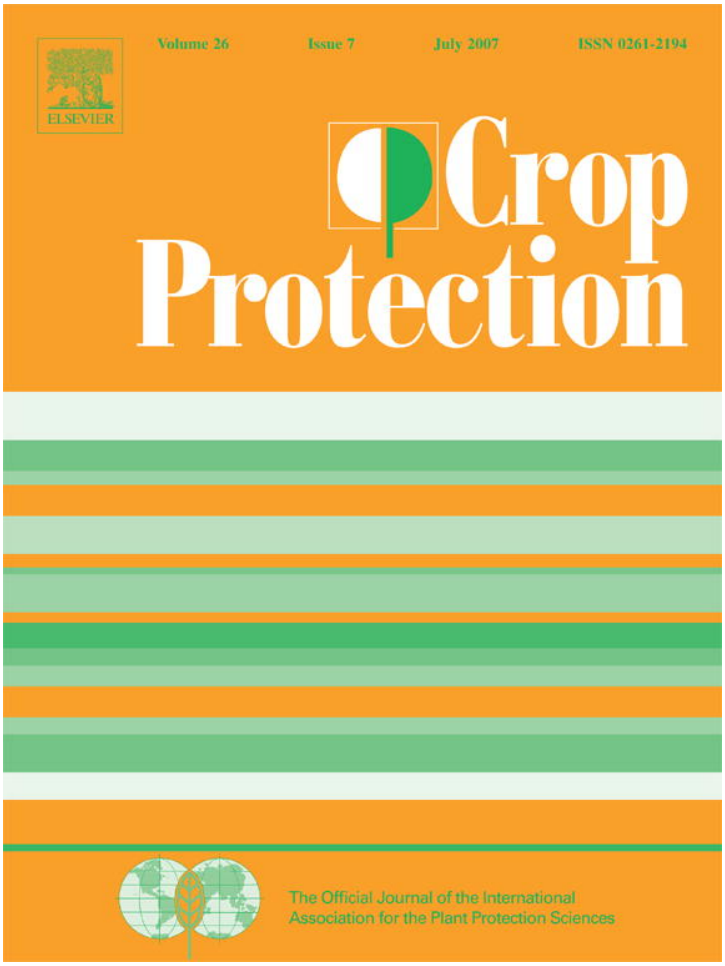

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Review

Fusarium culmorum: Infection process, mechanisms of mycotoxin production and their role in pathogenesis in wheat

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Abstract

Fusarium culmorum is an important pathogen of wheat causing seedling blight, foot rot, and head blight (*Fusarium* head blight (FHB)) or scab. The pathogen is dominant in cooler areas like north, central and western Europe. The fungus reproduces asexually by means of conidia, which form the main mode of dispersal. Head blight is by far the most serious concern of *Fusarium* infection on pre-harvest wheat and other small grain cereals. The significance of *F. culmorum* in wheat production is attributed to both head blight and mycotoxin contamination of the grain harvested from infected ears. Ear infection mainly occurs during anthesis and is favoured by wet weather or high humidity and warm temperatures. The major mycotoxins produced by *F. culmorum* are deoxynivalenol, nivalenol and zearalenone, which are a potential health hazard for both humans and animals. The mycotoxins, especially deoxynivalenol, are believed to play a role in disease development. Available options of managing FHB include use of fungicides, cultural practices, resistant cultivars and biological agents. However, no wheat cultivar is completely resistant to FHB while fungicides are at most 70% effective against natural infection. This review seeks to document and infer information on *F. culmorum*, with special emphasis on wheat head blight infection process, mechanisms of mycotoxin production, the role the mycotoxins play in pathogenesis, and the possible management options.

Keywords: *Fusarium culmorum*; Mycotoxins; Wheat; Head blight; Pathogenesis

Contents

1. *Fusarium culmorum* (W.G. Smith) Saccardo

The genus *Fusarium* currently contains over 20 species (De Hoog et al., 2000). Several taxonomic keys and manuals for identification of Fusaria have been developed. Examples of such keys and manuals include those

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developed by Wollenweber and Reinking (1935), Snyder and Hansen (1940, 1941, 1945), Booth (1971), Gerlach and Nirenberg (1982), Nelson et al. (1983). Identification of *Fusarium* species is difficult due to great variation in morphological and nonmorphological characteristics, including virulence. Separation of *Fusarium* species is based on primary and secondary characteristics. Primary characteristics include shape of the macroconidia, presence or absence of microconidia and their shape, whether or not microconidia are borne in chains, and the type of microconidiophores (Windels, 1991). Secondary characteristics include presence or absence of chlamydospores and their configuration and position, and presence or absence of sclerotia or sporodochia. Colony morphology, pigmentation, and growth rate can be useful if based on standardized procedures (Nelson et al., 1983; Burgess et al., 1988).

