Bacterial Sheath Brown Rot of Rice Caused by *Pseudomonas fuscovaginae* in Latin America

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ABSTRACT

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Many types of symptoms associated with discolored grain have been observed on rice (*Oryza sativa*) sheaths, leaves, and grain in Mexico, Guatemala, Panama, Surinam, Colombia, Peru, and Brazil. These include brown necrotic lesions ranging from small specks to large brown to maroon sheath blotches. Symptoms were also expressed as a brown stripe on the sheath that could extend along the midrib of the leaf lamina for nearly its entire length. Panicles failed to emerge properly from the boot when the flag leaf sheath was severely affected, producing discolored and poorly filled grain. Grain from panicles of badly affected plants showed variable severity of symptom expression. A fluorescent pseudomonad was consistently isolated from affected tissue collected in Colombia and Surinam and reproduced symptoms upon inoculation. The pathogen was identified as *Pseudomonas fuscovaginae*, the causal agent of bacterial sheath brown rot of rice first described in Japan. Comparison of the physiological characteristics of isolates from Latin America, Asia, and Africa, *P. fuscovaginae*, and *P. marginalis* suggests that the designation of *P. fuscovaginae* as a separate species may not be appropriate. The bacterium was seed-transmitted, with seedlings showing symptoms on sheaths and leaves after 10–20 days. Heat treatment at 65 C for 6 days eradicated the pathogen from infected seed. The possibility that this pathogen is a principal causal agent of the dirty panicle disease (grain spotting, *manchado de grano*) is discussed.

Additional key words: glume discoloration, seed treatment

In recent years, necrotic leaf and sheath symptoms and grain discoloration have been observed on rice (*Oryza sativa* L.) in Latin America. Discolored and poorly filled grain cause the most direct economic losses to seed producers and rice farmers. Government agencies are reluctant to certify discolored seed, and mills may levy a substantial discount against moderately or severely spotted and poorly filled grain. Because different fungi may be isolated from discolored grain (4,12,15,17,19,24), farmers apply fungicides beginning at the late boot stage to try to avoid mill discounts. However, there is ample evidence to indicate that fungicide applications offer little or no protection (12,17,19) and thus represent indirect losses.

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The following rice diseases caused by fluorescent *Pseudomonas* spp. affecting the sheath, leaves, panicles, and grain have been reported (15,21):

1. Bacterial stripe (9), caused by *P. syringae* pv. *panici* (Elliott) Young et al (= *P. setariae*), is recognized by dark green water-soaked lesions 0.5–1.0 mm wide on the sheath and may extend up to the full length of the sheath. The lesions turn reddish to dark brown with age. It is reported to affect only seedlings and has been observed in Asia (10).

2. Bacterial sheath rot, caused by *P. oryzicola* Klement, has been reported in Hungary and Asia (15). This disease is characterized by indistinct brown or black spots on the sheaths and stem, with grain discoloration and sterility in severe cases. The sheath spots may elongate, turn dark brown to reddish, and result in necrosis and drying of the entire sheath. Japanese isolates were found to be similar to *P. marginalis* (8).

3. Bacterial grain rot, caused by *P. glumae* Kurita & Tobei (nonfluorescent), is characterized by grain discoloration and sterility (15).

4. Bacterial sheath brown rot of rice (21) is characterized by longitudinal brown to reddish brown necrosis 2–5 mm wide extending the entire length of the leaf sheath and blade. Affected sheaths enclosing the panicle may show extensive water-soaking and necrosis with poorly defined margins. Glumes discolor before emerging from such panicles. Grain on affected tillers may be completely discolored and sterile to nearly symptomless with only small brown spots. The disease was first reported to be caused by *P. marginalis* (8), but the pathogen was subsequently renamed as the new species *P. fuscovaginae* and judged distinct from the previously mentioned pathogens (14,15,21). This bacterial disease has been reported from northern Japan and Burundi highlands (Central Africa) and causes appreciable losses (1,21). A disease with identical symptomatology has been reported from Brazil; however, the pathogen was not positively identified (7).

This study was undertaken at the Centro Internacional de Agricultura Tropical (CIAT) to determine to what extent the symptoms described above from Latin American rice are caused by one or more of the bacteria previously believed to be absent from the Americas. Seed transmission and heat therapy studies were also conducted.

