Chapter 6
Diseases Which Challenge Global Wheat Production - Root, Crown, and Culm rots

Richard W. Smiley, David Backhouse, Philippe Lucas, and Timothy C. Paulitz

SUMMARY

1) With few exceptions, root, crown, and culm rots are especially prevalent in cropping systems characterized by high residue retention, reduced tillage, or high frequency of host crops. Most of these diseases are not yet effectively managed by genetic resistance, fungicides, or biological agents. Optimal disease management generally requires changing the soil environment to reduce survival of the pathogens between susceptible host crops, or the virulence of pathogens during the infective stage.
2) Rotation to non-hosts is an effective management strategy for common root rot, take-all, Cephalosporium stripe, and eyespot but not crown rot, Pythium root rot, or Rhizoctonia root rot.
3) Other management practices that reduce damage caused by several of these diseases include preventing growth of volunteer cereals and weed hosts during the interval between crops, banding a portion of the fertilizer below the seed, adjusting the planting date, using a seed drill that causes intense soil disturbance in the seed row, and protecting seedlings by applying a fungicide to the seed. Only eyespot can be controlled by applying fungicide to the foliage. Crop management systems to control take-all and eyespot are optimized using models.
4) Severity of take-all and Rhizoctonia root rot may increase at the beginning of a wheat monoculture and then begin to decline in severity. These disease decline phenomena are mediated through influences of the soil microbiota.
5) Wheat cultivars with useful levels of resistance are available to suppress damage by common root rot, crown rot, Cephalosporium stripe, and eyespot. Molecular markers are used to detect resistance genes in seedlings, and DNA-based real-time polymerase chain reaction (PCR) assays are available to identify and quantify these pathogens in soil or plants. These assays plus data interpretation based on disease epidemiology have been used commercially to predict potential grain yield loss from several of these diseases.

INTRODUCTION

Most wheat (*Triticum* sp.) diseases caused by root-, crown- and lower culm-infecting fungi are not yet effectively managed by genetic resistance or by application of a fungicide or biological control agent. The best management strategy for many of these diseases continues to depend upon changing the soil environment in ways that either influence the survival of the pathogen between

summarized in this chapter (Table 6.1) are heavily influenced by soil physical and chemical properties, by interactions with associated microbes and micro-fauna in soil and on plant surfaces, and by the capacity of plants to serve as hosts for growth and multiplication. The complexity of factors affecting these pathogens before and during pathogenesis is immense.

Updated summaries of these diseases cannot be complete without acknowledging the great contributions that have been made by large numbers of practicing farmers, soil microbiologists, soil ecologists, soil physicists, soil chemists, agricultural engineers, agronomists, plant pathologists, botanists, geneticists, and wheat breeders. An introduction to these founding contributions can be found in Butler (1961), Baker and Snyder (1965), Garrett (1970), Griffin (1972), Bruehl (1975, 1987), Schippers and Gams (1979), Krupa and Dommergues (1979), Cook and Baker (1983), and Parker et al. (1985). Additional reference books are cited in appropriate sections of this chapter.

Many similarities occur among pathogens and diseases of wheat and other grasses. Complementary insights into the biology of pathogens causing four of these wheat diseases are summarized from a different perspective in treatises by Smith et al. (1989), Clarke and Gould (1993), Couch (1995), and Smiley et al. (2005a).

The content of this chapter also would be incomplete without referencing guidelines for studying these pathogens and gaining a greater visual appreciation of disease symptoms and pathogen characteristics. Methods to isolate and study the pathogens are provided by Singleton et al. (1992), among others. Excellent color images are available in Zillinsky (1983), Murray et al. (1998), Wallwork (1992, 2000), Bailey et al. (2003), and Bockus et al. (2009).

COMMON ROOT ROT

Common root rot is a name originally applied to a complex of diseases caused by several species of *Bipolaris* (‘*Helminthosporium*’) and *Fusarium* (Butler 1961). Current usage restricts this name to root and stem base diseases caused by *Bipolaris sorokiniana*, although this pathogen frequently occurs in association with Fusarium crown rot (Windels and Wiersma 1992; Smiley and Patterson 1996; Fernandez and Chen 2005). Common root rot was considered a very serious disease, especially in Canada and Australia, in the early- to mid-20th century (Butler 1961; Tinline et al., 1991) and can cause grain yield losses as great as 25% (Wildermuth et al., 1992). Common root rot has received less attention in recent years, possibly because of the increasing importance of Fusarium crown rot, and because most research on this pathogen now focuses on the foliar disease, spot blotch (Kumar et al., 2002). The significance of common root rot for modern cultivars in contemporary cropping systems is not well known and is probably underestimated.

Symptoms and epidemiology

All parts of the wheat plant can be infected. Initial symptoms are dark necrotic lesions. The classic symptom in soilborne infections is a dark lesion on the subcrown internode (Color Plate 12a), which can extend up to the crown, and in severe infections, up the lower internodes on the stems. Plants with severe subcrown internode symptoms have reduced root growth, especially of crown roots (Kokko et al., 1995). The roots are not usually the major site of infection, although they may show some browning (Fedel-Moen and Harris 1987; Kokko et al., 1995). Lesions of common root rot (Color Plate 12a) are much darker than those of Fusarium crown rot (Color Plate 12b). On the stems, they appear streaky rather than uniform around the circumference of the stem. Severe common root rot is often associated with water stress (Piccinni et al., 2000). Plants may be stunted, and whiteheads (Color Plate 13a) form if plants are water-stressed late in the season.

The main source of inoculum is the conidia, which are large and strongly pigmented and can survive for several years in soil (Wildermuth and McNamara 1991). *Bipolaris sorokiniana* can be seedborne (Couture and Sutton 1980), but seed transmission is unlikely to occur from common

Table 6.1. Synopsis of primary pathogens, infection sites, symptoms, and effects of crop management practices for diseases addressed in Chapter 6.

Disease	Principal pathogen(s)	Primary infection site	Primary symptoms	Conditions leading to highest disease severity	Practices that generally minimize crop damage	Efficacy of seed-applied fungicides	Efficacy of genetic resistance
Common root rot	*Bipolaris sorokiniana*	stem base, crown, subcrown internode	lesion on subcrown internode; stunting; whiteheads	cultivation; planting wheat annually; drought	crop rotation; summer fallow; shallow sowing; delay autumn planting date; plant resistant cultivar	poor to fair	poor to fair
Fusarium crown rot	*Fusarium culmorum*, *F. pseudograminearum*	stem base, crown, subcrown internode	browning of lower leaf sheath; lesion on subcrown internode; dry rot of crown and roots; whiteheads	no-till or minimum tillage; host crops annually or 2-year rotations; planting winter wheat too early; high N fertility; drought	destroy infested crop residue; crop rotation; judicious application of N; delay autumn planting date; plant resistant cultivar; shallow sowing	poor; more effective for spring than autumn plantings	poor to fair; currently not widely deployed
Pythium root rot	*Pythium ultimum, P. irregulare,* and others	seed, roots	seed rot; damping-off; root rot; stunting; delayed maturity	cool, wet soil; planting host crops annually; planting winter wheat too late or spring wheat too early	plant when soil temperature favors rapid seed germination; band a starter fertilizer below seed; control green bridge; plant fresh seed	poor to fair; more effective for spring than autumn plantings	not available
Rhizoctonia root rot	*Rhizoctonia solani* AG-8, *R. oryzae*	roots	root rot; stunting; delayed maturity	no-till; planting host crops annually; planting winter wheat too late or spring wheat too early	control green-bridge; disruption of seed row at planting; pre-plant tillage; band a starter fertilizer below seed; summer fallow	poor for wheat planted in autumn; poor to fair for spring wheat	not available
Take-all	*Gaeumannomyces graminis* var. *tritici*, *G. graminis* var. *avenae*	roots	root rot; stunting; blackened stem base in wet or humid sites; whiteheads	planting host crops annually; planting winter wheat too early; major increase in soil pH after lime application	crop rotation; control green-bridge; manage form of N absorbed by roots; delay autumn planting date	poor to fair; more effective for spring than autumn plantings	not available
Cephalosporium stripe	*Cephalosporium gramineum*	roots & crown	long leaf stripes merging into dark veins in leaf sheath; stunting; whiteheads	no-till or minimum tillage; acid soil; host crops annually or 2-year rotations; planting winter wheat too early; freeze/thaw cycles during winter	crop rotation; plant resistant winter wheat; plant spring wheat; destroy infested crop residue; lime acid soils; control green-bridge; delay autumn planting	not effective	fair to good
Eyespot	*Oculimacula yallundae*, *O. acuformis*	stem base	elliptical lesions on lower culm; plant lodging; whiteheads	host crops annually; planting winter wheat too early	plant resistant winter wheat; delay autumn planting date; plant spring wheat; crop rotation; apply foliar fungicide	fair to good; not necessary for spring plantings	good

root rot-infected plants unless spot blotch is also present.

Incidence of infection is related to the density of conidia in soil (Tinline et al., 1988). Spore density, and disease incidence and severity, are higher in cultivated soils than in no-tillage systems where seed is directly drilled into residue of a previous crop (Reis and Abrao 1983; Mathieson et al., 1990; Wildermuth et al., 1997). This may be related to dispersion of spores within the soil during cultivation. A high proportion of spore production from wheat residues is from crowns (Duczek 1990), and removal of residue by burning has been shown to reduce the severity of common root rot (Wildermuth et al., 1997).

High spore populations and disease severity occur under continuous wheat (Wildermuth and McNamara 1991; Conner et al., 1996). Continuous wheat also increases the average aggressiveness of field populations towards wheat (El-Nashaar and Stack 1989). Wheat and barley (*Hordeum vulgare* L.) are among the most susceptible hosts, with oat (*Avena sativa* L.) being less susceptible while legumes are generally resistant (Wildermuth and McNamara 1987). Spore populations decline under fallow or non-hosts, such as oilseeds or legumes, compared with cereals (Wildermuth and McNamara 1991).

The effect of nutrients on common root rot is equivocal. Most attention has been paid to chloride, which reduces disease severity in some experiments but has no consistent effect in others (Windels et al., 1992; Tinline et al., 1993). Low nitrogen levels possibly reduce disease severity, so that severity in continuous wheat with no added nitrogen is less than in wheat-legume sequences, despite the rotation effect (Dalal et al., 2004; Fernandez and Zentner 2005). This is probably due to an interaction between high nitrogen levels and water use (Dalal et al., 2004).

Incidences of common root rot and Fusarium crown rot are often inversely related when both diseases are present. Severity of common root rot declined as incidence of Fusarium crown rot increased in a long-term trial (Wildermuth et al., 1997). This may represent competition between the pathogens, because *B. sorokiniana* is a poor competitor with *Fusarium* species in plant tissue (Tinline 1977). However, *B. sorokiniana* is strongly antagonized by the crown rot *Fusarium* species in culture, and symptoms of the two diseases are similar. It is therefore possible that *B. sorokiniana* is difficult to detect by isolation or symptoms in the presence of *Fusarium* species. Because of possible interactions, reports of common root rot when it co-occurs with Fusarium crown rot must be interpreted with caution.

Causal organism

Bipolaris sorokiniana (Sacc.) Shoemaker [teleomorph = *Cochliobolus sativus* (S. Ito and Kurib) Drechsler ex Dastur] has a worldwide distribution and a wide host range among small grain cereals and grasses (Kumar et al., 2002). This pathogen is widely reported in older literature as *Helminthosporium sativum* Pammel, C.M. King & Bakke. The sexual state is readily produced in the laboratory (Singleton et al., 1992) but has not been reported in the field.

Some evidence exists for host specialization within the species. Isolates from barley are more virulent to barley roots than to wheat roots, and vice versa (Conner and Atkinson 1989). Wheat isolates vary greatly in their virulence on wheat leaves (Duveiller and Garcia Altamirano 2000) but no clear evidence for races has been found.

Bipolaris sorokiniana produces several sesquiterpenoid toxins, the most important of which is prehelminthosporol (Kumar et al., 2002). Isolates with low prehelminthosporol production in culture tend to have reduced virulence on barley roots (Apoga et al., 2002), but evidence for a major role of toxins in pathogenesis is not available.

Disease management

Rotation to non-hosts is the primary management strategy for common root rot. This must be combined with effective management of grassy weeds. Some benefit may also be gained by rotation among cereal hosts, because this acts against selection for highly aggressive strains (Conner et al., 1996). Zero-tillage (no-till or direct-drill) reduces common root rot severity,

and shallow sowing can also reduce contact with inoculum (Tinline and Spurr 1991). Avoidance of stress in the crop reduces severity. In general, management practices directed at more conspicuous diseases like take-all and Fusarium crown rot will also reduce common root rot in most areas.

A moderate level of resistance is available among wheat cultivars (Wildermuth et al., 1992). Most current breeding work is directed against spot blotch, but there is evidence that at least some sources of resistance to spot blotch will also be effective against common root rot (Arabi et al., 2006).

FUSARIUM CROWN ROT

Fusarium crown rot is a generic term for diseases of stem bases caused by several species of *Fusarium*. These diseases are also widely known as foot rot. Crown rot or similar diseases have been reported from all areas where wheat is grown (Nelson et al., 1981; Summerell et al., 2001b). The importance of this disease complex has risen with increasing adoption of residue retention and reduced tillage practices, which favor the buildup of inoculum and greater levels of infection (Windels and Wiersma 1992; Burgess et al., 1993; Smiley et al., 1996a).

The fungi which cause Fusarium crown rot can also incite Fusarium head blight. The focus of most community concern and research in recent years has been on head blight because of the potential for mycotoxin contamination of food and feedstuffs. However Fusarium crown rot remains a serious problem, especially given the difficulties in managing it within the constraints imposed by modern cropping systems.

Symptoms and epidemiology

Symptoms of crown rot are first seen as necrotic lesions or more general browning on leaf sheaths and stem tissue. Infections from inoculum in the soil may appear first as brown lesions on the subcrown internode (Color Plate 12b) while those from surface residue occur through the crown (Color Plate 13b; Summerell et al., 1990). Infected crown roots exhibit a dry, brown discoloration.

Infection from rain-splashed conidia may be through leaf sheaths above the soil surface, leading to browning first appearing at nodes above the crown (Jenkinson and Parry 1994). For all sites of primary infection, the near-uniform browning may then progress for several internodes up the stem (Color Plate 13c, left side). A pink discoloration under leaf sheaths or in other tissues may also be seen, and orange sporodochia (conidial masses) can form on nodes under high humidity. If infected plants are water-stressed during grain filling, premature ripening may occur, leading to whiteheads (Color Plate 13a). The whitehead symptom generally does not occur when moisture is adequate for optimal plant growth.

Following physiological maturity the fungi aggressively colonize stem tissue and survive as mycelium in the infested residues. *Fusarium culmorum* can also survive as chlamydospores in the soil.

Fusarium crown rot in most environments behaves as a monocyclic disease with incidence being dependent on initial inoculum in the soil or crop residue (Backhouse 2006). Secondary spread appears to be limited in dry environments, but splash dispersal of conidia can be significant in more humid environments (Jenkinson and Parry 1994). It is not known what role ascospores may play in infections of stem bases.

High nitrogen levels favor disease in two ways. The increased leaf area predisposes plants to late-season water stress under dry conditions, increasing severity (Cook 1980). High nitrogen levels also increase incidence of infection (Smiley et al., 1996a), presumably by increasing susceptibility. Zinc deficiency may also increase susceptibility (Grewal et al., 1996).

Causal organisms

A large number of *Fusarium* species are capable of causing stem base disease in wheat (Akinsanmi et al., 2004). Of these, *F. culmorum* (W. G. Smith) Sacc., *F. pseudograminearum*

O'Donnell & T. Aoki (teleomorph = *Gibberella coronicola* T. Aoki & O'Donnell), *F. graminearum* Schwabe [teleomorph = *G. zeae* (Schwein.) Petch], and *F. avenaceum* (Fr.) Sacc. (teleomorph = *G. avenacea* R.J. Cook) have been considered the most important species worldwide.

Fusarium culmorum has the most widespread recorded distribution from wheat stem bases among these species, being found on all continents. Parry et al. (1994) suggested that *F. culmorum* was typically found in warmer, drier cereal growing areas. However, surveys in Australia and North America indicate that it is most prevalent in the cooler or higher rainfall parts of the wheat growing areas in these regions (Smiley and Patterson 1996; Backhouse et al., 2004). *Fusarium culmorum* differs from the other species in that chlamydospores play an important part in epidemiology (Sitton and Cook 1981). Infection rates are less affected by surface plant residues compared to other species associated with crown rot (Windels and Wiersma 1992). No teleomorph is known, but evidence for recombination has been found in field populations (Tóth et al., 2004), and it is likely that a sexual state does exist.

