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REVIEW

Biology and control of cephalosporium stripe of wheat

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Cephalosporium stripe, caused by the fungus *Cephalosporium gramineum*, is the only known vascular wilt disease of small grain cereals. The pathogen causes characteristic striping of leaf blades and sheaths, but can also result in seedling death, stunting, and sterile seed heads (white heads). Cephalosporium stripe is a disease of autumn (fall)-sown wheat, especially in cool and wet production regions. The disease is further favoured by early sowing, reduced tillage practices, low pH soils, and by frost heaving that damages roots. Infections occur almost entirely from spores produced on surface crop debris that are washed into the soil, although a low level of seed transmission can also occur. The pathogen colonizes root epidermis and cortical cells, subsequently moves into the vascular tissue, and eventually spreads throughout the entire plant. Production of fungal toxin(s) and extracellular polysaccharides probably play an important role in pathogenesis. Cultural practices such as delayed sowing, crop rotation, destruction of crop debris, liming of soil and fertilizer management all have potential to reduce the incidence of cephalosporium stripe. All of these cultural practices have negative economic impacts and/or increase soil erosion, and thus there is much interest in the development of resistant cultivars. There is potential for introgression of highly effective resistance from wild species into cultivated wheat. Genes for quantitatively inherited resistance can also be accumulated within cultivated wheat to attain moderate resistance. The continued use of cultivars with moderate resistance will probably be sufficient for long-term control of the disease.

Keywords: *Cephalosporium gramineum*, cereals, cultural practices, disease resistance, soilborne pathogens, toxins

Introduction

Cephalosporium stripe of wheat is caused by *Cephalosporium gramineum* (syn. *Hymenula cerealis*) (Ellis & Everhart, 1894; Nisikado *et al.*, 1934; Bruehl, 1956). This soilborne pathogen has a wide range of grass hosts, mainly winter cereals (wheat, oats, barley and rye), but it is also pathogenic on other grass species (e.g. *Bromus*, *Dactylis* and *Poa*) (Bruehl, 1957; Howell & Burgess, 1969; Willis & Shively, 1974). However, *C. gramineum* is generally of economic importance only in winter wheat. It is the only known true vascular wilt of wheat (Mundt, 2010). Other economically important species in the genus *Cephalosporium* include *C. maydis*, the cause of late wilt of maize (*Zea mays*), which is very similar to cephalosporium stripe (Samra *et al.*, 1963; Molinero-Ruiz *et al.*, 2010), and *C. acremonium*, which causes black bundle disease of maize and is a source of *β*-lactam antibiotics that are of great importance in both human (Dancer, 2001) and veterinary (Caprile, 1988) medicine.

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Geographic distribution

Cephalosporium stripe was first observed and thoroughly described in Japan (Nisikado *et al.*, 1934; Nisikado & Higuti, 1938). The disease was subsequently identified in Scotland in 1952 (Gray & Noble, 1960), in the Palouse region in the state of Washington, USA in 1955 (Bruehl, 1956), and in England in 1960 (Slope, 1962). Today, *C. gramineum* is found in every winter wheat growing region of the world, with the exception of Oceania (Gray & Noble, 1960; Slope & Bardner, 1965; Hawksworth & Waller, 1976; Kobayashi & Ui, 1979). Cephalosporium stripe is widespread in the Pacific Northwest of the USA, where the disease can be quite severe, and in western provinces of Canada (Bruehl, 1957; Mundt, 2010). In the USA, it is also frequent in Montana, the Great Plains, the Midwest (Sharp, 1959; Gerdemann & Weibel, 1960; Smith *et al.*, 1966; Fernandez & McShane, 1980), and some eastern states, including the Virginias and New York (Tyler & Dickens, 1957; Willis & Shively, 1974; Mathre & Johnston, 1975b; Hawksworth & Waller,

1976; Jones *et al.*, 1980; Schmale *et al.*, 2007). Oxley (2009) recently reported that cephalosporium stripe is becoming an increasing problem on winter wheat in short crop rotations in Scotland, and suggested that this increase may be exacerbated by increasing rainfall.

Life cycle

Cephalosporium gramineum survives between host crops as conidia and mycelium in association with host residues on or near the soil surface (Lai & Bruehl, 1966; Fig. 1). The pathogen can survive saprophytically on undisturbed host crop residue for as long as three years, but cannot survive unprotected in soil for more than a few months (Wiese & Ravenscroft, 1975). Both mycelial growth and sporulation are influenced by soil fungistasis (Mathre & Johnston, 1975b), soil pH and moisture (Murray, 1988a; Specht & Murray, 1989; Murray & Walter, 1991; Blank & Murray, 1998). The fungus reproduces asexually by means of unicellular phialospores (conidia) in sporodochia on leaf sheath and stem surfaces, and blastogenously inside host xylem vessels (Bruehl, 1963; Wiese & Ravenscroft, 1978). No sexual stage has been reported.

