BIOSCIENCES BIOTECHNOLOGY RESEARCH ASIA, August 2015. Vol. **12**(2), 1233-1246

Colletotrichum gloeosporioides: An Anthracnose Causing Pathogen of Fruits and Vegetables

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DOI: http://dx.doi.org/10.13005/bbra/1776

(Received: 30 October 2014; accepted: 10 December 2014)

Colletotrichum species **are present in both tropical and subtropical regions of the world. But *Colletotrichum gloeosporioides* is most important pathogen and belongs to order melanconiales. The complete genome of this pathogen is not yet sequenced but various genes are identified which involved in pathogenesis and host defense. The optimum temperature for growth of this pathogen is 25-28°C, and pH 5.8-6.5. It is usually inactive in dry season but during favorable conditions it causes anthracnose disease to large number of economic crops amongst which mango anthracnose is important as far as losses caused by pathogen is concerned. First of all pathogen establish interaction with host by producing melanized appressorium and then penetrate the host cuticle. After penetration, infection vesicles and primary hyphae are formed. Later, secondary hyphae developed and spread to kill the host cell. *Colletotrichum gloeosporioides* follows the hemibiotrophic mode of infection where, biotrophic and necrotrophic phases are sequentially occur. The pathogen produced lesions on leaves, fruit and other parts of plant. Finally these lesions become dark and form concentric ring pattern. *Colletotrichum gloeosporioides* is also known to infect humans but only few incidents of such infections are known. A number of fungal genes have been identified using mutant screen, which plays role in different stages of infection and can be used as potential targets to devise strategies for controlling anthracnose disease in fields. This review focuses on up to date knowledge of all aspects of *C. gloeosporioides* biology.**

Key words: *Colletotrichum gloeosporioides*, anthracnose, pathogenicity genes.

Colletotrichum gloeosporioides is a ubiquitous pathogen. It belongs to the order melanconiales. This fungus infects monocotyledons (turf grass) to higher dicothyledons (cashew trees). *C. gloeosporioides* is widely distributed and common plant pathogen in the world (Sutton, 1992; Cannon *et al*., 2000). The fungus is more abundant in tropical and subtropical regions than in temprate (CAB international 2005).This pathogen infects about 470 different host genera. The pathogen also causes post-harvest problems (Prusky and Plumbley,1992) and also act as endophytic strains which are isolated from symptomless plant parts (Cannon and Simmons, 2002; Lu *et al*., 2004; Photita *et al*., 2004, 2005).

C. gloeosporioides was proposed for the first time as *Vermicularia gloeosporioides* by Penzig 1882. *C. gloeosporioides* was first reported at Deodoro, Brazil in 1937 on *S. humilis* and in India, it was first reported by Butler 1918 on coffee. *Glomerella cingulata* is the sexual stage (teleomorph) while the asexual stage (anamorph) is called *C. gloeosporioides* (Schrenk

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and Spaulding, 1903). There are various species come under genus *Colletotrichum* but only *C. graminicola* and *C. higginsianum* genomes were completely sequenced. *C. gloeosporioides* genome is under study but various genes have been identified which involve in pathogenesis and host defense mechanism.

It requires 25-28°C temperature, pH 5.8-6.5 for better growth. This pathogen is inactive in dry season and switches to active stages when encountered favorable environmental conditions. It involves hemibiotrophic mode of infection where both phases, biotrophic and necrotrophic phases occur sequentially. Various medium preparations were employed for the growth and sporulation of *C. gloeosporioides* including Potato dextrose agar, lima bean agar, malt extract agar and oat meal agar.

