Rhizoctonia Bare Patch of Cereals

An Australian Perspective

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Rhizoctonia bare patch disease, caused by *Rhizoctonia solani* Kühn AG-8, was first described in Australia in the late 1920s (11,53). Since then, it has been reported in England (7), Canada (1), Scotland (32), and the United States (61). *R. solani* AG-8 has been confirmed as the causal organism in all locations except Canada.

Although bare patch is the most striking form of Rhizoctonia root rot and has probably caused the most interest and controversy, it is not the only syndrome on cereals caused by *R. solani.* There is also the less obvious and less studied root rot on plants outside patches or in crops with no patches (12).

In the 1920s and 1930s, Rhizoctonia bare patch was investigated by Samuel and Garrett (54) and Hynes (12), but subsequently interest waned, with little further research until the mid-1950s. At that time, Flentje and colleagues (9,10) at the Waite Agricultural Research Institute initiated studies on the genetics, infectivity, growth, and survival of the pathogens in the *R. solani* complex. There has been another resurgence of interest in this disease in Australia since the 1970s. This renewed interest, especially in understanding the disease in the field and methods of control, is a result of the increase of this disease (18,22) in all southern cereal-growing regions of Australia (Fig. 1).

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Publication no. D-1996-0516-05F

Rhizoctonia bare patch was a problem in the 1920s and 1930s (11,12,53,54), presumably because the use of horses and old methods of cultivation did not provide sufficient disturbance of the soil for control (see below). The introduction of tractors and the widespread use of multiple cultivations prior to sowing could explain a subsequent drop in importance of this disease. More recently, increases in the disease since the 1970s appear to be due to changes in cultural practices (38) following the adoption by Australian farmers of reduced tillage and soil conservation cropping systems (18). Losses of 25% (22) or more have been recorded, and the disease has the potential to be moderate to severe in many areas across the southern cereal-growing regions of Australia (34).

In this paper we update established knowledge of this disease and review recent work emanating from the resurgence of interest in Rhizoctonia bare patch in Australia. We examine the interesting symptoms of this disease and look at its importance in Australia. We also discuss

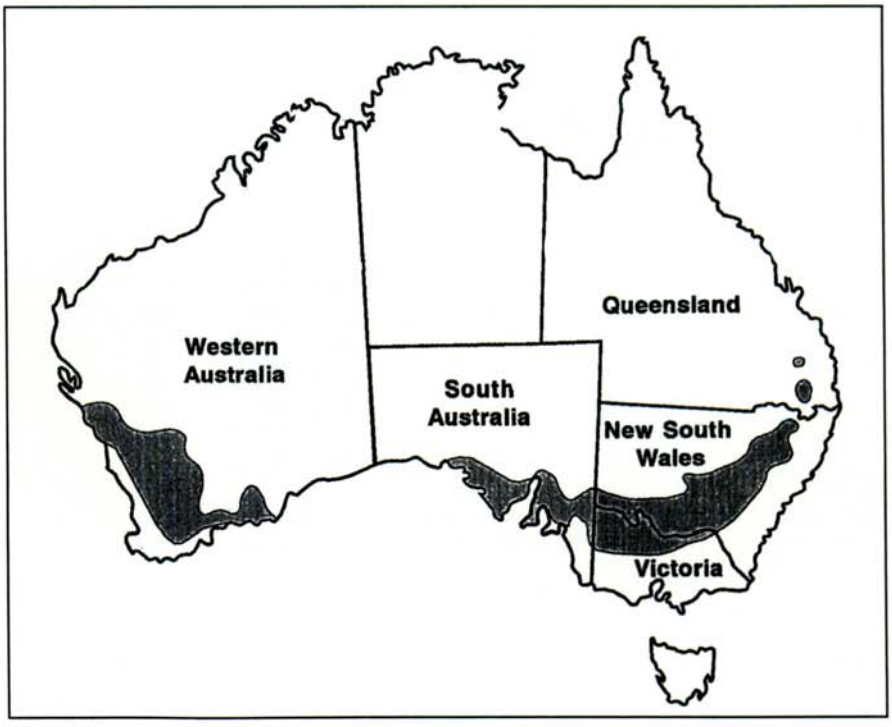

Fig. 1. Map of Australia showing the states and the main cereal-growing regions.

the latest work on disease management and finish with our recommendations for future research.

