Role of Natural Seed Infection by the Web Blight Pathogen in Common Bean Seed Damage, Seedling Emergence, and Early Disease Development

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ABSTRACT

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The web blight pathogen, *Thanatephorus cucumeris* (*Rhizoctonia solani*), was seedborne in both white- and pigmented-seeded genotypes of common bean *(Phaseolus vulgaris)* and was associated with blemishes and discoloration on the seed coat. The pathogen was consistently isolated from blemished seeds of six genotypes grown in fields where the predominant AG type of the pathogen was AG-1-IB (microsclerotial). Ninety-five percent of the isolates from seeds were AG-1-IB, and the remainder were AG1-IB (macrosclerotial), AG-2, and AG-4. The pathogen was isolated less frequently from unblemished seeds. *T. cucumeris* was isolated from up to 17% of unblemished seeds from black-seeded genotypes, including the reported web blight resistant breeding line HT-7719. All seedborne isolates were pathogenic to bean seedlings. When seedborne in genotypes with white, red mottled, and black seed, *T. cucumeris* significantly ($P < 0.05$) reduced seedling emergence and establishment. Isolates AG-1-IB and AG-2-2 added to the soil caused only slight injury to bean seedlings, whereas soil isolate AG-4 was very detrimental to seedling emergence and growth.

Web blight (WB) of common beans, *Phaseolus vulgaris* L., caused by *Thanatephorus cucumeris* (Frank) Donk (anamorph: *Rhizoctonia solani* Kühn), is a yield-limiting disease in Central America and the Caribbean (14,29). WB causes mild to severe foliage blight resulting in bean seed yield losses up to 90% (14).

The disease is spread by airborne basidiospores (6,10), mycelial bridges between plants, rain-splashed sclerotia, and infested soil debris (13,14). However, the role of seed infection in WB epidemics is unknown. Rates of natural seed infection by *T. cucumeris* (AG-1 and AG-1-IB) range from 1.5 to 65% in bean genotypes of Middle American and Andean origin, respectively (13,16), but there are no reports on damage to seed appearance.

Isolates of *T. cucumeris* causing web blight differ in various cultural, ecological, and pathological characteristics from those causing seedling diseases (3,8,11,22,23). Most of the isolates causing web blight of dry beans belong to intraspecific groups AG-1-IB (including microsclerotial and macrosclerotial types) and AG-2-2 (6,12, 15,26), whereas those causing damping-off or root rot are mainly of groups AG-3, AG-4, and AG-5 (22,33).

Seed infection by soil-inhabiting isolates of the anamorph, *R. solani*, results in severe pre- and postemergence damping-off or root rots (4,7,20), which reduce plant stands (25). Resistance to seed infection and subsequent seedling damage has been associated with seed coat color (9,28). Resistance is attributed to the higher phenol content and integrity of the seed coat of colored-seeded compared to white-seeded genotypes (28). No association between seed coat color and resistance to isolates of *T. cucumeris* has been found.

The objectives of this study were to determine (i) if natural infection by *T. cucumeris*, belonging to *R. solani* AG-1-IB, can cause bean seed coat damage and reduce subsequent seedling emergence and establishment, and (ii) if white- and pigmented-seeded genotypes differ in their degree of susceptibility to this pathogen.

MATERIALS AND METHODS

Seed collection. Seeds were collected in 1992 and 1993 from pods (not in contact with soil to prevent soil contaminants) of WB-infected plants of six bean genotypes grown in replicated plantings in Buena Vista in the San Juan Valley (400 masl) of the Dominican Republic. In Buena Vista, the predominant *T. cucumeris* isolates that cause WB belong to AG-1-IB (microsclerotial type) (15). Genotypes represented both Andean and Middle American common bean gene pools; those of Middle American origin were white-seeded Arroyo Loro and Anacaona and black-seeded lines H-270 and HT-7719. Red mottled line PR-PC-450 and cultivar PC-50 were Andean. HT-7719 and Anacaona were reported to be moderately resistant to web blight (14,17), whereas the other genotypes were susceptible in the field.

Seeds collected from WB-infected plants were divided into two categories: (i) seeds blemished with yellow-tan spots or discolored areas on the seed coat, and (ii) unblemished seeds with normal color and no obvious discoloration. Seeds with normal seed coat color were obtained from healthy plants in a field without a history of WB and were used as controls.

