Reaction of a Resistant Breeding Line and Susceptible California Rice Cultivars to *Sclerotium oryzae*

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ABSTRACT

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Resistance to *Sclerotium oryzae* (= *Magnaporthe salvinii*), the causal agent of stem rot of rice, was found in a wild species of *Oryza* (*O. rufipogon*) and incorporated into the high-yielding line, 87Y550. *S. oryzae* isolates were screened against this line and two susceptible cultivars. No isolate was able to differentially attack the resistant line. There were significant year, isolate, cultivar, and year × cultivar effects for both stem rot score and number of sclerotia per tiller ($P = 0.05$). Cultivars differed in stem rot score and number of sclerotia formed per tiller. There was a significant year × isolate effect for stem rot score ($P = 0.05$) but a slightly less significant effect for year × number of sclerotia per tiller ($P = 0.1$). Variability due to year effects was greater than that attributable to *S. oryzae* isolates. Stem rot score and number of sclerotia per tiller were highly correlated ($r^2 = 0.74$–0.78, $P < 0.001$). If the stem rot score was less than 3 (top four leaf sheaths healthy), no sclerotia were produced. Sclerotial production was greatest in leaf sheaths 2 and 3 (counting from the flag leaf down) and the culm.

Stem rot, caused by *Magnaporthe salvinii* (Cattaneo) Krause & R. K. Webster (sclerotial state = *Sclerotium oryzae* Cattaneo, conidial state = *Nakataea sigmoidea* (Cavara) K. Hara), occurs worldwide and is a major disease of rice. Field losses have been estimated as high as 80% (25). The disease is now widespread in California (33), and yield reductions of 12–22% under inoculated and 6–23% under natural conditions have been documented (13,20). Yield losses have been correlated with numbers of sclerotia in the seedbed at planting time, with amount of applied artificial inoculum, and with stem rot severity ratings at harvest (20). The disease increases proportionally to the amount of inoculum in the seedbed but levels off when infestation levels exceed those required for maximum infection (18,20). Sclerotia are the principal survival structures of *S. oryzae*, are long-lived in the soil, and are primary inoculum (5,20,32). Conidia and ascospores are produced approximately 60 and 100 days after planting, respectively, but apparently do not reduce yield within seasons (20). They may, however, initiate secondary infections, which in turn may increase the level of overwintering sclerotia. Stem rot is a late-season disease, developing rapidly only after plants shift to the reproductive stage and most rapidly around physiological maturity (3,10,11, 23,24).

Inheritance of resistance to *S. oryzae* in two United States cultivars was shown to be quantitative (7). *Oryza rufipogon* W. Griffith, accession 100912 of the International Rice Research Institute, is more resistant than California cultivars (10,11). At least two genes control inheritance; they are partially dominant, and resistance is of the rate-reducing type (10,24). *O. rufipogon* accession 100912 was also more resistant than Italian cultivars when exposed to *S. oryzae* isolates in Italy (M. Moletti, *personal communication*). Recently, high-yielding lines with resistance to *S. oryzae* derived from *O. rufipogon* have been developed in California (S. T. Tseng, C. W. Johnson, K. S. McKenzie, and J. Oster, *unpublished*). Cultivars reported to have high resistance elsewhere (25,27) had no better resistance than California cultivars when grown in California (J. Oster, *unpublished*).

Pathogenic specialization has been reported in *S. oryzae* (12,14,22). One report (12) describes specialization in *S. oryzae* when tested against cultivars of varying genetic backgrounds. Five cultivars were highly resistant to all isolates of the fungus. One of these, Caloro, is progenitor to all current medium- and short-grain California cultivars (21). *S. oryzae* is heterothallic, and the sexual stage has been found in California rice fields. Sexual recombination could produce races adapted to resistant cultivars (19). An evaluation of 69 isolates of *S. oryzae* for virulence showed that intermediate virulence was most common (9), implying that isolates with extremes of virulence were less fit. But predominance of isolates of intermediate virulence was apparently not a result of growth rate, sporulation rate, or sporulation capacity when measured on agar or water (9). Wide variation in virulence existed among *S. oryzae* isolates, and transgressive segregation for virulence occurred in progeny when certain isolates were mated (7).

