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Anthracnose, a Prevalent Disease in Capsicum.

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ABSTRACT

Chilli (*Capsicum annuum* L) is the major spice crops used all over the world. Anthracnose disease caused by *Colletotrichum* species is one of the chief hindrances for chilli production. *Colletotrichum* is a large genus of Ascomycete fungi, containing species that cause anthracnose disease on wide range crops of economic value. Though disease is being managed by chemical control agents, environmental concern calls for the usage of ecofriendly methods. Inspite of extensive research, anthracnose resistant chilli cultivar has not been developed and commercialized. Breeding for resistance is still challenging because of presence of several *Colletotrichum* species in a given pathosystem. This paper reviews importance of chilli, anthracnose disease and causative organism, plant pathogen interactions, disease control strategies. Emphasis is laid on molecular approaches for disease management.

Keywords: Anthracnose, Colletotrichum, Molecular Approaches.

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INTRODUCTION

Pepper is an important vegetable as well as spice crop, cultivated world wide. It is not only used in many cuisines but also found to have many medicinal properties. It belongs to genus *Capsicum*. *Capsicum* is a genus of Flowering plants that belongs to the family Solanaceae. Though it was originated in the American tropics, it is widely propagated [1]. In world, chilli is raised over an area of 1832 thousand hectares producing 2959 thousand tons. India is largest producer, with 36% share in global production. Andhra Pradesh, Orissa, Maharashtra, West Bengal, Karnataka, Rajasthan and Tamil Nadu are found to be important states growing chilli in India.

The genus *Capsicum* comprises about 20- 25 species, out of which *C. annuum, C, baccatum, C. chinense, C. frutescens and C. pubescens* are cultivated. *Capsicum annuum* is widely cultivated variety [2], second being *C. frutescens* [3]. Chilli is called variously with different names depending on the place viz, Pimento (Spanish), Puvre de Guinee (French), Paparika (German), Spaanse Peper (Dutch) etc. Commonly used term is Chilli, which refers to hot types of Capsicum.

Chilli is found to be comprised of many plant derived chemical compounds that promote health. The strong spicy taste comes due to the presence of active alkaloid compounds capsaicin, capsanthin, capsorubin. Chilli contains steam volatile oils, carotenoids, fatty oils, vitamins, mineral elements etc., [3]. Chilli reduces platelet aggregation; they also act as vasodilators stimulating blood circulation. Chilli helps in reducing calories by increasing thermogenesis. Chilli reduces risk of cancer by preventing carcinogens from binding to DNA. They contain pain alleviating salicyclate compounds. In addition, consumption of chilli itself releases endorphins in the body which help in reducing pain. In folk medicine, capsicum preparations are used in rheumatic disorders, pharyngitis, asthma, cough, anorexia, hemorrhoids. Vitamin C is present in more quantities in fresh green chilies than citrus fruits and Vitamin A is high in red chilli than carrots [4, 5]. Colour of the chilli is due to presence of carotenoids and presence of numerous chemicals, mineral elements impart nutritional value to chilli [6, 7, 8].

Chilli is an important commercial crop grown in India. India emerged as leading producer and exporter of chilli contributing one fourth of world's production. Although production is high in India, the average productivity is less (1ton/ha), when compared to other important producers of chilli viz, China, Mexico, Taiwan where the productivity is 3 tons/ha [9]. Cultivation of open pollinated varieties which lack the capacity to overcome yield barriers is one of the reasons for low productivity [10]. Another important contributor for this low productivity is biotic stress resulting in diseases. Several diseases caused by bacteria, fungi and viruses affect the production of chilli.

Among all the diseases, anthracnose disease is the major constraint to chilli production worldwide resulting in high yield losses [11]. This fungal disease caused by *Colletotrichum* species drastically reduces the quality and yield of fruit resulting in low returns to farmers. 10-

80% of marketable yield is reduced in Thailand [12], about 13% in Korea [13]. This die back/ fruit rot/ anthracnose disease is seen on mature fruits resulting in both pre harvest and post harvest fruit loss [14, 3]. In India, in severe cases, pre harvest and post harvest losses comprise up more than 50% [15]. Significant yield losses were reported from Punjab and Haryana (20-60%) and Assam (12-30%) [16, 17].

Anthracnose:

The word anthracnose is a Greek word meaning 'coal'. It is commonly used for plant diseases which are characterized by dark sunken lesions having spores [18]. Anthracnose disease is one of major constrains that restricts profitable production of chilli. It is caused by *Colletotrichum* species. It directly reduces the quantity and quality of the harvested yield. Small lesions on chilli fruits also affect the profits [19]. Post harvest damage is more as infection remains latent in plant cells [20] and symptoms appear once the fruit is matured. Symptoms include sunken necrotic lesions with concentric rings which produce conidial masses. Under severe conditions, lesions fuse and conidial masses may occur in concentric rings on lesions.

Colletotrichum:

Causal agent of chilli anthracnose disease is *Colletotrichum*. Genus *Colletotrichum* belongs to Kingdom-Fungi, Phylum-Ascomycota, Class-Sordariomycetes, Order- Phyllachorales and Family- Phyllachoraceae. *Colletotrichum* genus comprises a number of plant pathogens, effecting woody to herbaceous plants. Fruits are majorly affected in the disease. *Colletotrichum* species are pathogenic to commercial crops (strawberry, pepper, citrus), and cereals (maize, sugarcane, sorghum). It is the 8th most important plant fungal pathogenic group [21]. *Colletotrichum* species are known as broad range pathogens as a single species is capable of infecting diverse hosts and numerous species infect a single host [22]. Despite the fact that *Colletotrichum* species are responsible for causing anthracnose disease, some other diseases such as red rot of sugar cane, coffee berry disease, crown rot of banana were also reported [23]. Seldom, in human diseases like keratitis, sub-cutaneous infection, *Colletotrichum* species have been implicated [24, 25, 26, 27]. Mycotic infection of a sea turtle by *Colletotrichum* species was also reported [28]. Furthermore *Colletotrichum fioriniae* infecting hemlock scale insects in England and *Colletotrichum gloeosporioides* on citrus scale insects in Brazil [29] were reported. Though the infection process is yet to be understood, it was seen that insects became infected after conidial suspension was sprayed [30].

Pepper anthracnose caused by *Colletotrichum* species is the reason for severe yield losses in many Asian countries. Causal agents of chilli anthracnose, at different places were tabulated in Table 1. In *Colletotrichum* patho system, several *Colletotrichum* species can be associated with anthracnose of same host [31, 32]. Five species of *Colletotrichum* are found to be associated in causing anthracnose disease in chilli world wide. They are *C. capsici, C. acutatum, C. gloeosporioides, C. coccoides*, and *C. graminicola* . As per Kim et al., [33], different species infect chilli plant at different stages. Leaves and stems are damaged by *C. coccodes* and *C. dementium,* where as *C. acutatum* and *C. gloeosporioides* infect chilli fruits. *Colletotrichum*

capsici is found to be prevalent in red chilli fruits where as *C. acutatum* and *C. gloeosporioides* cause infection both in young and mature fruits ([34, 35, 15, 36, 37]. In Thailand, *C. capsici* is found to be great menace and found to contain three pathotypes [38].

Table 1- Reported causal agents of chilli anthracnose [11].

Countries and regions	Causal agent	Reference
Australia	Colletotrichum acutatum, C. atramentarium, C. demantium, C. gloeosporioides var. minor, C. gloeosporioides var. gloeosporioides	[31]
India	C. capsici	[158, 159]
Indonesia	C. acutatum, C. capsici, C. gloeosporioides	[122]
Korea	C. capsici, C. gloeosporioides, C. coccodes, C. demantium	[160]
Myanmar (Burma)	Gloeosporium piperatnum E. and E., C. nigrum E. and Hals	[161]
Papua New Guinea	C. capsici, C. gloeosporioides	[162]
New Zealand	C. coccodes	[163]
Taiwan	C. acutatum, C. capsici, C. gloeosoprioides	[164]
Thailand	C. acutatum, C. capsici, C. gloeosoprioides	[11]
UK	C. acutatum, Glomerella cingulata	[165]
USA	C. acutatum	[78]
Vietnam	C.acutatum, C.capsici, C.gleosporioides, C.nigrum	[166]

Many species of *Colletotrichum* are seed borne and they may survive in soil on debris and may be spread by water splash dispersal of conidia and transmission of ascospores through air [39]. They are capable of growing in and on seeds as acervuli and micro sclerotia [40]. Disintegration of parenchymatous layers of seed coat and even food materials in endosperm and embryo are depleted in highly colonized seeds [41]. Appresorium that developed from spore on plant surface becomes route of infection and is followed by cuticle penetration [42]. Once the pathogen is penetrated, establishment of fungus in plant tissues is aided by host induced virulence effectors. Fungal colonies enter biotrophic phase involving dormancy [43], followed by necrotic phase resulting in death of plant cells. Severe post harvest losses are due to delayed onset of disease symptoms [43]. Biotrophic life style adapted by *Colletotrichum* species also contribute to their standing as symptomless endophytes of living plant tissues [44, 45, 46] Fungi can grow on alternative hosts like other solanaceous or legume crops, rotten fruits [47].