Symptoms

The most characteristic field symptom of bare patch disease is the occurrence of distinct patches of stunted plants within an otherwise apparently healthy crop (Figs. 2 and 3). In Australia, this disease was originally called stunting disease or no growth patches (53), and subsequently purple patch (11) and bare patch (17). Samuel and Garrett (54) reported that "by far the most common effect of the disease...is the killing of all the plants when still very small, so that by harvest time the 'hole' in the crop is filled with weeds." This death of plants within patches probably led to the name bare patch. Although many plants within severe patches can die, it is rare under present Australian cultural practices for all plants to die. This may be due to better nutritional status and less weed competition than was the case in the 1920s.

Fig. 2. Aerial photograph showing Rhizoctonia bare patch in barley at Esperance, Western Australia.

Fig. 3. Aerial photograph of bare patch in oats at Esperance, Western Australia.

Patches usually have a very distinct margin between the stunted plants in the patches and normal size plants in the surrounding crop (Fig. 4). Patches in crops are most clearly observed from about tillering on but can be detected as early as 2 to 3 weeks after sowing (14) (Fig. 5).

Plants within the patch are stunted and show a range of symptoms associated with poor nutrition and moisture stress. Plants immediately outside the patch usually show no evidence of lack of nutrients or moisture stress. Patch plants may show yellowing of lower leaves, stiff upright habit, rolled leaf blades, spindly growth, failure to produce tillers, and under cold conditions, a dark green coloration and purplish tinge (12) (Fig. 6).

Stunted plants show little or no growth during the growing season in some locations, while at others they show some growth but always remain stunted or spindly compared to the rest of the crop. Plants within patches often remain green longer than the surrounding crop (Fig. 7), possibly due to a delay in maturity (7,54) or to a greater availability of soil moisture in the patch because of less plant competition. Weeds within patches often remain green longer than those in the healthy crop, presumably for the same reason. If the plants within the patches did not suffer from severe stunting, they may recover toward the end of the growing season (Fig. 8). These plants, however, usually have fewer tillers than normal and produce a poor yield.

Patches vary in size from less than 30 cm in diameter to large, irregular areas up to 0.4 ha (11,20,54). They are sometimes circular but more often elongated in the direction of sowing (17,20,44) (Fig. 5). This elongation suggests the pathogen is spread by tillage (presumably by movement of infested material), but there is also the possibility that the spread of the pathogen is enhanced by a host bridge along the drill row. Patches often occur in clusters (Fig. 9) rather than randomly (5,20). Within clusters, the patches often coalesce to form larger, irregular-shaped patches (20).

Plants removed from within patches have a range of root rot symptoms (15,44,49). The seminal roots of young seedlings show water-soaked lesions (54), either confined to one side of the root or often girdling the entire root, causing the cortex to slough off and leave the stele exposed. The stele eventually rots through, leaving a pointed stub or spear tip. The points are usually yellowish brown but often become dark brown in older plants. *R. solani* AG-8 can be easily isolated from early infections, but it is increasingly difficult to isolate as the infections become older (12,14,54). The disease is so severe on some plants that the entire root system is truncated (Fig. 6). Disease severity may vary among the different seminal roots on a plant, and occasionally plants are seen with apparently healthy seminal roots but severely damaged coronal roots.

Although plants within patches usually have considerable root rot, there may be a few plants with apparently intact root systems. Despite this, these plants will remain stunted like the surrounding plants within the patch. The reason for this occasional phenomenon is unknown. This may suggest the involvement of a toxin or abiotic factors in patch formation. Kirkegaard et al. (16) showed that the interaction between the fungus and roots causes greater stunting than can be attributed to the loss in root length alone, and they suggest a root message may be involved. Plants from outside the patches or plants from crops without patches may have considerable root rot but little or no stunting. This is probably due to late infection by *R. solani* AG-8 or infection by other *Rhizoctonia* spp. Our experience suggests that stunting is seen mainly in cereals that have been infected by *R. solani* AG-8 at the seedling stage.