F. culmorum belongs to section Discolour, whose other species include *F. graminearum* Schwabe, *F. sambucinum* Fuckel, *F. crookwellense* Burgess, Nelson & Toussoun, *F. heterosporum* Nees, and *F. reticulatum* Mont. (Nelson et al., 1983). Members of the discolour section are often referred to as the cereal fusaria (Booth, 1975). Cereal fusaria do not form microconidia, except under certain cultural conditions, and are generally distinguished on the basis of morphology of the macroconidia. Macroconidia are comparatively thick walled, distinctly septate, fusiform to falcate with beaked or fusoid apical cell. Chlamydospores are usually present and may form either from hyphae or from the cells of the macroconidia.

F. culmorum produces short, stout, thick-walled macroconidia that have curved ventral and dorsal surfaces (Nelson et al., 1983). Chlamydospores generally form abundantly and quickly and may occur singly, in chains or in clumps. On potato dextrose agar, growth is rapid, with dense aerial mycelium. The mycelium is generally white but often yellow to tan. Orange to red-brown sporodochia appear as the culture ages. The underside is carmine red. The fungus is relatively stable in culture, but mutants may occur. The mycelial mutants lack colour in the aerial mycelium, sporodochial formation is suppressed and the carmine red pigment may be decreased. In pionnotal mutants the aerial mycelium is not present, and the yeast-like surface of the colony on potato dextrose agar is a sheet of macroconidia on pionnotes. Both surfaces of the colony are then carmine. The fungus teleomorph is not known.

2. Wheat head blight and distribution of *F. culmorum*

Fusaria have long been recognized as pathogens of many plant species. Wheat and other small grain cereals may be attacked by a wide range of *Fusarium* spp. and on different plant organs. However, infestation of the ears appears to be the most critical, leading to *Fusarium* head blight (FHB), also known as scab. FHB is a pre-harvest disease, but *Fusarium* species can grow post-harvest if wet grain is not dried efficiently and quickly. *F. graminearum* and *F. culmorum* are the predominant *Fusarium* species infecting wheat (Parry et al., 1995) *F. graminearum* predominates in the warmer, humid areas of the world such as USA (Goswami and Kistler, 2004; Vigier et al., 1997) whilst *F. culmorum* has been shown to be one of the predominant *Fusarium* spp. in the cooler areas such as north, central and west Europe (Parry et al., 1995) and Canada (Demeke et al., 2005). Predominance of a specific species in a region is mostly influenced by climatic conditions especially temperature requirements (Parry et al., 1995).

F. culmorum has been identified as the predominant species in Western Germany (Muthomi et al., 2000) and in the Rhineland region, Germany together with *F. avenaceum* (Lieneman, 2002). Kosiak et al. (2003) reported *F. culmorum* to be among the four most frequently isolated *Fusarium* spp. from wheat, barley and oats in Norway. Others were *F. avenaceum*, *F. poae* and *F. tricinctum*. A recent study by Clear and Patrick (2006) ranked *F. culmorum* (besides *F. graminearum* and *F. avenaceum*) among the three most dominant FHB causing fusaria in cereals in Canada. However, recent surveys in some countries where *F. culmorum* was predominant have reported *F. graminearum* to be predominating (Jennings et al., 2004a, b). Waalwijk et al. (2003) reported replacement of *F. culmorum* by *F. graminearum* as the predominant trichothecene producing ear blight pathogen in the Netherlands. *F. graminearum* has recently also been more common on wheat grain in Germany (Obst and Fuchs, 2000) where harmful levels of deoxynivalenol (DON) have been found (Placinta et al., 1999). Shah et al. (2005) found *F. graminearum* to be the dominant species in Italy in a study conducted from 1999 to 2002. This trend could be indicative of a change to warmer weather, a genetic change in the *F. graminearum* population or changes in cropping practices (Bateman, 2005). The trend could also reflect a changing trend of dominance of *Fusarium* spp. in different parts of the world. However, under suitable environmental conditions, *F. culmorum* is capable of causing severe disease and loss of useable grain (Lacey et al., 1999). The pathogen is a soil-inhabiting fungus that is a competitive saprophyte and facultative parasite. Its populations in wheat field soil have been shown to fluctuate greatly throughout the season, increasing greatly in dry conditions that favour its pathogenic activity on stem bases (Goswami and Kistler, 2004; Bateman and Murray, 2001; Bateman et al., 1998; Vigier et al., 1997).

