Suriachandraselvan and Seetharaman (2003), cotton (Monga & Raj 1994) and mustard (Rana & Tripathi 1984). Sandhu and Singh (1999) found progressively decreased seed germination and increased disease incidence in cowpea with increased inoculum level of *M. phaseolina* (*R. bataticola*).

7.2. Overwatering and longevity of pathogen

The fungus survives in the soil and infected host crop debris as sclerotia. These sclerotia act as the primary source of inoculum (Francl et al. 1988) and have been found to persist within soil up to 3–6 years (Baird et al. 2003). The sclerotia are black, spherical to oblong structures that are produced in the host tissue and released in to the soil as the infected plant decays. These multi-celled structures allow the persistence of the fungus under adverse conditions such as low soil nutrient levels and temperature above 30°C. Microsclerotial survivability is greatly reduced in wet soils than the dry soils (Suriachandraselvan & Seethraman 2000; Songa & Hillocks 1998; http://www.cals.ncsu.edu/course/pp728/Macrophomina/macrophominia_phaseolinia.HTM).

Cardona (2006) investigated the vertical distribution of the sclerotia on naturally infected soil and found it to be influenced by soil, temperature and humidity. He found the distribution of sclerotia was higher at 0–5 cm than the deeper depths. Population of sclerotia in the soil was higher at the end of post-rainy season compared with population at sowing time. The rainy season crops had a differential effect on population of sclerotia, which was also affected by the cropping systems and rotations. Compared with the single (mono) cropping systems, higher counts of sclerotia were recorded in inter-cropping systems of sorghum or cowpea with legumes. An increase in the population of sclerotia in the soil and the disease can be redistributed by tillage equipment from field to field (Singh et al. 1990b; Songa & Hillocks 1998).

Survival of the fungus has also been reported on seeds (Raabe 1985; Abawi & Corrales 1990). Infected seeds do not germinate or produce seedlings and die soon after emergence. Transmission of the pathogen through the seed has been known as a means of spread of disease to new areas and new countries. Histopathological studies of diseased seed and component plating of artificially inoculated germinating seeds revealed that the pathogen invades the seed coat, cotyledons, plumule and radicle, thus causing pre-emergence rot on chickpea (Singh et al. 1990a) and cowpea (Sandhu & Singh 1998). Post-emergence mortality with dark brown lesions on affected roots was also observed (Singh & Chohan 1973). The viability of infected seeds of bean and okra and healthy seeds showed that seed rot due to fungus in naturally infected plants is more

than the artificially inoculated [(Songa & Hillocks 1998 (bean); Pun et al. 1998 (okra)]. The infection of *M. phaseolina* in various seed components was located by Arya et al. (2004) on soybean.

7.3. Association with other micro-organism

The infected chickpea plants showed maximum association between *F. oxysporum* f. sp. *ciceris* (56%) and *R. bataticola* (17%) from the root isolations of wilted chickpea plants (Srivastava et al. 2002). The combined inoculation of pathogens *F. oxysporum* f. sp. *ciceris* and *R. bataticola* resulted in higher DRR incidence in chickpea compared with single inoculation of the pathogen (personal observation, data not presented). Relatively higher incidence may be attributed to the additive effect of both the pathogens (Patel & Anahosur 2001). It has been repeatedly observed that plants get infected with wilt pathogen initially and later on DRR pathogen under favourable environmental conditions aggravate the combined effect. Our recent investigations suggested a clear difference in temperature-mediated responses of DRR and wilt in susceptible and resistant genotypes. Wilt-resistant genotypes have been found susceptible to DRR at higher temperature (Personal observation).

8. Managing dry root rot: search for answers

It is not possible to control the weather and also large shift in cultivation practices to grow chickpea on residual soil moisture particularly in semi-arid tropics is not probable. Therefore, we must search for other solutions for managing DRR. The current status of various management practices useful for DRR is described in the following section.

