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Biology of Rhizoctonia Species Associated with Turfgrasses

Few fungi have fostered the interest of turfgrass pathologists as have Rhizoctonia species. In 1914, R. solani Kühn was identified as a pathogen of creeping bentgrass (Agrostis palustris Hudson) by C. D. Piper, director of the United States Golf Association (39). The disease was named "brown patch" by F. W. Taylor, whose turf garden served as the source of the original diagnostic material. This marked the beginning of modern turfgrass pathology. In 1917, John Monteith and Arnold S. Dahl of the United States Department of Agriculture demonstrated control of brown patch with Bordeaux mixture, and by 1919 the fungicide was in general use on golf courses (26).

Brown patch, or Rhizoctonia blight, remains a serious disease in the warm humid and warm tropical climatic zones. The disease has been observed on at least 12 species of turfgrasses (12). In the southern United States, brown patch is a major factor limiting successful growth and maintenance of tall fescue (Festuca arundinacea Schreb.) (23), St. Augustinegrass (Stenotaphrum secundatum (Walter) Kuntze) (14,15,51), zoysiagrasses (Zoysia Willd. spp.) (44), and creeping bentgrass (23).

Prior to 1980, R. solani was considered to be the only species within the genus that caused disease of turfgrasses (11). As research led to more fully defined species concepts (37), however, pathologists began to identify additional Rhizoctonia species as turfgrass pathogens (6,13,33,42). Rhizoctonia species are imperfect fungi with teleomorphs assigned to the Basidiomycotina. Important characteristics of the genus are: 1) the absence of conidia, clamp connections, and rhizomorphs and 2) sclerotia undifferentiated into rind and medulla (some isolates and species do not form sclerotia) (31,37). Within the genus, taxa are delineated by the number of nuclei in cells of vegetative hyphae and by the color and morphology of hyphae, monilioid cells (short, broad hyphal cells), and sclerotia (37). Groups at the subspecies level are delineated by affinities for hyphal anastomosis (8,9,30, 32,38) and by differences in morphology, pathogenicity, physiology, and/or ecology (31).

Most Rhizoctonia species can be assigned to one of two groups on the basis of number of nuclei in vegetative hyphal cells (Fig. 1). Species with more than two nuclei per cell (multinucleate species) include R. solani (teleomorph: Thanatephorus cucumeris (A.B. Frank) Donk), R. oryzae Ryker & Gooch (teleomorph: Waitea circinata Warcup & Talbot), and R. zeae Voorhees (teleomorph: W. circinata) (31). Binucleate species comprise more than 40 taxa, several with teleomorphs in the genera Ceratobasidium D.P. Rogers and Tulasnella J. Schröt. (31).

In a recent revision, Moore (27) placed the anamorphs of *Thanatephorus* species (e.g., *R. solani*) in *Moniliopsis* Ruhland, those of *Ceratobasidium* in *Epulorhiza* Moore, and those of *Tulasnella* in *Ceratorhiza*. These changes resolved longstanding taxonomic problems, but the extensive literature on *R. solani* and its familiarity to plant pathologists would create potential chaos if the name *Rhizoctonia* is abandoned. In this discussion, therefore, we retain the name *Rhizoctonia* for *Moniliopsis*, *Epulorhiza*, and *Ceratorhiza* species.

Both multinucleate and binucleate Rhizoctonia species cause diseases of turfgrasses. Furthermore, turfgrasses serve as a niche for one or more non-pathogenic species of Rhizoctonia (23), and strains of these fungi may play a role in biological control of brown patho (7). Isolates of Rhizoctonia species may be visually distinct in culture (Fig. 2), but identification often requires additional morphological and physiological characteristics (Table 1).

Multinucleate Species

Taxonomic characters. Isolates of R. solani, R. oryzae, and R. zeae have been collected from turfgrasses. R. solani forms buff to brown colonies on potato-dextrose agar (PDA) (young colonies may be white) and a teleomorph of T. cucumeris (37). Hyphae of most isolates are >5 µm in diameter. Most of the

isolates studied are necrotrophic parasites, but the species also includes nonpathogenic strains, some of which form mutualistic relationships with plants (46). R. oryzae and R. zeae form white to buff to salmon-colored colonies on PDA (41,49). These species are closely related, as indicated by their common teleomorph (W. circinata), but can be distinguished in culture by the morphology and color of sclerotia. Sclerotia of R. oryzae are salmon-colored, vary in size (<1 to >3 mm in diameter) and shape (41), and usually form on the agar surface and are submerged in the medium (Martin and Burpee, unpublished). In contrast, sclerotia of R. zeae are 0.5-1.0 mm in diameter and more uniformly spherical, turn from white to orange, red, or dark brown (49), and frequently form submerged in agar and not on the agar surface (23). R. oryzae and R. zeae are necrotrophic pathogens whose host ranges probably are more confined to the Poaceae than is the host range of R. solani.

