Stewart's Bacterial Wilt of Corn

EVAN H. PEPPER

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EVAN H. PEPPER

Department of Plant Pathology
North Dakota State University
Fargo, North Dakota

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Contents

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Introduction

Stewart's bacterial wilt, caused by *Erwinia stewartii* (E. F. Sm.) Dye, can be a devastating disease of both sweet and field corn. Once it was understood, resistant varieties introduced, and effective control measures devised, the disease was reduced to lesser importance. An indication of research interest is given by the relative number of publications from the first report of the disease in 1897 through 1964 (Fig. 1). That this interest was inspired by the economic importance of the disease is evident by a comparison of disease index and the number of publications in the same period (Fig. 1).

The purpose of this monograph is to bring together the important information on this disease, and to critically evaluate these reports. The subject illustrates several important aspects of plant diseases: genetics of the causal organism; insect-vector relationships; successful breeding for disease resistance in relation to the development of hybrid corn; disease forecasting. Because the literature on this disease is voluminous, only papers that present original information can be included.

While no recent review article on Stewart's bacterial wilt of corn is available, there are several general references (75, 92, 94, 107, 116, 129, 171, 205, 270, 273, 289, 302, 305, 340, 371, 374, 375). The "Maize Bibliographies" are useful compendia of references on the disease (89-91).

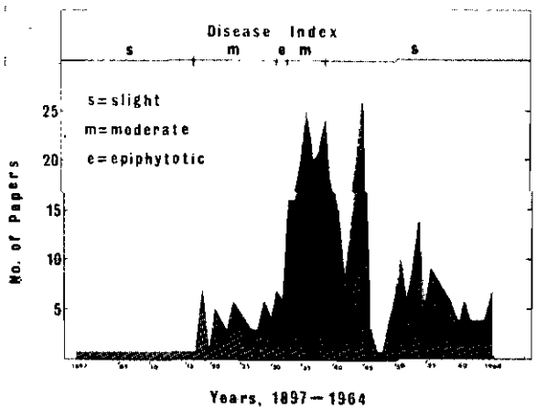


Figure 1. Relationship of incidence of Stewart's bacterial wilt to research publications on the disease, 1897-1964. Disease severity shown as s (slight), m (moderate), and e (epidemic).

Common Names

The disease was called "bacterial wilt of sweet corn" by its discoverer, F. C. Stewart, in 1897 (347). E. F. Smith variously termed the disease, "the sweet corn disease of Long Island" (301), and "Stewart's disease of sweet corn" (302). Other modifications of these common names include "Stewart's wilt," "Stewart's leaf blight," and "bacterial leaf blight." In Europe the disease is frequently referred to as "maize bacteriosis" (250-254). To avoid confusion with other bacterial diseases of corn, and because of the long-standing use of Stewart's name, it seems desirable to designate the disease by the name, Stewart's bacterial wilt of corn.

Host Range

All agronomic types of corn, *Zea mays* L., are attacked by the pathogen, with sub-species, inbreds, hybrids, and varieties varying in susceptibility. Two other hosts, native to the Western Hemisphere, have been reported for the disease. Lesions were found on teosinte (*Euchlaena mexicana* Schrad.), and bacteria isolated from these lesions produced typical wilt symptoms upon subsequent inoculation of sweet corn (263). Natural lesions were discovered in 1939 on eastern gama grass (*Tripsacum dactyloides* L.) in Arlington, Virginia, along with flea-beetle feeding scars (119, 261). The lesions were small and inconspicuous and, while inoculation with bacteria isolated from them produced infection in sweet corn, inoculation to *T. dactyloides* and three other species of *Tripsacum* did not lead to infection.

Artificial inoculation, on the other hand, has been accomplished with a number of diverse host plants. Ivanoff inoculated sorghum (*Sorghum vulgare* Pers.), Sudan grass [*S. vulgare* var. *sudanense* (Piper) Hitchc.], sugarcane (*Saccharum officinarum* L.), yellow foxtail [*Setaria lutescens* (Weigel) F. T. Hubb.], German foxtail millet [*Setaria italica* (L.) Beauv.], and common millet (*Panicum miliaceum* L.) with the bacterium (173, 174). Leaf symptoms, which resembled in type and shape those typically produced on corn, were obtained on all plants except sugarcane. Job's-tears (*Coix lacryma-jobi* L.), teosinte, and corn were infected, both with needle and insect inoculations, while negative results were obtained following inoculation of other grass species (264). Wellhausen noted a slight dwarfing and discoloration of the stems of Golden Cluster beans (*Phaseolus vulgaris* L.) after inoculation with *E. stewartii*, and brown, water-soaked, irregular stria-

