

Management of Blight in Chickpea Caused by *Ascochyta rabiei*: A Review

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ABSTRACT

Chickpea is the world's second-largest pulse crop and ascochyta blight caused by *Ascochyta rabiei* is the most destructive disease which occurs in all its growing areas. Particularly during the flowering and podding stages, it causes enormous economic losses. Infected seed and crop residue play an important role in the survival of pathogen from time to time. All the above-ground plant parts get affected in form of necrotic lesions, which girdle the stem in susceptible cultivars and lead to reduced yield even under favourable conditions. *A. rabiei* pathogen is highly variable in its genotype making it very difficult to control. The available resistance sources are not enough and it is important to explore new sources since from time to time there has been a breakdown of resistance in existing chickpea varieties. This majorly occurs due to the ongoing evolution of new pathotypes. Thus, we have attempted to cover loss, disease distribution, symptoms, epidemiology, and disease control in this review.

KEY WORDS: EPIDEMIOLOGY, IPM, BIOLOGICAL CONTROL, FUNGICIDE, TRICHODERMA, DISEASE CYCLE.

INTRODUCTION

The causative agent of *Ascochyta* blight is *Ascochyta rabiei* Labrousse (Teleomorph: *Didymella rabiei* (Kovachevski) Arx.). It is one of the important foliar phytopathogens of chickpea. The main incident of this disease occurs in the region where the chickpea growing season coincides with cool and humid weather. This disease is regarded as calamitous and ubiquitous diseases in the chickpea growing regions causing severe crop losses. Due to the persistent incidence of this disease, the area of chickpea production in Western Canada has decreased from over

500,000 ha in 2001 to less than 130,000 ha in 2006 (Statistics Canada, 2001, 2007). When environmental conditions are ideal, the yield loss may exceed up to 100 per cent. This disease is a seed and stubble-borne disease which develops both airborne ascospores and water-splashed conidia during the cropping season (Armstrong et al., 2001; Gossen and Miller, 2004).

Development of chickpea may be restricted by *Ascochyta* blight worldwide as it is documented from all over the world (Nene and Reddy, 1987; ICARDA, 1996; Akem, 1999; Khan et al., 1999; Kaiser et al., 2000; Chongo et al., 2003). Some techniques, such as crop rotation, can contribute to the epidemic management of this disease. Many areas of the world, including Australia (Ackland et al., 1998; Knights and Siddique, 2002), Canada (Chongo and Gossen, 2001), Latin America (Kaiser et al., 2000), Southern Europe (Trapero-Casas and Jimenez-Díaz, 1986), the United States (Kaiser and Muehlbauer, 1984), are under a serious effect of this disease and suffer significant economic losses. The early symptoms include epinasty and loss of turgor, accompanied by water-

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soaked lesions followed by infection on petioles, leaflets, and young branches of chickpea. The phytopathogen additionally secretes a symptom-causing toxin. The disease spreads primarily by infected seeds developed from infected crops (Weltzien and Kaark, 1981).

The use of contaminated crop seeds was responsible for the introduction of this disease in Australia (Cother, 1977), Canada (Morrall and McKenzie, 1974), Iran (Kaiser, 1972), and the USA (Kaiser and Muehlbauer, 1984). Chickpea seeds with mild *A. rabiei* infections appear to be discoloured and have a very low seed weight. However, the pathogen occurs on the seed coat, cotyledons, and embryo axis during serious infection (Dey and Singh, 1994). The mycelium of *A. rabiei* develops on the seed coat of the infected seed (Luthra and Bedi, 1932), on cotyledons, (Lukashevich, 1958) and the emerging seedlings (Kaiser and Hannan, 1988). Pycnidium is the dormant survival structure of *A. rabiei* on soil surface debris (Galloway and MacLeod, 2003). It also can colonize naturally infested debris as a teleomorph and can form viable pseudothecia on the uninfected portions of the debris. Under favourable conditions, pycnidium develops conidia and serves as the primary inoculum for the initiation of the disease in the succeeding crop. The teleomorph, however, grows on chickpea debris present in the field and eventually produces ascospores for the long-distance spread of disease under favourable conditions (Navas-Corte's et al., 1998).

Geographical distribution: The following countries have been reported with the presence of this blight causing fungus: Algeria, Australia, Bangladesh, Bulgaria, Canada, Cyprus, Ethiopia, France, Greece, Hungary, India, Iran, Iraq, Israel, Italy, Jordan, Lebanon, Mexico, Morocco, Pakistan, Romania, Spain, Syria, Tanzania, Turkey, and Tunisia (Nene, 1980; Kaiser et al., 1998). In Iraq, Cyprus, Greece, Algeria, Bulgaria, Israel, Lebanon, Jordan, Morocco, Pakistan, Romania, Spain, Syria, Tunisia, Turkey and the USSR, the disease has been seen more to occur frequently and significantly (Nene, 1982).

Symptoms: The symptoms of this disease in different countries have been identified by several researchers which showed remarkable similarities. All the above-ground components of the plant are under attack by this phytopathogen. The lesions are circular or elongated on the leaflets, holding irregularly depressed brown dots and surrounded by a red or brownish border. The lesions are typically circular with dark margins on the green pods and have pycnidia formed in concentric circles. The affected seeds also bear lesions. The lesions on the stem and petiole are brown, elongated (3-4 cm), bear black dots, and sometimes girdle the affected portion. The section above the point of attack easily dies as lesions girdle the stem. The entire plant dies if the primary stem is girdled in the collar area. As the disease progresses, diseased plant patches become prevalent in the field and expand gradually, covering the entire area. (Atanasoff

and Kovacevski, 1929; Benlloch and Del Canizo, 1931; Labrousse, 1930; Luthra and Bedi, 1932).

