Diseases of Chickpea

Fernando S. Rocha, Mamta Sharma, Avijit Tarafdar, Weidong Chen, Daiana M. Q. Azevedo, Pablo Castillo, Cândido A. Costa, and Devashish R. Chobe

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F. S. Rocha (✉)
Universidade Federal de Minas Gerais-UFMG, Montes Claros, Brazil
e-mail: rochafs@ufmg.br

M. Sharma
Principle Scientist and Theme Leader, Integrated Crop Management, International Crops Research Institute for the Semi-Arid Tropics-ICRISAT, Hyderabad, India
e-mail: mamta.sharma@cgiar.org

A. Tarafdar
Consultant, Legumes Pathology, Integrated Crop Management, ICRISAT, Patancheru, Hyderabad, India

W. Chen
USDA Agricultural Research Service, Department of Plant Pathology, Washington State University, Pullman, WA, USA
e-mail: w-chen@wsu.edu

D. M. Q. Azevedo
Departamento de Fitopatologia, Universidade Federal de Viçosa, Viçoca, MG, Brazil

P. Castillo
Institute for Sustainable Agriculture (IAS), Spanish National Research Council (CSIC), Madrid, Spain
e-mail: pcastillo@ias.csic.es

C. A. Costa
Universidade Federal de Minas Gerais-UFMG, campus Montes Claros, Montes Claros, MG, Brazil
e-mail: candido-costa@ufmg.br

D. R. Chobe
Legumes Pathology Division, Integrated Crop Management, ICRISAT, Patancheru, Hyderabad, India
e-mail: D.Chobe@cgiar.org

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Abstract

Chickpeas are one of the most economically important legumes and a rich source of carbohydrates, proteins, vitamins, minerals, and fiber. Chickpea is grown mainly in the tropics, in arid and semiarid countries in Asia and Africa, but it is also present in Europe, Oceania, North America, and Latin America. The varieties available for cultivation vary in productivity according to edaphoclimatic conditions and the incidence of diseases. The incidence and severity of diseases in chickpeas vary in relation to the planting time, cultivated variety, and the causal agent. Among the main diseases with major economic importance causing losses in productivity are those caused by soil-borne fungal and nematode pathogens, and diseases of the aerial plant portion caused by viruses and fungi. Integrated management and preventive measures such as pathogen identification, field selection, seed quality, use of fungicides, and crop rotation are effective practices for managing chickpea diseases. This chapter will address the main characteristics of the economically important diseases affecting chickpeas in several prominent and expanding production countries.

Keywords

Fusarium · *Pythium* · *Sclerotinia* · *Sclerotium* · *Rhizoctonia* · *Meloidogyne* · *Pratylenchus* · *Botrytis* · *Ascochyta* · Viruses

1 Introduction

Chickpeas (*Cicer arietinum* L.) is an annual herbaceous plant with habitually undetermined growth belonging to the Fabaceae family. This crop is part of the group called pulses, which includes legumes with dry grains such as lentils, peas, and beans. Chickpea is also called Bengal gram, Egyptian pea, and garbanzo or garbanzo bean. Chickpea grains are known for their high nutritional value, in particular protein, for human consumption. The crop originates in the Middle East region, more precisely in Southeastern Turkey and close to Syria (Ladizinsky and Adler 1976).

Chickpeas are propagated exclusively by seeds, with a crop cycle ranging from 95–110 days across five continents. Two main varieties of chickpeas are available for cultivation. The "Kabuli" type has white, common flowers; large, round, white to beige seeds; and it is customarily planted in the USA. The "Desi" type has pink or purple flowers with irregular shapes; small seeds with a color ranging from beige to yellow, green, or brown to black, and it is often used in India and the Middle East.

The largest planting and production areas are concentrated in Asia. India is the world's largest producer and consumer of chickpeas. In the last decades, there has been an expansion of planted areas in Eastern Europe, to meet the ever-increasing demands, resulting in an increased need for research to identify pathogens and adapt the culture to produce this grain in other potential countries. Although chickpeas are adapted to climates under low rainfall, it is successfully cultivated in tropical and subtropical countries during winter, spring, or summer.

Various pathogens such as fungi, bacteria, nematodes, and viruses have been reported to be associated with chickpeas, causing damage at different stages of the crop cycle. Some diseases can limit cultivation in the planting area, causing yield losses of up to 100% depending on the pathogen, period of infection, cultivated variety, and climatic conditions. The main diseases of chickpeas worldwide are Fusarium wilt, black root rot, wet root rot, dry root rot, Pythium root and seed rot, collar rot, Ascochyta blight, Botrytis gray mold, stunt (*Bean leaf roll virus*), mosaic (*Alfalfa mosaic virus*), chlorotic dwarf (*Chickpea chlorotic dwarf virus*), proliferation (*Cucumber mosaic virus*), narrow leaf (*Beet western yellow virus*), and disease caused by root-knot nematodes, root lesion nematode, cyst nematode, and reniform nematode. Bacterial spot caused by *Xanthomonas campestre* pv. *cassiae* is a low-incidence disease in producing areas, due to conditions of low humidity required for cultivating chickpeas. Sections of this chapter will address the main diseases of great economic importance caused by fungi, nematodes, and viruses, with information on geographic distribution and impact, symptoms, biology of the pathogen, epidemiology, and control measures for the management of these diseases in chickpeas.

2 Fungal and Fungus-Like Diseases

2.1 Ascochyta Blight (*Ascochyta rabiei* [Pass.] Labr.)

Geographic Occurrence and Impact. The fungal pathogen *Ascochyta rabiei* causes Ascochyta blight of chickpea, which is a major disease in Southern Europe, West Asia, and Northern Africa. In Pakistan and Northern India, the disease usually develops in February and March when the crop canopy is dense and temperature is favorable for its development. In Southern Europe, West Asia, and Northern Africa, such conditions frequently prevail in March, April, and May. Blight symptoms on chickpeas growing in the Mediterranean region can be found in November and December, when the weather is wet, cool winter, and with windy conditions (Nene et al. 1981; Pande et al. 2010a). Ascochyta blight of chickpea has been reported in 35 countries across six continents and occurs on chickpea essentially everywhere the crop is grown. All above ground parts of the plant are affected by this disease (Bayaa and Chen 2011). The symptoms are most severe in areas where cool, cloudy, and humid weather occurs during the crop season and can cause complete yield loss. Ascochyta blight mostly develops during flowering and pod development. The disease can cause grain yield and quality losses up to 100%.

Symptoms/Signs. The disease is frequently observed during the flowering and podding time of chickpea as a patch of blighted plants in the field. Symptoms can develop on all above-ground parts of the plant. However, the disease can also be observed at a very early stage of crop growth. Emerging seedlings develop dark brown lesions at the base of the stem when the pathogen is seed borne and environmental conditions at the time of germination are favorable for disease development. Affected seedlings may also show damping-Geographic symptoms. When the primary inoculum is airborne conidia or ascospores, the main symptoms are numerous small water-soaked necrotic spots on the tender leaves of almost all branches. These spots enlarge rapidly and merge when conditions are favorable for disease development. Pycnidia can be easily observed in leaf spots when examined with a microscope. Entire plant dries up suddenly when foliar infection is severe. Round leaf spots with concentric rings brown margins and grey centers are the typical symptom of Ascochyta blight (Fig. 1). Lesion size varies from 3–4 cm long on stems and often girdle the affected portion resulting in stems and petioles usually breaking. There may be a chance of development of several lesions on a single pod if infection occurs in the early stages of pod development. On pods, lesions are similar to those found on leaves (Fig. 1), and often are round with pycnidia in concentric rings. Pods infected early may not develop any seed. Seeds can be infected when conditions are favorable for disease development during seed formation, causing discoloration of the seed or cankers. Late infection of pods can cause infected seed to be shriveled (Pande et al. 2010a). But sometimes infected seed develop no symptoms (Pande et al. 2005).

Fig. 1 Ascochyta blight lesions on chickpea pod and leaves caused by *Ascochyta rabiei*

Biology and Epidemiology. The pathogen *Ascochyta rabiei* (teleomorph: *Didymella rabiei*) belongs to Ascomycota (Trapero-Casas and Kaiser 1992b). It can infect chickpea and some other legumes like Lucerne (*Medicago sativa*) and sweet white clover (*Melilotus alba*), but causes major damage mainly on chickpea (Kaiser 1991; Bayaa and Chen 2011). *Ascochyta rabiei* is a necrotrophic pathogen and can grow on a variety of artificial or natural media. It readily produces pale brown pycnidia that contain countless conidia. Ascochyta blight is a seed-borne disease. Leftover diseased debris in chickpea fields can serve as a primary source of inoculum. It was observed that ascospores can play a role in the initiation of disease epidemics. Pycnidia readily form on lesions and the conidia in the pycnidia are responsible for the secondary spread of the disease during the growing season (Pande et al. 2005; Bayaa and Chen 2011). Infected debris left in the field overwinter will produce sexual spores (ascospores) in pseudothecia. Ascospores are discharged from pseudothecia during storms and are responsible for the primary infection (Navas-Cortes et al. 1995). The most favorable conditions for infection are 15–25 °C and 6–12 h of high relative humidity (Trapero-Casas and Kaiser 1992a). Lesions become visible in 4–5 days and pycnidia become visible in 7–10 days. Conidia formed in the pycnidia can be spread by rain splash and cause new infections. Multiple cycles of infections can occur during a growing season (Trapero-Casas and Kaiser 1992a). *Ascochyta rabiei* produces solanopyrones, but these phytotoxins are not required for the pathogen to cause disease. Cloudy, cool, and wet climate favors pathogen growth. The pathogen inoculum builds up very fast when night temperature is about 10 °C and day temperatures around 20 °C, and rains are accompanied by cloudy days. Excessive canopy growth also favors Ascochyta blight development (Armstrong et al. 2001; Pande et al. 2013).

Management.

- ***Cultural practices*:** Infected seeds can act as a primary source of inoculum, avoiding contamination by planting certified disease-free chickpea seed. Seed treatment with fungicides is always recommended (Bayaa and Chen 2011). Seed infection levels of less than 0.3% are considered acceptable. Implement crop rotation such that chickpea is grown only on the same ground once every 3 years. If possible, also avoid planting chickpea adjacent to fields that were planted to chickpeas the previous year. Crop rotation helps to reduce sources of *A. rabiei* inoculum present on the soil or in crop residue. Removal or burying of contaminated crop residue can also be useful to break the pathogen cycle. The pathogen can easily be spread through spore transfer via people, clothing, vehicles, machinery, and animals; therefore, if it is identified, ensure all clothing and machinery is sanitized and disinfected.
- ***Chemical and biologicals***: Under favorable environmental conditions for disease, fungicide applications are an integral component of control. During the growing season, timely application of foliar fungicides is effective in reducing infection (Pande et al. 2005). However, fungicide selection combined with cultural practices is recommended to obtain disease control and to delay *A rabiei* from developing fungicide resistance (Sharma et al. 2020). Already there are some reports of resistance development in *A. rabiei* against QoI fungicides. *A rabiei* isolates collected from North Dakota and Montana were found to exhibit a mean 100-fold decrease in sensitivity to both azoxystrobin and pyraclostrobin compared to sensitive isolates, and were considered to be resistant to azoxystrobin and pyraclostrobin. Under greenhouse conditions, these QoI-resistant isolates caused significantly higher amounts of disease than sensitive isolates on azoxystrobin- or pyraclostrobin-treated plants (Kiersten et al. 2009). Since the pathogen is seed borne, it is recommended to treat seeds with a fungicide (Table 1).
- ***Resistance sources*:** Chickpea varieties with complete resistance to *A rabiei* have not been identified yet. However, some varieties having moderate levels of resistance are available (Nene et al. 1981; Pande et al. 2010b; Sharma and Ghosh 2016) (Table 2).

