Bacterial Vascular Necrosis and Rot of Sugarbeet: General Description and Etiology

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ABSTRACT

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Vascular necrosis and rot of sugarbeet in California is caused by specific strains of the *Erwinia carotovora* group of soft-rotting pathogens which can be distinguished from members of the group on the basis of host reaction and biochemical tests. The bacteria invade the vascular tissue of the petiole and roots and usually cause an extensive rot. Vascular bundles of infected roots are necrotic and areas surrounding the infected bundles turn pink upon exposure to air. The disease is found in most California sugarbeet plantings. The incidence of infection is usually 3-5%, but sometimes occurs in excess of 40%. Injury is necessary for infection, and temperatures of 25-30 C favor rapid disease development. Root yield was reduced from 76.8 metric tonnes/hectare (ha) in control plots to 37.9 metric tonnes/ha in plots where plants were injured and inoculated when 8 wk old. Infections which occur early in the season are more important since the reduction in root and sugar yield was significantly greater when 8-wk-old plants were inoculated in the field than when 12 or 15-wk-old plants were inoculated. The pathogen also infected tomato and chrysanthemum plants in greenhouse inoculations and caused typical blackleg symptoms on potatoes grown at 18 C. Some strains of *Erwinia carotovora* var. *atroseptica*, *Erwinia carotovora* var. *carotovora*, and *Erwinia chrysanthemi* also infected sugarbeet. A few strains of the latter two species caused blackleg of potatoes in greenhouse tests at 18 C. Populations of the sugarbeet *Erwinia* sp. in soils planted to sugarbeets ranged from 2.1×10^2 to 2.8×10^6 colony-forming units/g of soil. Populations declined rapidly after harvest and the organism was not detected in soils throughout the winter. The *Erwinia* sp. was not isolated from sugarbeet seed.

An unusually high incidence of bacterial root rot occurred in a number of sugarbeet (*Beta vulgaris* L.) plantings in the San Joaquin Valley of California in 1972. The incidence of the disease was generally 3-5%, although it reached 30-40% in some fields. Because of the symptomology, the disease was named vascular necrosis and rot of sugarbeet. It subsequently has been found in Washington (15, 17), Arizona (M. E. Stanghellini, *personal communication*), and Idaho (D. L. Mumford, *personal communication*).

Bacterial decay of sugarbeet roots has been reported for many years, and the causal organisms have been ascribed to the genera *Erwinia* (5, 13, 16) and *Pseudomonas* (2, 5, 14, 21). One of these, *Erwinia betivora*, is listed as a synonym of *Erwinia carotovora* in Bergey's Manual (11). Our investigation of beets exhibiting vascular necrosis and rot revealed that specific strains of bacteria belonging to the *Erwinia carotovora* group of soft-rotting pathogens always were associated with the disease. A description of the symptoms and etiology of this disease and results of several tests to identify the pathogen are presented in this paper. Preliminary reports of this disease have been published (17, 18, 20).

MATERIALS AND METHODS

Identification.—Thirty-one isolates were obtained from diseased sugarbeets in California over a 3-yr period. Isolations were made from necrotic vascular bundles of diseased roots and petioles using modified Miller-Schroth selective medium (19) with mannitol as the carbon source (MSM). Tentative identification of the sugarbeet *Erwinia* (SBE) was made by comparing colony morphology on MSM and hypersensitivity on tobacco according to Klement (9). Isolates of *E. carotovora* var. *atroseptica*, *E. carotovora* var. *carotovora*, and *E. chrysanthemi* were obtained from The International Collection of Phytopathogenic Bacteria, Hertfordshire, England; The National Collection of Plant Pathogenic Bacteria, Davis, California; New Zealand Plant Diseases Division, Auckland, New Zealand; S. M. Alcorn, Tucson, Arizona; R. S. Dickey, Ithaca, New York; A. Kelman, Madison, Wisconsin; T. D. Miller, Wooster, Ohio, and D. C. Sands, New Haven, Connecticut.

Tests used for distinguishing the various species and varieties of soft-rot erwinias included the oxidase test, anaerobic fermentation of glucose, growth at 36 C, formation of reducing compounds from sucrose (11), and utilization of various substrates for growth. Substrate utilization was determined by incorporating 0.1% (w/v) filter-sterilized substrates into the mineral base of Ayers

et al. (1), placing approximately 0.03 ml of a bacterial suspension of 10^7 colony-forming units (cfu)/ml on the medium and observing growth of the bacteria for periods up to 3 wk. Ten isolates each of *Erwinia carotovora* var. *atroseptica*, *Erwinia carotovora* var. *carotovora*, and *Erwinia chrysanthemi* were compared with 10 isolates of the SBE.