3. Infection process and epidemiology

Most epidemiological research on head blight has focused on *F. graminearum* as the pathogen. Unlike *F. graminearum*, *F. culmorum* is not known to produce ascospores (teleomorph). It produces asexual spores (conidia), which are the main mode of dispersal. The conidia are dispersed either by wind or rain splashes to the wheat heads (Fernando et al., 1997; Jenkinson and Parry, 1994).

The conidia infect ears of wheat mainly during a short period of high susceptibility during anthesis (Bai and Shaner, 1996). The success of infection depends on many factors including climatic conditions, mainly temperature (Brennan et al., 2005; Cowger, 2005; Stein et al., 2005; Mentewab et al., 2000) and humidity (Cowger, 2005; Nita et al., 2005), cultivar resistance level (Cowger, 2005; Nita et al., 2005; Llorens et al., 2004; Bai and Shaner, 1999) and nitrogen fertilization (Doohan et al., 2003) among others. However, it is the availability of moisture, which is the overriding factor (Lacey et al., 1999).

Despite a number of research undertakings, the routes by which conidia of *F. culmorum* reach the ears are not clearly understood. According to Zange et al. (2005) the infection process on untreated spike tissue revealed conidia had germinated on the inner surfaces of the lemma and palea as well as on the ovary within 12–24 h after inoculation (hai). The fungus had formed a dense mycelial network 2 hai. Following a short inter-cellular growth, hyphae of *F. culmorum* developed both inter- and intracellularly and caused severe damages in colonized host tissues. A study by Jackowiak et al. (2005) using scanning electron microscopy confirmed localization of *F. culmorum* hyphae on the surface and inside the tissue of wheat kernels and presence of fungal hyphae in the endosperm. Even though systemic infection has been reported for *F. culmorum* (Xi and Turkington, 2003) there is no evidence that seedborne inoculum has a contribution to FHB (Bateman, 2005). However, Mishraa et al. (2002) suggested possibility of seedborne dispersal of *F. culmorum*.

Temperatures above 25 °C and moist periods of longer than 24 h favour infection and mycotoxin production by *F. culmorum* (Mentewab et al., 2000; Campell and Lipps, 1998) and wheat heads are most susceptible at anthesis (Lacey et al., 1999). Temperature has also been shown to influence the incidence and severity of FHB diseases with higher temperatures (>25 °C) causing greater disease than lower temperatures (Mentewab et al., 2000). The in vitro growth rate of *F. culmorum* was found to increase between 25 and 30 °C while optimal growth occurred at 20–25 °C. Brennan et al. (2005) reported greater visual disease symptoms and loss in yield caused by *F. culmorum* at 20 than at 16 °C even though the fungal DNA was not significantly different at the two temperature levels. Severe ear blight in a field experiment in England developed only in plots that received either rainfall or were mist irrigated (Bateman, 2005).

4. Mycotoxins biosynthesis in *F. culmorum*

The main mycotoxins produced by *F. culmorum* include the trichothecenes (DON, nivalenol (NIV), 3-acetyldeoxynivalenol and acetyl T-2 toxin), zearalenone (ZON) and fusarins (Llorens et al., 2006; Demeke et al., 2005). Trichothecenes are the largest group of mycotoxins. Chemically, trichothecenes are a large group of sesquiterpenes epoxides, and are characterized by the presence (type A trichothecenes) or absence (type B trichothecenes) of a keto group at the C-8 position. The trichothecenes, including DON, acetyldeoxynivalenol, NIV, and fusarenone X, are common fungal contaminants of cereals (Magan and Olsen, 2004; Jennings et al., 2000) and occur naturally worldwide on cereals (Dalcero et al., 1997; Muller et al., 1997; Park et al., 1996; Ryu et al., 1996; Kim et al., 1993; Fujisawa et al., 1992; Abbas et al., 1988). Consumption of these toxins is a potential problem for humans and farm animals (Eriksen and Alexander, 1998; Rotter et al., 1996).