The fungus sporulates during cool, wet periods in the autumn and winter (Bruehl, 1968; Wiese & Ravenscroft, 1978). Conidia are then washed into the root zone by rainwater, becoming the infective propagules for the next crop (Bruehl, 1957, 1963; Wiese & Ravenscroft, 1973; Mathre & Johnston, 1975b; Zillinsky, 1983; Mundt, 2010). Conidia of *C. gramineum* enter wheat roots through wounds caused by freeze injury, frost heaving of soil, insects, nematodes or other mechanical injury. Although the role of freeze injury has been emphasized and is important, infection can also occur in the absence of freeze injury (Slope & Bardner, 1965; Bailey *et al.*, 1982; Stiles & Murray, 1996; Douhan & Murray, 2001).

After entering roots, conidia germinate and the fungus becomes established in the vascular system of the host plant. With crop growth in the spring, the fungus moves upward through the xylem vessels into leaves and elongating tillers, where it can extend for several internodes up the stem. It continues to multiply, and can colonize the entire plant. This creates a considerable amount of potential inoculum for the next season, which is key to pathogen survival and spread. Additionally, *C. gramineum* produces toxic metabolites that block the vascular system (Bruehl, 1957; Spalding *et al.*, 1961; Bruehl & Lai, 1966; Lai & Bruehl, 1966; Mundt, 2010).

Although the major source of inoculum for cephalosporium stripe is infested debris from previous crops, seed transmission of *C. gramineum* may be important in fields where the pathogen does not occur or where other control measures have greatly reduced inoculum loads (Arneson & Stiers, 1977; Murray, 2006). Since the first reports on cephalosporium stripe, seed has been implicated as a potential source of inoculum (Nisikado *et al.*, 1934). In his initial study on cephalosporium stripe, Bruehl (1957) could not isolate *C. gramineum* from seeds of the winter wheat cultivar Elmar harvested from a naturally infected crop. In his second attempt, he suc-

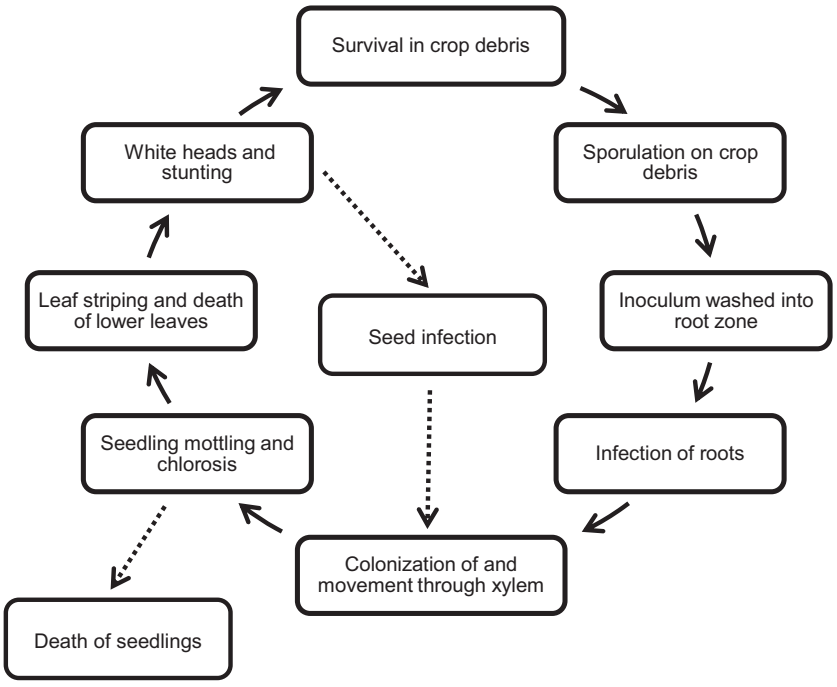

Figure 1 Disease cycle of cephalosporium stripe of wheat. Solid arrows indicate predominant pathways; dotted arrows indicate less frequent pathways.

ceeded and concluded that the pathogen was seed-transmitted, but at a very low rate, insufficient to produce an epidemic. In contrast, Ozaki *et al.* (1987) reported high incidence of embryo infection, i.e. up to 20%, and that symptoms appeared in up to 1·1% of plants grown from infected seed. More recently, Murray (2006) reported seed infection up to 0·9% and found cephalosporium stripe developed in up to 0·55% of plants grown from seed lots infected by *C. gramineum*, a level sufficient 'to allow the pathogen to become established in fields where it is not present and become a significant problem in subsequent crops'. A recently developed, PCR-based detection method for *C. gramineum* may prove useful in detecting the pathogen in both seeds and in symptomless plants (Klos *et al.*, 2012).