Fusarium pseudograminearum was formerly known as *F. graminearum* Group 1 (Aoki and O'Donnell 1999). The teleomorph, *G. coronicola*, is rarely found in the field (Summerell et al., 2001a). This species is the most important cause of crown rot in Australia and South Africa (Burgess et al., 1975; Van Wyk et al., 1987; Backhouse and Burgess 2002; Chakraborty et al., 2006). It is also prevalent in western North America (Smiley and Patterson 1996; Clear et al., 2006), occurs at low frequency in the Mediterranean region and Asia (Bentley et al., 2006; Tunalı et al., 2008), and has not been reported from South America or Europe north of the Alps.

The role of *F. graminearum* as a cause of Fusarium crown rot is unclear because of uncertainty about the identity of fungi reported under this name. In literature prior to the 1980s *F. pseudograminearum* was reported as *F. graminearum*, and even recently the two species have not always been distinguished. O'Donnell et al. (2004) segregated *F. graminearum* from eight cryptic sister species, many of which also occurred on cereals. *Fusarium graminearum* in the strict sense is best known as a head blight pathogen. It is frequently isolated from wheat stem tissue, and shows a similar range of aggressiveness to wheat crowns as *F. pseudograminearum* in pathogenicity tests under controlled conditions (Akinsanmi et al., 2004). However, surveys in several countries which have used modern taxonomic concepts have generally failed to report *F. graminearum* as a significant component of the Fusarium crown rot complex compared with other species (Pettitt et al., 2003; Akinsanmi et al., 2004; Backhouse et al., 2004; Smiley et al., 2005b). The teleomorph, *G. zeae*, occurs readily in the field, unlike other species in the complex.

Fusarium avenaceum is typically prevalent in cooler climates such as eastern Canada and northern Europe (Hall and Sutton 1998; Pettitt et al., 2003), although it occurs more widely as a minor component of the disease complex. Pathogenicity to wheat subcrown internodes and crowns is similar to that of *F. culmorum*, *F. graminearum*, and *F. pseudograminearum* (Fernandez and Chen 2005; Smiley et al., 2005b). The teleomorph, *G. avenacea*, has not been found in the field. *Fusarium avenaceum* appears to have a broader host range than the other species but is more frequently associated with diseases of legumes (Satyaprasad et al., 2000).

Disease management

Chemical control of Fusarium crown rot is generally unsuccessful. Management therefore depends mainly on cultural practices and resistance. Because incidence of disease caused by *F. pseudograminearum*, *F. graminearum*, and *F. avenaceum* is strongly correlated with the quantity of infested residue (Windels and Wiersma 1992; Smiley et al., 1996a; Wildermuth et al., 1997; Backhouse 2006), practices which either remove the residue or allow its decomposition are effective management tools. Stubble burning, either after harvest or

immediately before sowing, can maintain the disease at low levels even in continuous wheat production (Burgess et al., 1993, 1996). However, this practice has become unacceptable in many areas. Amounts of surface residue and stubble burning may have less effect where *F. culmorum* is present (Windels and Wiersma 1992; Smiley et al., 1996a), presumably because of the presence of chlamydospores in the soil (Bateman et al., 1998).

Rotation with non-hosts allows time for natural mortality of the pathogen. The rotations that can be used, and their effectiveness, differ between pathogens. *Fusarium pseudograminearum* has the narrowest host range, and almost any non-cereal host, including grain legumes and oilseeds as well as sorghum [*Sorghum bicolor* (L.) Moench] can be used (Burgess et al., 1996; Kirkegaard et al., 2004). Maize (*Zea mays* L.) has not been recorded as a host for *F. pseudograminearum*, but would be a poor choice if *F. culmorum* or *F. graminearum*, which do infect maize, are present. Crop rotation may have less effect on *F. culmorum* than on *F. pseudograminearum*, presumably because of the longevity of chlamydospores relative to mycelium in stubble (Sitton and Cook 1981; Summerell and Burgess 1988).

Nitrogen management has a strong impact on disease incidence and severity, particularly in low-rainfall environments where plants mature without the benefit of late-season rainfall (Cook and Veseth 1991; Smiley et al., 1996a). Application of nitrogen at rates greater than required for the expected or attained grain yield typically increases the expression of whiteheads, reduces grain yield and test weight, and increases grain protein content. In low-rainfall regions where low nitrogen inputs are required to produce low-protein soft-white wheat, severity of crown rot is greatly increased when growers apply higher rates of nitrogen to produce high-protein market classes of wheat.

Wheat cultivars differ in susceptibility to crown rot, although the range available among released cultivars is usually small (Wallwork et al., 2004). Moreover, cultivars or lines expressing resistance in some instances are often highly variable in responses over seasons and geographic areas (Smiley, unpublished data). Durum wheat (*T. turgidum* ssp. *durum*) tends to be more susceptible than bread wheat (*T. aestivum* L.) (Kirkegaard et al., 2004) and should be avoided in high disease-risk situations. Wheat lines with higher levels of resistance have been identified in a wide range of backgrounds including bread wheat, *T. zhukovskyi* Menabde et Ericzjan, *T. dicoccum* Schrank, and synthetic wheat (Wallwork et al., 2004; Nicol et al., 2007). Resistance appears to be expressed against all pathogen species (Miedaner 1997; Wallwork et al., 2004). Both seedling and adult-plant forms of partial resistance have been identified for *F. pseudograminearum*. For screening purposes, seedling but not adult-plant resistance has been inversely correlated with the genetically determined depth at which crown tissue is formed for each wheat genotype (Wildermuth et al., 2001). Genotypes with seedling resistance form crowns at more shallow depth than susceptible genotypes, possibly enabling them to partially escape or delay infection. Most work on resistance to *Fusarium* in wheat has been done with head blight, but the lack of correlation between resistance for head blight and resistance for crown rot in rye (*Secale cereale* L.) (Miedaner et al., 1997) suggests that head blight resistance will not necessarily be effective against crown rot.

PYTHIUM ROOT ROT

Pythium root rot of wheat is caused by numerous species of *Pythium*. These species have a broad host range including maize, barley, oat, rye, and many broadleaf crops such as pea (*Pisum sativum* L.), chickpea (*Cicer arietinum* L.), lentil (*Lens culinaris* Medik.), and soybean [*Glycine max* (L.) Merr.].

Pythium species have a worldwide distribution and are found in most agricultural soils (Hendrix and Campbell 1970; Martin and Loper 1999). As many as six species have been isolated from a single soil sample and more than 30 species have been isolated from wheat (Farr et

al., 2007), although not all species are equally virulent (Chamswarng and Cook 1985; Ingram and Cook 1990; Higginbotham et al., 2004b). Because of the ubiquitous nature of these pathogens and the chronic nature of the disease, Pythium root rot was termed "the common cold of wheat" (Cook and Veseth 1991).

The impact of *Pythium* on wheat was not realized until field trials were conducted with the fungicide metalaxyl. This oomycete-specific fungicide increased yields as much as 0.8 t ha^{-1} in the Pacific Northwest USA (Cook et al., 1980; Smiley et al., 1996b). Treatment of *Pythium*-infested soil with fumigation increased yields 13% to 36% (Cook et al., 1987). *Pythium* root rot has also been reported on wheat in Australia (Pankurst et al., 1995), southeast USA (Milus and Rothrock 1997), and Turkey (Tunalı et al., 2008) but can probably be isolated from soils in most wheat growing areas.

Symptoms and epidemiology

Pythium primarily infects juvenile tissues including embryos, emerging seedlings, root tips, lateral roots, and root hairs. High pathogen populations can reduce emergence of seedlings and stands of wheat (Color Plate 14a), due to death of the seedling from infection either before emergence (pre-emergence damping-off) or after emergence (post-emergence damping-off). Rotted seeds can also be found in soil. However, with cereals such as wheat, these symptoms are rare. Usually, an embryo-infected seedling will emerge successfully and then remain stunted (Fukui et al., 1994).

Diagnosis of *Pythium* is difficult in wheat because of the lack of distinctive above-ground symptoms. In general, wheat will appear stunted, but without disease-free plants for comparison, this generalized mild stunting may not be noticed in the field. However, because the pathogen destroys root tips, feeder roots, and root hairs, the ability of the plant to take up water and nutrients is reduced and symptoms of nutrient deficiency or water stress can become apparent. Maturity can be delayed, plant height is reduced, plants have fewer tillers, and heads are poorly filled. Infected roots may appear yellow-brown in color, but usually the rotted roots quickly disintegrate and are not recovered.

Pythium species survive as thick-walled oospores or sporangia that are produced in infected roots. When roots decay the inoculum is released into the soil. Most inoculum is present in the top 10 to 15 cm of soil. The pathogen can colonize clean wheat straw, chaff, or green manure as a nutrient source to support mycelium growth and to increase inoculum density (Cook et al., 1990). In the Pacific Northwest, *Pythium* populations averaged 350 to 400 propagules per gram of soil (Cook et al., 1990), exceeding the threshold of 200 propagules per gram of soil needed to cause growth reductions (Fukui et al., 1994). When a seed is placed in the soil, or a root tip grows near a *Pythium* spore, seed or root exudates stimulate the germination of the spore, resulting in rapid chemotrophic attraction to and infection of the seed or root (Hering et al., 1987; Fukui et al., 1994; Martin and Loper 1999). Spore germination can occur within a few hours and infection within 10 to 24 hours. Many *Pythium* spp. are also capable of rapid mycelial growth. In wet soils, some species can form motile swimming spores (zoospores) which are chemotactically attracted to root tips and seeds.

Pythium diseases are favored by cool, wet, poorly-drained soils with high clay content and low pH (Fukui et al., 1994). These cool wet conditions are often associated with delayed sowing of fall-planted crops (Smiley et al., 1996b) or result from excessive crop residue in no-till systems, which reduce the warming and drying of the soil during the spring (Cook et al., 1990). However, improved water infiltration often associated with long-term no-till may reduce the occurrence of *Pythium* diseases. Maximum infection by *P. ultimum* and *P. irregulare* occurs at 10°C and 5°C (Ingram and Cook 1990), but some species can act as snow molds, infecting under snow cover at 0°C to 3°C.

Causal organisms

Although 30 species of *Pythium* have been associated with wheat, most reports focus upon *P.*

arrhenomanes Drechs., *P. graminicola* Subr., *P. ultimum* Trow, *P. aristosporum* Vanterpool, *P. irregulare* Buisman, *P. torulosum* Coker & Patterson, *P. sylvaticum* Campbell & Hendrix, and *P. heterothallicum* Campbell & Hendrix. *Pythium debaryanum* Hesse is frequently cited in older literature but this is not currently recognized as a valid species and many records may be incorrect. Recent surveys using classical and molecular techniques have identified 13 species of *Pythium* on wheat in the Pacific Northwest USA, including a new species, *P. abappressorium* (Paulitz and Adams 2003, Paulitz et al., 2003a). Most species are capable of causing significant reductions in root biomass (Higginbotham et al., 2004b), but the most virulent were *P. ultimum*, *P. irregulare* group 1, and *P. irregulare* group IV sensu Matsumoto (identified as *P. debaryanum* in that paper).

Pythium species produce oospores, which result from the fertilization of oogonia by antheridia. Oospores are generally spherical, from 15 to 40 µm in diameter, and result from sexual recombination. Asexual sporangia can germinate directly to form hyphae or indirectly to form zoospores, and can be spherical, filamentous, or lobed in shape. Identification is based on morphology of sporangia, oospores, oogonia, and antheridia (van der Plaats-Niterink 1981), or on molecular methods based on sequencing of the internal transcribed spacer (ITS) region of the rDNA (Lévesque and de Cock 2004; Schroeder et al., 2006).

Disease management

Although no high-level resistance or tolerance is found in adapted wheat cultivars, minor differences do occur in susceptibility among cultivars (Higginbotham et al., 2004a). Resistance is not a management option at the present time. Crop rotation is not used for management because of the wide host range of *Pythium* species. However, different hosts select for different *Pythium* species (Ingram and Cook, 1990) and recent surveys have demonstrated shifts in species composition resulting from different crop rotations or cropping systems (Schroeder et al., 2007). For example, *P. irregulare* Group I is strongly associated with legume rotations (lentil and pea). Because of long survival of *Pythium* oospores in dry soils during the summer, traditional summer fallow may not be effective, although many species decline to low numbers in a fallow system (Schroeder and Paulitz 2006).

Seed treatments reduce early damage to seed and seedlings (Smiley et al., 1996b; Cook et al., 2002b) and can increase grain yield through improved stand establishment (Color Plate 14a), but they will not reduce root rot in mature plants because no commercially registered fungicide is systemically translocated downward into the roots. Effective seed treatments include the oomycete-specific metalaxyl and mefanoxam, and the broad-spectrum thiram. Biological seed treatments with bacteria (*Pseudomonas*, *Bacillus*, *Enterobacteria*) have controlled Pythium root rot in the greenhouse and have resulted in some yield increases in the field (Weller and Cook 1986; Kim et al., 1997; Cook et al., 2002b; Kageyama and Nelson 2003).

Some management practices can mitigate the effects of Pythium root rot, such as banding a starter fertilizer directly below the seed to maintain seedling vigor when a portion of the root system becomes rotted (Cook et al., 2000). Because old seed germinates more slowly, giving a greater window of opportunity for *Pythium* to infect, only new seed should be planted. Volunteer crop plants and weeds should be killed with pre-plant herbicides at least three weeks before planting to minimize the "green-bridge" effect (Smiley et al., 1992; Pittaway 1995). Plants that are dying from treatment with the herbicide glyphosate can serve as reservoirs of *Pythium* inoculum, because the necrotrophic pathogen can extensively colonize the root system when the plant defense system is reduced by inhibition of a key enzyme in the shikimate pathway by this herbicide (Lévesque and Rahe 1992).

RHIZOCTONIA ROOT ROT AND BARE PATCH

Rhizoctonia root rot and bare patch of wheat occur throughout the world (MacNish and Neate 1996; Mazzola et al., 1996a). The disease is caused by a complex of *Rhizoctonia* species that infects roots and seeds of wheat, barley, and other cereals, resulting in above-ground stunting, pruning of root tips, reduced tillering, and reduced yield. Some groups of *Rhizoctonia*, including *R. solani* AG-8 and *R. oryzae*, have a wide host range and will also infect broadleaf rotation crops (Cook et al., 2002a; Paulitz 2002; Paulitz et al., 2002a).

Symptoms and epidemiology

The main above-ground symptom of *Rhizoctonia* root rot is plant stunting. Plants can be stunted in patches or individually. When in patches, called bare patch, the classic symptom is expressed as severe stunting of nearly all plants in roughly circular patches that can be small or up to many meters in diameter. Patches are first noticeable about one month after planting spring wheat and as late as early spring for winter wheat (Color Plate 14b). Bare patch is associated with *R. solani* anastomosis group 8 (AG-8) and occurs most commonly in areas with low rainfall and lighter-textured soils such as sandy loam soils with low organic matter. Roots of young seedlings are pruned off, resulting in plants deficient in nutrients such as phosphorus, which can cause purpling of the leaves in phosphorus-deficient soils -- hence the name "purple patch" in some regions. *Rhizoctonia* can also cause generalized stunting and uneven plant heights without bare patches, especially in areas of higher precipitation with continuous annual cropping.

Rhizoctonia solani AG-8 causes a characteristic "spear tipping", where the root tips appear reddish-brown and are tapered to a fine point (Color Plate 12c). Brownish lesions 1 to 3 mm long are present on the root. The pathogen can rot the root cortex, leaving a temporarily intact stele, giving the roots a constricted or pinched appearance at the site of infection (Paulitz et al., 2002a). Seminal and crown root growth is inhibited because of death of root tips. Maturity is delayed and tiller formation is reduced (Smiley and Wilkins 1993). *Rhizoctonia oryzae* can cause pre-emergence and post-emergence damping-off, but this is not common with *R. solani* AG-8.

Rhizoctonia primarily survives from year to year in intact roots and crop debris. *Rhizoctonia oryzae* can also survive as microsclerotia. Hyphae of *Rhizoctonia* species are capable of spreading long distances in soil from a food base (Garrett 1970; Bailey et al., 2000), moving through soil pores and along the surfaces of soil particles. Most of the inoculum is present in the top 10 to 20 cm of soil (Neate 1987). *Rhizoctonia* is not uniformly or randomly distributed in the field but has an aggregated spatial structure (Paulitz et al., 2003b). Roots are infected by hyphae growing out from crop debris or infected roots (Gill et al., 2002), and infections of seedling roots causes more damage to plants than infections of mature plant roots.

Greenhouse studies have shown that *Rhizoctonia* spreads faster in sandy soil than in finer-textured soil (Gill et al., 2000). *Rhizoctonia* in Australia is associated with light-textured soils, especially the sandy calcareous soils of South Australia, but has also been found in red clays and acid soils (MacNish and Neate 1996). In the Pacific Northwest USA, bare patch is more common in sandy loam soils with low soil organic matter than in silt loam soils.

