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Epidemiology of *Fusarium* diseases and their mycotoxins in maize ears

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Abstract

Fusarium species cause two distinct diseases on ears of maize, Fusarium ear rot (or pink ear rot) and Gibberella ear rot (or red ear rot), both of which can result in mycotoxin contamination of maize grain. The primary causal agent for Fusarium ear rot is *Fusarium verticillioides*, but *F. subglutinans* and *F. proliferatum* are also important. Gibberella ear rot is caused primarily by *F. graminearum*, but *F. culmorum* can also be important, especially in Europe. Aspects of the epidemiology of both diseases have been studied for decades, but only recently have efforts been made to synthesize this information into comprehensive models of disease development. Much of the work on *F. graminearum* has focused on Fusarium head blight of small-grain crops, but some of the results obtained are also relevant to maize. The primary mycotoxins produced by these fungi, fumonisins and deoxynivalenol, have differing roles in the disease-cycle, and these roles are not completely understood, especially in the case of fumonisins. Progress is being made toward accurate models for risk assessment of both diseases, but key challenges remain in terms of integrating models of pre- and post-infection events, quantifying the roles of insects in these diseases, and characterizing interactions among competing fungi and the environment.

Introduction

Fungi in the genus *Fusarium* are the most common causes of ear and kernel rot diseases of maize. The importance of these diseases has been recognized for many decades, but they remain difficult problems because high levels of genetic resistance have not been discovered and incorporated into high-yielding, agronomically desirable hybrids. The complexity of developing resistant maize hybrids is related to the multi-faceted epidemiology of *Fusarium* diseases. With a more complete knowledge of *Fusarium* epidemiology with respect to maize comes a greater probability of identifying effective resistance mechanisms.

Fusarium infection of maize ears and kernels comprises two distinct diseases that differ, but also overlap, in their epidemiological characteristics. Gibberella ear rot or 'red ear rot' usually initiates from the tip of the ear and develops a red or pink mold covering a large proportion of the ear. Usually, it is caused by *Fusarium graminearum* (teleomorph *Gibberella zeae* (Koehler, 1959; Miller, 1994; Smith and White, 1988), although in Europe several other *Fusarium* species may be associated with this disease, especially *F. culmorum* (Logrieco et al., 2002; Miller, 1994). Gibberella ear rot predominates in cooler areas or those with higher precipitation during the growing season (Bottalico, 1998; Koehler, 1959; Logrieco et al., 1993; Smith and White, 1988). Fusarium ear rot typically occurs on random kernels, groups of kernels or on physically injured kernels (Koehler, 1959; Miller, 1994) and consists of a white or light pink mold. Identical symptoms are caused by *F. verticillioides* (syn. *F. moniliforme*), *F. proliferatum* or *F. subglutinans*, but occasionally other *Fusarium* species are associated with these symptoms (Miller 1994; Smith and White, 1988). Historically, *F. moniliforme* has been reported as the most common pathogen causing Fusarium ear rot; currently, its synonym *F. verticillioides* is considered the predominant species, and it seems clear that some previous reports of *F. moniliforme* included other species such as *F. subglutinans* and *F. proliferatum*.

In the United States, Fusarium ear rot is the most common disease associated with corn ears and it can be found at low severity levels in nearly all corn fields late in the season (Koehler, 1959; Kommedahl and Windels, 1981). Aside from causing ear rot symptoms, *F. verticillioides* frequently can be isolated from symptomless kernels (Foley, 1962; Smith and White, 1988).

The epidemiology of Gibberella ear rot has been the subject of at least two reviews (Miller, 1994; Sutton, 1982), and a third review (Parry et al., 1995), focusing on Fusarium head blight of wheat, also contains information relevant to maize. The general epidemiology of Fusarium ear rot has received relatively less thorough attention; some aspects were reviewed by Miller (1994; 2001) and by Nelson (1992). In this paper, the objective is to summarize key points from the previous reviews, discuss the results of recent research not covered in those reviews (with emphasis on the infection process), and explore the epidemiology of these diseases in relation to their primary associated mycotoxins (deoxynivalenol and fumonisins).