Symptoms

Seedling blight can occur when inoculum density is high. Infected seedlings first show a mild mosaic-like yellowing and then wilt and die (Wiese, 1972). The most recognizable symptom, chlorotic leaf striping, appears early in spring but is most apparent during jointing and heading. One to three distinct yellow stripes, often with a narrow brown centre stripe, appear on leaf blades (Fig. 2) and continue on leaf sheaths and stems. Symptoms are most obvious on the younger, upper leaves, as the lower leaves may die prematurely. Stripes might not develop on all tillers of an infected plant. Nodes on stems can darken as plants mature. Severely infected stems become stunted and ripen prematurely, producing white seed heads that often are sterile or produce a small number of shrivelled seeds (Fig. 3). A large number of stunted stems results in a 'double canopy', with a shorter layer of infected heads and a taller layer of healthy stems (T. D. Murray, unpublished data). The greatest yield losses occur when the disease is sufficiently severe to cause stunting and white heads (Nisikado *et al.*, 1934; Johnston & Mathre, 1972; Mathre & Johnston, 1975a; Morton *et al.*, 1980; Mundt, 2010).

Figure 2 Leaf symptoms of cephalosporium stripe include broad, yellow stripes with brown centres and premature death of lower leaves. Photo by Tim Murray, Washington State University, USA.

Figure 3 Severe infection by *Cephalosporium gramineum* results in stunting of tillers, sterile seed heads (white heads), and premature death of leaves. Photograph by Martin Quincke, Instituto Nacional de Investigación Agropecuaria, Uruguay.

Pathogenesis

Infection

Although for many years mechanical damage of roots – by frost heaving, insects, or other means – was believed to be necessary for infection by *C. gramineum* (Mathre & Johnston, 1975b; Morton & Mathre, 1980a), more recent research has shown that the pathogen is capable of direct penetration of intact tissues in both the roots and the crown (Douhan & Murray, 2001). Freeze–thaw cycles, once thought to allow infection by creating wounds in root tissue, may encourage infection of unwounded roots by inducing production of root exudates that stimulate germination of conidia and growth of mycelia (Bailey *et al.*, 1982). However, Anderegg & Murray (1988) demonstrated in greenhouse experiments that neither root breakage nor soil freezing is a prerequisite for severe disease to develop. Although there is little doubt that wounding increases susceptibility to infection by *C. gramineum* (Slope & Bardner, 1965; Specht & Murray, 1990), it remains unclear whether this mechanism dominates in field situations.

In order to visually determine how the fungus infects and colonizes wheat, Douhan & Murray (2001) transformed a strain of *C. gramineum* with the β-glucuronidase (GUS) reporter gene. The GUS-transformed isolate

colonized stems and roots tissues of plants in the field well before the occurrence of soil freezing, confirming that freeze injury is not required for infection. Colonization occurred as early as 15 days post-inoculation in roots, and by 20 days post-inoculation in vascular tissues. The pathogen directly penetrated stems through leaf sheaths and at sites of tiller emergence. It was able to gain access to the vascular system through root cap cells and meristematic tissues near root tips, although adventitious roots were found to be a more important entry point than other parts of the root system. Appressorium-like structures were found within cells of stems and roots, and were hypothesized to aid in penetration.

Symptom development

The symptoms of cephalosporium stripe suggest that toxins or xylem-plugging compounds may be involved in pathogenesis, prompting investigations of antibiotics, toxins and extracellular polysaccharides produced by the pathogen. Bruehl (1957) suggested that toxic metabolites of the fungus might play a role in pathogenesis. Other researchers proposed that an extracellular polysaccharide produced by the fungus resulted in plugging of the xylem (Spalding *et al.*, 1961). Later reports indicated that vascular occlusions were due to fungal proliferation that developed only after lateral extension of leaf striping (Wiese, 1972).

Graminin A was isolated and characterized from culture filtrates of *C. gramineum* by Kobayashi & Ui (1977). This toxic compound caused yellowing at concentrations of 25 μg mL^{-1} in excised leaves (Kobayashi & Ui, 1979). Graminin A possesses antimicrobial activity and affects stomatal function in the same manner as infection by *C. gramineum* (Creatura *et al.*, 1981). However, pathogenicity and virulence of *C. gramineum* have been found to be independent of *in vitro* production of either extracellular polysaccharides or graminin A (Van Wert & Fulbright, 1986).

Epidemiology

Inoculum density

In general, cephalosporium stripe symptoms increase with increasing levels of *C. gramineum* inoculum until relatively high incidences are reached (Mathre & Johnston, 1975a; Bruehl *et al.*, 1986). The relationship may be either linear or logarithmic. For resistant varieties, the response to varying levels of *C. gramineum* inoculum, measured as either percentage infection or grain yield reduction, followed a linear function, whereas for susceptible genotypes of winter wheat a logarithmic relationship was found to fit better (Mathre & Johnston, 1975a). In greenhouse studies, a logarithmic relationship was found between disease incidence and inoculum density (Specht & Murray, 1990). This implies that large reductions in inoculum are necessary in order to affect the prevalence of the disease, and that variation in environmental conditions and root wounding may have larger impacts on disease prevalence than will modest reductions in soil inoculum levels (Specht & Murray, 1990).