MATERIALS AND METHODS

Potential pathogens were isolated from fresh and air-dried grain, leaf

sheaths, leaf blades, and culms collected in Colombia and Surinam after washing 1 hr under tap water. Small sections of affected sheaths and leaves were placed on media or first macerated in sterile distilled water and the resulting suspension poured over the isolation medium. Whole grains were partially embedded in the isolation medium. Isolation media were V-8 juice agar, nutrient agar, and King's B medium. Fungi isolated from the samples were inoculated by spore aspersion (10^5 spores per milliliter) and bacteria by leaf clip, stem puncture, and stem injection (10^9 cfu/ml) at 24–27 C, 100% relative humidity for 72–96 hr. Test-plant seed was treated at 65 C for 6 days before planting. Plants used were of cultivars Bluebonnet 50, CICA 8, Oryzica 1, and Fanny 20, 40, and 60 days old and Oryzica 1 at late booting stage. Characteristics of the pathogenic bacterial isolates (Table 1) were determined by standard microbiological techniques (2,6,13,18,20,23). Flagella number was determined by electron microscopy after first staining with uranyl acetate (18).

To determine if the pathogen is seed-transmitted, discolored seed from affected tillers (taken from the field) were collected from cultivar IAC 1246 plants showing leaf and glume symptoms. Seeds were treated at 27, 35, 45, 50, 55, and 60 C for 4, 24, 48, 96, and 144 hr, then planted in steam-sterilized soil to determine if relatively mild heat therapy could eradicate the pathogen. Symptomless leaves were removed from apparently healthy seedlings grown from infected seed to determine if they harbored the pathogen. These and leaves from seedlings showing longitudinal necrosis of the midrib were washed and placed on King's B medium. Discolored mill grain with 50% contamination by fluorescent *Pseudomonas* spp. and seed from plants showing sheath necrosis and glume discoloration (samples of 40–50 g) were treated with dry heat at 55, 60, and 65 C for 0, 1, 3, and 6 days, placed on King's B medium, and incubated 48 hr at 27 C to determine at which treatment no fluorescent bacteria were recovered. Samples of fluorescent bacteria from untreated lots were tested for pathogenicity.

RESULTS

Bacteria producing fluorescent pigment on King's B medium were consistently isolated from rice plants showing a range of sheath, leaf, and grain symptoms. Leaf and sheath symptoms included longitudinal necrotic, reddish brown stripes extending the full length of the sheath and often the entire length of the leaf lamina (Fig. 1B). Other sheath symptoms were small, brown (1–5 mm) elongated spots that expanded or coalesced to form large indistinct blotches on the sheath and sometimes the culm (Fig. 1C). Symptoms on seedlings were usually limited to brown, water-soaked necrosis on the sheaths. Occasionally, necrotic brown stripes were observed on leaves. Seedlings showing symptoms early in the field often died. When older sheaths were affected, the flag leaf sheath enclosing the emerging panicle sometimes showed water-soaking, blotching, and brown necrosis. In most cases, the glumes of most of the enclosed developing florets were discolored brown and the panicle often only partially emerged from the boot (Figs. 1A and 2). Most florets that did emerge often did not fill normally, and grain was spotted or almost completely discolored and sterile (Figs. 2 and 3). In severe cases, the flag leaf sheath and/or collar became necrotic and dry. Even panicles that emerged normally usually showed spotted or discolored grain. Grain discoloration associated with sheath and leaf symptoms generally was more severe when grain matured under conditions of high humidity and rainfall and/or was from fields with soil nutrient imbalances. Roots often showed brown discoloration and also yielded the pathogen.

Fluorescent bacteria were always isolated from the distinct longitudinal necrosis of the sheath and leaf and associated discolored grain. They were commonly isolated from discolored glumes, and blotchy, necrotic flag leaf sheaths and collars not associated with

Table 1. Comparative characters of 29 pathogenic isolates of the rice bacterial sheath brown rot bacterium from Colombia, Asia, and Africa compared with published characters of *Pseudomonas fuscovaginae* and *P. fluorescens* biovar II and isolates of the two previously reported Latin American bacterial rice pathogens

