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A review: Chilli anthracnose, its spread and management

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Abstract
Chilli anthracnose, caused by *Colletotrichum* spp, is one of the main causes for post-harvest decay of chilli. It can develop on the field, during long distant transport, cold storage and shelf-life. In conventional agriculture, the whole plant including the fruits, are sprayed with fungicides as a prerequisite for post-harvest control of chilli anthracnose. Due to consumer concerns regarding the use of synthetic fungicides and the demand for safer storage methods, the use of synthetic fungicides is no longer allowed for the post-harvest control of chilli anthracnose. As a result, studies on alternative methods to control post-harvest decay have been developed over the years along with the demand for safer storage methods. In this review, results published within the last decade have been summarized and alternative approaches to synthetic fungicides for post-harvest control of chilli anthracnose were discussed in detail. Overall, the use of natural antimicrobials, biocontrol agents, resistant cultivars and ozone shows promise as treatments that can be adopted on a commercial scale to control post-harvest chilli anthracnose caused by *Colletotrichum* species.

Keywords: bioagents, plant extract, variability, post harvest

Introduction
Chilli (*Capsicum annum* L.) is one of the most important constituent of the cuisines of tropical and subtropical countries and the fourth major crop cultivated globally. Around 400 different varieties of chilies are cultivated throughout the globe. The hottest variety being "Carolina Reaper" developed by a grower Ed Currie of West Indies having the maximum pungency of about 2.2Chilli (*Capsicum annum* L.) is one of the most important constituent of the cuisines of tropical and subtropical countries and the fourth major crop cultivated globally. Around 400 different varieties of chilies are cultivated throughout the globe. The hottest variety being "Carolina Reaper" developed by a grower Ed Currie of West Indies having the maximum pungency of about 2.2 million SHU (Scoville Heat Units; PuckerButt Pepper Company, 2013). One of the hot chilli varieties of the world "Naga Jalokia," is the native of Tezpur in Assam, India. Numerous varieties of chilli are grown for vegetables, spices, condiments, sauces, and pickles occupying an indispensable position in Indian diet. Apart from the explicit importance of the crop in the diet, chilli is also used in other forms like medicines and beverages and also as an ornamental plant in the gardens. Nutrition wise these are enriched with high Vitamin A and C content; high iron, potassium, and magnesium content with the ability to boost the immune system and lower the cholesterol levels (Grubben and Mohamed El, 2004). India has been a leading producer, consumer and exporter of chilli especially in dried form. Various varieties of the crop are found in India and its quality varies among the states of the country.

Host: Chilli
The genus *Capsicum* was originated in the American tropics and has been propagated throughout the world including the tropics, subtropics, and also temperate regions (Pickersgill, 1997) [49]. The fruit of *Capsicum* has a variety of names, such as 'chilli', 'chilli pepper' or 'pepper' depending on place (i.e., most serious destructive diseases of chilli (Isaac, 1992) [27]. Anthracnose disease caused by *Colletotrichum* species is one of the most economically important diseases reducing marketable yield from 10% to 80% of the crop production in some developing countries, particularly in Thailand (Poonpolgul and Kumphai, 2007) [50]. Anthracnose is mainly a problem on mature fruits, causing severe losses due to both pre- and post-harvest fruit decay (Hadden and Black, 1989; Bosland and Votava, 2003) [21, 9].

Anthracnose Disease
Anthracnose, derived from a Greek word meaning 'coal', is the common name for plant diseases characterized by very dark, sunken lesions, containing spores (Isaac, 1992) [27].

Generally, anthracnose disease is caused by *Colletotrichum* species which belongs to the Kingdom Fungi; Phylum Ascomycota, Class Sordariomycetes; Order Phyllachorales; and Family Phyllachoraceae. The anamorphs are *Glomerella* species. Anthracnose of chilli was first reported from New Jersey, USA, by Halsted (1890) [22] in 1890 who described the causal agents as *Gloeosporium piperitum* and *Colletotrichum nigrum*. These taxa were then considered as synonyms of *C. gloeosporioides.* Anthracnose causes extensive pre- and postharvest damage to chilli fruits causing anthracnose lesions. Even small anthracnose lesions on chilli fruits reduce their marketable value (Manandhar *et al*., 1995) [34]. Many post-harvest diseases of fruit exhibit the phenomenon of quiescence in which symptoms do not develop until the fruit ripens. *Colletotrichum* species are the most important pathogens that cause latent infection (Jeffries *et al*., 1990) [29]. Appressoria are known to form adhesive disks that adhere to plant surfaces and remain latent until physiological changes occur in fruits (Bailey and Jeger, 1992) [7]. Appressoria that formed on immature fruits may remain quiescent until onto genic changes occur in the fruits (Prusky and Plumbley, 1992) [52]. Anthracnose disease can occur on leaves, stems, and both pre- and post-harvest fruits (Isaac, 1992) [27]. Typical fruit symptoms are circular or angular sunken lesions, with concentric rings of acervuli that are often wet and produce pink to orange Anthracnose caused by *C. coccodes* does not result in severe epidemics on chilli fruits (Hong and Hwang, 1998) [23]. *C. gloeosporioides*, the predominant species on chilli in Korea, was differentiated into G and R strains by isozyme analysis of esterase, leucine amino peptidase, phosphatase and glutamine oxaloacetic transaminase (Park *et al*., 1987) [41]. *Colletotrichum* species can survive in and on seeds as acervuli and micro-sclerotia (Pernezny *et al*., 2003) [46]. Survival of mycelia and stomata in colonized chilli seeds had been reported (Manandhar *et al*., 1995) [34]. It has been shown that the pathogen readily colonizes the seed coat and peripheral layers of the endosperm even in moderately colonized seeds. Heavily colonized seeds had abundant inter- and intracellular mycelia and acervuli in the seed coat endosperm and embryo, showing disintegration of parenchymatous layers of the seed coat and depletion of food material in endosperm and embryo (Chitkara *et al*., 1990) [10].

