Anthracnose of cereals and grasses

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Anthracnose impacts on the health of cereals and grasses worldwide and is caused by a monophyletic group of taxa in the genus *Colletotrichum*. During the past decade there have been important changes in our knowledge of how the grass-associated *Colletotrichum* have evolved, and an increased understanding of the mechanisms by which these fungi engage in hemibiotrophic interactions with their host plants. Several new species of graminicolous *Colletotrichum* have been described from both cool- and warm-season grasses, revealing a pattern of distinctly specialized relationships between these fungi and their hosts. In this review, an overview of the *Colletotrichum* species that cause anthracnose disease in cereals and grasses is provided. The current knowledge of how these organisms interact with and impact their hosts, their evolutionary histories, population processes, and how these factors come together to influence the diversity and ecology of the graminicolous *Colletotrichum* group are highlighed.

Key words: anthracnose, *Colletotrichum*, corn, disease epidemics, endophyte, oats, plant disease, phylogenetics, Poaceae, prairie, sorghum, sugarcane, turfgrass, wheat.

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Introduction

Colletotrichum species cause anthracnose diseases of cereals and grasses worldwide, infesting at least 42 genera of plants in the family *Poaceae* (Table 1). Representative illustrations of disease symptoms caused by these fungi are shown in Fig. 1. Although best known as plant pathogens, naturally occurring endophytic strains of *Colletotrichum* in the species *C. cereale* have recently been reported from grasses in the subfamily Pooideae, including wheat, orchardgrass, Canadian wildrye and smooth brome grass (*Triticum aestivum*, *Dactylis glomerata*, *Elymus canadensis* and *Bromus inermis*; Crouch *et al*., 2009c).

In their sexual state, these fungi are members of the ascomycete genus *Glomerella*, although with the exception of *C. falcatum*, teleomorphs are rare or absent, and have never been reported from the field. Like all members of the genus, graminicolous *Colletotrichum* produce erumpent acervuli containing heavily melanized, sterile hairs called setae (Fig. 2), a character that distinguishes the group from the morphologically similar genus *Gloeosporium*. *Colletotrichum* species associated with graminicolous plants have falcate-shaped asexual conidia, a morphological feature that is also shared by a number of *Colletotrichum* found on dicotyledonous and non-graminicolous monocotyledonous hosts, including *C. dematium*, *C. capsici*, *C. circinans* and *C. trichellum* (Sutton, 1980; Shenoy *et al*., 2007). With the exception of a single report of the fusoid-spored *C. acutatum* inhabiting *Calamagrostis* x *acutifolia* as a non-pathogen or the rare pathogenic association of *C. gloeosporioides* with local landraces of sorghum, only *Colletotrichum* with falcate conidia have been identified from grass hosts (Mathur *et al*., 2002; Crouch and Inguagiato, 2009). Conversely, only one grass-associated species, *C. graminicola*, has been reported from a host other than graminicolous plants, causing keratitis in humans (Ritterband *et al*., 1997). Given the frequent inaccurate application of the name *C. graminicola* prior to 2006 (see taxonomy

Table 1. The 14 species of *Colletotrichum* associated with cereal and grass hosts, with host range and *Glomerella* teleomorphs.

Species	Teleomorph	Host genera
C. axonopodi	–	*Axonopus*
C. caudatum	–	*Andropogon, Agropyron, Aristida, Bothriochloa, Cymbopogon, Eragrostis, Eremochloa, Eulaliopsis, Koeleria, Imperata, Roetboellia, Schizachyrium, Setaria, Sorghastrum, Sporobolus, Zoysia*
C. cereale	–	*Agrostis, Avena, Bromus, Calamagrostis, Dactylis, Elymus, Festuca, Hierochloe, Holcus, Hordeum, Lolium, Poa, Polypogon, Triticum*
C. echinochloae	–	*Echinochloa*
C. eleusines	–	*Eleusines*
C. falcatum	*G. tucamensis*	*Saccharum*
C. graminicola	*G. graminicola*	*Zea*
C. hanaui	–	*Digitaria*
C. jacksonii	–	*Echinochloa*
C. miscanthi	–	*Miscanthus*
C. navitas	–	*Panicum*
C. nicholsonii	–	*Paspalum*
C. paspali	–	*Paspalum*
C. sublineola	*G. sorghi*[1]	*Sorghum, Eremochloa*

[1]The teleomorph of *C. sublineola* has been generated *in vitro* by Vaillancourt and Hanau (1992) but is not yet formally described. A forthcoming paper is in preparation, describing the fungus as *Glomerella sorghi* (Lisa Vaillancourt, pers. comm.).

section, below), it is currently unknown whether the keratitis-associated strain of the fungus is a true member of *C. graminicola*.

Although anthracnose diseases in cereals and grasses have been well-studied since the beginning of the 20th century, recent research has shown that the group is larger and more complex than previously expected. Periodic outbreaks of anthracnose disease in important cereal and grass crops over the course of the past 70 years attests to the adaptive potential of these organisms, but additional research is needed in this area if we are to understand the underlying mechanisms responsible for disease emergence. In this review, we present an overview of the graminicolous *Colletotrichum* group, emphasizing recent findings about its evolution and phylogeny, biology and host-pathogen interactions, novel anthracnose disease outbreaks in new grass systems, and practical considerations for the molecular discrimination of these fungi. We also provide a synthesis of several older studies for the first time, providing connections that would not have been possible prior to the current advances in taxonomy and nomenclature for the group. The review is organized in three major sections, in order to explore some of the major themes that underlie the diversity of these fungi: (1) life cycle and biology; (2) species boundaries and taxonomy; and (3) ecology of the graminicolous *Colletotrichum*, as influenced by the environment, physiological races, population dynamics and disease.

I. Biology and lifecycle of the graminicolous *Colletotrichum*

Much of what is known concerning the biology, epidemiology and the interaction of *Colletotrichum* species with cereal and grass hosts is drawn from just two species: *C. graminicola* and *C. sublineola* which cause anthracnose pathogens of corn (*Zea mays*) and sorghum, respectively. In particular, beginning with the onset of a devastating corn anthracnose epidemic during the early 1970s, *C. graminicola* has emerged as a model system for the biology and epidemiology for the entire graminicolous *Colletotrichum* group. *Colletotrichum graminicola* also holds the distinction of being the first *Colletotrichum* species with a sequenced genome (http://www.broadinstitute.org/annotation/genome/colletotrichum_group/MultiHome.html). Bergstrom and Nicholson (1999, 2000) provided comprehensive

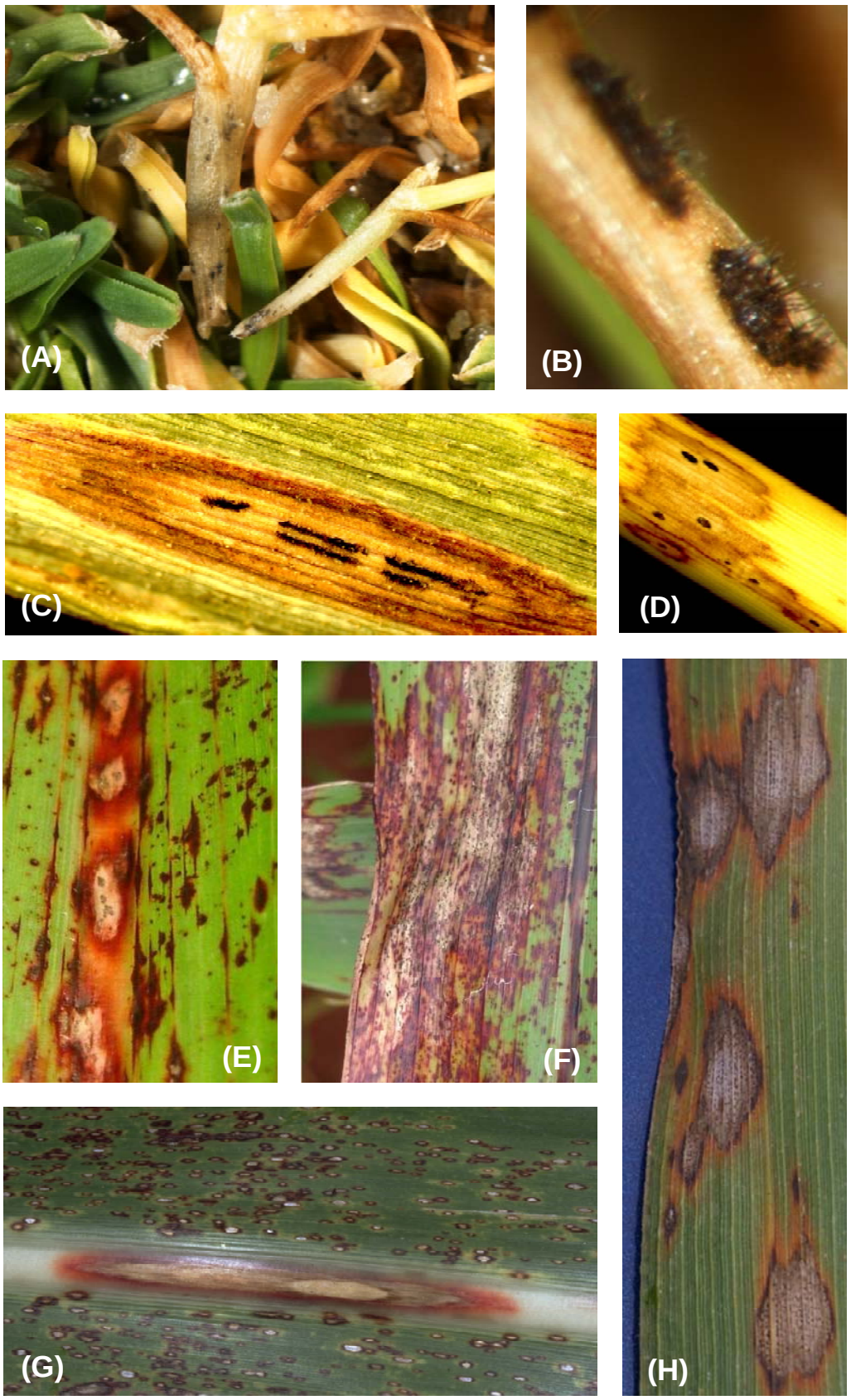

Fig. 1. Anthracnose disease symptoms on cereals and grasses caused by *Colletotrichum* species. **A, B.** anthracnose basal stem rot of *Poa annua* turf; **B** shows a close-up of setae emerging from acervuli on plant surface; **C, D.** *C. navitas* on *Panicum virgatum* leaves and stem; **E, F.** *C. sublineola* on *Sorghum bicolor* leaves; **G.** Red rot of *Saccharum officinarum* caused by *C. falcatum*; and **H.** *C. caudatum* on *Sorghastrum nutans*. Photos courtesy of John Inguagiato (**A, B**); Ester Buiate (**E, F**); Eric McKenzie (**G**) and Gary Bergstrom (**H**).