Character[a]	CIAT isolates of the rice sheath brown rot bacterium	*Pseudomonas fuscovaginae*[b]	*P. fluorescens* biovar II (*P. marginalis*[b])	*Xanthomonas campestris* pv. *oryzae*[c]	"Brown blotch" isolate[d]
Flagellation	>1, polar	>1, polar	>1, polar	1, polar	1, polar
Gram reaction (18)	−	−	−	−	−
Yellow pigment	−	−	−	+	+
Fluorescent pigment (18)	+	+	+	−	−
Arginine dihydrolase (18)	+	+	+	−	−
Gelatin liquefaction (18)	+	+	+	−	−
Lipase (23)	+	+	+	±	−
Oxidase (18)	+	+	+	−	+
Levan formation from sucrose (18)	+	−	+	+	+
Starch hydrolysis (2)	−	(±)	v	+	−
NO_3-reduction (20)	v	−	+	−	+
Acid from					
Sucrose (18)	+	−	+	±	−
Sorbitol (18)	+	v	+	±	−
Inositol (18)	+	−	+	±	−
Raffinose (18)	+	−	ND	±	+
Potato rot (18)	+	−	+	−	−
Carbon source utilization					
Polygalacturonic acid (13)	−	−	+	−	−
2-Ketogluconate (13)	−	−	+	−	−
Pit formation on polypectate gel (18)	−	−	+	ND	ND
Tobacco hypersensitivity (18)	+	ND	ND	+	+
Growth at 41 C	−	ND	−	−	−
Growth at 4 C	−	ND	+	−	−

[a] + = Positive reaction, ± = weakly positive, (±) variable and only weak when positive, − = negative reaction, v = variable reaction among isolates, ND = not done.
[b] Reactions for *P. fuscovaginae* and *P. fluorescens* biovar II taken from references 21 and 16, respectively.
[c] CIAT isolate 1167.
[d] CIAT isolate 1173.

