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Strategies for the control of *Fusarium* head blight in cereals

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Abstract

Fusarium head blight (FHB) is a widespread and destructive disease of small grained cereals caused by a number of *Fusarium* species and *Microdochium nivale.* In addition to causing significant reductions in grain yield, FHB can result in the reduction of grain quality, either by affecting grain processing qualities or by producing a range of toxic metabolites that have adverse effects on humans and livestock. Control of FHB can be achieved by a number of cultural, biological and chemical strategies along with the exploitation of host plant resistance. In recent years, much of the research undertaken for the control of FHB has been concentrated on understanding and exploiting the genetic resistance of cereal plants to FHB-causing pathogens. Although, a brief overview of genetic resistance is presented, this review seeks to summarise the significance of FHB and review the effectiveness of cultural, biological and chemical control strategies that have been investigated for the control the disease.

Introduction

Fusarium head blight (FHB), also known as scab, is a significant disease of small grain cereals and has been reported throughout the world. The disease is caused by five major species, *Fusarium avenaceum* (teleomorph, *Gibberella avenacea*), *F. culmorum*, *F. graminearum* (teleomorph, *G. zeae*), *F. poae* and *Microdochium nivale* (teleomorph, *Monographella nivalis*) (Parry et al., 1995). Initial symptoms of FHB appear as slightly brown water-soaked spots present on the glumes. The lesions increase in size until the whole spikelet is covered and, depending on weather conditions, spreads to the neighbouring spikelets. Infected plant tissues senesce, taking on the typical colour of ripe heads in contrast with green uninfected heads. In some cases, infection of the rachis causes blighting or death to those spikelets situated above the point of infection. The production of sporodochia at the base of infected glumes gives rise to a pinkish colour on severely infected ears (Atanasoff, 1920). Grain harvested from FHB-affected ears is often shrivelled and may have a red discolouration due to the presence of fungal growth. In North America, *Fusarium* damaged kernels (FDK) are assessed and categorised according to their colour: 'white tombstone' (shrivelled, white and chalky) and 'pink tombstone' (shrivelled, with pinkish appearance) (Sinha and Savard, 1997).

Significance of *Fusarium* head blight

Effect on grain yield

According to McKay (1957), a severe head blight outbreak in Ireland in 1942, decreased yield in wheat by between 21% and 55%. A second outbreak during 1954 was responsible for yield reductions in wheat and oat crops by up to 50%. An extensive field survey of wheat crops in the Atlantic Provinces of Canada during 1980 (Martin and Johnston, 1982) revealed that FHB was responsible for between 30% and 70% yield loss. Scab epidemics in wheat and barley occurred in southern Idaho in 1982 and 1984 and resulted in estimated yield losses as high as 50% (Michuta-Grimm and Foster, 1989). In China, the largest area

affected by FHB is in the mid and lower regions of the Yangtze river valley. Surveys carried out between 1951 and 1985 recorded 19 FHB outbreaks with grain yields of wheat reduced by 5–15% in years when moderate epidemics of FHB were recorded and up to 40% in years when disease epidemics were severe (Zhuping, 1994). According to Sayler (1998), in nine US states between 1991 and 1996, wheat producers lost 501 million bushels of grain, equivalent to $2.6 billion. Hard red spring wheat crops were worst affected with ca. 52% production losses, whilst soft red wheat and durum wheat experienced 38% and 10% production losses. During head blight epidemics in the Northern Argentinean Pampas areas, yield losses between 10% and 50% were recorded (Moschini et al., 2001).

These surveys provide an indication of the potential yield loss that may be associated with FHB, but they provide no indication as to how the disease reduces grain yield. More precise data on the effect of FHB on grain yield have been obtained from artificially inoculated field trial studies. Arseniuk et al. (1993) for example reported that under experimental conditions, 1000-grain weight, the number of grains per head and the head weights in four triticale cultivars were reduced by 15%, 18% and 22%, respectively. Following the artificial inoculation of double haploid barley genotypes with *F. culmorum,* Surma et al. (2000) observed significant reductions in grain number per ear (4–31%) and 1000-grain weight (14–31%) when compared to uninoculated plots of the same barley genotypes.