Morphological Characterization:

For effective disease management, accurate identification of *Colletotrichum* species is very much essential. Classically, identification of *Colletotrichum* is done by morphological characteristics like size and shape of conidia, appressoria and cultural characters like, colony outline, shape, colour and texture [48, 49]. Most often considered morphological characters include: culture colony characteristics, growth rate, conidial morphology, appressorial morphology. Than et al., [50] reported grayish white to dark grey *C. gleosporioides*. Some isolates of *C. gleosporioides* showed pale grey to black aerial mycelium while some produced even mycelia mass. Summary of morphological data of *Colletotrichum* species studied in Thailand was presented in Table 2. *C. capsici* colonies were white to grey with little aerial

mycelium while *C. acuatatum* produced pale orange colonies, showing sparse aerial mycelium. Most of *Colletotrichum capsici* colonies showed cottony growth with regular- irregular margins showing colour variation from light to dark grey having whitish to brownish tinge [51]. Further, conidial shape was reported to be fusiform to falcate. Optimum temperarure for *C. capsici* is 28°C while that of *C. gloeosporioides* is 32^{0}C [52].

Table 2- Morphological data for Colletotrichum species [11]

S. No	Host	Species	Colony Character	Conidia			Appressoria	
				Length (µm)	Width (µm)	Shape	Length (µm)	Width (µm)
1	Chilli	C.gloeosporioides	Pale grey to black zonated colonies with abundant orange conidial masses near the centre	13.5	4.5	Cylindrical	9.0	6.3
2	Chilli	C.acutatum	Orange coloured colony with slight mycelium	14.0	3.5	Fusiform	6.5	6.0
3	Chilli	C.capsici	White to grey colour with dark green centre and cottony mycelium	21.0	3.0	Falcate	9.5	6.5

Molecular Characterization:

Nevertheless, morphological examination followed by species identification is not adequate. This may be due to changes in the morphological characteristics due to variation in environment. DNA sequence analysis method is being used to characterize the species to overcome problems in traditional methods. Cannon et al., [32] emphasized that data derived from DNA analysis is the most reliable for classifying *Colletotrichum* as DNA is not directly influenced by environmental factors. For fungal phylogenetic studies, mostly utilized sequences are from ribosomal gene cluster, as they are present in large numbers and evolved as a single unit [53]. Because of their comparative variability, sequence analysis of internal transcribed spacer (ITS) regions lying between 18S and 5.8S genes and 5.8S and 28S genes, proved to be useful in studying phylogenetic relationships of *Colletotrichum* species [54, 55, 56, 57]. Restriction Fragment Length Polymorphisms (RFLP) studies of ITS regions from AluI. RasI and BamHI digestions were used to differentiate *Colletotrichum* species causing anthracnose in chilli in Taiwan region [58]. *Colletotrichum capsici* and *Colletotrichum gloeosporioides* causing chilli anthracnose in Thailand were distinguished using RAPD markers [59]. According to Cannon et al., [32] integrated approach, where there is application of molecular diagnostic tools along with traditional morphological characterization is more accurate and reliable approach for studying *Colletotrichum* species.

Plant- Pathogen Interactions:

It is often observed that a pathogen which causes disease in certain plant may not cause disease in another plant. In general, plants are resistant to many diseases. Pathogens are able to cause disease, only when they evade plants immune responses. According to Flor [60]

inheritance of resistance in host and the ability of pathogen to cause disease is controlled by pair of corresponding genes, one being resistance gene present in plant and the other being *Avr* gene of pathogen. *Avr* genes of pathogens are accountable for the production of proteins that are either directly or indirectly recognized by only those plants that possess complementary R gene. In the evolution, plants have evolved many strategies to protect themselves from pathogens. At the same time, even pathogens developed many mechanisms to overcome host immune responses and to establish themselves in host. These resulted in detailed raise of attack and counterattack strategies.

The first barrier that will be encountered by pathogen is rigid plant cell wall. In order to gain entry into the host plant and establish infection, pathogens need to hydrolyze the cell wall. The ability of the pathogens to secrete hydrolytic enzymes to degrade components of plant cell wall is one of important virulence factors of plant pathogenic fungi and bacteria [61]. Cellulases, xylanases, polygalacturonases and pectate lysases initially degrade polysaccharide components of cell wall, followed by cleavage of ester cross links between polysaccharide fibrils by the enzymes like pectin esterases. This results in loosening of cell walls [62]. Cell wall degrading enzymes serves dual function, as they not only allow pathogen's entry into the host, but also alarms host. Degradation products released due to cell wall degradation induce plant's immune response [63, 64]. The immune responses may include production of antimicrobial compounds, strengthening of plant cell wall, inducing programmed cell death etc. Some pathogens are successful in causing disease, as these pathogens encompass the ability to overcome immune responses [65].

Despite the fact that pathogens developed diverse strategies to successfully overcome host defense responses, plants also improved their strategies to protect themselves from the effects of pathogens. Pathogen strategies may include interference or disruption of host defensive mechanisms, avoiding detection and elimination by host immune response by preventing antigen presentation, blocking apoptosis, mimicking molecules [66, 67]. Plants naturally comprise immune system that helps to protect itself from several microbial infections. One of the effective methods through which the defense system is mediated in plants is by Resistance (R) genes. They are capable of recognizing specific pathogen derived avirulence (*Avr*) factors [68, 69]. Resistance mediated by R genes is due to the highly specific interaction between plant R gene and corresponding pathogen *Avr* genes [60]. Hosts protect themselves either by resistance or by tolerance [70, 71]. Plant resistance is due to the presence of resistant traits. Resistant traits reduce damage by restraining multiplication of pathogen and finally eliminating it. On the other hand, tolerance is achieved by thinning the consequences of infection without eliminating the pathogen [70, 71]. During their co evolution, plants and pathogens maintained a long standing relationship allowing them for mutual co- existence. This might lead to one of the possible mechanisms i.e. exchange of genetic material called horizontal gene transfer [72].

Once pathogen enters into host, obtaining nutrition from the host is crucial. Pathogens developed many mechanisms all the way through to obtain nutrition from the plants. When we take into consideration of fungi, fungi form several infection structures such as appressoria,

hyphae to get nutrition from the host. Fungi change the structures of the hyphae they produce in response to changes in host plant cell. These infection structures help at different stages of pathogenesis like attachment to host, penetration, multiplication, establishment of infection, obtaining nutrition. Plant pathogenic fungi are classified based on the mode through which they obtain nutrition from plants. Fungi, obtaining nutrition from the living cells are Biotrophs. While fungi obtaining nutrients from host cells after killing them are called Necrotrophs. Necrotrophs immediately kill plant cells and subsequently lead a saprotrophic life. Biotrophs, maintain a relation with host cell and by their feeding activities, they deplete nutrients in the cell, thus exploiting the host but not killing it. Different biotrophic ways include intercellular, sub cuticular, inter and intracellular, extracellular with haustoria, intracellular with haustoria. Apart from these two groups, there is a group of fungi, which come under hemibiotrophs. They use both the modes of nutrition at different stages of their life. *Colletotrichum* is best example for hemibiotrophic life style.

For many years, *Colletotrichum* species proved to be an excellent model for studying cellular and molecular ways of fungal pathogenicity [73]. Most species of *Colletotrichum* are hemibiotrophs but some (*Colletotrichum capsici*) express 'subcuticular intramural necrotrophy' [74], *Colletotrichum gloeosporioides* display both strategies based on the host plant [75]. Some *Colletotrichum* species form long term quiescent infections [76, 77].

They are capable of growing in and on seeds as acervuli and micro sclerotia. Micro-sclerotia produced by *Colletotrichum* species allow dormancy in stressfull conditions. During conditions that favour, conidia from acervuli and micro sclerotia are splashed by rain. Even diseased fruit acts as inoculums and spread the disease from one plant to other [78]. The first phase of fungal- plant interaction is the adhesion of spores to host surface. Conidia of *Colletotrichum* species dispersed, quickly adhere to the plant surfaces [79, 80]. Conidia are produced in acervuli which are protected by mucilage that covers them. Mucilage comprise of several different enzymes and they may help in protecting conidia from odd conditions and plant metabolites [81]. It was identified that hydrophobic interactions play role for initial attachment of conidia to the host cuticle [80]. Conidial adhesion also requires some proteins on the surface of spores apart from hydrophobic interactions. This was proved in the case of *C. musae* and *C. graminicola*, when conidial adhesion was inhibited following proteolytic treatment [82, 83, 84]. Studies have exposed that the surface of *C. lindemuthianum* conidia has a covering of brush like layer made of fibrillar material [85]. A study revealed that at higher concentrations of capsaicin, (principle component imparting pungency to chilli) conidial germination was completely inhibited [86]. *Colletotrichum* spores after adhering on to the plant surface, sense physical and chemical signals communicated by plant and start germinating giving rise to appressoria. Germination is an important stage in fungal development. Studies revealed that mostly expressed genes during conidial germination of *C. acutatum* belonged to those encoding histone protein, ATP synthease, 14-3-3 protein, MAP kinase and ABC transporter [87]. One of the abundantly expressed genes encoding 14-3-3 protein was identified in several other fungi. It is a member of putative kinase regulators which was characterized in mammalian brain tissue [88]. The role of this gene during conidial germination in *C. acutatum* is so far not studied. After germination, conidia forms a melanised dome shaped

appressorium. Formation of appressorium is significant for penetration into host. In apprressorium, turgor pressure created by accumulation of glycerol allows apprressorium to make a way into the host followed by instigation of infection [89].