Greenhouse studies of infection.—Pathogenicity of isolates was determined by inserting a small amount of inoculum into a petiole near the crown of 1-mo-old sugarbeet seedlings (cultivar US H9) using a sterile toothpick. Plants were grown in the greenhouse at a temperature range of 25-30 C. Symptoms were compared with those induced by a representative culture of SBE (UCBPP193). The effect of temperature on disease development and the effect of injury on infection were investigated by inoculating 1-mo-old US H9 sugarbeet seedlings in the greenhouse with UCBPP193 by spraying foliage to run-off with a suspension of 10^6 cfu/ml in sterile tap water (STW). Inoculum was grown on Luria slants (LU) (12). Some beets were injured prior to inoculation by puncturing two petioles in the crown area with a toothpick. Plants were maintained in the greenhouse at 18, 24, and 30 C. Control plants were sprayed with STW. This experiment was performed three times with 10 beets per treatment.

Pathogenicity to other plants.—To compare the effects of this pathogen and related bacterial spp. on sugarbeet and other hosts, 2-mo-old sugarbeet (*Beta vulgaris* L. 'US H9'), tomato (*Lycopersicum esculentum* Mill. 'Bonnie Best'), potato (*Solanum tuberosum* L. 'White Rose'), and chrysanthemum (*Chrysanthemum morifolium* Ramat. 'Torch') were inoculated with 57 isolates of *E. carotovora* var. *carotovora*, 31 isolates of *E. carotovora* var. *atroseptica*, 34 isolates of *E. chrysanthemi*, and 31 isolates of the SBE (Table 1). A small amount of inoculum was taken from a 24-hr LU slant on a sterile toothpick and inserted into the stem near the axil of the leaf. Three to five plants of each variety were inoculated with each isolate. Potato plants were incubated in the greenhouse at 18 and 24 C; all other plants were incubated at 24 C.

Seed isolation.—The possibility that the SBE was seed-borne was determined by grinding 1 g of untreated beet seed in 10 ml sterile distilled water (SDW) with mortar and pestle. The slurry was allowed to settle for 1 min and 0.1 ml of a 10^{-2} dilution was spread with a bent glass rod on MSM plates. This procedure was repeated with nine additional 1 g samples from different seed lots.

As an additional check for possible seed infestation, 1,000 untreated beet seed were planted in pasteurized vermiculite, grown in the greenhouse at a temperature range of 25-30 C, and observed for disease at emergence and again after 3 wk. Four seed lots were assayed in this way.

Enrichment for low populations of the SBE in seed was attempted by placing 50 untreated seed on a moistened filter paper in a petri dish and incubating at 28 C. A total of 500 beet seeds were assayed in 10 tests. When approximately 90% of the seeds had germinated, they were washed in STW and 0.1 ml aliquots of 10^{-1}, 10^{-2}, and 10^{-3} dilutions were spread on MSM. Plates were incubated at 28 C for 48 hr and suspect colonies were tested for hypersensitivity on tobacco, the production of reducing substances from sucrose, and pathogenicity on sugarbeet seedlings.

Recovery efficiency of the selective medium and isolation from soil.—The selective medium, MSM was compared with nutrient agar (NA) and LU to determine the efficiency of recovery of the SBE on various media. Aqueous suspensions (10^2 to 10^3 cfu/ml) of six different SBE isolates were prepared from a 24-hr-old LU slant and 0.1 ml aliquots were pipetted onto five Petri dishes, each containing MSM, NA, or LU. Bacteria were distributed with a bent glass rod and incubated at 28 C for 2 to 3 days.

The recovery efficiency of the SBE on MSM was tested with Lost Hills sandy loam soil from three different locations. One ml of various concentrations of aqueous suspensions of a 24-hr LU culture of UCBPP193 were mixed thoroughly with 100 g of soil in a plastic bag to obtain populations of 10^1 to 10^6 cells/g of soil. Soils had about 15% moisture after adding the bacterial suspension. A 1-g sample was immediately removed, added to 10 ml SDW and 0.1-ml aliquots from aqueous dilutions were pipetted to MSM. Plates were incubated at 28 C for 2-3 days.