Effect on grain quality

The presence of *Fusarium* spp. in wheat can cause deleterious effects on grain processing qualities. Bechtel et al. (1985), for example, found that *F. graminearum* was capable of destroying starch granules, storage proteins and cell walls during invasion of wheat grains. Dexter et al. (1997) showed that Canadian hard red spring wheat grain samples that contained *Fusarium* damaged grains exhibited weak dough properties and unsatisfactory baking quality. Following a study of the effects of fungal proteases on wheat storage proteins, Nightingale et al. (1999) suggested that *F. graminearum* and *F. avenaceum* produced proteolytic enzymes. These enzymes hydrolyse endosperm proteins during dough mixing and fermentation and result in weaker dough and decreased loaf volume. In barley, infection of grains with *Fusarium* spp. reduces malt yield and quality, as well as causing uncontrolled foaming of beer (gushing) during the malting process. (Narziss et al., 1990; Schwarz et al., 2001; 2002).

Seed quality

Fusarium head blight can result in grain becoming infected by the causal pathogen. Such infections can result in seedling blight if seed is subsequently sown (Winson et al., 2001). The drilling of *Fusarium*-infected cereal seed has been linked with the subsequent development of seedling blight and foot rot in growing crops (Nelson, 1929), as well as a reduction in plant establishment, number of ears m^{-2} and grain yield (Wong et al., 1992; Humphreys et al., 1995; 1998).

Mycotoxins

Apart from the effects on seed and grain processing qualities, *Fusarium* species produce a range of toxic metabolites. These include a number of mycotoxins belonging to the trichothecene group. The different trichothecenes produced by members of the *Fusarium* are classified as type A or type B according to the structural components (Krska et al., 2001). Type A trichothecenes includes T-2 and HT-2 toxins whilst type B trichothecenes are represented by deoxynivalenol (DON), nivalenol (NIV), 3-acetyldeoxynivalenol (3-AcDON), 15-acetyldeoxynivalenol (15-AcDON) and fusarenon-X (FUS-X). *F. graminearum*, *F. culmorum* and *F. crookwellense* also produce an oestrogenic mycotoxin zearalenone (ZEN), also known as F-2 toxin which does not belong to the trichothecene group of toxins (Hussein and Brasel, 2001).

If grain contaminated with *Fusarium* toxins is used as feed for animal or human consumption, a range of adverse toxicosis as well as other health disorders are observed. In the middle of the last century, in Russia, the consumption of food prepared from over-wintered cereals, contaminated with *F. poae* and *F. sporotrichioides*, caused human poisoning known as Alimentary Toxic Aleukia (ATA). Symptoms of ATA include fever, necrotic angina, leukopenia, haemorrhaging and exhaustion of bone narrow. In some cases there are fatalities (Joffe, 1978). In 1987, an outbreak of a gastrointestinal disorder in the Kashmir Valley, India was associated with the ingestion of *Fusarium* mycotoxins (Bhat et al., 1989). Similarly, in China, 53 outbreaks of human food poisoning was associated with scabby and mouldy cereals occurred between 1960 and

1991 (Luo, 1992). In the Anhui Province of China in 1991, approximately 130,000 people were affected by gastrointestinal disorders, accompanied with abdominal pain, nausea, vomiting, fatigue and fever (Huang, 1992). Analysis of eight wheat and two barley samples revealed that DON was present in all samples at concentrations ranging from 0.016 to 51.45 mg kg^{-1}. NIV was also detected in all these eight wheat samples and one of the barley samples (0.001–6.93 mg kg^{-1}). Furthermore, both barley samples and six wheat samples contained ZEN at concentrations of between 0.046 and 0.3 mg kg^{-1} (Li et al., 1999) In 1998 and 1999, Li et al. (2002) analysed wheat samples taken from crops in the Henan Province of China, where cases of human toxicosis had been reported (Luo et al., 1987). Thirty samples out of the 31 tested (97%) from the Puyang area of this province contained DON, and 21 of them (70%) exceeded the Chinese advisory limit of 1 mg DON kg^{-1} grain.

The effect of DON-contaminated feed grain on domestic livestock is dependent on the animal species involved and on the severity and duration of exposure to contaminated grain (Rotter et al., 1995; Rotter et al., 1996). Among farm animals, pigs show greatest sensitivity to DON, while poultry and ruminants appear to show higher tolerance to the toxin (Trenholm et al., 1984). The oestrogenic compound ZEN, causes a range of reproductive disorders in young pigs ranging from vulva vaginitis and vaginal prolapses to enlargement of the uterus and atrophy of the ovaries (Mirocha et al., 1971). Consumption of grain contaminated with ZEN by pregnant sows resulted in an increase in stillborn pigs and small litters (Miller et al.,1973). T-2 toxin reduces feed consumption and weight gain in chickens due to severe oral lesions (Kubena et al., 1994). The toxin has also been associated with coagulopathy (Doerr et al., 1981) and altered feathering (Wyatt et al., 1975).

