Pathogenic and Nutritional Variation in the Halo Blight Group of Fluorescent Pseudomonads of Bean

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ABSTRACT

There is no clear distinction in host ranges of *Pseudomonas phaseolicola, P. glycinea,* and *P. mori,* because some strains of each nomenspecies are capable of infecting certain cultivars of both the common bean and lima bean. Certain strains of *P. phaseolicola* also infected soybean. There was, however, a pronounced tendency for most strains to segregate along the commonly accepted host lines. Strains of *P. phaseolicola* attacked fewer soybean cultivars, and were less virulent than *P. glycinea* strains; the reciprocal also was true. The lima bean cultivars Fordhook 242, Fordhook Concentrate C-2, and King of the Garden were universally infected by all strains of the three nomenspecies. None of the tested *P. phaseolicola* and *P. glycinea* strains infected mulberry. Virulence among *P. phaseolicola* strains varied in two ways: (i) Certain strains were less virulent than others in all hosts tested; whereas (ii) virulence of some strains depended solely on the host. Neither race 1 nor race 2 strains of *P. phaseolicola* as defined by their reaction on Red Mexican UI-3 were homogeneous as to host range or virulence. Many so-called race 1 strains are merely low in virulence to all common bean cultivars, and did not infect the relatively more resistant ones. There was considerable variation in symptomatology on bean plants infected with *P. phaseolicola.* Some strains produced systemic symptoms on many hosts, others on a few hosts; whereas others produced only water-soaked lesions. The effects of infection by *P. phaseolicola* on plant growth depended upon bean cultivar, temperature, and bacterial strain. Nutritional studies of the nomenspecies indicated that strains with dissimilar host ranges differed in capacity to utilize substrates. Phytopathology 61:852-857.

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A taxonomic study of *Pseudomonas* plant pathogens revealed that *Pseudomonas phaseolicola, P. glycinea,* and *P. mori* have many nutritional characters in common, suggesting that they might belong to the same pathogenic population (10). A study of DNA hybridization has furthermore indicated that considerable homology exists among these organisms. There was 75-81% homology when *P. phaseolicola* DNA was hybridized with DNA of *P. mori* or *P. glycinea* with temperature at 80 C as compared with 40-52% homology with *P. tomato* and *P. syringae* DNA (1).

Phenotypic similarity of these organisms suggested a re-examination of their virulences and host ranges. Patel & Walker (7) showed marked pathogenic differences among isolates of *P. phaseolicola,* and reported the existence of two races. They did not test *P. glycinea* or *P. mori.* Cross et al. (3) found seven races of *P. glycinea,* but did not test *P. phaseolicola* or *P. mori.*

We therefore broadened these studies by initiating a comparative investigation of the virulences and host ranges of diverse strains in these nomenspecies. Variation of symptoms elicited by the strains was also noted. On the basis of host range and virulence, we subsequently selected several strains for a study of their nutritional spectra to determine whether or not differences could be detected among strains of similar and dissimilar host ranges. The importance of these findings in relation to taxonomy are discussed. An abstract of this work has been published (12).

MATERIALS AND METHODS.—Identity and source of bacterial isolates of beans are listed in Table 1. Tests were made with two genera and four species: *Phaseolus vulgaris* L. (common bean); *P. limensis* Macf. (large-seeded lima bean); *P. lunatus* f. *macrocarpus* (Benth.) Van Ess. (small-seeded lima bean); and *Glycine max* (L.) Merr. (soybean). Limited tests were also made on *Phaseolus aureus* Roxb. (mung bean) and *Morus alba* L. (mulberry).

Inoculation methods.—Plants (two/pot) grown in 4-inch pots were inoculated in a greenhouse with a temperature of ca. 21 ± 4 C. Selection of an inoculation method for evaluating virulence, host range, and symptomatology was determined by comparison of the following commonly used techniques: seed inoculation; needle puncture of the epicotyl; vacuum infiltration; water pressure spray; light spray followed by incubation in a moist chamber for 24 hr; and the abrasive grit (corundum) method, where leaves are dusted with grit, then rubbed lightly with a cotton swab dipped in a bacterial suspension of about 10^9 cells/ml.

