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Post Flowering Stalk Rot Complex of Maize - Present Status and Future Prospects

Mukesh K Khokhar[1]*, Karambir S Hooda[1], Shyam S Sharma[2], Vimla Singh[1]

[1]Directorate of Maize Research, Pusa Campus, New Delhi-110012, India
[2]Department of Plant Pathology, Rajasthan College of Agriculture, Maharana Pratap University of Agriculture and Technology, Udaipur, Rajasthan-313001, India
*Corresponding author: E-mail: khokharmk3@gmail.com

Abstract

Post flowering stalk rot complex is one of the most serious, destructive and widespread group of diseases in maize and yield losses range from 10 to 42% and can be as high as 100% in some areas. PFSR nature is often complex as a number of fungi (like *Fusarium verticillioides* cause Fusarium stalk rot, *Macrophomina phaseolina* cause charcoal rot, *Harpophora maydis* cause late wilt) are involved in causation of the diseases. To combat this problem, identification of quantitative trait loci for resistance to PFSR would facilitate the development of disease resistant maize hybrids. Moreover, various chemical and biological control methods have been developed but major emphasis is on development of maize cultivars with genetic resistance to for environment friendly control of the Post flowering stalk rot complex. The current paper reviews the information on distribution, impact of the disease, symptoms, epidemiology, disease cycle; genetics of resistance and integrated disease management approaches has been enumerated to understand the present status of knowledge about PFSR complex and will try to focus on the future perspectives available to improve PFSR management.

Keywords: maize, yield losses, post flowering stalk rot, late wilt, charcoal rot, integrated management

Introduction

Among major cereal crops in production, corn (*Zea mays* L) is the world's third leading crop after wheat and rice grown in different agro-ecologies of the world. It has highest genetic yield potential amongst the cereal crops. Diseases are one of the major constraints in realizing the potential yield of this crop. It suffers from a number of diseases but turcicum leaf blight (*Exserohilum turcicum*), maydis leaf blight (*Drechslera maydis*), polysora rust (*Puccinia polysora*), brown stripe downy mildew (*Sclerophthora rayssiae* var *zeae*), sorghum downy mildew (*Peronosclerospora sorghi*), Rajasthan downy mildew (*Peronosclerospora heteropogoni*), banded leaf and sheath blight (*Rhizoctonia solani* f sp *sasakii*), bacterial stalk rot (*Erwinia chrysanthemi* pv *zeae*), post-flowering stalk rots (*Fusarium verticillioides*, *Macrophomina phaseolina*), Curvularia leaf spot (*Curvularia lunata*) are the important constraints ones in globe responsible for yield losses.

They reduce both quantity and quality of the grain and may increase cost of cultivation. Globally, about 9% yield losses have been estimated in maize due to diseases (Oerke, 2005). This varied significantly from 4% in northern Europe and 14% in West Africa and South Asia (http://www.cabicompendium.org/cpc/economic.asp). In Southeast Asia, hot, humid conditions have favored disease development while economic constraints prevent the deployment of effective protective measures.

Post flowering stalk rot (PFSR) complex is one of the most serious, destructive and widespread groups of diseases in maize. The disease causes internal decay and discoloration of stalk tissue, directly reducing yield by blocking translocation of water, nutrient and can result in death and lodging of the plant during the cropping season. The term «stalk rot» is often used to include stalk breakage, stalk lodging, premature death of plant and occasionally root lodging. The PFSR is a complex of disease and difficult to characterize because a number of fungi, bacteria and nematodes are involved in the decay of pith. The disease is prevalent in most of the maize growing areas of world particularly where there is scarcity of irrigation especially after post flowering stag of the crop growth.

Causal agents of PFSR complex:

Fusarium stalk rot - *Fusarium verticillioides* (Saccardo); Gibberella stalk rot - *Gibberella zeae* (Schwein) ptec; Charcoal rot - *Macrophomina phaseolina* (Tassi) Goidanich; Diplodia stalk rot - *Stenocarpella maydis* (Berk) Sutton; Anthracnose stalk rot - *Colletotrichum graminicola* (Ces) Wils; Black Bundle diseases - *Cephalosporium acrimonium;* Late wilt - *Cephalosporium maydis* (Samra, Sabet, and Hingorani).

Among all of these pathogens of PFSR, Fusarium stalk rot, charcol rot and late wilt are most prevalent and destructive. The information on different aspects of post flowering stalk rot complex was reviewed keeping in view objective to assess the present status of PFSR diseases complex which would further

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Table 1 - Some important diseases of maize crop along with their losses and causal agent.

No	Disease	Causal agent	% Losses	Reference
1	Northern corn leaf blight	*Setosphaeria turcica*	13–50	Tefferi et al,1996
2	Southern corn leaf blight	*Cochliobolus heterotropus*	15–46	Zwonitzer et al, 2009
3	Gray leaf spot	*Cercospora zeae*	5–30	Ward et al, 1999
4	Curvularia leaf spot	*Cochliobolus lunatus*	10–60	Akinbode 2010
5	Brown spot	*Physoderma maydis*	6–20	Lal and Chakarvati, 1976
6	Southern corn rust	*Puccinia polysora*	20–80	Liang and Wu, 1993
7	Common corn rust	*Puccinia sorghi*	18–49	Groth et al, 1983
8	Eye spot	*Aureobasidium zeae*	14–44	Chang and Hudon, 1990
9	Alternaria leaf spot	*Alternaria tenuissima*	3–7	Ward et al, 1999
10	Head smut	*Sporisorium reilianum*	Up to 30	Njuguna, 2001
11	Common smut	*Ustilago zeae*	40–100	Pope et al,1992
12	Ear rot	*Fusarium verticillioides*	5–15	Ako et al, 2003
13	Downy mildew	*Peronosclerospora*, *Sclerophthora*	10-30	Spencer and Dick, 2002
14	Banded leaf and sheath blight	*Rhizoctonia solani* f sp *sasaki*	0–60	Tang et al, 2004
15	Fusarium Stalk rot	*Fusarium verticillioides*	10-42	Harlapur et al, 2002
16	Charcoal rot	*Macrophomina phaseolina*	25-32	Krishna et al, 2013
17	Late wilt	*Harpophora maydis*	51	Johal et al, 2004
18	Root rot	*Fusarium graminearum*	25–30	Hebbar et al, 1992
19	Maize dwarf mosaic	Maize dwarf mosaic virus	0–90	Goldberg and Brakke, 1987
20	Maize rough dwarf	Maize rough dwarf virus	10–70	Dovas et al, 2004
21	Bacterial stalk rot	*Erwinia carotovora* p var *zeae*	85	Thind and Payak, 1985

courtesy: Ali and Yan, 2012

necessitate the development of biorational and climate resilient integrated disease management (IDM) schedule.