Pathogen isolation and strain identification. One hundred seeds from each combination of year, genotype, and seed category and from controls were tested for the presence of the WB pathogen. The fungus was isolated on 2% water agar (WA) amended with metalaxyl (Ridomil 2E, 25% a.i.) at 30 µl/liter and streptomycin sulfate at 60 µg/liter (1 g/ml of distilled water). The embryo end of each seed was placed within a shallow pit made in the agar surface by a microspatula. Plates were incubated at 25 to 28°C for 48 h, after which Rhizoctonia-like colonies were counted and transferred to potato-dextrose agar (PDA) for AG-typing (18) and determination of cultural characteristics (24,31). Tester strains were kindly provided by A. Matsuda (Japanese International Cooperation Agency).

Pathogenicity tests. Twenty isolates from blemished seeds of each genotype were selected for pathogenicity tests. Isolates WB-BV of *T. cucumeris* (AG-1-IB from leaf lesions) and SD-SCR of *T. praticola* (AG-4) from a decayed bean seed (15) were included as controls. Isolates from unblemished seeds were also tested.

Pathogenicity was assessed on 2-week-old PC-50 seedlings grown in polystyrene cups (9 cm diameter) containing a heat-pasteurized soil mixture (sand:clay loam 1:1 vol/vol, pH = 8.3). Two plants per isolate were inoculated at the base of the petiole of each expanded unifoliolate. Inocula were 0.5-cm mycelial plugs taken from the edge of colonies grown on PDA for 48 h.

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Agar plugs without mycelia were used as controls. Inoculated plants were placed in a mist chamber at 25 to 28°C for 48 h, then rated for disease severity using a scale of 1 to 5, where 1 = no disease; 2 = small, discrete, irregular water-soaked and necrotic lesions; 3 = coalescent water-soaked and necrotic lesions; 4 = leaf defoliation and stem girdling; and 5 = dead plant.

Seedling infection tests. The effect of seedborne *T. cucumeris* on seedling emergence and growth was tested in a screenhouse in San Juan de la Maguana, Dominican Republic, in 1992 and 1993. For both years, the screenhouse temperature ranged from 10 to 28°C, relative humidity was 30 to 90%, and the day length of 12 h was not supplemented with artificial light. Seed representing the controls and both categories of the six genotypes were arbitrarily selected from the field collections and planted in 20-cm-diameter plastic pots. Each genotype-seed category combination was replicated seven times. Each pot contained 15 seeds. The pots were arranged in a randomized complete block design with a factorial arrangement of genotypes and seed categories within seven blocks. The test was irrigated no less than three times daily to favor disease development (14). Seedlings were counted 7 to 9 days after emergence. Seedling survival, height, fresh top weight, and fresh root weight were determined 2 weeks after emergence. Lesions of diseased plants were assayed for the pathogen.

In 1993, another experiment was conducted in the screenhouse to determine the effect of *T. cucumeris* soil inoculum on seedlings of PC-50, H-270, and Anacaona. Two isolates, WB-BV and WB-LV, representing the AG-1-IB and AG-2-2 types, respectively, and one AG-4 isolate, SD-SCR, were grown on PDA. Mycelial plugs were transferred to autoclaved wetted (200 g of seed + 120 ml of water) oat seeds covering the bottom of 500-ml flasks. After 2 to 3 weeks incubation, the colonized oat seed was air-dried and separated. The oat-seed inoculum containing mycelia or sclerotia of the isolates was thoroughly mixed with sterile sandy loam soil to obtain 50 or 100 infested oat seeds/kg of soil. The control soil contained sterile oat seed. Unblemished seeds of PC-50, H-270, and Anacaona, collected from healthy plants, were planted in plastic pots filled with infested or control soil. Fifteen seeds of each line or cultivar were planted per pot. Pots were arranged in a completely randomized block design with a factorial arrangement for isolate, inoculum level, and genotype. Each treatment was replicated seven times. The pots were irrigated daily in a manner that avoided splashing of soil and inoculum. Seedling emergence and growth were evaluated as previously described.

Statistical analysis. Weighted least squares analysis for category responses and contrasts were used to evaluate the effects of blemished and unblemished seeds and genotypes on the recovery of the WB pathogen (1). Analysis of variance and contrasts were used to evaluate main effects and interactions in the screenhouse experiments (32). Data from each year were analyzed separately.