Some strains of *F. culmorum* are able to produce type B trichothecenes, such as DON and acetyldeoxynivalenol, while other species are not (Marasas et al., 1984). According to the findings of O'Donnell et al. (1998) working with *F. venatum*, these two types of *Fusarium* strains can be distinguished on the basis of DNA polymorphism in the β-tubulin gene as well as in the large ribosomal subunit or the internal transcribed spacer (O'Donnell et al., 1998; Mulè et al., 1997; Guadet et al., 1989). Nicolaisen et al. (2005) has also reported the feasibility of oligonucleotide microarrays for parallel detection, identification and differentiation of trichothecene producing and non-producing *Fusarium* spp.

According to trichothecene production, *F. culmorum* (and *F. graminearum)* strains have been divided into two chemotypes: the NIV chemotype, which includes isolates producing nivalenol and fusarenone X, and the DON chemotype, which includes isolates producing DON and acetyldeoxynivalenol (Bakan et al., 2001; Sydenham et al., 1991; Ichinoe et al., 1983). Recent studies have shown that optimum production of DON and NIV by *F. culmorum* isolates occurs at sub-optimal water activity conditions, and sometimes only after 7–14 days incubation (Hope and Magan, 2003). It has also been observed that the pathogen chemotypes are distributed independently of geographical and wheat cultivar origin (Bakan et al., 2001). Llorens et al. (2006) did not observe any relationship between *F. culmorum* (together with *F. graminearum* and *F. cerealis*) with different cereal hosts, geographical origin of the isolate and mycotoxins-producing capacity. There have, however, been conflicting results about the correlation between trichothecene and ZON production. Whereas Sydenham et al. (1991) and Molto et al. (1997) reported a correlation (positive or negative), Bakan et al. (2001) reported lack of a strict correction between production of the two groups of mycotoxins. Using the Aspin–Welsh test, mean ZON production by the DON-producing strains was significantly higher than the ZON production by NIV-producing strains (Bakan et al., 2001). However, the substrate on which a *Fusarium* strain is grown could influence mycotoxin production (O'Neill et al., 1993).

In addition, it has been demonstrated that, within the same species and in the same culture conditions, toxin production by *Fusarium* strains may vary sharply; some strains produce large amounts of trichothecenes, whereas others produce small or undetectable amounts of

trichothecenes (Llorens et al., 2006; Walker et al., 2001; Muthomi et al., 2000; Langseth et al., 1999; Gang et al., 1998; Atanassov et al., 1994; Miller et al., 1991; Sydenham et al., 1991; Blaney and Dodman, 1988). According to the findings of Llorens et al. (2006) there is a positive correlation between amount of toxins produced and aggressiveness of a *F. culmorum* isolate. Until now, no method except in vitro culture has been available to distinguish high producing from low-producing *Fusarium* strains. However, variation among the toxigenic potential of *Fusarium* isolates may contribute to the wide range of mycotoxin content in cereals.