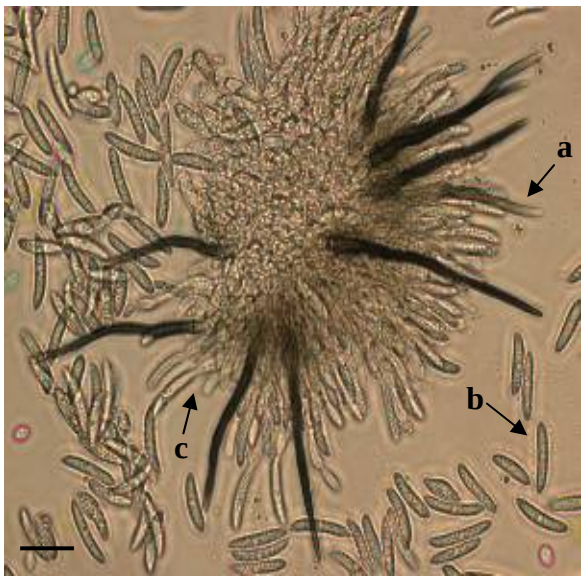

Fig. 2. *Colletotrichum cereale* grown on potato dextrose agar. Shown: diagnostic black setae emerging from a central mass of hyphae, surrounded by conidia which have been produced from conidiogenous cells. Bar = 25 μm.

reviews of *C. graminicola* as a pathogen of corn in (1999, 2000) provided comprehensive reviews of *C. graminicola* as a pathogen of corn in The following section provides an overview of the biology and lifecycle of the graminicolous *Colletotrichum*, updated to incorporate research that has been conducted during the past ten years and not included in earlier reviews. Unless otherwise noted, details of the life cycle and infection process are derived from experiments conducted using the *C. graminicola*/corn pathosystem. A summary of the graminicolous *Colletotrichum* life cycle is illustrated in Fig. 3. For reviews of the *Colletotrichum* infection process, refer to Perfect *et al*., 1999 and Latunde-Dada, 2001.

Overwintering and primary innoculum sources

Colletotrichum graminicola, *C. sublineolum* and *C. falcatum* overwinter in soil and decaying plant residues as mycelium, acervuli, melanized hyphopodia, sclerotia and microsclerotia (Singh and Singh, 1982; Casela and Frederiksen, 1993; Bergstrom and Nicholson 1999; Sukno *et al*., 2008). Little is known about the overwintering cycle of *C. cereale*, but there is data to show that the fungus can maintain dormancy as sclerotia on rhizomes of oat and barley plants (*Avena sativa* and *Hordeum vulgare*; Sanford, 1935; Caglevic, 1960) or overwinter as living mycelia in winter grains (Selby and Manns, 1909). Disease can be initiated from any of these sources; however, *C. graminicola* has been shown to cause more destruction when primary inoculum initiates from saprotrophic residues on the soil surface (Naylor and Leonard, 1977; Casela and Frederiksen, 1993; Lipps, 1983, 1985, 1988). In the absence of decaying plant material, lysing of *C. graminicola* spores and mycelium occurs rapidly due to competition from other fungal soil inhabitants (Vizvary and Warren 1982; Lipps, 1983, 1985). Survival in the soil is heavily dependent on environmental conditions, temperature, and other soil microflora. When ample debris is present, fungal material can effectively overwinter for lengthy periods and provide a source of primary inoculum for the following season. *Colletotrichum sublineola* is capable of surviving on crop debris for 18 months (Casela and Frederiksen, 1993) and *C. graminicola* for 20 months (Naylor and Leonard, 1977). Corn kernels stored at 4°C may also harbor *C. graminicola* for more than three years (Warren, 1977), while *C. sublineola* survives in sorghum seed at room temperature for up to 2.5 years (Mishra and Siradhana, 1957). *Colletotrichum falcatum* is frequently spread through the use of infected canes or seed in propagation, as dormant infections in sugarcane nodes are difficult to detect without expert inspection (Singh, 2008).

Persistance of *C. sublineola* capable of infecting cultivated sorghum has been suggested to occur through infection of wild sorghum species such as johnsongrass (*Sorghum halapense*; Edmunds and Zummo, 1975; Warren, 1986), but Rosewich (1996) showed that populations of the fungus from wild and cultivated sorghum species are genetically differentiated, demonstrating that weedy hosts do not act as a reservoir for cultivated sorghum anthracnose (Gale, 2002). Similarly, Crouch and colleagues (2009c) found significant differentiation between populations of *C. cereale* from cereal crops, prairies, and fine turfgrass, and concluded that non-turfgrasses do not act as alternate hosts or provide a secondary source of innoculum for turfgrass anthracnose disease.

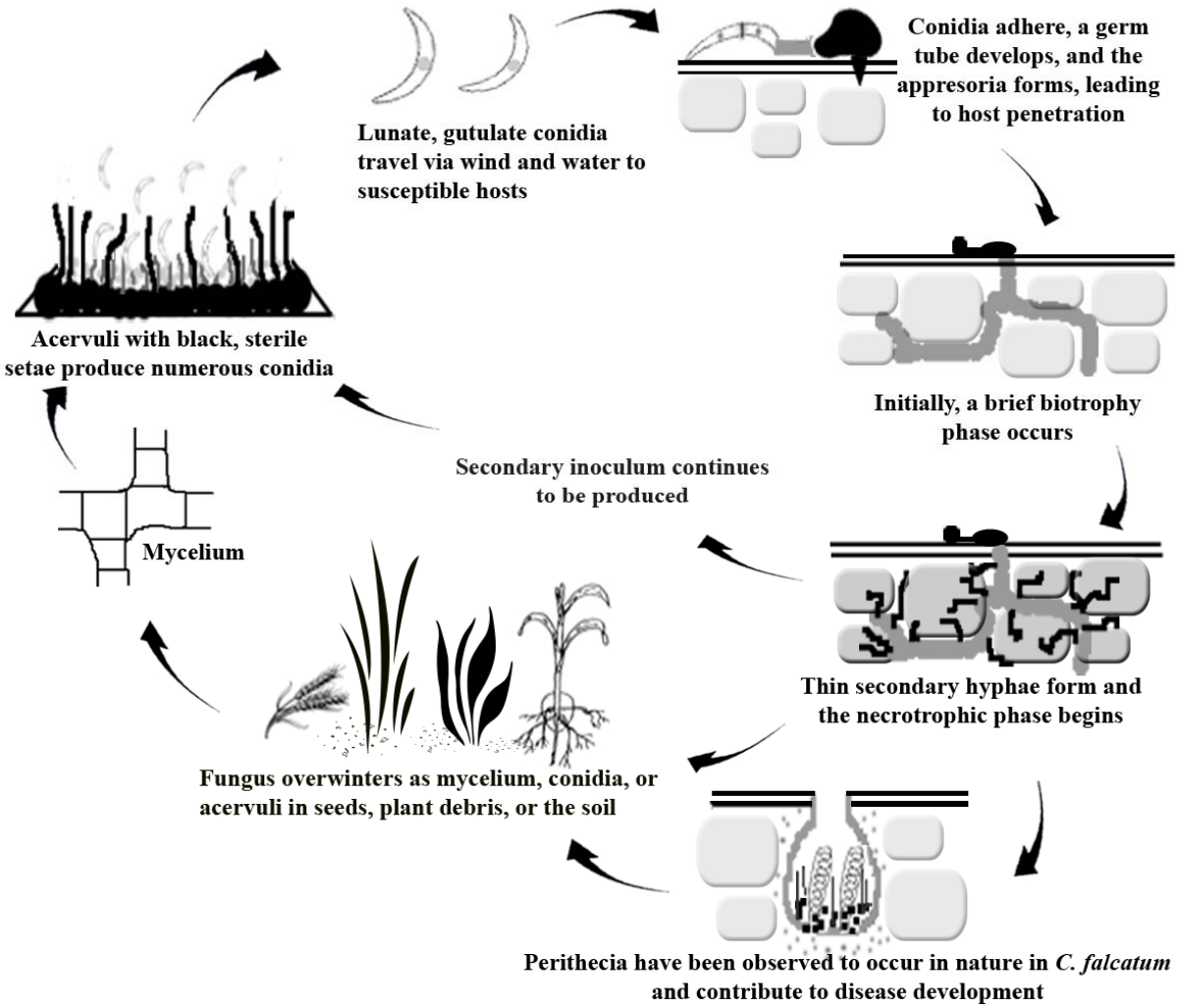

Fig. 3. Lifecycle of the graminicolous *Colletotrichum*.

Dissemination

Transmission of *C. cereale*, *C. graminicola*, *C. falcatum* and *C. sublineola* from plant to plant is known to occur through the transfer of falcate-shaped, asexual conidia, a process which is primarily dependent on water splash and blowing raindrops (Bergstrom and Nicholson, 1999; Viswanathan and Samiyappan, 2001; Murphy *et al*., 2008). Aerial transfer of dessicated *C. falcatum* and *C. graminicola* spore masses has been reported by some sources, but has not been experimentally verified (Nicholson and Moraes, 1980; Bergstrom and Nicholson, 1999; Viswanathan and Samiyappan, 2000). Acervuli are formed erumpently from necrotic plant tissue; these structures produce numerous falcate-shaped conidia blastically from distinct conidoigenous cells (Fig. 2) that serve as a source of secondary inoculum in the disease cycle.