Disease Cycle and Epidemiology: The pathogen overwinters in the residue and seed of infected chickpea crop. As the pathogen is easily transferred from the seed to seedlings, infected seeds play an important role, both in the introduction of *A. rabiei* to newer areas and also in the early development of the disease. The residue of crop may also serve as a source for the production of both asexual spores (conidia, spread by rain-splash) and sexual spores (ascospores, spread by wind). Sexual reproduction yields pseudothecia that house the ascospores in late fall and early spring. Under sufficient moisture and moderate temperatures (near 10°C), production of pseudothecia takes place during five to seven weeks. Mature pseudothecia release ascospores into the air in spring and early summer, that can move to several miles. The initial cause of infections in the spring is thought to be airborne ascospores, although the rain-splashed conidia are also considered to be involved (Bogdan, 2018). Infection and development of Ascochyta blight disease occur at a temperature frame of 5-30°C with an optimal of 20°C, which produce serious infections after 17 hrs of wetness. Dry phase (6-48 hrs) directly after inoculation often maximizes severity of the disease, but dry periods >12 h after an early wetting duration of 6 h typically provide a detrimental impact on the development of disease (Trapero-Casas and Kaiser, 1992a).

Management: Ascochyta blight management includes fungicide application, cultural management, and host resistance development. While, some chickpea cultivars have been reported to have genetic resistance to this pathogen, but the resistance is only partial and begins to break down during the plant's flowering period (Nene and Reddy, 1987; Chongo and Gossen, 2001). The successful control of this pathogen has been documented by a variety of fungicides, but the residual effects of these fungicides lead to contamination of the environment thus, affecting the natural world. The main key for effective chickpea cultivation and development against this disease is integrated disease management (IDM). One of the methods of minimizing the loss caused by Ascochyta blight is the treatment of chickpea seeds with effective chemical fungicides. Numerous chemical and physical methods for seed treatment and disease minimization, such as copper sulphate (Sattar, 1933), malachite green (Zachos, 1951), pimaricin (Zachos et al., 1963) and, hot water (Sattar, 1933) were applied from 1930 to early 1960. These seed treatments, however, were found to be generally less effective in controlling the seed-borne disease transmission.

The microbial community residing in the plants also gets badly affected due to the intensive use of fungicides. This

is well known because legumes are capable of fixing nitrogen (N) in the atmosphere (Kyei-Boahen et al., 2001; Gan et al., 2005). Symbiotic N-fixing microorganisms such as rhizobium live in close contact with the plants in the field. The fungicide application decreases the survival of rhizobium and affects the symbiosis between the plant root and the microorganism (Revellin et al., 1993; Kutcher et al., 2002). The rate of application of fungicides to the seed is the main factor affecting the viability of rhizobium as a biofertilizer added to the seed coat. The major cause of the decrease in the rhizobial population may be excessive use of fungicides which are recommended for seed treatment (Kyei-Boahen et al., 2001).

The length of incubation with fungicide can also affect the survival of rhizobium (Matus et al., 2003; Gan et al., 2005). The use of chemical pesticides for IDM should be limited, for the development of a technique with a combined use of pesticides and rhizobium (Welty et al., 1988). Before rhizobium inoculation, fungicides should be applied. Rhizobium and fungicides should be applied in a manner that helps to enhance the survival of seed coat rhizobium. The use of granular rhizobial inoculants may decrease the risk of fungicide physical contact, thereby raising symbiosis (Kyei-Boahen et al., 2002; Gan et al., 2005).

The use of biocontrol agents has become a safer choice for the management of this disease. Several researchers are currently working in this field to discover environmental friendly biocontrol agents having stronger capabilities against this phytopathogen. Few fungal antagonists such as *Chaetomium globosum*, *Trichoderma viride*, and *Acremonium implicatum* have been studied for their biological control activity, under in-vitro and in-vivo conditions. The mycelium was inundated by *A. implicatum* isolate-1 and caused its breakdown. A clear zone was formed by *A. implicatum* isolate-2. However, the mycelium of *A. rabiei* is covered by *C. globosum* and *T. viride*. A noticeable impediment to pycnidiospore germination and colony expansion of *A. rabiei* was observed while applying culture filtrates of antagonist microorganisms. Additionally, the culture filtrates of all three antagonists were found to be efficient in bringing a reduction in the production of disease under glasshouse condition. *C. Globosum* was found to be the most successful one with a disease index deduction of around 73.12 per cent (Rajakumar et al., 2005). Dugan et al. (2009) confirmed the natural occurrence of *Aureobasidium pullulans* in the post-harvest debris of chickpea. *A. pullulans* spore suspension spray onto chickpea debris contributes to a 38 per cent lower incidence of Ascochyta blight.

CONCLUSION

Ascochyta management is an important component of chickpea to grow successfully. A mixture of cultivar tolerance, seed and crop hygiene, seed and foliar fungicides, and suitable sowing dates are used for integrated disease management. Due to the complexity of the pathosystems and the inter-relationship with resistance and the environment, choosing the most successful strategies can be challenging. Therefore, a greater understanding of the factors that affect pathogen population survival and fitness and their study of diversity can help in the development of proper management practices. Further investigations are needed for a thorough study of the climatic factors responsible for the incidence and severity of this disease. The information presented in this review on the condition of ascochyta blight will be helpful for growers to prepare and implement strategies for management for reducing the blight below threshold levels.

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