Fig. 4. Bare patch in young wheat crop at Ceduna, South Australia.

Fig. 5. Bare patch in 3-week-old seedling oats at Cungena, South Australia. Distinct edge of patch is marked in the soil. Note elongation of patch in direction of sowing.

Pathogen

In the past, the members of *Rhizoctonia* associated with root disease of cereals in Australia were all considered to be *R. solani* Kühn (teleomorph *Thanatephorus cucumeris* (A.B. Frank) Donk). However, recently, *Rhizoctonias* with a *Ceratobasidium cornigerum* (Bourd.) D.P. Rogers and *Ceratobasidium* spp. teleomorph, and which are mildly pathogenic to wheat, have been associated with diseased cereals (36,44,56).

In Australia, prior to the adoption of anastomosis groups (AG) as a basis for identification of *R. solani*, cultures of *Rhizoctonia* were often grouped into pathogenic strains. Neate and Warcup (43) showed that the root strain (9,14,54) is AG-8. AG-8 is highly pathogenic to roots of cereals and other plant species, and is regularly associated with diseased plants from within Rhizoctonia bare patches (28,36,43).

Identification of *Rhizoctonia* cultures by induction of teleomorphs or determination of anastomosis groups can be slow and is not always successful. Pectic zymogram patterns produced on pectin acrylamide gels provide a rapid and reliable procedure for separating groups within *R. solani* (40,56). Through use of this technique, it has been established that *R. solani* AG-8 is composed of at least five zymogram groups (ZG1-1 to 1-5) (29,40,56). These five groups appear to be very stable (29,43), and some have been isolated from all states in Australia where the disease is found. Two (ZG1-1 and 1-2) have also been detected in the United States (25).

Host Range

R. solani AG-8 has a very wide host range, being recorded on wheat (*Triticum aestivum* L.), oats (*Avena sativa* L.) (53), barley (*Hordeum vulgare* L.) (54), cereal rye (11), and triticale (×*Triticosecale* Wittm.) (18). Other hosts include lupines (*Lupinus angustifolius* L.), rapeseed (*Brassica napus* L.) (18) (Fig. 10), grasses (11,14), subterranean clover (*Trifolium subterraneum* L.) (Fig. 11), lucerne (*Medicago sativa* L.), and annual medics (*Medicago* spp.) (14,17).

Distribution in Australia

Rhizoctonia bare patch and root rot have been recorded in the southern cereal-growing regions of Australia, most of which have a Mediterranean climate (Fig. 1). The incidence and severity, however, differ between regions. A recent estimate of incidence and severity of Rhizoctonia bare patch in Australia (34) indicates that in South Australia the disease is often widespread and has the potential to be very severe. In the other southern states, the disease is observed in localized areas in most seasons and has the potential to be moderate to severe. In subtropical Queensland, the disease is rare.

Economic importance. The economic loss caused by *R. solani* in Australia is unknown. Hynes (12) reported patch areas of 8 to 35% of individual fields, with yields within patches ranging from 14 to 40% of yield outside patches. In one study of patches within a field, MacNish (22) demonstrated that yields within patches ranged from 0 to 0.3 t ha^{-1}; whereas outside the patches the yield was 1.6 to 3.0 t ha^{-1}. The average yield within patches was 8% of that outside. As patches occupied 26% of the field, the estimated reduction in yield was approximately 25%. Rovira (49) reported a yield of 0.2 t ha^{-1} within patches and 1.9 t ha^{-1} outside patches, with patch areas as high as 45% of the crop. MacNish and Fang (27) demonstrated that there was a 17.3-kg ha^{-1} drop in wheat yield for each 1% increase in root rot. They made no attempt, however, to separate the contribution of root rot inside patches and outside patches to yield reduction.

The spectacular symptoms of Rhizoctonia bare patch, especially if viewed from the air (Figs. 2 and 9), may convince farmers that the losses due to patches are greater than is really the case. On the other hand, there is a possibility that in some circumstances, insidious losses from Rhizoctonia root rot on plants outside patches may be greater than the losses within patches.