The objectives of this research were to compare sclerotial production in a high-yielding resistant breeding line with production in current susceptible cultivars and to evaluate whether this resistance is maintained when comparing a resistant line with susceptible cultivars for disease levels caused by different California isolates of *S. oryzae*.

MATERIALS AND METHODS

Rice culture. All research was conducted at the Rice Experiment Station, Biggs, California, on Stockton clay adobe soil. Standard cultural practices for rice production in California were followed (26), except as noted below. In 1988, 224 kg/ha of 16-20-0 plus 616 kg/ha of ammonium sulfate were preplant incorporated (165 kg of N/ha), and 160 kg/ha of ammonium sulfate (34 kg of N/ha) were topdressed by airplane 50 days after seeding. In 1989, practices were similar, except 280 kg/ha of urea were preplant incorporated (166 kg of N/ha) in place of ammonium sulfate, and topdressing occurred 54 days after seeding.

In 1988 and 1989, areas enclosed by 2.44-m-diameter aluminum rings were divided into thirds and sown with 35 g of each of the California cultivars M-101 (CI 9970), M-201 (CI 9980), and the breeding line 87Y550. Historically, the very early-maturing M-101 has been more susceptible (higher stem rot score) than other cultivars, and the early-maturing M-201 has been less susceptible than other cultivars. The early- to intermediate-maturing 87Y550 is a high-yielding breeding line with stem rot resistance derived from *O. rufipogon*, a wild relative of cultivated rice (11). Soil was raked to cover the seed, flooded to saturation, and drained to promote seed germination. The grass herbicide Bolero (thiobencarb) was sprayed on the soil at 4.5 kg a.i./ha 18–24 days after seeding. Fields were then flooded for the remainder of the growing season. The broadleaf herbicide MCPA was sprayed at 0.84 kg a.i./ha in 1988, and the broadleaf herbicide Londax (bensulfuronmethyl) was broadcast at 0.07 kg a.i./ha in 1989.

Inoculation. Inoculum production techniques involved growth of single-spore subisolates on autoclaved rough rice medium and harvest with a grain sheller and screens of various pore sizes (20,24). Inoculum consisted almost

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entirely of sclerotia. Cultures were prepared for long-term storage by growing on a soil–wheat bran medium, drying, and placing in a freezer at 0 C (6). In 1988 and 1989, nine *S. oryzae* isolates were used that had been collected from nine separate fields in Butte County, California. Two single-spore subisolates derived from conidia were taken from each isolate to test for possible variability within isolates. A highly virulent, similarly single-spored reference isolate (D-30) derived from a conidium (7,9) and an uninoculated (but naturally infested) check were included, for a total of 20 treatments. Each ring plot was inoculated by hand with 50 ml of sclerotia (12.5 g or 9.4×10^5 sclerotia) of one single-spore subisolate at both 62 and 76 days after seeding in 1988, but 65 and 79 days after seeding in 1989. Plots were inoculated twice to ensure presence of high inoculum levels at the sensitive early internode elongation stage for cultivars of different maturities (20). Sclerotia dispersed on the water surface and contacted plants at the waterline.

Disease evaluation. In 1988 and 1989, treatments were replicated three times in a factorial design, with isolates as the main plots, subisolates as the subplots, and cultivars as sub-subplots. Fifteen tillers per sub-subplot replicate were taken at flowering and at 35 and 77 days after flowering. They were frozen until evaluation. The final sampling was delayed until plants were completely dried out, and further within-season sclerotial formation was unlikely to occur. Plants were rated for stem rot severity on a scale of 0–10, where 0 represented no infection, and 10 represented culm penetration and tiller death (20,24). For all sampling times, stem rot score and numbers of sclerotia per tiller were recorded. In addition, number of lesions per tiller and lesion size were recorded for samples taken at flowering. For each year, all sclerotia were counted in situ for each leaf sheath and culm with the aid of a binocular microscope equipped with an eyepiece grid. Results were subjected to analysis of variance (2).