A series of special structures are produced by *Colletotrichum* species. They include germ tubes, appresoria, intracellular hyphae, necrotrophic hyphae [89]. The pathogens either colonize by establishing intracellularly or in subcuticular tissues. Chemical inducers for the production of appressoria were studied on *C. gloeosporioides* infecting avocado and C. musae infecting banana [90, 91]. In *C. trifolii*, inhibitor studies have shown that cAMP and cAMP dependent protein kinase are necessary for germination and for the formation of appressorium [92]. In pathogenesis of *Colletotrichum* species, secretion of hydrolytic enzymes plays a vital role [93, 94].

Although the mechanisms developed by *Colletotrichum* species for interaction with hosts seem to be similar, there are some variations between species. Host- pathogen interactions of *C. acutatum* appear to be more biotrophic than that of *C. gloeosporioides* [95]. Most of the *Colletotrichum* species establish biotrophic interaction with the host. It is assumed that during the establishment of biotrophic interaction, pathogen develops many strategies to evade defense mechanisms. For example, pathogen masks its surface by converting chitin by deacetylation process. This prevents plant chitinases from recognizing and degrading chitin [96]. During biotrophic phase, the nutrition of *Colletotrichum* species is dependent on living host cells. *Colletotrichum* species develop and maintain efficient transport system which directs nutrients from host cell to fungal cell [97]. Depending on the environmental conditions and based on the species infecting the host, biotrophic stage transitions to necrotrophic phase. During necrotrophic phase, pathogen concentrates on killing the host rather than protecting itself from host defenses [98]. On the whole, life cycle of *Colletotrichum* includes the following stages: adhesion, germination and appressorium formation, appressorium differentiation and development, biotrophic development and necrotrophic development.

Disease Management:

Anthracnose disease is one of the major diseases of chilli. Under moisture conditions, spores expand very quickly spreading the disease. There are various methods of controlling disease. Few of them are discussed below. As no single strategy is found to be effective in controlling chilli anthracnose disease, Bailey [99] and Agrios [100] recommended integrated disease management approach.

As pathogen is capable of remaining in soil, plant debris, soil must be deeply ploughed before planting [100]. Disease free seeds are to be used to reduce infection. Crop rotation is done with non Solanaceous plants [101]. Proper sanitation and pretreatment of seeds with fungicides may reduce the risk of disease. Wounds are to be avoided while handling, as pathogen finds entry through wounds.

Use of chemicals is widely used disease control strategy which increased the yield [102, 103, 104] Fungicide widely recommended for anthracnose chilli is Manganese ethylenebisdithiocarbamate (Maned) [105]. Soaking of chilli seeds for 12hrs in 0.2% thiram is best way to control *Colletotrichum capsici* [106]. When seeds were treated with emisan effective control of *Colletotrichum* species was observed [107]. Usage of Companion, JKstein and Bavistin along with thiram was effective in eliminating infection from seeds [108]. High cost of chemicals and toxic effect of chemicals on farmers and other environmental concerns raise questions on usage of chemicals.

Biological Control:

For many years control of chilli anthracnose relied on chemicals. Indiscriminate use of these chemicals gave up new challenges like development of pest resistance, food poisoning, environmental pollution, negative effect on farmer's health, and increase in cost. To overcome the undesirable effects of chemical usage, use of plant extracts to control the infection came at rescue. Antimicrobial activity of *Nigella sativa* against *Colletotrichum capsici* was reported [109]. Investigations proved that *Azadirachta indica, Datura stramonium, Ocimum sanctum, Polyalthial longifolia* and *Vinca rosea* were fungitoxic against *C. capsici* [110]. When the extracts of garlic bulb at 3% concentration was used, complete inhibition of fungal growth and spore germination was achieved [111]. It was reported that crude extracts from different parts of Sweet flag, Palmorosa oil, Neem oil confined the growth of anthracnose fungus [112, 113]. Leaf extracts of *Solanum torvum, Datura metel* and *Prospopis juviflora* are effective in inhibiting conidial germination [114].

Fresh and dry weight of *C. capsici* was reduced when antifungal activity of fruit and flower extracts of *Datura innoxia* were used against *C. capsici* in vitro [115]. *P. fluorescens* isolate pf1 inhibited mycelia growth of *C. capsici* invitro effectively [116]. Under in vitro conditions 3% nimbicidin (Neem kernel extract) inhibited growth of *C. capsici* and under green house conditions, less mortality rate was observed in nimbicidin sprayed plants [117]. Ethanolic extracts of *Abrus drecatorius* and *Rauvolfia tetraphylla* showed inhibitory effects on conidial germination and radial growth of *C. capsici* [118]. Increase in the yield of chilli by decreasing the incidence of fruit rot was reported when 40day old seedlings were treated with *P. fluorescens* sol (1%) [119]. Using *Saccharomyces cerevisae* and *Bacillus subtilis* as biological control of the organism was also reported [120].

Molecular Approaches for Disease Management:

Eco- friendly and affordable and beneficial method by which anthracnose disease is managed, is to use of resistant varieties. Several sources for resistance to *Colletotrichum capsici* were found [121, 15, 36, 122]. Inheritance patterns are being studied to locate and map quantitative trait loci for resistance.

'PBC80' and 'PBC81; are resistant sources in *Capsicum baccatum* [123, 124], 'PBC32' is a resistant source of *Capsicum chinense* [125, 15] Inheritance of resistance genes have been

studied which revealed that inheritance patterns depend on source of resistance and Colletotrichum species. In a study, resistance to anthracnose disease caused by *Colletotrichum capsici* and *Colletotrichum acutatum* was studied in *Capsicum baccatum* PBC80 and PBC1422 and *C. chinense* PBC932. PBC80 and PBC1422 intraspecific cross populations(F2, BC1) were developed and inheritance pattern of resistance was determined. A single recessive gene responsible for the resistance at mature green fruit stage and a single dominant gene for the resistance at ripe fruit stage. Linkage analysis revealed that identified genes co4 and Co5 from PBC80 are different loci from co1 and co2 identified in PBC932 [126].

Capsicum annuum 83-168 breeding line is resistant to *Colletotrichum capsici 158ci*. The resistance is inherited by a single dominant gene. Resistance to *Colletotrichum dematium* in *Capsicum annuum* chungryong is by partial dominance [36]. *Capsicum chinense* Jacq 'PBC932' is resistant to *Colletotrichum capsici* and the resistance is inherited through a recessive gene [15]. Resistance in *Capsicum baccatum* 'PBC80' to *Colletotrichum accutatum* 'Ksca-1' is governed by a dominant gene [127]. Resitance in AR line which is derived from *C. chinense* Jacq 'PBC932' to *Colletotrichum accutatum* is governed by a recessive gene [128]. *Capsicum annuum* Chungryong, is resistant to *Colletotrichum capsici* and this resistance is govered by partially dominant gene [121, 36]. Resistance in Daepoong- Cho variety against C. capsici is controlled by recessive gene. It was reported that 'Daepoong Cho' and 'AR' lines pocess same resistance gene to *Colletotrichum capsici* even though, the source of resistant genes were different Capsicum spp., *C. annuum* and *C. chinense* respectively [129]. When QTL mapping was performed in *Capsicum chinense* 'PRI95030' resistant to *Colletotrichum gloeosporioides* and *C. capsici*, one major QTL (B1) and three minor QTLs (B2, H1, D1) for *Colletotrichum gloeosporioides*, one major (B1) and one minor QTL for *Colletotrichum capsici* were mapped [122]. QTLs for resistance to *Colletotrichum acutatum* and *C. capsici* were analysed in which it was reported that CaR12.2 QTL (for *C. acutatum*) and CcR9 QTL (for C. capsici) are positioned differently. But close links between minor QTL CcR12.2 and major QTL Car12.2, and major and minor QTLs of CcR9 were found [130]. EtagMcgg05e, EtacMccg13, EtagMcgt04, EacgMcgg02 AFLP markers are closely linked to major QTL CaR12.2, and EtacMccg13 is closely linked to CcR9 (C. annuum x C. baccatum, PBC81) [131].