Survival and sources of inoculum

Currently, maize is typically grown in a short rotation with one or two other crops in crop production areas with little overall crop diversity. As a result, most fields where the crop is grown have some remnants of maize plant residue in or on the soil, or such residue is present in nearby fields. This residue is the primary source of inoculum for infections of maize kernels (Smith and White, 1988). Recent studies on Fusarium head blight of wheat also confirm that maize residue is a much more important source of *F. graminearum* inoculum than wheat residue (Dill-Macky and Jones, 2000; Schaafsma et al., 2001). *Fusarium* species survive well on maize crop residue (Nyvall and Kommedahl, 1970; Sutton, 1982) as mycelium or other survival structures. *F. graminearum* produces chlamydospores that can survive periods between host crops (Sutton, 1982), whereas *F. verticillioides* (reported as *F. moniliforme*) can produce thickened hyphae which also have survival capabilities (Nyvall and Kommedahl, 1968). Additionally, existing populations in the soil can colonize senescent maize tissues that are deposited there without prior infection (Cotten and Munkvold, 1998). *Fusarium* species can colonize senescent tissues of other crop and weed species that are not considered hosts for these pathogens (Parry et al., 1995). Several types of infectious propagules can be generated from these residues. *F. graminearum* forms perithecia on the residue, and these fruiting structures forcibly discharge ascospores into the air. A great deal of research has been conducted in order to characterize the temporal patterns of ascospore dispersal and the effects of environmental variables on their formation and release. Perithecial development occurs over a wide range of temperatures, but the optimal temperature is about 28 °C, whereas the optimal temperature for ascospore release is about 16 °C (Tschanz et al., 1976). Ascospore release requires a period of perithecial dehydration and follows a diurnal pattern, with release occurring primarily at night. Paulitz (1996) suggested that 'perithecial drying during the day followed by sharp increases in RH, may provide the stimulus for release of ascospores.' Rain events are not needed for ascospore release; in fact, heavy rainfall can inhibit release. *F. graminearum* also produces macroconidia in sporodochia on crop residue. In general, it appears that ascospores are the more important infectious type of propagule for Fusarium head blight of wheat, but the situation for maize has not been investigated as intensively. *F. verticillioides* and the other species causing Fusarium ear rot do not form perithecia as readily as *F. graminearum*. These species are heterothallic and while sexual reproduction probably plays an important role in genetic recombination, it is less important in the overall epidemiology of this disease, compared to its role in Gibberella ear rot. *F. verticillioides*, *F. subglutinans* and *F. proliferatum* produce large numbers of microconidia and macroconidia on crop residues, and these asexual spores comprise the most important inoculum for Fusarium ear rot and symptomless kernel infection.

The role of seed as an inoculum source for subsequent ear infection has been controversial. *Fusarium* species are commonly seedborne in maize (McGee, 1988), although current seed production practices such as gravity separation can reduce the incidence of seedborne *Fusarium* infection (Gillette, 1999). *F. graminearum*, *F. subglutinans* and *F. verticillioides* can all be transmitted from seeds to seedlings (Cotten, 1996; Kabeere et al., 1997). *F. verticillioides* can also systemically colonize the entire maize plant without causing symptoms, and in this way can be transmitted from seed to plant to kernels (Foley 1962; Munkvold and Carlton, 1997; Munkvold et al., 1997). A similar mechanism of kernel infection has recently been reported for *F. subglutinans* (Wilke et al., 2001). In general, seed appears to be a minor source of inoculum in relation to airborne spores infecting through the silks

(Munkvold et al., 1997), but this issue is still under investigation.

Kernels can also be infected by *F. verticillioides* strains that originate from the rhizosphere. The pathway for this type of infection is probably a symptomless systemic infection of the plants, initiated through the seedling roots. This has been demonstrated in experiments where toothpicks infested with marked strains of *F. verticillioides* were placed in the soil adjacent to the seed and the strains were later recovered from kernels (Desjardins et al., 1998).

Dispersal

Recent research focused on Fusarium head blight of wheat has also yielded valuable information about the dispersal of *F. graminearum* by air. Within-field spread of ascospores has been described using a two-dimensional Gaussian model (Paulitz et al., 1999). Gradients varied from steep to relatively shallow, depending on wind direction. However, ascospores and macroconidia that escape the crop canopy can be distributed long distances. Effective inoculum can be detected at kilometer distances from any known inoculum source (Bergstrom and Shields, 2002; Francl et al., 1999).