Fusarium stalk rot

Among the stalk rots of maize, Fusarium stalk rot, caused by *Fusarium verticillioides* (Saccardo) Nirenberg [*Fusarium moniliforme* (Sheldon) (Seifert et al, 2004] was first reported from United States of America by Pammel in 1914 as a serious root and stalk disease. Later Valleau (1920) indicated that *Fusarium moniliforme* was a primary cause of root rot and stalk rot of maize. Subsequently, this disease has also been reported from several countries like Canada (Conner, 1941), UK (Butler, 1947), Hungary (Podhradszky, 1956), North America (Kucharek and Kommedahl, 1966), Russia (Ivaschenko, 1989), and China (Wu et al, 1973). In India, Fusarium stalk rot was first reported from Mount Abu, Rajasthan (Arya and Jain, 1964). Fusarium stalk rot was observed in the plant age group of 55 to 65 days which coincides with tasselling and silking and immediately followed grain formation stage. At these stages the stem reserves are depleted and most of the carbohydrates are translocated to developing sinks and stalks are predisposed to the fungi (Desai et al, 1992).

Distribution

Fusarium stalk rot is one of the most devastating soil-borne diseases of maize, occurring in all continents of the world (Figure 1), including USA (Koehler, 1960), Europe (Ledencan et al, 2003), Africa (Chambers, 1988), Asia (Lal and Singh, 1984), and Australia (Francis and Burgess, 1975). In India, the disease is prevalent in most of the maize growing areas, particularly in rainfed areas viz., Jammu and Kashmir, Punjab, Haryana, Delhi, Rajasthan, Madhya Pradesh, Uttar Pradesh, Bihar, West Bengal, Andhra Pradesh, Tamil Nadu and Karnataka, where water stress occurs after flowering stage of the crop (Singh et al, 2012).

Impact of disease

The stalk rot usually occurs after flowering stage and prior to physiological maturity, which reduces yields in two ways:i) affected plants die prematurely, thereby, producing lightweight ears having poorly filled kernels and ii) plants with stalk rot easily lodge, which makes harvesting difficult, and ears are left in the field during harvesting (Singh et al, 2012). Stalk rot reduces maize yield directly by affecting the physiological activity of the plants and finally results in lodging, which is the main cause of economic losses (Ledencan et al, 2003).

Lal et al (1998) reported that incidence of post flowering stalk rot complex (Charcoal rot, Fusarium stalk rot, late wilt) varying from 5 to 40% in different parts of the country. The annual loss due to maize diseases in India was estimated to the tune of 13.2 to 39.5% (Payak and Sharma, 1985). The disease was reported to cause a reduction of 18.7% in cob weight and 11.2% in 1000-grain weight in the infected plants (Cook, 1978). The disease incidence ranged from 10 to 42% in Karnataka (Harlapur et al, 2002). Hooker and Britton (1962) estimated the reduction in grain weight by 5-20%, whereas the estimated loss due to fusarium stalk rot has been reported as 38% in total yield (AICRP, 2014).

Associated species

As different species of pathogens have been iso-

Figure 1 - Geographical distributions of Fusarium stalk rot of maize. Courtesy CIMMYT, 2004.

lated from diseased maize stalks in different parts of the world, therefore, it appeared to be a complex disease (Chambers, 1987). Among the variety of pathogens, Fusarium is considered as a devastating fungal menace of the most prevalent fungus on maize. Reports of surveys conducted in African countries showed Fusarium as the most prevalent fungus on maize (Baba Moussa, 1998). Doko et al (1996) reported *F. verticillioides* as the most frequently isolated fungus from maize and maize-based commodities in France, Spain and Italy. Likewise, Orsi et al (2000) found *F. verticillioides* as the predominant species on maize in Brazil. Dorn et al (2009) surveyed the prevalence of Fusarium species and its impact between the north and the south regions of Switzerland and between kernel and stem piece samples. Several species of Fusarium have been reported to cause stalk rots like, *F.* (*F. semitectum*), *F. avenaceum*, *F. sulphurcum*, *F. acuminatum*, *F. roseum*, *F. merismoides*, *F. nivale*, and *F. solani* (Rintelen, 1965; Kommedahal et al, 1972; Nur Ain Izzati et al, 2011). In India, so far only *F. moniliforme* and *F. semitectum* are reported to be widespread in Western Uttar Pradesh, Punjab and Rajasthan (Lal and Diwivedi, 1982).

Symptoms

The disease becomes apparent when the crop enters senescence phase and severity increases during grain filling stage. The stalk rot symptoms are observed during post flowering and pre-harvest stage (Lal and Singh, 1984). The rotting extends from infected roots to the stalk and causes premature drying, stalk breakage and ear dropping, thus significantly reducing maize yields (Colbert et al, 1987). The disease causes internal decay and discoloration of stalk tissues, directly reducing yield by blocking translocation of water and nutrients, thus resulting in death and lodging of the plant (Dodd, 1980). Symptom development depends on several stress factors including an excess or lack of moisture, heavy and continuous cloudiness, high plant density, foliar diseases, and corn borer infestation (Parry et al, 1995).

Epidemiology of Fusarium stalk rot

Temperature may be one factor that determines the extent of invasion of the stalk rot fungi of maize (Williams and Munkvold, 2008). *F. verticillioides* is more common in regions with hot and dry growing conditions (Doohan et al, 2003), especially before or during pollination (Pascal et al, 2002). Reid et al (2002) observed that hot and dry conditions, especially at maize silking stage predisposes the plants to infection by *F. moniliforme* and *F. proliferatum*. Williams and Munkvold (2008) reported the role of high temperatures in promoting systemic infection of maize by *F. verticillioides*, but plant-to-seed transmission may be limited by other environmental factors that interact with temperature during the reproductive stages. Higher temperature reduces the time between wilting and lodging because heat increases the metabolic rate of fungi. After flowering, a major shift in carbohydrate flow towards the ear reduces the availability to the tissues resulting in senescence of root cells. Hence, insufficient water is moved to the leaves to meet the demands of transpiration causing the wilting of plants. Dead rind tissues are invaded by other fungi like Fusarium, Diplodia, Gibbrella, Colletotrichum, Macrophomina etc. Cellulase and pectinases enzymes from these fungi further weaken the stalk tissues (Dodd, 1980).