The teleomorphic genera of Rhizoctonia species are assigned to the family Ceratobasideaceae, order Tulasnellales of the Basidiomycotina (48). These resupinate hymenomycetes produce subspherical to broad, unbranched basidia with usually four stout, fingerlike or inflated sterigmata (25). T. cucumeris is characterized by the formation of short $(10-25 \times 6-19 \mu m)$, barrel-shaped to subcylindrical basidia that are about the same diameter (up to 17 µm) as supporting hyphae (47). Basidia are often formed in discontinuous clusters. W. circinata is differentiated from T. cucumeris by the formation of: 1) irregular, contorted branching of hyphae in the hymenium; 2) suburniform basidia; 3) small, curved sterigmata, one-quarter to one-fifth the length of the metabasidia; and 4) nonrepetitive spores (47).

In addition to morphology, physiological differences between isolates of *R. solani* and those of *R. zeae* and *R. oryzae* may be of value in identifying these fungi. Isolates of *R. zeae* and *R. oryzae* form dark brown reaction zones on agar media within 3 days of exposure to phenol or catechol, whereas isolates of *R. solani* and binculeate *Rhizoctonia* species form only a light brown pigment (23). Isolates of *R. zeae* (24) and *R. oryzae* (Martin,

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unpublished) are less sensitive to benomyl (EC₅₀>50 mg a.i./L) than isolates of R. solani (EC₅₀<10 mg a.i./L). Rhizoctonia species also differ in antibiotic production and in electrophoretic patterns of pectic enzymes (10).

Anastomosis groups. An affinity for hyphal fusion (anastomosis) among isolates of Rhizoctonia species has been used to assign these fungi to specific anastomosis groups (AG) (31,38). Isolates in a common AG are believed to be more genetically homogeneous than isolates in different AGs (31). To date, 10 AGs are recognized for R. solani (31), including two subgroups of AG-2 (AG-2-1 and AG-2-2) that differ in cultural morphology, thiamine requirement, and frequency of anastomosis between subgroups (31). Isolates in AG-2-2 have been divided further into those causing root rots, primarily in the Chenopodiaceae (AG-2-2 IV), and those infecting mainly shoots and leaves of the Poaceae (AG-2-2 IIIB) (31). In addition, AG-1 includes three subgroups (IA, IB, IC) that differ in morphology and symptoms induced (31,50).

Isolates of R. solani from turfgrasses have been assigned to the following anastomosis groups: AG-1 (subgroups not designated) (23), AG-2-2 (subgroups not designated) (14,15), AG-4, and AG-5; some isolates are unassigned because they fail to anastomose with isolates from described groups (Burpee, unpublished). Isolates that represent a specific AG may be associated with a specific turfgrass species, plant tissue, or perhaps geographic region. For example, most isolates collected from foliage of tall fescue in North Carolina were assigned to AG-1 (23), whereas isolates from diseased leaf sheaths of St. Augustinegrass in Texas (15) and South Carolina (14) were assigned to AG-2-2.

Isolates of *R. oryzae* from rice and soil and of *R. zeae* from rice, millet, pine, and soil have been assigned to *Waitea* anastomosis groups WAG-O and WAG-Z, respectively (35). An association of turfgrass isolates with these groups has not been reported.

Pathology. Diagnostic symptoms of turf diseases caused by R. solani, R. oryzae, and R. zeae are primarily on plant tissues at or above the soil surface. In fact, the potential impact of these fungi on roots of turfgrasses is uncertain. A root disease of turfgrass supposedly caused by R. solani (19) has not been confirmed because the species of grass was not reported and conclusive etiologic evidence was lacking. In Japan, foliar brown patch caused by R. solani AG-1 or AG-2-2 IIIB is distinguished from a root disease designated "large patch" and caused by R. solani AG-2-2 IV (33). In North America, Rhizoctonia species are considered to be primarily pathogens of aboveground parts of turfgrasses (11,45). However, Rhizoctonia species sometimes are not isolated from diseased foliage and stolons of some warm-season grasses, e.g., St. Augustinegrass and bermudagrass (Cynodon dactylon (L.) Pers.), showing brown, patchlike symptoms (Burpee, unpublished). The potential for Rhizoctonia species to induce root disease in turfgrasses requires further investigation.