As a result of the adverse effects induced by *Fusarium* toxins, several countries have adopted advisory limits to ensure minimum levels of DON in finished products intended for human consumption and for animal feeds (Van Egmond, 1989). For example, the Food and Drug Administration (FDA) in the United States recommends that DON levels should not exceed 1000 μg kg^{-1} in finished wheat products and should not exceed 5000 or 10,000 μg kg^{-1} for feed intended for swine and cattle, respectively. The proposed advisory limits for trichothecene mycotoxins to be adopted within the European Union are 500 μg kg^{-1} for retail products such as breakfast cereals, bread and pasta and 750 μg kg^{-1} for flour and grain (Pricket et al., 2000). Although the development of trichothecene mycotoxins occurs primarily under field conditions, inappropriate grain storage can result in further increases in mycotoxin content (Birzele et al., 2000; Homdork et al., 2000).

Control of *Fusarium* head blight

Cultural control

In order to reduce the risk of FHB epidemics, several cultural control techniques can be employed, including suitable crop rotation, appropriate use of fertilisers and weed control. Crop rotation is one of the most effective cultural control measures that can be adopted. A survey involving 28 wheat crops grown in Illinois and Indiana (Holbert et al., 1919) showed that when wheat was sown following maize, 15% became infected by FHB. However, when wheat was grown following either alfalfa or oats, only 4% of crops became infected. Similar observations were later recorded by Koehler et al. (1924). Where wheat was grown after maize, up to 43% ears were observed to be affected by FHB. In wheat crops grown following rye or oats, 27% and 23% of ears showed symptoms, whilst following clover or timothy grass, only 11% and 8% of infected ears were observed. The average incidence of wheat crops affected by FHB on 72 farms in southwestern Ontario was six times greater when wheat followed maize than when wheat crops followed soybeans or cereals (Teich and Nelson, 1984). Recent studies on the effect of previous crop residues on FHB development also demonstrated that the incidence and severity of FHB was greater when wheat followed corn and lower when wheat followed soybeans (Dill-Macky and Jones, 2000).

Removing or burying crop residues leads to a reduced source of FHB inoculum. When wheat was planted after maize plots were ploughed, only 99 scabbed heads 10^{-5} were observed, whilst in disc-cultivated plots 209 wheat heads 10^{-5} showed symptoms. Such observations appeared to be due to the greater quantity of maize residue left on the soil surface (Teich, 1989). Other workers have also demonstrated that removal or ploughing in of crop debris reduces the incidence of FHB in cereals. For example, in Germany it has been observed wheat following grain maize had 0.5 mg kg^{-1} DON in comparison to that found in wheat following forage maize (0.3 mg kg^{-1})

(Obst et al., 1997). This effect was probably due to less crop debris remaining on the field after forage maize. In Canada, during a 3 year field study on the effect of tillage practices on FHB in wheat, Miller et al. (1998) isolated *F. graminearum* from 79%, 55% and 46% of kernels in year one, two and three, respectively, in no-till plots, whilst the incidence of infected kernels was 20%, 40% and 13% in the 3 years when plots were tilled.

The incidence of FHB can also be affected by fertiliser applications. During field trials investigating the effect of nitrogen inputs on the development of FHB, Martin et al. (1991) observed that increasing nitrogen applications from 70 to 170 kg N ha^{-1} resulted in increases in the incidence of *Fusarium*-infected grain in wheat, barley and triticale. In 1985 in Lambton County, Ontario, Canada, a comparative study showed that applications of ammonium nitrate resulted in 79 FHB-infected heads 10^{-5} whilst applications of urea resulted in 59 infected heads 10^{-5} (Teich, 1987). Applications of nitrolime to wheat plots reduced the incidence of FHB by 59% when compared to plots treated with calcium ammonium nitrate (Yi et al., 2001). However, there was no significant effect on DON concentration in harvested grain. It is not clear how nitrogen affects FHB development, however, several hypotheses can be postulated, including nitrogen influencing the water potential of the plant which in turn could influence the susceptibility of ears to infection by *Fusarium* species.