The water stress at flowering and high soil temperature help in increasing of the magnitude of the stalk rot symptoms at post flowering stage of maize crop (Smith and McLaren, 1997). The PFSR is more severe under moisture stress condition after flowering (Khokhar et al, 2014). Schneider et al (1983) observed that pre-tasselling moisture stage resulted in higher stalk rot incidence compared to moisture stress at post pollination and grain filling stages. Mews et al (1988) opined that pre-tassel moisture stress reduced the stalk rot during later season by reduced photosynthetic sink because the plants are subjected to the highest moisture stress and did not produce any grains. Soil texture affected the incidence of *F. verticillioides* on maize when it was grown alone or intercropped with cowpeas and soybeans. Disease incidence was greater in sandy soil than in loam or clay soils (Mohamed, 1991). The influence of climatic factors on Fusarium caused complications as they can cause disease complex infections and there are numerous reports on how species differentially respond to different environmental variations, particularly temperature and humidity (Doohan et al, 1998).

In general, stalk rot incidence and severity increase with increased fertility. There is evidence that potassium fertilizers reduces the severity of stalk rot and that nitrogen ones, especially if in excess compared with potash, increases the severity of stalk rot (Abney and Foley, 1971). A balanced and continuous nitrogen supply helps to explain the reduction of stalk rot with the use of nitrification inhibitors such as 2-chloro-6-(trichloromethyl) pyridine (nitrapyridin) when mixed with anhydrous ammonia (White et al, 1978). Potassium is involved in stomatal functions as well as metabolic pathways. When plants are deficient in potassium, the photosynthesis rate is lower and may result in pith senescence. Hence, maintaining a sufficient supply of potassium to prevent lodging needs more attention in maize hybrids. The response to phosphorus varies with the season, cultivar and the pathogen while higher level of phosphorus does not decrease stalk rot severity however, it seems to afford some protection against the stalk rot (Thayer and Williams, 1960).

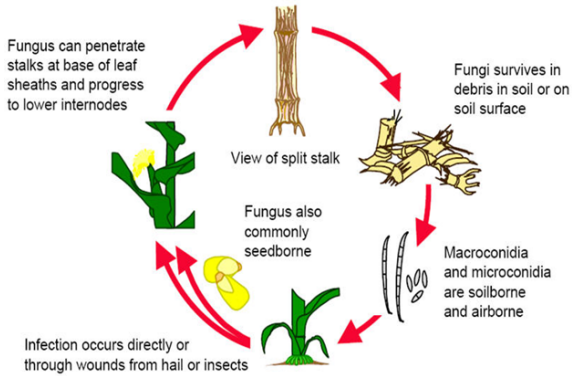

Figure 2 - Fusarium cycle. courtsey www.pioneer.com/home/site/us/agronomy/crop-management/corn-insect-disease/fusarium-stalk-rot.

Disease cycle

As shown in Figure 2, *F. verticillioides* survives on crop residue in the soil or on the soil surface (Nyvall and Kommedahl, 1970). Under favorable condition, it may infect roots as well as stalk (Lipps and Deep, 1991). *F. verticillioides* may be present throughout the life cycle of the plant, originating from infected seed (Headrick and Pataky, 1990).

Genetics of resistance to FSR

Due to its soil-borne infection pathway, fungicidal control of Fusarium stalk rot is not effective. Alternatively, discovery/utilization of resistance gene(s) to improve maize tolerance to stalk rot is a cost-effective and environment friendly approach for its management. Substantial numbers of maize germplasm have been evaluated for stalk rot resistance and some have demonstrated high levels of resistance (Ledencan et al, 2003; Afolabi et al, 2008). Resistance to Fusarium stalk rot disease involves several physiological, morphological and functional traits. Maize stalk strength is determined by two main factors, the mechanical structure of the stalk and abiotic stress factor (Singh et al, 2012). The degree of stalk rot infection depends greatly on environmental factors, the genotype and environment interaction (GxE) and the resistance of the given maize genotypes to the pathogens (Szoke et al, 2007). Ledencan et al (2003) have showed the resistance of maize inbreds and their hybrids to natural and artificial stalk infection with Fusarium spp. and compared the response of inbreds and their test cross hybrids to the pathogen. Inbreds and hybrids differed significantly in resistance and infection types and disease scores of hybrids were generally lower than that of inbreds. This would allow for identifying potential resistance genes/QTLs for resistance to stalk rot in maize by either genetic transformation or marker-assisted selection (MAS). As several factors impact symptom development, such as pathogen population, varied environmental conditions, and plant growth status, proposed genetic models have been inconsistent. Both qualitative and quantitative genetic loci have been reported to confer resistance to stalk rot. Studies have indicated that resistance to stalk rot is quantitatively inherited and controlled by multiple genes with additive effects. After evaluating 150 $F_{2:3}$ families from a cross between the maize susceptible line 33-16 and resistant line B89, Pè et al, (1993) identified five resistance QTL loci to Gibberella stalk rot, located on chromosomes 1, 3, 4, 5, and 10. In another study, a major resistance QTL (Rcg1) against Anthracnose stalks rot, located on the long arm of chromosome 4, has been identified and cloned (Jung et al, 1994; Frey, 2005). Whereas, a single resistance gene against *Pythium inflatum* has been mapped on chromosome 4 and located within a genetic distance of 5.7 cM flanked by simple sequence repeat (SSR)

markers bnlg1937 and agrr286 (Yang et al, 2005). Likewise, a single resistance gene to *F. graminearum* is located on chromosome 6 at a genetic distance of 5.0 cM flanked by markers mmc0241 and bnl3.03 (Yang et al, 2004). Yang et al (2010) detected two loci QTLS qrfg1 and qrfg 2, conferring resistance to Fusarium stalk rot. Report from Egypt indicated that resistance to Fusarium stalk rot was controlled by two genes and was dominant in expression. These two genes were located on the short arm of chromosome 7 and long arm of 10. Resistance to Fusarium stalk rot in inbred 61 C was also attributed to two genes. Sources of resistance against Fusarium stalk rot of maize identified were CM 103, CM 119, CM 125, CI 21 E, CML 31, 77, 79, 85, 90 and CML 381 (Kumar and Shekhar, 2005; Hooda et al, 2012).