tions on the veins of Early Pearl oats (*Avena sativa* L.) and Proso millet (*Panicum miliaceum* L.), as on corn (392). No infection resulted from similar inoculations to cahbage (*Brassica oleracea* var. *capitata* L.) and tomato (*Lycopersicon esculentum* Mill.). Wilt symptoms were obtained hy Poos after inoculating sweet corn with juice obtained from a number of infected grass species upon which insects had been feeding (261). The plants from which wilt bacteria were obtained were: sweet corn, field corn, *Digitaria* sp., *Panicum dichotomiflorum* Michx., *P. capillare* L., *Coix lacryma-jobi* L., *Poa pratensis* L., *Dactylis glomerata* L., *Agrostis alba* L., *Sorghum vulgare* var. *sudanense* (Piper) Hitchc., *Triticum* sp., and *Setaria lutescens* (Weigel) F. T. Hubb. In another inoculation experiment, Elliott and Poos were ahle to infect *Euchlaena perennis* Hitchc., *E. mexicana* Schrad., and *Coix lacryma-jobi* L. with *E. stewartii*, using flea beetles. *Tripsacum dactyloides* (L.) L., *T. pilosum* Scribn. & Merr., *T. lanceolatum* Rupr., and *T. latifolium* Hitchc. were immune (118).

These data indicate that a relatively large number of plant species may he infected by the organism, at least under artificial conditions. Non-corn hosts may act, therefore, either as symptomless carriers, or, if symptoms are produced, they may be so inconspicuous as to be overlooked. Such symptomless carriers may, therefore, he important in the spread of the disease when insect vectors are present (261).

History of the Disease

Some early reports of injury to field corn in Illinois described symptoms that suggested the presence of bacterial wilt. Burrill (66) described a new bacterial disease of corn in 1889 which showed some of the symptoms of bacterial wilt (107). This disease was probably not bacterial wilt, although it may have been present in Illinois at that time. The corn flea beetle, the common wilt vector, was then commonly reported from Illinois (107).

Stewart reported a bacterial wilt disease from New York in 1897 that had caused considerable damage to sweet corn grown on Long Island (347). The disease was first observed by Stewart in 1895, causing losses on sweet corn of 20 to 100%. He gave an accurate account of the symptoms and isolated the bacterium. Inoculations with the isolated bacterium produced the disease in sweet corn, but dent corn, popcorn, and teosinte resisted infection. A description of the cultural characteristics of the bacterium was given in his report, although he did not name it.

A culture of the bacterium was sent to Erwin F. Smith, who described and named it *Pseudomonas stewarti* in honor of F. C. Stewart in 1898 (299). Smith described its cultural characteristics (300), established its infectious nature by inoculation with pure cultures (301), and supported Stewart's suggestion that the organism was seed-borne (303). He showed that infection was through stomata and hydathodes of seedlings, and that wounding was not required. He also recommended a mercuric chloride seed treatment (301, 302).

During the first 20 years of this century, investigations were largely concerned with cultural studies,

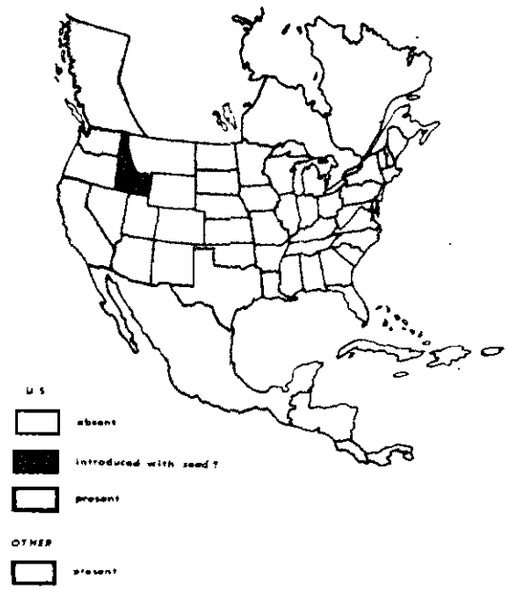


Figure 2. Geographical distribution of Stewart's bacterial wilt of corn in the Western Hemisphere.

distribution and taxonomy of the bacterium, and losses produced by the disease. Later studies by Rand, Cash, Reddy, Ivanoff, Poos, and Elliott were concerned with dissemination of the bacterium by soil, insects, and seed, and with fluctuations in disease incidence. These studies provided an explanation for the observed correlation between winter temperatures and disease severity (323). A series of experimental disease forecasts was subsequently initiated for wilt, based on a winter temperature index (242, 345).

The epidemics of 1932 and 1933 stimulated intensive studies of the disease (Fig. 1) (106). The development of resistant corn hybrids, along with increased knowledge of the pathogen, decreased the importance of the disease. Research interest waned, however, as the losses from the disease were reduced. Most of the research on *E. stewartii* in recent years has been taxonomic or physiologic.