8.1. Host plant resistance

For chickpea-DRR pathosystem, very limited information is available on the defence enzymes activated during host × pathogen × environment interactions. Singh, Prabhjot, et al. (1982) found that there is a little difference in amounts of phenols in the extracts of seed coat and naked seed (cotyledons and embryos) of susceptible (L-550) and resistant varieties (BG 203 and Hare cholle-1) against *R. bataticola* infection in chickpea. The studies showed that even though extracts contained phenols, growth of the pathogen was not inhibited, which indicated that perhaps factors other than total phenols play a role in the resistance of chickpea to *R. bataticola*. Possible involvement of phytoalexin such as glyceolin and peroxidase and polyphenoloxidase system and antigenic substances in susceptibility or resistance to *M. phaseloina* in soybean has been reported (Lygin et al. 2010; Gupta et al. 2012a).

We know that the development of resistant cultivars is the best option for the management of any diseases as it is economical and eco-friendly. Several screening techniques have been developed for DRR at ICRISAT such as “paper towel screening technique and sick pot technique” (Nene et al. 1981; Pande et al. 2012). In paper towel screening technique, roots of eight-day-old seedling of the test chickpea cultivars were dipped in *R. bataticola* inoculum for 2–3 min and placed on a blotter paper towel. The paper towels having inoculated seedlings were moistened by sprinkling sterile distilled water and were kept in an incubator at 35°C for disease development. In each paper towel, 8–10 seedlings were kept with three replications. Seven days after inoculation, disease was observed and the severity was recorded on a 1–9 scale (Pande et al. 2012).

In "sick pot technique", soil was made sick by preparing the fungus–soil mixture followed by sowing of susceptible cultivar and checking the disease severity. Once the disease severity in susceptible cultivar reached 9 on a 1–9 rating scale, screening for the test material was done. Detailed step-wise procedure for these techniques has been described by Pande et al. (2012).

Search for specific resistance to DRR in chickpea has been unsuccessful so far, as none of the lines showed consistent resistant reactions to DRR. Several germplasm and breeding lines were evaluated using these two screening techniques by various researchers (Nene et al. 1981; Gurha et al. 2003; Ashraf et al. 2005). Jayalakshmi et al. (2008) found none of the chickpea cultivars highly resistant to chickpea; however, four genotypes (GCP- 101, GBM-2, GBM-6 and ICCV-10) were found tolerant. Iftikhar and Ilyas (2000) screened 108 chickpea germplasm cultivars to DRR and found only one chickpea line (ICCV 97112) resistant. Few resistant sources for DRR have also been reported by Gangwar et al. (2002); Prajapati et al. (2003); Pande et al. (2006); Gupta et al. (2012b); and Khan et al. (2013). Baker and Ahmed (1991) from Bangladesh reported few resistant sources to DRR/wilt complex. Few germplasm lines such as ICCV 05530, ICCV 08305, ICCV 05529, ICCV 05532, ICCV 07117 and ICCV 07112 had shown a moderate level of resistance to DRR with a disease score ≤ 4 on a 1–9 scale (Mamta Sharma, not published). Resistance identified so far in improved chickpea germplasm and breeding lines as well as wild accessions and its relatives under field and greenhouse conditions needs to be reconfirmed under epidemiologically sound, repeatable resistance screening technique with the existing variability in *R. bataticola* isolates.

8.2. *Inheritance of resistance*

The knowledge about the variation in host and pathogen is a pre-requisite for a successful breeding programme (Porta-Puglia et al. 1996). There is only a single report available on the inheritance of resistance to DRR, where crosses of Parental F_1 and F_2 populations of two resistant (H208 and K850) and two susceptible parents (C104 and P165) used revealed that the resistance to DRR is monogenic, showing a 3R:1S segregation ratio in the F_2 population (Rao & Haware 1987). It was observed that even the resistant parents developed symptoms of the disease if the plants were grown for a longer period in infected soil. Studies on host plant resistance and inheritance of resistance to DRR suggested that the screening techniques for this disease need further refinement, and sources of resistance need further confirmation under controlled environment and field. This puts the breeding for resistance in chickpea to DRR in a state of uncertainty, particularly now when we are experiencing uncertainty in weather. Further, there is no report of any molecular markers linked to the DRR resistance gene identified so far.