Mycotoxin biosynthesis is also influenced by environmental factors (Bakan et al., 2001). Most studies indicate that the optimum temperature for trichothecene and ZON production in *Fusarium* infected grain appears to be specific to the substrate, species and individual metabolites (Doohan et al., 2003). Doohan et al. (2003) showed that production of trichothecenes by *F. culmorum* and *F. graminearum* is favoured by warm and humid conditions. Minimum temperatures for DON production have been reported to be 11 °C, dependent on time of incubation (Versonder et al., 1982). A recent study by Hope et al. (2005) showed that growth of *F. culmorum* occurred at water activity (a_w) greater than 0.90 while DON production was optimum at 25 °C. Llorens et al. (2006) reported 20 °C to be the optimum temperature for ZON production for *F. culmorum*. Additionally, Llorens et al. (2004) reported optimum temperature values of 28, 20 and 15 °C for DON, NIV and 3-acetyl DON, respectively, for *F. culmorum* and *F. graminearum*. *F. culmorum* produces no DON at below 0.90 a_w. Recent studies on wheat-based media showed that *F. culmorum* had differential a_w and temperature optima for DON and NIV suggesting that production by this fungus may respond differently to a_w and temperature stress (Hope and Magan, 2003). The fungus may produce NIV under sub-optimal conditions for improving competitiveness. However, while it produces less NIV than DON, the former metabolite is more toxic than the latter (Hope et al., 2005). A recent study by Llorens et al. (2006) suggested existence of significant differences regarding the susceptibility of three cereal grains (wheat, corn and rice) to ZON production by *F. culmorum*. Studies by Birzele et al. (2000) suggested that the pathogen produced DON at 17% moisture content (0.80–0.85 a_w) in natural wheat grain.

The biosynthesis of *Fusarium* trichothecenes has been studied in *F. sporotrichioides*, which produces T-2 toxin, and in *F. culmorum*, which produces 3-acetyldeoxynivalenol (McCormick, 2003). Several genes involved in the biosynthesis of trichothecenes have been described, most of them localized in a gene cluster. The Tri5 gene encodes the trichodiene synthase, which catalyzes the first step in the biosynthesis of trichothecenes. The nucleotide sequence of the Tri5 gene has been characterized in several *Fusarium* species (Fekete et al., 1997; Hohn and Desjardins, 1992). The Tri6 gene encodes a protein that regulates the trichothecene biosynthesis genes (Proctor et al., 1995) and has been sequenced in *F. sporotrichioides* (Matsumoto et al., 2004; Proctor et al., 1995), *Giberella zeae* (*F. graminearum*) (Matsumoto et al., 2004; Brown et al., 2001; Lee et al., 2001), and *F. cerealis* (Matsumoto et al., 2004). For several *Fusarium* species, it has been shown that the Tri5 (Hohn and Desjardins, 1992; Hohn and Beremand, 1989) and Tri6 (Matsumoto et al., 2004) genes were present in single copy. Functioning of Tri13 and Tri7 genes are required for the production of NIV and 4-acetylnivalenol, respectively, in *Fusarium* spp. producing type B trichothecenes. Genes Tri13 and Tri7 from the trichothecene biosynthetic gene cluster convert DON to NIV (Tri13) and NIV to 4-acetyl-NIV (Tri7) (Chandler et al., 2003). Mutations have been identified in isolates which are able to produce DON but unable to convert this to NIV. In such isolates of *F. culmorum*, the Tri7 gene is deleted entirely (Jennings et al., 2004a). Presence or absence as well as functionability of any of the fore-mentioned genes determine the final toxin, which is produced by a particular *Fusarium* isolate species (Hestbjerg et al., 2002).

5. Role of trichothecene mycotoxins in pathogenesis

The production of DON by *F. culmorum* is believed to play a role in pathogenesis (Hestbjerg et al., 2002; Mesterhazy, 2002; Eudes et al., 2001). This relationship has been studied almost exclusively in connection with head blight. *F. culmorum* isolates of the DON chemotype were found to be more aggressive towards barley seedlings (Hestbjerg et al., 2002). Muthomi et al. (2002) reported more aggressiveness for DON-producing *F. culmorum* isolates than those producing NIV in wheat. These findings strengthen involvement of DON in pathogenicity of *F. culmorum* in different cereal crops.

DON, which is a potent inhibiter of protein synthesis and is postulated to inhibit activation of defence response genes, can induce complete loss of chloroplast pigments at sub-lethal concentrations (Rotter et al., 1996). Eudes et al. (2001) made similar observations confirming that trichothecenes are a principal determinant of *F. graminearum* aggressiveness on most spring wheat cultivars. They further found out that lack of trichothecene synthesis capacity of *F. graminearum* resulted in susceptible plants being able to slow down and even stop *Fusarium* spread which remained restricted to the infected florets. Ittu et al. (1995) reported yield reductions in spikes treated with a low concentration of crude DON or NIV, in the absence of the pathogen, suggesting a strong toxicity of these compounds to the plant tissues in growth. It is possible that in the presence of trichothecene, plant defence mechanisms are not triggered fast enough, thereby leading to an increased aggressiveness of the pathogen. However, Langevin et al. (2004) found out that the role of trichothecenes in FHB pathogenesis differs among species. The researchers found out that non trichothecene producing *F. graminearum* strain failed to spread within the inflorescence of wheat, triticale, rye and

barley while its spread was significantly reduced in durum wheat spikes. Other researchers such as Muthomi et al. (2000) have reported involvement of the trichothecenes produced by *F. culmorum* isolates in pathogenesis. This suggests that trichothecenes are a major determinant of fungal spread and disease development in triticeae.