Resistance Gene Analogs (RGAs):

Though there are many advances in plant disease control strategies, production is still at threat by several pathogens and pests. Initially, chemical control methods served the purpose. Soon, their indiscriminate usage questioned environmental safety. To overcome these problems, much attention is being focused on investigating, understanding plant innate resistance mechanisms. Plants are capable of activating cascade of defense responses. This in turn, triggers a long lasting systemic response that enables plant for gaining resistance against a broad spectrum of pathogens [132, 133] R gene mediated resistance is very advantageous to plant as it can eliminate the pathogen without harming the plant, and also it is ecofriendly. But R genes are often defeated by pathogens as a part of coevolution [134]. Another main concern is durability of R genes. Many R genes become non functional, because of single mutation in associated *Avr* gene. This results in non recognition of pathogen. Traditionally, breeding

strategies focused introgression of R genes, one at a time. This homogenous host population often exerts selection pressure resulting in mutation in corresponding *Avr* genes. This renders host very susceptible to the pathogen. Durability can be enhanced by introducing multiple R genes (gene pyramiding) into individual plant lines [134].

Almost 60 resistance genes have been identified and cloned from variety of plants (monocots and dicots) [135]. Cloning R genes from variety of crops and transferring them into useful cultivars has become technological advance [136]. For example, Pepper gene Bs2 provided sustainable resistance against Bacterial spot disease caused by Xanthomonas campestris. This gene is found to encode a NB- LRR protein [137]. This pepper Bs2 transgene effectively works in tomato conferring resistance to tomato against X. campestris. Several R genes against fungal pathogens were identified like barley Rpg1 gene [138, 139].

But transfer of R genes from model crops into other crops that are distantly related can be hampered due to 'restricted taxonomic functionality' (RTF) [137].Bs2 and several R genes from tomato can function as transgene in plants within same family (tobacco, potato, pepper) [140] but does not function in Arabidopsis. Even Arabidopsis RPS2 gene does not confer resistance in tomato [137].

Most of the R genes cloned belong to family that encodes proteins with NBS (Nucleotide Binding Site) and LRR (Leucine rich repeat) domains [141, 142]. Products of NBS-LRR genes are comprised of three main domains, (1)a variable N-terminal domain of about 200 amino acid, (2) a NBS domain of 300 amino acids and (3) variable tandem arrangement of about 10-40 LRR motifs [142]. NBS domain is implicated in signal transduction, where as LRR domain in ligand binding and pathogen recognition. Because of their sequence similarity with known R genes, these are called Resistance gene homologs (RGHs) or Resistance genes analogs (RGAs). Relation between R genes and RGAs are described in different ways. According to He et al., [143] RGAs are actual R genes while according to Radwan et al., [144] and Yan et al., [145] RGAs are linked to R genes, and they segregate along with R genes. PCR strategy, in which by using degenerated primers designed from these conserved motifs, resulted in identification of many resistance gene analogs (RGAs) from different plant species like potato [146], bean [147], rice [148]. Expressional studies of RGAs enable to determine whether RGAs play a key role in conferring resistance or they are simply linked to R genes [149]. RGAs were studied more in Solanaceae family [150]. RGAs can be identified in many crops as certain functional domains are highly conserved in R genes. Further, studies revealed that NBS-LRR families are ubiquitos in plants [151].

Egea- Gilabert et al., [152] developed an efficient technique in pepper, for isolation of RGAs from silver stained denaturating polyacrylamide gel using modified Amplified Fragment Length Polymorphism (AFLP) strategy. Wan, H.et al., [153] identified 78 RGAs in pepper by using degenerate PCR amplification. Further, they were grouped into non- Toll interleuking-1 receptor (TIR)- NBS- LRR and TIR-NBS-LRR subfamilies.

RGAs can be cloned by PCR based approaches and can genetically mapped. Functionality cn be tested if RGA maps to known resistance locus. Once identified, RGAs can be used as probe in the process of searching R genes, can also be used in Marker Assisted Selection. Isolation of RGAs became increasingly imperative as Resistant Gene Analog Polymorphism technique is proved to be efficient technique [154] in identifying molecular markers for disease resistance. Identification of RGAs against *Colletotrichum* will open up avenue for developing resistant chilli cultivars.

MAB:

One of the age old practices which is even now employed to produce high yielding and resistant varieties is Breeding. One of the major objectives of plant breeding is the development of cultivars and hybrids with multiple resistances or tolerances to stresses (both biotic and abiotic). Conventional breeding involves crossing of entire genome and relines on visual selection, which is time taking process. But with the development of molecular tools, plant breeding is becoming much quicker and easier, more effective and efficient. One such method which reduces the time lapse in conventional breeding method by replacing the phenotypic selection by genotypic selection is Marker Assisted Selection.

Finding sources of resistance and introducing the resistant traits in other varieties helps in the development of resistant varieties. Due to advancement of molecular markers, phenotypic screening of population is replaced by marker assisted screening. AFLP technique developed by Vos et al., [155] was used widely to identify molecular markers linked to traits of interest. As it is time consuming, during marker assisted selection, these AFLP markers are being converted to SCAR or CAPS [156, 157]. Reports of Marker Assisted Breeding for Chilli against Colletotrichum were not yet reported. Mapping QTLs and further identification of markers help in pyramidizing genes, thus highly resistant varieties can crop up which efficiently fight back anthracnose disease.

CONCLUSION

Despite of extensive research being carried out in anthracnose disease of chilli, resistant variety is not yet commercialized. This may be due to lack of information concerning interactions of different species related with chilli anthracnose. Deep insight into plant pathogen interactions is required in order to understand pathosystem of *Colletotrichum*. Although there are diverse strategies for disease management, use of resistant cultivars is ecofriendly and breeder friendly. Using of molecular approaches for the development of resistant varieties should be focused as it provides long lasting resistance. Major reports on anthracnose, plant pathogen interactions are still needed. This review article will be helpful to the researchers for better understanding.

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REFERENCES

[1] Pickersgill, B., 1997. Genetic resources and breeding of *Capsicum* spp. Euphytica, 96 (1):129-133.

[2] Tong, N., Bosland, P.W., 1999. *Capsicum tovarii*, a new member of Capsicum complex. Euphytica, 109(2): 71-72.

[3] Bosland, P. W., Votava, E.J., 2003. Peppers: Vegetable and Spice Capsicums. CAB International, England,p.333.

[4] Osuna- Garcia, J.A., Wall, M.W., Waddell, C.A., 1998. Endogenous levels of tocopherols and ascorbin acid during fruits ripening of New Mexican- type chilli (*Capsicum annuum* L.) cultivars. Journal of Agricultural and Food Chemistry 46(12): 5093-5096.

[5] Martin, A., Ferreres, F., Tomas Barberan, F.A., Gil, M., 2004. Characterisation and quantization of antioxidant constituents of sweet pepper (*Capsicum annuum* L.). Journal of Agricultural and Food Chemistry, 52(12): 3861-3869.

[6] Britton, G., Hornero- Mendez, D., 1997. Carotenoids and Colour in Fruits and Vegetables. In: Tomoas- Barberan, F. A., Robins, R.J. (Eds), Phytochemistry of Fruits and Vegetables. Clarendon Press, Oxford, England, p.11-28.

[7] Hornero- Mendez, D., Coasta- Garcia, J., Minguez- Mosquera, M.I., 2002. Characterisation of carotenoids high producing *Capsicum annuum* cultivars selected for paprika production. Journal of Agricultural and Food Chemistry, 50(20): 5711- 5716.

[8] Parez- Galvez, A., Martin, H.D., Sites, H., Stahl, W., 2003. Incorporation of carotenoids from paprika oleoresin into human chylomicron. British Journal of Nutrition, 89(6): 787-793.

[9] Peter, K.V., 1998. Recent advances in chilli breeding, Indian Species,35: 3-5.

[10] Kaur, N., Dhiman, J.S., Khurana, D.S., 2011. Physiological and biochemical traits analysis of Capsicum annuum L. germplasm for resistance to *Colletotrichum capsici*. Cell and Plant Science, 2(3): 12-21.

[11] Than, P.P., Prihasturi, H., Phoulivong. S., Taylor, P.W.J., Hyde, D., 2008. Chilli anthracnose disease caused by *Colletotrichum* species. J Zhejiang Univ Sci 9: 764-778.

[12] Poonpolgul, S., Kumphai, S., 2007. Chillli Pepper Anthracnose in Thailand. Country Report. In: Oh, D.G., Kim, K.T. (Eds.), Abstracts of First International Symposium on Chilli Anthracnose. National Horticultural Research Institute. Rural Development of Administration, Republic of Korea, p.23.

[13] Yoon, J.B., Yand, D,C., Lee, W.P., Ahn, S.Y., Park, H.G., 2004. Genetic resources resistant to anthracnose in the genus Capsicum. J Korean Soc Hort Sci, 45:318- 323.

[14] Hadden , J. F., Black, L.L., 1989. Anthracnose of Pepper caused by *Colletotrichum* spp. Proceeding of the International Symposium on Integrated Management Practises: Tomato and Pepper Production in the Tropics. Asian Vegetable Research and Development Centre, Taiwan, p. 189-199.