The populations of the SBE in natural field soils

TABLE 1. Substrate utilization and other characteristics of the sugarbeet *Erwinia* and other soft-rot *Erwinia* species[a]

Substrate	*E. carotovora* var. *carotovora*	*E. carotovora* var. *atroseptica*	Sugarbeet *Erwinia*	*E. chrysanthemi*	*E. cypripidii*[b]	*E. rhapontici*[b]
Citrate[c]	+	+	−	+	+	+
Galacturonate	+	+	−	+	+	+
α-Methyl glucoside	−	v[d]	+	−	−	−
Lactose	+	+	+	v	−	+
Malonate	−	−	−	+	v	+
Melezitose	−	−	−	−	−	+
Melibiose	+	+	−	v	v	+
m-Tartrate	−	−	−	+	+	−
Xylose	+	+	+	+	+	−
Growth at 36 C	+	−	+	+	+	−
Reducing compounds from sucrose	−	+	+	v	−	−

[a]Ten isolates of each species and the sugarbeet *Erwinia* were tested.
[b]Data from Bergey's Manual (11).
[c]Readings taken at 3 days.
[d]v = variable results; 21-79% of these strains were positive.

planted to sugarbeets were determined by taking soil samples at random from one sugarbeet field in Kern County and one in Yolo County, California, with incidences of 30% and 5% diseased beets, respectively. Samples (400 g) were collected from the top 15-cm of soil at least 15 cm away from the sugarbeet plants 20 days before harvest, 1 day after harvest, and 1 and 2 mo after harvest. Soil also was assayed from six other sugarbeet fields throughout the state with <5% incidence of disease. The soils were transported to the laboratory in plastic bags and stored at 5 C. All samples were assayed within 48 hr. Each sample was mixed thoroughly and assays were made from five subsamples per soil. Subsamples were equivalent to 1 g oven dry soil. These subsamples were suspended in 10 ml of SDW in 125-ml Erlenmeyer flasks and shaken on a Burrill wrist-action shaker for 15 min and assayed by standard dilution plating on MSM. Plates were incubated for 3 days at 28 C. Representative orange colonies were tested for pathogenicity to sugarbeet and positive isolates were recorded as the number of *Erwinia* sp. per gram of oven dry soil.

Field study.—A field trial was conducted at Davis, California, to determine the pathogenicity of an SBE isolate from sugarbeet and the effects of wounding the