The costs of attempting to control Rhizoctonia bare patch and root rot are significant. The use of cultivation for control, discussed below, may add directly to the cost through the application of the treatment and indirectly through soil erosion and loss of soil structure.

Fig. 6. Plants from a severe bare patch (left) showing severe root truncation and leaf purpling. Plants from a mild bare patch (right) showing seminal root damage and early signs of root rot on coronal roots.

Fig. 7. Bare patch in barley at Esperance, Western Australia, showing diseased plants in patch remaining green as healthy barley matures.

Fig. 8. Patch crossing boundary between two- and six-row barley at Lake Grace, Western Australia. Note diseased plants about the same height as healthy crop but showing spindly growth.

Fig. 9. Aerial photograph of wheat crop at Ceduna, South Australia, showing clustering of patches and coalescing of patches within the clusters.

Influence of Environment on Rhizoctonia Bare Patch

Soil type. Rhizoctonia bare patch was found traditionally on sandy, calcareous type soils in South Australia (5,14,54). However, since the introduction of reduced-tillage cropping systems, the disease has been reported on a larger range of soil types, including clay loams and red-brown earths (S. M. Neate, *unpublished*). In Western Australia (19), the disease was most severe on sand to loamy sand soils, and in New South Wales it was found on calcium deficient, acid soils (12). De Beer (5) found eight times as many patches in a light soil compared to a nearby heavier soil. A. D. Rovira (*personal communication*) has also shown marked differences in patch expression in long plots transecting three soil types. Root rot severity was similar for all the soil types, but patches were common on the light soil, decreasing in number and finally disappearing as the soil became heavier. Thus, it is likely that while Rhizoctonia root rot is present in heavier soils, it is rarely expressed as the bare patch form.

Soil water. Using a bioassay, Kerr (14) found that maintaining field soil in a moist condition generally resulted in a decline in disease. He tested moist soil monthly for 6 months and compared it to the same soil maintained in a dry state.

Temperature. The optimum temperature for growth of *R. solani* AG-8 in agar media is between 23 and 27°C, with growth occurring between 3 and 33°C (2,54). The pathogenicity of *R. solani* AG-8 on wheat in artificially infested soil maintained at 12, 17, 22, 27, or 32°C varies from almost total destruction of roots at 12°C to only slight damage at 27 and 32°C (54). Using undisturbed soil cores removed from patch and nonpatch soil, Dubé (8) found that the greatest difference in root damage between patch and nonpatch soils occurred at 15°C (Fig. 12).

Fig. 10. Bare patch in rapeseed crop at Esperance, Western Australia.

Fig. 11. Bare patch in subterranean clover at Esperance, Western Australia. Clover within patch senesced before healthy clover.

Disease Management

At present, there are no completely satisfactory methods for controlling Rhizoctonia bare patch and root rot, and what methods are available should all be used simultaneously for best effect. The various suggested methods of reducing the impact of this disease are briefly reviewed below.

Soil disturbance and cultivation. Wheat seedlings grown in undisturbed cores of soil removed from bare patches have root rot symptoms similar to those on field plants removed from patches (8). However, if soil from a patch is thoroughly mixed prior to sowing wheat, there is little or no root rot (14). MacNish (19) used soil cores to demonstrate that shallow mixing (0 to 2.5 cm) had little effect; whereas deep mixing (5 cm or greater) reduced root rot. As most of the inoculum of *R. solani* AG-8 is in the topsoil (0 to 7.5 cm) (5, 26, 37), it appears that mixing could have a deleterious effect on the inoculum.

Field experiments have shown that cultivation reduces Rhizoctonia bare patch and root rot compared to zero or reduced tillage (13,21,35,42,52). MacNish (21) demonstrated that cultivation did not eliminate the pathogen. A return to zero tillage in areas cultivated for the previous three seasons caused an immediate return to patch levels similar to those measured in adjacent zero-tillage areas.