Increased disease is associated with reduced-tillage or no-till (Weller et al., 1986; Pumphrey et al., 1987; Smiley and Wilkins 1993; Roget et al., 1996), at least in the initial years of no-till following conversion from conventional tillage (Schroeder and Paulitz 2006). The mechanism for this increase is unknown. Tillage may disrupt hyphal networks (Gill et al., 2001a) or promote flushes of microbial activity that inhibit *Rhizoctonia*.

In moist soil, root rot caused by *R. solani* AG-8 is most severe at 12°C to 15°C (Smiley and Uddin 1993; Gill et al., 2001c). However, in dry soil the pathogen is equally and highly virulent at temperatures from 10°C to 25°C (Gill et al.,

2001c). Pathogen virulence in warm, moist soils is somewhat suppressed by a high level of activity by associated soil microorganisms (Gill et al., 2001b). *Rhizoctonia oryzae* has a higher temperature optimum for disease (Ogoshi et al., 1990; Smiley and Uddin 1993), causing maximum disease at 20°C to 27°C.

Reduction in root mass can result in nutrient deficiency. Increasing nutrient availability with starter fertilizer banded below or beside the seed enables plants to tolerate the disease even where seedling infection rates are amplified by the starter fertilizer (Smiley et al., 1990; Cook et al., 2000). Application of nitrogen has shown variable effects on disease, and application of zinc reduced bare patch in zinc-deficient soils in Australia (MacNish and Neate 1996) but not in zinc-sufficient soils in the USA (Cook et al., 2002a).

Infected roots of grassy weeds and crop volunteers enable *Rhizoctonia* to survive and expand inoculum density between crops. When these plants are killed with pre-plant or in-crop herbicides, this nectrophic pathogen can extensively colonize the dying root system, serving as a bridging reservoir of amplified *Rhizoctonia* inoculum. If a pre-plant herbicide is applied to infected plants soon before planting, the newly planted wheat seed germinates and seedlings emerge at a time when the *Rhizoctonia* inoculum level is at a maximum level (Smiley et al., 1992). This phenomenon is known as the *green bridge* and is especially evident following application of glyphosate, which curtails plant defenses because of inhibition of a key enzyme in the shikimate pathway (Lévesque and Rahe 1992). Residual levels of certain other herbicides in soil from a previous crop may also increase Rhizoctonia disease (Smiley and Wilkins 1992).

Causal organisms

A complex of species causes root rot on wheat, including *Rhizoctonia solani* Kühn (teleomorph = *Thanatephorus cucumeris* Donk), *R. oryzae* Ryker and Gooch (teleomorph = *Waitea circinata* Warcup and Talbot), and binucleate species with a sexual stage in the genus *Ceratobasidium*. *Rhizoctonia solani* is divided into a number of subgroups called anastomosis groups (AGs), based on fusion of hyphae between isolates (Sneh et al., 1991); AG-8 is associated with stunting and bare patch symptoms on wheat and barley. Other weakly virulent AGs also have been isolated from wheat, including AGs 2, 2-1, 4, 5, 9, and 10 (Ogoshi et al., 1990; T.C. Paulitz, unpublished data).

Rhizoctonia oryzae also is an important cause of root rot in the Pacific Northwest USA (Ogoshi et al., 1990; Smiley and Uddin 1993; Mazzola et al., 1996b; Paulitz et al., 2002a,b). The sexual state, *W. circinata*, consists of subgroups that attack rice (*Oryza sativa* L.; *W. circinata* var. *oryza*e) and turfgrasses (*W. circinata* var. *circinata* and *W. circinata* var. *agrostis*). The most pathogenic isolates on wheat in the Pacific Northwest appear to be var. *circinata*, based on DNA sequencing (P.A. Okubara, pers. comm.).

Binucleate *Rhizoctonia* AGs CI, E, H, K, and D have also been isolated from wheat, although their pathogenicity on wheat roots is not well known (Mazzola et al., 1996a; Tunalı et al., 2008). Most of these binucleate isolates appear to be weakly virulent on cereals and more virulent on broadleaf rotation crops (Paulitz, unpublished data). AG-D is also known as *Rhizoctonia cerealis* Van der Hoeven, which causes sharp eyespot, a basal culm disease similar in appearance to eyespot caused by *Oculimacula* species and discussed later in this chapter.

Rhizoctonia species can be difficult to identify because they do not produce spores. *Rhizoctonia solani* forms rather thick hyphae (4-15 µm diam.) with characteristic right-angle branching, dolipore septa near each hyphal branch, and a slight constriction at the branching point (Sneh et al., 1991). Some species form microsclerotia in culture, which are irregularly shaped dark-brown aggregations of thick-walled, monilioid cells. However, these are not common with *R. solani* AG-8. *Rhizoctonia solani* and *R. oryzae* are multinucleate. *Rhizoctonia oryzae* branches at 30°C to 50ºC from the main hyphae and forms abundant irregularly shaped orange-pink or salmon to brown-colored sclerotia in culture, 1 to 3 mm in diameter. Both species can

survive in root pieces as rounded, thick-walled monilioid cells.

Rhizoctonia is difficult to quantify because of low population densities in soil. Rhizoctonia propagules can be extracted by sieving of organic matter, plating of soil pellets, and elutriation or baiting with various plant materials (Singleton et al., 1992). A semi-quantitative method using wood toothpicks as bait has been particularly useful (Paulitz and Schroeder 2005) and quantitative DNA-based methods using real-time polymerase chain reaction (PCR) have been developed to identify and quantify several *Rhizoctonia* species from soil and plants (Okubara et al., 2008).

Disease management

The most effective cultural management practice involves controlling the green-bridge by killing volunteer crop plants and weeds with a pre-plant herbicide at least three weeks before planting (Smiley et al., 1992). Keeping the field fallow without a living host also reduces the severity of disease, but only if the fallow period is long enough for inoculum levels of the fungus to be reduced (Roget et al., 1987). Paulitz (unpublished data) showed that chemical fallow over two consecutive years was not enough to reduce inoculum of *R. solani* AG 2-1 or *R. oryzae* in a higher precipitation area of the Pacific Northwest, but could reduce hyphal activity in low-rainfall areas by the end of the fallow season, and that this effect could be carried over to the following crop. Reduced mechanical fallow was more effective than chemical fallow.

Increased disturbance in the seed row can also reduce disease (Roget et al., 1996). Farmers in the USA greatly reduced the impact of Rhizoctonia bare patch by using very heavy direct-seed drills that caused extensive and deep disruption of soil in the seed row. However, such drills are now seldom used because they required large tractors and high amounts of energy. A lighter, paired-row direct-seed drill configuration also reduced Rhizoctonia disease, possibly because of a more open canopy and quicker soil warming between pairs of rows and residue removal between paired rows (Cook et al., 2000).

Burning or otherwise removing stubble from no-till fields does not generally reduce disease severity (Smiley et al., 1996a; T.C. Paulitz, unpublished data) or the amount of *Rhizoctonia* inoculum (T.C. Paulitz, unpublished data), possibly because the pathogen mainly survives in the root system. However, stubble or crop residue does affect the soil temperature and moisture during the spring, often resulting in cooler soils which are more conducive for *Rhizoctonia* damage to young seedlings.

Protective seed treatments with chemicals such as tebuconazole, difenoconazole, thiram, and fludioxonil often result in better seedling health, expressed as more tillers, roots, and greater plant height, but in most cases grain yield is not statistically increased (Mazzola et al., 1996a; Paulitz and Scott 2006). Effects of crop rotation have been variable because of the wide host range of *Rhizoctonia* on other rotation crops (MacNish and Neate 1996; Mazzola et al., 1996a; Cook et al., 2002a). No genetic resistance to Rhizoctonia root rot has been detected in adapted cultivars of wheat but resistance appears to reside in wild relatives of wheat that have not yet been exploited (Smith et al., 2003a,b).

TAKE-ALL

Take-all is the most damaging root disease of wheat worldwide and can cause severe grain yield losses when consecutive cereal crops are grown (Asher and Shipton 1981; Hornby et al., 1998). Take-all is the most important limiting factor for winter wheat production in Western Europe. The pathogen causes root necrosis of wheat and, to a lesser extent, of barley, rye, and some grasses.

Symptoms and Epidemiology

The take-all fungus survives during the intercrop period on root and shoot debris of a previously infected crop, or on grasses and volunteer cereals. It infects seminal roots, causing characteristic

black necrosis, sometimes after root surface colonization by brown runner hyphae of the fungus. Secondary infections occur mainly from root-to-root contact, extending disease to the neighboring plants, but spreading only a short distance resulting in a patchy distribution of the disease within a crop (Cook 2003; Gosme et al., 2007). The root system can become entirely affected. In moist conditions a black necrosis can develop on the lower stem (Color Plate 12d), but this symptom seldom occurs in low-rainfall regions where wheat matures with little or no summer rainfall or irrigation. Perithecia (sexual stage) may form on the lower stem and discharge ascospores, but the importance of ascospores in the existing crop is likely limited because perithecia are formed late in the growing season. Due to restricted capture of nitrogen and water by roots (Schoeny et al., 2003), infected plants develop poorly and appear as stunted patches in spring. Premature ripening in summer is expressed as whiteheads (Color Plates 13a, 14c) bearing shrivelled grains.

Severe disease that develops early in the season causes dramatic yield losses by reducing all yield components (Schoeny et al., 2001). Even when disease severity is low or moderate, major yield losses are to be expected when a dry period occurs during grain formation and filling. Take-all is severe when the autumn and winter are mild and humid, allowing early infections to greatly increase the level of primary inoculum (Hornby 1978). As the take-all fungus is not a good saprophyte, high levels of inoculum are the result of host plants being infected. Wheat monocultures increase inoculum levels through several seasons, increasing disease severity to a maximum generally between the second and fourth year. In regions where soil organic matter is plentiful and the soil environment is favorable for microbial growth, continued production of wheat then leads to a decline of disease severity until a balance is reached at a severity less than that during years of maximum disease expression. Several biological hypotheses were proposed to explain this phenomenon, named take-all decline (Hornby 1979). Current agreement focuses on the important role of fluorescent pseudomonads as biological antagonists of the take-all pathogen (Weller et al., 2007).

Mathematical models (Brasset and Gilligan 1989) incorporate components for primary and secondary infection, together with reduction of inoculum potential over time. A routine DNA-based assay recently refined as a real-time PCR test is provided as a service to Australian farmers to quantify take-all pathogens in soil samples and to serve as a basis for predicting potential yield loss (Herdina and Roget 2000).

Causal organism

Gaeumannomyces graminis (Sacc.) Arx & Olivier var. *tritici* Walker (Ggt) is responsible for take-all of wheat, triticale (*Triticosecale rimpaui* Wittm.), barley, and rye, in decreasing order of susceptibility. These crops can also be attacked by *G. graminis* var. *avenae* (*Gga*), which causes take-all of oat and take-all patch of turfgrass (Smiley et al., 2005a). Varieties *Ggt* and *Gga* can be differentiated by host range and pathogenicity tests or by measuring the mean length of ascospores: 70 to 105 μm for *Ggt* and 100 to 130 μm for *Gga* (Freeman and Ward 2004). Two other *G. graminis* varieties are known to colonize roots of wheat but are considered as weakly or non-pathogenic, *G. graminis* var. *graminis* causing dieback of Bermudagrass [*Cynodon dactylon* (L.) Pers.], and *G. graminis* var. *maydis* causing take-all on maize (Freeman and Ward 2004).

Gaeumannomyces species such as *Ggt* and *Gga* are homothallic, meaning individual isolates are able to produce perithecia. When *Gaeumannomyces*-like fungi are isolated from plants with take-all symptoms but fail to produce perithecia in culture, the isolates should be examined for the possibility that they are an anamorphic state (*Phialophora* or *Harpophora* spp.; Gams 2000) of recently described heterothallic species requiring the presence of both mating types to produce the sexual stage. These fungi seldom produce a sexual stage in nature and require mixtures of mating types to produce the sexual stage in culture. They are easily misidentified as *Ggt* or *Gga*, may be under-

reported, and are likely to occur on cereals in nature. *Gaeumannomyces incrustans* Landschoot & Jackson and *Magnaporthe poae* Landschoot & Jackson were the first-described heterothallic species of *Gaeumannomyces*-like fungi (Clarke and Gould 1993). Anamorphs of *M. poae*, misreported initially as *P. graminicola* and later shown to be individual mating types of *M. poae* (Clarke and Gould 1993), cause summer patch of perennial grasses (Smiley et al., 2005a). Anamorphic states of *G. incrustans*, *M. poae*, and related fungi cause a high-temperature form of take-all on wheat and other cereals in pot tests (Smiley et al., 1986; Elliot 1991).

DNA probes have also been developed to identify isolates of *G. graminis* varieties and related species (Clarke and Gould 1993; Augustin et al., 1999; Herdina and Roget 2000; Rachdawong et al., 2002; Freeman and Ward 2004). Molecular tools have revealed genetic polymorphism within *Ggt* populations (Ward and Gray 1992; Bateman et al., 1997). Characterizations of *Ggt* populations from monoculture wheat crops, using both restricted fragment length polymorphism (RFLP) and random amplified polymorphic DNA (RAPD) markers, have revealed two genetic groups called G_1 and G_2 (Lebreton et al., 2004). Isolates of G_1 were dominant in the first and sixth wheat crops, and G_2 isolates were dominant in the third and fourth wheat crops. Aggressiveness of group G_2 was significantly greater than that of group G_1, which corresponds with observed peaks of disease during a wheat monoculture. A linear relationship between G_1 and G_2 frequencies and disease severity on wheat roots occurred in fields monitored over three consecutive seasons (Lebreton et al., 2007).

Disease management

The most important cultural practice used to control take-all is crop rotation (Asher and Shipton 1981; Hornby et al., 1998; Ennaïfar et al., 2007). Other practices that influence take-all severity following the build-up of inoculum during preceding host crops include tillage (Ennaïfar et al., 2005), sowing date and density, application of fertilizer or lime, and grass weed control. Fungicide seed treatment provides consistent but partial efficacy by reducing primary infections (Schoeny and Lucas 1999; Bailey et al., 2005; Ennaïfar et al., 2005). No resistant cultivar is currently available and sources of resistance are scarce (Cook 2003).

Effects of soil cultivation have been variable. Direct-seeded crops have had a lower disease incidence in Britain (Brooks and Dawson 1968) and either a higher level of disease (Moore and Cook, 1984) or no effect (Schroeder and Paulitz 2006) in the Pacific Northwest USA.

Late sowing will allow a longer period of inoculum reduction and less favorable temperature conditions at the time of possible infections, thus reducing the frequency of primary infections. Reducing the sowing density reduces the amount of primary infection as well as secondary infections capable of transmitting the disease from plant to plant (Colbach et al., 1997).

Take-all generally becomes more severe immediately following application of lime to acid soils, particularly when applications causing a large change in acidity occur during intervals in which environmental conditions and host frequency are also conducive to disease expression (Asher and Shipton 1981). However, host genotypes, isolates of Ggt, and other soil microbes each vary in tolerance to acidity. In some regions the occurrence of severe take-all has been associated more with acid than neutral soils, resulting in less severe take-all following application of lime (Hornby et al., 1998).

Application of the ammonium-ion form of nitrogen fertilizer generally leads to a reduction of take-all severity compared to the nitrate form or a mixture of these ions (Huber et al., 1968; Lucas et al., 1997). When absorbed by roots, the ammonium ion reduces rhizosphere pH and stimulates antagonistic components of the root-surface microflora, such as fluorescent pseudomonads (Smiley 1978; Sarniguet et al., 1992a,b).

Efficient control of grass weeds and volunteer cereals is important for eliminating additional inoculum build-up (Ennaïfar et al.,

2005; Gutteridge et al., 2005) that may occur without contributing to take-all decline, as reported for blackgrass (*Alopecurus myosuroides* Hudsen), barren brome [*Anisantha sterilis* (L.) Nevski] and rye brome (*Bromus secalinus* L.) (Dulout et al., 1997; Gutteridge et al., 2005).

Attempts have been made to develop a biological method to control the disease based on various hypotheses proposed to explain the phenomenon of take-all decline (Lucas and Sarniguet 1998). The most intensive work on this topic has involved fluorescent pseudomonads that produce antibiotic compounds (Weller et al., 2007). Due to often inconsistent performance when these biocontrol agents are applied to soil in an inundative biological control, Cook (2007) stressed the importance of managing the resident rhizobacteria with the cropping system to achieve a conservation biological control.