Symptoms of brown patch caused by R. solani can differ with respect to turfgrass species, mowing height, and degree of turf maintenance, i.e., irrigation, fertilization, etc. For example, an irregular, silver-gray to light brown foliar lesion with a thin brown border (Fig. 3A) is a good diagnostic symptom on most nonstoloniferous species—e.g., tall fescue and Kentucky bluegrass (*Poa pratensis* L.)—that are mowed at a height of not less than 5 cm and maintained as prescribed (2) for lawn turf. In contrast, brown lesions on stolons and brown discoloration of leaf sheaths (Fig. 3B) are diagnostic on most stoloniferous species, including St. Augustinegrass and centipedegrass (*Eremochloa ophiuroides* (Munro) Hack.).

Mowing height affects symptoms in grasses such as creeping bentgrass and annual bluegrass (P. annua L.) that can be maintained at heights as low as 3.2 mm. When these grasses are mowed to a height <13 mm (e.g., golf and bowling greens), a dark gray ring or arc of mycelium (Fig. 4A), observed when the turf is wet, is a good diagnostic character of brown patch. The ring of mycelium, or "smoke ring," usually is not observed on turfgrasses maintained at >13 mm (e.g., golf fairways and home lawns). Decreased density of the foliar canopy probably limits the density of mycelium on and between leaf blades, resulting in the absence of a smoke ring. On all susceptible grasses, extensive foliar necrosis results in brown or strawcolored patches or rings a few centimeters to >1 m in diameter (Fig. 4A and B). On warm-season grasses, brown patch, caused by R. solani AG-2-2, rarely forms a smoke ring because infections that occur near the leaf sheaths induce a sheath rot symptom (Fig. 3A). Thus, mycelium of the fungus is not usually present in the higher turf canopy. Patches of infected turf can be several meters in diameter (Fig. 4C and D).

Brown patch occurs commonly on cool-season grasses (e.g., creeping bent-grass, tall fescue, and Kentucky bluegrass) during warm (>25 C), humid weather in summer. In contrast, on warm-season grasses (e.g., St. Augustinegrass, bermudagrass, and zoysiagrass), brown patch occurs commonly in spring as the plants break dormancy or in fall as they approach dormancy. Isolates from warm-season grasses have been identified as *R. solani* AG-2-2 (14,15). It is interesting to speculate that these

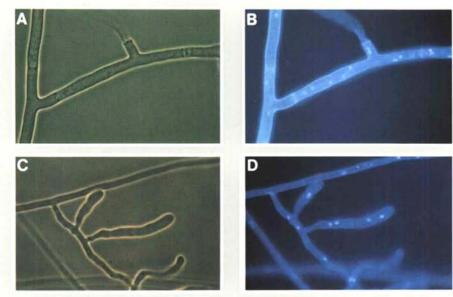


Fig. 1. Delineation of *Rhizoctonia* species on the basis of number of nuclei in hyphal cells: (A) *R. solani*, phase-contrast. (B) *R. solani* stained with 1 ppm 4',6'-diamidino-2-phenylindole (DAPI) and viewed with fluorescence microscopy. (C) *R. cerealis*, phase-contrast. (D) *R. cerealis* stained with 1 ppm DAPI and viewed with fluorescence microscopy.



Fig. 2. Cultures of Rhizoctonia species pathogenic to turfgrasses after 4 wk of incubation at 28 C in the dark. Top row, left to right: R. zeae, R. oryzae, and R. cerealis. Bottom row, left to right: R. solani AG-2-2 and AG-1A.

Table 1. Some morphological and physiological characteristics of Rhizoctonia species associated with turfgrasses

Character	R. solani	R. zeae	R. oryzae	R. cerealis	Rhizoctonia AG-Q
Nuclei per cell	>2	>2	>2	2	2
Colony color ^a	Buff to brown	White to salmon	White to salmon	White to buff	White to buff
Temperature optimum ^a	18-28 C	~32 C	~32 C	~23 C	2
Anastomosis groups	AG-I to AG-10	WAG-Z	WAG-O	AG-D (CAG-1)	AG-O
EC ₅₀ of benomyl	<10	>10	>10	>10	20-0
Phenol reaction ^b	+	+++	+++	2 10	2
Teleomorph	Thanatephorus cucumeris	Waitea circinata	W. circinata	Ceratobasidium cereale	C. cornigerum
Disease incited	Brown patch	Leaf and sheath spot	Leaf and sheath spot	Yellow patch	Yellow patche

On potato-dextrose agar (PDA).

Color surrounding colony on PDA containing phenol or catechol: + = light brown, +++ = dark brown.

Reported as incited by Rhizoctonia AG-Q only in Japan.