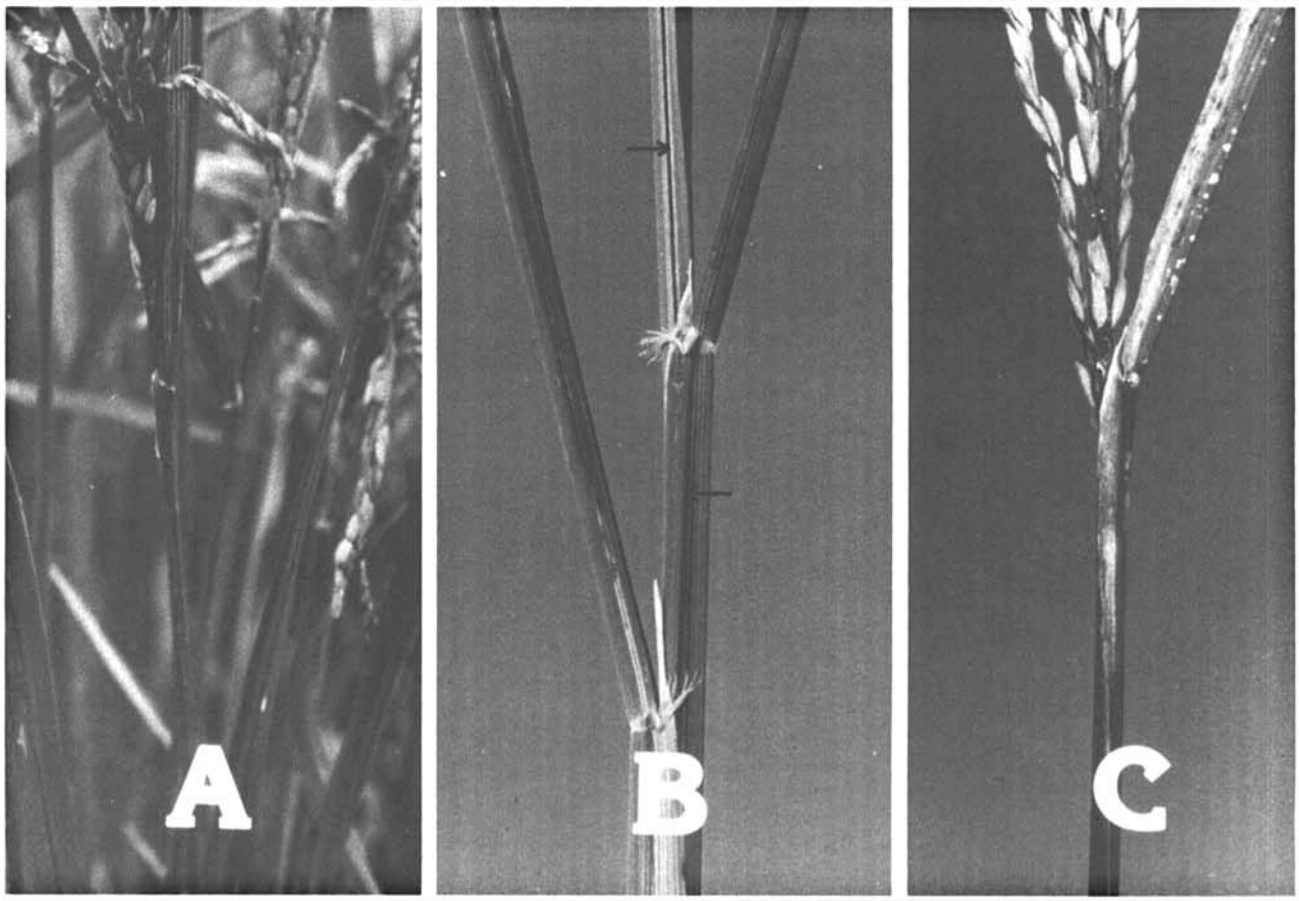

Fig. 1. Leaf and sheath symptoms on plants infected by *Pseudomonas fuscovaginae*. **(A)** Poorly emerged panicle with discolored grain, **(B)** longitudinal necrotic stripe (arrow), and **(C)** sheath rot similar to that caused by *Sarocladium oryzae*.

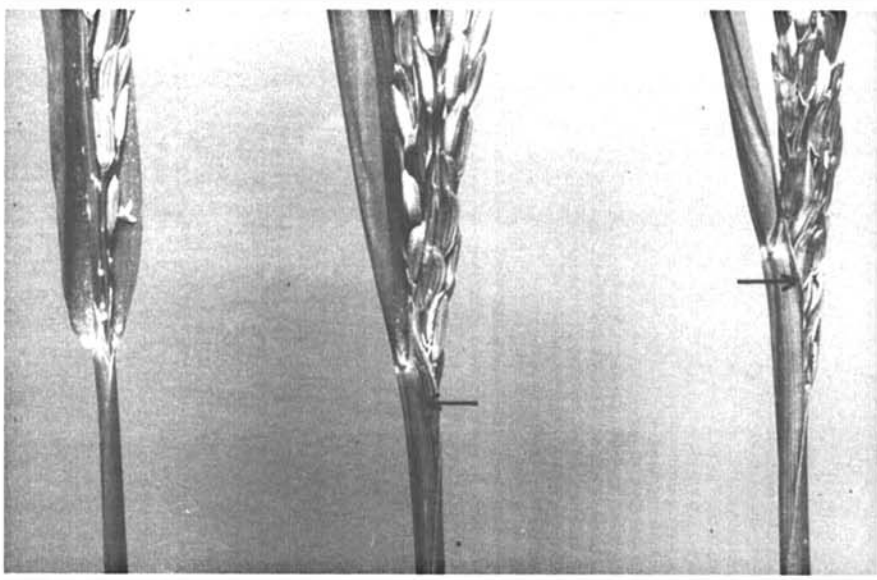

Fig. 2. Sheath and panicle symptoms on rice caused by *Pseudomonas fuscovaginae*. Note poorly emerged panicles and discolored grain. Arrows indicate sheath necrosis.

the pronounced longitudinal leaf and sheath necrosis as well as from relatively clean grain from plants showing sheath blotching and/or necrotic stripe (Fig. 3). Plants inoculated by stem puncture or injection with purified isolates of the fluorescent bacteria showed progressive water-soaking in the inoculated region after 2–4 days. The lesions became necrotic and progressed up and down the inoculated sheaths and stems, occasionally killing the seedlings. Most isolates, including those originally from plants showing only glume discoloration and/or sheath blotching, induced the necrotic stripe on the sheaths and lamina. Leaf clip of young leaves resulted in progressive necrosis and discoloration of the vascular tissue of the inoculated leaf, particularly the midrib. Symptoms were visible after 2–5 days. Plants inoculated at the boot stage by stem puncture and leaf clip developed typical blotching on the flag leaf sheath and severely discolored glumes. The panicles often did not emerge completely. Regardless of the isolate used, these plants never developed the necrotic stripe, although marginal necrosis of the flag leaf and its sheath and/or collar was common.