Disease management

Since the stalk rot of maize is a complex disease involving more than one organism, it is very difficult to manage the disease with single control measure. Hence, efforts are needed to explore the feasibility of combination of various control measures for integrated management of stalk rots (Kulkarni and Anahosur, 2011). Trivedi et al (2002) evaluated systemic and non systemic fungicides viz., bavistin, dithane M-45, blitox 50, hexacap-75, TMTD, topsin-M, and apron-35SD against *F. pallidoroseum* causing post flowering stalk rot of maize in vitro at different concentration viz. 100, 250, 500, and 1000 ppm. All the fungicides completely inhibited the growth at 500 and 1000 ppm, though Topsin-M recorded the highest growth inhibition (70% and 98%) at low concentrations i.e. 100 and 250 ppm, respectively. Chandra et al (2008) evaluated two fungicides viz., tebuconazole and thiabendazole for their ability to inhibit the growth of toxigenic *F. verticillioides* and found that tebuconazole 5% aqueous solution effectively reduced ear rot disease and fumonisins accumulation to a maximum extent compared to other fungicides. Bioagents are useful for the effective management of soil borne pathogen propagules like chlamydospores of Fusarium species. The isolation and identification of effective bio control agents against PFSR is urgently required for use in integrated disease management. For decades, various Trichoderma species had shown antagonistic activities against many pathogens, both in vitro and in vivo (Howell, 2003). Successful growth suppression of *F. verticillioides* (*in vitro*) and its subsequent significant exclusion from internodes of maize stem in the field (*in vivo*) by strains of *Trichoderma pseudokoningii* had been reported by Sobowale et al (2005). Shekhar and Kumar (2010) reported the native isolate of *Trichoderma harzianum* resulted in good plant health and reduced post-flowering stalk rot of maize. Patil et al (2003) reported the seed treatment with *T. harzianum* (4g kg^{-1} seed) along with soil application of castor or neem cake (250 kg ha^{-1}), 15 days prior to sowing gave an effective control to stalk rot disease and gave better cost benefit ratio.

Integrated disease management

Integration of biological and chemical control seems to be a promising way of controlling many pathogens with minimum interference in the biological equilibrium in soil (Papavizas, 1973). Since soil is highly complex and biologically active substrate through which the fungicide act against fungi, fungitoxicants often give viable success in controlling seedling disease of crops in diverse agro-climatic regions of the world (Khan et al, 2008). The use of fungicides and tolerant genotypes has been reported to be effective method to manage stalk rot of maize which holds some promise. Kulkarni and Anahosur (2011) reported that application of farm yard manure and neemcake along with *T. harzianum* 15-20 days before sowing with two additional irrigation at tasselling and silking stage reduced the disease incidence from 70.08 to 13.24%. Thori et al (2011) reported that maximum germination (90%) with minimum mortality (0.0 and 2.5%) at 35 and 70 DAS and least percent disease index (PDI) of 23.2% was recorded by integration of *Trichoderma viride* (drenching), with bavistin seed treatment, followed by tebuconazole (ST) + *T. viride* (drenching). Among the individual treatments, seed treatment with bavistin and *T. viride* drenching showed good effects and resulted in 75% germination with 3.3% and 7.1% mortality after 35 and 70 day after sowing followed by 72.5% germination in tebuconazole seed treatment. Integration of plant resistance with these components was useful for reducing the losses caused by PFSR pathogen.

Late wilt of maize

Late wilt or black bundle disease of corn caused by the soil-borne and seed-borne fungus, *Harpophora maydis* (Gams, 1971; Michail et al, 1999; Samra et al, 1966; Degani and Cernica, 2014) with synonyms *Cephalosporium maydis* (Samra, Sabet and Hingorani) and *Acremonium maydis* (El-Shafey and Clafin, 1999). This disease was first reported as a vascular wilt disease of corn in Egypt in 1960 (Sabet et al, 1961) and was isolated from the roots and stems of wilting maize. The only reported hosts of *H. maydis* are corn (Samra et al, 1962), lupine (Sahab et al, 1985), and cotton (Sabet et al, 1966a).

Distribution

This disease has been reported to occur in the tropics of Tanzania, Pakistan, Hungary, Kenya (Freeman and Ward, 2004), Egypt, and India (Samra et al, 1963; Payak et al, 1970; Pecsi and Nemeth, 1998; Ward and Bateman, 1999) for some decades. It is more recently reported from Portugal, Spain (Molinero-Ruiz et al, 2010), Romania (Bergstrom et al, 2008), and Israel (Drori et al, 2012). This widely scattered locations suggest its probable transmission by seed, but also a failure to distinguish its symptoms from those of other diseases or stress (Freeman and Ward, 2004) and is now considered endemic throughout

maize growing areas. Although the known host, maize, originated in Central America (Maiti and Ebeling, 1998), the relatively per cent appearance of this disease in the widely grown crop may indicate a different source for the fungus.

Symptoms

Late wilt disease is characterized by relatively rapid wilting of maize plants typically at the age of 70 to 80 days, before tasselling and until shortly before maturity (Degani and Cernica, 2014). First symptoms appear approximately 60 days after sowing (Sabet et al, 1970b). Leaves become dull green, eventually lose color and become dry and include the development of light green stripes on the leaves, the stripes become translucent, and the entire leaf rolls inward from the edges. Later, drying-out ascends upwards in the plant and includes leaf yellowing and dehydration, color alteration of the vascular bundles to a yellow-brown hue and then the appearance of red-brown stripes on the lower internodes, the symptoms advancing to the fifth internode or further (Sabet et al, 1966a). With disease progression, the lower stem dries out (particularly at the internodes) and has a shrunken and hollow appearance, with dark yellow to brownish macerated pith and brownish-black vascular bundles. Because of the delay in appearance of initial symptoms until about flowering, this disease has been designated as «late wilt» (Samra et al, 1963). According to Jain et al (1974) a sweet smell often accompanies the wet rot. After the first wilt symptoms appear, progress of the disease is relatively rapid. Late wilt is often associated with infection by secondary invaders such as *H. acremonium*, *Sclerotium bataticola*, *F. verticillioides* and various bacterial rots to present a «stalk rot complex» (Samra et al, 1962; Drori et al, 2012).