Environment

Infection of wheat by *C. gramineum* and the development of disease are highly influenced by environmental factors (Bruehl & Lai, 1968; Pool & Sharp, 1969; Martin *et al.*, 1989). Disease is most severe in plants grown in cool, wet, low pH soils (Blank & Murray, 1998).

Soil pH

Greater severity of cephalosporium stripe occurs in acidic soils with pH 4·5–5·5 than in soils with pH 6·0 or higher (Bockus & Claassen, 1985; Love & Bruehl, 1987; Anderegg & Murray, 1988; Specht & Murray, 1989; Murray *et al.*, 1992). The mechanisms by which soil pH influences disease are unknown; however, one plausible explanation is that low pH favours growth, sporulation and survival of *C. gramineum* in soil and, hence, the subsequent development of disease (Murray, 1988a; Specht & Murray, 1989; Murray & Walter, 1991). Survival of the pathogen in infested wheat straw is enhanced at low soil pH, perhaps as a result of increased antibiotic production (Bruehl *et al.*, 1972). Sporulation of the pathogen on colonized oat kernels and wheat straw was two- to three-fold greater at soil pH 4·5–5·5 than at pH 6·5–7·5 (Murray & Walter, 1991). Growth of *C. gramineum* is also enhanced by low pH on artificial media (Murray, 1988a). However, these increases in survival and sporulation are probably not sufficient to explain the five-fold increase in disease associated with acidic soils (Love & Bruehl, 1987; Anderegg & Murray, 1988; Specht & Murray, 1990; Blank & Murray, 1998). Blank & Murray (1998) reported a lack of a pH effect on spore germination. It has been proposed that acidic soil may favour cephalosporium stripe by promoting increased host susceptibility to root infection, possibly as a result of greater root stress and damage and/or slower wound healing (Specht & Murray, 1990). Soil pH has the greatest influence on cephalosporium stripe incidence under field conditions in years when root injury from other causes is relatively minor. Disease severity, in contrast to incidence, is not significantly affected by soil pH (Murray *et al.*, 1992; Stiles & Murray, 1996). Instead, severity may be more influenced by temperature and rainfall in the autumn and winter, or by cultural practices (Murray *et al.*, 1992).

Soil moisture

Incidence of cephalosporium stripe was found to be greater with high soil moisture in both the field and greenhouse (Bruehl, 1957; Bruehl & Lai, 1968; Anderegg & Murray, 1988; Specht & Murray, 1989), and the disease most severe in years with cool, wet autumns (Love & Bruehl, 1987; Anderegg & Murray, 1988). These conditions are often associated with increased soil heaving

during freeze–thaw cycles, which can create root wounds vulnerable to infection. However, individual processes in the life cycle of *C. gramineum* are not necessarily optimized in wet soil. For example, the relationship between sporulation and matric potential is not clear: laboratory experiments using infected oat kernels in soil have resulted in contradictory conclusions (Specht & Murray, 1989; Murray & Walter, 1991). Also, conidial germination (Blank & Murray, 1998) and survival (Specht & Murray, 1989) increased as soil moisture decreased from near-saturation to −0·06 MPa, which is still very wet. On the other hand, sporodochial production may be favoured by wet, cool weather (Wiese & Ravenscroft, 1975), and wetter soils may increase host susceptibility (Pool & Sharp, 1969).

Temperature

Both growth and survival of *C. gramineum* are favoured by low temperatures (Murray, 1988a; Murray & Walter, 1991). Sporulation per unit area and hyphal growth were less at 5°C than at 20°C on artificial media, and sporulation of the pathogen on oat kernels or straw buried in soil was 28–50 times greater at 5°C than at 15°C, perhaps as a result of increased biological competition at the higher temperature (Murray & Walter, 1991). The survival of conidia, free in the soil, is temperature-dependent and limited. Conidia have a half-life of 0·5–2·5 weeks at 23°C in autumn-collected field soil, and a half-life of 17 weeks if the soil is allowed to dry at 7°C (Wiese & Ravenscroft, 1975).

Population structure and genetic variation

Little is known about the extent of genetic variation in *C. gramineum*. Because the pathogen has no known sexual stage, it is likely to be highly clonal with a limited spectrum of genetic variation within each lineage (Anderson & Kohn, 1995). A recent study of restriction fragment length polymorphism (RFLP) analysis of ribosomal DNA (Wafai Baaj & Kondo, 2011) indicated that the internal transcribed spacer (ITS) region was nearly identical among 40 *C. gramineum* isolates from Japan, Europe and USA. RFLP analysis of the intergenic spacer (IGS) region of the same 40 isolates identified only four genotypes. In contrast, Klos *et al.* (K. L. E. Klos, J. G. Evans and T. D. Murray, Washington State University, Pullman, WA, USA, unpublished data) demonstrated high phenotypic and genotypic diversity among a collection of 270 isolates of *C. gramineum* based on cultural morphology and amplified fragment length polymorphism (AFLP) analysis of the genomic DNA.