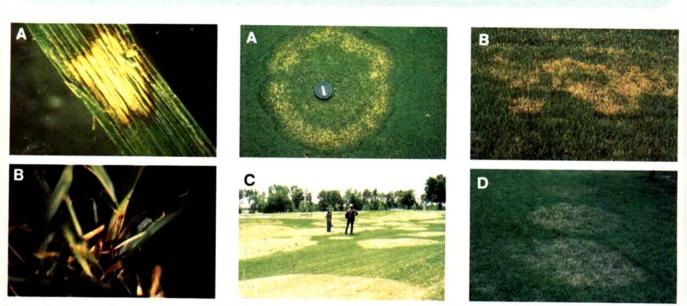


Fig. 3. (A) Foliar lesion on cool-season Kentucky bluegrass and (B) leaf sheath lesion on warm-season St. Augustinegrass induced by *Rhizoctonia solani*.

Fig. 4. Brown patch caused by *Rhizoctonia* species on: (A) creeping bentgrass, (B) tall fescue, (C) Tifton 419 bermudagrass, and (D) centipedegrass.

isolates may represent a cool-weather (<20 C) biotype of R. solani.

Symptoms induced by R. zeae and R. oryzae are less well defined than those caused by R. solani. On tall fescue, R. zeae has been isolated from lesions similar to those that are diagnostic for brown patch (Fig. 3A). On creeping bentgrass maintained at the mowing height of a putting green, R. zeae has been isolated from turf showing dark gray-brown or yellow arcs or circles; a smoke ring does not form. The role of R. zeae in the etiology of these symptoms remains to be determined. Occasionally, during periods of high temperature (>32 C), white to cream-colored mycelia of R. zeae can be observed growing from the foliage of infected cool-season grasses (Martin, unpublished). In these cases, the disease may be mistaken for Pythium

On centipedegrass and St. Augustinegrass, R. zeae and R. oryzae induce water-soaked lesions at the base of leaf sheaths (Fig. 3B) similar in appearance to lesions induced by R. solani (14). Isolates of R. zeae and R. oryzae also induce foliar lesions on centipedegrass and St. Augustinegrass (14); similar lesions are not induced by R. solani AG-2-2. R. zeae has been isolated frequently from the leaf sheaths of bermudagrass showing a diffuse foliar blight (Martin, unpublished) but has not been found associated with the large brown patches typical of infection by R. solani AG-2-2 on southern grasses (Fig. 4C and D). R. zeae is isolated frequently in Florida from warm-season turfgrasses (A. Chase, personal communication).

Penetration and colonization of turfgrasses by *Rhizoctonia* species have not been studied in detail. Shurtleff (43) observed that foliar penetration of turfgrasses by *R. solani* was primarily stomatal or cuticular, or both, depending on the species of turfgrass and the fungal isolate. Cut and uncut grasses had similar amounts of disease, and infection through hydathodes was of minor importance (43). In contrast, Rowell (40) reported that disease was more severe on creeping bentgrass cut to a height of 1.3



Fig. 5. Yellow patch caused by R. cerealis on creeping bentgrass.

cm than on uncut grass. His interpretation that wounds are significant sites of penetration may be correct, but histological evidence is lacking.

Penetration and colonization of turfgrasses by *Rhizoctonia* species are probably similar to the events on rice leaves inoculated with *R. solani* (20,21) or *R. oryzae* (21). Hyphae that originate from sclerotia or plant debris grow longitudinally along leaf sheaths. Some of the branches produced from these "runner hyphae" continue longitudinal, epiphytic growth and the remainder

develop into infection cushions, lobate appressoria, or both. Infection cushions, $100-400~\mu m$ in diameter and $80-170~\mu m$ in height, are formed via the aggregation of hyphal branches. Lobate appressoria are composed of short, swollen branches with apical lobes. The percentage of hyphae that form infection structures and the dominance of one type of structure over another appear to be functions of the genotype of the host-parasite, i.e., isolate-cultivar, combination (21).

Infection structures penetrate through or between epidermal cells or through stomates. Penetration pegs arise from lobes on lobate appressoria and from lobed areas of cells in infection cushions that are aligned perpendicular to the plant surface (21). Hyphae may penetrate stomata in the absence of infection structures, but this form of penetration appears not to be common in compatible host-parasite combinations (21). Direct penetration probably results from a combination of mechanical pressure and enzymatic degradation of the cuticle and cell wall (1).

Colonization of grasses by Rhizoctonia species has not been studied in detail, but observations of R. solani on other hosts indicate that the process usually involves a combination of intercellular and intracellular hyphal growth (1). The tissue necrosis resulting from enzymatic dissolution of middle lamellae and cell walls (1), and possibly the production of toxins (29), is characteristic of infection caused by R. solani. The origin and possible function of the thin necrotic border surrounding foliar lesions on turfgrasses (Fig. 3A) are unknown; the border may constitute wound periderm and function as a resistance mechanism (4).