Field surveys in southwestern Ontario showed that fields with high weed densities had twice as many heads with FHB symptoms compared to weed-free fields (Teich and Nelson, 1984). The potential significance of weeds in the development of FHB epidemics has also been demonstrated by Jenkinson and Parry (1994), who isolated *Fusarium* species, which proved to be pathogenic to wheat, from 14 species of common broad-leaved weeds. These workers suggested that weeds provide an alternative source of inoculum for FHB epidemics and that weed control may reduce inoculum availability.

Cultivar resistance

The importance of wheat cultivars, which are resistant to FHB has been recognised since the early part of the last century (Dickson and Mains, 1929; Christensen et al., 1929). As a result, screening for resistance to FHB is conducted in most major wheat-growing countries. A simplified model for FHB resistance was proposed by Schroeder and Christensen (1963) who suggested that resistance was of two types; resistance to initial infection (Type I) and resistance to colonisation within the ear (Type II). Two further types of resistance based on the degradation of the mycotoxin DON (Type III) (Miller and Arnison, 1986) and tolerance to high DON concentrations (Type IV) (Wang and Miller, 1988) have also been proposed.

Types I and II resistances have been associated with certain morphological characters of wheat cultivars. Hilton et al. (1999) observed a significant negative relationship between plant height and resistance to FHB following artificial inoculation of ears of 17 cultivars of winter wheat with *F. culmorum*. Furthermore, while assessing plant height and disease severity on segregating populations from tall × short-strawed cultivars, a clear tendency was observed for tall-strawed lines to show less severe symptoms of FHB than shorter strawed lines. Such observations are supported by genetic mapping of quantitative trait loci (QTL), where QTLs for plant morphological traits such as plant height and heading date coincide with QTLs for lower FHB severity and DON concentration (Zhu et al., 1999; Ma et al., 2000).

It is beyond the scope of this paper to review the considerable amount of literature that has been published on host resistance to FHB and DON accumulation. The authors, therefore, refer the reader to Kolb et al. (2001) who have reviewed information on molecular markers associated with QTL for resistance to FHB in wheat and barley and the use of those markers for marker-assisted selection, and Ruckenbauer et al. (2001) who recently reviewed breeding strategies in resistance breeding against FHB.

Biological control

There is limited information on the control of FHB by biocontrol agents, although recent reports demonstrate that biocontrol of FHB pathogens has potential. For example, field antagonists of *G. zeae* affected the production of perithecia and ascospores of this pathogen (Bujold et al., 2001). *In vitro* studies on wheat and maize residues (straw/stalk and grain) showed that inoculating residues with a *Microsphaerosis* species (isolate P130A) significantly reduced *G. zeae* ascospore production by 73% (Bujold et al., 2001). When applied to crop residues in the field, the *Microsphaerosis* species had no effect on the pattern of perithecial formation, but significantly reduced

perithecial production. Under glasshouse conditions, the inoculation of wheat ears with *Phoma betae* at anthesis, reduced the severity of FHB symptoms caused by *F. culmorum* by 60% (Diamond and Cooke, 2003). In addition to these observations, it was also recorded that inoculating wheat ears with either *Pythium ultimum* or cell-free germination fluids obtained from a number of FHB-causing pathogens, significantly increased the latent period of *M. nivale*.

Bacterial biocontrol agents have also been investigated. Applications of the bacterial strain AS 43.4 (*Bacillus* spp.) isolated from wheat anthers decreased disease severity of FHB under glasshouse conditions by 67–95% and DON concentration in grain by 89–97% (Khan et al., 1999). In another glasshouse investigation, three (*Bacillus* strains 43.3; 43.4 and *Cryptococcus* strain OH 182.9) out of seven FHB antagonists reduced disease severity by 48–95% and decreased DON quantity in grain by 83–98% (Schisler et al., 2002). Unfortunately, under field conditions, the same antagonistic strains gave variable results. The *Bacillus* strains had no effect on either FHB severity or DON concentration in grain, while strain OH 182.9 reduced FHB and DON by 50%. Field studies undertaken by McMullen et al. (2002) showed that whilst the fungicide tebuconazole provided significant control of FHB, strain OH 182.9 had no effect on disease development.

Unfortunately, the discrepancy between the performance of biocontrol agents under environmentally controlled and field conditions is an issue that is commonly observed, and which provides a major obstacle to the development of commercial biocontrol products.