8.3. *Cultural control*

With the help of cultural practices, population levels of pathogen can be lowered and soil moisture can be retained to some extent, which in turn can culminate in a reduced incidence of DRR. Manipulation in date of sowing is a good option to escape the hot weather conditions at the time of crop maturity (Singh et al. 1990b; Gurha et al. 2003). Timely or early sowing of early maturing cultivars with timely irrigation can avoid high temperature during maturity, thereby reducing DRR. Study conducted by Patel and Anahosur (2001) with two chickpea cultivars ICC 4951 (susceptible) and Bheema (resistant), three sowing times and four available soil moistures in sick pots revealed that

both cultivars showed maximum disease incidence at the lowest soil moisture level, although incidence was higher in ICC 4951 than Bheema.

In the absence of high resistant cultivars, crop rotation with non-host crops is helpful in reducing the population of *M. phaseolina* sclerotia in the soil (Singh et al. 1990b). Deep ploughing and removal of infected host debris from the soil may reduce the further multiplication of sclerotia in its saprophytic phase and also reduce the inoculum levels, resulting in reduced disease severity. Research in the Pacific Northwest of USA showed that tillage and residue management can markedly influence the severity of root rot in pea (Kraft et al. 1988). The no-tillage had fewer sclerotial populations than the conventional tillage. The no-tillage may provide a less conducive environment to support the *R. bataticola* population (Mengistu et al. 2009). Tillage practices which reduce soil moisture stress may reduce disease potential. Further, maintaining good soil moisture with irrigation from planting to pod fill may reduce disease potential due to *R. bataticola* (http://www.cals.ncsu.edu/course/pp728/Macrophomina/macrophominia_phaseolinia.HTM).

8.4. Biological control

Seed treatment with biocontrol agents like *Trichoderma viride* has shown some benefits in managing the DRR of chickpea (Singh et al. 1998; Indira & Thiribuvanamala 2002; Gurha et al. 2003). Antagonistic effect of four bacterial and six actinomycetes isolates on *R. bataticola* of chickpea was reported by Singh and Mehrotra (1980). Biological control of *R. bataticola* of black gram was most effective by the integration of antagonist (*Trichoderma virens*) and organic amendments (FYM) and significantly improved the plant growth, grain yield, dry matter production and reduced disease incidence than application of *T. virens* alone (Christopher et al. 2007). A combination of biocontrol agents of *T. viride*, *Pseudomonas fluorescens* and *Bacillus subtilis* improved the management of *R. bataticola* in mung bean (Thilagavathi et al. 2007). In the case of chickpea, there is a need to further strengthen the investigations on this aspect based on thorough understanding of the biology of the pathogen and host × plant × environment interactions.

8.5. Chemical control

Seed treatment by fungicides is effective to some extent in reducing losses caused by *R. bataticola* in crops which are particularly vulnerable at the seedling stage. Fungicide seed treatment with carbendazim and thiophanate methyl and vitavax reduced the DRR of chickpea significantly over untreated check (Taya et al. 1990; Bhardwaj 1995; Singh & Sindhan 1998; Rathore & Rathore 1999; Sharma & Gupta 2004). The combined use of host resistance with fungicide treatment resulted in better seedling emergence and delayed the onset of root rots. Treating the seeds with captan or thiram is also helpful in reducing the disease (Gurha et al. 2003). Practice of seed treatment with fungicide (bavistin and thiram) in farmers' field reduced the DRR incidence in central and southern parts of India (Ghosh et al. 2013). Taya et al. (1990) in greenhouse tests found the best control of *R. bataticola* was given by carbendazim alone or in combination with thiram as seed treatment, pre-sowing, soil–drench and seed treatment + drenching after sowing. Vijay-Mohan et al. (2006) reported management of DRR using carbendazim (0.2%) and etaconazole (0.1%) as seed treatment, soil drenching and seed treatment plus soil drenching.