Distribution of the Disease

Stewart's bacterial wilt is distributed generally throughout the corn-growing areas of the United States (Fig. 2), and in restricted regions elsewhere in the world (83). Summarized listings of the distribution of the disease have appeared periodically since its discovery (8, 107, 186, 271, 273, 289, 302, 324, 340).

The following list gives references to information on: (a) occurrence, (b) losses, (c) varietal relations, (d) insect vectors, and (e) influence of the weather, for the United States and foreign countries.

Alabama: (a) 9

Arkansas: (a) 9, 128, 343; (b) 53, 204, 343; (c) 128; (d) 53

California: (a) 8, 20, 24, 404; (b) 102
Connecticut: (a) 76, 82, 260, 405; (b) 279, 343, 404, 405; (c) 279, 405; (d) 405
Delaware: (a) 8, 53; (b) 8
District of Columbia: (a) 302
Florida: (a) 99
Georgia: (a) 8, 9
Idaho: (a) 123
Illinois: (a) 100, 144, 147, 149, 186, 274, 312, 314, 349, 355, 404; (b) 23, 36, 53, 143-145, 147, 196-198, 239, 245, 307, 342, 343, 366, 411; (c) 145, 147, 197, 243, 366; (d) 36
Indiana: (a) 24, 127, 143-145, 147, 186, 239, 260, 309, 314; (b) 22, 53, 100, 102, 136, 143, 144, 163, 243, 307, 341-343, 365, 367, 372, 403, 405; (c) 24, 144, 145, 163, 243, 308, 309, 369
Iowa: (a) 18, 186, 265, 347; (b) 55, 102, 243, 266, 295, 378-380, 403, 404; (c) 266, 378
Kansas: (a) 102, 143, 147, 186, 239, 253; (b) 100, 404
Kentucky: (a) 102, 130; (b) 8, 53, 127, 165, 166, 243, 376, 404; (c) 376, 377
Maine: (a) 223, 224, 324
Maryland: (a) 100, 127, 149, 260, 302; (b) 8, 53, 102, 143-145, 147, 185, 194, 239, 243, 274, 314, 351, 383, 384, 403, 405, 406; (c) 127, 147, 162, 163, 185, 194, 351, 384, 385, 404, 406
Massachusetts: (a) 8, 9, 24, 46-48, 50-52, 102, 132, 149, 260, 341; (b) 48, 100, 145, 343, 404; (d) 132; (e) 49, 50
Michigan: (a) 8, 9, 24, 100, 102, 149, 163, 249, 302, 403; (b) 8, 53, 149, 163, 343, 367, 404
Mississippi: (a) 9, 147, 239, 406; (b) 351; (c) 406
Missouri: (a) 24, 56-58, 143, 147, 271, 294, 366; (b) 294, 362; (c) 143, 294, 362; (d) 24
Nebraska: (a) 42
New Hampshire: (a) 324
New Jersey: (a) 8, 9, 11, 100, 138, 145, 260, 274, 342; (b) 8, 15, 149, 274, 342, 405; (c) 15, 194, 405; (d) 194, 405; (e) 194
New Mexico: (a) 8; (b) 8
New York: (a) 8, 26, 67-69, 71-74, 79, 81, 102, 127, 131, 132, 143, 144, 147, 149, 194, 215, 239, 244, 260, 268, 302, 341, 347, 405; (b) 8, 79, 282-286, 341-343, 404, 405; (c) 68, 73, 79, 127, 132, 144, 147, 284, 285, 405; (d) 72, 79, 132, 286, 405; (e) 194, 244
North Carolina: (a) 314, 406; (c) 406
North Dakota: (a) 147, 186, 239, 394
Ohio: (a) 8, 9, 24, 127, 140, 143-145, 147, 149, 239, 260, 302, 314, 350, 354, 362, 363, 405; (b) 4, 102, 141, 143, 145, 243, 343, 350, 354, 358, 368, 403, 404; (c) 10, 127, 143, 144, 147, 243, 362
Oklahoma: (a) 8, 142, 363; (b) 78; (c) 78
Pennsylvania: (a) 8, 24, 80, 192, 194, 260; (b) 8, 80, 81, 102, 191, 342, 343, 403, 411
Rhode Island: (a) 12; (b) 12
South Carolina: (a) 271
South Dakota: (a) 9, 314
Tennessee: (a) 8, 9, 142, 165, 167, 274, 314, 406; (b) 8, 53, 165, 167, 274; (c) 142, 164, 406
Texas: (a) 5, 24, 52, 100, 128, 403; (b) 100, 102; (c) 52, 102; (e) 52
Vermont: (a) 9
Virginia: (a) 8, 9, 11, 80, 100, 121, 122, 143-146, 287, 302, 314, 403, 406; (b) 8, 52, 53, 80, 102, 142, 147, 185; (c) 9, 80, 110, 142, 147, 185, 399, 406; (e) 287
Washington: (a) 9
West Virginia: (a) 19, 53, 100, 144, 145, 194, 239, 251, 302, 320, 404; (b) 24, 102, 149, 194, 338, 342; (c) 24, 145, 149, 320, 338; (e) 24
Wisconsin: (a) 9, 159; (b) 102, 403
Canada: (a) 84, 85, 88, 125, 293, 342, 404
China: (a) 298, 385
Costa Rica: (a) 393
Italy: (a) 29-31; 254
Mexico: (a) 111
Poland: (a) 114
Puerto Rico: (a) 369
USSR: (a) 14, 43, 115, 118
Switzerland: (a) 410
Yugoslavia: (a) 396