6. Management of FHB of wheat

The available management options for FHB may be classified as short and long term. The short-term management options include use of fungicides, biocontrol and cultural practices while the use of resistant genotype is the most promising long-term management option. The main goal is to prevent the introduction and establishment of *Fusarium* species in areas where it does not occur and where it occurs, to reduce inoculum available for dispersal, prevention of dispersal of inoculum and prevention of infection of spikelets if the inoculum is present. Therefore, early detection and control of trichothecene-producing *Fusarium* spp. is critical to prevent toxins entering the food chain.

Chemical control: Control of FHB using fungicides has provided inconsistent results due to the complexity of causal organisms, influence of N-fertilization, timing of application and masking control of one *Fusarium* species by the subsequent growth of another species (Heier et al., 2005; Parry et al., 1995). Use of fungicides in the management of FHB has been shown to be at most 77% and 89% effective in reduction of disease severity and mycotoxins content, respectively (Haidukowski et al., 2004). These figures were reported for control of *F. culmorum* and *F. graminearum* on artificially inoculated wheat under field conditions. Lower levels of at most 70% effectiveness have been reported for fungicide control in field conditions for naturally infected wheat (Stack, 2000). Fungicides that have been used in control of FHB include prochloraz, propiconazole, epoxyconazole, tebuconazole cyproconazole and azoxystrobin (Haidukowski et al., 2004; Matthies and Buchenauer, 2000; Hutcheon and Jordan, 1992). A recent study by Balmas et al. (2005) reported a significant reduction of *F. culmorum* caused foot and crown rot incidence and improved grain yield after tebuconazole in cyclodextrin was applied as a seed dresser in vivo. Ramirez et al., (2004) reported effectiveness of fungicides against *F. graminearum* to be influenced by complex interactions between water, temperature, fungicide concentration and the time of inoculation. Although some of the fungicides are ineffective against FHB, some have been shown to stimulate DON and NIV production particularly at sub-optimal fungal growth conditions and low fungicide dosage (Ramirez et al., 2004; Magan et al., 2002; Jennings et al., 2000; D'Mello et al., 1999). In addition, food safety concerns limit the chemical management option due to fungicide residues in grain and wheat products (Jones, 2000).

Biocontrol: Because of the low effectiveness of fungicides to control members of the *Fusarium* spp., there have been efforts to identify biological antagonists, which could be used in integrated pest management (IPM) strategies. The short time period during anthesis when wheat ears are most susceptible to FHB could offer an ideal opportunity for biological control, thus avoiding the hazards associated with late fungicide application (Parry et al., 1995). Isolates of *Clostachys rosea* have been shown to consistently suppress sporulation of *F. culmorum* and *F. graminearum* on wheat straw and of *F. culmorum*, *F. graminearum*, *F. proliferatum* and *F. verticillioides* on maize stalks (Luongo et al., 2005). A strain of *F. equiseti* has been shown to consistently decrease DON (>70%) on wheat inoculated with *F. culmorum* with similar performance to a standard fungicide tebuconazole (Dawson et al., 2004). Diamond and Cooke (2003) reported a 60% reduction in FHB symptoms relative to control treatment after 25 days on ears pre-inoculated with *Phoma betae* and challenged with *F. culmorum*. They further reported a significant increase in number of grains per ear of wheat ears pre-inoculated with *Pythium ultimum* and *Phoma betae*. Two strains of *Pseudomonas fluorescens* have been reported to inhibit growth of *F. culmorum* both in vivo and *in* vitro (Kurek et al., 2003).

