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Number 1567

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# Blind Seed Disease



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Number 1567

# Blind Seed Disease

Stephen C. Alderman

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## Abstract

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In blind seed disease, unfertilized or developing seed of susceptible grasses are colonized by the fungus *Gloeotinia temulenta*. Infection results in loss of seed germination. About 56 species of grasses are susceptible, including important forage and turf grasses such as ryegrass and tall fescue. The disease occurs in all areas of production of cool season grasses grown for seed. Germination in infected seed samples has been reported as low as 1 percent in New Zealand, 13 percent in the United States, and 50 percent in Great Britain. Blind seed disease continues to periodically plague growers in New Zealand, and a recent reappearance of blind seed in the United States has renewed interest in the disease. This monograph provides a comprehensive review of our understanding of *G. temulenta* and blind seed disease, including host and geographical distribution, taxonomy, biology, and control.

Keywords: Disease management, disease distribution, *Gloeotinia*, grass seed, host range, seed production, seed quality

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## Contents

Introduction and historical overview .....	1
Geographical distribution and host range .....	1
Yield loss and economic impact .....	2
Symptoms .....	3
Causal agent .....	3
Synonymy .....	4
Technical description .....	4
Growth on media .....	6
Similar-looking fungi .....	13
Biology and epidemiology .....	13
Overwintering and production of apothecia .....	13
Production and release of ascospores and primary infection .....	13
Secondary infection .....	13
Disease development and spread .....	13
Histopathology .....	16
Fungal genetics and physiology .....	16
Toxicity .....	16
Disease management .....	17
Disease resistance .....	17
Field location .....	17
Seed source .....	17
Seed treatments .....	17
Planting .....	18
Time of closing .....	18
Nitrogen fertilization .....	18
Stand density .....	19
Fungicide sprays .....	19
Harvest .....	19
Straw residue removal .....	19
Postharvest plowing .....	19
Crop rotation .....	19
Field burning .....	19
Seed cleaning .....	20
Methods for detection and assessment .....	20
Postharvest disease detection and assessment .....	20
Preharvest testing .....	21
Outlook .....	21
References .....	27

## Tables

Table 1. Geographical and host distribution of <i>Gloeotinia temulenta</i> .....	22
Table 2. Relative susceptibility of grass species to <i>Gloeotinia temulenta</i> .....	24





## Introduction and Historical Overview

During the early 1920s, growers of perennial ryegrass (*Lolium perenne* L.) seed in New Zealand were troubled by poor germination of their seed crops. A systematic inquiry initiated in 1923 associated reduced germination with humid conditions during seed development (Foy 1927), but the cause was unknown. By 1926, germination was as low as 19 percent, and in the southern region of New Zealand 95 percent of the seed lots tested had germination of 90 percent or less (Foy 1927). Ungerminable seed had an abnormal appearance characterized by opacity, roughness, and a reddish caryopsis surface (Hyde 1932). These symptoms were difficult to see unless the lemma and palea, which cover the caryopsis, were removed. The difficulty in visual detection of the ungerminable (diseased) seeds led Neill and Hyde (1939) to propose “blind seed” as the common name of the disease.

By 1932, it was apparent that a conidia-producing fungus was associated with affected seeds (Hyde 1932), but it was not until 1937 that pathogenicity of the blind seed fungus (tentatively identified as a *Pullularia* sp.) was established (Hyde 1937). However, another fungus, distinct from *Pullularia*, was also found associated with infected seed. After careful study, this second fungus, not *Pullularia*, was found to cause blind seed disease (Muskett and Calvert 1940, Wilson et al. 1940).

In 1942, after an investigation of the life history of the blind seed fungus, Neill and Hyde (1942) determined that a fungus called *Phialea temulenta* Prill. & Delacr. was identical to the blind seed fungus on ryegrass in New Zealand. *P. temulenta* was previously reported on seed of rye (*Secale cereale* L.) in France in 1891 (Prillieux and Delacroix 1891, 1892b). In 1945, Wilson et al. (1945) reviewed the taxonomic placement of *P. temulenta* and erected a new genus, *Gloeotinia*, to accommodate it. Thus the blind seed fungus became *Gloeotinia temulenta* (Prill. & Delacr.) M. Wilson, Noble, & E.G. Gray.

The effects of blind seed disease on the production of grass seed can be tremendous. Germination in infected seed samples has been as low as 1 percent in New Zealand (Greenall 1943), 13 percent in the United States (Hardison 1945), and 50 percent in Great Britain (Noble and Gray 1945). Blind seed continues to periodically plague growers in New Zealand (Skipp and Hampton 1996), and its recent reappearance in the

United States (Alderman 1996) has renewed interest in the disease here. This monograph provides a comprehensive review of our understanding of *G. temulenta* and blind seed disease, including host and geographical distribution, taxonomy, biology, and control.

## Geographical Distribution and Host Range

The blind seed fungus was first recorded on infected seeds of rye (*Secale cereale* L.) in France in 1891 (Prillieux and Delacroix 1891, Neill and Hyde 1942). Although first reported on rye, its subsequent occurrence on this crop is very rare. Blind seed disease is primarily a problem of forage and turf grasses grown for seed.

Blind seed disease was unknown in Great Britain until after its discovery in New Zealand. However, the connection between blind seed and low germination in ryegrass (*Lolium* sp.) was suspected to be of long standing in Great Britain, since low germination in some years was well known (Calvert and Muskett 1944, 1945). Proof of the long-standing occurrence of blind seed was established when conidia of *G. temulenta* were found among stored seeds from a 1909 ryegrass crop grown in Ireland (Lafferty 1948). The identification of blind seed disease in the United States in 1944 established that the fungus was widely distributed on ryegrass grown for seed, a distribution likely established through the international grass seed trade.

Blind seed has been reported from Australia, including Tasmania, Victoria, and New South Wales (Neill and Hyde 1939, Wade 1949, Anonymous 1955, Wade 1957, Anonymous 1962, McGee 1971a, Munro 1978); Denmark (Noble 1939, Gemmell 1940, Lafferty 1948, Kristensen and Jørgensen 1960); England, including Kent, Sussex, Hereford, and the Isle of Man (Neill and Hyde 1939, Gemmell 1940, Glasscock 1940); Ireland (Gemmell 1940, Lafferty 1948); France (Prillieux and Delacroix 1891, 1892a); The Netherlands (de Tempe 1950, 1966); New Zealand (Gorman 1939; Neill and Hyde 1939; Blair 1947, 1948; Latch 1966; Hampton and Scott 1980a); Northern Ireland (Neill and Hyde 1939; Gemmell 1940; Calvert and Muskett 1944, 1945); Scotland, including Ayrshire and the Shetland Islands (Neill and Hyde 1939, Gemmell 1940, Noble and Gray 1945, Dennis and Gray 1954); Sweden (Neill and Hyde 1939); United States, including

Oregon (Fischer 1944, Hardison 1945, Alderman 1988); and Wales (Neill and Hyde 1939).

Worldwide, 56 host species have been reported as susceptible to *G. temulenta* (table 1). Most hosts are in the subfamily Pooideae, tribes Avenae and Poeae, with heaviest infections reported in the genera *Agrostis*, *Festuca*, *Lolium*, and *Poa* (Hardison 1962) (table 2). *Lolium perenne* is widely recognized as susceptible and has been identified as a host from all countries reporting blind seed disease. In the Triticeae, moderate to heavy infections were observed on *Psathyrostachys*, *Pseudoroegneria*, and *Secale* species. Grasses in the Bromeeae appear less susceptible, with little to no infection observed among species of *Bromus*.

In the United States, blind seed disease was found on species of *Agrostis*, *Aira*, *Alopecurus*, *Bromus*, *Cynosurus*, *Deschampsia*, *Danthonia*, *Festuca*, *Glyceria*, *Hordeum*, *Holcus*, *Lolium*, *Phleum*, and *Poa* (table 1). Despite the susceptibility of many common grasses in the United States, *G. temulenta* has been reported only from Oregon. In New Zealand, blind seed was reported on *Agrostis*, *Cynosurus*, *Festuca*, *Holcus*, *Lolium*, *Poa*, and *Secale cereale*. In Northern Ireland, blind seed was found on *Agrostis*, *Cynosurus*, *Festuca*, *Holcus*, *Lolium*, and *Poa*. Additional host reports include *Calamagrostis* from Germany, *Elytrigia* from Norway, and *Secale* from France and Germany.

Most of these U.S. hosts were reported in a comprehensive host range study by Hardison (1962) (tables 1 and 2). However, there is one discrepancy in the U.S. host range. Fischer (1944) reported *G. temulenta* on *Danthonia californica* Boland (subfamily Arundinoideae, tribe Danthoneae). Hardison (1962), however, did not observe infection on *D. californica* inoculated with *G. temulenta* under natural or artificial conditions. Additional studies are needed to determine all grasses that are susceptible to *G. temulenta* and their relative susceptibility.

## Yield Loss and Economic Impact

In the production of grass seed, loss from blind seed disease occurs through a reduction in germinable seed since infected seeds are ungerminable. In addition, seed lots with germination below certification limits, or below seed contract standards, are of less value and

in some countries are unmarketable. Presence of *G. temulenta* in import seed shipments may result in rejection of the seed by some countries (Halfon-Meiri 1978).

**Australia.** Blind seed was reported to cause few crop failures, although in 1969, 2,400 out of 9,000 acres could not be certified because of blind seed disease (McGee 1971a).

**Denmark.** A low level of blind seed was found in 6 percent of ryegrass samples exported from Denmark to Ireland (Lafferty 1948). A low level of blind seed was also found in 1957 (Kristensen and Jørgensen 1960).

**England.** In 1938 and 1939, germination as low as 50 percent was common in ryegrass (Noble and Gray 1945). In 1940, an average of 26 percent of ryegrass seed from south England was infected (Gemmell 1940).

**The Netherlands.** In 1965, the level of infection with the blind seed fungus ranged from 0 to 94 percent, with an average infection rate of 19.2 percent (de Tempe 1966).

**Scotland.** In 1938–1939, infection of ryegrass seed as great as 50 percent was reported (Gemmell 1940, Noble and Gray 1945). Average infection in samples from Ayrshire was 26.4 percent (Gemmell 1940).

**New Zealand.** Between 1931 and 1934, cost of seed lost to blind seed was estimated at £1,975–4,382 (Gorman 1939). In 1938, average germination of ryegrass was 67–76 percent in Christchurch, Canterbury, and South Canterbury (Hyde 1938b). Greenall (1943) reported germination of ryegrass as low as 1 percent. Greenall also noted that the severity of blind seed disease depended on environmental conditions, and he expected that in South Otago one year in every two or three would be accompanied by poor germination. During 1944–1946, 45–84 percent of samples from the South Island had more than 20 percent blind seed and 10–22 percent of samples had 70–100 percent diseased seed (Blair 1947). Stocks of seed throughout New Zealand had germination below 40 percent—in some lots as low as 5 percent (Osborn 1947). In seed exported from New Zealand to Ireland, 26 percent of samples had a low level of blind seed disease (Lafferty 1948).

Between 1948 and 1960, 70 percent of seed samples tested positive for blind seed disease; the average was 12 percent (Hampton and Scott 1980a). Levels of infection declined after 1960. During 1976–1978, 27 percent of samples tested positive, with a mean of 4 percent infected seed. The disease declined between 1964 and 1974 to the point that preharvest testing was stopped (Scott 1974). Hampton and Scott (1980a) related decreased levels of blind seed to the increased use of nitrogen fertilizers. In 1980–1990, only low levels of blind seed were detected (Skipp and Hampton 1996). In 1993, environmental conditions were favorable for blind seed development, and 100 percent of seed lots were infested, with a mean of 13.5 percent infected seed (Skipp and Hampton 1996). Low levels of blind seed infection returned in 1995 when conditions were again less favorable for blind seed development (Skipp and Hampton 1996).

**Northern Ireland.** By 1944, infection levels ranged from 31 to 55 percent and were as high as 70 percent in perennial ryegrass (Calvert and Muskett 1944). During 1947–1948, 60–70 percent of samples had trace to 60 percent infected seeds (Lafferty 1948).

**United States.** In the Willamette Valley of Oregon, low germination in ryegrass was first noticed in 1941 (Hardison 1957). Blind seed disease was positively identified in 1943 (Hardison 1948, 1949). By 1944, the disease was found in 85 percent of certified samples (Hardison 1945), and about one-quarter of the seed crop could not be certified (Hardison 1948).

U.S. levels of infection with blind seed disease declined during the late 1940s after the introduction of field burning to control the disease (Hardison 1976, 1980). During the 1950s, blind seed increased as growers explored alternatives to field burning. During the 1960s, when field burning was again widely practiced, blind seed occurrence returned to trace levels. Low levels of the disease were detected during 1986–1989 (Alderman 1991a,b).

In 1991, the Oregon State legislature mandated an incremental reduction in postharvest burning of grass fields to a maximum of 16,000 hectares after 1997. The area burned declined from about 80,000 hectares in 1987 to about 28,000 in 1993 (Young et al. 1994). In 1995, a high level of blind seed (20 percent infected seeds) was found in several fields of tall fescue in Oregon (Alderman 1996). However, surveys from 1995–1997 (Alderman 1999) indicate that blind seed disease levels in most fields in Oregon remain low.