RESULTS

Isolation of the WB pathogen from blemished or discolored seeds. *T. cucumeris* was isolated more frequently from blemished or discolored seeds collected from infected plants (Table 1). No other pathogen was isolated from blemished or discolored seeds. In 1992, WB was severe and the pathogen was isolated from a large percentage of the blemished seeds of the six genotypes. The WB pathogen also was found in 13 and 17% of unblemished seeds of black-seeded HT-7719 and H-270, respectively. Even though the disease was less severe in 1993, many blemished seeds contained the pathogen. In both 1992 and 1993, there were significant differences among genotypes in frequency of isolation of the pathogen from seed. The WB pathogen was isolated from less than 1% of unblemished seeds in all the genotypes in 1993. In 1993, the seed category-by-genotype interaction was significant for frequency of recovery of the pathogen. This interaction was due to differences in pathogen recovery frequency among the genotypes with blemished seed. Neither *T. cucumeris* nor *Rhizoctonia* species were isolated from control seeds collected in 1992 and 1993.

Pathogen identification: anastomosis groups. Fungi isolated from seeds of WB-infected bean plants had mycelial characteristics typical of the anamorph *Rhizoctonia solani.* Ninety-five percent of these isolates were assigned to group AG-1-IB. Linear growth ranged from 20 to 29 mm/day on PDA at 25°C. Mycelial color was initially light brown but turned dark brown with age. Spherical microsclerotia of 0.5 to 1.0 mm in diameter were produced abundantly on the surface of amended WA and PDA. Microsclerotia initially were white and turned dark brown when mature. These characteristics are the same as those of AG-1-IB isolates from leaf lesions. In a few instances, other types were isolated along with the microsclerotial type. These were assigned to AG-1-IB (macrosclerotial type > 2 mm in size), AG-2-2 and AG-4. Because these groups were infrequently found, they were not considered further.

Pathogenicity tests. *T. cucumeris* isolates from seed in either year were pathogenic. All isolates produced high disease severity ratings of 4 or 5. Symptoms on leaves 36 h after inoculation were initially water-soaked lesions, which turned olive green and later brown. Lesions on the hypocotyl extended 1 to 2 cm in both directions from the inoculation point and were light to dark brown. Further disease development resulted in defoliation or plant death (rating 4 or 5).

Seedling infection tests. Blemished or discolored seeds had significantly ($P < 0.05$) lower seedling emergence and produced seedlings with reduced survival and growth when compared to plantings of unblemished and control seeds (Table 2). Seedlings grown from blemished seeds of Arroyo Loro, Anacaona, and H-270 had dry cankers at the base of the stems that caused death of the seedlings. Isolates from these cankers resembled in appearance and pathogenicity (rating of 5) those of AG-1-IB causing WB. Root weight of seedlings from blemished, unblemished, or control seeds was not affected. Seed category-by-genotype interaction was significant in both years for all variables except root weight in 1992. Interactions indicated that seed blemishing affected the genotypes differently. Foliar symptoms of WB did not develop in any of the seedlings.

Isolates WB-BV (AG-1-IB) and WB-LV (AG-2-2) in soil did not significantly reduce either seedling emergence or seedling survival (Table 3). High inoculum levels of both WB isolates reduced seedling development. In contrast, the seed isolate SD-SCR (AG-4), normally found in soil, reduced seedling emergence and seedling development of the three bean genotypes.

Table 1. Percent seed yielding web blight fungus, *Thanatephorus cucumeris* (*Rhizoctonia solani*) AG1-IB, from blemished and unblemished seed collections from six bean genotypes

	1992 seed[w]			1993 seed[w]		
Genotype	Blemished	Unblemished	Mean[x]	Blemished	Unblemished	Mean[x]
Arroyo Loro	70[y]	1	35.5 a	50	1	25.5 d
Anacaona	84	2	43.0 b	20	0	10.0 b
HT-7719	80	13	46.5 b	6	0	3.0 a
H-270	94	17	55.0 c	58	1	29.5 d
PR-PC-450	81	2	41.5 ab	28	1	14.5 bc
PC-50	81	1	41.0 ab	35	1	18.0 c
Check	0	0	0	0	0	0
Mean[z]	81.7	6.0		41.2	0.7	

[w] Seed were collected from web blight infected plants.
[x] Genotypes with different letters differed significantly ($P < 0.05$).
[y] Percent seed yielding the web blight fungus.
[z] Blemished versus unblemished means differed significantly ($P < 0.01$).

For all genotypes, high inoculum levels of the SD-SCR isolate depressed emergence, survival, height, and top and root weight. Anacaona seedlings sustained the most injury from this isolate.