Observations of infection by R. zeae have not been reported, but study of the mechanisms is needed so that the factors leading to infection are sufficiently understood.

Ecology. Few reports document saprophytic growth and survival of multinucleate *Rhizoctonia* species in turfgrass swards. This is unfortunate because turfgrass with its perennial habit and potential to form thatch (a layer of living and dead roots, rhizomes, or stolons) creates a favorable environment for the saprobic development of fungi. Oval, light tan to black sclerotia of R. solani (1-2 mm long) have been found attached to leaf sheaths and grass-blades of Agrostis spp. showing brown patch (43). On turf inoculated in a greenhouse, sclerotia were formed on the adaxial and abaxial sides of leaf sheaths, in the crown, and in the area where the roots are attached to the crown (43). The number of sclerotia formed on inoculated grasses was positively correlated with the severity of foliar blight, i.e., 10, 7, 1, and 0 sclerotia per tiller on colonial bentgrass (A. tenuis Sibth. 'Astoria'), creeping bentgrass (cv. Seaside), Kentucky bluegrass, and creeping red fescue (Festuca rubra L.), respectively.

Sclerotia produced by an isolate of *R. solani* from colonial bentgrass did not show dormancy (43) and remained viable after 25 successive germinations on agar media within a 9-month period. Isolates have survived in dry grass clippings for as long as 4 months. Isolates survived longer in partially diseased turf than in completely necrotic tissue (43).

R. solani, R. zeae, and R. oryzae have been isolated from the thatch layer of tall fescue (22). Mycelia originated primarily from colonized organic matter and occasionally from sclerotia. Propagule density of the three species in a horizontal plane 0.5 cm deep was not correlated with disease severity (22).

Some aspects of the ecology of *Rhizoctonia* species can be inferred from studies of soils cropped with plants other than turfgrasses (17,36), but definitive studies of these fungi in thatch and the thatch-soil interface are required.

Binucleate Species

Taxonomic characters. Two binucleate Rhizoctonia species (BNR), R. cerealis Van der Hoeven (5) and a fungus designated Rhizoctonia AG-O (33), have been identified as turfgrass pathogens. Colonies of R. cerealis are white to buff on PDA, with hyphae $2.8-6.2 \mu m$ in diameter (3). Monilioid cells are 17-30 \times 7-15 μ m. Sclerotia are 0.3-1.2 mm in diameter, globose to irregular, and white to yellow, turning brown with age (3); some isolates, however, fail to form sclerotia on PDA (5). Optimum temperature for growth in culture is approximately 23 C (5). Colonies of Rhizoctonia AG-Q also are white to buff on PDA and are without sclerotia (Burpee, unpublished). Rhizoctonia AG-Q is slower growing (1.3 mm/day) than R. cerealis (about 5 mm/day) at

The teleomorph of R. cerealis is Ceratobasidium céreale Murray & Burpee (28), although Oniki et al (34) designated C. gramineum (Ikata & T. Matsura) Oniki, Ogoshi, & Araki as the teleomorph. The justification for this has been questioned, and the relationship between C. cereale and C. gramineum will remain uncertain until teleomorphic stages of additional isolates of R. cerealis are examined. The teleomorph of Rhizoctonia AG-Q is C. cornigerum (Bourd.) D.P. Rogers (34), but morphological differences between C. cornigerum and C. gramineum are not very distinct (34).

In addition to *R. cerealis*, fungi representing less clearly defined species of BNR have been isolated frequently from turfgrass tissues (15,23,42) or soils from turfgrass swards (22). The limited

number of isolates tested were nonpathogenic or weakly virulent on turfgrasses (15,23,42). They differ from *R. cerealis* in one or more morphological or physiological traits, including hyphal diameters, temperature optima for growth, morphology of teleomorphs, and anastomosis reactions (Burpee, *unpublished*). The relationship of these fungi to *Rhizoctonia* AG-Q (33) is unknown. Comprehensive taxonomic studies are needed, because some strains may function as biocontrol agents (7).