The seed inoculation and needle puncture techniques were rejected, as they were useful only with isolates which systemically invaded the plant. The water pressure technique was not used because there was too much variation in infection, and a hypersensitive reaction often was obtained. Vacuum infiltration was too time-consuming for the inoculation of many plants, and the requirement for incubation in moist chambers made

TABLE 1. Strains of *Pseudomonas phaseolicola, P. glycinea,* and *P. mori* used in nutritional and pathogenicity studies

Strain[a]	Source[b]	Year isolated
P. phaseolicola		
HB-1B	W. J. Virgin; WHB, CPC[b]	1964
HB-2	R. G. Grogan; G16, 507-2	
HB-8	L. L. Dean	1966
HB-9	R. G. Grogan	
HB-11	R. W. Goth	1966
HB-13	R. G. Grogan; 507-1, SS-2-2	1967
HB-14	R. G. Grogan; G77, 507-1	1964
HB-16	R. G. Grogan; G75, 507-1	1964
HB-18	R. G. Grogan; G75a, 507-1	1964
HB-19	R. G. Grogan; G101	
HB-20	R. G. Grogan; G106, 507-1 (race 1)	
HB-23	R. G. Grogan; G114, 507-1, SS-102	
HB-24	R. G. Grogan; G115, 507-1, SS-2-1	
HB-25	R. G. Grogan; G123, 507-1, SS-2-1	
HB-26	R. G. Grogan; G124	1963
HB-27	R. E. Stall; 65-4	1967
HB-28	J. J. Natti; HB Std. race 1	
HB-29	J. J. Natti; HG-2	1963
HB-30	J. J. Natti; HG 9A-1	
HB-31	J. J. Natti; Wallace race 1	<1962
HB-32	J. J. Natti; Wisc. race 2	<1963
HB-33	J. J. Natti; W. Burkholder race 1	<1944
HB-35	M. N. Schroth	1964
HB-36	M. N. Schroth	1964
HB-37	ATCC 7-E	
HB-38	J. W. Guthrie	1965
HB-39	NCPPB; 1102	
HB-40	NCPPB; 1103	
HB-41	NCPPB; 1341	
HB-42	W. J. Zaumeyer	1967
HB-43	M. N. Schroth; race 1	1967
HB-45	A. Kelman; race 1	1968
HB-46	D. J. Hagedorn; race 1	1968
HB-47	D. C. Graham	1966
HB-48	D. C. Graham	1966
P. glycinea		
R-1	B. Kennedy	1962
R-2	B. Kennedy	1962
R-4	B. Kennedy	1964
R-6	B. Kennedy	1964
P. mori		
PM-2	NCPPB; 1413	1958
PM-3	NCPPB; 1415	1958
PM-4	NCPPB; 1445	1958
PM-5	Grogan; 18-526	1966
PM-6	Grogan; 112-547-1	1966
PM-7	Grogan; 126-547-2	1966
PM-8	ICPPB; PM135	
P. mori var. huszi		
PM-1	NCPPB, 1037	1958

[a] Our strain designation.
[b] ATCC = American Type Culture Collection, NCPPB = National Collection of Plant Pathogenic Bacteria, ICPPB = International Collection of Plant Pathogenic Bacteria; other designations are those of the donor.

the light spray technique unsuitable. The abrasive grit technique was selected because it enabled a rapid inoculation of large numbers of plants and could be standardized.

Exploratory tests of differences in the susceptibility of leaves indicated that there was little difference between primary and trifoliolate leaves, provided primary leaves were inoculated shortly after they unfolded. To maintain the greatest rate of plant turnover, therefore, the primary leaves were inoculated. With *P. aureus* Roxb., however, primary leaves were resistant, and inoculations were made on the trifoliolate leaves.