## Symptoms

The seed is the only component of the host plant infected by *G. temulenta* (Wilson et al. 1945). Infected caryopses appear shriveled, rough on the surface, and rusty brown or pinkish in color (Gemmell 1940, Calvert and Muskett 1945, Hyde 1945, Noble and Gray 1945, Wilson et al. 1945, Blair 1947). Conidia accumulate on the seed surface in a spore secretion (slime), which may be waxy and clear or pale pink in color (Hyde 1938a) or may appear as a reddish-brown crust (Calvert and Muskett 1945, Hyde 1945). Healthy caryopses normally appear golden brown, plump, and smooth (Calvert and Muskett 1945). However, infected seeds covered by the lemma and palea are difficult to discern from normal seeds (Gemmell 1940, Neill and Hyde 1942, Hyde 1945).

A consequence of blind seed infection is reduced germination, and the correlation between percentage of infected seed and percentage germination in ryegrass is well established (Hyde 1932; Gemmell 1940; Greenall 1943; Calvert and Muskett 1944, 1945; Hyde 1945; Lafferty 1948; Chestnutt 1958; Hardison 1963; de Tempe 1966; Matthews 1980). Germination of infected seed is rarely greater than 10 percent (Gemmell 1940, Lafferty 1948).

## Causal Agent

The taxonomic placement of *Gloeotinia* is not clearly established. Wilson et al. (1954) placed *G. temulenta* within the family Sclerotiniaceae, based on its occurrence as a plant pathogen, presence of spermatia and macroconidia, and formation of a fleshy cupulate apothecium from a stroma. Although *G. temulenta* shares many features of the Sclerotiniaceae, it develops only an interwoven mycelium within the infected seed and does not form the true sclerotium that is characteristic of the Sclerotiniaceae. Ellis (1956) described *Gloeotinia* as structurally similar to *Symphyosirinia*, a member of the family Leotiaceae. Similar views were stated by Baral (1994) who considered *Gloeotinia* and *Symphyosirinia* related and members of the Leotiaceae, subfamily Hymenoscyphoideae. In 1997, Holst-Jensen et al. (1997) provided data from DNA analysis that *Gloeotinia* was distinct from other fungi within the Sclerotiniaceae. These studies support the concept that *Gloeotinia* should be considered a member of the Leotiaceae, subfamily Hymenoscyphoideae.

Schumacher (1979) reported that a specimen described on *Bromus erectus* by Quelet (1883) as

*Phialea granigena* was conspecific with *G. temulenta* and therefore represented an older name of the fungus. Alderman (1997) recognized *G. temulenta* and *G. granigena* as separate species, based on host range and morphological differences. *Bromus erectus* is not believed to be a host for *G. temulenta* (Hardison 1962, Alderman 1997). Little is known about *G. granigena*. Additional studies concerning species identity and their associated host range in the genus *Gloeotinia* are needed. Unfortunately, specimens of *Gloeotinia* from outside areas of commercial seed production are very rare in nature.

Two other species of *Gloeotinia* from Germany have been described: *G. aschersoniana* (P.C. Hennings and T. Ploettner) H.O. Baral on *Carex* and *G. juncorum* (J. Velenovsky) H.O. Baral on *Juncus* (Baral and Krieglsteiner 1985). Nothing is known of the life history of these species.

## Synonymy

### Teleomorph:

*Gloeotinia temulenta* Prill. & Delacr. (Wilson et al. 1954)

*Phialea temulenta* Prill. & Delacr. (Prillieux and Delacroix 1892b)

*Peziza (Phialea) temulenta* Prill. & Delacr. (Prillieux and Delacroix 1892a)

*Ciboria (Stromatinia) temulenta* Prill. & Delacr. (Prillieux and Delacroix 1893)

*Stromatinia temulenta* Prill. & Delacr. (Prillieux 1897)

*Sclerotinia secalincola* Rehm (Rehm 1900)

*Sclerotinia temulenta* (Prill. and Delacr.) Rehm (Höhnelt 1903)

*Stromatinia secalincola* (Rehm) Boudier (Boudier 1907)

*Phialea mucosa* Gray (Gray 1942)

*Gloeotinia granigena* (Q.) Schumacher for hosts other than *Bromus* (Alderman 1997)

### Anamorph:

*Endoconidium temulentum* Prill. and Delacr. (Prillieux and Delacroix 1891)

## Technical Description

**Stroma.** Infection of the grass caryopsis results in the mummification of the caryopsis, creating a substratal stroma (Spooner 1987, Williams and Spooner 1991). Hyphae, 3–4 µm wide, ramify throughout the pericarp, teste, and endosperm and are not differentiated into rind and medullary parenchyma (Gray 1942,

Wilson et al. 1945). A true sclerotium does not develop, although the infected seed functions similarly to a sclerotium as a means of survival through the winter.

**Sporodochia.** In late winter or early spring, pinkish, pulvinate, gelatinous sporodochia form either on the surface of the pales or between the pales and caryopsis (Neill and Hyde 1939, Gray 1942, Calvert and Muskett 1945, Griffiths 1959b). They are 0.4–1.3 0.5–1.5 mm in size (Prillieux 1897, Neill and Hyde 1939, Gray 1942, Calvert and Muskett 1945). Sporodochia consist of a core of closely septate, branching hyphae (Neill and Hyde 1939, Griffiths 1959b) with the terminal cells of each branch bearing 1–4 microconidiophores (Gray 1942, Griffiths 1959b).

## Microconidiophores and microconidia (spermatia).

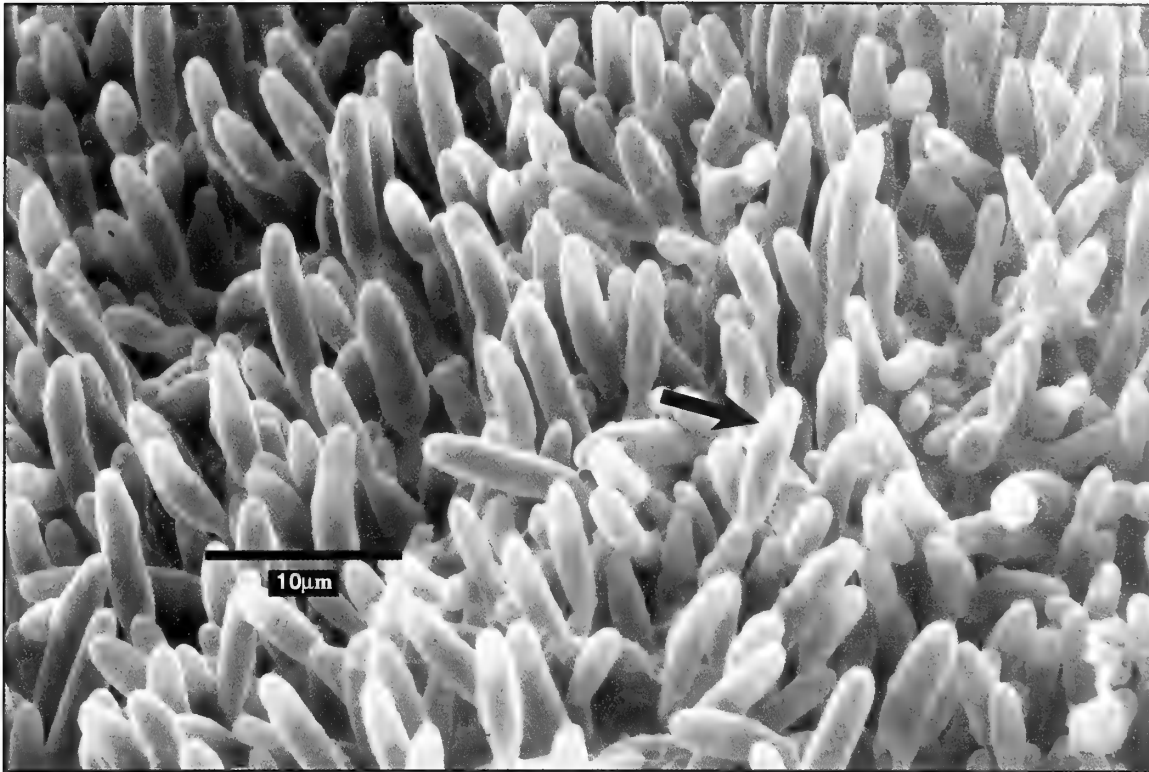
Microconidiophores are 2–5 µm in diameter and 5–9 µm long, septate, guttulate, hyaline, and penicillate (branched 2 or 3 times) (Neill and Hyde 1939, Gray 1942, Griffiths 1959b). Microconidia are first formed by a constriction below the apex of the microconidiophore. The rest bud off in succession inside a tube formed by the terminal portion of the microconidiophore (Prillieux and Delacroix 1892b; Neill and Hyde 1939; Gray 1942; Wilson et al. 1945, 1954; Griffiths 1959b).

Microconidia are unicellular, uninucleate, ovoid, guttulate or biguttulate, hyaline, 1.8–3.0 2.3–6.0 µm (Gray 1942, Calvert and Muskett 1945, Griffiths 1959b). In microconidial germination, a terminal germ tube forms; or if a transverse septum forms, a terminal or lateral germ tube will be produced (Griffiths 1959b).

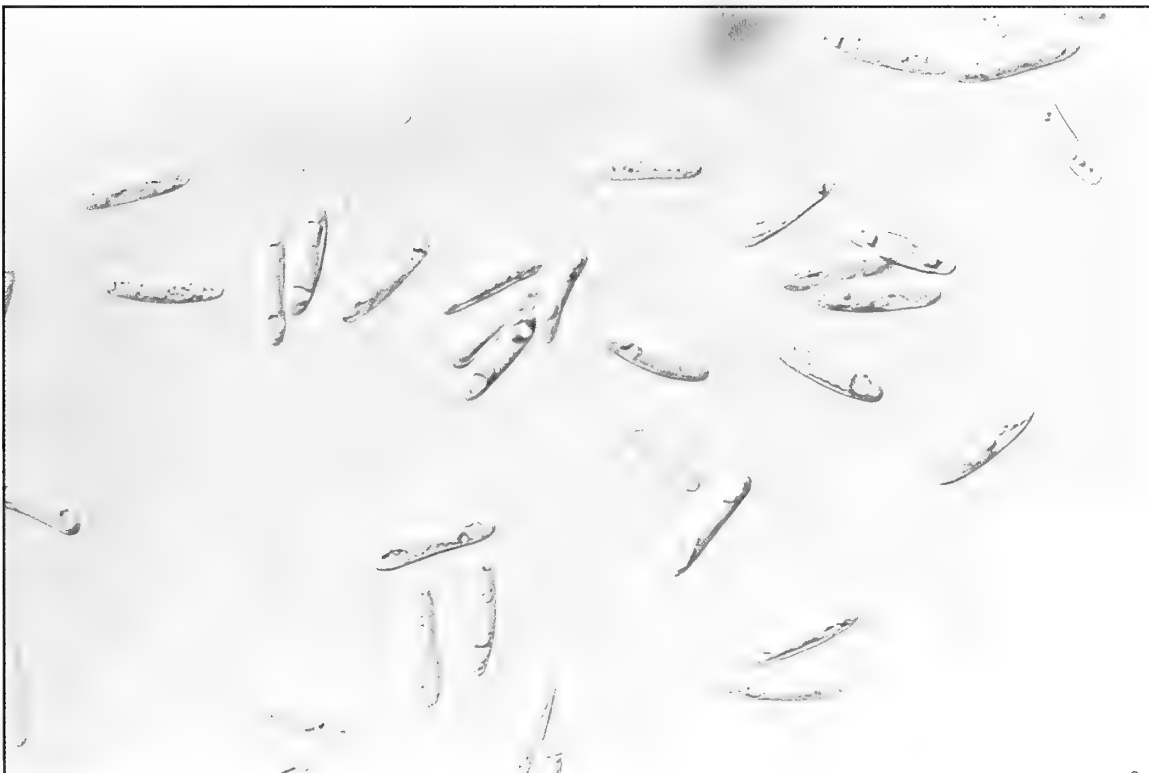
## Macroconidiophores and macroconidia.

Macroconidiophores are short barrel-shaped cells, 2–3 µm wide and 5–15 µm long, that arise laterally on the hyphae (Neill and Hyde 1939, Griffiths 1959b). Macroconidia are budded from the apex of the macroconidiophores (Griffiths 1959b) (figure 1) and arrange in clusters perpendicular to the hypha (Calvert and Muskett 1945, Wilson et al. 1945). Up to 30 macroconidia develop per conidiophore (Wilson et al. 1945).

Macroconidia are smooth, unicellular, uninucleate, hyaline, cylindrical to slightly crescentic with rounded ends, and usually biguttulate (figure 2) (Gray 1942; Calvert and Muskett 1945; Wilson et al. 1945, 1954; Spooner 1987). They are 2.5–6.3 11–21 µm in size.



**Figure 1.** Scanning electron micrograph of conidia of *Gloeotinia temulenta* being produced on the surface of an infected seed. Arrow points to macroconidium.



**Figure 2.** Macroconidia of *Gloeotinia temulenta*.

The vegetative nucleus is 3–5  $\times$  2  $\mu\text{m}$  and the nucleolus may be as large as 2  $\mu\text{m}$  (Griffiths 1959b).

On the surface of the caryopsis, macroconidia are embedded in a pinkish, slimy mass (Spooner 1987) that dries to form a hard reddish-brown crust (Calvert and Muskett 1945, Hyde 1945) (figures 3–5). When germinating, macroconidia swell and produce one or two germ tubes (Griffiths 1959b).