2.2 Black Root Rot (*Fusarium solani* [Mart.] Sacc.)

2.2.1 Geographic Occurrence and Impact

Black root rot has already been reported in Argentina, Brazil, Chile, India, Mexico, Spain, Syria, and the USA (Nene et al. 2012; Azevedo et al. 2017). Although losses in areas where the fungus is present in the soil are significant, there is no precise information about the damage caused by the disease.

Table 1 Details of commercial fungicides and its dosages used to control major diseases in chickpea in India

Recommended dosages of fungicides against major diseases of chickpea		Mode of application
Vascular wilt	Carbendazim @ 2.5 g kg^{-1} seed; Benlate T® (Benlate 50% + Thiram 50% mix) @ 1.5 g kg^{-1} seed	Seed dressing
	Carbendazim @ 1 g L^{-1}	Spot drenching
Dry root rot	Methyl benzimidazole carbamates @ 2 g kg^{-1} seed; Captan/ Thiram/PCNB @ 3 g kg^{-1} seed	Seed dressing
Collar rot	Rizolex®/ Vitavax 200®/ Captan/ Orthiram @ 3 g kg^{-1} seed	Seed dressing
Ascochyta blight	Phenthiuram/Thiram/Benomyl @ 2 g kg^{-1} seed; Calixin M® 3 g kg^{-1} seed	Seed dressing
	Zineb/Ferban/Maneb/Captan/Captafol/Daconil®/ Bravo 500® @ 3 g L^{-1}	Foliar spray
Botrytis gray mold	Ronilan®/Daconi® @ 3 g L^{-1}	Foliar spray
	Dithane M 45®/Iprodione/Thiram @ 3 g kg^{-1} seed	Seed dressing

2.2.2 Symptoms/Signs

Chickpeas affected by black root rot turn yellow, wilt, and dry out prematurely. The root system rots with most of the fine roots destroyed due to the action of the fungus (Fig. 2). The remaining roots become blackened and necrotic (Harveson 2011). Drying plants are seen scattered throughout affected fields. The disease can happen at any growth stage, and under conditions of high soil moisture. Seeds may rot before they emerge, especially in Kabuli genotype.

2.2.3 Biology and Epidemiology

Black root rot of chickpea is caused mainly by the soil dweller fungi *Fusarium solani* species complex. Some authors attribute the disease etiology to *Fusarium solani* f. sp. *pisi* (Hasanzade et al. 2008; Westerlund et al. 1974) and different lineages of *Fusarium solani* have also been reported as causal agents of the disease (Cabral et al. 2016; Azevedo et al. 2017). The pathogen survives in infested crop debris, parasitizing other crops planted in rotation with chickpeas and volunteer chickpea plants. In Brazil, for example, the disease occurs in chickpea plantations planted in succession with soybeans. *F. solani* produces chlamydospores that can remain viable for several years in the soil before they germinate, invade the host plant, and grow on the root system. Hypha penetrate and branch into the root cortical tissue where the disease develops. Excessive moisture and temperatures around 25–30 °C as well as stress factors such as drought and soil compaction favor diseases development.

Table 2 Details of resistant cultivars against major diseases of chickpea

Disease	Country	Resistant cultivar
Wilt	*India*	Pusa 212, Avrodhi, JG 315, JG 14, JG 11, JGK 2, KAK 2, Vijay, Vaibhav, JG 63, Birsa canna-3, WR 315, JG 74, JAKI 9218, Vihar, JG 1265, BG 1053, PDG 4, Gujarat gram 4, Gujarat gram 1, BGM 47, COG 29–1, L551, ICCV 98505, ICCV 07105, ICCV 07111, ICCV 07305, ICCV 08113, ICCV 05530, ICCV 05534
	Mexico	Surutato 77, Sto. Domingo, Senora, UC 15 and UC 27
	Pakistan	Punjab 2000, CM 98
	Sudan	ICCV 2, UC 15, FLIP 85-20C, FLIP 85-29C and FLIP 85-30C
	Ethiopia	Chefe
	Spain	FLIP 84–43, FLIP 85–20, FLIP 85-29C, ILCs 127, 219, 237, 267, 513 and CA 2954
Collar Rot	–	–
Dry Root Rot	*India*	ICCV 05530,[a] ICCV 08305,[a] RSG 991[a]
Ascochyta blight	*Bulgaria*	Plovdiv 019, Obraztsov chijlik 1, Plovdiv 8 (All genotypes are moderately resistant)
	Cyprus	ILC 3279 (All genotypes are moderately resistant)
	Italy	Ali, Sultano, Califfo (all genotypes are moderately resistant)
	India	F8, C 12/34, C 235, G 543, H 75–35, GG 688,GNG 146, Gaurav, BG 261, GG 588, Hima chana-1, Gaurav, Vardan, Samrat, PBG 1 and BG 261 ICCV 04052, 04509, 04505, 04512, 04513, 04523, 04524, 04525, 04526, 04530, 04537, 05502, 05503, 05511, 05512, 05513, 05514, 05515, 05523, 05530, 05531, 05546, 05551, 05571, 98,811, 98,816, 98,818[b]
	Morocco	Pch 4
	Pakistan	F8, C 12/34, C 727, C 235, CM 72, C 44[b]
	Syria	ILC 482, Ghab 2[b]
	Turkey	Guney Sarisi 482, ILC 195[b]
	Russia	VIR 32, Nut Zimistoni[b]
Botrytis Gray Mould	*Bangladesh*	Barichola 5, ICCL 87322[b]
	India	BG 276, GL 90159, GL 91040, GL 91071 and GL 92162[b]
	Nepal	ICC 14344[b]

[a]Data not published
[b]Genotypes having moderate level of resistance

2.2.4 Management

- ***Cultural practices***: Avoid planting in high moisture soil, minimize stress conditions such as soil compaction and waterlogging, use disease-free seeds, crop rotation, and avoid planting chickpeas in succession with another pulse crop.
- ***Chemicals and biologicals***: Seeds treatment with thiram + benomyl or thiram + captan can be used to prevent this disease (Nene et al. 2012).
- ***Genetic resistance***: Cultivars with general resistance to root rot can be helpful to control the disease (Nene et al. 2012).

Fig. 2 Symptoms of black root rot on chickpea

2.3 Botrytis Gray Mold (*Botrytis cinerea* Pers. ex Fr)

2.3.1 Geographic Occurrence and Impact

Botrytis gray mold is a severe disease in various parts of Argentina, Australia, Bangladesh, India, Nepal, and Pakistan. It has also been reported in Canada, Chile, Colombia, Hungary, Mexico, Myanmar, Spain, Turkey, the USA, and Vietnam. It can cause yield losses up to 100% (Nene et al. 1981).

2.3.2 Symptoms/Signs

Botrytis gray mold is more severe on the portions of plant concealed in the canopy. The infection is typically observed at flowering time when the crop awning is fully developed. Gray or dark brown lesions (10–30 mm) covered with mouldy sporophores appear on flowers, leaves, stems, and pods when humidity is optimum (up to 70%) and can be seen on plant parts as signs of the disease (Fig. 3). Tender twigs break Geographic at the point where the gray mold fungus has caused infection. Pretentious flowers and leaves turn into a rotting mass. Infected pods contain either

Fig. 3 Botrytis gray mold symptoms on tender twigs, stem, flower, and pod of chickpea (**a**, **b,** and **c**) and spores under microscope (**d**)

small, shrivelled seeds or no seeds (Pande et al. 2002). Pathogen may develop on infected dead tissue and are capable of producing spores on their surface.

2.3.3 Biology and Epidemiology

The Botrytis gray mold pathogen has an extensive host range. The pathogen produces white fungal growth which turns gray due to the production of enormous numbers of spores produced in bunches at the ends of dark stalks. Over ten million spores can be formed on a single two-centimeter long lesion on the host stem. Spores of *B. cinerea* can be dispersed in wind gusts many kilometers, and if dropped on chickpea plants, they can remain latent until environmental conditions favor spore germination. Free moisture is essential for incubation and infection (Pandey 1988). Disease development is favored by excessive vegetative growth due to frequent irrigation or rain, dense spacing, and varieties with a spreading growth type. Excessive humidity around flowering and podding time and temperature between 20 and 25 °C also favors disease development (Pande et al. 2006). Temperature favorable for *B. cinerea* is slightly higher than *A rabiei*. Ascochyta blight and Botrytis gray mold may occur one after the other with Ascochyta blight appearing first. There are also some reports regarding seed-borne nature of Botrytis gray mold (Rewal and Grewal 1989).

2.3.4 Management

- ***Cultural practices***: It is possible that the pathogen can remain viable in infested stubble, which remains on the soil surface. Burning of stubble can reduce the inoculum potential to some extent but it will considerably increase the risk of soil erosion and reduce water infiltration. Removal of volunteer chickpea plants can also be a useful practice when near production fields in locations where Botrytis gray mold is a significant problem.
- ***Chemicals and biologicals***: A preventive spray of a recommended fungicide (Table 1) just prior to awning closure, followed by another fungicide application after 2 weeks, will contribute to managing Botrytis gray mold. However, fungicides will not eradicate already established infections. Therefore, regular and timely application is critical (Singh and Bhan 1986). While in case of seed-borne nature of Botrytis gray mold, obtain seed from a commercial supplier, or from a source known to have minimal levels of *B. cinerea* contamination. Nevertheless of the sources, seeds must be systematically treated with a registered fungicide (Table 1).
- ***Genetic resistance***: The nature of infection progression and genetic base of pathogen variability have not been established yet for *B. cinerea*. However, effective and repeatable controlled-environment and field-screening techniques have been developed for identification of resistant materials by various research institutes (Kaur et al. 2013; Sharma and Pande 2013). By using such phenotyping facilities only a few accessions (Table 2) belonging to both cultivated and wild *Cicer* spp. were found tolerant to *B. cinerea*, and the search for higher levels of disease resistance in present and cultivated genepool is in progress. Meanwhile, germplasm with moderate levels of resistance to *B. cinerea* has been identified (Pande et al. 2003, 2006).

2.4 Collar Rot (*Sclerotium rolfsii* Sacc.)

2.4.1 Geographic Occurrence and Impact

Collar rot of chickpea is an incipient soil-borne disease and may result in 55–95% mortality of chickpea seedlings under favorable environmental conditions. Climatic factors such as heavy rainfall and high soil temperature (25–30 °C) are favorable for this disease (Sharma et al. 2017). Collar rot occurs in each province where chickpea is cultivated in India. Losses due to collar rot may be significant depending upon the environment, cultivar, and field history; however, data of actual yield losses due to collar rot are not available. Due to the death of the seedling in early crop stages, it is assumed that death of plants caused by collar rot are largely compensated by the increase in the growth of the neighboring plants.

2.4.2 Symptoms/Signs

Collar rot infection can be diagnosed when observing drying plants scattered throughout a field whose foliage turns slightly yellow before death. Collar rot is observed particularly when the soil moisture is high (up to 80%) at the seedling stage

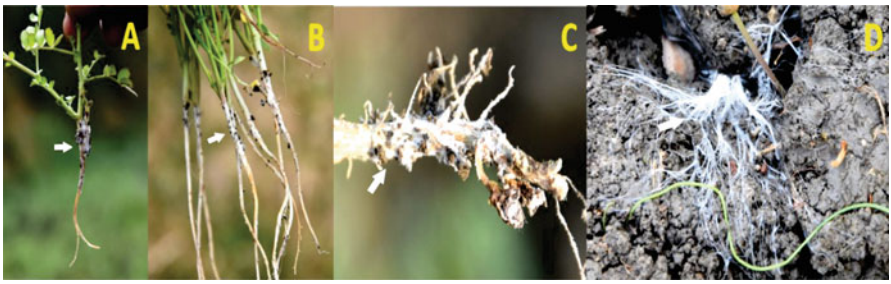

Fig. 4 Characteristic symptoms of collar rot infection (**a**, **b,** and **c**) and *Sclerotium rolfsii* mycelia development on rhizoplane nearby chickpea plant (**d**)

(Tarafdar et al. 2018). Young seedlings may collapse, but older seedlings may dry without subsiding. Uprooted seedlings may show rotting at the collar region and downward. The pathogen produces densely floccose, white, septate mycelium that covering the rotten portion is the typical collar rot symptom seen when a seedling is uprooted (Fig. 4). If affected seedlings are uprooted from moist soil in the earlier stages of infection, rapeseed-like sclerotia can be observed. The sclerotia are numerous globose hard fungal bodies produced on host tissues and in culture, with color olive brown to clove brown and measuring 0.8–2 mm. A white mycelial covering can be seen on the root of completely dried seedlings, even several days after death.

