Fungal Biology

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11 About the Series

Fungal biology has an integral role to play in the development of the biotechnology 12 and biomedical sectors. It has become a subject of increasing importance as new 13 fungi and their associated biomolecules are identified. The interaction between 14 fungi and their environment is central to many natural processes that occur in the 15 biosphere. The hosts and habitats of these eukaryotic microorganisms are very 16 diverse; fungi are present in every ecosystem on Earth. The fungal kingdom is 17 equally diverse, consisting of seven different known phyla. Yet detailed knowledge 18 is limited to relatively few species. The relationship between fungi and humans has 19 been characterized by the juxtaposed viewpoints of fungi as infectious agents of 20 much dread and their exploitation as highly versatile systems for a range of 21 economically important biotechnological applications. Understanding the biology 22 of different fungi in diverse ecosystems as well as their interactions with living and 23 non-living is essential to underpin effective and innovative technological 24 developments. This series will provide a detailed compendium of methods and 25 information used to investigate different aspects of mycology, including fungal 26 biology and biochemistry, genetics, phylogenetics, genomics, proteomics, molecular 27 enzymology, and biotechnological applications in a manner that reflects the many 28 recent developments of relevance to researchers and scientists investigating the 29 Kingdom Fungi. Rapid screening techniques based on screening specific regions in 30 the DNA of fungi have been used in species comparison and identification, and are 31 now being extended across fungal phyla. The majorities of fungi are multicellular 32 eukaryotic systems and therefore may be excellent model systems by which to 33 answer fundamental biological questions. A greater understanding of the cell 34 biology of these versatile eukaryotes will underpin efforts to engineer certain fungal 35 species to provide novel cell factories for production of proteins for pharmaceutical 36 applications. Renewed interest in all aspects of the biology and biotechnology of 37 fungi may also enable the development of "one pot" microbial cell factories to meet 38 consumer energy needs in the 21st century. To realize this potential and to truly 39 understand the diversity and biology of these eukaryotes, continued development of 40 scientific tools and techniques is essential. As a professional reference, this series 41 will be very helpful to all people who work with fungi and should be useful both to 42 academic institutions and research teams, as well as to teachers, and graduate and 43 postgraduate students with its information on the continuous developments in 44 fungal biology with the publication of each volume. 45

46 More information about this series at http://www.springer.com/series/11224

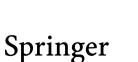
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Management of Fungal Pathogens in Pulses

Current Status and Future Challenges

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55 ISSN 2198-7777

ISSN 2198-7785 (electronic)

56 Fungal Biology57 ISBN 978-3-030-35946-1

ISBN 978-3-030-35947-8 (eBook)

- 58 https://doi.org/10.1007/978-3-030-35947-8
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Preface

Pulses, due to their rich protein content, play an important role in maintaining the 76 nutritional balance, and have become an integral part of versatile diets, including 77 vegetarian diets, across the globe. The yield and quality of these crops are adversely 78 affected due to various fungal pathogens, amounting around 100% yield losses in 79 certain crops. Pulses are infected by approximately 100 fungal diseases all around 80 the world. This book houses information on major fungal pathogens that cause sig-81 nificant losses and on management strategies to reduce the incidence and severity of 82 fungal diseases in pulse crops. 83

The present volume has 12 chapters dealing with major pathogens to pulses and 84 their management. Chapter 1 deals with the role of plant growth-promoting rhizo-85 bacteria in the management of soil-borne fungal pathogens. Chapter 2 presents sec-86 ondary metabolites which have also been proven to have antagonistic potential and 87 are considered to have the ability to control fungal pathogens affecting pulses and 88 other crops. Chapter 3 focuses on the management of fungal foliar diseases of arid 89 legumes using integrated approach. Chapter 4 discusses omics approach to control 90 Fusarium wilt of chickpea. Chapter 5 gives an overview of the management of fun-91 gal pathogens of chickpea, whereas Chapter 6 discusses the detection of wilt and 92 root rot complex of important pulse crops with strategies to control them. Chapter 7 93 discusses the management strategies and diversity of Phytophthora, causing stem 94 blight of pigeonpea. Chapter 8 reviews important foliar fungal diseases of pulses 95 and their management strategies. Similarly, Chapter 9 talks about the role of soil 96 and crop health management for cultivation of pigeon pea. Chapter 10 deals with 97 the vital foliar diseases of chickpea with its science, epidemiology, and manage-98 ment practices. Chapter 11 focuses on the management of wilt in pigeonpea mainly 99 caused by Fusarium udum. Lastly, Chapter 12 elaborates the use of biofertilizers as 100 a sustainable tool for the management of fungal pathogens. 101

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- To sum it up, the present book volume gives comprehensive information about the prevalent fungal pathogens affecting pulses and their management approaches for sustainable agriculture.
- 105 Mizoram, India
- 106 Mizoram, India
- 107 Kanpur, India
- 108 Mysore, India
- 109 New Delhi, India

Bhim Pratap Singh Garima Singh Krishna Kumar S. Chandra Nayak N. Srinivasa

Acknowledgments

Our sincere gratitude to the contributing authors who have contributed chapters in 111 this book volume, Management of Fungal Pathogens in Pulses. Current Status and 112 Future Challenges, on different aspects dealing with fungal pathogens of pulses and 113 their management. We are also thankful to the series editors, Dr. Vijai Kumar Gupta 114 and Prof. Maria G. Tuohy, for accepting our proposal and giving us a chance to 115 bring out this volume. We are equally thankful to Springer Publishing for their con-116 tinuous support and help received throughout from conceptualizing to production. 117 The production team of Springer Nature is also acknowledged for their support and 118 guidance. At last, we do agree that it is very much possible that some mistakes 119<mark>AU3</mark> might be detected and we remain responsible for any mistake this volume may con-120 tain. Please feel free to inform us the same. 121

Mizoram, India Mizoram, India Kanpur, India Mysore, India New Delhi, India Bhim Pratap Singh 122

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Chapter 1 Plant Growth-Promoting Rhizobacteria in Management of Soil-Borne Fungal Pathogens

Parishmita Gogoi, Priyanka Kakoti, Juthika Saikia, Rupak K. Sarma, Archana Yadav, Bhim P. Singh, and Ratul Saikia

1.1 Introduction

Plant diseases reduce crop yield, product quality and contaminate food grains 8 with toxic chemicals, causing a great economic loss (Zaidi et al. 2014). Soil-borne 9 fungal pathogens cause root rot, leaf fall, wilting, etc. in plants and are responsi-10 ble for the decline of yield in highly cultivated areas. These pathogens feed on 11 organic soil residues which results in root rot, leading to death, and the growth 12 rate of plants depends on their susceptibility to various environmental factors and 13 hosts (Redman et al. 2001). Pythium spp., Rhizoctonia spp., Fusarium spp., 14 Sclerotinia sclerotiorum, Phoma spp., and Cylindrocarpon spp. are few of the 15 common pathogens of soil affecting most of the agricultural crops. The epidemi-16 ology of these pathogens is caused by a large number of physiochemical and 17 biological factors. Most root rot-causing fungal pathogens can colonize and sur-18 vive in soil (Pettitt et al. 1996). Development of a large number of fungicides has 19 occurred due to numerous varieties and complexities of fungal diseases; unfortu-20 nately, resistance has already been developed by pathogens against these fungicides 21 (Agrios 2005). The genetically resistant cultivar is another approach, but this is not 22 feasible with time (Fry 2008). 23

Literature review for the last 50 years has shown that several microorganisms 24 have grown competence against soil-borne pathogens and nematodes. PGPRs are 25 studied and used in managing soil-borne fungal diseases in plants as they reduce 26

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B. P. Singh et al. (eds.), *Management of Fungal Pathogens in Pulses*, Fungal Biology, https://doi.org/10.1007/978-3-030-35947-8_1

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diseases by acting as biocontrol agents (Shaikh and Savved 2015). The PGPR 27 stimulates other beneficial symbionts and protects plants in inhibiting the contami-28 nated soils by degrading xenobiotics (Jacobsen 1997). Recently, researchers are 29 working with beneficial microbe's potential for measuring of plant protection. The 30 biocontrol agent use easy delivery, provides resistance mechanisms in the host, 31 improves plant growth, and increases yield. These antagonists operate through para-32 sitism, mycolytic enzymes, antibiosis, and competition for nutrients and space, 33 secretion of volatile toxic metabolites, etc. Thus, PGPR biocontrol is recommended 34 as a green approach; the commercial availability is very slow for a proportion of 35 registration as biocontrol agents. Therefore, future research aims to develop geneti-36 cally modified (GM) strains of PGPR to enhance plant growth-promoting activity 37 and additional mechanisms for biocontrol (Glick and Bashan 1997; Blouin-38 Bankhead et al. 2004); it is necessary to understand the environmental factors that 39 adequately act upon activity of PGPR and mechanism for biocontrol of some wild 40 strains (Landa et al. 2004a, b; Berg and Smalla 2009) as this would be acting upon 41 their inconsistent performance. 42

Biology of Soil-Borne Pathogen 1.2 43

Soil-borne pathogens survive as soil inhabitants (retain in the soil for comparably 44 longer periods) and also as soil transients (retain in the soil for shorter periods). 45 Soil-borne pathogens are survived in saprobes form. They are distributed in soil 46 depending upon the history of cropping, production practices, and various other 47 attributes. The root pathogen inoculum is present generally at the top 10 inches of 48 the soil profile, in the vertical plane, whereas field inoculums are collected from a 49 susceptible crop that grows in the horizontal plane. Soil type, pH, texture, moisture, 50 temperature, and nutrient levels are some of the factors affecting the distribution of 51 soil pathogens. Soils with poor irrigation facilities allow the growth of several soil-52 borne pathogens like Phytophthora, Pythium, and Aphanomyces. Similarly, 53 Fusarium and Verticillium wilt also occur more frequently in damp soils rather than 54 in dry soils (Deketelaere et al. 2017). Streptomyces scabies is one among the other 55 pathogens occurring in wet soil. Some of the predominant soil-borne pathogens are 56 cited in Table 1.1. 57

Table 1.1 Some predominant	Fungi	Bacteria	Nematodes	t1
soil-borne pathogens	Sclerotium rolfsii	Erwinia	Meloidogyne	t1
	Rhizoctonia solani	Ralstonia	Heterodera	t1
	Fusarium spp.	Rhizomonas	Longidorus	t1
	Pythium spp.	Agrobacterium	Paratrichodorus	t1

t1.1

t1.2

1.3 Diseases Caused by Soil-Borne Pathogen

A diverse group of fungi and other organisms are the causal agents of soil-borne 59 diseases. Genera *Pythium, Phytophthora, Rhizoctonia, Cylindrocladium*, and 60 *Armillaria* are the most important which leads to root rots. The root rot diseases are 61 distinguished by root system decay; some pathogens attack the juvenile roots, while 62 others infect mature portions of the root system. Root rot symptoms include death 63 of leaf, leaf fall, wilting, limb and branch death, and in extreme conditions full plant 64 trends to die. 65

Root rot caused by *Rhizoctonia* is well-known as wire stem, damping-off, and crown or head rot. When the mature seedling is attacked by the fungus, the effect is less in the outer cortical tissues which produce elongate drab to the reddish-brown lesion. Infected area increases in length and width, spreading the disease to the whole plant causing death (Gonzalez et al. 2011). 70

Stem rots, head rots, and collar rot are incited by Phytophthora, Fusarium, 71 Rhizoctonia, Sclerotinia, and occasionally Aspergillus niger, and the major symptom 72 of these diseases is stem rot at ground level subsequently in death of leaves and the 73 plant. Fusarium oxysporum and Verticillium spp. are the major fungi that cause wilts. 74 Symptoms of wilt include internal necrosis of stems, vascular tissue and wilting of 75 foliage. Similarly, bacterial pathogens also cause wilt disease in plants, resulting in 76 loss of yield. Seedling blights and damping-off are caused by some fungal patho-77 gens, Phytophthora, Pythium, Sclerotium rolfsii, Fusarium, and rarely Rhizoctonia 78 spp. The fungi infect in different establishment stages of pre-emergence, post-emer-79 gence, or germination of the seedling. Damping-off disease by Pythium species like 80 P. debaryanum, P. graminicola, P. aphanidermatum, and P. ultimum occurs in circu-81 lar patterns as the fungi grow radically from the point of origin. Phytophthora damp-82 ing-off disease, a low stem rot, is caused primarily by P. fragaria, P. palmivora, 83 P. cactorum, and P. syringae where warmer soil temperatures (15-23 °C) are needed 84 by the fungus for their rapid activity (Deadman 2017). 85

1.4 Management of Soil-Borne Disease

Management of soil-borne diseases require a thorough knowledge of host, patho-87 gen, and environmental conditions. These three factors are responsible for the 88 development of soil-borne diseases. The pathogens require viable inoculums to 89 infect the host. The host needs to be exposed to the pathogen inoculums. For plant 90 infection and pathogen growth, the environmental conditions should be suitable. 91 These pathogen-host-environment dynamics help in constructing a disease manage-92 ment strategy (Shafique et al. 2016). For making a disease management strategy 93 economical, potential crop loss, disease incidence assessments, and severity of dis-94 eases are key factors. It also needs regular and careful examination of symptomatic 95 plants and fields. Disease management is also critical, e.g. the management of 96

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97 *Phytophthora* root rot that requires early implementation of control measures. A management strategy in spite of being economically sound must also be safe, 98 simple, and sufficiently effective to reduce diseases to acceptable levels. Management 99 strategies of soil-borne diseases could be exclusion, eradication, and inoculum 100 reduction. Use of resistant varieties, agronomic practices, chemical control, and 101 biological control is useful for controlling this disease. Among those PGPR, ISR 102 and systemic acquired resistance (SAR) are some of the important techniques 103 (Beneduzi et al. 2012). 104

105 1.4.1 Soil-Borne Fungal Pathogens and PGPR

Substantial yield loss is caused by soil-borne the fungal pathogen (Oerke 2006). 106 PGPR are environmental friendly management strategies (Weller et al. 2007). The 107 usage of PGPR explicitly soil-borne fungal plant pathogen agents is a complemen-108 tary strategy (Haas and Défago 2005; Weller 1998). Study shows that a wide range 109 of PGPR protects against soil-borne fungal diseases (Saikia et al. 2003). The use of 110 PGPR for their biocontrol effect in field conditions is often not steady enough which 111 is one of its major limitations (Saikia et al. 2004a, b). Hence, some of the limitations 112 of applying PGPR strains are sometimes not capable of surviving in their applied 113 place or are not able to execute the specific biocontrol activity (Landa et al. 2001). 114 One of the main reasons for their inconsistency that their survival rate is not the 115 same in all types of ecosystems (Kravchenko et al. 1993; Picard and Bosco 2008; 116 Berg and Smalla 2009). Biocontrol provided by PGPR involves competition, para-117 sitism, antibiosis, etc. which comes under natural processes and is affected by abi-118 otic and biotic factors (Weller et al. 2002, 2007; Haas and Défago 2005). The abiotic 119 and biotic factors usually modify the interactions between plant, pathogen, and 120 antagonist; thus, biocontrol agent efficiency is reduced on pathogens (Berg and 121 Smalla 2009). Even if many abiotic soil factors influence the biocontrol mechanism 122 (e.g. moisture, texture, pH, temperature, organic and inorganic constituents, etc.), 123 there are very few experimental data of the interactions between antagonists and 124 their soil-borne pathogens (Picard and Bosco 2008; Berg and Smalla 2009). Factors 125 influencing the dynamics of populations in PGPR are not always affected by the 126 biocontrol mechanisms governing PGPR efficacy. Pathogen suppression by PGPR 127 occurs mainly by the activities involved in PGPR growth (Pathak et al. 2017). 128

Plant growth-promoting rhizobacteria enhance plant growth and development, 129 also increases crop productivity. Rhizobacteria (PGPR) stimulate mechanisms that 130 are broadly categorized as direct or indirect (Glick 1995). PGPR contributes directly 131 to plant growth through phytohormone production like cytokines, gibberellins, and 132 auxins, improving plant nutrition uptake by solubilizing minerals like iron and phos-133 phorus, siderophore and enzyme productions, induction of systemic resistance, and 134 lowering of ethylene level (Bhattacharyya and Jha 2012). The plant is indirectly 135 benefited by PGPR as they enhance plant growth by controlling harmful microorgan-136 isms, including parasitism, antibiotic production, synthesis of extracellular enzymes 137

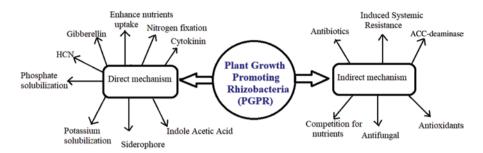


Fig. 1.1 Mechanism of plant growth promotion by rhizobacteria. Plant growth-promoting rhizobacteria (PGPR) promote plant growth directly by either assisting resource utilization of nitrogen, phosphorus, and other essential minerals or indirectly modulating plant hormone levels or by reducing the inhibitory effects of diverse pathogens on plant growth and development in the forms of biocontrol agents

for hydrolysing cell wall of fungi, decreasing pollutant toxicity, competition for138nutrients and niches within the rhizosphere (Podile and Kishore 2006; Bhattacharyya139and Jha 2012). The direct mechanism of PGPR includes the synthesis of plant hor-140mones, nitrogen fixation, and phosphate solubilization (Ahemad and Kibret 2014).141The indirect mechanism also includes biological controls, induced systemic resis-142tance (ISR), antibiotics, competition for nutrients (Fig. 1.1).143

Plant growth-promoting rhizobacteria involve various plant growth-promoting 144 mechanisms and bacterial features that are important in facilitating the growth of 145 the plant. It is controlled by 1-aminocyclopropane-1-carboxylate (ACC) deaminase 146 enzyme as it cleaves ethylene precursor of plant, ACC into ammonia and 147 α -ketobutyrate (Honma and Shimomura 1978). Plant ethylene level is decreased by 148 ACC deaminase-producing organisms by lowering ACC level in plants, while eth-149 ylene present in maximum concentrations lead to growth inhibition or death in 150 plants (Saikia et al. 2018). 151

In response to pathogen infections, plants produce an excess amount of ethylene in 152 various stresses (Abeles et al. 1992). Symptoms shown by infection-causing patho-153 gens which are seen in an infected plant appear as a direct result of pathogen imposing 154 stress (Van Loon 1984). Increase in stress ethylene level of plants infected by patho-155 gen generally results in damage to plants. Chemical inhibitors of ethylene synthesis 156 decrease the severity of the infections, while severities of pathogen infections are 157 increased by exogenous ethylene. Pretreating plants with ACC deaminase-producing 158 rhizobacteria protects ethylene-caused damage in plants (Saikia et al. 2018). 159

1.4.2 Factors Influencing on Pathogen-PGPR Interactions

The factor of climate change specifically the increase in temperature has a link 161 between PGPR and soil-borne pathogens and also on biocontrol efficacy interceded 162 by PGPR (Table 1.2). 163

Sl. no.	Abiotic factors	Biotic factors
1	Soil physical and chemical characteristics	Target pathogen
2	Temperature	Host plant
3	Water availability	Insects
4	рН	Allelopathy
5	Moisture	Weeds
6	Quality and type of pesticides applied to the soil	Phytopathogens

1.4.3 Induced Resistance 164

Microorganisms are the environment-friendly approach used in controlling soil-165 borne diseases as biological control. The major approaches of biocontrol activity 166 in PGPR are competition for antifungal metabolite production, nutrients, niche 167 exclusion, and induced systemic resistance (ISR) (Lugtenberg et al. 2001). Plant 168 growth-promoting rhizobacteria acting as biocontrol agents and its chief indirect 169 mechanism. Antifungal metabolites produced by rhizobacteria are HCN, pyrrolni-170 trin, 2,4-diacetylphloroglucinol, phenazines, tensin, pyoluteorin, and viscosin-171 amide (Bhattacharyya and Jha 2012). Rhizobacteria provide resistance against 172 some pathogenic fungi, bacteria, and viruses interacting with plant roots 173 (Lugtenberg et al. 2001). 174

Plant growth-promoting rhizobacteria trigger ISR in plants. Physical charac-175 teristics of ISR are similar to systemic acquired resistance (SAR). Plants activate 176 their defence mechanisms against infection caused by a pathogenic agent, SAR 177 (Pieterse et al. 2009). ISR is effective at managing diseases caused by various 178 pathogens; it does not target specific pathogens (Saikia et al. 2005; Romera et al. 179 2019). ISR involves jasmonate and ethylene signalling pathways within the plant, 180 and these hormones stimulate the host plant's defence responses to a range of 181 pathogens (Verhagen et al. 2004). Other molecules, like O-antigenic side chain of 182 the bacterial outer membrane protein lipopolysaccharide, cyclic lipopeptide sur-183 factants, pyoverdine, chitin, flagellar proteins, β -glucans, and salicylic acid, have 184 been summarized to act as signals for ISR. Van Peer et al. (1991) observed ISR in 185 carnation plants protected systemically by P. fluorescens strain WCS417r against 186 F. oxysporum f. sp. dianthi. ISR was also studied in cucumber plants (Wei et al. 187 1991). In cucumber leaves, rhizobacterial strains protect the leaves from anthrac-188 nose disease caused by Colletotrichum orbiculare. ISR mediated by rhizobacteria 189 is similar to SAR induced by pathogens (Van Wees et al. 1997; Kannojia et al. 190 2019), involving viral, bacterial, and fungal pathogens, and also by insects and 191 nematodes (Zehnder et al. 1997; Pozo and Azcon-Aguilar 2007; Bent 2006). It 192 was also reported that in the same plant, the same strain provides resistance 193 against several pathogens (Somers et al. 2004). The most studied rhizobacteria 194

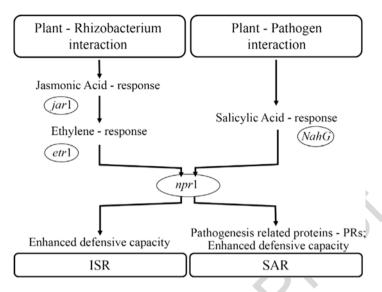


Fig. 1.2 Signal transduction pathways leading to pathogen-induced systemic acquired resistance (SAR) and rhizobacteria-mediated induced systemic resistance (ISR) in *Arabidopsis thaliana*. In ISR the jasmonate and ethylene responses are involved consecutively to trigger a defence response that is regulated by NPR1; however, in SAR the salicylic acid triggers the defence response regulated by NPR1 (Source: Van Loon et al. 1998)

that trigger ISR are *Pseudomonas* and *Bacillus* spp. (Van Wees et al. 2008; 195 Monaim 2012).

Systemic acquired resistance (SAR) and ISR are mediated by two different 197 signalling pathways (Fig. 1.2). ISR requires ethylene (ET)- and jasmonic acid 198 (JA)-mediated signalling pathways, whereas SAR uses salicylic acid (SA) (Saikia 199 et al. 2006). The signalling molecules accumulate and counter the defence responses 200 (Ryals et al. 1996). ISR provides significantly lesser protection compared to SAR 201 (Van Loon 2000). ISR depends on plant genotyping degree (Bloemberg and 202 Lugtenberg 2001). According to Van Wees et al. (2000), SAR and ISR when used 203 collaboratively provide better protection rather than acting alone upon pathogens. 204 The utilization of exogenous SA also induces SAR in many plant species. Tissue 205 necrosis is a common symptom for SAR activation (Vleesschauwer and Höfte 2009; 206 Mishina and Zeier 2007). Pathogenesis-related (PR) proteins are specific sets of 207 defence-related genes responsible for activating SA. Normally, ISR does not act 208 upon the activation of PR genes. PR proteins are responsible for the enhanced 209 defensive property of SAR (Van Loon 2007). The ethylene precursor, ACC, and also 210 the methyl jasmonate (MeJA) provide pathogen resistivity (Shine et al. 2019). 211 Different plant species studies have shown its ability to produce ISR in response to 212 different PGPRs, and also the specific interaction among rhizobacteria and plants 213 was studied (Van Loon 2007). 214

215 1.4.4 Other Control Methods

216 1.4.4.1 Cultural Method

Irrigation and fertilizer, when used together, improve the health of the plant. The use of ammonium bicarbonate, phosphatic fertilizer, phosphoric acid, and gypsum reduces the effect of soil-borne diseases in plants. The reduction of the disease requires good air circulation and good soil drainage within plants. Timely removal of dead or infected plants when disease occurs reduces inoculum build-up potential.

222 1.4.4.2 Crop Rotation

Soil-borne pathogens can exist in plant and soil debris for up to many years. Crop
rotation can be applied to evade this problem as it helps in controlling the soil-borne
inoculums. Pathogens are soil invaders that can help give the best result in crop rotation. However, crop rotation becomes less impractical when the pathogen resides in
soil. In some causes of cropping systems, field tilting and field fallow are done for
6 months or a year (Veena et al. 2014).

229 1.4.4.3 Tillage Practices

Soil tilting can reduce the pathogen population by its burial or are dried in the
exposed out layers. Deep ploughing is very useful in reducing the infection source.
Before planting subsoiling is done to increase the yields of root rot-infected plants
(Singh 2017).

234 1.4.4.4 Soil Amendments

Sawdust, straw, oil cake, etc. are organic amendments that are used effectively to
manage diseases caused by *Aphanomyces*, *Pythium*, *Verticillium*, *Phymatotrichum*, *Macrophomina*, and *Phytophthora*. Useful microorganisms multiply in soil and
help to suppress pathogens. Lime usage increases soil pH to 8.5 which reduces
cabbage clubroot. Castor cake and neem leaves play a crucial role in reducing the
foot rot of wheat.

241 1.4.4.5 Soil Solarization

Soil solarization is rise of soil temperature by sunlight. Various soil-borne pathogens like bacteria, fungi, and nematodes reduce the potential and inoculum for disease by inactivating near the soil surface due to soil solarization. *Verticillium* and *Fusarium*

wilts are controlled by soil solarisation (Veena et al. 2014).

1.4.4.6 Chemical Control

The application of chemical fungicides is done to defend the plant from disease or 247 eliminate a pathogen infecting the plant. Chemical control includes soil treatments, 248 disinfestations of warehouses, cleaning of equipment, etc. Application of fungicides 249 is in the form of liquid drenches, granules, or dust to the soil to eradicate diseases. 250 They are applied in the fields through the irrigation system available. Nematodes are 251 treated by chemical controls and volatile substances. Chemical fungicides mainly 252 act as toxic barriers between host and pathogens. They are used as soil drenching, 253 seed treatment, and soil fumigation. Propamocarb, prothiocarb, and metalaxyl are 254 some of the frequently used fungicides. Chemical fungicides cause a lot of harm to 255 the soil and plant along with reduction of diseases (Mahmood et al. 2016). 256

1.4.4.7 Resistance of Host Plant

Making a resistant plant is the most cost-effective and adequate method. It reduces the258loss of yield, and also it reduces pollution and cuts off the disease controlling effort.259Monogenic (vertical) resistance is a gene- or race-specific resistance that is capable of260controlling only a few pathogens. On the other hand, polygenic (horizontal) resistance261is a quantitative or non-specific resistance. It lasts longer and is not so adequate. Host262resistance is useful when used together with chemical and cultural methods.263

In transgenic approaches, genes are transformed for tolerating detrimental abiotic and biotic conditions, and for genes encoding enzymes like glucanases and chitinases acting upon fungi, viruses, and bacteria by using DNA technology. 266 Various PR proteins, glucanases, and chitinase-coding genes are cloned, isolated, and expressed in plants; thus, the development of pathogens is resisted along with plant resistance. 269

1.4.4.8 Aerial Photography

1.5 Conclusion

Management of soil-borne diseases can be successful and cost-effective if we have 278 a detailed information/knowledge regarding crop, disease history, resistant levels, 279 and environmental conditions. The increasing concern about nature and understanding 280

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the adverse effect of chemical use in the environment, non-chemical methods have 281 been developed for the prevention of soil-borne diseases. PGPR offers an attractive 282 alternative for sustainable approaches to agriculture. Credentials of diverse mecha-283 nisms involved in plant-rhizosphere microorganism interactions opened new possi-284 bilities to design strategies for improving crop yields. Subsequently, microbial 285 strains that have plant growth-promoting traits are improved with the use of a bio-286 technological approach to create transgenic strains with multiple mechanisms of 287 action. Comprehensive knowledge of plant-microbe interactions in the rhizosphere 288 is necessary before utilizing PGPR as biofertilizers which establish a sustainable 289 promotion of plant growth. Genes providing resistance to common and widely 290 occurring soil-borne fungal pathogens normally lack economic importance in most 291 cultivated plants. Alternatively, a strategy is evolved in plants that stimulate and 292 support specific antagonistic microorganisms groups from lots of deleterious, ben-293 eficial, and neutral species in the environment of the rhizosphere. Thus, PGPRs are 294 the most important antagonistic microorganisms selected since they are rich in 295 nutrients released from plant roots, and they provide the first line of defence against 296 soil-borne diseases (Weller et al. 2007; Cook et al. 1995). Identifying environmental 297 factors influencing the disease management capability of these PGPRs would cater 298 a base for enhanced alliance treatments of biocontrol with different control prac-299 tices that are environmental friendly, both under climate scenarios of the present and 300 future, making the farmers capable of managing soil-borne diseases and reducing 301 the use of chemical pesticides. 302

Acknowledgement The work is supported by a Network Project, MLP-1005, sponsored by the Council of Scientific and Industrial Research, Ministry of Science and Technology, Government of India, New Delhi. The authors are also thankful to the Director of CSIR-NEIST, Jorhat, Assam, for providing necessary facilities to carry out the work and DBT-BIF Centre, CSIR-NEIST, Jorhat, for providing the computational facilities.

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Chapter 2 Exploration of Secondary Metabolites for Management of Chickpea Diseases

Deepika Sharma, Sachin Gupta, Moni Gupta, and Baby Summuna

2.1 Introduction

Pulses are important components of the farming system both ecologically and 6 nutritionally (human and animal). Although pulse crops are more important due to its 7 nutritional value, there has not been any remarkable increase in area under its cultiva-8 tion and production during 1950–2010. However, a significant increase in area under a pulse crop cultivation and production has been recorded from 2010 to 2011 onward. 10 The production of pulse crops has increased by approximately 68% at 764 kg/ha dur-11 ing the year 2014 from 441 kg/ha during 1951. Over a dozen pulse crops are grown 12 annually all over the country in about 22-23 million hectares of area, producing 13 13-15 million tons of pulses. However, the prices of pulses have skyrocketed over 14 the last few years making life difficult for the poor peoples to afford. One of the 15 important reasons behind the price rise has been the fact that over the years, the pro-16 duction of pulses has declined due to the attack of diseases and insects. Around 17 8–10% of pulse crops are lost every year due to ravages of diseases alone costing 18 nearly 1000 crores to the National Exchequer. The reduction of losses caused by 19 diseases is, therefore, an important component of crop production technology. 20

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B. P. Singh et al. (eds.), *Management of Fungal Pathogens in Pulses*, Fungal Biology, https://doi.org/10.1007/978-3-030-35947-8_2

Among pulses, chickpea (Cicer arietinum L.) is the world's fourth significant 21 pulse crop after peas, common bean, and soybean. Chickpea is a rich supplement to 22 the cereals in developing countries due to its high nutritional value. Chickpea is 23 considered to be important because of the high level of protein content present in it, 24 i.e., about 40% of its weight. Moreover, chickpea crop has various health benefits 25 such as lessening the danger of cardiovascular diseases, cancer, diabetes, and other 26 health problems. Chickpea alone contributes the largest share of $\approx 85.64\%$ and 27 84.87% in India's export market of pulses during the year 2014-2015 and 28 2015–2016, respectively. This crop is mainly grown for its edible seeds which are 29 high in protein content and forage production (Yadav et al. 2011). India contributes 30 75% of the world's total production of chickpea (Mahajan et al. 2018), and the crop 31 accounts for 48% of the total pulse production in India (Anonymous 2015). 32

Chickpea's productivity remained stagnant from the last few decades due to the 33 susceptibility of cultivars to various soilborne diseases and insects. In temperate 34 regions, yield losses due to insects and diseases range from 5% to 10%, whereas in 35 tropical regions, it is 50% to 100% (Van Emden et al. 1988). In this context, disease 36 management in cereals and pulse crops is very important to alleviate the problem of 37 shortages of food to feed the ever-growing population and to improve food produc-38 tion efficiency. Many microbial pathogens including airborne and soilborne patho-39 gens have been reported to affect the chickpea crop. In chickpea, the development 40 of resistance against the soilborne fungal pathogens is the major research efforts 41 that have been made as compared with the foliar fungal pathogens. Foliar pathogen 42 management gained the least important because they don't cause much yield loss. 43 The list of common fungal diseases of chickpea is summarized in Table 2.1. 44

45 2.2 Role of Endophytic Bacteria in the Management of Plant 46 Diseases

Endophytes are microorganisms, both bacteria and fungi, that reside within the 47 plant host tissues without causing any harm to the host (Hallmann et al. 1997). 48 Many endophytic bacteria are being used as promising biocontrol agents against the 49 plant pathogens (Passari et al. 2015a, 2016, 2017). Endophytic bacteria colonize in 50 the internal tissues of the host plant for improving crop health and its protection 51 (Pavlo et al. 2011). Endophytic bacteria can promote the growth of the plant by 52 altering its physiology which includes osmotic regulation, increased uptake of cer-53 tain minerals, changes in stomatal responses, and nitrogen accumulation and metab-54 olism (Compant et al. 2005). AitBarka et al. (2002) reported that endophytic bacteria 55 trigger induced systemic resistance (ISR)-based plant growth promotion. 56

As have been reported by Pleban et al. (1995), *P. fluorescens* and *Bacillus* sp. effectively inhibited the growth of *Rhizoctonia solani* (46–56%, in bean), *Pythium ultimum*, and *Sclerotium rolfsii* (26–79%) plant pathogens. Experiments that have been conducted on various crops such as oilseed rape (Alstrom 2000), tomato (Chen et al. 1995), cotton (Liu et al. 1995), cucumber (Safiyazov et al. 1995), and

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Table 2.1

S. No	Disease	Causal organisms	Host	Symptoms
-:	Ascochyta blight	Didymella rabiei (anamorph: Ascochyta rabiei)	Chickpea	Chickpea First gray circular spots appear on leaves and pod that later turn dark brown with black borders. Black dots (pycnidia) are also present in advanced lesions in concentric rings
5	Fusarium wilt	Fusarium oxysporum f. sp. ciceris	Chickpea	Chickpea Leaves droop and appear pale and plants collapse and lie flat on the ground. Often, brown discoloration of internal root tissue is visible when the root splits into two vertically from the collar region
ю.	Powdery mildew	Leveillula taurica	Chickpea	Small patches of white powdery coating initially develop on both surfaces of the older leaves. Affected leaves turn purple and then die. Stems, young leaves, and pods are also covered with the powdery coating The plant may also lose leaves too early in the season and produce seeds that are smaller than normal
4.	White mold stem and crown rot of chickpea	Sclerotinia (S. minor, S. sclerotiorum, and S. trifoliorum)	Chickpea	Chickpea Visible white mycelium grows around the stem on the soil surface Black bodies (sclerotia) appear in various shapes and sizes on dead or dying chickpea stems Infected stems become pale in color, like bleaching, and the symptoms spread both upward and downward along the stems
5.	Black root rot	Fusarium solani	Chickpea	Chickpea General symptoms include yellowing and wilting of scattered plants, rotten root system, shedding of finer roots, and remaining roots turning black
ó.	Pythium seedling and root rot	Pythium sp.	Chickpea	Chickpea Root tissue may die and become discolored, leading to less branching and fewer feeder roots. Low emergence and seed rot could occur. Discoloration of the crown and hypocotyl's tissue may be observed as rotting progresses. Stunting of plants is common and some plants can die before flowering, leading to reduced yield
				(continued)

S. No	S. No Disease	Causal organisms	Host	Symptoms
.7	Downy mildew	Peronospora sp.	Chickpea	The disease is often exhibited in a few branches, leading to curled or twisted leaves and dwarfed tips The symptoms may appear on any aerial part of the plant with white mycelial patches appearing first on the lower leaf surfaces and then chlorotic to yellow spots on the upper surface Fine, dirty, pinkish tufts of fungal growth are often formed on leaf surfaces under cool and humid conditions, which may disappear when dry conditions take over, resulting in yellowing symptoms The chlorotic spots then become dark and brittle Stunting and bushy apical growth with small leaflets is typical The affected plants can also lose all their leaves, resulting in reduced yield and seed size
∞́	Gray mold(<i>Botrytis</i> stem and pod rot)	Botrytis cinerea	Chickpea	Chickpea Water-soaked lesions on any aerial parts of the plant are indicative of infection, with the growing tips and flowers being the most susceptible After some time, the lesions change to gray or dark brown and take on a fuzzy appearance as a result of the hairy sporophores and masses of conidia The stem may be girdled by the lesions and the leaves often turn into a rotting mass. The dead tissues could have tiny, black sclerotia that form on them If the disease moves to pods, the seed may not form or they may shrivel or become discolored Frequently flowers drop and the pod formation could be unfavorable, leading to low grain yields
9.	Rust	Uromyces ciceris-arietini hellow	Chickpea	Chickpea At first, small, round, brown spots (pustules) appear The pustules are sometimes surrounded by chlorotic halos. They often appear in a ring pattern. These may combine later and turn dark If the infection is severe the layes may drop off

peas (Sturz et al. 1999) by using endophytes such as *Pseudomonas* and *Bacillus* spp. 62 against the fungal pathogens provide evidence of plant growth protection and 63 promotion by the introduced endophytic bacteria. 64

2.3 Secondary Metabolite Production

Interspecies interactions in nature are often exhibited by microorganisms. 66 Competition for space and nutrients results in interspecies interaction prompting the 67 generation of secondary metabolites for improving their growth and development 68 (Passari et al. 2019; Calvo et al. 2002). Competition among microbes for space and 69 resources serves to be the major driving force for secondary metabolite production 70 (Oh et al. 2005). Studies on secondary metabolite production by microbes and their 71 application in suppressing plant diseases are gaining much significance in farming 72 systems (Gohain et al. 2019). Because of the increased concerns on environmental 73 pollution, pathogen resistance, and high plant security costs, secondary metabolites 74 produced by microbes have been developed as commercial pesticides and can be 75 used as an alternative to chemical fungicides. These metabolite products can also be 76 utilized as bactericides, fungicides, and insecticides (Singh et al. 2019). 77

2.3.1 Secondary Metabolites Associated with Rhizobacteria in the Management of Plant Diseases

Biocontrol using microbial antagonists is becoming a critically needed component 80 of plant disease management, particularly in reducing the risk of soilborne diseases 81 using potential microorganisms (Mishra et al. 2016; Nautiyal 2000; Meki et al. 82 2009). At present, control of soilborne and seed-borne pathogens has been achieved 83 mainly through the use of bacterial and fungal antagonists. Some rhizobacteria 84 especially *Pseudomonas* spp. and *Bacillus* spp. from the plant rhizosphere are effec-85 tive against the plant pathogens and also help the plants to acquire nutrients 86 (Gopalakrishnan et al. 2011). Moreover, the use of biological control agents is much 87 safer for the environment than synthetic or chemical pesticides. 88

Various mechanisms have been involved in antagonism, like cell wall-degrading 89 enzymes (pectolytic enzymes, cellulases, xylanases, and glycosidic hydrolases) and 90 siderophores that cannot only bind iron but also contribute to suppression of diseases 91 of the plant (Passari et al. 2015b; Deshwal et al. 2003). Kravchenko et al. (2002) sug-92 gested that siderophores produced by microbes may also enhance plant growth by 93 competitively inhibiting iron uptake system by fungal pathogens. Biological control 94 agents also produce different types of volatile and diffusible antifungal metabolites 95 which have the potential to suppress diseases caused by a fungal pathogen in various 96 pathosystems (Yang et al. 2009). Trichoderma sp. has greater potential to control 97 chickpea wilt under field as well as in polyhouse conditions, but its efficacy is not 98 almost the same everywhere (Kaur and Mukhopadhayay 1992). 99

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Rhizobacteria are ideal biocontrol agents that reside in the rhizosphere that give 100 frontline protection to the roots against the pathogen entry. Rhizobacteria have 101 received special attention as they are excellent root colonizers and have the potential 102 to induce plant's defense mechanism through the production of various 103 pathogenesis-related (PR) proteins (Kumar et al. 2010). Bacillus spp., a gram-posi-104 tive rhizobacteria, are potential biocontrol agents because of its abundance in the 105 rhizosphere and have the potential to produce active secondary metabolites (Milner 106 et al. 1996). Improvements in the plant disease management and productivity are 107 mainly mediated through pathogen antagonism, plant growth promotion, and stimu-108 lation of defense response in host plant against the pathogen. Plant growth-promot-109 ing rhizobacteria (PGPS) suppress the growth of soilborne phytopathogens through 110 the production of allelochemicals such as siderophores, antibiotics, and mycolytic 111 enzymes, viz., chitinases, β-1, 3-glucanase, proteases, lipases, etc. (Whipps 2001). 112 Rhizobacteria association with plant roots may increase plant yield through mecha-113 nisms that help in improved nutrient uptake, plant disease suppression, or produc-114 tion of phytohormone (Defago and Keel 1995). Plant rhizobacteria maintain a 115 symbiotic association with the surface of plant roots (Lutenberg and Dekkers 1999). 116 Decreased biocontrol activity may be associated with poor root colonization by 117 rhizobacteria (Schippers et al. 1987). 118

2.4 Production of Secondary Metabolites by *Pseudomonas fluorescens*

Use of P. fluorescens has revolutionized the field of biological control in suppressing 121 soilborne plant pathogens by producing antibiotics such as phenazine (Toohey et al. 122 1965), pyrrolnitrin (Burkhead and Geoghegan 1994), siderophores (Sakthivel et al. 123 1986), and phloroglucinol (Howell and Stipanovic 1980) that can help in controlling 124 wilt (Fridlender et al. 1993). The biocontrol activity of *Pseudomonas* spp. is mainly 125 mediated via the production of secondary metabolites and hydrolytic enzymes and 126 through competitive exclusion (Elasri et al. 2001). P. fluorescens produce various 127 secondary metabolites including antibiotic compounds that have been evaluated for 128 biocontrol activity against plant pathogens mainly by genetic techniques. Antibiotics 129 produced by *Pseudomonas* spp. inhibit metabolic activities and growth of pathogens. 130 Antifungal secondary metabolites, viz., 2,4-diacetylphloroglucinol, pyoluteorin, 131 phenazines, pyrrolnitrin, and HCN, contribute to the suppression of disease incidence 132 in various host-pathogen systems. Howell and Stipanovic (1980) studied the impor-133 tance of antibiotics secreted by P. fluorescens Pf-5 in the suppression of Pythium 134 ultimum causing damping-off in cotton seedlings. Various secondary metabolites 135 such as pyrrole-type antibiotics, phenazines, pyo-compounds (pyocyanin or pyover-136 dine), and indole derivatives have been characterized. Metabolites such as (amino-2-137 chloro-3-phenyl)-4-pyrrole-2-carboxylic acid, 7-chloroindole-3-acetic acid, and 138 3-chloroanthranilic acid produced by Pseudomonas aureofaciens at an early stage of 139 fermentation have been reported by Salcher et al. (1978). The two-component global 140

regulatory system GacS/GacA is known to control secondary metabolite production, 141 viz., pyoluteorin, 2,4-DAPG, pyrrolnitrin, phenazine, HCN, exoprotease, and chitinase compound as well as siderophores (Chin-A-Woeng et al. 2000). 143

Enzymes produced by pseudomonads can lyse fungal cell walls but not plants, 144 thereby preventing proliferation of plant pathogens. Hydrolytic enzymes, viz., chitin-145 ases, β -1,3-glucanases, lipases, proteases, etc., are produced by pseudomonads which 146 are known to digest fungal cell walls, thus using them as an energy source (Leah et al. 147 1991) and thus making them as potential biocontrol agents (Garbeva et al. 2004). 148 Synergistic effects have been observed on nodulation and plant growth of legume 149 crops by inoculation of mixtures of *B. japonicum* and *P. fluorescens* in soybean 150 (Li and Alexander 1988), R. leguminosarum and P. fluorescens strain F113 in pea 151 (Andrade et al. 1998), and Bradyrhizobium/Mesorhizobium and Pseudomonas sp. in 152 chickpea and green gram, respectively (Goel et al. 2000; Sindhu et al. 2002). 153

2.5 Mode of Action of Secondary Metabolites Produced by Pseudomonads

Biological control of plant pathogens by PGPR generally includes the production of 156 antibiotics (Haas and Defago 2005), HCN (Dowling and O'Gara 1994), cell wall-157 degrading enzymes, viz., chitinase, protease, β -1-3-glucanase, and lipase, which 158 can lyse the cell walls of the fungal pathogen (Chet and Inbar 1994). Characterizing 159 potential biocontrol candidates against soilborne pathogens is more important for 160 carrying out a successful action against plant pathogens in a dynamic and complex 161 rhizosphere condition. A brief description of the mechanisms through which pseu-162 domonads function to control plant pathogen and thus ultimately plant diseases is 163 described herewith. 164

2.5.1 Through Antibiotic-Mediated Suppression of Plant 165 Diseases 166

2.5.1.1 2, 4-Diacetylphloroglucinol (2,4-DAPG)

2,4-DAPG is a natural phenol specifically produced by gram-negative bacterium, 168 i.e., P. fluorescens, and is responsible for its biocontrol and antiphytopathogenic 169 properties. 2, 4-DAPG is the best-known phloroglucinol compound that includes 170 monoacetylphloroglucinol and diacetylphloroglucinol formed by uncharacterized 171 condensation of phloroglucinol and monoacetylphloroglucinol in a family of related 172 molecules (Mavrodi et al. 2001). Troppens et al. (2013) proposed that 2,4-DAPG 173 acts as a proton ionophore which dissipates the proton gradient across the mito-174 chondrial membrane. The uncoupling of ATP synthesis and respiration ultimately 175 leads to inhibition of plant pathogen which is the lethal effect of 2,4-DAPG. 176

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177 **2.5.1.2 Pyoluteorin** (**Plt**)

Pyoluteorin is an aromatic chlorinated polyketide compound mainly produced by *P. fluorescens* and is effective against oomycetes like *Pythium ultimum*. Bender et al. (1999) isolated pyoluteorin from *P. fluorescens* Pf-5 and *P. aeruginosa* for the first time. Howell and Stipanovic (1980) reported that its antimicrobial properties and its application suppressed the cotton damping-off in cotton seeds caused by pathogen *Pythium ultimum*.

184 2.5.1.3 Pyoverdine (Pvd) or Siderophores

Siderophores are low-molecular-weight extracellular compounds having a high 185 affinity for ferric ions (Fe³⁺) and bind with Fe³⁺ ions to form a ferric-siderophore 186 complex that cannot be utilized by the pathogen but the producing organism can use 187 it via specific receptors in their outer cell membrane. The ability to bind Fe³⁺ ions 188 provides a competitive advantage to microorganisms. The siderophores produced by 189 P. fluorescens play an important role in the promotion of plant growth (Kloepper 190 et al. 1980). Pseudomonas fluorescens is also known to produce siderophores which 191 are fluorescent and yellowish-green water-soluble pigments under iron deficit condi-192 tions (Sullivan and Gara 1992). Moreover, P. fluorescens is known to produce several 193 types of siderophores, i.e., salicylic acid, pyoverdine, and pyochelin (Dave and Dube 194 2000), and to control chickpea wilt by induced systemic resistance (ISR) via the 195 production of salicylic acid (SA) as a signaling molecule in a medium as well as in 196 the rhizosphere (Saikia et al. 2003). Induction of ISR via salicylic acid-dependent 197 pathway in chickpea plants by *Pseudomonas* spp. via the production of phenolic 198 compounds has been reported by Singh et al. (2003). 199

200 2.5.1.4 Phenazine (Phz)

Phenazines are nitrogen-containing heterocyclic compounds produced by 201 Pseudomonas spp. Phenazines are produced by certain members of the pseudomo-202 nads that are redox agents and are toxic to competing organisms. As has been 203 reported by Wienberg (1969), P. fluorescens produced phenazine derivative, i.e., 204 PCA (phenazine-1-carboxylic acid), whereas P. aureofaciens produced two phen-205 azine derivatives, i.e., PCA and 2- hydroxyphenazines. Almost all phenazine com-206 pounds exhibited a broad spectrum of antimicrobial activity against phytopathogens. 207 P. fluorescens is among the first few microbes from which phenazine compounds 208 were isolated and purified and reported to exhibit activity against fungal pathogens 209 (Gurusiddaiah et al. 1986). It is largely unknown how pseudomonads themselves 210 respond and survive in the presence of these compounds. 211

2.5.1.5 Pyrrolnitrin (Prn)

Pyrrolnitrin is an antifungal metabolite produced by members of the genus213Pseudomonas spp. Arima et al. (1964) first described phenyl pyrrole derivative used214as fungicide in agriculture. A four-gene cluster (prnABCD) responsible for pyrrol-215nitrin synthesis was first reported in Pseudomonas aurantiaca BL915, earlier iden-216tified as Pseudomonas fluorescens (Gross and Loper 2009).217

2.5.1.6 Hydrogen Cyanide (HCN)

Hydrogen cyanide is mainly produced by plant growth-promoting rhizobacteria 219 which plays an important role in biological control (Defago et al. 1990). HCN is 220 weakly acidic and partially ionizes in water to give cyanide ions (CN⁻). Cyanide 221 ions from HCN interfere with the enzymes of the respiratory system and inhibit the 222 action of cytochrome oxidase of the electron transport chain (Gehring et al. 1993). 223 The energy supply to the cell is disrupted which leads to the death of the invading 224 organism. It also inhibits the activity of enzymes and natural receptors via reversible 225 inhibition (Corbett 1974). As have been reported by Voisard et al. (1989), fluores-226 cent pseudomonads isolated from potato and wheat rhizosphere can produce HCN. 227

2.5.2 Through Cell Wall-Degrading Enzymes/Hydrolytic Enzymes

2.5.2.1 Chitinases

Chitinases fall into three classes, viz., endochitinases, 1,4-β-N-231 acetylglucosaminidases, and exochitinases or chitobiosidases, depending on the 232 mechanism of chitin degradation (Viterbo et al. 2002). Chitin is a polymer made 233 of N-acetyl-D-glucosamine (GlcNAc) units linked through β -1,4 glycosidic bonds 234 which are mainly degraded by chitinases. Nandakumar et al. (2007) reported the 235 production of chitinases by strains of P. fluorescens, viz., PF1, PB2, and FP7, on 236 the addition of chitin source in culture medium and maximum chitinase (31.2%)237 is recorded by strain FP7. The addition of chitin results in a significant increase of 238 chitinase activity (Nandakumar et al. 2007). P. fluorescens is known to have strong 239 antimicrobial activity against Rhizoctonia solani, Pyricularia oryzae, Xanthomonas 240 oryzae, and Fusarium oxysporum under in vitro and field conditions (Vidhyasekaran 241 et al. 2001; Nandakumar et al. 2001). Expression of enzymes, viz., chitinases and 242 β -1,3-glucanases, was reported in chickpea by Vogelsang and Barz (1993), and the 243 presence of four isoforms of these enzymes in stems and roots of chickpea crop 244 induced by wounding or by ethephon has been reported by Cabello et al. (1994). 245

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250 against *Fusarium* wilt under in vitro conditions.

251 2.5.2.2 Lipases

Lipase hydrolyzes triacylglyceride into fatty acids, di-acylglycerols, and monoacylglycerols and also catalyzes esterification and trans-esterification reactions (Fernandes et al. 2007). Prasad (2014) isolated lipase-producing microorganisms from different soil samples that are rich in lipid content like oil mills, and the maximum lipase activity by the isolate *Pseudomonas aeruginosa* was reported at pH 7 at 35 °C for 45 hours.