Fungi can overwinter on alternative hosts such as other solanaceous or legume crops, plant debris and rotten fruits in the field (Pring *et al*., 1995) [51]. *Colletotrichum* species naturally produce micro-sclerotia to allow dormancy in the soil during the winter or when subjected to stressful condition, and these micro- sclerotia can survive for many years (Pring *et al*., 1995) [52]. During warm and wet periods, conidia from acervuli and micro-sclerotia are splashed by rain or irrigation water from diseased to healthy fruit and foliage. Diseased fruit acts as a source of inoculum, disease development (Roberts *et al*., 2001) [56]. *Colletotrichum* species utilize diverse strategies for invading host tissues, which vary from intracellular hemibiotrophic to subcuticular intramural necrotrophic (Bailey and Jeger, 1992) [7]. *Colletotrichum* species produce a series of specialized infection structures such as germ tubes, appressoria, intracellular hyphae, and secondary necrotrophic hyphae (Perfect *et al*., 1999) [45]. These pathogens infect plants by either colonizing subcuticular tissues intramurally or being established intracellularly. The pre infection stages of the both are very similar, in which conidia adhere to and germinate on the plant surface, producing germ tubes that form appressoria which in turn penetrate the cuticle directly (Bailey and Jeger, 1992) [7]. Following penetration, the pathogens that colonize the intramural region beneath the cuticle invade in an ectotrophic manner and spread rapidly throughout the tissues (O′Connell *et al*., 1985) [38]. There is no detectable biotrophic stage in this form of parasitism. In contrast, most anthracnose pathogens exhibit a biotropic infection strategy initially by colonizing the plasmalemma and cell wall intracellularly. After the biotrophic state, intracellular hyphae colonize one or two cells and subsequently produce secondary necrotrophic hyphae (Bailey and Jeger, 1992) [7]. These pathogens are therefore regarded as hemi biotrophs or facultative biotrophs (Kim *et al*., 2004) [31]. For example, *C. gloeosporioides* on avocado, chilli and citrus can produce both types of colonizations: intracellular biotrophy at an early stage and intramural necrotrophic later (O′Connell *et al.*, 2000) [39].

Although the mechanisms developed by *Colletotrichum* species appear similar in pre penetration events, there are differences between species in the later mechanisms such as spore adhesion, melanization and cutinization in penetration of the plant cuticle by the appressoria. For example, the host-pathogen interaction of *C. acutatum* appears to be more biotrophic than that of some other species such as *C. gloeosporioides* (Wharton and Diéguez-Uribeondo, 2004) [72]. Based on studies with *C. acutatum* on specific hosts, four types of interactions or infection strategies were described by Peres *et al*. (2005) [44] as follows:

(1) Biotrophic growth of *C. acutatum* with secondary conidiation in which conidia germinate to form appressoria and quiescent infections, and secondary conidia are formed after germination of the germ tube that cause anthracnose in chilli. There are still several questions to be answered.

Epidemiology and disease symptoms

Environmental factors play an important role in deciding the severity and spread of any disease. The favorable host pathogen and weather conditions lead to establishment of disease (Agrios, 2005) [3]. Thus, before proposing the management strategy of the disease, a thorough knowledge regarding the epidemiology of the disease should be studied. Anthracnose disease of chilli is generally most common among the tropical and sub-tropical countries. Hot and humid environmental conditions support the spread of the disease.

Other important environmental factors governing the severity of the disease include rainfall intensity and duration, humidity, leaf surface wetness and light. Amongst them leaf surface wetness has been directly linked with the severity of the disease owing to the better establishment of the pathogen in respect of germination, attachment and penetration into host tissues (Than *et al.,* 2008) [68]. The relationship between the environmental factors like rainfall intensity and duration and the prevailing temperature and humidity along with the crop geometry and inoculum spread leads to possible development of disease as well (Dodd *et al.,* 1992) [15]. Temperature also affects the development of the disease and presence of surface wetness and competitive microbiota further favors the disease development (Royle and Butler, 1986) [57]. Temperature around 27◦C with relative humidity of 80% have reported to be the most optimum conditions for successful establishment of the disease in a given area (Roberts *et al.,* 2001) [56]. The development of the disease also depends on the host cultivar, along with its resistance against the pathogen.