**Apothecia.** Apothecia are small, fleshy, and cup-shaped. One to 7 (usually 1 to 3) apothecia emerge from each infected seed (Prillieux 1897; Gray 1942; Calvert and Muskett 1945; Wilson et al. 1945, 1954) (figure 6). The stipe is smooth, velutinous under magnification, externally white or gray, internally pinkish brown, enlarging upward (Neill and Hyde 1939), and longitudinally furrowed (Spooner 1987). The stipe varies from 1 to 10 mm in length and from 0.2 to 0.5 mm in diameter (Prillieux and Delacroix 1892b, Rehm 1900, Gray 1942, Calvert and Muskett 1945) and is composed of hyaline, parallel hyphae, 4–6  $\mu\text{m}$  in diameter, occasionally intertwining and seldom branched (Gray 1942, Calvert and Muskett 1945).

Apothecia emerge from the caryopsis and elongate (figure 7). The disk of the apothecium is at first closed (Gray 1942) but opens to cup-shaped and with age becomes saucer-shaped and then flat (Gray 1942, Calvert and Muskett 1945, Spooner 1987) (figures 8 and 9). The disc diameter ranges from 1.0 to 7.0 mm (Prillieux and Delacroix 1892b, Rehm 1900, Neill and Hyde 1939, Gray 1942, Calvert and Muskett 1945). The disk color changes from light pinkish brown to deep brown (Calvert and Muskett 1945), orange brown (Spooner 1987), or pale pinkish cinnamon, darkening to cinnamon when old (Neill and Hyde 1939, Gray 1942). The margin is smooth and entire (Neill and Hyde 1939, Gray 1942, Calvert and Muskett 1945, Spooner 1987) and is radially wrinkled around the stipe apex (Spooner 1987).

**Hymenium.** The hymenium is 100–140  $\mu\text{m}$  deep (Williams and Spooner 1991). The subhymenium consists of intricately intertwined and coiled hyphae 2.5–3  $\mu\text{m}$  in diameter. The subhymenium blends into the medullary excipulum, a 22–27  $\mu\text{m}$  deep layer composed of fine, densely intertwining hyphae 2–5  $\mu\text{m}$  broad (Neill and Hyde 1939, Gray 1942, Williams and Spooner 1991). The outermost layer (the ectal excipulum) is 35–40  $\mu\text{m}$  thick and is composed of

parallel to somewhat interwoven hyphae 3.5–4.5  $\mu\text{m}$  in diameter (Williams and Spooner 1991) (figure 10).

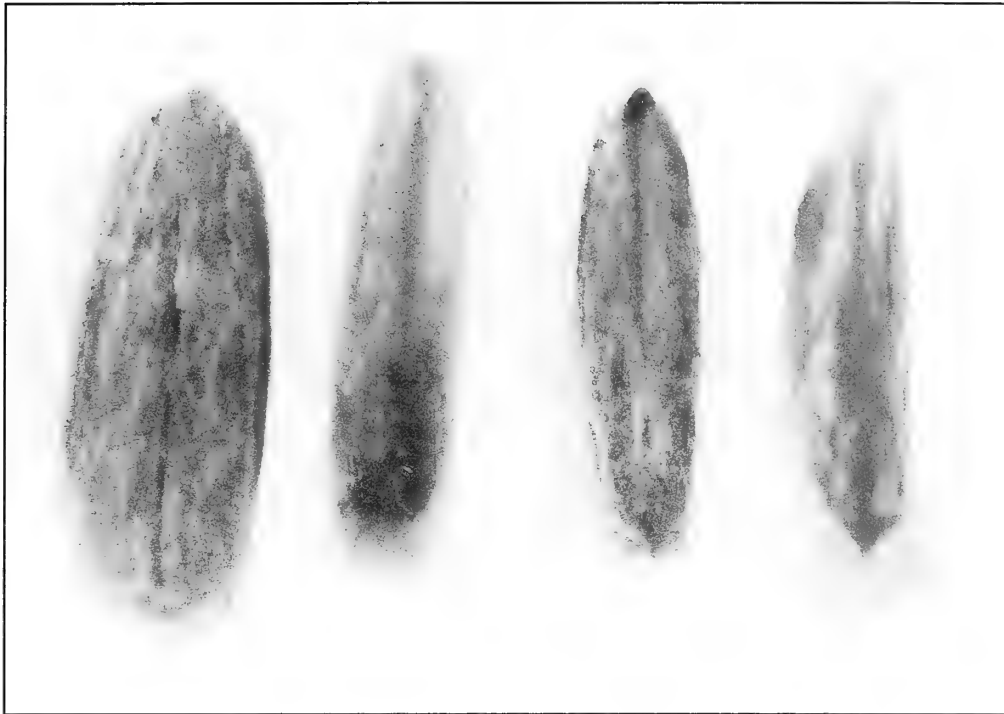
**Asci.** The asci are cylindrical and clavate, with 8 spores obliquely placed in a single row (uniseriate) in the upper two-thirds of the ascus (Neill and Hyde 1939, Gray 1942, Calvert and Muskett 1945, Spooner 1987) (figure 11). Ascus size is variable but falls within the range of 66–120  $\mu\text{m}$  long  $\times$  3–8  $\mu\text{m}$  wide. The ascus base tapers to about 2–5  $\mu\text{m}$  (Spooner 1987, Williams and Spooner 1991). The apical cap is 1–3  $\mu\text{m}$  thick (Alderman 1997), and the apical plug does not stain blue with iodine (Prillieux and Delacroix 1892b, Neill and Hyde 1939, Gray 1942, Calvert and Muskett 1945, Wilson et al. 1954, Spooner 1987).

**Ascospores.** Ascospores are hyaline, smooth, elliptical, fusoid to broadly fusoid, and usually biguttulate (Neill and Hyde 1939, Gray 1942, Calvert and Muskett 1945). One side is often flattened, or curved, continuous, or rarely developing a central septum (Spooner 1987, Williams and Spooner 1991). Ascospore size is variable, 7–14  $\times$  2.5–4.5  $\mu\text{m}$ . Germinating ascospores swell to about 10  $\times$  5  $\mu\text{m}$  (Neill and Hyde 1939) (figure 12). The first germ tube is terminal, followed by a second that is frequently lateral in position and usually constricted at the point of origin. They normally develop a central septum and two polar hyphae, but often lack a septum and have a single polar or lateral hypha (Neill and Hyde 1939, Calvert and Muskett 1945)

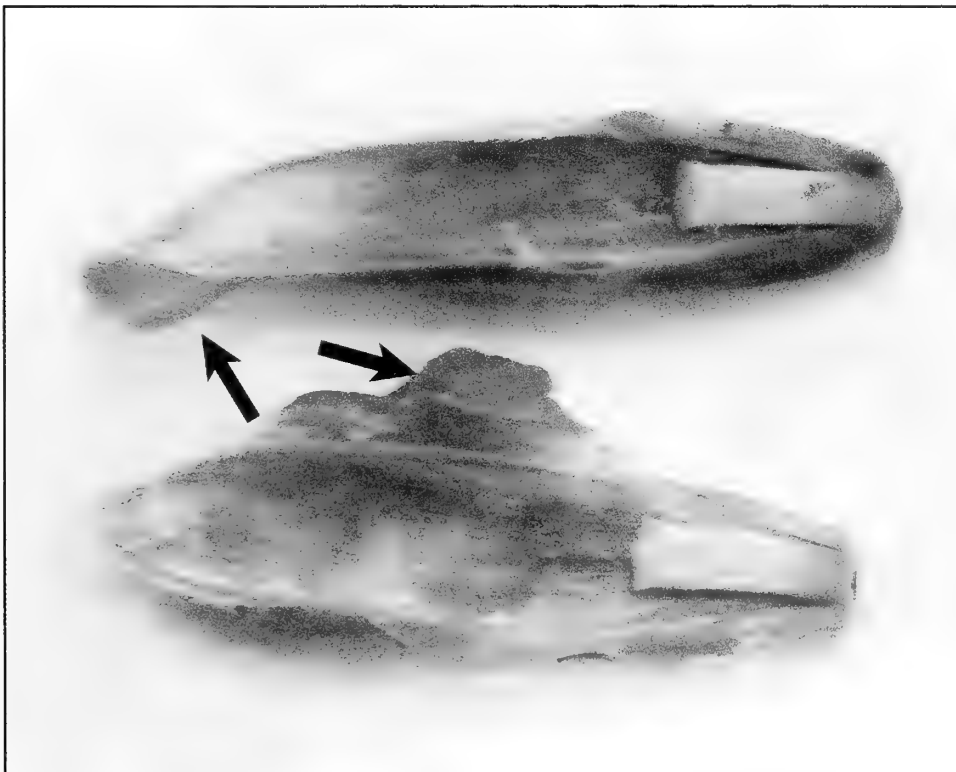
**Paraphyses.** Paraphyses are fusiform, hyaline, nonseptate (Neill and Hyde 1939, Gray 1942, Calvert and Muskett 1945) or sparsely septate (Spooner 1987) and 1.5–4  $\mu\text{m}$  wide (Neill and Hyde 1939, Gray 1942). Spooner (1987) described the paraphyses as enlarging at the apex to 2.5–3.0  $\mu\text{m}$ , but others (Neill and Hyde 1939, Gray 1942, Calvert and Muskett 1945) reported that the apex was not swollen. Paraphyses are as long as or slightly longer than the asci (figure 13).

### Growth on Media

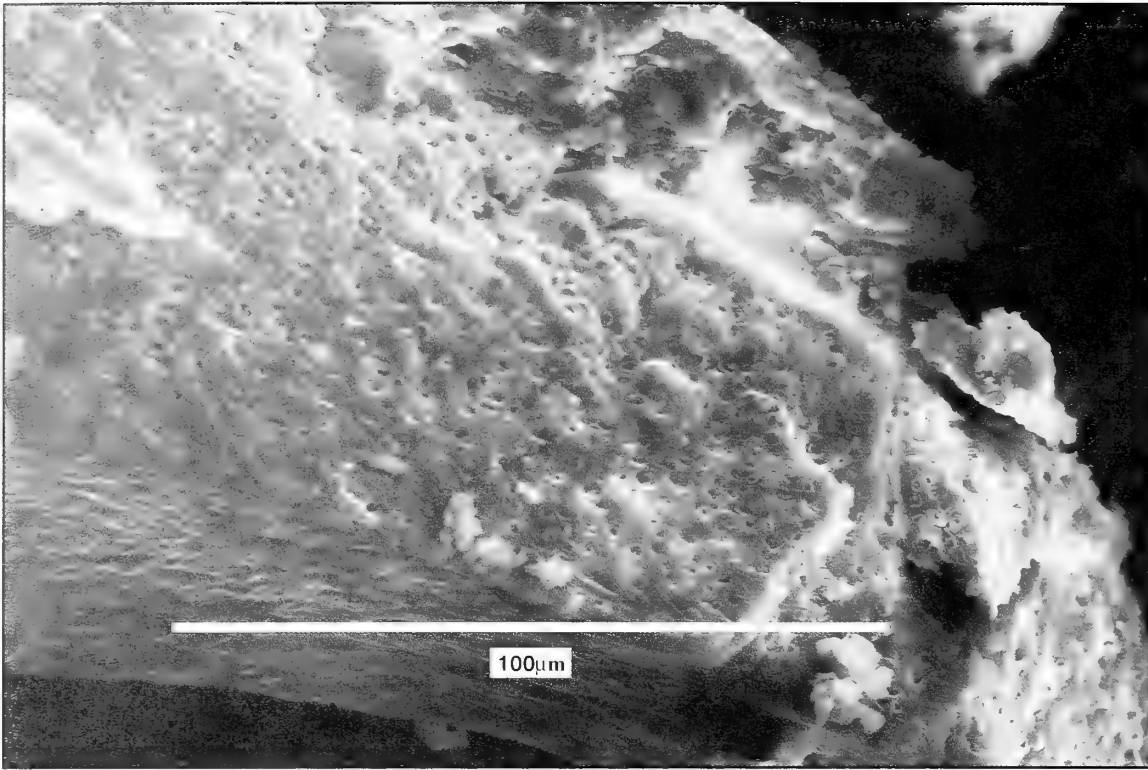
On a nutrient medium such as potato dextrose agar, *G. temulenta* grows slowly and produces a partly submerged, branching, hyaline, septate mycelium (Neill and Hyde 1939, Calvert and Muskett 1945). Sporulation and slime production occur after 7 days (Calvert and Muskett 1945, Wilson et al. 1945, Hair 1952) and in culture appears reddish brown (Neill and Hyde 1939) or chocolate brown (Wilson et al. 1945). The



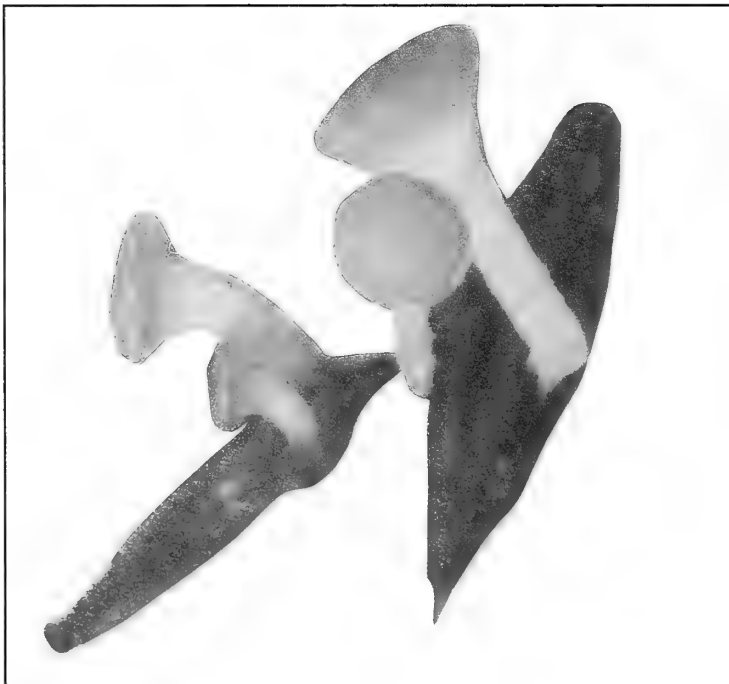
**Figure 3.** Seeds of *Lolium multiflorum* infected with *Gloeotinia temulenta* (lemma and palea removed). Healthy seed is on left.