258 **2.5.2.3 Proteases**

Proteases are enzymes that hydrolyze proteins into its constituent amino acids. 259 These proteases are also known as proteolytic enzymes or systemic enzymes. 260 Proteases can hydrolyze proteins as long as they are not part of living cells. Normal 261 living cells are protected from lysis via the inhibitor mechanism. As have been 262 reported by Giri et al. (1998), the differential expression of proteinase inhibitors and 263 its accumulation are induced by wounding in Helicoverpa armigera against the 264 production of proteases, which is not sensitive to inhibition by protease and can 265 degrade them. 266

267 **2.5.2.4** β-1,3-Glucanases

Glucanases hydrolyze the glycosidic bond in glucan, a polysaccharide of several glu-268 cose sub-units. β-1,3-Glucan commonly known as laminarin is a polymer of D-glucose 269 that is arranged as helical coils in a β -1,3 configuration. Cell walls of fungi contain 270 about 60% laminarin that is mainly hydrolyzed by glucanases or laminarinase (Onsori 271 et al. 2005). Glucanases are mainly produced by microbes (fungi and bacteria) (Zhu 272 et al. 2008). Exo-β-1,3-glucanases break glucose residues into monosaccharide from 273 nonreducing ends, whereas endo-\beta-1,3-glucanases cleave polysaccharide chain into 274 oligosaccharides at random sites (Vazquez-Garciduenas et al. 1998; Vijayendra and 275 Kashiwagi 2009). β -1,3-Glucanases from bacterial and fungal sources are known to 276 be involved in the degradation of polysaccharides into its constituent sub-units and 277 used them as an energy source (Planas 2000). 278

Induction of phytoalexins and pathogenesis-related proteins, i.e., β -1,3glucanases, may be associated with a reduction in disease incidence in chickpea (Kuc 2006). Under in vitro conditions, the purified chitinases and β -1,3-glucanases exhibited antifungal activity against β FOC (Saikia et al. 2005) indicating their 282 direct effect on the pathogen growth. Harsha et al. (2012) reported the antifungal 283 activity of glucanase enzyme produced by *P. fluorescens* and its use as biocontrol 284 agent in agriculture. 285

2.5.2.5 Xylanases

Xylanases are enzymes that degrade linear polysaccharide, i.e., β -1,4-xylan, into xylose sub-units and hydrolyze hemicellulose which is the major component of plant cell walls. It helps in the degradation of plant matter into useful nutrients by microorganisms, viz., fungi, bacteria, and yeast. The filamentous fungi are the commercial source of xylanase (Beg et al. 2001). 287 289 289 290 290 291

2.5.3 Production of Plant Growth-Promoting Substances (PGPS)

2.5.3.1 Indole Acetic Acid (IAA)

Indole-3-acetic acid is a naturally occurring phytohormone (auxins) and is commonly295produced by plant growth-promoting rhizobacteria (Barazani and Friedman 1999).296IAA is involved in the root initiation, enlargement of the cell, and cell division297(Salisbury 1994). Biofertilizing PGPR plays an important role in the production of298IAA and its implications in plant growth promotion (Passari et al. 2015a; Vessey2902003). IAA is believed to enhance root growth, resulting in a greater area of the root300surface, and thus helps the plants to acquire more nutrients from the rhizosphere.301

Barea et al. (1976) isolated bacteria from the rhizosphere which can produce 302 IAA, gibberellins, and cytokinins and found that out of the total, 17 isolates belong 303 to the Pseudomonas spp. Production of IAA and GA by Pseudomonas striata was 304 also reported by Sattar and Gaur (1987). It has been reported that IAA production is 305 the inherent mechanism of PGPRs like *Pseudomonas* spp. (Mazumdar et al. 2007). 306 As have been reported by Kumar et al. (2007), P. fluorescens strain Pf4–99 is capa-307 ble of producing IAA in culture medium and is most effective in the improvement 308 of chickpea crops under controlled greenhouse conditions and natural field condi-309 tions. Rhizobacteria from the roots of legume crops such as pea, lentil, and chickpea 310 are capable of producing IAA (Hynes et al. 2008). 311

2.5.3.2 Gibberellins (GA)

Gibberellic acid commonly known as gibberellins is a phytohormone mainly found in plants and is capable of promoting plant growth and cell elongation. It helps in the stimulation of cells of germinating seeds to produce mRNA molecules encoding for 315

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hydrolytic enzymes. It is associated with the modification of plant morphology by the
elongation of plant tissue (Salisbury 1994). The evidence for the production of GA by
PGPRs has been provided by Gutierrez-Manero et al. (2001). The production of plant
hormones such as IAA, GA, and cytokinins by PGPRs played a direct role in plant
growth promotion and also helps in nitrogen fixation (Patten and Glick 1996).

As have been reported by Siddiqui et al. (1998), P. fluorescens can control wilt 321 disease in pigeon pea caused by *H. cajani* when used alone or in combination with 322 pesticides. *Pseudomonas* spp. have the potential to increase plant growth, nodula-323 tion in leguminous plants, and phosphorus solubilization and decrease nematode 324 multiplication, thereby suppressing wilting in infected plants. Saikia et al. (2004) 325 found that *P. fluorescens* isolated from rhizosphere of broad bean has antagonistic 326 activity against fungal pathogens, viz., Rhizoctonia solani and Macrophomina pha-327 seolina, and also reported the suppression of *Fusarium* wilt and charcoal rot in 328 chickpea by P. aeruginosa strain RsB29. 329

2.6 Role of Biocontrol Agents in Induced Systemic Resistance (ISR) and Systemic Acquired Resistance (SAR)

Induced systemic resistance is the enhanced defensive ability developed within the 333 host plant by nonpathogenic forms of rhizobacteria (Van Loon et al. 1998). ISR in 334 carnation plants was induced by P. fluorescens strain WCS417r against F. oxyspo-335 rum f. sp. dianthi (Van Peer et al. 1991). In cucumber plants, it was induced by 336 rhizobacterial strains against the anthracnose caused by Colletotrichum orbiculare 337 (Wei et al. 1991). ISR mediated through rhizobacteria resembles pathogen-mediated 338 systemic acquired resistance (SAR) that render resistance in uninfected plant parts 339 against plant pathogens (Van Wees et al. 1997). Bacillus spp. and Pseudomonas spp. 340 are the most widely studied rhizobacteria that induce the ISR (Van Wees et al. 2008). 341 ISR is induced by PGPR or nonpathogenic rhizobacteria, whereas SAR is triggered 342 by a localized infection. ISR and SAR are mediated through a different set of signal-343 ing pathways. SAR is mediated through salicylic acid (SA) pathway, whereas two 344 signaling pathways, i.e., jasmonic acid (JA) and ethylene (ET) pathways, are 345 involved in ISR (Van Loon et al. 1998). The defense responses are induced by these 346 signaling molecules when they are applied exogenously (Ryals et al. 1996). ISR-347 mediated resistance is significantly less than that of SAR-mediated resistance (Van 348 Loon 2000). ISR and SAR jointly provide a better resistance response which indi-349 cates that they act in coordination in inducing the resistance response against patho-350 gens (Van Wees et al. 2000). 351

The high concentration of ET and JA is a sign of defense response in infected plants (Mauch et al. 1984). In *Arabidopsis*, JA and the ET response mutants (jar1 and etr1) were tested in the induction of ISR against *P. syringae* pv. tomato by Pieterse et al. (1998) and found that these mutants were unable to induce ISR-mediated resistance in tomato upon colonization of the roots by rhizobacteria WCS417r. Methyl jasmonate (MeJA) and the ethylene precursor 1-aminocycloprop

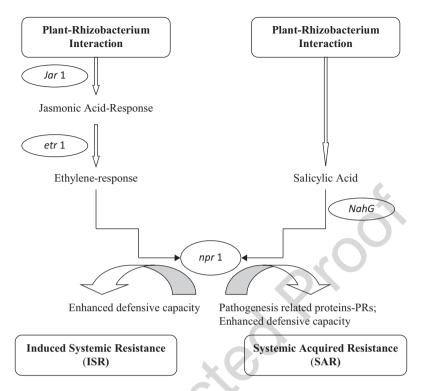


Fig. 2.1 Signal transduction pathways leading to rhizobacteria-mediated induced systemic resistance (ISR) and pathogen induced systemic acquired resistance (SAR) in *Arabidopsis thaliana*. (Source: Modified from: Van Loon et al. 1998).

ane-1-carboxylate (ACC) promote resistance against *P. syringae* pv. tomato in sali-358 cylic acid non-accumulating (NahG) plants. MeJA-mediated resistance is blocked 359 in etr1-1, npr1-1, and jar1-1 plants, while ACC-mediated resistance is affected in 360 npr1–1 and etr1–1 plants, but not in jar1–1 plants. Thus, WCS417r-mediated ISR 361 follows JA- and ethylene-mediated signaling pathways, and these signaling mole-362 cules are successively coordinated to induce a defense mechanism like SAR which 363 is regulated by NPR1 (Pieterse et al. 1998). Signal transduction pathways leading to 364 rhizobacteria-mediated ISR and pathogen-mediated SAR in Arabidopsis thaliana 365 are summarized in Fig. 2.1. 366

2.7 Future Perspective

The area under the legume crop cultivation and its production has not been 368 increased in the last few years. Fungal pathogens and pests are recurrent problems 369 for pulse crops. The chickpea pulse crop is widely grown under diverse climate 370 conditions ranging from temperate to subtropical climates. The exploitation of the 371

372 plant-microbe interaction will benefit us to identify novel secondary metabolites

having antagonistic activity against disease-causing pathogens. Biological control

agents are commercially available now, and these are formulated to control diseases

caused by pathogens through nutrient competition and increasing resistance in plants. Biocontrol agents could be used to reduce the intensive use of agrochemicals

plants. Biocontrol agents could be used to reduce the intensive use of agrochemicals and synthetic pesticides as they contain potential active ingredients. Thus, a strategy

and synthetic pesticides as they contain potential active ingredients. Thus, a strategy including the exploitation of secondary metabolites by biocontrol agents needs to be

developed for integrated disease management.

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Chapter 3 Integrated Fungal Foliar Diseases of Arid Legumes: Challenges and Strategies of Their Management in Rain-Fed Areas

Udaya Kumar Vandana, P. Bijoya Singha, Sharmista Chakraborthy, and P. B. Mazumder

3.1 Introduction

Grain legumes play a major role in improving food and nutritional security of farm-8 ers and populations, covering up to 45% of arid and semiarid regions of the world 9 (Sprent and Gehlot 2010). Some of the globally important grain legumes which are 10 grown worldwide and economically important are chickpea (Cicer arietinum L.), 11 lentil (Lens culinaris Medik), cowpea (Vigna unguiculata (L.) Walp), and faba bean 12 (Vicia faba L.) (Cernay et al. 2016; Raseduzzaman and Jensen 2017). These legumes 13 are severely damaged by numerous plant pathogens from bacteria to fungi and 14 viruses to nematodes causing economic losses globally (Jones et al. 2013). Among 15 these pathogens, fungi are the largest group that affects all parts of the plants, 16 majorly foliar parts. Fungal foliar diseases such as Ascochyta blight (Ascochyta 17 rabiei) and Botrytis gray mold (Botrytis cinerea) affect chickpea (Cicer arietinum). 18 In lentils, Ascochyta blight is caused by Ascochyta lentis and rust is caused by 19 Uromyces viciae-fabae Pers. Anthracnose (Colletotrichum lindemuthianum Sacc. & 20 Magn.) and Cercospora leaf spot (Cercospora canescens Fellis & Martin and 21 Cercospora cruenta Sacc.) affect cowpea, respectively. Chocolate leaf spot (Botrytis 22 fabae and B. cinerea) and rust (Uromyces viciae-fabae) affect faba bean (Girish 23 et al. 2019). Challenges in sustainable management are lack of understanding of 24 integrated pest management while adopting biopesticides in underdeveloped coun-25 tries conquer the disease and are not effective as chemical fungicides and hence the 26 farmers are not willing to use the products (Parsa et al. 2014; Peshin et al. 2009; 27 Vandana et al. 2017). The integrated disease management (IDM) of legumes in a 28 particular area depends upon the genetic resistance and other components of disease 29 management (Coakley et al. 2002; Isman 2000). IDM program lies in identifying, 30 evaluating, merging, and locating distinct components (D'Mello et al. 1998; 31

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B. P. Singh et al. (eds.), *Management of Fungal Pathogens in Pulses*, Fungal Biology, https://doi.org/10.1007/978-3-030-35947-8_3

arid legumes, affected by important fungal foliar diseases, and strategies of IDM for
 the control of fungal diseases. Approaches to sustainable management including
 cultural and physical practices, exploitation of host resistance, and protection with
 a synthetic fungicide are also discussed in the chapter.

37 3.2 Chickpea

Chickpea is a staple grain legume, the most prevalent food legume in the world. It 38 serves as a major source of human diets rich in nutrients (protein) and high-quality 39 crop residues for animal feed as well. Some of the crucial facets of chickpea are to 40 maintain the fertility of soil via biological nitrogen fixation, furthermore in contrib-41 uting to the sustainability of cropping structures by approaching practice like cereal-42 legume rotations. The significance of chickpea among temperate pulses is its 43 tolerance to heat and drought in low fertility soils. Some of the important diseases 44 affecting chickpea crop are: 45

46 3.2.1 Ascochyta Blight (Ascochyta rabiei)

Ascochyta rabiei comes under the most devastating fungal diseases of chickpea in
numerous countries (Pande et al. 2005), favoring disease development and spread
particularly by environmental conditions (cool and wet weather).

50 3.2.1.1 Diagnosis and Epidemiology

The fungal pathogen outbreaks parts above the ground of the plant. Fungi thrive on 51 infected seeds, crop residues, and volunteer seedlings starting from one growing 52 season to the next. When conditions are favorable and the prime source of inoculum 53 is a seed, some dark brown lesions develop in the stem. When it comes to the air-54 borne spores, initial indications emerge as tiny necrotic specks on aerial parts of the 55 primordial leaves. These specks under cool and wet conditions rapidly become 56 enlarged and cohere, with the blighted portions having pycnidia formed all over the 57 plant. In a susceptible culture, the necrosis progressively grows down, thereby kill-58 ing the infected plant. Lesions are inversely ovate to extend and bear pycnidia on the 59 stems and petioles. Generally, there is a breakage in stems and petioles due to engir-60 dle. The round lesions develop on pods with some pycnidia, generally arranged in 61 concentric rings, where the pod wall is penetrated by a fungus, infecting the seed on 62 which lesions develop. Crop infection may emerge from seed-borne inoculum and 63 from conidia of rain-splashed or windborne ascospores from infested parts. It was 64 displayed that the teleomorph (the sexual reproductive stage of any fungus of phyla 65

Ascomycota and *Basidiomycota*) has a crucial portion in the epidemiology of the 66 infection and played important role in controlling the disease in Spain and the 67 United States (Kaiser et al. 2011). 68

3.2.1.2 Control

Disease control can include approaches such as burying the harvest debris, abolition 70 of seed-borne inoculum, and establishing disease-resistant varieties. ICARDA and 71 ICRISAT released numerous blight-resistant cultivars (Nene et al. 2011) which 72 involve methods such as seeding blight-free seed, application of foliar fungicides 73 and seed treatments and rotation of crop for 3 years, controlling diseased debris, and 74 finally implanting blight-resistant varieties. 75

3.2.2 Botrytis Gray (Botrytis cinerea)

Botrytis grey mould is the common plant diseases in India, Nepal, Pakistan, and Bangladesh which is caused by *Botrytis cinerea*, which is reported to reduce yields in Australia and Argentina as well (Pande et al. 2006). Favorable conditions for the pathogen can substantially lead to major yield loss (Rashid et al. 2014).

3.2.2.1 Diagnosis and Epidemiology

A minimum of five diverse pathogen types of B. cinerea were identified (Kaiser 82 et al. 2011). Furthermore, studies in pathogenic variability are mandatory. The 83 inceptions of infection take place in the lower portions of the infected plant initially 84 and later, under favorable condition, extend to the upper leaves. Often, there is a 85 development of soft rot, and fungus sporulation can be noticed at the plant basal part 86 in the seedlings which were seed infested with B. cinerea. Plant parts cultivated 87 symptoms like dark-colored lesions mainly shielded with moldy fungal develop-88 ment. Changes such as complete engirdling of stems by lesions and breaking off of 89 tender branches at the site where gray mold causes decomposition can be observed. 90 Damaged leaves and flowers eventually turn into a decaying mass, and pods almost 91 disappear or left with less quantity, withered spores (having lost all moisture). 92 Immature seeds develop grayish-white mycelium. B. cinerea has a broad range of 93 host, there is almost always a presence of the inoculum in the environment, and it 94 can survive with other crops and weeds. Kaiser et al. (2011) conducted some experi-95 ments in a glasshouse, where they found that the fungus is being potential on 8 dif-96 ferent crop species and 21 weed types. Feasibility of seed-borne source greatly 97 reduces when kept in room storage. However, there is a prompt diminution in the 98 sustainability of the fungus throughout stowage. The disease is mainly favored by 99 moist and moderate temperatures. The respective significance of seed-borne inocu-100 lum and additional causes needs to be explored in different parts. 101

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102 3.2.2.2 Control

Voluminous lines of chickpea with moderate resistance to gray mold were found 103 although lines of resistance with increased levels have not been found. They found 104 22 lines with valuable resistance out of 8500 accessions evaluated. Despite a huge 105 degree of flower infection, numerous chickpea lines produce good yields (Kaiser 106 et al. 2011). The severity of gray mold can be reduced by the late sowing of chick-107 peas, but it leads to reduced yields in normal years (Kaiser et al. 2011). Gray mold 108 can be efficiently reduced by seed treatment trialed by three sprays of carbendazim 109 (Kaiser et al. 2011). The effectiveness of foliar sprays of vinclozolin was reported 110 as well (Kaiser et al. 2000). Seed treatment with the spraying of triadimefon, car-111 bendazim + thiram, mancozeb, or triadimefon was useful in checking seed-borne 112 infection ([>]94%) (Kaiser et al. 2011) followed by observation 50 days post-sowing 113 or at the advent of indications which resulted in comprehensive control of both pri-114 mary and secondary infections. However, at present, disease resistance at a high 115 level is not found in chickpea cultivars. Therefore, moderately resistant cultivars are 116 necessary to be developed in combination with an integrated disease management 117 program with critical chemical use, and improved cultural procedures appear to 118 minimize crop loss devastated by gray mold. 119

120 **3.3 Lentil**

Lentil is regarded as one of the important legumes considering its nutritive value. 121 It is an outstanding source of molybdenum and folate and also serves as a rich 122 source of copper, phosphorus, manganese, and dietary fiber (Hall et al. 2017). It 123 serves as a staple food in countries like India, Canada, Turkey, the United States, 124 and Nepal. According to the USDA National Nutrient Database, 353 calories can 125 be produced from 100 g of raw lentil (Agriculture 2014). Lentils are rich in water 126 (8%), carbohydrates (63%), dietary fiber (11%), protein (25%), and fat (1%). They 127 are also rich in phosphorus (40% DV), iron (50% DV), zinc (35% DV), folate 128 (120% DV), thiamin (76% DV), pantothenic acid (43% DV), and vitamin (42% 129 DV (Faris et al. 2013). 130

131 3.3.1 Rust (Uromyces viciae-fabae Pers.)

One of the serious diseases of lentils is caused by rust (*Uromyces fabae*), which is particularly damaging the crops in countries like India, Chile, Pakistan, Ethiopia, Morocco, and Ecuador (Kaiser et al. 2011).

3.3.1.1 Diagnosis and Epidemiology

Environmental conditions (temperatures varying between 20 and 22 °C and wet 136 weather) favour the initial infection and disease development, resulting in crop loss. 137 All the green plants, including plant parts and pods, are infected. Early symptoms 138 of yellowish-white pycnia (spermatogonia) and aecia (individually or in small 139 groups) appear on the undersurface of pods and leaflets and eventually turn brown. 140 Dark brown to black teliospores are observed to be developed on leaves and on 141 stems and petioles. Crop genera including Lathyrus, Lens, Pisum, and Vicia are 142 infected by the pathogen majorly. Before the establishment of a favorable and effec-143 tive pathogenic relationship, there is a necessary association between the patho-144 genic cell surfaces and its host. Following the contact between the two faces, 145 pre-penetration is a basic necessity for the events that lead to disease development 146 (Negussie and Pretorius 2012). Many pathogenic fungi such as U. viciae-fabae pro-147 duce substances that are generally present in the extracellular matrix which facili-148 tate adhesion of gremlins and ungermlins spores. 149

Moreover, to extracellular matrix materials, adhesion pads of germinating urediniospores recognized to aid in the addition to the spores on the surface of the host by intensifying the part of interaction for substratum (Negussie and Pretorius 2012). 152 The fungus thrives on infested lentil debris from season to season via teliospores. 153 The diseased debris, when mixed with seed, became infected (Negussie and Pretorius 2012). During the growing season, aeciospores have a vital role in spreading the infection. 156

3.3.1.2 Control

Numerous approaches are attempted to control the disease which includes field 158 sanitation, crop rotation, seed treatment, and use of foliar fungicides (Nene et al. 159 2011), and most resistance variety (Kaiser et al. 2011). ICARDA identified novel 160 sources of rust resistance to one or more diseases by screening lentil germplasm in 161 various parts of the world, namely, in Ethiopia, Morocco, and Pakistan, where rust 162 epidemics are frequent. There have been several lines that have moderate resistance. 163 Seed treatment with diclobutrazol compels in annihilating seed-borne inoculum 164 effectively (Nene et al. 2011), and it was also reported with the efficiency of foliar 165 sprays with mancozeb. However, some new inputs in this area of research are 166 required to control this disease. 167

3.3.2 Ascochyta *Blight* (Ascochyta lentis)

Ascochyta blight caused by Ascochyta lentis, is one of the most devastating fungal 169 diseases that restrains lentil production. It was first reported from the USSR (Nene 170 et al. 2011). 171

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172 3.3.2.1 Diagnosis and Epidemiology

A favorable environmental condition such as cool and wet weather leads to disease 173 development and spread of A. lentis. It is a seed-borne disease that affects all the 174 aboveground parts of the host plant, creating tiny, spherical gray- to dark-colored 175 lesions along with the dark margins in the vicinity of lacerations on the leaflets. Tiny 176 dark brown to black pycnidia appear in the abrasions on leaflets and pods. Pedersen 177 et al. (1994) reported that although under rain-splashed condition, it leads to conidia 178 dispersion, conditions such as wetness periods of 1-2 days will lead to infection 179 under favorable temperature (10–15 °C). The dispersion of pathogens may also take 180 place via wind-blown infected leaflets and seeds. The fungus thrives in crop debris. 181 Disease epidemiology is needed to be investigated by researchers. 182

183 **3.3.2.2 Control**

184 The strategies for controlling *Ascochyta* blight in economical and sustainable ways

can be via resistance breeding and cultural practices. Practices including crop rotation, early seeding for evasion of damp weather at harvest, and employing diseasefree seed can be applied to minimize crop losses (Nene et al. 2011). Numerous

fungicides are evaluated to control seed-borne infection with thiabendazole, benomyl, carbathin, and carbendazim having effective manifold degrees.

190 **3.4 Cowpea**

Cowpea (*Vigna unguiculata*) is a widely adapted legume. Cowpea has important
nutritional content; thus, it is widely consumed by millions of people. The crop is
cultivated in warm regions of the world on around seven million hectares (Adebanjo
and Bankole 2004). Cowpea is produced in Asia, in North America (southeastern
and southwestern regions), and largely in semiarid northeastern Brazil.

196 3.4.1 Anthracnose (Colletotrichum lindemuthianum Sacc. 197 & Magn)

Cowpea is prone to outbreak by several pathogens such as anthracnose from seeding
to harvest affected by *Colletotrichum lindemuthianum* (Saccardo and Magnus)
Briosi and Cavara, which is first recorded in Nigeria in 1969 (Adebanjo and Bankole
2004). Anthracnose causes a 50% yield loss in cowpea under wet and damp conditions in the regions ranging from Nigerian rainforest belt to other parts of Nicaragua;
Eastern, Western, and Southern Africa; and Brazil (Williams 1975).

3.4.1.1 Diagnosis and Epidemiology

The disease is prompted to spread under cool, wet weather and particularly damage 205 monocropped cowpeas and affect all aboveground plant parts. Individual lesions 206 vary in shape, generally, from biconvex to circular, and color, turning tan to dark. 207 Lines with high susceptibility can develop lesions that spread largely in number, 208 rapidly leading to coalescing stems and twigs and petioles engirdle. Later, they 209 appear almost completely brown. Resistant lines appear to have relatively small nar-210 row lesions than hypersensitive lines which range from tiny necrotic flecks to shiny 211 reddish-brown lenticular lesions of 5 mm long without sporulation. About 40% of 212 the pathogen is seed-borne in cowpea (Adebanjo and Bankole 2004). Reduction of 213 35–50% in grain yield of a highly susceptible line has been measured in a monocrop 214 culture when introduced with the disease at an initial stage during crop growth 215 (Adebanjo and Bankole 2004). Nonetheless, the disease breakthrough is taking a 216 relatively prolonged time in mixed-cropped cowpeas. 217

3.4.1.2 Control

The most endeavoring approach to control the disease is the utilization of host plant 219 resistance. The cowpea germplasm is collected and screened at IITA where two 220 types of resistance have been identified: (1) hypersensitive reactions make cowpea 221 lines functionally immune, and (2) field resistance allows less or null anthracnose 222 development in nurseries. Nature along with inheritance of this resistance is studied 223 at IITA to produce cowpea with varying degrees and high level of stable resistance 224 to anthracnose. 225

3.4.2 Cercospora Leaf Spot (Cercospora canescens Fellis & 226 Martin and Cercospora cruenta Sacc) 227

Cercospora leaf spot is a foliar fungal disease that affects a vast number of legumes
including cowpea. *Cercospora canescens* and *Cercospora cruenta* (Williams 1975)
both cause *Cercospora* leaf spot. They cause severe loss of yield of <40% in cowpea.
Although there are not only a variety of resistant lines but also susceptible ones, there
is a necessity to identify suitable varieties for cultivation (Booker and Umaharan 2007).
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3.4.2.1 Diagnosis and Epidemiology

The initial symptom of *Cercospora* leaf spot in cowpea is the development of tiny, 234 light-colored spot (almost yellow) which later turned to bronze and then dark 235 grayish circular spot. The fungus produces windborne spores in bulk on the abaxial 236

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surface of leaf which gives the spots a gray to dark powdery appearance. Symptoms
are not usually observed during flowerin4g time. *C. cruenta* occurs in the leaf with
more intensity, as it occurs in all seasons when the susceptible lines are planted.
Both species are found to be sporulating on pods as well, favored by wet weather
(Ratnadass et al. 2012). Yield reductions of cowpea grain attributed by *C. canescens*and *C. cruenta* are about 20% and 40%, respectively, by IITA (1973) (Vaghefi
et al. 2018).

244 3.4.2.2 Control

Crop practices such as intercropping can be applied which includes planting cow-245 peas in alternate rows along with another suitable nonlegume crop, such as maize, 246 which can limit or eradicate the spread of disease within a field. Chemical approaches 247 include the fungicide's utility to control disease outbreaks when favorable condi-248 tions enable disease establishment. The disease develops on older leaves, but early 249 crop survey is difficult to monitor due to complication in distinguishing symptoms 250 from other types of damage. Mancozeb is applied with a maximum of 2-3 applica-251 tions subsequently after crop flowering and pod development per planting season 252 (Devasirvatham et al. 2012). 253

254 3.5 Faba Bean

Faba bean (Vicia faba L.) is another important legume seed rich in protein which 255 can adapt to most of the European climatic conditions. Several faba bean cultivars 256 are characterized by varying amount of diets of nutritional value which contain 257 high and/or reduced levels of tannins and a combination of high or low levels of 258 vicine and convicine (VC) (Crépon et al. 2010). This nutritional value was exam-259 ined in ruminants and monogastric animals. Faba bean has common usage as a 260 staple food in many emerging countries including countries of Asia and Africa 261 (Gago et al. 2014). 262

263 3.5.1 Chocolate Leaf Spot (Botrytis fabae and Botrytis cinerea)

Chocolate leaf spot of faba bean is caused by *Botrytis fabae* and *B. cinerea*. The disease affects many parts of the world, reducing faba bean yields (Sahile et al. 2008).
Serious epidemics were reported in the UK, Tunisia, and Syria (Nene 2003). Fifty percent of faba bean yield loss has been reported in Egypt which is due to chocolate leaf spot and rust diseases, occurring regularly together (Jensen et al. 2010).

3.5.1.1 Diagnosis and Epidemiology

Generally, symptoms include brown-colored spots on the leaves, strips on the stems 271 and petioles, comprehensive darkening of the infected plant, and ultimately death of 272 the infected plant (Motilal and Sreenivasan 2013). The following symptoms are 273 linked to considerable yield losses during extended rainy periods. The age of faba 274 bean influences the severity of chocolate leaf spot (Plantegenest et al. 2007). When 275 observed under artificial conditions, relatively 7-week-old plants had shown more 276 severe disease development than 2-week-old plants. The optimum temperature for 277 infection is around 20 °C and relative humidity is 85% (Nene et al. 2011). 278

3.5.1.2 Control

The method of breeding disease-resistant cultivars is mostly practiced. Two-cycle 280 procedure has been followed at ICARDA (Nene et al. 2011). In the first cycle, a 281 broad mixture of *B. fabae* isolates with germplasm lines was evaluated, which were 282 collected from leaves of naturally infected plants from the local susceptible cultivars 283 of Syria (Sari et al. 2018). A couple of coalesced-sporulating lesions were developed 284 in the resistant lines, which were detected in the first cycle and then mixed with the 285 isolates collected from such abrasions. Isolates were later eventually inoculated back 286 in the post-screening cycle to the progenies of the resistant lines identified in the first 287 cycle. Subsequently, the outcome of these screenings gave three lines identified as 288 possessing wide-based and stable resistance (Davidson et al. 2016; Sari et al. 2018). 289

3.5.2 Rust (Uromyces viciae-fabae)

The rust occurring in most faba bean-growing areas is triggered by Uromyces291viciae-fabae (syn. U. fabae). It is considered to be the most severe constraint of292faba bean in Egypt and is conjoint all over the Mediterranean province. Rashid and293Bernier (1991) reported faba bean losses of up to 50%.294

3.5.2.1 Analysis and Epidemiology

Rust of faba bean is homoecious and two stages are commonly evident: uredial and 296 teleuto. The development of red pustules occurs on either leaves, stems, or petioles, 297 which exhibited small circles. However, the teleutopustules arise on the leaves, and 298 they are commonly present on the stems. They appear to be brown to black. The rust 299 in faba bean crops results in defoliation. The pathogen is also known to infect pea, 300 lentil, and wild-cultured species of Vicia and Lathyrus. And detailed epidemiologi-301 cal studies are necessary (Eshetu et al. 2018; Hanounik and Hawtin 2011; Zhang 302 et al. 2019). 303

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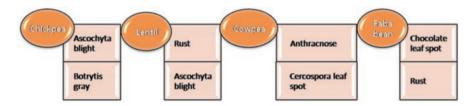


Fig. 3.1 An overview of legumes mentioned in the chapter along with their fungal foliar diseases

	-	e	e e	
Sl. no.	Legumes	Fungal diseases	Disease-causing agent	References
1.	Chickpea	Ascochyta blight	Ascochyta rabiei	Pande et al. (2005)
		Botrytis gray mold	Botrytis cinerea Pers. ex Fr.	
2.	Lentil	Rust	<i>Uromyces viciae-fabae</i> (Pers.) Schroet	
		Ascochyta blight	Ascochyta lentis Bond & Vassil	
3.	Cowpea	Anthracnose	Colletotrichum lindemuthianum (Sacc. & Magn.)	
		Cercospora leaf spot	Cercospora cruenta (Sacc.)	
4.	Faba bean	Chocolate leaf spot	Botrytis fabae and B. cinerea	Nene et al. (1988)
		Rust	Uromyces viciae-fabae	

 Table 3.1
 Fungal diseases of legumes and their causal organisms

304 **3.5.2.2 Control**

Practical methods can be applied by utilization of resistant cultivars. There is still an ongoing work at ICARDA and in Canada, where many lines were identified to be resistant. When tested via international nurseries, most of these culture lines were evident with only location-specific resistance. The exceptional case is the resistance of BPL 1179–1 (in Syria, Egypt, and Canada) (Cetin et al. 2002) (Fig. 3.1 and <u>Table 3.1</u>).

311 3.6 Disease Management of Fungal Foliar Disease

Among the paramount food legumes that are grown globally, the one found in cool season is *Cicer arietinum L.* (chickpea), *Lens culinaris* Medik. (lentil), and *Vicia faba* (faba bean), whereas the one found in warm season is *V. unguiculata* L. (cowpea). Organic pressure markedly minimized the yield of those legumes noticeably. Fungi and viruses are the massive deteriorating factors that affect plants at different growth phases of the legumes (Chen et al. 2006; Ghanem et al. 2015; Walley et al. 2007). Foliar diseases like gray mold and *Ascochyta* blight spawned via varieties of Botrytis and Ascochyta are of vast significance to faba bean, lentil, and chickpeas.319In lentil, the genus Stemphylium induces foliar disease and in cowpea, Septoria species gives rise to leaf spots. Based on published reports, it is found that approximately 45 viruses infect legumes worldwide, but only a few are of economic threat320with esteem to certain regions (Gaur et al. 2012; Muehlbauer et al. 2006; Rodda et al. 2017).324

In this chapter, a great effort has been made to mark the management of foliar 325 disease of food legumes in both seasons. A successful integrated disease manage-326 ment scheme for economically prime foliar diseases of cowpea, chickpea, faba 327 bean, and lentil has been explored with an allusion to the investigation results on 328 biology, pathogen, and etiology. Integrated disease management strategy (IDM) is 329 the process in which legumes are safeguarded from the yield-reducing consequences 330 of the infectious agent and providing the after commercial insignificance. In this 331 particular system, a discrete constituent of disease controlling plant resistance, 332 backwoods practices, sensible use of fungicides, etc., have to be specific or 333 complementary. 334

3.6.1 Foliar Disease Management of Food Legumes

Throughout research and development, the prime emphasis to inhibit legume infections is laid upon host resistance and chemical management. The principle of IPM 337 (integrated pest management) has been taken into consideration by IDM (integrated disease management) (Abdullah et al. 2015). The IDM of legumes in a particular 339 area depends upon the genetic resistance, in addition to other components of disease management. Based on the environment, IDM may require a lot of or different components to inhibit foliar diseases (Hema et al. 2014). 342

In the production of food legumes, the elements of IDM are cataloged in this 343 fashion: 344

A host plant resistance
Disease pressure
Biotic control
Agronomic practices
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3.6.1.1 Cool Season Legumes

Chickpea The most common foliar diseases in chickpea are *Ascochyta* blight and 350 *Botrytis* gray mold (BGM). This decrepitude was appraised by various workers. 351 Chickpea diseases and their management have been discussed in detail by Varshney 352 et al. (2012). IDM practices are economically vital in potent control of AB 353 (*Ascochyta* blight) and BGM (*Botrytis* gray mold). According to studies in specific 354 areas, several provenances of reluctance to AB were found and the developed 355

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genotypes aid to grow the yield during winter in Mediterranean provinces, resulting
in the twofold construction potential of chickpeas. And under a high disease pressure, a sufficient level of genetic resistance to BGM is not handy in the cultivated
genotype (Tribe et al. 2006). Therefore, the use of handy management options by
IDM is vital to mitigate the disease and reduce yield losses.

A union of a fairly resistant type and two chemicals, one during the seedling 361 period and the other at early podding period, issued the best efficient turf control for 362 AB in Syria and Australia (Owati et al. 2017). An IDM package for AB management 363 was initiated by ICARDA in alliance with the Syrian national program. A higher 364 chickpea yield using local variety without other methods was observed with this 365 package. Agronomic and ethnic management of BGM has been exhibited in several 366 countries like India, Bangladesh, and Nepal (Davidson and Kimber 2007; 367 Schreinemachers et al. 2015; Varaprasad et al. 2011; Yadav et al. 2010). 368

369 IDM practices for location-specific AB include:

- The seed used that must be free of pathogens
- Treatment of seed with fungicides
- 372 Crop rotation practices
- Deep plowing for burying crowded debris
- Use of disease-resistant genotypes

Lentils The economically vital foliar diseases of lentil are Ascochyta blight and 375 rust. Ascochyta blight is caused by A. lentils producing conidia. It involves the use 376 of resistant cultivators, aiding seed, and seed analysis by foliar spray. It can be main-377 tained by the application of fungicides (Peever et al. 2004). Lentil rust is fostered by 378 Uromyces viciae-fabae (Pers.) de Bary, which is an atrocious fungus. The disease 379 arises in the early podding phase as aecia and then into secondary aecis which rap-380 idly shows up a little delay in crop season followed by evaluation of Telia. Integrated 381 management of rust controls volunteer plants in summer and infected lentil debris. 382 It includes the use of clean seeds, suitable fungicide treatment, and host plant resis-383 tance. Various rust-resistant cultivators are deployed in different countries, with 384 resistance at CARDIA, Syria, and India (Ammar et al. 2017). 385

Faba Bean The vital diseases of faba bean are chocolate leaf spot and rust. Another 386 paramount disease of faba bean is brown rust which is spawned by fungus Uromyces 387 viciae-fabae Schroet (Mahuku et al. 2016). For controlling the foliar disease of faba 388 bean, the IDM strategy comprises the usage of the disinfected seed, avoiding the 389 spread of disease too quickly, and pursuing crop rotation. In order for the spray 390 program is to be fruitful, regular crop monitoring is crucial. Fungicide application 391 timing depends on the level of disease observed. When high chocolate spot pressure 392 occurs, carbendazim is used, and when rust or Ascochyta blight is the problem, then 393 chlorothalonil or mancozeb is used (Varaprasad et al. 2011). 394

Chocolate spot disease is spawned by *Botrytis fabae*. Initially, chocolate
spot occurs on leaves, stem, flowers, etc. as small reddish-brown circular spots.
The spot then turned into a gray dead center with a red-brown margin. This disease

kills flowers and stems. When the disease spread under favorable conditions, it 398 causes severe defoliation, flower drop, and plant death. The major component of 399 disease management includes resistance because cultural practices and fungicides 400 only give partial crop protection. To take the benefits of high priced fungicides, the 401 faba bean must be grown in early seasons. Chocolate spot control and faba bean 402 yield can be increased by using vinclozolin 50WP, once every 2 weeks. For better 403 management of this disease, different types of fungicides are used such as manco-404 zeb, chlorothalonil, carbendazim, and procymidone (Elliott and Whittington 1979; 405 Noorka and El-Bramawy 2011). 406

Rust is spawned by *Uromyces viciae-fabae* Pers. Schroet. This rust completes its 407 entire life cycle on faba bean itself. It infects many species. *Uromyces fabae* is short, 408 whitish, and cup-shaped (Barilli et al. 2014). 409

To reduce the inoculums and avert the disease and future pollution, numerous 410 cultural methods such as suitable plant spacing, appropriate crop rotation, and elim-411 ination and burning of crop debris are employed (Sparkes 2016). Field sanitation is 412 vital for reducing losses from faba bean rust. To reduce the chances of primary 413 infection, elimination of infected plant debris and faba bean rotation with nonhost 414 crops play a vital role (Lemke et al. 2007; Rótolo et al. 2015; Wesche et al. 2012). 415 Several control measures are taken to minimize crop losses like the application of 416 mancozeb (0.2%), bayleton (0.05%), and calixin (0.2%) which are fungicides that 417 control pathogenic diseases. The triazole fungicides provide excellent control when 418 applied 72 hours after inoculation. Foliar sprays of mancozeb or chlorothalonil and 419 copper product are valuable in controlling at the time of disease occurrences by a 420 chocolate spot in the same field (Godoy et al. 2016; Hartman et al. 2011) (Fig. 3.2). 421

3.6.1.2 Warm Season Legume

Cowpea It is the most important legume. *Cercospora* leaf spot, cowpea golden 423 mosaic, and cowpea aphid-borne mosaic are likely of commercial significance. In 424 growing areas of cowpea, Cercospora leaf spot is observed. The two most critical 425 diseases in cowpea are cowpea aphid-borne mosaic and cowpea golden mosaic 426 virus. Under field condition, the virus-infected seed gives the basic inoculums, and 427 aphids are accountable for the ancillary extent of the disease. ELISA is one of the 428 important methods for detection of both the seeds and the plant tissue for seed cer-429 tification project (Nautiyal 2002). 430

3.7 Sustainable Management of Fungal Foliar Disease

Sustainable management can be defined as a long-term plan of an organized system 432 of plant production practices that will satisfy the present human needs without compromising the economy of future generations and also enhancing environmental 434

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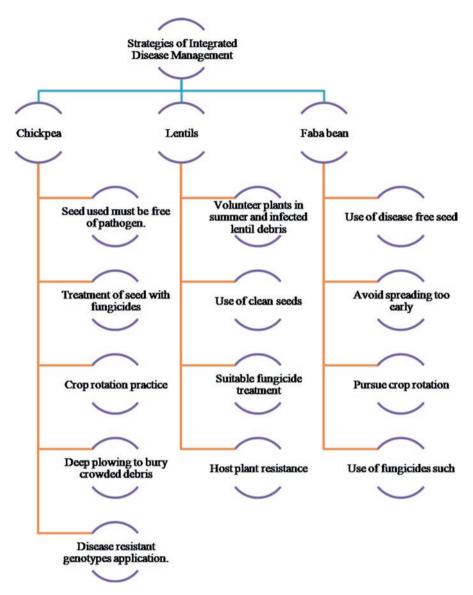


Fig. 3.2 Strategies of integrated disease management in chickpea, lentils, and faba bean

- 435 quality. Sustainable agriculture management is carried out for future generations in
- 436 the form of farming (Folgarait 1998). Sustainable agriculture management com-
- 437 prises the following:
- 438 (a) Meet human needs
- 439 (b) Natural resources are protected
- 440 (c) Prevent degradation of water quality, etc.
- 441 (d) Nonrenewable resources efficiently used

Along with sustainability, new technologies have also improved agricultural production. BMPs are used presently by targeting the applications rather than broadcasting. Cultural practices, biological pest control, new disease resistance hybrids, and many more ways are being implemented (Liang et al. 2016). 445 446 447

3.8 An Outlook for Sustainable Disease Management

3 Integrated Fungal Foliar Diseases of Arid Legumes: Challenges and Strategies...

Sustainable management of fungal diseases includes exploitation of host resistance, 450 use of synthetic fungicides, and cultural and physical methods, which is discussed below. 451

The exploitation of host resistance: To control fungal diseases, host resistance is 453 used as an efficient, inexpensive, and effective way. In this segment, available infor-454 mation is integrated for identification of resistance source; molecular markers com-455 bine with disease defiance gene identification and improved disease resistance genes 456 (Toyoda et al. 2002). Mainly cultivars are used in host-plant resistance which can 457 tolerate pathogen attack. The interaction between genetic factors in the pathogen and 458 the plant determines the expression of plant resistance. Host-plant resistance could 459 become a deficit when exposed to unsuitable environmental conditions (Andersen 460 et al. 2018). As observed on phoma stem canker (Leptosphaeria maculans) of oilseed 461 rape, disease resistance can be dependent on temperature (West et al. 2001) where 462 resistance is expressed at 15 °C but not at 25 °C (Mitrousia et al. 2018). 463

Protection with fungicides: The usual approach for fungal disease management is 464 the application of fungicides. Disease management in a traditional way is the use of 465 immense spectrum of fungicides as seed treatment chemicals and foliar sprays. 466 Numerous testing were focused on *Cercospora* leaf spot, anthracnose, and powdery 467 mildew, and some trials were on Macrophomina blight, web blight, and dry root rot. 468 DMI (demethylation inhibitors) and MBC (methyl benzimidazole carbamate) are 469 the effective fungicides that control foliar diseases. Instantly, after the appearance of 470 disease symptoms, foliar spray was applied followed by second and third sprays 471 after15–20 days from the first spray for anthracnose, powdery mildew, and 472 Cercospora leaf spot. Counter to wet and dry root rot seed treatment is applied. 473 Carbendazim is an effective fungicide against dry and wet root rot disease (Rathore 474 et al. 2008; Sumrra et al. 2015). As recommended by the Fungicide Resistance 475 Action Committee (FRAC), various management strategies, markedly, rotation of 476 treatments of a fungicide tank mix of broad spectrum and the fungicides that are 477 selected and integrated fungicide spray program along with elements of disease 478 controlling practices are executed at various levels of organizing bodies of many 479 countries (Vincelli 2002). However, sometimes disease management failures are 480 observed. For example, isolates of C. kikuchii (Cercospora leaf spot) from soybean 481

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fields in the USA were reported to be unaffected by thiophanate-methyl (Soares
et al. 2015). Isolates of *Ascochyta* blight of chickpea also reported being unresponsive to chlorothalonil, fluxapyroxad, prothioconazole, and pyraclostrobin. Nextgeneration fungicides are therefore used which are the derivatives of natural
products. These are ecologically safer and effective at reduced doses (Khani et al.
2016; Salam et al. 2011; Pande et al. 2005).