Anastomosis groups. Isolates of BNR with Ceratobasidium teleomorphs consist of at least 17 AGs, designated AG-A through AG-Q (30,32,34). These groups correspond to the Ceratobasidium anastomosis groups (CAG) established by Burpee (6) as follows: AG-A = CAG2, AG-D = CAG 1, AG-E = CAG 3and 6, and AG-F = CAG = 4 (30,32); groups CAG 5 and CAG 7 do not correspond to any of the Japanese groups. Isolates of R. cerealis belong to a single group, AG-D or CAG 1 (5,6,18), whereas isolates causing yellow patch symptoms in Japan have been assigned to AG-Q (33). Anastomosis affinities of other BNR from turf have not been characterized well

Pathology and ecology. R. cerealis causes yellow patch of turfgrasses (5), and a similar disease is incited by Rhizoctonia AG-Q in Japan (33). Yellow patch, familiar to turf managers as coolweather brown patch, develops at temperatures <20 C on grasses that are wet for extended periods (5). Susceptible grasses include creeping bentgrass, annual bluegrass, Kentucky bluegrass, tall fescue, zoysiagrass, and bermudagrass (12). In a controlled-environment test, Burpee et al (9) recorded mean disease indices (0 = no disease, 10 =100% disease) of 8.0, 4.7, 5.0, and 1.5 for creeping bentgrass, perennial ryegrass, tall fescue, and Kentucky bluegrass, respectively.

Detailed descriptions of preinoculation events or the mode of infection by R. cerealis are lacking. Infected grasses show foliar chlorosis and necrosis, resulting in yellow to straw-colored patches or, more commonly, irregular rings in turfgrass swards (Fig. 5). The patches and rings range from a few centimeters to more than I min diameter. The ring pattern probably develops as a result of the growth of symptomless foliage from noninfected meristems in the center of the patches (Burpee, unpublished).

Grasses infected with R. cerealis seldom show distinct foliar lesions, and the fungus does not form a dense ring of mycelium in turf similar to the smoke ring formed by R. solani. This lack of distinct signs and symptoms complicates diagnosis. Furthermore, the yellow to straw-colored rings produced in turf by R. cerealis can be similar to those

associated with certain type B superficial fairy rings (45). Therefore, diagnosis of yellow patch is usually reserved until the pathogen is isolated and identified (5). R. cerealis can be isolated from plant tissue by incubation at 15-20 C on water agar acidified with lactic acid (Burpee, unpublished). Recently, a selective medium was developed for isolation of this fungus from soil (16).

Virtually nothing is known about the ecology of BNR in a soil. Analogies drawn from information on the saprophytic activities of R. solani (36) can be misleading. The frequent isolation of BNR from roots and rhizosphere soil (Martin and Burpee, unpublished), even when isolation of R. solani is expected (22), suggests that they are competitive soil inhabitants, perhaps to the exclusion of R. solani. Detailed studies on the ecology of BNR are required.

BNR as biocontrol agents. Nonpathogenic strains of BNR from turfgrass have been used experimentally to suppress brown patch (7). Creeping bentgrass treated with strain Bn 165 24 hours before inoculation with R. solani showed 83% less disease than bentgrass treated with inoculum of R. solani only. The mechanism of this suppression is unknown, but it appears to be something other than antibiosis or parasitism (7).

Future Directions

Knowledge of *Rhizoctonia* species associated with turfgrasses has increased markedly since 1980, especially taxonomic aspects. Fundamental information on the biology of these fungi is lacking, however. For example, do *Rhizoctonia* species cause root rots of turfgrasses? How effectively do *Rhizoctonia* species function as saprobes in turfgrass thatch and soil? We propose the following areas as potentially productive for future research:

- 1. A comparative study of the potential of different species and strains of *Rhizoctonia* to induce root rots in various species of turfgrasses;
- 2. A study of saprobic growth and survival of species and strains of *Rhizoctonia* in turfgrass thatch and soil;
- 3. An investigation of possible pathological and ecological differences among anastomosis groups and subgroups of *R. solani* associated with turfgrasses; and
- 4. Further studies on the pathology, ecology, host range, and anastomosis groups of *R. zeae* and *R. oryzae* in North America.