2.4.3 Biology and Epidemiology

The disease is favored by the presence of decomposed organic matter on the soil surface and excessive moisture at the time of sowing and seedling stage. As for the infection process of this fungus, the oxalic acid present in *Sclerotium rolfsii* is responsible for the initial breakdown of the structural defense mechanism and outer plant tissue maceration, following which the fungus invades the host.

2.4.4 Management

- ***Cultural practices***: Management of collar rot is challenging because of its wide host range. *Sclerotium rolfsii* infects near 500 species, which include legumes, crucifers, and cucurbits. However, the pathogen can be controlled to some extent by using soil pulverization with fungicides.
- ***Chemicals and biologicals***: Seed treatment with fungicides like carbendazim, thiram, captan, and thiphenylmethoate are generally recommended to reduce the disease (Table 1). Fungicides like carbendizim can also be used for drenching purposes when the disease is observed in small patches.
- ***Genetic resistance***: Since collar rot is an emerging threat, efforts are in progress to search for resistant materials. There is a possibility of resistance in wild species and land races of chickpea against this disease, but to introgress such trait with positive genome plasticity is a big challenge for plant breeders. Combining resistant cultivars with use of a biocontrol agent or fungicide can be a consortium approach to manage this disease.

2.5 Dry Root Rot (*Rhizoctonia bataticola* [Taub.] Butler; Syn: *Macrophomina phaseolina*)

2.5.1 Geographic Occurrence and Impact

Dry root rot is the most important root rot disease in chickpea particularly in the semiarid tropics of Central and Southern India and Ethiopia. It has also been reported from Australia, Bangladesh, Iran, Kenya, Lebanon, Myanmar, Mexico, Nepal, Pakistan, Spain, Sudan, Turkey, and the USA. The disease was first identified in India, and subsequently reported from Iran, USA, and several other countries in Asia and Africa (Nene et al. 1996; Westerlund et al. 1974). The pathogen is also known to have a wide host range causing root rots and seedling blights in many legumes as well as other economically important crops. *Rhizoctonia bataticola* is a soil inhabiting, necrotrophic fungus infecting chickpea at any crop stage, but predominantly affects during post reproductive stage when the climate remains dry and warm (Sharma et al. 2015). Over the past few decades, continuous rise of ambient temperature and depletion of soil moisture has been providing favorable condition for the emergence of dry root rot in chickpea at various locations in arid and semiarid countries of Asia and sub-Saharan Africa, where the trend of crop losses tends to be high (Sharma et al. 2015).

2.5.2 Symptoms/Signs

Dry root rot symptoms are most commonly observed in chickpea during the flowering and podding stages. Symptoms include sudden drying and blackening of roots followed by plants becoming straw colored and defoliated. During later stages, the entire root system rots and further prevents the growth of lateral and finer roots. Post-flowering stage symptoms include drooping and chlorosis of petioles and leaflets, initially confined to top leaves of the plant (Fig. 5). The disease occurs in an irregular pattern and distributes both temporally and spatially (Savary et al. 2011). In chickpea, dry root rot is easily mistaken for Fusarium wilt as well as other root rot diseases, as the general symptoms of these diseases are similar (Sharma and Pande 2013).

2.5.3 Biology and Epidemiology

The pathogen is a facultative saprophyte and is both seed borne and soil borne. Maximum ambient temperatures above 30 °C, minimum above 20 °C, and moisture stress (dry conditions) at the reproductive stages favor disease development. *Rhizoctonia bataticola* infection is initiated generally by soil-borne inoculum present in the form of hyphae and sclerotia (Fig. 5). The pathogen causes destruction of epidermal cells and penetrates through the roots. Mechanical plugging of xylem vessels by microsclerotia, toxin production, enzymatic action, and mechanical pressure during penetration leads to disease development (Sharma et al. 2015) in addition to direct secretion of macerating enzymes. However, infection of chickpea by *R. bataticola* may also occur through cotyledons during emergence, through small rootlets or through small wounds on the root surface. The fungus grows inter- and intracellularly and invades the cortical cells. It primarily grows intercellularly

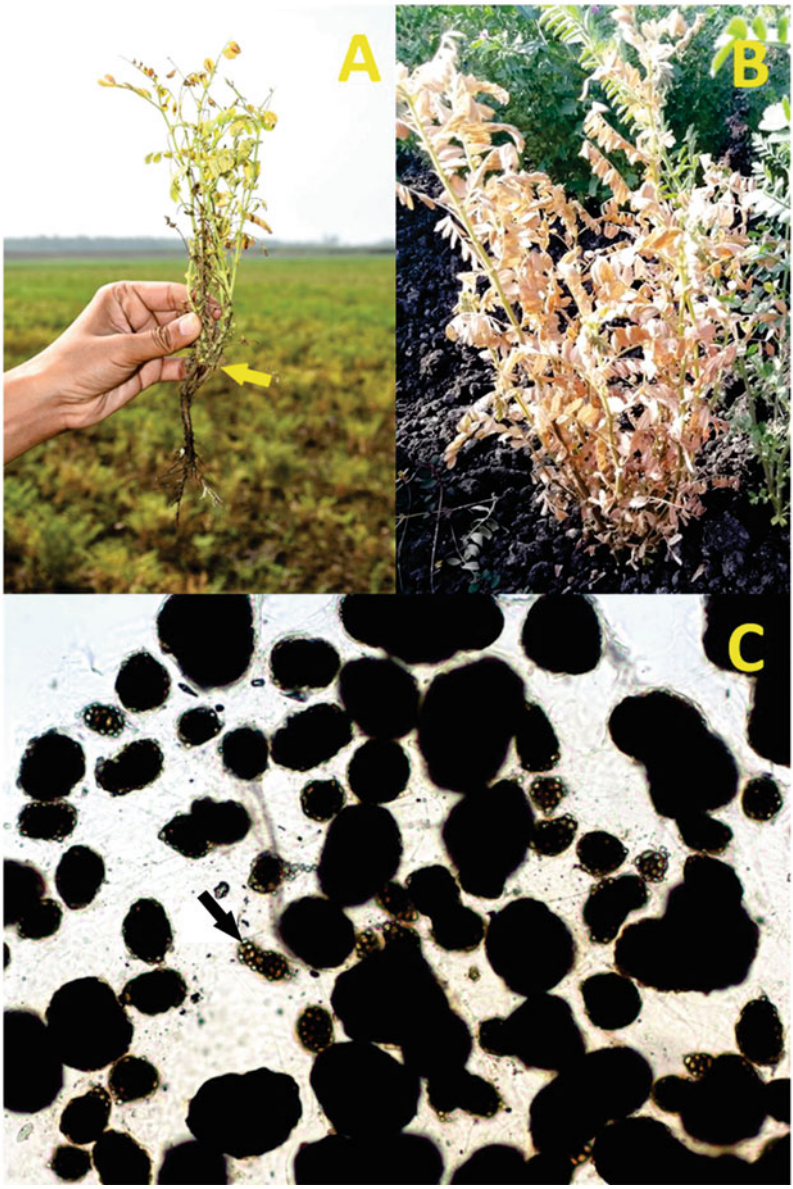

Fig. 5 Dry root rot symptoms (**a** and **b**) and microscopic view (**c**)

forming thick, short, and dark colored cells that result in the formation of large depressed necrotic lesions. The invaded cortical cells result in disintegrated or severe rotting of the roots. Being mainly a soil-inhabiting pathogen, many environmental and soil factors are responsible for the development of disease. In recent years, high

temperature stress (>30 °C) at the propagative phase and terminal drought stress (<60% soil moisture content) are occurring simultaneously during the crop growth period. These conditions are favorable for the development and spread of dry root rot, which can result in severe disease. No systematic research related to the biology, ecology, and epidemiology of dry root rot in chickpea has been conducted so far (Chobe et al. 2019).

2.5.4 Management

- ***Cultural practices***: The incidence of dry root rot can be considerably reduced by lowering the pathogen population and by maintaining soil moisture to some extent using certain physical and cultural practices. To escape hot weather conditions at the time of maturity, altering the date of sowing is a viable option. The population of the pathogen can be lowered by following crop rotation with nonhost crops. Early sowing of early maturing cultivars of chickpea, like JG-11 and ICCV 2, with timely application of irrigation practices could help in avoiding dry spells during crop maturity, thereby reducing disease severity. Further, multiplication of sclerotia while *R. bataticola* is in the saprophytic phase can be lessened by using deep plowing and proper crop sanitation. It is quite interesting that zero-tillage practices have been shown in several instances to greatly lower sclerotial populations of *R. bataticola* compared to conventional tillage practices, due to the less conducive environment provided the by former (Mengistu et al. 2009). Inoculum potential can be reduced to some extent with soil solarization practices (Gill et al. 2017). Soil solarization is the heating of soil beneath a clear plastic trap. It is usually only economical with high-value crops and in areas of abundant sunshine. In soils which are amended with nitrogen and manure, application of irrigation during summer months can result in partial control of *R. bataticola* (Lodha et al. 2003). As *R. bataticola* is a soil-borne pathogen there is little knowledge available regarding effective physical management practices, as proper crop sanitation practices can restrict the pathogen to some extent.
- ***Chemicals and biologicals***: There are various chemicals reported so far for the management of dry root rot (Table 1). However, achieving effective control with chemicals is challenging because the pathogen survives in the soil and crop debris predominantly as sclerotia, which acts as primary source of inoculum and having longevity of up to 3–5 years in the soil (Baird et al. 2003). And these resting structures permit the persistence of the fungus under adverse climatic conditions such as low soil moisture and temperatures above 30 °C (Suriachandraselvan and Seethraman 2000). As the fungus is necrotrophic and soil borne in nature, it is impractical and uneconomical to drench the whole field with chemicals.
- ***Genetic resistance***: Lack of resistant sources to dry root rot is a big challenge. Genetic enhancement of host plant resistance using conventional breeding approaches has been hampered in chickpea against dry root rot due to lack of sufficient genetic variability. There is a need to examine a large number of germplasm and wild species against dry root rot and confirm resistance found under epiphytotic conditions. Research is needed to improve the identification and characterization 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 (Table 2). New and innovative approaches like mutagenesis, induced systemic resistance (ISR), and marker-assisted selection (MAS) are becoming areas of interest of researchers to trigger the desired resistance. Mutagenesis and ISR using phytohormones like methyl jasmonate and salicylic acid etc. are simple and economic ways to induce resistance in various crop plants. Efforts are underway to further evaluate the promising lines for dry root rot and also decipher the role of phytohormones in chickpea against dry root rot. There is a high necessity for genetic gains in enhancement of host plant resistance; new and innovative approaches will be the game changing tools to obtain a stable, true resistance against dry root rot of chickpea (Chobe et al. 2019).

