

# A SHORT REVIEW OF FUSARIUM SECTION LISEOLA: IMPLICATIONS FOR CONIFER SEEDLING PRODUCTION<sup>1</sup>

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## ABSTRACT

Species of *Fusarium* in the section *Liseola* have the same teleomorph (*Gibberella fujikuroi*) and similar anamorphic characteristics, including production of microconidia in chains or false heads borne on mono- or polyphialides. None of the species normally produce chlamydospores. Using anamorphic characteristics to separate taxa is difficult, so species have been delineated based on "mating populations," i.e., production of the ascigerous stage when paired with known testers. Anamorphic species designated as *F. proliferatum* reside in mating populations C and D; all isolates tested from Pacific Northwest conifer seedlings are in mating population D. Some pathogens formerly designated as *F. moniliforme* may actually be *F. proliferatum*; ambiguity can be reconciled using mating population tests and molecular nucleic acid analyses. Based on molecular analyses, *F. oxysporum*, an important pathogen of bareroot forest nurseries, is most closely related to fungi in the section *Liseola*.

## REVIEW

*Fusarium proliferatum* (Matsushima) Nirenberg is one of several taxa placed in the *Fusarium* section *Liseola* (Leslie 1995; Nelson et al. 1983). *Liseola* contains several closely-related anamorphic taxa, all having the teleomorph *Gibberella fujikuroi* (Correll et al. 1989; Joffe 1974; Klittich and Leslie 1992; Kuhlman 1982; Puhalla and Spieth 1985; Tudzynski et al. 1996), sometimes previously called *G. moniliforme* (Snyder and Hansen 1945). Most workers now regard it as *G. fujikuroi* (Klittich and Leslie 1988; Sidhu 1983; Windels 1991). Biological species within *G. fujikuroi* have been designated as "mating populations" based on production of the sexual (ascigenous) stage when isolates are crossed with known testers (Klittich and Leslie 1992; Leslie 1991). From such crosses, several mating populations

have been designated for fungi in *G. fujikuroi* (Klittich and Leslie 1992; Leslie 1991; Xu et al. 1995). Fungi with *F. proliferatum* anamorphs are found in two mating populations: C and D (Leslie 1991; 1995; Xu et al. 1995). All evaluated isolates obtained from conifer seedlings and evaluated belong in mating population D (Leslie, personal communication).

Undoubtedly, some fungi in *Liseola* were previously misidentified based solely on anamorphic characteristics (Leslie 1991, 1995). For example, *F. moniliforme*, a species very commonly isolated from maize (Logreico and Bottalico 1988), sorghum (Jardine and Leslie 1992), corn (Headrick et al. 1990; Lim 1967; Nava Saucedo et al. 1989; Ooka and Kommedahl 1977), figs (Michailides and Morgan 1994; Subbarao and Michailides 1992), asparagus (Elmer and Ferrandino 1992; La Mondia and Elmer 1989), cassava (Msikita et al. 1996) and occasionally from conifer seedlings (Chakravarty and Unestam 1986; Hildebrand 1985; Huang and Kuhlman 1990; Unestam et al. 1987) differs from *F. proliferatum* only on the basis of formation of microconidia exclusively on monophialides (Nelson et al. 1990; Tiedt and Jooste 1988). *F. proliferatum* forms microconidia on either mono- or polyphialides (Fisher et al. 1983; Klittich and Leslie 1992; Leslie 1991; Nelson et al. 1983). Conidiophores may sometimes be difficult to find and some workers have had to reidentify *F. moniliforme* isolates as *F. proliferatum* (Abbas et al. 1988; Elmer 1990; Marasas et al. 1988; Schreuder et al. 1995; Viljoen et al. 1995; Wagih et al. 1989). Because of this ambiguity, several reports designating *F. moniliforme* as the causal agent of a specific plant disease may actually have dealt with *F. proliferatum* or some other member of *Liseola* (Elmer 1991; Logreico et al. 1995; Puhalla and Spieth 1983). Fortunately, future use of the mating population concept should reduce this taxonomic ambiguity (Elmer 1995a; Leslie 1995; Plattner et al. 1996).

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Molecular analyses have simplified isolate differentiation within *Fusarium* section *Liseola*. These analyses usually evaluate nucleic acid similarities and determine if polymorphisms occur at specific sites in either nuclear, ribosomal, or mitochondrial DNA or RNA (Ellis 1988a, 1988b, 1989; Guadet et al. 1989; Leslie and Dickman 1991; Lodolo et al. 1992; Manicom et al. 1987; Marshall and Partridge 1981a, 1981b; Peterson 1991). Depending on the technique, molecular results generally substantiate identifications based on mating populations (Klittich and Leslie 1992; Leslie 1995; Xu et al. 1995). Genetic work with this group of commercially-important fungi will undoubtedly continue to clarify phylogenetic relationships.