Causal agents of chilli anthracnose

In the *Colletotrichum* patho-system, different *Colletotrichum* species can be associated with anthracnose of the same host (Simmonds, 1965; Freeman *et al*., 1998) [61, 18]. *Colletotrichum* species causing anthracnose of chilli have been reported from different countries and regions (Table 1). Although these species have been the subject of numerous investigations, there remain many gaps in the knowledge of the disease process and understanding of the complex relationships between the species involved. Kim *et al*. (2004) [31] reported that different species cause diseases of different organs of the chilli plant; for example, *C. acutatum* and *C. gloeosporioides* infect chilli fruits at all developmental stages, but usually not the leaves or stems, which are mostly damaged by *C. coccodes* and *C. dematium*. Leaf anthracnose of chilli seedlings caused by *C. coccodes* was first reported in chilli growing in a field in Chungnam Province of Korea in 1988 (Hong and Hwang, 1998) [23]. Different *Colletotrichum* species may also play an important role in different diseases of mature stages of chilli fruit as well. For example, *C. capsici* is widespread in red chilli fruits, whereas *C. acutatum* and *C. gloeosporioides* have been reported to be more prevalent on both young and mature green fruits (Hong and Hwang, 1998; Kim *et al*., 1999) [23]. Allowing the disease to spread from plant to plant within the field (Roberts *et al*., 2001) [56]. Initial infection by *Colletotrichum* species involves a series of processes including the attachment of conidia to plant surfaces, germination of conidia, production of adhesive appressoria, penetration of plant epidermis, growth and colonization of plant tissue and production of acervuli and sporulation (Bailey and Jeger, 1992; Prusky *et al*., 2000) [7]. Anthracnose is mainly a problem on mature fruits, causing both pre- and post-harvest fruit decay resulting severe economic losses (Hadden and Black, 1989; Bosland and Votava, 2003) [21, 9]. Appressoria that formed on immature fruits may remain quiescent until the fruits mature or ripen.

Table 1: Reported causal agents of chilli anthracnose

S. No.	Country	Species associated	References
1.	Australia	*C. brisbanense*	Damm *et al*., 2009 [12]
2.	Brazil	*C. boninense*	Tozze and Massola, 2009 [69]
3	. India	*C. capsici*, *C. acutatum*	Ranathunge *et al*., 2012; Saxena *et al*., 2014 [54, 58]
4.	Indonesia	*C. acutatum*, *C. gloeosporioides*, *C. nymphaeae*, *C. capsici*	Damm *et al*., 2009; Voorrips *et al*., 2004 [12, 70]
5.	Korea	*C. acutatum*, *C. gloeosporioides*, *C. coccodes*, *C. dematium*	Park and Kim, 1992 [40]
6.	Mexico	*C. capsici*	Damm *et al*., 2009 [12]
7.	New Zealand	*C. kartsii*, *C. novae-zelandiae*, *C. nigrum*, *C. coccodes*	Damm *et al*., 2012b; Liu *et al*., 2013 [14, 32]
8.	Papua New Guinea	*C. capsici*, *C. gloeosporioides*	Pearson *et al*., 1984 [43]
9.	Sri Lanka	*C. acutatum*	Damm *et al*., 2012a [13]
10.	Taiwan	*C. acutatum*, *C. capsici*, *C. gloeosporioides*	Manandhar *et al*., 1995 [34]
11.	Thailand	*C. acutatum*, *C. capsici*, *C. gloeosporioides*, *C. siamense*, *C. scovillei*, *C. asianum*	Than *et al*., 2008; Damm *et al*., 2009; Phoulivong *et al*., 2012; Weir *et al*., 2012 [68, 12, 48, 71]
12.	United States	*C. capsici*, *C. gloeosporioides*, *C. acutatum*, *C. coccodes*,	Harp *et al*., 2008 [27]
13.	Vietnam	*C. acutatum*, *C. capsici*, *C. gloeosporioides*, *C. nigrum*	Don *et al*., 2007
14.	Zimbabwe	*C. nymphaeae*	Damm *et al*., 2009 [12]

Colletotrichum

Colletotrichum is one of the most important phytopathogens worldwide causing the economically important disease anthracnose in a wide range of hosts (Bailey and Jeger, 1992) [7]. The causal agent of chilli anthracnose disease is *Colletotrichum* which belongs to Kingdom-Fungi, Phylum-Ascomycota, Class-Sordariomycetes, Order-Phyllachorales, and Family-Phyllachoraceae. Causal agents of chilli anthracnose in different countries are tabulated in Table 1 (Than *et al.,* 2008) [68]. Kim *et al*. (2004) [31] reported that different species of *Colletotrichum* affect different organs of the chilli plant; for examples, *C. acutatum* and *C. gloeosporioides* infect chilli fruits at all developmental stages, but not usually the leaves or stems, which are mostly damaged by *C. coccodes* and *C. dematium*. Leaf anthracnose of chilli seedlings caused by *C. coccodes* was first reported in chilli growing in a field in Chungnam Province of Korea in 1988 (Hong and Hwang, 1988) [23].

Characterization of *colletotrichum* species

Morphological characterization

For effective disease management, accurate identification of *Colletotrichum* species is essential. Classically, identification and characterization of *Colletotrichum* species have primarily relied on morphological characters such as colony color, size and shape of conidia, optimal temperature for growth, growth rate, presence or absence of setae, and existence of the teleomorph, Glomerella (Freeman *et al*., 1998) [18]. Conidial morphology has been traditionally emphasized over other taxonomic criteria, although conidia of *Colletotrichum* are potentially variable. Several researchers reported that the growth rate of *C. gloeosporioides* was higher than that of *C. acutatum* (Agostini *et al*., 1992; Liyanage *et al*., 1992) [2, 33]. Adaskveg and Hartin (1997) [1] reported that, considering mycelial growth responses to temperature, *C. acutatum from* strawberry, almond, and peach grew well at 25°C while *C. gloeosporioides* from citrus and papaya grew well at 30°C. Table 2 shows the morphological data of *Colletotrichum* species (Sutton, 1992) [66].