Symptoms ranged, as generally described by Patel & Walker (7), from highly resistant, necrotic lesions to susceptible, transparent, water-soaked lesions. A reaction was considered as susceptible only if water-soaking occurred during symptom development. The severity of disease was indicated by a rating of 0-3, with *1* representing a lesion count ca. $< 2/cm^2$; *2* a count ca. 2-10 lesions/cm^2; and *3* with lesions ca. $> 10/cm^2$. A tolerant reaction was characterized by the appearance of lesions with a greasy appearance or by small, water-soaked spots that became dry and necrotic 12 hr or less after their appearance. Following Patel & Walker (7), no attempt was made to relate the different types of water-soaked lesions to susceptibility, such as the occurrence of halos, necroses surrounding water-soaked lesions, or the sizes of the lesions. Although amount of inocula appeared to affect systemicity, we considered that the inoculation technique was sufficiently standardized to enable the determination of the potential of a strain to cause systemic chlorosis. In general, systemic symptoms were noted more commonly with host-pathogen combinations where there was severe infection of leaves. The ability of strains to cause stunting (recorded only when plant size was $< 75\%$ of the control) varied greatly; some produced a severe reaction that prevented the unfurling of trifoliolate leaves.

RESULTS.—*Host range.*—Results from challenging diverse strains of *P. phaseolicola, P. glycinea,* and *P. mori* against common bean, lima bean, and soybean varieties did not reveal a clear distinction in the host range among strains of the nomenspecies (Table 2 [the complete table is available upon request to the senior author]). In general, however, there was a pronounced tendency for strains to segregate among commonly accepted host lines. Although most *P. vulgaris* cultivars were immune to infection by *P. glycinea,* several cultivars were tolerant, and some were susceptible. *Pseudomonas glycinea* R-2, when inoculated into Red Kidney, was the only strain that produced the typical systemic symptoms common to many *P. phaseolicola* strains.

Five of 35 *P. phaseolicola* isolates were pathogenic on some soybean cultivars. Systemic symptoms, however, were produced only by strains HB-28 and HB-31, and only on variety Hawkeye.

Lima bean cultivars, in general, were the most susceptible of the legumes to infection by strains of the three nomenspecies. Fordhook 242, Fordhook Concentrated C-2, and King of the Garden were infected by all strains of *P. phaseolicola, P. glycinea,* and *P. mori.* The limited tests with mung bean indicated that only the strain isolated from it (HB-48) produced severe symptoms.

The *P. mori* strains indicated further diversity in the legume group of pseudomonads; they infected some cultivars of common bean and lima bean, but not soybean; strain PM-7 produced systemic symptoms on

TABLE 2. Reaction of common bean, lima bean, and soybean cultivars to various strains of *Pseudomonas phaseolicola*, *P. glycinea*, and *P. mori*[a]

