Characterization of the Bacterium Inciting Bean Wildfire in Brazil

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ABSTRACT

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The bacterium that incites bean wildfire and a foliar blight of garden peas in Brazil was identified as a strain of *Pseudomonas tabaci*. The organism conformed with this nomenspecies on the basis of in vitro biochemical properties, including the type of phytotoxins formed (tabtoxins). The Brazilian pathogen and a strain of *P. tabaci* originally isolated from soybean induced typical wildfire lesions on bean but were avirulent on tobacco. However, all the tobacco wildfire stains tested induced a hypersensitive response on bean. Cowpea, lima bean, and soybean developed wildfire symptoms with all strains of *P. tabaci*, regardless of their original hosts. The *P. tabaci* strains reached populations substantially higher in cowpea leaves than in leaves of their respective natural hosts (tobacco and bean). The bean wildfire organism multiplied in tobacco leaves much less than did the tobacco wildfire pathogen. The bean wildfire bacterium did not infect bean seeds. Differential symptoms produced on inoculated bean pods were useful in distinguishing between bean wildfire and bean halo blight, because the foliar symptoms of the two diseases were similar. The Brazilian pathogen and other strains that can induce wildfire symptoms on *Phaseolus* bean and are avirulent on wildfire-susceptible tobacco cultivars constitute a separate group within *P. tabaci*.

Additional key words: bacterial leaf spot, *Phaseolus vulgaris*.

A bacterial leaf spot of bean (*Phaseolus vulgaris* L.), with symptoms (light to dark brown lesions surrounded by pronounced chlorotic halos, Fig. 1) closely resembling those of bean halo blight, was first reported in 1974 from São Paulo, Brazil (23). The disease, bean wildfire (BW), was caused by a fluorescent pseudomonad in group *Ia* of Lelliott et al (19), according to the LOPAT characteristics. It differed from *Pseudomonas phaseolicola*, the incitant of bean halo blight, in certain bacteriological tests such as gelatin liquefaction, β-glucosidase activity, and utilization of $NaNO_3$ as sole nitrogen source (23). The pathogen also has been associated with a foliar blight of garden peas (*Pisum sativum* L.) that occurs in the same region (22).

The purpose of this paper is to establish the relationship of the causal organism to other closely related bacteria in terms of physiological characteristics, including toxin production and pathogenicity. A preliminary report was published (22).

MATERIALS AND METHODS

The bacterial strains used were: (i) six strains of the BW bacterium (BW 1 to 6), including five strains from bean and one from pea, were collected in São Paulo, Brazil; (ii) *Pseudomonas coronafaciens*, PC-27 from M. P. Starr (Bacteriology Department, University of California, Davis); (iii) *Pseudomonas tabaci*, 12 strains including ATCC 11528, ATCC 17914, ICPPB-PT 113, ICPPB-PT 13, ICPPB-PT 127A, ICPPB-PT 127B, ICPPB-PT 15, four tobacco wildfire strains (TOB-B, WIS-BELL, VIR-24, and VIR-8.2.74) from R. W. Fulton (Department of Plant Pathology, University of Wisconsin, Madison), and a soybean wildfire strain (No. 0152) from Melda L. Moffett (Department of Primary Industries, Plant Pathology Branch, Indooroopilly, Queensland, Australia); (iv) *P. phaseolicola*, NY-race 1 from M. L. Schuster (Department of Plant Pathology, University of Nebraska, Lincoln), HB-race 2 from bean halo blight in Wisconsin, and a strain (15A) virulent on mung bean (25) from H. A. J. Hoitink (Department of Plant Pathology, Ohio Agricultural Research and Development Center, Wooster); and (v) *P. syringae*, three strains (BS 1 to 3) from bacterial brown spot of bean in Wisconsin and one strain (B-397) that incites holcus spot of corn from J. E. DeVay (Department of Plant Pathology, University of California, Davis).