[15] Pakdeevaraporn, P., Wasee, S., Taylor, P.W.J., Mongkolporn, O., 2005. Inheritance of resistance to anthracnose caused by *Colletotrichum capsici* in *Capsicum*. Plant Breed, 124: 206-214.

[16] Bansal, R.D., Grover, R.K., 1969. Reaction on chilli (*Capsicum fruitenscens*) varieties to *Colletotrichum capsici*. Journal of Research PAU, 6: 345-348.

[17] Chowdhry, S., 1957. Studies on the development and control of fruit rot of chillies. Indian Phytopathology, 10: 55-62.
[18] Issac, S., 1992. Fungal Plant Interaction. Chapman and Hall Press, London, p.115.
[19] Manandhar, J.B., Hartman, G.L., Wang, T.C., 1995. Anthracnose development on pepper fruits inoculated with Colletotrichum gloeosporioides. Plant Disease, 79:380-383.
[20] Bailey , J.A., and Jeger, M., 1992. *Colletotrichum*: Biology, Pathology and Control. CAB International. Wallingford.
[21] Dean, R., Van, J.A.L., Pretorious, Z.A., Hammond- Kosack, K.E., Di Pietro, A., 2012. The top 10 fungal pathogens in molecular plant pathology. Molecular Plant Pathology, 13: 414-430.
[22] Freeman, S., Katan, T., Shabj, E., 1998. Characterisation of Colletotrichum species responsible for anthracnose disease of various fruits. Plant Dis, 82: 596-605.
[23] Lenne, J.M., 2002. Some major plant diseases. In: Plant Pathologist's Pocketbook (Waller, J.M., Lenne, M., Waller, S.J., eds). 3rd edn. CABI, Wallingford, UK: 4-18.
[24] Ritterband, D.C., Shah, M., Seedor, J.A., 1997. *Colletotrichum graminicola*: a new corneal pathogen. Cornea, 16: 362-364.
[25] Guarro, J., Svidzinski, T.E., Zaror, L., Forjaz, M.H., Gene, F., 1998. Subcutaneous hyalohyphomycosis caused by *Colletotrichum gloeosporioides*. Journal of Clinical Microbiology, 36: 3060- 3265.
[26] Shiraishi, A., Araki- Sasaki, A., Mitani, A., Miyamoto, H., Sunada, A., 2011. Clinical characteristics of Keratitis due to *Colletotrichum gloeosporioides*. Journal of Ocular Pharmacology and Therapeutics, 27: 487- 491.
[27] Shivaprakash, M.R., Appannanavar, S.B., Dhaliwal, M., Gupta, A., Gupata, S., 2011. *Colletotrichum truncatum*: an unusual pathogen causing mycotic keratitis and endophtalmitis. Journal of Clinical Microbiology, 49: 2894- 2898.
[28] Marine, C.A., Rhinehart, H.L., Sutton, D.A., Thompson, E.H., Rinaldi, MG., 2002. Journal of Clinical Microbiology, 40: 4273- 4280.
[29] Marcelino, J., Giordano, R., Gouli, S., Gouli, V., Parker, B.L., et al., 2008. *Colletotrichum* var. floriniae (telemorpj: *Glomerella acutata* var. floriniae var. nov) infection of a scale insect Mycologia, 100: 353-374.
[30] Marcelino, J., Gouli, S., Parker, B.L., Skinner, M., Giordano, R., 2009. Entomopathogenic activity of a variety of fungus, *Colletotrichum acutatum* recovered from elongate hemlock scale, Florina externa. 11 pp. Journal of Insect Science 9: article 13.
[31] Simonds, J.H., 1965. A study of the species of *Colletotrichum* causing ripe furit rots in Queenslands, Queensland Journal of Agriculture and Animal Sciense, 22: 437-459.
[32] Cannon, P.Fp., Bridge, P.D., Monte, E., 2000. Linking the past, present and future of *Colletotrichum* Synstematics. In: Prusky, D., Freeman, S., Dickman, M. (Eds.), *Colletotrichum*: Host specificity, Pathology and Host- Pathogen Interactions. APS Press, St. paul, Minnesota, p.1-20.
[33] Kim, K.H., Yoon, J.B., Park, H.G., Park, E.K., Kim, Y.H., 2004. Structural modifications and programmed cell death of chilli pepper fruit related to resistance responses to *Colletotrichum gloeosporioides* infection. Phytopathology, 94: 1295-1304.

[34] Hong, J.K., Hwang, B.K., 1998. Influence of inoculums density wetness duration, plant age, inoculation method, and cultivar resistance on infection of pepper plants by *Colletotrichum coccodes*. Plant Disease, 82(10): 1079-1083.

[35] Kim., K.D., Oh, B.J., Yang, J., 1999. Differential interactions of a *Colletotrichum gloeosporioides* isolate with green and red pepper fruits. Phytoparasitica, 27: 1-10.

[36] Park, H.K., Kim, B.S., Lee, W.S., 1990. Inheritance of resitance to anthracnose (*Colletotrichum* spp.) in pepper (*Capsicum annuum* L.) II. Genetic analysis of resitance to *Colletotrichum dematium*. Hort. Environ. Biotechnol, 31: 207-212.

[37] Than, P.P., Jeewon, R., Hyde, K.D., Pongsupasamit, S., Mongkolporn, O., Taylor, P.W.J., 2008b. Characterisation and pathogenecity of *Colletotrichum* species associated with anthracnose on chilli (Capsicum spp) in Thailand. Plant Pathol, 57: 562-572.

[38] Montri, P., Taylor, P.W.J., Mongkolporn, O.,2009. Pathotypes of *Colletotrichum capsici* the causal agent of chilli anthracnose in Thailand. Plant Dis. 93:17-20.

[39] Nicholson, R.L., Moraes, W.B.C., 1980. Survival of *Colletotrihcum graminicola*: importance of the spore matrix. Phytopathology, 70: 255-261.

[40] Pernezny, K., Roberts, P.D., Murphy, J.F., Goldberg, N.P., 2003. Compendium of Pepper Diseases. The American Phytopathological Society, St. Paul, Minnedota, p.73.

[41] Chitkara, S., Singh, T., Singh, D., 1990. Histophathology of *Colletotrichum dematium* infected chilli seeds. Acta Botanica Indica, 18: 226-230.

[42] Deising, H.B., Wernitz, M., (2000). The role of funal appressoria in plant infection. Microbes and Infection, 2: 1631-1641.

[43] Prusky, D., Plumbley, R.A., (1992). Quiscent infections of *Colletotrichum* in tropical and sub tropical furit. In: *Colletotrichum*. Biology, Pathology and Control (Bailey, J.A., Jeger, M.J., eds). CBAI, Wallingford, UK: 289-307.

[44] Joshee, S., Paulus, B.C., Park, D., Johnston, P.R., 2009. Diversity and distribution of fungal foliar endophytes in New Zealand Popocarpaceae Mycological Research, 113: 1003-1015.

[45] Rojas, E.I., Rehner, S.A., Samuels, G.I., Van Bael, S.A., Herre, E.A., et al., 2010. *Colletotrichum gloeosporioides* s.I. associated with Theobroma cacao and other plants in Panama: Multilocus Phylogenies distinguish pathogen and endophyte clades. Mycologia, 102: 1318-1338.

[46] Yuan, Z.L., Su, Z.Z., Mao, L.J., Peng, Y.Q., Yang, G.M., et al., 2011. Dustinctive endophytic fungal assemblage in stems of wild rice (*Oryza granulate*) in China with special reference to two species of Muscodor (Xylariaceae). Journal of Microbiology, 49: 15-23.

[47] Pring, R.J., Nash, C., Zakaria, M., Bailey, J.A., 1995. Infection process and host range of *Colletotrichum capsici*.Physiological and Molecular Plant Pathology, 46(2): 137-152.

[48] Von Arx, J.A., 1957. Die Arten der Gattung Colletotrichum Cda. Phytopathologicshe Zeitschrift, 29: 414-468.

[49] Smith, B.J., Black, L.L., 1990. Morphologicial, cultural and pathogenic variation among *Colletotrichum* species isolated from strawberry, Plant Disease, 74(1):69-76.

[50] Than, P.P., Jeewon, R., Hyde, K.D., Pongsupasamit, S., Mongkolporn, O., Taylor, P.W.J., 2007. Characterisation and pathogenecity of *Colletotrichum* species associated with anthracnose on chilli (*Capsium* spp.) in Thailand. Plant Pathology, 57: 562-572.

[51] Lubna Masoodi, Ali Anwar, Shahzad Ahmed, Sofi, T.A., 2013. Cultural, Morphological and Pathogenic Variability in Colletotrichum capsici causing Die- back and Fruit Rot of Chilli. Asian Journal of Plant Pathology 7(1): 29-41.

