Pseudomonas avenae, Causal Agent of Bacterial Leaf Stripe of Pearl Millet

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ABSTRACT

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The causal agent of bacterial stripe disease of pearl millet (*Pennisetum glaucum*) in Nigeria was identified as *Pseudomonas avenae*. Most strains of *P. avenae* obtained from culture collections and all six strains from pearl millet were positive for acid production from arabinose, fructose, galactose, glucose, glycerol, and sorbitol. None of the strains utilized sucrose as the sole source of carbon, but most utilized citrate and all grew at 41 C. In the greenhouse, all strains of *P. avenae* were pathogenic in corn, millet, and sorghum and most were pathogenic in sugarcane.

Additional keywords: *Pseudomonas andropogonis, P. rubrisubalbicans, Saccharum officinarum, Sorghum bicolor, Zea mays*

Millets are a diverse group of plants within the Gramineae family that are grown in various parts of the world for food and fodder (21). Pearl millet (*Pennisetum glaucum* (L.) R. Br.) is the most widely grown species (21), with lesser production of proso (*Panicum miliaceum* L.), finger (syn. ragi; *Eleusine coracana* (L.) Gaertn.), yellow foxtail (*Setaria glauca* (L.) P. Beauv.), Japanese (*Echinochloa crusgalli-edulis* (L.) P. Beauv.), and Italian (syn. German; *Setaria italica* (L.) P. Beauv.). Millets are particularly well adapted for growth in harsh environments, including arid areas, and in some areas may be the only commodity that can prevent famine on a local scale or, in the event of unfavorable climatic conditions in numerous countries, alleviate hunger for millions of people.

Numerous bacterial diseases of millet have been reported, but only limited or no significant losses have been attributed to such diseases, probably because millets are commonly grown in hot, dry environments. Pathogenic bacteria isolated from diseased millet include *Pseudomonas eleusineae* of finger millet in India (1), *Bacterium panici* of proso millet in the United States (6), *P. syringae* van Hall of pearl and foxtail millets in the United States (13,23), *Xanthomonas translucens* on Japanese millet in Australia (17), *P. alboprecipitans* Rosen of finger millet in Japan (18) and foxtail millet in the United States (23), *P. setariae* (Okabe) Savulescu of Italian millet in Formosa (19), *X. penniseti* of pearl millet in India (22), *X. campestris* (Pammel) Dowson pv. *pennamericanum* in Nigeria (20), and a *Pseudomonas* spp. (33) of pearl millet in West Africa. Pseudomonads pathogenic to the millets previously named include *B. panici, P. eleusineae, P. alboprecipitans, P. setariae*, and *P. panicimilliacei*. These were determined to be synonymous and were grouped first into *P. panici* (8) and subseguently into *P. avenae* Manns (18,24).

In 1984, symptoms typical of a bacterial disease were observed on nearly every plant in almost all fields surveyed over an area of nearly 700 km from Sokoto to Jos in northern Nigeria. Water-soaking usually occurred at the ends of advancing interveinal lesions varying in length from several to more than 25 cm (Fig. 1). Older lesions usually were a uniform light brown.

Leaves were collected from diseased plants in various fields and brought to the laboratory for isolation and identification of the causal agent. The pathogen was identified on the basis of biochemical, physiological, serological, and pathogenicity tests. Results of these investigations are reported herein. A portion of this research was reported previously (2).

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MATERIALS AND METHODS

Isolation of the pathogen. Pearl millet leaves with symptoms were sectioned across the veins in 2- to 3-mm sections. The sections were triturated in sterile 10 mM phosphate-buffered (pH 7.2) saline (PBS). Samples of the ground tissue-buffer suspension were streaked on yeast-dextrose-calcium carbonate (YDCA) (5) and King's medium B (KB) (14) agars. Plates were incubated at 28 C for 96 hr. Suspect colonies were streaked for isolation on YDCA. Known cultures of bacteria used for comparison tests (Table 1) and the unknown strains were freeze-dried and stored in ampules.

Biochemical and physiological tests. The bacteria were grown overnight in nutrient yeast extract broth (NBY) (31) in shake cultures. A flamed transfer needle was dipped into the culture and stabbed into Thornley's medium 2A (30) to test for production of arginine dihydrolase.