Cultural control: Cultural management options include crop rotation, appropriate use of fertilizers, irrigation, weed control, proper land preparation, and timely harvesting. Rotation should be with a non-cereal crop. *Fusarium* species that infect cereals are capable of surviving saprophytically on crop debris (Jones, 2000; Parry et al., 1995) and ploughing to bury crop debris removes the source of inoculum from the soil surface, which could be available for dispersal to ears. Bateman and Kwasna (1999) reported increase in *F. culmorum* population with increased number of crops over a 3-year period. Management practices, such as tillage systems and crop rotation, are important factors influencing contamination with *Fusarium*. Use of reduced tillage practices increases incidence and severity of FHB (Pereyra and Dill-Macky, 2004). The effect of soil preparation (tillage), nitrogen fertilization levels, crop rotation, previous crop, intensity of cultivation and weed control play an important role in FHB infection (Oerke et al., 2002). It is critical to avoid planting wheat adjacent to fields with large amounts of small grain or corn residue remaining on the soil surface. No-till planting wheat into corn residues substantially increases FHB infection. Rotation to a legume crop between corn and small grain crops provides time for the residues to break down and the pathogen population decline (Champeil et al., 2004).

Resistant cultivars: Growing of wheat cultivars resistant to *Fusarium* spp. should be the most economic, environment-friendly and effective method of disease control. Two types of resistance to FHB of wheat have been reported; resistance to primary infection and resistance to spread of the disease within a spike (Schroeder and Christensen,

1963). Kang and Buchenauer (2000) reported that FHB resistant cultivars were able to develop active defence reactions during infection and spreading of *F. culmorum* in the host tissues. The researchers also reported lower accumulation of DON in the tissues of infected spikes of resistant wheat cultivars. Miller et al. (1985) found that resistant cereal lines inoculated with *F. graminearum* contained lower concentrations of DON in the grain than susceptible cultivars. This suggests that it could be possible that a resistance mechanism that neutralizes DON production exists and DON may play a role in FHB pathogenesis (Snijders and Krechting, 1992). It is now generally agreed that FHB resistance is controlled by a polygenic system. Effects of dominance of genes probably influence FHB resistance, but additive effects appear to be important, and resistance genes can be accumulated (Bai et al., 1999; Snijders, 1990). Recent studies on chromosomal location of FHB resistance genes/loci in the wheat genome have used RFLP and AFLP methods and recombinant inbred lines as a mapping population. Quantitative trait loci of resistance to FHB have been preliminarily mapped on the following chromosomes: 1B, 2AL, 3 BS, 3A, 5A and 6B (Buertsmayr et al., 2002; Anderson et al, 2001; Ruckenbauer et al., 2001; Bai et al., 1999, Waldron et al., 1999). Sources of resistance have been found in China, South America and Czech Republic (Mesterhazy et al., 1999; Mesterhazy, 1995; Snijders, 1990). Currently, there are no wheat cultivars with high level of resistance to FHB although some cultivars have useable levels of partial resistance that limit yield loss and mycotoxins accumulation (Pereyra and Dill-Macky, 2004). Wisniewska and Kowalczyk (2005) have recently reported a breeding line with useable resistance to *F. culmorum* and other *Fusarium* spp.

The best approach in managing *F. culmorum* and FHB in general should focus on an integrated approach. Proper timing and application of fungicides and/or biocontrol products is critical. Fungicides with no harmful effects on biological antagonists against *F. culmorum* should be used. In the medium term, cultural practices such as crop rotation and proper tillage should be incorporated to maintain low inoculum levels. Breeding for resistance is necessary as a long-term strategy.

7. Further work

F. culmorum is globally ranked among the three most important FHB pathogens (with *F. graminearum* and *F. avenaceum*). Despite the global importance of *F. culmorum* on wheat and other cereals, there is little documented research information. Most of the available information focuses on *F. graminearum*. It is important to study the infection pathways, genetic structure, mycotoxin production and whether they play specific roles in pathogenicity of *F. culmorum*. The infection path to the ears needs to be studied further. It is crucial to establish whether there is a correlation between trichothecene and ZON production by *F. culmorum*. Additionally, since most of the previous studies have focused on cereal grain contamination by *Fusarium* spp., studies need to be conducted to determine the level of contamination of the vegetative parts and the effects these may have on animal and human health.

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