RESULTS

Sampling time and disease evaluation. Figures 1 and 2 illustrate sclerotial formation on three cultivars in 1988 and 1989, respectively. Note the rapid increase in sclerotial formation on M-101 and M-201 as plants matured and senesced (35–77 days after flowering), as previously reported (24). The increase for 87Y550 was considerably slower. Stem rot score increase was similar to that of sclerotia per tiller. Neither lesion number per tiller (range of 0.5–1.5) nor lesion size at flowering (range of 0.2 cm × 0.4 cm to 0.9 cm × 3.0 cm) correlated with stem rot score nor sclerotia per tiller at 35 or 77 days after flowering (*data not shown*).

Interaction of *S. oryzae* with cultivars. Disease developed more in 1989 than 1988, as reflected by numbers of sclerotia formed per tiller (Figs. 1 and 2), and reached highest levels by the final sampling date. In 1988, M-101 and M-201 did not differ significantly in number of sclerotia produced, but both had 4.0–4.5 times more than 87Y550 by 77 days after flowering. In 1989, M-101 had 4.4 times and M-201 had 2.4 times more sclerotia than 87Y550. This produced a significant year × cultivar interaction for M-101 and M-201. Results were similar for stem rot score.

The most prolific isolate (number 7) produced an average of 2.7–2.9 times as many sclerotia as the least prolific isolates (numbers 1 and 2) (Fig. 3). Stem rot score followed a similar pattern, with isolate 7 producing an average score of 7.6 versus 5.6 and 5.0 for isolates 1 and 2, respectively. There were significant year × isolate interactions for isolate 5 and the naturally infested check for both stem rot score and sclerotial production (Fig. 3). There were no significant differences among subisolates, so data were pooled for a total of six replications per isolate.

A significant isolate × cultivar interaction occurred only in 1989 for sclerotial production (Fig. 3), but there was no significant interaction for stem rot score. Isolates 6, 9, and the uninoculated (but naturally infested) check did not produce significantly more sclerotia on M-101 than M-201. All other isolates produced

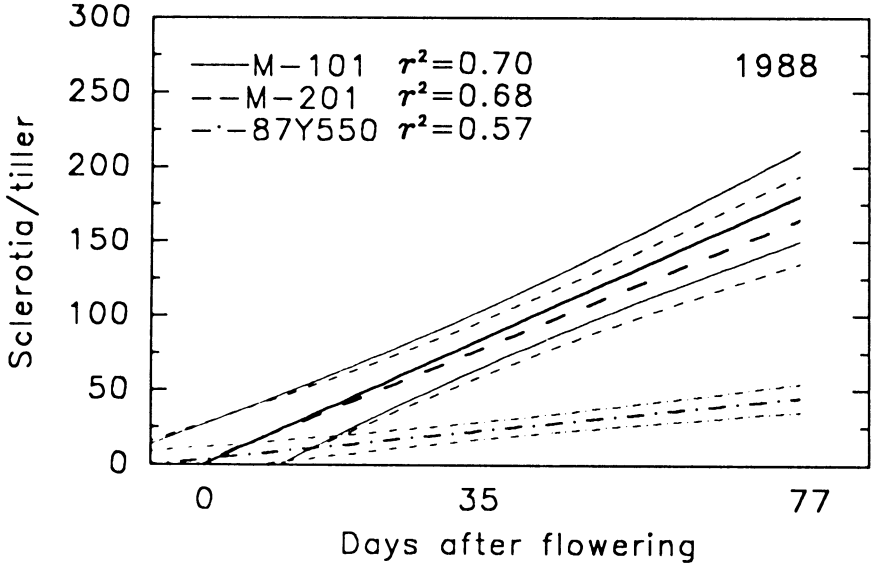

Fig. 1. Progress of sclerotial formation with time in 1988. There are 33 data points per regression (three sampling times, 10 isolates plus the check). The bold regression line for each cultivar is surrounded by thinner lines representing a 95% confidence interval. Differences in regression line slopes and levels are significant ($P < 0.0001$) for M-101 and M-201 versus 87Y550.