Cultivar	Reaction to indicated strains				
	None[b]	Tolerant	<2 Lesions/cm²	2-10 Lesions/cm²	>10 Lesions/cm²
			Common bean variety		
Blue Lake White Seeded	R6[c]	*11*[d], 26, *28*, *29*, *31*, *46*, R1, R2, R6	*8*, 24, 27, *35*, *38*, *40*, 41, 42, *43*	1B, 16[SD], 23, *32*, 36[S]	*33*
Bountiful Green Pod			*8*, *11*, 23, *28*, *31*, *32*, *35*, *38*[S], 41, R1, R2	16[SD], 24[S], *29*[S], *33*[SD], 36[S], *40*[S], *43*[S], *46*, R6[S]	1B, 26[S], 27[SD], 42[S]
Common Red Mexican UI-3	*8*, *11*, *25*, *32*, *35*, PM1, PM7, PM8, R1, R2, R6	*28*, *29*, *31*, *32*, *33*, *38*, *40*, *43*, *46*, *48*	16, 23, 24, 26, 27, 41, 42	1B, 36	
Dark Red Kidney			1B, 46, PM8, R1, R6	27, *28*[SD], *29*, 36[S], *40*, *43*[SD], *48*, PM1	26[SD], *31*[SD], *33*, PM7[S], R2[S]
Great Northern US-1140	R1, R2, R6	*11*, *28*, *33*, *40*, *43*, *46*	*8*, *29*, *31*, *32*, *35*, 41, 42	1B, 16[SD], 23, 24[S], 26, 36[S], *38*	
Improved Super Green		11, R1, R2, R6	*31*, *35*	1B, *8*, 16[S], 24[S], *28*, 29[S], *32*, *33*, 36[S], *38*, *40*[S], 41	23, 26, 27, 42, *43*[S]
Improved Topnotch	*29*, R1, R2, R6	*11*, *28*, 41, *46*	1B, 23, *32*, *35*	*8*, 16, 24[SD], *31*, *33*[SD], *40*[SD], *43*[SD]	26[S], 27[SD], 36[SD], *38*[SD], 42[SD]
Pencil Pod Wax	R4	*28*, 41, R1, R6	1B, *11*, *31*, *32*, *35*, *38*[S], *46*, R2	*8*, 16[S], 23, 24[S], *29*[S], *33*[S], *40*[S], *43*[S]	26[S], 27[S], 36[S], 42[S]
Pinto UI-111	*46*, PM1, PM8, R1, R2	*11*	*8*, *28*, *31*, *32*, *33*, *35*, 41, 42, *43*, R4, R6	1B, 16[S], 23, 24, 26[S], 27, *29*, 36[S], *38*, *40*	
Red Kidney-Red Kote	R6	23, *28*, *31*, *33*, *35*, R1	1B, *11*, 16, 26, *32*, 36, *40*, 41, *43*, *46*, *48*, R2	*8*, 24, 27[S], *29*[S], *38*[S], 42[S]	
Romano		*35*, *46*, R1, R2, R6	26[S], 27, *28*, *29*, *31*, *32*	1B[SD], *8*, *11*, 16[SD], 23, 24[SD], *33*[SD], 41[SD], 42[SD]	36[SD], 38[SD], *39*[SD], *40*[SD], *43*[SD]
Small White 59	*11*, *33*, *46*, *48*, R1, R2, R6	29, *38*, *40*, 41, *43*	1B, *8*, 23, 24, 27, *28*, *31*, *32*, 42	16, *35*	36
Wren's Egg Burpee		*11*, 27, *28*, *29*	*8*, 23, 24[SD], *31*, *32*, *35*, 42[S], *43*[SD]	1B, 16[SD], 33[SD], *40*[SD], 41[S], *46*	26[SD], 36[SD], *38*[SD]
			Lima bean variety		
Florida Butter	PM1, PM8, R1, R2, R6	1B, 16, 23, *28*, *29*, *32*, *35*, *38*, *40*, 42, *46*, PM7	*8*, 24, 27, 36, 41	26, *31*, *33*, *43*	*11*
Fordhook 242			29, PM8	*11*, 23[S], 26[S], *35*[S], PM1, R1, R2, R6	1B, *8*[S], 16[S], 24[S], 27[S], *28*, *31*, *32*, 33[S], 36, *38*[S], 40, 41[S], 42, *43*[S], *46*, *48*
King of the Garden			35[S], R6	23[S], 26, 27[S], *28*, *41*, *43*[SD], *46*	1B, *8*[S], *11*[S], 16[S], 24, *29*[S], *31*[S], *32*[S], *33*[S], 36[S], *38*[S], *40*, 42[SD], *42*[S], PM1, PM7[S], PM8, R1, R2, R6
Sieva Pole	*8*, R1, R2, R6	1B, 23, 24, 26, 27, *32*, *35*, 36	*33*, *38*	*11*, *31*[S], *40*[S], *43*, *46*	16[S], *28*[S], *29*
			Soybean variety		
A100	23, *29*, *32*, *33*, PM1, PM8	26, *28*, *31*, 36			*48*
Flambeau	PM1, PM8	*29*	23, 36, R1, R2	*28*, *31*, *32*, 33, *48*	R6
Hawkeye 63	23, PM1, PM8, R1, R2		26, R6	*18*, *29*	*28*[S], *31*[S], 32, *33*, 36, *48*
Merit	*29*, *32*, PM1, PM8, R1, R6	*28*, *31*, *33*, R2	*48*		
			Mung bean variety		
Mung Bean	*29*, 42	*28*, *31*, *33*, *43*, *46*		*48*	

[a] This table represents partial data to save space. The complete table is available upon request to M. N. Schroth.
[b] Lesions per unit area are approximate. [S] = systemic chlorosis; [SD] = systemic chlorosis with stunting.
[c] Strain designations: R = *P. glycinea*; PM = *P. mori*; and numbers only = *P. phaseolicola*.
[d] *Pseudomonas phaseolicola* race 1 strains are italicized.