Cultural and physical practices: To terminate seed-borne pathogens, various cul-488 tural and physical methods are used to control Cercospora foliar blight. In foliar 489 diseases, field cleanliness, crop rotation, etc. is important (Tagne et al. 2008). For 490 example, mung bean seed analysis with gamma rays and storage of 90 days at a sub-491 duing effect on root rot fungi (Ikram and Dawar 2017). Computing diversity in the 492 crop rotations maintains the sustainable management of soil-borne diseases. Crop 493 rotation, plant residue management, etc., are productive for controlling diseases in 494 climatic surroundings (Chakraborty 2013; Juroszek and Von Tiedemann 2015). 495

496 3.8.1 Challenges for Sustainable Management

Ouite a lot of challenges prevail in the enactment of unified supervision, and a lack 497 of suitable understanding of integrated pest management exists among the farmers. 498 For example, gamma rays are used for seed treatment in eliminating the seed-borne 499 pathogen, but in the case of smallholder farmers, it's ineffective because the produc-500 tion of seeds in their farm is done on a small scale. With several studies, disease 501 resistance genotypes were assessed in limited localities or seasons. The pathogen 502 population varies among dissimilar geography, and for that reason, screening of 503 emerging breeding lines for disease resistance should be done in multiple locations 504 (Rebaudo and Dangles 2013;) (Crowder and Harwood 2014). 505

Various attempts are implemented for the production and application of biopes-506 ticides in the undeveloped countries. Several biopesticides just conquer the disease 507 and not effectual as chemical fungicides, and hence the growers are unwilling to use 508 the products. The farmers in those countries are not well equipped with knowledge 509 about the influence of global climate change in disease management which affect 510 the improvement and durability of plant protection chemicals and biocontrol agents 511 which can be a vital task to manage foliar imminent diseases (Afreh-Nuamah and 512 Akotsen-Mensah 2015; Heong et al. 2013). 513

514 **3.9 Conclusion**

Legumes such as chickpea, lentil, faba bean, and cowpea are consumed by the major population worldwide. This chapter dealt with the diagnosis and epidemiology of the fungal foliar diseases such as *Ascochyta* blight, *Botrytis* gray mold, rust,

chocolate leaf spot, and Cercospora leaf spot and how to control them. In this 518 chapter, the development of the management of foliar diseases of both cold and warm 519 season legumes has been explored. Previous researches were based on resistant 520 sources and chemical control of scarce diseases, whereas the present IDM program 521 lies in identifying, evaluating, merging, and locating distinct components. In spite of 522 the various IDM modules developed to tackle diseases of legumes, but, a gap exists 523 between farmers and scientists. Therefore, IDM technology might be expanded by 524 increasing farmer awareness and the crop residue quality of food legumes which are 525 the vital components of the mixed crop-livestock system. 526

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Chapter 4 Omics Approaches in Chickpea *Fusarium* Wilt Disease Management

Abeer Hashem, Baby Tabassum, and Elsayed Fathi Abd-Allah

4.1 Introduction

Chickpea is an important founder of crops in agriculture, having diploid (2n = 16)6 chromosome number. It belongs to legumes and papilionoid (subfamily) from its 7 wild Cajanus reticulatus ancestor present in Turkish Kurdistan dating back 8 (8000–9000) years (Lev-Yadun et al. 2000) and considered a major source of human 9 food due to the presence of lysine-rich protein. It is an important legume and pulse 10 crop in the world having 41–50.8% carbohydrates, 3–6% oil, 17–24% protein, and 11 considerable amount of other minerals like phosphorus, magnesium, calcium, 12 potassium, iron, zinc, and manganese. Chickpea also plays an important role as an 13 alternate rotation crop followed by cereals and manages soil fertility and productiv-14 ity by improving the N fertilization (nitrogen-fixing ability) from the atmosphere 15 (Jiménez Díaz et al. 2015). Over the past few years, it is stated that chickpea pro-16 ductivity has been marginal decreases due to the effect of biotic factors (Fusarium 17 wilt and pod borer) and abiotic factors. Reducing the pressure of these factors (biotic 18 and abiotic) is important to increase production. Chickpea ranked second among the 19

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© Springer Nature Switzerland AG 2020 B. P. Singh et al. (eds.), *Management of Fungal Pathogens in Pulses*, Fungal Biology, https://doi.org/10.1007/978-3-030-35947-8_4 1

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important food legume crops in tropical, subtropical, South, and West Asia. Overall, 20 about 1.35×10^7 ha of chickpea are growing and yield about 1.31×10^7 in more than 21 50 countries. Chickpea is used not as a valuable crop for export in developed coun-22 tries but a good source of protein supplement in cereal-based diets in developing 23 countries. Chickpea is generally grown under the rainfed condition and depends on 24 available soil water showing drought tolerance over the year. Fusarium oxysporum 25 f. sp. *ciceris* (FOC) affects the chickpea crop by inducing wilt disease, more damag-26 ing worldwide for their occurrence, and accounts 90% annual yield losses world-27 wide. The disease was first reported by Butler in 1918, but etiology was not 28 confirmed until 1940 and later was spread in Americas, Europe, and Africa but not 29 reported in Australia. Fusarium wilt has become a limiting factor for chickpea pro-30 duction in the Mediterranean basin, the Indian subcontinent, and America. The most 31 important symptoms of wilt, i.e., the patch in group form and occurs at any stage 32 and spread across a field (Haware 1990). The main reason for Fusarium wilt is soil-33 borne pathogen and observing signs like delaying crown, leaf anomalies, and rolled 34 brown leaves. The number of strains is unknown to the soilborne pathogen and is 35 difficult to control without solid information and identification of the pathogen 36 (Cha et al. 2016). 37

The susceptible varieties showed symptoms in 25 days after sowing such as 38 including flabbiness in leaves tailed by a dull green streak, dehydration, and down-39 fall of the plant. Though disease marks are commonly more visible at the initiation 40 of flowering for 6-8 weeks, in some studies, it is reported that it appeared at the 41 podding stage. The leaves dropping has occurred in the upper part of the plant, but 42 within a few days, it ensures on the whole plant. In partial wilt, few branches were 43 affected initially, but later roots of affected material affect the nearby plants. In par-44 tial wilting, no color discoloration was recorded visually. In general, symptoms of 45 the disease occur at any stage of plant growth (Jiménez Díaz et al. 2015) while more 46 visible at the early stage of flowering and appears at the podding stage (late wilt). 47 Late wilted plants exhibited falling of petioles, rachis, and leaflets as well as necro-48 sis and discoloration of foliage (Jiménez Díaz et al. 2015). Early Fusarium wilt 49 affects more than late wilting. However, late wilted plants produce lighter, rougher, 50 and duller seeds as compared to normal (Haware and Nene 1980; Navas-Cortés 51 et al. 2000). If the cross-sectional study was done on the affected plant, a dark 52 brown color discoloration was observed in xylem tissues. The discoloration was 53 also recorded in vascular tissues of roots as well as in stems. The symptoms were 54 also recorded as cavity formation among xylem and phloem, medulla and cortical 55 parenchyma, and cell proliferation in vascular cambium. 56

During the defense mechanism, the plant uses many molecular signals or protein 57 receptors to know the presence of microbes. Two modes of pathogen recognition 58 used by the host, i.e., effector-triggered immunity (ETI) and pathogen-triggered 59 immunity (PTI). The invariant epitope types are called microbe-associated molecu-60 lar patterns (MAMPs) and are composed of flagellin, chitin, and lipopolysaccha-61 rides that help spread the disease. Moreover, pathogen-induced danger-associated 62 molecular patterns are composed of fructans, callose, and glucans. As a result, host 63 secretes effector R protein domains have nibblers act as PTI. Studies also reported 64

that the sensing of bacteria produce siderophores and fungi serve as MAMPs and hydroxyproline and rapid alkalization factors, but their role was not clear yet in defense mechanism. The current has described the chickpea *Fusarium* wilt etiology, occurrence, and management practices including the most recent molecular breeding, high-throughput sequencing techniques, as well as identification of transcription factors that could favor the crop and enhance the tolerance mechanism to control the disease. 71

4.2 Casual Organism and Symptoms

It is caused by *Fusarium oxysporum* f. sp. ciceris [Fusarium oxysporum Schlecht, f. 73 sp. concerns (Padw.) Matuo & Sato] (Jimenez-Fernandez et al. 2011; Haware 1990). 74 The aerial mycelium in the first appearance was whitish and cotton, on potato 75 sucrose agar, potato dextrose agar and under UV light, but turn into salmon in color 76 and some cases, remain white (Jimenez Diaz et al. 2011). Fusarium wilt of chickpea 77 produces microconidia, macroconidia, and chlamydospores. The microconidia are 78 elliptical or tubular and straight. Macroconidia are thinner than microconidia and 79 typically 3–5 septate or fusoid, while chlamydospores are produced in 15-day-old 80 cultures and infected chickpea tissues, smooth or rough-walled (Castro et al. 2012; 81 Jimenez Diaz et al. 2011). Maximum sporulation was recorded at pH ranges from 82 7.1 to 7.9 (Jimenez Diaz et al. 2011). Hyphae are septate and split abundantly. 83 Optimum growth was recorded at 25–27 °C and pH 5.1–5.9 and liable on strains. 84

4.3 Epidemiology

The severity of the chickpea wilt is depending upon the pathogen, genotypes, patho-86 genic races, inoculum density, environmental condition, and cultivar sensitivity. The 87 activity of the wilting disease was triggered by a combination of pathogen activities. 88 It includes fungus mycelium in the xylem that produced contaminant components 89 that affect host defense response, production of gels, teloses, and vessel crushing by 90 the propagation of linked parenchyma cells (Beckman 1987). The mycelium might 91 survive as a pathogen in seed, soil and toxic residues (crop), roots, and stem tissue 92 concealed in the soil for more than 6 years or even in absence of host (Singh et al. 93 2008). Dicotyledonous weeds that don't show the symptoms but have the infection 94 that could enhance the pathogen activity and survived in fallow soils. Moreover, 95 infected soil is an important source of primary inoculum for the development of 96 *Fusarium* wilt (Al-taae et al. 2013). The transmission can also be done by the seed 97 and can survive in plant debris as well as in the soil. Moreover, it also observed that 98 fungus chlamydospore was present in soil freely (Haware et al. 1996), seed hilum 99 (Haware et al. 1978), and cotyledon axis (Shakir and Mirza 1994). Chlamydospores 100 or mycelia are the main and basic sources of infection, even the conidia of the fungus 101

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are short-lived, while chlamydospores can remain feasible up to the next available 102 crop in the field (Chand and Khirbat 2009). Chlamydospore production is contin-103 gent on the nutrient availability of the inoculum. Fungal inoculum may be exposed 104 to lower nutrient levels in the field condition as compared to grow under well-fed 105 macroconidia form under agar media (Schippers and Van Eck 1981). The pathogen 106 grows very well in roots and stems in apparently looking good condition but con-107 cealing adequate fungus (Trapero Casas and Jimenez Diaz 1985). The pathogen 108 remains dormant until triggered to germinate when carbohydrate is released from 109 decaying tissue or roots, present in the form of chlamydospores (Schippers and Van 110 Eck 1981). The provocation for germination could be the host or non-host plant 111 roots or plant wreckage (Nelson 2012), after the germination of chlamydospores, 112 conidia, hyphae, and new chlamydospores is formed. After conidia and hyphae pro-113 duction, thallus formation took place and leads to chlamydospore production in 114 2-3 days if suitable condition prevailed (Beckman and Roberts 1995). By penetra-115 tion of the epidermal cells, attack on the roots occurs on the host or non-host plants 116 (Beckman and Roberts 1995) and caused vascular disease (Stover 1970). The infil-117 tration occurs directly or by wounds (Nelson 2012), the common sites for infiltra-118 tion are the root tip of both tap and lateral roots (Lucas 1998). The infiltration is 119 stopped by different factors, such as fungal compounds, and inhibits the spore for-120 mation (fungal), plant surface structures, and germ tube production (Mendgen et al. 121 1996). The more adverse form is, mycelium moved through intercellular root cortex 122 and finally reaches to xylem vessels during colonization and remains within the 123 xylem vessels and colonize in the host (Bishopt and Cooper 1983). 124

Breeding 4.4 125

rec The Fusarium wilt activity can be reduced in the host using breeding approaches in 126 chickpea crop. Breeding approaches involved availability of genetic diversity con-127 sidered the most important step for a breeding program, wild relatives, and selection 128 of desirable plant for trait and disease resistance and evaluate the plant for commer-129 cial production (Salimath et al. 2007). As chickpea is a self-pollinated crop, it 130 requires genes to fix the breeding problems by pure lines development. Initial 131 screening was done by mass or pure line selection and later crossing programs and 132 alteration in pedigree and bulk methods were employed for segregating generation 133 (Gaur et al. 2012; Millan et al. 2015). In the intraspecific hybrid program, the single 134 cross method was used in desi and Kabuli chickpea genotypes with variant genetic 135 history (Berrada et al. 2007). Parents from desi varieties have been used for gene 136 transfer in Kabuli varieties against Fusarium wilt resistance, as parents from Kabuli 137 parents are used to improve large size seed and seed quality in desi variety (Gaur 138 et al. 2007). The breeding development efforts were also made for interspecific 139 crosses and enhance genetic diversity and interrogate useful genes from wild cicer 140 into cultivated spp. The FOC resistance has been recognized from desi germplasm 141

as well as in wild Cicer spp. (Kaiser et al. 1994). For genetic gains enhancement, 142 there is a need precise and efficient selection of segregating populations (Gaur et al. 143 2012). For successful wilt, sick breeding programs hot spot location, field, green-144 house and laboratory methods have been used for the selection of resistance varieties 145 (Gaur et al. 2007). It has been reported about 5174 Kabuli genotypes were screened 146 against Fusarium wilt resistance at ICARDA, and about 110 genotypes were recog-147 nized as resistant. Fusarium wilt resistance depends upon monogenic or oligogenic 148 depending upon the resistance resource (Sharma and Muehlbauer 2007; Upadhyaya 149 et al. 1983; Sharma et al. 2005). It is also reported that FOC genetic resistance culti-150 var contains three independent genes (h1, h2, and h3) (Singh et al. 2014). Moreover, 151 it is also suggested that late wilting was controlled by the presence of any one gene 152 nut combination of two genes confirm the wilt resistance in chickpea (Castro et al. 153 2012; Jiménez Díaz et al. 2015). Similar results also stated that resistance was con-154 firmed by the presence of these genes in the combine or individual form (Tullu et al. 155 1999). Some ICARDA lines, i.e., WR-315, CA-1938, and CA2139, contain these 156 genes (Halila et al. 2009; Rubio et al. 2003). However, the genetic of resistance for 157 some chickpea races like 1B/C and 6 is still unknown. 158

4.5 Genetic and Pathogen

The first name of the fungus was *fusarium orthoceras* apple and swollen. var. cice-160 rone by Padwick and modified by Chattopadhyay and Sen Gupta and was renamed 161 as F. oxysporum Schl. f. sp. ciceri (Padwick) Snyder and Hansen. Fusarium oxyspo-162 rum is among the monophyletic origin in the Fusarium oxysporum complex of the 163 gibberella clade and considered as polyphyletic and currently known as *Fusarium* 164 oxysporum (Schlechtend, Fr.) f. sp. ciceris (Padwick) Matuo & K. Sato, Fusarium 165 is the only pathogen in *Cicer* sp. (Kaiser et al. 1994), and *oxysporum* is an attack on 166 root tissue in faba bean, lentil, and pea and recorded as symptomless carters for the 167 pathogen (Trapero Casas and Jimenez Diaz 1985). Yellow or wilting syndromes 168 along with brown discoloration were recognized based on two pathotypes and 169 induce in sensitive chickpeas. The recorded symptoms are considered slow, foliar 170 yellowing and death of plant at a later stage while wilting is considered reckless, 171 adverse chlorosis, flabbiness, and plant death during an early stage of growth 172 (Trapero Casas and Jimenez Diaz 1985). The susceptibility of the pathogen depends 173 upon the races and efficient use of available resources for the chickpea breeding 174 program. The identification of the races against pathogens is simple but depends 175 upon the cost, available resources, and facilities. So, there is a dire need to develop 176 new methods that are more rapid and effective, and reproducible identification of 177 pathogen and races is used to determine the diversity and resistance among the 178 genotypes. Polymerase chain reaction (PCR)-based molecular markers have been 179 used to determine the Fusarium oxysporum f. sp. ciceris and its related pathogen 180 races identified by the method developed by Jiménez-Gasco et al. (2001). 181

The screening and legacy of the gene of interest (GOI) and traits are possible 182 now with the development of marker-assisted selection (MAS) and provide ben-183 eficial information to exploit the genes useful for agronomic traits (Allahverdipoor 184 et al. 2011). Molecular markers are an important tool for identification, character-185 izing, and screening and determine the diversity among the pathogens and diseases. 186 Commonly, internal transcribed spacer (ITS) markers are used for classification and 187 screening of the fungi (White et al. 1990), while ITS data is not enough for com-188 plex identification and diverse gene information; therefore, it is not suitable for 189 genetic diversity or characterization of fungus. The Fusarium genus is improbable 190 as compared to the genetic study of F. oxysporum f. sp. fragariae has not vet been 191 reported. Among the various available technologies, restriction fragment length 192 polymorphism (RFLP) markers are important rDNA used to determine the genetic 193 diversity of plant pathogenic fungi. It is also used to group the isolated strains with 194 low cost (Kachuei et al. 2015). Based on symptoms, the two pathogens were geneti-195 cally distinguished by random amplified polymorphic DNA markers (RAPD) and 196 sequence characterized amplified region (SCAR). The specific Fusarium assays 197 were successfully characterized using RAPD and SCAR molecular markers. 198 Another study stated that evaluation and screening of resistant wilt lines were done 199 against Fusarium by using RAPD and SSR molecular markers. The results repre-200 sent that about 70% cultivars were resistant to disease while 30% showed suscep-201 tibility for wilt response. SSR marker (TA194) recorded an 85% probability locus 202 at wilt resistant among the total primer used, and it was later reconfirmed by the 203 receiver operating characteristic curve (Ahmad et al. 2014). Gowda et al. (2009) 204 former designing the linkage map for FOC 1-5 gene resistance races with SSR 205 and RAPD in recombinant inbred lines (RILS) developed by sensitive and resistant 206 parents. About eight races were recognized as the specific fungus, out of which six 207 are more infectious (Jimenez-Diaz et al. 1993). Introgression of Ascochyta blight 208 resistance with double podding traits in chickpea was confirmed by marker-assisted 209 backcrossing. SSR markers are used in separate backcross generation to assist in 210 selection against the resistance of Fusarium (Varshney et al. 2014). SCAR markers 211 are used for Ascochyta blight resistance to determine the OTLs in chickpea, and 212 respected OTLs were identified, i.e., SCY17590 and SCAE19336, tightly linked 213 with Ascochyta blight resistance gene at QTLAR2 location (Iruela et al. 2006) and 214 later on successfully used for tagging in chickpea resistance lines for germplasm 215 collection (Imtiaz et al. 2008). Combinations of SCAR with a codominant marker 216 (CaETR) linked with QTLAR1 for Ascochyta tagging and help to identify the 217 resistance alleles from a core collection of resistant cultivars (Madrid et al. 2013). 218 Near-isogenic lines (NILs) were developed by using STMS markers that are tightly 219 linked FOC 5 and FOC 01 for the selection of susceptible genotypes and resistant 220 genotypes in LG2 and LG5 (Castro et al. 2010). Moreover, NILs are used as a valu-221 able tool for mapping, refining the target region and selection of the desired gene 222 for resistance to foc0 (Jendoubi et al. 2016). Jendoubi et al. (2016) reported that the 223 results obtained from the population were useful for position refining of the target 224 area involved in resistance mechanisms. Similar results were obtained by Ali et al. 225 (2015) that identify the target regions associated with growth habit and double-226 podding base morphological position-based markers that are used in chickpea. 227

4.6 Integrated Genomic Approaches

The identification and construction of the genetic map of the segregating population 229 is the foremost objective of the breeders. Efforts have been made to construct the 230 genetic map using molecular markers for tagging traits and site-specific gene of inter-231 est in chickpea (Millan et al. 2010; Millan et al. 2015). The first maps were con-232 structed using the isozymes F2 population from interspecific crosses (Gaur and 233 Slinkard 1990). Many researchers reported identified genes regarding flower color, 234 wilt resistance (Fusarium), double pod, and growth habit (Gaur and Slinkard 1990; 235 Kazan et al. 1993; Cobos et al. 2005), and other agronomic characters and Ascochyta 236 blight resistance linked QTLs were identified on these maps (Lichtenzveig et al. 237 2006). The larger numbers of maps were derived from crosses with C. reticulatum as 238 well as many markers identification related to specific traits. However, the populations 239 derived from interspecific crosses were made due to microsatellite markers and exploit 240 more genetic polymorphisms among the chickpea genotypes (Cobos et al. 2007). The 241 first transcriptome study for the chickpea genome was done with the advancement of 242 next-generation sequencing (Hiremath et al. 2011). With the advancement of tran-243 scriptome information, detail genetic maps were made using large-scale molecular 244 markers (Hiremath et al. 2012; Thudi et al. 2011). The availability of the draft genome 245 sequencing in desi and Kabuli varieties would also facilitate the genetic population 246 used for mapping and positioning of the QTLs in chickpea genome (Ali et al. 2015). 247 Omics approaches gathered genomic information and triggered molecular markers 248 development of tightly linked QTLs (Kumar et al. 2011). 249

4.7 Transcription Factors

Recent advances in molecular plant sciences boost the knowledge, and transcrip-251 tomic emerged as a powerful method to understand differential genic response over 252 specific time-bound fashion. Transcriptomic is the techniques used to study the 253 whole set of RNA transcripts (coding and non-coding) of a cell at a specific time 254 and conditions. Expression analysis of tissue under different growth conditions 255 reveals the regulatory network of the responsive gene for that specific stage or con-256 ditions, it could also help to annotate those genes which were previously unanno-257 tated due to lack of information. TF has the function to regulate the cell development, 258 differentiation, and growth by tagging specific site with DNA or multiple sites and 259 triggered the activation or repression of the TF through various mechanism and 260 interaction, i.e., DNA-protein, protein-protein, and alteration in chromatin structure 261 (Kusuya et al. 2018). The soilborne fungus is a causal agent of chickpea wilt dis-262 ease. The infection includes root identification, colonization, penetration, adhesion, 263 and penetration of the root cortex, and hyphal proliferation within the xylem vessels 264 are controlled by transcription factors (TFs). Transcriptome analysis based on RFLP 265 and RAPD-based cDNA techniques were used and identified many defense-related 266 genes in chickpea (Gurjar et al. 2012). Moreover, next-generation sequencing 267

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identified microRNA responsive genes regulating plant development and pathogen
growth depending on target genes (Kohli et al. 2014). *Fusarium* spp. produced
about 50 unique types of secondary metabolites, i.e., growth regulators, pigments,
and mycotoxins, that are important for feed and food concerns. TFs have been
shown to manage the mycotoxin biosynthesis compound that is favorable for other
pathogenic *Fusarium* species (Brown et al. 2014).

Identification of FolCZF1 encoded for (C2H2) transcription factor. It is also 274 known to affect pathogenicity in wheat (F. graminearum) and rice (Magnaporthe 275 oryzae). The critical role of gene FolCZF1 is to produce fusaric acid and regulate 276 the expression of fusaric acid biosynthesis. Fusaric acid (FA) taking part in the 277 severity of Fusarium diseases, i.e., damping off, vascular wilt, and root rot (Ding 278 et al. 2015). Fusaric acid is linked with vascular wilt symptoms caused by F. oxys-279 porum; some transcription factors are involved in the regulation of virulence and FA 280 biosynthesis. *FolCZF1* affects the FA and influence the virulence (Yun et al. 2019). 281 Moreover, *FolCZF1* is also reported that it requires secondary metabolism and early 282 host infection (Yun et al. 2019). Zinc finger proteins (C₂H₂) are widely studied in 283 filamentous fungi. 284

A similar study was conducted to determine the molecular basis of wilt disease 285 in chickpea by comparing the analysis of the transcriptome of resistant and suscep-286 tible wilt cultivars under Fusarium oxysporum f. sp. ciceri and controlled condition. 287 Analysis results stated that novel genes with differential or unique expression caus-288 ative to lignification, hormonal balance, plant defense signaling, and ROS. Moreover, 289 the study also provides information about the functional characterization of the 290 genes involved in resistance mechanism and their use in a breeding program against 291 wilt resistance and tolerance mechanism as well as target pathogen identification for 292 the facilitation of the development of novel control management strategy (Upasani 293 et al. 2017). Microscopic, proteomic, and metabolic approaches are also used to 294 characterize the chickpea cultivars under *Fusarium oxysporum* interaction. The 295 resulting expression at the microscopic level stated that differential colonization of 296 FOC was present in susceptible and resistant genotypes. It is also reported that 297 resistant host severely restricted the pathogen growth while opposite results were 298 observed in susceptible cultivars. Moreover, proteomics and metabolomics results 299 notified that the upregulation of several metabolic pathways was observed in resis-300 tant genotypes (Kumar et al. 2015; Kumar et al. 2016; Upasani et al. 2016). 301

ROS played an important role in recognized insight and defense signaling, but 302 their redox relation in plant is still unknown for the defensive network. A study was 303 conducted to determine the role of FOC 1 by inducing redox-responsive transcript 304 for regulating defense signaling in chickpea. Microscopic studies emphasized inva-305 sion and colonization along with tissue damage and confession of degraded prod-306 ucts at the xylem vessels in diseased roots area. Due to confession clogging of the 307 xylem vessels incompatible hosts while resistant plant not. Assays related to lipid 308 peroxidation represent membrane injury, and other remarkable changes were 309 recorded such as cell shrinkage and gradual nuclear depression in fungal ingress. 310 Moreover, qPCR results showed expression of redox regulators, cellular transport, 311 and transcription factors in FOC 1 analysis. Functional analysis results stated that 312

respiratory homolog, vacuolar sorting receptors, and zinc finger domain TF provide 313 deep insight regarding the complex structure of wilt disease defense mechanism in 314 chickpea as well as other legume crops (Gupta et al. 2013). The study also reported 315 that chickpea transcript is used for involvement to regulate the redox state when 316 infection occurs due to FOC 1 races (Gupta et al. 2009; Ashraf et al. 2009; Gupta 317 et al. 2010; Garcia-Limones et al. 2002). Moreover, it is also reported that modifica-318 tion in the RBOH recorded regulatory role during an invasion in resistant plants 319 while sensitive plants do not show similar variation. The other modification in OCP 320 and FSD has a role in ROS signaling and OCP considered as ABA-dependent TF 321 regulator, recorded down regulation in Arabidopsis thaliana (62). Also reported that 322 cationic peroxidase has the function to accumulate in the xylem vessels in rice plants. 323

Genome-wide analysis of chickpea genotypes against Fusarium oxysporum was 324 done and transcriptome study conducted by illumining technology at conidial ger-325 mination stage at variant points. The results revealed that; genes linked to fungal 326 developments are transcribed at consecutive ways were discovered. It was also 327 reported that genes related to secret effectors, cell wall degrading, metabolism, pep-328 tidases, and transporters-related enzymes were determined at the germination stage 329 of conidial growth. Moreover, metabolism genes are upregulated at germination, 330 while secondary metabolites and transporters genes were upregulated at a later 331 stage (Sharma et al. 2016). The root structure and colonization (hypocotyl) and their 332 expression profiling in infected genotypes and plant response factors were deter-333 mined using two Fusarium oxysporum. The results revealed that less colonization in 334 xylem vessels was recorded in weekly infected genotypes. After the analysis of 335 virulent genes, the expression profiling results represent that two genes (SIX1, 336 SIX6) include TF (FTF1) were upregulated in root crown and hypocotyl. Both 337 strains performed differently, the virulent strain showed strong transcription in PR1 338 gene while other strains respond to ethyne factor ERF2 (Niño-Sánchez et al. 2015). 339

In general plant colonization by fungal vascular wilt pathogens after invasion 340 colonization was done in cortical cells, latterly hyphae intercellularly move toward 341 vascular parenchyma cells and occupied xylem vessels. Once reached to xylem, 342 mycelium is restricted in the vessels; as a result necrosis occurs in host tissue for 343 general colonization (Yadeta and Thomma 2013). Ma et al. (2010) also reported that 344 Fusarium oxysporum-specific sequences present in replaceable chromosomal posi-345 tion are the basis of host specialization and polyphyletic origins of most formae 346 specials. 347

4.8 Exclusion and Eradication of the Pathogen

The exclusion and eradication of the pathogen is the basic paradigm for crop 349 improvement programs. For this purpose, integrated approaches have been used to 350 exclude and eradicate crop diseases, pests, and weeds. Though disease control by 351 the integrated management approach is no cure for plant disease control, it is considered as an ecology approach by which different disease control measures are 353

adopting such as pathogen-free planting material, avoiding planting in high-risk 354 soil, exclusion and eradication of F. oxysporum inoculum from rhizosphere, and 355 using of biocontrol measures for healthy planting materials. It is transmitted through 356 virulent seeds and plant residues (Jimenez Diaz et al. 2011; Nelson et al. 1981), 357 infected materials than propagating into pathogen-free soils. For this purpose, strict 358 legislation and inspection of the seeds material and planting area and optimize the 359 use of FOC spp. in the non-virulent area (Jimenez Diaz et al. 2011). For quantifica-360 tion, evaluation, inspection, and legislation of the quarantine measurement, Jiménez-361 Fernández et al. (2011) established a gPCR protocol that permits to measure the 362 DNA quantity in root and stems from infected asymptomatic chickpea. Seed dress-363 ing with Benlate could be used to remove seed borne inoculum (Haware et al. 1978). 364

Soil having problems of *Fusarium oxysporum* can be reclaimed by reducing or 365 lessening the initial inoculum or reducing the disease potential (Passari et al. 2017; 366 Jimenez Diaz et al. 2011), and this can be achieved by various methods, i.e., bio-367 logical, physical, and chemical means. A most important method is soil solarization, 368 and Fusarium wilt can be controlled in many crops in this way (Stapleton and de 369 Vay 1986). By solarization, pathogen not only kills but also weakens and reduces 370 the severity and increases the availability of other components in soil microbiota 371 (Strange 2003). Moreover, soil pathogen can also be controlled by flooding (Strange 372 2003), by removing the plant residue from wilt affected crop, by killing the FOC 373 chlamydospore, and by limiting the severity of the disease for the next crop (Jiménez 374 Díaz et al. 2015). During biological control, use bio-agents to reduce the pathogen 375 activity by making colonization in the rhizosphere while no toxic residue remains in 376 the soil (Dubey et al. 2007). Trichoderma has been used against Fusarium wilt in 377 greenhouse and field condition and gives tremendous result to control the disease 378 (Kaur and Mukhopadhyay 1992). 379

Moreover, the application of Pseudomonas restricted the FOC in vitro and 380 allowed significant growth in shoot length, dry weight, and yield (Nautiyal 1997). 381 Application of nonpathogenic type strains such as Bacillus sp. and Pseudomonas 382 recorded a significant reduction in the severity of *Fusarium oxysporum* f. sp. ciceris 383 (Nautival 1997). Another practice could also reduce the severity of plant pathogen 384 effect on the chickpea crop. An adequate amount of cultural practices takes the ben-385 efit of Fusarium management. A study reported that Fusarium can live about 6 years 386 and 3 years of crop rotation but is not effective to reduce the effect of Fusarium 387 incidence (Haware et al. 1996). Moreover, widespread disease development is due 388 to the sowing date (Navas-Cortés et al. 1998); sowing chickpea from early spring to 389 early winter could slow the Fusarium wilt development and ultimately enhance the 390 yield (Landa et al. 2004). Along with the sowing date, the use of resistant cultivars 391 also appears to be benefitted to control the wilt disease. Resistant varieties played an 392 important role in an integrated disease management program (Landa et al. 2004; 393 Jimenez Diaz et al. 2011; Jiménez Díaz et al. 2015). Resistant desi genotypes have 394 been identified against FOC that reduced the disease incidence in wild and desi 395 chickpea varieties (Jiménez Díaz et al. 2015). The availability of high genetic diver-396 sity in pathogenicity reduces the effectiveness and extensive use of present resis-397 tance (Bavraktar and Dolar 2012). 398

4.9 Conclusion

Fusarium oxysporum f. sp. ciceri (FOC) affects the chickpea crop causing wilt 400 disease, more damaging and worldwide in occurrence. The main reason of Fusarium 401 wilt is soilborne pathogen and showed symptoms, i.e., delaying crown, leaf anoma-402 lies, and rolled brown leaves. The number of strains is unknown of the soilborne 403 pathogen and is difficult to control without solid information and identification of 404 the pathogen. In general, symptoms of the disease occur at any stage of plant growth 405 while more visible at the early stage of flowering and appears at the podding stage 406 (late wilt). Late wilted plants exhibited falling of petioles, rachis, and leaflets as well 407 as necrosis and discoloration of foliage. Early Fusarium wilt affects more than late 408 wilting. However, late wilted plants produce lighter, rougher, and duller seeds as 409 compared to normal. During the defense mechanisms, the plant uses many molecu-410 lar signals or protein receptors to know the presence of microbes. Two modes of 411 pathogen recognition are used by the host, i.e., effector-triggered immunity and 412 pathogen-triggered immunity (PTI). The invariant epitope types are called microbe-413 associated molecular patterns and are composed of flagellin, chitin, and lipopoly-414 saccharides that help spread the disease. Studies also reported that the sensing of 415 bacteria produce siderophores and fungi serve as MAMPs and hydroxyproline and 416 rapid alkalization factors, but their role was not clear yet in defense mechanism. The 417 severity of the chickpea wilt is depending upon the pathogen, genotypes, pathogenic 418 races, inoculum density, environmental condition, and cultivar sensitivity. The 419 activity of the wilting disease was triggered by a combination of pathogen activities. 420

Breeding approaches involved genetic diversity the most important step for a 421 breeding program, selection of desirable plants for trait resistance and disease resis-422 tance and evaluation of the plant for commercial production. In an intraspecific 423 hybrid program, the single-cross method was used in desi and Kabuli chickpea geno-424 types with variant genetic history. Molecular markers are an important tool for iden-425 tification, characterizing, screening, and diversity among the pathogens and diseases. 426 Commonly, internal transcribed spacer (ITS) markers are used for classification and 427 screening of the fungi. While data regarding pathogen diversity is compulsory to 428 comprehend pathology and development for control measures, SSR markers are 429 used in separate backcross generation to assist in the selection against the resistance 430 of *Fusarium*. Many pathogenic FOC spp. cause alike symptoms in chickpea crop as 431 with FOC. For this purpose, screening, identification, and insight are more important 432 among the pathogen FOC spp. This approach provides a deep understanding of the 433 epidemiology of the disease and triggered the development of elite resistant geno-434 types by adopting breeding, molecular, and plant omics technology. QTLs linked 435 molecular markers would also facilitate to identify the desired traits is the basic 436 requisite for the application of molecular markers in the breeding program and 437 enhance the selection process. Combinations of SCAR with a codominant marker 438 (CaETR) linked with QTLAR1 for Ascochyta tagging and help to identify the resis-439 tance alleles from a core collection of resistant cultivars. Moreover, NILs are used as 440 a value able tool for mapping, refining the target region and selection of the desired 441 gene for resistance to FOC 0. Efforts have been made to construct the genetic map 442

using molecular markers for tagging traits and site-specific gene of interest in chick-443 pea, However, the population derived from interspecific crosses was made due to 444 microsatellite markers exploiting more genetic polymorphisms. Recent advances in 445 molecular plant sciences boost the knowledge, and transcriptomic emerged as a 446 powerful method to understand differential genic response over specific time-bound 447 fashion. TF has the function to regulate the cell development, differentiation, and 448 growth by tagging specific site with DNA or multiple sites and triggered the activa-449 tion or repression of the TF through various mechanisms and interactions, i.e., DNA-450 protein, protein-protein, and alteration in chromatin structure. The infection includes 451 root identification, colonization, penetration, adhesion, and penetration of the root 452 cortex and hyphal proliferation within the xylem vessels are controlled by transcrip-453 tion factors (TFs). The functional characterization of the genes would also facilitate 454 resistance mechanisms and their use in the breeding program against will resistance 455 and crop tolerance mechanism along with target pathogen identification for the facil-456 itation of the development of novel control management strategy. 457

Acknowledgments The authors would like to extend their sincere appreciation to the Deanship
of Scientific Research at King Saud University for funding this research group NO
(RG-1435-014).

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Chapter 5 Integrated and Sustainable Management of Fungal Diseases of Chickpea: Current Status and Challenges

Babu Nagabhushan Motagi, M. S. Laxminarayan Rao, and Akshay Mathad

5.1 Introduction

Chickpea is an important commercial rabi pulse crop of the globe and India. India 7 is a leading producer of chickpea ranked first both in an area with 99.27 lakh ha and 8 production of 98.80 lakh tonnes of chickpea, followed by Pakistan, Iran, and 9 Australia. The highest productivity of 3759 kg ha⁻¹ is observed in China followed 10 by Israel, the Republic of Moldova, and Bosnia and Herzegovina. However, Indian 11 chickpea productivity is only 995 kg ha⁻¹ (Anonymous 2016). The low productivity 12 observed in India is mainly attributed to the increasing pests and diseases with poor 13 management practices coupled with climate change. Chickpea crop is mainly 14 affected by fungal diseases like fusarium wilt (Fusarium oxysporum. f. sp. ciceris), 15 ascochyta blight (Ascochyta rabiei), rust (Uromyces ciceris-arientini), dry root rot 16 (Rhizoctonia bataticola), gray mold (Botrytis cinerea) and powdery mildew 17 (Leveillula taurica), leaf spot (Alternaria sp.), phytophthora root rot (Phytophthora 18 medicaginis), damping off (Pythium debaryanum), foot rot (Sclerotium rolfsii), and 19 sclerotinia wilt (Verticillium albo-atrum). 20

Fusarium is both soil and seed borne disease and is very hard to handle only by chemicals and also often breakdown of resistance owing to the presence of new virulent races, poses a true challenge for farmers and pathologists as a result of the scenario remains unchanged for last ten years, although attempts have been made in breeding and selecting several chickpea varieties with elevated disease-tolerant yield capacity. Epidemics of Fusarium Biodiversity can devastate plants and trigger 26

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B. P. Singh et al. (eds.), *Management of Fungal Pathogens in Pulses*, Fungal Biology, https://doi.org/10.1007/978-3-030-35947-8_5

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up to 100% losses in extremely infested areas and favorable circumstances. Resistant 27 cultivars are the most efficient way of managing the disease and helping to stabilize 28 the returns of chickpea. The development of fusarium-resistant strains is focused 29 primarily on standard choice in various breeding programs. This process takes time 30 and relies on inoculum load and certain environmental influences on the growth of 31 the disease. Using molecular techniques provides a good opportunity for enhance-32 ment of chickpea, in particular by defining molecular markers associated tightly to 33 genes / QTLs that control fusarium wilting (Warda et al. 2017). Biological control 34 seems to be a better option and novel methods like 'Bio-priming' is being tested for 35 the sustainable and eco-friendly management of diseases like Fusarium and 36 Sclerotium wilt of both Chickpea (Vidhyasekaran and Muthamilan 1995). Ascochyta 37 blight is the most serious disease causing up to 100% losses in Northern India, 38 Pakistan, U.S.A. and Middle East (Smithson et al. 1985). Chickpea rust is also pos-39 ing a serious threat and epidemics have been reported in several states like Karnataka, 40 Andhra Pradesh, Maharashtra, etc. Further studies need to be carried out for a clear 41 understanding of the biology of this pathogen, the role of alternate hosts like 42 Trigonella polycerata survival of the pathogen in the, etc. Integrating bio-chemical 43 monitoring seems to be an excellent way to combat many pathogenic agents with 44 minimum intervention with the soil biological balance (Papavizas 1973). 45

Sequencing of reference genomes of CDC Frontier genotype in chickpea 46 (Varshney et al. 2013a, b) and mapping of about 50 chickpea traits including blight, 47 wilt, and gray mold diseases at ICRISAT helped in understanding the function of 48 genes and pathways besides translating genomics research into product develop-49 ment in these important pulse crops. Superior chickpea line C 2014 with wilt and 50 blight resistance is in multilocation trials for evaluation and release (ICRISAT 51 2017). Integrated and sustainable management of important fungal diseases of 52 chickpea is discussed in the book chapter. 53

54 5.2 Fusarium Wilt

It is one of the most significant fungal diseases that can cause significant loss to chickpea crop worldwide. Butler first recorded it in India in 1918, but Padwick did not determine its etiology properly until 1940. The disease is now common in most of the Asian, African, Southern European, and American countries (Cunnington 2007). In India, it is widely distributed across Indo-Gangetic regions and elsewhere in Southern India.

61 5.2.1 Symptoms

The main symptom of fusarium wilt in the field is drooping and the death of plants.
The leaves turn yellow and drop off prematurely. In the wilted plants, necrosis of the
collar region and discoloration are seen. The diseased plants can be easily removed

from the soil, and most of the lateral roots are infected and become weak and remain 65 in soil when plants are uprooted. The transverse section of the basal stem/roots 66 revealed masses of hyphae under the microscope in the vascular bundles and discoloration of vascular cells. 68

The disease symptoms can be seen at any stage of the plant, and affected plants are 69 in patches or spread across the whole field (Trapero-Casas and Jiménez-Díaz 1985). 70 Sensitive cultivars may have signs of premature wilting, with flaccidity of individual 71 plants and a dull green coloration following complete plant desiccation within 25 days 72 after the sowing period. Late wilting signs, however, are generally most visible at 73 flowering, and even appear until podding, when the petioles and leaflets drop, accom-74 panied by yellowing and necrosis of foliage. In the upper part of the plant, drooping is 75 seen first but occurs over the whole plant within a couple of days. Symptoms may only 76 affect a few plant stalks that trigger partial wilting. The xylem of roots and stems 77 develops dark-brown coloration and seen when made vertical/cross sections (Fig. 5.1). 78 Fusarium decreases the production of chickpea by reducing both the yield and weight 79

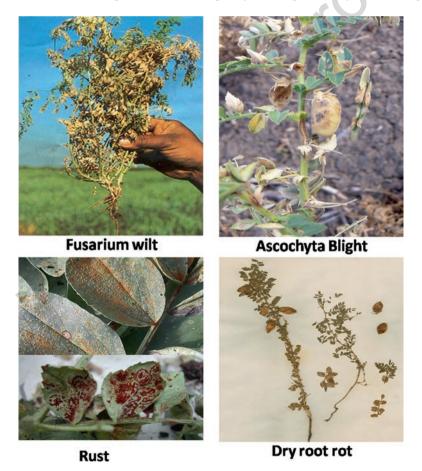


Fig. 5.1 Field view of disease symptoms of major fungal diseases of chickpea and name of pathogens are marked in figure

(Nene and Haware 1980). Yield loss due to fusarium wilt in India and Spain is 10–15%
(Singh and Dahiya 1973) and 40% in Tunisia (Bouslama 1980) have been reported.
Early wilting had greater yield reduction (77–94%) than yield reduction (24–65%)
due to late wilting (Nene and Haware 1980).

84 5.2.2 Causal Organism

Fusarium oxysporum f. sp. *orthoceras* (Appel & Wollenweber) Bilay (class,
Deuteromycetes; order, Moniliales; family, Tuberculariceae).

The fungus produces both inter- and intracellular hyaline mycelia in the infected 87 tissue most abundantly in the vascular bundles. The fungus produces both macro-88 and microconidia in the host tissues as well as in cultures. Microconidia are small, 89 thin-walled, hyaline, elliptical 1–2 celled, measuring $4-6 \times 2-4 \mu m$. Macroconidia 90 are long, curved (fusiform or sickle-shaped) pointed at both ends, septate, and mea-91 sure $25-40 \times 3-4$ µm. Chlamydospores, the surviving structures, are also formed in 92 the host as well as in old cultures, which develop from any cell of the hypha. The 93 cells round off and become thickly walled to form chlamydospores; they are spheri-94 cal or oval single or, in chains, terminal or intercalary. 95

Although monophyletic, F. oxysporum f. sp. ciceris shows considerable patho-96 genic variation. Different pathogen syndromes with brown vascular discoloration 97 were noticed depending upon the unique vellowing or wilting syndromes which 98 make chickpea genotypes susceptible. Pathotypes, which are genetically diverse, are 99 being placed in two separate groups depending on fingerprint assays RAPD, SCAR, 100 and DNA (Jimenez-Gasco et al. 2001). Haware and Nene (1982b) reported that there 101 is lot of variation in symptom types because of the presence of the eight races of 102 pathogen (races 0, 1A, 1B/C, 2, 3, 4, 5, and 6), which were identified by reactions on 103 a set of differential cultivars of chickpea. Races 0 and 1B/C induce the yellowing 104 syndrome (yellowing pathotype), whereas races 1A, 2, 3, 4, 5, and 6 induce the wilt-105 ing syndrome (wilting pathotype), and all the races have distinct geographic distribu-106 tions. Haware and Nene (1982a) reported only four races (Races1A, 2, 3, and 4) in 107 India, whereas races 0, 1B/C, 5, and 6 are found mainly in the Mediterranean region 108 and the USA (Jimenez-Gasco et al. 2001). Three new races were reported from India 109 based on old differentials (Honnareddy and Dubey 2006). The isolates from each 110 state of India were highly variable, and based on the reactions on international dif-111 ferentials, more than one race were found to be prevalent in every state (Dubey and 112 Singh 2008). Dubey et al. (2012), based on new differential set of chickpea cultivars, 113 reported that all eight races were found in India. 114

115 5.2.3 Disease Cycle

The wilt-causing fungus survives saprophytically being facultative saprophyte and on dead organic matter in soil when the crop is harvested the diseased roots are left over in the soil. It also produces chlamydospores that survive in soil and becomes active in the next cropping season. The perfect stage is unknown.

5.2.4 Integrated Management

Fusarium wilt being both soil- and seed-borne is difficult to manage by chemical121alone which may not be practically feasible. Accordingly, this calls for an integrated122approach, involving chemical, biological, and genetic approaches. Several attempts123have been made by several workers to manage this disease biologically.124

Chickpea fusarium wilt is mainly driven by the pathogen inoculum as it is a 125 monocyclic disease. Therefore, its management should aim at excluding the pathogens and decreasing the original inoculum quantity by using measures like (i) 127 pathogen-free seeds; (ii) avoiding sowing in disease-affected soils; (iii) elimination 128 or reducing of soil inoculum; (iv) resistant varieties; (v) seed treatment with biocontrol agents or fungicides; and (vi) avoiding cropping patterns which favor infection 130 by the pathogen (Jiménez-Díaz et al. 2015). 131

Bio-priming of seeds with P. fluorescens effectively controlled chickpea wilt dis-132ease in addition to increased yield. The seed treatment of P. fluorescens followed by133its application in the root zone has not only increased the efficacy of P. fluorescens134formulations but also enhanced the chickpea yields. Pseudomonas fluorescens does135not have any adverse effect on the beneficial N-fixing bacteria, viz., Rhizobium and136Azospirillum, and P. fluorescens were not inhibited by the thiram and carbendazim137seed treatment fungicides (Vidhyasekaran and Muthamilan 1995).138

The practical and cost-efficient individual measures for wilt management include 139 developing and using high-yield cultivars which are resistant to the common patho-140 genic races(s) of fusarium wilt in a specified region. Fusarium wilt management 141 could be helped by the use of plants that do not have any pathogens (Pande et al. 142 2007), sanitary procedures and soil inoculum reductions, selection of sites, and atten-143 tion to reducing the disease capacity and the protection of plants with fungicides. 144 For the characterization and tracking of Fusarium, molecular protocols are accessible. 145 In the course of the integrated management strategy, the improved management of 146 these disease control interventions can be further achieved through mixing slow-147 wilting cultivars (Jiménez-Díaz et al. 2015). 148

Effective test fungicides, bioagent, and organic amendments were evaluated for 149 integrated management of fusarium wilt. The seed treatment with the combination 150 of carbendazim, thiram, *Trichoderma viride*, and *P. fluorescens* followed by soil 151 application with neem seed cake powder was found to be an effective treatment 152 which resulted in significantly higher seed germination, lower incidence of wilt, and high seed yield compared to control treatment (Thaware et al. 2016). 154

There have been significant advances in identifying the desi and kabuli chickpea 155 germplasm types and in developing productive high-performance 'Kabuli' culti-156 vars with full resistance to more strains of the pathogen. There have also been 157 substantial advances in the breakdown of racial resistance genes. This would allow 158 further advancement in pyramiding of various strain-specific resistances in chick-159 pea, which would increase the efficiency in multilocations and possibly merge this 160 with resistance to other major diseases, viz., root-knot and cyst nematodes and 161 blight, and tolerance to drought. But resistance hasn't been broken up to date by 162 the use of racially specified resistant cultivars. Pre-planting of the existing patho-163 gen with molecular protocols would assist to prevent the affected soils. In chickpea 164

165 germplasm, slow-wilting resistance is also recognized. Increased effectiveness of 166 the integrated wilt management in chickpea would be combined with other pre-167 planting disease control practices, viz., pathogen-free seeds, avoiding sowing in 168 disease-affected soils, elimination or reducing of soil inoculum, resistant varieties, 169 and seed treatment with biocontrol agents or fungicides, which would control the 170 fusarium wilt in chickpea.

Marker-assisted introgression was performed with foreground selection with SSR markers TA 37 and TA110 in Pusa 256 (elite desi cultivar) and with background selection with 45 SSRs accommodating 8 multiplexes to get the higher recovery of recurrent parent genome. Finally, there have been acquired 17 BC3F4 and 11 BC3F3 lines that have resulted in the detection of 5 high-resistance Pusa 256 strains with Foc 2 genes. This will assist the development of chickpea horizontally and vertically in India (Aditya Pratap et al. 2017).

178 5.3 Ascochyta Blight

It is the most important disease reported from 25 countries around the world (Singh 179 et al. 1984) that includes Europe, North Africa (bordering Mediterranean Sea, Iran, 180 Iraq, Pakistan, Portugal, Romania, Spain, the USA, USSR (formerly), Mexico, 181 Tanzania, Bangladesh, and India, while it is not reported in chickpea areas of Nepal, 182 Myanmar (Burma), Argentina, Bolivia, Peru, Chile, Libya, Columbia, Malawi, Zambia, 183 Sudan, Uganda, and Yugoslavia. In India chickpea blight is common in Punjab, 184 Haryana, Himachal Pradesh, Northwest Uttar Pradesh, and Bihar, Madhya Pradesh, 185 but not from Andhra Pradesh. Recently its incidence has been observed from Karnataka 186 state also. During the 1930s total loss due ascochyta blight in Spain was reported, and 187 losses up to 25–50% were reported during 1922–1933 from undivided Punjab (before 188 partition of Pakistan). In Rajasthan, 5-75% losses have been observed in 1982, under 189 favorable environment disease severity increases resulting in losses up to 100%. 190

191 5.3.1 Symptoms

It occurs in all parts of the plant above ground. On the leaves and pods, circular 192 spots develop and elongated spots on the petioles and stems. These leaf spots can 193 have brown dots with a brown-red margin. On coalescence, the places turn whole to 194 leaf gray with a scorched look. The lesions on green pods are curved and dark in the 195 edges and are placed in a concentrated circle with pycnidia. In the clusters of seeds, 196 lesions can also appear. In stems and petioles, the red with black dots are elongated 197 that may cover the impacted area. The sections above these lesions drop out and die 198 when such places girdle the stem entirely (Fig. 5.1). The whole plant dries when the 199 main stem is located at the bottom (neck area). As the disease progresses, patches of 200 drooping and wilting crops can subsequently spread to whole areas. The distribution 201 may be limited in dry weather, but it extends quickly in moist conditions. 202

5.3.2 Causal Organism

Ascochyta rabiei (Pass.) Labrousse. Also referred to as *Phyllosticta rabiei* (Pass.) or
204 *Phoma rabiei* (Pass.), the pathogen belongs to subdivision, Deuteromycotina; class,
Coelomycetes; order, Sphaeriales; and family, Sphaeropsidaceae in which globose,
dark pycnidia with hard textured walls are formed.
207

The pathogen produces hyaline to brownish septate mycelium. The pycnidia are 208 produced on leaves, stem petioles, and pods including seeds which are erumpent, 209 globose dark brown 140-200 µm in diameter with a prominent ostiole. The perfect 210 stage (observed in Bulgaria by Kovachevski in 1936) described as Mycosphaerella 211 rabiei Kov. (later renamed as Didymella rabiei (Kov.)) belongs to the family, 212 Dothideaceae; order, Dothideales; and class Loculoascomycetes of Ascomycotina. 213 The pseudothecia (perithecia in locules) contain eight small ascospores, immersed 214 in host tissues (dead parts or in crop debris) dark brown or black globose and mea-215 sure $120-250 \times 75-152 \mu m$. They contain cylindrical-clavate asci slightly curved 216 pedicellate which measures $48-70 \times 9-13.7 \mu m$. The ascospores are one septate and 217 one cell is bigger than the other prominently formed at the septum and measure 218 $12.5-19.0 \times 6.7-7.6 \mu$ m; however, in Indian conditions, these perfect stages are not 219 observed as hot summer conditions prevail after the cropping season. 220

5.3.2.1 Races

Based on the reactions of the cultivars, the population of A. rabiei were grouped 222 into seven races, and differential cultivars for each race were identified. The iso-223 lates were also analyzed for their genetic diversity using ITS, URP, and SSR mark-224 ers (Baite and Dubey 2015). The presence of races could not be found by Luthra 225 and others in 1939 and by Arif and Jabbar (1965). An anonymous study from India 226 (1963) indicated that genotype C-12/34 broken its resistance due to a new strain. In 227 controlled environments, scientists examined variations in fungal isolates. Based 228 on the symptoms, the pycnidial formation and the pathogenic behavior of the 229 eleven isolates were found and several races exists in Panjab, India. Further, find-230 ings from the Chickpea International Ascochyta Blight Nursery were also indi-231 cated the presence of races. Intensive race studies are needed to identify stable host 232 resistance (Nene, 1981). 233

5.3.3 Disease Cycle

Blight pathogen survives as pycnidia in seeds and plant debris that is a major source235under Indian conditions. However, pycnidia survive for more than 2 years in crop236debris depending on temperatures (10–35 °C) and RH 65–100%. The fungus survives on the seed coat, cotyledons, and embryo for >5 months.238

The pathogen spreads from these sources (infected debris and seeds) by rain 239 droplets in windy weather, by insects and contact between leaves, and by movement 240

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of animals through the field. The 22–26 °C temperatures and high rainfall conditions are conducive for disease development at all crop growth (seedling to pod
formation) stages. The pathogen has been noticed on berseem also with cross inoculums from these counterpart hosts, besides common bean (*Phaseolus vulgaris*).

245 5.3.4 Integrated Management

Genetic resistance to ascochyta blight: The resistance to G-52 isolate of ascochyta blight in chickpea was under the control of single dominant gene pair in the
I-13 resistant variety (Satya Vir et al. 1975).

There have been efforts to identify sources of resistance, resistance breeding, and genetic variability between the blight pathogen races. Importance of the genotype x environment interaction in elucidating aggressiveness of isolates from different places and identifying pathotypes and stable sources of resistance has been recognized. The current blast resistance breeding programs rely on crossing durable and adaptive cultivars, stable performance of breeding lines through multilocation testing, and the marker-assisted selection (Sharma and Ghosh 2016).

Molecular diversity analyses of Indian isolates of Ascochyta rabiei: About 11 256 AFLPs and 20 SSR markers were evaluated in 64 isolates obtained from various 257 agroclimate areas in North Western Plains Zone (NWPZ) India for the study. 258 Some 9 polymorphic AFLP primer pairs produced 317 fragments with a median 259 PIC value of 0.28, 130 of them are polymorphic. Of the SSR markers, 12 were 260 polymorphic and had an average PIC value of 0.35 with a total of 29 alleles. This is 261 the first AFLP and SSR diversity assessments in A. rabiei in the best of our under-262 standing. The dendrograms were created respectively and placed the series of AB 263 isolates in geographical areas based on AFLP and SSR information and the merged 264 variable dataset. The population structure assessment model disclosed that 4 separate 265 populations of different concentrations of ancient admixtures were explored between 266 64 isolates. Interestingly, several SSR markers and AFLP primer combinations 267 showed the locus/allele specific to AB isolates from certain regions, viz., Gurdaspur, 268 Hisar, Sundarnagar, and Sriganganagar. Genetic variability found in Indian NWPZ 269 AB isolates indicates that modifications in A. rabiei population should be monitored 270 continuously to prevent the collapse of resistance in chickpea cultivars. 271

272 Management

- Good Agronomic Practices (GAP) such as deep plowing, deep sowing, removal and destruction of crop debris, and crop rotation need to be followed.
- Intercropping with cereals reduces the disease spread (chickpea-barley).
- Application of 40–60 kg potash +20 kg nitrogen +40 kg phosphorus was reported to reduce the disease severity and increase grain yield (Tripathi et al 1987).
- Seed treatment with copper sulfate, thiram, or Calixin M (this last named fungicide completely eradicates the seed inoculum). Tripathi et al. (1987) have

reported successful control, by seed treatment with carbendazim + thiram (1:3 280 ratio) @2.5 g/kg seeds followed by three times spraying of carbendazim 281 @0.5 kg/ha at 10-day interval. 282

- In mild infections, spraying of zineb, ferbam, maneb, or captan and Daconil, 283 Rovral, Calixin M, tebuconazole, difenoconazole, chlorothalonil (Bravo), or azoxystrobin (Amistar 250 SC) can be taken @0.1% to 0.2%. Four to six sprays may be required depending upon disease severity and stage of the crop. 286
- Use of resistant varieties (ICRISAT and other centers in the country) such as F-8, 287 C 325, C 727, I 13, EC 26414, 26,435, and 26,446. The Kabuli types ILC 3664, 288 3870 and 4421, and C 215 have been reported to be resistant to blight. Generally, 289 Kabuli types are more resistant than desi chickpea or gram. It has observed that 290 the resistant genotypes are hairier than susceptible plants that produce more 291 maleic acid than healthy plants. Erect growth, less lateral spread, high hairiness, 292 high peroxidase activity, lesser maleic acid content, higher L-cystine, and pheno-293 lic contents are the attributes of resistant varieties. 294

5.4 Rust

The rust of gram is reported from >15 countries. The disease is widespread in sev-296eral parts of India including Maharashtra, Tamil Nadu, Bihar, West Bengal, Uttar297Pradesh, and Punjab and recently in many places of Karnataka.298

5.4.1 Symptoms

The rust appears around 4-month-old crop (January–February) on small leaves and 300 light-dark brown pustules which tend to coalesce to form bigger pustules which 301 may develop on either side of the leaf preferably on the lower surface and covers the 302 entire leaf area later. Often the pustules appear on the stem, petioles, pods, and floral 303 parts. In advanced stages, dark telial stages appear in rust pustules (Fig. 5.1). 304

5.4.2 Causal Organism

Uromyces ciceris-arietini (Gregnon) Jacs. The pathogen was first detected and 306 described in France in 1863. The pycnidial and aecial stages of rust pathogen are 307 unknown. The uredia are hypophyllous, scattered minute round powdery when 308 mature light brown. The urediospores are globose, loosely echinulate, 20-28 µm in 309 diameter, and yellowish brown in color. The telia appear late in the season (March-310 April) and resemble uredia except for dark brown color. The teliospores are round 311 or oval or warty or angular with a roundish unthickened apex. The wall is brown and 312 warty and measures $18-30 \times 8-24 \mu m$ with short hyaline pedicel. 313

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314 5.4.3 Disease Cycle

Rust fungus will survive by repeating its uredial stage, while the role of telia is 315 unknown. The pathogen is known to infect the legume weed *Trigonella polycerata* 316 and Lathyrus spp. and collateral hosts of Uromyces ciceris-arietini on high hills in 317 summer and provide inoculums to the main host. The disease is favored by tempera-318 tures of 11–30 °C; the uredospore germination takes place in leaf exudates of sus-319 ceptible varieties as resistant varieties are low in leaf exudates. Leaves of resistant 320 varieties Nandrival 49 contain more of maleic acid and sucrose than susceptible 321 genotype Agra local (Bahadur and Sinha 1970). 322

323 5.4.4 Integrated Management

Image processing of rust disease: Automatic plant disease detection is an impor-324 tant aspect, which can demonstrate advantages in the surveillance of wide crop 325 areas and therefore automatically identify disease symptoms when they appear on 326 plant leaves. Costs and inaccuracies may be the problem with sheer naked eve 327 monitoring for the detection and classification of diseases. The model suggested 328 offers a software alternative for the traditional techniques integrated into the iden-329 tification of programmed crop diseases with the use of the picture handling method. 330 This system is beneficial for farmers to control the disease spread. It also offers 331 precise outcomes with naked eyes. The method commences with chickpea leaving 332 from the field being captured. Captured pictures are filtered, and then the green 333 pixels are disguised and deleted with a certain limit value. The complete area on 334 the disease-affected leaf and the good region is calculated based on the result. 335 Texture characteristics are finally obtained (Shivanand et al. 2014). 336

Rust resistance in chickpea germplasm collection: A collection comprising 140 337 chickpea lines and 109 related wild (Cicer spp.) species has been screened for 338 chickpea rust resistance. Different levels of partial resistance have been identified 339 based on reduced disease seriousness and disease progression area, curve, and host 340 cell necrosis macroscopically visible. In wild Cicer species, higher rates of resis-341 tance but not linked with hypersensitivity were found macroscopically and micro-342 scopically, and resistant components were researched in chosen C. arietinum 343 accessions. During the long latent period a reduced infection was expressed that 344 are associated with a greater percentage of early colonies aborted, a decrease in 345 the amount of haustorial colony and mother cell and a reduction in the size of the 346 colony (Sillero et al. 2012). 347

348 Management

- 1. Early sowing is known to provide disease escape mechanism.
- 2. Many antagonistic fungi suppress spore germination of *Uromyces ciceris-arietini*.