Some plants inoculated at 40 days were left to flower and produce grain. Bluebonnet 50, which developed the typical necrotic brown stripe on sheaths and lamina of inoculated tillers and on subsequent developing leaves, gradually "outgrew" the symptoms such that the flag leaf showed only very fine discoloration along the midrib and principal veins. However, the glumes were moderately spotted. All of the 50 seeds sampled from the panicle on King's B medium yielded pathogenic fluorescent bacteria.

The symptoms described were observed on commercial and experimental rice in Mexico, Guatemala, Panama, Surinam, Colombia, Peru, and Brazil. Pathogenic isolates were obtained from symptomatic plants and discolored grain from Colombia and Surinam and from

experimental seed from Asia and Africa received in routine germ plasm exchange.

Sheath symptoms of affected plants from the field and those inoculated in the boot stage strongly resemble "sheath rot," attributed to *Sarocladium oryzae* (Sawada) Gums & Hawks. (= *Acrocylindrium oryzae* Sawada) (15) and *Myrothecium verrucaria* (Alb. et Schu.) Ditm. ex Fr. (3). In only one case was *S. oryzae* isolated from the more than 50 samples with sheath blotching and necrosis. In this case, it was only isolated from the sheath and not from the associated discolored grain. The fluorescent bacterium was recovered from both sheath and grain in this sample. *M. verrucarria* was never isolated from symptomatic plants. *Cochliobolus miyabeanus* Ito & Karuibayashi (*Helminthosporium oryzae* Breda de Haan) was never isolated from sheath lesions. It was, however, commonly isolated from spotted grain but caused only local lesions upon reinoculation, with no systemic development.

Colonies of the mobile, bacilliform (2.5–3.5 × 0.5–1 μm) bacterium grown on nutrient agar were cream-colored, circular, and convex with erose margins. The physiological and morphological characteristics of 29 pathogenic isolates from commercial Colombian grain production fields, CIAT experimental plots, and experimental seed from Africa and Asia are compared with those of the other bacterial pathogens of rice already reported in Latin America (11,23), *P. fuscovaginae* and *P. fluorescens* biovar II (*P. marginalis*) (Table 1). The CIAT isolates obviously fall within the genus *Pseudomonas* (16), and if the designation of *P. fuscovaginae* as a separate species is accepted, they may be considered to be *P. fuscovaginae*. However, the CIAT isolates, including these causing the longitudinal necrosis typical of that caused by *P. fuscovaginae*, fall between *P. fluorescens* biovar II (16) and *P. fuscovaginae* in tests considered to be critical by Tani et al (21). The differential characters presented by them were levan formation from sucrose, nitrate reduction, acid-free sucrose, inositol, sorbitol, and raffinose, potato rot, carbon source utilization, and pit formation on polypectate gel. The isolates in this study were similar to *P. marginalis* for levan formation and acid production from the carbohydrates. They were similar to *P. fuscovaginae* for pit formation on polypectate gel and carbon source utilization. Nitrate reduction was variable.

The effects of mild heat treatment on seed transmission via seed from field-infected plants are summarized in Table 2. Symptoms first appeared after 10–20 days as necrotic spots on the sheaths surrounding the first internodes. Typical veinal necrosis appeared after 30–35 days. Many seedlings remained symptomless; however, the pathogen could be recovered from leaf and sheath tissue. Symptoms developed on these plants at varying stages in their development but were most severe on the flag leaf sheath. The rate of seed transmission via freshly harvested seed was very high, as determined by pathogen recovery, occurring in more than 30% of a small (25-seed) sample.