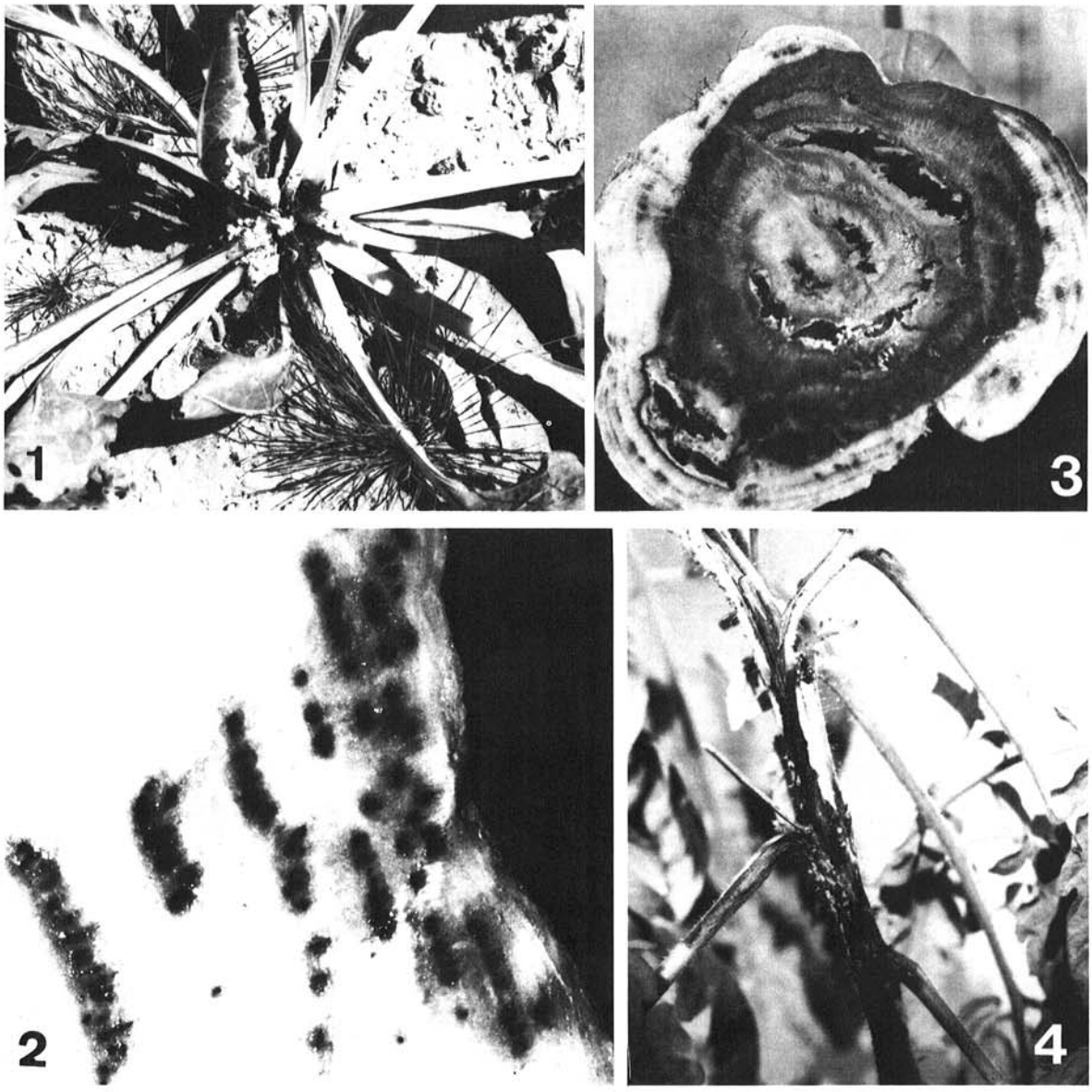

Fig. 1-4. Disease symptoms caused by the sugarbeet *Erwinia* sp. on sugarbeet and tomato. **1)** An infected sugarbeet plant showing necrotic petioles, rotting, and a whitish foam at the base of the petioles. **2)** A cross section of an infected sugarbeet root with necrotic vascular bundles. **3)** Cross section of a sugarbeet root exhibiting advanced rotting in the center of the root and the characteristic necroses of vascular bundles near the periphery. **4)** Infection of a tomato stem (cultivar Bonnie Best) showing necrosis and wilting 7 days after inoculation with the sugarbeet *Erwinia* sp.

host plant and timing of inoculation upon disease development. Treatments included combinations of three dates of inoculation, and wounding versus not wounding. Controls consisted of wounded, noninoculated plants and noninoculated, nonwounded plants. The experimental design was a randomized complete block with six replications. Individual plots were four rows wide and 9.2 m long. The two center rows were treated leaving two buffer rows between each plot.

Inocula of the SBE isolate (UCBPP193) were prepared from a 24-hr culture on LU to provide a final concentration of 10^8 cfu/ml and this was applied with a CO_2 pressurized sprayer to deliver approximately 0.1 ml into the crown of each plant.

Sugarbeets (cultivar US H9) were planted 14 May and inoculated when either 8, 12, or 15 wk old. Beet plants were wounded by forcing six nails protruding 5 cm from a wooden disk (8 cm diameter) into the crown of the beet by means of an attached handle. The nails punctured the petioles and the pressure of the disk cracked petioles at the crown region.

Beets were harvested 8 October, when 21 wk old. The beets in the treated rows of each plot were dug, topped, and weighed, and a random sample of 10 roots was taken for sugar and tare analyses. The remaining roots were cut and rated on a scale of 0, 15, 50, 85, and 100% rot.

RESULTS

Identification of the pathogen.—The strains of bacteria isolated from diseased sugarbeet possessed peritrichous flagella, were Gram-negative, oxidase-negative, fermented glucose within 48 hr and therefore were considered *Erwinia* sp.

TABLE 2. Effect of temperature and injury on the incidence of vascular necrosis and rot of 1-mo-old sugarbeet seedlings inoculated with the sugarbeet *Erwinia* sp. (UCBPP193) and grown in the greenhouse

	Temperature		
Treatment	18 C	24 C	30 C
Not inoculated	0/30[a]	0/30	0/30
Inoculated	0/30	1/30	0/30
Injured	0/30	0/30	0/30
Injured and Inoculated	22/30	30/30	30/30

[a]The fraction represents (number of infected sugarbeets)/(number of sugarbeets inoculated with the sugarbeet *Erwinia* sp.).