Macroconidia of *F. graminearum* are dispersed initially by water splashes, but they are a component of the atmospheric population of *F. graminearum* propagules (Bergstrom and Shields, 2002), indicating that once they are dispersed from the sporodochium, they can be dispersed relatively long distances by air.

Fusarium verticillioides and other species causing Fusarium ear rot are dispersed primarily as microconidia, although macroconidia also act as infectious propagules. Microconidia typically are more numerous and more easily wind-dispersed than macroconidia. Propagules of *F. verticillioides*, *F. subglutinans* and *F. proliferatum* are common in the air within fields of maize (Gillette, 1999), and also at locations distant from maize fields. Ooka and Kommedahl (1977) estimated that viable spores of *F. verticillioides* (reported as *F. moniliforme*) traveled as much as 300–400 km.

Insects play a key role in the dispersal of *F. verticillioides*, but less so for *F. graminearum*. The role of insects in dispersing other *Fusarium* spp. has not been reported, but it is likely that other microconidial species such as *F. proliferatum* and *F. subglutinans* have dispersal mechanisms very similar to *F. verticillioides*. A variety of insect species have been implicated in the dispersal of *F. verticillioides*, including European corn borers (*Ostrinia nubilalis*), sap beetles (*Carpophilus* spp. and *Glischrochilus quadrisignatus*), western flower thrips (*Frankliniella occidentalis*), and corn rootworm beetles (*Diabrotica* spp.) (Dowd, 1998; Gilbertson et al., 1986). Several lepidopteran and coleopteran species were associated with increased ear rot and *F. verticillioides* infection in Africa (Cardwell et al., 2000). European corn borer larvae can acquire spores of *F. verticillioides* from leaf surfaces and transport them to kernels (Sobek and Munkvold, 1999). Adult rootworm beetles and sap beetles commonly carry spores of *F. verticillioides* and *F. graminearum*. Rootworm beetles feed on maize silks, where spores of either *Fusarium* species may be deposited and cause kernel infection. Sap beetles are attracted to maize ears that have been damaged by other insects such as the European corn borer or corn earworm. In addition, they are attracted to volatile compounds produced by *F. verticillioides* (Bartelt and Wicklow, 1999). The beetles, therefore, may be well-situated for acquiring *Fusarium* spores from infested plant material and delivering them to wounded kernels, which are very susceptible to infection. Attraction of insects to *F. verticillioides*-infected plant material may not be limited to sap beetles. Schulthess et al. (2002) reported that several lepidopteran and coleopteran insect species were more numerous on *F. verticillioides*-inoculated plants than on non-inoculated control plants.

Deposition and infection pathways

The primary infection pathway for infection of maize kernels by *F. graminearum* is via the silks, which are highly susceptible during the first 6 days after silk emergence, but less so afterwards (Reid and Hamilton, 1996). Spores reach the silks by splashing, wind dispersal or insect vectors. Some infections by *F. graminearum* are clearly initiated by direct injury to the kernels by lepidopteran insects, but this appears to be a less important pathway than silk infection.

For *F. verticillioides*, several infection pathways have been identified including silk infection, insect injury and systemic transmission from seeds or roots to kernels (Munkvold et al., 1997; Sobek and Munkvold, 1999). The relative importance of different pathways may vary among geographic areas. In the central United States, severity of Fusarium ear rot and symptomless kernel infection are closely correlated with insect injury

Table 1. Linear correlation coefficients for insect injury (kernels/ear), Fusarium ear rot severity (kernels/ear), and fumonisin concentrations (mg/kg $FB_1 + FB_2 + FB_3$) for maize grain from field experiments in Iowa, USA, from 1996 to 2001. All correlations were highly significant ($P \leq 0.001$)