Traditionally the identification and characterization of *Colletotrichum* spp were relied on differences in morphology features such as colony color, size, and shape of conidia and appressorium, optimal temperature for growth, growth rate, presence or absence of setae (Von Arx, 1957; Smith and Black, 1990; Gunnel and Gubler, 1992; Sutton, 1992). Now molecular techniques provide alternative methods for taxonomic studies and are important tools in solving the problem of species delimitation (Maclean *et al*., 1993)

Germination in *C. gloeosporioides* follows two routes: "pathogenic" and "saprophytic" (Barhoom and Sharon, 2004). Pathogenic germination takes place on plants or on a hydrophobic surface and is characterized by fast mitosis followed by development of a single germ tube. This process is initiated immediately and results in the formation of appressoria. Saprophytic germination occurs in rich medium. It takes a much longer period of time and is characterized by development of two germ tubes that emerge from opposite sides of the spore. These germ tubes do not form appressoria, and these germinated spores do not infect plants. These two germination routes in *C. gloeosporioides* are regulated by different signaling pathways such as: saprophytic germination involved cAMP pathways while pathogenic germination is cAMP independent.

C. gloeosporioides causes anthracnose disease on a wide variety of fruits, including almond, avocado, apple, Arabica coffee, guava, mango, strawberry, papaya, banana, passion fruit, citrus, grapes and cashews (Simmonds, 1965; Hartill, 1992 ; Alahakoon *et al*., 1994 ;Timmer *et al*., 1998; Agwana *et al*., 1997; Freeman *et al*., 1998; Martinez-Culebras *et al*., 2000, 2003; Sanders and Korsten 2003; Xiao *et al*., 2004; Amusa *et al*., 2005; Nelson 2008). It causes considerable damage to large number of crops such as cereals, coffee, legumes (Bailey and Jeger, 1992;Lenne, 1992) and tropical, subtropical fruits such as avocado, banana, mango (Mordue, 1967; Jeffries *et al*., 1990). *Colletotrichum spp* are also found on decaying wild fruits (Tang *et al*., 2003). Under a high concentration of CO_2, there is increase in fecundity (spores produced per lesion area) observed and this may increase the severity and spread of disease (Chakraborty and Datta, 2003)

Colletotrichum species that cause serious plant disease are also commonly isolated as endophytes from healthy plants, and have been identified as saprobes on dead plant material (Photita *et al*., 2001, 2004; Promputtha *et al*., 2002; Toofanee and Dulymamode, 2002; Kumar and Hyde, 2004). The symptoms such as small, dark lesions appear on leaves, fruits and flowers of the infected plant which finally produce concentric ring pattern.

Phylogenetic relationships in *Colletotrichum* genus is successfully achieved by using ITS1 and ITS2 regions (Sherriff *et al*., 1994; Sreenivasaprasad *et al*., 1996; Freeman *et al*., 2001; Hsiang and Goodwin, 2001; Denoyes Rothan *et al*., 2003; Martinez-Culebras, *et al*., 2000, 2003).

Morphology

There are more than 600 synonyms of *C. gloeosporioides* showed many morphological and physiological variations reported by Von Arx (1957). Palo (1932), described the morphology of the fungus and the spores were irregular and appear as brown to black dots. The acervuli were highly variable in size, shape and exude pink masses of conidia when mature under moist conditions (Sattar and Malik, 1939).

Conidia were straight, cylindrical and oval and borne on distinct well developed hyaline conidiophores (Sattar and Malik, 1939). Bose *et*

al., 1973, observed the size of conidia varied from 11-16 x 4-6 µm and 13.8 x 4.8 µm, broad oblong with rounded ends 14.0 x 3.7 µm reported by Simmonds, 1965, formae speciales of *C. gloeosporioides* was observed by Sutton, 1992 and also recognized the species as a heterogeneous group with a great variation in morphology.

Ji and Guo, 1992 described the current method for the detection and identification of *C. gloeosporioides* and *C. oleifera*. This method depends on isolation of pure cultures on nutrient media followed by morphological examination of the isolates.

Baxter *et al*., 1985 defined *C. gloeosporioides* aggregate by using morphological methods and reported that conidia were cylindrical with rounded ends and less than 4.5 ¼m in diameter. These features are not reliable because *Colletotrichum spp* frequently produce different shape and sizes secondary conidia.