2.6 Fusarium Wilt (*Fusarium oxysporum* Schlechtend. emend. Snyd. et Hans. f. sp. *ciceri* [Padwick] Matuo et K. Sato)

2.6.1 Geographic Occurrence and Impact

Wilt of chickpea occurs in about 32 countries across six continents. The disease was first reported in India by Butler in 1918. Subsequently, the pathogen has extensively spread to most chickpea cultivating areas of Asia, Africa, Southern Europe, and the Americas. Eight races of *Fusarium oxysporum* f. sp. *ciceris* (FoC) have been reported, viz. 0, 1A, 1B/C, 2, 3, 4, 5, and 6. In India, Races 1A (also known as race 1), 2, 3, and 4 have been reported, whereas in the Mediterranean region and the USA, races 0, 1B/C, 5, and 6 were found. Race 1 is more widespread among all the races, and has been reported from various regions like the USA (California), India, and the Mediterranean region. Chickpea wilt epidemics can be devastating and cause up to 100% loss under favorable conditions. In India and Spain, the annual yield loss due to chickpea wilt is estimated to be up to 10–15%, with losses mounting up to 70–100% in years of severe outbreaks of the disease (Nene et al. 1981; Kaiser et al. 1994; Chobe et al. 2016).

2.6.2 Symptoms/Signs

Depending on the resistance level of the cultivar, wilt symptoms can progress at any stage of plant growth, and the affected plants may appear in patches or be spread across a field. In susceptible genotypes, wilt symptoms can be observed within 25 days after field sowing (designated “early wilt”). However, the symptoms are more prominent in the genotypes at flowering usually 6–8 weeks after sowing (designated “late wilting”). During late wiling, symptoms can develop to the pod-ding stage and include drooping of the petioles, rachis, and leaflets, followed by yellowing and necrosis of foliage (Fig. 6). As compared to late wilting, early wilting causes more loss due to complete mortality of plants. Seeds harvested from late-wilted florae are lighter, rougher, and duller than those from healthy plants. This pathogen gives differential reaction to different cultivars owing to its race-specific disease symptomatology. Races 0 and 1B/C induce yellowing symptoms, whereas

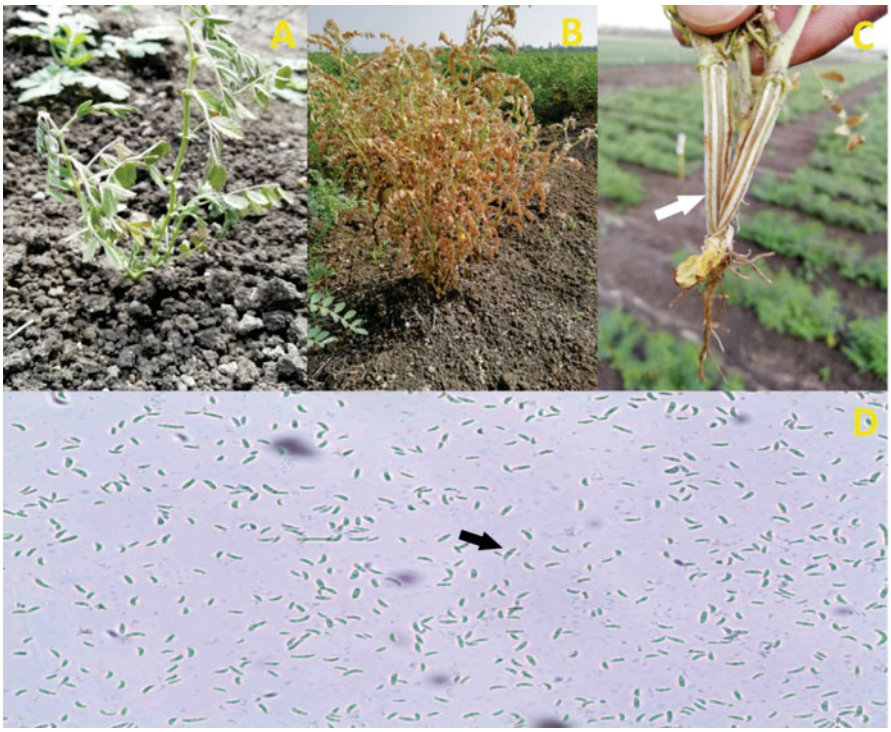

Fig. 6 Furarium wilt symptoms early wilting (**a**), late wilting (**b**), xylem browning due to FoC colonisation (**c**), and conidia (**d**)

the remaining races (1A, 2, 3, 4, 5, and 6) induce wilting (Chobe et al. 2016; Sharma et al. 2019).

2.6.3 Biology and Epidemiology

Chickpea is grown in warmer and drier climates, it was observed that the wilt incidence is generally higher at high temperature (>25 °C) and in soils where crop rotations are generally not practiced. This pathogen can be transmitted by seed and may survive in plant debris existing in soil. The chlamydospores produced by the pathogen can be observed free in soil, in cotyledons, and in the hilum of the seed. Primary infection of this pathogen occurs through chlamydospores or by the mycelia. Additionally, it was observed that the chlamydospores of this pathogen can remain viable up to the next crop season, while the conidia are generally short lived. It was also found that the nutrient status of the inoculum regulates chlamydospore formation (Nene et al. 1981, 1996). The pathogen stays in dormant condition through chlamydospores in plant debris until forced to germinate. Conditions such as release of carbohydrates from plant debris favors germination of conidia. After germination of chlamydospores, new conidia may form from the actively growing hyphae. Fungal hyphae penetrate through epidermal cells of the host roots which is

later followed by the development of a systemic vascular disease in host plants. This process of invasion can either occur directly or through wounds. As far as direct penetration by the fungus is concerned, it could occur through root tip of both taproots and lateral roots. Pathogenicity is regulated through amalgamation of different factors, which include various fungal compounds, plant surface structures, activators or inhibitors of fungal spore germination, and germ tube formation. At the time of pathogen colonization, mycelium spreads intracellularly through the root cortex until it reaches the xylem vessels and enters them through the pits. Though the formation of chlamydospore is thought to be contingent on the nutrient status of inoculum, under natural field conditions, pathogen inoculum is frequently subjected to much inferior nutrient levels compared to "well-fed" macroconidia (Fig. 6d) produced on any suitable artificial medium.

2.6.4 Management

- ***Cultural practices***: Use of disease-free seed should be adopted as a regular practice. Also avoid sowing when temperature is high (late sowing). Soil solarization during summer months and regular crop rotation practices can reduce disease incidence to some extent.
- ***Chemicals and biologicals***: Seed treatment or soil drenching with carbendazim, Benlate T (benomyl + thiram), can be done as a preventive measure for Fusarium wilt (Table 1).
- ***Genetic resistance***: Host plant resistance is the most reliable approach to manage Fusarium wilt in chickpea. There are a number of genotypes available having high level of resistance to Fusarium wilt in chickpea. Some of the resistant sources identified against Fusarium wilt are listed in Table 2.

2.7 Phytophthora Root Rot (*Phytophthora medicaginis* E. M. Hans and D. P. Maxwell)

2.7.1 Geographic Occurrence and Impact

Phytophthora root rot is a potential threat to chickpea production in wetter regions, causing significant yield losses. Phytophthora root rot is a serious disease mainly in Australia, but it has also been reported in Argentina, India, Pakistan, Spain, and the USA (Harveson 2011; Nene et al. 2012).

2.7.2 Symptoms/Signs

Symptoms of Phytophthora root rot in chickpea can occur at any stage of plant development. During seedling emergence, the disease can cause seed decay and pre- and postemergence damping-Geographic (Harveson 2011; Nene et al. 2012). In adult plants the disease leads to a premature yellowing and drying of the foliage with loss of basal leaves and rotting of the lateral roots and taproot tips. Dark brown to black lesions can occur at the upper side of the taproots, extending to the base of the stem. As the disease is usually fatal, it is common to observe patches of dead

plants in the field. The symptoms may be confused with those caused by *Rhizoctonia solani, Fusarium solani* species complex, and *Pythium* spp. or even waterlogging.

2.7.3 Biology and Epidemiology

The disease is caused by the oomycete *Phytophthora medicaginis.* This pathogen was first identified in Australia as *P. megasperma* var. *sojae* (Vock et al. 1980) and *P. megasperma* f. sp. *medicaginis* (Irwin and Dale 1982) and then reclassified as *P. medicaginis* according to Hansen and Maxwell (1991). *Phytophthora medicaginis* can survive in soil, plant debris, and on other hosts such as lucerne and leguminous plants such as *Medicago* spp., *Hedysarum* spp., and *Sesbania* spp. The thick-walled oospores, sexual spore of the pathogen, can survive in the soil for at least 10 years. In saturated soil, the oospores germinate and produce sporangia, from which zoospores (asexual spores) are released. The zoospores "swim" toward developing chickpea roots, where infection starts. After colonization, new sporangia and zoospores are produced and spread, starting new infection cycles. If the soil remains wet, the pathogen rapidly spreads to other plants. The disease is favored by warm temperatures (28–32 °C) and saturated, heavy textured soils.

2.7.4 Management

- ***Cultural practices***: Avoid planting in poorly drained and acid soil or areas with a history of Phytophthora root rot. Avoid sowing chickpeas in succession with lucerne or other leguminous plant hosts. Alleviate soil compaction.
- ***Chemicals and biologicals***: Although it does not provide season-long protection, seed treatment with metalaxyl can Geographicer some level of control (Moore et al. 2015).
- ***Genetic resistance***: Choose varieties with the highest level of resistance. Some commercial varieties, mainly of desi genotypes, can Geographicer moderate resistance (Moore et al. 2015).

2.8 Pythium Damping-Off (*Pythium ultimum* Trow and *Pythium* spp.)

2.8.1 Geographic Occurrence and Impact

Pythium damping-off of chickpea is a widespread disease reflecting ubiquitous presence of the pathogens and low temperature favorable for them at planting time. It has been reported from a number of countries including Brazil, Canada, India, Iran, Spain, and the USA (Westerlund et al. 1974; Nene and Reddy 1987; Kaiser and Hannan 1983).

2.8.2 Symptoms/Signs

Emergence failure is the major symptom of Pythium damping-off. Unemerged seeds often rot and have soil stuck on the seeds and appear as soil balls. Large-seeded chickpea seeds with thin seed coat are most susceptible to Pythium damping-off (Fig. 7).

Fig. 7 Pythium damping-off: A germinating chickpea seed colonized by *Pythium ultimum* causing preemergence damping-off

2.8.3 Biology and Epidemiology

A number of *Pythium* species are reported to cause chickpea damping-off, including *P. ultimum* and *P. irregulare* and other *Pythium* spp. All the *Pythium* species form thick-walled oospores in infected root tissue and some produce thick-walled sporangia, which can survive in soil for years. The surviving oospores and sporangia germinate in response to seed exudates and infect seeds. Generally, Pythium damping-off is favored by low temperature and high-water potentials (Bhatti and Kraft 1992).

2.8.4 Management

- ***Cultural practices***: These include later planting, selecting well-drained soil, and choosing small-seeded chickpea varieties.
- ***Chemicals and biologicals***: Treat seeds (especially varieties with large-sized seeds) with metalaxyl or mefenoxam that are specific for *Pythium* spp. and other oomycetes (Trapero-Casas et al. 1990; Kaiser and Hannan 1983). In locations where metalaxyl-resistance is known, seed treatments should include fungicides that are effective against metalaxyl resistant isolates, such as active ingredient ethaboxam (Wang et al. 2021).

2.9 Sclerotinia White Mold (*Sclerotinia sclerotiorum* [Lib.] De Bary and *Sclerotinia* spp.)

2.9.1 Geographic Occurrence and Impact

There are three species of *Sclerotinia* in different parts of the world that are known to cause Sclerotinia white mold of chickpea, with *Sclerotinia sclerotiorum* being the most common. The disease has been reported from many countries including

Australia, Bangladesh, Egypt, India, Italy, Nepal, and the USA (Bolton et al. 2006; Njambere and Chen 2011).