**Figure 4.** Seeds of *Lolium multiflorum* infected with *Gloeotinia temulenta*. Arrow points to conidial slime.

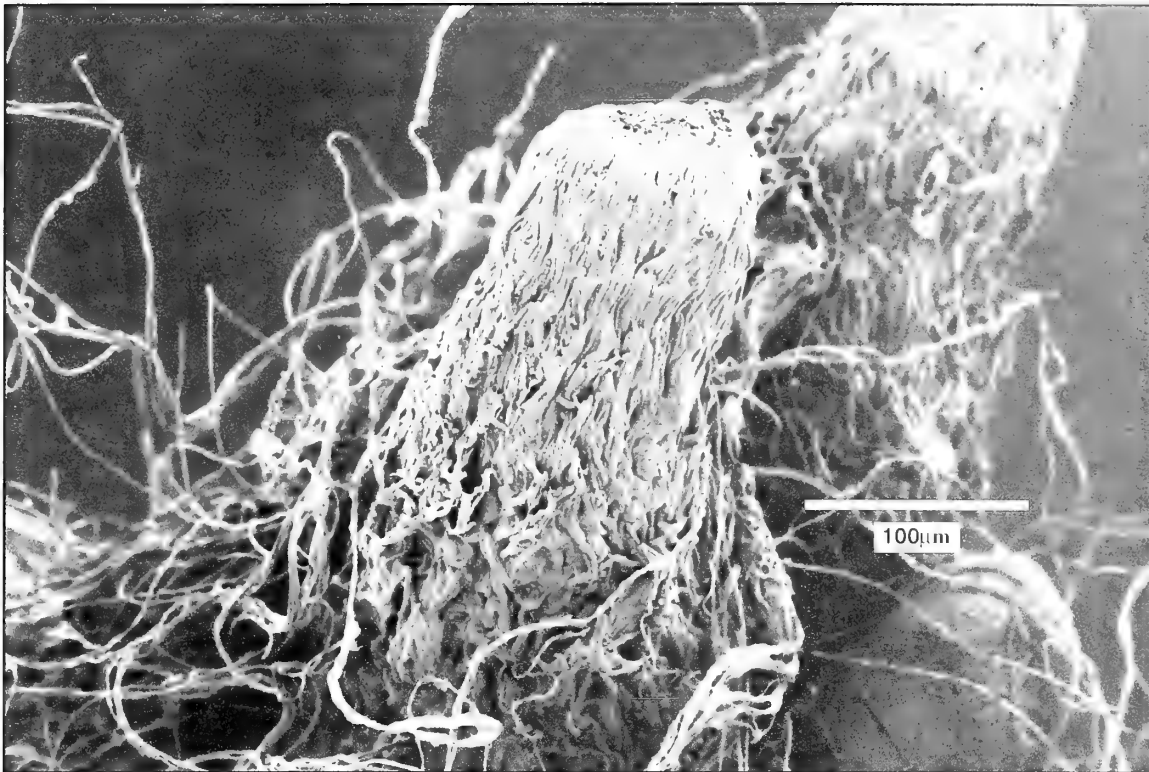


**Figure 5.** Scanning electron micrograph of the surface of conidial slime of *Gloeotinia temulenta*.

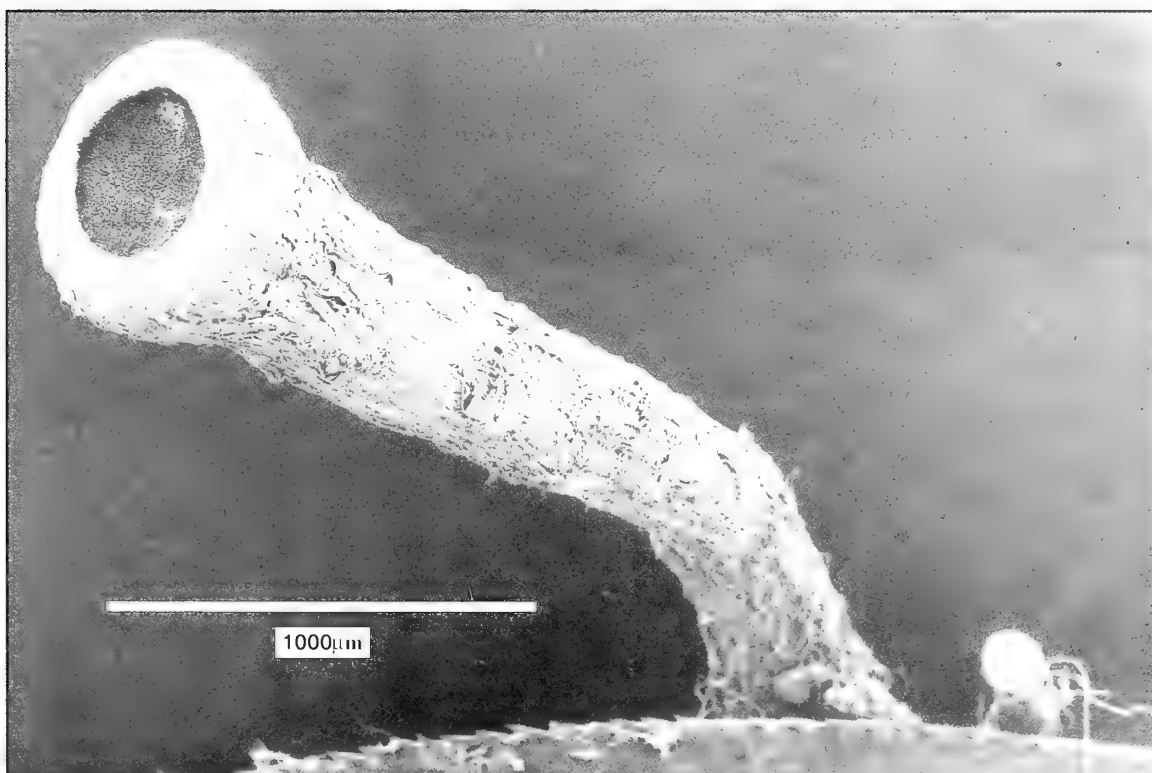


**Figure 6.** Apothecia of *Gloeotinia temulenta*.





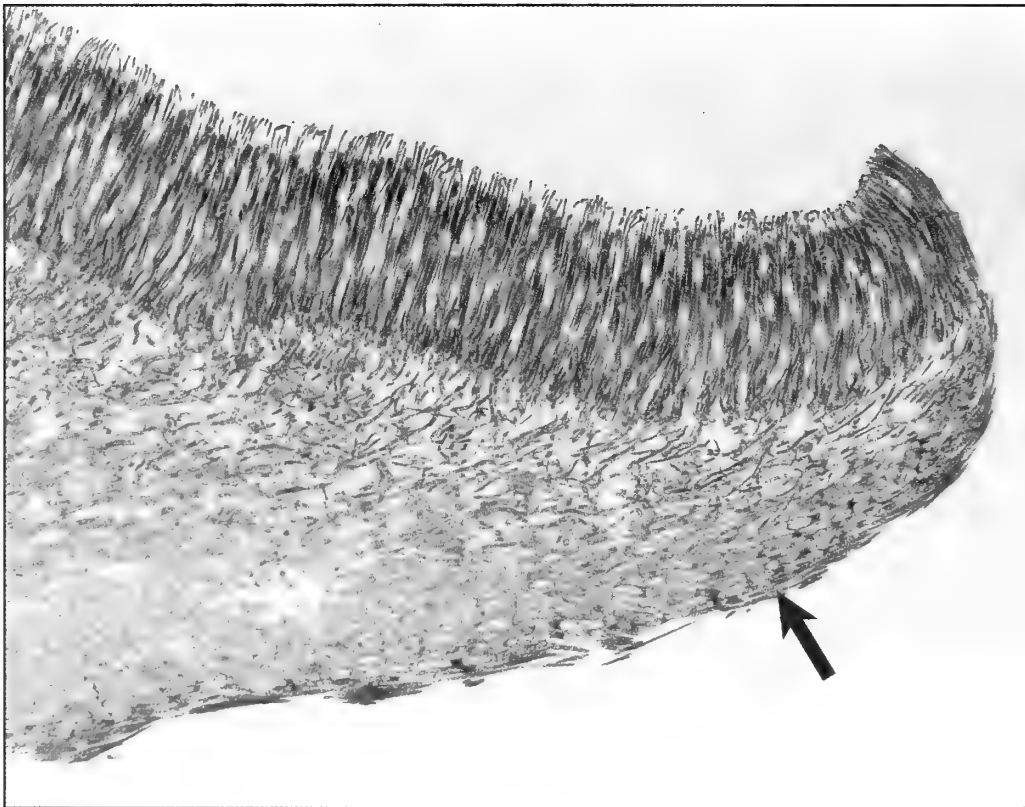
**Figure 7.** Scanning electron micrograph of the early stage of apothecium development of *Gloeotinia temulenta*.



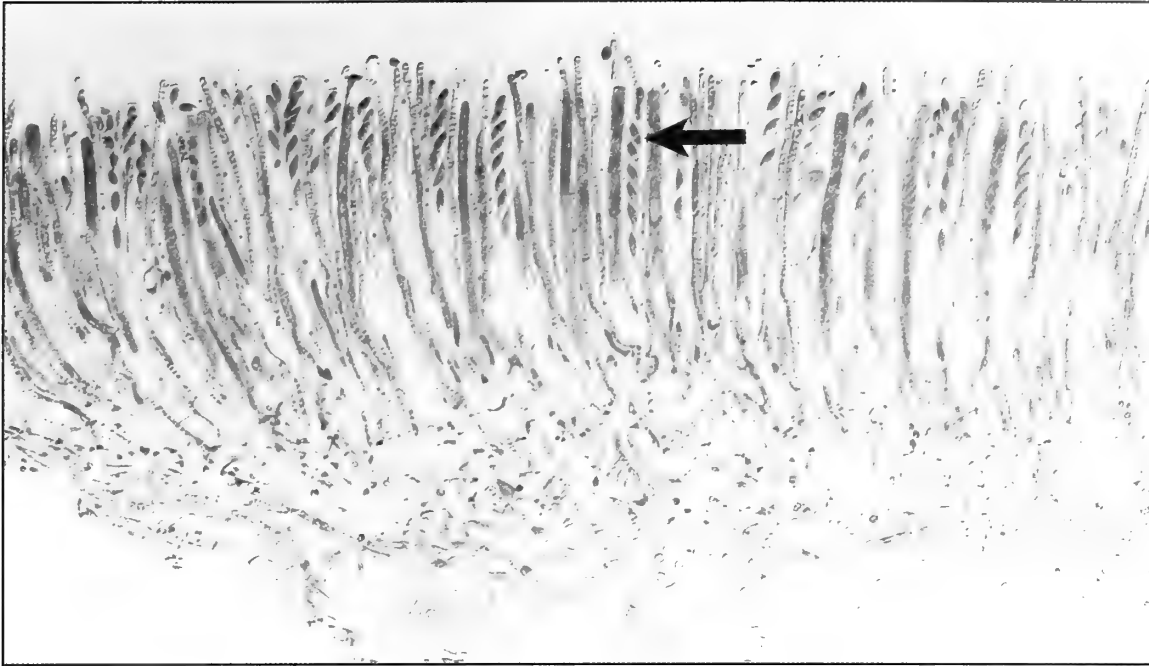
**Figure 8.** Scanning electron micrograph of developing apothecium of *Gloeotinia temulenta*.



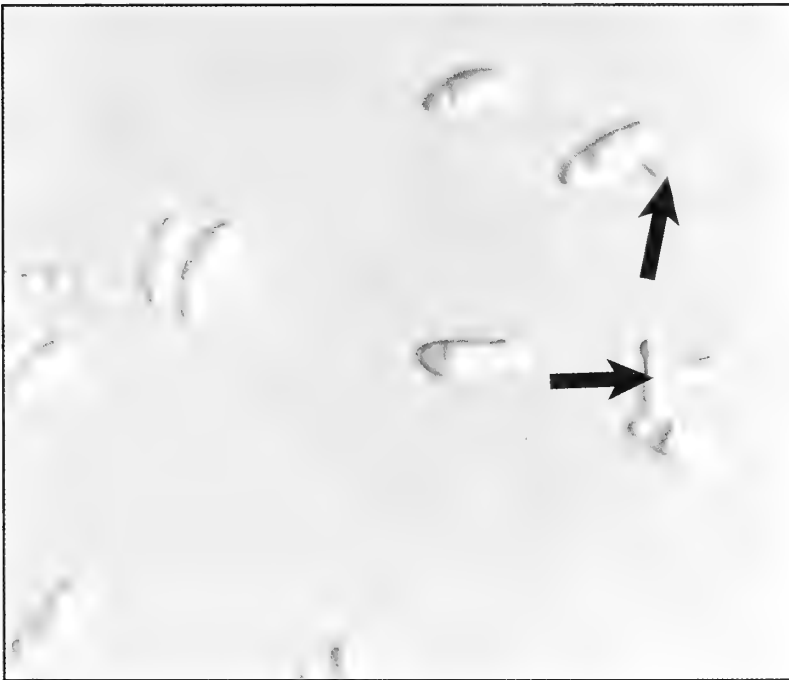
**Figure 9.** Scanning electron micrograph of mature apothecium of *Gloeotinia temulenta*.