Bacteriological tests performed were the ability to cause pitting on a crystal violet-polypectate medium (7) at pH 7.0, hydrolyze aesculin (28), and utilize certain organic acids and polyalcohols as sole carbon sources (24). Production of syringomycin was determined with still cultures in potato-dextrose-broth as described by Gross and DeVay (14); *Geotrichum candidum* Link ex Pers. was the test organism. The in vitro formation of chlorosis-inducing toxins was tested with filtrates from 5 day old cultures grown on a modified Woolley's medium (11). Some of the filtrates were eluted in a column containing Amberlite CG-120 (H^+ form) as previously described (21); the toxins then were separated by ion-exchange chromatography (30). Chlorosis-inducing activity of crude filtrates and partially purified preparations was tested on bean seedlings (cultivar Bush Blue Lake 274) by prick inoculation of primary leaves (21).

Pathogenicity tests were performed in the greenhouse (20–30 C) or, when so specified, in controlled environment chambers (12-hr day and 10,000 lux). Plants were grown in a mixture of compost soil, peat moss, and sand (3:3:1, v/v). Unless otherwise mentioned, the cultivars were: Bush Blue Lake 274 and Topcrop (bean); Blackeye (cowpea); Early Thorogreen and Fordhook 242 (lima bean); Clark, Arksoy, Roanoke, and Shore (soybean); and Havana 38, Xanthi, and Burley 21 (tobacco). Inocula consisted of suspensions prepared from 1 day old cultures grown on glycerol nutrient agar slants. Bacterial cells were washed by centrifuging and resuspending them twice in sterile, distilled water. Cell concentration was estimated with a Bausch & Lomb Spectronic-20

colorimeter at 600 nm. Inoculations were performed by pricking leaves or pods with a sterilized needle through droplets of inoculum or by light spraying or watersoaking leaves forcibly with a Ceccato 2701-2711 (1.5 nozzle) paint gun connected to compressed air. Detached bean pods were prick-inoculated and incubated in a moist chamber at 24 C. Inoculum dosages varied from about 10^5 viable cells per milliliter (watersoaking) to about 10^7 viable cells per milliliter (prick and light spray). Bean flowers also were inoculated using Kauffman and Leben's method (17) and suspension of 10^7 viable cells per milliliter.

Populations of bacteria in leaves were monitored with a modification of Ercolani's method (13). Six leaf disks (5 mm in diameter) were harvested from the prick-inoculated areas, bulked, thoroughly rinsed, and ground aseptically in a mortar with 4 ml of sterile, distilled water. After the tissue debris settled, 1 ml of the supernatant fluid was pipetted and mixed with 9 ml of sterile, distilled water. From this suspension, 10-fold dilutions were prepared and 0.2-ml samples of selected dilutions were spread on petri dishes containing King's medium B (18). The plates were dried (10–15 min at 40 C) with the lids open, then closed and incubated at 24 C. The numbers of typical (fluorescent) colonies were recorded 72 hr later, and the populations of viable cells recovered per leaf disk were estimated.

RESULTS

By the bacteriological tests (Table 1), the BW bacterium was indistinguishable from *P. tabaci*, sensu Hildebrand and Schroth (15). The organism differed from *P. phaseolicola* in its ability to hydrolyze aesculin and to utilize L(+)-tartrate or polyalcohols as sole carbon sources for growth; it differed from *P. syringae* in its ability to utilize L(+)-tartrate and to cause pitting on polypectate gel and in its inability to grow on DL-lactate. *P. coronafaciens* did not utilize L(+)-tartrate or induce pitting on polypectate gel. The Brazilian pathogen also could be distinguished from *P. syringae* and *P. phaseolicola* by the type of phytotoxins it produced in culture. All *P. syringae* strains formed syringomycin, as indicated by inhibition of *G. candidum* in the bioassy, but the BW, *P. tabaci*, *P. coronafaciens*, and *P. phaseolicola* strains did not. On the other hand, culture filtrates from the BW strains contained toxins that induced chlorotic lesions with necrotic centers and well-defined margins in bean leaves. This effect was similar to that obtained with filtrates from *P. tabaci* and *P. coronafaciens*. The toxic material was heat-labile and the chlorotic effect was light-dependent, as is characteristic of the tabtoxins (10,26). Filtrates from *P. phaseolicola* also induced chlorotic lesions in bean leaves; these lesions were devoid of necrotic centers, however, and had diffuse margins. In addition, the effect was light-insensitive and the toxic material was heat-stable (100 C for 10 min), which is characteristic of the chlorosis-inducing toxin formed by *P. phaseolicola* (10,16). Ion-exchange chromatography of partially purified preparations from the BW strains, *P. tabaci* (ATCC 11528), and *P. coronafaciens* revealed two peaks of chlorosis-inducing activity. These peaks corresponded in elution time to those of the three tabtoxins reported for *P. tabaci* and *P. coronafaciens* (11,21). Upon acid hydrolysis (6N HCl, 1 hr at 100 C) the first peak (50–60 min retention) yielded only tabtoxinine, as determined by chromatographic and mass spectral procedures (30); the second peak (100–120 min retention) yielded tabtoxinine, threonine, and serine, as determined by the same procedures.