2.9.2 Symptoms/Signs

Initial symptoms of *Sclerotinia* stem rot consist of small brown lesions on the stem. Lesions elongate along the stem, causing wilting and stem breakage. Under moist conditions, white fluffy mycelium may develop on lesions. The fungus produces black irregularly shaped sclerotia on the stem base that fall onto the ground and can survive in soil for years in the absence of a host plant (Njambere and Chen 2011). Infection may also occur on stems near the soil line causing crown rot as a result of myceliogenic germination of sclerotia or direct spread from adjacent infected plants (Njambere et al. 2008).

2.9.3 Biology and Epidemiology

Three species of *Sclerotinia* are known to cause Sclerotinia white mold of chickpea: *Sclerotinia sclerotiorum, S. minor*, and *S. trifoliorum* (Matheron and Porchas 2000; Chen et al. 2006; Njambere et al. 2008). The species are characterized by sclerotial production and can be identified based on DNA ITS sequences. The three species have similar life cycles (Kohn 1979). The sclerotia formed on infected plants can survive in soil for several years. Sclerotia may germinate directly by means of mycelium or carpogenically through apothecia and ascospores. The ascospores can be forcibly discharged and initiate infection (Kohn 1979; Bolton et al. 2006). *S. sclerotiorum* is known to infect more than 600 plant species, whereas *S. minor* and *S. trifoliorum* have narrower host range. *S minor* can cause significant economic losses in crops like peanut (*Arachis hypogaea*), sunflower (*Helianthus annuus*), and lettuce (*Lactuca sativa*) (Fuhlbohm et al. 2003). *S. trifoliorum* causes disease mainly on legume crops, particularly forage legumes such as alfalfa. Sclerotinia white mold is generally favored by cool moist weather. A crop with closed canopy provides ideal conditions for disease development. Seeds harvested from an infected crop may carry sclerotia and may carry the inoculum along with the seeds.

2.9.4 Management

- ***Cultural practices****:* Sclerotinia white mold of chickpea can be managed through cultural practices, like deep plowing to bury sclerotia, selecting chickpea cultivars with upright growth and wide row spacing to delay canopy closure (Njambere and Chen 2011; Bolton et al. 2006).
- **Genetic resistance:** Currently there are no chickpea cultivars known to be resistant to Sclerotinia white mold.

3 Viral Diseases

3.1 Alfalfa Mosaic Virus (AMV)

3.1.1 Geographic Occurrence and Impact

Alfalfa mosaic virus in chickpea was first reported from California, USA. The disease has been found to be distributed in different countries of Africa, America, Asia, and Europe and in Australia. In Asia, AMV has been reported in chickpea from India and Iran. The disease has also been reported from Algeria, Canada, Hungary, Morocco, New Zealand, and elsewhere in the USA.

3.1.2 Symptoms/Signs

AMV is not considered an economically important virus in chickpea and incidence typically is low. The first visible symptoms are usually mosaic or mottle under field conditions. Under some conditions, the symptoms may become very mild or could disappear. In severe infection in chickpea, the symptoms can include chlorosis, twisting of terminal buds, and plant dwarfing followed by necrosis and the initiation of secondary branches (Fig. 8). The new secondary branches become erect and stiff with mild mottle and smaller leaflets. With kabuli types the mosaic symptom is clear with larger leaflets. Infected plants produce very few pods. Premature drying of the pods is very common.

3.1.3 Biology and Epidemiology

Lucerne crops are one of the important hosts and inoculum sources of AMV for chickpea. AMV is spread to chickpea by aphids (e.g., *Acyrthosiphon pisum*, *Aphis craccivora*, and *A. fabae*). Beczner (1968) found that incidence of AMV in chickpea

Fig. 8 Alfalfa mosaic viral (AMV) disease in chickpea with chlorosis, twisting of terminal buds and plant dwarfing symptoms

was high, with 30% of plants infected, in the region of Hungary where the virus had earlier been very common in Lucerne crops. AMV may also infect other leguminous crops like faba bean, lentil, and pea. The virus can be transmitted in chickpea from infected but symptomless hosts through sap or aphid. The virus may also infect several nonleguminous weed or wild hosts including clovers (arrow leaf clover, crimson clover, red clover, and white clover). AMV is also reported to be seed borne in those weeds, and it may persist in a field for several years through infested seeds.

3.1.4 Management

Although the virus is widespread, until now the virus has not been a major threat to chickpea cultivation. No source of resistance has been identified for AMV in chickpea. Since the virus is reported to be seed transmitted, virus-free seed could be used as a control measure. Spatial separation of plants can reduce disease incidence. Overwintering of chickpea should be avoided where there is aphid activity. Some aphicides can be applied to control the aphid population to avoid rapid spread of AMV.

3.2 Beet Western Yellow Virus (BWYV)

3.2.1 Geographic Occurrence and Impact

The beet western yellow virus (BWYV) was first identified in California, USA (Duffus 1961). Later the virus was reported from different region of Africa, Asia, Australia, and elsewhere in the USA (Makkouk et al. 2002).

3.2.2 Symptoms/Signs

The main symptoms of BWYV infected chickpea are yellowing and stunting with brown phloem discoloration.

3.2.3 Biology and Epidemiology

BWYV is reported to infect several legume crops especially those grown in winter, e.g., chickpea, lentil, and faba bean. But the virus is not restricted to leguminous crops. It infects many nonleguminous crops and several weeds belonging to the families Asteraceae and Brassicaceae. Chickpea grain yield losses can vary widely depending upon cultivars and plant stage of infection. The virus is aphid-transmitted to chickpea in persistent and nonpropagative manner. The main aphid vectors are *Acyrthosiphon pisum*, *Aphis craccivora* (Fig. 9), and *A. fabae*. BWYV is not known to be mechanically transmitted.

3.2.4 Management

There have been no specific studies on host plant resistance to BWYV in chickpea. Effective cultural practices include increasing plant spacing and reducing plant density, rouging of infected plants, managing weed hosts, and adjusting sowing date to when aphids are less active.

Fig. 9 Colonization of *Aphis craccivora,* one of the main aphid vectors of beet western yellow virus (BWYV) in chickpea. (Photo courtesy: ICRISAT)

3.3 Chickpea Chlorotic Dwarf Virus (CpCDV)

3.3.1 Geographic Occurrence and Impact

Chickpea stunt disease caused by chickpea chlorotic dwarf virus (CpCDV) was first reported from India (Horn et al. 1993). Presently the disease occurs in Africa, Asia, Australia, and the Middle East. It has been reported from Egypt, Iran, Iraq, Pakistan, Syria, and Yemen in Asia, and from Ethiopia and Sudan in Africa.

3.3.2 Symptoms/Signs

The main symptom of CpCDV is reddening (desi type) or yellowing (kabuli type) of the leaflets. Infected leaflets become very small with bushy appearance and plants exhibit stunted growth.

3.3.3 Biology and Epidemiology

In the early 1970s, CpCDV was recognized as a serious endemic problem in India. The virus is transmitted by the leafhoppers *Orosium orientalis* and *Orosius albicinctus* in nonpropagative and persistent manner. The virus is not transmitted by sap or mechanical inoculation. CpCDV is causing extensive yield losses in economically important crops of the families Amaranthaceae, Asteraceae, Caricaceae, Chenopodiaceae, Cucurbitaceae, Cruciferae, Fabaceae, Malvaceae, Pedaliaceae, and Solanaceae. Chickpea yield loss due to CpCDV is proportional to disease incidence in the field, as infected chickpea plants produce no or few grains. An estimation of 5–10% yield losses was reported from some regions of India and Pakistan (Horn et al. 1996), whereas 50% losses was estimated in some chickpea-growing regions of Sudan. Severe yield losses due to severe infection and high

incidence of CpCDV were also recorded in Iran. It is reported that crop losses can reach up to 75–95% in chickpea when CpCDV infection is very severe in the field.

3.3.4 Management

There are no known sources of resistance to CpCDV in chickpea. Delaying the sowing date by up to 3–4 weeks combined with short irrigation interval was found effective in reducing the disease in Sudan (Hemad and Makkouk 2002).

3.4 Chickpea Stunt or Bean Leaf Roll Virus (BLRV)

3.4.1 Geographic Occurrence and Impact

Stunt or bean leaf roll virus (BLRV) is the most important viral disease in chickpea occurring in most of the chickpea-growing countries. All the viral diseases of chickpea are considered minor. The name chickpea stunt was first used by Nene and Reddy (1976) in India. The disease is widely distributed throughout the world and has been reported from several countries across four continents. Bangladesh, India, Lebanon, Myanmar, Pakistan, Syria, and Turkey on the Asia continent are most affected by the disease. Apart from those Asian countries, the disease was also reported from Africa (Algeria, Ethiopia, Kenya, Morocco, Sudan, Tunisia, and Zambia), Europe (New Zealand and Spain), and the USA. But the disease has not yet been reported from Australia.

3.4.2 Symptoms/Signs

Symptoms of the disease were first described extensively by Nene et al. (1978). When infection occurs early in plant development, the stunting or dwarfing symptom is most visible. It occurs due to shortening of the internodes of the plants. Affected plants can be easily distinguished from healthy plants by their orange, yellow, or brown discoloration as well as stunted growth (Fig. 10). In latter stage infections, the plant discoloration symptom and phloem browning at the collar region may occur while stunting may not be obvious. The margins and tips of the leaflets often show brittleness and become chlorotic before turning red, or reddish brown in the case of desi type, and leaf yellowing in kabuli types.

3.4.3 Biology and Epidemiology

BLRV is not transmitted through seed or sap. It is mainly transmitted by aphids (e.g., *Acyrthosiphon pisum*, *Aphis craccivora*, *A. fabae*, and *Myzus persicae*) in a persistent, nonpropagative manner. Wider plant spacing and early sowing were found to favor stunt disease in India. Aphid activity influences BLRV incidence.

3.4.4 Management

Spread of BLRV within a crop can be reduced using aphicides. Seed treatment with imidacloprid before sowing was found effective in reducing disease incidence (Makkouk et al. 2002). Sowing should be started when the aphid population is low. The sowing time could be changed to either early or late depending upon the

Fig. 10 Stunt or bean leaf roll viral (BLRV) disease in chickpea with yellow or brown discoloration and stunted growth symptoms

environment and locations. The chickpea line ICC 10466 was selected because of its combined resistance to BLRV and Fusarium wilt (Nene, 1988).

3.5 Cucumber Mosaic Virus (CMV)

3.5.1 Geographic Occurrence and Impact

Cucumber mosaic virus (CMV) in chickpea was first reported in Iran (Kaiser et al. 1968). The disease was subsequently reported from India, Pakistan, and Morocco. It is reported that the virus causes economic losses in chickpea in Australia. The less natural incidence of CMV is found in almost all cool-season legume crops in Africa, Asia, Europe, and the USA.

3.5.2 Symptoms/Signs

The general symptom of CMV infection in chickpea is clear mottling or mosaic pattern on leaves. Sometimes it can be characterized in chickpea through stunting and leaf malformation. The plant become bushy in severe infection of CMV. Diseased plants produce very few flowers and pods, and most of them die prematurely. In some cultivars of chickpea, drooping of the terminal bud occurred within 4–5 days after artificial inoculation, and later the plants showed necrosis as well as wilting symptom of terminal or axillary buds and ultimately died within 10 days of infection. The proliferation of branches with necrotic symptom was also reported in some chickpea cultivars (Fig. 11).

Fig. 11 Proliferation of branches due to cucumber mosaic virus (CMV) infecting in chickpea

3.5.3 Biology and Epidemiology

CMV has a wide natural host range and infects more than 800 species in over 85 families including both dicotyledonous and monocotyledonous plants. Nevertheless, it is reportedly more prevalent in leguminous crops, e.g., chickpea, lentil, faba bean, and pea. It can be transmitted through more than 60 species of aphid. The virus is also spread via seeds mostly in chickpea and lentil. Mechanical transmission of CMV has also been reported in some cases.