The SBE strains formed a distinct group which were distinguishable from *E. carotovora* var. *atroseptica*, *E. carotovora* var. *carotovora*, *E. chrysanthemi*, *Erwinia cypripidii*, and *Erwinia rhapontici*, on the basis of utilization of citrate, galacturonate, α-methyl glucoside, lactose, malonate, melezitose, melibiose, m-tartrate, and xylose. Differences also are apparent in growth at 36 C and the formation of reducing compounds from sucrose (Table 1).

Symptoms.—Diseased beets are not easily recognized in the field until they are severely rotted. Examination reveals black longitudinal lesions on petioles with occasional exudation of a foamy ooze in the crown area (Fig. 1). The disease is characterized by necrotic or discolored vascular bundles in the root (Fig. 2). Areas surrounding these diseased vascular bundles usually turn pink to reddish after exposure to air. Rots which vary from a soft wet rot to a dry rot usually follow the vascular invasion (Fig. 3). Infections usually begin in the crown and may progress toward the root tip until the entire beet is infected. Severely infected roots may become "hollowed" but plants remain alive. Diseased and rotted beets are frequently still intact at harvest and such beets adversely affect sugar refining and processing if present in high numbers (E. D. Whitney, *unpublished*).

Studies of sugarbeet infection in a greenhouse.—Injury apparently is necessary for the infection of 1-mo-old sugarbeets by the SBE (Table 2). The primary infection always was noted at the site of wounding. Most of the inoculated sugarbeets incubated at 24 and 30 C were dead within 3 to 21 days after inoculation and symptom development was similar to that described for naturally infected sugarbeets. The SBE was reisolated from diseased beets in all cases.

Symptoms usually were evident within 2-3 days on beets incubated at 30 C but were not apparent until 5-10 days on beets incubated at 24 C. Beets incubated at 18 C did not show symptoms until 10-14 days and usually did not die. Transfer of infected beets from 19 to 30 C induced severe symptoms and death, usually within 1-2 days. All control plants, whether injured or not, remained healthy throughout the study.

Pathogenicity to other plants.—One-mo-old sugarbeet seedlings usually were killed within 1-3 wk by isolates of the SBE. Vascular necrosis and rot as described previously occurred in the petioles, crowns, and roots of 97% of the inoculated plants. However, only 23, 2, and 6% of the *E. carotovora* var. *atroseptica*, *E. carotovora* var. *carotovora*, and *E. chrysanthemi* isolates, respectively,

TABLE 3. Infection of sugarbeet, tomato, potato, and chrysanthemum plants with *Erwinia* spp.

	Number and percentage of *Erwinia* spp. producing disease			
Plant	Sugarbeet *Erwinia*	*E. carotovora* var. *atroseptica*	*E. carotovora* var. *carotovora*	*E. chrysanthemi*
Chrysanthemum	24/31 (77)[a]	2/31 (6)	24/57 (42)	19/34 (56)
Potato[b]				
18 C	23/31 (74)	24/31 (77)	14/57 (25)	4/34 (12)
24 C	31/31 (100)	25/31 (81)	44/57 (77)	17/34 (50)
Sugarbeet	30/31 (97)	7/31 (23)	1/57 (2)	2/34 (6)
Tomato	30/31 (97)	12/31 (39)	19/57 (33)	6/34 (18)

[a]Ratio and percentage of isolates producing disease.
[b]Potato plants were grown at 18 and 24 C; all others were grown at 24 C.

induced symptoms on sugarbeet similar to the SBE (Table 3).

Tomato plants usually were killed within 1 wk by 97% of the SBE isolates. Stem tissues surrounding the point of inoculation became necrotic and black and a profuse exudate from the lesions ran down the stem and produced necrosis of tissues that it contacted (Fig. 4). The vascular tissue was necrotic 10-15 cm from the inoculation site. Symptoms also were induced on tomato stems by 39, 33, and 18% of the *E. carotovora* var. *atroseptica*, *E. carotovora* var. *carotovora* and *E. chrysanthemi* isolates, respectively (Table 3). However, the symptoms consisted of a localized necrosis adjacent to the site of inoculation and the black exudate was not produced.