A comparative study of the pathogenicity of the Brazilian bacterium and strains of *P. tabaci* from tobacco and soybean (Table 2) revealed the following: (i) On bean leaves all strains from tobacco wildfire induced a typical hypersensitive reaction characterized by small necrotic flecks that appeared within 24 hr after watersoaking or light spray inoculations (Fig. 2). Such flecks did not expand and chlorotic halos did not develop. The response was identical for the two reported "races" of the tobacco wildfire organism (6,29,31). Prick inoculation of bean leaves with all the tobacco strains produced only a local, incipient necrotic reaction without chlorotic halos. Identical responses on bean leaves also were obtained by inoculation with *P. coronafaciens*. Conversely, inoculation of beans with the BW strains induced the typical wildfire disease; lesions appeared in 3–4 days and were at first watersoaked, then necrotic, but always surrounded by the characteristic broad, circular, bright yellow halos, which reached maximum size in 7–10 days. (ii) Tobacco leaves developed an incompatible reaction to all strains isolated from legumes. Prick

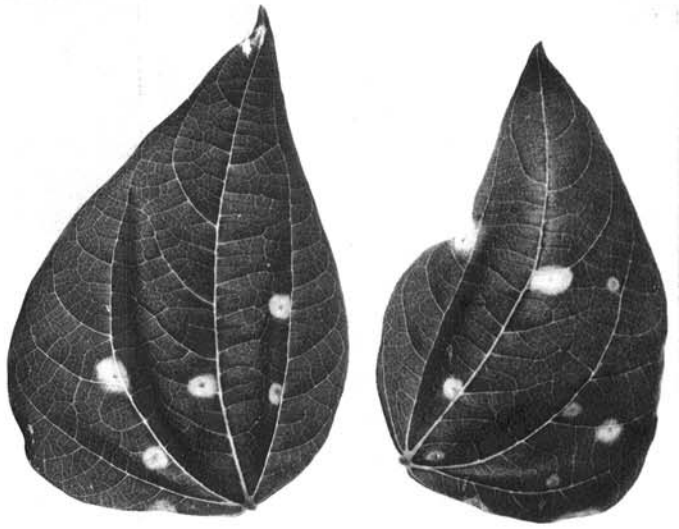

Fig. 1. Leaves of bean (cv. Bush Blue Lake 274) 7 days after inoculation with washed cell suspensions of bean wildfire strains. Left, strain BW 1 (from bean). Right, strain BW 6 (from pea).

TABLE 1. Comparison of bacteriological properties of bean wildfire (BW) strains with four nomenspecies of *Pseudomonas*

Test	Strain or nomenspecies				
	BW 1 to 6[a]	*P. syringae*[a]	*P. tabaci*[a]	*P. coronafaciens*	*P. phaseolicola*[a]
Growth on DL-lactate	–[b]	+	–	–	–
Growth on L(+)-tartrate	+	–	+	–	–
Growth on erythritol	+	+	+	NT	–
Growth on sorbitol	+	+	+	NT	–
Pitting on polypectate	+	–	+	–	+
Hydrolysis of aesculin	+	+	+	NT	–

[a] All strains reacted identically.
[b] + = positive reaction, – = negative reaction, and NT = not tested.