DISCUSSION

The WB pathogen *T. cucumeris* (*R. solani* AG-1-IB microsclerotial type) infects white- and pigmented-seeded common beans, causing unusual yellow to tan pigmentation in the seed coat of white and red-mottled seed types and discoloration of black seed. Blemishes, stains, or discoloration of bean seed coats can reduce the USDA seed grade (34) and the commercial value of seeds. In other legumes, such as soybeans, discoloration of the seed coat is also an important indicator of seed quality (30). Therefore, *T. cucumeris* not only reduces dry bean seed yield but also may reduce market value of harvested seeds.

The WB pathogen visibly affected the bean seed coat regardless of seed color, genotype, or reported resistance. Previous reports (9,27,28) suggested that phenolic compounds in seed coats of pigmented-seeded bean genotypes conferred resistance to seedling diseases caused by *R. solani*. Our results suggest that seed coat color does not confer resistance to seed infection by the WB pathogen. The different results may be due to the different AG groups involved in each study.

All of the seedborne AG-1-IB isolates obtained from the six bean genotypes were pathogenic to bean seedlings. The fact that pathogenic isolates exist on or in symptomless seeds of WB-resistant genotypes may have serious implications in the spread of the pathogen from one region to another in beans and other legumes. Isolates of AG-1 cause WB of common beans, cowpeas, and soybeans in tropical and subtropical regions worldwide (2,14).

Intraspecific groups of *Rhizoctonia* spp. are ecologically specific in relation to the different plant organs attacked (22). WB isolates would be predicted to cause more damage to the foliage of mature bean plants than to emerging or developing seedlings. In our study, the WB pathogen, when seedborne, reduced seedling emergence and also seedling survival and development. In cowpea, seedborne WB isolates incited postemergence damping-off in the field as well as in the greenhouse

Table 2. Effect of seedborne *Thanatephorus cucumeris* from blemished and unblemished seed of six common bean genotypes on seedling emergence and growth in screenhouse pot experiments

		1992					1993				
Genotype	**Seed category**	**SE[x]**	**SV**	**TH**	**TW**	**RW**	**SE**	**SV**	**TH**	**TW**	**RW**
Arroyo Loro	Blemished[y]	13.57	12.57	10.09	1.19	0.93	9.57	9.57	8.65	1.41	0.56
	Unblemished	13.29	13.29	12.97	1.56	0.97	13.86	13.57	8.96	1.71	0.68
	Control	15.00	14.86	12.79	1.47	0.91	14.86	14.86	9.53	1.49	0.54
Anacaona	Blemished	10.57	9.57	7.39	1.22	0.86	10.29	10.29	8.60	1.03	0.56
	Unblemished	14.29	13.29	9.74	1.37	0.76	11.86	11.71	10.31	1.16	0.58
	Control	14.29	14.29	10.71	1.69	0.86	14.57	14.57	9.73	1.11	0.58
H-270	Blemished	12.57	11.71	10.57	1.29	0.59	13.29	12.86	13.64	1.53	0.53
	Unblemished	14.86	14.43	12.89	1.59	0.60	14.71	14.43	13.88	1.65	0.56
	Control	14.29	14.57	12.31	1.53	0.64	14.14	14.14	13.24	1.59	0.60
HT7719	Blemished	12.43	12.00	12.11	1.20	0.73	11.57	11.26	9.21	1.04	0.39
	Unblemished	13.71	13.43	13.53	1.40	0.64	15.00	15.00	10.59	1.57	0.47
	Control	14.86	14.71	14.27	1.40	0.81	14.86	14.86	11.60	1.60	0.56
PC-50	Blemished	12.29	12.00	12.44	1.87	0.94	9.71	8.86	11.16	2.20	0.59
	Unblemished	12.00	11.71	13.96	2.23	1.01	13.71	13.43	11.74	2.33	0.61
	Control	14.57	14.57	13.79	2.17	0.93	15.00	14.86	13.24	2.60	0.77
PR-PC-450	Blemished	13.00	13.00	11.06	2.30	0.90	9.71	9.57	8.73	2.47	1.12
	Unblemished	13.86	13.86	12.57	2.51	0.93	13.57	13.57	10.00	2.53	0.81
	Control	14.86	14.86	12.91	2.33	0.91	14.57	14.57	9.54	2.07	0.67
	LSD[z]	1.05	1.22	0.74	0.19	0.18	1.30	1.39	0.89	0.20	0.14

[x] SE = emerged seedlings out of 15 seeds; SV = number of surviving seedlings; TH = top height in cm; TW = top weight in g; RW = root weight in g.
[y] Blemished = seeds from infected plants showing yellow to tan spots or discolored areas on the seed coat; unblemished = seeds from infected plants with no discoloration; control = seeds with normal seed coat color obtained from healthy plants.
[z] Least significant difference ($\alpha = 0.05$) for comparing means of genotype, seed category combinations.