 Growing rust resistant chickpea line viz., NRC 34, NEC 249, JM 583, and 2649, 352 HPC 63, HPC 136 and HPC 147 is recommended in rust epidemic area. 353

5.5 Dry Root Rot of Chickpea

5.5.1 Symptoms

This disease usually occurs as scattered dead plants around flowering and podding 356 time. Petioles and leaflets are drooped at the bottom of the plant. Uppermost leaves 357 are chlorotic when the remaining are dry on the plant. The taproot is pale and has 358 indications of drying, and most of its lateral and finer branches are empty. 359

5.5.2 Causal Organism

In the altered climate situation, chickpea dry root rot induced by *Rhizoctonia batati*-361 cola (Macrophomina phaseolina) is gaining significance when increasing crops are 362 exposed to elevated temperature and water stress. Many soil and climate variables 363 are accountable for disease growth as these are primarily soilborne pathogens. So 364 far, there has been no systemic ecological, biological, and epidemiologic study 365 linked to dry root rot in chickpea. Investigations are required to enhance the charac-366 terization and identification of variation within its pathological and epidemiological 367 niches. A limited accessible manuscript on HPR of dry root rot indicates that the 368 disease has no resistant sources (Sharma et al. 2016). 369

The DRR was initially reported by Mitra (1931) in India subsequently, in Iran 370 (Kaiser et al. 1968), the USA (Westerlund et al. 1974), and several Asian and 371 African countries (Nene et al. 1996). The disease was formally recognized in 372 chickpea as "rhizoctonia wilt," but was subsequently called as "dry root rot." In the 373 recent years, changes in weather conditions, especially owing to a long drought, it 374 has become an extremely serious risk to chickpea production. Chickpea is predis-375 posed to DRR utilizing elevated temperature and depletion of soil water during 376 plant development, especially post-harvest stages (Sharma and Pande 2013). The 377 wide and enhanced prevalence of DRR in Central and Western India was stated in 378 recent 2010–2013 studies (Ghosh et al. 2013). Regardless of soil, cultivars, and 379 cropping systems, diseases were detected, and their prevalence ranged from 5 to 380 50% in poorly affected soils. 381

Dry root rot is an important biotic limitation for chickpea production. A total of 94 isolates from various agroclimate areas of India were analyzed with AFLP. Distinct morphological characteristics were evaluated to identify the variety of *Rhizoctonia bataticola* species in India. *Rhizoctonia bataticola* species were varied in terms of distinct moral and cultural parameters from various agroecological areas such as colony color, development pattern, development frequency, mycelial characteristics, 387

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sclerotial intensity, sclerotial initiation time, and sclerotial morphology. A total of 388 121 fragments were obtained from five AFLP primer combinations. All fragments 389 were found to be polymorphic with an average value of 0.213 for polymorphic 390 data content. Based on AFLP assessment, the dendrogram found that the highest 391 amount of isolates of Rhizoctonia bataticola was varied and did not rely on geo-392 graphical origin. Morphological and molecular information linked and endorsed 393 the diversity and independence of the Rhizoctonia bataticola found in India 394 (Sharma et al. 2012). 395

Dry root rot external expression: phenotypical modification: DRR signs are 396 most often seen in the afterblown phase of chickpeas, which includes drooping and 397 chlorosis of leaflet which is restricted to top plant leaves. The plant leaf and stalks 398 are generally straw-colored and the reduced branches and stalks are gray in some 399 cases. The root of the tap has red symbols that become black and absent in most of 400 the lateral and softer components. The radicals that died are quite fragile and bark 401 tipped. The roots revealed and the inner part of the bark, or when divided up verti-402 cally on the collar region, are observed with dark sclerotic minute bodies (Sharma 403 et al. 2016). 404

405 5.5.3 Disease Cycle and Histopathology

DRR is usually caused by the presence of hyphae and sclerotia in the soilborne 406 inoculum. The pathogen creates epidermal cell death and penetrates the roots. 407 Mechanical plugs of the xylem cells by micro-sclerotia, enzyme action, toxin pro-408 duction, and mechanical stress lead to disease development and direct secretion of 409 macerating enzymes (Sharma et al. 2004). The pathogen may also cause disease 410 during the formation of cotyledons, through tiny rootlets/injuries on the root sur-411 face. The fungus develops within the cell as well as between the cells of cortical 412 tissue. It mainly grows intercellular, forming thick and dark-colored cells which 413 lead to large necrotic lesions that are depressed. Invaded cortical cells cause the 414 roots to decay or to rot severely (Singh and Mehrotra 1982). The vascular system 415 and the sclerotic bodies of the pathogen are colonized by hyphae. The level of root 416 necrosis rises gradually over time without obvious signs in the above ground until 417 blooming and podding. 418

419 5.5.4 Integrated Management

Host plant resistance: There have been so far researches on DRR resistance in
chickpea, as neither demonstrated significant resistance to DRR. Comprehensive
list of scientists worked on DRR resistance breeding and their findings on sources
of DRR resistance/tolerance (Table 5.1).

Table 5.1 Resistance sources for dry foot for disease in chickpea		
Chickpea lines	DRR disease reaction	Reference
GCP-101, GBM-2, GBM-6, and ICCV-10	Tolerant	Jayalakshmi et al. (2008)
ICCV-97112	Resistant	Iftikhar and Ilyas (2000)
ICCV-05530, ICCV-08305, ICCV-05529, ICCV-05532, ICCV-07117, and ICCV-07112.	Moderately resistant	Sharma et al. (2016)

+1 1

t2.1

Table 5.1 Resistance sources for dry root rot disease in chickness

 Table 5.2 Important cultural practices to avoid the DRR incidence in chickpea

Sl. no	Cultural method	Reference
1	Manipulation in the date of sowing, i.e., timely or early sowing followed by scheduled irrigation can avoid the elevated	Singh et al. (1990)
	temperatures thereby reducing the DRR	
2	Crop rotation with non-host crop plants	Singh et al. (1990)
3	No-tillage	
4	Deep plowing and removal of infected debris for the reduced sclerotial multiplication	Ð

Table 5.3	Important biological control measures to avoid the DRR	incidence in chickpea	t3.1
Sl. no.	Biological control	Reference	t3.2
1	Seed treatment with Trichoderma viride	Sharma and Gupta (2004)	t3.3
2	Application of antagonistic <i>Trichoderma virens</i> and organic amendments like FYM	Thilagavathi et al. (2007)	t3.4 t3.5
3	A combination of biocontrol agents, viz., T. viride, Pseudomonas fluorescens, and Bacillus subtilis	Thilagavathi et al. (2007)	t3.6 t3.7

Inheritance of DRR resistance: DRR resistance inheritance study reveals that it is 424 controlled by dominant monogenic genes (Rao and Haware 1987), in which two resis-425 tant (H-208 and K-850) and two sensitive relatives (C-104 and P-165) have been used. 426 Even resistant parents had signs of the disease if the crops were cultivated in infected 427 soil for a longer period. More refinement of screening techniques is needed as well as 428 further confirmation of resistance sources in regulated environments and field. 429 This brings the breeding of chickpea resistance to DRR in a scenario of uncertainty, 430 especially now when the climate is unsafe. Besides, no study has been recognized so 431 far regarding any molecular markers associated with the DRR gene. 432

Cultural control: The incidence rates of the disease can be decreased by cultural 433 methods as listed below that can lead to a decreased occurrence of DRR (Table 5.2). 434

Biological control: Some of the important biological control measures are given in 435 Table 5.3. 436

Chemical control: Seed treatment with fungicide is effective in reducing the 437 losses due to Rhizoctonia bataticola. Some of the chemical control measures are 438 listed in Table 5.4. 439

Table 5.4 Important chemical control measures to avoid the DRR incidence in chickpea		
Sl. no.	Seed treatment chemicals	Reference
1	Carbendazim, thiophanate-methyl, and Vitavax	Sharma and Gupta (2004)
2	Carbendazim or in combination with thiram (soil drench and seed treatment + drenching after sowing)	Sharma and Gupta (2004
3	Bavistin and thiram	Ghosh et al. (2013)

Table 5.4 Important chemical control measures to avoid the DRR incidence in chickness

Botrytis Gray Mold 5.6 440

5.6.1 **Symptoms** 441

At any stage of development, plants can be targeted by the pathogen gray mold, 442 which is most probably found at the bottom of the stalk of the collar region as soft 443 rot. In the beginning, the tissues in the injured condition are coated with a fuzzy 444 gray mold, and as the disease develops, plants will be desiccated and die. On the 445 surface of the affected tissue, small black sclerotia can occur when the plant dies. In 446 older crops, only a few parts of the plant are occasionally damaged, and the remain-447 ing appears to be quite regular. The disease with seedlings can trigger damping and 448 significant thinning. 449

Causal Organism 5.6.2 450

A fungus known as *Botrytis cinerea* causes this disease. The disease can grow quickly, 451 distributed extensively, and trigger a complete loss of yield under favored circum-452 stances. Genotypes of chickpea with strong seedling development, early flowering, 453 and early canopy closure are amenable for disease development compared to other 454 varieties. Total crop failure is reported during the use of heavily infected seeds and 455 when seed treatment is not followed in some cases. Crop losses in moist periods are 456 highest, especially when plants are developing very thick canopies. 457

Disease Cycle 5.6.3 458

As soilborne sclerotia and saprophyte grow on decaying crop waste, the fungus sur-459 vives on infected plants. The disease often occurs through the sowing of infected 460 seeds in fresh fields. On infected crops, masses of spores are generated. The fungal 461 spores can be transmitted through air currents from one crop to another crop and dis-462 tributed quickly. The hot, damp circumstances under the plant canopy offer perfect 463 circumstances for infection and disease propagation once the plant has established. 464 Botrytis Grey Mould management in chickpea is indicated in Table 5.5. 465

	5 5		
Sl. no.	Methods	Practices	t5.2
1	Cultural method	By use of disease-free seeds	
		Low seed rates	t5.4
		Wider row spacing	t5.5
2	Biocontrol method	Soil or seed or foliar treatment of Trichoderma harzianum	t5.6

t5.1

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Table 5.5 Botrytis Grey Mould management in chickpea

5.7 Other Minor Fungal Diseases of Chickpea

5.7.1 *Powdery Mildew* (Leveillula taurica)

Symptoms	468
 Oidiopsis type of powdery mildew in which the mycelium is endophytic. 	469
• The affected leaf shows powdery patches on the lower surface corresponding	470
with yellowing on the upper surface.	471
Older leaves show symptoms first.	472
• There will be premature defoliation of affected leaves.	473
• Airborne	474
Management	475
Spray carbendazim 1 g/lit or carbendazim + mancozeb (1 g/it) or wettable sulfur	476
2.5 g/lit.	477
SO	
5.7.2 Blight (Alternaria alternata)	478
Symptoms	479
• The disease occurs at flowering stage.	480
 Leaves are infected most. 	481
Shedding of infected lower leaves.	482
• Small, circular, water-soaked, and purple lesions are seen on leaflets	483

Small, circular, water-soaked, and purple lesions are seen on leafletsInfected pods become blackish and seeds shriveled.

Management

•	Space planting	486
•	Reduced vegetative growth	487
•	Intercrop with linseed	488
•	Limited irrigation	489
•	Compact varieties	490

t6.1

• Mancozeb @ 2.5 g/lit or carbendazim @ 1 g/lit

492 5.8 Host Plant Resistance and Molecular Markers for Major 493 Fungal Diseases of Chickpea

The chickpea has a limited genetic base and often does not have sources of resis-494 tance to several stresses including major fungal diseases in the cultivated germ-495 plasm. Thus, it is critical for the development of different cultivars to diversify and 496 broaden the genetic base using wild relatives. In the past, some attempts have been 497 done to monitor germplasm samples for valuable DNA to resist ascochyta blight. 498 fusarium wilt, botrytis gray mold, and other diseases under field and controlled 499 circumstances. Through such attempts, precious resistance sources have been iden-500 tified to these major fungal diseases in chickpea (Table 5.6). Efforts to develop 501 genomic resources resulted in the identification of molecular markers for agronomic 502 and biotic stresses, enabling the use of genomics-assisted breeding in chickpea crop 503 (Varshney et al. 2013a). In the recent past, marker-assisted selection tool using SSR 504 and SNP resources and density genetic map of chickpea have significantly aug-505 mented the chickpea breeding programs effectively and efficiently (Varshney et al. 506 2010; Kumar et al. 2011). Furthermore, genome sequencing of 90 chickpea has 507

Major disease	Resistance sources	References
Ascochyta blight	ILC 72, ILC 191, ILC 196, ILC 201, ILC 202, ILC 2506, ILC 2956, ILC 3274, ILC 3279, ILC 3346, ILC 3856, ILC 3956, ILC 3996, ILC 4421, ICC 3634, ICC 4200, ICC 4248, ICC 4368, ICC 5124, ICC 6981, ILWC 7–1, ILWC 33/S-4, 03039, 03041, 03053, 03115, 03131, 03133, 03143, 03159, 93A-086, 93A-111, 93A-3354	Malhotra et al. (2003) Ilyas et al. (2007) Kumar et al. (2011)
Fusarium wilt	JG 16, JG 62, ILC 482, C-104, GJ 74, WR 315, K-850, KWR 108, L-550, BG 212, BG 215, Ghaffa, CPS-1, UC 27, Vardan, Vijay, Vishal, Annigeri, ILWC 7–1, ILWC 33/S-4, CM 368/93, CM 444/92, FLIP 00-17C, FLIP 02-7C, FLIP 02-9C, FLIP 02-40C, FLIP 02-47C, FLIP 03-26C, FLIP 03-29C, FLIP 03-57C, FLIP 03-108C, FLIP 03-127C, FLIP 05–28, FLIP 05-68C, FLIP 05-72C, FLIP 05-85C, FLIP 05-106C, FLIP 90-131C, FLIP 99-66C	Sharma et al. (2005) Sharma and Muehlbauer (2007) Singh et al. (2009) Ali et al. (2011) Kumar et al. (2011)
Botrytis gray mold	ICCV 2, Pusa 209, Gaurav	Singh et al. (2009)
Rust	FLIP05-74C, PI 593072, PI 642748	Rubiales et al. (2001)

 Table 5.6
 Resistance/tolerance sources to major fungal diseases of chickpea

accelerated the development of disease resistance lines from molecular breeding efforts (Varshney et al. 2013b). However, this has some limitations, viz., not all the genes or QTLs for major diseases are fine-mapped and new sources of resistance need to be genotyped (Zhu et al. 2008).

5.9 Future Prospects

The cultivated chickpea has limited variability that necessitated using wild *Cicer* 513 species having a high degree of resistance to many biotic and abiotic stresses. 514 Transferring resistance and other desirable gene complexes from such unexploited 515 wild to cultivated species through hybridization are limited by reproductive barriers 516 that can be overcome by using novel biotechnological approaches. Further, a greater 517 understanding of the genetic bases of virulence, mechanism of resistance, and host-518 pathogen interactions is required to enhance the breeding efficacy in chickpea. 519 Minor diseases have been poorly studied due to difficulty in resistance screening 520 and other reasons which require much more attention in the context of the climate 521 change scenario. 522

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Chapter 6 Wilt and Root Rot Complex of Important Pulse Crops: Their Detection and Integrated Management

Nidhi Kumari and Shabnam Katoch

6.1 Introduction

Attainment of self-sufficiency in crop production is the only way to fulfil the food 7 requirement of expanding population; though India has come a long way from a 8 pulse-deficient country to self-reliant one, still there are so many factors which con-9 tribute towards low production of agricultural goods. Among different cultivated 10 crops, pulses are in the midst of imperative sources which have a say to the nutri-11 tional security of a country (Singh et al. 2015). Pulses are protein-rich commodity 12 which in addition to the fulfilment of protein requirement also improves the soil 13 fertility (Narayan and Kumar 2015; Singh et al. 2018). Throughout the world, 14 India has the biggest contribution in the production and consumption of tropical and 15 sub-tropical pulse crops such as gram, red gram, black gram, green gram, field pea 16 and lentil (Srivastavaa et al. 2010; Singh et al. 2017a; Hasan and Khan 2018). In 17 India, pulses are cultivated in 294.65 Lakh hectares of land with the annual pro-18 duction of 22.95 MT (http://agricoop.nic.in/sites/default/files/Krishi%20AR%20 19 2017-18-1%20for%20web.pdf), out of which the maximum share of 77.0% is col-20 lectively from Madhya Pradesh, Maharashtra, Uttar Pradesh, Karnataka, Andhra 21 Pradesh and Rajasthan followed by only 23% from Gujarat, Chhattisgarh, Bihar, 22 Orissa and Jharkhand (Trivedi et al. 2017; Singh et al. 2018). In spite of country's 23 autonomy in pulse production, there are some supply and demand affecting ambi-24 guities like unpromising weather, several agronomic limitations, insect-pests and 25 diseases and inappropriate marketing. Out of all, the effect of microbes on plant 26 growth and development has arrived as a major apprehension among the pulse 27

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B. P. Singh et al. (eds.), *Management of Fungal Pathogens in Pulses*, Fungal Biology, https://doi.org/10.1007/978-3-030-35947-8_6

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growers of India. Out of ~100 fungal pathogens, the soilborne disease incidents 28 causing wilts and root rots have been a matter of worry for sustainable production 29 of pulses since couple of years and are reported to cause considerable yield losses 30 in pulses (Trivedi et al. 2017), sometimes even up to 100% (Sinha et al. 2018). The 31 complex nature of these diseases further aggravates the problem. The vascular wilt 32 caused by Fusarium sp. is one of the major threats to pulse growers throughout the 33 world (Sinha et al. 2018). Keeping in view the importance of pulses, different man-34 agement strategies, viz. cultural, physical, biological and chemical control methods, 35 have been used to manage wilt and root rot diseases/complex, but till date, apart 36 from the high cost and deleterious effects of chemicals, their use is considered as the 37 quick and accurate way of disease management. But as far as soilborne pathogens 38 are concerned, the sole use of fungicides may not lead to their proper management. 39 And due to the increasing awareness regarding health hazards caused by the intake 40 of food with pesticide residues, scientists are looking for integrated management 41 strategies which are not solely dependent on the use of chemicals. Before the imple-42 mentation of any management strategy, the accurate detection of diseases is also 43 very important. The early detection of diseases at their onset helps farm scientists 44 and farmers to plan as well as execute effective integrated management strategies. 45 From the last many years, the conventional methods are in use for pathogens detec-46 tion, but these methods sometimes lead to confusing conclusions and are not as 47 accurate as DNA and protein-based methods (Katoch et al. 2019). In this chapter, 48 we will discuss the different wilts and root rots (Table 6.1) causing considerable 49 losses to important Indian pulse crops and their integrated/holistic management. 50 In addition to this, recent diagnostic methods used for early and timely detection 51 will also be discussed. 52

53 6.2 Root Rot of Pulse Crops

Root rots are the diseases of utmost importance impacting a wide range of crops 54 worldwide. Often root rot is a complex disease where more than one pathogen is 55 involved. Fungi, oomycetes, bacteria and viruses have been reported to cause root 56 rots (El Karkouri et al. 2010; Legg et al. 2011; Heffer Link et al. 2002; Cleary 57 et al. 2011; Cui et al. 2014). Frequently nematodes have been reported to aggra-58 vate the problem by facilitating the entry of other pathogens through wounds 59 made by nematodes while feeding (Back et al. 2002). Initial symptoms appear on 60 the roots of the affected plants which go unnoticed or are not visible. Till the 61 aboveground symptoms become noticeable, sufficient losses to the plant health 62 have already occurred; thus it becomes almost impossible to recover the plants. 63 Root rot is favoured by poor drainage conditions, moderate to high soil moisture, 64 monocropping, etc. 65

Crop	Disease	Causal organism	References
Gram	Dry root rot	Rhizoctonia bataticola (taub) Butl. (Pycnidial stage: Macrophomina phaseolina Tassi Goid)	Pandey et al. (2017); Kadam et al. (2018); Sunkad et al. (2018)
	Collar rot	Sclerotium rolfsii (Teleomorph: Athelia rolfsii (Curzi) Tu and Kimbrough)	Ghosh et al. (2013); Ahsan et al. (2018)
	Black root rot	Fusarium solani	Ghosh et al. (2013)
	Wilt	<i>Fusarium oxysporum</i> f. sp. ciceris	Pandey et al. (2017); Sankar et al. (2018)
Red gram	Dry root rot	Rhizoctonia bataticola (Taub.) Butler (Macrophomina phaseolina (Tassi) Goid)	Maruti et al. (2017)
	Wilt	Fusarium udum Butler	Chennakesavulu et al. (2013); Singh et al. (2016); Saxena et al. (2012); Sharma et al. (2018)
Black gram	Dry root rot	Rhizoctonia bataticola (Taub.) Butler (Macrophomina phaseolina (Tassi) Goid)	Tetali et al. (2015)
Green gram	Root rot	Rhizoctonia bataticola (Taub.) Butler (Macrophomina phaseolina (Tassi) Goid)	Sarkar and Bhattacharyya (2008); Mallaiah and Rao (2016); Shahid and Khan (2016)
	Seedling rot and web/ leaf blight	Rhizoctonia solani Kuhn (Thanatephorus cucumeris)	Singh et al. (2013)
Field pea	Rhizoctonia root rot	Rhizoctonia solani	Rawat et al. (2014)
	Wilt/root rot complex	<i>Fusarium oxysporum</i> f. sp. <i>pisi</i> <i>Fusarium solani</i> f. sp. <i>pisi</i> (Jones) Synder and Hansen	Rao (2014); Thakur et al. (2016); Nongmaithem et al. (2017)
Lentil	Collar rot or root rot	Sclerotiun rolfsii (Athelia rolfsii)	Surulirajan et al. (2007); Kushwaha (2016); Tiwari et al. (2018)
	Rhizoctonia root rot	Rhizoctonia solani	Tiwari et al. (2018)
	Wilt	Fusarium oxysporum f. sp. lentis	Garkoti et al. (2013); Singh et al. (2017a, b); Arya and Kushwaha (2018)

Table 6.1 List of important wilt and root rots of major pulse crops in India

6.2.1 Dry Root Rot of Chickpea

Chickpea (*Cicer arietinum* L.) is grown over an area of 9.53 million hectare with 67 9.07 million tonnes in India (FAOSTAT 2017). Dry root rot (DRR) of chickpea 68 caused by *Rhizoctonia bataticola* (Taub.) Butler. (synonym: *Macrophomina pha-*69 *seolina*) has emerged as a serious problem of world's second largest and India's 70 largest produced pulse crop (Sharma et al. 2015). The life cycle of *Rhizoctonia*71

t1.1

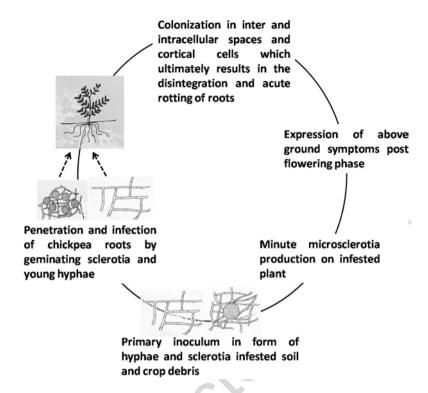


Fig. 6.1 Disease cycle of dry root rot of chickpea caused by Rhizoctonia bataticola

- *bataticola* is being illustrated in Fig. 6.1. As per recent reports after *Fusarium* wilt
- 73 (*Fusarium oxysporum* f. sp. *ciceris*), DRR is imposing humongous hazard on chick-
- pea production worldwide (Ghosh et al. 2013; Sharma et al. 2015).

Pathogen:	Taxonomic position of <i>Rhizoctonia bataticola</i> (Taub.) Butler	t2.1
Kingdom:	Fungi	t2.2
Division:	Basidiomycota	t2.3
Class:	Agaricomycetes	t2.4
Order:	Cantharellales	t2.5
Family:	Ceratobasidiaceae	t2.6
Genus:	Rhizoctonia	t2.7

Augustin Pyramus de Candolle descried the fungus Rhizoctonia (means "root 76 killer") in 1815 as plant pathogenic fungi capable of producing hyphae and sclero-77 tia. The fungus is predominantly saprophytic in nature but acts as facultative para-78 site causing diseases to many economically important crops (Ram and Singh 2018). 79 Though the accurate taxonomic name recognized is M. phaseolina (CMI descrip-80 tion of pathogenic fungi and bacteria No.275), R. bataticola is referred for the 81 sclerotial phase of the fungus (Holliday and Punithalingam 1970). R. bataticola is a 82 serious soilborne pathogen capable of infecting greater than 500 cultivated and wild 83

host plants (Maruti et al. 2017). In DRR of chickpea, only sclerotial phase is present; therefore the pathogen is referred as *R. bataticola*.

Symptoms: Symptoms are generally not visible at seedling stage. Older plants are 86 more prone to the disease (Sharma and Pande 2013). Symptoms are more evident 87 during post-flowering period as chlorosis of petioles and leaflets followed by droop-88 ing at the top of the plant. The leaves and stem become straw coloured, while few 89 times the lower stem and leaves become brown coloured (Sharma et al. 2015; Ram 90 and Singh 2018). Upon uprooting the diseased plant, blackened and rotten tap roots 91 with fewer or no lateral and finer roots are observed. These dead roots become 92 brittle with shredded bark. Microsclerotia can be clearly seen underneath the bark. 93

Disease cycle: The primary inoculum remains in soil in the form of hyphae and 94 sclerotia. The epidermal cells are dismantled by the enzymatic actions and mechani-95 cal pressure exerted by the pathogen followed by penetration of roots, though the 96 infection may also take place during emergence of seedlings through cotyledons or 97 through wounds on root surface and small rootlets. Mechanical plugging of xylem 98 vessels due to microsclerotia and toxin production also takes place during disease 99 development along with the secretion of macerating enzymes (Bhatt and Vadhera 100 1997; Sharma et al. 2004). After penetration the hyphae grow inter- and intracellu-101 larly and spread through the cortical cells which ultimately results in the disintegra-102 tion and acute rotting of roots (Singh and Mehrotra 1982). The colonization of 103 vascular system by hyphae and plugging of xylem vessels by sclerotia is observed 104 in this disease (Singh et al. 1990). As the disease advances, the root necrosis con-105 stantly extends without any evident aboveground symptoms till flowering and pod-106 ding stage (Fig. 6.1). 107

6.2.2 Dry Root Rot of Pigeon Pea

Red gram (pigeon pea, *Cajanus cajan* (L.) Millsp., 2n = 22), after chickpea, is the 109 second predominant pulse crop in India and can be cultivated in low fertilizer input 110 land or even in drought conditions. This pulse crop is famous among the small and 111 marginal farmers due to its hardy, wide adapting and drought-tolerating nature. In 112 India the cultivation of this pulse crop is expanded over 5.38 million hectare land with 113 4.87 million tonnes production (FAOSTAT 2017). Among various constraints in 114 achieving maximum productivity of pigeon pea in India, one is dry root rot of pigeon 115 pea which is distributed in Uttar Pradesh, Madhya Pradesh, Karnataka, Maharashtra, 116 Tamil Nadu and Delhi states of the country. Disease comes in severe form in late-sown 117 or summer pigeon pea as well as in perennial or rationed pigeon pea. Under favour-118 able conditions the disease may result in 100% yield loss (Smitha et al. 2015). 119

Causal organism: Sclerotial stage, *R. bataticola*; pycnidial stage, *M. phaseolina* 120

Symptoms: There is drooping and drying of leaves followed by sudden drying and 121 death of the plants. During early disease stage, on stems and branches, spindle-122 shaped lesions surrounded by brown margins with grey centres and pycnidial bodies 123

scattered all over are formed which later on coalesce resulting in drying and ultimately death of the branches or even the whole plant. The infected plants have rotten, shredded and brittle roots. Underneath the bark of finer roots, dark, blackened streaks with dark sclerotial bodies are quite evident. Prolonged hot and dry weather or drought followed by irrigation and rains favours the disease development.

6.2.3 Root Rot and Leaf Blight of Black Gram (Vigna mungo) and Green Gram (Vigna radiata)

Black gram and green gram belong to Fabaceae and are widely cultivated in Indian 131 subcontinent as sole, mixed, catch or sequential crop in kharif or summer season 132 under rainfed or semiarid conditions. In India, black gram is popularly known as 133 "urad dal" and is one of the highly prized pulse crops in India. In India, it is con-134 sumed in the form of dal (husked or non-husked, whole or split). In Indian subcon-135 tinent, green gram/golden gram also called as mung or mung bean is widely 136 cultivated as short-duration pulse crop grown in kharif, summer and spring seasons. 137 Root rot and leaf blight also called as web blight is one of the major constraints in 138 the production of black and green gram. The distribution of the disease is wide-139 spread and has been reported from India, Malaysia, the Philippines, Iran and Taiwan. 140

141 Causal organism: *Rhizoctonia solani* {Perfect stage: *Thanatephorus cucumeris*142 (Frank) Donk}

The fungus is omnipresent and can be easily isolated from infected plant part and
soil. The fungus has characteristic septate mycelium, white to deep brown in colour
with right angled branching.

Symptoms: During initial phase of the disease, the symptoms are damping off, seed and root rot, seedling blight, stem canker and web blight. On seedling hypocotyls, reddish brown sunken lesions which later on enlarge and coalesce lead to girdling of stems which ultimately results in death of the affected seedlings.

Thanatephorus cucumeris (Frank) Donk causes the web blight symptoms on the 150 foliage of black and green gram. The symptoms include yellowing of leaves fol-151 lowed by appearance of brown irregular lesions initiating from the apical portion of 152 the leaflets later on covering the entire leaf blades and then advances to the petiole 153 and stem part. The fungal runner hyphae can be seen on affected leaves, petioles and 154 stem, thus causing the typical web blight symptoms. Under severe infection, the 155 affected plant die prematurely even before the commencement of flowering stage. 156 Fewer numbers of pods with brown necrotic lesions on their surface are produced 157 by the infected plants. As the disease advances, on affected plant parts and fallen 158 leaves, an abundant number of sclerotia which are initially white in colour but later 159 on turn brown are formed. 160

Disease cycle: The fungus grows saprophytically in the soil enriched with adequate 161 amount of organic matter. The wide host range and regular addition of organic matter 162 in the soil allows the survival of pathogen in soil for longer duration. Sclerotia pro-163 duced by fungi persist in soil, and its germination is stimulated by the root exudates of 164 the host plants under favourable humidity and temperature conditions. The soil inocu-165 lum is disseminated by flooding, irrigation, movement of contaminated soil and plant 166 debris. Basidiospores are produced by T. cucumeris on healthy areas adjoining the 167 infected part which cause the aerial infection on plants. Temperature around 20 °C as 168 well as wet and alkaline soil favours the rapid disease development. 169

6.2.4 Root Rot and Damping Off of Cowpea

Cowpea [Vigna unguiculata (L.) walp] is mainly cultivated in northern and central171part of India as annual leguminous fodder crop. Cowpea is susceptible to many172insects, bacteria, fungi and viruses that are capable of infecting at all growth stages173of the crop. The root rot and damping off of cowpea caused by *R. solani, M. phaseo-*174*lina* and *Pythium ultimum* are the most devastating disease occurring as a complex.175

Symptoms: The disease is mainly characterized by rapid death of young plants.176The other symptoms include yellowing and drying of leaves, rooting of taproots177with longitudinal cracks on stems which ultimately results in poor yields.178

6.2.5 Charcoal Rot, Ashy or Stem Blight or Dry Root Rot of Soybean

Soybean (Glycine max L. Merril) is an important oilseed crop contributing about 181 25% of global edible oil (Agarwal et al. 2013). The USA, Argentina and Brazil 182 occupy top three positions of leading soybean producers in world. In India soybean 183 has been introduced by China and now is being cultivated on an area of 10.60 mil-184 lion hectare with 10.98 million tonnes production (FAOSTAT 2017) in Madhya 185 Pradesh, Maharashtra and Rajasthan which together contribute for more than 90% 186 of total production from the country. Charcoal rot, also known as DRR, dry weather 187 wilt, ashy stem blight and seedling blight disease, caused by *M. phaseolina* (Tassi) 188 Goid is one of the major diseases of soybean (Su et al. 2001). In India the disease 189 was of minor importance till 2004 but acquired the status of major disease due to 190 altered weather conditions (Agarwal et al. 2013). 191

Causal organism: M. phaseolina (Tassi) Goid

The pathogen has a wide host range which includes major field and pulse crops 193 like common bean, soybean, mungbean, cotton, maize, sorghum, sesame, peanut, 194 cowpea and chickpea (Dhingra and Sinclair 1977; Diourte et al. 1995). 195

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Symptoms: The pathogen is predominant soilborne pathogen but also seed borne 196 in nature, capable of infecting the crop at any growth stage. The symptoms on coty-197 ledons appear as dark brown spots after emergence with brown to black margins, 198 and they shed at an early stage. After the emergence of unifoliate leaf, reddish 199 brown, circular to oblong lesions which after several days may turn dark brown to 200 black appear on the emerging hypocotyls of infected seedlings. Lesions appear on 201 roots, stems, pods and seeds. Lower leaves become chlorotic and later on wilt and 202 dry. As the disease advances, reddish brown discolouration of the vascular elements 203 of roots and lower stems followed by premature vellowing of plants is observed. 204 Blackening and cracking of roots is the most common symptom of this disease. 205 Diseased plants show poor seed-setting in pods with reduced seed size, which ulti-206 mately lead to heavy yield losses. 207

Disease cycle: Pin-sized microsclerotia are produced in abundance underneath the 208 epidermal tissue of the affected lower stems and roots after the death of plants. 209 These microsclerotia are capable of long survival up to 2–12 years in soil and initi-210 ate the disease by acting as primary inoculum (Meyer 1974). The pathogen has also 211 been associated with seed when detected using agar plate, blotter paper and modi-212 fied potato-sucrose-agar [PSA + Penta Chloro Nitro Benzene (PCNB)] methods by 213 Kushi and Khare (1978). The germination of microsclerotia is induced by the root 214 exudates of host plants present in the vicinity. Heavily infected plants die at early 215 stage due to the accumulation of fungal toxin, viz. botryodiplodin or phaseolinone 216 (Ramezani et al. 2007). Microsclerotia are released into the soil after the death of 217 the plant and the cycle continues. 218

219 6.3 Wilt Diseases of Major Pulse Crops

220 6.3.1 Fusarium Wilt of Chickpea

221 Butler (1918) first time reported the occurrence of gram wilt from India, and later in 1940, Padwick identified Fusarium orthoceras var. ciceri as the incitant of chickpea 222 wilt (Chand and Khirbat 2009). Due to the complex nature of gram wilt, Dastur 223 (1935) came to the conclusion that the drooping of plants was because of *Rhizoctonia* 224 wilt caused by Rhizoctonia bataticola. In 1940, Synder and Hansen renamed 225 Fusarium orthoceras var. ciceri as F. oxysporum f. sp. ciceri, and it is now world-226 wide accepted. All over the world, gram wilt alone is known to cause 10-50% yield 227 losses, while from India 10–15% losses were observed (Kheni et al. 2017). 228

Symptoms: Conducive conditions to *Fusarium* wilt pathogen initially results in drooping, yellowing and drying of the leaves followed by the wilting of entire plant (Lodhi et al. 2006; Kumari and Khanna 2018). Most of the times, disease appears in scattered patches of yellow colour, but under favourable conditions, wilting of entire field may occur. Sometimes, the infection starts after 25 days of sowing and that diseased condition is known as early wilt. In early wilt, the seedlings lose their turgor, further collapse and lie flat on the field. In most of the cases, the prominent disease 235 symptoms appear at 6-8 weeks after sowing when flowering starts and during pod 236 formation stage; that situation is known as late wilt (Jimenez-Díaz et al. 2015; 237 Arunodhayam et al. 2014). F. oxysporum usually results in discolouration, desiccation 238 and collapse/crumpling of entire plant following the drooping of leaves. The drooping 239 starts from the upper portion of the plant, and within no time, entire plant becomes 240 wilted. The cross-sectioned or vertically splitted roots/stems of infected plants shows 241 dark brown discolouration of xylem vessels. The pathogen results in the development 242 of histological distortions of vascular tissues along with the formation of occlusions 243 and gel in the xylem cells (Patil et al. 2017). These histological distortions lead to the 244 clogging of vascular tissues and retard the vascular flow of water, and ultimately, the 245 affected plant wilts. Actually, the toxins produced by F. oxysporum f. sp. ciceri are 246 responsible for wilting of plants (Chand and Khirbat 2009). 247

Causal organism:Fusarium oxysporumSchlecht and Emnd Snyd. & Hans. f. sp.248ciceri (Padwick)Snyd. & Hans249

F. oxysporum f. sp. ciceris produces three types of asexual spores, i.e. macro- and 250 microconidia and chlamydospores. Under in vitro conditions, white mycelial growth 251 with different pigments, viz. pink, pale yellow, light yellow, etc. has been observed 252 (Patra and Biswas 2016). The macroconidia are $25.00-55.00 \ \mu m \times 2.50-6.00 \ \mu m$ in 253 size, straight to slightly curved, thin walled usually with 3-5 septa, a foot-shaped 254 basal cell and a tapered and curved apical cell, while the microconidia are 255 $5.00-15.00 \ \mu\text{m} \times 2.00-5.00 \ \mu\text{m}$, ellipsoidal with single or no septum (Nath et al. 256 2017). The chlamydospores are thick walled, globose, formed singly and in pairs or 257 in chains on hyphae or alternatively by the modification of hyphal cells and are 258 important source of primary infection. In the absence of host plant, chlamydospores 259 of the fungus can survive up to 6 years in the soil. In lab, the chlamydospore forma-260 tion has been observed by several workers in 15-day-old culture and infected tis-261 sues. The teleomorph or sexual reproductive stage of F. oxysporum is unknown. The 262 variability among different isolates could be studied morphologically by using the 263 size and shape of asexual spores (Sinha et al. 2018). Its growth is primarily depen-264 dent on the type of soil, pH, moisture content and temperature. The optimum tem-265 perature for disease development is 25 °C, but fungus can grow within a range of 266 7-35 °C in the soils having pH 4-9.4. Many researchers reported different pH, i.e. 267 5.1-5.9 and 7.1-7.9 for mycelial growth and sporulation, respectively (Jendoubi 268 et al. 2017). 269

Disease cycle: The pathogen survives as chlamydospores or mycelium on seed, 270 soil (for up to 6 years) and crop residues buried in the soil (Lodhi et al. 2006). Initial 271 infection starts with the germination of chlamydospores/mycelia after getting stim-272 ulus (phytoalexins and flavonoids) from the roots of host/non-host plants. After ger-273 mination, the germtube directly or through wounds invades the roots and enters into 274 the epidermal cells of the plant. Following penetration, the fungus starts colonizing 275 the root cortex intracellularly and eventually grows to clog xylem vessels. Ultimately 276 F. oxysporum results in wilting of plants, and it is dependent on pathogen activities 277

284 6.3.2 Wilt of Pigeon Pea

Pigeon pea wilt is one of the devastating diseases causing even 100% yield losses 285 under favourable disease conditions (Pande et al. 2013). In addition to predisposing 286 factors, the losses are also dependent upon the stage of plant at which pathogen 287 establish itself. There are reports where 30%, 67% and even 100% losses are 288 recorded when infection occurs at preharvesting, maturity and pre-podding stage, 289 respectively. The disease was first time reported by Butler (1906) from Bihar, India, 290 and later in 1910, the causal organism was named as Fusarium udum. Thereafter, 291 Rai and Upadhyay (1982) reported its perfect stage *Gibberella indica* Rai and found 292 that perfect stage formed on exposed roots and collar region up to the height 293 of 35 cm. 294

Symptoms: The prominent symptoms are drooping of plants due to turgidity loss 295 and clogging of xylem vessels. Infected plants show partial wilting, mild interveinal 296 chlorosis, discolouration of xylem vessels and purplish bands on the stem which 297 extends in the upward direction. In addition to this, drying of plants from top 298 towards base following the yellowing and chlorosis is also common. Generally, the 299 wilting of plants is due to the presence of mycelial clumps in the xylem vessels 300 (Chaudhary 2016; Meena 2016). The cross-sections of the main root and base of the 301 stem show tissue discolouration, and in case of partial wilting, plant tissues are 302 discoloured and show wilting from one side, while the rest of the plant escapes. 303

304 Causal organism: Fusarium udum Butler

The pathogen is host specific and soilborne in nature and can survive on the crop 305 debris for 3 years. Like F. oxysporum, it also produces macro- and microconidia and 306 chlamydospores, but presence of prominent apical hook cell of macroconidia makes 307 it different from F. oxysporum. The fungus produces septate hyaline mycelium, 308 which grows inter- and intracellularly in xylem vessels to obstruct the water flow. 309 The microconidia formed by F. udum are unicellular, 1 or 2 septate and small in size 310 which varies from 5 to 15×2 to 4 μ m, while the macroconidia are long, and slightly 311 curved, 3–4 septate and $15-50 \times 3-5 \mu m$ in size. In addition to these, persisting/ 312 perennating structures, i.e. chlamydospores, are also formed which are formed at 313 either terminal ends or intercalary regions from any cell of hyphae or from macro-314 conidium. The optimum temperature for its growth and development ranges between 315 17 and 29 °C with soil pH of 4.6-9.0. 316

Disease cycle: Fusarium wilt of red gram is soilborne in nature, but there exist few 317 reports which confirm its survival in seed. Wilt pathogen has two phases in its life 318 cycle, i.e. pathogenic and saprophytic. In first phase pathogen remains attached to the 319 host plant, while in the later, it survives on dead host plants/parts as conidia or mainly 320 as the chlamydospores. Generally, infected seeds, soil and roots of previous year 321 crop serve as the main source of inoculum. Conidia/chlamydospores germinate and 322 penetrate the rootlets of pigeon pea. After that, the fungal mycelium grows in inter-323 and intracellular spaces and ultimately clogs the water-conducting vessels of host 324 plant. Following the clogging of xylem vessels, symptoms appear on the infected 325 plant, and on the infected portion, macro- and microconidia and chlamydospores are 326 formed which serve as a both primary and secondary inoculum. The fungus produces 327 different pectic enzymes (pectin methyl esterase, polygalacturonase and cellulase) 328 and toxins (Fusaric acid) which are involved in the pathogenesis. 329

6.3.3 Wilt of Field Pea

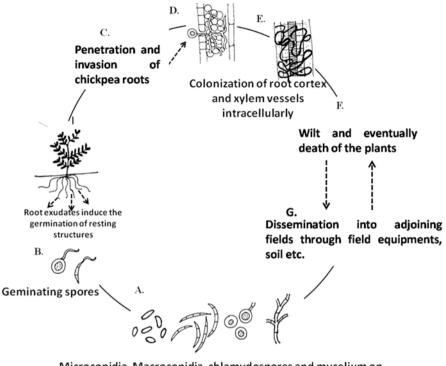
The production and productivity of pea is adversely affected by number of plant 331 pathogens, but *Fusarium* wilt and root rot diseases are of considerable importance 332 (Sharma 2011). The wilt disease for the first time was reported by Jones and Linford 333 (1925) from the USA. Linford (1928), in the initial years of discovery, named the 334 suspected wilt causing entity as *F. othoceras* App. and *Wr.* var. *pisi*. Thereafter in 335 (1935, the pathogen was renamed as race 1 of *F oxysporum* Schlecht f. sp. *pisi* (van 346 Hall) Snyd and Hans. 337

Symptoms: In pea generally two forms of wilt are known, i.e. wilt and near wilt. 338 Like other wilts, infected plants show drooping and change in colour of foliage from 339 green to pale yellow, and ultimately due to loss of turgidity, the entire plant topples 340 down. Tissue discolouration is also very common and could be observed after cross-341 sectioning of root or the lower stem. However, in case of near wilt (race 2), the 342 disease appears later at late blossom stage or at pre-pod or full pod development 343 stage. Near wilt is different from normal wilt, as it appears in scattered manner in 344 the field rather than being concentrated in specific areas as in case of race 345

Causal organism: Fusarium oxysporum f. sp. pisi (Linford) Snyder & Hansen

The formation of macro- and microconidia and chlamydospores is common like 347 other wilts. In this case the microconidia are oval to cylindrical and $5-12 \times 2.2-3.5 \,\mu\text{m}$ 348 in size, while macroconidia are 3–5 septate, fusoid, pointed at both of the ends and 349 27–46 × 3–4.5 μ m in size. Chlamydospores are also formed. 350

Disease cycle: Most of the *Fusarium* causing wilt diseases are soilborne in nature 351 and survive for a longer period in a soil by means of chlamydospores. *F. oxysporum* 352 f. sp. *pisi* is reported to remain viable for more than 10 years, and once the pea crop 353 is available in the field, the pathogen penetrates into the rootlets and ultimately 354



Microconidia, Macroconidia, chlamydospores and mycelium on seed, soil (for upto 6 years), crop residues buried in the soil

Fig. 6.2 Disease cycle of *Fusarium* wilt of chickpea caused by *Fusarium oxysporum* f. sp. ciceris

enters into the vascular system of the plant. The seed-borne nature of the pea wilt pathogen has also been reported. After entering into the roots or cortex, the pathogen established it in the xylem vessels, and further pathogen enters into the system of plants and results in seed-borne infections. The fungus continues to grow on the crop debris left after the death of plants and resulting in the establishment of soilborne inoculum. The fungus is monocyclic in nature (Fig. 6.2).