As indicated previously, *F. moniliforme* may infect several grain crops (Kingsland and Wernham 1962) and be particularly detrimental because some isolates produce mycotoxins (Abbas et al. 1995; Jardine and Leslie 1992; Marasas et al. 1979, 1986, 1988; Miller et al. 1993; Van Asch et al. 1992). Several mycotoxins have been identified; some may cause serious diseases to mammals (Klittich and Leslie 1989). Likewise, *F. proliferatum* has also been implicated as causing diseases of assorted plants including sorghum, maize, corn, asparagus, gladiolus, and taro (Elmer 1990, 1992, 1995b; Magie 1980; Ocumb and Kommedahl 1994; Schreuder et al. 1995; Wagih et al. 1989; Wall and Cruz 1991 and some isolates produce important mycotoxins (Logreico and Bottalico 1988; Logreico et al. 1995; Marasas et al. 1986; Marin et al. 1995; Van Asch et al. 1992). *F. proliferatum* has also been reported as a biological control agent, controlling grape powdery mildew (Falk et al. 1996).

There are several references of conifer diseases associated with members of the *Fusarium* section *Liseola*. Perhaps the most common is pitch canker disease caused by *F. subglutinans* f. sp. *pini* (Correll et al. 1991; Kuhlman et al. 1978; Storer et al. 1995) which occurs in epidemic proportions in the Southeast U.S. and along the California coast. The pathogen normally causes most damage to older, natural or plantation, trees (Barrows-Broadus 1990; Storer et al. 1995), although damage in nurseries has been recorded (Blakeslee et al. 1981; Rowan 1982; Viljoen et al. 1994). Several other conifer seedling diseases have been attributed either to *F. moniliforme* and *F. proliferatum* (Chakravarty and Unestam 1986; Fraedrich and Miller 1995; Hildebrand 1985; Huang and Kuhlman 1990; James 1991, 1992, 1993, 1995; Unestam et al. 1987; Viljoen et al. 1995).

Experience in Pacific Northwest nurseries indicates that the most common pathogen in the genus *Fusarium* causing root diseases in bareroot nurseries is *F. oxysporum* (James 1996; James et al. 1991, 1996). Experience in container nurseries indicates that *F. oxysporum* is often a major seed-borne pathogen (James 1987; James et al. 1987, 1991) and an important cause of both pre- and post-emergence damping-off (James 1993; James et al. 1987, 1991). However, as the container seedling growth cycle progresses, occurrence of *F. oxysporum* on roots of seedlings decreases, whereas infection by *F. proliferatum* often increases (James et al. 1995). By the time seedling height growth is terminated and seedlings are stressed to initiate bud set and dormancy, *F. proliferatum* commonly colonizes roots of many seedlings (James et al. 1991, 1995). During this period of seedling stress, root disease symptoms may become common on infected seedlings. However, not all infected seedlings display symptoms, even though much of their root systems may be colonized with potentially-pathogenic *F. proliferatum* (James et al. 1995). When non-diseased Douglas-fir seedlings are outplanted on forest sites, *F. proliferatum* tends to gradually decline on root systems; the fungus usually does not readily colonize new roots being formed after outplanting and gradually dies out as "plug" roots become replaced (Dumroese et al. 1993). However, when infected container seedlings are transplanted in nursery soil, especially soil fumigated with general biocides, *F. proliferatum* may become active and contribute to seedling mortality (James 1991, 1995). Introducing infected seedlings into soil that may be naturally suppressive to the pathogen (forest soil) probably results in inactivation and replacement of the pathogen by other mycoflora (Dumroese et al. 1993). However, when introduced into disease-conducive nursery soil, there may be an absence of competing mycoflora (James 1991, 1995).

Occurrence of *F. proliferatum* is rare in nursery soil and on conifer seed. *F. proliferatum* is likely introduced into greenhouses from several inoculum sources. Reused plastic and styrofoam containers are important inoculum sources (Dumroese et al. 1995; James and Woollen 1988; James et al. 1988). However, other sources such as organic debris located within greenhouses or "alternate" hosts located near greenhouses may also provide primary inoculum.

Molecular analyses indicate that isolates of *F. oxysporum* are most closely related to fungi in the section *Liseola* (Donaldson et al. 1995; Ellis 1988a,

1988b; Guadet et al. 1989; Waalwijk et al. 1996). Some workers believe that the teleomorph for *F. oxysporum*, when found, will be a species of *Gibberella* (Guadet et al. 1989). Fungi in both sections *Liseola* and *Elegans* (*F. oxysporum*) share the propensity to colonize cortical cells of conifer seedlings (James et al. 1991). Likewise, they may be present on seedlings that display disease symptoms as well as those lacking symptoms (James et al. 1987, 1991). Occurrence of a wide range of virulence in isolates of *F. oxysporum* has been well documented (Bloomberg 1976; Gordon and Okamoto 1992; James et al. 1989). However, our tests on *F. proliferatum* although limited, indicated that most isolates were capable of eliciting germinant and seedling diseases readily under experimental conditions without the variability of virulence experienced with *F. oxysporum* isolates (James et al. 1997).

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