The preliminary results from the seed transmission-heat therapy experiments indicated that seed treated at 60 C for 6 days was pathogen-free. The results of heat treatment of larger contamined seed lots are presented in Table 3. Dry heat treatment at 65 C for 6 days eradicated the bacteria completely from two samples, since no fluorescing bacteria

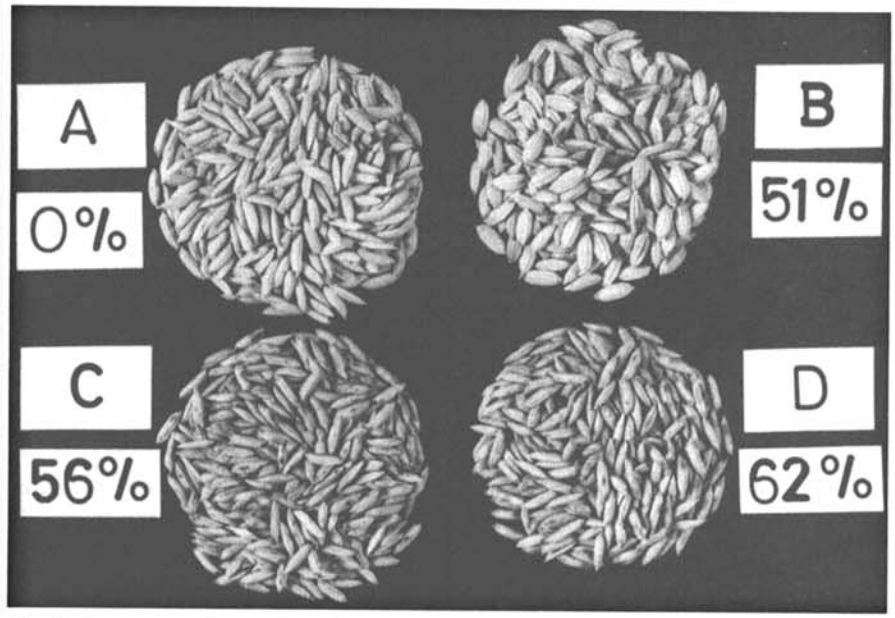

Fig. 3. Comparison of cleaned paddy with 0 and 51–62% seed contaminated with *Pseudomonas fuscovaginae*. A = Oryzica 1, B = IRAT 170, C = CICA 8, and D = Oryzica 1; B–D are from the same seed lots used in the heat-treatment experiment (Table 3), C and D matured during a rainy period, and B matured under irrigation during a dry period.

Table 2. Seed transmission of *Pseudomonas fuscovaginae* in the rice cultivar IAC 1246 after various heat therapy treatments

Temperature (C)	Treatment period (hr)[a]				
	4	24	48	96	144
35	+	−	+	+	+
45	−	−	+	+	+
50	+	+	+	+	+
55	−	−	+	+	+
60	+	−	−	−	−

[a] + = One or more of three seedlings per treatment showed symptoms 35 days after emergence; − = all plants symptomless until maturity.

Table 3. Recovery of fluorescent *Pseudomonas* spp. on King's B medium from grain lots infected with *P. fuscovaginae* after dry-heat therapy treatments

Temperature (C)	Cultivar[a]	Treatment period (hr)[b]			
		0 (control)	24	72	144
55	Oryzica 1	683/1,110	102/421	105/338	12/374
	CICA 8	648/1,166	107/396	235/418	24/333
	IRAT 170	485/943	415/779	126/835	33/784
60	Oryzica 1	683/1,110	47/377	38/369	5/234
	CICA 8	648/1,166	153/591	56/443	9/350
	IRAT 170	485/943	260/850	51/859	82/814
65	Oryzica 1	683/1,110	18/375	12/345	0/403
	CICA 8	648/1,166	22/584	2/277	0/401
	IRAT 170	485/943	25/857	33/812	5/950

[a] Grain from experimental farm.
[b] Number of seeds yielding fluorescent bacteria on King's B medium/number of seeds tested.

were recovered, and reduced the level in the third sample to less than 1%. It is possible that those fluorescent bacteria recovered from the shorter treatments and lower temperatures were not pathogenic, but because of the large numbers and contaminants, this proved impossible to test in all cases.

Pathogenic isolate was obtained from the highest treatment level. Regardless, heat treatment commonly used to break dormancy in experimental lines (55 C for 5 days) does not sufficiently reduce seed infection. Although 65 C dry heat for 6 days is a harsh treatment, germination was not significantly reduced by treatments up to 70 C dry heat for 6 days.