The type and depth of cultivation appear to influence the effectiveness of the treatment. Jarvis and Brennan (13) demonstrated that wide tine points (15 cm), giving good soil disturbance, had little or no effect on patch when used at 3-cm depth. When used at 10-cm depth, however, patch area was reduced by 55% compared to a triple-disk no-till control. In the same experiment, when narrow-point (6 cm) and wide-point (15 cm) tines were compared at 10-cm depth, patch area was 12% with the narrow points and 6% with the wide points. The effects of number of cultivations and time of application are variable (6,13,21,22). The trend appears to be that two cultivations are superior to a single cultivation. In general, the closer the cultivation is to sowing, the better the chance of success. Deep ripping to 27 cm was also shown to reduce patch severity, possibly because the loosened soil may allow faster root growth, which helps the plant to compensate for root damage caused by *R. solani* (13).

Both modified combine drills and modified narrow sowing points were developed in Australia to allow one pass cultivation to 10 cm while sowing seed at 3 cm (13,46). These machine modifications provide cultivation for bare patch control while reducing erosion risks.

The reason(s) cultivation reduces this disease is not completely clear. McDonald and Rovira (31) suggested that cultivation severs the contact between hyphae and their energy source in the organic matter from previous crops (37). Presumably, the pathogen would then have less energy for growth and infection (60). They also suggested that the fragments of hyphae may be "prone to attack by soil micro-organisms and thus less infective." Cultivation may also prevent a buildup of organic matter that would otherwise provide a substrate for *R. solani* (52).

Fig. 12. Wheat seedlings grown for 21 days at 15°C in undisturbed soil cores removed from the center (left), inside edge (center), and outside (right) of a bare patch.

Adequate nutrition. Application of N (as ammonium sulfate, urea, or sodium nitrate) was shown to reduce Rhizoctonia bare patch or root rot (2,5,12,21). Wall et al. (59) showed that the critical level of tissue N for optimum growth of wheat was the same in diseased and healthy plants, although the response to added N was less in diseased plants. There are situations, however, where nitrogen has failed to reduce Rhizoctonia root rot or bare patch (23).

There is little published information on the effect of other nutrients on Rhizoctonia root rot. In the calcium-deficient soils studied by Hynes (12), the application of calcium reduced disease but several other minerals had no effect. Recent work has shown that in zinc-deficient soils, applications of this element can reduce bare patch and root rot, but applications to zinc-sufficient plants did not further reduce disease (57,58).

Rotations. Pasture and crops grown in rotation with wheat may play a role in inoculum multiplication and survival. Samuel and Garrett (54) pointed out that pasture could not be used as a break crop to control Rhizoctonia bare patch because some pasture species are susceptible to this pathogen. The effect of rotation on disease appears to be variable, the effect changing with season and tillage treatment (35,49,52). Neate (35) found generally that wheat following a medic-dominant pasture had a higher root rot rating than wheat following a grass-dominant pasture–wheat rotation. Less Rhizoctonia root rot has also been reported in wheat after certain pulses than in wheat after wheat or pasture (15,49). Patch area was higher when wheat followed grass–medic pasture than when it followed grass-free medic pasture, peas, or wheat (49). MacNish (22) reported fewer patches in pasture than in crop, but found little effect on patch area within the crop regardless of whether wheat followed 1, 2, or 3 years of grass–medic pasture.

Pasture manipulation and fallows. Because closely grazed pastures in the year prior to sowing wheat are reportedly less susceptible to bare patch than pastures carrying large amounts of dry herbage (17), this has been suggested as a method of checking the disease. New volunteer pasture growth after the opening rains of the season in the period just prior to sowing of wheat has been investigated in both South and Western Australia. In South Australia, Neate (35) found that mowing pasture after the break of the season reduced the total length of roots in the soil but did not reduce root rot in that season's wheat crop. However, a 32-day chemical bare fallow in the field, using quick-acting herbicides such as paraquat and diquat, reduced root rot rating and increased yield compared to no treatment (47). In a subsequent field experiment in 1987, Roget et al. (46) confirmed these results when they found that a 3-week chemical fallow reduced the root rot rating from 2.0 to 1.3. Roget et al. (47) proposed that this effect could be due to both a reduction in plant root material that would provide a substrate for *R. solani* and provision of sufficient time for some plant debris to decompose. Volunteer pasture before cropping could also reduce soil N and thus exacerbate root rot (12,21).

