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Management of Fusarium head blight of wheat and barley

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ABSTRACT

Fusarium head blight (FHB) is an economically devastating disease of small grain cereal crops. It is caused by species of fungi in the genus *Fusarium*, of which *Fusarium graminearum*, *Fusarium culmorum*, and *Fusarium avenaceum* predominate. The disease is responsible for worldwide losses in excess of a billion dollars annually, and the majority of these losses occur in wheat (*Triticum* spp.) and barley (*Hordeum vulgare*) production. Losses are manifested as reduction in yield and grain quality and the presence of mycotoxins in grain, the most common of which is deoxynivalenol (DON). Due to the devastating nature of the disease, it is essential for growers to have in place strategies to mitigate losses due to FHB and DON. These strategies include a combination of cultural practices, planting resistant or tolerant cultivars, chemical control, biological control, use of forecasting systems, and harvesting strategies. This review examines these techniques individually and emphasizes the integration of two or more of them to optimize the effectiveness of managing FHB and DON during the growing season.

1. Introduction

Fusarium head blight (FHB), also known as scab, is an economically devastating disease of small grain cereal crops. It affects all small grains including wheat (*Triticum* spp.), barley (*Hordeum vulgare*), rye (*Secale cereale*), oats (*Avena sativa*), and triticale (x *Triticosecale*). The majority of economic losses occur in wheat and barley production. FHB was first described in England by W.G. Smith in 1884 and several years later it was reported in the United States by F.D. Chester in 1890 and J.C. Arthur in 1891 (Parry et al., 1995; Stack, 2003). Since then several major epidemics have occurred worldwide (Parry et al., 1995; Stack, 2003). In the U.S., there were five major FHB epidemics in the late 1910s to early 1930s (Stack, 2003). The disease re-emerged in the 1980s and 1990s and since then epidemics of varying intensity have occurred, more frequently in the eastern half of the U.S. (McMullen et al., 1997, 2012). This re-emergence of FHB in the U.S. is attributed in part to a combination of favorable weather conditions (wet, humid, and warm weather) before and during anthesis and an abundant supply of inoculum due to reduced tillage practices that leave crop residue on the soil surface to reduce erosion and conserve soil moisture (Shaner, 2003).

Globally, there is evidence that climate change, by modifying the environment to favor disease development, is associated with increased frequency and severity of FHB epidemics (Jeger and Pautasso, 2008; Garrett et al., 2014; Hernandez Nopsa et al., 2014a; Kriss et al., 2012; Parikka et al., 2012). A weather-based model developed using data in the United Kingdom suggested a slight increase in FHB severity as a direct effect of climate change (West et al., 2012). Using cross-spectral analysis, Kriss et al. (2012) determined that winter and spring Oceanic Niño Indices were significantly coherent with FHB in Ohio, USA. The Oceanic Niño Index is a measure of the El Niño-Southern Oscillation, the Pacific-North American pattern, and the North Atlantic Oscillation, known to strongly influence climate in the Northern Hemisphere. Crop and disease models are predicting more severe FHB epidemics, higher levels of mycotoxin contamination of grain, and higher yield losses in places where the disease is present (Madgwick et al., 2011). Locations or areas that historically were not affected by the disease

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are likely to become new hotspots for FHB.

FHB is caused by species of fungi in the genus *Fusarium*, of which *Fusarium graminearum*, *Fusarium culmorum*, and *Fusarium avenaceum* predominate (Dill-Macky, 2010; Parry et al., 1995). *F. graminearum* and *F. culmorum* are the most common and most virulent, and their geographical distribution appears to be related to temperature and moisture. *F. graminearum* occurs mostly in warmer and wetter climatic regions of the world including North America, Eastern Europe, Australia, and Southern China whereas *F. culmorum* occurs mostly in cooler climatic regions such as Western Europe (Miller, 1994; Parry et al., 1995).

FHB-causing *Fusarium* spp. survive intercrop periods as mycelium, perithecium initials, or chlamydospores in host crop residues. Corn (*Zea mays*), wheat, and barley residues are especially suitable for survival and reproduction of FHB-causing fungi (Khonga and Sutton, 1988; Pereyra and Dill-Macky, 2008; Trail, 2009). In the spring, mature perithecia discharge ascospores which are considered the primary inoculum. The ascospores are carried by air currents, land on spikes and infect the spikelets. Infections can occur any time from full spike emergence until maturity; however, most infections occur during anthesis (Andersen, 1948; Dill-Macky, 2010) in part because anthers contain stimulants for spore germination and pathogen growth (Strange and Smith, 1971). Symptoms are manifested as bleaching of one or more spikelets on a spike. The bleaching can start anywhere on the spike, but frequently begins in the center of the spike and can continue until the entire spike is whitened. Bleached spikes are randomly scattered in the field and appear suddenly so that a crop that appeared healthy only a few days earlier can show widespread symptoms. FHB is considered to be a monocyclic disease implying secondary spread is of minor or negligible importance (Fernando et al., 1997).