Chemical control

In vitro work determining the efficacy of fungicides to inhibit the growth and mycotoxin production of *Fusarium* spp. has been undertaken. Moss and Frank (1985) studied the effect of various concentrations of tridemorph on T-2 toxin and diacetoxyscirpenol (DAS) production by *F. sporotrichioides in vitro*. At low concentrations (6–8 mg kg^{-1}) tridemorph caused slight enhancement of fungal growth, whilst significantly reducing the production of T-2 toxin and DAS. However, at higher concentrations of tridemorph (30–50 mg kg^{-1}), fungal growth was inhibited by ca. 50%, but T-2 toxin production was stimulated fivefold. Dichloran, iprodione and vinclozolin were also effective against *F. graminearum* (Hasan, 1993). All three fungicides, when added to potato-dextrose broth at 10, 100, 150 and 500 $\mu g\,ml^{-1}$, produced a significant reduction of mycelial mass, and in production of DAS and ZEN. Dichloran eliminated DAS production at 500 $\mu g\,ml^{-1}$ and eliminated ZEN at 250 $\mu g\,ml^{-1}$. Iprodione reduced DAS production at 100 $\mu g\,ml^{-1}$ and prevented it at 250 $\mu g\,ml^{-1}$. Vinclozolin prevented DAS production at 250 $\mu g\,ml^{-1}$ and significantly reduced the production of ZEN at 500 $\mu g\,ml^{-1}$. In contrast, *F. graminearum* reacted in various ways when incubated in the presence of sub-lethal concentrations of some fungicides such as prochloraz, tebuconazole, benomyl, carbendazim and thiabendazole (Matthies et al., 1999). These workers showed that prochloraz inhibited mycelial growth of *F. graminearum* and reduced 3-ADON production. Tebuconazole inhibited fungal growth at 0.1, 0.5 and 1.0 $\mu g\,ml^{-1}$, however, at 0.5 $\mu g\,ml^{-1}$, 3-ADON production was increased fourfold when compared to control treatments. At 0.1 $\mu g\,ml^{-1}$, benomyl increased mycelial growth of *F. graminearum* by 22% and reduced 3-ADON production by 22% compared to the untreated control. Dose-related inhibition of mycelial growth and mycotoxin production was observed when carbendazim was added to the media at 0.5, 0.7, 1, 1.5 and 2 $\mu g\,ml^{-1}$. Guazatine and iminoctadine significantly reduced mycelial growth of *F. graminearum in vitro*, but increased 3-ADON production by up to 200%. The influence of fungicides on fungal growth and toxin production by *F. sporotrichioides* is affected by temperature (Placinta et al., 1996). In a laboratory study, the fungus was placed on carbendazim-amended potato dextrose agar at concentrations ranging from 1.0 to 10 $\mu g\,ml^{-1}$ and incubated at 25 °C. After 5 days of incubation, half the plates were incubated at 11 °C. The results indicated that at 5 $\mu g\,ml^{-1}$ carbendazim and 25 °C, production of T2-toxin increased, however, no effect on ZEN or neosolaniol (NEO) production was observed. Conversely, following the 25/11 °C temperature regime, dose-related inhibition of ZEN and T-2 toxin was observed. One possible explanation for these variable reactions of *F. sporotrichioides* is the existence of fungicide-resistant strains within the species. D'Mello et al. (2000) demonstrated that when two strains of *F. sporotrichioides,* a control strain (CS) and a strain resistant to carbendazim (RS), were grown at 25 °C in a peptone broth containing 1, 2, or 4 $\mu g\,ml^{-1}$ carbendazim, the CS showed dose-related effects on inhibition of fungal growth and T-2 toxin production, whilst 2 $\mu g\,ml^{-1}$ carbendazim enhanced T-toxin production by the RS, with no effect on mycelial mass.