Dark Red Kidney and King of the Garden. *Pseudomonas phaseolicola* strains HB-20, HB-26, HB-36, HB-41, and HB-42, and *P. glycinea* strains R-1, R-4, and R-6 did not infect mulberry. These representative strains were the only ones tested.

Virulence and symptomatology.—Virulence among *P. phaseolicola* strains varied in two ways. Certain strains were less virulent than others on all hosts tested; e.g., HB-35 consistently produced a less severe reaction than HB-36. The virulence of other strains, however, depended solely upon the host; e.g., HB-38 was considerably more virulent than HB-27 on Wren's Egg, but less virulent on Bountiful Green Pod. Neither race 1 nor race 2 isolates of *P. phaseolicola* were homogeneous as to host range or virulence (Table 2). Although race 1 isolates were not virulent, or only slightly so, on Red Mexican UI-3, they varied considerably in virulence to other cultivars.

Variation in symptomatology occurred in degree and type of injury. Some isolates under the test conditions produced systemic symptoms on many hosts (HB-16, HB-26, HB-40), others on a few hosts (HB-8, HB-11, HB-23); and some caused only water-soaked lesions without producing systemic symptoms (HB-1B, HB-32). Some strains produced systemic symptoms and caused dwarfing of the host (HB-24, HB-36, HB-38), whereas others produced systemic symptoms without apparent dwarfing (HB-29, HB-41, HB-42, HB-43).

Effect of temperature and disease on plant growth.—The quantitative evaluation of *P. phaseolicola* infection with various temperatures on plant growth was determined by selecting host-pathogen combinations characterized by (i) systemic symptoms with stunting (Pencil Pod Wax and HB-16); (ii) systemic symptoms without stunting (Sieva Pole and HB-16); and (iii) local lesions without systemic symptoms (Improved Super Green and HB-28). The plants were grown at both 21 and 27 C, since temperature influences systemic symptoms (5). Plant growth was measured by excising the plants at the cotyledon scar 1 month after inoculation and weighing the fresh tops. Each treatment consisted of 25 plants.

The effect of *P. phaseolicola* infection on plant growth depended upon the bean variety, temperature, and bacterial strain. Systemic symptoms were produced in Pencil Pod Wax infected with HB-16 when grown at 21 C, but not at 27 C. The incidence of infection on the primary leaves was about equal at both temperatures. The mean wt of plant tops grown at 21 C was 2.0 g for diseased and 9.0 g for healthy ($T = 2.01$; $t = 3.5$); at 27 C, 6.0 g for diseased and 6.7 for healthy plants ($T = 2.01$; $t = 0.36$).

Infection of Sieva Pole with HB-16 resulted in production of systemic symptoms at 21 C only. The primary leaves were equally infected at both 21 and 27 C. In contrast to the HB-16-Pencil Pod Wax combination at 21 C, systemic invasion did not influence growth of Sieva Pole. Mean wt of plants grown at 21 C was 11.1 g for diseased and 12.4 for healthy plants ($T = 2.01$; $t = 0.42$); at 27 C, 9.3 g for diseased and 8.4 g for healthy plants ($T = 2.01$; $t = 0.31$).

Growth of Improved Super Green, when infected with the nonsystemic strain HB-28, was not significantly influenced by disease at either 21 or 27 C, although primary leaves had been severely infected at both temperatures. The mean of plants at 21 C was 16.2 g for diseased and 14.3 g for healthy plants ($T = 2.01$; $t = 0.04$); at 27 C, 10.0 g for diseased and 12.8 g for healthy plants ($T = 2.01$; $t = 0.81$).

Nutritional differences among isolates with varying host ranges.—We regard a host plant as a complex diagnostic medium, and the series of observable events in symptomatology which occur after inoculation with a pathogen, a reflection of the physiological properties of the pathogen. If this hypothesis is correct, strains of a pathogenic species that have a similar host range and produce equivalent host reactions should be more closely related physiologically than strains which differ in these aspects.