9. Future outlook

The current understanding of the chickpea–dry root rot–*R. bataticola* interaction has provided significant information and paved a way for working on this emerging disease. We now have evidences that climate has a significant role in DRR emergence as frequent high temperature and low moisture conditions lead to increased incidence of DRR in chickpea. More studies are needed to determine the temporal and spatial distributions of the pathogen. Proper and rapid characterisation of symptoms of the disease for timely scouting is needed. Pathogenic characterisation based on morphology and genetic makeup will provide ample opportunities to understand the population genetics. Additionally, information on biology and epidemiology of DRR will further strengthen the screening procedures required to identify resistant sources. This in turn will assist breeders in optimising breeding strategies that will enable long-term resistance over broader geographical areas. Till date, no study on the defence mechanism of *R. bataticola* in chickpea for DRR has been done. The pathogenic and genetic variability has been described earlier; however, these are some gaps to be filled.

Despite the extensive investigations in other hosts, the infection process of *R. bataticola* on chickpea has not been studied. Also, very little is known about the resistance mechanisms of chickpea against *R. bataticola*. Understanding the genetics, behaviour of host and pathogen in the process of disease development and host–pathogen relationship are crucial for reliable breeding programmes for disease resistance. Genetics of resistance against *R. bataticola* have not been clearly demonstrated and controversies are found in the findings of various researchers. There is also a need to map the genetic profile of the few chickpea accessions that have shown traces of resistance to DRR. This will identify the putative molecular marker to identify the gene (s) responsible for the resistance. Further, knowledge of infection process and host defence mechanisms will help in devising effective management strategies to DRR.

To the best of our knowledge, this is the first review on DRR of chickpea which describes the overall status on biology, mode of infection, epidemiology and available information on resistant sources to the disease.

Acknowledgements

The authors are thankful to the Department of Science and Technology, Climate Change Division for partial funding support provided to conduct various research activities reported in this review.

Disclosure statement

No potential conflict of interest was reported by the authors.

References

Abawi GS, Corrales MAP. 1990. Seed transmission and effect of fungicide and seed treatments against *Macrophomina phaseolina* in dry edible beans. Turrialba. 40:334–339.

Anilkumar TB, Sastry MNL. 1982. Nutritional and physiological variation among isolates of *Rhizoctonia bataticola* from sunflower. Zbl Mikrobiol. 137:8–232.

Arya VK, Vishunavat K, Himanshu N. 2004. Detection, location and transmission of seedborne inoculum of *Macrophomina phaseolina* in charcoal rot in soybean. J Mycol Plant Pathol. 34:233–236.

Ashraf MS, Khan TA, Hasan S. 2005. Reaction of chickpea varieties to *Macrophomina phaseolina* and their effect on peroxidase activity. Pakistan J Bot. 37:761–767.