A platinum loopful of each culture was streaked onto filter paper, and a solution of 1% (w/v) *p*-aminodimethylaniline oxalate (Difco) (4) was added; the development of a purple color within 60 sec was oxidase-positive. For lipase activity, a peptone agar medium containing 1% (v/v) Tween 80 was streaked with each culture (27); a positive test was recorded if an opaque halo was observed around the colonies. Tubes of nutrient gelatin (Difco) were stab-inoculated and incubated at 28 C for 3 wk. Every third day, the tubes were cooled to 20 C and visually checked for liquefaction, evidence for gelatin hydrolysis (4). Hydrolysis of starch was determined by adding 0.2% (w/v) soluble potato starch (Sigma, St. Louis, MO) to nutrient agar (Difco) supplemented with yeast extract (5 g/L). Bacterial cells were streaked on the plates and incubated for 4 days at 28 C. The plates then were flooded with 1% iodine.

Reduction of nitrate to nitrite was detected by adding a loopful of cells to nitrate broth (Difco) (4). Nitrite production was tested after 2, 4, and 6 days of incubation by adding a few drops of sulfanilic acid and α-naphthylamine to the broth; a pink or red reaction was positive for nitrite. Growth at 4 and 41 C was determined by streaking 3-day-old cultures on YDCA and incubating them at those temperatures for a maximum of 1 mo. Plates were examined weekly for growth, and a positive reading was noted if isolated single colonies were observed.

Acid production from various carbohydrate and carbon sources was tested with an agar basal medium (Difco) supplemented with the test compounds at a concentration of 1.0% (w/v) (4). Tubes of the basal medium were stab-inoculated and incubated at 28 C. Acid production was recorded as positive if green changed to yellow through the length of the tube after 7, 14, or 21 days. Utilization of malonate and citrate was determined by a previously published procedure (4). All tests were repeated at least four times.

Growth curves. The bacterial strains were grown overnight in NBY in shake cultures at 28 C. Then, 1 ml of each culture was added to Nephelo sidearm culture flasks (Bellco Glass Inc., Vineland, NJ) containing 10.0 ml of NBY and adjusted to 10–20 Klett units with NBY broth. The flasks were placed on a reciprocal shaker (New Brunswick Scientific Co., New Brunswick, NJ) and incubated at 28 C. Klett readings were taken every 45 min, and eight readings were taken of each culture to determine the doubling time.

Hypersensitivity. Hypersensitive reactions were determined on tobacco (*Nicotiana tabacum* L. 'Samsum') plants as described by Klement et al (15). Bacterial strains were harvested from NBY shake cultures by centrifugation at 13,000 *g* for 10 min. Pellets were resuspended in 12.5 mM phosphate buffer (pH 7.2), centrifuged, and washed two additional times. The final cell suspension was diluted with PBS to approximately 10^9 cfu/ml. This suspension was injected into parenchyma of mature (about 8-wk-old) tobacco leaves with a 3-ml syringe. Plants were maintained under ambient conditions (about 22–30 C) in a greenhouse with a minimum of 12 hr of light. Leaves were observed for confluent necrosis 24 hr after infiltration. Hypersensitivity experiments were repeated four times.

Host range. Corn (*Zea mays* L. 'Pioneer 3195' and 'Gold Cup'), grain sorghum (*Sorghum bicolor* L. Moench

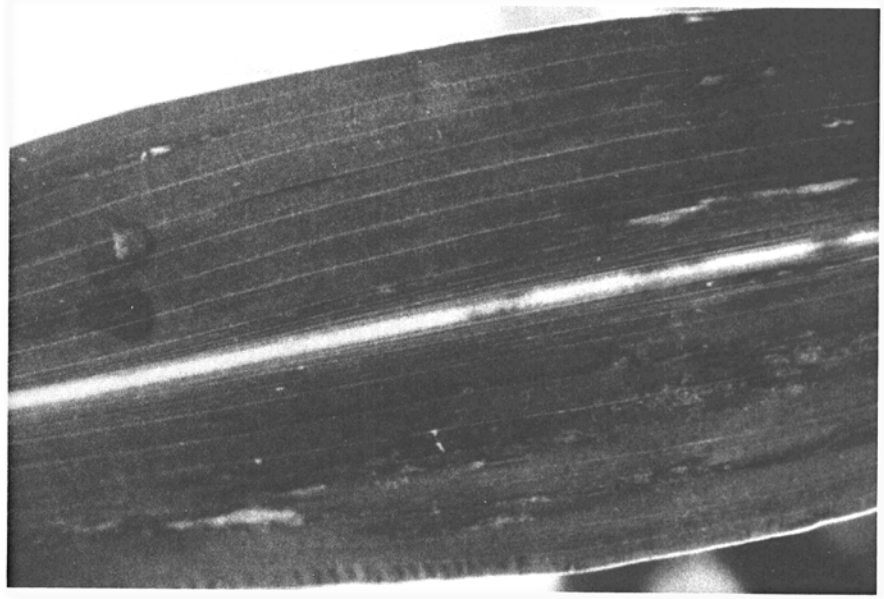

Fig. 1. Lesions on pearl millet caused by *Pseudomonas avenae*.