Pseudomonas phaseolicola strains HB-29, HB-36, HB-43, and HB-48 and *P. glycinea* strains R-1 and R-2 were selected to make this comparison. *Pseudomonas phaseolicola* HB-36 was the only strain to produce a sensitive reaction on Red Mexican UI-3, and thus would be considered race 2, using the system of Patel & Walker (6). HB-48, although designated as *P. phaseolicola* race 1 because of its reaction on Red Mexican, was isolated from mung bean and appeared to differ considerably in host range. *Pseudomonas glycinea* R-1 and R-2 were selected because they infected several varieties of common bean. Growth of the six strains was tested on the 135 different organic carbon sources used by Ballard et al. (2) in their study of *Pseudomonas*.

The nutritional tests support the hypothesis that strains which produce similar plant reactions and possess a similar host range have a more similar nutritional range than those with a different pathogenic potential. All strains grew on L-arabinose, D-glucose, D-mannose, D-galactose, gluconate, saccharate, mucate, pelargonate, succinate fumarate, L-malate, hydroxymethylglutarate, citrate, α-ketoglutarate, glycerol, quinate, L-aspartate, γ-aminobutyrate, and L-proline, and all were negative on 99 substrates [see Ballard et al. (2) for substrates not listed here or in Table 3]. Growth on an additional 16 substrates was variable among the strains (Table 3). An inspection of the growth pattern on the 16 variable substrates indicated that strains with similar host ranges had the most similar nutritional range. Accordingly, *P. phaseolicola* strains isolated from the common bean (HB-29, HB-36, and HB-43) were more similar to each other (differing in 1 to 2 tests), regardless of their race designation, than to the *P. glycinea* strains (differing in 7 to 10 tests). Strain HB-48, the unusual mung bean strain, differed in 10 to 11 tests from the other *P. phaseolicola* strains, and in 11 to 12 tests from *P. glycinea* strains.

DISCUSSION.—We view *P. phaseolicola, P. glycinea,* and *P. mori* as one population which should not be separated into distinct species. In the original description of these organisms, their distinction was based mostly on the symptoms produced and the species of

TABLE 3. Variability in utilization of substrates for growth by *Pseudomonas phaseolicola* (HB) and *P. glycinea* (R)

Substrates	HB29[a]	HB36	HB43	HB48	R1	R2
D-Xylose	−	−	+	+	−	+
Acetate	−	−	−	+	−	±
Propionate	+	+	±	−	−	±
Butyrate	−	−	−	+	−	−
Caprylate	±	−	−	±	−	−
Malonate	−	−	−	−	+	±
D-Malate	+	+	+	−	−	+
Pyruvate	+	+	+	−	+	+
Aconitate	+	+	+	+	−	−
Mannitol	−	−	−	+	+	+
meso-Inositol	−	−	−	−	+	+
p-Hydroxybenzoate	+	+	+	−	+	±
L-Serine	+	+	−	+	−	−
L-Tyrosine	−	−	−	+	−	−
Trigonelline	+	+	±	−	−	−
Sucrose	−	−	−	−	−	+

[a] *Pseudomonas phaseolicola* strains HB29, HB43, and HB48 are race 1, and HB36 is race 2. HB29 causes a tolerant reaction on Red Mexican, whereas race 1 strains produce no reaction. HB48 differs from the other strains by infecting mung bean.

plants attacked without extensive testing of host ranges. Speciation resulted in part from too small a sampling of strains and the inadvertently biased selection of strains from the population because of the use of particular plant cultivars which function as a selective isolation medium. Although there are host range and nutritional differences among strains in the population, the differences are too few to justify separation into three separate species.

The inadequate testing of the pathogenicity of many bacterial plant pathogens, coupled with the absence of physiological tests for diagnostic purposes, historically have contributed to synonymy and confusion in speciation. Recently, however, several systematic studies on the taxonomy of bacterial plant pseudomonads have been made. Lelliott et al. (4) and Misaghi & Grogan (5) showed that there was a clear distinction between the saprophytic fluorescent pseudomonads and the group-I fluorescent plant pathogens. We also recognized this group of organisms as distinct from saprophytes, both on the basis of cytochrome (9) and nutritional tests (10), and recommended that this group be designated as one species, *P. syringae*. Our work further indicated that subgroups exist within this group of pathogens and that the subgroups in general correspond to current nomenspecies. One of these subgroups, 1-B, consisted entirely of *P. phaseolicola* (8 isolates); and another, 1-C, of *P. phaseolicola* (one isolate), *P. glycinea* (one isolate), *P. morsprunorum* (one isolate), and *P. mori* (four isolates). Two isolates of *P. glycinea* existed as intermediate between these groups. All of these organisms were similar to each other but were nutritionally distinguishable from isolates of other subgroups. It now is clear that these organisms share the same hosts as well. We therefore believe that these bacteria have sufficient characters in common to be regarded as one species. However, as stated above and also in Sands et al. (10), there is an absence of physiological tests at this time to indicate the separation of these organisms from all other group-I plant pathogens. Therefore, we still recommend that the nomenspecies representing the group-I pathogens excluding *P. cichorii* be designated as pathotypes for utilitarian purposes until their status can be fully determined.