	Fusarium ear rot	Fumonisins
Insect injury – 1996	0.66	0.50
Insect injury – 1997	0.89	0.69
Insect injury – 1998	0.81	0.77
Insect injury – 1999	0.92	0.77
Insect injury – 2000	0.80	0.58
Insect injury – 2001	0.73	0.72
Fusarium ear rot – 1996	—	0.69
Fusarium ear rot – 1997	—	0.76
Fusarium ear rot – 1998	—	0.73
Fusarium ear rot – 1999	—	0.89
Fusarium ear rot – 2000	—	0.76
Fusarium ear rot – 2001	—	0.62

(Table 1), primarily due to *O. nubilalis*, and this appears to be the most important infection pathway in this area. The relative importance of lepidopteran insects in disease development for *F. graminearum versus F. verticillioides* can be illustrated by how insect control affects the two diseases. Transgenic Bt maize hybrids, which are highly resistant to European corn borer injury, have much lower levels of Fusarium ear rot and fumonisins (up to 90% reduction), compared to conventional hybrids (Munkvold et al., 1999). Bt hybrids have also been shown to have lower levels of DON (as a result of reduced *F. graminearum* infection), but the reductions have been more modest (up to 59% reduction) (Schaafsma et al., 2002). A similar situation exists in Europe. As early as 1991, Lew et al. predicted, 'measures to combat the European corn borer will mainly reduce moniliformin and fumonisin contamination, but will affect zearalenone, deoxynivalenol and nivalenol contents of the ears to a much lesser extent.' Recent results have confirmed the association of mycotoxin contamination with insect injury due to the insects *O. nubilalis* (Bakan et al., 2002) and *Sesamia nonagrioides* (Avantaggiato et al., 2003) in Europe. As predicted by Lew et al. (1991), reductions in deoxynivalenol, nivalenol and zearalenone were not as great as reductions in fumonisins when these insects were controlled with transgenic insect resistance in the field in France and Spain (Bakan et al., 2002).

Infection through silks is also a significant source of Fusarium ear rot and symptomless infection by *F. verticillioides* (Desjardins et al., 2002; Munkvold et al., 1997; Nelson, 1992). In the absence of, or in addition to, insect injury, infection through silks appears to be the most important infection pathway for *F. verticillioides* (and probably *F. proliferatum* and *F. subglutinans*). Most methods for screening maize hybrids for resistance to Fusarium ear rot have employed silk inoculation or a method that wounds the kernels (Clements et al., 2003). Resistance factors have thus been identified both in the silks (Headrick and Pataky, 1991) and in the pericarp of the kernels (Hoenisch and Davis, 1994; Scott and King, 1984), which may reflect relative ability to avoid kernel injury.

Systemic transmission of the fungus seems to be of lesser importance, but the frequency of systemic transmission has varied widely among reports (Desjardins et al., 1998; Munkvold and Carlton, 1997). There may be environmental conditions under which systemic transmission is more prevalent, but this has not yet been demonstrated. Systemic transmission also has been reported for *F. subglutinans* in maize (Wilke et al., 2001), but not for other *Fusarium* species.

Factors affecting disease development and mycotoxin production

One of the most important factors that determines the level of disease and mycotoxin accumulation is genetic resistance of maize hybrids. Hybrids differ significantly in this trait, for both Gibberella ear rot (Reid and Hamilton, 1996) and Fusarium ear rot (Clements et al., 2003). In addition, physical traits of hybrids, such as husk coverage (Warfield and Davis, 1996), are related to disease susceptibility. Genetic variability in the pathogen population is a relatively unexplored factor that also may contribute to variability in disease and mycotoxins (Carter et al., 2002; Melcion et al., 1997).

Fusarium ear rot and Gibberella ear rot are favored by distinctly different conditions. Fusarium ear rot is more common in warmer and drier areas, in comparison with Gibberella ear rot (Bottalico, 1998; Miller, 1994). This pattern is associated with different temperature optima for *F. graminearum versus F. verticillioides*. The optimum temperature for *F. graminearum* has been reported as 24–26 °C (Booth, 1971) or about 28 °C (Reid et al., 1999), whereas the optimum temperature for *F. verticillioides* is reported as about 30 °C (Marín et al., 1999; Reid et al., 1999). Gibberella ear rot is favored by high levels of moisture around silking, followed by moderate temperatures and high rainfall during the maturation period (Sutton, 1982). Fusarium ear