Table 3. Effect of soil infestation with two isolates of the web blight pathogen *Thanatephorus cucumeris* and a soil isolate of *Thanatephorus praticola* on seedling emergence and growth

Isolate	**Emergence[x]**			**Survival[x]**			**Top height[x]**			**Top weight[x]**			**Root weight[x]**		
Inoc. level[w]	**1[y]**	**2**	**3**	**1**	**2**	**3**	**1**	**2**	**3**	**1**	**2**	**3**	**1**	**2**	**3**
AG-1 (WB-BV)															
0	15.00	15.00	13.33	15.00	15.00	12.67	12.03	13.10	10.90	2.16	1.26	1.26	0.66	0.50	0.73
50	15.00	15.00	14.33	15.00	15.00	14.33	11.63	12.27	10.70	2.00	1.20	1.23	0.53	0.50	0.73
100	14.67	15.00	14.67	14.67	15.00	14.67	11.33	11.60	10.20	1.60	1.23	1.06	0.90	0.56	0.53
AG-2 (WB-LV)															
0	14.67	15.00	13.33	14.67	15.00	12.67	12.03	13.10	10.90	2.16	1.26	1.26	0.66	0.50	0.73
50	14.33	14.67	14.67	14.33	13.67	14.67	12.07	11.50	10.70	1.83	1.06	1.10	0.50	0.50	0.46
100	15.00	14.00	15.00	14.67	12.33	14.00	10.33	10.67	9.50	1.43	0.86	0.93	0.70	0.60	0.53
AG-4 (SD-SCR)															
0	14.67	15.00	13.33	14.67	15.00	12.67	12.03	13.10	10.90	2.16	1.23	1.26	0.66	0.50	0.73
50	15.00	13.33	9.67	15.00	13.33	9.67	9.73	9.73	7.10	1.60	1.16	0.96	0.30	0.40	0.40
100	10.33	7.33	1.00	10.00	7.33	1.00	6.97	7.00	1.00	1.13	0.73	0.50	0.26	0.23	0.10
LSD[z]		2.04			2.23			1.31			0.18			0.14	

[w] Inoculum levels in propagules/kg of soil.
[x] Emergence = emerged seedlings from 15 seeds; survival = surviving seedlings; top height in cm; top weight in g; root weight in g.
[y] Genotypes: 1 = PC-50; 2 = H-270; 3 = Anacaona.
[z] Least significant difference ($\alpha = 0.05$) for comparing means of genotype, isolate, inoculum level combinations.

(23). Plant emergence rates in our study were 70 to 80% in 1992 and 65 to 89% in 1993 for blemished white- and pigmented-seeded genotypes. However, this was higher than emergence rates for seeds that had *R. solani* isolates causing seed decay or damping-off (7,20). Although WB isolates are less specific than originally predicted, damage caused by these isolates is more pronounced aboveground than below. This supported our finding that isolates of the WB pathogen added to soil had little effect on seedling emergence when compared to a seed isolate of AG-4. Similar results were obtained with other WB isolates (17). Other reports state that WB isolates in soil had little effect on the number of seedlings emerging and surviving in field plots as well as in the greenhouse (35), and that isolates producing microsclerotia, when added to soil, were less pathogenic than those isolated from root rots or damped-off seedlings (3).

In contrast with the knowledge that intraspecific groups of *R. solani* are epidemiologically and ecologically specific (22), isolates of the WB pathogen were reported to cause severe root rot and hypocotyl damage in common beans and soybeans using artificial soil infestation (6,12,21). Factors such as growth media, inoculation methods, amount and type of inoculum, isolate variability, and host genotype susceptibility could explain the damage recorded on roots and hypocotyl by an isolate that only causes WB in the field.

In Colombian and Dominican bean fields, WB isolates of AG-1-IB and AG-2-2 were associated mainly with the aerial portion of the plant and were found at very low population densities in soil (6,17). Isolates of *R. solani* causing brown patch and foliar blight of tall fescue also had low propagule densities in soils (19,36). Thus, seed appears to be a more favorable ecological niche for the spread and survival of aerial isolates of *R. solani*, which may have only a cursory association with soil. In some cases, the pathogen may lead an aerial existence independent of soil, as suggested by other investigators (5,23).

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