Molecular Characterization

One of the most serious problems in chilli anthracnose is that two pathogens, *C. acutatum* and *C. gloeosporioides* cannot easily be differentiated based on morphological and cultural characteristics due to environment-induced changes in morphological characteristics. Therefore, to overcome this problem, DNA sequence analyses have been used to characterize and analyze the taxonomic complexity of *Colletotrichum*. Canon *et al.,* (2000) stated that data derived from DNA analyses is the most reliable framework for classifying *Colletotrichum* as DNA is not directly influenced by environmental factors. In particular, sequence analysis of the internal transcribed spacer (ITS) regions lying between the 18S and 5.8S genes and the 5.8S and 28S genes, has proved very useful in studying phylogenetic relationship samong *Colletotrichum* species (Sreenivasa prasad *et al.,* 1996; 1996; Moriwaki *et al.,* 2002; Photita *et al.,* 2005) [63, 37, 47]. Sequence analysis of protein coding genes such as partial β-tubulin gene

and introns from two genes (glutamine synthetase and glyceraldehyde-3-phosphate dehydrogenase) were also useful in resolving the phylogenetic relationships among *C. acutatum* species (Sreenivasa prasad and Talhinhas, 2005; Guerber *et al.,* 2003) [64, 20]. Although ITS sequences do not separate the *C. gloeosporioides* complex, some single genes or combination of genes, glutamine synthetase, and glyceraldehydes-3-phosphate dehydrogenase (GAPDH), can be used to differentiate *Colletotrichum* species (Weir *et al.,* 2012) [71]. Isolates of *C. acutatum* were phylogenetically separated from A1 to A4 subgroups based on sequences in partial β -tubulin 2 (exons 3-6). According to Canon *et al.* (2000), an integrated approach, where molecular diagnostic tools are applied along with morphological characterization, is a more accurate and reliable approach for studying *Colletotrichum* species.

Pathogenic Variability

When any of the progeny exhibits a characteristic that is different from those present in the ancestral individuals, this individual is called a variant (Agrios, 2005) [3]. Compatibility of plant-pathogen interactions is often governed by the gene-for-gene model in many pathosystems (Flor, 1971) [17]. Some pathogen populations are known to be pathogenically diverse and the diversity seems to be due to continuous generation of novel pathogenic variations (Taylor and Ford, 2007) [67]. A genotype with partial resistance would result in lower levels of infection which eventually would decrease the amount of inoculum in the field and limit the potential of epidemics. Several studies (AVRDC, 1999; Yoon *et al*., 2004) [4, 73] have screened *C. acutatum*, which is a very virulent species (Than *et al.,* 2008) [68] against chilli genotypes and found that *Capsicum baccatum* genotype 'PBC 80' is a genetic resource pool for resistance to anthracnose. However, another genotype of *C. baccatum*, 'PBC81' showed high susceptibility to some *C. acutatum* isolates. In contrast to *C. baccatum,* the susceptibility of the cultivar *Capsicum annuum* has been reported in several studies (Mongkolporn *et al.,* 2004; Park, 2007) [35, 42]. Moreover, *Capsicum chinense* 'PBC932' has been reported as a resistant variety to *C. capsici* (AVRDC, 2003) [5]. However, to date, there has not been any strong resistance found in *C. annuum*, which is the only species grown worldwide (Park, 2007) [42].

Infection stages and disease cycle

Colletotrichum employs different strategies for causing infection to the host plant which initiate from the intracellular hemi biotrophic mode to the intramural necrotrophic mode of nutrition (Bailey and Jeger, 1992) [7]. An intermediate stage showing partial endophytic life style of the pathogen before adapting to the necrotrophic mode of nutrition in the host plant was seen. Different species of this genus exhibit different mechanism of infection depending on the host infected. For instance, Peres *et al*., (2005) [44] reported the epiphytic or endophytic mode of survival of *C. acutatum* in an orchard infected with the bitter rot of apple. Also, intramural necrotrophy by *C. capsici* was reported by Pring *et al*., (1995) [52] while infecting cowpea leading the formation of appresoria from the conidia is followed by the formation of secondary conidia which further infects and spreads the pathogen inside the host leaves (e.g., The biotrophic disease cycle in citrus leaves).The second is the subcuticular intramural necrotrophy with the development of wide and swollen hyphae in the anticlinal and periclinal wall so host epidermal cells (e.g., The necrotrophic disease cycle on strawberry). The third strategy is the hemi biotrophic mode of infection where the pathogenic hyphae interact with the infection vesicles within the host cells (e.g. The biotrophic disease cycle on blue berry fruits).The fourth type of interaction is the combination of hypertrophic and subcuticular intra and intercellular development of the pathogen generally observed during infestation of almond leaves and fruits. As far as studies related to infection and colonization by *Colletotrichum* species i.e., *C. gloeosporioides* on susceptible chilli variety is considered, no biotrophic stage in for no infection vesicle has been found during the infection (Kim *et al*., 2004) [31]. An increased number of small vacuole with the condensed cytoplasm in the epidermal cells followed with cell destruction extending to the sub epidermal cells of the plant due to the action of pathogen enzyme has been noticed during the early stages of infection in chilli plant. During the later stages, inter and intra cellular for controlling the anthracnose disease in chilli from different parts of the world.