[52] Hartman, G.L., Wang, T.C., 1992. Characteristics of two Colletotrichum species and Evaluation of resistance to anthracnose in Pepper. Conference on Plant Protection in Tropics, Kuala Lumpur, Malaysian Plant Protection Society.

[53] Mitchell, J.R., Roberst, P.J., Moss, S.T., 1995. Sequence or structure? A short review on the application of nucleic acid sequence information to fungal taxonomy. Mycologist, 9: 67-75.

[54] Sreenivasaprasad, S., Mills, P., Brown, A., 1994. Nucleotide sequence of the rDNA spacer 1 enables identification of isolates of *Colletotrichum* as *C. acutatum*. Mycological Research, 98: 186-188.

[55] Sreenivasaprasad, S., Mills, P., Meehan, B.M., Brown, A., 1996. Phylogeny and systematic of 18 *Colletotrichum* species based on ribosomal DNA spacer sequences. Genome, 39(3): 499-512.

[56] Moriwaki, J., Tsukiboshi, T., Sati, T., 2002. Grouping of *Colletotrichum* species in Japan based on rDNA sequences. Journal of General Plant Pathology, 68(4):307-320.

[57] Photita, W., Taylor, P.W.J., Ford, R.,Lumyong, P., McKenzie, H.C., Hyde, K.D., 2005. Morphological and molecular characterization of *Colletotrichum* species from herbaceous plants in Thailand. Fungal Diversity, 18: 117-133.

[58] Sheu, Z., Chenand, J., Wang, T., 2007. Appllication of ITS- RFLP analysis for identifying *Colletotrichum* species associated with Pepper Anthracnose in Taiwan. In:Oh, D.G., Kim, K.T. (Eds.), Abstracts of the First International Symposium on Chilli Anthracnose. National Horticultural Research Institute, Rural Development of Administration. Republic of Korea, p.32.

[59] Ratanacherdchai, K., Wang, H.K., Lin, F.C., Soytong, K., 2007. RAPD analysis of *Colletotrichum* species causing chilli anthracnose disease in Thailand. Journal of Agricultural Technology, 3: 211-219.

[60] Flor, H.H., 1971, Current status of the gene- for –gene concept. Annu. Rev. Phytopath, 9: 275-296.

[61] Albersheim, P., Jones, T.M., English, P.D., 1969. Biochemistry of cell wall in relation to the infective processes. Annu. Rev. Phytopathol, 7: 171-194.

[62] Esquerre- Tugaye, M.T., Boudart,G., Dumar, B., (2000). Cell wall degrading enzymes, inhibitory proteins and oligosaccharides participate in the molecular dialogue between plant pathogens. Plant Physiol. Biochem, 38: 157-163.

[63] Darvill, A.G., Albersheim, P., 1984. Phytoalexins and their elicitors: A defense against microbial infection in Plants. Annu. Rev. Plant Physiol. 35: 243-275.

[64] Ryan, C.A., Farmer, E.E., 1991. Oligosaccharide signals in plants: A current assessment. Annu. Rev. Plant Physiol. Mol. Bio. 42: 651-674.

[65] Jha, G., Rajeshwari, R., Sonti, R.V., 2005. Bacterial type two secretion system secreted proteins: double- edged swords for plant pathogens. Mol. Plant Microbe Interact, 18: 891-898.

[66] Lalani, A.S.; Mc Fadden, G., 1999. “Evasion and exploitation of chemokines by viruses” Cytokine and Growth Factor Reviews, 10(3-4): 219-233.

[67] Haig, D.M., 2001. "Subversion and piracy: DNA viruses and immune evasion," Research in Veterinary Science, 70(10): 205-219.
[68] Keen, N.T., 1990. Gene- for- gene complementarity in plant- pathogen interactions. Annu Rev Genet, 24: 447-463.
[69] Dangl, J.L. and Jones, J.D.G., 2001, Plant Pathogens and integrated defense responses to infection. Nature, 441: 826-833.
[70] Roy, B.A., and Kirchner, J.W., 2000. "Evolutionary dynamics of pathogen resistance and tolerance," Evolution, 54(1): 51-63.
[71] Miller, M.R., White, A., Boots, M., 2005. "The evolution of host resistance: tolerance and control as distinct strategies," Journal of Theoretical Biology, 236 (2): 198-207.
[72] Keeling, P.J., 2009. "Functional and ecological impacts of horizontal gene transfer in eukaryotes." Current Opinion in Genetics and Development, 19 (6):613-619.
[73] Bailey, J.A., O' Connell, R.J., Pring, R.J., Nash, C., 1992. Infection strategies of Colletotrichum species. In Colletotrichum: Biology, Pathology, Control (J.A. Bailey and M.J. Jeger Eds.). pp. 88, 120. CAB International, Wallingford.
[74] O'Connelle, R.J., et al., 2000. Dissecting the cell biology of *Colletotrichum* infection processes. In *Colletotrichum*: Host specificity, Pathology, and Host Pathogen Interaction (Prusky, D. et al., Eds.).pp. 57-77.
[75] Perfect, S.E., and Green, J.R. (2001). Infection structures of biotrophic and hemibiotrophic fungal plant pathogens. Mol. Plant Pathol. 2: 101-108.
[76] Reedman et al., 1999. Conversion of the pathogenic fungus *Colletotrichum magna* to a non pathogenic endophytic mutualist by gene disruption. Mol. Plant Microbe Interact, 12: 969-975.
[77] Beno- Moualem, D., Prusky, D., 2000. Early events during quiescent infection development by *Colletotrichum gloeosporioides* in unripe avocado fruits. Phytopathology 90: 553-559.
[78] Roberts, P.D., Pernezny, K., Kucharek, T.A., 2001. Anthracnose caused by *Colletotrichum* sp. on pepper. Journal of University of Florida/ Institute of Food and Agricultural Sciences.
[79] Nicholson, R.L., 1996. Adhesion of fungal propagules. In Histology, Ultrastructure and Molecular Cytology of Plant-Microorganism Interactions (Nicole, M, and Gianinazzi-Pearson, V., Eds.) pp.117-134. Kluwer Academic Publishers, Dordrecht/ Norwell, MA.
[80] Mercure, E.W., Leite, B., and Nicholson, R.L. 1994. Adhesion of un germinated conidia of *Colletotrichum graminicola* to artificial hydrophobic surfaces. Physiol. Mol. Plant Pathol, 45: 421- 440.
[81] Nicholson, R.L. 1992. *Colletotrichum graminicola* and the anthracnose disease of corn and sorghum. In *Colletotrichum*: Biology, Pathology and Control (J. A. Bailey and M.J. Jeger, Eds.), pp. 186- 202. CAB International. Wallingford.
[82] Young, D.H., and Kauss, H., 1984. Adhesion of *Colletotrichum lindemuthianum* sportes to Phaseolus vulgaris hypocotyls to polystyrene. Appl. Env. Microbiol. 47: 616- 619.
[83] Sela- Buurlage, M.B., Epstein, L., and Rodriguez, R.J. 1991. Adhesion of ungerminated *Colletotrichum musae* conidia. Physiol Mol. Plant Pathol, 39: 345-352.

[84] Mercure, E.W., Kunoh, H., and Nicholson, R.L., 1994a. Adhesion of *Colletotrichum graminicola* conidia to corn leaves: A requirement for disease development. Physiol. Mol. Plant Pathol. 45: 407-420.

[85] O' Connell, R.J., Pain, N.A., Hutchison, K.A., Jones, G.L. and Green, J, R., 1996. Ultrastructure and composition of the cell surfaces of infection structures formed by the fungal plant pathogen *Colletotrichum lindemuthianum*. J. Microsc. 181: 204-212.

[86] Wilawan, K., Somsiri, S., Sutevee, S., 2008. Effect of Capsaicin on germination of Colletotrichum capsici conidia. Kasetsart J (Nat. Sci) 42:417-422.

[87] Kim, J.H., Lee, J.H., Choi, W., 2013. Identification of genes expressed during conidial germination of the pepper anthracnose pathogen, *Colletotrichum acutatum*. Journal of Life Science, 23(1): 8-14.

[88] Umahara, T., Uchihara, T., Tsuchiya, K., Nakamura, A., Iwamoto, T., (2007). Intranuclear localization and iso-form dependent translocation of 14-3-3 proteins in human brain with infarction. J. Neurol Sci 260: 159-166.

[89] Perfect, S., Hughes, H., O'Connell, R., Green, J., 1999. *Colletorichum*: a model genus for studies on pathology and fungal-plant interactions. Fungal Genetics and Biology,27: 186-198.

[90] Podila, G.K., Rogers, L.M., and Kolattukudy, P.E. 1993. Chemical signals from avocado surface wax trigger germination and appressorium formation in *Colletotrichum gloeosporioides*. Plant Physiol. 103: 267- 272.

[91] Kolattukudy, P.E., Rogers, L.M., Li, D., Hwang, C.S., and Flaishman, M.A., 1995. Surface signaling in pathogenesis. Proc. Natl. Acad. Sci. USA. 92: 4080-4087.