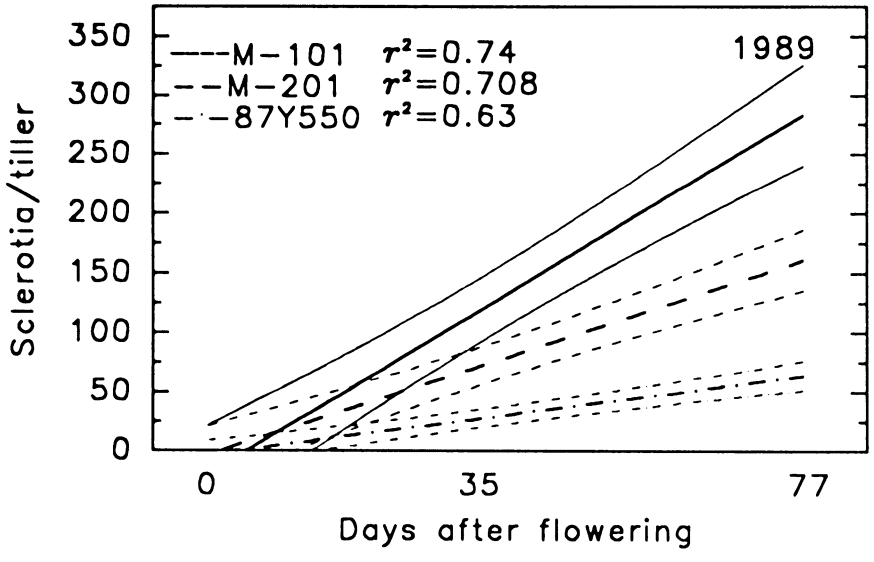

Fig. 2. Progress of sclerotial formation with time in 1989. There are 33 data points per regression (three sampling times, 10 isolates plus the check). The bold regression line for each cultivar is surrounded by thinner lines representing a 95% confidence interval. Differences in regression line slopes and levels are significant ($P < 0.0001$) for all cultivars.

more sclerotia on M-101 than M-201. Year × isolate and year × cultivar interactions were consistent from year to year, larger, and more significant than the isolate × cultivar interaction (Figs. 1–3).

Correlation of stem rot score with sclerotial production. For both cultivars and line 87Y550, there were high correlations between stem rot score and number of sclerotia per tiller (r^2 = 0.74–0.78) (Fig. 4). High correlations for a wider range of cultivars have also been reported (24). Slopes and intercepts of the regression lines reflected susceptibility of the cultivars. Tillers with low stem rot scores (less than 4) produced very few sclerotia.

Location of sclerotia. Sclerotia were found only in sheaths 2–4 and in the culm (Table 1). Leaf sheath 1 was above the zone colonized by the pathogen. Sheaths 2 and 3 and the culm contained, on average, more than 95%of all sclerotia. Total number of sclerotia per tiller was less in 87Y550 than in cultivars.