Disease management

There are various methods of controlling plant disease. As no single strategy is found to be very effective in controlling chilli anthracnose disease, Agrios (2005) [3] recommended an integrated disease management approach. Effective approaches for disease management usually involve the combined use of intrinsic resistance along with cultural, mechanical, biological, and chemical control (Wharton and Dieguez-Uribeondo, 2004) [72]. Using resistant varieties may eliminate losses from diseases as well as chemical and mechanical expenses of diseases control (Agrios, 2005) [3]. The use of shorter ripening period cultivars may allow fruits to be harvested earlier in order to prevent infection by the fungus. Crop rotation should be done at least 2 years with crops that are not Solanaceae plants. As the pathogen is capable of remaining in the soil and in plant debris, soil must be deeply ploughed to completely bury the crop residues containing the pathogens (Agrios, 2005) [3]. Among disease control management approaches, the use of resistant cultivars is the cheapest, easiest, safest, and most effective means of controlling diseases.

Chemical control

Use of chemicals is a widely used disease control strategy and a practical method to control anthracnose disease. However, fungicide resistance often arises quickly, if a single compound is relied upon too heavily (Staub, 1991). A fungicide widely recommended for anthracnose management in chilli is manganese ethylene bis dithiocarbamate (Maneb) (Smith, 2000) [62]. Soaking of chilli seeds for 12 hours in 0.2% thiram is best way to control *Colletotrichum* species. The strobilurin fungicides azoxytrobin (Quadris), trifloxystrobin (Flint), and pyraclostrobin (Cabrio) have recently been recommended for the control of chilli anthracnose (reviewed by Than *et al.,* 2008) [68]. Moreover, various fungicides have been found to be effective, including 0.2% mancozeb, 0.1% ziram, Blitox 50, 0.1% Bavistin and 0.5% or 1% Bordeaux mixture; benlate and Delsene M are used as seed dressings (CPC, 2007). However, there are numerous undesirable effects of using chemicals such as on farmers' income, the toxic effects of chemicals on farmers, and other environmental concerns, particularly in developing countries (Voorrips *et al.,* 2004) [70].

Biological control

To overcome the negative effect of chemical usage, use of plant extracts and biocontrol agents to control infection have

become a solution. Complete inhibition of fungal growth and spore germination were achieved with the use of 3% garlic bulb extract concentration (Singh, 1997) [60]. Crude extracts from different parts of Sweet flag, Palmorosa oil, Neem oil have been reported to be effective in curbing the growth of anthracnose fungus (Jayalakshmi and Seetharaman, 1998) [28]. An effective approach for eco-friendly management of chilli anthracnose is the combined application of plant extract of neem (*Azadirachta indica),* mahogany *(Swietenia mahagoni*), and garlic *(Allium sativum*). The combination of extracts from these plants showed significant impact on disease reduction as well as on yield of chilli (Rashid *et al.,* 2015) [55]. Trichoderma species have been reported to effectively control *Colletotrichum* species in chilli with concomitant disease reduction (Boonn ratkwang *et al.,* 2007) [8]. Moreover, antagonistic bacterial strains (DGg13 and BB133) were found to effectively control *C. capsici* (Intanoo and Chamswarng, 2007) [8]. Other biological control agents such as *Bacillus subtilis* and *Saccharomyces cerevisiae* have been reported as antagonistic to microorganisms (Jayelakshmi and Seetharaman, 1998) [28]

Use of resistant varieties

Developing resistance against the pathogen in the host is seeking to be the most important and sustainable approach for managing the disease. This strategy not only eliminates the losses caused due to the disease, but also remove the chemical and mechanical expense of the disease control (Agrios, 2005) [3]. The principle behind the use of resistant cultivars is to trigger the host defense response that in turn would inhibit or retard the growth of the pathogen involving the use of a single gene pair: a host resistance gene and the pathogen avirulence gene (Flor, 1971) [17]. Inlieu of the existing biotechnological approach to manage diseases, certain successful resistant varieties of chilli against *C. capsici* have been reported from different parts of the world (Yoon, 2003; Voorrips *et al.,* 2004; Garg *et al.*, 2014) [73, 70]. Though, not much success has been sought in developing resistant chilli varieties in the species *Capsicum annum* L.,which is the only species grown worldwide (Park, 2007) [42]. The two major requirements before proceeding for developing the cultivar is the knowledge of the resistant varieties of *Capsicum* occurring wildly in the region and the different Pathotypes of the pathogen found in that region. Many varieties resistant to *Colletotrichum* spp. and information regarding the pathotypes of the pathogen has been reported and is available AVRDC, 2003; Babu *et al.,* 2011) [5]. However, the challenging task of resistant breeding is exceptionally difficult in *Colletotrichum*-chilli pathosystem due to the association of more than one species of the pathogen with the disease (Sharma *et al.,* 2005; Saxena *et al.,* 2014) [58] along with the differential ability of the pathogenic virulence (Montri *et al.,* 2009) [36].

Future prospects

Though the epidemic nature of the disease has been studied for ages, many are still unexplored in terms of host- pathogen interaction, its spread and effective management strategies. There lies an urgent need to develop an efficient integrated management strategy keeping in concern the different environmental factors and pathogenic resistance, driving the successful colonization of the pathogen in the host tissues. An insight into the pathogen's lifestyle would provide valuable information required to develop targets for developing resistant varieties of chilli against the pathogen. Also, modifications in conventionally recommended cultural practices suiting to a particular agro- climatic region will prove helpful in better management of the disease. More studies are required for acquiring in-depth information regarding various modes of infection by the pathogen and the pathogenic variability associated within a region with the post-harvest as well as pre-harvest loss in the crop production. The overall knowledge about the key aspects of a disease triangle will enable better management of the disease keeping track of the quality and quantity of the crop produced thereby contributing efficiently to the country's economy.