The question of races in the *P. phaseolicola-P. glycinea-P. mori* group is of particular importance in breeding plants for resistance. Cross et al. (3) described seven races of *P. glycinea*, based on pathogenicity to soybean. Earlier, Patel & Walker (7) described two races of *P. phaseolicola*, based essentially on whether or not Red Mexican cultivars were infected. Our studies suggest that there is an infinite number of races within the *P. phaseolicola-P. glycinea-P. mori* group when we consider the virulence of a strain towards a given bean cultivar as a criterion for establishing a race. Neither *P. phaseolicola* race 1 nor 2 is homogeneous with regard to virulence when tested on a number of bean cultivars. For example, race 1 strains are easily distinguished from each other by their reactions on the bean cultivars Romano, Pinto UI-111, Blue Lake White Seeded, Improved Topnotch, and Red Kidney-Red Kote. The hazard of relying on one or several host-pathogen combinations to identify races as discrete genotypes is exemplified by the reaction of strains R-4 and HB-46 on Red Mexican UI-3, Dark Red Kidney, or Pinto UI-111. The host reactions suggest that they were of similar virulence, and should be designated as *P. phaseolicola* race 1. However, use of other bean and soybean cultivars indicate otherwise.

An inspection of data in Table 2 suggests that the separation of *P. phaseolicola* into race 1 and race 2 on their reaction on Red Mexican UI-3 is in part a separation of populations on the basis of virulence (11). Strains exhibiting moderate to weak virulence toward most bean cultivars do not infect the more resistant Red Mexican varieties, whereas the strains with high virulence do. We therefore consider the designation of races on the basis of the horticultural variety that is attacked as being rather pointless, unless it has economic value. Rather, we emphasize the importance of not relying on several isolates to provide an example

of the pathogenic potential of a species when testing plants for resistance.

In view of the lack of physiological tests, the best method to determine whether or not an unknown isolate belongs to the *P. phaseolicola-P. glycinea-P. mori* group is to test it on one of the "universal hosts" such as Fordhook 242 or King of the Garden, mindful that our study of strains may still represent only a portion of the population. Some isolates of *P. syringae* produce water-soaking on lima beans (the brown spot disease) and should not be confused with the *P. phaseolicola-P. glycinea-P. mori* group.

It is interesting to speculate why *P. mori* has the capacity to infect legumes, whereas none of the tested legume pseudomonads infects mulberry. Perhaps these pseudomonads evolved from a strain with a broader pathogenic potential, and *P. mori* retained a potential to infect legumes; whereas *P. glycinea* and *P. phaseolicola* became more specialized, losing pathogenic versatility. This capacity of *P. mori* to infect legumes might be termed as abeyant potential; i.e., the bacterium has the pathogenic potential to infect a greater spectrum of plant varieties than is normally expressed, because it possesses a complement of genes relating to disease initiation greater than that required for pathogenesis in mulberry. Abeyant potential would give pathogens greater capacity to perpetuate themselves as they encounter a favorable habitat, such as a new plant cultivar. We speculate, then, that the ascribed development of new races in some cases are in effect the reflection of an increase in a population of a clone which hitherto did not have a susceptible plant in which to express its pathogenic potential, and had existed either as a saprophyte or as a quasi pathogen.

It is noted that *Fusarium pisi,* a common cause of pea root rot, curiously has a peculiar host range similar to *P. mori.* The perfect stage of this fungus occurs on mulberry in Japan (8).

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