[92] Yang, Z., Dickman, M.B., 1997. Regulation of cAMP and cAMP dependent protein kinase during conidial germination and appressorium formation in Colletotrichum trifolii. Physiol. Mol.. Plant Pathol. 50: 117- 127.

[93] Herbert, C., O'Connell, R., Gaulin, E., Salesses, V., Esquerre- Tugaye, M., Dumas, B., 2004. Production of cell wall- associated endopolygalacturonase by *Colletotrichum lindemuthianum* and pectin degradation during bean infection. Fungal Genetics and Biology, 41:140-147.

[94] Acosta- Rodriguez, I., Pinon- Escobedo, C., Zavala- Paramo, M., Lopez- Romero, E., Cano-Camacho, H., 2005. Degradation of cellulose by the bean pathogenic fungus *Colletotrichum lindemuthianum*. Production of extracellular cellulolytic enzymes by cellulose induction. Antonie van Leeuwenhoek, 87: 301-310.

[95] Wharton, P.S., Dieguez- Uribeondo, J., 2004. The biology of *Colletotrichum acutatum.* Anales del Jardin Botanico de Madird, 61: 3-22.

[96] Gueddari, N.E., Rauchhaus, U., Moerschbacher, B.M., Deising, H.B., 2002. Developmentally regulated conversion of surface exposed chitin to chitosan in cell walls of plant pathogenic fungi. New Phytol, 156: 103-112.

[97] Mendgen, K., Hahn,M., 2002. Plant infection and the establishment of fungal biotrophy. Trends Plant Sci, 7: 352-356.

[98] Thines, E., Aguirre, J., Foster, A.J., Deising, H.B., 2006. Secondary metabolites as virulence determinants of fungal plant pathogens. In: Esser, K., Luttge, U.E., Beyschlag, W., Murata, J., editors. Progress in botany. Berlin, Heidelberg, Springer; p. 132-159.

[99] Bailey, J.A., 1987. Phytoalexins: A Genetic View of Their Significance. In: Day, P.R., Jellis, G.J. (Eds.), Genetics and Plant Pathogenesis, Blackwell, Oxford, p. 13-26.
[100] Agrios, G.N., 2005. Plant Pathology, 5th Ed. Academic Press. San Diego,p.922.
[101] Roberts, P.D., Pernezny, K., Kucharek, T.A., 2001. Anthracnose caused by *Colletotrichum* sp. on pepper. Journal of University of Florida/ Institute of Food and Agricultural Sciences.
[102] Jayasekhar, M., Eswaramurthy, S., Natarajan, S., 1987. Effect of certain fungicides on chilli fruit rot. Madras Agricultural Journal, 74: 10-11, 479-480.
[103] Hedge, G.M., Srikanth, K., Kulkarni, S., 2001. Vulnerable infection stage of chilli fruit by *Colletotrichum capsici* (sydow) Butler and Bisby. Karnataka Journal of Agricultural Sciences, 14: 162- 163.
[104] Ekbote, S.D., 2002. Bioefficacy of copper hydroxide (Coxid) against anthracnose of chilli. Karnataka Journal of Agricultural Sciences, 15: 729-730.
[105] Smith, K.L. 2000. Peppers. In: Presheur, R.J. (Eds.), Ohio Vegetable Production Guide. Ohio State University Extension. Columbus, Ohio, p.166-173.
[106] Chakravarthy, B.P., Anil Kumar, T.B., 1975. Control of seed borne infection of *Colletotrichum capsici* in chillies. Curr. Res., 4: 172.
[107] Shetty, T.A.S., Uthaih, B.C., Rao, K.B. and Indiresh, K.M., 1988, Chemical control of seed microflora on chilli. *Pl. Path. Newslet.,* Univ. Agric. Sci., Dharwad, 6: 22.
[108] Kumudkumar, Singh, J. and Khare, A., 2004, Detection, location transmission and management of seed borne *Colletotrichum dematium* causing dieback and anthracnose in chilli. *Farm Sci. J.,* 13(2): 152-153.
[109] Rathee, P.S., Mishra, S.H. and Kaushal, R., 1982, Antimicrobial activity of essential oil, fixed oil and unsaponifiable matter of *Nigella sativa* Linn. *Indian J. Pharmac. Sci.,* 44: 8-10.
[110] Shivapuri, A., Sharma, O.P. and Jhamaria, S.L., 1997, Fungitoxic properties of plant extracts against pathogenic fungi. *J. Mycol. Pl. Path.,* 27: 29-31.
[111] Singh, S.N., Yadav, B.P. Sinha, S.K. and Ojha, K.L., 1997, Efficacy of plant extracts in inhibition of radial growth and spore germination of *Colletotrichum capsici*. *J. Appl. Biol.,* 7(1/2): 58-61.
[112] Jayalakshmi, C., Durairaj, P., Seetharaman K., Sivaprakasam, K., 1998. Biocontrol of fruit rot and die back of chilli using antagonistic microorganisms. Indian Phytopathology, 51: 180-183.
[113] Korpraditskul, V., Rattanakreetakul, C., Korpraditskul, R., Pasabutra, T., 1999. Development of Plant Active Substances from Sweetflag to Cotrol Fruit Rot of Mango for Export. In: Proceedings of Kasetsart University Annual Conference. Kasetsart University, Bangkot, p.34.
[114] Gomathi, V. and Kannabiran, B., 2000, Inhibitory effects of leaf extracts of some plants on the anthracnose fungi infecting *Capsicum annuum* L. *Indian Phytopath.,* 53: 305-308.
[115] Chitra, H. and Kannabiran, B., 2000, Antifungal effect of *Datura innoxia* on anthracnose fungus *Colletotrichum capsici in vitro*. *Adv. in Pl. Sci.,* 14: 317-320.
[116] Ramamoorthy, V. and Samiyappan, R., 2001, Induction of defense related genes in *Pseudomonas fluorescens* treated chilli plants in response to infection by *Colletotrichum capsici*. *J. Mycol. Pl Path.,* 31(2): 146-155.

[117] Hegde, G.M., Anahosur, K.H. and Srikant Kulkarni, 2002, Biological control of *Colletotrichum capsici* causing fruit rot of chilli. *Pl. Path. Newslet.,* 20: 4-5.
[118] Kumaran, R.S., Gomathi, V. and Kannabiran, B., 2003, Fungitoxic effects of root extracts of certain plant species on *Colletotrichum capsici* causing anthracnose in *Capsicum annuum*. *Indian Phytopath.,* 56(1): 114-116.
[119] Ekbote, S.D., 2005, Effect of *Pseudomonas fluorescens* on anthracnose of chilli caused by *Colletotrichum capsici*. *Karnataka J. Agric. Sci.,* 18(1): 162-165.
[120] Jayelakshmi, C., Seetharaman, K., 1998. Biological controls of fruit rot and die back of chilli with plant products and antagonistic microorganisms. Plant Disease Research, 13:46-48.
[121] Lin, Q., Kanchana- udomkarn, C., Jaunet, T., Mongkolporn, O., 2002. Genetic analysis of resistance to pepper anthracnose caused by *Colletotrichum capsici*. Thai J. Agric. Sci. 35: 259-264.
[122] Voorrips, R.E., Finkers, R., Sanjaya, L., Groenwold, R., 2004. QTL mapping of anthracnose (Colletotrichum spp.) resistance in a cross between *Capsicum annuum* and *C. chinense*. Theor. Appl. Genet. 109: 1275-1282.
[123] AVRDC. 1999. Studies on pepper anthracnose. In AVRDC Report 1998, Shanhua, Taiwan: AVRDC- the world vegetable centre. Pp.27-30.
[124] Yoon, J.B., Yand, D.C., Lee, W.P., Ahn, S.Y., Park, H.G., 2004. Genetics resources resistant to anthracnose in the genus Capsicum. Journal of Korean Society and Horticultural Science, 45: 318-323.
[125] AVRDC. 2003. Host resistance to pepper anthracnose. In AVRDC Report 2002, Shanhua, Taiwan: AVRDC- the world vegetable centre, pp 29-30.
[126] Mahasuk, P., Taylor, P.W.J., Mongkolporn, O., 2009. Identification of two new genes conferring resistance to *Colletotrichum acutatum* in *Capsicum baccatum*.The American Phytopahological Society, 99(9): 1100-1104.
[127] Yoon, J.B., Park, H.G., 2005. Trispecies bridge crosses (*Capsicum annuum x C. chinense*) x *C. baccatum*, as an alternative for introgression of anthracnose reistance from *C. baccatum* into *C. annuum*. Hort. Environ. Biotechnol. 46: 5-9.
[128] Kim, S.H., Yoon, J.B., Do, J.W., Park, H.G., 2007. Resistance to anthracnose caused by *Colltetorichum acutatum* in chilli pepper (*Capsicum annuum* L.) J. Crop. Sci. Biotech. 10: 277-280.
[129] Kim, S.H., Yoon, J.B., Do, J.W., Park, H.G., 2008. A major recessive gene associated with anthracnose resistance to *Colletotrichum capsici* in chilli pepper (*Capsicum annuum* L.). Breeding Science, 58: 137- 141.
[130] Lee, J., Hong, J.H., Do, J.W., Yoon, J.B., 2010. Identification of QTLs for resistance to anthracnose to two *Colletotrichum* species in pepper. J. Crop. Sci. Biotech. 13:227-133.
[131] Lee, J., Do, J.W., Yoon, J.B., 2011. Development of STS Markers Linked to Major QTLs for resistanct to Pepper Anthracnose Caused by *Colletotrichum acutatum* and *C. capsici*. Hort. Environ, Biotechnol. 52(6): 596-601.
[132] Dong, X., 2001. Genetic dissection of systemic acquired resistance. Curr. Opin. Plant Biol.4: 309-314.
[133] Metraux, J.P. 2001. Systematic acquired resistance and salicylic acid: current state of knowledge. Eur. J. Plant Pathol. 107: 13-18.