CEPHALOSPORIUM STRIPE

Cephalosporium stripe is a vascular wilt caused by a pathogen with a host range within the Poaceae (Farr et al., 2007). Spring cereals are susceptible but most economic damage occurs on winter cereals (wheat, barley, oat, rye, and triticale) in cool, temperate regions of North America, Europe, Africa, and Japan.

Symptoms and epidemiology

One or more distinct longitudinal chlorotic stripes appear in leaves during jointing (Color Plate 15a). A dark brown leaf vein (Color Plate 15b) extends from the base of each leaf stripe, through the leaf sheath, and into the culm. Leaf striping often does not occur on all leaves and tillers of affected plants. Affected leaves senesce prematurely, plants are typically stunted, and heads may ripen prematurely to produce whiteheads (Color Plate 13a). As plants mature the culm of infected tillers may darken at and below nodes. When seedlings are heavily infected they may exhibit a mosaic-like yellowing in late winter or early spring and may die before stripes develop.

The pathogen is disseminated in infested seed (Murray 2006), but most disease is caused by infection of roots by soilborne conidia (Mathre and Johnston 1975). The pathogen directly penetrates adventitious (coronal) roots and lower stems and moves into xylem vessels (Stiles and Murray 1996; Douhan and Murray 2001). Conidia produced in the xylem and those entering directly through wounds are carried upward in the transpiration stream and lodge and multiply at stem nodes and in leaf veins. Occlusion of xylem vessels by conidia impedes the transport of water and nutrients (Wiese 1972).

The pathogen produces a chlorosis-inducing toxin Graminin A and an exogenous polysaccharide that are not required for pathogenicity and virulence (Van Welt and Fullbright 1986). They are important to development of disease symptoms (Kobayasi and Ui 1979; Creatura et al., 1981; Rahman et al., 2001) and survival of the pathogen in dead straw (Bruehl 1975; Wiese and Ravenscroft 1978).

Foliar tissue infested during the parasitic phase on living plants is returned to the soil during harvest and tillage. The pathogen does not persist in root tissue. In regions where summer rainfall is common the fungus can survive for up to three years in infested residue but is mostly destroyed within one year if the residue is buried by tillage. In regions where most precipitation occurs during the winter period the rate of straw decomposition is slower and survival of the pathogen longer. Acid soils favor saprophytic survival in straw and production and survival of conidia (Murray and Walter 1991).

Reduction in grain yield is correlated with numbers of spores in soil (Martin et al., 1986; Specht and Murray 1990; Bockus et al., 1994). Cool, wet weather during autumn and winter favors profuse sporulation on infested plant debris at or near the soil surface (Wiese and Ravenscroft 1975, 1978; Mathre and Johnston 1977). Disease incidence is often more prevalent when seed is planted into wet soil (Bruehl 1968; Pool and Sharp 1969; Anderegg and Murray 1988). Numbers of propagules in soil decline rapidly during spring.

Pathogen entry into the plant does not require pre-existing tissue damage (Anderegg and Murray 1988) but is strongly amplified by injuries caused by emerging secondary roots and tillers (Douhan and Murray 2001), freezing of roots (Bailey et al., 1982; Martin et al., 1989), root breakage during freeze and thaw cycles (Bruehl 1968), insects (Slope and Bardner 1965), and acid soils (Bockus and Claassen 1985; Anderegg and Murray 1988; Stiles and Murray 1996).

Causal organism

Cephalosporium gramineum Y. Nisik. & Ikata produces small unicellular conidia in a slimy exopolysaccharide matrix (Stasinopoulos and Seviour 1989). The *Cephalosporium* stage occurs in the xylem of living plants and on the surface of dead straw at or near the soil surface. In most but not all regions the fungus also produces a saprophytic sporodochial stage (*Hymenula cerealis* Ellis & Everh.) mostly near nodes of dead straw that was infested while living (Wiese and Ravenscroft 1978). Sporodochia are formed during cool, wet periods from late autumn to early spring. Hyaline conidia are produced in great abundance on moistened sporodochia.

Disease management

Cephalosporium stripe incidence and severity increase with frequency of winter wheat production (Latin et al., 1982; Bockus et al., 1983). Disease is greatest where wheat is grown annually and is much less damaging in two-year rotations where summer rainfall is common and in three-year rotations where precipitation occurs mostly during the winter. Rotations are most effective when volunteer cereals and grass weeds are controlled during the over-winter fallow period to prevent an increase in pathogen inoculum density. Cephalosporium stripe is especially damaging in direct-drill (no-till) planting systems (Latin et al., 1982; Bockus et al., 1983). The amount of pathogen inoculum can be greatly reduced by burning, removing, or deeply burying infested residue (Pool and Sharp 1969; Wiese and Ravenscroft 1975; Bockus et al., 1983; Christian and Miller 1984). Planting as late as possible during the autumn reduces disease incidence and severity (Bruehl 1968; Pool and Sharp 1969; Martin et al., 1989) by limiting the colonization of root and crown tissue by the pathogen (Pool and Sharp 1969; Douhan and Murray 2001), provided planting is not delayed such that yield potential is reduced more than may be caused by Cephalosporium stripe in earlier plantings (Raymond and Bockus 1984). Disease severity on winter wheat grown on acid soils can be reduced by applying lime (Bockus and Claassen 1985; Anderegg and Murray 1988), but the benefit is mostly limited to years when root wounding from frozen soil is minor (Murray et al., 1992).

Winter wheat cultivars with partial resistance are available (Bockus 1995; Murray et al., 2001; Mundt 2002). Susceptible cultivars are consistently susceptible and resistant cultivars vary widely in disease reaction from year to year (Martin et al., 1989). Repeated plantings of moderately resistant cultivars reduce the level of pathogen inoculum in soil and adequately manage the loss of yield over time (Shefelbine and Bockus 1989; Murray et al., 2001). Two mechanisms of resistance have been described. The pathogen may be excluded from entering the plant, resulting in lower levels of disease incidence, or the pathogen may have restricted ability to move through root and crown tissue, resulting in fewer infected tillers and delayed symptom development (Morton and Mathre 1980; Mathre and Johnston 1990; Douhan and Murray 2001). Genes conveying a high level of resistance to *C. gramineum* were derived from a wheat-*Thinopyrum* amphiploid (Mathre et al., 1985), were characterized (Cai et al., 1996, 1998), and are being introgressed into commercial cultivars.

Fungicide and microbial products do not effectively suppress Cephalosporium stripe. Spring cereals are susceptible but mostly escape infection.

EYESPOT

Eyespot is caused by a fungus which produces lesions on the lower culms, just above the soil surface. The disease is also called strawbreaker foot rot because, when severe, it causes the stem to break and the plant to lodge. Eyespot occurs commonly on fall-planted wheat but can also be observed on barley and oat, and occasionally on wheat planted during early spring. Although reported in many countries, eyespot is most important in regions with temperate climates such as in the Pacific Northwest USA and Western Europe (Nelson and Sutton 1988).

Symptoms and Epidemiology

Initial symptoms appear on seedlings during autumn or early spring as dark lens-shaped lesions with diffuse margin and occasionally a central black 'pupil' on the outer leaf sheaths, just above the soil. The infection progresses inward from sheath to sheath and into the stem (Color Plate 13c, right side). Invasion of the stem reduces translocation of water and nutrients, reducing yield mainly through a reduction of kernels per head and kernel weight (Ponchet 1959). Severe eyespot lesions (Color Plate 13d) weaken the stems to the extent that they collapse (Ray et al., 2006), causing plants to lodge (Color Plate 14d).

The main source of inoculum is mycelium of the fungus surviving on infested crop debris remaining from a previous crop. Infection occurs via spores formed on the debris, which are mostly spread over short distances in rain-splash droplets. The optimum temperature for sporulation is 5°C (Fitt et al., 1988). Frequent rains are necessary to assure inoculum dispersal, and high humidity is required for sporulation and infection (Rowe and Powelson 1973).

Growth of the fungus inward through successive leaf sheaths on an infected tiller is a function of accumulated temperature and differences in susceptibility between cultivars (Ponchet 1959; Rapilly et al., 1979). The earliest infections lead to earlier and more severe penetrations of the stems and the greatest reduction in yield. Forecasting models have been developed (Rapilly et al., 1979; Siebrasse and Fehrmann 1987), but most of them do not take into account the effects of cultivar resistance, diversity within the fungus populations, and crop management practices (Colbach and Saur 1998). The models are mainly used at a regional level as an alert system to enable advisors and farmers to optimize fungicide applications. Typical decisions at the field level call for a justification to apply a fungicide if the disease exceeds a threshold of at least two outer leaf sheaths penetrated on more than 20% of the tillers at Zadoks growth stage 30 (Fitt et al., 1988). A model defining crop management systems that reduce the risk of eyespot is also available (Colbach et al., 1999).

Causal organisms

Eyespot is caused by *Oculimacula yallundae* (Wallwork & Spooner) Crous & W. Gams, and *O. acuformis* (Boerema, R. Pieters & Hamers) Crous & W. Gams (Crous et al., 2003). These fungi were previously classified as *Tapesia yallundae* and *T. acuformis*, respectively (Robbetse et al., 1995), and are the teleomorphic stages of *Helgardia herpotrichoides* (Fron) Crous & W. Gams and *H. acuformis* (Niremberg) Crous & W. gams (Crous et al., 2003). Before being considered as two species these fungi were described as *Pseudocercosporella herpotrichoides* var. *herpotrichoides* and *P. herpotrichoides* var. *acuformis* (Niremberg 1981). These two varieties were initially thought to correlate with two pathotypes known as the wheat-type (W-type) and rye-type (R-type), respectively, in reference to their pathogenicity on wheat only or on wheat and rye (Priestley et al., 1992). They were also distinguished as the N- and L-type in reference to normal or slow mycelium growth (Cavelier et al., 1987), but further examination of more strains found this distinction to be incomplete (Lucas et al., 2000).

Globose, greyish brown apothecia bearing ascospores of *O. yallundae* develop on decaying stems and leaf sheaths late in the season and are mainly found on stubble during the intercrop

period (Wallwork and Spooner 1988; Hunter 1989). Although *O. yallundae* has been considered the more important causal agent of eyespot, *O. acuformis* increased significantly to become the dominant species during the 1990s in the Pacific Northwest USA (Douhan et al., 2003) and Western Europe (Lucas et al., 2000).

The forms which are more commonly observed in a growing wheat crop are the anamorphs *H. herpotrichoides* and *H. acuformis*. They can be differentiated through examination of conidial and cultural characteristics: *H. herpotrichoides* produces either curved or curved and straight conidia (4 septate, 35-80 μm x 1.5-2.5 μm) on fast growing, even-edged colonies and squirrel-grey or olive-grey mycelia, and *H. acuformis* with only straight conidia (4-6 septa, 43-120 μm x 1.2-2.3 μm) on slow-growing, feathery or uneven-edged colonies and grey to brown-grey mycelia (Nirenberg, 1981). The two species can be identified more rapidly and accurately by molecular markers used in a PCR assay combined with restriction enzyme digestion of an amplified ribosomal DNA fragment (Gac et al., 1996a,b). A real-time PCR assay now allows workers to simultaneously identify and quantify *O. yallundae* and *O. acuformis* (Walsh et al., 2005).

Disease management

Crop rotation remains the best preventive method for managing this disease. Intensification of agriculture in Western Europe led to an increase in grain yield losses due to eyespot and this disease became, from the 1970s, the main target of fungicides applied between the tillering and stem-extension stages of wheat growth. The most active fungicides have been the antimicrotubular benzimidazole group, especially carbendazim. In the early 1980s, because of the selection of resistant strains, the benzimidazoles were replaced by C-14 demethylation inhibitors (DMIs) such as prochloraz (imidazole group) or flusilazole (triazole group). In the early 1990s the efficiency of these DMIs was also compromised by fungicide resistance in some regions (Leroux and Gredt 1997). The most-used current fungicide is cyprodinil, but decreased sensitivity from repeated applications has also been observed with this fungicide (Babij et al., 2000).

Moderate resistance to eyespot, provided by the gene *Pch2*, was first incorporated in the French winter wheat cultivar Cappelle-Desprez (Muranty et al., 2002). This gene remained durable despite widespread exploitation and has been transferred into many cultivars. The gene *Pch1* was transferred (Maia 1967) from *Aegilops ventricosa* Tausch. into *Triticum persicum* Vavilum ex Zhuk., and the F_1 hybrid was backcrossed with the bread wheat cultivar Marne, producing the resistant line VPM_1; initials refer to ventricosa, persicum, and Marne. The resistance to eyespot conferred by *Pch1* is higher than that conferred by *Pch2* (Hollins et al., 1988; Jahier et al., 1989), which only acts at the seedling stage (Muranty et al., 2002), and now is being emphasized in wheat breeding programs in Europe and the USA (Allan et al., 1993). These eyespot resistance genes remain durable and there is no evidence of differences with respect to *Oculimacula* species.

FUTURE PERSPECTIVES

Knowledge of the etiology and control of root, crown, and culm rots continues to improve with advances in technology. The following examples illustrate promising areas of emerging research.

Development of molecular procedures is greatly expanding the precision of pathogen identification and, consequently, also supporting a constant evolution in pathogen taxonomy and phylogeny. Examples include identification of DNA sequences of the ITS region of *Pythium* (Martin 2000; Lévesque and de Cock 2004; Schroeder et al., 2006), *Rhizoctonia* (González et al., 2001), *Gaeumannomyces* (Freeman and Ward 2004), and *Fusarium* (O'Donnell et al., 2004). Results of these tests suggest that many *Pythium* species reported in the literature may be conspecific with others, and other described species may actually be complexes of cryptic species. Many sequences of *Pythium* species on GenBank may also be misidentified. Likewise,

newly recognized polymorphism within *G. graminis* var. *tritici* has been revealed (Lebreton et al., 2004), and it appears that anastomosis groups of *R. solani* may function as phylogenetic and biological species which have evolved separately on different host plants and no longer exchange genetic material (González et al., 2006). PCR techniques were used to show that *Cephalosporium gramineum* can become seedborne in wheat (Vasquez-Siller and Murray 2003), and real-time PCR procedures were developed to identify and quantify several of these pathogens in DNA extracts from soil and plants (Lees et al., 2002; Schroeder et al., 2006; Okubara et al., 2008).

Real-time PCR is also now used to make routine farm management decisions. A commercial soil diagnostic laboratory in South Australia uses a DNA extract from soil to identify and estimate population levels of several nematode species and inoculum levels of fungal pathogens including *F. culmorum*, *F. pseudograminearum*, *G. graminis* var. *tritici*, *G. graminis* var. *avenae*, and *R. solani* AG-8 (Ophel-Keller et al., 2008). Predictions of potential disease risk are communicated back to farmers through a network of agronomic advisors (Herdina and Roget 2000). This application of modern technology will undoubtedly facilitate more rapid and effective surveys of species distribution and inoculum density.

The growing body of literature on molecular aspects of pathogenicity will continue to refine understanding of pathogenic variation within pathogen populations, as well as to improve development of genetic resistance. Considerable new molecular information is available regarding the pathogenicity and genetic structure of *B. sorokiniana* (Kumar et al., 2002), *Fusarium* species causing crown rot (O'Donnell et al., 2004; Monds et al., 2005; Chakraborty et al., 2006), and *G. graminis* var. *tritici* (Lebreton et al., 2004). All *Fusarium* populations studied thus far appear to be recombining, suggesting an unrecognized role for ascospores in epidemiology. Current studies of the roles of avenacinase, melanin, and laccases in take-all, and the link between *G. graminis* var. *tritici* and *Magnaporthe grisea* (Cook 2003; Freeman and Ward 2004) are likely to provide new options for achieving genetic resistance. Likewise, despite the lack of specific genetic resistance to *Pythium* in wheat, a better understanding is emerging for innate resistance and the role of signaling pathways, especially the jasmonic acid and ethylene pathways (Vijayan et al., 1998; Okubara and Paulitz 2005). Elicitors produced by *Pythium* have also been identified, which may be perceived by the host plant (Veit et al., 2001). Higher levels of eyespot resistance may result from studies of the determinants of pathogenicity by *Oculimacula yallundae* and *O. acuformis*, including analysis of the infection process and importance of tissue susceptibility.

Identification and deployment of genetic resistance has been especially difficult for species of *Pythium*, *Rhizoctonia*, *Fusarium*, and *Gaeumannomyces*. Strong advances are currently being made in mapping QTLs for partial seedling resistance to Fusarium crown rot (Boville et al., 2006; Collard et al., 2006). This work is also being extended to include adult-plant resistance. However, these pursuits need to be balanced by a deeper understanding of the components of resistance, including resistance to penetration, resistance to stem colonization, plant reaction to infection, and sensitivity to toxins to enable a more definitive identification of the QTL.