Both *C. graminicola* and *C. sublineola* also produce a second type of asexual spore blastically from hyphae lacking conidiogenous cells: an oval conidia, smaller in size than the falcate conidia (Panaccione *et al*., 1989). In liquid culture, oval conidia are found prior to the production of the falcate conidia, but this observation has not been observed in nature (Panaccione *et al*., 1989, Thomas and Frederiksen 1995). The role of oval conidia in the disease cycle remains unknown, but they have been observed in host lesions, xylem tissue, root epidermal cells and root hairs (Panaccione *et al*. 1989; Vernard and Vaillancourt, 2007; Sukno *et al*., 2008).

Warm, humid weather is conducive for falcate conidia development in *C. cereale* (29-35°C; Danneberger *et al*., 1984) and *C. graminicola* (25°C; Lipps, 1988). Increased light is known to greatly enhance conidia

production by *C. cereale*, *C. falcatum*, *C. graminicola*, *C. navitas* and *C. sublineola* (Bell, 1949; Singh, 1973; Panaccione *et al*., 1989; Rosewich, 1989; Crouch *et al*., 2010; J.A. Crouch, unpublished data), although germination is decreased in *C. falcatum* if it is exposed to high intensity light during sporulation (Singh, 1973). Within the acervulus, conidia are excreted in a water-soluble spore matrix of polysaccharides and proteins that allows spore survival until a susceptible host is present (Nicholson and Moraes, 1980). This mucilaginous matrix contains glycoproteins that provide antidessicant activity, allowing the conidia to survive adverse conditions while associated with the acervuli (Leite and Nicholson, 1992). The matrix also possesses a high affinity for binding phenolic compounds, indicating that these glycoproteins aid in virulence by sequestering toxic phenolics produced on the leaf surface (Nicholson *et al*., 1986, 1989). In addition, the conidial mucilage contains laccase, an enzyme capable of oxidizing phenol (Anderson and Nicholson, 1996). Plants inoculated with spores lacking this matrix often develop lesions at a much later time than those suspended in the mucilage (Bergstrom and Nicholson, 1999), demonstrating the importance of the spore matrix in pathogenicity.

Water also plays an important role in the infection process, in that the mucilaginous conidial matrix also contains the water-soluble compound mycosporine-alanine at high concentration (4 mM), a powerful inhibitor of germination (Leite and Nicholson, 1992). Rain splash reduces the concentration of the inhibitor, and germination can proceed once mycosporine-alanine levels fall below 0.5 mM (Leite and Nicholson, 1992). Water also uniquely facilitates infection of sugarcane by *C. falcatum.* During monsoon seasons when sugarcane plants are partially submerged, *C. falcatum* conidia are placed in physical proximity with nodes, providing an optimal point for germination and infection by the fungus (Viswanathan and Samiyappan, 2000).

Attachment and penetration of the host

Following conidia dissemination and contact with the rigidity of a susceptible host or hydrophobic surface, conidia adhere within minutes of contact (Mercure *et al*., 1994). Conidia and conidial germlings produce an extracellular matrix composed of mannose and heavily glycosylated glycoproteins that permit adhesion to take place on hydrophobic surfaces (Sugui *et al*., 1998). Fastening of conidia to host leaves prevent the spores from being washed away by rain, allowing germ tube formation and increased disease development (Sugui *et al*., 1998, Mercure *et al*., 1994).

Approximately six hours after adhesion, conidia of *C. graminicola* begin to germinate (Mercure *et al*., 1994). Germination can be induced by both hydrophobic substrates and host surface rigidity (Chaky *et al*., 2001). The conidia swell, and mitotic division of the nuclus occurs, followed by the formation of a septum that serves to separate the two daughter nuclei (Politis and Wheeler, 1973); this process has also been documented during the infection of barley plants by *C. cereale* (Skoropad, 1967). From one of the two cells, a germ tube is produced (Politis and Wheeler, 1973). In *C. cereale*, *C. graminicola* and *C. sublineola*, germ tube development leads to the formation of a globose or pear-shaped, heavily melanized appressoria within 24 hours after contact with the plant; these structures are filled with lipid bodies, mitochondria, polyribosomes, and glycogen granules (Skoropad, 1967; Politis and Wheeler, 1973; Kozar and Netolitzky, 1978; Wharton *et al*., 2001; Mims and Vaillancourt, 2002). The appressoria adhere to host tissue by means of hemicelluloses, making a semi-permanent attachment (Lapp and Skoropad, 1978). In order for appressoria to form, germ tubes of *C. graminicola* require more than 4µm of continuous contact with a host (Apoga *et al*., 2004) and temperatures between 15-20°C (Skoropad, 1967).

Following appressorium formation and adhesion, a penetration peg forms near the base of the appressorium. This structure is surrounded by an electron-lucent layer in *C. graminicola* and *C. sublineola* (Politis and Wheeler, 1973, Wharton *et al*., 2000; Mims and Vaillancourt, 2002). When temperatures reach 25-30°C, turgor pressure builds within the appressoria and penetration of the cuticle by the penetration peg occurs (Skoropad, 1967). Turgor pressure within the appressoria is enormous (17 micronewtons), providing

sufficient physical force for the fungus to mechanically penetrate the underlying host cell (Bechinger *et al*., 1999). Melanization of the appressoria is known to play an important role in the development of turgor pressure during penetration (Kubo and Furusawa, 1991). Host penetration by *C. graminicola* has been observed within 24-36 hours of the conidia coming into contact with the plant surface (Mims and Vaillancourt, 2002), and 42 hours in the *C. sublineola*/sorghum interaction (Wharton *et al*., 2000). As host plants and conidia mature, adhesion rates drop, most likely a factor of differing leaf topography in more mature plants and decreased conidium viability (Mercure *et al*., 1994).

In both sorghum and corn, the host responds to the fungus by forming papillae between the plasmalemma and the host cell wall to prevent fungal penetration and expansion (Wharton *et al*., 2000; Mims and Vaillancourt, 2002). Typically, these papillae are not successful at halting entrance of the penetration peg, but may house toxic phenols and peroxidases that aid in preventing fungal spread (Cadena-Gomez, 1987). In the case of *C. sublineola* infection, sorghum plants form vesicle-like structures near the infection peg that produce numerous amount of phytoalexins that function in a similar way (Snyder *et al*., 1991). Pre-penetration host responses by sorghum to *C. sublineola* include the accumulation of H_2O_2, phytoalexins and hydroxyproline-rich glycoproteins (Basavaraju *et al*., 2009).

Indirect penetration may also occur through wounds, and in the case of *C. graminicola*, actually results in more efficient tissue colonization by the fungus (Venard and Vallancourt, 2007). Quite commonly, corn may become easily infected with *C. graminicola* after entry and feeding of the stalk-boring European corn borer, *Ostrinia nubilalis* (Bergstrom and Nicholson, 1999). Other forms of wounding, such as mechanical damage and hail, are likely to increase susceptibility to anthracnose, but must be evaluated individually as wounding does not always facilitate the disease process. For example, in the association between *C. cereale* and annual bluegrass turf (*Poa annua*), quantitative measures of disease severity show that wounding of the host plant through processes such as verticutting, rolling, and compaction do not enhance the infection process and may even decrease symptoms (Inguagiato *et al*., 2008, 2009; Roberts, 2009).

Post-penetration*: *Intracellular hemibiotrophic infection of the host

Both *C. graminicola* and *C. sublineola* employ an intracellular hemibiotrophic strategy during the infection process, in common with several other species in the genus *Colletotrichum* (reviewed in (Perfect *et al*., 1999). There are also indications that *C. cereale*, *C. falcatum* and *C. navitas* are hemibiotrophic, but additional study is required in these systems (Edgerton and Carvajal, 1944; Hsiang and Goodwin, 2001; Crouch *et al*., 2010). After penetration of the host plant by the fungus, a short period of biotrophy occurs, during which the fungus grows between plant cell walls and plasma membranes without entering the cell or inducing specific host defense responses (Wharton and Julian, 1996). The biotrophic phase is brief. Approximately 24 hours after penetration by *C. sublineola* (Wharton *et al*., 2000) and less than 12 hours after penetration by *C. graminicola* (Mims and Vaillancourt, 2002), the host plant cells originally invaded by the fungus begin to degrade and primary hyphae start to expand and enter nearby cells. Primary hyphae along with haustoria-like structures (Münch *et al*., 2008), invade epidermal and schlerenchyma cells, but do not rupture the plasma membrane or enter mesophyll cells (Wharton *et al*., 2001; Mims and Vaillancourt, 2002). Infected plants show no symptoms at this stage, possibly because the fungus is masking its invasion by deacetylating the chitin on exposed hyphae and secreting proteins that may hinder the host's response (Münch *et al*., 2008).

The necrotrophic infection phase occurs when secondary hyphae begin to ramify throughout host tissue. Secondary hyphae are distinctively thinner than primary hyphae. In *C. sublineola*, necrotrophy begins 66 hours after penetration by *C. sublineola* (Wharton *et al*., 2001) and 48-72 hours after penetration by *C. graminicola* (Mims and Vaillancourt, 2002). At this point, the fungus begins to produce various phytotoxins and depolymerases that begin degrading tissue (Münch *et al*., 2000). Host tissue rapidly turns necrotic, and it is at this

point that acervuli and dense hyphae can now be observed on the plant surface (Wharton *et al*., 2000). Characteristic setae can be seen protruding from acervuli along with conidiophores, bearing diagnostic falcate conidia (Edgerton and Carvajal, 1944).