Environment condition for growth of pathogen

Environmental conditions favors the pathogen growth are temperature, 25-28° C being optimum, pH range of 5.8 to 6.5 and high humidity. Activity of this pathogen depend upon weather, *Colletotrichum* is inactive in dry season. Ponte 1996, observed that sunlight, low humidity and temperature extremes (below 18^0 C or greater than 25^0) rapidly inactivate spores. Spores are released from acervuli when there is an abundance of moisture. The pathogen persists on and in seed, trash and weed hosts and is dispersed locally by water splash, air currents, insects, or other forms of contact (CAB international 2005 crop protection compendium). In general, infection is favored at temperatures ranging from 20 to 30°C. Davis *et al*., 1987 reported the range between 20-30°C as the optimum temperature for the growth and sporulation of *C. gloeosporioides* on mango.

C. gloeosporioides required free water or relative humidity above 95 per cent for conidial germination and appressorium formation. Pandey 2011, observed that temperature and moisture requirements for infection have also been used to build forecasting systems for mango anthracnose a vital component for the disease management.

***In vitro* culture**

Various growth parameters of *C. gloeosporioides* were studied using solid media such as effect of concentration and composition of media, inoculums density and temperature on the spore carrying capacity and microcycle conidiation. Slade *et al.*, 1987, compared spore production of *C. gloeosporioides* on solid media with liquid media.

C. gloeosporioides grow well on PDA (potato dextrose agar) and CWA (coconut watery endosperm) which contain appropriate amounts of carbohydrates, proteins, minerals and lipids (Santoso *et al*., 1996).

Aerial mycelium growth is better on the Richard's, Brown's agar and better sporulation occur on oat meal, corn meal agar along with abundant development of acervuli in rings and few setae in *C. gloeosporioides.* Glutamic acid and alanine supported maximum growth and sporulation of *C. gloeosporioides*. The growth is completely inhibited at 10°C.

Light is not necessary but enhance sporulation, pH 6 (for growth and sporulation) and germination is better on a more acidic medium. Czapek's and yeast extract agar medium give maximum growth.

Mode of infection

C. gloeosporioides follows the hemibiotrophic mode of infection where, biotrophic and necrotrophic phases are sequentially occur. First of all pathogen establish interaction with host by producing melanized appressorium and then penetrate the host cuticle. After penetration, infection vesicles and primary hyphae are formed. These structures are somewhat similar to haustoria (formed by powdery mildews and rust fungi) do not cause any harm to host. This stage of infection is called biotrophic phase. Later, necrotrophic secondary hyphae developed and spread to kill the host cell (Munch *et al*., 2008)

Anthracnose caused by *Colletotrichum gloeosporioides*

C. gloeosporioides causes anthracnose disease to variety of crops worldwide. It is a disease of the foliage, stems, fruits and causes pre-harvest and post-harvest losses in mango, papaya, guava, custard apple, pomegranate and other subtropical fruit crops. Anthracnose is favored by wet, humid, warm conditions and

spread by infected seeds, rain splash and moist winds. It often result in fruit drop and fruit rot (*infonet-biovision.com*).

Anthracnose is caused by fungi that produce conidia within black fungal fruiting bodies called acervuli. Other species are also responsible for most anthracnose disease. First, lesion appears as small, dark spots on stolons and petioles. With age these lesions become large in diameter. Brownish areas are formed by the conidial masses that cover the lesion center and are frequently produced in a concentric ring pattern (Ponte, 1996) (*infonet-biovision.com*).

Host range and crop loss

Life cycle of this pathogen starts by germination of spores on the plant surface to form melanized infection structures called appressoria followed by penetration of host tissue. At this point thick infection hypaes are produced in primary infected cells, this stage is called as biotrophic stage of infection. After this the fungus suddenly switches to necrotrophic phase of infection which is characterized by formation of thin secondary hyphaes, which originated from the primary hyphaes and it is these secondary hyphae which starts colonizing the nearby cells, and ultimately leads to development of visible lesions at the site of infection. Finally the spores are formed on the surface of infected tissue and then they are dispersed by insect, air current and water splash to start another infection cycle.