Impact of the disease

Among the 112 diseases reported in maize, on a global scale, late wilt cause yield losses up to 100 per cent (Galal et al, 1979) and is a potential threat to maize cultivation. The disease has been reported to occur and cause severe damage in Indian states of Andhra Pradesh and Rajasthan (Singh and Siradhana, 1986). Serious economic losses from late wilt have been reported in Egypt, where 100% infection occurs in some fields, and in India, with an incidence as high as 70% and economic losses up to 51% (Johal et al, 2004). In susceptible varieties, the disease affected 70% of the plants and caused 40% loss of grain yield (Labib et al, 1975). The saprobic organisms cause the stem symptoms to become more severe. Fewer ears are produced, and kernels that form are poorly developed (Drori et al, 2012) and may be infected with the pathogen. Seed quantity is correlated negatively to disease severity (Shehata, 1976). Payak et al (1970) reported that the fungus caused seed rot and a low percentage of emergence, and plants that did emerge were delayed (Degani et al, 2014). These researchers also reported that seeds taken from infected plants showed similar symptoms. In severe cases, no cobs were formed. Payak et al (1970) reported that infesting soil with Harpophora maydis caused an increased rate of seed rot, and reduced emergence. Seeds obtained from infected plants of the composite variety «Ambar» also had reduced emergence and lower seedling vigor compared to seeds from unwilted plants.

The pathogen

The fungus was described initially as *Cephalosporium maydis*, based on its production of «heads» of hyaline, non-septate conidia from simple phialides (Samra et al, 1963). Gams (2000) observed that it is similar to anamorphs of species of Gaeumannomyces and Magnaporthe in conidiogenous cell morphology and in that its colonies are fast-growing, thin and pigmented in culture. He transferred it to the new genus Harpophora comprised of those anamorphs. The divergent collarettes, which Gams (2000) observed on the phialides are not apparent in the earliest photographs of the species (Samra et al, 1963). Molecular studies indicate that *H. maydis* is closely related to species of Gaeumannomyces, a genus in the ascomycete family Magnaporthaceae, but support it as a distinct species (Ward and Bateman, 1999; Saleh and Leslie, 2004). *H. maydis* reproduces asexually, and no perfect stage has been identified (Zeller et al, 2000). Saleh et al (2003) and Zeller et al (2002) showed that the pathogen is clonal in Egypt and that the Egyptian population contained four lineages, three of which were widely distributed throughout the country. The fourth lineage was the most virulent but was the least competitive on susceptible maize accessions when inoculated as a mixed inoculum of all four isolates (Zeller et al, 2002).

Epidemiology of late wilt

Optimum temperature and moisture conditions for corn growth are also optimal for disease development (Warren, 1983). The minimum temperature for growth has been reported as 6°C (Pecsi and Nemeth, 1998), between 10 and 15°C (Singh and Siradhana, 1985), 12°C (Samra et al, 1963) and 10 to 13°C (Sabet et al, 1966b). The optimum for growth has been reported as 30°C (Samra et al, 1963), 25°C (Singh and Siradhana, 1985) and between 27-30°C (Sabet et al, 1966b).The maximum temperature for growth has been reported as 38°C (Pecsi and Nemeth, 1998), above 34°C (Samra et al, 1963), between 35 and 40°C (Singh and Siradhana, 1985) and between 33 and 35°C (Sabet et al, 1966b). A field study using controlled heated enclosures suggests that wilt infection occurred between 20 and 32°C with an optimum at 24°C (Singh and Siradhana, 1987). Thus, late wilt develops rapidly at 20˚C - 32˚C, with optimum disease development at 21˚C - 27˚C (Singh and Siradhana, 1987a). Growth of *H. maydis* in soil is sharply inhibited above 35˚C, but this fungal pathogen can grow over a wide range of soil pH from 4.5 - 10, with an optimum at pH 6.5 (Singh and Siradhana, 1987b).

Spread is primarily through movement of infested soil, crop residue, or seed borne inoculum. Spread within a field is often associated with mechanical operations such as cultivation that moves soil. *H. maydis* can persist on corn stubble for 12 - 15 months (Sabet et al, 1970; Singh and Siradhana, 1987b). Sclerotia are produced under low humidity, which ensures long-term survival of *H. maydis* (up to 15 months) in no-till residues on the soil surface. Lupine facilitates parasitic survival of the pathogen under at least some field conditions (Botros et al, 1990). *H. maydis* can survive in seeds for 10 months at high temperatures and low humidity in India, but longer survival is predicted at low temperatures (Singh and Siradhana, 1987b). Infected seeds can produce plants with late wilt symptoms, infest soil and result in subsequent development of late wilt in healthy seeds grown in that soil (Degani and Cernica, 2014).

Disease cycle

H. maydis is soil-borne and infect maize seedlings through the root or mesocotyl (Sabet et al, 1970b). As plants mature, few plants are infected and the rest became immune about 50 days after sowing. Initially, the fungi grow superficially on roots, produce hyphae with short, thick walled and swollen cells. After penetration, the fungus colonizes xylem tissue and is rapidly translocated to upper parts of the plant. When infection is severe, the fungus colonizes the kernels, and resulting in seed borne nature, and also sometimes causes seed rot (Kumar and Shekhar, 2005). Singh and Siradhana (1987a) found that three irrigations at an interval of 8 hours after inoculation supported maximum rate of disease development. As plants mature, fewer plants are infected, and they become immune about 50 days after planting. After penetration, *H. maydis* colonizes xylem tissue and is rapidly translocated to the upper parts of the plant. When infections are severe, the fungus colonizes the kernels, resulting in seedborne dissemination and also causes seed rot and damping off (El-Shafey and Claflin 1999; Michail et al, 1999). The pathogen can remain viable in the soil for several years in the absence of a host. Lupine, an alternate host, can play a role in the survival of the pathogen. The pathogen is most common in hot and humid environments and in heavy textured soils rich in clay or silt. Saturated soils lessen the incidence of Harpophora maydis (Kumar and Shekhar, 2005; Degani and Cernica, 2014).

Genetics of resistance to late wilt

Most studies have used traditional quantitative genetic approaches and find that resistance is under polygenic control; however, one study claimed resistance was controlled by a single dominant gene (Shehata, 1976; García-Carneros et al, 2012). Shehata and Salem (1971) analyzed the genetics of resistance to late wilt of maize in the field. They utilized six generations resulting from two resistances and two susceptible inbred lines. They found additive gene effect to be significant in two crosses and dominance gene effect in one cross. Evidence for epistatis varied in a non significant way from cross to cross. Resistance has been reported as being partially dominant with five loci controlling resistance, additive with at least three loci controlling resistance, or involving three major genes (El-Itriby et al, 1984). Dominance and epistasis have been cited as major contributors to resistance, with additive effects of lesser importance (Shehata, 1976). The development of specific genetic markers for resistance to late wilt would greatly facilitate incorporation of resistance into adapted hybrids (Drori et al, 2012).