3.5.4 Management

Management of CMV is mainly preventive. Conventional control measures of virus are difficult to apply for CMV due to its wide host range. The virus can also infect several weeds which then act as virus reservoirs and facilitate spread to adjacent production fields. There are no true sources of genetic resistance for CMV in chickpea. But chickpea lines ICC 1781 and ICC 8203 were found disease free when artificially inoculated during a host plant resistance screening trial (Chalam et al. 1986). Sticky traps and planting barrier crops can reduce aphid activity. Some aphicide could be used to control aphids and thus CMV indirectly.

4 Nematode Diseases

4.1 Cyst Nematode (*Heterodera ciceri*)

4.1.1 Geographic Occurrence and Impact

Heterodera spp. and *Globodera* spp. are the main genera of cyst nematodes causing severe damage worldwide. In chickpeas, the genus *Heterodera* is the most important due to the severe damage caused to the crop. Nematodes of the genus *Heterodera* are obligatory parasites and have more than 40 species that attack specific crops, often causing huge economic damage to farmers. The species *H. ciceri*, *H. cajani*, *H. goettingiana*, and *H. swarupi* have been reported to be associated with chickpeas (Castillo et al. 2008). Among these species, *H. ciceri* is considered the most economically important and aggressive in the main chickpea-producing areas worldwide. In Syria and Turkey, *H. ciceri* causes losses in chickpeas and lentils of 20 and 50%, respectively, under low management (Greco et al. 1988a; Di Vito et al. 1994b). In addition to Syria and Turkey, *H. ciceri* has also been found in Jordan, Lebanon, and Italy (Greco et al. 1992; Di Vito et al. 2001; Greco and Inserra 2007). Under conditions of water stress and high infestation of *H. ciceri*, total losses in chickpea production can occur in soils with good fertility and reduce the rate of grain yield. In India, *H. cajani* and *H. swarupi* have been reported to infect chickpeas in several locations (Ali 1995; Sharma et al. 1998; Ali and Sharma 2003), and *H. goettingiana* has been detected in Northern Africa (Di Vito et al. 1994a). Although these species are associated with chickpeas, no damage to chickpeas has been observed under field conditions in these countries.

4.1.2 Symptoms/Signs

Cyst nematodes affecting chickpea cause a reduction in the development of the root system and nitrogen-fixation nodulation in the roots, preventing the absorption of water and nutrients by the plants (Castillo et al. 2008). In infected roots it is possible to see cysts or adult white females of *H. ciceri* due to parasitism. Adult females that are lemon-shaped, vary in size, and are white, cream, or light brown in color are visible 19 days after sowing, and brown cysts are observed 38 days after sowing (Greco et al. 1988a; Kaloshian et al. 1986) (Fig. 12). Chickpeas infected by *H. ciceri* become stunted, with light green leaves that progress to chlorosis, making diagnosis difficult in the field as the symptoms are similar to the effects of water stress, nutritional deficiency, and herbicide toxicity. Initial symptoms occur in plants in small patches, usually circular, with greater evidence in the flowering phase, consequently a reduction in the number of flowers, pods, and grain is observed (Greco et al. 1988a). When no management strategy is adopted, symptoms spread quickly in the production field.

4.1.3 Biology and Epidemiology

The life cycle of *H. ciceri* from egg to adult phase is similar to that reported for *Meloidogyne* spp., showing only small peculiarities. Infective J2 emerges from the cyst after the first rains or irrigation moves in the soil and penetrates the tips of new

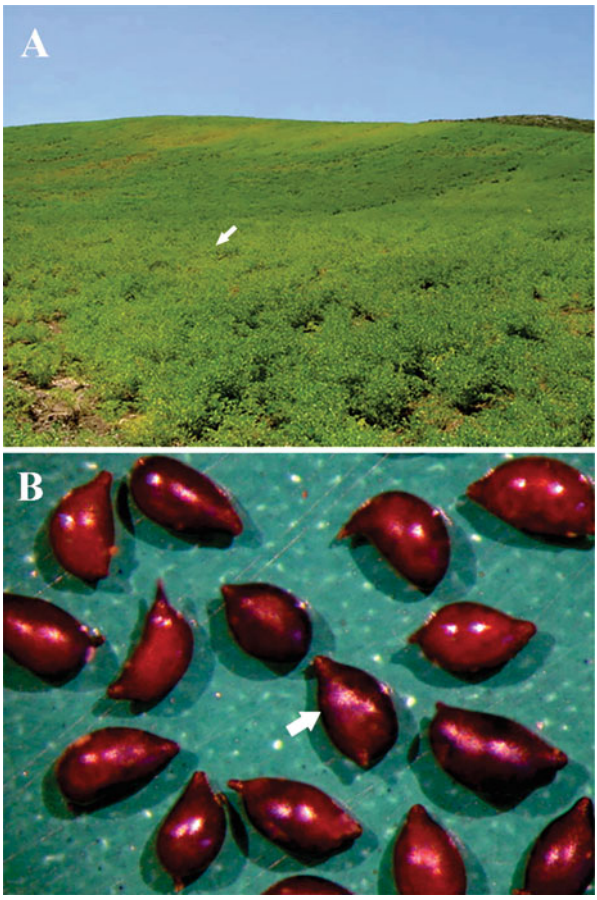

Fig. 12 Field symptoms caused by infections of chickpea with *Heterodera ciceri* (**a**) and brown mature cysts of *H. ciceri* (**b**)

roots, migrates intercellularly and establishes the feeding place (syncytial cells). Juveniles of *H. glycines* penetrate through the root cap and 15 mm in length from the root tip (Campos et al. 2011). This suggests that *Heterodera* species can penetrate both new and older roots. Temperatures below 8 °C and above 30 °C inhibit the hatching of juveniles and penetration into the root. The females, when feeding on the roots, become obese and break the cortex of the root, leaving part of the body outside the root and visible externally. At 37 days after sowing, the adult female of *H. ciceri* releases a small gelatinous matrix containing an average of 256 eggs (Kaloshian et al. 1986), with the majority of the eggs being retained inside the female. When the female dies, the wall of the cuticle becomes rigid and the body turns into a cyst. The life cycle of *H. ciceri* is completed between 25 and 32 days with a base temperature of 10 °C, and an optimum temperature of 20–25 °C for greater reproduction of the nematode (Kaloshian et al. 1986; Greco et al. 1992). The eggs inside the cysts are resistant to dehydration, remain viable, and survive in the soil for up to 8 years in the

absence of the host. In soils with water deficiency during the growth stage of chickpeas, it allows only one generation of *H. ciceri* (Kaloshian et al. 1986), but it also interferes in the grain yield of the crop. The cysts of most species of *Heterodera* are disseminated mainly by agricultural machinery, rainwater or irrigation, swirling winds that suspend and disperse the cysts to other locations, soil particles, and infested plant tissue.

4.1.4 Management

- ***Cultural practices***: One strategy that allows the population reduction of *H. ciceri* and a financial return to the farmer is crop rotation with poor hosts or nonhost crops, requiring 3–4 years of rotation to control the nematode. Clean fallow is also a common practice by chickpea farmers in some countries, allowing the population reduction of the nematode in the soil by 35–50% per year (Greco et al. 1988a). Another tactic control that would be promising for irrigated areas is the "humid fallow," which would reduce the time for planting and reduce the threshold of economic damage. Clean agricultural equipment before entry into production fields. Use clean seed sources (certified seeds) for planting.
- ***Chemicals and biologicals***: The application of commercial products based on biocontrol agents, such as fungi and bacteria, is a promising alternative for management.
- ***Genetic resistance***: There is no commercial variety resistant to *H. ciceri*. The use of tolerant varieties and poor host crops such as alfalfa, bean, lupine, and *Vicia* spp. are recommended.

4.2 Root-Knot Nematode (*Meloidogyne* spp.)

4.2.1 Geographic Occurrence and Impact

The genus *Meloidogyne* is one of the main sedentary plant-endoparasitic nematodes of economic importance on horticultural, vegetable, and other field crops. *Meloidogyne* spp., commonly known as root-knot nematodes, include about 100 species described worldwide that infect a wide range of host plants, including more than 3000 herbaceous and woody monocotyledonous and dicotyledonous species, most of which are cultivated agricultural plants. Among these species, *M. incognita*, *M. javanica*, *M. arenaria,* and *M. artiellia* have been reported to cause severe damage to chickpeas (Sikora et al. 2005; Castillo et al. 2008). These species can be divided into two groups: species adapted to warm regions and species adapted to cool weather. In the first group are *M. incognita*, *M. javanica,* and *M. arenaria*. *Meloidogyne incognita* and *M. javanica* are prevalent in tropical countries that cultivate chickpeas such as Ethiopia, Zimbabwe, and Malawi in Africa, India, Nepal, Pakistan, Bangladesh, and Brazil (Sharma et al. 1992; Castillo et al. 2008; Zwart et al. 2019). In Brazil, *M. incognita* and *M. javanica* are considered the most important species due to damage caused and their distribution in the main agricultural soybean fields in rotation with chickpeas. In addition, six chickpea genotypes were susceptible to *M. enterolobii* (Neto et al. 2019), and may be a problem for crop

rotation management due to the low availability of resistance sources or tolerance to *M. enterolobii* in soybean germplasm (Dias et al. 2010). On the other hand, *M. artiellia* is predominant and widely distributed in areas cultivated with chickpeas in cool and humid climates in the Mediterranean Basin, and in countries such as Italy, Spain, Syria, Turkey, Morocco, Algeria, Tunisia, and Egypt (Thompson et al. 2000; Castillo et al. 2008; Zwart et al. 2019). In India, *M. incognita* and *M. javanica* cause losses in chickpeas ranging between 19–40% and 24–61%, respectively (Ali and Sharma 2003; Ali et al. 2010). In Southern Italy, *M. artiellia* can reduce production by 50% in winter and 80% in spring (Di Vito and Greco 1988b). Losses in production due to root-knot nematodes are mainly influenced by the amount of initial soil inoculum density, spread in the cultivated area, variety of chickpeas, and the management strategies adopted. The geographic distribution and damage caused by the species of root-knot nematodes in chickpeas seems to be greater than that reported in literature.

4.2.2 Symptoms/Signs

Root galls are the primary and characteristic symptoms of parasitism by the species of root-knot nematodes that infect chickpea roots. However, a difference has been observed in the size of the root galls developed by the different species of root-knot nematodes (Fig. 13). *M. incognita*, *M. javanica*, and *M. arenaria* induce larger galls than *M. artiellia* (Vovlas et al. 2005; Castillo et al. 2008). In addition, the body of adult females of *M. artiellia* in infected roots show a protrusion similar to the parasitic position of adult females of cysts nematodes (Fig. 13d). The other species of root-knot nematodes have a pear-shaped body and the female remains with practically the entire body internal to the root tissue. Another evident factor in root galls is the egg masses produced by light to dark brown nematodes. In soils with high root-knot nematode infestation, this usually leads to reflex symptoms that appear as patches in a field, with stunted plants growing in contrast to healthy plants, light green leaves progressing to yellowing, and fewer branches in chickpea (Fig. 14). In soil with low infestation, the plants show mild yellowing, develop normally with no growth reduction, but there is a reduction in the production and an increase in the formation of root galls.