The blackening, wilting, and soft-rot of potato stems, typical of blackleg, was produced at 18 C by 74% and 77% of the SBE isolates and the *E. carotovora* var. *atroseptica* isolates, respectively. However, only 12 and 25% of the *E. chrysanthemi* and *E. carotovora* var. *carotovora* isolates, respectively, produced blackleg symptoms at 18 C. Most of the isolates produced blackleg symptoms at 24 C in potato plants (Table 3).

Isolation of the pathogen from seed.—The SBE was not isolated from sugarbeet seed regardless of method attempted. Other bacteria were present in seed but these were not pathogenic on sugarbeet seedlings.

Recovery efficiency of the selective medium and isolation from soil.—The average recovery efficiency of the six SBE isolates on MSM was 91% of that on LU and 92% of that on NA.

The SBE always was recovered from artificially infected soils that contained at least 10^3 cells/g of soil. The efficiency of recovery was 48% from soils infested with 10^6 cells/g of soil and only 5% from soils infested with 10^3 cells/g of soil.

The population of SBE in soil from field B (30% incidence) averaged 10^6 CFU/g 3 wk before harvest and was slightly higher 1 day after harvest (Table 4). The population began to decline after harvest and was not detected in soils 2 mo after harvest. The population of SBE was much less in soils from field A (5% disease incidence) when compared to soils from field B (30% incidence). Two days after harvest the SBE population was 7×10^5 CFU/g of soil. One mo later, SBE could not be detected (Table 4). The SBE were isolated only infrequently from soil sampled at six other sugarbeet fields which had <5% disease.

Field study.—The amount, type and extent of rotting varied in inoculated beets but vascular necrosis was the universal symptom. Injury increased the incidence and severity of disease in inoculated sugarbeets, especially early in the season. Sugarbeets inoculated at 8 wk were more likely to be killed than those inoculated at 12 or 15 wk. Root yield was reduced from 76.8 metric tonnes/ha in the control plots to 37.9 metric tonnes/ha in plots in which plants were injured and inoculated at 8 wk (Table

TABLE 4. Populations of sugarbeet *Erwinia* sp. in soil from sugarbeet fields with a 5% and 30% incidence of bacterial vascular necrosis

Sample date (1973)	Colony forming units/gram of soil	
	Field A - 5% Diseased	Field B - 30% Diseased
16 September	2.5×10^{4}[a]	1×10^{6}
7 October[b]	7×10^{5}	2.8×10^{6}
6 November	0	2.1×10^{2}
6 December	0	0

[a]Data presented as number of colony-forming units of *Erwinia* sp. per g of oven dry soil (100 C, 24 hr).
[b]Sugarbeets were harvested on 5 October in Field A and 6 October in Field B.

TABLE 5. Incidence of vascular necrosis and rot, and effect on subsequent root and sugar yield when injured and noninjured beets were inoculated at three different stages of growth with the sugarbeet *Erwinia* isolate UCBPP193

Inoculation	Age (wk)	Injury	Roots diseased (%)	Rot/root (%)	Yield (metric tonnes/hectare)	
					Roots	Sucrose[a]
No	...	No	36	14	76.8	9.76
No	8	Yes	48	23	65.0	8.50
No	12	Yes	38	15	76.8	9.93
No	15	Yes	30	13	70.0	9.31
Yes	8	No	46	26	68.5	9.11
Yes	8	Yes	81	54	37.9	4.57
Yes	12	No	41	19	71.8	9.01
Yes	12	Yes	67	32	63.0	7.73
Yes	15	No	42	22	68.7	8.68
Yes	15	Yes	57	22	70.2	9.23
LSD, $P = 0.05$, between treatments:						
not inoculated			12	10	8.3	1.40
inoculated			9	7	7.0	1.05
not inoculated vs. inoculated			11	9	5.8	1.28

[a]Sugar yield was determined by submitting a random sample of 10 roots per replication to a sugar refinery for standard sugar and tare analysis.

5). The percent rot per root was approximately 4-fold higher in beets injured and inoculated at 8 wk compared to control plants. Injury alone increased disease and resulted in significant reductions in root yield (Table 4).