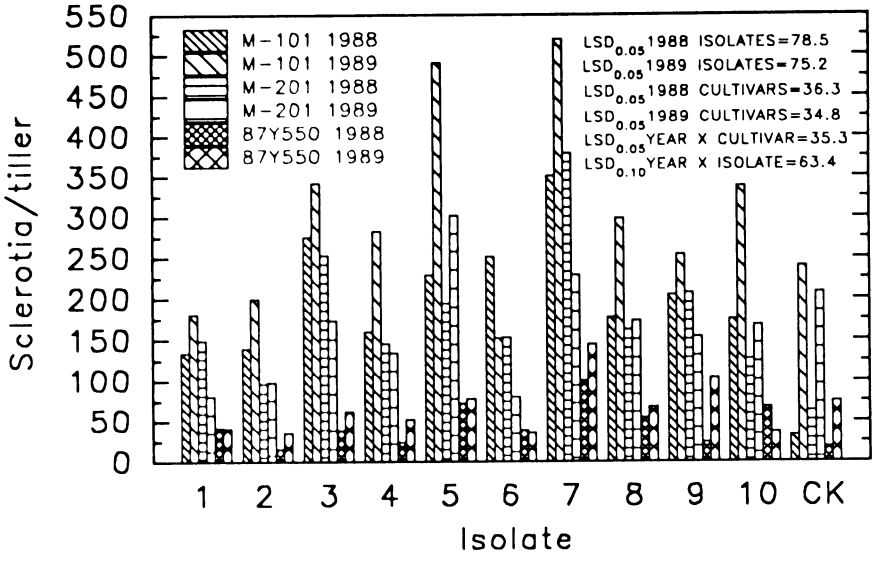

Fig. 3. Sclerotial production by *Sclerotium oryzae* isolates on M-101, M-201, and the resistant line 87Y550 77 days after flowering. Each isolate is represented by two subisolates for each year, except isolate 10 (reference isolate D-30) and the uninoculated (naturally infested) check (CK). For isolates, $P \leq 0.0003$ for both years. For cultivars, $P \leq 0.00001$ for both years. For the year × cultivar interaction, $P < 0.00001$. For the year × isolate interaction, $P = 0.07$.

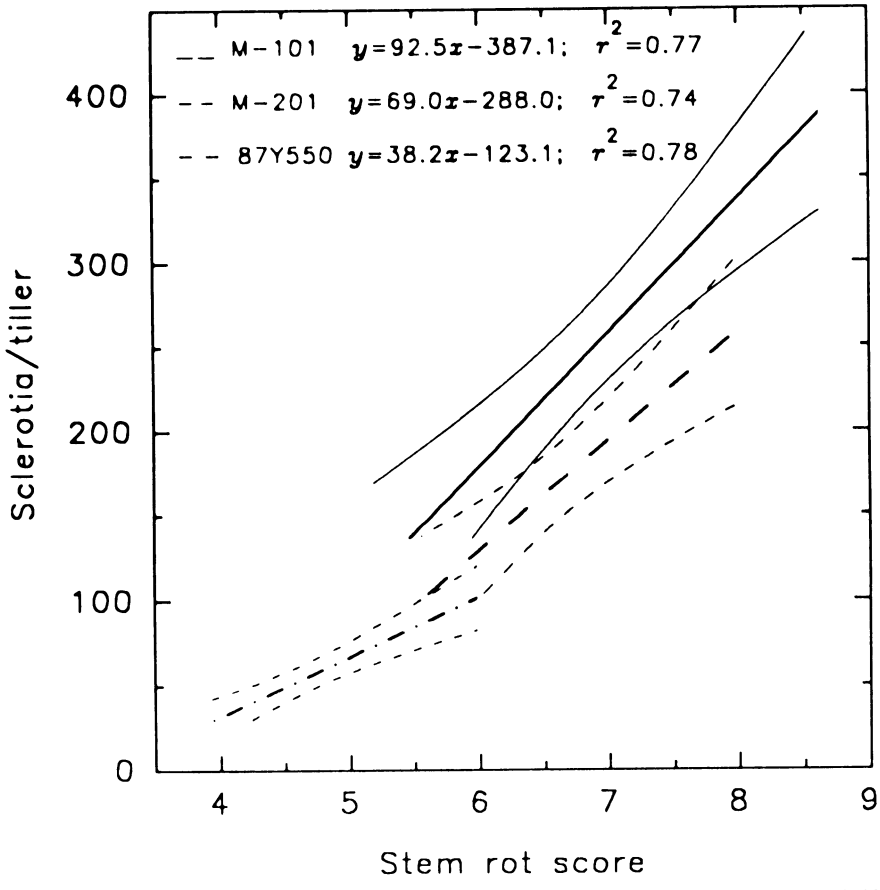

Fig. 4. Correlation of stem rot score with numbers of sclerotia per tiller for 1988 and 1989. Ratings were taken 35 and 77 days after flowering and represent the average of all subisolates and years for each *Sclerotium oryzae* isolate (10 isolates plus the check, 2 yr for a total of 22 data points per regression). The bold regression line for each cultivar is surrounded by thinner lines representing a 95% confidence interval. Differences in regression line slopes and levels are significant ($P < 0.0001$).