C. gloeosporioides infects about 470 different host genera but some economic important crops such as: avocado, mango, beans, cashews, cassava, citrus plant, cotton, cow-pea, cucumber, eggplant, green gram, mango, onion, pepper, pumpkin, papaya, sorghum, soybean, tomato, watermelon, wheat, yam, zucchini/ courgette, cucurbit, cereals, legumes and spinach. Amongst them mango anthracnose is very important from Indian prospective.

Anthracnose caused by *C. gloeosporioides* was reported from several parts of the world. Bitter rot of apple *(Malus sylvestris* Mill) caused by *Glomerella cingulata* and *C. gloeosporioides* was reported in North Carolina orchards. The disease was first observed during the end of June and may cause 100% fruit rot by mid-august (Shane and Sutton, 1981).

Fruit rot of apple and pear was caused by *C. gloeosporioides* and *C. acutatum* in the Southern, Central and Mid Atlantic regions of the United States and in most countries where these fruits are grown (Sutton 1990).

C. gloeosporioides was reported to cause both pre and post harvest anthracnose on avocado in several countries including Australia (Fitzell, 1987), Israel (Binyamini and Nadel, 1972), South Africa (Darvas and Kotze, 1987) and Sri Lanka (Sivanathan and Adikaram, 1989) primarily as a quiescent pathogen (Jefferies *et al*., 1990).

Almond and avocado are also reported to be infected by *C. gloeosporioides* in Israel (Binyamini and Nadel, 1972; Shabi and Katan, 1983). This fungus acts as a post-harvest pathogen in avocado and in almond it infect the young fruits). Prusky and Saka, 1989; Prusky *et al*., 1991 observed that germination and appressorium formation of *C. gloeosporioides* spores in avocado fruits may be triggered by chemical signals from the surface wax .The pathogen causes severe yield losses and having different optimal growth temperature (Freeman *et al*., 1995). The site of infection in avocado is primarily the fruits, but infections may also appear on leaves and stems but it does not attack avocado flowers (Nelson, 2008). Anthracnose caused by *C. gloeosporioides* was also reported on avocado in Australia, South Africa (Giblin and Coates, 2007) and banana (Jeger *et al*., 1995).

In Belize, the three strains of *C. gloeosporioides* such as: *cgm*, *cgc* and *cgp* was observed on citrus. *cgm* and *cgc* were non-pathogenic to citrus flowers (Fagan, 1980). In Florida, the fast growing and slow growing strains of *C. gloeosporioides* caused post bloom fruit drop (Sonoda and Pelosi, 1988). *C. gloeosporioides* also causes infection on Dragon fruit (Hylocereus *spp*.) in Peninsular Malaysia (Masyahit *et al*., 2009).

Trichosanthes kirilowii Maxim, a species within the gourd family, is cultivated in China for its edible seeds and medicinal roots. In 2000, there was a heavy loss due to fruit rot caused by *C. gloeosporioides* (Li and Zhang, 2007).

Anthracnose is highly destructive disease of *lupins*. It was first found in Western Australia in 1996 and infect almost all species of *lupins.*

But 10-100% crop loss was observed in albus *lupins* and 10-50% in narrow leafed *lupins* (www.hannafords.com).

Post harvest disease of mango was also reported to be caused by *C. gloeosporioides* (Ploetz and Prakash, 1997). It was first reported from Puerto Rico (Collins, 1903) and later from Hawaii (Higgins, 1906), Florida (Fawcett, 1907), Cuba (Cardin, 1910), Philippines (Wester, 1911), Columbia (Taro, 1929), South Africa (Doidge, 1932), Brazil (Bitancounrt, 1938), United States (Traub and Robinson, 1938) and Pakistan (Sattar and Malik, 1939). In India, this disease of mango was reported by Stevens and Pierce, 1933 and currently, it is widely distributed in the entire mango growing states of the India causing huge economic loss.

It affects both vegetative and reproductive structure. Initial infection starts from leaves and spreads to flowers causing blossoms blight, which destroys inflorescence leading to considerable reduction in fruit set and yield loss. The disease incidence from different countries has been reported to be 32% in South Africa (Sanders *et al*., 2000), 64.6% in Cost Rica during 1990 (Arauz *et al*., 1994) and could reach almost 100% in fruit produced under wet or very humid condition (Arauz, 2000). In India, Himachal Pradesh, during 1990-92, post harvest decay due to anthracnose was 29.6% (Sharma *et al*., 1994). *C. gloeosporioides* causes anthracnose which is the most important biological constraint to mango production in South East Asia resulting in substantial yield loss (Dodd *et al*., 1991).