References

1. Adaskaveg JE, Hartin RJ. Characterization of *Colletotrichum acutatum* isolates causing anthracnose of almond and peach in California. Phytopathology. 1997; 87:979-987.
2. Agostini JP, Timmer LW, Mitchell DJ. Morphological and pathological characteristics of strains of *Colletotrichum gloeosporioides* from citrus. Phytopathology. 1992; 82:1377-1382.
3. Agrios GN. Plant Pathology, 5th Ed. Academic Press, San Diego, 2005, 922.
4. AVRDC (Asian Vegetable Research and Development Centre). Off-season Tomato, Pepper, and Eggplant. Progress Report for 1998. Asian Vegetable Research and Development Centre, Taiwan, China, 1999.
5. AVRDC (Asian Vegetable Research and Development Centre), AVRDC Progress Report for 2002. Asian Vegetable Research and Development Centre, Taiwan, China, 2003.
6. Babu BS, Pandravada SR, Rao RDVJP, Anitha K, Chakrabarty SK, Varaprasad KS *et al*. Global sources of pepper genetic resources against arthropods, nematodes and pathogens. Crop Prot. 2011; 30:389-400.
7. Bailey JA, Jeger MJ (Eds.). *Colletotrichum*: Biology, Pathology and Control. Commonwealth Mycological Institute, Wallingford, 1992, 388.
8. Boonnratkwang C, Chamswarng C, Intanoo W, Juntharasri V. Effect of Secondary Metabolites from *Trichoderma Harzianum* Strain Pm9 on Growth Inhibition of *Colletotrichum Gloeosporioides* and Chilli Anthracnose Control. pp. 323-336. Proceeding of the 8th National Plant Protection Conference. Naresuan University, Phisanulok, Thailand, 2007.
9. Bosland PW, Votava EJ. Peppers: Vegetable and Spice Capsicums. CAB International, England, 2003, 233.
10. Chitkara S, Singh T, Singh D. Histopathology of *Colletotrichum dematium* infected chilli seeds. ActaBotanica Indica. 1990; 18:226-230.
11. CPC. Crop Protection Compendium, 2007.
12. Damm U, Woudenberg JHC, Cannon PF, Crous PW. *Colletotrichum* species with curved conidia from herbaceous hosts. Fungal Divers. 2009; 39:45-87.
13. Damm U, Cannon PF, Woudenberg JHC, Crous PW. The *Colletotrichum acutatum* speciescomplex. Stud. Mycol. 2012a; 73:37-113.
14. Damm U, Cannon PF, Woudenberg JHC, Johnston PR, Weir BS, Tan YP *et al*. The *Colletotrichum boninense* speciescomplex. Stud Mycol. 2012b; 73:1-36.
15. Dodd JC, Estrada A, Jeger MJ. Epidemiology of *Colletotrichum Gloeosporioides* in the Tropics. In: Bailey JA, Jeger MJ. (Eds.), *Colletotrichum*: Biology, Pathology and Control. CAB International, Wallingford, 1992, 308-325.