Effective chemical control of FHB under field conditions has generally been inconsistent. Glasshouse and field trials conducted to assess the efficacy of fungicides against FHB have yielded conflicting results. Jacobsen (1977) studied the effect of benomyl (0.55 kg a.i ha^{-1}), mancozeb (1.76 kg a.i ha^{-1}), mancozeb + benomyl (1.1 kg a.i ha^{-1} + 0.27 kg a.i ha^{-1}) and benomyl + carbendazim (0.55 kg a.i ha^{-1} + 1.1 kg a.i ha^{-1}) on septoria leaf and glume blotch, and FHB caused by *F. graminearum,* on grain yield and test weight of wheat. Fungicide applications resulted in an increase in test weights by up to 2% over unsprayed control plots for benomyl, by 1.2% for the mixture benomyl + mancozeb and by 1.7% for mancozeb. All treatments reduced the percentage of scab-infected grain by 50%. Similarly an application of benomyl + thiabendazole applied 5 days before expected flowering date reduced the incidence of wheat grain infected with *F. graminearum* by 88%, and increased grain germination by 65% and 1000-grain weight by 48% (Carranza, 1988). In contrast, Michail (1989), who observed the incidence of *Fusarium*-infected seed in 32 samples of 16 wheat cultivars from crops sprayed with a range of fungicides (triadimefon, captfol + triadimefon, fenpropimorph, carbendazim, propiconazole, captafol + pyrazofos and prochloraz) at growth stages between GS 32 and GS 50 (Zadoks et al., 1974) failed to find any significant effect of the fungicide treatments on grain infection.

Carbendazim, prochloraz and propiconazole were effective against *F. avenaceum*, *F. culmorum* and *F. graminearum* by reducing them by 70% in the wheat spikelets over the control treatment (Hutcheon and Jordan, 1990). In naturally infected trials in the Atlantic Provinces in Canada, propiconazole, at a rate of 250 g a.i ha^{-1} applied at GS 51 and GS 73 (Zadoks et al., 1974) provided good control of FHB and increased yield by 34% compared to the control (Martin and Johnston, 1982). No effect of the fungicide treatments was observed on the concentration of DON in grain. Milus and Parsons (1994) studied the effects of benomyl, chlorothalonil, fenbuconazole, flusilazole, myclobutanil, potassium bicarbonate, propiconazole, tebuconazole, thiabendazole and triadimefon + mancozeb. These fungicides had no effect on FHB, mycotoxin levels or yield of harvested grain. Fungicides have, however, been shown to affect mycotoxin concentration in harvested wheat grain following FHB infection in the field. A 16-fold increase in NIV concentration occurred in harvested grain when the fungicide mixture (tebuconazole + thiabendazole) was applied 3 h post-inoculation and a sixfold increase when applied 24 h post-inoculation (Gareis and Ceynova, 1994). The severity of FHB symptoms was reduced by 54%. It is difficult to draw firm conclusions from just one trial, however, such observations raise the question of how each of the trichothecenes produced by *Fusarium* spp. are affected by different fungicides.

During fungicide efficacy field trials in Hungary, triadimefon, carbendazim, bromuconazole, cyproconazole + carbendazim, propiconazole, tebuconazole + triadimenol and tebuconazole were applied to plots of the FHB susceptible wheat cultivars Zombor and Csaba 1 day after artificial inoculation of ears with *F. graminearum* and *F. culmorum* (Mesterhazy and Bartok, 1996). Results showed that fungicide treatments that included the triazole tebuconazole, reduced grain infection by between 97% and 99% and DON contamination by 100%. Conversely, those treatments, which did not include tebuconazole, reduced, grain infection by 43–87% and DON contamination by 50%. The efficacy of 'azole' fungicides against FHB was confirmed by Ellner (1997). The fungicides tebuconazole, tebuconazole + triadimenol and prochloraz, significantly reduced FHB severity by 50% and DON concentration by 85%. One possible reason for the inconsistent control of FHB achieved by fungicides under field conditions is the complex interaction, which may occur between fungicide *Fusarium* species and other ear colonising fungi such as *Alternaria, Septoria, Cladosporium* and *Botrytis cinerea*. For example, Bateman (1979) studied the relationship between saprophytic ear colonising species and *M. nivale* on wheat ears and wheat seed. Grain collected from wheat ears which were artificially inoculated with either *Alternaria* spp., *Cladosporium* spp. or *Sporobolodomyces* spp. at anthesis prior to the inoculation of ears with *M. nivale,* yielded significantly less *M. nivale* in comparison with saprophyte-free ears. Similar results were obtained by Liggitt et al. (1997) during glasshouse studies where wheat plants were inoculated with either *A. alternata*, *B. cinerea* or *C. herbarum* at GS 59 prior to inoculation with *F. culmorum* at GS 65. The presence of any one of the three saprophytic species reduced FHB severity by between 46% and 78% compared to plants inoculated only with *F. culmorum*. However, when each of the saprophytic species were introduced to ears after their inoculation with *F. culmorum*, neither *B. cinerea* nor *C. herbarum* had any effect on FHB severity, although *A. alternata* significantly increased disease symptoms by 34%. Liggitt et al. (1997) also

demonstrated that fungicides had different effects. For example, pyrimethanil reduced mycelial growth of *A. alternata* by up to 92%, but failed to reduce growth of *F. culmorum*, *B. cinerea* or *C. herbarum* by more than 27%. Conversely, flusilazole reduced mycelial growth of *F. culmorum* by up to 90%, but failed to reduce mycelial growth of *B. cinerea* or *C. herbarum* by more than 59%. These workers suggested that the application of fungicides which have limited activity against *F. culmorum*, but which have significant activity against saprophytic species, may lead to greater colonisation of wheat ears by the pathogen, due to the removal of antagonistic saprophytes.