Disease management

Various cultural measures such as soil solarization, balanced soil fertility and flood fallowing can reduce disease severity and losses. Inoculum survival is restricted to the top 20 cm of soil, and survival depends primarily on the persistence in infected crop residues (Sabet et al, 1970). Crop rotation with rice provides some control, but the fungus may survive several years in soil in Egypt (El-Shafey et al, 1988; El-Shafey and Claflin, 1999). In India, it remained viable in stem pieces on the surface of the soil for 12 months, but could not be recovered after 10 months from pieces buried at 10 cm (Singh and Siradhana, 1989). High soil moisture favours disease, but saturated soil reduces it (El-Shafey and Claflin, 1999). Moisture management and flood fallowing may be useful cultural controls for late wilt where they are economically practical (Singh and Siradhana, 1988). Balanced fertility can reduce disease severity, although it does not provide complete control. Low levels of nitrogen fertilization (60 kg ha^{-1}) increased wilt even though yields were increased overall; however, higher nitrogen levels (120 kg N ha^{-1}) needed for optimal yield reduced late wilt (Singh and Siradhana, 1990). A physiological sufficiency of potassium is also reported to reduce late wilt in low K fields of India (Singh and Siradhana, 1990) but not in the higher K soils of Egypt (Samra et al, 1966). Phosphorus, organic amendments (straw, cotton cakes, and brodret) and micronutrients (Cu, Fe, Mn, and Zn) also reduce disease severity (Singh and Siradhana, 1990).

A number of organisms have shown promise as control agents. Six isolates of actinomycetes (*Streptomyces graminofaciens*, *S. gibsonii*, *S. lydicus*, *S. nogalater*, *S. rochei*, and *S. anulatus*) and five isolates of yeasts (*Candida maltosa*, *C. glabrata*, *C. slooffii*, *Rhodotorula rubra*, *Trichosporon cutaneum*) from the rhizosphere of maize in Egypt were antagonistic to *H. maydis in vitro*, and, when applied to the seed, significantly reduced the incidence of late wilt of maize planted in *H. maydis* infested sterilized soil in the greenhouse (El-Mehalawy et al, 2004). The fungus, *Trichurus spiralis* was also found to inhibit the growth of *H. maydis* in liquid culture, on solid medium, and in soil in pots (Abdel et al, 1981). Suspensions of the antagonistic bacterium, *Bacillus subtilis*, or its culture filtrate, reduced infection when added to infested soil

in pots either at the time of sowing or after sowing (Sellam et al, 1978).

Seed treatment with carbendazim or captan gave effective control of late wilt of maize in India (Satyanarayana and Begum, 1996), but benomyl was not effective as a dust or dip in Egypt (Sabet et al, 1972). When applied to seeds, certain actinomycete and yeast isolates were antagonistic to *H. maydis* and significantly reduced the incidence of late wilt disease under controlled greenhouse conditions (El-Mehalawy et al, 2004). Benomyl controlled the pathogen in pots and in culture, but was not effective when applied to soil (Sabet et al, 1972). In India, significant reduction of late wilt incidence was obtained with 0.1% Benlate, 0.1%. Bavistan, or 0.2% Bayleton applied to the soil as a drench after stem-inoculation of 60-day old plants in pots (Singh and Siradhana, 1989). Degani et al (2014) reported that fungicide azoxystrobin, inhibited the development of wilt symptoms and recovered cob yield by 100%.

The use of resistant varieties is considered to be the best, most practical method of control (Samra et al, 1963; El-Shafey et al, 1988). Resistance to stalk rot caused by *H. maydis* and other fungi was observed in different maize varieties, inbred lines and hybrids, by Mohamed et al (1966). The Egyptian resistant hybrid DC-19 was introduced by Labib et al (1975). Hybrid varieties have been reported to be more susceptible than open-pollinated ones (Sabet et al, 1961). In India, during 2001 to 2004, a breeding program in collaboration with Asian Regional Maize Program of CYMMYT evaluated two-hundred inbred lines for sources of resistance against post-flowering stalk rots of maize, caused among others fungi by Harpophora maydis, *F. verticillioides* and *Macrophomina phaseolina* (Shekhar et al, 2010). Three resistant maize lines, namely PFSR-13-5, JCY2-2-4-1-1-1-1, and JCY3-7-1-2-1-b-1, were identified. Additionally, resistance level of five pools/populations was improved to an overall acceptable level [PFSR (Y)-C1, PFSR (white), extra-early (white), P-100, P-300, and P-345]. Resistant lines developed in India include X102, CM111, CM202, and (CM104×WL) (Satyanarayana, 1995). Resistance is polygenic, quantitatively inherited, and due to additive gene effects (Shehata, 1976; Galal et al, 1979; El-Shafey et al, 1988). Observing four lineages with different regional distributions among Egyptian isolates of the pathogen, Saleh et al (2003) suggested that resistant lines could be deployed according to the lineages present in a region. Resistance would need to be tested with all four lineages, individually as well as in the usual combination, because virulence and competitiveness were not linked among the isolates (Zeller et al, 2002).

Charcoal rot

The charcoal rot of maize, caused by *Macrophomina phaseolina* (Tassi) Goid, is an important disease of this crop (Shekhar, 2004; Gill et al, 2005; Shekhar et al, 2010). The pathogen is reported to infect nearly 500 species of plants in tropical and subtropical countries (Figure 3; Ghaffar, 1988). The pathogen produces numerous black sclerotia on diseased plant parts, which are globular to irregular

Figure 3 - Geographical distributions of charcoal rot of maize. courtesy: CIMMYT 2004.

in shape. The fungus is composed of many strains, differentiated by sclerotial size and the presence or absence of pycnidia. *Rhizoctonia bataticola* is considered to be the sclerotial and mycelial stage of *M. phaseolina* (Malcom, 1980).

In India, the charcoal rot disease was observed in an epidemic form during 1960 Kharif season in Kashmir valley. Later it was noticed at Hyderabad (Andhra Pradesh) during 1965-66 rabi season and at Pantnagar (Uttar Pradesh) in 1966 kharif (Payak and Renfro, 1969). This disease has also been found to be prevalent in Jammu and Kashmir, Punjab, Haryana, Delhi, Rajasthan, Madhya Pradesh, Uttar Pradesh, Bihar, Andhra Pradesh, Tamil Nadu Karnataka and West Bengal (Kaiser, 1982).