16. Don LD, Van TT, Phuong VYTT, Kieu PTM. *Colletotrichum* spp. attack in gon chilli pepper growing in Vietnam, Country Report, in Abstracts of the First International Symposium on Chilli Anthracnose, eds DG. Ohand KT Kim (Seoul: Seoul National University), 2007.
17. Flor HH. Current status for the gene-for-gene concept. Annu. Rev. Phytopathol. 1971; 9:275-296.
18. Freeman S, Katan T, Shabi E. Characterization of *Colletotrichum* species responsible for anthracnose diseases of various fruits. Plant Disease. 1998; 82(6):596-605.
19. Garg R, Loganathan M, Saha S, Roy BK. Chilli Anthracnose:a review of causal organism, resistance source and mapping of gene, in Microbial Diversity and Biotechnology in Food Security, eds Kharwar RN, Upadhyay R, Dubey NR. Raguwanshi (Springer), 2014, 589-610.
20. Guerber JC, Liu B, Correll JC, Johnston PR. Characterization of diversity in *Colletotrichum acutatum* sensu lato by sequence analysis of two gene introns mtDNA and intron RFLPs and mating compatibility. Mycologia. 2003; 95:872-895.
21. Hadden JF, Black LL. Anthracnose of Pepper Caused by *Colletotrichum* spp. Proceeding of the International Symposium on Integrated Management Practices: Tomato and Pepper Production in the Tropics. Asian Vegetable Research and Development Centre, Taiwan, 1989, 189-199.
22. Halsted BD. A new anthracnose of pepper. Bulletin of the Torrey Botanical. Club. 1890; 18:14-15.
23. Hong JK, Hwang BK. Influence of inoculum density, wetness duration, plant age, inoculation method, and cultivar resistance on infection of pepper plants by *Colletotrichum cocodes*. Plant Disease. 1998; 82(10):1079-1083.
24. Harp TL, Pernezny K, Ivey MLL, Miller SA, Kuhn PJ, Datnoff L. The etiology of recent pepper anthracnose out breaks in Florida. Crop Prot. 2008; 27:1380-1384.
25. Hong JK, Hwang BK. Influence of inoculum density, wetness duration, plant age, inoculation method, and cultivar resistance on infection of pepper plants by *Colletotrichum cocodes*. Plant Disease. 1998; 82(10):1079-1083.
26. Intanoo W, Chamswarng C. Effect of antagonistic bacterial formulations for control of anthracnose on chilli fruits. Proceeding of the 8th National Plant Protection Conference. Naresuan University, Phisanlok, Thailand, 2007, 309-322.
27. Isaac S. Fungal Plant Interaction. Chapman and Hall Press, London, 1992, 115.
28. Jayalakshmi C, Seetharaman K. Biological control of fruit rot and die-back of chilli with plant products and antagonistic microorganisms. Plant Disease Research. 1998; 13:46-48.
29. Jeffries P, Dodd JC, Jegerand MJ, Plumbley RA. The biology and control of *Colletotrichum* species on tropical fruit crops. Plant Pathology. 1990; 39(3):343-366.
30. Kim KD, OH BJ, Yang J. Differential interactions of a *Colletotrichum gloeosporioides* isolate with green and red pepper fruits. Phytoparasitica. 1999; 27:1-10.
31. Kim KK, Yoon JB, Park HG, Park EW, Kim YH. Structural modifications and programmed cell death of chilli pepper fruits related to resistance responses to *Colletotrichum gloeosporioides* infection. Genetics and Resistance. 2004; 94:1295-1304.
32. Liu F, Cai L, Crous PW, Damm U. Circumscription of the anthracnose pathogens. *Colletotrichum lindemuthianum* and *C. nigrum. Mycologia.* 2013; 105:844-860.
33. Liyanage HD, McMillan RT, Kistler HC. Two genetically distinct populations of *Colletotrichum gloeosporioides* from citrus. Phytopathology. 1992; 82:1371-1376.
34. Manandhar JB, Hartman GL, Wang TC. Anthracnose development on pepper fruits inoculated with *Colletotrichum gloeosporioides*. Plant Disease. 1995; 79:380-383.
35. Mongkolporn O, Dokmaihom Y, Kanchana-udomkarn C, Pakdeevaraporn P. Genetic purity test of F1 hybrid Capsicum using molecular analysis. Journal of Horticultural Science and Biotechnology. 2004; 79:449-451.
36. Montri P, Taylor PWJ, Mongkolporn O. Pathotypes of *Colletotrichum capsici,* the causal agent of chili anthracnose, in Thailand. Plant Dis. 2009; 93:17-20.
37. Moriwaki J, Tsukiboshi T, Sato T. Grouping of *Colletotrichum* species in Japan based on rDNA sequences. Journal of General Plant Pathology. 2002; 68:307-320.
38. O′Connell RJ, Bailey JA, Richmond DV. Cytology and physiology of infection of *Phaseolus vulgaris* by *Colletotrichum lindemuthianum*. Physiologial Plant Pathology. 1985; 27(1):75-98.
39. O′Connell RJ, Perfect S, Hughes B, Carzaniga R, Bailey JA, Green J. Dissecting the cell biology of *Colletotrichum* infection processes in *Colletotrichum*: Biology, Pathology, and Control, eds JA Bailey and MJ Jegar (Walling fords: CAB International), 2000, 57-77.
40. Park KS, Kim C. Identification, distribution, and etiological characteristics of anthracnose fungi of red pepper in Korea. Korean J Plant Pathol. 1992; 8:61-69.
41. Park WM, Park SH, Lee YS, Ko YH. Differentiation of *Colletotrichum* spp. causing anthracnose on *Capsicum annuum* L. by electrophoretic method. Korean Journal of Plant Pathology. 1987; 3:85-92.
42. Park HG. Problem of anthracnose in pepper and prospects for its management. In: Oh Dg, Kim KT. (Eds.), Abstracts of the first international symposium on chilli anthracnose. p. 19. National Horticultural Research Institute, Rural Development of Administration, South Korea, 2007.
43. Pearson MN, Bull PB, Speke H. Anthracnose of *Capsicum* in Papua, New Guinea; varietal reaction and associated fungi. Trop. Pest Manag. 1984; 30:230-233.
44. Peres NA, Timmer LW, Adaskaveg JE, Correll JC. Life cycles of *Colletotrichum acutatum*. Plant Disease. 2005; 89(8):784-796.
45. Perfect SE, Hughes HB, O′Connell RJ, Green JR. *Colletotrichum*: a model genus for studies on pathology and fungal-plant interactions. Fungal Genetics and Biology. 1999; 27(2-3):186-198.
46. Pernezny K, Roberts PD, Murphy JF, Goldberg NP. Compendium of Pepper Diseases. The American Phytopathological Society, St. Paul, Minnedota, 2003, 73.
47. Photita W, Taylor PWJ, Ford R, Lumyong P, McKenzie HC, Hyde KD. Morphological and molecular characterization of *Colletotrichum* species from herbaceous plants in Thailand. Fungal Diversity. 2005; 18:117-113.