Mathre *et al.* (1977) determined the virulence of 25 isolates of *C. gramineum* from various areas in North America on winter wheat. Most of the isolates (18) were highly virulent, causing yield reductions of 50% or more in susceptible wheat cultivars. A few isolates from Montana and New York were weakly virulent. Van Wert *et al.* (1984) tested six isolates of *C. gramineum* on 15 winter wheat lines and noted apparently different virulence patterns produced by two wildtype Michigan isolates, suggesting that races of *C. gramineum* may exist. Cowger & Mundt (1998) conducted two growth chamber experiments to assess whether isolates from different regions would rank cultivars differently. Winter wheat cultivars from the US Southern Plains and Pacific Northwest were inoculated with isolates from both regions in a complete factorial design. No significant cultivar × inoculum source or cultivar × isolate interactions were found, demonstrating an absence of pathogenic variability or virulence per se for *C. gramineum* in that study.

Impacts on crop yield and quality

Cephalosporium gramineum limits the movement of water and nutrients within stems and leaves (Bruehl, 1957), resulting in loss of both grain yield and quality. Yield reductions of up to 80% can occur with widespread infection of a susceptible cultivar in a conducive environment (Slope & Bardner, 1965; Richardson & Rennie, 1970; Johnston & Mathre, 1972; Mathre *et al.*, 1977; Morton & Mathre, 1980a; Martin *et al.*, 1989; Bockus *et al.*, 1994). Yield components most affected are kernel number and kernel weight (Slope & Bardner, 1965; Richardson & Rennie, 1970; Johnston & Mathre, 1972; Mathre *et al.*, 1977; Morton & Mathre, 1980a). Greater yield loss is related to degree of host colonization, which is reflected in the number of leaves expressing symptoms. Bockus & Sim (1982) developed a disease rating system for cephalosporium stripe in which the number of colonized leaves during early stages of kernel development (Feeke's stage 10–10·5) was positively correlated with increasing yield loss. Disease effects on kernel weight exacerbate overall yield loss because many of the light kernels can be expelled from the combine during harvest (Richardson & Rennie, 1970; Johnston & Mathre, 1972), and reductions in test weight can decrease crop value (Mathre *et al.*, 1977; Quincke *et al.*, 2012). Kernel shrivelling alters the carbohydrate to protein ratio, causing the percentage protein to increase (Johnston & Mathre, 1972; Quincke, 2009).

Mathre *et al.* (1977) determined the effect of *C. gramineum* on flour quality of four wheat lines with different levels of resistance. Overall, quality deteriorated as a consequence of cephalosporium stripe, as evidenced by reductions in test weight, flour yield and dough water absorption, as well as increases in flour ash, dough strength (farinograph peak time), farinographic stability time, and volume. However, these effects were not great enough to significantly affect baking parameters (loaf volume or grain and texture characteristics).

Control

Chemical

No chemicals are currently available commercially for the control of cephalosporium stripe, either as foliar sprays, soil applications or seed treatments. Application

of benzimidazole fungicides as soil drenches and 'in-furrow' treatments significantly reduced the incidence of cephalosporium stripe in greenhouse studies. However, in-furrow treatments in the field provided disease and yield effects that were too inconsistent to be useful commercially (Murray, 1988b).

Cultural

Cephalosporium stripe can be partially controlled by cultural practices such as crop rotation, crop residue management, altering planting date, liming and fertilizer management (Pool & Sharp, 1969; Mathre & Johnston, 1975b; Latin *et al.*, 1982; Bockus *et al.*, 1983; Raymond & Bockus, 1984; Martin *et al.*, 1989; Murray *et al.*, 1992).

Crop residue management

Destruction of infested crop residue reduces inoculum density and, therefore, disease incidence. In Kansas, a 3-year field experiment was conducted to compare the effect of five different wheat residue management practices on the incidence of cephalosporium stripe (Bockus *et al.*, 1983). Burning wheat stubble was the most effective method and deep ploughing was the second most effective method to minimize disease incidence after a severe outbreak under a continuous winter wheat production regime. However, cephalosporium stripe incidence after three consecutive years of ploughing (3·6%) was similar to that after three years of burning (3·0%). This is consistent with previous research, which showed reduced incidence of cephalosporium stripe after conventional ploughing (Pool & Sharp, 1969; Wiese & Ravenscroft, 1975; Latin *et al.*, 1982; Bockus *et al.*, 1983; Christian & Miller, 1984). Ozaki *et al.* (1988) similarly concluded that removal or burning of residue infested by *C. gramineum* resulted in lower soil inoculum density, less disease and greater grain yield than chopping the residue and leaving it on the soil surface. Severe disease incidence in no-till cropping systems was also consistently reported (Latin *et al.*, 1982; Bockus *et al.*, 1983). Therefore, residue management practices applied to control cephalosporium stripe should destroy, remove, or reduce the amount of straw left on the soil surface or in the top layer of soil to limit disease incidence during the next cropping season. However, these recommended practices conflict with attempts to reduce soil erosion.