There are several mango varieties like Alphonso, Baramasi, Carabao, Carrie Early Gold, Keaw, Kent, Kishen Bhog, Rad, Saigon, Tommy Atkins and Van Dyke are resistant to infection caused by *C. gloeosporioides.* (Peterson, 1986; Dinh *et al*., 2003).

Infection on oil coffee berries in Vietnam was observed and finally pathogen characterize as *C. gloeosporioides* by employed morphological and molecular methods (Nguyen *et al*., 2009). In September 1995, *C. gloeosporoides* was observed on olive on the southern Montenegrin coast near Ulcinj (Latinovic and Vucinic, 2002). Brazil, one of the largest onion producers in the world observed onion anthracnose caused by *C. gloeosporioides* (Barbosa, 2001). 17% of papaya fruits were affected by anthracnose disease in Hawaii, rounded, water soaked, and sunken lesions appeared on the body of the ripened fruits. These lesions are referred to as "chocolate spots" (Dickman and Alvarez, 1983).

Microsclerotia formed sparsely by *C. gloeosporioides* and play an important role in survival (Baxter *et al*., 1985). Survival of mycelia and stromata in colonized pepper seeds have also been reported (Manandhar *et al*., 1995). The pathogen readily colonizes the seed coat and peripheral layers of endosperm even in moderately colonized seeds. Heavily colonized seeds had abundant inter and intra-cellular mycelium and acervulli in 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). *C. gloeosporioides* was reported that it transmits from endosperm tissue to hypocotyls and radicals in red pepper (Lee and Chung, 1995).

Pepper anthracnose is caused by *C. capsici* and *C. gloeosporioides* in the hot humid tropics of Asia. They reduce marketable yields of pepper (Manandhar *et al*., 1995). Recently, Park and Kim reported that five anthracnose fungi- *C. gloeosporioides, C. dematium, C. coccodes, C. acutatum* and *Glomerella cingulata* are pathogenic to different tissue of pepper plants. Among these species *C. gloeosporioides* was the predominant species causing anthracnose on pepper fruits. Purple and ripe red fruit stage developed more anthracnose than the immature stages (Oh *et al*., 1999). During 2005 and 2006, *C. gloeosporioides* was isolated from diseased samples of bell pepper (*Capsicum annuum*) collected from various districts of Himachal Pradesh, India. This was the first report of *C. gloeosporioides* on bell pepper from Himachal Pradesh (Gupta *et al*., 2009).

Three species of *Colletotrichum* such as: *C. gloeosporioides, C. fragariae,* or *C. acutatum* were observed to cause *c*rown rot of strawberry (Freeman and Katan, 1997; Howard and Albregts, 1984; Howard *et al*., 1992). In United State (southeastern) infection by *C. fragariae* and *C. gloeosporioides* is favored by warm, moist conditions (Mori, 1998; Smith and Black, 1987).

Tulip tree (*Liriodendron chinense*) has been widely cultivated in Korea and infection of *C. gloeosporioides* was detected by mycological characteristics, pathogenicity, internal transcribed spacer sequence. This was the first report on anthracnose disease caused by *C. gloeosporioides* on tulip trees in Korea (Choi *et al*., 2012). Yam a stable crop in tropical, subtropical Africa, Central South America, parts of Asia, the Carribean and Pacific islands (Coursey, 1967; Adelusi and Lawanson, 1987). Water yam (*D. alata*) is thought to be more susceptible to anthracnose than other yams (Plant Protection Service Secretariat of the Pacific Community, 2002). Other fungi were also associated with yam leaf-spot (Amusa *et al.*, 1996).