Table 1. Strain number, host, and origin of various strains of *Pseudomonas avenae*, *P. andropogonis*, and *P. rubrisubalbicans* used in identification of the pearl millet pathogen

Bacterium	Strain number	Host	Origin	Donor[a]
Pseudomonas avenae	ATCC 19860	*Zea mays*	Indiana	1
	PA134	*Z. mays*	Japan	4
	PA135	*Z. mays*	Japan	4
	PA136	*Euchlaena mexicana*	Japan	4
	PA138	*E. mexicana*	Japan	4
P. andropogonis	ATCC 23061	*Sorghum bicolor*	Indiana	1
	ATCC 23062	*Z. mays*	Indiana	1
	KS 74	*S. bicolor*	Kansas	2
	RF 4	*S. bicolor*	Kansas	2
	RF 10	*S. bicolor*	Kansas	2
	RF 12	*S. bicolor*	Kansas	2
	AF 1	*S. bicolor*	Kansas	2
	TX-5	*S. bicolor*	Texas	2
P. rubrisubalbicans	ATCC 19308	*Saccharum officinarum*	United States	1
	PDDCC 793	*Saccharum officinarum*	Australia	3
	PDDCC 2850	*Z. mays*	Florida	3
	PDDCC 3109	*Saccharum officinarum*	Sri Lanka	3
	PDDCC 6268	*S. bicolor*	New Zealand	3
	PDDCC 3110	*Saccharum officinarum*	Tanzania	3

[a] 1 = American Type Culture Collection, Rockville, Maryland; 2 = Department of Plant Pathology, Kansas State University, Manhattan; 3 = Plant Disease Division Culture Collection, Auckland, New Zealand; 4 = M. P. Starr, International Collection of Plant Pathogenic Bacteria, Davis, California.

'80B3039'), pearl millet cultivars Sidney Dwarf and Serere 3A, and sugarcane (*Saccharum officinarum* L. '67500') were inoculated with cell suspensions prepared as described above. A Hagborg device (10) was used to force bacteria through stomates into the mesophyll of leaves. Inoculated plants were maintained in a greenhouse (22–28 C) with automated misting (6 min/hr for 6 hr/day). Symptoms were recorded 2 wk after inoculation. Each strain was inoculated in four plants, and the test was replicated four times.

Antisera production and dot-immunobinding assay. Antisera were produced against *P. avenae* (syn. *P. alboprecipitans*; ATCC 19860 and ICPB PA134), *P. andropogonis* (ATCC 23061 and 23062), *P. rubrisubalbicans* (ATCC 19308), and *P. syringae* pv. *syringae* (ICPB PS 146 and PS 296) as described by Leach et al (16), except that rabbits were injected intramuscularly with 1 ml of the emulsified suspension at weekly intervals. These injections were continued until serial twofold agglutination titers exceeded 1:1,280. Sera were collected 3–4 hr after bleeding and stored at −20 C without preservatives. The dot-immunobinding assay was conducted as previously described (3,16).

RESULTS

Biochemical and physiological tests. Isolations from the millet lesions yielded cream-colored, nonfluorescing colonies on both media within 3 days and are hereafter referred to as the W-6 strains. Morphologically, the colonies resembled those of *P. andropogonis*, but there were significant differences between *P. andropogonis* and W-6 in physiological and biochemical test results (Table 2). Few differences were observed between test results of W-6 and those of *P. avenae* and *P. rubrisubalbicans*. In particular, *P. avenae* and *P. rubrisubalbicans* utilized malonate as sole source of carbon, whereas W-6 did not.

The average doubling time for strains of *P. avenae, P. andropogonis, P. rubrisubalbicans*, and the W-6 pathogen were 103, 148, 110, and 108 min, respectively (*data not shown*).