The extent of correlation between the well-studied lifecycles of *C. graminicola* and *C. sublineola* and the twelve other species in the graminicolous group is currently undetermined, with only fragments of the process understood for the majority of these organisms. Little research in this area has been conducted for other graminicolous species, even those that have been taxonomically known for almost a century (*e.g*., *C. caudatum, C. falcatum*), likely reflecting the economic importance of the most understudied taxa. The general infection process of *C. cereale* using detached leaves has been described through inoculations of turfgrass hosts (Khan and Hsiang, 2003) and barley (Skoropad, 1967), showing that infection by *C. cereale* proceeds in a manner similar to that of *C. graminicola*. Further experiments using living hosts will be required to fully elucidate the *C. cereale*/grass interaction, as the defense responses utilized by living plants against hemibiotrophic *Colletotrichum* are not reflected in senescing detached leaves (Liu *et al*., 2007). It has also been observed that the timing of events in the *C. graminicola*/corn infection process vary based on whether plant tissue was living or detached (Mims and Vaillancourt, 2002; Politis and Wheeler, 1973).

Sexual reproduction and its role in the graminicolous Colletotrichum lifecycle

Of the 14 graminicolous *Colletotrichum* species, the teleomorph has been characterized from only three: *C. falcatum*, *C. graminicola* and *C. sublineola* (Carvajal and Edgerton, 1944; Arx and Muller, 1954; Politis, 1975; Vaillancourt and Hanau, 1992), and of these, only *G. tucamensis*, the sexual state of *C. falcatum*, has been described outside of the laboratory. Even in these cases the teleomorph is not linked to the anamorph and so the linkage may be suspect. Although the physical absence of the sexual state indicates that the teleomorph plays little or no role in the lifecycle of these fungi, research of physiological races and genetic diversity has shown that recombination is a factor in the ecology of *C. cereale*, *C. falcatum* and *C. sublineola* (Duttamajumder *et al*., 1990; Souza-Paccola *et al*., 2003; Crouch *et al*., 2006, 2008a, 2009c; but see Gale, 2002; for additional discussion of this topic, refer to the section on populations and races below). In addition, the signature of repeat-induced point (RIP) mutation was identified from the genome of *C. cereale* (Crouch *et al*., 2008a,b), a genome defense mechanism that occurs only during sexual reproduction (Cambareri *et al*., 1991). RIP mutated sequences have also been observed in the genome sequence of *C. graminicola* (L. Vaillancourt, pers. comm.). These data indicate that a more thorough investigation of sexual reproduction during the lifecycle of the graminicolous *Colletotrichum* should be undertaken.

The genetic control underlying sexual development in the genus *Colletotrichum* does not conform to any known system known in the fungal kingdom. Both *C. falcatum* and *C. graminicola* (*G. graminicola*) are essentially homothallic, able to complete the sexual cycle independently (Arx and Muller, 1954; Politis, 1975; Vaillancourt and Hanaui, 1991). However, *G. graminicola* perithecia are produced in greater abundance when outcrossing occurs, and many strains are strictly heterothallic (Vaillancourt and Hanaui, 1991; Vaillancourt *et al*., 2000). Furthermore, it is quite common for homothallic *C. graminicola* to produce heterothallic offspring (Vaillancourt *et al*., 2000).

The teleomorph of *C. sublineola* has only been generated through outcrossing and therefore may be strictly heterothallic (Vaillancourt and Hanaui, 1992). Crosses between *C. graminicola* and *C. sublineola* do not produce sexual progeny, supporting the distinction between these two species (Vaillancourt and Hanaui, 1992).

The enigma of observing both homothallic and heterothallic strains of *C. graminicola* is not yet fully resolved. Investigations of *C. graminicola* fertility through crosses of two obligate outcrossing parental strains (M1.001 and M5.001) led to the exclusive production of heterothallic progeny, consistent with true heterothallism (Vaillancourt *et al*., 2000). The segregation patterns of these progeny illustrated an unsual pheneomenom not previously

known in the ascomycete fungi, where two unlinked loci controlled compatibility (Vaillancourt *et al*., 2000). For an extended review of cross fertility and mating in the genus *Colletotrichum*, refer to Vaillancourt *et al*. (2000).

In the bipolar mating system typically used by ascomycetes to regulate sexual compatibility between individuals, mating typically can only occur when both alleles of the *Mat1* gene, termed idiomorphs because of their dissimilar sequences, are present (*Mat1-1* and *Mat1-2*). In compatible heterothallic matings, two different strains, each bearing one of the two idiomorphs, are required. In homothallic interactions, a single fungal strain carries copies of both idiomorphs, either linked at the *Mat1* locus, or, less commonly, in another area of the genome at a second locus *Mat2* (*e.g*., *Aspergillus nidulans*; Galagan *et al*., 2005). Using this paradigm to develop degenerate primers at highly conserved regions of the two idiomorphs, molecular approaches have been used to elucidate the mechanism underlying control of the graminicolous *Colletotrichum* sexual cycle. In both the original experiments using *C. graminicola* and subsequent surveys of *C. cereale* and *C. sublineola*, only the conserved HMG-box of the *Mat1-2* has been identified (Vaillancourt *et al*., 2000; Chen *et al*., 2002; Du *et al*., 2005; Crouch *et al*., 2006; Moriwaki and Tsukiboshi, 2009). To date, the conserved alpha domain of the *Mat1-1* idiomorph has not been detected in any isolate of *Colletotrichum* through degenerate PCR or Southern blot analysis, even in homothallic strains of *C. graminicola* (Vaillancourt *et al*., 2000) or in more distantly related species such as *C. acutatum*, *C. gloeosporioides* and *C. lindemuthianum* (Du *et al*., 2005, Rodríguez-Guerra, *et al*., 2005; García-Serrano *et al*., 2008). The ubiquitous presence of the *Mat1-2* idiomorph at the *Colletotrichum* mating type locus is consistent with the *C. graminicola* genetic data, and further supports the hypothesis that the regulation of mating in the genus *Colletotrichum* involves a second gene or genes situated at a different locus. Not all available data is in agreement with this theory, with inheritance patterns produced through crosses of *C. gloeosporioides* strains suggestive of a single locus with multiple alleles exerting control over compatibility (Cisar and TeBeest, 1999). Thus, for the moment, the mechanism(s) utilized to regulation of sexual development in the genus *Colletotrichum* remains an unsolved question.

II. Taxonomy, evolution and species determination in the graminicolous *Colletotrichum*

The distinction between species boundaries in the graminicolous *Colletotrichum* has long been problematic for the group, confounding efforts to understand the biology of these organisms and, by extension, control the disease they cause in cereals and grasses. The taxonomy of the graminicolous group has been widely studied during the past several years, as reported in a series of papers combining multilocus molecular phylogenetics, morphological characters and host range association (Crouch *et al*., 2005; Crouch *et al*, 2006; Crouch, 2008; Crouch *et al*., 2009a,b,c; Crouch *et al*. 2010; Du *et al*., 2005; Moriwaki and Tsukiboshi, 2009). At the time this review was written, 14 species of *Colletotrichum* described in association with 42 grass genera are accepted (Table 1). For an overview of valid names for the graminicolous group, refer to Hyde *et al*., (2009).

Morphology, host range, and molecular data for the diagnosis of species boundaries

Prior the application of molecular phylogenetic methods, the taxonomy and nomenclature of *Colletotrichum* were well known as inadequate to describe the organisms that comprised the genus (reviewed in Cannon *et al*., 2000). As summarized by Sutton (1980, 1992), *Colletotrichum* morphological structures and cultural features provide few characters informative for taxonomic purposes, and the rarity and/or absence of the teleomorph in most populations largely precludes the use of sexual structures for this purpose. In the graminicolous group, the majority of taxa are morphologically cryptic. Ten of the 14 currently accepted graminicolous species were described as phylogenetic species diagnosed through monophyly (Crouch *et al*., 2006, 2009b, 2010; Moriwaki and Tsukiboshi, 2009). Fig. 4 illustrates the phylogeny of the graminicolous

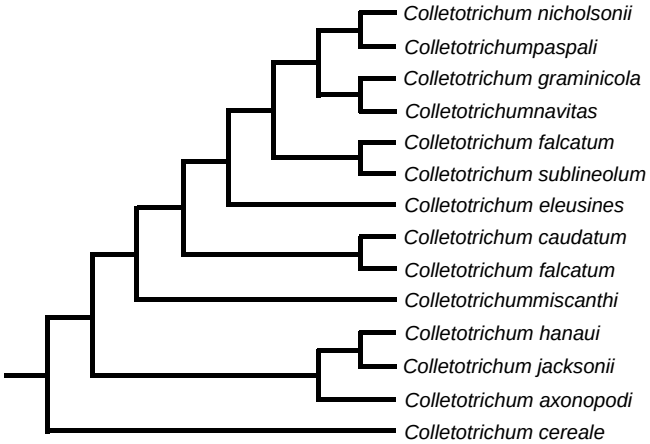

Fig. 4. Cladogram illustrating the evolutionary history of the graminicolous *Colletotrichum* group. Tree topology is adapted from the multilocus phylogenies reported in Crouch *et al*., 2009b and 2010.

Colletotrichum group.

Only two morphological characters have been shown capable of broadly diagnosing species in the graminicolous group, and even these two characters provide limited information. The first character, conidia shape, is capable of distinguishing two taxa from other species in the group: *C. caudatum* possesses a unique appendage on one end of the conidia and *C. navitas* conidia are comparatively larger than other species in the group, possessing a distinctive hockey-stick shape (Fig. 2). Hyphal appressoria, or hyphopodia, are the second character used for species discrimination, with *C. graminicola sensu stricto* Sutton exhibiting exceptionally large and irregularly shaped hyphopodia that have been used since the 1960s to discriminate *C. graminicola* from all other graminicolous *Colletotrichum* (Sutton, 1968), although some overlap in this character has been observed between *C. graminicola* and *C. sublineola* (Crouch *et al*., 2006, 2009b). By mapping hyphopodia shape and size against phylogenetic trees, it was determined that this character does not reflect the evolutionary history of the group, with hyphopodial size and shape subject to convergent evolution (Crouch *et al*., 2009a).