inoculation produced very small necrotic lesions devoid of chlorotic halos. Watersoaking and light spray inoculations induced no visible response in most cases. In a few instances, faint, chlorotic flecks were detected, but they disappeared completely in 2–3 days. These reactions were identical on the three tobacco cultivars. A similar response was obtained by inoculating the Burley 21 cultivar with some of the tobacco strains of *P. tabaci*; however, these strains induced typical wildfire symptoms on the cultivars Havana 38 and Xanthi. The remaining strains of tobacco origin were virulent on all three tobacco cultivars. (iii) The Australian strain of *P. tabaci* from soybean induced wildfire symptoms on bean but was much less aggressive than the BW strains. In six separate tests, the mean number of lesions elicited by the soybean strain on leaves of Bush Blue Lake 274 bean was roughly one-fifth of that elicited by the Brazilian bacterium. Furthermore, the average diameter of the yellow halos induced by the soybean strain was less than one-half that of the halos produced by the BW strains. (iv) All the strains tested, regardless of their original hosts, produced wildfire symptoms on cowpea, lima bean, and soybean. For these three species, prick inoculation was more effective than light spray or watersoaking inoculations. On soybean, watersoaking inoculation produced infection erratically and light spray inoculation was completely ineffective.

Cowpea was particularly susceptible to *P. tabaci* and the BW organism; large halos formed around leaf lesions. The tobacco and the bean wildfire pathogens reached populations considerably higher in cowpea leaves than in leaves of their respective natural hosts (Table 3). In bean leaves, the tobacco wildfire organism multiplied much less than the BW bacterium did, and conversely, in tobacco, the BW pathogen reached a much lower population level than that of the tobacco wildfire organism.

In bean, lesions surrounded by chlorotic halos were produced by the BW organism at all temperatures tested (Table 4); the halos increased in size with increasing temperatures from 16 to 28 C. In contrast, *P. phaseolicola* induced large halos at the cooler temperatures but not at 28 or 32 C.

Pods from 65 dry and snap bean cultivars (including 54 commercial varieties from Brazil), inoculated with the BW pathogen, developed minute, light brown spots at the pricked regions only. Under similar conditions, all *P. phaseolicola* strains induced watersoaked, expanding lesions with bacterial exudation, typical of the halo blight disease. All *P. syringae* strains of bean origin induced the sunken, watersoaked lesions characteristic of the brown spot disease.

TABLE 2. Comparison of pathogenicity to selected hosts of bean wildfire (BW) strains with eight strains of *Pseudomonas tabaci*

Strain[a]	Original host	Response of test plant					
		Tobacco		Bean	Lima bean	Soybean	Cowpea
		Havana 38 and Xanthi	Burley 21				
BW 1 to 6	bean/pea	–[b]	–	+	+	+	+
ATCC 11528	tobacco	+	–	–	+	+	+
ICPPB-PT 113	tobacco	+	–	–	+	+	+
ICPPB-PT 13	tobacco	+	–	–	+	+	+
VIR-8.2.74	tobacco	+	–	–	+	+	+
VIR-24	tobacco	+	+	–	+	+	+
TOB-B	tobacco	+	+	–	+	+	+
WIS-BELL	tobacco	+	+	–	+	+	+
0152	soybean	–	–	±	+	+	+

[a] Strains ATCC 17914, ICPPB-PT 127A, ICPPB-PT 127B, and ICPPB-PT 15 were nonpathogenic on all test plants.
[b] + = virulent (typical wildfire lesions with pronounced yellow halos), – = avirulent (no visible response or nonexpanding flecks), ± = weakly virulent on bean.