Pathogenicity tests. In corn, all strains caused lesions with translucent to light tan centers and dark brown margins (Table 3). Water-soaked areas usually were present around the margin of the lesion, which extended several centimeters and was confined to interveinal tissue. In sorghum, the lesions had tan centers with dark red margins, and leaf veins adjacent to the margin were dark red. In pearl millet, the lesions were light brown with dark brown margins, up to 10 cm long, and confined to interveinal tissue. Water-soaking was observed only at the edge of the margin. On sugarcane, elongated dark red lesions were interveinal and up to 45 cm long. Symptoms in corn, sorghum, millet, and sugarcane inoculated with the strain of W-6 were identical to those incited by *P. avenae* (Table 3).

Most strains of *P. rubrisubalbicans* were pathogenic in sugarcane, although several caused weak reactions (Table 3). In corn, sorghum, and millet, however, five strains were nonpathogenic or induced only a hypersensitive reaction, whereas PDDCC 3109 caused typical mottled stripe symptoms (12).

Strains of *P. andropogonis* were highly aggressive in corn and sorghum, mildly aggressive in sugarcane, and weakly aggressive in millet.

Dot-immunobinding assays. These assays with *P. avenae* (ATCC 19860) antiserum did not distinguish *P. avenae* from W-6 isolates at dilutions of 10^5 cfu (Fig. 2). Faint cross-reactions were observed at a dilution containing 10^8 cfu for *P. s.* pv. *syringae* and at dilutions containing 10^7 and 10^8 cfu for *P. rubrisubalbicans*. In several experiments, *P. avenae* and the W-6 isolates provided faint readings at dilutions of 10^5 (approximately 800 cfu per application).

Table 2. Phenotypic characteristics of *Pseudomonas avenae*, *P. andropogonis*, *P. rubrisubalbicans*, and millet strains from Nigeria

Characteristic	Millet strains	*P. avenae*	*P. andropogonis*	*P. rubrisubalbicans*
Acid production from:				
Arabinose	+[a]	+	+	+
Cellobiose	−	−	−	−
Fructose	+	+	+	+
Galactose	+	+	+	+
Glucose	+	+	+	+
Glycerol	+	+	−	+
Lactose	−	−	−	v
Maltose	−	−	−	−
Melibiose	−	−	−	−
Myo-inositol	−	−	+	v
Raffinose	−	−	−	−
Rhamnose	−	−	−	−
Salicin	+	v	−	−
Sorbitol	+	+	−	+
Utilization of:				
Citrate	+	v	+(weak)	+
Malonate	−	+	+	+
Sucrose	−	−	−	−
Hyrolysis of:				
Gelatin	−	−	−	−
Starch	−	−	−	−
Growth at:				
4 C	−	−	−	−
41 C	+	+	−	+
Arginine dihydrolase	−	−	−	+
Lipase production	+	+	−	−
Nitrate reduction	+	+	−	v
Oxidase production	v	+	−	+
Tobacco hypersensitivity	+	+	+	+

[a] + = Positive for 80% of all strains, − = negative for 80% of all strains, v = variable results.

DISCUSSION

The bacterial pathogen isolated from diseased millet in northern Nigeria was identified as *P. avenae* on the basis of results of biochemical, physiological, and immunological tests. The bacterium was indistinguishable from *P. avenae* but clearly distinct from *P. rubrisubalbicans* and *P. andropogonis*.

Numerous species of the Gramineae family have been reported as susceptible hosts of *P. avenae*, including barley (*Hordeum vulgare* L.) (9,28), wheat (*Triticum aestivum* L.) (9,28), oats (*Avena sativa* L.) (9,28), Italian millet (9), barnyard millet (*Panicum crusgalli* var. *frumentaceum*) (9), sorghum (9), corn (7,9,11,12,28), proso millet (6,9,28), foxtail millet (28), finger millet (9), rice (*Oryza sativa* L.) (8,26), rye (*Secale cereale* L.) (28), and Vasey grass (*Paspalum urvillei* Steud.) (7). We found that sugarcane is also susceptible to *P. avenae* in the greenhouse and might be an important host for bacterial survival between cropping seasons in tropical areas.

Although *P. avenae* is not known to be seed-transmitted in corn or pearl millet, the high incidence of bacterial leaf stripe of pearl millet in Nigeria could be due to infested seed. The majority of farmers maintain their own seed stocks from year to year, and very limited amounts of new seed are introduced. In a recent study (26), *P. avenae* was detected in 55 rice seed samples from 28 countries. Infestation levels ranged from 1 to 75%, and *P. avenae* remained viable in rice seed stored for 8 yr (25). Gitaitis et al (7) isolated *P. avenae* from Vasey grass seed in Florida and surmised that the seedborne phase would be important in spreading and increasing initial bacterial inoculum.