Host range has some utility for the diagnosis of species in the graminicolous group, especially for *C. cereale* (Pooid grasses), *C. eleusines* (*Eleusine* sp.), *C. graminicola* (corn), *C. hanaui* (crabgrass; *Digitaria* sp.), *C. miscanthi* (*Miscanthus sinensis*) and *C. navitas* (switchgrass; *Panicum virgatum*) (Crouch *et al*., 2009b). Definition of other species within the graminicolous group using host range criteria may be less reliable due to shared hosts for multiple species (e.g.; *C. nicholsonii* and *C. paspali* on *Paspalum* sp.) or multiple hosts for some species (*e.g.*; *C. caudatum*, *C. sublineola*). Isolates of *Colletotrichum* found in association with grass hosts not part of the 42 known grass host genera listed in Table 1 should be referred to as *Colletotrichum* sp. until systematic research can be undertaken.

Historical perspective*: *The "group species" concept in Colletotrichum taxonomy

The first report of *Colletotrichum* pathogenic to grasses was made for the taxon currently known as *C. graminicola*. The fungus was originally described as *Dicladium graminicolum* in 1852 from diseased corn and barnyard grass (*Echinochloa crus-galli*) plants (Cesati, 1852). Subsequent reports of *Colletotrichum* from indiangrass (*Sorghastrum nutans*), sugarcane, several cool-season grasses including wheat, oats, and smooth brome, and sorghum resulted in the establishment of *C. caudatum*, *C. falcatum*, *C. cereale* and *C. sublineola*, respectively (Went, 1893; Selby and Manns, 1909; Kabát & Bubák, 1913); these morphologically defined species have been upheld through recent molecular systematic research (see Hyde *et al*., 2009 for review). Additional graminicolous *Colletotrichum* species were erected between 1852–1914, but these taxa, along with *C. cereale*, were synonymized by Wilson as *C. graminicola* (Wilson, 1914) due to similar morphology and common grass host range. The next major advance in the systematics of the graminicolous *Colletotrichum* took place in 1957, when von Arx (1957) synonomized all 35 graminicolous species as *C. graminicola* in an attempt to unify the newly characterized teleomorph, *Glomerella tucamensis*, with the presumed anamorphic states. Sutton's morphological studies during the 1960s began to slowly break apart the "group species" concept of *C. graminicola sensu* Arx, reestablishing *C. caudatum*, *C. falcatum* and *C. sublineola* as species distinct from *C. graminicola* (Sutton, 1965, 1966, 1968). Importantly, Sutton (1966) found that the appressoria produced from hyphae were large and

distinctly shaped in isolates of *Colletotrichum* from corn, resulting in the proposal to limit the application of the name *C. graminicola* to *Colletotrichum* associated with this host, in accordance with his examination of the lectotype material for the species. The contraction of *C. graminicola sensu* Sutton has been repeatedly upheld through subsequent biochemical, physiological, morphological and molecular study (Le Beau, 1950; Dale, 1963; Baxter *et al*., 1983; Jamil and Nicholson, 1991; Vaillancourt and Hanau, 1991, 1992; Sherriff *et al*., 1995; Sreenivasiprasad *et al*., 1996; Hsiang and Good-win, 2001; Moriwaki *et al*., 2002, Du *et al*., 2005; Crouch *et al*., 2005, 2006, 2008a,b, 2009a,b,c; Moriwaki and Tsukiboshi, 2009). A distinct teleomorph for *C. graminicola* (*G. graminicola*) was described within 10 years of Sutton's work, and shown to be distinct from *G. tucamensis*, the teleomorph of *C. falcatum* (Politis and Wheeler, 1972; Politis, 1975).

Sutton's treatment of the graminicolous *Colletotrichum*, and in particular, his finding that *C. graminicola* is limited to corn hosts, left the status of unknown numbers of *Colletotrichum* associated with grasses in an indeterminate state. Sutton (1980, 1992) acknowledged that additional work would be required to address these unnamed fungi, but with the limited morphology available, left further resolution of the group for future researchers. As a result, the name *C. graminicola* was continually applied to describe any *Colletotrichum* associated with grass hosts other than sugarcane, sorghum or indiangrass for the next four decades.

Interest in taxonomic and nomenclatural issues in the graminicolous group was renewed at the beginning of the 21st century with the onset of damaging turfgrass anthracnose disease outbreaks on annual bluegrass and creeping bentgrass (*Poa annua* and *Agrostis stolonifera*) golf course greens that began in the mid-1990s. The causal organims was at that time referred to as *C. graminicola* (Smiley *et al*., 2005). Initial research using genetic markers and ITS gene trees to analyze small populations supported the morphological distinction between *C. graminicola* from corn and populations of *Colletotrichum* isolated from these diseased turfgrass hosts (Backman *et al*., 1999; Browning *et al*., 1999; Hsiang and Goodwin, 2001; Crouch *et al*., 2005). Crouch and colleagues (2006) adopted a multi-locus phylogenetic approach to define species in the graminicolous group through evolutionary history rather than morphological criteria, resurrecting *C. cereale* as a taxa associated with grasses of the Pooideae, including the turfgrass pathogens and endophytic inhabitants of numerous cool-season grasses such as wheat, oats and orchardgrass (Crouch *et al*., 2009c). Later, eight additional new phylogenetic species were described from warm-season (C4 physiology) grasses through genealogical concordance of molecular phylogenies (Crouch *et al*., 2009b, 2010; Moriwaki and Tsukiboshi, 2009). Given the high level of correspondence of fungal species boundaries with grass host range for the majority of the graminicolous taxa (Crouch *et al*., 2009c), it is likely that additional species will be named and described as further grass hosts are studied.

The relationship of the graminicolous Colletotrichum to other members of the genus

Phylogenetic trees constructed using multilocus sequence data demonstrated that the graminicolous *Colletotrichum* make up a monophyletic group (Crouch *et al*. 2009a,c), consistent with the groups shared falcate-shaped conidial morphology. The monophyly of the graminicolous taxa indicates that the association of these fungi with grass hosts occurred just once during the evolution of *Colletotrichum*, but studies including a more comprehensive sampling of other members of the genus are still needed to test this prediction. In particular, additional work is needed to clarify the relationship of the graminicolous *Colletotrichum* relative to other falcate-spored species associated with non-graminicolous hosts. A possible common evolutionary origin between *C. cereale* and isolates of two falcate-spored species associated with non-grass hosts, *C. dematium* and *C. truncatum*, has been reported (Crouch *et al*., 2009a). Because the tree topologies were constructed from ITS sequences alone, the dataset was insufficient to strongly support the relationship between *C. cereale* and *C. dematium*/*C. truncatum* without corroboration from additional sequence data (Crouch *et al*., 2009a).

In many systematic studies of the graminicolous group performed to date, the closely related taxon *C. acutatum* has been used to root the phylogenetic trees. *C. acutatum* is a fusoid-spored species of *Colletotichum* shown to possess a wide host range of dicot hosts, including many important fruit crops (Than *et al*., 2008), but is now being shown to be a species complex (Shivas and Tan, 2009). The use of *C. acutatum* as an outgroup taxa to the graminicolous group has been shown to provide good quality, largely unambiguous nucleotide alignments without incurring loss of informative variable regions (Crouch *et al*., 2006). ITS gene trees show that the graminicolous group and *C. acutatum* are likely sister taxa (Sreenivasaprasad *et al*., 1996; Moriwaki *et al*., 2002; Du *et al*., 2005; Crouch *et al*., 2006), but further study using additional sequence data from multiple loci is needed to convincingly support this predicted relationship.

Evolutionary relationships between species of graminicolous Colletotrichum

In the graminicolous group, two major subgroups have been identified through molecular phylogenetic study, reflecting a divergence between the species that are associated with cool-season hosts (C3) and those associated with warm-season hosts (C4). The C3 lineage is comprised of a single species – *C. cereale* – a wide host range taxa found in association with 14 host genera, and responsible for anthracnose disease in turfgrass, wheat, rye, oats, barley and orchardgrass (Crouch *et al*. 2006, 2009c). In contrast, the C4 group is subdivided into 13 species, most known from narrow host ranges (Crouch *et al*., 2009a, c, 2010; Moriwaki and Tsukiboshi, 2009). With the exception of *C. caudatum* and *C. sublineola*, the C4 taxa studied to date have only been found in association with single host plant species. Given the diversity observed from strains of *C. caudatum*, it is possible that this species could be further subdivided, but epitypification to stabilize the nomenclature and additional extensive sampling of this understudied taxon is required before this determination can be made.

III. Ecology of the graminicolous *Colletotrichum*, as influenced by environment, physiological races, population dynamics and disease

Although the 14 species of *Colletotrichum* known from grass and cereal hosts share a common ancestry, and in many instances, are morphologically indistinguishable from one another, there are additional characteristics that serve to discriminate between them. Specifically, differences between the graminicolous species are evident in the way in which they interact with and adapt to their environments, the hosts they associate with, and how these fungi influence the fitness of their hosts.