Atiq M, Shabeer A, Ahmed I. 2001. Pathogenic and cultural variation in *Macrophomina phaseolina*, the cause of charcoal rot in sunflower. Sarhad J Agric. 2:253–255.
Baird RE, Watson CE, Scruggs M. 2003. Relative longevity of *Macrophomina phaseolina* and associated mycobiota on residual soybean roots in soil. Plant Dis. 87:563–566.
Baker MA, Ahmed F. 1991. Additional sources of resistance to wilt/root rots of chickpea in Bangladesh. Int Chick News. 25:28–29.
Bhardwaj CL. 1995. Charcoal rot incidence and efficacy of seed treatment with carbendazim in frechbean relative to variety and environment. Indian J Mycol Plant Pathol. 25:246–249.
Bhatt J, Vadhera I. 1997. Histopathological studies on cohabitation of *Pratylenchus thornei* and *Rhizoctonia bataticola* on chickpea (*Cicer arietinum* L.). Adv Plant Sci. 10:33–37.
Bhatti MA, Kraft JM. 1992. Influence of soil moisture on root rot and wilt of chickpea. Plant Dis. 76:1259–1262.
Cardona R. 2006. Vertical distribution of sclerotia of *Macrophomina phaseolina* in a natural infested soil in Portuguesa state. Rev Fac Agron (LUZ). 23:283–290.
Christopher DJ, Raj TS, Udhayakumar R. 2007. Biological control of dry root rot (*Macrophomina phaseolina*) of urdbean by the integration of antagonist and organic amendments. J Plant Dis Sci. 2:153–156.
Dhingra OD, Sinclair JB. 1973. Variation among isolates of *Macrophomina phaseolina* (Tassi.) Goid. causing root rot/ charcoal rot disease of castor. Indian J Plant Pathol. 6:97–98.
FAOSTAT. 2012. Agriculture. Available from: http://faostat.fao.org.
Ferguson BJ, Indrasumunar A, Hayashi S, Lin MH, Lin YH, Reid DE, Gresshoff PM. 2010. Molecular analysis of legume nodule development and autoregulation. J Integr Plant Biol. 52:61–76.
Fernandez RB, De Santiago A, Delgado SH, Perz NM. 2006. Characterization of Mexican and non- Mexican isolates of *Macrophomina phaseolina* based on morphological characteristics, pathogenicity on bean seeds and endoglucanase gene. J Plant Pathol. 88:53–60.
Francl LJ, Willie TD, Rosembrok SM. 1988. Influence of crop rotation on population density of *Macrophomina phaseolina* in soil infested with *Heterodera glycines*. Plant Dis. 72:760–764.
Gangwar RK, Prajapati RK, Srivastava SSL, Kumar K. 2002. Resistance in chickpea germplasms against the dry root rot. Ann Plant Protect Sci. 10:393–394.
Ghosh R, Sharma M, Telangre R, Pande S. 2013. Occurrence and distribution of chickpea diseases in central and southern parts of India. Am J Plant Sci. 4:940–944.
Gupta GK, Sharma SK, Ramteke R. 2012a. Biology, epidemiology and management of the pathogenic fungus *Macrophomina phaseolina* (Tassi) Goid with special reference to charcoal rot of soybean (*Glycine max* (L.) Merrill). J Phytopathol. 160:167–180.
Gupta JP, Gupta GK. 1986. Effect of tillage and mulching on soil environment under arid condition. Soil & Tillage Res. 7:233–240.
Gupta O, Rathi M, Mishra M. 2012b. Screening for resistance against *Rhizoctonia bataticola* causing dry root-rot in chickpea. J Food Legumes. 25:139–141.
Gurha SN, Singh G, Sharma YR. 2003. Diseases of chickpea and their management. In: Ali M, Kumar S, Singh NB, editors. Chickpea research in India. Lucknow: Army Printing Press; p. 195–227.
Hwang SF, Gossen BD, Chang KF, Turnbull GD, Howard RJ, Blade SF. 2003. Etiology, impact and control of rhizoctonia seedling blight and root rot of chickpea on the Canadian prairies. Canadian J Plant Sci. 83:959–967.
Holliday P, Punithalingam E. 1970. *Macrophomina phaseolina*. CMI Description of pathogenic fungi and bacteria. No. 275. Farnham Royal: Commonwealth Agricultural Bureaux.
Hooda I, Grover RK. 1988. Effect of age, quantity of inoculum and isolates of *Macrophomina phaseolina* on the pathogenesis of mungbean and its control by chemicals. Indian Phytopathol. 41:107–117.
Iftikhar K, Ilyas MB. 2000. Screening of chickpea germplasm against dry root rot disease (*Macrophomina phaseolina*) in pots/glass house. Pakistan J Phytopathol. 12:66–70.
Indira N, Thiribuvanamala G. 2002. Antagonism of Trichoderma spp. Against Macrophomina phaseolina causing root rot of black gram. Plant Dis Res. 17:142–144.
Jana T, Sharma TR, Prasad RD, Arora DK. 2003. Molecular characterization of *Macrophomina phaseolina* and *Fusarium* species by a single primer RAPD technique. Microbiol Res. 158:249–257.