DISCUSSION

For the 1988 and 1989 experiments, year effects in interactions with isolates and cultivars were large, applied to both stem rot score and sclerotial production, and were more significant than inconsistent (1989 only) isolate × cultivar effects. Nongenetic variation played a larger role in data variability than genetic makeup. Part of this variability could be due to supplying nitrogen in different forms from year to year. Urea was applied preplant in 1989; 16-20-0 or ammonium sulfate, in 1988. Londax was applied in 1989, and MCPA in 1988. Londax enhances nitrogen uptake by the plant (D. M. Brandon, *personal communication*), and increased nitrogen has been shown to increase stem rot severity (15,16,17). MCPA, especially if applied late (close to panicle initiation), can increase stem rot severity (34). In this research, MCPA was applied late in the season in 1988, and injury to rice plants was noted, but greater disease severity was experienced in 1989 on M-101 and 87Y550. This could have been due to more nitrogen uptake as a result of the use of Londax. Disease was more severe on M-101 than M-201 in 1989 but not in 1988. This could have resulted in lesser nitrogen uptake by M-101 as a result of MCPA injury and therefore less stem rot damage. M-201, being of later maturity, may not have been so affected. If so, this would be contrary to existing research findings (34).

Experiments were conducted in different naturally infested fields each year. The fields were, however, very close to each other geographically and had similar management histories. Disease in the uninoculated check was greater in 1989 than in 1988. However, there were still highly significant differences among isolates in virulence and the ability to produce sclerotia above this background level of disease for both years. In a few instances, stem rot score or sclerotial production was lower than that of the check. The lack of significant differences among subisolates may indicate low variability of this fungus, but the number of subisolates representing each isolate was small.

The fungus was not able to differentially overcome the resistance of 87Y550. Even though there was a significant isolate × cultivar interaction in 1989, it consisted only of differences in sclerotial formation on M-101 and M-201, but no differences were found in 1988. No interaction was observed for stem rot score. Maybe large differences in isolate × cultivar interactions should not be expected. Very few cultivars and stem rot isolates were used, M-101 and M-201 are closely related, and the research was performed only in and with isolates from Butte County. Reports in which larger numbers of cultivars with more diverse origins were tested indicated a larger amount of pathogenic specialization in the stem rot fungus (12,14). Since stem rot has been introduced into California (31), maybe only a portion of its pathogenic potential is present. But one of the studies (12) found that Caloro, a progenitor to all present California short- and medium-grain cultivars, was equally resistant to all stem rot isolates. Moletti (*personal communication*) found *O. rufipogon* (the source of resistance in 87Y550) highly resistant to stem rot in Italy. In California, then, the wild species resistance may be broadly based, and it has been transferred into backgrounds likely to have broadly based but lesser resistance. The inheritance of the wild species resistance is quantitative (10), and the lower level of resistance in susceptible California cultivars may also be quantitative (7), making breakdown of resistance less likely. However, the presence of a sexual stage (19) and a report of transgressive segregation for virulence in the stem rot fungus (7) are reasons for concern. In these experiments, variation for stem rot score (virulence) and sclerotial-forming ability were observed, confirming previous reports (7,8,9). The most virulent isolate produced 2.3–6.3 times as many sclerotia (depending on cultivar) and a 15–31% difference in stem rot score (highest score − lowest score/maximum score of 10) compared to the least virulent isolates. This compares with a 3.6-fold difference in sclerotial production on agar (9) and a 16% difference in stem rot score (8) previously reported. However, high correlations between these parameters were observed here, in contrast to previous in vitro tests in California indicating that they were not associated (8). If sclerotial formation is preeminent in determining pathogen fitness, then highly virulent isolates should be the most common in nature. But isolates of intermediate virulence are most frequently found, as determined in seedling tests (9). Therefore, other major fitness mechanism(s) besides sclerotial production must determine the distribution of virulence levels among *S. oryzae* isolates.