361 6.3.4 Wilt of Lentil

In India, *Fusarium* wilt is a major constraint behind low production of lentil, and from 50% to complete yield losses are reported under favourable conditions (Tiwari et al. 2018). The severity of lentil wilt is dependent on different factors including crop stages, predisposing factors and variety sown in the field. Chaudhary et al. (2009) reported the association of three fungal pathogens *Fusarium oxysporum* f. sp. *lentis*, *Sclerotium rolfsii* and *Rhizoctonia bataticola* with the wilt/root rot complex from Indian conditions and found the dominance of wilt pathogen. 368

Symptoms: Like other wilts, lentil wilt can be prevalent at seedling and adult plant 369 stages. The infection at seedling stage leads to the drooping and toppling of lentil 370 seedlings, and the condition is referred to as early wilting. At this stage the roots 371 appear healthy and no tissue discoloration is observed, while the infection at adult 372 stages of plant, i.e. flowering to pre-podding stage, results in either partial or com-373 plete wilting of infected plants. Flowering to pre-podding stage is considered as the 374 crucial stage, and infection at these stages leads to complete crop loss. The optimum 375 temperature for pathogen ranges between 22 and 25 °C (Tiwari et al. 2018). The 376 infection at later stages is characterized by dull green foliage, sudden drooping of 377 top leaves and branches followed by wilting of the entire plant. 378

Causal organism:Fusarium oxysporumSchlecht. emend Snyder & Hansen f.sp.379lentisVasudeva and Srinivasan380

Lentil wilt causing entity, i.e. Fusarium oxysporum f. sp. lenis, was first time 381 reported by Booth in 1971. Similar to other Fusarium spp., the fungal mycelium is 382 septate, and all the three asexual spores are formed in F. o f. sp. lentis. The microco-383 nidia are straight or curved and $5-11 \times 2.5-3.5$ µm in size while, macroconidia are 384 fusoid, 1–6 septate and $25-65 \times 3.5-4.5 \mu m$. The chlamydospores' formation under 385 in vitro conditions (on old cultures) has also been observed. The host range studies 386 by many workers on different crops concluded that the *Fusarium oxysporum* f. sp. 387 lentis produces disease only on lentil. 388

Disease cycle:The pathogen is soilborne and known to survive in soil for 3–4 years389without its host.The primary infection is through chlamydospores which remain390viable for the next season or for longer periods.Secondary spread is through conidia391by irrigation water, cultural operations and implements.392

6.4 Recent Advances in Detection and Diagnosis of Plant 393 Diseases 394

Fungi are the most diverse plant pathogens with a wide host range accounting for 395 70-80% of diseases infecting field crops, vegetables, fruit trees and ornamental plants 396 (Ray et al. 2016). Till date fungal disease management is still a challenge due to wrong 397 diagnosis of disease, resistance breakdown in host plants, development of fungicide 398 resistance in pathogens, residual effect of fungicide in environment, etc. Soil has a 399 complex environment, thus forge myriad of challenges in detection, isolation and 400 quantification of soilborne pathogens. Timely and accurate disease detection in case 401 of soilborne pathogens in the absence of their hosts has always remained a limitation 402 (DeShields et al. 2018). The soilborne pathogens infect the plants resulting in early 403 symptomless infection phase and express the symptoms when sufficient impact on 404 plant yield and productivity has already taken place. Accurate disease detection and 405

diagnosis to clearly define the plant disease and causal agent is the first and foremost 406 step in the integrated disease management. This allows preventing the introduction 407 and establishment of a soilborne pathogen to newer areas and losses due to planting of 408 healthy planting material in already infested soil, restricting the movement of infested 409 soil and water to a possible extent. The current detection methods include conven-410 tional and advanced molecular methods (Fang and Ramasamy 2015; Balodi et al. 411 2017). Conventional methods include identification based on diseased symptoms, iso-412 lation and culturing of the pathogen on artificial regular or selective media followed 413 by microscopic observations, growing of healthy plants on soil under test, etc. But all 414 these methods are time-consuming and laborious, require skilled laboratory staff and 415 often lead to incorrect diagnosis or wrong interpretation (Tsedaley 2015). Molecular-416 based approaches are competent strategies in case of early-stage detection and are 417 very helpful in undertaking prophylactic measures. 418

419 6.5 Molecular Approaches in Plant Disease Diagnosis

420 6.5.1 Polymerase Chain Reaction (PCR)

PCR method that involves in vitro replication of DNA was first invented by Kary 421 Mullis in 1984 for which in 1993 he received Nobel Prize in Chemistry. Since then, 422 PCR is extensively used in molecular detection as well in studying the phylogeny of 423 plant pathogens (Henson and French 1993; Caruso et al. 2003; Pandey et al. 2015; 424 Fang and Ramasamy 2015; Balodi et al. 2017). Different variants of PCR, viz. co-425 operational PCR, multiplex PCR, multiplex nested RT-PCR and real-time PCR, are 426 proficient in rapid and accurate plant disease diagnosis (Pandey et al. 2015; Yang and 427 Juzwik 2017). Most wilt diseases of pulse crops are caused by *Fusarium* spp. in which 428 conventional approaches of identification are time-consuming and require eminent 429 competence in Fusarium taxonomy and physiology (Leslie and Summerell 2006; 430 Thokala et al. 2015). Apart from detection, phenotypic and genotypic characterization 431 of pathogen variants prevalent in particular area is also of great significance in plant 432 disease management. In 2015, Chitten et al. identified Fusarium spp. associated with 433 root rot of field peas in North Dakota through PCR using translation elongation factor 434 alpha 1 (TEF-1 α) region. Jimenez-Gasco and Jimenez-Diaz (2003) developed PCR-435 based detection assay for Fusarium oxysporum f. sp. ciceris, chickpea wilt pathogen 436 to selectively differentiate pathogenic and nonpathogenic F. oxysporum isolates as 437 well as other species and formae speciales of *Fusarium* and *F. oxysporum*, respec-438 tively, and each of the F. oxysporum f. sp. ciceris pathogenic races 0, 1A, 5 and 6. 439 Apart from detection PCR has been employed for studying the diversity of pathogens 440 affecting pulse crops. Dubey et al. (2012) studied the diversity present in Rhizoctonia 441 solani infecting different pulse crops in different Indian agroecological regions at 442 molecular level using 23 inter-simple sequence repeats (ISSR) markers, 12 universal 443 rice primers (URPs) and 22 random amplified polymorphic DNA (RAPD). 444

6.5.2 Real-Time PCR (RT-PCR)

Among PCR techniques, real-time PCR (RT-PCR) has been proven as one of the 446 reliable, sensitive and easy to perform techniques for detection and quantification 447 of soilborne pathogens. This technique allows the real-time monitoring of PCR 448 reaction. RT-PCR-based quantification of soilborne pathogens can provide more 449 accurate and authentic estimation of inoculum load in soil unlike culturing meth-450 ods which are comparatively less reliable and inaccurate (Mirmajlessi et al. 2015). 451 One of the significant applications of RT-PCR in plant disease diagnostics is 452 simultaneous detection of more than one pathogen when lots of samples are 453 involved (Cooke et al. 2007). Vandemark and Grunwald (2005) applied RT-PCR 454 to establish the relationship between disease severity of Pea root rot and 455 Aphanomyces euteiches DNA in soil. Gangneux et al. (2014) developed a rapid 456 and sensitive assay for reliable detection and quantification of Aphanomyces in 457 soil. RT-qPCR assay was developed for rapid detection and quantification of 458 Fusarium wilt pathogen of Phaseolus vulgaris, F. oxysporum f. sp. phaseoli 459 (Sousa et al. 2014). 460

6.5.3 Loop-Mediated Isothermal Amplification Assay

Loop-mediated isothermal amplification (LAMP) is an one step amplification assay 462 with great sensitivity and specificity which takes less than an hour to make multiple 463 copies of DNA/RNA (up to 10^9) from very few copies of template under isothermal 464 conditions (Notomi et al. 2000). Four different primers, viz. Forward Inner Primer 465 (FIP), Forward Outer Primer (FOP), Backward Inner Primer (BIP) and Backward 466 Outer Primer (BOP), targeting six distinct region of target gene are used in a LAMP 467 reaction. The overview of different stages of LAMP is available at http://loopamp. 468 eiken.co.jp/e/lamp/anim.html. The technique has been used as a rapid and accurate 469 method for plant disease detection and diagnosis (Tomlinson and Boonham 2008; 470 Khan et al. 2018; Huang et al. 2017). Ghosh et al. (2017) used this novel technique 471 to develop a rapid and sensitive diagnosis for dry root rot of chickpea caused by 472 R. bataticola (Taub.) Butler targeted the 5.8S rDNA region of fungus. Rapid diag-473 nosis for Ascochyta blight of chickpea pathogen, Ascochyta rabiei L. (A. rabiei), 474 was also developed through LAMP method with 6.01×10^{-6} ng/µl detection limit 475 based on internal transcribed spacer (ITS) region (Chen et al. 2016). Rapid detec-476 tion of F. oxysporum f. sp. ciceris (Foc), chickpea wilt pathogen through LAMP 477 combined with hydroxynaphthol blue (HNB) was performed which developed sky 478 blue colour with Foc DNA but not with negative control (without DNA) or with 479 other fungal DNA (F. acuminatum, F. udum, F. solani, R. bataticola, Alternaria 480 alternata and Phytophthora cajani) (Ghosh et al. 2015). 481

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482 6.6 Management of Root Rots and Wilt Diseases of Pulse 483 Crops

For disease development by any biotic factor, successful interaction between sus-484 ceptible host, virulent pathogen and favourable environment is required which 485 remains for a sufficient period of time. The interference and manipulation of any of 486 these components during disease development before the occurrence of sufficient 487 loss to reduce the disease level below economic injury level with minimum harm to 488 the environment is the basic principle of plant disease management (Katan 2017). 489 This cannot be achieved by just adopting a single tactics but to amalgamate all the 490 approaches, viz. cultural and mechanical methods, chemical methods, biological 491 control, using host plant resistance, etc. in the framework of "integrated plant dis-492 ease management (IDM)". IDM can be defined as a "decision-based process involv-493 ing coordinated use of multiple tactics for optimizing the control of all classes of 494 pests (insects, pathogens, weeds, vertebrates) in an ecologically and economically 495 sound manner" (Prokopy 2003). As soilborne pathogens produce their resting struc-496 tures in soil, therefore they are influenced by biotic and abiotic factors of soil which 497 changes with agricultural practices that are applied to the soil. In case of soilborne 498 pathogens, it is not always necessary that soil inoculum is the only and major source 499 of inoculum and thus makes the management even more tedious. There are four 500 foremost steps in soilborne disease management, viz., prevention of introduction 501 and establishment of pathogen to newer cultivating areas, reduction of pathogen 502 population below economic injury level, improvement of natural suppressiveness of 503 soil and least manipulation of natural biological and physical properties of the soil 504 (Chellemi et al. 2016). 505

506 6.6.1 Cultural and Mechanical Methods

By adopting good cultural practices, one can maintain an environment favourable 507 for crop but not for the disease development. The present cultural practices or tradi-508 tions which are followed today to control soilborne pathogens are the result of 509 numerous observations and long-term experience generated through trials and 510 errors though due to the availability of effective chemical, the interest in cultural 511 practices is lost among the growers (Katan 2010). Howbeit, with increasing concern 512 of deteriorating environment and popularization of IDM concept, the interest in 513 cultural practices has been again emerged. Application of diverse cultural practices, 514 viz. intercropping/mixed cropping, crop rotation, field sanitation, adjusting sowing 515 times, etc., are advocated as effective tools for soilborne disease management 516 (Juroszek and von Tiedemann 2011; Pandey et al. 2018). However the survival 517 period of wilt and root rot pathogens in soil is very long, therefore at least 5 or 518 7 years of rotations are required to prevent the building up of pathogen population 519 to a level causing damage above economic injury level. Intercropping of pigeon pea 520

with sorghum at 1:1 was found effective in managing Fusarium wilt when inte-521 grated with other management approaches (Prasad et al. 2012). The sorghum's root 522 exudates which include hydrocyanic acid and tannins are reported to affect the 523 mycelial growth and conidial germination of *Fusarium* spp. in soil (Rangaswami 524 and Balasubramanian 1963; Odunfa 1979). High temperature during maturity of 525 chickpea can be prevented by timely or early sowing of chickpea; moreover, when 526 supplemented with timely irrigation, DRR incidence is further reduced (Sharma 527 et al. 2015). Yaqu and Shahzad (2009) has observed less disease incidence due to the 528 use of plastic mulching which led to sclerotial mortality of *M. phaseolina*, the dry 529 root rot pathogen. In lentil, sowing in the first week of December at 2 cm depth 530 results in least wilt severity and highest grain yield (Sallam and Monaim 2012). 531

6.6.2 Chemical Control

The soilborne nature of both wilt and root rot causing pathogens, development of 533 resistance to chemicals in pathogenic isolates over the time and zero possibility of 534 treating soil at a large scale make chemical management less worthy than cultural 535 practices. But still there are reports where chemicals are in use. Generally, foliar 536 sprays are found to be less effective in management of soilborne pathogens as com-537 pared to seed and soil treatment. The management of soilborne pathogens starts 538 with the chemical treatment of soil and commonly used chemicals are metalaxyl, 539 diazoben, pentachloronitrobenzene, captan and chloroneb (Veena et al. 2014). The 540 foliar spray of Fosetyl-aluminium has been reported to control soilborne pathogens. 541 After soil treatment, the seed treatment with various fungicides alone or in combi-542 nation is also in use; seed treatment with tebuconazole at 1 ml/kg (for gram wilt), 543 difenoconazole, carbendazim, thiram, mixture of benomyl and thiram and a 544 combination of carbendazim + thiophanate (0.15 + 0.10%), carbendazim 545 12% + mancozeb 63% WP has been recommended by various workers for the con-546 trol of root rots and wilts (Sinha et al. 2018; Golakiya et al. 2018; Durga et al. 2014). 547 Seed treatment with thiram + PCNB or thiram + carboxin was reported to keep 548 check over lentil wilt, while for the management of pea wilt tebuconazole/metalaxyl 549 M + difenoconazole/imidacloprid + tebuconazole were recommended. For the con-550 trol of DRR of chickpea, seed treatment with carbendazim and thiophanate methyl 551 was found to be effective (Sharma and Kumara 2017). Overall, the management of 552 both wilts and root rot pathogens are the same. 553

6.6.3 Biological Control

Keeping in view the environmental losses, reduction in beneficial soil microflora and microfauna, residual effects due to excessive use of pesticides and development of resistance in pathogens, the biological control offers an attractive and ecofriendly 557

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alternative approach for plant disease management (Chandrashekara et al. 2012; 558 Singh 2014). Biological control has been defined as "the action of parasites, preda-559 tors, or pathogens in maintaining another organism's population density at a lower 560 average than would occur in their absence" (DeBach 1964). Several potential bio-561 control agents (BCAs) have been identified for the management of soilborne dis-562 eases. The potential BCAs identified are Gliocladium, Trichoderma, nonpathogenic 563 Fusarium, Bacillus, fluorescent Pseudomonas, Streptomyces, etc. (Harman and 564 Kubhicek 1998; Benhamou et al. 2012; Bochow et al. 1997; Weller 1988). King and 565 Parke (1993) applied *Pseudomonas cepacia* strain AMMD as seed treatment and 566 achieved control in case of four pea cultivars against *Pythium* sp. causing damping 567 off and Aphanomyces root rot. Root rot disease in chickpea due to Meloidogyne 568 incognita and Macrophomina phaseolina was least when all the three phosphate 569 solubilizing bacteria, viz. Pseudomonas aeruginosa (isolate Pa28), Aspergillus 570 awamori, and Glomus intraradices were inoculated together greatest increase in the 571 plant growth (Siddiqui and Akhtar 2007). Shahid and Khan (2016) evaluated the 572 biocontrol efficiency of different fungi and bacteria, viz. Trichoderma harzianum, 573 T. reesei, Aspergillus niger and Bacillus subtilis against M. phaseolina, DRR of 574 mungbean pathogen, and found T. harzianum and B. subtilis as best BCA in manag-575 ing the disease as well as improving the plant growth and yield of mungbean. 576 Actinobacteria Streptomyces has also been identified as potential BCA against 577 Aphanomyces euteiches, the causal agent of pea root rot based on in vitro antimicro-578 bial activity assay followed by identification based on 16S rDNA analysis and mor-579 phological and chemical characteristics (Oubaha et al. 2018). For the management 580 of wilt and root rot diseases of pulse crops, seed treatment with T. viride at 4 g/kg or 581 P. fluorescens at 10 g/kg of seed or spot drenching with P. fluorescens / T. viride 582 2.5 kg/ha with 50 kg Farm Yard Manure (FYM) has been recommended. 583

584 6.6.4 Host Plant Resistance

Host resistance offers the most economic and environment-friendly method of plant 585 disease management. In case of soilborne diseases, use of resistant varieties is the 586 most practical approach for their management. Serious efforts are being taken in the 587 direction of finding new sources of resistance in wild relatives of cultivated pulse 588 crops, mapping of resistance genes/quantitative trait loci (QTL) and identifying 589 genetic markers linked with identified resistant (R)genes/OTLs for application of 590 marker-assisted selection (MAS) in resistance breeding programmes. The applica-591 tion of MAS by identifying molecular markers linked to R genes against different 592 pathogen races can accelerate the resistance breeding programme (Winter and Kahl 593 1995). For resistance breeding programmes, a clear picture of the existing patho-594 genic variability and races present in the target area is the prerequisite. A lot of con-595 ventional as well as molecular breeding programmes have been conducted worldwide 596 in developing resistant chickpea cultivars. The existence of race 1, 2, 3 and 4 was 597 confirmed by Haware and Nene (1982) in India using ten chickpea differential lines. 598

Jimenez-Diaz et al. (1993) studied the pathogenic variability of 107 F. oxysporum f. 599 sp. ciceris (Foc) isolates from Algeria, California, Morocco, Tunisia, Spain and Italy 600 and screened 2702 kabuli lines procured from ICARDA for resistance against 601 Fusarium wilt. Different workers have mapped resistant genes for Foc race 1, 2, 4 602 and 5 on the same linkage group (Simon and Muehlbauer 1996; Ratnaparkhe et al. 603 1998). Benko-Iseppon et al. (2003) identified molecular markers closely linked to 604 Fusarium R genes in chickpea through bulked segregant analysis (BSA) which 605 showed significant alignments to pathogenesis-related (PR) genes located on 1 and 5 606 chromosomes of Arabidopsis. Iftikhar and Ilyas (2000) found only ICCV 97112 was 607 found resistant out of 108 chickpea germplasms screened for resistance against 608 DRR. Gangwar et al. (2002), Prajapati et al. (2003), Pande et al. (2006) and Khan 609 et al. (2013) reported few resistance sources in chickpea against DRR. Marker-610 assisted backcrossing programmes were undertaken to introgress resistance against 611 Ascochyta blight and Fusarium wilt in chickpea cultivar, C 214 targeting two QTL 612 regions, viz. ABOTL-I and ABOTL-II and foc1 locus. Foreground selection for foc1 613 locus in case of Fusarium wilt Race 1 was conducted using six markers, viz. TA194, 614 TR19, GA16, TAA60, TS82 and TA110, while in case of Ascochyta blight, eight 615 markers, viz. GAA47, TA2, TA194, TR58, TS82, TA130, SCY17 and GA16, linked 616 to ABQTL-I and ABQTL-II were used (Varshney et al. 2014). 617

In mungbean breeding programmes against disease resistance, MAS has not 618 been much exploited; however molecular markers against major resistant (R) genes 619 or QTLs against fungal diseases like powdery mildew and Cercospora leaf spot 620 have been identified, but no associated molecular markers or R gene or OTLs were 621 reported for DRR of mungbean (Pandey et al. 2018). In case of Fusarium wilt of 622 pea, four races, viz. 1, 2, 5 and 6, of F. oxysporum f.sp. pisi (Fop) were recognized 623 by Kraft and Pfleger (2001). Fusarium wilt resistance against majority of Fop races 624 is governed by single gene (Coyne et al. 2000; Grajal-Martin and Muehlbauer 2002; 625 McClendon et al. 2002; Kwon et al. 2013). However, resistance against Fop race 2 626 is quantitative (Bani et al. 2011; McPhee et al. 2012). Single gene Fw was located 627 on linkage group III which confers resistance against Fop race 1 in pea (Kwon et al. 628 2013). Kwon et al. (2013) identified three tightly linked markers to Fw locus, viz. 629 Fw_Trap_480, Fw_Trap_340 and Fw_Trap_220, which were only 1.2 cM away 630 from the locus. These markers were found to be suitable for their use in MAS for 631 Fop race 1 breeding programmes. A genetic linkage map was constructed for 632 Fusarium wilt resistance and localized on linkage group 6 in lentil based on micro-633 satellite markers mapping identified from genomic library of lentil (Lens culinaris 634 Medis.) (Hamwieh et al. 2005). 635

6.7 Conclusion

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The Indian agriculture is still struggling due to incidence of pests and diseases 637 resulting in huge crop losses. Though fungal pathogens are known to incite plant 638 diseases since 1807, still phytopathogenic fungi take a heavy toll on crop production 639

worldwide. Out of different classes of fungal pathogens, soilborne pathogens man-640 age to remain as the most notorious one due to various factors like challenges that 641 are there in timely detection and therefore management, long survival period in soil, 642 complex nature of diseases caused by them due to involvement of multiple microor-643 ganisms and nematodes as well. Root rots and wilts are the major limiting factors of 644 pulse crop production in India. Despite the considerable application of chemicals 645 and other management approaches that include cultural, biological and exploitation 646 of host resistance, these diseases continue to be a constraint in pulse crop produc-647 tion. The nature of these pathogens, various climatic factors affecting the incidence 648 and disease development caused by these pathogens are already known; however 649 extensive studies are required to elucidate the infection process and determine the 650 pathogenic and genetic variation, spatial and temporal distribution of causal patho-651 gens and resistance mechanism in host plants. Moreover, the application of advanced 652 molecular tools in timely and precise detection and diagnosis of root rots and wilt 653 pathogens is very limited. For sustainable management of root rot and wilt in pulse 654 crops, reliable marker-assisted resistance breeding programmes suitable for broader 655 geographical areas using tightly R-gene linked markers are required. Different 656 omics approaches must be employed to identify the molecular mechanism of resis-657 tance and key molecular factors playing role in governing resistance against root 658 rots and wilts in already identified resistant lines. 659

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Chapter 7 Diversity of *Phytophthora* Stem Blight of Pigeonpea and Its Sustainable Management

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7.1 Introduction

Pigeonpea (Cajanus cajan (L.) Millisp.) is called by different vernacular names 8 (arhar, tur, redgram, togari, kandalu, etc.), and it is an economically important grain 9 legume of the small and marginal farmers in India. Pigeonpea is one of the major 10 and inseparable dietary protein sources to the large mass of the Indian population 11 (Varshney et al. 2010). Pigeonpea is cultivated as a sole crop and intercrop with 12 rainfed cereals, millets, oils seeds, and other pulses; thereby, it enhances the system 13 productivity and net income to the small and marginal farmers. The differences in 14 the maturity duration of pigeonpea allow it to grow in diversified cropping systems 15 and patterns in varied agro-eco regions of the country. 16

This has been a matter of concern since the per capita protein availability in India 17 is declining steadily from 27.30 kg/year in 1950 to 10 kg/year in 2009 (Saxena et al. 18 2014). At present, the national harvest accounts for about 4.25 million tonnes of 19 pigeonpea grains (http://agricoop.gov.in). However, this quantity is not sufficient to 20 meet the domestic needs; about 0.41 million tonnes of pigeonpea is imported annu-21 ally. The prevailing situation is not likely to improve in the near future by considering 22 the 1.1% annual growth in population (World Bank 2017), plateau of pulse produc-23 tion, inherent low genetic variability for high yield and its attributing traits among 24 the cultivars used in breeding programme and susceptibility of pigeonpea to major 25

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B. P. Singh et al. (eds.), *Management of Fungal Pathogens in Pulses*, Fungal Biology, https://doi.org/10.1007/978-3-030-35947-8_7

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diseases and insect pests (Ariyanayagam et al. 1995; Yang et al. 2006; Mallikarjuna
et al. 2007; Naik Satheesh et al. 2012; Bohra et al. 2014a; Mishra et al. 2016). This
opens the new avenue to use the elite genotypes and wild species into the breeding
program to create unexplored genetic variability in pigeonpea through pre-breeding
(Sharma and Upadhyaya 2016; Saxena and Kumar 2003; Saxena et al. 2010).

In India, the majority of the pigeonpea production comes from states like Madhya 31 Pradesh, Maharashtra, Gujarat, Karnataka, Andhra Pradesh, Telangana, and Uttar 32 Pradesh. In these states, medium- and long-duration pigeonpea cultivars are grown 33 as intercrop, and it is unlikely that the cultivated pigeonpea area will increase by any 34 significant extent to meet the entire need of the country. Hence, new production 35 niches with early-maturing cultivars were explored. As a follow-up pigeonpea, 36 wheat rotation was successfully introduced in the states of Punjab, Harvana, and 37 Western Uttar Pradesh. However, the new varieties which are resistant to 38 *Phytophthora* stem blight disease and photothermal insensitive, a major production 39 constraint, are being marketed through local agro-dealers (Varshney et al. 2014). 40

The diverse growing conditions expose the pigeonpea to different biotic and abi-41 otic stresses during its life cycle. Pigeonpea get infected by different diseases and 42 insect pests; however, few of them only cause considerable economic losses (Nene 43 et al. 1996; Dhar et al. 2004). After wilt (C.O: Fusarium udum) and sterility mosaic 44 disease (SMD) (C.O: Pigeonpea Sterility Mosaic Virus), Phytophthora stem blight 45 (PSB) caused by Phytophthora drechsleri Tucker f. sp. cajani is the third most 46 important disease of pigeonpea in India (Kannaiyan et al. 1984; Mishra et al. 2016) 47 causing complete crop loss upon its infection. PSB has also been reported as the 48 most important production constraint in northeastern states of India (Mishra and 49 Shukla 1987; Chauhan et al. 2002). 50

7.2 Economic Importance of *Phytophthora drechsleri* Tucker f. sp. *cajani*

The fungus, *Phytophthora drechsleri*, attacks to young (1–7-week-old) plants of pigeonpea, which in turn kills the young plants at the early stage of crop stand to leave large gaps in plant stands (Fig. 7.1). Yield losses are generally higher in early maturing pigeonpea in comparison to medium- and long-duration varieties, because of favorable disease triangle components in early pigeonpea.

58 7.3 Disease Epidemiology

The *Phytophthora drechsleri* Tucker f. sp. *cajani* survives in soil and infected plant parts as chlamydospores, oospores, and dormant mycelium. Chlamydospore is thick-walled long-term survival spores, as they are produced through asexual means of reproduction. Whereas oospores are sexual spores, these are produced from



Fig. 7.1 *Phytophthora* stem blight infected field of pigeonpea at the early stage (a) and later stage (b) leaving the large gap in the plant stand

fertilization of the oogonium by an antheridium. Mycelium of *Phytophthora* is coenocytic, aseptate, hyaline, and profusely branching mainly of monopodial branches. The septa are formed at the time of reproduction.

For a successful disease triangle, moist cloudy conditions with drizzling rain are 66 prerequisite, and temperatures between 25 and 28 °C favor rapid infections in young 67 seedlings. The infection requires continuous wetness of plants for about 8 hours to 68 start. As plants grow older, they gradually develop tolerance/resistance to the dis-69 ease incidence, and they are generally not infected after they are 60 days old. The 70 PSB infection occurs more in organic matter-enriched clay soil in comparison to 71 clayey soil with little organic matter. The disease symptom appears first in low-72 lying areas of the field where water stagnates. High-density planting, coupled with 73 low availability of resistant varieties, leads to enhanced PSB buildup in early matu-74 rating pigeonpea. Warm and humid conditions followed by start-up of an infection 75 of PSB would result in rapid disease development and eventually lead to plant death. 76 Further, speedy wind and rain splashes help to disseminate zoospores. Phytophthora 77 drechsleri Tucker f. sp. cajani lives on different wild hosts of pigeonpea, for 78 instance, Cajanus scarabaeoides var. scarabaeoides, a wild relative of pigeonpea, 79 act as a collateral host for *drechsleri* Tucker f. sp. *cajani*. 80

7.4 Disease Symptoms and Progress of Disease on Pigeonpea 81

Phytophthora drechsleri present symptomless in the rhizosphere of pigeonpea, and 82 the infection was only evident when the favorable disease triangle exists (Stanier et al. 83 1971; Lewis 1973). The symptoms of the *Phytophthora* blight disease on pigeonpea 84 have been described in detail by Pal et al. (1970) as stem rot, by Williams et al. (1975) 85 as stem blight, and by Kaiser and Melendez (1978) as a stem canker. The most 86 commonly preferred name for *Phytophthora* infection is the term blight to describe 87 the disease; because all aboveground parts of the pigeonpea plant are affected, further 88 the roots of diseased plants show no symptoms until the plant dies. 89

Sarkar (1988) reported that the development of PSB is positively correlated with 90 its soil inoculum potential. Bisht (1985) and Sharma et al. (2015) found that zoo-91 spores are the primary source of inoculums. Speedy wind helps in spore dispersal 92 over short distances during rain splash. Williams et al. (1975) found high disease 93 incidence due to poor soil surface drainage; in contrary Singh and Chauhan (1985) 94 reported PSB developing to an epidemic level in well-drained fields. Therefore, 95 drainage alone is not the deciding factor for PSB epidemics. Further, Sharma et al. 96 (2006) reported an outbreak of PSB in well-drained, partially drained, and tempo-97 rarily waterlogged fields irrespective of cropping systems, soil types, and crop cul-98 tivars in the Deccan Plateau of India. 99

Phytophthora stem blight resembles damping off disease at the early stage of 100 infection that causes young seedlings to die after infection. Further infected plants 101 have water-soaked lesions on their leaves and brown to black spots, slightly sunken 102 lesions on their stems and petioles. Infected plant parts lose turgidity and become 103 desiccated. Lesions strap the affected main stem or a branch which leads to break at 104 that infected point, causing the foliage above the lesion to dry up and lodging. 105 Pigeonpea plants that are infected by blight, but not killed, often produce large galls 106 on their stems especially at the edges of the lesions (Fig. 7.2). 107

Singh and Chauhan (1985) reported more rapid development of PSB at night in 108 the field due to favorable disease development conditions; this hypothesis was con-109 firmed under artificial darkness conditions in the greenhouse. Reddy et al. (1991a, 110 b) confirmed the PSB infection usually occurs when there is a decrease in day tem-111 peratures of the previous week, and the difference between the maximum and mini-112 mum temperatures are the least. Studies on relationships between PSB incidence 113 and soil nutrition indicated that in the absence of potassium (K) and high doses of 114 nitrogen (N), PSB incidence increased (Pal and Grewal 1975). Nevertheless, the 115 addition of K decreased disease incidence regardless of the presence of N or phos-116 phorus (P) in the soil (Fig. 7.3). 117



Fig. 7.2 Phytophthora infected pigeonpea plants at the early stage (a) and later stage with large galls on the stems (b)

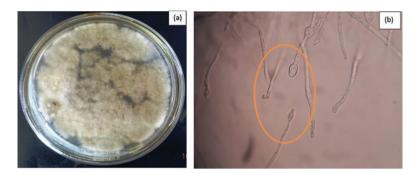


Fig. 7.3 (a) Cottony mycelial growth of PSB on V8 juice agar. (b) The hypal structure and $40 \times$ magnified papiliate hybphae of PSB

7.5 Morphological Features of Phytophthora

The cell wall of *Phytophthora* is made up of cellulose. *Phytophthora drechsleri* 119 Tucker f. sp. cajani resembles true fungi because they grow using fine filaments 120 called hyphae and produce spores. Phytophthora hyphae lack cross wall septa and 121 diploid phase. The Phytophthora drechsleri Tucker f. sp. cajani has terminal papil-122 late hyphae which in turn produces the spores. The sizes of sporangia of *Phytophthora* 123 *drechsleri* var. *cajani* ranging from 42 to 83×29 to 48 µm (average 61.8×37.3 µm) 124 and the sporangial stalks is either narrowly tapered or widened somewhat at the base 125 of the sporangium (Fig. 7.4b). 126

Phytophthora produces several types of substructure that are specialized for survival during the adverse condition of their life cycle. Chlamydospores and oospores127vival during the adverse condition of their life cycle. Chlamydospores and oospores128are prominent spores of Phytophthora produced during the adverse conditions of129their growth and development. Chlamydospores are thick-walled long-term survival130spores produced by asexual means of reproduction, while oospores are sexual131spores, which are produced from fertilization of the oogonium and antheridium.132

7.6 Disease Management Techniques

In any disease management, host plant resistance is the primary step for exploring 134 available germplasm stocks and breeding lines to identify donors. Different tech-135 niques for PSB resistance screening under field and greenhouse conditions have 136 been reported by various researchers. Pal et al. (1970) used a "leaf scar" method 137 to inoculate 30- to 60-day-old seedlings which are grown in pots under greenhouse 138 conditions. This method consisted of inoculating plants at the point of attachment 139 of leaf after its removal with mycelial mats of the fungus multiplied on potato dex-140 trose agar. Kannaiyan et al. (1981) standardized the pot-culture drench inoculation 141 and foliage inoculation techniques. In drench inoculation, 5- to 10-day-old seed-142 lings raised in pots filled with sterilized field soil are drench-inoculated with the 143

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Fig. 7.4 (a) Ridge planting of pigeonpea at early seedling stage. (b) Established pigeonpea crop on ridge planting method

macerated mycelial suspension of the fungus multiplied on V-8 juice medium (one 144 mycelial mat in 200 ml of water). Inoculum (100 ml) was poured around seedlings. 145 Pots were liberally watered three times a day to assure adequate development of 146 the disease. In this technique, the disease developed after 7–10 days of inoculation. 147 In the foliage inoculation technique, the inoculum is sprayed on 15- to 30-day-old 148 plants grown in a pot, the plants covered with polythene bags for 48 h, kept on glass-149 house benches, and later sprayed with water for 10 days. Typical blight symptoms 150 appeared within 10 days after the inoculations. 151

The sick field screening of pigeonpea genotypes for Phytophthora blight resis-152 tance was standardized at ICRISAT and ICAR-IIPR, Kanpur, including planting of 153 test entries with 30 cm row spacing and interplanting a susceptible cultivar (e.g., 154 ICP 2376, UPAS 120, ICP 1134, and ICP 7119) to serve as an indicator line after 155 every 2-4 rows. The sick field was prepared by incorporating diseased debris of 156 susceptible cultivars; further, the inoculum load in the sick field is maintained 157 through periodical soil sample analysis of PSB sick field. Additional sickness in the 158 field is created by incorporating infected plant debris. 159

Agronomic intervention plays an important part in the management of PSB dis-160 ease. The desiccation of pathogenic spore and dormant mycelium through summer 161 solarization or summer ploughing of field is being done to avoid the inoculum load. 162 Practicing the ridge planting method is highly advantageous to drain excess rainwa-163 ter since pigeonpea requires well-aerated soil for its growth and development. After 164 the onset of monsoon, timely sowing is highly advisable for establishing early 165 growth and in turn keeping away the disease incidence, because older plants are 166 more resistant to *Phytophthora* blight disease due to systemic acquired resistance. 167 Select fields with no previous record of PSB, and avoid sowing pigeonpea in fields 168 with low-lying patches that are prone to temporary waterlogging. Use wide inter-169 row spacing for good aeration and plant growth. 170

Although several fungicides have proved effective in the control of PSB, how-171 ever, systematic studies on the control of soilborne diseases like PSB using fungi-172 cides are limited. In a pot experiment, Pal and Grewal (1983) reported Brestan-60 173 effective in controlling PSB in 1-month-old plants when applied before inoculating 174 with PDC. Significant control of blight (>90%) was achieved with metalaxyl (1.75 g 175 a.i kg¹ seed) in a greenhouse experiment (Agarwal 1987; Bisht and Nene 1988). 176 However, Chaube et al. (1987) reported the poor efficacy of metalaxyl applied as a 177 seed dressing in protecting older pigeonpea plants against PSB. At the later stage of 178 PSB, the infection plant develops galls and makes them susceptible to lodging dur-179 ing intercultural operation and speedy wind. Sheila and Nene (1987) reported 180 reduced PSB incidence with the spray or soil drench with two phytoalexins like 181 Phytoalexin-84 and Induce. Park et al. (2007) claim that the direct application of 182 slow-releasing phosphorous acid formulations (curdlan or pestan) using a carrier 183 coated with polysaccharides resulted in an excellent control of PSB disease of pep-184 per. They further suggested that the application of formulation product once or 185 twice during crop season can control Phytophthora diseases on various crops. 186 However, there is no evidence in pigeonpea to say this product can be used for the 187 management of PSB in pigeonpea. 188

Practicing of the integrated disease management (IDM) technology is essential for 189 economical and sustainable means to control PSB. Moderate levels of host plant resis-190 tance-bred varieties can be combined with other cultural practices, and application of 191 minimal dosage of fungicide for control of PSB would save large input cost to farm-192 ers. The recommended IDM practices include (a) use of pathogen-free seed, (b) seed 193 treatment with fungicide, (c) crop rotation, (d) raised bed planting, (e) adequate field 194 drainage, and (f) use of disease resistant variety, and strategic application of fungi-195 cides will help in the management of disease in a sustainable manner. 196

7.7 Future Prospective and Conclusion

Phytophthora blight (*Phytophthora drechsleri* f. sp. *cajani*) is one of the major 198 yield limiting factors of short-duration varieties of pigeonpea (*Cajanus cajan*). 199 For eco-friendly and sustainable management of the disease, antagonists 200

(Pseudomonas fluorescens, Bacillus subtilis, Trichoderma viride, and T. hamatum) 201 were evaluated widely and used as bioagents and can be integrated with fungicides 202 for effective management of PSB disease. Commercially available metalaxyl for-203 mulation - Ridomil MZ - is also at a par with apron in respect to efficacy against 204 P. drechsleri f. sp. cajani, and they could be integrated with P. fluorescens and 205 T. viride for better and eco-friendly management of Phytophthora blight of pigeon-206 pea. Ridomil MZ has an additional advantage that it possesses different modes of 207 action and there is a lower chance of cross-resistance with metalaxyl-resistant popu-208 lations. Mancozeb in combination with metalaxyl was found to be highly effective at 209 reducing disease. However, the chemical method of controlling PSB is not economi-210 cal and eco-friendly. Therefore more focus is needed for the development of resis-211 tant varieties for sustainable management and for higher productivity per unit area. 212

Acknowledgments We are thankful to the Director, ICAR-IIPR, Kanpur, for extending
 facilities.

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Chapter 8 Foliar Fungal Diseases in Pulses: Review and Management

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8.1 Introduction

Pulses play a major role in the nutritional security of people having a cereal-based 7 diet. The United Nations Food and Agriculture Organization (FAO) recognizes 11 8 types of pulses in India as chickpea (brown and green), lentil (masoor), faba bean 9 (broad beans), field pea (matar), cowpeas, black gram, black-eyed bean, pigeonpea 10 (arhar), and red kidney beans (rajma) (Busby et al. 2016). Besides their value as diet 11 and having nitrogen fixation ability, pulses also play an important role in flourishing 12 intensive agriculture by improving the physicochemical and biological properties of 13 soil. Aerial fungi attack causes diseases like gray and chocolate spots, Ascochyta 14 blight, anthracnose, leaf rot, powdery mildew, leaf yellowing, stem canker, and 15 downy mildew. These diseases are caused by a fungus that can be necrotrophic or 16 biotrophic, e.g., Botrytis cinerea, B. fabae, Ascochyta rabiei, Colletotrichum, 17 Puccinia triticina, Erysiphe polygon, and Perenospora (Trivedi et al. 2017). 18 However, some of them affect larger areas among several countries where the culti-19 vation of legumes occurs and cause degradation in quantity and quality. 20

Development of disease by fungi in host plants is a stepwise phenomenon, starting from the contamination phase, following contact between the host plant and spores of the fungus. Depending on adequate receptivity and compatibility, spore germination occurs and forms appressoria that allow the fungus to penetrate directly the host plant or by cuticle, stomata, or tissues wounded. Infection follows penetration, where the fungus settles and invades the host tissue, enhancing its 26

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B. P. Singh et al. (eds.), *Management of Fungal Pathogens in Pulses*, Fungal Biology, https://doi.org/10.1007/978-3-030-35947-8_8

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development either on a dead (necrotrophic fungus) or on living tissue (biotrophic 27 fungus), resulting in the development of symptom (Sinha et al. 2018). The fungi 28 then develop specialized structures to carry out the production of secondary inocu-29 lum by sporulation that contributes to starting another infection cycle. Measures 30 for controlling these diseases including identification of resistant germplasm, 31 choosing varieties resistant to fungus by screening and experimentation, cultural 32 management, chemicals, genetic resistance, or combination of such approaches are 33 required, and attention has been given in this direction by the researchers (Pautasso 34 et al. 2012). 35

36 8.2 Fungi Affecting Foliar Parts

Pulses are consumed as a chief source of plant protein. Consisting of amino acids, 37 they have medicinal properties as well. Pulse crops are cultivated during rabi, zaid, 38 and kharif seasons of the agricultural year. Rabi crops require mild cold climate 39 during sowing to pod development and warm climate during maturity/harvesting, 40 e.g., chickpea, lobia, moong, pigeonpea, urad, masur, etc., affected by fungi such as 41 Colletotrichum (Dilani et al. 2017), Uromyces, Cylindrosporium, etc., whereas 42 kharif pulse crops require warm climate throughout their life that is from sowing to 43 harvesting, e.g., arhar, black gram, cowpea, moong, and urad, usually attacked by 44 Ervsiphe, Cercospora, Fusarium, Ascochyta, Alternaria, Phoma, etc. (Tivoli et al. 45 2006). In most of the fungal groups, the temperature varies according to species. 46 Some diseases and their common causal organisms are cited in Table 8.1. 47

Disease/		
symptom	Causal agent	References
Blight	Alternaria alternate, Ascochyta fabae, Stemphylium botryosum	Akem (1999), Davidson and Kimber (2007)
Anthracnose	Colletotrichum truncatum	Kim et al. (2015), Than et al. (2008)
Leaf spot	Cercospora lentis, C. cruenta, C. zonata, Cylindrosporium sp., Helminthosporium, Phoma medicaginis, Pestalotia sp.	Suterman et al. (2011), Ringer and Grybauskas (1995)
Stem canker	Cylindrosporium sp.	Nikmaram et al. (2017)
Downy mildew	Perenospora lentis, P. viciae	Madden et al. (2007), Farouk et al. (2017), Xin et al. (2011)
Wilt	Fusarium oxysporum	Oumouloud et al. (2013)
Leaf rot	Choanephora sp.	Gossen et al. (2016)
Leaf yellowing	Cladosporium herbarum, Pyrenophora tritici-repentis	Raimondo and Carlucci (2018)
Powdery mildew	Erysiphe polygoni, Podosphaera xanthii	Sparks and Kelly (2017), Caffarra et al. (2012)

 Table 8.1
 Some pulse diseases and their causal agents

8.3 Overview of Common Foliar Diseases of Pulses

8.3.1 Blight Disease

Blight can be considered as complete chlorosis, which includes browning and 50 death of plant tissues such as leaves, twigs, branches, and floral organs and fruits. 51 Blight mostly appears as water-soaking spots, toward the edge of lower leaves 52 where dew or water gets collected (Davidson and Kimber 2007). Under cool and 53 moist conditions, water-soaking spots enlarge faster, and a vellow broad portion 54 might be seen around the lesion. While on the underneath, white mold growing 55 zone producing spore (approx. 0.1-0.2 inches wide) may appear at the lesion bor-56 der. Under a wet environment, disease flourishes faster. Dry and warm weather 57 slow down or stop disease development; however, it resumes with weather condi-58 tions being moist again (Akem 1999). Spores are readily disseminated by rain 59 splashing, over-irrigation, or wind. Repeating cycles of production of spores, dis-60 semination, and production of extra spores give blight disease its explosive poten-61 tial. Late blight is most aggressive among all because of its polycyclic nature as it 62 goes through several disease cycles within a year. Seven fungicides were evaluated 63 in vitro against *Exserohilum turcicum* that causes leaf blight (Reddy et al. 2013). 64 The mancozeb (0.25%) alone or combination with carbendazim reduced the dis-65 ease up to 72-73%. 66

8.3.2 Anthracnose

This fungal disease mostly attacks plants during the spring with cool and wet weather, on leaves and twigs. Cool, rainy weather creates favorable conditions for the spores to spread. Fruiting bodies appear as tiny dispersed black-colored flecks, and pink masses of spores are seen at the center of the old black spot. 71

Colletotrichum uses different strategies to cause infection of the host plant which 72 starts from the hemibiotrophic intracellular mode up to the necrotrophic nutrition 73 mode (Bailey and Jeger 1992). But different species undergo diverse infection 74 mechanisms depending on the host plant infected. The initial infection starts as the 75 conidia attach to the host surface, germinate, and produce appressoria following 76 penetration of host epidermis. Fungus colonizing plant tissue results in the forma-77 tion of certain structures called acervuli that contain spores. The pathogen stays in 78 the inert state sometimes in the form of appressoria in tissues of unripe fruits, and 79 infection is caused after it ripens. The management and control of anthracnose dis-80 eases are still being studied. Many studies have concluded that disease management 81 practices are often insufficient to eradicate these diseases. Breeding techniques to 82 develop long-lasting resistant varieties are also not successful due to the involve-83 ment of multiple Colletotrichum species in anthracnose infection (Than et al. 2008). 84 Different species are reported to attack different organs of the host plant, e.g., 85

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C. acutatum and *C. gloeosporioides* infect fruits of host plant at all developmental
 stages, and the leaves or stems are mostly damaged by the species such as
 Colletotrichum coccodes and *Colletotrichum dematium* (Kim et al. 2015).

89 8.3.3 Leaf Spot Disease

Leaf spots, round in shape are found on the leaves of many plant species, mostly 90 caused by fungi that are parasitic in nature. A typical spot has a defined edge and is 91 often dark at the border. When many spots are present, together they can grow and 92 form a blotch or blight. Spots of fungi are usually of free form or round in shape. In 93 spring, when conditions are in favor of the fungus, ascospores discharge from peri-94 thecia and infect young leaves of plants. Once infected, a leaf serves as a good nutri-95 ent source for the fungus to produce secondary inoculum (conidia) inside pycnidia 96 (the surviving structure that protects the spores). Conidia are capable of undergoing 97 several repeated secondary cycles and re-infect other plants nearby. When the leaves 98 of the plant start falling, asci and ascospores are produced within perithecia and are 99 protected until the following spring. The ascospores are characterized by a cylindri-100 cal, curved shape, pointed at both ends with four septa (Ringer and Grybauskas 101 1995). A temperature of 30 °C is favorable for maximum colony growth and acer-102 vuli production. The optimum temperature for growth and sporulation of *Pestalotia* 103 sp. was 25 °C. Germination of spores requires 30 °C, and they don't germinate 104 below 15 °C or above 40 °C (Ramaswamy and Sohi 1984; Naqvi 2004). It was 105 reported that rainfall, relative humidity, and temperature are the weather compo-106 nents significantly affecting the increase of disease severity of Pestalotia (Suterman 107 et al. 2011). 108

109 8.3.4 Stem Canker

Stem canker is often confused with Phytophthora stem rot. Green stem tissue 110 appears below the canker, while it is not present in root rot, there is none. Necrosis 111 and interveinal foliar chlorosis may occur as a result of fungus producing toxins. 112 These symptoms may be similar to those of sudden death syndrome and brown rot. 113 Stem canker is noticed at the latter half of the growing season. During the early 114 reproductive stages of plants, reddish-brown stem lesions appear which are in the 115 portion of the stem node (Backman et al. 1985). The pathogen can survive in the 116 residue of host or the soil for many years in the form of spores which act as the 117 primary source. During rainy weather, spores are produced in the early vegetative 118 stages which splash onto plant tissue causing infection. Until the plant enters the 119 reproductive stage, cankers are not visible on plant tissue where secondary spore 120 production may take place. Infection can occur over temperatures of a wide range, 121 but the fungus needs the moist condition to infect (Nikmaram et al. 2017). 122

8.3.5 Downy Mildew

Downy mildew is caused by oomycete organism. It is spread by airborne spores. 124 The infection is enhanced by prolonging wetness of leaf. Spore formation can occur 125 within 4 days after the initial infection. However, the typical period for germination 126 of spores is 7-10 days (Madden et al. 2007). There are many downy mildew species 127 capable of spore germination by the creation of a germ tube that enters the host. 128 Some species also germinate by zoospores. However, some downy mildew species 129 cannot handle the cool weather and so are reintroduced to another area for another 130 infection to occur (Vittorio et al. 2007). Chitosan application was significantly supe-131 rior to other elicitors to increase shoot length, nitrogen and phosphorus percentage, 132 photosynthetic pigment, and ascorbic acid, proline, and phenolic compounds of the 133 leaves (Xin et al. 2011). The silicon reduces downy mildew disease severity (Farouk 134 et al. 2017) 135

8.3.6 Wilt

Wilt disease affects the vascular system of plants. It starts with vein clearing on 137 younger leaves and dropping the old ones toward the lower side, followed by stunt-138 ing, defoliation, marginal necrosis, yellowing of leaves toward the lower side, and 139 death of the plant. The most abundant is microconidia. Chlamydospores can survive 140 in the soil for a longer time. The mycelium grows intracellularly into the xylem 141 through the root cortex, exclusively within the vessels, and produces microconidia 142 (Saikia et al. 2004a, b). It enters the stream sap and is upwardly transported and 143 germinates where the flow stops. Eventually, the mycelia and the spores clog the 144 vessels of xylem, which prevents the plant from translocating nutrients and their 145 uptake. In the end, the plant transports less and transpires more resulting in stomatal 146 closure, wilting of leaves, and death of the plant. After the plant's death, the fungus 147 sporulates by invading all tissues and continues infecting other nearby plants. The 148 development and deployment of resistant cultivars are generally considered to be 149 the best approach to control Fusarium wilt. Two dominant resistance genes fom-1 150 and fom-2 play an important role in controlling resistance in various races of the 151 host (Oumouloud et al. 2013). 152

8.3.7 Leaf Rot

In leaf rot, lesions are water soaking with various colors and shape formed on the appearing spindle and young leaves; thus the leaflet does not open fully. Central shoots are affected, and further, all the crown leaves get rotted (Gossen et al. 2016). 156 The lesions enlarge and fuse leading to extensive rot of spindle leaves. Rotting 157

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results in the decay of buds as it extends toward the interior of the spindle which 158 further causes the yellowing of leaves. Further infections of the emerging spindles 159 result in the appearance of symptoms in most of the crown leaves. The pathogen 160 survives as long as debris of the infected plant remains. The remaining debris lying 161 on the soil is often the source for primary inoculation that infects other plants of 162 upcoming seasons. High humidity and moisture (dew) on the leaves are needed for 163 the pathogen to infect the host. Cercospora zeae-maydis is an atypical pathogen 164 (Aref and Anderson 1973), whose conidia before penetration can grow and survive 165 for many days. But most spores need to be penetrated within hours of germination 166 for ensuring survival. Considering the weather favoring conditions, the conidia for 167 upper leaf regions may also serve as secondary inoculum. Additionally, heavy rains 168 and wind tend the conidia to disperse during many secondary cycles to other parts 169 of the field causing more secondary infection cycle. In adverse conditions, the 170 pathogen undergoes an interstate and reactivates when conditions are favorable. 171

172 8.3.8 Leaf Yellowing

According to the recent report, P. pauciseptata and P. ramiseptata are the most 173 aggressive species causing leaf vellowing in plants (Raimondo and Carlucci 2018). 174 Yellowing of leaves may be caused by manganese, zinc, or nitrogen deficiencies. It 175 is widely known as chlorosis. The yellow spot of disease, caused by *Pyrenophora* 176 tritici-repentis, is a stubble-borne disease. The fungus survives on stubble in small 177 fruiting black bodies, asci, from season to season. They contain ascospores in large 178 numbers which are in humid conditions ejected forcibly. However, at wet conditions 179 and temperatures between 10 and 25 °C, the second type of spore, conidia, is pro-180 duced. Disease development in higher plants, pulses, and other crops can occur by 181 the secondary spore. It is one kind of secondary infection that leads to loss of 182 high vield. 183

184 8.3.9 Powdery Mildew

Mildew is marked by a white floury covering comprising of conidia. The lower 185 leaves are mostly affected, but it is also seen aboveground part of the plant as well. 186 As the disease progresses, the spots get larger and denser as large numbers of asex-187 ual spores are formed, and the mildew spreads on the entire host including pods. All 188 species of powdery mildew fungus require living tissues of plant for growth. They 189 survive on stem and bud tissue in perennials. The optimum temperature between 68 190 and 77 °F and relative humidity between 40% and 100% are favorable for spore 191 germination. Powdery mildew development is also favored by low, diffuse light 192 (Caffarra et al. 2012). Powdery mildew in pulses (mungbean) is caused by the 193

Podosphaera xanthii, responsible for yield losses of up to 40% (Sparks and Kelly1942017). The mildew spreads faster as the disease cycle can be finished in about19572 hours. However, it takes 7–10 days from the time of infection to the development196of symptoms and the production of secondary spores.197

8.4 Management

The major prominence in research and development to mitigate pulse diseases is 199 chemical control and resistance of host against pathogens. Recently, a shift in man-200 agement practices of pulse diseases is seen, and emphasis was given on identifying, 201 evaluating, and integrating components specific to location for integrated disease 202 management (IDM). In general, IDM follows certain principles (Bailey and Jeger 203 2000). Single component or in combination of other components (fungicide and seed 204 treatment), are used adequately to mitigate pulse diseases. The major components of 205 IDM are the resistance of host plant, disease modeling for the avoidance of high risk 206 or pressure of disease, use of chemicals, biological control, and cultural agronomic 207 practices (Pandel et al. 2009). 208

8.4.1 Resistance of Host Plant

The interaction between the pathogen and the host defines race specificity or non-210 race specificity of resistance and is based on the presence or absence of statisti-211 cally significant interaction between host and pathogen genotypes. It is hard to 212 identify the clear host-fungus interaction or relationship in nature that entirely fits 213 these definitions. Most plant pathogenic fungi show different interactions with 214 their host plants, changing their relationship at different stages of their life cycle 215 depending on the resistance of the host, physiology, the environment, and associ-216 ated virulence genes of the pathogens. Each intracellular structure also prevents 217 non-specific defense of plants triggered by activities of fungi, possibly by intru-218 sion with the system signaling rather than the expression of defense. In resistant 219 cultivars of the host plant, rapid cell death is triggered by the cellular invasion that 220 shares some features with apoptosis of plant tissues and is controlled by resistant 221 genes that are parasite-specific which resemble genes that defend plants against 222 other types of pathogens (Oumouloud et al. 2013). Evidence suggests that a fun-223 gal peptide elicit this response which does not involve the oxidative burst typical 224 of expression of resistance in other pathogen and plant interactions (Heath 1997). 225 However, in general, few of the molecules involved in any fungi and plant interac-226 tions have been characterized completely, and much is left to be discovered 227 (Farouk et al. 2017). 228

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229 8.4.2 Disease Modeling

Disease models help to understand how the disease develops and approaches to 230 test potential mitigation steps. Plant diseases account for about 16% or more of 231 the total vield losses every year. To forecast the spread of these diseases both 232 locally and over long distances, numerical models and monitoring networks have 233 been developed (Knogge 1996). The epidemics of these airborne diseases depend 234 on the production of infectious propagules, their aerial transport, specific infec-235 tiousness, and finally their reproduction (Pan et al. 2010). For modeling disease 236 development, various approaches such as statistical modeling, growth curve mod-237 eling, and mechanistic modeling are developed. A common core of disease epi-238 demic models is the relationship between disease intensity (y) and time (t), which 239 is given by dy/dt, e.g., dy/dt = rL; rL is the parameter determining how fast the 240 disease develops and is dependent on environmental conditions (Maanen and 241 242 Xu 2003).