Genes involved in host defence and in pathogenesis of *Colletotrichum gloeosporioides*

During colonization in host tissue, *C. gloeosporioides* create alkalinizes surroundings. The transcription factor, pacC, is a regulator of pH-controlled genes and is essential for successful colonization. PacC up-regulates 478 genes and down-regulates 483 genes, comprising 5% of the fungal genome including; transporters, antioxidants and cell wall degrading enzymes (Alkan *et al*., 2013).

Genes involved in host defense

Pel-B gene is a virulent gene and encodes for pectate lyase (Yakoby *et al*., 2001). This enzyme degrades the plant cell wall and its expression can be easily seen in necrotrophic phase of infection. The expression of this gene also induce host defense mechanism. Pectate lyase expression is strongly affected by alkalinization. Alkalinization occurs naturally during fruit ripening, where the pH of the pericarp increases from 5.2 to 6.1 and pathogen also helps in increase the amount of ammonia accumulated by the host. This increase pH may change the expression of pacC, the terminal component of

Table 1. Showing various genes involved in host defense as well as in pathogenesis of *Colletotrichum gloeosporioides*

S.No	Gene	Function	Encode (Reference)
1	Shpx2,Shpx5,Shpx6,Shpx12	Host defense	Shpx2,5,6 12 protein (Harrison et al ., 1994)
2	PepCYP	Host defense	PepCYP protein (Hutvagner et al., 1997)
3	Cap 20	Host pathogenesis	Cap-20 protein (Chilly et al., 1998)
4	CgDN3	Host pathogenesis Pathogenesis,	CgDN3 protein (Stephenson et al., 2000)
5	Chip 6	conidial germination, appressorium formation	Chip 6 protein (Kim et al., 2002)
6	Pnl-1,Pnl-2	Pathogenesis	Cellulose binding domain, pectin lyase (Wei et al., 2002)
7	Pel-B	Degrade plant cell wall	Pectate lyase (Drori et al., 2003; Kramer-Haimovich et al., 2006)
8	CgDN24	Pathogenesis , hyphal development	Stephenson et al., 2005
9	Bcl-2	Apoptosis, mycelium, conidia, germination, pathogenesis	Bax and antiapoptotic protein (Barhoom and Sharon, 2007)
10	CgCTR2	Putative copper transporter	CgCTR2 protein (Barhoom et al., 2008)
11	Pel-1,Pel-2	Pathogenesis	Pectic lyase (Shih et al., 2008)
12	CgOPT1	Spore germination, mycelium growth	CgOPT1protein (Chague et al., 2009)
13	CgRac1	Morphogenesis, nuclear division, pathogenesis	CgRac1 protein (Nesher et al., 2011)
14	GDH2,GS1,GLT,MEP	Induce ammonia accumulation, pathogenesis	GDH2,GS1,GLT,MEP proteins (Miyara et al., 2012)
15	PacC	Create alkalinize environment and regulate activity of several genes	Pac C protein (Alkan et al., 2013)

the pH-dependent genes which is known to regulate the expression of Pel-B gene (Kramer-Haimovich *et al*., 2006; Drori *et al*., 2003).

Pel-B mutants do not secrete PLB and exhibited 25% lower pectate lyase and pectin lyase activities and 15% higher polygalacturonase activity. In addition, these Pel-B mutants induced a significantly higher host phenylalanine ammonia lyase activity as well as the antifungal diene, which is indicative of higher host resistance (Yakoby *et al*., 2001).

PepCYP gene encodes a protein homologous to cytochrome P450 containing a'heme-binding domain. PepCYP gene expression is higher in the incompatible interaction than in the compatible interaction. The induction of PepCYP gene is up-regulated by wounding or jasmonic acid treatment during ripening. PepCYP gene product play a role in the defense mechanism when the fungus invades and colonizes the epidermal cells of fruits in the incompatible interaction during the early fungal infection process. Sequence comparison showed that PepCYP protein shared highest homology to the CYPs.ch from a *Solanum Chacoense* line rich in glycoalkaloids (Hutvagner *et al*., 1997).