Correlations of number or size of lesions at flowering and stem rot score or sclerotia produced per tiller at either 35 or 77 days after flowering were very low. Physiological changes in the plant after initiation of the flowering stage favor pathogen development. Disease evaluations should be performed late in the season, when cultivar differences in stem rot score and sclerotial production are more evident (20,24). In this way, both damage in the current season (stem rot score) and potential damage in future seasons (sclerotial production) could be evaluated. Final sampling was performed when plants were desiccated and further within-season sclerotial production was unlikely. However, some sclerotial production in already-infected straw is possible after rains rewet soil-incorporated straw (5). At this time, sclerotial production may no longer be restricted by host resistance, but low winter temperatures during most of the rainy season and the low saprophytic ability of *S. oryzae* sclerotia restrict colonization of additional straw (4).

Because sclerotia are produced mainly in leaf sheaths 2–4 and in the culm, sclerotial formation will be almost completely suppressed within the growing season if resistance protects this area. Reports from wheat and rice on the relative importance of these sheaths and the leaves they support indicate little yield loss will result if they remain undamaged (1,29). Sheaths and leaves 1 and 2 contribute most to yield. However, for stem rot, sheath 3 should also be protected, since penetration of this sheath can result in stem damage and yield loss (18,20). This level of resistance could severely impair the survival ability of the stem rot fungus, since sclerotia are overwintering structures and initiate the phase of the disease primarily responsible for yield loss within a season. The resistance of the high-yielding line 87Y550 reduced sclerotial production significantly under severe disease pressure. At more normal disease levels, within-season sclerotial formation could be nearly eliminated. However, the effect of this resistance on other spore forms is not known. In brown stem rot of soybeans and Cephalosporium stripe of wheat, even moderate disease resistance significantly reduced yield loss, especially through the interaction of less severe disease symptoms with reduction in overwintering spore load over time (28,30).

Table 1. Location of *Sclerotium oryzae* sclerotia on tillers of three rice cultivars[a]

Cultivar	Year	Sheath 5[b]	Sheath 4	Sheath 3	Sheath 2	Culm	Total
M-101	1988	0.0 (0.0)[c]	0.7 (0.3)	13.8 (6.8)	35.4 (17.1)	156.7 (75.8)	206.4
	1989	0.0 (0.0)	0.3 (0.1)	17.5 (5.9)	59.8 (19.8)	223.4 (74.2)	301.0
	Average	0.0 (0.0)	0.5 (0.2)	15.6 (6.4)	47.6 (18.4)	190.0 (75.0)	253.7
M-201	1988	0.0 (0.0)	4.7 (2.6)	41.8 (22.9)	42.5 (23.2)	93.9 (51.3)	182.9
	1989	0.0 (0.0)	3.0 (1.9)	57.3 (35.5)	59.2 (36.7)	41.7 (25.9)	161.2
	Average	0.0 (0.0)	3.8 (2.2)	49.6 (29.2)	50.8 (30.0)	67.8 (38.6)	172.0
87Y550	1988	0.0 (0.0)	4.4 (9.7)	20.0 (44.0)	9.2 (20.3)	11.8 (26.0)	45.4
	1989	0.0 (0.0)	1.6 (2.4)	20.0 (29.5)	17.8 (26.3)	28.3 (41.8)	67.7
	Average	0.0 (0.0)	3.0 (5.3)	20.0 (35.4)	13.5 (23.9)	20.0 (35.4)	56.5

[a] Seventy-seven days after flowering.
[b] Flag leaf sheath is sheath 1.
[c] Number of sclerotia per tiller, and percent of total sclerotia per tiller (in parentheses). Data are means of six replicates and 10 treatments (nine isolates plus check). LSD for 1988 = 54.0; LSD for 1989 = 52.0; $P = 0.05$.

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