243 8.4.3 Chemical Control

Chemical fertilizers provide nutrients for healthy plant growth which are a com-244 bination of synthetic primary nutrients as nitrogen, potassium, and phosphorus. 245 They provide the benefit of having more nutrients than organic ones. The different 246 types of chemicals used in agriculture are insecticides, herbicides, soil fumigants, 247 desiccants, fungicides, plant growth regulators, and harvest aids because natural 248 pesticides are not enough for conventional agriculture (Meyer et al. 2016). 249 Organic farmers use a wide range of natural pesticides for controlling weeds, 250 insects, and diseases. The benefits of using chemicals include increasing yield 251 potential that allows farmers to farm more acres of land and protects the soil 252 through conservation methods. Chemical fertilizers and pesticide use peaked in 253 the 1980s but are declining as farmers and scientists are inclined to eco-friendly 254 control methods. 255

256 8.4.4 Biological Control

Many microbes show antagonistic activity against fungal pathogens which could be
used to prepare solid or liquid microbial formulations to apply on sensitive and
diseased plants (Passari et al. 2017, 2019). The use of these microbes also helps the
plants in developing resistance against the fungal infections, e.g., *Bacillus* sp., *Pseudomonas* sp., *Ochrobacterium* sp., etc., which also helps plant growth promotion (Saikia et al. 2003, 2005, 2018). They also induce a defense mechanism against
the pathogens in host plants through induced systemic resistance (ISR) (Saikia

et al. 2003, 2006). The antagonistic activity of some microbes showed prominent 264 inhibition against the pathogen. This would be helpful for the detection and control 265 of the devastating disease (Chowdhury et al. 2018). In general biocontrol agents 266 suppress pathogens and other organisms. However, the interrelationships of many 267 environmental factors can result in multiple interactions among organisms and 268 their environment, several of which might contribute to effective biological control. 269 Furthermore, some natural products also lead to the development of biorational 270 pesticides (Passari et al. 2017; Gardener and Fravel 2002). Prospects for using bio-271 logical control are to limit the damage of plant pathogens in both conventional and 272 organic agriculture (Singh 2014). 273

8.4.5 Cultural Practices

Cultural practices can control fungal diseases in pulses and other plants. The selec-275 tion of resistant varieties of plants is necessary and is selected by proper screening 276 in the field. Plantation needs to be done in a well-drained area, with full sunlight. 277 Airflow and ventilation discourage fungal growth, so crowding of plants should be 278 avoided (Bennett et al. 2012). Diseases such as powdery mildew flourish where the 279 nitrogen rate is higher. It promotes tender leaf formation that causes dense strands 280 that are more susceptible to infection. Thus, organic fertilizers or slow-release for-281 mulations are good choices. If the infestations are severe, the removal and destruc-282 tion of the infected plants are effective. Watering plants in the morning is important 283 as it gives the rest of the day time to dry, so that establishment of fungal disease 284 flourishing in wet conditions could be discouraged. Among the treatments of bio-285 fungicide, leaf extracts of neem (Azadirachta indica), datura (Datura stramonium), 286 and debdaru (Polyalthia longifolia) showed excellent performance in controlling 287 disease (Hasan et al. 2014). 288

8.4.6 Organic Control Agent

Sulfur is highly effective against foliar fungal disease including mildews. So, it can 290 be used at a minimum of 7–14 days interval as a protectant. Garlic naturally consists 291 of high levels of sulfur, which can be added with a few cloves crushed in water, like 292 a homemade spray. It is applied as organic fungicide at the first emergence of patho-293 gens and can be repeated if necessary (Djeugap et al. 2014). However, proper timing 294 is vital for successful control, so it should be made sure to begin at the first sign of 295 the disease. Sulfur can cause damage to other edible varieties such as squash; thus 296 another option is to spray it with a solution of baking soda once in a week. It makes 297 the leaf surface unsuitable for the growth of fungal spores by increasing the pH. 298

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8.5 Conclusion 299

In different aspects of biological control of pulse diseases caused by fungi, a signifi-300 cant improvement has been made, but this area still needs much more investigations 301 and development for the existing problems to be solved. To have strategies in the 302 future with more effective biological control, it is critical for more research to be 303 carried out. On some aspects, novel formulation development, understanding envi-304 ronmental factors' impact on biocontrol agents, mass production of biocontrol 305 agents, and the use of nanotechnology and biotechnology can be used for improving 306 strategies and biocontrol mechanisms (Howell 2007). Future perspectives of pulse 307 disease control are promising and brighter. It is possible to use biological control as 308 a strategy effective for managing diseases of plants, environmental protection, and 309 vield increase and is a sustainable system for agriculture. 310

Acknowledgment The work is supported by a Network Project, MLP-1005, sponsored by the 311 Council of Scientific and Industrial Research, Ministry of Science and Technology, Government 312 of India, New Delhi. The authors are also thankful to the Director, CSIR-NEIST, Jorhat, Assam, 313 for providing necessary facilities to carry out the work and DBT-BIF Centre, CSIR-NEIST, 314

315 Jorhat, for providing the computational facilities.

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Chapter 9 Soil and Crop Health Management for the Cultivation of Pigeon Pea: An Overview of Management Practices

Christy B. K. Sangma

9.1 Background to the Pigeon Pea Crop

Pigeon pea (Cajanus cajan L. Millsp.) is a herbaceous pulse crop, under the 7 Leguminaceae family (Fabaceae), predominantly cultivated in tropical and subtrop-8 ical climatic areas. The crop ranked fifth among the pulse crops in the world con-9 tributing 91% to the world's production. In India, it ranked second next to chickpea 10 (occupying 5.13 million hectares area of total 25 million hectares pulse area, 4.23 11 million tonnes of 18 million tonnes total pulse production and 824 kg ha⁻¹ produc-12 tivity; Anonymous 2015). India is the largest grower of this crop contributing 66%13 of total production, with the larger portion of production coming from seven states 14 (Maharashtra, Uttar Pradesh, Madhya Pradesh, Karnataka, Rajasthan, Andhra 15 Pradesh and Gujarat). There are largely four types of pigeon pea varieties available, 16 extra-short-duration varieties (<100 days), short-duration viz. varieties 17 (100–120 days) grown in the north-western region, medium-duration varieties 18 (140–180 days) grown in Central India and South India and long-duration varieties 19 (>200 days) grown in the north-eastern plain zone (Singh et al. 2013a; Singh et al. 20 2013b). The crop is mostly grown as an annual (var. *flavus*) and as a perennial crop 21 (var. *bicolor*) with the rainfed condition in *Kharif* season. Pigeon pea is a drought-22 enduring crop having a high source of proteins (21-22.3%), vitamins (traces) and 23 minerals such as calcium, magnesium, potassium, phosphorus, iron and fewer 24 amounts of copper and zinc (Saxena et al. 2002). Its carbohydrate content is around 25 57.2% and very less fat content (around 1.7%), and the crop is largely consumed as 26 "dal" (Singh et al. 2004). 27

Pigeon pea is a short-day deep-rooted crop and can proliferate as deep as 1.9 m, 28 which enables the plant to explore moisture from deeper soil layers and can bind the 29 soil and reduce erosion (Singh and Russell 1981). It is a widely spaced crop attaining 30

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B. P. Singh et al. (eds.), *Management of Fungal Pathogens in Pulses*, Fungal Biology, https://doi.org/10.1007/978-3-030-35947-8_9 2

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a height of 1.5–4 m, grown mostly in less fertile soils and marginal areas with row
spacing of 60 cm apart, and comparatively inefficient when grown as the sole crop,
due to various reasons, viz. slow initial growth rate, indeterminate growth habit,
poor source-sink relationship, poor harvest index, poor biomass production, etc.

35 (Reddy et al. 2011; Nandhini et al. 2015).

9.2 Biological Nitrogen Fixation in Pigeon Pea Crop

Pigeon pea crop has the specialty of biological nitrogen fixation (BNF) and estab-37 lishes symbiosis with *Bradyrhizobium* spp. (gram-negative, slow grower) which 38 provides more than 90% of nitrogen (N) requirement for its vegetative growth 39 depending on the conduciveness of the growing environment, variety of crop and 40 type of soil (Nambiar et al. 1988; La Favre and Focht 1983). Pigeon pea is known 41 to be the *promiscuous legume*, which is the capability of the crop to form nodules 42 and nitrogen fixation in symbiotic association with one or more indigenous strains 43 of Rhizobium. But results have shown that Rhizobium strains are less competent 44 than Bradyrhizobium isolates for N₂ fixation in pigeon pea (Anand and Dogra 1991, 45 1997). In a given season, the crop can fix approximately 40–90 kg ha⁻¹ N, and under 46 most favourable environmental conditions, it can fix up to 200 kg ha⁻¹ (Kumar Rao 47 and Dart 1987; Adu-Gyamfi et al. 1997; Anonymous 2010). Mhango et al. (2017) 48 reported that the ability to fix total N differed with cropping systems as well, and it 49 is well understood that under intercropping agricultural system, very low level of N 50 was fixed (15 kg N ha⁻¹) as compared to sole pigeon pea (32 kg N ha⁻¹) crop grown 51 in the field. Other than fixing nitrogen, pigeon pea crop is well-known to add bio-52 mass to the soil through leftover crop residues (up to 3.1 t ha⁻¹), and the roots of the 53 plant help in mineralizing phosphorus which will be available to the plants. 54

Temperature is the main factor bi-directionally affecting the legume-55 Bradyrhizobium symbiosis, viz. (i) restricts the development of microsymbionts 56 and (ii) regulates the growth of the acrosymbiont (Hashem et al. 1998; Kuykendall 57 et al. 2000). At low temperature, the height of pigeon pea was reduced and N₂ fixa-58 tion was hampered. The most favourable temperature was found to be 59 20-30 °C. Besides temperature, variations in soil pH also influence the survivability 60 of rhizobia. The optimum pH for the rhizobial population is neutral to slightly 61 acidic, and extreme soil pH, viz. acidity, alkalinity and salinity, severely affects the 62 legume production and survival of Rhizobium spp. in soil (Slattery et al. 2004). Salt 63 stress and alkalinity also interrupt nodulation, nitrogenase activity and symbiotic N2 64 fixation as a whole (Tejera et al. 2006). Though many studies have been conducted 65 for the effects of salinity on N₂ fixation in various leguminous crops, the physiologi-66 cal mechanisms linked are ambiguous. Likewise, in the same manner, acidity also 67 limits the survival of the rhizobial population and reduced nodulation (Taurian 68 et al. 1998). 69

Bradyrhizobium, an important member of PGPR (plant growth-promoting rhizo bacteria), not only carried out nitrogen fixation but also showed indirect effects like

phytohormone production, iron chelation, phosphorus solubilization, HCN production, chitinase production, etc. (Deshwal et al. 2003). *Bradyrhizobium* was also found to have an antagonistic effect on soil-borne pathogens (Deshwal et al. 2003). 74

9.3 Stressors to Pigeon Pea Production

The productivity of pigeon pea in India is 24.7% lower than the world's average. In 76 general, this low productivity is attributed to major barriers including abiotic and 77 biotic factors limiting the maximum yield potential. The major abiotic stresses 78 affecting the crop are temperature, soil acidity, salinity, etc., whereas biotic stresses 79 include the diseases, viz. wilt, sterility mosaic, Phytophthora blight, Alternaria 80 blight, etc. The crop is also susceptible to various parasitic nematodes, viz. 81 Meloidogyne javanica of Meloidogyne spp. (root-knot nematode), Heterodera 82 cajani (pigeon pea cyst nematode), Rotylenchus spp., Helicotylenchus spp., etc. 83 (Sharma and McDonald 1990). 84

9.3.1 Common Diseases of Pigeon Pea in India

Diseases are the main setback in pigeon pea production. The crop is sensitive to 86 hundreds of diseases caused by fungi, bacteria, viruses, mycoplasma-like organisms 87 and nematodes (Reddy et al. 1993). Among the major diseases (n = 210) affecting 88 pigeon pea, fungal pathogens are responsible for around 83 diseases, and bacterial 89 diseases are reported to be only 4, whereas the viral and mycoplasma causes 19 and 90 104 diseases, respectively. Among the pathogens affecting the crop, 98 nos. of 91 pathogens are reported from India (Nene et al. 1989, 1996). Among these patho-92 gens, only a few can cause severe economic losses. Major diseases of pigeon pea 93 which are common in India are given in Table 9.1. Other than these diseases on the 94 standing crops, infected or contaminated seeds also prove hazardous as they cause 95 pre- and post-emergence losses resulting in reduced germination of seeds and reduc-96 tion of yield and spoiled the quality of seeds during storage. Some researchers 97 (Jalander and Gachande 2011) reported fungal species, viz. Fusarium oxysporum, 98 Fusarium udum, Alternaria alternata, Aspergillus flavus, A. niger, etc., on stored 99 seed samples of pigeon pea. 100

Among these diseases, the fungal disease Fusarium wilt caused by Fusarium 101 udum Bulter was reported to be the highly devastating soil- and seed-borne disease 102 and widely spread in all pigeon pea-growing areas (with maximum damage in states 103 like Maharashtra, Uttar Pradesh, Madhya Pradesh, Bihar and Tamil Nadu) leading 104 to serious yield losses (Pande et al. 2013). Symptoms of this disease include wilting 105 followed by drying up of the crop under field conditions, which show black lines 106 when the infected plant is cut vertically. According to many researchers (Sarojini 107 1955; Vishwa et al. 2005; Khadse et al. 2015), wilting in pigeon pea was also due to 108

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Sl. No.	Disease	Causal organism	Type of damage	Literature
Fungal				
1.	Seedling or seed rot	Aspergillus flavus	Reduces protein content in seeds	Sinha and Prasad (1977)
2.	Stem canker, anthracnose	Colletotrichum capsici	36.6% yield loss	Tucker (1927)
3.	Fusarium wilt	Fusarium udum	30–100% yield loss depending on the growth stage of crop	Reddy et al. (1990); Okiror (2002)
4.	Neocosmospora root rot	Neocosmospora vasinfecta	72.4% wilting percentage	Khadse et al. (2015)
5.	Phoma stem canker	Phoma cajani	5–50% mortality in plants at maturity stage	Behera et al. (2017)
6.	Phytophthora (stem) blight	Phytophthora drechsleri f. sp. cajani	26.3–98% yield loss	Kannaiyan et al. (1984)
7.	Dry root rot	Macrophomina phaseolina	The disease will infect quickly and cause huge economic losses ranging from 10% to 100%. Disease incidence 9–24%	Smita et al. (2015) Maruti et al. (2017)
8.	<i>Alternaria</i> blight	Alternaria alternata	Disease incidence 20–80% in any kind of cultivar	Sharma et al. (2012)
9.	Wet root rot	Rhizoctonia solani	10-50% yield loss	Singh et al. (2009)
Bacteric	ıl			
1.	Bacterial leaf spot and canker	Xanthomonas axonopodis pv. cajani	40% of disease incidence	Gaikwad and Kore (1981)
2.	Leaf spot	Cercospora indica	Yield losses up to 85% and losses are severe when defoliation occurs before flowering and podding	Reddy et al. (1993)
Viruses/	mycoplasma			
1.	Sterility mosaie	Virus	With early infection, 95% yield losses occur	Dahal and Neupane (1991)
2.	Phyllody	Mycoplasma-like organism	NA	NA
3.	Pigeon pea mosaic mottle	Viroid	NA	NA
4.	Rosette	Mycoplasma-like organism	NA	NA
			yield losses annually in pigeo ena and Reddy 1987b)	on pea) (Sasser and
1.	Root-knot nematode	Meloidogyne spp.	Yield losses range from 8% to 35%	Sharma et al. (1993)
2.	Pearly root (cyst nematode)	Heterodera cajani	Suppresses plant growth by 28% and reduces grain yield by 24% and yield loss up to 49%	Saxena and Reddy (1987a, b); Reddy (1997)

 Table 9.1 Common diseases of pigeon pea, their causal organism and type of damage to the crop
 t1.1

(continued)

	Sl. No.	Disease	Causal organism	Type of damage	Literature
t1.51	3.	Root rot	Helicotylenchus	NA	NA
t1.52			spp.		
t1.53	4.	Lance nematode	Hoplolaimus spp.	NA	NA
t1.54	5.	Dirty root	Rotylenchus spp.	14-29% yield losses in	Saxena and Reddy
t1.55				pigeon pea	(1987a)
t1.56	6.	Pigeon pea cyst	Heterodera cajani	Suppresses plant growth by	Saxena and Reddy
t1.57		nematode		28% and reduces grain	(1987b)
t1.58				yield by 24%. Yield losses	
t1.59				over 30%	

Table 9.1 (continued)

Neocosmospora vasinfecta (Anamorph, Acremonium spp.). Besides wilt, 109 Phytophthora blight is another major foliar disease of pigeon pea plant which occurs 110 in the seedling stage as well as in the grown-up plants (Pande et al. 2011). In the 111 seedling stage, the symptoms are similar to the damping-off disease, with water-112 soaked lesions on leaves and breaking of stems, whereas the cankerous outgrowth 113 or galls developed in the stem of the mature plants. The disease is favoured by the 114 high humidity and mostly appears in the low-lying regions of the field and 115 water paths. 116

9.3.2 Abiotic Stresses Affecting Crop Health

Moisture stress (waterlogging or drought), temperature stress (cold or heat), acidity, 118 alkalinity, salinity, nutrient deficiencies and toxicities, photoperiod, etc. are some of 119 the abiotic factors which affect the production of pigeon pea. Among these stresses, 120 moisture stress is common because pigeon pea is mainly cultivated as a rainfed 121 crop. These abiotic stresses contribute 30-100% of yield losses in the pigeon pea 122 crop (Shabala et al. 1998; Sultana 2010; Choudhary 2013; Pooniya et al. 2015). 123 These stress conditions not only affect the crop directly but also indirectly change 124 the quality and quantity of the microflora of the rhizosphere, adversely affecting the 125 growth and nodulation in the plant. The possibility of the damage and the sensitivity 126 to the diseases, e.g. disease caused by *Macrophomina phaseolina*, also increase 127 under stress conditions. 128

9.3.2.1 Drought

Although pigeon pea is considered to be a hardy *Kharif* legume crop because of its vigorous root system, the crop usually suffers from early, intermittent and terminal drought stress, with reduced in yield of about 50% or more (Choudhary 2013; 132 Pooniya et al. 2015). The crop has the four maturity groups, from which extra early and early types complete their life cycle just after the recession of the monsoon season encountering terminal drought in the reproductive phase only. But the medium-

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and long-duration pigeon peas face acute soil moisture deficit during the flowering 136 and pod-filling stages which reduced nodule nitrogenase activity (70–90%), followed 137 by the rate of photosynthesis (50-71%) and root and nodule respiration (31-45%). 138 Such a shortage of soil moisture during crucial developmental stages of the plant, 139 like the flowering and pod development stage, decreases the grain yield significantly 140 (Sharma et al. 2012). Drought stress was also found to decrease the rate of photo-141 synthesis (Kawamitsu et al. 2000) and impairs mitosis and cell elongation with a 142 considerable decrease in the number and size of leaves and overall poor perfor-143 mance of the plant as a whole (Hussain et al. 2008). Small-seeded pigeon pea culti-144 vars were reported to be more drought tolerant than the large-seeded cultivars 145 (Kuhad et al. 1989). 146

147 9.3.2.2 Waterlogging

Waterlogging is another major limitation for crop production and the productivity of 148 pigeon pea in India. Waterlogging accounts for 1.1 Mha of pigeon pea crop area out 149 of the total area, causing an annual loss of 25-30% (Sultana 2010) and a yield loss 150 of 80–100% (Shabala et al. 1998). Soil types that contribute easily to waterlogging 151 are Vertisols and alluvial soils, with characteristics of high water holding capacity, 152 surface crusting and formation of subsoil pan. Waterlogging can affect pigeon pea 153 during germination and early and late seedling stages and can decrease the height of 154 the plant and delays flowering in surviving plants, ultimately reducing the pod's 155 formation, the number of seeds per pod or the seed yield as a whole. 156

Pigeon pea requires well-drained soils and is found to be highly susceptible to 157 waterlogging conditions leading to the sudden death of crop (Choudhary et al. 158 2011). Roots are highly sensitive to anaerobic conditions. The severity of the plants 159 affected due to waterlogging was found to be lower in the intercropped field than 160 sole-cropped fields. The death due to waterlogging may often be confused with the 161 wilt disease of pigeon pea (no sudden death), which can be differentiated with the 162 easy peeling off of bark and presence of brown patches in the collar region in case 163 of waterlogging. Mature plants were found to be more susceptible to waterlogging 164 than the seedlings. 165

166 9.3.2.3 Nutrient Stress (Deficiencies and Toxicities)

Nutrient stress occurs due to imbalance application of chemical fertilizers like nitro-167 gen (N), phosphorus (P) and potassium (K), growing of high-yielding varieties, 168 intensive cropping without addition of secondary and micronutrients, no or less use 169 of organic manures, leaching of nutrients under high rainfall and irrigation, conver-170 sion of nutrients to unavailable form in problem soils, use of high-analysis fertiliz-171 ers, negative (-) interaction of micronutrients with other macro-/micronutrients and 172 soil degradation like soil erosion, soil salinity, soil alkalinity, etc. (Reddy et al. 173 2011; Junjittakarna et al. 2013). Micronutrient deficiencies or toxicities are other 174

limitations for pulse crop production. Restriction of growth and development 175 because of boron (B) toxicity or deficiency is common in leguminous crops (Poulain 176 and Al Mohammad 1995), and these deficiencies or toxicities are more critical in 177 the case of root nodulation than the overall plant growth (Rahman et al. 1999). 178 These micronutrient deficiencies like iron, molybdenum (Mo) and zinc (Zn) or toxicities (boron (B)) can reduce the yield of legume crops at varying magnitudes (Ali 180 et al. 2002). 181

9.3.2.4 Temperature Stress

Pigeon pea, being a warm-season pulse, an optimum temperature requirement 183 during germination is 30–35 °C, during vegetative stage is 20–25 °C and during 184 flowering and pod-filling stages 15-18 °C and 35-40 °C at maturity, cannot with-185 stand chilling (<15 °C) and frost (Sultana et al. 2014; Rana et al. 2016). The stress 186 considerably upsets the growth, survival and reproductive capacity of the plant 187 when the temperature is lesser than 5 °C. At the freezing temperature, intracellu-188 lar water gets converted into ice, which in turn causes shrinkage of cells inside the 189 plant, resulting in wilting and death of plants. Singh et al. (1997) studied the 190 effects of low temperature on floral buds and flower drop in the pigeon pea germ-191 plasm and observed that long-duration cultivars are well-adapted to cold situa-192 tions because of their inherent genetic mechanism to cope with very low 193 temperature during reproductive stages. Choudhary (2007) and Sultana et al. 194 (2014) also observed that low-temperature stress (11.4 °C) reduces the number of 195 buds and flowers in pigeon pea. 196

9.3.2.5 Soil Salinity/Alkalinity Stress

Soil salinity is another major constraint to pigeon pea in regions where it is pre-198 dominantly grown (Subbarao et al. 1991). Crops cultivated in salt-affected soils 199 experience high osmotic stress conditions, while in alkali soils, nutritional disorders 200 and poor soil physical condition decrease the productivity of the crop. Pigeon pea is 201 very vulnerable to soil salinity and the threshold limit is <1.3 dS m⁻¹. However, 202 some varieties of pigeon pea endured 6-12 dS m⁻¹ and even tolerated 3.5 dS m⁻¹ 203 salinity through the adaptive mechanisms of the plant (Tayyab et al. 2016). Saline 204 soils can impair the growth and development of the plant, and these cases are mostly 205 observed in irrigated and dryland agriculture. Salinity was found to prolong 50% 206 flowering stage by 1-2 weeks and also delays the peak flowering stage. It stimulates 207 increased flower shedding, reducing the effective number and weight of the pods 208 (Vadez et al. 2007) finally reducing the seeds (Promila and Kumar 1982). During 209 salt stress, improper flower, pollen grain and embryo formation inhibited proper 210 ovule fertilization. Salinity also is known to obstruct the germination of seeds and 211 decreases nodule numbers, ultimately hindering the plant growth and crop yield of 212 pigeon pea (Singh et al. 2016). 213

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214 9.3.2.6 Soil Acidity

Acid soils occupy considerable areas in different parts of the world. This type of soil 215 is represented by low productivity and infertile areas owing to the toxicities of alu-216 minium (Al) and manganese (Mn) along with deficiency of nutrients, viz. phospho-217 rus (P), calcium (Ca), magnesium (Mg), etc. The key growth-limiting factor in this 218 type of soils is the excess of Al (Singh and Choudhary 2009). In India, acid soils 219 occupy 49 million hectares (Mha), of which 24 Mha have pH below 5.5 (Mandal 220 1997). Pulses are highly susceptible to soil acidity, and pH less than 5.5 leads to 221 restricted root growth because of Al, Fe and Mn toxicity. Slightly acidic to slightly 222 alkaline soils containing 50% or more sand particles were found to favour disease 223 incidence in susceptible cultivars, and it is also noted that a higher proportion of 224 sand in soil favours occurrence of wilt disease (Shukla and Gupta 1975). 225

226 9.3.2.7 Other Constraints

Other limitations to pigeon pea production include faulty sowing practices and seed rate, absence of irrigation facilities, timely availability of quality seeds and use of chemical fertilizers, pesticides, etc. (Ramakrishna et al. 2000; Reddy 2009; Singh et al. 2013a).

In India, pulse crops are cultivated in different agro-climatic regions with varied soil types, rainfall, thermal regimes, topography, etc. This requires precise production techniques with location-specific crop varieties resistant to biotic and abiotic stresses existing in the area. Even the strains used in biofertilizers, biopesticides or biocontrol agents should originate from areas of corresponding agro-climatic regions to be effective and also equally applicable for production technologies like tillage and seeding devices (Singh et al. 2012).

9.4 Soil and Crop Health Management Practices of Pigeon Pea

India is the leading producer (25% of global production), the consumer (27% of 240 world consumption) and the importer (14%) of pulses in the world. Estimates indi-241 cate that the country needs a 4.2% growth rate in pulse production annually to 242 ensure the projected demand of 30 million tonnes by 2030. In 2008–2009, the pro-243 duction of pulses was 14.57 million tonnes (Mt) from an area of 22.09 million 244 hectares (FAO 2016). Since then, the acreage under pulse crops remain stagnated 245 for many years and had failed to surpass the demand. As a result, India is compelled 246 to do heavy imports of pulse every year to meet the pulse demand. This situation is 247 likely to get worse shortly considering the increase in population in the country, 248 decrease in the per capita availability of land, competition from other crops and 249 short of advances in technologies. Considering these facts, the Government of India 250

launched various schemes (National Food Security Mission 2007-2008, Accelerated 251 Pulses Production Programme (A3P), Integrated Schemes of Nutrient and Pest 252 Management Programmes, Price Support Policies, etc.) for the promotion of pulses 253 and to increase its productivity and meet the gap between the demand and the sup-254 ply. Globally, the Food and Agriculture Organization (FAO) had declared 2016 as 255 the "International Year of Pulses" during the 68 Session of the United Nations 256 General Assembly on December 20, 2013 (FAO 2016). This was declared to create 257 awareness about the dietary benefits of pulse crops, increase and sustain the pulse 258 production and ensure self-sufficiency aiming towards food security and nutrition. 259

Three possible options are available to increase production in pulses (including pigeon pea) and to meet the demands, and these are (1) soil health management, (2) 261 crop health management and (3) increase in acreage under pulses. In this chapter, 262 the increase in acreage under pulses to increase production will not be touched in 263 detail, as it is beyond the scope of this section. 264

9.4.1 Soil Health Management of Pigeon Pea Crop

Soil is a complex ecosystem in itself, and functioning processes (viz. nutrient 266 cycling and transformations including mobilization, fixation and mineralization, 267 rate of residue decomposition, soil structure formation, etc.) which are governed 268 largely by soil biota community in the ecosystem are the main drivers in regulating 269 the nutrient supplying capacity or fertility of soils. Soil fertility or health depends 270 not only on elemental constituents of soil but also on the quality and quantity of 271 microbes residing in it. These microorganisms are key component of soil biota com-272 munity, and they are mainly of two types, i.e. the positive effect type or beneficial 273 (PGPRs) and negative effect type or disease-causing organisms, which affect 274 directly or indirectly the productivity and health of any crop (Kennedy and Papendick 275 1995; Pankhurst et al. 1996). This is true, as the plant-derived nutrients and growth 276 factors, attractants or even inducers of enzymes for microbial colonization from the 277 soil. So, maintaining the soil health by supplying all the necessary elements in the 278 form of organic or inorganic manures is crucial for the crop to remain healthy and 279 productive. 280

9.4.1.1 Nutrient Management Practices in Pigeon Pea

The poor yield of pigeon pea crop is mainly attributed to their farming in marginal soils with poor management practices of inadequate and imbalanced nutrient application, no application of organic manures and macro- and micronutrients like phosphorus (P), sulphur (S), zinc (Zn), iron (Fe), etc. Hence, nutrient management is found to exert a great influence not only on growth and yield attributes of crops but also for obtaining sustained productivity of pigeon pea. In pigeon pea, the nutrient requirement (recommended dose of fertilizer (RDF) is 20:40:30 or 20:60:30 kg 288

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NPK ha⁻¹ depending on the region) is much lesser than cereals due to symbiotic N₂ 289 fixation. But, P deficiency could reduce pigeon pea yields by over 30% (Chauhan 290 et al. 1992). The yield can be increased by about 70% by P application @20 kg ha⁻¹ 291 alone, which can be boosted by rhizobial inoculation as well. The pigeon pea crop 292 was reported to consume 56 kg of nitrogen, 5 kg of phosphorus and 22 kg of potas-293 sium to produce 1 tonne of grains (Kanwar and Rego 1983). This indicates that the 294 continued crop production without proper nutrient management practices can remove 295 the huge quantity of nutrients leaving the soil deteriorated in due course of time. 296

Leguminous crop pigeon pea requires a comparatively higher amount of micro-297 nutrients, viz. molybdenum (Mo) and iron (Fe), as they are an integral part of the 298 nitrogenase enzyme and required for N fixation (Choudhary et al. 2014). Besides 299 this, boron (B), zinc (Zn) and sulphur (S) deficiencies are reported to be common in 300 pulse-growing areas (Singh et al. 2013a). To tackle some of these deficiencies, 301 application of gypsum or single superphosphate at sowing was carried out, which 302 supply sulphur up to 20–40 kg/ha, and application of ZnSO₄ @25–50 kg/ha once in 303 2 years also addresses these problems effectively and boosts the crop production 304 (Singh et al. 2013b). A balanced dose of nutrients is also important in increasing the 305 vield of pigeon pea. Application of 25:50:25:20 of N:P₂O₅:K₂O:S in kg ha⁻¹ and 306 $ZnSO_4$ @15 kg ha⁻¹ with organic manures is found optimum for pigeon pea 307 (Anonymous 2012). An unconventional way of nutrient management is to employ 308 soil test-crop response (STCR)-based targeted precision nutrient management prac-309 tices for higher crop productivity with economic use of chemical fertilizers (Suri 310 and Choudhary 2013). Meena et al. (2012) suggested that the rate of fertilizer appli-311 cation based on soil test yield is found to be higher as compared to conventional 312 methods. Acute deficiency can also be managed by foliar spray of nutrient solu-313 tions, e.g. 2% N at flower initiation coupled with manure and fertilizer application 314 (Sharma et al. 2010). Verma et al. (2004) also reported that Zn application in terms 315 of foliar spray @0.5% ZnSO₄ also supplements the nutrient requirement directly, 316 which increases plant height (115.5 cm), pods per plant (185 nos.) and seed yield of 317 the crop (1942 kg ha^{-1}) in comparison to other treatments. 318

Efficient integrated nutrient management practices, especially nitrogen along 319 with biofertilizers, hold a great promise for maintaining the soil health along with 320 the steady supply of nutrients to the plant. Subba Rami Reddy et al. (2011) found 321 that 50% RDF + Rhizobium @200 g/kg seed application as basal dose gives better 322 seed yield of pigeon pea. Inoculation of seeds with a combination of biological 323 fertilizers (viz. Rhizobium + Pseudomonas striata) considerably improved the 324 accumulation of dry matter, the nodulation and the overall yield of pigeon pea 325 (Patil and Padmani 2007). Economic viability of pigeon pea was proved superior 326 with vermicompost application @5 t ha⁻¹ plus RDF (20:50 of N and P_2O_5 kg ha⁻¹), 327 gypsum (100 kg ha⁻¹), ZnSO₄ @25 kg ha⁻¹ and borax @10 kg ha⁻¹ and *Rhizobium* 328 (as seed treatment) in Vertisols of Karnataka (Somashekar et al. 2017). DAP appli-329 cation @20 kg P_2O_5 ha⁻¹ along with *Bacillus polymyxa* also increases the yield. 330 Application of 40 kg P_2O_5 ha⁻¹ through rock phosphate along with either *B. poly*-331 myxa or Aspergillus awamori (P solubilizers) was also found to be effective 332 (Anonymous 2001). 333

Organic components such as enriched composts, FYM, green manure, soil 334 amendments like biofertilizers, etc. affect soil microbial activity, diversity, biomass, 335 respiration and fertility improving the physicochemical characteristics of soils 336 (Grayston et al. 2004). The organic matter also plays a crucial role in maintaining 337 soil physical conditions. Researchers have shown that pulse crop diseases could be 338 reduced significantly with the addition of organic manures, crop residues and 339 organic amendments. These amendments can also reduce the impact of abiotic 340 stresses especially drought stress, salinity conditions (Mayur and Deshmukh 2003), 341 etc. Mayur and Deshmukh (2003) reported that legume wilt incidence was signifi-342 cantly reduced by incorporating de-oiled mustard cake, groundnut cake and FYM 343 into the soils. Kumar et al. (2014) also showed that inoculation of arbuscular mycor-344 rhizal (AM) fungi imparts tolerance to water stress besides phosphorus nutrition in 345 rainfed regions. 346

Leguminous crops perform well under neutral pH soil condition, and nodulation 347 significantly reduces under the acidity and salinity/alkalinity soil conditions. Liming 348 of acid soils plays the main role in neutralizing the acidity. Liming with dolomitic 349 limestone of 80.3% relative total neutralizing capacity, with an assumption of 60% 350 base saturation for 30 days, is the best way for correcting soil acidity (Singh et al. 351 2013a). Throughout this phase, soil moisture content of 60% can be maintained for 352 increasing effectiveness. Other soil amendments that can be utilized for correcting 353 soil acidity are basic slag, paper mill sludge, etc. Band application @1/10 of lime 354 requirement plus required doses of fertilizers annually is also found to be economi-355 cal, practical and effective than lime requirement based on laboratory tests. Furrow 356 application @2-4 q ha⁻¹ (particle size below 80 mesh) before planting a crop is the 357 alternate method of application. 40-100% of yield benefit was observed with liming 358 in furrows alone in leguminous crops like pigeon pea, black gram and cowpea 359 grown in low pH soils. Biochar is another such amendment that can ameliorate soil 360 acidity and can reduce the excess of Al. Besides this, biochar is rich in several 361 nutrients, viz. macronutrients (N, P, K), secondary nutrients (Ca, Mg) and micronu-362 trients (Fe, Mn, Zn and Cu), improve water retention and improve soil conditions 363 (Purakayastha et al. 2013). Biochar is applied in many ways, e.g. broadcasting, 364 banding, spot placement, etc. 365

In the same way, the soil types with pH more than 8 with exchangeable sodium 366 >12-15% require an appropriate management practice for successful cultivation. In 367 such type of soils, mineral calcium helps regulate ion transport into cells of the plant 368 and inhibits Na+ absorption in pigeon pea (Subbarao et al. 1990). Amendments 369 used for chemical amelioration of saline/alkaline soils are those containing soluble 370 calcium ion in it like gypsum and phosphogypsum which is readily available and 371 cost-effective or acid-forming amendments, viz. pyrites, sulphuric acid, aluminium 372 sulphate, sulphur, etc. These chemical ameliorants are incorporated followed by 373 leaching. For cultivation of crops, gypsum or phosphogypsum is applied at 374 15-30 days ahead of sowing @75% of gypsum requirement (GR). According to the 375 crop and available sulphur (S) status in soil, gypsum requirement varies from 100 to 376 200 kg ha⁻¹. Change in yield from 20% to 30% in pulses can be observed with 377 gypsum application alone in soils deficient in sulphur content. 378

379 9.4.1.2 Soil Moisture Conservation Practices

Merely 12% of the area under pulses is irrigated in India (Reddy and Reddy 2010), 380 and the major areas come under the rainfed cultivation system. Therefore, soil mois-381 ture is the major constraint for pigeon pea in dryland agriculture. Adoption of suit-382 able cultivation techniques is the pre-requisite for conserving soil moisture for 383 maximizing productivity under moisture stress conditions. In drylands, a deep sum-384 mer ploughing coupled with levelling is essential for moisture conservation; and 385 similarly, supplementary life-saving irrigation during the post-rainy season would 386 be beneficial for increasing productivity. Chaudhary et al. (2003) suggested that in 387 red lateritic areas, grass, *Gliricidia* or *Lantana* mulch applied @8 t ha⁻¹ retained 388 significantly higher soil moisture and thereby enhanced pulse crop yield by 2-3 389 times compared to no-mulch under rainfed conditions. Fertigation also holds a 390 promise for widely spaced crops like pigeon pea, and through this method, 30–50% 391 more area can be irrigated (Singh et al. 2013a, b). 392

Foliar application of anti-transpirants in pulses is recommended for low productivity of pulses due to erratic and scanty rainfall and prolonged dry spell during flowering and pod-formation stages. Foliar spray of kaolin (6%) with FYM + dust mulch was reported to have a desirable change in the productivity of the pigeon pea + mungbean intercropping system besides reducing evapotranspiration losses of water, suppression of weeds and conservation of soil moisture (Kumar and Rana 2007).

Cover crop also called the *living mulch* also gains considerable attention because 400 of the many benefits it provides for the main crop. It acts as the cover to the soil 401 reducing the erosion as well as reducing evaporation. It accelerates the infiltration 402 of rainwater, improves organic matter content and reduces high temperatures. Cover 403 crops can also suppress soil-borne pathogens, as well as the annual weeds up to a 404 certain extent, and also increase microbial activity. In widely spaced crops like 405 pigeon pea, the cover crop is also a potential option to grow as an intercrop in 406 between main crops. The thick mat of dead plants and residue also acts as the natu-407 ral mulch for the crop. Examples of cover crops are clovers, hairy vetch, field peas, 408 alfalfa, etc. 409

410 9.4.1.3 Manipulation of Rhizospheric Soil for Fungal Disease 411 Management

Soil amendments with decomposable crop residues and oil cakes have been recog-412 nized as the most effective method of changing soil and rhizosphere environment, 413 thereby affecting the quality and quantity of soil microflora and fauna, and have 414 already been reported to reduce nematode infestation, viz. Heterodera cajani 415 (Pandey and Singh 1990). The application of nitrogen-rich organic amendments 416 releases allelochemicals in the soil through microbial decay, thereby reducing the 417 soil-borne diseases. It also has the potential to suppress the plant pathogens and 418 enhance plant growth-supporting microbes, thereby improving the health of the soil 419

as well as the crop (Papavizas and Lumsden 1980). Oil cakes of neem, mustard, 420 mahua, coconut, linseed and sesame at different concentrations (0.25%, 0.5%, 1.0% 421 and 2.0%) were tested against radial growth of *Fusarium udum* (wilt of pigeon pea). 422 Neem, mustard and mahua oil cakes were found most effective botanicals in reduc-423 ing fungal growth. The best growth of pigeon pea plants was recorded with mahua 424 oil cake, but the neem oil cake was most effective in controlling Fusarium wilt 425 incidence and germination of sclerotia of Macrophomina phaseolina (Dwivedi and 426 Dubey 1986). Devadason and Subramanian (2012) observed that the mycelial 427 growth of Macrophomina phaseolina can be subdued by the application of a 10% 428 mahua cake. Neem seed oil (Azadirachta indica) is well-known for its antiviral, 429 antibacterial, antiprotozoal, anti-insecticidal and antifungal (Murthy and Sirsi 1958; 430 Singh et al. 1980) properties. 431

PGPRs (plant growth-promoting rhizobacteria Rhizobium, Bradyrhizobium, 432 Bacillus, Pseudomonas, Azospirillum, Azotobacter, Enterobacter, Arthrobacter, 433 Burkholderia, etc.) can influence several plant development mechanisms, viz. atmo-434 spheric nitrogen fixation, chelation of iron through siderophore production and 435 making it accessible to the plant root, solubilization of certain minerals (like phos-436 phorus, zinc, potassium, etc.) increasing the mineral uptake by plants and increase 437 in yield by 10-30% and synthesis of phytohormones like indole acetic acid, abscisic 438 acid, gibberellic acid, cytokinins, ethylene, etc. (Gupta et al. 2012; Kennedy et al. 439 2004; Patten and Glick 2002; Zahir et al. 2003). It suppresses the phytopathogens 440 and synthesizes antifungal metabolites like antibiotics. Root-nodulating rhizobia 441 are also known to reduce the soil-borne root-infecting fungi. Co-inoculation of 442 P. aeruginosa and Bradyrhizobium has the potential in curbing the root rot disease 443 (M. phaseolina, R. solani and F. solani) on pulses (Ehteshamul-Haque and Ghaffar 444 1993; Siddiqui et al. 1998). 445

In general, the soil microflora increases with the addition of nutrients like nitro-446 gen, phosphorus and potassium. This increase of microflora in the rhizosphere zone 447 plays an important role in the disappearance of pathogenic soil Fusarium as they are 448 unable to sporulate well. Colonization of Fusarium was also found to be low in the 449 presence of minerals like aluminium (Al), cobalt (Co), molybdenum (Mo) and 450 nickel (Ni) (Sulochana 1952). The addition of the solution of micronutrients boron, 451 manganese and zinc is also reported to develop resistance in the host against 452 Fusarium udum. Zinc, on the other hand, inhibits spore germination of pathogens 453 and eliminates pathogens quickly from the soil. Similarly, pre-treatment of seeds in 454 Mn solution provides resistance to the plant against infection, or soil amendments 455 at 100 and 200 ppm of Mn exclude fungal spores in the soil (Subramanian 1956). 456

The cultural operations, viz. deep summer ploughing, soil solarization and adop-457 tion of organic amendments, have been reported to control soil-borne diseases 458 (Pande et al. 2013). Soil solarization is a technique of increasing soil temperature 459 during hot summer days usually by covering or mulching the moist soil with a trans-460 parent polythene sheet. The idea behind soil solarization is to increase the tempera-461 ture (35 °C) of moist soil to a lethal range that destroys the soil-borne pathogens 462 directly and indirectly by destroying the resting structures of the soil borne pathogens. 463 The practice of soil solarization is usually very useful under the organic farming 464

system. The wilt disease (*Fusarium udum*), being the soil-borne pathogen, can also
be managed by soil solarization. Mihail and Alcorn (1984), on the other hand,
reported that soil solarization alone was not effective for controlling *M. phaseolina*in field soils. So, the combined effect of different neem products with an increase in
duration of soil solarization gradually decreases propagules of *M. phaseolina*(Dubey et al. 2009). Lodha (1995) also reported reducing the population of *M. pha- seolina* by 25–42% by summer irrigation alone.

Fusarium wilt of pigeon pea generally develops in the low-lying regions and in 472 water paths and proliferates rapidly in high humidity areas. The best possible way 473 to reduce mortality by the disease are by sowing pigeon pea on ridges avoiding the 474 maximum exposure to rains and allowing better drainages. Another potential 475 approach to decrease yield losses is by growing varieties of pigeon pea resistant to 476 wilt. Umesha et al. (2017) also reported that the ridge sowing or planting method 477 gives higher grain yield and helps in overcoming the *Phytophthora* blight during 478 waterlogging and avoids wilt disease along with seed treatment with *Rhizobium* + PSB 479 which is found to be beneficial to get a higher yield. 480

481 9.4.2 Plant Health Management of Pigeon Pea

The plants' health is usually determined by its environment. Plant environment is in turn comprised of abiotic and biotic factors, which are major constraints in crop production. These factors must be analysed, and effective steps must be undertaken to harness the maximum achievable yields. Since pigeon pea is the second key crop among the pulses in India, crop health management practices are the priority areas, which can be achieved by various following approaches.

488 9.4.2.1 Intercropping

Pigeon pea is a wide-spaced crop having a deep root system, and the initial slow rate 489 of growth offers a good scope for intercropping with short-duration crops like green 490 gram, black gram or sesame. Intercropping is one of the potent means of increasing 491 total pulse production and income per unit area. In the intercropping system, inter-492 crop has a lower plant population than its sole crop; thus, higher dose of nutrients 493 may help improve yield. Mixed cropping or intercropping of pigeon pea (1:1 and 494 1:2) with sorghum (Sorghum vulgare Pers.) provides the most effective and practi-495 cal solution by substantially reducing the incidence of wilt (reduce to 4.3% disease 496 incidence) and *Phytophthora* blight incidence (reduce to 1.2% disease incidence) in 497 pigeon pea which is due to the inhibitory effects of exudates and root secretion of 498 hydrocyanic acid (HCN) of sorghum on pathogen (Singh 2000; Agrawal and 499 Tripathi 2003). Intercropping pigeon pea with other crops can also reduce weeds. 500 Kaur et al. (2015) stated that mixed cropping of pigeon pea with soybean (2:4) can 501 subdue the weed growth resulting in more grain yield by 32% when pigeon pea is 502 503 grown as the sole crop.

One such example is the intercropping of pigeon pea + green gram/black gram 504 which is also helpful in total pulse production and pigeon pea + sesame for enhanc-505 ing the production of pulses and oilseed (Kumar and Kushwaha 2018). For success-506 ful cultivation of any intercropping, plant geometry, suitable varieties and fertilizer 507 management of component crops become important which may vary with crop 508 combination, varieties and location. Pigeon pea crops are fertilized @20 kg N ha⁻¹ 509 for sole whereas for intercropping system @20 kg N + 60 kg P2O5 + 20 kg K2O 510 ha⁻¹ (Kumar and Kushwaha 2018). Patil et al. (2008) otherwise suggested that for 511 integrated nutrient management system, 50% RDF + vermicompost @3 t ha-1 or 512 FYM @5 t ha⁻¹ + biofertilizers was found best for intercropping of pigeon pea with 513 pearl millet. 514

Crop Rotation 9.4.2.2

The rule of thumb for crop rotation is that the same crop should not be grown mul-516 tiple times. The continuity of the same crop in the same piece of land helps in build-517 ing up pathogens, insects, weeds, etc. which reduces the yield of the crop. This calls 518 for higher plant protection measures, viz. herbicides, insecticides, pesticides, etc. 519 involving huge cost. Crop rotation is also called *break crop* as it provides a break in 520 a pest, disease or weed through the removal of suitable host and environment. This 521 cropping system also helps in the conservation of soil moisture and building up of 522 organic matter in soil and improves the physical conditions of the soil. The choice 523 of crops in the rotation should include: 524

(i)	N-demanding and N-fixing crop	525
(ii)	Shallow- and deep-rooted crop	526
(iii)	Large root and small root biomass	527
(iv)	Weed-susceptible and weed-suppressing type	528
(v)	Crops with different pest and disease sensitivity	529
(vi)	Grow catch crops, green manures, etc.	530

(vi) Grow catch crops, green manures, etc.

Crop rotation is one of the best ways of suppressing the wilt of pigeon pea. 531 Nevertheless, along with crop rotation, field sanitation and deep summer ploughing 532 play a major role in successfully curving the wilt disease. A crop rotation of 533 4–5 years was noticed to free the field completely of the wilt pathogen. The duration 534 of rotation can be decreased by eliminating the affected roots. Sorghum, pearl mil-535 let, cotton and resistant pigeon pea cultivars are recommended as rotation crops 536 (Singh 2000). Natarajan et al. (1985) studied and recorded the impact of cropping 537 systems on the disease. In continuous cropping of pigeon pea, the incidence was as 538 high as 64-69%. A rotation of sorghum and fallow reduced it to 16-31%, and two 539 cycles of sorghum followed by pigeon pea reduced the incidence to 16%. The root 540 exudates of sorghum had a suppressive effect on the pathogen in the soil, thereby 541 suppressing infection of pigeon pea (Singh 2000). Some researchers (Sikora and 542 Greco 1990) have reported reducing the population of nematodes (e.g. M. incognita, 543 *M. javanica*, *H. cajani*, etc.) upon the practice of crop rotation. 544

545 Usually, the conventional pigeon pea varieties or landraces are long maturing 546 types so normally intercropped with the early-duration cereals and other pulses. 547 Extra short and short varieties have the potential for inclusion as the sole crop into 548 the rotation as an alternative to rice within the rice-wheat systems, especially during 549 periods of water shortage, price incentives and problems of soil fertility.

550 9.4.2.3 Biopesticides

Constant application of fungicides harms the environment, as the toxic remains 551 persist in soil polluting the entire surroundings. Fungicides also wipe out both the 552 beneficial and disease-causing types and in certain cases even develop resistant spe-553 cies of the pathogen. It also has the chance of exposure to an applicator, and if the 554 fungicides stay in food chains, it is also a threat to the consumer (Hemanth et al. 555 2016). Biopesticide is a potential substitute for the use of synthetic pesticides in 556 plant disease management. It is eco-friendly with the goal of sustainable agriculture 557 means to control plant pathogens through the use of indigenous or genetically modi-558 fied organisms (Taylor et al. 1994). 559

Biocontrol as a component of integrated disease management (IDM) can also be 560 employed effectively to control the pathogen population in the soil. Some of the 561 well-recognized promising biocontrol agents are Trichoderma species, Gliocladium 562 spp., Chaetomium spp., Pseudomonas fluorescens and Bacillus subtilis. Biocontrol 563 agents efficiently suppress pathogens by suppressing the inoculums potential of 564 the pathogen (Baker and Drury 1981) in forms of antagonism as competition, anti-565 biosis or exploitation. It provides resistance to the host plant by indirectly altering 566 its microenvironment (Mclaughlin et al. 1990). Several researchers have reported a 567 decrease in the incidence of diseases after inoculation of soils or seed treatment 568 with non-phytopathogenic fungi, bacteria and actinomycetes (Chalutz and Wilson 569 1990; Mandeel and Baker 1991). Biocontrol agent Trichoderma viride present in 570 the rhizosphere soil of pigeon pea was found to be efficient in managing the dis-571 ease caused by Aspergillus niger, Streptomyces spp., Penicillium spp. and Bacillus 572 spp. Bapat and Shah (2000) also reported that the strain of *Bacillus brevis* has 573 biological potential against Fusarium wilt in pigeon pea. Aspergillus niger, A. fla-574 vus, A. terreus, Penicillium citrinum, Trichoderma harzianum (suppress mycelia 575 growth by 17.52%), T. viride (suppress mycelia growth by 43.13%), T. virens (sup-576 press mycelia growth by 31.79%) and Streptomyces griseus were also demon-577 strated as potent antagonists for control of pigeon pea wilt disease (Upadhyay and 578 Rai 1987; Chaudhary et al. 2017). Sharma et al. (2018) had observed that inte-579 grated disease management by seed treatment with thiram + carben-580 dazim + Trichoderma viride + Rhizobium + soil application of Trichoderma viride, 581 resulting in higher germination percentage (96.8 and 97.2) of pigeon pea, wilt inci-582 dence per cent at 60 DAS (2.97 and 3.15), wilt incidence per cent at 150 DAS (9.68 583 and 7.65) and seed yield (15.10 and 16.28 q ha⁻¹) at two consecutive years, respec-584 tively, was found superior over the rest of the treatments. T. harzianum application 585

@ 10 and 20 g also control the disease by 42.9% and 61.5%, respectively, and *T. harzianum* @ 10 g can reduce disease by 30% even at the high level of pathogen
density (Prasad et al. 2002).