4.2.3 Biology and Epidemiology

The life cycle of root-knot nematodes starts in the egg, followed by the embryonic development and formation of first-stage juveniles (J1) of *Meloidogyne*. Inside the eggs, J1 undergo their first molt and hatch as a second-stage juvenile (J2) that moves in the soil, penetrating in the region of the root cap (Campos et al. 2011). J2 is also called the juvenile infective stage. Temperature of 28–30 °C favors the hatching, the muscular activity, the migration, and the host penetration for *M. incognita*, *M. javanica*, and *M. arenaria*. On the other hand, *M. artiellia* has greater activity for root penetration at a temperature of 10 °C (Di Vito and Greco, 1988a). After J2 establishes the feeding site, giant cells are formed in the root cortex, followed by three molts reaching the third and fourth juvenile stages and then adult. Adult male and female of *Meloidogyne* spp. exhibit a clear sexual dimorphism, with females

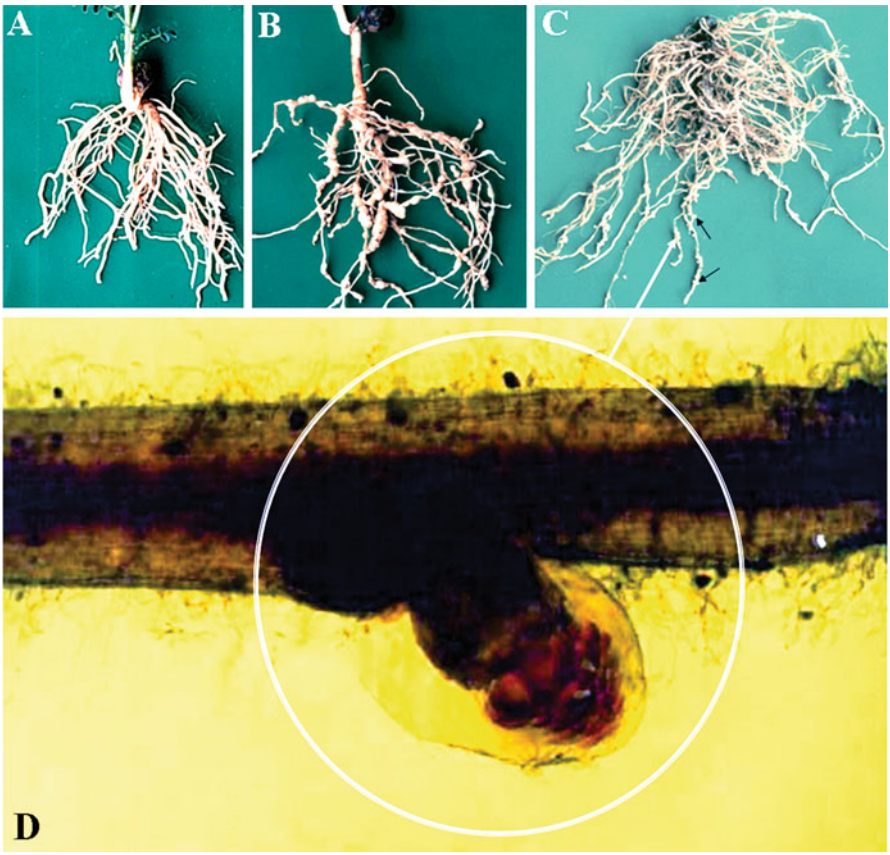

Fig. 13 Chickpea root systems. Healthy (**a**) and severely infected by root-knot nematodes (B and C). Galled roots caused by *Meloidogyne incognita* (**b**) and *M. artiellia* (**c**). Inset shows closer view of mature female (♀) of *M. artiellia* attached to a root stained with phloxine B (D)

displaying pear-shaped bodies and vermiform males. Parasitism of root-knot nematodes causes hypertrophy and hyperplasia of cells around the feeding sites. In *M. artiellia* the number of nuclei per giant cell is lower compared to *M. incognita*, *M. javanica*, and *M. arenaria* (Vovlas et al. 2005). The limited hyperplasia in the vascular cylinder and the cortical cells that surround the feeding site of *M. artiellia* seem to determine the small gall size of *M. artiellia*-infected chickpea roots (Vovlas et al. 2005). Adult females are sedentary and milky-white in color, and remain feeding on the giant cells until the eggs reproduce. Three days after reaching adult female, oviposition occurs and egg masses form on root galls. The species of root-knot nematodes adapted to warm climates complete the cycle in about 4 weeks when average soil temperature is 28–30 °C and soil moisture is at field capacity. Low temperatures (10–15 °C) favor the survival of embryos and J2 in the soil. Eggs are more resistant to water losses than J2 in the soil. Free J2 in the soil is the most

Fig. 14 *Meloidogyne artiellia*: infected chickpea plants showing stunted growth and leaves with pale green to yellow color

susceptible phase to break the life cycle of *Meloidogyne* spp., while the egg phase is the main form of nematode survival in the soil in the absence of the host plant. On the other hand, *M. artiellia* develops well at temperatures between 15 and 25 °C, completing just one generation per growing season (Di Vito and Greco, 1988a; Thompson et al. 2000). Temperatures of 30 °C slow down its development (Sikora et al. 2005), surviving during the dry and warm summer season under anhydrobiotic conditions (Di Vito and Greco 1988a). *Meloidogyne incognita*, *M. javanica*, and *M. arenaria* are more adapted and thus more abundant in soils with a medium to sandy texture, while *M. artiellia* occurs in soils with 30–40% clay (Sikora et al. 2005).

4.2.4 Management

- ***Cultural practices***: Crop rotation with nonhost crops constitutes one of the most efficient control measures for root-knot nematodes. Avoid cropping for three consecutive times with suitable host plants, aiming to reduce population density and, consequently, avoid reaching the threshold level of economic damage. Several species of *Crotalaria* have antagonistic action to the root-knot nematodes, and can be an alternative for management in soils with high infestation. The combined effect of soil harrowing and irrigation "humid fallow," in medium to sandy soils during a warm and dry season, leaving the soil free of weeds, followed by fallow 14 days before sowing is a promising measure for the control of *M. incognita* in field conditions (Dutra and Campos 2003). The addition of organic materials in the soil contributes to the population reduction of root-knot nematodes, improves the physical structure of the soil, stimulates the survival of antagonistic microorganisms, and increases productivity. No-tillage prevents soil disturbance, consequently reducing the spread of the nematode, in addition to favoring nematode antagonistic agents, maintaining soil moisture, and recycling

nutrients in the field. Weed management of suitable root-knot nematodes host crops also reduces the source of inoculum and the survival of root-knot nematodes.

- ***Chemical and biologicals***: Nematicides and soil solarization, although shown effective for controlling root-knot nematodes, are not recommended for their control in chickpea because their use is prohibitively expensive (Sikora et al. 2005). However, biocontrol products based on antagonistic bacteria (*Bacillus* spp., *Pseudomonas* spp., and *Pasteuria penetrans*) and fungi to root-knot nematodes (*Trichoderma* spp., *Purpureocillium lilacinum*, and *Pochonia chlamydosporia* var. *chlamydosporia*) are also efficient measures for managing root-knot nematodes under field conditions.
- ***Genetic resistance***: Some crops such as groundnut and winter cereals are not suitable hosts for *M. arenaria*, *M. incognita*, and *M. javanica* and could therefore be included in rotation with chickpea in warm climates (Sikora et al. 2005; Castillo et al. 2008). However, in the Mediterranean Basin, nonhost of *M. artiellia*, such as cotton, sugar beet, maize, or lentil, can be used for managing this nematode (Sikora et al. 2005; Castillo et al. 2008). Although resistant genotypes have been identified in wild *Cicer* species with resistance to *M. incognita*, *M. javanica*, and *M. artiellia*, some challenges restrict their use as sources of resistance to root-knot nematodes at the level of applicability for the farmer (Sikora et al. 2005; Castillo et al. 2008).

4.3 Root Lesion Nematode (*Pratylenchus* sp.)

4.3.1 Geographic Occurrence and Impact

Among the root lesion nematodes that parasitize chickpeas, the genera *Pratylenchus*, *Pratylenchoides*, and *Zygotylenchus* are the most widespread. However, *Pratylenchus* is commonly the most present in the main agricultural regions cultivated with chickpeas and can cause severe damage to the crop. In the Mediterranean region, the species *P. thornei, P. penetrans*, *P. zeae*, *P. mediterraneus*, *P. crenatus*, and *P. pinguicaudatus* were identified (Di Vito et al. 1994a, b; Greco et al. 1992; Castillo et al. 2008). In Australia, *P. thornei* and *P. neglectus* are the most widespread species attacking chickpeas grown in rotation with wheat (Sikora et al. 2005). In Brazil, *P. brachyurus* and *P. zeae* are the most widespread in soybean fields rotated with corn and chickpeas, with emphasis on the first species that causes significant damage to soybeans and corn, however, there has been little investigation of *P. brachyurus* on chickpeas. The genus *Pratylenchus* has a wide range of hosts, with *P. thornei* and *P. penetrans* being the main species identified in the semiarid and dry regions with high infestation, reducing the root system of chickpeas (Sikora et al. 2005; Castillo et al. 2008). *Pratylenchus thornei* has a cosmopolitan distribution, with a high incidence ranging from 61–92% in chickpea production fields in the Mediterranean Basin and 28–61% in North Africa (Di Vito et al. 1994a, b; Greco et al. 1992). Areas of cultivation of chickpeas in Turkey, Lebanon, and North African countries show high infestation and severe symptoms due to attack by root lesion

nematodes (Sikora et al. 2005). In Syria, in soil infested with *P. thornei,* losses in production due to damage caused vary between 25% and 75% (Greco et al. 1988b; Di Vito et al. 1992). In Australia, *P. thornei* causes yield losses of approximately 25% in intolerant chickpea cultivars (Reen et al. 2019).

4.3.2 Symptoms/Signs

Root lesion nematodes cause brown necrotic lesions in the cortex of chickpea roots. The large cavities left by the nematode in the root may allow secondary penetration of bacteria and fungi from the soil, leading to root rot. *Pratylenchus* parasitism causes a reduction of the root system (lack or delay of secondary-lateral roots), decreasing the absorption of water and nutrients by the plant. In the aerial part, therefore, there is a gradual loss of vigor and slow decline of the plant, with reduced growth, yellowing of the leaves, and withering of the plant in the hottest times of the day (Fig. 15). Symptoms occur in uneven patches in chickpea production fields, with the initial infestations present on the margins of the cultivated areas where nematode introduction and crop infestation normally occur. In areas with fusariosis attack, diagnosis in the field is difficult, causing symptoms and more severe damage to chickpeas. Soils with high infestations of nematodes and *Fusarium* also cause severe damage and death of plants, reducing the stand of the chickpea cultivated area.

4.3.3 Biology and Epidemiology

As in the other life cycles of plant-parasitic nematodes, *P. thornei* begins its cycle in the egg phase, hatching as J2, followed by three moltings until it reaches male and adult females, all morphologically vermiform and infective. After hatching, the J2 of *P. thornei* moves in the soil and penetrates into the root of chickpeas. *Pratylenchus thornei* as well as the other root lesion nematodes are migrating endoparasites, and therefore move intracellularly in the region of the cortical parenchyma when feeding, causing large necrotic areas of the epidermal, cortical, and endodermal cells (Castillo and Vovlas 2007). Penetration and internal migration at the root occur due to a set of physical and enzymatic processes. Eggs are deposited along the roots by the females, and all stages after J2 can parasitize and feed on the host. *Pratylenchus thornei* females reproduce by mitotic parthenogenesis and complete their life cycle in 4 weeks under favorable conditions. Soil temperature of 20 °C favors hatching of *P. thornei* in chickpeas (Castillo et al. 1996a, b). In new roots, *Pratylenchus* species complete several generations during the vegetative stage growth of chickpeas, with low population densities at the end of the crop cycle and in the absence of host plants. In the absence of chickpeas in the field, species of *Pratylenchus* survive in the soil in host weeds, in the egg stage in the cultural remains, and in the state of anhydrobiosis, being reactivated with humid rain (Castillo and Vovlas 2007). *Pratylenchus thornei* is more common in clay soils with warm and dry climates, characteristic of semiarid climates with seasonal rains and planted under irrigation.