Interactions between fungicides and FHB pathogens have also been observed. Applications of the strobilurin fungicide, azoxystrobin, may have a significant effect on the interaction between *F. culmorum* and *M. nivale* colonising wheat ears. During 2 years of field studies, applications of tebuconazole, metconazole or carbendazim caused a significant reduction in both DON concentration and the extent of grain colonisation by *Fusarium* spp. quantified by a competitive PCR assay (Jennings et al., 2000). Conversely, applications of the same fungicides resulted in an increase in the extent of grain colonised by *M. nivale.* In the first year of the study, effective reduction of *M. nivale* on wheat ears, achieved following applications of azoxystrobin, alleviated competition between *M. nivale* and *Fusarium* spp. As a result, greater colonisation of ears by *Fusarium* spp. was observed and DON contamination was increased by 41%. In the second year, *M. nivale* was not present and no significant increase of DON concentration in grain was detected after treatment with azoxystrobin. More recently, Simpson et al. (2001) also associated applications of azoxystrobin with increased DON concentrations in harvested wheat grain. During a field trial where wheat ears were artificially inoculated with a mixture of *F. avenaceum*, *F. culmorum* and *M. nivale* at GS 65 and then sprayed with an application of azoxystrobin three days later, a 40% increase in DON concentration was observed in harvested grain, compared to grain harvested from unsprayed plots. The quantification of *F. culmorum* DNA did not indicate an increase of this species in grain, although *M. nivale* DNA was significantly reduced by azoxystrobin.

It can thus be postulated that applications of azoxystrobin might have a direct effect on DON production, through imparting a stress factor on *F. culmorum*, inducing the pathogen to produce more mycotoxin. However, by quantifying the amount of the *Tri*5 gene present in grain following fungicide efficacy trials under both glasshouse and field trials, azoxystrobin appeared to have no direct effect on DON production (Edwards et al., 2001; Pirgozliev et al., 2002). Indeed, when the concentration of *Tri*5 DNA was related to DON content, a significant and strong positive linear relationship was observed between the two variables. Plotting *Tri*5 DNA and DON concentrations for each of a range of fungicides and dose rates revealed that those fungicides which were ineffective at controlling *F. culmorum*, such as azoxystrobin, allowed greater colonisation of grain by the fungus and hence more *Tri*5 DNA: this was reflected in a greater DON content. None of the fungicide treatments resulted in an elevated production of DON per copy of *Tri*5 DNA, an observation that would be expected if a particular fungicide directly influenced DON production. It would appear, therefore, that fungicides can influence DON content in harvested grain, and that this influence is indirect by affecting the extent of grain colonisation by toxin-producing *Fusarium* species.

In contrast to the observations of Jennings et al. (2000) and Simpson et al. (2001), Jones (2000) observed a decrease in FHB severity of 12% compared to unsprayed controls and a reduction of DON by 25% when azoxystrobin was applied during field trials inoculated with *F. graminearum* between 1995 and 1997. Siranidou and Buchenauer (2001) also showed that applications of azoxystrobin reduced FHB severity, although DON concentration was similar to that in unsprayed controls. Similarly, azoxystrobin provided some control of FHB, without any detrimental effects on the mycotoxin production (Cromey et al., 2001). Azoxystrobin, tebuconazole and carbendazim were applied at GS 59 or 65 on winter wheat plots naturally infected with *Fusarium* spp. (predominantly *F. graminearum*). Tebuconazole reduced disease severity by 41%, whilst azoxystrobin and carbendazim reduced FHB by 29% compared to the control treatment. Tebuconazole and carbendazim significantly reduced DON and NIV in grain, whilst azoxystrobin did not have any effect on these mycotoxins.