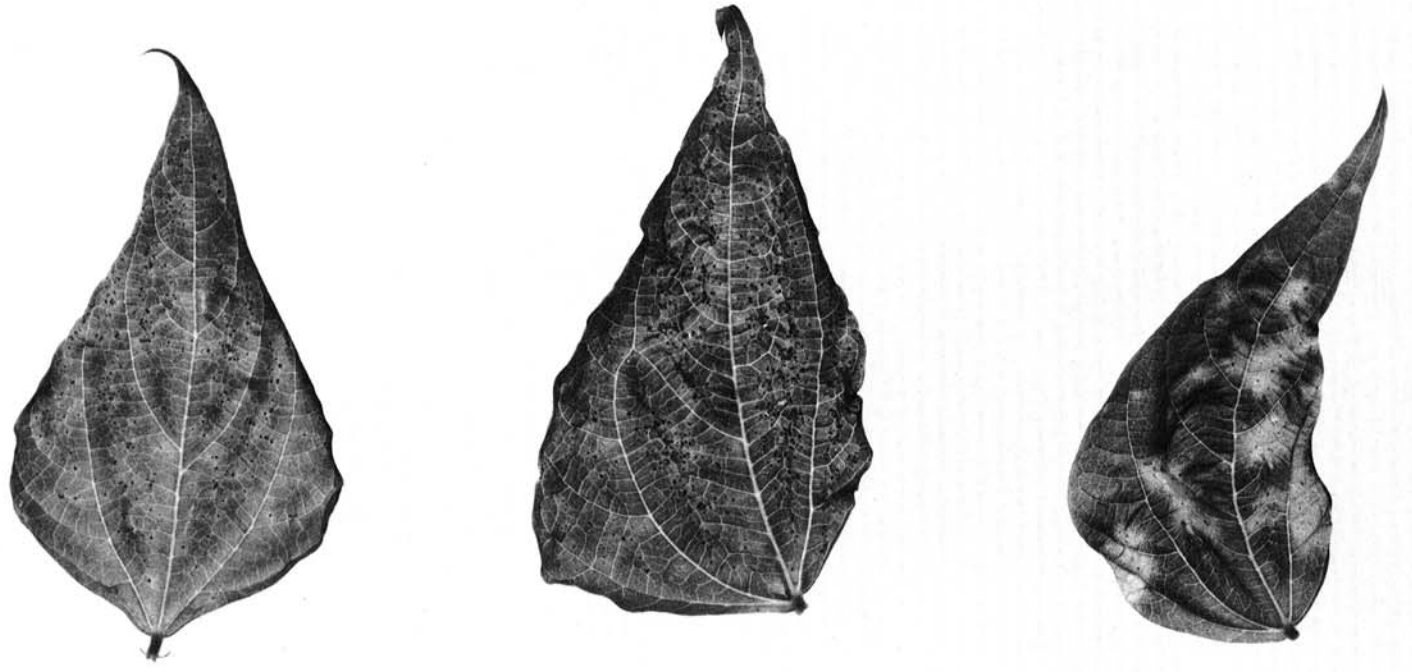

Fig. 2. Leaves of bean (cv. Bush Blue Lake 274) 7 days after watersoaking inoculation with a strain of bean wildfire (right), *Pseudomonas coronafaciens* (center), and a strain of *Pseudomonas tabaci* from tobacco wildfire (left).

TABLE 3. Comparison of populations of bean wildfire (BW 2) and tobacco wildfire (ATCC 11528) strains in prick-inoculated leaves of selected test plants

Plant species and cultivar	Days after inoculation	No. of viable cells per leaf disk (×100)[b]	
		BW 2	ATCC 11528
Bean (Bush Blue Lake 274)	0[a]	65 ± 8	81 ± 5
	2	3,393 ± 110	410 ± 28
	4	3,501 ± 125	129 ± 19
	6	3,512 ± 112	90 ± 7
	8	3,224 ± 141	9 ± 2
	10	2,840 ± 95	5 ± 2
Tobacco (Havana 38)	0[a]	69 ± 12	98 ± 8
	2	337 ± 17	2,292 ± 60
	4	85 ± 6	3,077 ± 110
	6	74 ± 6	2,813 ± 82
	8	63 ± 7	1,994 ± 30
	10	27 ± 3	1,819 ± 43
Cowpea (Blackeye)	0[a]	59 ± 5	54 ± 9
	2	4,328 ± 116	3,957 ± 68
	4	5,647 ± 132	5,105 ± 109
	6	20,802 ± 178	19,898 ± 123
	8	4,923 ± 89	2,468 ± 70
	10	1,266 ± 37	2,203 ± 65

[a] Disks collected 2 hr after inoculation.
[b] Average values of three tests.