4.3.4 Management

- ***Cultural practices***: Most species of *Pratylenchus* have a wide range of hosts, which makes rotation difficult, especially with winter cereals that are also good

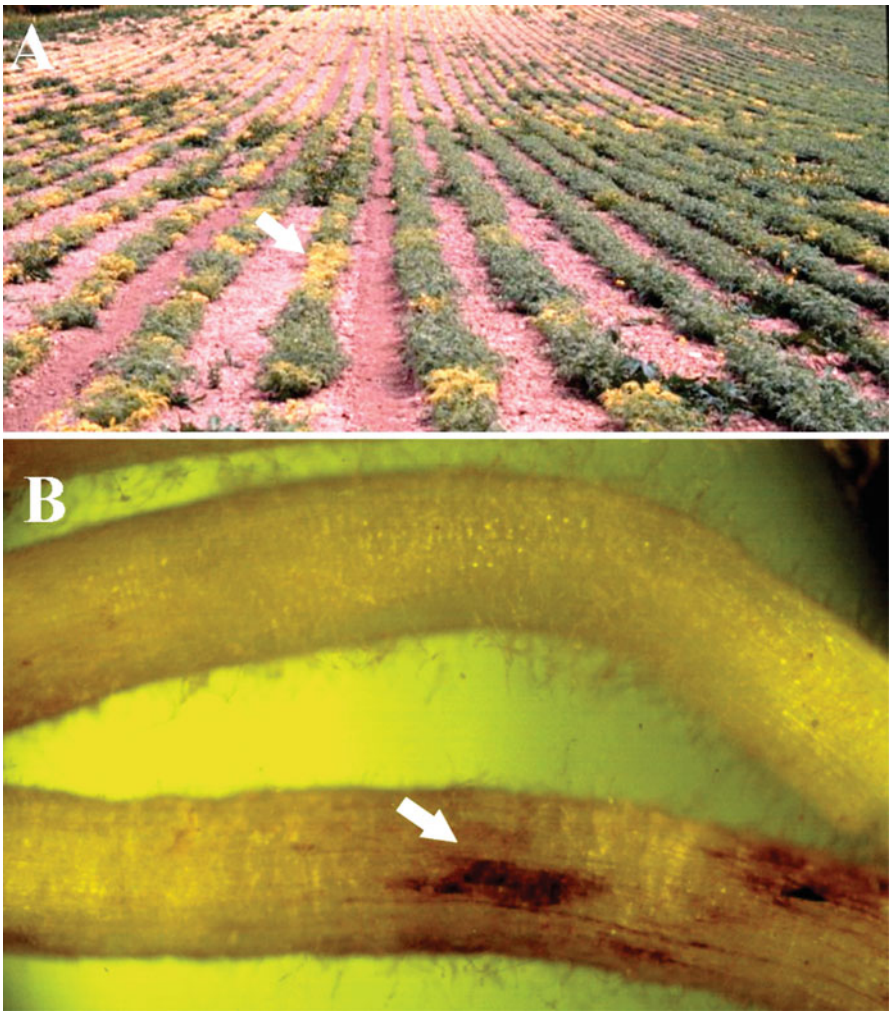

Fig. 15 Stunted and chlorotic chickpea plants infected by *Pratylenchus thornei* (**a**) and root necrosis on roots caused by infections with the nematode (**b**)

hosts. However, the choice of tolerant varieties and rotation with nonhost cultures (sorghum, millet, cotton, pigeon pea, oats, linseed, lablab, and canary) or poor hosts (Canola, maize, wheat, triticale, and sunflower), together with "clean" fallow between the cool and warm seasons, can contribute to the management of *P. thornei*. Managing weeds in the field impacts nematode survival and reduces the population density between harvests. Performing a balanced fertilization may not control root lesion nematodes, but allows greater tolerance of plants to nematode attack.

- ***Chemicals and biologicals***: There are no nematicides registered for root lesion nematodes in commercial chickpea cultivations. However, the use of biocontrol agents (*Bacillus* spp., *Trichoderma harzianum*, *Purpureocillium lilacinum, and Pochonia chlamydosporia*) is an important strategy in the management of these nematodes.
- ***Genetic resistance***: There are no chickpea cultivars resistant to *P. thornei* available to farmers, although studies have shown that 30% of wild accessions are more resistant than the cultivars currently being grown (Reen et al. 2019). Tolerant varieties such as Canola, linseed, Canary seed or nonhost cover (*Crotalaria* species, *Stylosanthes* cv. Capitata, *Stylosanthes* cv. Macrocephala, millet, corn, and sorghum), or fallow crops can be used as strategy for management of *Pratylenchus* species in areas cultivated with chickpea.

4.4 Reniform Nematode (*Rotylenchulus reniformis*)

4.4.1 Geographic Occurrence and Impact

Reniform nematodes, genus *Rotylenchulus* spp., commonly occur in countries with tropical and subtropical climates, causing severe damage to several plant species cultivated worldwide. The species *R. reniformis* is found in countries in Africa, Asia, Europe, Oceania, North America, and South America, attacking species of economic importance such as legumes, cereals, cotton, vegetables, fruit, and oilseeds. In chickpeas, the species *R. reniformis* is the most harmful to the crop, occurring in producing countries such as India, Egypt, and Ghana (Ali 1995). Mahapatra and Pahdi (1986) demonstrated that in loamy sandy soil with an inoculum density of 0.5 nematodes/g of soil there was a reduction in the growth of chickpeas, and that with a population density of 10 nematodes/g of soil occurs greater disease severity with more obvious symptoms of *R. reniformis* and 80% reduction in plant growth and development.

4.4.2 Symptoms/Signs

In the roots, the main symptoms of *R. reniformis* observed are necrotic root lesions, root rot, and reduced chickpea root system. Mature females have a kidney-shaped body, which characterizes the term "reniform" to nematodes of the genus *Rotylenchulus*. The female projects approximately two-thirds of the body out of the root and at the posterior end of the female the gelatinous matrix, "egg mass," is retained (Fig. 16). In clay soils, the clay particles adhere to the brown colored egg masses, which makes it difficult to detect the female, and characterizes the symptom named "dirty root" on the root surface. In low infestation of *R. reniformis*, the symptoms observed are a small reduction in the growth of plants and light green leaves, turning yellow with new generations and an increase in the nematode population density in the soil. After several generations of the nematode, mainly in the flowering phase, the chickpeas become weaker, causing a great reduction in crop yield. Planting at high population levels of *R. reniformis* causes severe root necrosis, reduction of the root system due to root destruction, resulting in typical symptoms of

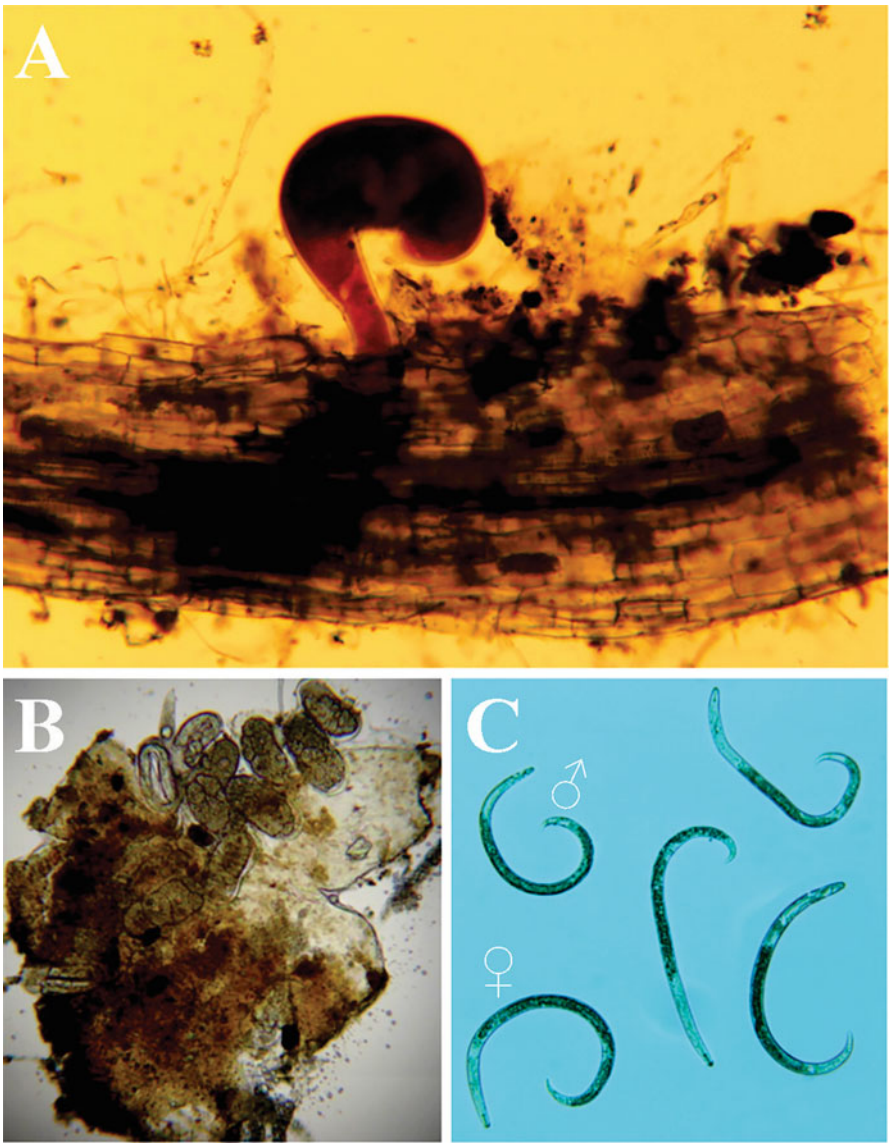

Fig. 16 Mature female of the reniform nematode *Rotylenchulus reniformis* infecting chickpea roots (**a**). Detail of an egg mass showing eggs (**b**). Immature females and males of the nematode (**c**)

the disease such as brownish discoloration of the roots, stunted growth, yellowing of the leaves, early senescence, wilt in the warm period with water deficit, dieback, and plant death. These symptoms are observed in patches distributed over the area cultivated with chickpeas, with variable sizes depending on the level of dissemination in the field.

4.4.3 Biology and Epidemiology

R. reniformis is a sedentary semi-endoparasitic nematode of roots of chickpea and several other economically important crops. After hatching, the J2 of *R. reniformis* undergoes three molts to form the immature female, which penetrates the epidermis and the cortical parenchyma of the chickpea root and establishes the feeding site in an endodermal cell (Castillo et al. 2008). J4 juveniles give rise to approximately equal numbers of vermiform parasitic females and nonparasitic males which do not feed on the plant (Castillo et al. 2008). As the female feeds on nourishing cells called syncytium, slightly hypertrophied, they become sedentary becoming a kidney-shaped body. At the end of the life cycle, mature females release between 60 and 200 eggs into a gelatinous matrix which flows out from the vulva, forming the egg masses (Castillo et al. 2008). *Rotylenchulus reniformis* completes its life cycle in 17–23 days at a temperature of 30 °C, but with thermal limits for reproduction varying between 15 and 36 °C (Castillo et al. 2008). Ali (1995) reports high infestations of *R. reniformis* in areas cultivated with chickpeas under irrigation, compared to nonirrigated areas in India (Ali 1995).

4.4.4 Management

- ***Cultural practices***: As *R. reniformis* has a wide host range, management with crop rotation becomes more difficult, but rotation with antagonistic plants (*Crotalaria juncea* and *Tagetes patula*) or cover crops (*Chloris gayana*) in combination with fallow can significantly reduce the nematode population density. Other management strategies include the choice of an area with low infestation with *R. reniformis*, soil solarization, and no-tillage to avoid the spread of the nematode in the cultivation area (Castillo et al. 2008).
- ***Chemicals and biologicals***: Nematicides are not recommended for control of *R. reniformis* in chickpeas. However, the application of biocontrol agents such as fungi and bacteria mentioned in the control of root-knot nematodes is an important tool in the population control of *R. reniformis*. Other management options are those recommended for root-knot nematodes.

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