The original discovery of race-cultivar specialization between fungi and host plants was made in the genus *Colletotrichum* through studies of the bean anthracnose pathogen *C. lindemuthianum* (Barrus, 1911). In the graminicolous group, the first reports of host-specificity are from studies of *C. falcatum* infecting sugarcane in 1920 (Edgerton and Moreland, 1920) and later from *C. cereale* infecting cereal crops by Sanford (1935), Bruehl (1948), Bell (1950), who all noted difficulties inoculating hosts with strains isolated from different cereal cultivars or species. Similarly, much has been written about the physiological specialization of the sorghum and sugarcane pathogens *C. falcatum* and *C. sublineola*; however, for these fungi, races are well-established through defined differentials (Ferreira and Casela, 1986; Ali and Warren, 1987; Cardwell *et al*., 1989; Pande *et al*., 1991; Casela and Fredriksen, 1994; Casela *et al*., 1996, 2000, 2004; Minnatullah and Kumar, 2005; Rao and Patro, 2005; Suman *et al*., 2005; Valèrio *et al*., 2005; Moore *et al*., 2008). In the following section, we provide an overview of the diversity of the four well-studied graminicolous species: *C. cereale*, *C. falcatum*, *C. graminicola* and *C. sublineola*, emphasizing the interaction of these fungi with their hosts and how this exchange impacts both organisms. Not surprisingly, all four of these fungal species are well-known pathogens of important crops, although *C. cereale* has also been identified as an endophyte of several species of cool-season grasses (Crouch *et al*., 2009c). The remaining nine species of the graminicolous *Colletotrichum* are poorly studied, likely because these fungi have been of little

economic importance, with the majority lumped into the group taxa *C. graminicola sensu* Arx until recently (Crouch *et al*., 2009b). As a result, these species are not surveyed in this section. It is worth noting, however, that several of these poorly studied graminicolous species, including *C. axonopodi*, *C. caudatum*, *C. hanaui* and *C. jacksonii*, are pathogens of undesirable, invasive weed grasses (Crouch *et al*., 2009a). Because of the high degree of inter- and intra-specific physiological specialization present in many of the graminicolous *Colletotrichum*, these fungi could potentially find application in the biological control of invasive grass species, including barnyard grass, crabgrass, cogongrass (*Imperata cylindrica*), and carpetgrass (*Axonopus affinis*). For example, experimental studies show that *C. jacksonii* (described by its former name, *C. graminicola*) is effective at killing barnyard grass in rice fields (Yang *et al*., 2000). Suggestions for the use of graminicolous *Colletotrichum* species with broad host ranges as biocontrol agents may prove less successful, as valuable non-target crop plants may also be impacted by the deployment of these organisms as herbicides. Proposals to control the spread of cogongrass with *C. caudatum* in Australia (Caunter, 1996) and the use of *C. sublineola* to control johnsongrass (Chiang *et al*., 1989) should be carefully evaluated to ensure adequate target specificity.

North American anthracnose epidemics leads to the establishment of Colletotrichum graminicola as a model organism

Anthracnose disease of corn caused by *C. graminicola* was first described by Cesati as *Dicladium graminicola* (1852). Although corn anthracnose was observed throughout the 20th century (see Sprague, 1950 or Farr and Rossman, 2009 for check-lists of incidence), *C. graminicola* was primarily studied due to its taxonomic significance, not because the fungus was overly important as a plant pathogen. This view of *C. graminicola* changed during the 1970s, when devastating outbreaks of corn anthracnose caused by *C. graminicola* unexpectedly began to sweep across the north-central and eastern regions of the USA, resulting in epidemic levels of disease (reviewed in Bergstrom and Nicholson, 1999, 2000). These epidemics persisted throughout the 1990s, and were initially so severe in some regions that sweet corn production was completely eliminated within two years of the disease outbreaks (Warren *et al*., 1973). These epidemics spurred an explosion of fundamental research in the pathosystem from groups led by Nicholson and Hanaui; and knowledge gained from these studies still provides the foundation for much of what is currently known about the biology and lifecycle of the graminicolous *Colletotrichum*. Today, corn anthracnose is well controlled in the USA through the use of resistant cultivars, but may still be problematic in developing countries. Bergstrom and Nicholson (1999) suggested possible reasons for the sudden North American outbreaks, including changes in the environment (*i.e*., cultivation practices), shifts in host genotypes, or the appearance of more virulent fungal strains or races; however, the underlying cause of these outbreaks has never been determined.

Colletotrichum graminicola can infect corn during several phases of plant growth, resulting in either seedling blight, leaf blight, or stalk rot (Bergstrom and Nicholson, 1999). Seedling blight may occur so rapidly in susceptible cultivars that death can occur, but most plants are capable of developing faster than the fungus and merely develop concentric oval lesions on infected leaf tissue (Bergstrom and Nicholson, 1999). Older plant tissue is less likely to escape damage. Infection of leaves by *C. graminicola* leads to the formation of chlorotic lesions within 1-2 days (Venard and Vaillancourt, 2007). Lesions become necrotic and coalesce upon the invasion of parenchyma cells by *C. graminicola* (Venard and Vaillancourt, 2007). Erumpent acervuli form in this degrading tissue, producing copious amounts of conidia when the environment is moist (Venard and Vaillancourt, 2007). Low-light conditions (Schall *et al*., 1980), and warmer temperatures (~30°C) (Leonard and Thompson, 1975) favor lesion expansion and the formation of acervuli in the lesion center. Lesions may spread to the corn stalks, facilitated primarily through hyphal growth (Venard and Vaillancourt, 2007; Venard *et al*., 2008), eventually causing the plants to lodge and develop a black pigmentation (Berstrom and Nicholson, 1999). Secondary lesions on stalks may also occur,

unattached to leaf lesions or primary stalk lesions. These secondary lesions may occur through infection of the tissue by oval conidia, but conclusive evidence to support this association is still required (Venard *et al*., 2008). Infection of root tissue is also possible and can lead to systemic spread throughout the host without causing widespread disease symptoms (Sukno *et al*., 2008).

Little is known about the ecology and evolution of *C. graminicola*, with questions about populations, races, and the role of endophytes almost entirely unstudied. Nicholson and Warren (1981) provided an account of physiological races of *C. graminicola*, but little research has been published since then, likely because the fungus is no longer destructive in corn crops in the USA On the genome scale, Rollins (1996) showed that *C. graminicola* chromosomes are highly polymorphic, with large differences found in overall genome size, individual chromosome sizes and the presence and/or absence of small chromosomes. Phylogenetic studies of the graminicolous *Colletotrichum* also provided a glimpse of the molecular variation present in populations of *C. graminicola* using nucleotide sequence data. In this research, two divergent lineages separated *C. graminicola* strains from the western hemisphere (USA and Brazil) from strains of the fungus originating from the eastern hemisphere (Japan, The Netherlands), but sampling was insufficient to be conclusive (Crouch *et al*., 2009c). In the clade derived from the western hemisphere, separate sub-clades correspondent to different geographic locations in the USA (Indiana, Missouri, Kentucky) were also present, but similarly, larger samples are required to evaluate these indications of population subdivision according to geographic derivation (Crouch *et al*., 2009c).

Despite the fact that *C. graminicola* has only been observed from nature interacting with corn hosts as a pathogen, it seems likely that the fungus may also be able to survive as an endophyte, but information about non-pathogenic strains in natural systems is lacking. Because *C. graminicola* is actively studied as a model for the hemibiotrophic infection of plants by fungal pathogens, details about endophytic strains are beginning to emerge from experimental research. Mims and Vaillancourt (2002) investigated the interaction between a non-pathogenic mutant strain of *C. graminicola* generated through restriction enzyme-mediated mutagenesis (Thon *et al*., 2002). This research showed that the cytology of infection was identical in mutant and wild-type strains until 48 to 72 hours after inoculation, when the wild-type strain switched to necrotrophic growth, and the mutant persisted in the host tissue biotrophically. In these experiments, the endophytic mutant was found to persist in the corn host asymptomatically for three weeks (Mims and Vaillancourt, 2002). Similarly, Sukno *et al*., (2008) documented the asymptomatic vascular movement of *C. graminicola* from root tissue into above-ground plant parts, indicating a possible role for roots as sites of non-pathogenic, endophytic symbiosis.

Red rot disease of sugarcane caused by Colletotrichum falcatum

Red rot disease of sugarcane, caused by *C. falcatum*, is one of the oldest known diseases of sugarcane, with the first scientific reports made in 1893 from Indonesia and in 1901 from India (Went, 1893; Barber, 1901), although much earlier indications of the disease can be found in Buddhist literature (reviewed in Viswanathan and Samiyappan, 2001). The first incidence of red rot disease in the USA was reported from a plantation in the Orleans parish of Louisiana in 1909 (Edgerton, 1910). Although in the USA the disease is not problematic, red rot continues to persist as one of the most destructive diseases of sugarcane in other regions of the world, especially in India, but also in countries such as Bangladesh, Pakistan and Taiwan (Viswanathan and Samiyappan, 2001; Singh, 2008; Subhani *et al*., 2008). An estimated 35 million farmers in India rely on sugarcane farming as a source of income, with millions more dependent on it as a source of food; thus, losses from red rot can be particularly devastating in this country (Singh, 2008). Although the disease was once limited to the subtropical regions of India, epidemics of red rot are now problematic throughout the entire country, with losses ranging from 15-20% in moderate seasons to 100% in severe seasons (reviewed in Singh, 2008). The reader is referred to the recent reviews of Singh (2008) and Viswanathan and

Samiyappan (2000) for a comprehesive overview of red rot caused by *C. falcatum* and the management of this disease in sugarcane crops.

Symptoms of the disease (Fig. 1) begin early in the growing season as minute reddish-brown lesions that form around cane nodes and progress into dark brown, heavily sporulating lesions (Singh, 2008). Although unsightly, the real destruction comes from damage to the pith. Inside the canes, reddish-brown decaying pith marked with white transverse bands can be found, often accompanied by a fermenting stench (Singh, 2008). Nodes can become completely rotted in severely diseased plants.

Physiological specialization of *C. falcatum* was first documented in 1920 in the USA (Edgerton and Moreland, 1920), with the formation of newly adapted races resulting in the failure of several important sugarcane varieties worldwide (reviewed in Singh, 2008). Ten physiological races have been recently documented across different regions of India using host differentials (Satyavir *et al*., 2000); but it is unknown how these races are geographically distributed outside of India; nor has it been established how race composition may have fluctuated throughout the past century. There are also reports in the older literature about differential pathogenicity exhibited by different morphological variants of *C. falcatum*. Following the first red rot epidemics of subtropical India, several researchers found that isolates with light-colored pigmentation in culture were extremely pathogenic, and the most common form of the fungus found in epidemic areas. In areas where red rot disease was uncommon, dark-coloured, poorly sporulating, less virulent strains were the dominant form of *C. falcatum* (Chona and Srivastava, 1960).