rot generally is favored by warm, dry weather during the grain-filling period (Marasas et al., 2000). Several lines of evidence indicate that drought stress is associated with elevated levels of *F. verticillioides* infection and fumonisin accumulation in kernels (Miller, 2001). Shelby et al. (1994) showed that, in a group of maize hybrids planted in different locations in the United States, fumonisin levels were inversely correlated with June rainfall. An earlier report from Illinois indicated that Fusarium ear rot was negatively correlated with rainfall during June and July but positively correlated with rainfall during the period from August to October (Koehler, 1959). These results are consistent, in that a dry period before or during grain filling favors more severe Fusarium ear rot and higher levels of fumonisins.

There may be an interaction between drought stress, insect populations, and Fusarium ear rot and fumonisins. During several years (e.g., 1989; 1997) when drought stress occurred in the central United States, fumonisin accumulation (Munkvold et al., 1999; Murphy et al., 1993) and *O. nubilalis* populations (Bullock and Nitsi, 2001) were higher than average. Although the hypothesis is difficult to test, annual fluctuations in insect populations are likely to be a significant factor in fumonisin content of maize grain.

Environmental influences on *F. graminearum* have been incorporated into risk assessment models for head blight of wheat in North America (DeWolf et al., 2003; Hooker et al., 2002). While this type of analysis could potentially be useful for risk assessment in maize, the efforts so far have not been sufficiently detailed to facilitate application to this crop. In both cases, environmental variables were empirically related to head blight severity or deoxynivalenol levels; environmental effects on components of the disease-cycle that apply to maize cannot be extracted from the existing risk assessment models for wheat.

Efforts to synthesize the effects of environment on *Fusarium* ear diseases of maize have been more limited. Sutton et al. (1980) correlated summer precipitation with incidence of zearalenone in maize. Vigier et al. (1997) developed a regression model for ear rot incidence (caused by *F. graminearum*, *F. subglutinans* and *F. verticillioides*) based on July rainfall and an examination of ear damage and incidence of fungal infection. This model did not have predictive capabilities, since it relied on assessment of the infected ears for physical injury and kernel infection. A mechanistic model of post-inoculation fungal growth was developed by Stewart et al. (2002). This model comprised differential equations relating growth rates of *F. graminearum* and *F. verticillioides* to temperature, relative humidity and precipitation, and to a non-linear silk function that described changes in silk susceptibility over time. The key determinants of fungal growth were temperature and ear wetness, which were estimated using precipitation and relative humidity data. This model effectively predicted ear rot severity following inoculation with a coefficient of determination of 0.89. However, its predictive capability is limited by the lack of assessment of pre-infection events.

Conditions for growth of *F. verticillioides* and production of fumonisins on solid corn kernels *in vitro* have been studied extensively, primarily in a series of papers by Marín and co-workers. Marín et al. (1999) developed a polynomial regression describing fumonisin production as a function of temperature and water activity. They reported optimal conditions of 30 °C and 0.97 a_w for fumonisin B_1 production by *F. verticillioides* and 15 °C at 0.97 a_w for *F. proliferatum*. Other studies confirmed 30 °C as the optimal temperature for *F. verticillioides* growth and fumonisin production, but indicated an optimal temperature for growth of *F. proliferatum* higher than 15 °C (Marín et al., 1995). The authors also pointed out that differences in optima occurred among isolates of the same species. There were significant interactions between the effects of temperature and water activity. Growth and fumonisin production for both fungi were greatly reduced at a_w values around 0.92. Germination of both fungi was optimal around 30 °C, but this was affected by water activity (Marín et al., 1996); the minimum a_w for germination was 0.88, and both species germinated rapidly at 0.94 and above. These results have relevance to predicting fumonisin development in field-drying grain, but have not been tested as such.