MacNish and Fang (27) were unable to show any relationship between short chemical fallow, grass production, and Rhizoctonia bare patch or root rot in two seasons of field experiments in Western Australia. The reasons for the differences between the South Australian and Western Australian results are unknown but could be related to different soil types, soil microbiology, pasture composition, climatic factors (27), or the generally much higher level of disease in their experiments.

It has been suggested that a long fallow is an effective method of controlling this disease (11,54). Prior to the introduction of modern herbicides, fallow involved regular cultivation, so it is impossible to partition out the relative importance of weed removal from the cultivation effect.

Chemical control. There is no economically practical method of chemical control. Several fungicides, the majority triazoles, were tested in experiments to control both natural and artificial epidemics of *R. solani* at seven field sites over two seasons. The chemical treatments were rarely effective at reducing either incidence or severity of the disease (3,4).

Biocontrol agents. Merriman et al. (33) applied *Streptomyces griseus* or *Bacillus subtilis* to wheat seed but found little or no effect on Rhizoctonia root rot. The earthworm *Aporrectodea trapezoides* was shown to have the potential to reduce *R. solani* damage on cereals grown in both clay and sandy soils (55). Unfortunately, the natural levels of the earthworm are very low in areas in which Rhizoctonia root rot is high, thus limiting potential usefulness of this biocontrol agent.

Natural suppression of Rhizoctonia bare patch has recently been identified in the field in Australia (23,45). Levels of disease rose to a peak over 4 or 5 years and then declined over the next few years to negligible levels. At one location where bare patch was recorded, the percent area of patch remained at zero for the following 3 years, and levels of root rot also remained low during that period (45). Research into the mechanisms of the suppression demonstrated that they are biological in origin (62).

Herbicides. Herbicides may have an indirect effect on bare patch by controlling grasses (47), but there are no reports of herbicides causing direct reductions in Rhizoctonia root rot. The herbicides commonly used in direct drilling practice (e.g., glyphosate, paraquat–diquat mixture) appear to have no effect on Rhizoctonia root rot (21,35). There is clear evidence, however, that the herbicide chlorsulfuron can exacerbate Rhizoctonia bare patch in the field when used in alkaline soil types (48,50), and pot tests have shown that both metsulfuron methyl and triasulfuron are also capable of increasing Rhizoctonia root rot (41).

Host resistance. There are small differences in reaction to *R. solani* AG-8, both within and between the cereal genera (39), but the differences are too small to be either useful to breeders or as alternative rotations. The usefulness of the differences is also reduced by variation in relative susceptibility to disease between seasons in the field and between controlled-environment and field tests (39).

Dynamics of Rhizoctonia Bare Patch

The dynamics of Rhizoctonia bare patch, including explanations for patch initiation, expansion, demise, coalescing, and changes in configuration between seasons, were reviewed by MacNish (24). In summary, he concluded that patches are caused by *R. solani* AG-8 and that each patch is the result of a single isolate colonizing the soil from a single infection focus. His review found no support for an aboitic cause for Rhizoctonia bare patch. Results from fumigation experiments also supported the claim that patches are of a biological nature (51). His review found only very limited support for the contention that *Rhizoctonias* other than AG-8, or other fungi, cause Rhizoctonia bare patch (44).

MacNish (24) proposed that changes in patch configuration between seasons can be attributed to differential growth rates of the colonizing pathogen, tillage effects, coalescing of patches, and decline or demise of patches. He felt, however, that these elements of patch dynamics could not explain all of the changes in patch configuration between seasons. He suggested that some changes can be explained by differences in seasonal suppressiveness. Under this proposal, interaction between changing levels of suppressiveness between seasons and the level of inoculum potential of the pathogen in each patch of colonized soil causes changes in the number and severity of patches. MacNish (24) proposed that "in a very suppressive season, those patches of soil colonised by a pathogen with a low inoculum potential will be hidden and will not be expressed as patches in the crop. Only those patches of soil colonised by a pathogen with a very high inoculum potential will be evident as patches in the crop and these may be expressed as mild patches. In a very conducive season, however, all the patches will be seen in the crop with a range from mild through to severe patches." He envisages that this

changing suppressiveness is controlled by seasonal changes and is not a general microbiological suppressiveness (45,62).