Identification of loci for resistance to *Fusarium* and other pathogens are complemented by improvements in the precision and speed of assays to detect disease resistance or to link phenotypic reactions to sources of genetic resistance (Cowger and Mundt 1998; Wildermuth et al., 2001; Mitter et al., 2006). Continued development of markers for detecting the presence of resistance genes in seedlings will further improve the efficiency of wheat breeding programs. Plant breeders in France (INRA) are also attempting to clone the *Pch1* gene for resistance to eyespot.

New insights into disease epidemiology are emerging through development of models to predict incidence and severity of take-all (Ennaïfar et al., 2007) and eyespot (Colbach et al., 1999). These models provide a tool to more

effectively identify and combine the most efficient methods that individually provide only partial disease control (Ennaïfar et al., 2005). They also provide a tool to more effectively respond to society's demand to reduce the use of pesticides. One example is the current multi-disciplinary emphasis on developing crop rotation and management systems to control eyespot, based on the use of multi-resistant, hardy winter wheat cultivars in France (Savary et al., 2006).

Extensive research continues to be focused on the pursuit of biological control and enhanced soil suppressiveness for diseases such as take-all, common root rot, Pythium root rot, and Rhizoctonia root rot. The greatest focus has been applied to take-all (Hornby et al., 1998; Weller et al., 2007). Additionally, *B. sorokiniana* is a poor saprophytic competitor, sensitive to suppression in soils (Bailey and Lazarovits 2003), and sensitive to several potential biocontrol agents (Kumar et al., 2002). Likewise, Rhizoctonia root rot often becomes severe during the initial transition from conventional tillage to no-till (Schroeder and Paulitz 2006), but long-term no-till farms and annual wheat experiments in the USA show little *Rhizoctonia* disease (Smiley et al., 1996a; T.C. Paulitz, unpublished data). Natural suppression to *R. solani* in cereal crops has been documented (Lucas et al., 1993; Roget 1995; Mazzola et al., 1996a). Wiseman et al. (1996) demonstrated that suppression was dependent upon a microbial component, and disease incidence and severity have been inversely correlated with microbial biomass (Smiley et al., 1996a). Many other instances of *Rhizoctonia*-suppressive soils have been described (Mazzola et al., 1996a; Sneh et al., 1996) but specific mechanisms for suppression are generally unknown.

Complementation and collaboration among regional or national research programs throughout the world have been highly effective for identifying and deploying germplasm with higher levels of resistance to common root rot, eyespot, and Fusarium crown rot. However, these efforts are not effectively funded and coordinated. Greater institutional collaboration and funding linkages are needed to improve the efficiency of coordination between international organizations such as CIMMYT and ICARDA, and public and commercial programs in countries where wheat is an important crop.

REFERENCES

Akinsanmi, O.A., V. Mitter, S. Simpfendorfer, D. Backhouse, and S. Chakraborty. 2004. Identity and pathogenicity of *Fusarium* spp. isolated from wheat fields in Queensland and northern New South Wales. Aust. J. Agric. Res. 55:97-107.

Allan, R.E., G.L. Rubenthaler, C.F. Morris, and R.F. Line. 1993. Registration of three soft white winter wheat germplasm lines resistant or tolerant to strawbreaker footrot. Crop Sci. 33:1111-1112.

Anderegg, J.C., and T.D. Murray. 1988. Influence of soil matric potential and soil pH on Cephalosporium stripe of winter wheat in the greenhouse. Plant Dis. 72:1011-1016.

Aoki, T., and K. O'Donnell. 1999. Morphological and molecular characterization of *Fusarium pseudograminearum* sp. nov., formerly recognized as the Group 1 population of *F. graminearum*. Mycologia 91:597-609.

Apoga, D., H. Akesson, H.B. Jansson, and G. Odham. 2002. Relationship between production of the phytotoxin prehelminthosporol and virulence in isolates of the plant pathogenic fungus *Bipolaris sorokiniana*. Eur. J. Plant Pathol. 108:519-526.

Arabi, M.I.E., A. Al-Daoude, and M. Jawhar. 2006. Interrelationship between spot blotch and common root rot in barley. Australasian Plant Pathol. 35:477-479.

Asher, M.J.C., and P.J. Shipton (ed.) 1981. Biology and control of take-all. Academic Press, London.

Augustin, C., K. Ulrich, E. Ward, and A. Werner. 1999. RAPD-based inter- and intravarietal classification of fungi of the *Gaeumannomyces-Phialophora* complex. J. Phytopathol. 147:109-117.

Babij, J., Q. Zhu, P. Brain, and D.W. Hollomon. 2000. Resistance risk assessment of cereal eyespot, *Tapesia yallundae* and *Tapesia acuformis*, to the anilinopyrimidine fungicide cyprodinil. Eur. J. Plant Pathol. 106:895-905.

Backhouse, D. 2006. Forecasting the risk of crown rot between successive wheat crops. Aust. J. Exp. Agric. 46:1499-1506.

Backhouse, D., A.A. Abubakar, L.W. Burgess, J.I. Dennis, G.J. Hollaway, G.B. Wildermuth, H. Wallwork, and F.J. Henry. 2004. Survey of *Fusarium* species associated with crown rot of wheat and barley in eastern Australia. Australasian Plant Pathol. 33:255-261.

Backhouse, D., and L.W. Burgess. 2002. Climatic analysis of the distribution of *Fusarium graminearum*,

F. pseudograminearum and *F. culmorum* on cereals in Australia. Australasian Plant Pathol. 31:321-327.

Bailey, K.L., B.D. Gossen, R.K. Gugel, and R.A.A. Morrall (ed.) 2003. Diseases of field crops in Canada. 3rd ed. Univ. Extension Press, Univ. Saskatchewan, Saskatoon, Canada.

Bailey, K.L., and G. Lazarovits. 2003. Suppressing soil-borne diseases with residue management and organic amendments. Soil Tillage Res. 72:169-180.

Bailey, J.E., J.L. Lockwood, and M.V. Wiese. 1982. Infection of wheat by *Cephalosporium gramineum* as influenced by freezing of roots. Phytopathology 72:1324-1328.

Bailey, D.J., W. Otten, and C.A. Gilligan. 2000. Saprotrophic invasion by the soil-borne fungal plant pathogen *Rhizoctonia solani* and percolation thresholds. New Phytol. 146:535-544.

Bailey, D.J., N. Paveley, C. Pillinger, J. Foulkes, J. Spink, and C.A. Gilligan. 2005. Epidemiology and chemical control of take-all on seminal and adventitious roots of wheat. Phytopathology 95:62-68.

Baker, K.F., and W.C. Snyder (ed.) 1965. Ecology of soil-borne plant pathogens: Prelude to biological control. John Murray, London, UK.

Bateman, G.L., G.M. Murray, R.J. Gutteridge, and H. Coskun. 1998. Effects of method of straw disposal and depth cultivation on populations of *Fusarium* spp. in soil and brown foot rot in continuous winter wheat. Ann. Appl. Biol. 132:35-47.

Bateman, G.L., E. Ward, D. Hornby, and R.J. Gutteridge. 1997. Comparisons of isolates of the take-all fungus, *Gaeumannomyces graminis* var. *tritici*, from different cereal sequences using DNA probes and non-molecular methods. Soil Biol. Biochem. 29:1225-1232.

Bentley, A.R., B. Tunalı, J.M. Nicol, L.W. Burgess, and B.A. Summerell. 2006. A survey of *Fusarium* species associated with wheat and grass stem bases in northern Turkey. Sydowia 58:163-177.

Bockus, W.W. 1995. Reaction of selected winter wheat cultivars to Cephalosporium stripe. p. 112. *In* Biological and cultural tests for control of plant diseases. Vol. 10. APS Press, St. Paul, MN.

Bockus, W.W., R.L. Bowden, R.M. Hunger, W.L. Morrill, T.D. Murray, and R.W. Smiley (ed.) 2010. Compendium of wheat diseases and insects. 3rd ed. APS Press, St. Paul, MN.

Bockus, W.W., and M.M. Claassen. 1985. Effect of lime and sulfur application to low-pH soil on incidence of Cephalosporium stripe in winter wheat. Plant Dis. 69:576-578.

Bockus, W.W., M.A. Davis, and T.C. Todd. 1994. Grain yield responses of winter wheat coinoculated with *Cephalosporium gramineum* and *Gaeumannomyces graminis* var. *tritici*. Plant Dis. 78:11-24.

Bockus, W.W., J.P. O'Connor, and P.J. Raymond. 1983. Effect of residue management method on incidence of Cephalosporium stripe under continuous winter wheat production. Plant Dis. 67:1323-1324.

Boville, W.D., W. Ma, K. Ritter, B.C.Y. Collard, M. Davis, G.B. Wildermuth, and M.W. Sutherland. 2006. Identification of novel QTL for resistance to crown rot in the doubled haploid wheat population 'W21MMT70' x 'Mendos'. Plant Breed. 125:538-543.

Brasset, P.R., and C.A. Gilligan. 1989. Fitting of single models for field disease progress data for the take-all fungus. Plant Pathol. 38:397-407.

Brooks, D.H., and M.G. Dawson. 1968. Influence of direct-drilling of winter wheat on incidence of take-all and eyespot. Ann. Appl. Biol. 61:57-64.

Bruehl, G.W. 1968. Ecology of Cephalosporium stripe disease of winter wheat in Washington. Plant Dis. Rep. 52:590-594.

Bruehl, G.W. (ed.) 1975. Biology and control of soil-borne plant pathogens. Am. Phytopathol. Soc., St. Paul, MN.

Bruehl, G.W. 1987. Soilborne plant pathogens. Macmillan Publ. Co., New York, NY.

Burgess, L.W., D. Backhouse, B.A. Summerell, A.B. Pattison, T.A. Klein, R.J. Esdaile, and G. Ticehurst. 1993. Long-term effects of stubble management on the incidence of infection of wheat by *Fusarium graminearum* Schw. Group 1. Aust. J. Exp. Agric. 33:451-456.

Burgess, L.W., D. Backhouse, L.J. Swan, and R.J. Esdaile. 1996. Control of Fusarium crown rot of wheat by late stubble burning and rotation with sorghum. Australasian Plant Pathol. 25:229-233.

Burgess, L.W., A.H. Wearing, and T.A. Toussoun. 1975. Surveys of Fusaria associated with crown rot of wheat in eastern Australia. Aust. J. Agric. Res. 26:791-799.

Butler, F.C. 1961. Root and foot rot diseases of wheat. Science Bull. 7. Dep. Agric., New South Wales, Sydney, Australia.

Cai, X., S.S. Jones, and T.D. Murray. 1996. Characterization of an *Agropyron elongatum* chromosome conferring resistance to Cephalosporium stripe in common wheat. Genome 39:56-62.

Cai, X., S.S. Jones, and T.D. Murray. 1998. Molecular cytogenetic characterization of *Thinopyrum* and wheat-*Thinopyrum* translocated chromosomes in a wheat *Thinopyrum* amphiploid. Chromosome Res. 6:183-189.

Cavelier, N., D. Rousseau, and D. Lepage. 1987. Variabilité de *Pseudocercosporella herpotrichoides*, agent du piétin-verse des céréales : Comportement in vivo de deux types d'isolats et d'une population en mélange. Z. Pflanzenkrankh. Pflanzenschutz. 94:590-599.

Chakraborty, S., L. Liu, V. Mitter, J.B. Scott, O.A. Akinsanmi, S. Ali, R. Dill-Macky, J. Nicol, D. Backhouse, and S. Simpfendorfer. 2006. Pathogen population structure and epidemiology are keys to wheat crown rot and Fusarium head blight

management. Australasian Plant Pathol. 35:643-655.
Chamswarng, C., and R.J. Cook. 1985. Identification and comparative pathogenicity of *Pythium* species from wheat roots and wheat-field soils in the Pacific Northwest. Phytopathology 75:821-827.
Christian, D.G., and D.P. Miller. 1984. Cephalosporium stripe in winter wheat grown after different methods of straw disposal. Plant Pathol. 33:605-606.
Clarke, B.B., and A.B. Gould (ed.) 1993. Turfgrass patch diseases caused by ectotrophic root-infecting fungi. APS Press, St. Paul, MN.
Clear, R.M., S.K. Patrick, D. Gaba, M. Roscoe, T.K. Turkington, T. Demeke, S. Pouleur, L. Couture, T.J. Ward, and K. O'Donnell. 2006. Trichothecene and zearalenone production, in culture, by isolates of *Fusarium pseudograminearum* from western Canada. Can. J. Plant Pathol. 28:131-136.
Colbach, N., P. Lucas, and J.M. Meynard. 1997. Influence of crop management on take-all development and disease cycles on winter wheat. Phytopathology 87:26-32.
Colbach, N., J.M. Meynard, C. Duby, and P. Huet. 1999. A dynamic model of the influence of rotation and crop management of the disease development of eyespot: Proposal of cropping systems with low disease risk. Crop Prot. 18:451-461.
Colbach, N., and L. Saur. 1998. Influence of wheat crop management on eyespot development and infection cycles. Eur. J. Plant Pathol. 104:37-48.
Collard, B.C.Y., R. Jolley, W.D. Bovill, R.A. Grams, G.B. Wildermuth, and M.W. Sutherland. 2006. Confirmation of QTL mapping and marker validation for partial seedling resistance to crown rot in wheat line '2-49'. Aust. J. Agric. Res. 57:967-973.
Conner, R.L., and T.G. Atkinson. 1989. Influence of continuous cropping on severity of common root rot in wheat and barley. Can. J. Plant Pathol. 11:127-132.
Conner, R.L., L.J. Duczek, G.C. Kozub, and A.D. Kuzyk. 1996. Influence of crop rotation on common root rot of wheat and barley. Can. J. Plant Pathol. 18:247-254.
Cook, R.J. 1980. *Fusarium* foot rot of wheat and its control in the Pacific Northwest. Plant Dis. 64:1061-1066.
Cook, R.J. 2003. Take-all of wheat. Physiol. Mol. Plant Pathol. 62:73-86.
Cook, R.J. 2007. Management of resident plant growth-promoting rhizobacteria with the cropping system: A review of experience in the US Pacific Northwest. Eur. J. Plant Pathol. 119:255-264.
Cook, R.J., and K.F. Baker. 1983. The nature and practice of biological control of plant pathogens. Am. Phytopathol. Soc., St. Paul, MN.
Cook, R.J., C. Chamswarng, and W.H. Tang. 1990. Influence of wheat chaff and tillage on *Pythium* populations in soil and *Pythium* damage to wheat. Soil Biol. Biochem. 22:939-947.
Cook, R.J., B.H. Ownley, H. Zhang, and D. Vakoch. 2000. Influence of paired-row spacing and fertilizer placement on yield and root diseases of direct-seeded wheat. Crop Sci. 40:1079-1087.
Cook, R.J., W.F. Schillinger, and N.W. Christensen. 2002a. Rhizoctonia root rot and take-all of wheat in diverse direct-seed spring cropping systems. Can. J. Plant Pathol. 24:349–358.
Cook, R.J., J.W. Sitton, and W.A. Haglund. 1987. Influence of soil treatments on growth and yield of wheat and implications for control of Pythium root rot. Phytopathology 77:1172-1198.
Cook, R.J., J.W. Sitton, and J.T. Waldher. 1980. Evidence for *Pythium* as a pathogen of direct-drilled wheat in the Pacific Northwest. Plant Dis. 64:102-103.
Cook R.J., and R.J. Veseth. 1991. Wheat health management. APS Press, St. Paul, MN.
Cook, R.J., D.M. Weller, A.Y. El-Banna, D. Vakoch, and H. Zhang. 2002b. Yield responses of direct-seed wheat to fungicide and rhizobacteria treatments. Plant Dis. 87:780-784.
Couch, H.B. 1995. Diseases of turfgrasses. 3rd ed. Keiger Publ., Malabar, FL.
Couture, L., and J.C. Sutton. 1980. Effect of dry heat treatments on survival of seed borne *Bipolaris sorokiniana* and germination of barley seeds. Can. Plant Dis. Survey 60:59-61.
Cowger, C., and C.C. Mundt. 1998. A hydroponic seedling assay for resistance to Cephalosporium stripe of wheat. Plant Dis. 82:1126-1131.
Creatura, P.J., G.R. Safir, R.P. Scheffer, and T.D. Sharkey. 1981. Effects on *Cephalosporium gramineum* and a toxic metabolite on stomates and water status of wheat. Physiol. Plant Pathol. 19:313-323.
Crous, P.W., J.Z. Ewald Groenewald, and W. Gams. 2003. Eyespot of cereals revisited: ITS phylogeny reveals new species relationships. Eur. J. Plant Pathol. 109:841-850.
Dalal, R.C., E.J. Weston, W.M. Strong, K.J. Lehane, J.E. Cooper, G.B. Wildermuth, A.J. King, and C.J. Holmes. 2004. Sustaining productivity of a Vertosol at Warra, Queensland, with fertilisers, no-tillage or legumes: 7. Yield, nitrogen and disease-break benefits from lucerne in a two-year lucerne-wheat rotation. Aust. J. Exp. Agric. 44:607-616.
Douhan, G.W., and T.D. Murray. 2001. Infection of winter wheat by a beta-glucuronidase-transformed isolate of *Cephalosporium gramineum*. Phytopathology 91:232-239.
Douhan, G.W., T.D. Murray, and P.S. Dyer. 2003. Population genetic structure of *Tapesia acuformis* in Washington State. Phytopathology 93:650-656.
Duczek, L.J. 1990. Sporulation of *Cochliobolus sativus* on crown and underground parts of spring cereals in relation to weather and host species, cultivar, and phenology. Can. J. Plant Pathol. 12:273-278.