Fusarium species that infect maize kernels clearly encounter other fungi, including other *Fusarium* species, and competition among these fungi can have a distinct effect on the eventual levels of infection and mycotoxin contamination. In mixed field inoculations, *F. verticillioides* interfered with the growth of *F. graminearum* and markedly reduced the levels of deoxynivalenol in grain compared to *F. graminearum* alone (Reid et al., 1999). *F. verticillioides* growth and fumonisin levels were affected inconsistently by the presence of *F. graminearum*. The correlation between ear rot symptoms and deoxynivalenol is typically high in *F. graminearum*-inoculated ears, but this correlation was poor in ears receiving a mixed inoculation

(Stewart et al., 2002). Competition among *Fusarium* species and with other fungi has also been studied on irradiated grain (Marín et al., 2001; Velluti et al., 2000). In contrast to the field results, deoxynivalenol production by *F. graminearum* was stimulated in the presence of *F. verticillioides* (Velluti et al., 2001). *Aspergillus parasiticus* reduced growth of *F. verticillioides* and *F. proliferatum* but did not affect their fumonisin production, whereas the *Fusarium* species inhibited production of aflatoxin but not growth of *A. parasiticus* (Marín et al., 2001). Interactions among all these fungi were influenced by complex interactions between temperature and water activity.

Role of mycotoxins in the disease-cycle

The ecological role of mycotoxins has been a subject of much speculation, in particular their possible role in pathogenesis in the plant. In this respect there seems to be a contrast between Gibberella ear rot and Fusarium ear rot. Initially, some evidence pointed toward a role for fumonisins in the infection process for *F. verticillioides* and *F. proliferatum*. Fumonisins are phytotoxic to maize (Lamprecht et al., 1994), and virulence of *F. verticillioides* isolates co-segregated with fumonisin production (Desjardins et al., 1995). Thorough tests of the possible role of fumonisins in ear rot disease have now been conducted. In contrast to the initial hypothesis, strains of *F. verticillioides* that lack fumonisin production due to a natural mutation or having a biosynthesis gene disrupted had the same capability to infect maize kernels and cause ear rot as wild-type, fumonisin-producing strains (Desjardins and Plattner, 2000; Desjardins et al., 2002).

Similar tests with *F. graminearum* strains that lacked trichothecene production yielded different results. On both wheat and maize, strains that lacked DON production due to disruption of the biosynthesis gene were less able to cause disease than their wild-type counterparts (Desjardins et al., 1996; Harris et al., 1999).

While DON appears to aid plant infection, the ecological role of fumonisins remains unknown. One possibility is that fumonisins aid during competition with other fungi. Fumonisins are toxic to some fungi (Keyser et al., 1999). Studies assessing competition between *F. verticillioides* and other fungi have shown that fumonisin production is not inhibited by competing fungi (Marín et al., 2001), and that *F. verticillioides* is dominant against *Penicillium* and *Aspergillus* species in grain (Marín et al., 2001) and against *F. graminearum* in inoculated maize ears (Reid et al., 1999).

A key element in eventual efforts to predict the development of mycotoxins in maize is the relationship between fungal growth and mycotoxin production. Can predictions of fungal growth or visible disease be assumed to also predict mycotoxin contamination? In general, conditions favorable for fungal growth are indeed favorable for production of deoxynivalenol and fumonisins. This is evident from the generally close correlations among ear rot symptoms, fungal growth (measured by species-specific DNA assays or ergosterol accumulation) and mycotoxin content (Bakan et al., 2002; Munkvold et al., 1999; Reid et al., 1996; Stewart et al., 2002), and the finding that the large majority of fumonisins and deoxynivalenol resides in the symptomatic kernels (Desjardins et al., 1998; Reid et al., 1996). However, it has not been demonstrated that growth and mycotoxin production by these fungi respond to temperature and water activity in an identical manner. Furthermore, correlations between fungal growth and mycotoxins are sometimes quite variable and affected by competition with other fungi (Melcion et al., 1997; Stewart et al., 2002).

Future efforts toward understanding the epidemiology of these diseases must focus on more precise relationships between environmental variables and specific components of the disease-cycle, possibly facilitated by a stronger linkage to wheat head blight research efforts already underway. For Fusarium ear rot, the role of systemic infection by *F. verticillioides* and *F. subglutinans* has not been completely elucidated; conditions under which this is an important disease-cycle component have not been identified. Perhaps more importantly, a greater understanding is needed of the conditions under which insect activity is or is not crucial, along with a connection to prediction models for insect activity.

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