Another possible reason for the inconsistent control of FHB achieved by fungicides under field conditions could be the timing of application. Studies where fungicides were applied between GS 32 and GS 50 failed to reveal any significant reduction of FHB (Michail, 1989; Hutcheon and Jordan, 1992). However, when fungicides were applied between GS 59 and 70, significant reductions were achieved in both the severity of FHB and concentration of mycotoxin in harvested

grain. For example, during a field trial where wheat plots were artificially inoculated with *F. graminearum* at anthesis, Boyacioglu et al. (1992) observed that triadimefon reduced grain infection and DON concentration when applied either 2 days pre-inoculation, at the time of inoculation or 2 days post-inoculation of ears. Propiconazole also reduced *F. graminearum* infection by 39–56% and DON concentration by 62–79% when applied at the time of inoculation and 2 days post-inoculation of ears. Thiabendazole was most effective, reducing DON concentration by 83% when applied 2 days pre-inoculation despite the fungicide having no effect on the incidence of grain infection. In Germany, Matthies and Buchenauer (2000) reported that in wheat inoculated with *F. culmorum,* applications of either tebuconazole or prochloraz, 2 days post-inoculation (GS 65), reduced disease severity by 56% and 41%, whilst applications 8 days pre-inoculation or 9 days post-inoculation were less effective. Tebuconazole and prochloraz applied 2 days post-inoculation reduced DON content in grain by 43% and 22%. However, an application of tebuconazole against FHB (*F. culmorum*) 3 days pre-inoculation reduced disease severity by 92% and DON concentration in grain by 69% in comparison with control treatment, whilst the same fungicide applied 5 days post-inoculation reduced FHB by 57% and DON in grain by 54% (Homdork et al., 2000). The greatest reduction of DON concentration in grain (80%) and FHB severity (90%) was achieved when tebuconazole was applied twice (pre- and post-inoculation). Suty et al. (1996), also reported that double treatment of tebuconazole at GS 55 and 69 was more effective than a single treatment at either GS 55 or 69. However, this approach may be uneconomical in commercial situations as it would result in increased fungicide input.

More recently, Siranidou and Buchenauer (2001) showed that applications of tebuconazole 2 days before and 2 days post-inoculation of wheat plots with *F. culmorum* reduced severity of FHB and DON content in wheat grain by 61–89% and 50–70%, respectively. Metconazole was applied at only 2 days pre-inoculation and reduced DON and FHB severity by 69% and 71% respectively. Chlorothalonil, prochloraz and benomyl failed to effectively control FHB.

Future developments

Due to the lack of consistently effective control measures, FHB continues to pose a significant threat to the yield and quality of small grained cereals. Although cultural control strategies such as rotation, land preparation and weed control can have an effect on inoculum load, the ubiquitous nature of the causal pathogens means that such control measures will always be limited. A range of fungicides have been identified with good activity against FHB pathogens. Unfortunately, the efficacy of these fungicides is significantly influenced by the dose rate used, time of application and perhaps even the method of application. Effective chemical control of FHB is further confounded by the fact that the disease is caused by a complex of pathogens which interact with one another and with saprophytic species such as *Alternaria* spp. and *Cladosporium* spp. (Liggitt et al., 1997; Pirgozliev, 2002). For example, glasshouse studies have shown that introducing either *Alternaria tenuissima, C. herbarum* or *M. nivale* to wheat ears before inoculation with *F. culmorum*, significantly increased FHB symptoms and DON content (Pirgozliev, 2002). Furthermore, applying azoxystrobin, a fungicide know to be effective against *M. nivale*, after the inoculation of wheat ears with this pathogen (Simpson et al., 2001) at GS 57 resulted in a 56% increase on DON content caused by *F. culmorum*. In order to optimise the control of FHB and mycotoxin contamination of grain, therefore, detailed studies on the effect of fungicide treatments on the cereal ear disease complex needs to be undertaken. The quantification of FHB-causing pathogens using molecular PCR assays following fungicide treatments will prove invaluable in advancing our understanding of the disease complex and in optimising disease and mycotoxin control.

The most reliable and consistent strategy for controlling FHB and mycotoxins is the exploitation of cultivars with good resistance. Large quantitative variation for FHB resistance in wheat has been described (Buerstmayr et al., 1996). Molecular mapping of QTL associated with FHB resistance, such as that carried out by Buerstmayr et al. (2002) for spring wheat, will prove invaluable in marker-assisted selection and will significantly speed up the development of cultivars with good resistance to FHB and mycotoxin contamination.

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