Jayalakshmi SK, Usharani S, Benagi MDD. 2008. Sources of resistance to dry root rot of chickpea caused by *Rhizoctonia bataticola*. Agric Sci Dig. 28:147–148.
Kaiser WJ, Danesh D, Okhovat M, Mossahebi H. 1968. Diseases of pulse crops (edible legumes) in Iran. Plant Dis Rep. 52:687–691.
Khan RA, Bhat TA, Kumar K. 2013. Screening of chickpea (*Cicer arietinum* L.) germplasm lines against dry root rot caused by *Rhizoctonia bataticola* (taub.) Butler. Asian J Pharm Clin Res. 6:211–212.
Kraft JM, Harware MP, Hussein MM. 1988. Root rot and wilt diseases of food legumes. In: Summerfield RJ, editor. World crops: cool season food legumes proceedings of the International Food Legume Research Conference on pea, lentil, fababean and chickpea; 1986 Jul 6–11; SPOKANE, Washington DC (USA). Dordrecht: Kluwer Academic Publishers; p. 565–575.
Lygin AV, Hill CB, Zernova OV, Crull L, Widholm JM, Hartman GL, Lozovaya VV. 2010. Response of soybean pathogens to glyceollin. Phytopathology. 100:897–903.
Maheswari U, Ramakrishnan G. 1999. Factors influencing the competitive saprophytic ability of *Macrophomina phaseolina* in groundnut. Madras Agric J. 86:552–553.
Manici LM, Cerato C, Caputo F. 1992. Pathogenic and biologic variability of *Macrophomina phaseolina* (Tassi.) Goid. isolates in different areas of sunflower cultivation in Italy. Proc Sunflower Conf. 13:779–784.
Mengistu A, Reddy KN, Zablotowicz RM. 2009. Propagule densities of *Macrophomina phaseolina* in soybean tissue and soil as affected by tillage cover crop and herbicide. Available from: http://www//planthealthprogress.
Mitra M. 1931. Report of the imperial mycologist. Sci Rep of the Agric Res Inst. 1929–1930:58–71.
Monga D, Raj S. 1994. Cultural and pathogenic variations in the isolates of *Rhizoctonia* species causing root rot in cotton. Indian Phytopathol. 47:403–408.
Nawaz KS. 2007. *Macrophomina phaseolina* as causal agent for charcoal rot of sunflower. Mycopathology. 5:111–118.
Ndiaye M. 2007. Ecology and management of charcoal rot (*Macrophomina phaseolina*) on cowpea in the Sahel. Ecology and management of charcoal rot (*Macrophomina phaseolina*) on cowpea in the Sahel. The Netherlands: Wageningen University; p. 109.
Nene YL, Haware MP, Reddy MV. 1981. Chickpea disease resistance-screening technique. ICRISAT Inf Bull. 28:28–29.
Nene YL, Shelia VK, Sharma SB. 1996. A world list of chickpea and pigeonpea pathogens. 5th ed. Hyderabad: International Crops Research Institute for Semi-Arid Tropics; p. 27.
Okwulehie IC. 2001. Physiological studies in groundnuts (*Arachis hypogea* L.) infected with *Macrophomina phaseolina* (Maub.) Ashby. Int J Tropical Plant Dis. 19:25–37.
Olaya G, Abawi GS. 1996. Effect of water potential on mycelial growth and on production and germination of sclerotia of *Macrophomina phaseolina*. Plant Dis. 80:1347–1350.
Pande S, Desai S, Sharma M. 2010. Impact of climate change on rainfed crop diseases: current status and future research needs. Lead Papers. National Symposium on Climate Change and Rainfed Agriculture, 2010 Feb. 18–20. Hyderabad: Indian Society of Dryland Agriculture, Central Research Institute for Dryland Agriculture; p. 55–59.
Pande S, Kishore GK, Upadhyaya HD, Rao JN. 2006. Identification of sources of multiple disease resistance in mini-core collection of chickpea. Plant Dis. 90:1214–1218.
Pande S, Sharma M, Avuthu N, Telangre R. 2012. High throughput phenotyping of chickpea diseases: stepwise identification of host plant resistance. Inf Bull No 92. Technical Report. Patancheru: International Crops Research Institute for Semi-Arid Tropics.
Patel ST, Anahosur KH. 2001. Influence of sowing time, soil moisture and pathogens on chickpea wilt and dry root rot incidence. Karnataka J Agric Sci. 14:833–835.
Pearson CAS, Schwenk FW, Crowe FJ. 1984. Colonization of soybean roots by *Macrophomina phaseolina*. Plant Dis. 68:1086–1088.
Porta-Puglia A, Crino P, Moscow C. 1996. Variability in virulence to chickpea of an Italian population of *Ascochyta rabiei*. Plant Dis. 80:39–41.
Prajapati RK, Gangwar RK, Srivastava SSL. 2003. Resistant sources of chickpea against dry root rot. Farm Sci J. 12:86.
Pun KB, Doraiswamy S, Valluvaparidasan V. 1998. Studies on seed-borne nature of *Macrophomina phaseolina* in okra. Plant Dis Res. 13:162–164.
Raabe RT. 1985. Association of soil particles with seeds and three pathogens of chickpea in California. Indian Phytopathol. 69:238–239.