Symptoms

The disease produces a variety of symptoms, which ranged from seedling blight, rotting of stalk, roots and kernels. It also produces brown, water soaked lesions on the roots that later turns black (Kumar and Shekhar, 2005). As the plants mature the fungus spreads into the lower internodes of the stalk, causing premature ripening, shredding and breaking at the crown. Numerous black sclerotia on the vascular strands give the interior stalks a charred appearance (Malcom, 1980; Kaur et al, 2008). Sclerotia may be found just beneath the stalk surface and on the roots. The fungus also infects the kernels turning them black completely (Prakasam et al, 1993; Shekhar et al, 2006). The fungus entering through the root epidermis was intracellular as well as inter-cellular. Hyphal colonization was generally much greater in roots than in stalks. Gum deposition was observed in the cortical tissues of roots (Singh and Kaiser, 1994).

Impact of the disease

The charcoal rot of maize caused by *M. phaseolina* has been reported to cause considerable yield loss in grain range from 25-32.2 % and deterioration in fodder quality (Krishna et al, 2013). Harris (1962) from Kano, Nigeria reported that charcoal rot caused considerable loss in yield. He recognized three factors in crop losses: i) loss in grain yield and quality due to stuntedness of stalks with premature drying; ii) loss of yield due to lodging of plants; iii) loss in quantity and quality of fodder due to infection and destruction of stalks by the pathogen. The yield reduction in susceptible genotype of maize due to this disease has been estimated to the tune of 39.5% (Payak and Sharma, 1978). In recent years, yield reduction has been reported to be as high as 63.5% (Desai et al, 1991; AICRP, 2014). In case of artificial inoculation studies with susceptible varieties 85.5% yield reduction over control recorded (Pareek, 1997).

The pathogen

M. phaseolina (Tassi) Goid (there are numerous synonyms such as *Tiarosporella phaseolina*, *Macrophoma phaseoli*, and *Rhizoctonia lamellifera*) is an anamorphic ascomycete of the family Botryosphaeriaceae and causes the disease charcoal rot on a broad range of plants in many areas of the world (Reichert and Hellinger, 1947). The lack of a known teleomorph has stalled its taxonomy over the years (Crous et al, 2006); however, a thorough phylogenetic study of 113 members of the family Botryosphaeriaceae using ribosomal DNA sequences was able to separate the genera Macrophomina and Tiarosporella (Crous et al, 2006). Although *M. phaseolina* is generally known as a plant pathogen, it is also an opportunistic human pathogen (Tan et al, 2008; Srinivasan et al, 2009) and, so far, the strains that invade plants and humans appear to be very similar (Srinivasan et al, 2009).

Epidemiology of charcoal rot

The diseases caused by the pathogen are more prevalent under the condition of high soil temperature 30 - 42°C, low soil moisture and low soil pH (5.4 - 6.0) or when plants are under water stress (Bremer, 1944; Dhingra and Sinclair, 1978; Kumar and Shekhar, 2005). The sclerotial and mycelial inoculum was equally effective in causing the disease (Meyer et al, 1974). In maize, the charcoal rot disease is reported to develop very fast under high temperature (Payak, 1975) and low humidity (McLean and Cook, 1965). The water stress and high soil temperature are reported to increase the charcoal rot symptoms in post flowering stage of maize (Kaiser and Das, 1988; Smith and McLaren, 1997; Kumar and Shekhar, 2005). Most of the commercially grown cultivars have shown a high level of disease incidence around grain filling stage.

Disease cycle

Seed borne nature of *M. phaseolina* in maize has been reported by many workers (Malcom, 1980; Payak and Sharma, 1985; Sandra et al, 2008). Savita et al (1996) reported that the pathogens survived for 12 months in seeds of maize. In soil, the fungus primarily survived as sclerotia released into the soil as host tissue decayed (Smith, 1969; Cook et al, 1973; Ilyas and Sinclair, 1974). The soil population of sclerotia increased if susceptible crops were grown annually (Watanabe et al, 1970; Meyer et al, 1974). The population of sclerotia as high as 1,000 per gram of soil has been reported by Papavizas and Klag (1975). The free, sclerotia of pathogen survived upto 30 days at extreme temperatures and moisture regimes in field soil (Banerjee et al, 1983). Root and seed exudates from germinated seeds of sesamum induced the germination of sclerotia in the soil and attracted growth towards the root region (Abdou et al, 1980). Mukherjee and Banerjee (1983) found that the sclerotial production was stimulated by low soil moisture (10 % holding capacity). In arid environments, the highest viable population of sclerotia was recorded at 0 to 5 cm level (Satish, 1995).

Disease management

Chandra et al (2008) evaluated two fungicides viz., tebuconazole and thiabendazole for their ability to inhibit the growth of toxigenic *F. verticillioides* and

found that tebuconazole 5% aqueous solution effectively reduced ear rot disease and fumonisins accumulation to a maximum extent compared to other fungicides. Disease intensity of *F. verticillioides* and *M. phaseolina* stalk rot can be reduced up to 25.6% through seed treatment with thiophanate methyl and increasing grain yield by 45.6% (Ahmad et al, 1992).

The common methods of disease management include chemical control, breeding for resistant varieties, cultural practices and use of biocontrol agents. It is very difficult to control by chemical treatment alone as it does not give protection throughout the crop growth period (Campbell, 1994). The chemical treatment also suffers from other disadvantages, like soil drenching with fungicides is not economically feasible, chemicals induce new strains of pathogen and environmentally hazardous (Hornby, 1990). Breeding for disease resistance for this disease has not shown a very encouraging result (Krishna et al, 2013).

Inhibition of microsclerotial production of *M. phaseolina* by *T. harzianum* has been recorded by Majumdar et al (1996) and Elad et al (1986). The reduction of disease incidence and improvement of plant health upon treatment with *T. viride* has been recorded by Harman and Taylor (1988). Seed coating with *T. viride* has been found to be effective at increasing the shoot dry weight, grains and nodules and improving plant growth (Kehri and Chandra, 1991). The seed treatment with *T. harzianum* (4 g kg^{-1} seed) along with castor and neemcake in furrow application gives effective control of stalk rot complex with a good cost benefit ratio (Patil et al, 2003). Shekhar and Kumar (2010) tested efficacy of six isolates of *T. harzianum* using dual culture plate technique and inhibition through volatile substances against charcoal rot pathogen of maize and found that Hyderabad isolate of *T. harzianum* caused maximum inhibition (62.3 %) of radial growth of *M. phaseolina* and regarded as potential bio-control agent for minimizing PFSR incidence on maize. Kleifeld and Chet (1992) proposed that *T. harzianum* can live in plants, where it enhances seed germination and promotes plant growth and flower production. Use of *T. harzianum* formulation as furrow treatment at 1 kg $acre^{-1}$ after mixing with 100 kg FYM at least 10-15 days before its use in the field, was found to be promising as it will be more effective in the field. These bioagents – FYM mixture can be used between the rows of standing maize crop after 30-35 days after planting to reduce the disease incidence of charcoal rot of maize (Kumar and Shekhar, 2005)