Based on estimates from natural FHB epidemics, fungicide trials, and more precise measurements in inoculation studies, yield reductions of up to 74% have been reported in small grain cereals (McMullen et al., 1997, 2012; Parry et al., 1995). Yield loss results mainly from sterility of infected spikelets and reduction in kernel size. Reduction in grain quality is due to i) presence of shriveled or discolored kernels known as *Fusarium*-damaged kernels (FDK) or 'tombstones' which are unsuitable for milling, baking, and malting (McMullen et al., 1997; Parry et al., 1995) and ii) presence of the trichothecene mycotoxins deoxynivalenol (DON) and nivalenol (NIV) and the sterol zearalenone (ZEA) produced by FHB-causing fungi (Desjardins, 2006; McCormick, 2003; Gale, 2003). These mycotoxins, of which DON is the most common, are toxic to humans and animals. Furthermore, grain with elevated mycotoxin levels is prone to discounts and/or rejection at the point of sale (Desjardins, 2006; McMullen et al., 1997; Dexter and Nowicki, 2003). Field and greenhouse studies have shown a positive, linear relationship between DON and FHB intensity (Panthi et al., 2014; Paul et al., 2005; Wegulo, 2012; Wegulo et al., 2011), implying that management measures that reduce FHB intensity will also reduce DON accumulation in grain. Some field and greenhouse studies have shown that DON can accumulate in grain to unacceptable levels when FHB intensity is relatively low or in the absence of symptoms, due to late infections well past the flowering growth stage when environmental conditions are favorable (Cowger and Arrellano, 2010; Yoshida and Nakajima, 2010; Del Ponte et al., 2007).

The acetylated derivatives of DON, 3-ADON and 15-ADON, are frequently detected in DON-contaminated grain (Mirocha et al., 2003). Some isolates of *F. graminearum* produce only 3-ADON and are designated as isolates with the 3-ADON chemotype whereas others produce only 15-ADON and are designated as isolates with the 15-ADON chemotype (Miller et al., 1991). Trichothecene mycotoxins have been shown to play a role in the aggressiveness or virulence of *F. graminearum* isolates on wheat. Desjardins et al. (1996) demonstrated that a mutant of *F. graminearum* that did not produce trichothecenes was less virulent than a wild-type, trichothecene-producing strain. Hernandez Nopsa et al. (2014b) and Panthi et al. (2014) found that isolates of *F. graminearum* collected from wheat grain elevators and fields in Nebraska, USA (all belonging to the 15-ADON chemotype), varied widely in the amount of DON they produced in vitro and in grain on wheat spikes. The investigators demonstrated in greenhouse studies that the isolates differed in aggressiveness on wheat spikes. Isolates that produced higher concentrations of DON were more aggressive than those that produced lower concentrations of the mycotoxin. In North Dakota, USA, Puri and Zhong (2010) found that *F. graminearum* isolates with the 3-ADON chemotype were more aggressive on wheat spikes of a susceptible and a moderately resistant (to FHB) wheat cultivar and produced more DON in grain than isolates with the 15-ADON chemotype. These observations indicate that *F. graminearum* isolates in a given population differ in aggressiveness and capacity to produce DON regardless of chemotype (3-ADON or 15-ADON). The recent discovery of the NIV chemotype in Louisiana and Arkansas, USA (Gale et al., 2011; Horevaj et al., 2011) highlights the diversity and complexity of *F. graminearum* populations in North America.

The evidence for DON as a virulence factor in other grains is less convincing. In Canada, Langevin et al. (2004) showed that when bread wheat, barley, oats, rye, and triticale were point-inoculated on the spikes with two isogenic strains of *F. graminearum*, the trichothecene-producing strain was more aggressive than the non-producing strain. However, Maier et al. (2006) found that DON-deficient mutants did not differ significantly from wild-type in their ability to cause disease in barley and maize, although they were unable to spread beyond the inoculated spikelet in wheat. Interestingly, a NIV-deficient mutant did show reduced virulence on maize and wheat, but not barley (Maier et al., 2006). Jansen et al. (2005) found limited spread of *F. graminearum* in barley, regardless of DON production. DON is not produced in experimental infection of rice (Goswami and Kistler, 2005), and is produced but is not essential for infection in maize (Adams and Hart, 1989; Proctor et al., 2005).

2. Management of FHB

There are several strategies for management of FHB in small grain production. Their effectiveness will vary depending on environmental conditions during the growing season, especially before, during, and after flowering, and cultivar susceptibility to FHB. They include cultural practices, including irrigation management, that reduce FHB intensity, planting resistant or tolerant cultivars, chemical control, biological control, disease forecasting, and harvesting strategies that minimize loss of grain quality and reduce DON. Integration of two or more management practices is more effective and therefore recommended.

2.1. Agronomic/cultural practices

2.1.1. Tillage and crop rotation

Tillage to bury host crop residue and crop rotation with non-hosts have been found to reduce FHB intensity and DON accumulation in grain. In Minnesota, USA, Dill-Macky and Jones (2000) found that FHB intensity was highest when wheat was planted following corn and least when wheat followed soybeans (*Glycine max*). They also found that FHB intensity was lower in moldboard-plowed plots compared to chisel-plowed or no-till plots and, averaged across tillage treatments, DON concentration in a soybean-wheat rotation was 25% lower than in a wheat–wheat