9.4.2.4 Microbial Consortium

Earlier the concept of disease management or biofertilization is to improve the 590 health of the crop or manage the soil health by applying the single antagonist to 591 suppress a single pathogen or to apply a single biofertilizer to enhance specific 592 nutrient requirement in a single cropping system. This concept is also beneficial for 593 the crops, but it is narrow and sometimes not applicable as the crop may suffer from 594 the series of different diseases or may have multi-nutrient deficiencies at a time. So 595 these constraints were analysed, and various microbial consortia have been devel-596 oped, which contain different compatible inoculants (whether bacteria or fungi), 597 viz. N fixer, P solubilizer, Zn solubilizer, biocontrol agents, etc., in a single product 598 and are available only for the research purpose at some leading agricultural insti-599 tutes (e.g. Arka Microbial Consortium of ICAR-IIHR, Bangalore; AAU, Jorhat, 600 Assam; etc.) and are not available commercially. These microbial consortia are 601 those PGPRs and biological control agents which possess the secondary effects and 602 otherwise can be applied as biofertilizers, plant strengtheners and biopesticides. For 603 example, Rhizobium sp. earlier is mainly used for promoting the soil and plant 604 health but now also has been recognized in decreasing diseases also. These products 605 are environmentally safe and can be used for organic agriculture systems. Rajasekhar 606 et al. (2016) evaluated Trichoderma harzianum (TH), Pseudomonas fluorescens 607 (PF), Rhizobium (Rh) and Bacillus subtilis (BS) at variable combinations for plant 608 disease management of pigeon pea or in the form of consortia and have observed 609 that the plant vigour improvement was noticeable and that all the four combinations 610 (TH + PF + BS + Rh) have shown 86%, TH + BS gives 82% and PF + Rh gives 77% 611 disease reduction. 612

9.4.2.5 Weed Management

Weeds served as the alternate host to most of the disease-causing pathogens and 614 nematodes and even directly reduce the yield of the crop. Pigeon pea is severely 615 infested by weeds mainly as it is a *Kharif* season crop with a slow initial growth rate 616 and wider spacing. This wide spacing allows the weed growth to come up very fast 617 and smothers the crop, which reduces the yield of the crop by 55-60% (Kandasamy 618 1999). The reduction in yield can go up to 79.93% if the weeds are allowed to grow 619 till the harvest (Talnikar et al. 2008). So, the initial period during the first 6–8 weeks 620 is a crucial phase, and clean cultivation is recommended during this period. Some 621 major weeds of pigeon pea are Cyperus rotundus, Digera alternifolia, Parthenium, 622 Ageratum conyzoides, Euphorbia hirta, etc. and some of these weeds known to have 623 an allelopathic effect on pigeon pea (Sukhadia et al. 2000). 624

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Field sanitation, clean seeds, application of organic manures, etc. are some of the 625 weed management practices. Besides these, weed destruction by cutting and 626 removal or hand hoeing, hand pulling, tillage, zero tillage, intercropping, crop rota-627 tion, closer spacing and flooding or desiccation and burning, soil sterilization and 628 mulching can be followed to decrease the weed infestation. Mulching is found to be 629 efficient in controlling annual weeds and some perennial weeds like Cynodon dac-630 tylon, Sorghum halepense, etc. (Talnikar et al. 2008). Sugarcane trash mulching 631 @8 t/ha is also effective for control of weeds, increasing yield, conservation of soil 632 moisture and moderation of soil temperature in pigeon pea (Gajera et al. 1998). 633 Chemical weed control (like pendimethalin @1.25 kg ha⁻¹ for broad-leaved weed or 634 fluchloralin 0.5–1.0 kg ha⁻¹ or oxadiazon 0.75 kg ha⁻¹ and quizalofop-p-ethyl 635 @0.5% or alachlor $@2 \text{ kg ha}^{-1}$ for duration legumes) is also found to be most prom-636 ising (Kaur et al. 2015). 637

638 9.4.2.6 Manipulation in Cultivation Practices

Sowing of pigeon pea by broadcasting on flatbed is the traditional method of 639 pigeon pea cultivation which produces low yield and is at the same time prone to 640 waterlogging conditions. This problem can be tackled by sowing crops on raised 641 broad bed furrow, which drains out excess water easily, also saves irrigation water 642 (16–20%) and induces less crop lodging. Ridge and furrow systems of planting are 643 usually beneficial when saline irrigation waters are used. This method is also suc-644 cessful in draining excess water from crop root zone, reduces the incidence of 645 insect pests and diseases and results in 25-30% higher yield in *Kharif* pulses over 646 flatbed planting (Das et al. 2014). Tillage is necessary for obtaining ideal condi-647 tions for proper seed germination, seedling establishment and growth of crops. For 648 pulses, deep ploughing results in better moisture conservation and better root pro-649 liferation. Deep ploughing in summer and exposing the soil to the sun effectively 650 reduce *Fusarium* wilt and root rot in chickpea and pigeon pea. Another option is 651 zero-tillage practices, which minimize the soil erosion, and conservation tillage 652 system which conserves soil moisture in moisture-deficit areas (Das et al. 2014). 653 Apart from this cultivation practices, plant diseases can be kept under control by 654 adopting good field sanitation by removing the infected plants and their debris 655 which keeps the primary inoculum at a low level. Practices like timely sowing of 656 the crop, proper spacing, proper depth of sowing, etc. are also helpful in reducing 657 the diseases. 658

659 9.4.2.7 Resistant Varieties

660 Selection of suitable varieties or cultivars of pigeon pea to different regions and 661 weather conditions, tolerant or resistant varieties to abiotic and biotic stresses, etc. 662 is an important option to improve plant growth, disease management and productiv-663 ity of pigeon pea in any condition as plant response to abiotic and biotic stresses is found to be variety- or cultivar-specific (Maheswari et al. 2015). In drought and heat664stress areas with low rainfall and terminal drought conditions, early maturing variet-665ies (short-duration crops) are widely used.666

AcknowledgementsThe author is thankful to ICAR Research Complex for NEH Region,667Nagaland Centre, Jharnapani, Medziphema, Nagaland 797 106 for the facilities and the financial668support.669

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Chapter 10 The Vital Foliar Diseases of *Cicer arietinum* L. (Chickpea): Science, Epidemiology, and Management

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10.1 Introduction

Chickpea production in the world has increased over the past two decades, ranking 8 third after dry bean (Phaseolus vulgaris L.) and field pea (Pisum sativum L.) 9 (Hirdyani 2014). It dominates other legumes in the international market, and its 10 trade traffic is more than 8 billion dollars annually (Stagnari et al. 2017). This crop 11 contributes to agricultural sustainability through N₂ fixation and allows agricultural 12 production by diversification. India is also the largest chickpea-producing country 13 with 9.33 million tonnes production in 9.48 million ha of cultivated areas (Pande 14 et al. 2005). The productivity in India is lesser in comparison to other chickpea-15 producing countries because of the biotic and abiotic stresses and also due to fungal 16 foliar diseases. Chickpea is grown commercially in soils having residual moisture 17 and with or without minimum irrigation in RRFL (rainfed rice fallow lands) (Pande 18 et al. 2012). The optimal conditions needed for growth and development of chick-19 pea include temperature around 18-26 °C during the night and 21-29 °C during the 20 day and a total of 560-660 mm of annual rainfall. Chickpea is broadly classified 21 into two types: desi type and kabuli type. Desi-type chickpea has seeds that are 22 small and have sharp angular edges, and the color of the seed varies from black to 23 almost cream color or yellow. The desi-type flowers are pink in color and produce 24 about 80-90% of the chickpea throughout the world. Dal (the splits) and besan 25 (flour) are made up of desi type (Purushothaman et al. 2014; Toker et al. 2007). The 26 kabuli type has large, rounded seeds that are head-shaped having cream beige seed 27 color and white seed coats (Pande et al. 2012). Production of chickpea is constrained 28 by foliar diseases as well as insect pests. In general, fungal foliar diseases like 29 Ascochyta blight, Botrytis gray mold, etc. are spread in northern, northern-western, 30 and eastern India (Bretag et al. 2008). 31

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B. P. Singh et al. (eds.), *Management of Fungal Pathogens in Pulses*, Fungal Biology, https://doi.org/10.1007/978-3-030-35947-8_10

32 10.2 History and Origin: Chickpea

Chickpea is a historical crop of the modern age; it was cultivated since 9500 years 33 ago in the Fertile Crescent, through Turkey to Iran (Harlan 1971). Chickpea is cul-34 tivated in association with other crops like wheat, pea, barley, lentils, flax, and vetch 35 as a part of agricultural evolution in the Fertile Crescent (Abbo et al. 2003a). The 36 large area spreading over Israel to Western Iran, from southeast Turkey to Jordan 37 and Iraq, ascertained a balanced collection of basic needs like carbohydrate, protein, 38 oil, and fiber (Diamond 1997). Wild plants were cultivated primarily in this region 39 and were observed archaeologically, and information from 7500 BC and recent 40 years remain feasible (Fuller and Harvey 2006). Chickpea is used as a food in the 41 eighth millennium BC (Tanno and Willcox 2006). Even though, archeological 42 records in chickpea are scarce because the seed is almost crushed down in the car-43 bonization of seed Neolithic chickpea supported the distribution which restricted 44 during the Fertile Crescent, especially at Anatolia and the eastern Mediterranean 45 (Van der Maesen 1972). Later, the Neolithic Period, chickpea expanded westward 46 to modern Greece. During the Bronze Age, chickpea has been spread widely to the 47 west of Crete, south of Upper Egypt, eastward through recent Iraq toward the Indian 48 subcontinent, where the other was found in Harappan community in Pakistan and 49 various sites in Maharashtra and Uttar Pradesh (Colledge et al. 2004). During the 50 Iron Age, chickpea was spread in South and West Asia and in Ethiopia. The crop 51 expanded with the group of originator crops from the Fertile Crescent toward West 52 Central Asia and also Europe from 5500 BC (Moreno and Cubero 1978). In the 53 sixteenth century AD, chickpea was produced by the Spanish region and Portugal; 54 and in the eighteenth century, kabuli type spread in the Indian region from the 55 Mediterranean region (van der Maessen 1972). Indian immigrants in the later nine-56 teenth century imported the desi chickpea to Kenya (van der Maessen 1972). At 57 present in the USA, Canada, and Australia, chickpea breeding programs have 58 started. The related species of chickpea is *Cicer reticulatum*, which is the only 59 related species in the gene pool and spread in southeast Turkey. Numerous addi-60 tional *Cicer* species of almanac and perennial are hereditarily found in the genetic 61 makeup as per AFLP (amplified fragment length polymorphism) analysis (Kumar 62 et al. 2016). The actual difference among the wild relatives and the native chickpea 63 is the loss due to vernalization which is a polygenetic attribute (Abbo et al. 2003a). 64 The most widespread production of chickpea occurs in North America and the 65 Middle East and un-moistured winter regions of India (Abbo et al. 2003b). 66

67 10.3 Center of the Diversity of Chickpea

The spread of old and wild type occurs in the main three areas from 8° N to 56° N
latitude and 8° W to 85° E longitude especially Ethiopia, Crete, Western
Mediterranean, Greece, the Caucasus Iran, Asia Minor, Central Asia, Himalayan

region, and Afghanistan. Domestic chickpea is presently highly nurtured in 71 Australia, southern South America, African Mediterranean regions, Ethiopia, the 72 European Mediterranean region, southern Asia toward Iran to Myanmar, and the 73 Middle East encompassing Turkey, Iraq, and Israel (Van der Maesen 1972). 74 International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), India, 75 is the largest GenBank for chickpea, which consists of 17,250 accessions and 6390 76 of Indian diversity, followed by 4850 of Iran, 930 of Ethiopia, 700 of Afghanistan, 77 480 of Pakistan, 470 of Turkey, 390 of Mexico, 220 of Syria, 139 of Chile, 133 of 78 Soviet Union, and many additional countries from Northern Africa, Southern 79 Europe, East Africa, North America, and South America (Abbo et al. 2003a). 80 International center for Agricultural Research in the Dry Areas (ICARDA), Aleppo, 81 Syria, for chickpea of kabuli type, the genebank consists of 12,070 accessions from 82 1780 of Iran; 970 of Turkey; 410 of India; 340 of Chile; 300 of Uzbekistan; 280 of 83 Spain; 270 of Tunisia; 230 of Morocco; 210 of Bulgaria; 170 of Portugal; 160 of 84 Russian Federation; 160 of Mexico; 150 of Jordan; 120 of the USA; 110 of 85 Bangladesh, Tajikistan, and Azerbaijan; and some further provinces lesser (100) 86 like Italy, Ethiopia, Palestine, South America, Algeria, North Europe, tropical 87 Africa, and Egypt (Diamond 1997). 88

10.4 Chickpea Production

Chickpea is also known by different local names: *hamas* (in the Arab world), *zimbra*90 (in Ethiopia), *nohud* or *lablabi* (in Turkey), *chana* (in India), and *garbanzo* (in Latin
91 America). Chickpea crop production spread from 6.6 million tonnes in the year
92 1998–1999 to 9.5 million tonnes by 2000–2001 (Moreno and Cubero 1978).
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10.5 Ecology of Chickpea

The chickpea evolution is different from the other wild type of the West Asian 95 Neolithic crops, and it shows a part in regulating the crop habitat. The chickpea 96 habitat can be characterized easily with the advanced high-resolution information of 97 the climate and geographical information system (GIS) software freely present in 98 the public databases (Hijmans et al. 2001). The areas like Egyptian Nile Valley, Iraq, 99 Pakistan, and central Iran retain the lowermost annual report precipitation in cold 100 winter and mix the midsummer heats with adequate winter temperatures (Rousta 101 et al. 2018). 102

Temperature and altitude of chickpea103Altitude and rainfall variableness remain less in Europe than in West Asia and104North. South Asia's yearly temperatures remain higher, earlier to the beginning105of the monsoon. It was observed minor dissimilarity between the mean106

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temperature of the warmest quarter in between northern and southern halves of
the subcontinental distribution of chickpea which ranges from 30.8 to 31.9 °C,
and the mean winter temperatures vary from the North (16.8 °C) to the South
(22.1 °C). In Central Asia chickpea is cultivated in areas with a high series of
temperature variation and rainfall unevenness, which leads to a hasty change in
the altitude region (Bhat et al. 2017).

113 • Summer-dominant rainfall region environments of chickpea

Chickpea-growing regions like South Asia, Peru, and East Africa are summer-114 dominant rainfall environments (Ahmed et al. 2016). There is a strong decrease 115 in rainfall in the Indian subcontinent from the southeast to northwest; Madhya 116 Pradesh, a central state with higher rainfall and summer-dominant rainfall region 117 in the subcontinent, produces 50% of the chickpea (Bhat et al. 2017). Chickpea 118 growing region in Mexico is arid from 119 to 284 mm/year and is a summer-119 dominant rainfall region, where summer and winter rainfall proportion increases 120 from 36% to 43% and 33% to 46%, respectively (Nicholson 2014). 121

10.6 Adaptation of Chickpea: Stresses, Cropping Systems, and Habitats

124 10.6.1 Stresses in Chickpea

Stresses in chickpea can be classified as biotic stress and abiotic stress based on coarse agro-climate divisions. In the Mediterranean rainfall region and summerdominant rainfall region, drought is dangerous and is intensified by heat pressure (Saxena et al. 1996). In India, for most summer-dominant rainfall region, Fusarium wilt-root rot complex, Ascochyta blight, and Botrytis gray mold are the biotic stresses which contribute to disease distribution and are estimated to cause 10% of the annual yield loss (Singh 1990).

132 10.6.2 Cropping Methods of Chickpea

Seeding methods for chickpea vary in various environments. The highest range of seeding approaches was found to be in the Mediterranean region, because of the comparative strength of the biotic and abiotic stress (Rasool et al. 2015).

Maturation of Chickpea in Late Spring or Early Summer of the Autumn-Sown Rainy Season

138 It is a regular chickpea cultivation system for regions with relatively warm winter 139 and less biotic stress or pressure because it works on intra-seasonal rainfall and 140 decreases the disclosure to drought. In West Asia and North Africa (WANA),

especially in warmer regions of Iran and in the Nile Valley, both countries use this

system to grow chickpea with supplementary irrigation in the areas to decrease 142 drought stress (Saccardo and Calcagno 1990). At present-day winter, sowing and 143 drill irrigation have been used by approximately 90% of Israeli farmers. Australia is 144 biotic stress-free until the mid-1990s, and later production of chickpea declined, but 145 it is recovered by the release of resistant variety and by adopting good management 146 practices (Hughes et al. 1987). In Mediterranean Australia, winter temperature is 147 moderate, and autumn sowing of chickpea is exposed to suboptimal temperature on 148 flowering and can delay pod set by 30 days more. Prompt flowering expands yield 149 constancy and attains alteration to water deficiency but expands the threat of encom-150 passing less temperature (Saccardo and Calcagno 1990). 151

Spring-Sown Chickpea in Post-Rainy Season Maturation in Summer

It is a regular chickpea cultivation system of Mediterranean climates in WANA and 153 minimizes the risk factor of winter frosts, disease stresses, and the farmers to take a 154 decision for planting based on soil moisture profile (Hamwieh and Imtiaz 2015). In 155 Tunisia, winter stress is lowered geographically, as the crops are grown in low ele-156 vation of <600 m deep clay loams in areas of semiarid, avoids heavy rainfall of 157 >1000 mm/year, along with areas and frost-prone areas. Chickpea is sown in the 158 middle of May to escape high temperatures which will occur post-October in north-159 eastern Australia (Saccardo and Calcagno 1990). Cultivation can be done in dry-160 sown if sufficient soil moisture is present, or as farmers delay for opening rain, 161 which leads postponement of sowing till August in a few of the regions (Hamwieh 162 and Imtiaz 2015). The chickpea crop can tolerate heat stress at phases of maturation, 163 normally in November (27-30 °C). Although rainwater tends to rise from October 164 in many regions, chickpea crop cannot enter the similar terminal drought stress in 165 South Asian environments (Kumar 2017). 166

10.6.3 Chickpea Habitat Range

Chickpea is grown in diverse habitat which consists of altered climate, cropping 168 system, and stress. Chickpea is essentially separated into definite ecotypes, showing 169 local selection pressures in the region of millennia. From the past 30 years, there has 170 been an evaluation of germplasm ranging from characterization and resistance 171 screening by many international centers and physiological studies based on acces-172 sion number (Upadhyaya 2003). Chickpea physiological and habitat understanding 173 is a must, and major stresses can be avoided by combinations of sowing strategies 174 and appropriate phenology (Berger et al. 2006). Chickpea phenology is increased 175 by drought stress as it decreases the thermal time for flowering, maturity, and pod 176 fill; it also lowers the water potential, photosynthesis pod number, and yield (Berger 177 et al. 2006). Chickpea is also having dehydration postponement and consistency of 178 tolerance, like deep rooting, extraction of high soil water, and adjustment of osmosis 179 (Summerfield et al. 1985). Chickpea is highly tolerable to heat stress than the other 180 cold grain legumes like field pea, lentils, and faba bean, and it also absorbs less 181

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incident radiation approximately <50% photosynthetically available radiation 182 (PAR) than other seasonal legumes. Kabuli-type characters were demonstrated in 183 East Asia, Europe, and the Mediterranean, while the desi character was common in 184 Africa and also in Southeast Asia (El-Amier et al. 2015). The vegetative phase, 185 when extended under long-season conditions, increased biomass accumulation and 186 reproduction and delayed flowering till the temperature becomes sufficiently warm 187 to aid the pod set. The difference among provinces has been detected in the assess-188 ment of Ethiopian drought-tolerant germplasm (Berger et al. 2006). 189

190 10.7 Uses, Consumption, and Utilization

From the beginning of agricultural time, legume crops have several uses depending 191 on the utilization of different plant parts. Dry or green seeds are applicable for ani-192 mal feed, fodder, and organic manure. It is also used as a whole and mixed with 193 other cereals (Kumar 2017). Legumes are eaten as a main course in the dish either 194 singly or with meat, fish, and snacks, green or dried. One of the examples of legume 195 is chickpea. Chickpea can be packaged, ice-covered, canned, and precooked. It is a 196 source of oil which is used in baking protein-rich cake (Venn and Mann 2004). It 197 contains protein and carbohydrates and has nutritive value. It can also fix nitrogen 198 from the atmosphere which is secreted into the soil. The cultivation is decreasing in 199 recent years due to the cause of their marginalization of a late entry in the market 200 (Rimal et al. 2015). The legume crop is the essential food of the vegetarian dietary 201 system, so it is directly linked with Indian civilization (Agbola et al. 2002). The 202 pulses or legumes can be dried properly and conserved to consume throughout the 203 year. Consumption per capita of pulses of 80 g/day is advised by the World Health 204 Organization (WHO) and consumption of 47 g/day by the Indian Council of Medical 205 Research (ICMR) (Misra et al. 2011). Consumption in India is less than 30-34 g/ 206 day/person because of the unavailability and price rise of pulses (Akinjayeju and 207 Ajayi 2011). 208

209 10.8 Nutritional Value of Chickpea

Nutrition through food is necessary for human life. Nutrition provides energy, macronutrients, micronutrients, etc. for growth, tissue maintenance, regulation of
metabolites, and physiological functions. Chickpea in many countries is a staple
food and plays an important element in the diet of vegetarians around the world.
Chickpea is a valuable source of minerals, vitamins, energy, fibers, and also healthbeneficial phytochemicals (Brenes et al. 2008).

216 Nutritional Composition

- 217 The nutritional composition can vary due to the environment, climate, soil biology,
- soil nutrient, stress factors, and agronomic factors (McCleary 2003).

• Energy 219 Energy is defined as gross energy (MJ/kg) or as a caloric value (kcal/100 g). 220 Chickpea has an energy value of 14–18 MJ/kg or 334–437 kcal/100 g for desi 221 types, and for kabuli type, it is 15-19 MJ/kg or 357-446 kcal/100 g. It showed 222 that the kabuli type has higher energy than the desi type due to the presence of 223 the seed coat component (Perttilä et al. 2005). 224 Protein and amino acid 225 The protein concentration of desi type ranges from 16.7% to 30.6% and for kab-226 uli type 12.6% to 29.0%. Chickpea is used for the treatment of malnutrition and 227 kwashiorkor in children because of its high protein content (Greenfield and 228 Southgate 2003). The body is also provided with amino acids to synthesize new 229 proteins for repairing and replacing damaged tissue and to synthesize enzymes, 230 hormones, and growth factors. Chickpea has a high amount of sulfur amino acid 231 than the lysine (Sotelo et al. 1987). 232 • Lipid and fatty acid 233 Chickpea consists of 2.9–7.4% lipid content for desi and 3.4–8.8% content for 234 kabuli (Jukanti et al. 2012). The total lipid content consists of 62-67% of poly-235 unsaturated, 19-26% of monounsaturated, and 12-14% of saturated fatty acids. 236 Essential fatty acids like linolenic and linoleic acid are supplied through the diet 237 (Trumbo et al. 2002). 238 Carbohydrates 239 Carbohydrates are the most important component in chickpea, having 54–71% 240 for desi type and 54–71% for kabuli type (Greenfield and Southgate 2003). The 241 key types of carbohydrates present are oligosaccharides (like raffinose (2.2%), 242 stachyose (6.5%), ciceritol (3.1%), and verbascose (0.4%)), polysaccharides 243 (like starch (30–57%)), monosaccharides (like glucose (0.7%), ribose (0.1%), 244 fructose (0.25%), and galactose (0.05%)), and disaccharides (like maltose (0.6%)) 245 and sucrose (1-2%)) (Joint FAO/WHO 1998). 246 • Minerals 247 Chickpea plants absorb the minerals (like, B, Fe, Mn, Zn, Cu, Ni, Ca, Mg, K, P, 248 S, Cl, and Mo) from the soil and transfer it to the seed performs a metabolic 249 activity like photosynthesis, respiration, chlorophyll synthesis, and cell division 250 (Sujak et al. 2006). 251 Vitamins 252 Chickpea comprises of a high source of water-soluble vitamins like the 253 B-complex vitamins (B1, B2, B3, and pantothenic acid) and vitamin C and lipid-254 soluble vitamins like vitamin A (provitamin A carotenoids), vitamin E (tocoph-255 erols and tocotrienols), and vitamin K (Who and Consultation 2003). 256 10.9 **Foliar Fungal Disease**

Chickpea is the most essential cool season pulse crop grown in dry regions. The 258 chickpea plant agonizes commencing fungal foliar diseases that distress the growth 259 stage of chickpea. The pathogens that infect the plant include bacteria, nematodes, 260

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viruses, fungi, and mycoplasma, which lead to severe crop yield loss. Among this,
fungi are the most threatening group that affect the roots, stems, flowers, leaves, and
pods of chickpea (Nene et al. 2012).

264 10.9.1 Ascochyta Blight (Ascochyta rabiei (Pass.) Labr.)

265 Distribution

Ascochyta blight (AB) is a viral disease found in West Asia, Southern Europe, and 266 Northern Africa. In Pakistan, it occurs in February and March and disease will 267 develop accordingly; and in Northern India, it happens when the crop canopy is 268 very dense. In West Asia, Northern Africa, and Southern Europe, such situations 269 usually occur from March to May. In winter chickpea is sown toward the 270 Mediterranean region, and the blight symptoms are found when the climate is wet 271 and warm in November and December. The disease has been found to develop 272 among 35 countries along 6 continents and presently seen in Canada and Australia; 273 it can expand swiftly to different areas of chickpea production (Nene et al. 2012). 274

275 Economic Importance

The fungal foliar disease causes crop yield loss and quality loss of up to 100%

277 (Nene et al. 2012).

278 Epidemiology

Ascochyta blight occurs through seed transmission of Ascochyta rabiei. Airborne 279 spores of A. rabiei are found to play a major vital role in epidemics of the disease 280 (Kaiser et al. 2000). A. rabiei either lives on the seed or inside it or can be found in 281 the plant debris of diseased left over in the fields as a mycelium and pycnidia or at 282 its teleomorph stages and can serve as an agent of the disease (Santra et al. 2001). 283 The secondary spread of this fungus occurs through conidia and ascospores. 284 Development of teleomorph, the stage of the sexual reproductive, appears due to the 285 mating of compatible new types in new areas spread through the air (Guarro et al. 286 1999). The teleomorph stage assists the pathogen in a longer duration of survival in 287 its host, though it has never been seen in the newly infected host. In many regions, 288 though, pseudothecia are found in infected plant wastes. Seed transmission in a field 289 causes pathogen distribution randomly, giving the cause of many initial infections. 290 Wet, cloudy, and cool weather is favorable for the disease development. In a cool 291 climatic condition, the density of asci and ascospore production per pseudothecium 292 are much higher than the warm condition (Daehler et al. 2004). Ascospores are also 293 necessary for dispersal of the pathogen to long distances. The ascospore gets 294 discharged to the air from pseudothecium during the wet condition. Production of 295 ascospore on largely infected crop residues can reach up to 1.5×10^4 ascospores/ 296 mm² on the tissue surface (Manstretta and Rossi 2015). The productions of conidia 297 per pycnidium are much more in cool regions compared to warmed counterparts. 298 Strong wind and rain can scatter conidia grown on diseased plant parts, provided if 299 conidia are present in water droplets or rain splash. Relative humidity compared to 300 temperature plays a more vital role as a critical factor in the determination of the 301

development of pseudothecia and pycnidia on crop debris (Vidal et al. 2017). The 302 disease best develops at low temperature, optimum being at 20 °C. The moist envi-303 ronment also acts as a vital factor to produce severe infection. Dry periods after 304 immediate inoculation may sometime induce disease severity though dry period 305 exceeding 12 hours after 6 hours of wet treatment may reduce the disease develop-306 ment. In tropical countries, A. rabiei by crop debris get influenced by the low rain-307 fall and high temperature during the out of season summer months, which is 308 detrimental for the survival of the pathogen A. rabiei. Impacts of light in in vitro 309 conditions reportedly have insignificant influence on pseudothecial development 310 and discharge of ascospores (Sehulster and Chinn 2003). 311

Symptoms

AB is typically seen during the flowering and podding stage as patches (Gurjar et al. 313 2012). The disease can be observed at an early stage of growth. When the pathogen 314 is seed-borne, the germination time is favorable for the development of disease at 315 the stem base with dark brown lesions (Lammerts van Bueren et al. 2004). The 316 seedlings which are affected can be collapsed and die due to the formation of pyc-317 nidia. The disease spread from the seedling to the flowering and podding which 318 results in patches of diseased plants. The disease appears in the form of spots of 319 small water-soaked in the young leaves in the branches when the origin is airborne 320 and conidia or ascospores (Nene et al. 2012). These spots enlarge and integrate 321 which blights the leaves and the buds that lead to disease development under favor-322 able conditions and also pycnidia presence on blighted leaves and buds. Because of 323 susceptible cultivation, the necrosis spread through the buds, which kill the plant. In 324 severe infection of the foliar disease, the entire plant gets dry and falls off. If the 325 temperature is hot, the condition is unfavorable for a disease formation, and the 326 infection remains in the leaves, stems, pods, and petioles as discrete lesions. The 327 symptoms appear like round spots that have brown margins where pycnidia are 328 presently showing a gray center that appears like concentric rings. Lesion size var-329 ies from 3 to 4 cm long on stems. If the disease arises during the pre-flowering stage 330 when conditions are unfavorable, the crop grows with the symptoms that are visible 331 on the older branches. Pods with fully developed lesions are round having 0.5 cm 332 diameter along with pycnidia arranged in concentric rings. The pod becomes 333 blighted and fails to grow any seed if infection occurs in the early developmental 334 stages of the pod growth. Shriveled seed and infected seed have resulted from late 335 infection. The seed shows symptoms of brown discoloration and visible pycnidia 336 which can be seen by the naked eye (Pande et al. 2012). 337

Pathogenesis

Ascochyta rabiei germinates after 12-48 hours of inoculation. Through leaflets, the 339 pathogen reaches to petiole and then attacks the stem. Following its germination, 340 the pathogen forms its germ tube and appressorium-like structure, which is a spe-341 cialized hyphal cell that occurs at the tip toward the germ tube required for penetrat-342 ing the plant cell. The appressorium is kept apart from the germ tube through a 343 septum and surrounded by mucilaginous exudates. The fungus at first penetrates its 344 hyphae through the cuticle and traversing the subcuticular region reaches the fore-345 front of epidermal cells. Penetration in the epidermal cells occurs through the wall, 346

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keeping the protoplasmic structures intact, reaches to the intercellular space, and 347 resides and grows between epidermal and palisade parenchymal cells (Pande et al. 348 2005). The diameter of hyphal cells varies in and out of the cell as $3.5 \,\mu\text{m}$ and $2 \,\mu\text{m}$. 349 respectively. Meanwhile, dark aggregates of mycelia start to grow at the subepider-350 mal portion. Subsequently, the structure of epidermal, palisade, and spongy paren-351 chyma starts to deteriorate and eventually gets disorganized. Infection near the 352 stoma occurs through penetration of hypha through the juncture of guard cells and 353 subsidiary cells regardless of whether the stoma is open or close. After the disorga-354 nization of leaf cells, pycnidium emerges from the damaged tissues. From pycnid-355 ium, conidiophores arise and subsequently conidium gets dispersed into the 356 surrounding environment and through which new chickpea crops get infected 357 (Galloway and MacLeod 2003). The pycnidia originate after the fifth day of inocu-358 lation. By the seventh day, non-lignified cells almost get deteriorated particularly 359 through necrosis, but lignified cells like xylem and tracheary elements remain 360 mostly unharmed. The pathogen while spreading from leaflet to stem through peti-361 ole infects the phloem vessels with less or no harm to xylem vessels, and conse-362 quently in some instances, the leaf breaks off from petiole. However, the fungal 363 hyphae colonize both the xylem vessels and phloem vessels in the stem, and the 364 walls of xylem and phloem vessels remain intact, while extensive damage happens 365 to parenchymatous tissues (Smith et al. 2017). Although pathogen infects stems 366 directly through its cuticle evading the usual route from the leaf, during pycnidia 367 formation, parenchymatous cortical degradation and tissues of the pith degradation 368 suggest that involvement of toxins and enzymes for cell wall digestion is inevitable 369 (van den Brink and de Vries 2011). Reportedly, in the process of the pathogenesis 370 of A. rabiei, solanapyrone A, solanapyrone B, and solanapyrone C are required. 371 Though under in vivo condition only solanapyrone C has been found and nonap-372 pearance of other toxins in experimentation probably due to their low concentration. 373 The application of solanapyrone in combination or independently results in promi-374 nent symptoms followed by an epidermal, palisade, and spongy parenchymal tissue 375 contraction due to the effect of toxins in the protoplasm. Solanapyrone A is said to 376 be the most toxic, resulting in shriveling, loss of turgor, broken stem, and chlorotic 377 leaves (Kim et al. 2015). Phytoalexins like pterocarpans get degraded by A. rabiei 378 through its conversion to 2-OH isoflavones and 1a-OH pterocarpans due to the 379 activity of reductase and hydroxylase enzymes. The two kinds of enzymes particu-380 larly act upon two isomeric forms of phytoalexins, namely, maackiain and 381 medicarpin. Apart from these enzymes, cutinase and polygalacturonase are also 382 found to act upon the host system (Uchida et al. 2017). 383

384 10.9.2 Botrytis Gray Mold (Botrytis cinerea Pers. ex Fr.)

385 Distribution

Botrytis gray mold (BGM) is a foliar disease found in Bangladesh, Nepal, India, Pakistan, Argentina, and Australia. BGM has also been observed in Canada, Chile,

Mexico, Hungary, Spain, Turkey, Vietnam, and the USA (Jain 2011).

Economic Importance

BGM fungal foliar disease causes yield losses of about 10% (Tivoli et al. 2006).

Epidemiology

Botrytis gray mold is the most detrimental crop disease after Ascochyta blight 392 (Shafique et al. 2014). The pathogen of this foliar disease has a very high host 393 range and can live on other crops as well as weeds, and hence the disease is wide-394 spread. Damages mostly occur during higher temperatures and humidity. The tem-395 perature required is greater than that of the optimum temperature needed for 396 Ascochyta blight development. BGM originates from seed, and the fungus has a 397 large range of hosts. The disease is generally observed during floral growth when 398 the canopy of the crop is fully matured. Excessive vegetation, too much irrigation, 399 rain, and close spacing are causes that favor disease growth and development. 400 Temperature ranges between 20 and 25 °C and high humidity during podding and 401 flowering period also favor disease growth. The disease may also occur subse-402 quently after the appearance of Ascochyta blight (Malhi et al. 1994). Botrytis cine-403 rea can inhabit on chickpea seed without showing any symptoms for more than 404 5 years. The period of survival gets largely affected by the storage temperature, 405 particularly between 5 and 10 °C being optimum for survival for up to 5 years. The 406 temperature at 20 °C has been observed to have reduced growth of the pathogen 407 from 95% to 2% at the duration of 12 months. Heating the infected seed at the 408 moist condition at a temperature of 50 °C resulted in a significant reduction of the 409 infection (Williamson et al. 2007). Studies showed that chickpea leaves infected 410 with the fungus get decomposed within a couple of days to months, but the deterio-411 ration of stems through infection requires longer duration. In India, the pathogen is 412 observed to survive for approximately 8 months in leftover infected crops on the 413 soil and is the principal source of the initial inoculum. Asexual sporulation of the 414 pathogen occurs on the stubble during higher temperatures and high humidity. 415 Spores get blown to the air from the debris of the infected crop and spread to other 416 places. The pathogen inhabits the soil in the form of mycelia and sclerotia (Bhaskar 417 et al. 2009). In crop stubbles, sclerotia occur in many host species, as the disease 418 has long-term survival on the host. However, in Australia, sclerotium does not 419 show long-term survival. In Europe, apothecia originate from fertilized sclerotia 420 (Cannon and Kirk 2007). Chlamydospore occurs during extreme conditions like 421 drought, nutrient deficiency, bacterial attack, and change of pH. Mycelium can be 422 produced through the germination of chlamydospores, which serves as secondary 423 inoculum (Stevens 2002). 424

Symptoms

The absence of pod setting is the primary symptom of the disease where leaves and 426 stems do not show symptoms. The disease shows symptoms under highly favorable 427 conditions and forms patches in the plant which often dies. The symptoms are vis-428 ible on stems, pods, leaves, and flowers as a dark brown or gray lesions layered with 429 sporophores under high humidity. 10 mm- to 30-mm-long lesions are present on the 430 stems which grid the stem fully. The branches break at the place of the gray mold 431 where it has caused rotting. The leaves and flowers which are affected become 432 a rotting mass. Lesions become water-soaked and shaped irregular on the pod. 433

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The pod consists of small and shriveled seeds or a lack of seed in the infected plants.

In the infected seeds, grayish-white mycelium is observed (Narayanasamy 2011).

436 Pathogenesis

The spore of *Botrytis cinerea* germinates after 6–8 hours of inoculation. The fungus 437 B. cinerea being a necrotrophic organism grows saprophytically on the leaf. The germ 438 tube develops and forms a mycelial connection on the leaf. The tip of the germ tube 439 forms appressorium, necessary for penetrating the plant cells. The pathogen pene-440 trates the host system through the cuticle of leaf and resides and formation of myce-441 lium at subcuticular or subepidermal layer. The penetration through stomata has been 442 observed in the spore of Botrytis cinerea which germinate after 6-8 hours of inocula-443 tion. The germ tube develops and forms a mycelial connection on the leaf. After estab-444 lishing itself at subcuticular or subepidermal position, the hyphae grow and reach to 445 mesophyll cells. The hyphae thicken and start branching at the mesophyll layer, con-446 sequently damaging mesophyll and epidermal cells. The degradation of the two layers 447 requires cell wall enzymes such as pectinases, cutinases, cellulases, and polygalactu-448 ronases. As the pathogen cannot degrade lignin, it does not affect the lignified cells 449 like xylem and tracheary elements. The degradations of mesophyll cells occur after 450 72–96 hours of inoculation. The total necrosis of the leaf takes place after 120 hours 451 of inoculation, and characteristic vellowing of the leaf is observed (Arranz et al. 452 2000). The reactive oxygen species (ROS) can be generated by B. cinerea during its 453 metabolic processes or with the help of NADPH oxidases (NOX). The NOX is a pro-454 tein of muti-subunit and can reduce superoxide anion from oxygen. The BcNoxA and 455 BcNoxB are catalytic subunits of NOX; BcNoxA helps pathogens to colonize on host 456 tissues, whereas BcNoxB is necessary for primary infection. Apart from these two 457 subunits, another regulatory subunit BcNoxR is responsible for the growth, sporula-458 tion, and increased virulence of the pathogen (Hua et al. 2018). Cell wall enzymes are 459 necessary for degrading the structural polysaccharides of the host cells. Cutinases are 460 responsible for degrading cuticles and cellulases for cellulose. Endo-β-1,4-xylanases 461 and pectin methylesterases found in the cell wall are necessary for degrading xylan 462 and dimethyl esterification of cell wall components like polygalacturonase, respec-463 tively, and therefore endorse the pathogen into its entry to host environment. Two 464 endo-polygalacturonases, BcPG1 and BcPG2, are required for virulence of the patho-465 gen. Both BcPG1 and BcPG2 are necessary for primary infection, while BcPG2 is 466 also involved in lesion expansion (Ten Have et al. 2010). 467

468 **10.10 Management**

469 10.10.1 Host-Plant Resistance

Host-plant resistance can be termed as the adaptation taken from different herbivores or pathogens for improvement in reproduction and sensitivity. Plants are sensitive; they produce several allelochemicals (secondary metabolites) which have

been used by the plant to inhibit the growth, behavior, and survival of different 473 pathogens (Pande et al. 2006). Pathogen inhibition can be also triggered by hyper-474 sensitivity (HR), reinforcement of cell wall by deposition of lignin, callose glyco-475 protein which is rich in hydroxyproline, polyphenols or cinnamic acid, etc. against 476 leaf cuticle thickening parasite by epithelium thickening, which provides a mechan-477 ical barrier. In the case of a disease like Ascochyta blight, resistance is also induced 478 by increasing the respiration rate and carbohydrate content of second days after 479 inoculation (DAI). It has resulted in a hypersensitivity response. Second DAI gives 480 resistance to ILC 32792 genotype by hypersensitivity response. Rather than hyper-481 sensitivity response, metabolic compounds like phytoalexin are involved in the 482 exertion of defense mechanisms toward photogenic fungi. It had been found that 483 when the crude culture filtrate (CCF) of the strain A. rabiei was applied, accumula-484 tion of medicarpin (phytoalexin) is increased in the culture. Accumulation of phe-485 nolic compounds like formononetin and biochanin A also helps in inducing plant 486 defense. Studies show that defense-related enzyme like hydrolytic enzymes and 487 phenylpropanoid pathway's enzymes also has their role in plant defense. 488 Accumulation of β -1,3-glucanase and peroxidase in the cell wall causes the hydro-489 lyzing of the cell of fungi. Ascochyta blight disease can be controlled by inducing 490 HPR (host-plant resistance) (Waliyar et al. 2016). In the case of Ascochyta blight, 491 there are several screening methods used in field and greenhouse conditions. 492 Screening in chickpea germplasm by HPR shows a high level of resistance against 493 BGM, by using this HPR, advanced chickpea breeding lines Australia evaluates 494 BGM resistance germ lines. These lines equally give resistance against Ascochyta 495 blight (AB) (Kumar et al. 2018). 496

10.10.2 Seed Treatment

In countries like Australia, Canada, Iran, the USA, etc., Ascochyta blight in chick-498 pea had been reported due to infected seed which results in the low seed weight and 499 discoloration. In the case of chickpea, blight-free seed productions are widely used 500 in disease management (Sharma and Ghosh 2016). The selection of larger-sized 501 seeds against smaller ones reduces the chances of blight disease as small-size chick-502 pea seeds have a higher level of Ascochyta infections. Seed immersion in the hot 503 water and chemicals like CuSO₄ solution, thiram, malachite green, etc. are used to 504 treat chickpea. Again fungicide dressing in the seeds of chickpea improves the resis-505 tance as it halts the spore germinations and mycelial growth on the surface of the 506 seed (Singh and Reddy 1996). But due to several factors like soil characteristics, 507 weather condition, and plant growth inhibition, it is found that blight disease is not 508 prevented against the phytotoxicity of fungicides which give adverse effect on seed 509 germination. It has been reported that treating chickpea using thiram, tridemorph, 510 imazalil, etc. causes the loss of vigor and hence is not practiced widely (Mohammed 511 et al. 2017). 512

513 10.10.3 Culture Control Method

The main concept of disease management is to produce pathogen-free seed. 514 Different practices like erect cultivars, manipulating in showing dates, etc. help in 515 reducing different foliar diseases. Late sowing lowers the vegetative growth and 516 thus reduces the disease incidence. To allow more aeration, wider row spacing is 517 practiced in the crop field, and it reduces leaf wetness, relative humidity, etc. Thus 518 it helps in the reduction of disease occurrence in plants. Another practice in the 519 plants with compact and erect growth also helps in reducing diseases than that 520 incuse of bushy spreading. Bushy spreading happened because of low aeration. By 521 practicing all the above, we can reduce the disease incidence in chickpea (Heydari 522 and Pessarakli 2010). 523

524 10.10.4 Cut-Twig Method

In the cut-twig method, test genotypes are grown in a plastic bag (45/30/5 cm) 525 which is filled with vermiculites (4:1) and sterilized sand and placed in a glasshouse 526 at 25 ± 2 °C with susceptible check H208/JG 62 used for artificial inoculums. 527 10–15-cm-long tender shoot of chickpea plant was cut with a sharp edge blade in 528 the evening. It is transferred to the test tube by wrapping the course portion with a 529 cotton plug containing fresh tap water. It inoculates in a test tube by the susceptible 530 check (G543 or H208 OR L3.0). The symptoms start to appear 24 hours, and after 531 6 days, 100% mortality of susceptible lines can be seen (Udall and Wendel 2006). 532

10.10.5 Resistance Sources and Studies on Disease Management

In reducing the control of Ascochyta blight, foliar spray of chlorothalonil and benomyl was used for increasing seed height and yield (Bretag et al. 2008). In Australia, they used thiabendazole and thiram for treating the chickpea seed which increases the yield by up to 20%. Complete resistance was seen in using inoculation of pregerminated seed and in the seed coat (1995). Benomyl or sulfur is used for spraying the foliages (Hagedorn 1996).