Kulkarni and Anahosur (2011) conducted an experiment for integrated management of charcoal rot of maize and reported that the plant stand of maize was maximum (97.33%) with treatment of *T. harzianum* + FYM and FYM + Neem cake + *T. harzianum* + *T. viride*. They also reported that the pre-sowing application of FYM neemcake and *T. harzianum* was most effective in avoiding the infection and it reduces the stalk rot at later stage. The treatment increased yield levels by reducing the lodging at 105 days to the extent of 10.19%. Significant effects of irrigation levels at 105th day with stress at tasseling and silking resulted in more disease ie. 39.51%. Treatment FYM + Neem cake + *T. harzinum* + *T. viride* recorded highest grains 1363.14 kg ha^{-1} followed by *T. harzinum* + FYM with 1253.89 kg ha^{-1} .

Integrated PFSR disease management

Moisture control

Moisture stress condition is favor by the *F. verticillioides* whereas, water stress for 30 days before harvest increased late wilt.

Temperature

Temperature has a marked influence on infection and development of stalk rot. Dry and hot weather during and after flowering favors development of disease Rot is favored by soil temperature of 30-45°C and low soil moisture.

Soil solarization

Soil solarization before sowing of crop cause deterrent effect on pathogen.

Plant maturity

Maize does not generally become susceptibility to stalk until silking time. Its infection increased with plant age from 5 to 95 days.

Plant population

A large plant population per hectare generally makes plants more prone to stalk rot. First, the stalks of plants grown under crowded conditions are smaller in diameter and, therefore, less rot is required to weaken the stalks to the breaking point.

Organic manure

Organic amendment stimulates the population of beneficial soil bacteria and actinomyceties resulting in a lower incidence of post flowering stalk rot in maize.

Soil fertility

In general, stalk rot incidence and severity increases with increased fertility. There is evidence that potassium fertilizers reduces the severity of stalk rot and that nitrogen fertilizers, especially if in excess compared with potash, increases the severity of stalk rot except in case of late wilt. Potassium plays a vital role in reducing stalk lodging in corn. Hence, maintaining a sufficient supply of potassium to prevent lodging needs more attention in maize hybrids

Biological control

Pseudomonas cepacia is a potential suppressor of maize soil borne diseases. The seed treatments with *T. harizianum* (4 g kg^{-1} seed) along with castor and neem cake gave an effective control of post flowering stalk rot diseases and gave better cost: benefit ratio.

Genetic resistance

Resistance to Fusarium stalk rot is controlled by two genes and is dominant in expression. These two genes are located in the short arm of chromosome 7

and long arm of 10. Resistance to Fusarium stalk rot in inbred 61 C is also attributed to two genes.

Resistant variety

Source of resistance against Fusarium stalk rot of maize identified are CM 103, CM 119, CM 125, CI 21 E, CML 31,77,79,85,90, and CML 3 81.

Chemical control

Diseases intensity of Fusarium stalk rot can be reduced by seed treatment with *T. viride* + bavistin along with two additional irrigation at tasselling and silking stage reduced the disease intensity from 70.08 to 13.24%.

Way forward

Stalk rot diseases of maize pose a threat to sustainable maize cultivation. Despite concerted studies on various aspects, more studies remain to be done. Some gaps in knowledge and need for future thrusts are listed below:

1. The survey and surveillance of the stalk rots should be a regular feature covering wider areas under maize cultivation;

2. The identified resistant inbred lines / genotypes may be used as the source of resistance in the development of single cross and other hybrids;

3. The proven resistant hybrids may be deployed in PFSR endemic areas after confirming their yield performance through multilocation yield trials;

4. Finger printing of inbreds and hybrids identified as donors of PFSR resistance and development of molecular markers for resistance to the disease can be undertaken;

5. Detection techniques to identify the pathogen and its variability using serological and molecular tools;

6. Use of isogenic lines in the race identification to be taken up;

7. Documentation of PFSR resistant lines and the registration with NBPGR;

8. Integrated management approaches need to be refined and output oriented research should be focused;

9. Construction of refined molecular linkage maps using DNA analysis, understanding species relationship through the analysis of mitochondria and genetic transformation. The greatest gains from biotechnology in near future can possibly come from work of defensive traits especially for complex diseases like stalk rot.

Conclusions

We have highlighted the progress made by different groups for a better understanding and control of PFSR using both conventional and modern technologies. These technologies provide valuable tools and resources for the agricultural sector and can contribute to yield and sustainability of maize production in world. Post flowering stalk rots cause comparatively more damage in tropical compared to temperate countries. The pathogen causes a permanent wilting, where leaves become flabby, basal stalk tissues turns to pinkish to purple tinge colourations. Late wilt kills the plant prematurely at or after the flowering stage. The infected plants are at first dull green, then yellow and eventually dry. In the advanced stage, the lower internodes become dry, shrunken and hollow. Charcoal rot is a common stalk rot disease in the warm and dry areas of the world. It occurs in areas where drought conditions generally prevail at or after flowering. The disease is favoured by high soil temperature ranging from 30°C to 42°C and low soil moistures. The pathogen overwinters as sclerotia in soil and may penetrate roots and lower stems during growing the season. A characteristic sign is the presence of numerous, minute and black sclerotia, particularly on the vascular bundles and outside the rind of the stalk. In the diseased plants, the outer rind and pith tissues are rotten, whereas the vascular bundles remain intact.

PFSR can be avoided by crop rotation, adequate potassium fertilization, appropriate plant populations, proper irrigation, and use of resistant hybrids with good stalk strengths. Although significant improvements in disease management have been made, the stalk rots continue to be a serious problem. Most of the commercially grown cultivars have shown a high level of disease incidence around the grain filling stage. Review of studies carried in affected locations revealed that none of disease management approaches is absolutely effective against it. Hence, immediate attention is required for identification of newer biorational and climate resilient components so as to integrate them together in a suitable combination(s). Hence it is necessary to develop an effective, economically feasible and environmentally safe method to manage this disease. The integrated management module evolved out of such studies particularly in the present changing climates would provide sustainable management of PFSR. Moreover, inheritance pattern of PFSR needs to be confirmed so as to develop resistant corn varieties/hybrids through conventional as well as biotechnological approaches.

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