Rana RS, Tripathi NN. 1984. Influence of inoculum, host and soil environment on the incidence of collar rot of Indian mustard. Indian J Mycol Plant Pathol. 14:23–28.
Rao APK, Haware MP. 1987. Inheritance of dry root rot (*Rhizoctonia bataticola*) resistance in chickpea (*Cicer arietinum* L.). Plant Breeding. 98:349–352.
Rathore BS, Rathore RS. 1999. Effect of seed dressers on *Macrophomina* root rot of mothbean. J Mycol Plant Pathol. 29:389–392.
Ratnoo RS, Jain KL, Bhatnagar MK. 1997. Variations in *Macrophomina phaseolina* isolates of Ash-gray stem blight of cowpea. J Mycol Plant Pathol. 27:91–92.
Sandhu A, Singh RD. 1998. Role of seed borne inoculum in development of charcoal rot of cowpea. J Mycol Plant Pathol. 28:193–195.
Sandhu A, Singh RD. 1999. Factors influencing susceptibility of cowpea to *Macrophomina phaseolina*. J Mycol Plant Pathol. 29:421–424.
Savary S, Nelson A, Sparks AH, Willocquet L, Duveiller E, Mahuku G, Forbes G, Garrett KA, Hodson D, Padgham J, et al. 2011. International agricultural research tackling the effects of global and climate changes on plant diseases in the developing world. Plant Dis. 95:1204–1216.
Sharma M, Ghosh R, Ramesh RK, Upala NM, Chamarthi S, Varshney R, Pande S. 2012a. Molecular and morphological diversity in Rhizoctonia bataticola isolates causing dry root rot in chickpea (*Cicer arietinum* L.) in India. Afr J Biotechnol. 11:8948–8959.
Sharma M, Ghosh R, Sharma TR, Pande S. 2012b. Intra population diversity in *Rhizocotonia bataticola* causing dry root rot of chickpea (*Cicer arietinum* L.) in India. Afr J Microbiol Res. 6:6653–6660.
Sharma M, Mangala UN, Krishnamurthy M, Vadez V, Pande S. 2010. Drought and dry root of chickpea (Abstract). 5th International Food Legumes Research Conference (IFLRC V), 2010 & 7th European Conference on Grain Legumes (AEP VII); 2010 April 26–30; Antlya: Akdeniz University & Ministry of Agriculture and Rural Affairs under Auspices of International Steering Committee of IFLRC & The European Association for Grain Legume Research (AEP).
Sharma M, Pande S. 2013. Unravelling effects of temperature and soil moisture stress response on development of dry root rot [*Rhizoctonia bataticola* (Taub.)] butler in chickpea. Am J Plant Sci. 4:584–589.
Sharma OP, Gupta RBL. 2004. Fungicides in the control of chickpea dry root rot caused by *Rhizoctonia bataticola*. J Mycol Plant Pathol. 34:321–322.
Sharma YK, Gaur RB, Bisnoi HR. 2004. Cultural, morphological and physiological variability in *Macrophomina phaseolina*. J Mycol Plant Pathol. 34:532–534.
Singh G, Sharma YR. 2002. Fungal diseases of pulses. In: Gupta VK, Paul YS, editors. Diseases of field crops. New Delhi: Indus publishing; p. 155–192.
Singh I, Chohan JS. 1973. Seed mycoflora of guar (*Cyamopsis tetragonoloba* L.) and their effect on germination and growth of seedlings. Ind J Mycol Plant Pathol. 3:86–92.
Singh PJ, Mehrotra RS. 1980. The influence of cultivar and temperature on carbohydrate and amino acid exudation from gram seeds and on pre-emergence damping-off by *Rhizoctonia bataticola*. Plant Soil. 55:261–268.
Singh PJ, Mehrotra RS. 1982. Penetration and invasion of gram roots by *Rhizoctonia bataticola*. Indian Phytopathol. 35:336–338.
Singh PJ, Prabhjot N, Mehrotra RS. 1982. Relation between phenol and *Rhizoctonia bataticola* infection in gram. Ind J Mycol Plant Pathol. 12:46–47.
Singh PJ, Yadav JP, Mehrotra RS. 1982. Comparison of mycelial and sclerotial inoculum of *Rhizoctonia bataticola* affecting damping-off and root rot of gram. Ind J Mycol Plant Pathol. 12:74–75.
Singh R, Sindhan GS. 1998. Effect of fungicides on the incidence of dry root rot and biochemical status of chickpea plants. Plant Dis Res. 13:14–17.
Singh SK, Nene YL, Reddy MV. 1990a. Some histopathological observations of chickpea roots infected by *Rhizoctonia bataticola*. Int Chick News. 23:24–25.
Singh SK, Nene YL, Reddy MV. 1990b. Influence of cropping systems on *Macrophomina phaseolina* populations in soil. Plant Dis. 74:812–814.
Singh R, Sindhan GS, Parashar RD, Hooda I. 1998. Application of antagonists in relation to dry root and biochemical status of chickpea plants. Plant Dis Res. 13:35–37.