DISCUSSION

Specific strains of *Erwinia* belonging to the *Erwinia carotovora* group caused a newly observed disease of sugarbeet in California. The results of several diagnostic tests, including utilization of α-methyl-glucoside and formation of reducing compounds from sucrose suggested a relationship between the SBE isolates and *E. carotovora* var. *atroseptica* (3, 4, 7). Our studies, however, indicated some substantial differences between the SBE isolates and *E. carotovora* var. *atroseptica* and *E. carotovora* var. *carotovora*. The intensity of the sucrose reduction test was markedly and consistently greater with SBE than with *E. carotovora* var. *atroseptica*. Also, a chromatographic analysis (Barbara Lund, *personal communication*) of the accumulated reducing compounds revealed that the SBE isolate, UCBPP193, produced fructose and glucose whereas 14 isolates of *E. carotovora* var. *atroseptica* accumulated palatinose and 1-*O*-α-glucosylfructose. The SBE isolates grew well at 37 C and most grew at 40 C which is indicative of a relationship to *E. carotovora* var. *carotovora* (7). On the basis of these tests, complemented by a comprehensive testing of the utilization of many carbon sources, a new name will be proposed elsewhere (Thomson, Schroth and Hildebrand, *unpublished*).

The principal diagnostic symptom of the disease is the necrotic vascular tissue in the root. The rots which follow are variable ranging from a soft wet rot to a dry rot. Much of this variability probably is caused by a wide range of secondary microorganisms that may invade the weakened tissues. In California, the disease was present in all sugarbeet fields investigated although the incidence usually was less than 5%. The etiological agent causing this disease probably has been overlooked in California because of the variability of the secondary rots which occur and because of the relatively low incidence of the disease.

The pathogenicity of SBE to tomatoes, potatoes, and chrysanthemums may be important when considering crop rotations and compatible plantings. However, the greenhouse pathogenicity tests are artificial in that succulent plants were inoculated with large dosages of inoculum. Thus, SBE may cause typical blackleg symptoms in greenhouse-grown potatoes at 18 C, but may not necessarily cause the disease in the field. Also, the fact that some isolates of *E. carotovora* var. *atroseptica*, *E. carotovora* var. *carotovora*, and *E. chrysanthemi* infect sugarbeet in the greenhouse is not surprising. We view this as an indication of an organism's potential to cause disease under artificial conditions and are reluctant to extrapolate how these data relate to an organism's capacity to cause disease in the field.

The sugarbeets inoculated in the field when 8 wk old were more severely infected than plants inoculated at 12 and 15 wk. This might be evidence of increased resistance as beets age but more likely is a result of the period of growth of the pathogen in the plant. For example, the plants inoculated at 8 wk were infected for 15 wk, whereas those inoculated at 12 and 15 wk were infected for 11 and 8 wk, respectively.

High temperatures appear to favor development of the disease. The first serious outbreaks of vascular necrosis and rot of sugarbeet occurred in the hot central valley of California. Our studies demonstrated that the pathogen grew at 37 C and that symptoms were produced much sooner at 30 C than at 24 C in the greenhouse. Infection may occur early in the season when cooler temperatures prevail and serious rots may not develop until later in the season when temperatures increase.

The evidence indicates that the bacterium is endemic throughout California soils. The disease frequently was most severe where sugarbeets were planted into areas cultivated for the first time and the pattern of disease could not be attributed to contamination of the seed lot. The SBE was not detected in any of the seed assays, and the disease has not been observed in the beet-seed producing areas of Oregon where the majority of the seed used in California is grown. Although it would not be surprising to detect the bacterium in a seed lot occasionally because of contamination with infested soil or beet refuse, this may be of little significance. The SBE may survive in the rhizosphere of plants other than sugarbeet. Several workers have shown that soft-rot *Erwinia* spp. are capable of surviving or are preferentially stimulated in the rhizospheres of nonhost plants (6, 8, 10).

There is an excellent likelihood that the bacterial rot of sugarbeet will be controlled in the near future by the development of resistant cultivars. During the development of the virus yellows-resistant cultivars, US H9 and US H10, a gene(s) for resistance apparently was deleted. However, a breeding program currently under way has shown that resistance to the bacteria is present in older varieties and that this resistance has been incorporated into the virus-resistant cultivars (E. D. Whitney, *unpublished*).