TABLE 4. Effect of incubation temperature on the diameter of chlorotic halos of lesions on bean leaves (cultivar Bush Blue Lake 274) inoculated with bean wildfire (BW) and bean halo blight (HB) strains

Temperature (C)	Diameter of chlorotic halos (mm)[a]	
	BW	HB
16	10.7 ± 1.6	17.2 ± 1.9
20	12.2 ± 1.8	15.9 ± 1.7
24	13.3 ± 1.5	13.5 ± 1.7
28	13.6 ± 1.6	0.0 ...
32	9.1 ± 1.6	0.0 ...

[a] Average values of three BW strains and three HB strains in two separate tests, 10 days after prick inoculation.

Flowers and pods of 40 bean plants (20 plants per cultivar) were inoculated with a mixed suspension of all BW strains. None of the 600 seeds harvested from those plants produced seedlings with symptoms of the disease.

DISCUSSION

The BW strains conformed with *P. tabaci* in all the bacteriological properties studied, including those previously reported (23), and in the type of toxins formed in culture (tabtoxins). Furthermore, the BW strains and *P. tabaci* induced identical symptoms on common hosts.

Clayton (4) showed that bean was not susceptible to *P. tabaci* isolated from tobacco wildfire. No reports were found on the pathogenicity of strains from soybean wildfire toward bean. We believe this is the first report that *P. vulgaris* is a host for *P. tabaci*.

Differences in pathogenicity between strains of *P. tabaci* isolated from tobacco and soybean wildfire were indicated clearly by early workers. Clayton (5) and Chamberlain (2) reported on strains from soybean that were virtually avirulent on tobacco. Clayton (5) suggested that strain differences could account for wildfire on soybean but not on tobacco in some areas of the United States where both tobacco and soybean are grown. Allington (1) and Chamberlain (3) found strains causing tobacco wildfire that were also pathogenic on soybean.

Doudoroff and Palleroni's treatment of the genus *Pseudomonas* (9) provisionally considered all oxidase-negative, fluorescent, plant-pathogenic pseudomonads to be nomenspecies with *P. syringae*. Later, Dye et al (12) suggested that a number of these nomenspecies, including *P. tabaci* should be retained until their taxonomic status can be more thoroughly assessed. More recently, Young et al (32) used the infrasubspecific epithet "pathovar" to reclassify many of the fluorsecent pseudomonads. In that scheme *P. tabaci* is reclassified as *P. syringae* pv. *tabaci*. However, because strains that incite typical wildfire diseases differ substantially in host range, they should be considered and designated as distinct pathovars. Therefore, a reassessment of the proposal by Young et al (32) for naming this particular group of pathogens may be needed in the future.

The populations of virulent *P. tabaci* strains in leaves of tobacco or bean were low compared with other compatible host/bacterium systems. However, similar populations of *P. tabaci* in leaves of wildfire-susceptible tobacco cultivars have been reported (2,8). Under greenhouse conditions, individual, well-isolated lesions induced by virulent *P. tabaci* strains on tobacco or bean leaves are rather small (except for the large yellow halos that surround them), and bacteria apparently are absent from the chlorotic areas, as indicated by microscopic examinations and reisolation attempts. It is therefore possible that the deleterious senescing effect of the tabtoxins on the host tissue might contribute to a decreased rate of in situ bacterial growth.

Unlike bean halo blight, bean wildfire is restricted to the leaves. Pod lesions were not found under natural disease conditions in Brazil. The results of flower and pod inoculations indicate that the BW pathogen cannot infect bean seeds. At an early stage of the bean crop, the two diseases can be easily confused. The differential symptoms produced on bean pods may be used as a simple, rapid, and reliable way to diagnose halo blight. This is likely to be of special interest in the case of crops grown for production of disease-free seed. The results of tests in a controlled environment confirmed previous observations that the BW strains could induce chlorotic halos at relatively high temperature (23). This also can be used for diagnostic purposes, because the bean halo blight bacterium induces the chlorotic effect at cooler temperature regimes only (20,27).

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