In Australia, the host plants which are resistant used in the industries are the best 541 for various conditions or option for controlling these diseases. Some of them use the 542 pathogen-free seed to break off at least 3 years between chickpea crops in the same 543 field. They keep it at least 500 m away from last year's crop in delaying sowing to 544 applying fungicide sprayed many times. Crop management practices where empha-545 sized to decrease or to reduce the damage occurred due to diseases. Pathogenicity is 546 the step of pyramiding resistance genes into genetic makeup. The key component of 547 disease management is host resistance. Fungicide dressings help to prevent the 548

spore germination and to eradicate the fungus from the seed coat. Another method 549 used is the crop rotation which helps in controlling the diseases (Salam et al. 2011). 550

10.10.6 Breeding for Disease Resistance

In single plant progenies and advanced breeding lines, they use field screening tech-552 niques and growth room for segregation. The deoxyribonucleic acid marker will 553 encourage using an exotic source of disease resistance. ICRISAT has seen the 554 growth of AB resistance lines in desi-type chickpea. From the diverse source, mul-555 tiple crosses are produced to accumulate resistance gene (Serraj et al. 2003). 556 Conventional breeding method NIFA-88 has been developed with the application of 557 propineb, zineb, ferbam, etc. This method helps to reduce the secondary spread of 558 AB in crops (Sarmah et al. 2012). 559

10.10.7 Biological Control

Studies show that the strains like Trichoderma harzianum Rifai and Trichoderma 561 viride give antagonistic effect on the B. cinerea. The growth of B. cinerea on the 562 hyphal tips is inhabited by T. viride species. Spraying of T. viride on the seeds helps 563 in the germination of the seeds. The T15 strain of Trichoderma species is used as an 564 effective biocontrol agent. T. viride and vinclozolin are found to be more effective 565 with the application of fungicides. To produce artificial resistance, it is treated with 566 T. viride and Gliocladium roseum (Monte 2001). This application is equivalent to 567 that of seed treated with thiram. Compounds like essential oil production in the 568 plants also reduce the infection of *B. cinerea* from 90% to 80%. These essential oils 569 include cinnamon oil, clove oil, etc. The essential oil effect is studied by an auto-570 matic microtiter plate. Bacterial species like Thymus zygis and Cymbopogon mar-571 tini help in the production of essential oil which is antagonistic against B. cinerea 572 (Wilson et al. 1997). Different techniques are involved in the study of growth inhibi-573 tion of fungi, and this includes the production of glyoxalate which helps to combat 574 different diseases. The biological control of foliar disease also helps in disease man-575 agement without applying chemicals to the crop field (Shamsi and Khatun 2016). 576

10.10.8 Resistance Sources and Disease Management

In reducing the control of Ascochyta blight, foliar spray of chlorothalonil and benomyl was used for increasing seed height and yield (Bretag et al. 2008). In Australia, they use thiabendazole and thiram for treating the chickpea seed which increases the yield by up to 20%. Complete resistance was seen in using inoculation of pre-germinated seed and in the seed coat. Benomyl or sulfur is used for spraying the

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foliages. In Australia, the industries use host-plant resistance as the best long-term 583 administration for diseases. Some of them use the pathogen-free seed to break off a 584 minimum of 3 years before sowing chickpea crops in the same field. They keep a 585 distance of 500 m from last year's crop in delaying the sowing to applying fungicide 586 spray for several times (Pande et al. 2005). Crop management practices were 587 emphasized to minimize the damage caused by these diseases. Pathogenicity is also 588 the method of pyramiding resistance genes into genetic materials. The key compo-589 nent of disease management is host resistance. Fungicide dressings help to prevent 590 the spore germination and to remove the fungal infections from the seed coat. 591 Another method used is the crop rotation which helps in controlling the diseases 592 (Johansen et al. 2008). 593

594 10.10.9 The Genetic Basis of Host-Pathogen Interaction

In the case of BGM, the gene control resistance was reported in 1985. In this, par-595 ents F1 and F2 and their backcross generation BC1 and BC2 screening for resis-596 tance against BGM under epiphytotic condition. A single dominant gene Bor1 gives 597 resistance to ICC 1069. The cross of ICC 1069 with BGM 413 and BGM 256 gives 598 the ratio like 13 resistances is to 1 susceptible plant. It shows that the two epistatic 599 interaction genes control resistance. Different studies on resistant varieties like ICC 600 1069, P 349, NEC 2451 and 2 susceptible genotypes JG 62 and T3 in India and 601 Australia produced BGM resistance cross. The resistance in the entire three parents 602 is controlled only by one single dominant gene. The F2 produces 15 resistances in 603 1 susceptible plant (Leroux et al. 2002). 604

605 10.10.10 Gene Plant Technology

Gene technology nowadays is used for crop/plant improvement. In the case of 606 chickpea, gene plant technology is used to treat diseases infected by both AB and 607 BGM. Production of antifungal metabolites by expressing different genes is one 608 such kind of gene plant technology. Different antifungal proteins and hydrolytic 609 enzymes like chitinase are also accumulated by gene plant technology which 610 degrades the cell wall of fungi. In the case of kiwi fruit, the production of β -1,3-611 glucanase reduced symptoms of B. cinerea infection. In the case of alfalfa ferritin, 612 an iron-binding protein is also produced which gives protection against oxidative 613 damage of necrotic pathogen. The transgenic plant which consists of 614 polygalacturonase-inhibiting protein (PGIP) gives resistance against B. cinerea. 615 The PGIP works against the PG that is secreted by the pathogen against the plant 616 cell wall. This PGIP is isolated from raspberry and kiwi fruit which is introduced in 617 different plants by gene plant technology. QTL mapping is used to study Ascochyta 618 blight disease in pea plants (Sagi et al. 2017). 619

10.10.11 Integrated Disease Management (IDM)

Integrated disease management is the technique that manages the disease and miti-621 gates yield at the same time. It involves the cultivation of pathogen-tolerant geno-622 type, application of diammonium phosphate in soil and of Carbendezim or Thiram 623 in seeds, and wider row spacing (0.6 m) against foliar diseases like Ascochyta blight 624 and BGM. It is reported that ICCL873 22 genotypes were controlled by chemicals 625 of BGM, wider row spacing is used, and T. viride is sprayed on the genotype (Pande 626 et al. 2006). The Nepal Agricultural Research Council (NARC) and Natural 627 Resources Institute (NRI), UK, reported the increase of health by 400% after the 628 IDM program (Pande et al. 2006). 629

10.10.12 Field and Control Environment Screening for Disease Resistance

Different techniques for screening are developed at different research centers for 632 chickpea, and it gives artificial resistance against foliar diseases like Ascochyta 633 blight. The field screening and control environment screening are two major 634 screening methods standardized by the ICRISAT (International Crops Research 635 Institute for the Semi-Arid Tropics) and ICAR (Bidinger et al. 2009) against 636 AB. This involves the planting of test material in a 40 cm row space. It also involves 637 independent cultivation that serves as the indicator or spirit line. In a cloudy day, the 638 spores are incubated in the plants at flowering time, and infected debris are spread 639 between rows. Again these inoculates are integrated during the dry weather for 640 approximately 15 days. In these plants, no visible lesions are found. Again, in the 641 environmental screening, air temperature is maintained at 20 ± 1 °C, 12 hours of 642 photoperiod, etc. (Landa et al. 2001). 643

10.11 Conclusion

Chickpea is a quantitative source of carbohydrates, proteins, minerals, vitamins, 645 and fibers. Chickpea also fixes atmospheric nitrogen and reduces the need for nitro-646 gen fertilizers. The crops are affected by serious foliar diseases, which affect the 647 development stages. Botrytis gray mold and Ascochyta blight are among the most 648 prominent diseases of chickpea. New and suitable understanding of the science, 649 ecology, distribution, symptoms, epidemiology, pathogenesis, economic impor-650 tance, and integrated management or control measures of the major foliar fungal 651 diseases of chickpea is studied or focused on this chapter. The foliar disease has 652 restricted chickpea production in many countries; therefore integrated management 653 or control strategies are needed to be adopted to prevent loss of crop and pulses. 654 Investigation of the pathogen's genetic basis of host-pathogen interaction and 655

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identification of the host-plant resistance will help in improving or breeding a resis-656 tant variety of chickpea and will be useful to farmers and researchers. Damage 657 caused by fungal foliar diseases can be reduced by using moderate integrated resis-658 tant cultivars with the strategies of agronomic management practices. The manage-659 ment practice will result in a better resistance for the host plant and will lead to 660 greater opportunities for sustainable agriculture and maximum productivity. 661 Agronomic options are added to management to decrease the damage which is 662 caused by the pathogen. 663

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Chapter 11 Management of *Fusarium udum* Causing Wilt of Pigeon Pea

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11.1 Introduction

Pigeon pea is an important source of protein and vitamin, and it is the second most 7 edible legume crop after chickpea and contributes about 90% production of the total 8 world production in India (Allen and Lenné 1998; Dhanasekar et al. 2010). Its pro-9 tein and essential amino acid content makes it an important food in a vegetarian 10 diet, with its seed and pod husk being the sources of feed (Varshney et al. 2010). In 11 addition to protein and amino acid, it also contains carbohydrates, minerals, and 12 fibers. Its plantation covered 4.3 million hectares globally (Anonymous 2007). In 13 India pigeon pea production and productivity are 2.76 metric tons and 762 kg/ha, 14 respectively, coming from an area of about 3.63 million hectare (the Year 2010, 15 ICAR Vision 2030/2010). Thirty-two species belong to the genus Cajanus, and 16 most of them are found in India and Australia, whereas only one species is native 17 from West Africa. Pigeon pea can be grown under drought conditions with signifi-18 cant return and minimum input. In India pigeon pea productivity is low due to the 19 lack of new cultivars and infection by plant pathogens (Nene et al. 1996). It is culti-20 vated with a minimum input of fertilizers and disease management strategies. 21 Pigeon pea production is affected by many biotic and abiotic stresses. Under biotic 22 stress, several pathogens such as fungi bacteria, viruses, nematodes, and 23

B. P. Singh et al. (eds.), *Management of Fungal Pathogens in Pulses*, Fungal Biology, https://doi.org/10.1007/978-3-030-35947-8_11 1

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mycoplasma-like organisms are responsible for the lower yield of pigeon pea (Nene 24 et al. 1989; Kannaiyan et al. 1984). Some important diseases responsible for legume 25 crop loss include fusarium wilt, sterility mosaic, phytophthora blight, macrophom-26 ina root rot, alternaria leaf spot, and cercospora leaf spot caused by *Fusarium udum*, 27 viruses, Phytophthora drechsleri f. sp. cajani, Macrophomina phaseolina, Alternaria 28 tenuissima, and Cercospora cajani, respectively (Kannaiyan et al. 1984). These dis-29 eases and other abiotic factors such as low moisture stress, waterlogging, and salt 30 stress are responsible for a significant reduction in yield of pigeon pea (Varshney 31 et al. 2007; Saxena 2008). The diseases fusarium wilt and sterility mosaic are eco-32 nomically important in our country. Fusarium wilt is a very severe disease, causing 33 yield loss of about US \$71 million annually in India. Wilt is a soilborne disease that 34 affects the yield of crop significantly especially in wilt-susceptible cultivars (Reddy 35 et al. 1990). Fusarium udum is soil inhabitant in nature and enters the vascular sys-36 tem of the plant through the root system. Because of the soilborne nature of wilt 37 disease, management through cultural practices is very difficult at a significant 38 level. Some chemical fungicides are effectively managing this disease, but the 39 extreme use of chemicals is harmful and noneconomical. Biocontrol strategies are 40 also in use through several antagonistic microorganisms for managing this disease 41 (Chaudhary and Kumar 1999). Many fungal and bacterial commercial products are 42 also developed for soilborne pathogen management (Kumar and Sarma 2016; 43 Kumar et al. 2017). Use of these biocontrol antagonistic microorganisms and their 44 commercial product in plant disease management is economical and risk-free con-45 cerning health hazards. In this chapter, we have discussed all the management strat-46 egies from conventional to advanced molecular technologies for wilt disease of 47 pigeon pea. 48

49 **11.2 History**

In 1809, Link was the first scientist to narrate about the genus Fusarium - the patho-50 gen with fusiform, nonseptate spores borne on a stroma. Later, a detailed account of 51 Fusarium species and pigeon pea wilt was first reported by Butler (1906). In India, 52 this destructive fungus was first described in 1906 by E.J. Butler in the pigeon pea 53 crop from Bihar and hence named as Fusarium udum Butler and later reported in 54 several other countries in Africa, South Asia, and Europe (Karimi et al. 2012). Then, 55 F. udum was established as a new species by Butler (1910), and isolation and iden-56 tification of the fungus were carried out. Previously, F. oxysporum f. sp. udum was 57 used frequently. Extensive characterization of fusarium-plant interaction in the 58 prospect of its biochemistry and physiology has been already done; however, recog-59 nition of vital molecules involved in the pathogenesis of Fusarium sp. did not start 60 till convenient molecular genetic techniques for filamentous fungi were available 61 (Timberlake and Marshall 1989; Datta and Lal 2013). Due to the soilborne nature of 62 the pathogen, chemical control is ineffective in many established cases, and manag-63 ing the disease seems to be very challenging. However, deployment of resistant 64

varieties is unlikely because of its high degree of genetic variability among the 65 pathogenic population (Kumar and Upadhyay 2014). At the present scenario, three 66 fungicides commonly used for the management of fusarium wilt are thiram, beno-67 myl, and bavistin (Vidhyasekaran et al. 1997; Meena et al. 2002; Melent'ev et al. 68 2006). Moreover, microorganisms producing various types of mycolytic enzymes 69 (chitinases, glucanase, and proteases) have shown a substantial impact on disease 70 development as they can degrade chitin and glucan present in the fungal cell wall 71 (Deshpande 1999; Hillocks et al. 2000; Hoster et al. 2005; Patel et al. 2007). 72

11.3 Distribution

Worldwide, pigeon pea wilt causes considerable devastation to the production of 74 pigeon pea (Kannaiyan et al. 1984). At crop blooming and maturity stages, 30-60% 75 of disease incidence has been recorded; on the other hand, yield losses may increase 76 up to 100% when susceptible cultivars were used (Okiror 2002; Dhar et al. 2005). It 77 is extensively occurring in India, Malawi, and East Africa leading to more than 50% 78 yield losses, and despite these, countries like Indonesia, Mauritius, Bangladesh, 79 Grenada, Myanmar, Venezuela, Trinidad, Nevis, Nepal, and Tobago are well-known 80 for incidence of Fusarium udum (Reddy et al. 2012; Marley and Hillocks 1996). In 81 the Indian context, this disease was reported in most of the pigeon pea-growing 82 states and caused about US\$ 71 million annual production losses (Reddy et al. 83 2012) except in southern states. However, the heavy incidence was reported in 84 Vidharbha (13.66%) followed by the Marathwada region where maximum severity 85 recorded up to 90% in the state of Maharashtra (Shinde et al. 2014). In other states 86 like Bihar, Jharkhand, Orissa, and West Bengal, fusarium wilt was effectively found 87 with a substantial range of cultural, morphological, and pathogenic variability in 88 maximum isolates collected from pigeon pea-growing regions (Kumar and 89 Upadhyay 2014). Mesapogu et al. (2012) have reported genetic diversity and patho-90 genic variability among 30 isolates of Fusarium udum collected from diverse agro-91 climatic conditions representing 7 states of India, i.e., Andhra Pradesh, Uttar 92 Pradesh, Jharkhand, West Bengal, Haryana, Rajasthan, and Punjab. 93

11.4 Symptoms

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The disease can be diagnosed by visualizing the gradual or sudden wilting of the pigeon pea plant. Similarly, the leaves show interveinal clearing followed by withering, yellowing, and drying of young leaves on the upper portion of the plant. Wilted plant loss their tugidity because off chlorisis and necrosis resulting in premature leaf drop and dropping of apical shoot followed by drying of entire shoot (Upadhyay and Rai 1992). As the pathogen survives in the soil and the nature of the infection is soilborne, it will infect the tap root system of pigeon pea plants resulting in wilting 101

of the whole plant instead of partial wilting. If the stem of infected plants is split
open, browning of vascular tissue mainly the xylem is the most common visible
symptom which differentiates it from other diseases. The wilting symptoms are the
most common and prominent during the flowering and pod maturation stages
(Reddy et al. 1990). Another visible symptom is purple banding, which extends
upward from the base of the plants and is easily seen on the stem portion. Purple
banding helps in differentiating healthy and infected plants (Sharma et al. 2016).

109 11.5 Disease Development and Pathogenicity

Fusarium wilt of pigeon pea is both a soilborne and seed-borne disease in which the 110 infection level of untreated seeds may range from 13% to 19% (Kannaiyan et al. 111 1984). The infected seeds thus serve as a primary vehicle for the spread of this dis-112 ease over long distances and/or to the newer areas. The pathogen, *Fusarium udum*, 113 survives in the soil for more than 3 years on the infected plant detritus. The disease 114 incidence and disease severity are principally dependent on the conditions of soil 115 and the genotype of the crop. The incidence of disease in susceptible cultivars is 116 facilitated by a slightly acidic to slightly alkaline soil having sand particles more 117 than half percentage in their soil texture (Singh and Hussain 1964; Upadhyay 1979). 118 A soil temperature of about 20-29 °C and soil moisture of about 6-16% are most 119 suitable for the development of wilt disease in pigeon pea (Upadhyay 1979). As per 120 the reports, disease incidence among different soils depends chiefly on the survival 121 and saprophytic activity of the pathogen in those soils that are ultimately favored by 122 the availability of the host substrate. The severity of the disease is dependent on the 123 duration of the pigeon pea varieties as very short-duration varieties suffer less than 124 the long-duration and medium-duration varieties. Growing of susceptible pigeon 125 pea varieties over the infested soils repeatedly increases the disease severity and 126 disease incidence. 127

Earlier the wilt of pigeon pea was known to be caused only by the imperfect state 128 of the pathogen (Fusarium udum), but the discovery of its perfect state, i.e., 129 Gibberella indica (Upadhayay and Rai 1983), is known to occur through both the 130 stages. As the perfect state is not known to be present frequently under natural con-131 ditions, the imperfect state is most common to incur the disease. In both the states, 132 the pathogen is known to grow externally and internally through the production of 133 a mycelial mass and conidia on the host's surface, majorly on the collar region and 134 roots (Upadhyay and Rai 1982). After the surface colonization, the fungal hyphae 135 invade the fine branches of roots that grow laterally and continue to proliferate in the 136 vessels of xylem. Even though the infection may take place in the seedling stage of 137 the plant, but the expression of disease is maximum during flowering and the pod-138 ding stage of plants (Reddy et al. 1998), which can be due to the longer time required 139 by the pathogen for colonization in the plants. It takes approximately about 140 3-4 months for the fungus to cause wilting in the infected plants which are when the 141 basal half of the main stem is colonized by the pathogen (Reddy et al. 1998). This 142

is the reason that can be understood as to why the short-duration crops have low 143 levels of wilt infestation when compared to long-duration crops as the former ones 144 are escaping the wilt incidence. 145

Once the infected plants wilt and die, the pathogen continues to live and survive 146 as a saprophyte for many years, mainly on the dead plant parts in its perfect form 147 (Upadhayay and Rai 1983) or imperfect form (Nene et al. 1980). Both the states of 148 the fungus survive simultaneously on the host plant. In addition to the confinement 149 of pathogen survival mainly on the dead roots and debris of infected plants, it may 150 survive on the other organic matter for a limited period. Apart from these, the fungus 151 Fusarium udum also survives on other fungi in the soil as mycoparasite as well as 152 on the bodies of termites that feed on the wilted host roots (Upadhyay and Rai 1982, 153 1983). The chlamydospores are also known to be formed in both the phases of the 154 fungus, i.e., the parasitic and the saprophytic phases, depending on the environmen-155 tal conditions from the hypha and the conidia (Sinha 1975). The fungus has been 156 also observed to produce a large number of dark violet perithecia on the exposed 157 roots and collar region of the host plant which also serves as resting structures. 158 These Fusarium udum perithecia produce ascospores in large numbers which 159 remain physiologically inactive in the soil for a limited period and after which they 160 produce either conidia or somatic hyphae on germination leading to infection of the 161 pigeon pea plants (Rai and Upadhyay 1982). 162

In recent years, many of the studies on morphological, cultural characterization 163 and the rate of reaction of the pathogen Fusarium udum have provided enough evi-164 dence for the existence of different virulence groups (Harlapur et al. 2007; Mahesh 165 et al. 2010; Karimi et al. 2010). The variable reactions of various tested resistant 166 pigeon pea varieties show the possibility of the presence of different physiological 167 forms of the pathogen (Muhammad et al. 2011). In a study, Reddy et al. (1998) 168 reported three strains of the pathogen which showed sensitivity/or resistance against 169 several pigeon pea differentials. 170

11.6 Mechanism of Host Plant Resistance

The employment and use of resistant varieties of the crop is the most economical, 172 effective, and eco-friendly strategy for the control of diseases even though their 173 response to the cultivating conditions will be a subject of concern (Saxena et al. 174 2012). To come up with a sound breeding program for the development of disease-175 resistant crop varieties, we need to understand the mechanism of host plant resis-176 tance and what mechanism to strengthen up in plants to restrict pathogen invasion. 177 There are mainly two mechanisms that constitute host plant resistance, viz., consti-178 tutive and induced defense mechanisms. The constitutive resistance mechanisms 179 contain all the preformed chemical factors and physical barriers that are present in 180 the host plant in advance to the attack of phytopathogens (Dangl and Jones 2001). 181 The physical barriers consist of the thick and/or hard cuticle, wax deposition in the 182 epidermal cells, stomatal shape and size, and the pericycle of the root (Keen 1992). 183

The chemical factors of the constitutive defense mechanism consist of peptides, proteins, protein inhibitors, preformed secondary metabolites, alkaloids, phenols, phytoanticipins, etc., which add up to the early barriers of defense being a part of plant's natural growth and development (Heath 2000; Dixon 2001; Grayer and Kokubun 2001). The plants are also reported to exudate some fungi toxic substances that restrict and/or inhibit the spore germination of the phytopathogen (Agrios 2004).

The induced defense mechanisms are the ones which get triggered on after the 190 attack of phytopathogen and involve both chemical and physical factors (Agrios 191 2004). The most important step of induced defense mechanism is the recognition of 192 the phytopathogen by the host plant so that it can conjure the defense reactions 193 (Dixon et al. 1994; Schenk et al. 2000). The process of reaction starts with the rec-194 ognition of the molecular pattern of the pathogen and is termed as pathogen-195 associated molecular patterns (PAMP) (Nürnberger and Lipka 2005). This 196 recognition of the pathogen leads to signal transduction involving a cascade of bio-197 chemical events which leads to incitation of defense responses (Keen 1992; Dixon 198 et al. 1994; Baron and Zambryski 1995). The most frequent defense response is the 199 hypersensitive response (De Wit 1992) which is a form of programmed cell death 200 (Greenberg and Yao 2004). The hypersensitive reaction restricts the growth of the 201 fungus to newer plant cells (Tomiyama 1982; Keen 1992; Schenk et al. 2000). In 202 addition to this, the other induced reactions include rapid oxidative burst, ion fluxes, 203 and strengthening of the cell wall by increased synthesis of cellulose, lignin, pheno-204 lic compounds, and hydroxyproline-rich glycoproteins (Bowels 1990; Agrios 2004). 205 The rapid oxidative burst is mainly through the production of hydroxyl radical 206 (OH), hydrogen peroxide (H_2O_2) , and superoxide (O_2^-) , and these reactive oxygen 207 species impart cross-linkage of the proteins present in the cell wall of the plant 208 resistant to fungal enzyme attack (Bradley et al. 1992; Keen 1999). These reactive 209 oxygen species are also known to induce hypersensitive cell death while working as 210 an agent in the cell signaling process (Levine et al. 1994; Alvarez et al. 1998). 211

There are other defense mechanisms which constitute in host plant resistance, 212 and it comprises of production of vascular occlusions such as tyloses and gels (Mace 213 1963) and defense-related gene expression involving the production of suberin and 214 lignin, signal transduction proteins, phytoalexins, and pathogenesis-related proteins 215 (Reymond and Farmer 1998; Greenberg and Yao 2004). The production of the sig-216 naling compounds in the host plant after the recognition of the phytopathogen attack 217 leads to the enactment of defense reactions systemically throughout the plant and is 218 termed systemic resistance (Ryals et al. 1994). 219

220 11.7 Management of Fusarium Wilt Disease

There are different methods for the control and management of *Fusarium udum* followed in agricultural technology with its positive and negative impacts. For complete resistance, single, race-specific resistance genes (R genes) could be used. For incomplete resistance, a bunch of minor genes work together for broad-spectrum.

Complete management of fungal disease is difficult due to lack of knowledge 225 regarding plant-pathogen interaction at genetic, histological, and molecular levels. 226 Thus, to protect pigeon pea from *Fusarium* in a sustainable way, it is necessary to 227 build a novel and potential approach by investigating the existing technologies. 228 Some of the important control methods are discussed here. 229

11.7.1 Cultural Management

For the formation of barrier in pigeon pea against fusarium wilt, numerous cultural 231 practices are used. Among them, crop rotation is one of the best control measures. 232 Crops like tobacco (Nicotiana tabacum L.), sorghum (Sorghum bicolor (L.) 233 Moench), or castor (*Ricinus communis* L.) are rotated with pigeon pea for 3 years to 234 wipe out the pathogen completely from the field. To reduce the infestation percent-235 age below 20%, cultivation of the main crop could be followed with a year break 236 with sorghum, or the land could be left fallow. The application of farmyard manure 237 or Crotalaria juncea as green manure also reduces the incidence of wilt to a signifi-238 cant level (Ingole et al. 2005). Another method is reducing *Fusarium* inoculums 239 from the field by solarization technique during the summer season (Reddy et al. 240 2012). Intercropping of sorghum with pigeon pea reduces incidences to 24% as 241 compared to the sole crop which gets 85% incidence (Natarajan et al. 1985). Mixed 242 cropping of *Crotalaria medicaginea* also has a positive impact on reducing wilt 243 (Upadhyay and Rai 1981). 244

11.7.2 Chemical Management

Chemical management is one of the most effective and common measures. An 246 equivalent mixture of benomyl and thiram is used for seed treatment and considered 247 effective (Reddy et al. 2012). Use of biocontrol agent like formulation of 248 Trichoderma viride and farmyard manure (2 kg and 125 kg, respectively) for one 249 square measure is also found to be very successful in reducing fusarium wilt 250 (Perchedpied and Pitrat 2004). Addition of mineral in the soil like boron (Bo), zinc 251 (Zn), manganese (Mn), and methyl bromide (CH₃Br) diminishes the disease event 252 of fusarium wilt (Maisuria et al. 2008). For effective management of this disease, 253 antibiotics like bulbiformin and griseofulvin have also been accounted. 254

11.7.3 Biological Management

As chemicals lead to undesirable and harmful effects on various living entities, 256 moreover it also causes an imbalance in the ecosystem. Thus, it creates a need for a 257 healthy control measure. The use of biological agents is thus a significant measure 258

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as it is a member of the ecosystem and a potential antagonist to pathogens. According 259 to a few reports, addition of antagonists in the soil diminishes the Fusarium udum 260 incidence (Maisuria et al. 2008; Bapat and Shar 2000; Singh et al. 2002; Anjaiah 261 et al. 2003). Various rhizobacteria as biocontrol agents are used for its management 262 (Siddiqui 2006; Siddiqui and Shakeel 2007; Pusey 1989; Bapat and Shar 2000; 263 Siddiqui et al. 2005). The addition of T. harzianum provides disease control of 264 22-61.5% at all pathogen levels (Prasad et al. 2002). According to reports popula-265 tion of F. udum is drastically reduced by antagonism of Aspergillus terreus, 266 Aspergillus niger, Micromonospora globosa, and Aspergillus flavus (Upadhyay and 267 Rai 1981) in a biocontrol experiment. In naturally infested soil, the addition of 268 Pseudomonas aeruginosa PAN1 significantly suppresses the incidence of Fusarium 269 in pigeon pea and chickpea (Anjaiah et al. 2003). A graphical representation of 270 direct and indirect mechanisms of biocontrol is presented in Fig. 11.1. 271

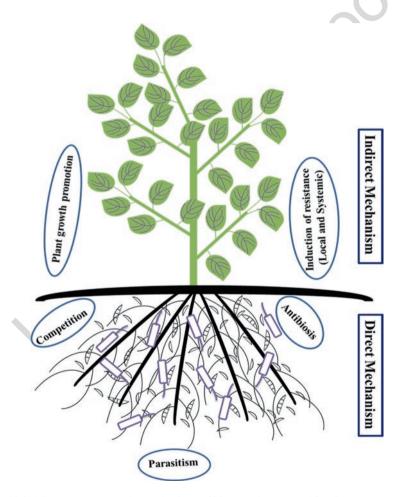


Fig. 11.1 Diagram represents the mechanisms of biocontrol agent used for disease management

11.7.4 Transcriptomics Approaches

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Plant receptor protein recognizes the pathogen-derived molecule which is the ini-273 tial step in defense response by activation of signal transduction cascades which 274 triggers expression of various plant defense genes (Barilli et al. 2014). The study 275 of gene expression provides a detailed knowledge regarding genes which were dif-276 ferentially expressed and various metabolic conduits at the time of host-pathogen 277 interfaces. It can jointly help to unveil candidate resistant genes collaborating in 278 every step of plant defense response (Ichinose et al. 2001). In the era of molecular 279 plant breeding, marker-assisted selection (MAS) could be highly useful by apply-280 ing the knowledge of the defense-responsive genes in legumes against fungal 281 pathogen attack to legume plants, and under transformation event, any change in 282 expression of such candidate genes could be linked with improved resistance. 283 There are certain techniques used in transcriptomics like enhancing the potential 284 number of defense-related genes by generating cDNA (complementary DNA) 285 libraries from plants under stress against pathogens inoculation or elicitor-treated 286 tissues or cells. The second one is the application of macro- or microarray designed 287 by using orthologue sequences from other legumes in the format of unigenes, 288 cDNA, expressed sequence tags (ESTs), or resistance gene analogs (RGAs) in the 289 query legumes like pigeon pea under specific fungal stress conditions. These meth-290 ods help to identify transcripts that are induced under pathogenic attacks and 291 majorly associated with candidate resistant genes with a certain level of expres-292 sion. Transcriptomics also helps to explore the information of genome sequence 293 information with the aid of new less expensive sequencing platforms (Illumina 294 (Solexa) sequencing, Roche 454 sequencing, Ion Torrent (Proton/PGM sequenc-295 ing), and SOLiD sequencing). NGS technologies decrease the complexity of tran-296 scriptome techniques like SSH, cDNA-AFLP, SuperSAGE (serial analysis of gene 297 expression), or MPSS (massive parallel signature sequencing), thereby increasing 298 the identified transcript amount devoid of cloning and Sanger sequencing. Now, 299 RNAseq technique allows building de novo transcriptomics that generates the tran-300 sition of the transcript in expression form of both plant host and the inoculated 301 fungal pathogen for examining plant-pathogen interactions, in addition to its basic 302 work of studying all expressed transcript's sequencing at that particular time 303 (Tadege et al. 2009). With the help of transcriptome profiling techniques, numerous 304 diverse expressed genes population across the genome can be easily generated 305 under pathogen attack. It is difficult to differentiate such a transcript associated 306 with defense response and resistant phenotypes. This can be resolved by studying 307 their co-localization with quantitative trait loci (QTLs) and exploring their func-308 tional analysis. Different advanced molecular techniques like gene silencing via 309 RNA interference (RNAi) and virus-induced gene silencing (VIGS) are also used 310 nowadays for knowing functional activities of PR proteins and biotic stress-induced 311 genes (Tadege et al. 2009). A generalized presentation of phases showing the 312 involvement of transcription factor in the induction of systemic acquired resistance 313 against pathogen stress is presented in Fig. 11.2. 314

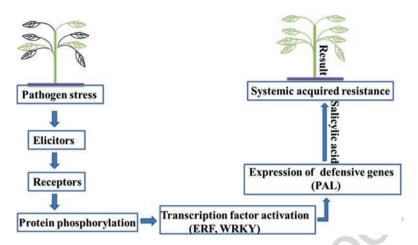


Fig. 11.2 A generalized presentation of phases showing involvement of transcription factor in induction of systemic acquired resistance against pathogen stress. Protein phosphorylation occurs early with the recognition of pathogen elicitor by host receptor. Further transcription factor activation induces expression of defense genes such as PAL. Salicylic acid biosynthesis and defense gene activate systemic acquired resistance during plant pathogen interaction

315 11.7.5 Proteomics Approaches

Protein expression and its functional activity rely on the extent of expression of 316 genes and posttranscriptional and posttranslational regulations. Therefore there 317 could be a large chance that all transcripts derived from the successful expression of 318 mRNA do not form successful protein accumulation and function. Thus, it is also 319 significant to study protein accumulation to get a clear picture of the mechanisms of 320 plant-pathogen interaction. Recent proteomic technologies provide opportunities 321 for large-scale protein profiling via quantitative and qualitative methods (Qin et al. 322 2013). In comparative proteomics, protein is separated by electrophoresis based on 323 their mass and isoelectric points followed by spectrometry techniques based on pro-324 tein identification like de novo sequencing or peptide mass fingerprinting. Another 325 technique is a separation of chromatography-based peptide mixtures continuing 326 their detection through mass spectrometry (Nautrup-Pedersen et al. 2010) and shot-327 gun proteomics which analyzes direct tandem mass spectrometric analysis that 328 includes chromatographic separation based on cell lysis (Qin et al. 2013). All these 329 techniques are practiced in legume particularly in the establishment of subcellular 330 localization of target proteins, thus forming reference protein maps (Salavati et al. 331 2012). But, in legumes after pathogen attack, the study of proteomics is quiet far 332 lacking behind as compared to other molecular advancements. But there is an exam-333 ple of a proteome study in chickpea – Fusarium oxysporum (Bourgeois et al. 2011). 334 To detect protein variation under biotic stresses, comparative proteomic approaches 335 are highly significant. Thus, there is a huge expectation from proteomic techniques 336 that might unveil endogenous elements that provide resistance to fungal diseases. 337

11.8 Conclusion

The use of resistant variety is the most effective way to restrict the incidence of a 339 disease. At present in the molecular biology and biotechnology era, it is possible to 340 know about the genes, enzymes, proteins, and transcription factors that show a 341 highly active defense response against pathogen attack. The study of resistances 342 sources (Genes, protein etc.) can be beneficial for developing resistace in crop plant. 343 For this purpose the current biotechnological and molecular biology techniques pro-344 vide knowledge on transcription factors to detect stress-responsive genes of the 345 plant. Further proteomics and genomics information is mandatory to know all cel-346 lular processes under stress response for better crop improvement. 347

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Chapter 12 Role of Biofertilizer in Biological Management of Fungal Diseases of Pigeon Pea [(*Cajanus cajan*) (L.) Millsp.]

Surbhi Gupta, Nidhi Didwania, and Srinivasa Nagappa Chowluru

12.1 Introduction

The world population is increasing at a high growth rate and is expected to reach 7 ~9.6 billion in 2050 according to a recent United Nations report (UNPAN 2010). 8 With a projected emphasis on sustainable genetic improvement of major staple 9 crops including rice, wheat and maize, it is also important to lay light on the produc-10 tion of protein-rich foods to reduce global malnutrition and hunger. Proteins are the 11 foremost building block of the human system. It is a known fact that developing 12 countries have only 33% of the normal requirement of protein, hence making it a 13 challenge for various nutritional development programs to fulfil the protein demand. 14

Leguminous plants (legumes or pulses) are one of the best available protein 15 sources that can contribute a handful amount of proteins in the diet of developing 16 countries as they require minimum care during cultivation and low inputs. Pigeon 17 pea or red gram (Cajanus cajan (L.) Millsp.) occupies a chief place in worldwide 18 agriculture among different legume crops (Saxena et al. 2010). It occupies 5.4 mil-19 lion hectares in 22 countries in the continents of Asia and Africa. Out of this India 20 alone has more than 3.9 million hectares, i.e. 72% of the area, of all the pigeon pea-21 growing countries of the world (FAOSTAT 2018). Uttar Pradesh is the largest pro-22 ducer of pigeon pea in India, but the average yield released by the crop is much less 23 than its other neighbouring states like Bihar and Jharkhand (Ahlawat et al. 2005; 24 Prasad et al. 2017). 25

Pigeon pea (*Cajanus cajan* (L.) Millsp.) is the most vital legume crop in the 26 world. India is one of the largest producers of pigeon pea commonly known as 27 "arhar" in its northern part followed by the eastern side of Africa and Central 28

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B. P. Singh et al. (eds.), *Management of Fungal Pathogens in Pulses*, Fungal Biology, https://doi.org/10.1007/978-3-030-35947-8_12 6

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America. It is roughly cultivated in at least 25 tropical and sub-tropical countries.

This crop is greatly influenced by weather conditions; it is well raised in semi-arid

- tropical areas which are rain-fed. Cropping of pigeon pea is intermixed with maize, sorghum, pearl millet and some other legume crops like groundnut etc. It supplements
- 33 soil through nitrogen fixation.

The term "biofertilizers" refers to live microbial culture, which when applied to 34 plants, soil or composting pits helps in mobilization of various nutrients by their 35 biological activity. Application of biofertilizers such as plant growth-promoting 36 rhizobacteria (PGPR) and arbuscular mycorrhizal fungi (AMF) in agricultural field 37 soils is well known. Assessment of native microbial field community is indispens-38 able for developing tracing tools to monitor the introduced biofertilizers. Pigeon pea 39 is affected by almost 60 plant pathogens comprising of bacteria, nematodes, fungi, 40 viruses, etc., but luckily, only a few of them are of economic importance. Out of 41 which, it is withered by numerous fungal diseases, viz. fusarium wilt, Phytophthora 42 blight, Phoma stem canker, Alternaria blight and Macrophomina root rot. 43

44 12.2 Some Major Fungal Diseases of Pigeon Pea

Diseases of economic importance in the country are fusarium wilt caused by 45 Fusarium udum Butler, Phytophthora blight caused by Phytophthora drechsleri 46 Tucker f. sp. cajani, Macrophomina root rot caused by Macrophomina phaseolina 47 (Tassi) Goid., stem canker caused by Phoma cajani (Rangel) and Alternaria blight 48 caused by Alternaria sp. Fusarium wilt caused by Fusarium udum Butler – a soil- as 49 well as seed-borne fungus spreads through wind, water and soil and can survive up 50 to 3 years on infected plant debris and is of great economic importance (Shinde et al. 51 2014). Symptoms of the disease appear during flowering when the plant is just 52 1-2 months old. Likewise, Phytophthora blight another fungal disease caused by 53 Phytophthora drechsleri Tucker f. sp. cajani is a common infection of Cajanus cajan 54 (L.) Millsp. (Pande et al. 2011). It is a soilborne fungus and thus is fast spreading, 55 surviving as dormant mycelia and chlamydospores in the soil. It is greatly affected 56 by the weather. Rainy season favours the growth of the fungus. The spores of the 57 fungus are spread through air and water. Warm and humid weather after the infection 58 has occurred is a serious concern as it damages the plant and facilitates infection. 59 Phoma stem canker of pigeon pea caused by Phoma cajani is one of the emerging 60 diseases of the crop. The symptoms of the disease first appear on the stems as a 61 necrotic spot and later turn into canker, resulting in the wilting of the whole plant. 62 Macrophomina root rot is also among one of the important fungal infections of 63 Cajanus cajan (L.) Millsp. caused by Macrophomina phaseolina (Tassi) Goid. 64 This disease along with Alternaria blight caused by Alternaria alternata is a major 65 problem for late-sown crops. Both these diseases are greatly affected by the weather. 66 They are more prominent in hot and humid season. Under these conditions, root rot 67 spreads to the base of the stem. The lesions further coalesce and cause the branches 68 and then the entire plant to dry up and die. 69

12.3 Management of Disease

12.3.1 Cultural Management

Cultural practices are the traditional practices used by farmers to overcome diseases 72 caused by pathogens in the crop. The commonly used practices include crop 73 rotation, intercropping, interrow spacing, removal of diseased plant, spraying of 74 nitrogen, etc. Verma and Rai in 2006 reported crop rotation with Sorghum bicolour 75 (L.) Moench (sorghum), Nicotiana tabacum L. (tobacco) or Ricinus communis 76 L. (castor) every 3 years terminates the pathogen from the field. They also stated 77 that growing sorghum or fallow for 1 year on the same field of pigeon pea reduces 78 the incidence of wilt disease up to below 20%. The spray of green manure with 79 Crotalaria juncea reduces rot and wilt diseases to a great extent (Upadhyay and Rai 80 1981). The application of nitrogen as farmyard manure has also been found to be 81 effective. One of the common and effective practices to control the diseases of 82 pigeon pea is intercropping. Growing of other crops like sorghum or black gram as 83 intercrop has proved to be effective (Table 12.1). 84

12.3.2 Chemical Management

Chemical management involves the treatment of the disease through chemical sprays. Numerous chemicals have been suggested for the management of fungal diseases of pigeon pea for long (Singh 1998). Pigeon pea seeds when treated with 88

Disease	Common cultural practice
Fusarium wilt	• A field with no previous record (up to 3 years) of fusarium wilt should be selected
	 Seeds used should be collected from disease-free fields of pigeon pea The intercropping pattern is preferred Rotation of 3 years and mixed cereal crops like sorghum, tobacco, etc. is beneficial Solarization of soil in summer is also encouraged to reduce disease incidence
Phytophthora blight	 Field with no previous disease record is preferred Sowing of seeds should be avoided in waterlogging areas like the low-lying patch Good drainage should be ensured through raised seedbeds Interrow spacing also proves to be helpful
Dry root rot	 Field with no previous disease record is preferred Late sowing of seeds should be avoided to reduce the risk of high temperature and drought conditions
Phoma stem canker	 Field with no previous disease record is preferred Infected plants should be removed subsequently to reduce the spread of infection
Alternaria blight	Seeds used for sowing should be taken from healthy fieldsAvoid late sowing of the crop

 Table 12.1
 Cultural practices for disease control against some major fungal diseases

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Disease	Chemical practice
Fusarium wilt	• Seed bacterization with Benlate and thiram in 1:1 (3 g per kg of seed)
Phytophthora blight	• Foliar spray at 15 days interval with Ridomil MZ (2 sprays)
Dry root rot	Dressing of seeds with tolclofosmethyl or thiram
Alternaria blight	Foliar spray with Indofil M45

 Table 12.2
 Chemical practices for disease control fungal diseases

an equal part mixture of benomyl and thiram eradicate the disease (ICRISAT 1987; Reddy et al. 1993). Supplementing soil with boron, manganese or zinc and methyl bromide (CH₃Br) reduces the incidence of fusarium wilt. Ingole et al. (2005) also reported similar findings with a mixture of carbendazim + thiophanate (0.15 + 0.10%)against wilt disease of pigeon pea. Few antibiotics like bulbiformin have also found to be an effective tool against pathogens (Table 12.2).

95 12.3.3 Biological Management

The application of hazardous fungicides affects the environment in adverse ways, 96 and moreover, chemical fertilizers are not targeted specifically. It not only degrades 97 the ecosystem but also has negative effects on human health. Fungicides affect the 98 food chain as they are toxic to species like earthworms and microorganisms and also 99 to an extent affect genotoxicity of humans (Shuping and Eloff 2017). They cause 100 water and soil pollution too. The solution to this above problem lies in sustainable 101 agriculture. The application of potential microorganisms which are part of the exist-102 ing ecosystem serves as an effective means against plant protection system. Biological 103 management of diseases has been reported by several workers and serves as an 104 attractive tool for eco-friendly management of soilborne as well as other pathogens 105 degrading the crop. Disease incidence of fusarium wilt has been reduced by the 106 application of antagonistic microorganisms like fungi and bacteria (Passari et al. 107 2017; Anjaiah et al. 2003; Mandhare and Suryawanshi 2005; Maisuria et al. 2008; 108 Singh et al. 2002). Out of cluster of scientific reports, few of them have notable bio-109 logical measures that are functional for the management of pigeon pea diseases. Seed 110 inoculation with rhizosphere bacteria like Bacillus subtilis, Pseudomonas fluores-111 cens and Pseudomonas aeruginosa is very effective against fungal disease of pigeon 112 pea (Mahesh et al. 2010). Integrated management strategies (IDM) which involve a 113 combination of fungicides and biocontrol agents also prove to be beneficial for the 114 management of Fusarium udum Butler (Pande et al. 2012). Oil formulations of 115 Trichoderma strains like Trichoderma harzianum reduce the traces of soilborne 116 pathogens from the diseased plants (Khan and Khan 2002). Siddiqui and Shakeel 117 (2007) suggested that various rhizobacteria are efficient biocontrol agents. Plant 118 extracts like neem and eucalyptus, garlic and henna, ginger and tulsi are also found 119 to have an inhibitory effect against Alternaria blight of pigeon pea (Rathore 120 et al. 2018). 121

12.4 **Biocontrol Agents**

The property of microorganisms to fight against phytopathogens is termed as a form 123 of biological control (Duffy and Defago 2009). This approach is eco-friendly, much 124 effective as well as cost-efficient. These PGPRs produce antifungal metabolites, cre-125 ating competition for nutrients that act as chief modes of biocontrol activity (Duffy 126 and Defago 2009). Rhizobacteria produce some antifungal metabolites like HCN, 127 phenazines, pyoluteorin and tensin which kill the fungal pathogen (Bhattacharyya 128 and Jha 2012). Bacillus spp. (Gong et al. 2006) and Pseudomonas (Leonardo et al. 129 2006) are two PGPRs that have been reported being effective biocontrol agents. 130 Among these bacterial species, Bacillus subtilis, Bacillus amyloliquefaciens and 131 Bacillus cereus are the most effective ones for controlling plant diseases through 132 various mechanisms (Passari et al. 2016a; Francis et al. 2010). PGPRs like Bacillus 133 spp. and *Pseudomonas* spp. have this ability to make endospores which allows them 134 to sustain in a wide range of environmental conditions and hence make them efficient 135 biofertilizers (Perez-Garcia et al. 2011). Application of T. harzianum, T. viride, 136 B. subtilis and P. fluorescens when mixed with neem or karanj cake and compost not 137 only reduces the diseases but also enhances the longevity of biocontrol agents 138 (Narayanan et al. 2015; Shanmugapackiam et al. 2016). 139 140

Application of biocontrol agents can be done in three forms:

- 1. By application of fungi
- 2. By application of AMF
- 3. By application of bacteria

By Application of Fungi 12.4.1

Trichoderma sp. secretes secondary metabolites which are antifungal and hence has 145 great potential to act as biocontrol agents. They reduce the fungal pathogen either 146 directly by mycoparasitism or through indirect mechanisms like competition for nutri-147 ents and space to survive and modifications of environmental conditions. They help in 148 the promotion of plant growth and also activate the defence mechanism of the plant. 149 Whipps and Lumsden (2001) stated that species of *Trichoderma* have been widely 150 accepted as biocontrol agents against numerous phytopathogens. Trichoderma spe-151 cies are useful virulent saprophytes that act as biocontrol agents against phytopatho-152 genic fungi by various mechanisms such as rhizosphere competition, mycoparasitism 153 and antibiotic and enzyme production and induce resistance. Growth promotion activ-154 ity of Trichoderma has also been reported (Cumagun 2012; Harman et al. 2004). 155 Strains of Trichoderma (T. viride, T. harzianum, T. virens) were evaluated under field 156 conditions against Fusarium udum; out of which T. viride was found to be most prom-157 ising at 15% concentration (Chaudhary et al. 2017). The inoculation of seeds with 158 antagonists helps in externally managing seed and soilborne pathogens. Talc-based 159 formulation of Trichoderma sp. has been used to coat seeds. 160

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161 12.4.2 By Application of AMF

AMF or arbuscular mycorrhizal fungi are the groups of fungi that act as promising 162 biofertilizers. Dumas-Gaudot et al. (2000), Garmendia et al. (2005) and Garcia-163 Garrido (2009), in their respective studies, reported that AMF-mediated bioprotec-164 tion is accepted as a key practice for disease control. AMF is currently exploited for 165 its anti-pathogenic properties. Linderman (2000) reported that induced systematic 166 resistance or ISF is the mechanism behind AMF phytoprotection. This mechanism 167 concentrates more on nutritional changes like competition with infection sites, 168 changes in the morphology of root and shoot tissues, abiotic stress reduction and 169 changes in the mycorrhizosphere and chemicals, constituting changes in plant tis-170 sues (Hause and Fester 2005). All these properties make AMF a good biofertilizer 171 also in the coming future. 172

173 12.4.3 By Application of Bacteria

Plant growth-promoting bacteria are the bacteria present in rhizospheric soil which 174 enhance the growth of the plant directly or indirectly. The awareness of PGPR is 175 increasing steadily in the world. They are applied to several economically important 176 crops to increase the yield of the crop by enhancing the growth of the plant and 177 protecting it from different pathogens. PGPR promotes plant growth by procure-178 ment of minerals like phosphorous, nitrogen, etc. directly from the soil (Gyaneshwar 179 et al. 1998) and also indirectly by acting against plant pathogens as a biocontrol 180 agent. Several reports suggest an increment in the quality and the number of differ-181 ent crops worldwide through the application of PGPRs under normal as well as 182 stressed conditions (Passari et al. 2019). The application of PGPR is encouraged 183 because it reduces the dependence on hazardous chemical fertilizers for improving 184 plant growth and helps in reducing plant pathogens, which destabilizes the 185 agriculture system. PGPR exhibits positive effect on the germination of the seeds, 186 the yield of the crop and their tolerance towards stresses like drought and salt 187 (Passari et al. 2019; Brown 1974). PGPR is an effective antagonist against plant 188 pathogens like Fusarium udum and Macrophomina phaseolina. Soil microbe's 189 interaction with the rhizosphere plays an important role in solubilizing and mobiliz-190 ing a limited amount of nutrients available and also their uptake by the plant (Bolton 191 et al. 1993; Mantelin and Touraine 2004). PGPR has beneficial effects as a biocon-192 trol agent to important crops like legumes, cereals, fruits, vegetables, etc. According 193 to reports, the exact estimate is unknown, but an average of more than 50% of crop 194 losses in pigeon pea is due to pathogenic microorganisms (Rajash 2005). Thus, the 195 need of the hour is to exploit and enhance the efficacy of soilborne control agents 196 and use their best possible combination against plant pathogens (Mishra et al. 2016; 197 Chang et al. 2005). The encouragement for the use of PGPR as biofertilizers against 198 plant pathogens will serve as a promising alternative to deadly chemical fertilizers 199

and pesticides (Goldstein 1995). Screening of soil for bacterial antagonist against 200 pathogens is a notable biological advancement (Passari et al. 2016a; Karimi et al. 2012; Siddiqui et al. 2005), mostly for PGPR as a biocontrol agent (Siddiqui and 202 Shakeel 2007; Prasad et al. 2002). Inoculation of *Pseudomonas aeruginosa* in the 203 seed is effective against fusarium wilt disease of pigeon pea (Mahesh et al. 2010). 204

12.4.3.1 Modes of Action of PGPR

The mechanism of action of PGPR is not completely known; however, they are 206 reported to exhibit several beneficial activities for plant growth promotion (Khan 207 et al. 2009; Zaidi et al. 2009). PGPR promotes plant growth in two ways: directly 208 and indirectly (Glick 2012). Pigeon pea is the most staple and proteinaceous food 209 available in many developing countries; hence, it becomes important to protect this 210 crop from damage. Root-nodulating bacteria Sinorhizobium inhibited the growth of 211 fusarium wilt of pigeon pea as it possesses chitinase and β -gluconase production 212 (Kumar et al. 2010). Plant growth promotion takes place indirectly when PGPR 213 increases plant growth by decreasing the activity of plant pathogens (Xiang 214 et al. 2017). 215

12.4.3.1.1 Nitrogen Fixation

Nitrogen is a vital nutrient required for the growth and productivity of the plant. The 217 atmospheric N₂ is converted into plant-utilizable forms by biological N₂ fixation during 218 which nitrogen gets converted into ammonia, and this is done with the help of nitrogen 219 fixation bacteria present in the rhizospheric soil catalysed by nitrogenase enzyme (Kim 220 and Rees 1994). Biological nitrogen fixation, also known as BNF, usually takes place 221 at mild temperatures, by widely spread nitrogen-fixing bacteria (Raymond et al. 2004). 222 This provides an economically beneficial and environmentally friendly alternative to 223 chemical fertilizers (Ladha et al. 1997). Nitrogen-fixing bacteria (symbiotic bacteria) 224 show symbiosis with plants belonging to leguminosae family like rhizobia (Ahemad 225 and Khan 2011; Zahran 2001) However, non-symbiotic nitrogen-fixing bacteria 226 provide only a small amount of the fixed nitrogen that bacterially associated host 227 plant requires (Glick 2012). 228

12.4.3.1.2 Phosphate Solubilization

After nitrogen, phosphorus is the second most vital nutrient required for plant230growth. This is also abundantly available both in an organic and inorganic form in231the soil (Khan et al. 2009). The low availability of phosphorous to the plants is due232to its presence in the insoluble form which plants are not able to absorb (Bhattacharyya233and Jha 2012). The only soluble form of phosphorous available for the use of plants is234monobasic and dibasic (Jha and Saraf 2015). To fulfil the phosphorous requirement,235

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phosphatic fertilizers are given as a supplement in the fields. As plants do not absorb
the full amount of applied fertilizer, the rest gets converted into insoluble complexes
in the soil (McKenzie and Roberts 1990). This practice not only affects the environment but is also not cost-effective. Hence finding a better reliable solution to this
problem is necessary. PGPR has coupled with phosphate solubilizing activity which
may provide the available phosphorous to the plants in a much eco-friendly way
(Khan et al. 2006).

243 12.4.3.1.3 Siderophore Production

Iron is a prominent nutrient available for all lives possible on earth. It is needed byall living beings.

In properly aerated soils, iron in the form Fe³⁺ (ferric iron), which is easily precipi-246 tated as iron oxide, is absorbed by plants (Duffy 1994). This property of microbes to 247 secrete siderophores makes them suitable biocontrol agents as they induce competition 248 for iron availability in the rhizosphere, hence restricting the proliferation of fungal 249 phytopathogens in the vicinity of the crop, because of less availability of iron. CAS or 250 chrome azurol agar media is used to isolate siderophore-producing bacteria. Rajkumar 251 et al. (2008) have reported the growth of the plant through siderophore, because of the 252 siderophore-producing bacteria in the rhizosphere. 253

254 12.4.3.1.4 Phytohormone Production

Microbes are known to synthesise phytohormones like auxins or IAA, i.e. indole 255 acetic acid, for a long time. About 80% of the microbes isolated from the rhizo-256 sphere, of many crops, secrete secondary metabolites like auxins (Patten and Glick 257 1996). Indole acetic acid has a prominent function in bacteria-plant interactions 258 (Passari et al. 2016a, b; Spaepen and Vanderleyden 2011). It is also reported that 259 IAA has a plant defence mechanism against plant pathogens, and it produces a sig-260 nalling effect to reduce the IAA production by the plant pathogen (Spaepen and 261 Vanderleyden 2011). 262

263 12.5 Microbial Consortium

Most applications of biocontrol of plant diseases use single biocontrol agents as the antagonist against plant pathogens. The microbial consortium works well as, biopesticides, against a wide spectrum of plant pathogens which is a little difficult to be fulfilled using a single biocontrol agent. Biocontrol agents individually or in consortium attack pathogens through antagonism effect. They act better and more effectively when combined and when belonging to the same ecosystem. Vital and future promising candidates of the microbial consortium are *Trichoderma* sp., *Pseudomonas* sp. and Bacillus sp. Seed bacterization with a consortium of Rhizobium and 271 Pseudomonas putida, P. fluorescens and Bacillus increased yield and biomass of 272 pigeon pea crop (Tilak et al. 2006). Trichoderma sp. in association with AMF has 273 great potential against plant pathogens (Wehner et al. 2010). The consortium of bio-274 organic (municipal waste) and applied organic (Rhizobium sp.) showed prominent 275 improvement in the growth of pigeon pea over control plant (Rizwan and Mahmood 276 2017). Didwania et al. (2019) have also reported integrated management for 277 Alternaria blight in oil-yielding crops. 278

12.6 Biotechnological Approaches to Biological Management 279

The detailed information on biotechnological techniques and genetics is important 280 for developing a mechanism against susceptible varieties. Numerous resistant theo-281 ries are known against fusarium wilt, and hence a single dominant gene has been 282 established (Owuoche and Silim 2010; Kotresh et al. 2006). Many well-characterized 283 or little-known genes, earlier reported being involved in legume crops, defend 284 against fungal infection in pigeon pea. Resistant varieties available in the market 285 against Phytophthora blight are Hy 4, ICPL 150, ICPL 288, ICPL 304, KPBR 286 80-1-4 and KPBR 80-2-1 (ICAR database). Out of 80 entries evaluated under sick 287 plot, 18 entries WRP-1, BDN-2004-1, MAHABEJ, BRG-14-2, PT-257, BRG-14-1, 288 MA-13, BWR-133, GRG-160, IPA8F, KA-12-03, ICPL-87119, KPL-44, KPL-43, 289 BSMR571, BSMR-846, BSMR-579 and BSMR-2 have showed moderate resistant 290 reaction with 0.00-10.00 per cent disease incidence. Similarly, Mishra and Dhar 291 (2005) reported the same findings in vitro. Prasanthi et al. (2009) have reported a 292 disease score of zero in treated and untreated pots of genotype ICP 8863, in pot 293 culture screening technique against fusarium wilt-resistant/fusarium wilt-susceptible 294 genotypes. IVT-520, IVT-509 and AVT-603 were found to be resistant against pod 295 bug damage among 29 genotypes screened (Singh et al. 2017). 296

12.7 Conclusion

With the increasing population of the world, the demand for staple food like 298 legumes, which are rich in protein, would also increase. Hence measures are 299 required to fulfil the demand of the crop. 300

Decades ago the green revolution happened which increased the agriculture supply globally. This revolution saved the then population from hunger and malnutrition but, in turn, also triggered the use of chemical fertilizer. These chemical fertilizers are very harmful to our environment as they enter the food chain. So it is the need of the hour that we adapt better means to improve the quality as well as quantity of the crop but keeping in mind the environment safety also. Biofertilizers are an excellent solution to this problem of chemical fertilizers. Biofertilizers help 307

- in the improvement of plant growth and also act as biocontrol agents. They are eco-
- 309 friendly and cost-effective means for crop improvement. Their use will serve as an
- instrument to ensure productivity and stability which will lead us to perfect agricul-
- tural practices in the world. A combination of biotechnological approaches with
- microbial consortium can contribute to go a long way in fighting with fungal dis-
- eases of pigeon pea and also to increase the yield.

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