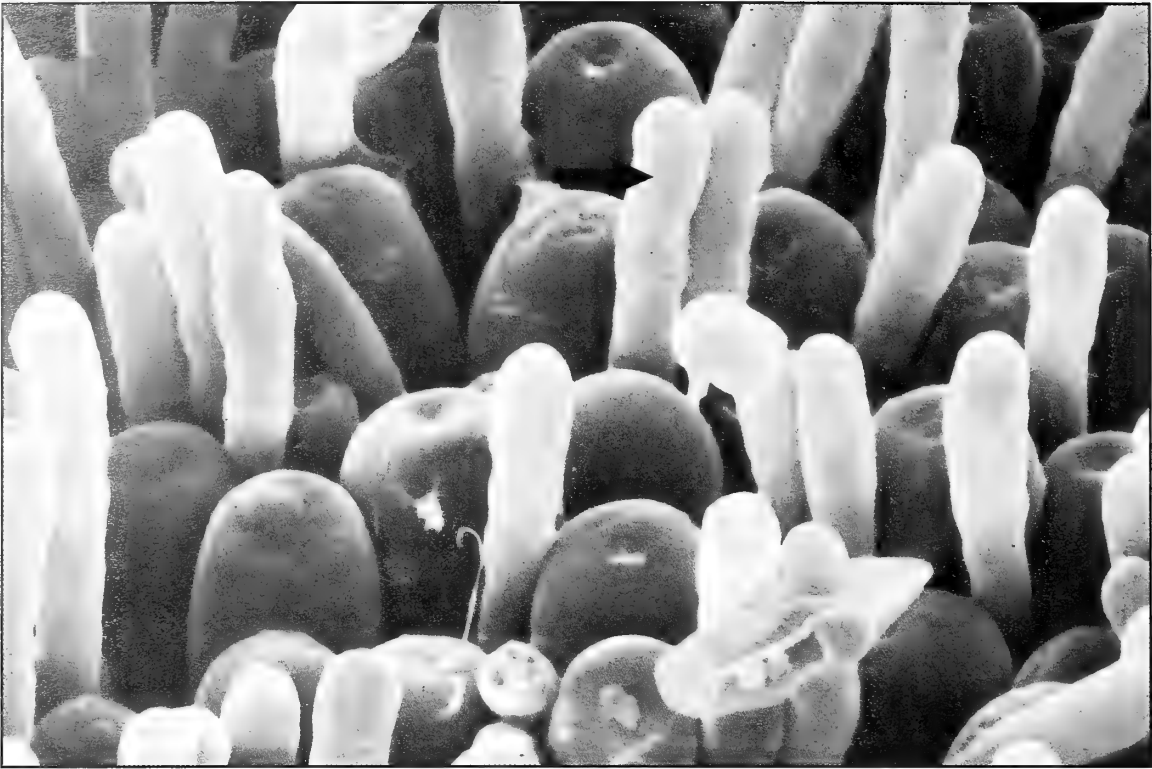
**Figure 10.** Cross section of apothecium of *Gloeotinia temulenta*. Arrow points to ectal excipulum.



**Figure 11.** Cross section of apothecium of *Gloeotinia temulenta*. Arrow points to ascus.



**Figure 12.** Germinating ascospores of *Gloeotinia temulenta*. Arrow points to germ tube.



**Figure 13.** Surface of hymenium of *Gloeotinia temulenta*. Arrow points to paraphysis.

addition of 1-percent peptone to PDA or malt agar increases spore mucilage production (Calvert and Muskett 1945). However, some cultures are predominantly mycelial while others are conidial (Wilson et al. 1945).

In culture, macroconidia are produced from short conidiophores formed at intervals perpendicular to the hypha (Calvert and Muskett 1945, Wilson et al. 1945). Conidia from culture may be larger (Wilson et al. 1945) or appear less regular than those from seed (Calvert and Muskett 1945). Growth is slow at 5 °C, optimal at about 20 °C, less at 27 °C, and restricted at 30 °C (Neill and Hyde 1939, Alderman 1992). Radial growth slows with decreasing water potential through -9.0 to -1.0 MPa (Alderman 1992).

Sporodochia develop in culture at 5 °C to room temperature after about 1–3 months (Calvert and Muskett 1945). Growth characteristics on various media were described by Neill and Hyde (1939) and Calvert and Muskett (1945).

### Similar-Looking Fungi

Calvert and Muskett (1945) collected other discomycetes associated with ryegrass and detritus that are similar to *G. temulenta* but differ in morphology in culture and do not produce spores. Unfortunately, neither species identification nor technical descriptions of these other fungi were recorded.

Neill and Hyde (1939) found a fungus on *Lolium* that is similar to *G. temulenta*. They defined it as *Lolium* fungus number 2. Unfortunately, the taxonomic description and species identity of this fungus was not established either.

## Biology and Epidemiology

### Overwintering and Production of Apothecia

The general life cycle of *G. temulenta* is illustrated in figure 14. The overwintering, or survival, unit of *G. temulenta* is the infected seed. Infected seeds reach the soil by shattering, by seed loss during harvest operations, by planting of diseased seeds, and by natural seed dispersal in harvested areas (Hardison 1945). Infected, ungerminable seeds resist attack by bacteria and molds and do not decay as they overwinter (Neill and Hyde 1939; Calvert and Muskett 1944, 1945). At or near the soil surface, *G. temulenta* continues to develop within the seed. Moist soil conditions with temperatures near 2 °C for about 8 weeks are required

to induce the sexual (apothecial) stage of *G. temulenta* (Griffiths 1958). The precise biochemical changes that occur or metabolic pathways affected during this conditioning have not been determined.

In spring or early summer, at or prior to flowering of perennial ryegrass, apothecia emerge from the overwintering infected seeds (Calvert and Muskett 1945, Wilson et al. 1945). Usually one to three, but as many as seven, apothecia can emerge from a single infected seed (Gray 1942, Calvert and Muskett 1945). Not all infected seeds will yield apothecia. In fact, only 5–30 percent of ungerminated seed produce apothecia (Calvert and Muskett 1945, Griffiths 1958).

### Production and Release of Ascospores (Primary Inoculum) and Primary Infection

Large numbers of ascospores are ejected from each apothecium in response to slight changes in relative humidity (Calvert and Muskett 1945). In New Zealand, spore release occurs between early November and middle December, with peak numbers coinciding with flowering in perennial ryegrass (Neill and Armstrong 1955). Most spores are airborne between 10:00 a.m. and 2:00 p.m. (Johnston et al. 1965).

Ascospores that land on flowers, including the stigma, ovary, or styles, will germinate and infect the host. However, seeds can be infected up to the time they reach their maximum size (Hyde 1937).

### Secondary Infection

Within about 7 days (Hyde 1937, 1945; Wilson et al. 1945) to 16–17 days (Calvert and Muskett 1945) after inoculation, the conidial stage is manifest—a pinkish slime in which conidia are embedded. These spores are relatively short-lived, about 1 month (Cunningham 1941, Neill and Hyde 1942). However, a few conidia may survive as long as 4–6 months if stored under cool, dry conditions (Calvert and Muskett 1945).

### Disease Development and Spread

Wet seasons, especially during anthesis in the grasses, are clearly supportive of blind seed infection (Foy 1927; Gorman 1940; Osborn 1947; Blair 1947, 1948; Lithgow and Cottier 1953; Chestnutt 1958; de Tempe 1966; Grant 1985). Based on field surveys in New Zealand, Lithgow and Cottier (1953) found that districts which produced ryegrass seed with high germination (low blind seed disease) had less than half the rain days during flowering than districts producing seed with low germination. Hardison

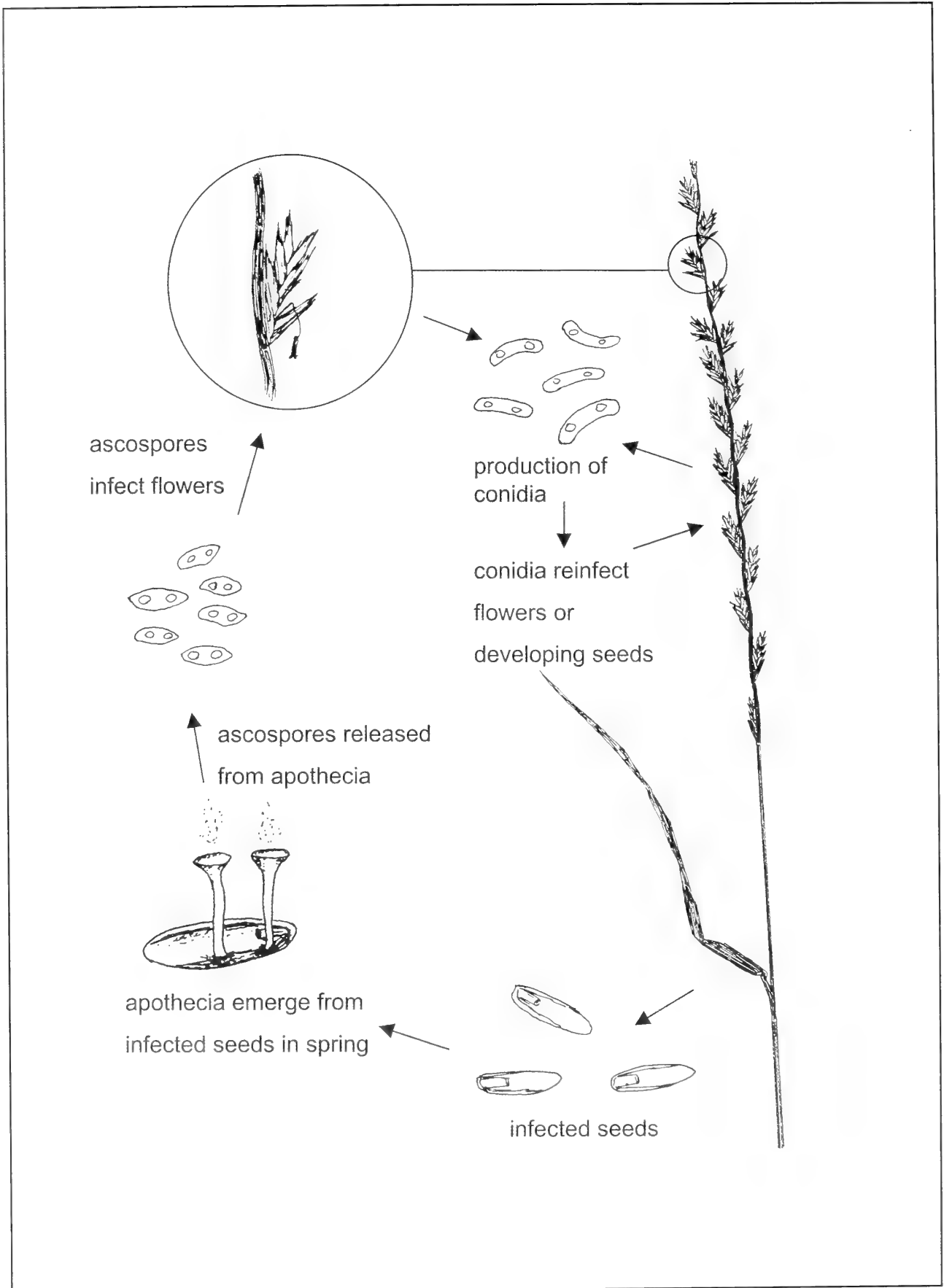


Figure 14. General life cycle of *Gloeotinia temulenta*.

(1957) concluded that blind seed in Oregon was not present in inflorescences formed in fields after the regular harvest because postharvest conditions in Oregon are typically dry with little precipitation.

Large numbers of apothecia can appear during wet weather. Blair (1948) counted 20 apothecia per square foot and observed subsequent severe disease development during a wet season in New Zealand. Under the dry conditions of 1947, no apothecia were found, and subsequent disease development did not occur. Hardison (1963) estimated that under favorable conditions in Oregon, 100 pounds of severely infected seed dispersed per acre would be expected to yield 10–50 apothecia per square foot.

Wet seasons, combined with low temperatures, extend the period of apothecial production and spore release. However, not all apothecia are produced at the same time. Some apothecia develop early, others late. Under cool (13 °C), wet conditions, apothecia can be produced over a 2-month time frame (Wright 1956). The expected lifespan of an individual apothecium is about 8–14 days, although they shrivel within a few hours in a dry atmosphere (Neill and Hyde 1939).

Temperatures of 10–16 °C and high humidity are considered ideal for blind seed development (Anonymous 1948, Alderman 1992). Infection does not occur under very warm (30 °C) temperatures (Alderman 1992).