Sobti AK, Sharma LC. 1992. Cultural and pathogenic variations in isolates of *Rhizoctonia bataticola* from groundnut. Indian Phytopathol. 41:149–151.
Songa W, Hillocks RJ. 1998. Survival of *Macrophomina phaseolina* in bean seed and crop residue. Int J Pest Manage. 44:109–114.
Srivastava A, Singh SN, Agarwal SC. 2002. Prevalence of chickpea wilt and dry root disease in Vindhyan plateau zone of Madhya Pradesh. Ind J Pulses Res. 15:192.
Suriachandraselvan M, Seetharaman K. 2003. Effect of culture media on growth and sclerotial production of different isolates of *Macrophomina phaseolina* infecting sunflower. J Mycol Plant Pathol. 33:226–229.
Suriachandraselvan M, Seethraman K. 2000. Survival of *Macrophomina phaseolina*, the causal agent of charcoal rot of sunflower in soil, seed and plant debris. J Mycol Plant Pathol. 30:402–405.
Taya RS, Tripathi MN, Pawar MS. 1988. Influence of soil type, soil moisture and fertilizers on the severity of chickpea dry root rot caused by *Rhizoctonia bataticola* (Taub.) Butler. Ind J Mycol Plant Pathol. 19:133–136.
Taya RS, Tripathi NN, Panwar MS. 1990. Influence of texture and nutritional status of soil on the efficacy of fungicides for the control of dry root-rot of chickpea. Indian J Mycol Plant Pathol. 20:14–20.
Than H, Thein MM, Myint SS. 1991. Relationship among *Rhizoctonia bataticola* isolates in rice based cropping systems based on colony fusion types. Int Chick News. 25:29–31.
Thilagavathi R, Saravanakumar D, Ragupathi N, Samiyappan R. 2007. A combination of biocontrol agents improves the management of dry root rot (*Macrophomina phaseolina*) in greengram. Phytopathol Mediterr. 46:157–167.
Thripathi NN, Sharma BK. 1983. Incidence of chickpea dry root rot (*Rhizoctonia bataticola*) in Southern Haryana. Int Chick News. 8:22–23.
Varshney RK, Song C, Saxena RK, Azam S, Yu S, Sharpe AG, Cannon S, Baek J, Rosen BD, Taran B, et al. 2013. Draft genome sequence of chickpea (*Cicer arietinum*) provides a resource for trait improvement. Nat Biotechnol. 31:240–246.
Vijay-Mohan SM, Prasad M, Barnwal MK, Kudada N. 2006. Fungicidal management of dry root rot disease and yield of chickpea. J Appl Biol. 16:42–44.
Westerlund FV, Jr-Cambell RN, Kimble KA. 1974. Fungal root rots and wilt of chickpea in California. Phytopathology. 664:432–436.