Dulout, A., P. Lucas, A. Sarniguet, and T. Doré. 1997. Effects of wheat volunteers and blackgrass in set-aside following a winter wheat crop on soil infectivity and soil conduciveness to take-all. Plant Soil 197:149-155.

Duveiller, E., and I. Garcia Altamirano. 2000. Pathogenicity of *Bipolaris sorokiniana* isolates from wheat roots, leaves and grains in Mexico. Plant Pathol. 49:235-242.

Elliott, M.L. 1991. Determination of an etiological agent of bermudagrass decline. Phytopathology 81:1380-1384.

El-Nashaar, H.M., and R.W. Stack. 1989. Effect of long-term continuous cropping of spring wheat on aggressiveness of *Cochliobolus sativus*. Can. J. Plant Sci. 69:395-400.

Ennaïfar, S., P. Lucas, J-M. Meynard, and D. Makowsky. 2005. Effects of summer fallow management on take-all of winter wheat caused by *Gaeumannomyces graminis* var. *tritici*. Eur. J. Plant Pathol. 112:167-181.

Ennaïfar, S., D. Makowsky, J.M. Meynard, and P. Lucas. 2007. Evaluation of models to predict take-all incidence in winter wheat as a function of cropping practices, soil, and climate. Eur. J. Plant Pathol. 118:127-143.

Farr, D.F., A.Y. Rossman, M.E. Palm, and E.B. McCray. 2007. Fungal databases [Online]. USDA-ARS, Beltsville, MD. Available at http://nt.ars-grin.gov/fungaldatabases/ (verified 13 June 2008).

Fedel-Moen, R., and J.R. Harris. 1987. Stratified distribution of *Fusarium* and *Bipolaris* on wheat and barley with dryland root rot in South Australia. Plant Pathol. 36:447-454.

Fernandez, M.R., and Y. Chen. 2005. Pathogenicity of Fusarium species on different plant parts of spring wheat under controlled conditions. Plant Dis. 89:164-169.

Fernandez, M.R., and R.P. Zentner. 2005. The impact of crop rotation and N fertilizer on common root rot of spring wheat in the Brown soil zone of western Canada. Can. J. Plant Sci. 85:569-575.

Fitt, B.D.L., A. Goulds, and R.W. Polley. 1988. Eyespot (*Pseudocercosporella herpotrichoides*) epidemiology in relation to prediction of disease severity and yield loss in winter wheat - A review. Plant Pathol. 37:311-328.

Freeman, J., and H. Ward. 2004. *Gaeumannomyces graminis*, the take-all fungus and its relatives. Mol. Plant Pathol. 5:235-252.

Fukui, R., G.S. Campbell, and R.J. Cook. 1994. Factors influencing the incidence of embryo infection by *Pythium* spp. during germination of wheat seeds in soils. Phytopathology 84:695-702.

Gac, M.L., F. Montfort, and N. Cavelier. 1996a. An assay based on the Polymerase Chain Reaction for the detection of N- and L-types of *Pseudocercosporella herpotricoides* in wheat. J. Phytopathol. 144:513-518.

Gac, M.L., F. Montfort, N. Cavelier, and A. Sailland. 1996b. Comparative study of morphological, cultural and molecular markers for the characterization of *Pseudocercosporella herpotrichoides* isolates. Eur. J. Plant Pathol. 102:325-337.

Gams, W. 2000. *Phialophora* and some similar morphologically little-differentiated anamorphs of divergent ascomycetes. Stud. Mycol. 45:187-199.

Garrett, S.D. 1970. Pathogenic root-infecting fungi. Cambridge Univ. Press, Cambridge, UK.

Gill, J.S., K. Sivasithamparam, and K.R.J. Smettem. 2000. Soil types with different texture affects development of Rhizoctonia root rot of wheat seedlings. Plant Soil 221:113-120.

Gill, J.S., K. Sivasithamparam, and K.R.J. Smettem. 2001a. Influence of depth of soil disturbance on root growth dynamics of wheat seedlings associated with *Rhizoctonia solani AG-8* disease severity in sandy and loamy sand soils of Western Australia. Soil Tillage Res. 62:73-83.

Gill, J.S., K. Sivasithamparam, and K.R.J. Smettem. 2001b. Soil moisture affects disease severity and colonisation of wheat roots by *Rhizoctonia solani AG-8*. Soil Biol. Biochem. 33:1363-1370.

Gill, J.S., K. Sivasithamparam, and K.R.J. Smettem. 2001c. Effect of soil moisture at different temperatures on Rhizoctonia root rot of wheat seedlings. Plant Soil 231:91-96.

Gill, J.S., K. Sivasithamparam, and K.R.J. Smettem. 2002. Size of bare-patches in wheat caused by *Rhizoctonia solani AG-8* is determined by the established mycelial network at sowing. Soil Biol. Biochem. 34:889-893.

González, D., D.E. Carling, S. Kuninaga, R. Vilgalys, and M.A. Cubeta. 2001. Ribosomal DNA systematics of *Ceratobasidium* and *Thanatephorus* with *Rhizoctonia* anamorphs. Mycologia 93:1138-1150.

González, D., M.A. Cubeta, and R. Vilgalys. 2006. Phylogenetic utility of indels within ribosomal DNA and beta-tubulin sequences from fungi in the *Rhizoctonia solani s*pecies complex. Mol. Phylogenet. Evol. 40:459-470.

Gosme, M., L. Willocquet, and P. Lucas. 2007. Size, shape and intensity of aggregation of take-all disease during natural epidemics in second wheat crops. Plant Pathol. 56:87-96.

Grewal, H.S., R.D. Graham, and Z. Rengel. 1996. Genotypic variation in zinc efficiency and resistance to crown rot disease (*Fusarium graminearum* Schw. Group 1) in wheat. Plant Soil 186:219-226.

Griffin, D.M. 1972. Ecology of soil fungi. Syracuse Univ. Press, Syracuse, NY.

Gutteridge, R.J., J.P. Zhang, J.F. Jenkyn, and G.L. Bateman. 2005. Survival and multiplication of *Gaeumannomyces graminis* var. *tritici* (the wheat take-all fungus) and related fungi on different wild and cultivated grasses. Appl. Soil Ecol. 29:143-154.

Hall, R., and J.C. Sutton. 1998. Relation of weather, crop, and soil variables to the prevalence, incidence, and

severity of basal infections of winter wheat in Ontario. Can. J. Plant Pathol. 20:69-80.

Hendrix, F.F., and W.A. Campbell. 1970. Distribution of *Phytophthora* and *Pythium* species in soils in the continental United States. Can. J. Bot. 48:377-384.

Herdina, and D.K. Roget. 2000. Prediction of take-all disease risk in field soils using a rapid and quantitative DNA soil assay. Plant Soil 227:87-98.

Hering, T.F., R.J. Cook, and W.H. Tang. 1987. Infection of wheat embryos by *Pythium* species during seed germination and the influence of seed age and soil matric potential. Phytopathology 77:1104-1108.

Higginbotham, R.W, K.K. Kidwell, and T.C. Paulitz. 2004a. Evaluation of adapted wheat cultivars for tolerance to Pythium root rot. Plant Dis. 88:1027–1032.

Higginbotham, R.W., T.C. Paulitz, and K.K. Kidwell. 2004b. Virulence of *Pythium* species isolated from wheat fields in eastern Washington. Plant Dis. 88:1021-1026.

Hollins, T.W., K.D. Lockley, J.A. Blackman, P.R. Scott, and J. Bingham. 1988. Field performance of Rendezvous, a wheat cultivar with resistance to eyespot (*Pseudocercosporella herpotrichoides*) derived from *Aegilops ventricosa*. Plant Pathol. 37:251-260.

Hornby, D. 1978. The problems of trying to forecast take-all. p. 151-158. *In* P.R. Scott and A. Bainbridge (ed.) Plant disease epidemiology. Blackwell Sci. Publ., Oxford, UK.

Hornby, D. 1979. Take-all decline: A theorist's paradise. p. 133-156. *In* B. Schippers and W. Gams (ed.) Soil-borne plant pathogens. Academic Press, London, UK.

Hornby, D., G.L. Bateman, R.J. Gutteridge, P. Lucas, A.E. Osbourn, E. Ward, and D.J. Yarham. 1998. Take-all disease of cereals: A regional perspective. CAB Int., Wallingford, UK.

Huber, D.M., C.C. Painter, H.C. McKay, and D.L. Petersen. 1968. Effect of nitrogen fertilization on take-all of winter wheat. Phytopathology 58:1470-1472.

Hunter, T. 1989. Occurrence of *Tapesia yallundae*, teleomorph of *Pseudocercosporelle herpotrichoides*, on unharvested wheat culms in England. Plant Pathol. 38:598-603.

Ingram, D.M., and R.J. Cook. 1990. Pathogenicity of four *Pythium* species to wheat, barley, peas and lentils. Plant Pathol. 39:110-117.

Jahier, J., A.M. Tanguy, and G. Doussinault. 1989. Analysis of the level of eyespot resistance due to genes transferred to wheat from *Aegilops ventricosa*. Euphytica 44:55-59.

Jenkinson, P., and D.W. Parry. 1994. Splash dispersal of conidia of *Fusarium culmorum* and *Fusarium avenaceum*. Mycol. Res. 98:506-510.

Kageyama, K., and E.B. Nelson. 2003. Differential inactivation of seed exudate stimulation of *Pythium ultimum* sporangium germination by *Enterobacter cloacae* influences biological control efficacy on different plant species. Appl. Environ. Microbiol. 69:1114-1120.

Kim, D.S., R.J. Cook, and D.M. Weller. 1997. *Bacillus* sp. L324-92 for biological control of three root diseases of wheat grown with reduced tillage. Phytopathology 87:551-558.

Kirkegaard, J.A., S. Simpfendorfer, J. Holland, R. Bambach, K.J. Moore, and G.J. Rebetzke. 2004. Effect of previous crops on crown rot and yield of durum and bread wheat in northern NSW. Aust. J. Agric. Res. 55:321-334.

Kobayasi, K., and T. Ui. 1979. Phytotoxicity and antimicrobial activity of Graminin A, produced by *Cephalosporium gramineum*, the causal agent of Cephalosporium stripe disease of wheat. Physiol. Plant Pathol. 14:129-133.

Kokko, E.G., R.L. Conner, G.C. Kozub, and B. Lee. 1995. Effects of common root rot on discoloration and growth of the spring wheat root system. Phytopathology 85:203-208.

Krupa, S.V., and Y.R. Dommergues (ed.) 1979. Ecology of root pathogens. Elsevier Sci. Publ. Co., Amsterdam, The Netherlands.

Kumar, J., P. Schafer, R. Huckelhoven, G. Langen, H. Baltruschat, E. Stein, S. Najarajan, and K.H. Kogel. 2002. *Bipolaris sorokiniana*, a cereal pathogen of global concern: Cytological and molecular approaches towards better control. Mol. Plant Pathol. 3:185-195.

Latin, R.X., R.W. Harder, and M.V. Wiese. 1982. Incidence of Cephalosporium stripe as influenced by winter wheat management practices. Plant Dis. 66:229-230.

Lebreton, L., M. Gosme, P. Lucas, A.Y. Guillerm-Erckelboudt, and A. Sarniguet. 2007. Linear relationship between *Gaeumannomyces graminis* var. *tritici* (Ggt) genotypic frequencies and disease severity on wheat roots in the field. Environ. Microbiol. 9:492-499.

Lebreton, L., P. Lucas, F. Dugas, A.Y. Guillerm, A. Schoeny, and A. Sarniguet. 2004. Changes in population structure of the soilborne fungus *Gaeumannomyces graminis* var. *tritici* during continuous wheat cropping. Environ. Microbiol. 6:1174-1185.

Lees, A.K., D.W. Cullen, L. Sullivan, and M.J. Nicolson. 2002. Development of conventional and quantitative real-time PCR assays for the detection and identification of *Rhizoctonia solani* AG-3 in potato and soil. Plant Pathol. 51:293-302.

Leroux, P., and M. Gredt. 1997. Evolution of fungicide resistance in the cereal eyespot fungi *Tapesia yallundae* and *Tapesia acuformis* in France. Pestic. Sci. 51:321-327.

Lévesque, C.A., and A.W.A.M. de Cock. 2004. Molecular phylogeny and taxonomy of the genus *Pythium*. Mycol. Res. 108:1363-1383.

Lévesque, C.A., and J.E. Rahe. 1992. Herbicide interactions with fungal root pathogens, with special reference to glyphosate. Ann. Rev. Phytopathol. 30:579-602.

Lucas, J.A., P.S. Dyer, and T.D. Murray. 2000. Pathogenicity, host specificity, and population biology of *Tapesia* spp., causal agents of eyespot disease in cereals. Adv. Bot. Res. 33:226-258.

Lucas, P., M.-H. Jeuffroy, A. Schoeny, and A. Sarniguet. 1997. Basis for nitrogen fertilisation management of winter wheat crops infected with take-all. Aspects Appl. Biol. 50:255-262.

Lucas, P., and A. Sarniguet. 1998. Biological control of soilborne pathogens with resident versus introduced antagonists: Should diverging approaches become strategic convergence? p. 351-370. *In* P. Barbosa (ed.) Conservation biological control. Academic Press, New York, NY.

Lucas, P., R.W. Smiley, and H.P. Collins. 1993. Decline of Rhizoctonia root rot on wheat in soils infested with *Rhizoctonia solani* AG-8. Phytopathology 83:260-265.

MacNish, G.C., and S.M. Neate. 1996. Rhizoctonia bare patch of cereals: An Australian perspective. Plant Dis. 80:965-971.

Maia, N. 1967. Obtention de blés tendres résistants au piétin-verse par croisements interspécifiques blés X *Aegilops*. Comptes Rendus Hebdomadaires des Séances de l'Académie d'Agriculture de France 53:149-154.

Martin, F.N. 2000. Phylogenetic relationships among some *Pythium* species inferred from sequence analysis of the mitochondrially encoded cytochrome oxidase II gene. Mycologia 92:711-727.

Martin, F.N., and J.E. Loper. 1999. Soilborne plant diseases caused by *Pythium* spp: Ecology, epidemiology, and prospects for biological control. Crit. Rev. Plant Sci. 18:111-181.

Martin, J.M., R.H. Johnston, and D.E. Mathre. 1989. Factors affecting the severity of Cephalosporium stripe of winter wheat. Can. J. Plant Pathol. 11:361-367.

Martin, J.M., D.E. Mathre, and R.H. Johnston. 1986. Winter wheat genotype responses to *Cephalosporium gramineum* inoculum levels. Plant Dis. 70:421-423.

Mathieson, J.T., C.M. Rush, D. Bordovsky, L.E. Clark, and O.R. Jones. 1990. Effects of tillage on common root rot of wheat in Texas. Plant Dis. 74:1006-1008.

Mathre, D.E., and R.H. Johnston. 1975. Cephalosporium stripe of winter wheat: Infection processes and host response. Phytopathology 65:1244-1249.

Mathre, D.E., and R.H. Johnston. 1977. Physical and chemical factors affecting sporulation of *Hymenula cerealis*. Trans. Brit. Mycol. Soc. 69:213-215.

Mathre, D.E., and R.H. Johnston. 1990. A crown barrier related to Cephalosporium stripe resistance in wheat relatives. Can. J. Bot. 68:1511-1514.

Mathre, D.E., R.H. Johnston, and J.M. Martin. 1985. Sources of resistance to *Cephalosporium gramineum* in *Triticum* and *Agropyron* species. Euphytica 34:419-424.

Mazzola, M., R.W. Smiley, A.D. Rovira, and R.J. Cook. 1996a. Characterization of *Rhizoctonia* isolates, disease occurrence and management in cereals. p. 259-267. *In* B. Sneh, S. Jabaji-Hare, S. Neate, and G. Dijst (ed.) *Rhizoctonia* species: Taxonomy, molecular biology, ecology, pathology and disease control. Kluwer Academic Publishers, Dordrecht, The Netherlands.