Red rot disease is thought to be caused primarily by the asexual state of the fungus, but the sexual morph, *G. tucamensis*, has been observed in natural infections of sugarcane (Chona and Bajaj, 1953; Mishra, 1957), distinguishing this fungus from other taxa in the graminicolous *Colletotrichum* group. Because *C. falcatum* has been observed to reproduce sexually in nature, this process may have played a major role in generating diversity in populations of this pathogen, leading to the continual development of new races able to overcome resistance. There is also evidence indicating that the exchange of genetic material by *C. falcatum* in nature, either through sexual recombination or some other mechanism such as conidial anastamosis tubes or fusion, may be an environmentally dependent process, reliant upon the presence of standing water. Waterlogging of sugarcane during rainy monsoon seasons (July to September in India) predisposes the plant to infection by *C. falcatum* (Viswanathan and Samiyappan, 2000), at least in part through stress-induced physiological changes (Papplis and Katsanos, 1965). Waterlogging is an unusual environmental feature in the graminicolous *Colletotrichum* group, and is only found in the *C. falcatum*/sugarcane pathosystem. Floating conidia are brought into direct contact with nodal regions where they are able to easily germinate and infect the host plant. Of greater importance is the observation that water-logged conditions favor the fusion of germinating conidia (Duttamajumder *et al*., 1990). Strains isolated from these post-fusion events have been shown to be more virulent than parental types, leading to the breakdown of resistance (Duttamajumder *et al*., 1990). Additional research is needed if we are to understand the relationship between standing water and the sexual cycle for *C. falcatum*, as well as the impact of this phenomenon on the ecology and evolution of this fungus.

Data generated from molecular phylogenetic studies show that there is not just one, but two morphologically indistinguishable phylogenetic species of *Colletotrichum* responsible for sugarcane red rot (Crouch *et al*, 2009a, c; Fig. 4). These two phylogenetic species were unquestionably distinct from each other, with each species grouped as part of two separate monophyletic lineages in the graminicolous clade. One lineage, comprised of *C. falcatum* isolates from Brazil and Japan, was sister to *C. sublineola*, pathogen of sorghum. A second *C. falcatum* lineage, most closely related to *C. caudatum* and *C. miscanthi*, comprised of isolates from Brazil, Japan and Nigeria, (Crouch *et al*, 2009a,c). Thus, it appears the divergence may not be geographically derived, as both of these phylogenetic species were found in Brazil and Japan. The taxonomy of these two species is currently unresolved, with the description of the new species postponed

until the nomenclature of *C. falcatum* could be addressed through epitypification with a specimen from the type sugarcane field, which is in progress (Crouch *et al*., 2009a; K.D. Hyde, pers. comm.).

Anthracnose disease in sorghum caused by Colletotrichum sublineola

Anthracnose disease caused by *C. sublineolum* occurs throughout sorghum growing areas worldwide, and is prevalent throughout Argentina, China, Brazil, Hawaii, India, Pakistan, eastern Africa and other central American countries (reviewed in Mathur *et al*., 2002). Sorghum anthracnose caused by *C. gloeosporioides* is also observed, although infrequently, and limited to localized outbreaks from land races (Mathur *et al*., 2000). In warm, humid climates such as those found in the tropics and subtropics, anthracnose is one of the most damaging diseases of sorghum, affecting all parts of the host plant. Susceptible cultivars have been reported to experience losses as high as 50% (Harris *et al*., 1964; Thomas *et al*., 1996). Infection of leaves, the plant part most commonly affected by *C. sublineola*, results in small (<5 mm) elliptical red lesions with tan centers (Fig. 1); lesions may eventually coalesce to cover large areas of the leaf surface. Severe infections may lead to defoliation and death for susceptible cultivars. When the fungus infects the stalks, the disease is known as red rot.

Two *formae speciales* of *C. sublineola* have been described based upon disease symptoms and cultural morphology: var. '*isolatum*' from Nigeria and var. '*zonatum*' from India (Rajasab and Ramalingan, 1981; Alawode *et al*, 1983), but it is unknown whether these subgroups are also evolutionarily distinct, as they have never been studied on the molecular level. Cultural characteristics of *C. sublineola* are highly variable (Pande *et al*., 1991; Casela and Frederickson, 1994; Rao *et al*., 1998; Mathur *et al*., 2002), but do not correspond with physiological races or genotype (Rao *et al*., 1998). Due to the taxonomic problems associated with the graminicolous *Colletotrichum* taxa during the 20th century, *C. sublineola* may also be found listed as the causal agent of anthracnose disease in several hosts, including *Axonopus affinis*, *Digitaria* sp., and *Eleusine indica*, but molecular phylogenetic analysis shows that the fungus is not associated with any of these plants (Crouch *et al*., 2009a). The host range of *C. sublineola* is limited to species of sorghum, including cultivated sorghums such as *S. bicolour*, and weedy relatives such as johnsongrass and maicillo landrace cultivars (Rosewich, 1996; Crouch *et al*., 2009a,c). Recently, however, *C. sublineola* has also been identified and confirmed as the causal agent of anthracnose disease outbreaks in centipedegrass lawns in the southern USA (*Eremochloa ophiuroides*) (J.A. Crouch and M. Tomaso-Peterson, unpublished data).

In areas of the world where the fungus has been studied – the USA, Hondorus and Zambia – the sexual state of this fungus is physically absent from natural populations, an observation that is supported through population genetic data (Rosewich, 1996). Nevertheless, the fungus is physically capable of generating sexual progeny in the laboratory through heterothallic crosses (Vaillancourt and Hanau, 1992). Laboratory studies also show that genetic recombination may occur through parasex (Souza-Paccola *et al*., 2003). It remains to be determined whether recombination occurs by either mechanism in locales where *C. sublineola* has not been well studied, especially where the disease is particularly devastating and new race formation is common.

Colletotrichum sublineola shows little variability at the genetic level, with the structuring of all populations examined to date clonal, regardless of geographic derivation (Rosewich, 1996). On the local scale, using a panel of seven RFLP markers, Rosewich *et al*. (1998) evaluated a population from a sorghum disease nursery in the USA over a three-year period. From a sample of 411 isolates, the population was found to be extremely stable, with the sample comprised of only nine haplotypes, and dominated by a single haplotype over a period of three years. From larger samples, long distance dispersal of high frequency *C. sublineola* haplotypes has been documented, with a single common haplotype observed in the southern USA, Zambia and Hondorus (Rosewich, 1996). Regional adaptation is also known to occur in the southern USA (Rosewich, 1996). Similarly, within the context of systematic study of the graminicolous *Colletotrichum* using nucleotide sequen-

ces from a four-gene dataset, seven samples of *C. sublineola* isolated from diverse locations (Burkina Fasso, Japan, South Africa, USA) between 1957 and 2005 were found to exhibit little variability (Crouch *et al*., 2009a,c). In this research, there were indications that the species might be differentiated according to geography, in that the Japanese isolates formed a sub-grouping separate from other isolates, and a Texas isolate from johnsongrass was almost indistinguishable from a sorghum isolate from Texas, but sample sizes were insufficient to draw any definitive conclusions. This observation that sorghum and johnsongrass isolates are so closely related is in conflict with the findings of Rosewich, who found that johnsongrass isolates of *C. sublineola* from Texas are more closely allied with isolates from Honduran maicillo landrace cultivars than they are to Texas strains of the fungus isolated from sorghum (Rosewich, 1996). Experimental inoculations, where most johnsongrass isolates of *C. sublineola* were shown to be only weakly virulent on most domesticated sorghums (Cardwell, 1989; Guthrie, 1993), support the later view, where physiological specialization exists between genetically distinct host-derived populations of the fungus.

Despite the presumed lack of sexual reproduction, physiological race formation is very common for this fungus, to the extent that multiple *C. sublineola* pathotypes may be isolated from an individual host plant (Rao *et al*., 1998; Mathur *et al*., 2002). Pathotypes have also been found to vary in individual fields from one year to the next, and multiple new races may develop very quickly (Moore *et al*., 2008). Numerous *C. sublineola* races have been documented since 1965 from both cultivated sorghum and johnsongrass (*e.g*., Ali and Warren, 1987; Cardwell, 1989; Cardwell *et al*., 1989; Pande *et al*., 1991; Moore *et al*., 2008; reviewed in Mathur *et al*., 2002), but there is currently no internationally standardized set of differentials in the sorghum anthracnose research community (Mathur *et al*., 2002), with the result that a comprehensive understanding of race variation for this fungus is incomplete. It remains an open question whether new *C. sublineola* races might be generated and dispersed over long distances from one or more recombinant disease foci in unsampled areas, a phenomenon that is known to occur in clonal populations of the fungus (Rosewich *et al*., 1996). Future work specifically designed to address these questions, in combination with the use of more variable markers such as microsatellites or SNPs may prove valuable for determining how *C. sublineola* populations are distributed within important sorghum growing regions and on a global basis.

Colletotrichum cereale: *Pathogen and endophyte of cool-season cereals, turfgrasses, field and forage grasses*

Colletotrichum cereale has a remarkably broad host range of C3 grasses, especially when one considers the high degree of host specialization corresponding to species boundaries in the C4 clade. This fungus has been identified from 14 genera of grasses (Table 1; reviewed in Hyde *et al*., 2009), living either as a pathogen, or as an endophyte (Crouch *et al*., 2009c). *Colletotrichum cereale* is also unique among the graminicolous *Colletotrichum* species because of its association with cool-season grass hosts of the subfamily Pooideae (Crouch *et al*., 2009c). The evolutionary divergence observed between *C. cereale* and the 13 species of *Colletotrichum* inhabiting warm-season grasses is not surprising, as the Pooid grasses inhabited by *C. cereale* differ significantly from their warm-season counterparts in many ways, including their ecology, phenology, physiology, photosynthetic rates and cycles (C3 vs. C4), leaf anatomy and vascular system architecture (Gibson, 2009).