There are four distinct cDNAs such as: Shpx2, Shpx5, Shpx6, Spx12 isolated from a cDNAs library of S. *humilis* contain deduced amino acid sequence motifs characteristics of peroxidases. The mRNAs of Shpx2 and Shpx6 but not Shpx5, Shpx12 are also induced by wounding (Harrison *et al*., 1994).

Gene involved in pathogenesis

CgRac1 gene is a major regulator of asymmetric development of *C. gloeosporioides* and encodes CgRac1 protein which is involved in the regulation of morphogenesis, nuclear division and pathogenic germination. CgRac1 protein is abundant in conidia and hyphal tips (Nesher *et al*., 2011).

During plant infection, *C. gloeosporioides* produces high level of IAA in axenic culture. CgOPT1 gene is necessary for full virulence and its expression can be enhanced by addition of IAA (auxin). CgOPTI gene encode a protein contains 752 amino acids and has mass of 84.9 kDa and pI of 8.89. The gene includes three exons separated by two introns of 58 and 73 bp. In resting spores the expression of CgOPT1 is very low and enhanced in spore germination and reduced again in mycelia development (Chague *et al*., 2009).

There are several nitrogen-metabolism genes such as: *GDH2*, *GS1*, *GLT*, and *MEP* are differentially expressed during colonization by *C. gloeosporioides* and induces ammonia accumulation and pathogenicity (Miyara *et al*., 2012).

Bcl-2 protein is observed in *C. gloeosporioides* and its expression determine the programmed cell death and fungal development. There are two Bcl-2 proteins such as: Bax protein and anti-apoptotic bcl-2 protein. The Bax protein expression leads to apoptotic-like cell death, while expression of the anti-apoptotic Bcl-2 protein leads to prolonged cell longevity and protected the fungus from stresses. In short these two proteins expression cause drastic changes in processes such as mycelium growth, conidia production, conidial germination, and fungal pathogenicity (Barhoom and Sharon, 2007).

C. gloeosporioides requires copper at the initial stages of pathogenesis, germination and the CgCTR2, a putative vacuolar copper transporter involved in regulating cellular copper balance during the process. This transporter is highly expressed in resting spores (Barhoom *et al*., 2008).

C. gloeosporioides expressed Pel-1 and Pel-2 during infection to host. These genes encodes for pectic lyase activity began at the end of the biotrophic phase and increased in the necrotrophic phase of infection. Initial pH condition and carbon sources affects the expression of Pel-1 and Pel-2. The expression of these genes are higher in MCWE (mallow cell wall extract) broth than in any other broths or MCWE is better inducer for the expression of Pel-1 and pel-2 (Shih *et al*., 2000).

Cap20 gene is expressed during appressorium formation induced by host signal in *C. gloeosporioides*. This gene encode 183 amino acid polypeptide and plays significant role in infection to host. The Cap20 expression was observed by a sensitive reverse transcriptase PCR method (Chilly *et al.*, 1998) in the various layers of the fruit as infection proceeded into the fruit. The mpg1 gene from *Magnaporthe grisea* is involved in appressorium formation (Talbot *et al*.,

1993) and did not show any homology with Cap20.

There are two pectin lyase genes, Pnl-1 and Pnl-2 cause infection to host. Pnl-1 encode a cellulose binding domain (CBD), which is common in cellulases and xylanases, where as Pnl-2 encodes a pectin lyase that lacks a CBD. The expression of Pnl-2 gene is observed in the necrotrophic phase of infection and expression of Pnl-1 is observed in both nectrotrophic and biotrophic phase of infection. Glucose affects the expression of these genes and expression of Pnl-2 is relatively low during infection, because it may be more sensitive to catabolite repression (Wei *et al.,* 2002).

Chip 6 gene encodes a protein with homology to sterol glycosyl transferase and induced by hard surface contact of the conidia of *C. gloeosporioides* and encodes a protein with homology to sterol glycosyl transferase. This transferase plays an important role in pathogenesis and involved in conidial germination, appressorium formation (Kim *et al.,* 2002).