Calvert and Muskett (1944, 1945) were the first to suggest that blind seed disease could spread from infested areas to noninfested areas, based on observations of commercial fields and field plots planted with pathogen-free seed. Additional sources of infection include seed for pastures (Hardison 1945), hedgerows with susceptible grasses, and waste ground (Calvert and Muskett 1945). Direct observations of spore movement were made by Neill and Armstrong (1955), who trapped spores of *G. temulenta* 18 m high and at ground level 1.6 km from the nearest infected field.

The highest rate of infection occurs while florets are open. The potential for infection reduces greatly after flowering (Calvert and Muskett 1944, 1945; Blair 1947). Corkill (1952) reported 90 percent infected seed when florets were open during inoculation, compared with 33 percent when florets were closed. Cool, moist weather conditions aid dispersal,

prolong the period of pollination (Calvert and Muskett 1945), and extend the period of greatest susceptibility of the plant.

Flowering in a ryegrass spike begins at the top and progresses downward over about 10 days (Noble and Gray 1945). Production of conidia begins within 6 days of infection and increases for about 16 days (Alderman 1992). Consequently, infection of upper florets by windborne ascospores may result in the spread of subsequently produced conidia to lower florets (Noble and Gray 1945) under rainy conditions. Rain dissolves the slime in which conidia are embedded and provides a vehicle for their secondary spread (Neill and Hyde 1939, Calvert and Muskett 1945, Hyde 1945).

Calvert and Muskett (1945) speculated that insects may be involved with transmission of the conidial slime. However, no observations or data on association of *Gloeotinia* with insects or their ability to vector *G. temulenta* has been published.

Infections occurring at flowering or prior to endosperm formation resulted in seeds that are thin and light in weight (Neill and Hyde 1939, Hyde 1945). These infected seeds may not be capable of supporting apothecial production (Wilson et al. 1945), although they may support development of macroconidia (Hyde 1945). Abundant production of macroconidia during early flowering or seed development provides inoculum for secondary spread and subsequent disease development.

Seeds infected during the early to middle stages of development are approximately normal size and weight (Neill and Hyde 1939, Hyde 1945, Wilson et al. 1945), and a large quantity of spores are produced (Hyde 1945). Seeds infected late in development may be capable of germination (Wilson et al. 1940, Calvert and Muskett 1945, Hyde 1945, de Tempe 1950). Fewer spores are produced from late infections than from early ones (Hyde 1945).

The potential for rapid increase in blind seed severity was emphasized by Hardison (1948, 1957), who noticed a rapid increase in disease over a 1- to 3-year period. De Tempe (1966) noted that seed with a 6.3 percent infection rate produced a crop with 26.7 percent seed infection.

## Histopathology

Detailed infection studies were conducted by Wilson et al. (1945) and Neill and Hyde (1939). Infections occurred at the base of the stigma in ovaries within 1 week of fertilization (Wilson et al. 1945). Hyphae invaded the inner epidermis, nucellus, and embryo sac. Within 9 days, conidia were produced between inner epidermis and outer integument and appeared on the surface. The endosperm and embryo filled with hyphae. The resulting grains were as long as healthy seeds but thinner. Hyphae invaded the embryo and endosperm when infections occurred after the embryo was differentiated into scutellum, plumule, radicle, and endosperm.

Neill and Hyde (1939) observed greater ramification and degradation of endosperm and embryonic tissues than Wilson et al. (1945), who observed extensive invasion of both embryonic and endosperm tissues. Wilson et al. (1945) observed hyphal penetration through the epithelial and aleurone layers, while Neill and Hyde (1939) reported that *G. temulenta* did not appear to penetrate cells of the aleurone layer. Systemic infections beyond the seed were not observed (Cunningham 1940, 1941; Neill and Hyde 1942; Wilson et al. 1945).

## Fungal Genetics and Physiology

*G. temulenta* is heterothallic—it requires genetic exchange between two different mating types for sexual reproduction and subsequent production of apothecia (Griffiths 1958). *G. temulenta* has two mating types that are identical in all morphological features. Within each apothecium half of the ascospores are of each mating type, arbitrarily called “a” and “b.” Apothecia will develop only after mating types a and b come into contact with one another and undergo fusion.

Conidia produced following infection from an ascospore of one mating type will produce only conidia of that mating type. Genetic exchange between types can occur through transfer of macroconidia from one infected seed to another or through transfer of microconidia, which can develop on the seed in spring after the seed has overwintered. A conjugation tube—a device to exchange genetic information—can form between pairs of macroconidia even before either conidium germinates (Wilson et al. 1945). As ex-

pected from the heterothallic requirement of *G. temulenta*, relatively few infected seeds produce apothecia.

The vegetative hyphae are uninucleate. Chromosome number in *G. temulenta* is  $n=15$ , and mitotic chromosomes range in size from 0.25 to 1.0  $\mu\text{m}$ . (Griffiths 1959b). In the microconidiophores the nucleolus is lacking, RNA is low, and the level of RNA depends on the level in the subtending cells (Griffiths 1959a). Microconidia have not been observed to germinate and produce a vegetative mycelium but can serve a sexual function (Griffiths 1958).

Little is known about variability in virulence of *G. temulenta*. Sproule and Faulkner (1974) reported variation in aggressiveness among strains of *G. temulenta*. Wright and Sproule (1969) reported that disease ranking of clones was the same when mixed blind seed isolates from The Netherlands or the British Isles were used.

Little is known about the physiology of *G. temulenta*. A cold conditioning period of about 8 weeks is required to induce the apothecial phase. The metabolic pathways or mechanism associated with the induction have not been investigated.

## Toxicity

Prillieux and Delacroix (1892a) and Prillieux (1897) described toxic properties associated with infection of rye by the asexual stage of the blind seed fungus, *Endoconidium temulentum* (anamorph of *G. temulenta*). Consumption of bread made from the flour induced dizziness, faintness, vertigo, and an intensive stuporous state lasting for several days. Dogs, pigs, and poultry that consumed the bread became depressed, numb, and refused to eat or drink for 24 hours. The symptoms in humans and animals differed from those produced after ingestion of ergot (*Claviceps purpurea*) or darnal (*Lolium temulentum*) (Prillieux and Delacroix 1892a, Prillieux 1897). This is the only known report of toxicity from seed infected with *G. temulenta*.

Cunningham (1958) conducted trials in which sheep were fed seed infected with *G. temulenta*. No abnormal symptoms or effects were observed.



## Disease Management

The survival propagule of *G. temulenta* is the infected seed. Control measures center around removing as many infected seeds as possible from the field during harvest and avoiding introduction of infected seed by using disease-free or treated seed. Maintaining a healthy stand through good fertilization practices also contributes to control of blind seed. An integrated approach to blind seed control should consider disease resistance, field location, seed source, seed treatments, planting, time of closing, fertilization, stand density, fungicide sprays, methods of harvest, postharvest residue management (straw residue removal, postharvest plowing, crop rotation, field burning), and postharvest seed cleaning.

## Disease Resistance

The search for resistance to blind seed began shortly after discovery of the disease. Early investigations in New Zealand compared indigenous grasses to commercial grasses (Hyde 1932, Calvert and Muskett 1944, Corkill and Rose 1945, Blair 1947). Differences in susceptibility were attributed to timing of flowering and favorability of climatic conditions during flowering (Gorman 1939, Gemmell 1940, Calvert and Muskett 1945, Corkill 1952, Wright 1956).

Early attempts at breeding ryegrass for resistance to *G. temulenta* were confounded by high variability and inconsistent results (Corkill 1952). Corkill and Rose (1945) examined progeny of crosses of resistant and susceptible ryegrass plants and concluded that resistance or susceptibility to the disease was inherited. Sproule and Faulkner (1974) reported that resistance was quantitative and repeatable across environmental conditions and fungal strains. Wright (1967) concluded that more than one gene was involved in resistance. Wright and Faulkner (1982) used a back-cross program to introduce resistance to *G. temulenta* into S24 perennial ryegrass. Cultivars Calan and Logan were found to have significantly greater resistance than S24. Unfortunately, little resistance is believed to be present in most cultivars of perennial ryegrass and tall fescue now grown commercially for seed.

## Field Location

Locating fields away from infested fields to avoid the introduction of inoculum from nearby sources is recommended (Blair 1947, 1948, 1952; Hardison 1949; Lithgow and Cottier 1953). To prevent estab-

lishment and persistence of infected seed, grazed areas not kept for seed should be topped when seed heads appear (Blair 1948). Surrounding fields with crops such as cereals or root or forage crops may provide a barrier to movement of spores into a field (Blair 1947), although long-distance (more than 1 km) airborne movement of ascospores can occur (Neill and Armstrong 1955).

## Seed Source

Since infected seed is the source of inoculum, planting disease-free seed is recommended (Calvert and Muskett 1944; Blair 1947, 1948; Hardison 1949). Osborn (1947) and Blair (1948) suggested that in New Zealand supplies of disease-free seed could be obtained in dry years when little disease develops.

Prillieux (1897) reported that in France the disease was scarce on rye (*Secale cereale* L.), but recommended that, where the disease is present, seed from regions free of contamination be used for planting.

## Seed Treatments

*G. temulenta* has limited survival in seed stored dry. Seed stored for 18 (Blair 1947), 21 (Calvert and Muskett 1945), or 20–22 months before spring planting (Hardison 1949, 1957) and 24 months before fall planting (Hardison 1949, 1957; Wade 1955) is considered safe to plant.

Calvert and Muskett (1944, 1945) controlled blind seed with a hot water treatment that included either a 4-hour pretreatment with tepid water, then 15 minutes at 50 °C, or no preimmersion treatment and 30 minutes at 50 °C. The treatments provided full control with little or no reduction in seed germination. After hot water treatment, infected seeds decayed in the soil (Calvert and Muskett 1944). Untreated infected seeds resisted decay. De Tempe (1966) reported complete blind seed control with no effect on germination when seed was treated with water at 45–46 °C for 2–2½ hours. Gorman (1940), however, reported lack of adequate control from hot water treatments.

Numerous fungicides have been evaluated for their efficacy as seed treatments for blind seed disease. Although Hair (1952) reported some success, most of the early research indicated that chemicals applied as seed protectants were not effective against blind seed disease (Gorman 1940; Calvert and Muskett 1944, 1945; Blair 1947; de Tempe 1966; Hardison 1975).

However, modern systemic fungicides such as benomyl have proven effective as a seed treatment (Hardison 1970, 1972; McGee 1971b). In New Zealand, seed treatment with fungicides has proven effective and is recommended for control of blind seed disease (Rolston and Falloon 1998).

## Planting

Calvert and Muskett (1944) reported that seed samples from fields sown with a high level of blind seed did not on average show a higher rate of infection than seed from fields sown with disease-free seed. Similarly, de Tempe (1966) found no association between severity of blind-seed-infected seed at planting and subsequent level of infection at harvest. However, the effect of infected seed introduced at the time of planting depends on the method of planting and planting depth. Hardison (1957) observed that maximum production of apothecia occurred when fields too small for drill planting were planted by broadcasting seed over the soil surface. When seeds are planted more than one-half inch deep, apothecia have difficulty reaching the soil surface (Hardison 1949, 1957). Good preparation of the seed bed facilitates planting at the proper depth and good coverage of seed (Hardison 1949, 1963).

Fields with heavy soils or poor drainage may be more favorable for blind seed development because they provide the prolonged moist conditions that are favorable for production of ascospores. Good soil drainage provides conditions that are less favorable for apothecial production (Hardison 1949, 1963).

Infected seed must undergo a cool, moist period for about 8 weeks to induce the reproductive (apothecial) phase of the pathogen. Wright (1956) found that when seed was planted in spring, apothecial production did not occur; the requirement for cold conditioning was not met. Similar results were reported by Fischer (1944), who detected no apothecia when seed was planted in spring but found 75.6 apothecia per square meter in fall-planted seed.

Planting a susceptible first-year companion crop such as *L. temulentum* is not recommended because of its potential to increase inoculum if seed becomes infected (Hardison 1949, 1957, 1963).

## Time of Closing (Grazing)

Crops in New Zealand that are closed to grazing very early or very late in the season may yield a crop that

escapes peak ascospore dispersal (Blair 1947). Early closing was recommended in New Zealand by Gorman (1940), Lithgow and Cottier (1953), and Lynch (1952).

## Nitrogen Fertilization

Numerous studies indicate a reduction in blind seed in response to manure or nitrogen fertilization. Chestnutt (1958) and Rutherford (1956) reported a significant reduction in blind seed in manured plots, compared with unmanured plots of perennial ryegrass. Lynch (1952) and Lithgow and Cottier (1953) observed that nitrogen improved yield and germination, although the effect of nitrogen on blind seed was uncertain. In a paired-plot experiment, Stewart (1963) found blind seed levels decreased in plots treated with nitrogen compared with untreated plots.

Hampton and Scott (1980a) established that a decline in blind seed between 1960 and 1980 in New Zealand correlated with the increased use of nitrogen fertilizer. In field trials, they demonstrated that as nitrogen rate increased, the rate of blind seed infection decreased, a result also reported by Hampton (1987) and de Filippi et al. (1996).

Under laboratory conditions, Hampton and Scott (1980a) observed that urea directly suppressed apothecial formation. However, in field plots, Hampton and Scott (1981) found no significant differences in number of apothecia among field plots treated with various levels of urea, although a reduction in blind seed infection was observed in urea treatments. They concluded that nitrogen fertilization altered the physiology of the plant, enhancing resistance to *G. temulenta* (Hampton and Scott 1980b).