Bacterial leaf stripe appears to be

present in most of the areas in Africa where pearl millet is grown, and it was detected in Niger (R. A. Frederiksen, *personal communication*) and Sudan (H. Omer, *personal communication*) in high incidence during the 1987 growing season. Bacterial diseases of pearl millet were not mentioned in earlier published surveys (29,32), possibly because of incorrect diagnosis, as bacterial stripe can be confused with other diseases, including downy mildew.

Zummo (33) described a yellow leaf blotch of corn, sorghum, and pearl millet in West Africa that partially resembled the symptoms we observed in pearl millet. The photographs reveal oval lesions closely resembling those caused by witchweed (*Striga* spp.), although they appear to be vein-limited. In our tests, symptoms were always interveinal and occurred more often at the leaf tips, suggesting that hydathodes were the portals of entry for the bacteria. It was impossible to compare the *Pseudomonas* spp. from yellow blotch with our strains, as only limited information was provided.

The bacterial stripe disease of proso millet described by Elliott (6) appears identical in symptomatology to the disease of pearl millet. Pearl millet was not tested, however, and foxtail millet, barnyard millet, and sorghum were resistant.

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Table 3. Pathogenicity tests of *Pseudomonas avenae*, *P. rubrisubalbicans*, and six Nigerian millet isolates (W-6) to four host plants

Strain	Host			
	Zea mays	*Sorghum bicolor*	*Pennisetum americanum*	*Saccharum officinarum*
P. avenae				
ICPB PA 117	+[a]	+	+	+
ICPB PA 134	+	+	+	+
ICPB PA 135	+	+	+	+
ICPB PA 138	+	+	+	+
P. rubrisubalbicans				
PDDCC 973	H	H	H	v
PDDCC 2850	H	H	−	+
PDDCC 3109	+	−	−	+
PDDCC 3110	−	−	−	v
PDDCC 6268	−	−	−	−
ATCC 19308	−	H	H	v
Nigerian isolates	+	+	+	+

[a] + = Distinct symptoms on leaf tissue 14 days after inoculation, − = no symptoms, v = variable results and/or indistinct symptoms, H = hypersensitive reaction.

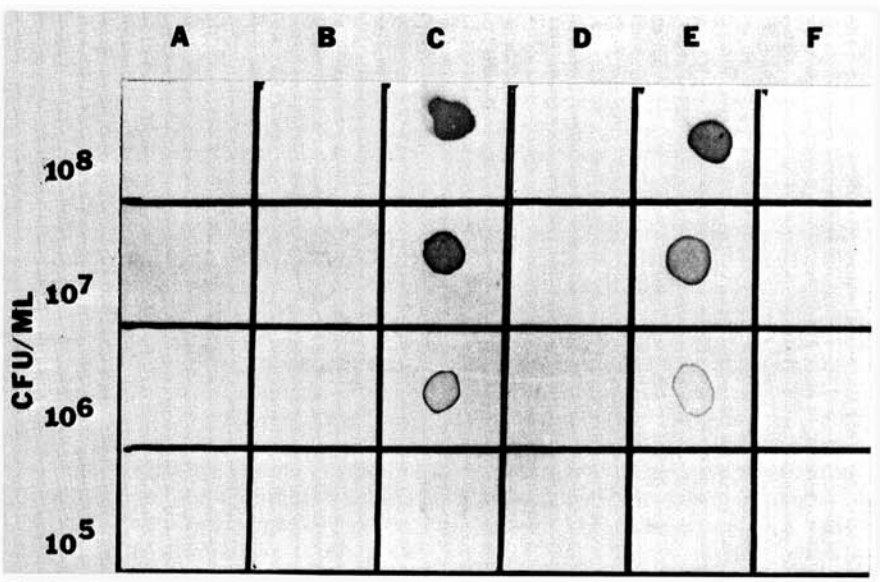

Fig. 2. Nitrocellulose membrane from a dot-immunobinding assay: lane A, *P. syringae* pv. *syringae*; lane B, *P. andropogonis*; lane C, *P. avenae*; lane D, *P. rubrisubalbicans*; lane E, *P. avenae* (W-6 isolate from millet); lane F, water control. *P. avenae* antiserum (ATCC 19860) diluted 1:1,000 was used.

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