CgDN3 gene of *C. gloeosporioides* plays an important role in pathogenesis and required to avert a hypersensitive-like response by a compatible host. This gene is induced by nitrogen starvation in axenic culture and is expressed at the early stages of infection. The gene encodes a protein of 74 amino acids that contains a predicted 18 amino acid signal sequence for secretion of a basic 54 amino acid mature protein with weak homology to an internal region of plant wall-associated receptor *kinases* (Stephenson *et al.,* 2000).

CgDN24 gene encodes cDNA and is induced by nitrogen starvation in axenic culture. The cDNA comprises of 905 bp and predicted a 215 amino acids protein. CgDN24 gene plays no role in pathogenesis and is necessary for normal hyphal development in axenic culture (Stephenson *et al.,* 2005). Table 1 demonstrates the current status of known genes involved in pathogenesis of *C. gloeosporioides* and host defense.

Infection in Humans

C. gloeosporioides is very rare pathogen in humans but there are few cases have been reported:

A patient of 56 years age, developed a subcutaneous nodule of left fore arm and elbow after traumatic injury. Numerous irregularly shaped, hyaline, septate, branched hyphae were observed throughout the tissue. When these hyphae get cultured on PDA and Sabouraud's glucose agar, fungus grew. Finally it was identified as *C. gloeosporioides* (Guarro *et al.,* 1988).

A patient of 82 years old was suffering from myelodysplastic syndrome and after contract surgery of left eye, patient developed fungal kerratitis. When corneal of patient cultured on media, it grew well and identified as *C. gloeosporioides* (Mitani *et al.,* 2009).

Management of *Colletotrichum gloeosporioides*

Non-chemical control

Non-chemical control involves the effective dips of infected plant or crop in hot water having temperature around 48^0C for approximately 20 minutes. This method is not that much effective to eliminate infection of *C. gloeosporioides* completely.

Chemical control

Chemical method involves the use of fungicide spray in orchard having infected plants or crops present. Fungicide spray was not recommended in rainy season. Fungicide spray applies at the interval of 14-28 days in the orchard is an effective control of this pathogen.

There are various types of fungicides used such as: post-harvest and pre-harvest. Post-harvest fungicide generally used as spray or dips to those crops which are already infected with *C. gloeosporioides*. This method is employed to those fruits and crops which are shipped to overseas market (Dickman, 1993).

There are various fungicides which are used as pre-harvest fungicides e.g. copper hydroxide, mancozeb, and copper sulfate products (these are routinely used from flowering through to harvest). Prohloraz fungicide is used when weather conditions favours the infection of *C. gloeosporioides* (Dirou and Stovold, 2005).

Azoxystrobin is one of the strobilurin class fungicide was evaluated both *in vitro* and *in vivo* conditions. Azoxystrobin completely inhibit mycelia growth. Azoxystrobin at 1, 2 and 4 ml/l suppressed the development of both panicle and leaf anthracnose. Sundravadana *et al*., 2006 observed total control of mango anthracnose with azoxystrobin.

CONCLUSION

Anthracnose disease caused by *Colletotrichum gloeosporioides* is a major cause of concern among farmers not only in India but around the world as it causes huge pre and post harvest looses to a number of fruit and vegetable crops. The only method to control anthracnose is by timely fungicide spray, which also raises environmental and health concerns. Another way is to use varieties resistant against the infection caused by *C. gloeosporioides*. In order to devise effective control measures the importance of better understanding about the mechanism of *Collecotrichum* infection has been felt and as a result of that a number of genes involved in *Colletotrichum* pathogenesis and host defense has been worked out. But still a lot of work needs to be done before any environment friendly and consistent control strategy to come into existence. The use of mycoviruses to control fungal infection has been proposed recently for a number of fungal diseases but it has never been worked out in case of anthracnose pathogen *Colletotrichum*. The study on mycoviruses infecting *Colletotrichum* is also need of the time as it has lot of potential for being an environment friendly method for fungal disease control.

ACKNOWLEDGEMENT

The authors would like to thank Prof. P. K. Khosla, Hon'ble Vice-Chancellor, Shoolini University of Biotechnology and Management Sciences, Solan and Foundation for Life Sciences and Business Management (FLSBM), Solan for providing financial support and necessary facilities.

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