Mazzola, M., O.T. Wong, and R.J. Cook. 1996b. Virulence of *Rhizoctonia oryzae* and *R. solani* AG-8 on wheat and detection of *R. oryzae* in plant tissue by PCR. Phytopathology 86:354–360.

Miedaner, T. 1997. Breeding wheat and rye for resistance to *Fusarium* diseases. Plant Breed. 116:201-220.

Miedaner, T., G. Gang, C. Reinbrecht, and H.H. Geiger. 1997. Lack of association between *Fusarium* foot rot and head blight resistance in winter rye. Crop Sci. 37:327-331.

Milus, E.A., and C.S. Rothrock. 1997. Efficacy of bacterial seed treatments for controlling Pythium root rot of winter wheat. Plant Dis. 81:180-184.

Mitter, V., M.C. Zhang, C.J. Liu, R. Ghosh, M. Ghosh, and S. Chakraborty. 2006. A high-throughput glasshouse bioassay to detect crown rot resistance in wheat germplasm. Plant Pathol. 55:433-441.

Monds, R.D., M.G. Cromey, D.R. Lauren, M. di Menna, and J. Marshall. 2005. *Fusarium graminearum*, *F. cortaderiae* and *F. pseudograminearum* in New Zealand: Molecular phylogenetic analysis, mycotoxin chemotypes and co-existence of species. Mycol. Res. 109:410-420.

Moore, K.J., and R.J. Cook. 1984. Increased take-all of wheat with direct drilling in the Pacific Northwest. Phytopathology 74:1044-1049.

Morton, J.B., and D.E. Mathre. 1980. Identification of resistance to Cephalosporium stripe in winter wheat. Phytopathology 70:812-817.

Mundt, C.C. 2002. Performance of wheat cultivars and cultivar mixtures in the presence of Cephalosporium stripe. Crop Prot. 1:93-99.

Muranty, H., J. Jahier, A.J. Tanguy, A.J. Worland, and C. Law. 2002. Inheritance of resistance of wheat to eyespot at the adult stage. Plant Breed. 121:536-538.

Murray, T.D. 2006. Seed transmission of *Cephalosporium gramineum* in winter wheat. Plant Dis. 90:803-806.

Murray, T.D., D.W. Parry, and N.D. Cattlin (ed.) 1998. A color handbook of diseases of small grain cereal crops. Iowa State Univ. Press, Ames, IA.

Murray, T.D., L. Pritchett, S.S. Jones, and S. Lyon. 2001. Reaction of winter wheat cultivars and breeding lines to Cephalosporium stripe. p. S21. *In* Biological and cultural tests for control of plant diseases. APS Press, St. Paul, MN.

Murray, T.D., and C.C. Walter. 1991. Influence of pH and matric potential on sporulation of *Cephalosporium*

gramineum. Phytopathology 81:79-84.
Murray, T.D., C.C. Walter, and J.C. Anderegg. 1992. Control of Cephalosporium stripe of winter wheat by liming. Plant Dis. 76:282-286.
Neate, S.M. 1987. Plant debris in soil as a source of inoculum of Rhizoctonia in wheat. Trans. Br. Mycol. Soc. 88:157-162.
Nelson, K.E., and J.C. Sutton. 1988. Epidemiology of eyespot on winter wheat in Ontario. Phytoprotection 69:9-21.
Nelson, P.E., T.A. Toussoun, and R.J. Cook (ed.) 1981. Fusarium: Diseases, biology, and taxonomy. Pennsylvania State Univ. Press, University Park, PA.
Nicol, J.M., N. Bolat, A. Bağcı, R.T. Trethowan, M. William, H. Hekimhan, A.F. Yildirim, E. Şahin, H. Elekçioğlu, H. Toktay, B. Tunalı, A. Hede, S. Taner, H.J. Braun, M. van Ginkel, M. Keser, Z. Arisoy, A. Yorgancılar, A. Tulek, D. Erdurmuş, O. Büyük, and M. Aydogdu. 2007. The international breeding strategy for the incorporation of resistance in bread wheat against the soil borne pathogens (dryland root rot and cyst and lesion nematodes) using conventional and molecular tools. p. 125-137. *In* H.T. Buck, J.E. Nisi, and N. Salomón (ed.) Wheat production in stressed environments. Springer, Dordrecht, The Netherlands.
Nirenberg, H.I. 1981. Differentiation of *Pseudocercosporella* strains causing foot rot diseases of cereals: 1. Morphology. Z. Pflanzenkrankh. Pflanzenschutz. 88:241-248.
O'Donnell, K., T.J. Ward, D.M. Geiser, H.C. Kistler, and T. Aoki. 2004. Genealogical concordance between the mating type locus and seven other nuclear genes supports formal recognition of nine phylogenetically distinct species within the *Fusarium graminearum* clade. Fungal Genet. Biol. 41:600-623.
Ogoshi, A., R.J. Cook, and E.N. Bassett. 1990. *Rhizoctonia* species and anastomosis groups causing root rot of wheat and barley in the Pacific Northwest. Phytopathology 80:784–788.
Okubara, P.A., and T.C. Paulitz. 2005. Root defense responses to fungal pathogens: A molecular perspective. Plant Soil 274:215-226.
Okubara, P.A., K.L. Schroeder, and T.C. Paulitz. 2008. Identification and quantification of *Rhizoctonia solani* and *R. oryzae* using real-time PCR. Phytopathology 98:837-847.
Ophel-Keller, K., A. McKay, D. Hartley, Herdina, and J. Curran. 2008. Development of a routine DNA-based testing service for soilborne diseases in Australia. Australasian Plant Pathol. 37:243-253.
Pankhurst, C.E., H.J. McDonald, and B.G. Hawke. 1995. Influence of tillage and crop rotation on the epidemiology of *Pythium* infections of wheat in a red-brown earth of South Australia. Soil Biol. Biochem. 27:1065-1073.
Parker, C.A., A.D. Rovira, K.J. Moore, P.T.W. Wong, and J.F. Kollmorgen (ed.) 1985. Ecology and management of soilborne plant pathogens. APS Press, St. Paul, MN.
Parry, D.W., T.R. Pettitt, P. Jenkinson, and A.K. Lees. 1994. The cereal Fusarium complex. p. 301-320. *In* J. P. Blakeman and B. Williamson (ed.) Ecology of plant pathogens. CAB Int., Wallingford, UK.
Paulitz, T.C. 2002. First report of *Rhizoctonia oryzae* on pea. Plant Dis. 86:442.
Paulitz, T.C., and K. Adams. 2003. Composition and distribution of *Pythium* communities from wheat fields in eastern Washington State. Phytopathology 93:867-873.
Paulitz, T.C., K. Adams, and M. Mazzola. 2003a. *Pythium abappressorium*-a new species from eastern Washington. Mycologia 95:80-86.
Paulitz, T.C., and K.L. Schroeder. 2005. A new method for quantification of *Rhizoctonia solani* and *R. oryzae* from soil. Plant Dis. 89:767-772.
Paulitz, T.C., and R.B. Scott. 2006. Effect of seed treatments for control of Rhizoctonia root rot in spring wheat, 2005. Fungic. Nematic. Tests 61:ST014.
Paulitz, T.C., R.W. Smiley, and R.J. Cook. 2002a. Insights into the prevalence and management of soilborne cereal pathogens under direct seeding in the Pacific Northwest, U.S.A. Can. J. Plant Pathol. 24:416–428.
Paulitz, T.C., J. Smith, and K. Kidwell. 2002b. Virulence of *Rhizoctonia oryzae* on wheat and barley cultivars from the Pacific Northwest. Plant Dis. 87:51-55.
Paulitz, T.C., H. Zhang, and R.J. Cook. 2003b. Spatial distribution of *Rhizoctonia oryzae* and rhizoctonia root rot in direct-seeded cereals. Can. J. Plant Pathol. 25:295–303.
Pettitt, T., X.M. Xu, and D. Parry. 2003. Association of *Fusarium* species in the wheat stem rot complex. Eur. J. Plant Pathol. 109:769-774.
Piccinni, G., C.M. Rush, K.M. Vaughn, and M.D. Lazar. 2000. Lack of relationship between susceptibility to common root rot and drought tolerance among several closely related wheat lines. Plant Dis. 84:25-28.
Pittaway, P.A. 1995. Opportunistic association between *Pythium* species and weed residues causing seedling emergence failure in cereals. Aust. J. Agric. Res. 46:655-662.
Ponchet, J. 1959. La maladie du piétin-verse des céréales: *Cercosporella herpotrichoides* Fron. Importance agronomique, biologie, épiphytologie. Annales des Epiphyties 10:45-98.
Pool, R.A.F., and E.L. Sharp. 1969. Some environmental and cultural factors affecting Cephalosporium stripe of winter wheat. Plant Dis. Rep. 53:898-902.
Priestley, R.A., F.M. Dewey, P. Nicholson, and H.N. Rezanoor. 1992. Comparison of isoenzyme and DNA markers for differentiating W-, R- and C-pathotypes of *Pseudocercosporella herpotrichoides*. Plant Pathol. 41:591-599.

Pumphrey, F.V., D.E. Wilkins, D.C. Hane, and R.W. Smiley. 1987. Influence of tillage and nitrogen fertilizer on Rhizoctonia root rot (bare patch) of winter wheat. Plant Dis. 71:125-127.

Rachdawong, S., C.L. Cramer, E.A. Grabau, V.K. Stromberg, G.H. Lacey, and E.L. Stromberg. 2002. *Gaeumannomyces graminis* vars. *avenae*, *graminis*, and *tritici* identified using PCR amplification of avenacinase-like genes. Plant Dis. 86:652-660.

Rahman, M., C.C. Mundt, T.J. Wolpert, and O. Riera-Lizarazu. 2001. Sensitivity of wheat genotypes to a toxic fraction produced by *Cephalosporium gramineum* and correlation with disease susceptibility. Phytopathology 91:702-707.

Rapilly, F., P. Eschenbrenner, E. Choisnes, and F. La Croze. 1979. La prévision du piétin-verse sur blé d'hiver. Perspectives Agricoles 23:30-40.

Ray, V.R., M.J. Crook, P. Jenkinson, and S.G. Edwards. 2006. Effect of eyespot caused by *Oculimacula yallundae* and *O. acuformis*, assessed visually and by competitive PCR, on stem strength associated with lodging resistance and yield of winter wheat. J. Exp. Bot. 57:2249-2257.

Raymond, P.J., and W.W. Bockus. 1984. Effect of seeding date of winter wheat on incidence, severity, and yield loss caused by Cephalosporium stripe in Kansas. Plant Dis. 68:665-667.

Reis, E.M., and J.J.R. Abrao. 1983. Effect of tillage and wheat residue management on the vertical distribution and inoculum density of *Cochliobolus sativus* in soil. Plant Dis. 67:1088-1089.

Robbertse, B., G.F. Campbell, and P.W. Crous. 1995. Revision of *Pseudocercosporella*-like species causing eyespot disease of wheat. S. Afr. J. Bot. 61:43-48.

Roget, D.K. 1995. Decline in root rot (*Rhizoctonia solani* AG-8) in wheat in a tillage and rotation experiment at Avon, South Australia. Aust. J. Exp. Agric. 35:1009-1013.

Roget, D.K., S.M. Neate, and A.D. Rovira. 1996. Effect of sowing point design and tillage practice on the incidence of Rhizoctonia root rot, take-all and cereal cyst nematode in wheat and barley. Aust. J. Exp. Agric. 36:683–693.

Roget, D.K., N.R. Venn, and A.D. Rovira. 1987. Reduction of Rhizoctonia root rot of direct-drilled wheat by short-term chemical fallow. Aust. J. Exp. Agric. 27:425-430.

Rowe, R.C., and R.L. Powelson. 1973. Epidemiology of *Cercosporella* footrot of wheat: Spore production. Phytopathology 63:981-984.

Sarniguet, A., P. Lucas, and M. Lucas. 1992a. Relationships between take-all, soil conduciveness to the disease, populations of fluorescent pseudomonads and nitrogen fertilizers. Plant Soil 145:17-27.

Sarniguet, A., P. Lucas, M. Lucas, and R. Samson. 1992b. Soil conduciveness to take-all of wheat: Influence of the nitrogen fertilizers on the structure of populations of fluorescent pseudomonads. Plant Soil 145:29-36.

Satyaprasad, K., G.L. Bateman, and E. Ward. 2000. Comparisons of isolates of *Fusarium avenaceum* from white lupin and other crops by pathogenicity tests, DNA analyses and vegetative compatibility tests. J. Phytopathol. 148:211-219.

Savary, S., B. Mille, B. Rolland, and P. Lucas. 2006. Patterns and management of crop multiple pathosystems. Eur. J. Plant Pathol. 115:123-138.

Schippers, B., and W. Gams (ed.) 1979. Soil-borne plant pathogens. Academic Press, London, UK.

Schoeny, A., F. Devienne-Barret, M.H. Jeuffroy, and P. Lucas. 2003. Effect of take-all infections on nitrate uptake in winter wheat. Plant Pathol. 52:52-59.

Schoeny, A., M.H. Jeuffroy, and P. Lucas. 2001. Influence of take-all epidemics on winter wheat yield formation and yield loss. Phytopathology 91:694-701.

Schoeny, A., and P. Lucas. 1999. Modeling of take-all epidemics to evaluate the efficacy of a new seed-treatment fungicide on wheat. Phytopathology 89:954-961.

Schroeder, K.L., P.A. Okubara, and T.C. Paulitz. 2007. Geographic distribution of *Rhizoctonia* and *Pythium* species in soils from dryland cereal cropping systems in eastern Washington. Phytopathology 97:S105 (abstract).

Schroeder, K.L., P.A. Okubara, J.T. Tambong, C.A. Lévesque, and T.C. Paulitz. 2006. Identification and quantification of pathogenic *Pythium* spp. from soils in eastern Washington using real-time PCR. Phytopathology 96:637-647.

Schroeder, K.L., and T.C. Paulitz. 2006. Root diseases of wheat and barley during the transition from conventional tillage to direct seeding. Plant Dis. 90:1247-1253.

Shefelbine, P.A., and W.W. Bockus. 1989. Decline of Cephalosporium stripe by monoculture of moderately resistant winter wheat cultivars. Phytopathology 79:1127-1131.

Siebrasse, G., and H. Fehrman. 1987. An enlarged model for the chemical control of eyespot (*Pseudocercosporella herpotrichoides*) in winter wheat. Z. Pflanzenkrankh. Pflanzenschutz. 94:137-149.

Singleton, L.L., J.D. Mihail, and C.M. Rush (ed.) 1992. Methods for research on soilborne phytopathogenic fungi. APS Press, St. Paul, MN.

Sitton, J.W., and R.J. Cook. 1981. Comparative morphology and survival of chlamydospores of *Fusarium roseum* 'Culmorum' and 'Graminearum'. Phytopathology 71:85-90.

Slope, D.B., and R. Bardner. 1965. Cephalosporium stripe of wheat and root damage by insects. Plant Pathol. 14:184-187.

Smiley, R.W. 1978. Antagonists of *Gaeumannomyces graminis* from the rhizoplane of wheat in soils fertilized

with ammonium or nitrate nitrogen. Soil Biol. Biochem. 10:169-174.

Smiley, R.W., H.P. Collins, and P.E. Rasmussen. 1996a. Diseases of wheat in long-term agronomic experiments at Pendleton, Oregon. Plant Dis. 80:813-820.

Smiley, R.W., P.H. Dernoeden, and B.B. Clarke. 2005a. Compendium of turfgrass diseases. 3rd ed. APS Press, St. Paul, MN.

Smiley, R.W., M.C. Fowler, and K.L. Reynolds. 1986. Temperature effects on take-all of cereals, caused by *Phialophora graminicola* and *Gaeumannomyces graminis*. Phytopathology 76:923-931.

Smiley, R.W., J.A. Gourlie, S.A. Easley, and L.M. Patterson. 2005b. Pathogenicity of fungi associated with the wheat crown rot complex in Oregon and Washington. Plant Dis. 89:949-957.

Smiley, R.W., A.G. Ogg, and R.J. Cook. 1992. Influence of glyphosate on Rhizoctonia root rot, growth, and yield of barley. Plant Dis. 76:937-942.

Smiley, R.W., and L. Patterson. 1996. Pathogenic fungi associated with Fusarium foot rot of winter wheat in the semiarid Pacific Northwest. Plant Dis. 80:944-949.

Smiley, R.W., L.-M. Patterson, and C.W. Shelton. 1996b. Fungicide seed treatments influence emergence of winter wheat in cold soil. J. Prod. Agric. 9:559-563.

Smiley, R.W., and W. Uddin. 1993. Influence of soil temperature on Rhizoctonia root rot (*R. solani* AG-8 and *R. oryzae*) of winter wheat. Phytopathology 83:777-785.