Literature Cited

1. Bateman, D. F. 1970. Pathogenesis and disease. Pages 161-171 in: Biology and

- Pathology of *Rhizoctonia solani*. J. R. Parmeter, Jr., ed. University of California Press, Berkeley.
- Beard, J. B. 1982. Turf Management for Golf Courses. Burgess, Minneapolis.
- Boerema, G. H., and Verhoeven, A. A. 1977. Checklist for scientific names of common parasitic fungi. Ser. 2b. Fungi on field crops: Cereals and grasses. Neth. J. Plant Pathol. 83:165-204.
- Bostock, R. M., and Stermer, B. A. 1989. Perspectives on wound healing in resistance to pathogens. Annu. Rev. Phytopathol. 27:343-371.
- Burpee, L. 1980. Rhizoctonia cerealis causes yellow patch of turfgrass. Plant Dis. 64:1114-1116.
- Burpee, L. L. 1980. Identification of Rhizoctonia species associated with turfgrass. Pages 25-28 in: Advances in Turfgrass Pathology. B. G. Joyner and P. O. Larsen, eds. Harcourt Brace Joyanovich, Duluth, MN.
- Burpee, L. L., and Goulty, L. G. 1984. Suppression of brown patch disease of creeping bentgrass by isolates of nonpathogenic *Rhizoctonia* spp. Phytopathology 74:692-694.
- Burpee, L. L., Sanders, P. L., Cole, H., Jr., and Sherwood, R. T. 1980. Anastomosis groups among isolates of *Cerato*basidium cornigerum and related fungi. Mycologia 62:689-701.
- Burpee, L. L., Sanders, P. L., Cole, H., Jr., and Sherwood, R. T. 1980. Pathogenicity of *Ceratobasidium cornigerum* and related fungi representing five anastomosis groups. Phytopathology 70:843-846.
- Burton, R. J., Coley-Smith, J. R., and Wareing, P. W. 1988. Rhizoctonia oryzae and R. solani associated with barley stunt disease in the United Kingdom. Trans. Br. Mycol. Soc. 91:409-417.
- Couch, H. B. 1973. Diseases of Turfgrasses. Krieger, New York. 348 pp.
- Couch, H. B. 1985. Turfgrass (several cultivated spp.). Plant Dis. 69:672-675.
- Dale, J. L. 1978. Atypical symptoms of Rhizoctonia infection on zoysia. Plant Dis. Rep. 62:645-647.
- Haygood, R. A., and Martin, S. B. 1990. Characterization and pathogenicity of species of *Rhizoctonia* associated with centipedegrass and St. Augustinegrass in South Carolina. Plant Dis. 74:510-514.
- Hurd, B., and Grisham, M. P. 1983. Rhizoctonia spp. associated with brown patch of Saint Augustinegrass. Phytopathology 73:1661-1665.
- Kataria, H. R., and Gisi, U. 1989. Recovery from soil and sensitivity to fungicides of *Rhizoctonia cerealis* and *R. solani*. Mycol. Res. 92:458-462.
- Lewis, J. A., and Papavizas, G. C. 1975. Survival and multiplication of soil-borne plant pathogens as affected by plant tissue amendments. Pages 84-89 in: Biology and Control of Soil-Borne Plant Pathogens. J. W. Bruehl, ed. American Phytopathological Society, St. Paul, MN.
- Lipps, P. E., and Herr, L. J. 1982. Etiology of *Rhizoctonia cerealis* in sharp eyespot of wheat. Phytopathology 72:1574-1577.
- Madison, J. H. 1971. Practical Turfgrass Management. Van Nostrand Reinhold, New York. 466 pp.
- Marshall, D. S., and Rush, M. C. 1980.
 Relation between infection by Rhizoc-

- tonia solani and R. oryzae and disease severity in rice. Phytopathology 70:941-946
- 21. Marshall, D. S., and Rush, M. C. 1980. Infection cushion formation on rice sheaths by Rhizoctonia solani. Phytopathology 70:947-950.
- 22. Martin, S. B., Campbell, C. L., and Lucas, L. T. 1983. Horizontal distribution and characterization of Rhizoctonia spp. in tall fescue turf. Phytopathology 73:1064-1068.
- 23. Martin, S. B., and Lucas, L. T. 1984. Characterization and pathogenicity of Rhizoctonia spp. and binucleate Rhizoctonia-like fungi from turfgrasses in North Carolina. Phytopathology 74:170-175.
- 24. Martin, S. B., Lucas, L. T., and Campbell, C. L. 1984. Comparative sensitivity of Rhizoctonia solani and Rhizoctonia-like fungi to selected fungicides in vitro.
- Phytopathology 74:778-781. 25. McNabb, R. F. R., and Talbot, P. H. B. 1973. Holobasidiomycetidae: Exobasidiales, Brachybasidiales, Dacrymycetales. Pages 317-325 in: The Fungi. Vol. 4B. G. C. Ainsworth, F. K. Sparrow, and A. S. Sussman, eds. Academic Press, New
- 26. Monteith, J., Jr., and Dahl, A. S. 1932. Turf diseases and their control. Bull. U.S. Golf Assoc. Green Comm. 12:85-187.
- 27. Moore, R. T. 1987. The genera of Rhizoctonia-like fungi: Ascorhizoctonia, Ceratorhiza gen. nov., Epulorhiza, gen. nov., Moniliopsis and Rhizoctonia. Mycotaxon 29:91-99.
- 28. Murray, D. I. L., and Burpee, L. L. 1984. Ceratobasidium cereale sp. nov., the teleomorph of Rhizoctonia cerealis. Trans. Br. Mycol. Soc. 82:170-172.
- 29. Nishimura, S., and Sabaki, M. 1963. Isolation of the phytotoxic metabolites of Pellicularia filamentosa. Ann. Phytopathol. Soc. Jpn. 28:228-234.
- 30. Ogoshi, A. 1985. Anastomosis and intraspecific groups of Rhizoctonia solani and binucleate Rhizoctonia. Fitopatol. Bras. 10:372-390.
- 31. Ogoshi, A. 1987. Ecology and pathogenicity of anastomosis and intraspecific groups of Rhizoctonia solani Kühn. Annu. Rev. Phytopathol. 25:125-143.
- 32. Ogoshi, A., Oniki, M., Sakai, R., and Ui, T. 1979. Anastomosis grouping among isolates of binucleate Rhizoctonia. Trans. Mycol. Soc. Jpn. 20:33-39.
- 33. Oniki, M., Kobayashi, K., Araki, T., and Ogoshi, A. 1986. A new disease of turfgrass caused by binucleate Rhizoctonia AG-Q. Ann. Phytopathol. Soc. Jpn. 52:850-853.
- 34. Oniki, M., Ogoshi, A., and Araki, T. 1986. Ceratobasidium setariae, C. cornigerum, and C. gramineum, the telemorphs of the pathogenic binucleate Rhizoctonia fungi from gramineous plants. Trans. Mycol. Soc. Jpn. 27:147-158.
- 35. Oniki, M., Ogoshi, A., Araki, T., Sakai, R., and Tanaka, S. 1985. The perfect state of Rhizoctonia oryzae and R. zeae and the anastomosis groups of Waitea circinata. Trans. Mycol. Soc. Jpn. 26:189-
- 36. Papavizas, G. C. 1970. Colonization and growth of Rhizoctonia solani in soil. Pages 108-122 in: Biology and Pathology of Rhizoctonia solani. J. R. Parmeter, Jr., ed. University of California Press, Berkeley.