In subsequent studies de Filippi et al. (1996) examined the level of blind seed in adjacent irrigated and nonirrigated field plots to which various rates of urea had been applied. In irrigated field plots, nitrogen application significantly reduced blind seed disease, but this did not occur in nonirrigated plots. As the inoculum source was external to the trial, they concluded that plants which are able to utilize available nitrogen develop a greater capacity to resist blind seed. The mechanisms associated with this resistance need to be determined.

Hampton (1987) reported there was no advantage to a split application of nitrogen (fall, spring) and recommended that all nitrogen be applied in spring. Blind

seed levels in the study were lowest when all of the spring nitrogen was applied at spikelet initiation.

In addition to increasing resistance, nitrogen applications can also increase lodging or increase stand density, providing a physical barrier to restrict spore movement up through the canopy (Gorman 1940, Noble and Gray 1945, Blair 1947).

### Stand Density

Movement of ascospores upward through ryegrass stands is believed to be reduced in a dense canopy, in stands that lodge, or where clover is planted with the ryegrass (Gorman 1940, Noble and Gray 1945, Blair 1947). Hampton (1987) reported that as lodging increased, blind seed disease decreased.

Lynch (1952) and Lithgow and Cottier (1953) found no evidence that germination was related to crop density or the extent of bottom growth, although they noticed improved germination in crops that lodged or those with increased percentages of grass in the sward. Wilson et al. (1945) observed that a ryegrass crop which remains standing until harvest was more likely to become infected by *G. temulenta* than a dense, heavily lodged crop. Noble and Gray (1945) found that acidic soils could contribute to poor stands of ryegrass and recommended replacement of ammonium sulfate with nitro chalk.

### Fungicide Sprays

Under field conditions, fungicides applied as foliar or inflorescence sprays were not demonstrated effective in blind seed control by Corkill and Rose (1945), Hair (1952), or Hardison (1970). However, recent research from the Foundation for Arable Research (Rolston and Falloon 1998) has established that fungicides such as tebuconazole or carbendazim are effective for blind seed control in New Zealand.

Sprays applied as soil drenches or to the soil surface have been shown effective in reducing the number of apothecia. McGee (1971b) observed that benomyl applied at 2.8 and 5.6 kg/ha reduced apothecia 80 and 90 percent, respectively. Hardison (1970) eliminated apothecia during April and May with a single application of benomyl (4.5 kg/ha) applied the previous November, December, or January. Hardison (1972, 1975) lists other fungicides effective against *G. temulenta* under greenhouse conditions.

### Harvest

Since the primary source of inoculum is the infected seed, early harvest to avoid excessive seed shatter is recommended. Osborn (1947) suggested early harvest under dry conditions as a source of disease-free seed, since late season disease could develop with a change in the weather to wet conditions. In Oregon, there is a narrow window of time in which swathing can occur to avoid seed shatter and obtain optimum seed yields.

Removal of lightweight or infected seeds during harvest reduces inoculum left in the field. Hardison (1949, 1957, 1963) recommends adjusting combines to retain lightweight seeds for removal from fields.

### Straw Residue Removal

Since dry soil conditions are unfavorable for apothecial development and spore release, Hardison (1949) recommended removing the straw after harvest to allow the soil surface to dry more rapidly in spring. In Oregon, residue is commonly baled and removed from the field. In some cases the straw is finely chopped with specialized flails. Residues that are not sufficiently chopped decompose slowly and can interfere with crop growth or development and may leave the soil wet for prolonged periods (Young et al. 1992).

### Postharvest Plowing

Plowing infested fields reduces the area of infestation by burying much of the inoculum source—the infected seeds (Hardison 1963). Hardison (1949) recommended plowing in Oregon before May 15 to prevent emergence of apothecia near the time of flowering in ryegrass. The effectiveness of plowing in control of blind seed in Oregon was demonstrated by Hardison (1949, 1957, 1963).

### Crop Rotation

Blair (1947) reported that less infection occurred in stands following 3–4 years of arable crops, suggesting that rotation with crops not susceptible to blind seed may provide a means to reduce inoculum within a field.

### Field Burning

The effectiveness of field burning in control of blind seed was established by Hardison (1949, 1980). Excellent control of blind seed is achieved with postharvest field burning. For optimal control, the entire dry-straw residue should be open burned. Burning by propane flaming after residue removal

(baling) is not as effective as open burning, since propane does not achieve the temperatures of open-grass burning (Johnston et al. 1996).

## Seed Cleaning

Recleaning of seed lots is not very effective in reducing the level of blind seed (de Tempe 1966). Hampton et al. (1995) reported that cleaning to a higher seed weight by removing infected seeds improved germination for some seed lots with a high level of infection; but in lots with a low level of blind seed, cleaning simply removed small but viable seed. A relationship between seed weight and germination could not be established.

Since infected seed are present in screenings, destroying the screenings destroys the inoculum. Destruction of screenings infested with blind seed was advocated by Hardison (1949).

## Methods for Detection and Assessment

### Postharvest Disease Detection and Assessment

Early methods of blind seed detection involved the direct observation of seed. Gemmell (1940) detected infection by looking for small pinkish spots on dehusked seed under a binocular microscope illuminated by direct light on a white background. At Lincoln College in New Zealand, the usual procedure was to place 100 paled seeds under magnification and examine them for infection (Blair 1947), although removing the lemma or palea to examine the caryopsis can be tedious. Sproule and Wright (1966) developed a manually operated apparatus to facilitate the removal of lemma and palea.

Infected seeds generally appear more opaque than healthy seeds. A diaphanoscope was used to differentiate infected and healthy seeds based on opacity (Noble 1939, Glasscock 1940, Hyde 1945, Muskett 1948). However, opaque seeds can also occur if the seed is weathered before threshing, in which case opacity increases due to pigmentation (Gemmell 1940, Calvert and Muskett 1945, Muskett 1948).

For estimation of total infection, Hyde (1945), Blair (1947), and Matthews (1980) believed that direct observations were not as reliable as placing seed in water and looking for spores under the microscope. A magnification of 1003 is suitable for examination for conidia of *G. temulenta* (Calvert and Muskett 1945). The lemma and palea may be removed (Calvert and

Muskett 1945, Hyde 1945, Sproule and Wright 1966) or left intact (Kolk and Rennie 1978). Kolk and Rennie (1978) soaked seed for 4 hours; Matthews (1980) soaked seed for at least 2 hours.

The number of seeds considered to provide an accurate estimate of rate of infection was reported as 100 (Calvert and Muskett 1945, Blair 1947, de Tempe 1966), 200 (Hyde 1945, Matthews 1980), or 500 (Muskett 1948). Matthews (1980) referred to the soaking and examination of seed as the “soaking test.” Matthews also performed a “droplet test,” in which 100 seeds were individually soaked in drops of water on microscope slides for at least 4 hours. The drops were examined at 1003 and classified subjectively as having light, moderate, or heavy spore concentration. However, Matthews did not find a significant correlation between the droplet test and ungerminated seed.

Rose (1945) correlated conidial numbers removed through soaking samples of 100 seeds with germination rate, but high variability in the number of conidia prevented accurate prediction of germination. Hardison (1957) mixed 18 ml of seeds and 18 ml of water in 250-ml flasks, soaked the seeds for 20 minutes, then counted conidia in a 0.0063-mm<sup>3</sup> hemacytometer chamber. The number of conidia per 0.0063 mm<sup>3</sup> corresponded to five infection classes ranging from trace to heavy. One to three conidia per 0.0063 mm<sup>3</sup> corresponded to a trace infection level, and more than 30 conidia corresponded to a heavy infection. Alderman (1999) used a similar seed-washing procedure and established a linear relationship between the number of conidia washed from a standardized seed sample and the percentage of infected seed.

Matthews (1980) described a detection method based on production of apothecia. In this test, 200 seeds were scattered over moist perlite in 14-cm-diameter petri dishes. The dishes were placed in plastic bags and stored at 5 °C for 12 weeks. Normal germinated seeds were removed. Dishes were transferred to 20 °C under a 12 hour light/12 hour dark cycle for a further 4–5 weeks. Seeds with apothecia were recorded. This procedure estimates the potential inoculum from seed, but since many infected seeds do not produce apothecia, the total number of infected seeds is greatly underestimated.

The number of seeds infected with viable *G. temulenta* can be assessed by isolating the pathogen

on nutrient media. In this test, the palea are removed from the seeds, the caryopsis is surface sterilized and bisected, and the halves are plated on malt-extract agar (Neill and Hyde 1942, Calvert and Muskett 1945, Muskett 1948).

### **Preharvest Testing**

Preharvest testing of blind seed was common during the 1940s in New Zealand (Scott 1974) to determine if the ryegrass seed crop should be harvested. Greenall (1943) sampled seed heads 2 weeks before harvest and found good correlation between the percentage of seed not infected (healthy seed) and germination of machine-dressed seed. However, samples should be taken within 1 week of cutting (Hyde 1942, 1945; Lithgow and Cottier 1953; Munro 1978; Alderman 1988, 1991b). Infection can occur up to the time of cutting, so samples collected too early could underestimate postharvest infection levels.

The number of seed heads believed to be representative of the area was reported as 50 (Wade 1949), 300 (Hyde 1945, Osborn 1947), or 400–500 (Lithgow and Cottier 1953; Alderman 1988, 1991b).

### **Outlook**

The past decade has seen considerable changes in the management of grass seed as growers moved away from open-field burning of postharvest residue.

Current management practices generally include baling and removing straw residue followed by flail chopping any remaining residue. In some cases, specialized flail choppers are used on the full straw load. Some growers practice no-till planting. It is not clear what long-term effect these practices will have on development of blind seed disease. Weather's role is significant. Several consecutive years of wet weather during flowering could be highly favorable for disease development.

Surveys of blind seed disease conducted over the past decade have established the presence of a low level in Oregon. The recent appearance of a high level of blind seed in some fields of tall fescue indicates the potential for development of the disease. The greatest risk will come from residue management practices that leave large numbers of seeds in the field. Practices such as field cleaning or late harvesting in which considerable seed shatter occurs will only encourage the disease under favorable conditions.

Although significant yield losses are possible, it is important to keep in mind that there can be a significant drop in seed value at relatively low levels of infection. Germination rates below 90 percent can significantly reduce the value of the crop. Thus, the presence of only 5 to 10 percent blind seed can hurt profits.

**Table 1. Geographical and host distribution of *Gloeotinia temulenta***

- Agropyron cristatum* (L.) Gaertn.: United States (Hardison 1962)
- Agrostis canina* L.: Northern Ireland (Calvert and Muskett 1944), United States (Hardison 1962)
- Agrostis capillaris* L. [= *A. tenuis* Sibth.]: United States (Hardison 1962, Alderman 1991a,b)
- Agrostis exarata* Trin. [= *A. exarata* Trin. var. *monolepsis* (Torr.) Hitchc.]: United States (Fischer 1944)
- Agrostis gigantea* Roth: New Zealand (Blair 1947)
- Agrostis stolonifera* L. [= *A. alba* L.; = *A. palustris* Huds.]: New Zealand (Blair 1947), Northern Ireland (Calvert and Muskett 1944), United States (Hardison 1962, Alderman 1991a,b)
- Aira caryophyllea* L.: United States (Fischer 1944)
- Alopecurus geniculatus* L.: United States (Fischer 1944)
- Alopecurus pratensis* L.: United States (Hardison 1962)
- Arrhenatherum elatius* (L.) Beauv. ex J. and C. Presl: United States (Hardison 1962)
- Bromus carinatus* Hook. and Arn.: United States (Hardison 1962)
- Bromus inermis* Leyss.: United States (Hardison 1962)
- Bromus racemosus* L.: United States (Fischer 1944)
- Bromus rubens* L.: United States (Hardison 1962)
- Calamagrostis bolanderi* Thurber in S. Watson [= *Calamagrostis varia* Bol. ex. Thurber]: Germany (Schmid-Heckel 1988)
- Cynosurus cristatus* L.: New Zealand (Blair 1947), Northern Ireland (Calvert and Muskett 1944)
- Cynosurus echinatus* L.: United States (Fischer 1944)
- Dactylis glomerata* L.: United States (Hardison 1962)
- Danthonia californica* Boland: United States (Fischer 1944)
- Deschampsia cespitosa* (L.) P. Beauv.: United States (Fischer 1944, Hardison 1962)
- Elymus elymoides* (Raf.) Swezey [= *Sitanion hystrix* (Nutt.) J.G. Sm.]: United States (Hardison 1962)
- Elymus glaucus* Buckley: United States (Hardison 1962)
- Elymus lanceolatus* (Scribn. and J.G. Sm.) Gould [= *Agropyron dasystachyum* (Hook.) Scribn.]: United States (Hardison 1962)
- Elymus repens* (L.) Gould [= *Agropyron repens* (L.) Beauv.; = *Elytrigia repens* (L.) Nevski]: Norway (Schumacher 1979), United States (Hardison 1962)
- Elymus trachycaulus* (Link) Gould ex Shinnars [= *Agropyron trachycaulum* (Link) Malte]: United States (Hardison 1962)
- Festuca idahoensis* Elmer: United States (Hardison 1962)
- Festuca nigrescens* Lam. [= *F. rubra* L. var. *commutata* Gaud.; = *F. fallax* auct. non Thuill.; = *F. rubra* L. subsp. *fallax* auct. non (Thuill.) Nyman]: New Zealand (Neill and Hyde 1942, Blair 1947), United States (Hardison 1962, Alderman 1991a,b)
- Festuca ovina* L.: Northern Ireland (Calvert and Muskett 1944), United States (Hardison 1962)
- Festuca rubra* L.: United States (Hardison 1962)
- Festuca trachyphylla* (Hackel) Krajina [= *F. ovina* var. *duriuscula* (L.) Koch]: United States (Hardison 1962)
- Glyceria borealis* (Nash) Batsch.: United States (Fischer 1944)
- Holcus lanatus* L.: New Zealand (Blair 1947), Northern Ireland (Calvert and Muskett 1944), United States (Fischer 1944, Hardison 1962)
- Hordeum marinum* Hudson subsp. *gussoneanum* (Parl.) Thell. [= *H. hystrix* Roth]: United States (Fischer 1944)
- Hordeum murinum* L. subsp. *leporinum* (Link) Arcang. [= *Hordeum leporinum* Link]: United States (Hardison 1962)
- Hordeum vulgare* L.: United States (Hardison 1962)
- Lolium arundinaceum* (Schreber) Darbysh. [= *Festuca arundinacea* Schreb.; = *F. elatior* L.]: New Zealand (Neill and Hyde 1942, Blair 1947), United States (Hardison 1962; Alderman 1988, 1991a,b)
- Lolium giganteum* (L.) Darbysh. [= *Festuca gigantea* (L.) Vill.]: United States (Hardison 1962)