Planting date

Delayed autumn planting has also been recommended for cephalosporium stripe control (Bruehl, 1968; Pool & Sharp, 1969; Mathre & Johnston, 1975b; Raymond & Bockus, 1984). Delayed planting results in plants with smaller root systems that constitute a smaller 'target' for infection by conidia and that are less susceptible to winter root injury. Mathre & Johnston (1975a) reported that early autumn seeding in Montana increased the number of infected tillers and white head counts under natural conditions, verifying earlier findings by Pool & Sharp (1969) and Bruehl (1968). In a 2-year trial in Kansas, Raymond & Bockus (1984) found a significant reduction in cephalosporium stripe incidence as a result of late planting in the first year but not in the second. Results from the same study also showed a 13·7% yield reduction for non-inoculated plots with every week of delay past the optimum planting date. For best economic return, careful crop management decisions must consider this and other factors that might also limit crop productivity. As with residue destruction, delayed planting increases the potential for soil erosion.

Crop rotation

Winter wheat suffers only minor damage from cephalosporium stripe when grown in rotations of appropriate length with spring cereals, non-host crops (e.g. legumes or corn), and weed-free fallow (Mundt, 2010). Rotations with three years between winter wheat crops can significantly reduce the incidence of cephalosporium stripe (Bruehl & Lai, 1968; Mathre *et al.*, 1977), although longer rotations may be required if inoculum is allowed to build to very high levels (C. C. Mundt, unpublished data) or if conditions during the rotation are very dry (T. D. Murray, unpublished data). In a study in the Palouse area of Washington State, rotations that had winter wheat every third year resulted in disease incidence of <10%, and in most cases <3% (Latin *et al.*, 1982). The generalization of three years between winter wheat crops as an adequate rotation may be strongly tied to the amount of time required for infested wheat straw to decompose (Mathre & Johnston, 1979; Murray & Bruehl, 1983).

Liming

Liming can reduce severity of cephalosporium stripe by increasing soil pH. Murray *et al.* (1992) conducted a study for four consecutive years in Washington State, USA to determine disease response to changes in soil pH in the field. In two of the four years, the incidence of cephalosporium stripe was reduced significantly by liming. Liming resulted in increased grain yield and test weight in three of the four years. However, the rates of lime necessary to raise pH levels from approximately 5·0 to approximately 6·5 or approximately 7·0 were 5·1 and 12·0 Mg ha^{-1}, respectively, making this practice economically infeasible to control cephalosporium stripe in that region.

Fertilizer management

Fertilization practices can have significant impacts on cephalosporium stripe severity. Pool & Sharp (1969) found that autumn fertilization increased disease incidence, probably due to an increase in root length and thus an increase in the number of potential infection sites. In their research, autumn fertilization increased infection by *C. gramineum* regardless of planting date. However, yield effects depended on planting date: fertilization decreased yield in the early plantings, and increased yield in later plantings. Declines in soil pH

caused by long-term application of ammonium forms of nitrogen have probably caused an increase in cephalosporium stripe severity in some wheat growing regions (Love & Bruehl, 1987). However, spring fertilization and use of alternative forms of nitrogen may be economically infeasible in many areas where cephalosporium stripe is a problem.

Host plant resistance

Variation for resistance

The identification of genetic variation in wheat for reaction to *C. gramineum* by Bruehl (1957) led to efforts to find a reliable source of resistance that can be broadly used in breeding programmes. Four resistant cultivars were identified by using hypodermic inoculations of a liquid conidial suspension into wheat culms above the crown. However, these cultivars later proved to be susceptible in the field under conditions of natural infection (Rivera & Bruehl, 1963). Artificial inoculation techniques were generally inadequate and inconsistent until the development of *C. gramineum* inoculum on oat kernels as an inoculation technique, in which a measured quantity of sterile oat kernels infested with *C. gramineum* was added with the seed at planting (Mathre & Johnston, 1975a); alternatively, oat kernel inoculum has sometimes been distributed on the soil surface (e.g. Wetzel & Murray, 2012). Using oat kernel inoculation techniques, over 1000 hard red winter wheat cultivars from the major winter wheat growing areas of the world were screened for resistance in the field (Mathre *et al.*, 1977). Although results revealed that most lines were highly susceptible, 29 cultivars showing either a low infection percentage or restricted symptom development in infected plants were selected for further characterization. Four lines with the highest tolerance to disease in terms of least reduction in yield, kernel weight, and kernels per head were considered to be useful to include in breeding programmes and three germplasm lines were subsequently registered (Mathre *et al.*, 1986). Although variation in resistance level among cultivars has frequently been demonstrated, complete resistance to *C. gramineum* has not been found in commercial cultivars (Mathre *et al.*, 1977, 1985; Martin *et al.*, 1983, 1986; Bruehl *et al.*, 1986; Morton & Mathre, 1980b; Wetzel & Murray, 2012).