48. Phoulivong S, McKenzie EHC, Hyde KD. Cross infection of *Colletotrichum* species; a case study with tropical fruits. Curr. Res. Environ. Appl. Mycol. 2012; 2:99-111.
49. Pickersgill B. Genetic resources and breeding of *Capsicum* spp. Euphytica. 1997; 96(1):129-133.
50. Poonpolgul S, Kumphai S. Chilli Pepper Anthracnose in Thailand. Country Report. *In*: Oh DG, Kim KT. (Eds.), Abstracts of the First International Symposium on Chilli Anthracnose. National Horticultural Research Institute, Rural Development of Administration, Republic of Korea, 2007, 23.
51. Pring RJ, Nash C, Zakaria M, Bailey JA. Infection process and host range of *Colletotrichum capsici*. Physiological and Molecular Plant Pathology. 1995; 46(2):137-152.
52. Prusky D, Plumbley RA. Quiescent Infections off *Colletotrichum* in Tropical and Subtropical Fruits. CAB International Wallingford, 1992, 289-307.
53. Prusky D, Koblier I, Aridi R, Beno-Moalem D, Yakoby N, Keen NT. Resistance Mechanisms of Subtropical Fruits to *Colletotrichum gloeosporioides*. In: Bailey JA, Jeger MJ. (Eds.), Colletotrichum: Biology, Pathology, and Control. CAB International Wallingford, 2000, 232-244.
54. Ranathunge NP, Mongkolporn O, Ford R, Taylor PWJ. *Colletotrichum truncatum* patho system on *Capsicum* spp.: infection, colonization and defence mechanisms. Australas. PlantPath. 2012; 41:463-473.
55. Rashid MM, Kabir MdH, Hossain MdM, Bhuiyan Md. R, Khan MAI. Eco-friendly management of chilli anthracnose (*Colletotrichum capsici).* International Journal of Plant Pathology. 2015; 6:1-11.
56. Roberts PD, Pernezny K, Kucharek TA. Anthracnose caused by *Colletotrichum* sp. on pepper [Online]. Journal of University of Florida/Institute of Food an Agricultural Sciences, 2001.
57. Royle DJ, Butler DR. Epidemiological Significance of Liquid Water in Crop Canopies and its Role in Disease Forecasting. *In*: Ayres PG, Boddy L. (Eds.), Water, Fungi and Plants. Cambridge University Press, Cambridge, 1986, 139-156.
58. Saxena A, Raghuvanshi R, Singh HB. Molecular, phenotypic and pathogenic variability in *Colletotrichum* isolates of subtropical region in North Eastern India, causing fruitrotofchillies. J Appl. Microbiol. 2014; 117:1422-1434.
59. Sharma PN, Kaur M, Sharma OP, Sharma P, Pathania A. Morphological, pathological and molecular variability in *Colletotrichum capsici*, the cause of fruit rot of chillies in the subtropical region of north western India. J Phytopathol. 2005; 153:232-237.
60. Singh SN, Yadav BP, Sinha SK, Ojha KL. Efficacy of plant extracts in inhibition of radial growth and spore germination of *Colletotrichum capsici.* Journal of Applied Biology. 1997; 7:58-61.
61. Simmonds JH. A study of the species of *Colletotrichum* causing ripe fruit rots in Queensland. Queensland Journal Agriculture and Animal Science. 1965; 22:437-459.
62. Smith KL. Peppers. *In*: Precheur, R.J. (Ed.), Ohio Vegetable Production Guide. Ohio State University Extension, Columbus, Ohio, 2000, 166-173.
63. Sreenivasaprasad S, Mills P, Meehan BM, Brown A. Phylogeny and systematic of 18 *Colletotrichum* species based on ribosomal DNA spacer sequences. Genome. 1996; 39:499-512.
64. Sreenivasaprasad S, Talhinhas P. Genotypic and phenotypic diversity in *Colletotrichum acutatum*, a cosmopolitan pathogen causing anthracnose in wide range of hosts. Molecular plant pathology. 2005; 64:361-378.
65. Staub T. Fungicide resistance: practical experience and anti-resistance strategies and the role of integrated use. Annu. Rev. Phytopathol. 1991; 29:421-442.
66. Sutton BC. The genus Glomerella and its anamorph *Colletotrichum*. In: *Colletotrichum* Biology, Pathology and Control (eds. JA. Bailey and MJ. Jeger) CAB International, Wallingford, 1992, 1-26.
67. Taylor PWJ, Ford R. Diagnostics, genetic diversity and pathogenic variation of ascochyta blight of cool season food and food legumes. European journal of Plant Pathology. 2007; 119:127-133.
68. Than PP, Jeewon R, Hyde KD, Pongsupasamit S, Mongkolporn O, Taylor PWJ. Characterization and pathogenicity of *Colletotrichum* species associated with anthracnose disease on chilli (*Capsicum* spp.) in Thailand. Plant Pathology. 2008; 57(3):562-572.
69. Tozze HJ, Massola NM. First Report of *Colletotrichum boninense* causing anthracnose on pepper in Brazil. Dis. Notes. 2009; 93:106.
70. Voorrips RE, Finkers R, Sanjaya L, Groenwold R. QTL mapping of anthracnose (*Colletotrichum* spp.) resistance in a cross between *Capsicum annuum* and *C. chinense*. Theoretical and Applied Genetics. 2004; 109:1275-1282.
71. Weir BS, Johnston PR, Damm U. The *Colletotrichum gloeosporioides* species complex. Stud. Mycol. 2012; 73:115-180.
72. Wharton PS, Diéguez-Uribeondo J. The biology of *Colletotrichum acutatum*. Anales del Jardin Botanico deMadrid. 2004; 61:3-22.
73. Yoon JB, Yand DC, Lee WP, Ahn SY, Park HG. Genetic resources resistant to anthracnose in the genus Capsicum. Journal of Koran Society Horticulture Science & Technology. 2004; 45:318-323.