DISCUSSION

It is clear that a seedborne and seed-transmitted bacterial pathogen or pathogens can cause the leaf, sheath, and grain symptoms discussed in this paper. The Latin American, Asian, and African isolates all fall within *P. fuscovaginae-P. fluorescens* biovar II (*P. marginalis*). This, combined with identical symptomatology, indicates beyond reasonable doubt that this pathogen, previously thought to be of limited distribution (1,21), is widespread in Latin America and probably elsewhere. It may well be the cause of brown stripe reported in Brazil (7). Characterization of the CIAT isolates calls into question the designation of *P. fuscovaginae* as a new species distinct from *P. fluorescens* biovar II (*P. marginalis*) and *P. oryzicola*. However, final judgment of this must await more sophisticated comparisons. Given the scant original physiological data available for "*P. stariae*" and "*P. oryzicola*" (5,15), the relationship among these pathogens, the CIAT isolates, and *P. fuscovaginae* is unclear. The wide range of distinct symptom expression (i.e., sheath blotching and grain discoloration to well-defined longitudinal leaf and sheath necrosis) described here and elsewhere (1,7,13) may result from a number of factors such as pathogen virulence, manner and timing of inoculation, variable host susceptibility, and environment during disease development. It is noteworthy that earlier reports of *P. fuscovaginae* are from cool environments (1,13).

The dirty panicle disease has perplexed rice pathologists for years (4,15,17,19,22). It is noteworthy that earlier studies of dirty panicle have focused exclusively on the grain, with little attention paid to the sheath, leaves, and culm. A number of weak fungal pathogens such as *Sarocladium oryzae, Alternaria* spp., *Curvularia* spp. and *Cochliobolus* spp. have been associated with the problem. However, fungicide applications generally fail to control dirty panicle disease (4,12,17,19), suggesting that the fungi may not be primary causal agents. The number and nature of the fungi implicated argues that they may be opportunistic pathogens. A fungal pathogen that infects rice in a semisystemic manner (i.e., not strictly local lesion) and that is commonly implicated in dirty panicle is *S. oryzae*, which causes sheath rot. However, it was rarely recovered from affected sheaths in this study and never from grain, as is corraborated by other studies (24). Furthermore, it is almost always associated with stem or sheath trauma (15). When we investigated reports of fungal sheath rot, bacterial sheath brown rot was almost always encountered. This is not surprising, given the striking similarity in symptoms (Fig. 2). These points suggest that the many reports of the increasing importance of fungal sheath rot (19,22,24) may actually reflect confusion in diagnoses based solely on symptomatology.

The results and observations presented here, with the preceding arguments, support the contention that a large portion of dirty panicle in Latin America and elsewhere is one of the complex of symptoms caused by *P. fuscovaginae* and/or closely related pathogens. The increase in dirty panicle and sheath rot (misdiagnosed?) in the last decade is consistent with a seed-transmitted bacterial pathogen, given the tremendous increase in the international exchange of rice germ plasm that has occurred. Furthermore, many of the other "minor" sheath and grain disorders caused by fluorescent pseudomonads may include *P. fuscovaginae* as a principal agent. Rice pathologists in Africa and Asia, as well as Latin America, should seriously consider the possibility that dirty panicle and sheath rot are not fungal but bacterial in nature.

That *P. fuscovaginae* is seedborne and seed-transmitted is particularly disturbing. Measures to reduce further spread of the pathogen should be undertaken. Treatment at 65 C for 6 days did not completely eradicate the pathogen from all samples; however, these samples were from seed lots or plots so badly affected that they would never be considered for distribution as seed. As of January 1986, all rice introduced to Colombia via CIAT and all material sent to cooperating institutions from CIAT undergoes heat therapy at 65 C for 6 days and monitoring to ensure that it is free of this pathogen. Other institutions involved in the international exchange of rice germ plasm should consider adopting similar measures.

Heat therapy to eradicate the pathogen, though appropriate for ensuring the cleanliness of experimental germ plasm, is obviously not practical at a commercial scale. Milder treatments for short periods (e.g., 60 C for 3 days) will not eradicate the pathogen but can substantially reduce the level of seed infection. Thus, relatively minor modifications of commercial seed-drying processes may offer a means of substantially reducing the initial level of inoculum at planting. Very little is known of the epidemiology of bacterial sheath brown rot other than the existence of numerous wild graminaceous hosts (14). Studies on transmission among plants and survival in the soil must be conducted before definitive statements can be made regarding the effect of reducing initial inoculum via seed treatment on overall disease incidences and economic losses.

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