Although highly effective resistance apparently does not occur within *Triticum aestivum*, such resistance does occur in wheat relatives (Jones *et al.*, 1995; Li *et al.*, 2008). Mathre *et al.* (1985) evaluated 12 wheat relatives and found that only tall wheatgrass (*Thinopyrum ponticum* syn. *Agropyron elongatum* and *Elytrigia elongata)* and intermediate wheatgrass (*Thinopyrum intermedium*, syn. *Agropyron intermedium* and *Elytrigia intermedia*) were highly resistant. Later work revealed that the *T. ponticum* chromosome 6Ae#2 was responsible for conferring resistance to the disease in a cultivated wheat background and that this chromosome showed close homoeology with wheat chromosome 6A (Cai *et al.*, 1996). Cox *et al.* (2002) evaluated 24 perennial wheat germplasm lines resulting from crosses between wheat and tall or intermediate wheatgrass and found that 13 of the lines were highly to moderately resistant to cephalosporium stripe. Translocations on wheat chromosomes 1D, 2B and 3D were found to be correlated with resistance to cephalosporium stripe in the highly resistant wheat–*Thinopyrum* amphiploid '*Agrotriticum* # 3425' (AT 3425) (Cai *et al.*, 1998).

Mechanisms of resistance

Seven hard red winter wheat cultivars varying in susceptibility to cephalosporium stripe were used to identify the types of resistance exhibited by resistant cultivars (Morton & Mathre, 1980b). Two types of resistance were observed. The first was expressed as a reduction in the percentage of diseased plants, presumed to be due to pathogen exclusion. The second was expressed as a reduction in the percentage of diseased tillers per infected plant and a reduced rate and severity of symptom development; this was presumed to be caused by restriction of pathogen spread after initial colonization. The two types of resistance appeared to be independent, suggesting that the highest levels of resistance would be obtained by combining the two types.

Research to elucidate the mechanisms of resistance and/or tolerance to this pathogen indicate that factors inherent to the roots, particularly crown tissue, of winter wheat plants and its wild relatives, might be directly associated with disease response. The difference between highly resistant wheat relatives and wheat was in the movement of *C. gramineum* through the transition zone from roots into the culm tissues, which is a common site of resistance to vascular wilt diseases. Movement of the pathogen in roots was also different, although not as distinctly so. It has also been suggested that a differential wound healing rate after root damage could be partially responsible for resistance (Mathre & Johnston, 1975b, 1990; Morton & Mathre, 1980b). Studies with a GUS-transformed isolate of *C. gramineum* showed that less crown colonization occurred in more resistant cultivars, although it was unclear if this was caused by initial exclusion of the pathogen from the crowns or by subsequent restriction in the degree of spread within the crown (Douhan & Murray, 2001).

The potential role of toxins in pathogenesis of *C. gramineum* (see Pathogenesis) suggests that toxin insensitivity may be a mechanism of resistance. Detached leaves exposed to a toxic fraction derived from *C. gramineum* showed wilting symptoms that identified toxin sensitivity within a group of 20 wheat genotypes (Rahman *et al.*, 2001). The degree of wilting in the toxin assay was highly correlated with disease reactions in the field. The assay was most useful in identifying differences among major germplasm groups (common, club, durum and a synthetic hexaploid wheat with D genome from *Aegilops tauschii*). In addition, progeny of a recombinant inbred line (RIL) population of wheat showed continuous variation for reaction to the toxic fraction (Rahman *et al.*, 2001). Further evidence that toxin insensitivity has

a role in resistance was contributed by Quincke *et al.* (2011), who found that a quantitative trait locus (QTL) on chromosome 5B had the highest additive effect and explained the greatest percentage of phenotypic variability for proportion of white heads among all QTLs identified. Chromosome 5B contains genes for insensitivity to toxins produced by other fungal plant pathogens of wheat, suggesting that toxin insensitivity may play a role in resistance to cephalosporium stripe.

Genetics of resistance

The genetics of quantitative resistance to plant disease is often less complex and more heritable than originally anticipated (Parlevliet, 1989; Young, 1996; Kover & Caicedo, 2001; Richardson *et al.*, 2006;), and this also seems to be true of resistance to cephalosporium stripe. Quincke (2009) evaluated 276 RILs derived from four crosses among Pacific Northwest × Western European genotypes. Broad-sense heritability of the four populations ranged from 0·83 to 0·93 among the eight population × year combinations. Rahman *et al.* (2001) exposed 112 RILs of the Opata 85 × M6 mapping population of the International Triticeae Mapping Initiative to a toxic fraction produced by *C. gramineum*. Using a variance components analysis, they calculated heritability on a genotype mean basis to be 0·88. In both the Quincke (2009) and Rahman *et al.* (2001) studies, frequency plots of progeny showed continuous variation, with some visual suggestion of bimodal distributions.