Colletotrichum cereale has a long history of inciting anthracnose disease outbreaks in several cultivated cereal crops and grasses, with reports spanning much of the 20th century. With the exception of recent, ongoing turfgrass anthracnose epidemic, outbreaks of anthracnose caused by this fungus have been of relatively short duration, lasting only a few years, often in geographically limited areas. In cereal crops such as wheat, oats, and barley, anthracnose outbreaks occurred intermittently during the first 50 years of the previous century. Notable cereal anthracnose outbreaks occurred in North American oat crops between 1933-1951; in wheat crops between 1911-1918 and 1951; and in rye crops between 1918-1926, 1961-1963 and during the 1940's (Selby and

Manns, 1909; Güssow, 1917; Sanford, 1933, 1935; Rosen, 1938, 1946, 1947, 1949; Vestal, 1944; Bruehl and Dickson, 1950; Goto and Moore, 1952; Luke and Sechler, 1963). Recent outbreaks of anthracnose from cereal crops have not been reported in North America, but occasional descriptions have come from elsewhere in the world (Kemp *et al*., 1991, Leyva-Mir *et al*., 2004; Iftikhar *et al*., 2008). Anthracnose disease of cool-season forage grasses such as orchardgrass, smooth brome and meadowgrass (*Festuca pratensis*) have also been infrequently observed (Hardison, 1943; Valleau, 1950; Bello and Sisterna, 1987). Today, populations of *C. cereale* are still found in cereal crops and forage grasses, as well as in native prairie grasses, but the fungus survives as an endophyte in these environments, inhabiting the host plant without causing anthracnose disease symptoms (Crouch *et al*., 2009c).

Outbreaks of anthracnose disease in turfgrasses cultivated as golf course greens – particularly annual bluegrass and creeping bentgrass (*Agrostis stolonifera*) – have been known to occasionally cause damage to turfgrasses in North America, the U.K. and other locations for most of the 20th century. During the past decade, however, turfgrass anthracnose has increased in incidence and severity across North America, the United Kingdom, Europe, and Australia to become one of the most destructive diseases of golf course turf (Murphy *et al*., 2008; Mann and Newell, 2005).

Symptoms of anthracnose disease caused by *C. cereale* vary depending on the host plant infected. On cereals, including rye, oats and wheat, the plant develops black streaks resembling scabs, extending along the culms, sheaths, roots, seed heads, stems, and panicles (Selby and Manns, 1909). These somewhat elliptical lesions resemble leaf blotch symptoms, but upon closer examination, acervuli are observed in parallel rows between the leaf veins once the plant matures (Bruehl and Dickson, 1950). Lesions and chlorotic areas may sometimes be absent, with entire leaves turning reddish-brown and dying from the tip back (Sanford, 1935). Infection by *C. cereale* in cereal grains often leads to smaller, shrunken seed heads that result in a diminished yield (Selby and Manns, 1909). Frequently, anthracnose stricken cereal hosts will ripen prematurely and produce shrivelled grain, or none at all (Bell, 1949). In severely infected rye fields, yield can be reduced by as much as 2/3 (Selby and Manns, 1909). At present, cereal anthracnose diseases are not actively managed, with standard cultural practices such as adequate fertilization, crop rotation and removal of residues sufficient to eliminate disease outbreaks.

Anthracnose disease of cultivated turfgrass species often results in plant death. Anthracnose foliar blight symptoms tend to develop during high temperatures in the summer (Smiley *et al*., 2005), producing yellow or reddish-brown leaf discoloration or, less frequently, oblong leaf lesions. Anthracnose basal rot symptoms can occur at any time during the growing season, with small patches (6-12 mm) of grass beginning to show yellow speckles that lead to large, reddish-brown patches up to several feet in diameter that may eventually die back completely (Smiley *et al*., 2005). When individual plants are examined, stems of diseased tissue can be easily pulled from the crown, revealing a water-soaked, black, rotting crown and stem base from which acervuli form (Fig. 1; Murphy *et al*., 2008). Fungicides are used frequently to control *C. cereale* in fine turf, resulting in rapid adaptation and the development of resistance by the fungus to several fungicide chemistries (Shane and Danneberger 1989; Avila-Adame *et al*., 2003; Crouch *et al*., 2005; Wong and Midland, 2007; Wong *et al*., 2007, 2008). Increasingly, management studies are revealing alternative practices to manage disease and minimize fungicide use (Inguagiato *et al*., 2008, 2009; Murphy *et al*., 2008, Roberts, 2009). Murphy *et al*. (2008) provide a recent overview of turfgrass anthracnose disease management.

Physiological races have never been characterized for *C. cereale* from any pathosystem, although Sanford (1935), Bruehl (1948), Bell (1949) all noted difficulties inoculating hosts with *C. cereale* strains isolated from different cereal cultivars or species. Each of these scientists suspected host-specificity or multiple races; however, formal race typing has never been developed for *C. cereale*, likely because disease outbreaks have been so transient in majority of economically important

systems. In turfgrass, development of a reliable growth chamber inoculation protocol has been problematic (Murphy *et al*., 2008), limiting the examination of race structure, although experimental inoculations are perfomed successfully under field conditions (Inguagiato *et al*., 2008, 2009). Browning *et al*., (1999) noted from greenhouse studies that *C. cereale* isolated from creeping bentgrass hosts were capable of causing disease in both creeping bentgrass and annual bluegrass plants, but that strains isolated from bluegrass only produce disease symptoms on annual bluegrass. In contrast, Backman *et al*. (1999) reported the absence of host-specificity in similar experiments, although for some strains of *C. cereale*, virulence was greater on the host plant from which the fungus was originally isolated.

Molecular research strongly supports intraspecific physiological specialization of *C. cereale* populations. Marker-based analyses of *C. cereale* from turfgrass identified a separation between isolates from creeping bentgrass and annual bluegrass hosts; however, exceptions were observed (Backman *et al*., 1999; Browning *et al*, 1999; Horvath and Vargas, 2004). Subsequent multilocus sequence analysis of populations from several grass hosts – including both pathogenic and endophytic isolates of the fungus from fine turf, prairie and cereals – showed that population structuring is relatively complex for *C. cereale*. The species is comprised of two major subgroups, designated clades "A" and "B" (Crouch *et al*., 2006, 2008a,b, 2009c). Clade A is further subdivided into ten highly differentiated subpopulations. A defining characteristic of the individual *C. cereale* "A" populations is ecosystem-level specialization, with boundaries between populations congruent with environment and sometimes specific hosts (Crouch *et al*., 2009c). In clade A, endophytic *C. cereale* isolated from prairie and cereal crops were shown to cluster separately from turfgrass pathogens. Notably, in this clade, North American endophytic *C. cereale* strains are members of the same populations comprised of endophytic strains collected from Japan, Germany and New Zealand – distinct from North American turfgrass isolates. Three of the ten populations in clade A are drawn from diseased turfgrass: two from annual bluegrass, and one from creeping bentgrass. Thus, it appears that lifestyle and habitat are the key factors defining population boundaries in *C. cereale* clade A, with limited gene flow occurring between the ten populations. In contrast, *C. cereale* clade B comprises both pathogenic strains isolated from turf and ornamental grasses, as well as endophytes from prairie grasses and cereal crops (Crouch *et al*., 2009c). Together, these data provide a possible explanation for the conflicting reports of differential infectivity in the turfgrass pathosystem, although this theory remains to be tested.

Gene flow is predicted to occur between *C. cereale* clade B and many of the individual clade A populations, unifying the two otherwise distinct lineages as a single species (Crouch *et al*., 2006, 2008a,b, 2009c). Although the sexual state of *C. cereale* has never been observed, recombination has been inferred through molecular analyses; with high levels of diversity, reticulate evolutionary relationships and statistical measures of recombination supporting the possibility of genetic exchange occurring in this species, particularly in clade B (Crouch *et al*., 2006, 2008a, 2009c). The signature of RIP mutation identified from several clade B transposons provides evidence of sexual reproduction for the lineage, since RIP mutation process is limited to meiosis (Crouch *et al*., 2009a), but to date, RIP has not been found in *C. cereale* clade A.

Concluding remarks

Future work in the graminicolous *Colletotrichum* group will continue to expand our understanding of the mechanisms that influ-ence the colonization of cereals and grasses by these fungi, and the ecological and evolutionary consequences of these interactions. In particular, the hemibiotrophic infection process employed by *Colletotrichum*, and the factors that control the transition from biotrophic colonization to necrotrophic pathogenesis, is currently an area of intensive research in the genus. It is presently not known what genetic elements are responsible for the shift from hemibiotrophy to necrotrophy, but it is hypothesized that pectolytic enzyme production, as seen in *C. lindemuthianum* and *C. gloeosporioides*, plays a role in the process

(Mims and Vaillancourt, 2002). Additionally, increased disease development has been linked to escalating leaf senescence, which indicates a relationship between host defense responses and the activation of pathogenicity genes, although the signals for this transition are not yet known (Münch *et al*., 2008). Ammonification plays an important role in the pathogenicity of several *Colletotrichum* species, including *C. acutatum*, *C. coccodes* and *C. gloeosporioides* (Prusky *et al*., 2001), and may also act during the shift of graminicolous species from biotrophy to pathogenicity, but this remains to be determined. The recent availability of genome sequences of both *C. graminicola* and *C. higginsianum* (R. O'Connell, pers. comm.), along with methodological developments capable of assaying stage-specific events, including a yeast two-hybrid signal sequence trap, laser microdetection and fluorescence-activated cell sorting (FACS) purification of hyphae from infected plant tissue, offer promising approaches for the identification of genes and effector proteins that are differentially expressed or secreted during the hemibiotrophic infection process of *Colletotrichum* species (Tang *et al*., 2006; Krijger *et al*., 2008; Takahara *et al*., 2009). With the well-resolved phylogeny of the graminicolous *Colletotrichum* now available, scientists will also be able to study the evolution of habitat adaptation, endophytism, pathogenicity and other traits in the group. Given the societal and economic implications of these fungi, it is hoped that continued research of the graminicolous *Colletotrichum* group will ultimately contribute to improved diagnosis and treatment of these important plant pathogens.

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