Outlook

An interesting aspect of work on Rhizoctonia root rot in Australia is the apparent contradictory observations of the ecology and epidemiology of the disease. In the early years of study of the disease, this was in part due to the lack of understanding of the existence of interspecific groups of *R. solani* with different pathogenic and ecological characteristics. As disease expression is a function of the effect of environment on both the host and the pathogen, an opportunist like *Rhizoctonia* that can tolerate a wide range of environmental conditions and quickly adjust from the role of saprophyte to parasite is well-suited to take advantage of any situation. More research is needed on the basic biology of the pathogen and on feasible approaches to control if the present unsatisfactory methods of controlling bare patch are to be improved. Although *R. solani* AG-8 causes Rhizoctonia bare patch and root rot, it is likely that changes in incidence and severity of this disease involve a complex interaction with other factors (24). These interacting factors need to be taken into account if a satisfactory control strategy is to be developed.

More specifically, we believe the following areas of research require attention.

Quantification of the extent of naturally suppressive soils. The extent of soils naturally suppressive to Rhizoctonia bare patch, the nature of the suppressiveness, and the conditions that led to its development need to be established. This may lead to management practices that enhance natural suppression as an alternative or additional means of control.

Identification and testing of foreign genes resistant to *Rhizoctonia* spp. Transgenic plants with resistance to anastomosis groups other than AG-8 have been developed. Technology is now developing to produce and screen transgenic cereals using the currently available genes, and further resistant genes may be obtained from other plant species and microorganisms. In conjunction with this, there is a need to establish the genetic diversity of field isolates of *R. solani* AG-8 to ensure the stability of resistance in transgenic resistant plants.

Development of molecular probes. There is a need for a wider range of specific molecular probes that can differentiate anastomosis groups and possibly even populations of specific anastomosis groups that are of interest, for example, populations of high and low virulence. This should be done in conjunction with further development in the speed, ease, and sensitivity of the current technique that quantifies *Rhizoctonia* spp. in soil using molecular probes (30).

Optimization of the current tillage techniques. Although it is well-established that cultivation has major effects on Rhizoctonia bare patch, further research is required on the optimization of the current tillage techniques. This may be achieved by defining the optimum timing and intensity of cultivation in relation to environmental factors such as rainfall prior to sowing.

A better understanding of patch dynamics. Despite several decades of investigation, the cause(s) of the development and decline of a patch of stunted *Rhizoctonia*-infected plants is still not clear. The relationship between patch development and factors such as genetic diversity or soil populations of the fungus, or soil type is also not clear.

Acknowledgments

We are pleased to acknowledge the continued funding support of the Grains Research and Development Corporation of Australia.

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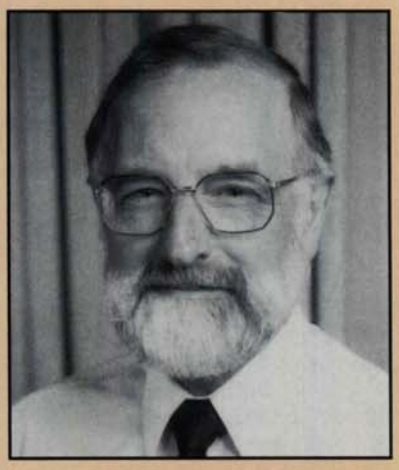

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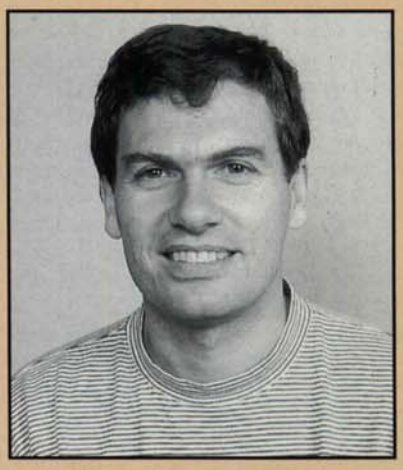

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