- 37. Parmeter, J. R., Jr., and Whitney, H. S. 1970. Taxonomy and nomenclature of the imperfect state. Pages 7-19 in: Biology and Pathology of Rhizoctonia solani. J. R. Parmeter, Jr., ed. University of California Press, Berkeley.
- 38. Parmeter, J. R. Jr., Whitney, H. S., and Platt, W. D. 1967. Affinities of some Rhizoctonia species that resemble mycelium of Thanatephorus cucumeris. Phytopathology 57:218-223.
- 39. Piper, C. V., and Coe, H. S. 1919. Rhizoctonia in lawns and pastures. Phytopathology 9:89-92.
- 40. Rowell, J. B. 1951. Observations on the pathogenicity of Rhizoctonia solani on bentgrasses. Plant Dis. Rep. 35:240-242.
- 41. Ryker, T. C., and Gooch, F. S. 1938. Rhizoctonia sheath spot of rice. Phytopathology 28:233-246.
- 42. Sanders, P. L., Burpee, L. L., and Cole, H., Jr. 1978. Preliminary studies on binucleate turfgrass pathogens that resemble Rhizoctonia solani. Phytopathology 68:145-148.
- 43. Shurtleff, M. C. 1953. Brown patch of turf caused by Rhizoctonia solani. Ph.D. thesis. University of Minnesota, St. Paul. 177 pp.

- 44. Smiley, R. W. 1983. Compendium of Turfgrass Diseases. American Phytopathological Society, St. Paul, MN.
- 45. Smith, J. D., Jackson, N., and Woolhouse, A. R. 1989. Fungal Diseases of Amenity Turf Grasses. E. & F. N. Spon Ltd., New York.
- 46. Sneh, B., Zeidan, M., Ichielevich-Auster, M., Barash, I., and Koltin, Y. 1986. Increased growth responses induced by nonpathogenic Rhizoctonia solani. Can. J. Bot. 64:2372-2378.
- 47. Talbot, P. H. B. 1965. Studies of 'Pellicularia' and associated genera of hymenomycetes. Persoonia 3:371-406.
- 48. Tu, C. C., and Kimbrough, J. W. 1978. Systematics and phylogeny of fungi in the Rhizoctonia complex. Bot. Gaz. 139:454-
- 49. Voorhees, R. K. 1934. Sclerotial rot of corn caused by Rhizoctonia zeae, n. sp. Phytopathology 24:1290-1303.
- 50. Yang, X. B., Berggren, G. T., and Snow, J. P. 1990. Types of Rhizoctonia foliar blight on soybean in Louisiana. Plant Dis. 74:501-504.
- 51. Zummo, N., and Plakidas, A. G. 1958. Brown patch of St. Augustine grass. Plant Dis. Rep. 42:1141-1146.



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