Smiley, R.W., W. Uddin, S. Ott, and K.E.L. Rhinhart. 1990. Influence of flutolonil and tolclofos-methyl on root and culm diseases of winter wheat. Plant Dis. 74:788-791.

Smiley, R.W., and D.E. Wilkins. 1992. Impact of sulfonylurea herbicides on Rhizoctonia root rot, growth, and yield of winter wheat. Plant Dis. 76:399–404.

Smiley, R.W., and D.E. Wilkins. 1993. Annual spring barley growth, yield, and root rot in high- and low-residue tillage systems. J. Prod. Agric. 6:270–275.

Smith, J.D., N. Jackson, and A.R. Woolhouse. 1989. Fungal diseases of amenity turf grasses. E. & F.N. Spon, London, UK.

Smith, J.D., K.K. Kidwell, M.A. Evans, R.J. Cook, and R.W. Smiley. 2003a. Assessment of spring wheat genotypes for disease reaction to *Rhizoctonia solani* AG 8 in controlled environment and no-till field conditions. Crop Sci. 43:694-700.

Smith, J.D., K.K. Kidwell, M.A. Evans, R.J. Cook, and R.W. Smiley. 2003b. Evaluation of spring cereal grains and wild *Triticum* relatives for resistance to *Rhizoctonia solani* AG 8. Crop Sci. 43:701-709.

Sneh, B., L. Burpee, and A. Ogoshi. 1991. Identification of *Rhizoctonia* species. APS Press, St. Paul, MN.

Sneh, B., S. Jabaji-Hare, S. Neate, and G. Dijst (ed.) 1996. *Rhizoctonia* species: Taxonomy, molecular biology, ecology, pathology and disease control. Kluwer Academic Publishers, Dordrecht, The Netherlands.

Specht, L.P., and T.D. Murray. 1990. Effects of root-wounding and inoculum density on Cephalosporium stripe in winter wheat. Phytopathology 80:1108-1114.

Stasinopoulos, S.J., and R.J. Seviour. 1989. Exopolysaccharide formation by isolates of *Cephalosporium* and *Acremonium*. Mycol. Res. 92:55-60.

Stiles, C.M., and T.D. Murray. 1996. Infection of field-grown winter wheat by *Cephalosporium gramineum* and the effect of soil pH. Phytopathology 86:177-183.

Summerell, B.A., and L.W. Burgess. 1988. Stubble management practices and the survival of *Fusarium graminearum* Group 1 in wheat stubble residues. Australasian Plant Pathol. 17:88-93.

Summerell, B.A., L.W. Burgess, D. Backhouse, S. Bullock, and L.J. Swan. 2001a. Natural occurrence of perithecia of *Gibberella coronicola* on wheat plants with crown rot in Australia. Australasian Plant Pathol. 30:353-356.

Summerell, B.A., L.W. Burgess, T.A. Klein, and A.B. Pattison. 1990. Stubble management and the site of penetration of wheat by *Fusarium graminearum* Group 1. Phytopathology 80:877-879.

Summerell, B.A., J.F. Leslie, D. Backhouse, W.L. Bryden, and L.W. Burgess (ed.) 2001b. Fusarium: Paul E. Nelson memorial symposium. APS Press, St. Paul, MN.

Tinline, R.D. 1977. Multiple infections of subcrown internodes of wheat (*Triticum aestivum*) by common root rot fungi. Can. J. Bot. 55:30-34.

Tinline, R.D., K.L. Bailey, L.J. Duczek, and H. Harding (ed.) 1991. Proc. Int. Workshop on Common Root Rot of Cereals, 1st, Saskatoon, Canada. 11-14 August 1991. Agric. Canada, Saskatoon.

Tinline, R.D., and D.T. Spurr. 1991. Agronomic practices and common root rot in spring wheat: Effect of tillage on disease and inoculum density of *Cochliobolus sativus* in soil. Can. J. Plant Pathol. 13:258-266.

Tinline, R.D., H. Ukrainetz, and D.T. Spurr. 1993. Effect of fertilizers and of liming acid soil on common root rot in wheat, and of chloride on the disease in wheat and barley. Can. J. Plant Pathol. 15:65-73.

Tinline, R.D., G.B. Wildermuth, and D.T. Spurr. 1988. Inoculum density of *Cochliobolus sativus* in soil and common root rot of wheat cultivars in Queensland. Aust. J. Agric. Res. 39:569-577.

Tóth, B., Á. Mesterházy, P. Nicholson, J. Téren, and J. Varga. 2004. Mycotoxin production and molecular variability of European and American isolates of *Fusarium culmorum*. Eur. J. Plant Pathol. 110:587-599.

Tunalı, B., J.M. Nicol, D. Hodson, Z. Uçkun, O. Büyük, D. Erdurmuş, H. Hekimhan, H. Aktaş, M.A. Akbudak, and S.A. Bağcı. 2008. Root and crown rot fungi associated with spring, facultative and winter wheat in Turkey. Plant Dis. 92:1299-1306.

van der Plaats-Niterink, A.J. 1981. Monograph of the genus Pythium: Studies in mycology 21. Centraalbureau voor Schimmelcultures, Baarn, The Netherlands.

Van Welt, S.L., and D.W. Fullbright. 1986. Pathogenicity and virulence of *Cephalosporium gramineum* is independent of in vitro production of extracellular polysaccharides and graminin A. Physiol. Mol. Plant Pathol. 28:299-307.

Van Wyk, P.S., O. Los, G.D.C. Pauer, and W.F.O. Marasas. 1987. Geographic distribution and pathogenicity of *Fusarium* species associated with crown rot of wheat in the Orange Free State, South Africa. Phytophylactica 19:271-274.

Vasquez-Siller, L.M., and T.D. Murray. 2003. Detection of *Cephalosporium gramineum* in wheat seed with PCR. Phytopathology 93:S87 (abstract).

Veit, S., J.M. Worle, T. Nurnberger, W. Koch, and H.U. Seitz. 2001. A novel protein elicitor (PaNie) from *Pythium aphanidermatum* induces multiple defense responses in carrot, *Arabidopsis*, and tobacco. Plant Physiol. 127:832-841.

Vijayan, P., J. Shockey, C.A. Lévesque, R.J. Cook, and J. Browse. 1998. A role for jasmonate in pathogen defense of Arabidopsis. Proc. Natl. Acad. Sci. USA 95:7209-7214.

Wallwork, H. 1992. Cereal leaf and stem diseases. South Aust. Res. Dev. Inst., Adelaide.

Wallwork, H. 2000. Cereal root and crown diseases. South Aust. Res. Dev. Inst., Adelaide.

Wallwork, H., M. Butt, J.P.E. Cheong, and K.J. Williams. 2004. Resistance to crown rot in wheat identified through an improved method for screening adult plants. Australasian Plant Pathol. 33:1-7.

Wallwork, H., and B. Spooner. 1988. *Tapesia yallundae* the teleomorph of *Pseudocercosporella herpotrichoides*. Trans. Brit. Mycol. Soc. 91:703-705.

Walsh, K., J. Korimbocus, N. Boonham, P. Jennings, and M. Hims. 2005. Using real-time PCR to discriminate and quantify the closely related wheat pathogens *Oculimacula yallundae* and *Oculimacula acuformis*. J. Phytopathol. 153:715-721.

Ward, W.E., and R.M. Gray. 1992. Generation of a ribosomal DNA probe by PCR and its use in identification of fungi within the *Gaeumannomyces-Phialophora* complex. Plant Pathol. 41:730-736.

Weller, D.M., and R.J. Cook. 1986. Increased growth of wheat by seed treatments with fluorescent pseudomonads, and implications of *Pythium* control. Can. J. Plant Pathol. 8:328-334.

Weller, D.M., R.J. Cook, G.E. MacNish, N. Bassett, R.L. Powelson, and R.R. Petersen. 1986. Rhizoctonia root rot of small grains favored by reduced tillage in the Pacific Northwest. Plant Dis. 70:70-73.

Weller, D.M., B.B. Landa, O.V. Mavrodi, K.L. Schroeder, L. De La Fuente, S. Blouin Bankhead, R. Allende Molar, R.F. Bonsall, D.V. Mavrodi, and L.S. Thomashow. 2007. Role of 2,4-diacetylphloroglucinol-producing fluorescent *Pseudomonas* spp. in the defense of plant roots. Plant Biol. 9:4-20.

Wiese, M.V. 1972. Colonization of wheat seedlings by *Cephalosporium gramineum* in relation to symptom development. Phytopathology 62:1013-1018.

Wiese, M.V., and A.V. Ravenscroft. 1975. *Cephalosporium gramineum* populations in soil under winter wheat cultivation. Phytopathology 65:1129-1133.

Wiese, M.V., and A.V. Ravenscroft. 1978. Sporodochium development and conidium production in *Cephalosporium gramineum*. Phytopathology 68:395-401.

Wildermuth, G.B., and R.B. McNamara. 1987. Susceptibility of winter and summer crops to root and crown infection by *Bipolaris sorokiniana*. Plant Pathol. 36:481-491.

Wildermuth, G.B., and R.B. McNamara. 1991. Effect of cropping history on soil populations of *Bipolaris sorokiniana* and common root rot of wheat. Aust. J. Agric. Res. 42:779-790.

Wildermuth, G.B., R.B. McNamara, and J.S. Quick. 2001. Crown depth and susceptibility to crown rot in wheat. Euphytica 122:397-405.

Wildermuth, G.B., R.D. Tinline, and R.B. McNamara. 1992. Assessment of yield loss caused by common root rot in wheat cultivars in Queensland. Aust. J. Agric. Res. 43:43-58.

Wildermuth, G.B., G.A. Thomas, B.J. Radford, R.B. McNamara, and A. Kelly. 1997. Crown rot and common root rot in wheat grown under different tillage and stubble treatments in southern Queensland, Australia. Soil Tillage Res. 44:211-224.

Windels, C.E., J.A. Lamb, and T.E. Cymbaluk. 1992. Common root rot and yield responses in spring wheat from chloride application to soil in northwestern Minnesota. Plant Dis. 76:908-911.

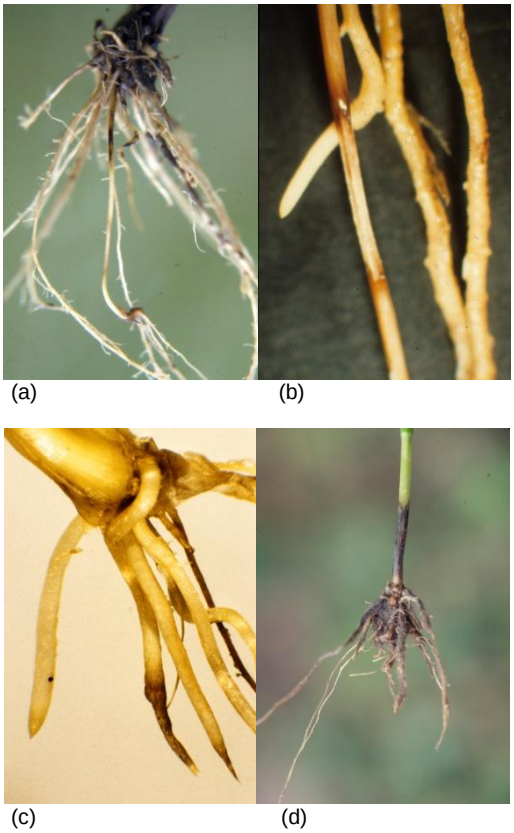
Windels, C.E., and J.V. Wiersma. 1992. Incidence of *Bipolaris* and *Fusarium* on subcrown internodes of spring barley and wheat grown in continuous conservation tillage. Phytopathology 82:699-705.

Wiseman, B.M., S.M. Neate, K.O. Keller, and S.E. Smith. 1996. Suppression of *Rhizoctonia solani* anastomosis group 8 in Australia and its biological nature. Soil Biol. Biochem. 28:727-732.

Zillinsky, F.J. 1983. Common diseases of small grain cereals: A guide to identification. CIMMYT., D.F., Mexico.

CITATION FOR THIS CHAPTER

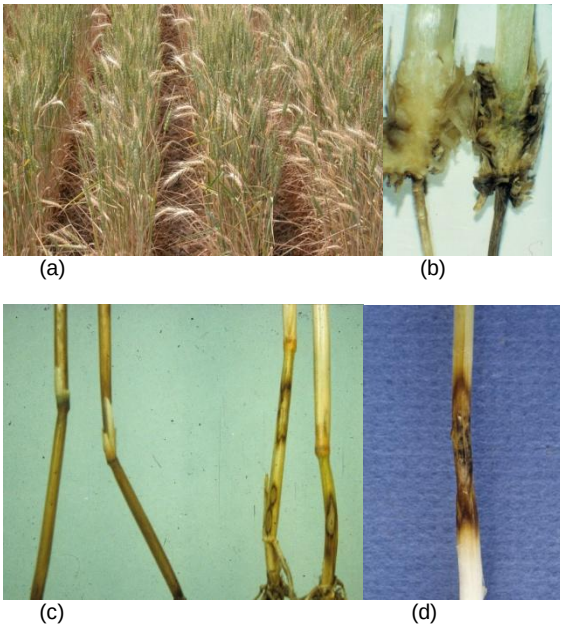
Smiley, R.W., D. Backhouse, P. Lucas, and T.C. Paulitz. 2009. Diseases which challenge global wheat production – root, crown, and culm rots. p. 125-153 *in* Wheat Science and Trade. B.F. Carver (ed.). Wiley-Blackwell, Ames, IA.



(a) (b)

(c) (d)

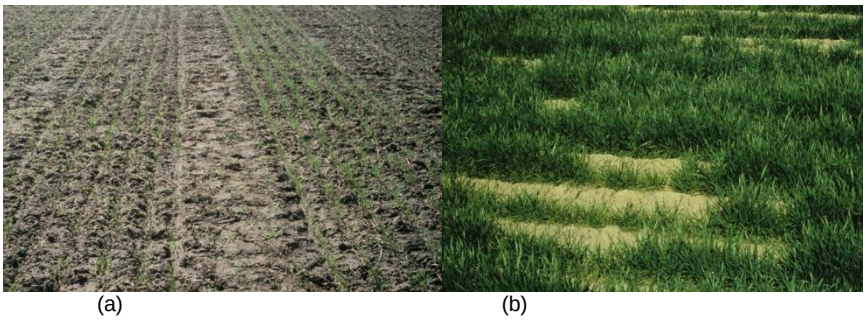
Plate 12 Lesions on subcrown internodes caused by (a) *Bipolaris sorokiniana* and (b) *Fusarium pseudograminearum*, (c) "spear tipping" of roots by *Rhizoctonia solani* AG-8, and (d) blackening of roots and basal stem by *Gaeumannomyces graminis* var. *tritici* . [(a & d) courtesy B.B. Bockus; (b & c) courtesy R.W. Smiley.]



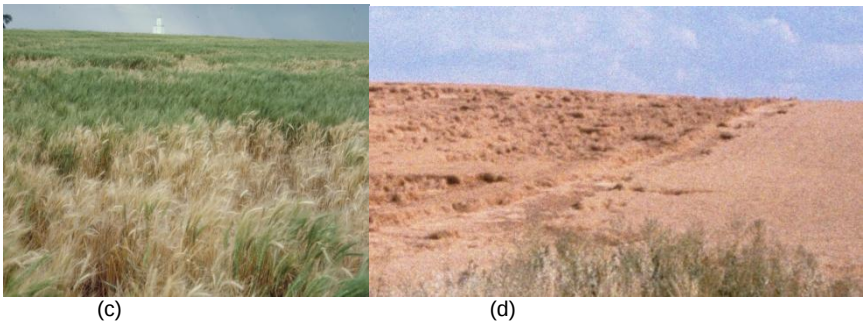
(a) (b)

(c) (d)

Plate 13 (a) Whiteheads caused by multiple root, crown, and culm rotting fungi and insect pests, (b) crown rot and (c, left) uniform browning of basal stem internodes by *Fusarium pseudograminearum*, and lesions of the culm mainstem (c, right) and leaf sheath (d) by *Oculimacula yallundae* ((a-c) courtesy R.W. Smiley; (d) courtesy T.D. Murray.)



(a) (b)



(c) (d)

Plate 14 (a) Improvement of wheat seedling stand by treating seed with metalaxyl (right) to reduce Pythium root-rot and damping-off (left), (b) Rhizoctonia bare patch symptoms in winter wheat during early spring, (c) patches of whiteheads caused by take-all, and (d) lodging of a winter wheat cultivar susceptible to eyespot (left) compared to a cultivar with a gene for resistance (right) [(a,b and d) courtesy R.W. Smiley; (c) courtesy W.W. Willis].

(a) (b)

Plate 15 Winter wheat with leaf stripes (a) and browning of vascular bundles (b) caused by *Cephalosporium gramineum*. (courtesy R.W. Smiley).