Quincke *et al.* (2011) conducted a QTL analysis of a RIL population derived from a cross between two commonly grown wheat cultivars from the Pacific Northwest region of the USA. Heritability for cephalosporium stripe resistance varied from 0·59 to 0·79 among the three environments. An analysis combined over three environments identified seven QTLs that accounted for 50% of the total phenotypic variance. Mean disease levels decreased with increasing number of resistance alleles, but there was still substantial variation among progeny with same number of resistance alleles. Interestingly, only three of the resistance alleles were contributed by the more resistant parent, whereas four originated from the more susceptible parent. However, the additive effects of all resistance alleles from the more resistant parent were larger than those derived from the more susceptible parent, resulting in a greater cumulative, additive effect for all resistance alleles contributed by the more resistant parent. As with other quantitative traits, the potential of marker-assisted selection for incorporating resistance to cephalosporium stripe in breeding programmes will depend on a number of factors, including whether the QTL can be validated in different host genetic backgrounds (Xu & Crouch, 2008). A recent comparison of three mapping populations of wheat identified two QTLs common to all three populations and a third QTL common to two of three populations, suggesting some potential for use of marker-assisted selection in breeding for resistance to cephalosporium stripe (Vazquez, 2014).

Integrated control and cropping system approaches

Growers often use a combination of practices to attain adequate levels of cephalosporium stripe control or to balance disease control against negative economic or soil conservation impacts. For example, it is common to grow a cultivar with moderate resistance in combination with a 1–2 week delay of autumn planting date, especially when disease pressure is high (Mundt, 2010). Controlling the disease on a susceptible cultivar, on the other hand, would require an even later planting date, risking large yield reductions and vulnerability to soil erosion.

Cultural practices that would be economically or environmentally unacceptable if practised continuously can sometimes still provide adequate control of cephalosporium stripe when implemented periodically. In dryland regions where wheat/summer fallow rotation is practised, it is common to burn wheat stubble or rotate to a spring cereal every seventh year (C. C. Mundt, unpublished data). This practice often reduces inoculum levels sufficiently to subsequently produce three cycles of winter wheat/summer fallow before again burning or rotating. The practice can also provide significant control of serious winter annual weeds, such as cheatgrass (*Bromus tectorum*). In Montana, researchers developed a more flexible decision aid to determine when it is economically appropriate to rotate to a spring-sown cereal crop to control cephalosporium stripe. This decision aid was based on actual disease levels in growers' fields, decay relationships of inoculum over time, and local economics of cereal production (Johnston & Mathre, 1985).

Cropping system practices also are relevant to the management of host plant resistance. The continued use of cultivars with moderate resistance to cephalosporium stripe may reduce field inoculum levels, and thereby increase disease control over years. Shefelbine & Bockus (1989) studied the impact of growing a monoculture of winter wheat cultivars with various levels of resistance on the intensity and progress rate of cephalosporium stripe over a 3-year period. Continuous planting of moderately resistant cultivars reduced the incidence and severity of cephalosporium stripe over the trial period. They suggested that the available levels of resistance were adequate to reduce inoculum over time and control disease in the long term, a view supported by field observations in the Pacific Northwest region of the USA (C. C. Mundt, unpublished data). Although such results might be expected to be driven by reduced inoculum production on moderately resistant cultivars, this mechanism was not confirmed in a subsequent glasshouse study (Shefelbine & Bockus, 1990). On-farm trials indicate that use of cultivar mixtures can sometimes provide yield advantages at sites where cephalosporium stripe pressure is high (Mundt & Karow, 1995; Mundt, 2002). This effect is probably due to some type of compensatory interaction between cultivars, as cultivar mixtures do not seem to lessen the incidence of cephalosporium stripe (Mundt, 2002).

Conclusions

Cephalosporium stripe will continue to be an important disease in regions where environmental conditions have historically been very favourable. The disease may also become important in other regions when reduced tillage methods become more widely adopted, and in areas where climate change tips the environmental balance in favour of the pathogen. Much has been learned in recent decades in terms of the pathogenesis and epidemiology of cephalosporium stripe. Although many cultural practices can influence the incidence of cephalosporium stripe, host plant resistance is the preferred control method for both economic and environmental reasons. There is potential to transfer highly effective resistance from related species of wheat. However, there also is substantial quantitative variation for resistance within the wheat gene pool that can be selected via traditional field methods and, perhaps in the future, via marker-assisted selection. Until sufficient levels of resistance are attained, integrated approaches and management of cropping systems will continue to be highly important to control cephalosporium stripe.

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