[134] Pink, D.A.C., 2002. Strategies using genes for non-durable resistance. Euphytica, 1:227-236.
[135] Martin, G.B., Bogdanove, A.J., Sessa, G., 2003. Understanding the functions of plant disease resistance proteins. Annu. Rev. Plant Biol. 54:23-61.
[136] Pink, D., Puddephat, I.I., 1999. Development of disease resistance genes by plant transformation- a mix and match approach. Trends Plant Sci. 4: 71-75.
[137] Tai, T.H. et al., 1999. Expression jof the Bs2 pepper gene confers resistance to bacterial spot disease in tomato. Prox. Natl. Accad. Sci. U.S.A. 96: 14153-14158.
[138] Horvath, H. et al., 2003. Genetically engineered stem rust resistance in barley using the Rpg1gene. Proc. Natl. Acad. Sci. U.S.A. 100: 364-369.
[139] Brueggeman, R. et al., 2002. The barley stem rust- resistance gene Rpg1 is a novel disease-resistance gene with homology to receptor kinases. Proc. Natl. Acad. Sci. U.S.A. 99: 9328-9333.
[140] Hulbert, S.H. et al., 2001. Resistance gene complexes: evolution and utilization. Annu. Rev. Phytopathol. 39: 285-312.
[141] Bai, J., Pennill, L.A., Ning, J., Lee, S.W., Ramalingam, J., Webb, C.A., Zhao, B., Sun, Q., Nelson, J.C., Leach, J.E., 2002. Diversity in nucleotide binding site-leucine-rich repeat genes in cereals. Genome Res,12:1871-1884.
[142] Cannon, S.B., Zhu, H., Baumgarten, A.M., Spangler, R., May, G., Cook, D.R., Young, N.D., 2002. Diversity, distribution and ancient taxonomic relationships within the TIR and non-TIR NBS-LRR resistance gene subfamilies. J. Mol. Evol, 54:548-562
[143] He, C.Y., Tian, A.G., Zhang, J.S., Zhang, J.Y., Gia, J.Y., Chen, S.Y., 2003. Isolation and characterization of a full length resistance gene homolog from soybean. Theor. Appl. Genet. 106: 786-793.
[144] Radwan, O., Bouzidi, M.F., Vear, F., Philippon, J., Tourvieille Labrouche, D., Nicolas, P., Mouzeyar, S., 2003. Identification of non TIR- NBS- LRR markers linked to the PI5/PI8 locus for resistance to downy mildew in sunflower. Theor. Appl. Genet. 106: 1438-1446.
[145] Yan, G., Chen, X., Line, R., Wellings, C., 2003. Resistance gene analog polymorphism markers co-segregating with the YR5 gene for resistance to wheat stripe rust. Theor. Appl. Genet. 106: 636-643.
[146] Leister, R.T., Ausubel, F.M., Katagiri, F., 1996. Molecular recognition of pathogen attack occurs inside of plant cells in plant disease resistance specified by the *Arabidopsis* genes RPS2 and RPM1. Proceedings or the National Academy Sciences, Washington, 93: 15497-15502.
[147] Ferrier- Can, E., Geffroy, V., Macadre, C., Creusot, F., Imbert- Bollere, P., Sevignac, M., Langin, T., 2003. Characterisation of expressed NBS-LRR resistance gene candidates from common bean. Theor. Appl. Genet, Berlin, 106(2): 251-261.
[148] Leister, R.T., Katagire, F., 2000. A resistance gene product of nucleotide binding site-leucine rich repeats class can form a complex with bacterial avirulence proteins in vivo. Plant J, 22: 345-354.
[149] Guiterrez, N., Gimenez, M.J., Torres, A.M., Atienza, S.G., Avila, C.M., Palomino, C.,2012. Upregulation of resistance gene analogs (RGA) in chickpea in the early response to Fusarium wilt. Euphytica. 186: 793-804.

[150] Leng, X.D., Xiao, B.G., Wang, S., Gui, X.J., Wang, Y., Lu. X.P., Xie, J.H., Li, Y.P., Fan, L.G., 2010. Identification of NBS-type resistance gene homologs in tobacco genome. Plant Mol. Biol. Rep. 28:152-161.

[151] Young, N. D., 2000. The genetic architecture of resistance. Curr. Opin. Plant Biol.3: 285-290.

[152] Egea- Gilabert, C.,Dickinson, M.J., Bilotti, G., Candela, M.E., 2003. Isolation of Resistance Gene Analogs in Pepper using Modified AFLPs. Biologia Plantarum, 47(1): 27-32.

[153] Wan, H., Yaun, W., Ye, Q., Wang, R., Ruan, M., Li, Z., Zhou,G., Yao, Z., Zhao, J., Liu, S., Yang, Y., 2012. Analysis of TIR- and non TIR-NBS-LRR disease resistance gene analogs in pepper: Characterisation, genetic variation, functional divergence and expression patterns. BMC Genomics, 13:502.

[154] Chen, X.M., Line, R.F., Leung, H, 1998. Genome scanning for resistance- gene- analongs in rice, barley, and wheat by high resolution electrophoresis. Theor Appl Genet. 97: 345-355.

[155] Vos, P., Rene Hogers, Majro Bleeter, Martin Reijans, Theo Van de Lee, Miranda Horner, Adrie Frijtes, Jerina Pot, Johan Peleman, Martin Kuiper and Marc Zabeau, 1995. AFLP: a technique for DNA fingerprinting. Nucleic Acids Research, 23(21): 4407-4414.

[156] Paran, I., Michelomore, R.W., 1993. Development of reliable PCR- based markers linked to downy mildew resistance genes in lettuce. Teheor. Appl. Genet. 85: 985- 993.

[157] Konieczny, A., Ausuble, F.M., 1993. A procedure for mapping *Arabidopsis* mutations using co- dominant ecotype- specific PCR based markers. Plant J. 4: 403- 410.

[158] Maiti, S., Sen, C., 1979. Fungal Diseases of Betel Vine. Proceedings of National Academy of Science of the United States of America. Vol. 25, p. 150-157.

[159] Paul, Y.S., Behl, M.K., 1990. Some studies on bell pepper anthracnose caused by Colletotrichum capsici and its control. Seed Research, 1: 656-659.

[160] Park, K.S., Kim, C.H., 1992. Identification, Distribution and Etiological Characterization of anthracnose fungi of red pepper in Korea. Korean Journal of Plant Pathology, 8: 61-69.

[161] Dastur, J.F., 1920. Glomerella cingulata (Stoneman) Spald and its conidial form, Gloeosporium piperatum E. and E. and Colletotrichum nigrum E. and Hals. On chillis and Carica papaya. Annals of Applied Biology, 6(4): 245-268.

[162] Pearson, M.N., Bull, P.B., Speke, H., 1984. Anthracnose of Capsicum in Papua, New Guinea, varietal reaction and associated fungi. Tropical Pest Management, 30: 230-233.

[163] Johnson, P.R., Jones, D., 1997. Relationships among Colletotrichum isolates from fruit rots assessed using rDNA sequences. Mycologia, 89(3): 420-430.

[164] Manandhar, J.B., Hartman, G.L., Wang, T.C., 1995. Anthracnose development on pepper fruits inoculated with *Colletotrichum gloeosporioides*. Plant Disease, 79: 380-383.

[165] Adikaram, N.K.B., Brown, A., Swinburne, T.R., 1983. Observations on Infection of Capsicum annuum fruit by Glomerella cingulata and Colletotrichum capsici. Transactions of British Mycological Society, 80:395-401.

[166] Don, L.D., Van, T.T., Phuong Vy, T.T., Kieu, P.T.M.,2007. Colletotichum spp. Attacking on Chilli Pepper Growing in Vietnam. Country Report. In: Oh, D.G., Kim, K.T., (Eds.), Abstracts of First Internationaal Symposium on Chilli Anthracnose. National Horticultural Research Institur, Rural Development of Administration, Republic of Korea, p.24.