**Table 1. Geographical and host distribution of *Gloeotinia temulenta* Continued**

- Lolium multiflorum* Lam.:** Denmark (Lafferty 1948), Ireland (Lafferty 1948), Scotland (Noble and Gray 1945), New Zealand (Hyde 1938b, Lafferty 1948, Latch 1966), Northern Ireland (Calvert and Muskett 1944, 1945), United States (Hardison 1962)
- Lolium perenne* L.:** Australia (Anonymous 1955, 1962; Wade 1957; McGee 1971a; Munro 1978), Denmark (Gemmell 1940, Lafferty 1948, Kristensen and Jørgensen 1960), England (Neill and Hyde 1939, Gemmell 1940, Glasscock 1940), Ireland (Gemmell 1940, Lafferty 1948), Netherlands (de Tempe 1950, 1966), New Zealand (Gorman 1939; Hyde 1942; Blair 1947, 1948; Lafferty 1948; Hampton and Scott 1980a; Neill and Hyde 1939, 1942), Northern Ireland (Neill and Hyde 1939, Calvert and Muskett 1944), Scotland (Neill and Hyde 1939, Gemmell 1940, Noble and Gray 1945, Dennis and Gray 1954), Sweden (Neill and Hyde 1939), United States (Fischer 1944; Hardison 1962; Alderman 1988, 1991a,b) and Wales (Neill and Hyde 1939)
- Lolium pratense* (Hudson) Darbysh. [= *Festuca pratensis* Huds.]:** Northern Ireland (Calvert and Muskett 1944), New Zealand (Neill and Hyde 1942)
- Lolium temulentum* L.:** New Zealand (Neill and Hyde 1942), United States (Fischer 1944, Hardison 1962)
- Lolium temulentum* L. subsp. *remotum* (Schrank) A. and D. Löve [= *Lolium remotum* Schrank]:** United States (Hardison 1962)
- Phleum pratense* L.:** United States (Fischer 1944, Hardison 1962)
- Poa ampla* Merr.:** United States (Hardison 1962)
- Poa arachnifera* Torrey in Marcy.:** United States (Hardison 1962)
- Poa compressa* L.:** United States (Hardison 1962)
- Poa nemoralis* L.:** United States (Hardison 1962)
- Poa pratensis* L.:** New Zealand (Blair 1947), Northern Ireland (Calvert and Muskett 1944), United States (Hardison 1962, Alderman 1991a,b)
- Poa secunda* J. Presl subsp. *juncifolia* (Scribner) Soreng [= *P. juncifolia* Scribn.; = *Poa nevadensis* Vasey ex Scribn.]:** United States (Hardison 1962)
- Poa trivialis* L.:** New Zealand (Blair 1947), Northern Ireland (Calvert and Muskett 1944), United States (Hardison 1962)
- Psathyrostachys juncea* (Fisch.) Nevski [= *Elymus junceus* Fisch.]:** United States (Hardison 1962)
- Pseudoroegneria spicata* (Pursh) A. Löve [= *Agropyron inerme* (Scribn. and J.G. Sm.) Rydb.; = *A. spicatum* (Pursh) Scribn. and J.G. Sm.]:** United States (Hardison 1962)
- Secale cereale* L.:** France (Prillieux and Delacroix 1891), Germany (Rehm 1900), New Zealand (Neill and Hyde 1942), United States (Hardison 1962)
- Thinopyrum intermedium* (Host) Barkworth and D.R. Dewey [= *Agropyron intermedium* (Host) P. Beauv.; = *A. trichophorum* (Link) Richt.; = *Elytrigia intermedia* (Host) Nevski]:** United States (Hardison 1962)
- Vulpia myuros* (L.) C.C. Gmelin [= *Festuca myuros* L.]:** United States (Fischer 1944, Hardison 1962)

Table 2. Relative susceptibility of grass species to *Gloeotinia temulenta*

Subfamily	Tribe	Species	Relative Infection	
Arundinoideae	Danthoneae	<i>Danthonia californica</i> Boland	none	
Pooideae	Aveneae	<i>Agrostis canina</i> L.	none to light	
		<i>Agrostis capillaris</i> L. [= <i>A. tenuis</i> Sibth.]	none to heavy	
		<i>Agrostis stolonifera</i> L. [= <i>A. alba</i> L.; = <i>A. palustris</i> Huds.]	none to moderate	
		<i>Aira caryophyllea</i> L. var. <i>capillaris</i> (Host) Mutel [= <i>Aira elegans</i> Willd. ex. Gaudin]	none	
		<i>Alopecurus aequalis</i> Sobol.	none	
		<i>Alopecurus arundinaceus</i> Poir. in Lam.	none	
		<i>Alopecurus pratensis</i> L.	none to light	
		<i>Anthoxanthum odoratum</i> L.	none	
		<i>Arrhenatherum elatius</i> (L.) Beauv. ex J. and C. Presl	none to trace	
		<i>Avena fatua</i> L.	none	
		<i>Avena sativa</i> L.	none	
		<i>Deschampsia cespitosa</i> (L.) P. Beauv.	none to trace	
		<i>Holcus lanatus</i> L.	none to light	
		<i>Phalaris aquatica</i> L. [= <i>Phalaris tuberosa</i> L.]	none	
		<i>Phalaris arundinacea</i> L.	none	
		<i>Phleum pratense</i> L.	none to light	
		<i>Trisetum flavescens</i> (L.) Beauv.	none	
		<i>Trisetum spicatum</i> (L.) Richter	none	
		Poeae	<i>Cynosurus cristatus</i> L.	none
			<i>Dactylis glomerata</i> L.	none to trace
	<i>Festuca idahoensis</i> Elmer		none to heavy	
	<i>Festuca ovina</i> L.		none to heavy	
	<i>Festuca rubra</i> L.		none to moderate	
	<i>Festuca rubra</i> subsp. <i>fallax</i> Thuill [= <i>F. rubra</i> L. var. <i>commutata</i> Gaud.]		none to trace	
	<i>Festuca trachyphylla</i> (Hackel) Krajina (= <i>F. ovina</i> var. <i>duriuscula</i> (L.) Koch)		light	
	<i>Lolium arundinaceum</i> (Schreber) Darbysh. (= <i>Festuca arundinacea</i> Schreb.; = <i>F. elatior</i> L.)		light to heavy	
	<i>Lolium giganteum</i> (L.) Darbysh. [= <i>Festuca gigantea</i> (L.) Vill.]		none to heavy	
	<i>Lolium multiflorum</i> Lam.		heavy	
	<i>Lolium perenne</i> L.	heavy		
<i>Lolium pratense</i> (Hudson) Darbysh. [= <i>Festuca pratensis</i> Huds.]	light			
<i>Lolium temulentum</i> L.	heavy			
<i>Lolium temulentum</i> L. subsp. <i>remotum</i> (Schrank) A. and D. Löve (= <i>Lolium remotum</i> Schrank)	heavy			



Table 2. Relative susceptibility of grass species to *Gloeotinia temulenta* Continued

Subfamily	Tribe	Species	Relative Infection	
		<i>Poa ampla</i> Merr.	trace to heavy	
		<i>Poa arachnifera</i> Torrey in Marcy	none to heavy	
		<i>Poa compressa</i> L.	heavy	
		<i>Poa nemoralis</i> L.	heavy	
		<i>Poa palustris</i> L.	none	
		<i>Poa pratensis</i> L.	none to heavy	
		<i>Poa secunda</i> J. Presl [= <i>P. canbyi</i> (Scribn.) Howell]	trace to heavy	
		<i>Poa secunda</i> J. Presl subsp. <i>juncifolia</i> (Scribner) Soreng [= <i>P. juncifolia</i> Scribn.; = <i>P. nevadensis</i> Vasey ex Scribn.]	light to heavy	
		<i>Poa trivialis</i> L.	none to heavy	
		<i>Vulpia myuros</i> (L.) C.C. Gmelin [= <i>Festuca myuros</i> L.]	light to heavy	
Triticoideae	Bromeae	<i>Bromus arvensis</i> L.	none	
		<i>Bromus carinatus</i> Hook. and Arn.	none to trace	
		<i>Bromus catharticus</i> Vahl	none	
		<i>Bromus commutatus</i> Schrad.	none	
		<i>Bromus erectus</i> Huds.	none	
		<i>Bromus hordeaceus</i> L. [= <i>B. mollis</i> L.]	none	
		<i>Bromus inermis</i> Leyss.	none to trace	
		<i>Bromus madritensis</i> L.	none	
		<i>Bromus marginatus</i> Nees in Steud.	none	
		<i>Bromus polyanthus</i> Scribn. in Shear	none	
		<i>Bromus rigidus</i> Roth	none	
		<i>Bromus rubens</i> L.	none to trace	
		<i>Bromus secalinus</i> L.	none	
		<i>Bromus squarrosus</i> L.	none	
	<i>Bromus tectorum</i> L.	none		
		Triticeae	<i>Aegilops cylindrica</i> Host	none
			<i>Agropyron fragile</i> (Roth) Candargy [= <i>A. sibiricum</i> (Willd.) P. Beauv.; = <i>A. fragile</i> (Roth) Candargy subsp. <i>sibiricum</i> (Willd.) Melderis]	none
			<i>Agropyron cristatum</i> (L.) Gaertn.	trace
			<i>Elymus canadensis</i> L.	none
			<i>Elymus caninus</i> (L.) L. [= <i>Agropyron caninum</i> (L.) Beauv.]	none
	<i>Elymus elymoides</i> (Raf.) Swezey [= <i>Sitanion hystrix</i> (Nutt.) J.G. Sm.]		trace	
	<i>Elymus glaucus</i> Buckley	light		
	<i>Elymus lanceolatus</i> (Scribn. and J.G. Sm.) Gould [= <i>Agropyron dasystachyum</i> (Hook.) Scribn.]	trace to light		

Table 2. Relative susceptibility of grass species to *Gloeotinia temulenta* Continued

Subfamily	Tribe	Species	Relative Infection
		<i>Elymus repens</i> (L.) Gould [= <i>Agropyron repens</i> (L.) Beauv.; = <i>Elytrigia repens</i> (L.) Nevski]	trace
		<i>Elymus sibiricus</i> L.	none
		<i>Elymus trachycaulus</i> (Link) Gould ex Shinnars [= <i>Agropyron trachycaulum</i> (Link) Malte]	trace
		<i>Elymus trachycaulus</i> (Link) Gould ex Shinnars subsp. <i>subsecundus</i> (Link) A. and D. Löve [= <i>Agropyron subsecundum</i> (Link) A.S. Hitchc.]	none
		<i>Hordeum brachyantherum</i> Nevski	none
		<i>Hordeum bulbosum</i> L.	none
		<i>Hordeum marinum</i> Hudson subsp. <i>gussoneanum</i> (Parl.) Thell. [= <i>H. hystrix</i> Roth]	none
		<i>Hordeum murinum</i> L. subsp. <i>leporinum</i> (Link) Arcang. [= <i>Hordeum leporinum</i> Link]	none to trace
		<i>Hordeum vulgare</i> L.	none to trace
		<i>Leymus triticoides</i> (Buckley) Pilg. [= <i>Elymus triticoides</i> Buckley]	none
		<i>Pascopyrum smithii</i> (Rydb.) A. Löve [= <i>Agropyron smithii</i> Rydb.]	none
		<i>Psathyrostachys juncea</i> (Fisch.) Nevski [= <i>Elymus junceus</i> Fisch.]	none to heavy
		<i>Pseudoroegneria spicata</i> (Pursh) A. Löve [= <i>Agropyron inerme</i> (Scribn. and J.G. Sm.) Rydb.; = <i>Agropyron spicatum</i> (Pursh) Scribn. and J.G. Sm.]	trace to moderate
		<i>Secale cereale</i> L.	none to heavy
		<i>Thinopyrum intermedium</i> (Host) Barkworth and D.R. Dewey [= <i>Agropyron intermedium</i> (Host) P. Beauv.; = <i>A. trichophorum</i> (Link) Richt.; = <i>Elytrigia intermedia</i> (Host) Nevski]	trace
		<i>Triticum aestivum</i> L.	none

Source: Based on data from Hardison (1962).

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