LITERATURE CITED

1. AYERS, S. H., P. RUPP, and W. T. JOHNSON, JR. 1919. A study of the alkali-forming bacteria found in milk. U.S. Dep. Agric. Bull. 782. 39 p.
2. BROWN, N. A., and C. O. JAMIESON. 1913. A bacterium causing a disease of sugar-beet and nasturtium leaves. J. Agric. Res. 1:189-210.
3. DYE, D. W. 1968. A taxonomic study of the genus Erwinia. I. The "amylovora" group. N. Z. J. Sci. 11:590-607.
4. DYE, D. W. 1969. A taxonomic study of the genus Erwinia. II. The "carotovora" group. N. Z. J. Sci. 12:81-97.
5. ELLIOTT, C. 1951. Manual of Bacterial Plant Pathogens. 2nd ed. Chronica Botanica, Waltham, Massachusetts. 186 p.
6. GOTO, M. 1972. The significance of the vegetation for the survival of plant pathogenic bacteria. Pages 39-53 *in* H. P. Maas-Geesteranus, ed. Proc. Third Int. Conf. Plant Pathogenic Bacteria, Centre Agric. Publ. Doc. (PUDOC), Wageningen, The Netherlands. 365 p.
7. GRAHAM, D. C. 1972. Identification of soft rot coliform bacteria. Pages 273-279 *in* H. P. Maas-Geesteranus, ed. Proc. Third Int. Conf. Plant Pathogenic Bacteria. Centre Agric. Publ. Doc. (PUDOC), Wageningen, The Netherlands. 365 p.
8. KIKUMOTO, T., and M. SAKAMOTO. 1969. Ecological studies on the soft-rot bacteria of vegetables. VII. The preferential stimulation of the soft-rot bacteria in the rhizosphere of crop plants and weeds. Ann. Phytopathol.

Soc. Japn. 35:36-40.
9. KLEMENT, Z., and R. N. GOODMAN. 1966. Hypersensitive reaction induced in apple shoots by an avirulent form of Erwinia amylovora. Acta Phytopathol. Acad. Sci. Hung. 1:177-184.
10. KLINGER, A. E., D. C. HILDEBRAND, and S. WILHELM. 1971. Occurrence of Erwinia carotovora in the rhizosphere of cotton plants which escape Verticillium wilt. Plant Soil 34:215-218.
11. LELLIOTT, R. A. 1974. Description of the species of genus Erwinia. Pages 332-340 *in* R. E. Buchanan and N. E. Gibbons, eds. Bergey's manual of determinative bacteriology. 8th ed. Williams and Wilkins, Baltimore, Maryland. 1,246 p.
12. LURIA, S. E., J. N. ADAMS, and R. C. TING. 1960. Transduction of lactose-utilizing ability among strains of E. coli and S. dysenteriae and the properties of the transducing phage particles. Virology 21:348-390.
13. MAGROU, J. 1937. Pages 195-219 *in* Hauduroy et al., eds. Dictionnaire des bacteries pathogenes. Masson and Co., Paris. 597 p.
14. MAZZUCCHI, U. 1975. Vascular blackening in sugarbeet taproots caused by Pseudomonas syringae van Hall. Phytopathol. Z. 84:289-299.
15. RUPPEL, E. G., M. D. HARRISON, and A. K. NIELSON. 1975. Occurrence and cause of bacterial vascular necrosis and soft rot of sugarbeet in Washington. Plant Dis. Rep. 59:837-840.
16. TAKIMOTO, S. 1931. A soft rot of sugarbeet and its causal organisms. Ann. Phytopathol. Soc. Japn. 2:350-356.
17. THOMSON, S. V., F. J. HILLS, and M. N. SCHROTH. 1975. Cultural procedures to reduce bacterial vascular necrosis and rot of sugarbeet. Proc. Am. Phytopathol. Soc. 2:119 (Abstr.)
18. THOMSON, S. V., and M. N. SCHROTH. 1972. Vascular necrosis and rot of sugarbeets. Calif. Plant Pathol. 12:1-2.
19. THOMSON, S. V., M. N. SCHROTH, W. J. MOLLER, and W. O. REIL. 1975. Occurrence of fire blight of pears in relation to weather and epiphytic populations of Erwinia amylovora. Phytopathology 65:353-358.
20. THOMSON, S. V., M. N. SCHROTH, E. D. WHITNEY, and F. J. HILLS. 1973. Bacterial rot–a new threat to sugar beet. Calif. Sugarbeet 1973:30-32.
21. TOMINAGA, T. 1967. Bacterial blight of sugarbeet caused by Pseudomonas aptata (Brown et Jamieson) Stevens. Ann. Phytopathol. Soc. Japn. 33:294-300.