The disease has not been reported in Alaska (218), Colorado (3), Hawaii (168), Montana (2), Nevada (220), Oregon (25, 407), Utah (70), and Wyoming (59). The pathogen has never become established in Idaho, and the disease has only been observed there on a few plants grown from seed introduced from other areas. Failure of the bacterium to persist in the state is due to dry climatic conditions and the absence of the corn flea beetle (123).

Since the principal natural hosts of the pathogen are native to the Western Hemisphere, it would be expected that *E. stewartii* also originated there. The most severe losses from this pathogen to date have occurred in the areas of most intensive cultivation of the crop (i.e., the Western Hemisphere).

Economic Importance

Reports concerning corn losses during the years prior to World War I are either lacking or are restricted to localized areas. Stewart in 1897, for example, stated that losses in Long Island market gardens frequently reached 20 to 40%, hut that in most cases these losses went unnoticed by growers. No extensive heavy losses from Stewart's bacterial wilt were reported until 1917, when severity of the disease was noted in several locations (7). Reports from West Virginia, Ohio, and Missouri in 1926 indicated that, while losses in sweet corn ranged from slight to severe, the disease was increasing on both field and popcorn (193). Three of seven reporting states (Missouri, Kansas, and Iowa) indicated that the disease had caused more damage in 1928 than in preceding years (148).

Losses continued to increase through 1929, 1930, and 1931, culminating in the widespread epidemic of 1932 (322). The epidemics of 1930-1932 were the most spectacular since Stewart's description 35 years earlier (400). The disease occurred in epidemic proportions in 1932 in Ontario (84), Illinois (202), Indiana (163), West Virginia (342), Michigan (163), Connecticut (321), and Iowa (267). Even

though an epidemic was raging in several states, not every corn-growing area was severely affected. Maryland reported only one case of the disease in 1932, although no observations were made of the market-garden crop (163). It was no more severe in 1932 than in the preceding 2 years in New Jersey and Pennsylvania (342). Stevens found no wilt in Maine or New Hampshire (321). Somers reported that heavy damage occurred in both 1931 and 1932 in Illinois (311); every inspected field showed bacterial leaf blight, and the bacteria were sometimes so profuse that they exuded from the stomata of the inner husks and covered the kernels.

The wilt epidemic continued into the 1933 growing season, but while some states reported increases in severity, losses were, in the aggregate, considerably reduced. States reporting severe incidence in 1933 included Massachusetts, Connecticut, New York, Pennsylvania, Ohio, Michigan, Indiana, Illinois, Arkansas, and Maine (first report, 223). Stevens pointed out that bacterial wilt was increasing on field corn in several states, and that the decrease in disease losses in certain areas resulted from the use of resistant corn strains and hybrids (326).

Most observers agree that the disease was much less severe in 1934 (324, 401). It is not known whether the reduction in severity was due to a less favorable environment or to the rapid replacement of susceptible sweet corn by the resistant hybrid, Golden Cross Bantam, developed in 1933 by G. M. Smith (308). Increased use of resistant hybrids and varieties in 1935 made assessment of the effects of the environment difficult. Losses due to wilt were significant in only 3 of the 9 reporting states (281, 328). Yield reduction for Indiana and Michigan in 1934 and 1935 was estimated by Stevens (330). Reductions in sweet-corn yields in Michigan declined from 3% in 1934 to a trace in 1935, while in Indiana they were estimated at 5% for both years. Only Virginia reported severe disease in 1936, the other nine states reported little or no wilt (101, 104, 344). The estimated reduction in sweet-corn yield caused by this disease, for the years 1931-1936, is shown in Fig. 3.

Wilt infection was generally of little importance in 1937 except in an area extending from southern Connecticut to southern Maryland (103, 402, 405). Pasinetti observed that the disease was increasing in severity in Italy, the 1936 losses amounting to 40-90% of the corn crop (255). The disease remained quiescent in the United States in 1938, with only occasional reports of increasing wilt severity (112, 196, 197). More damage was caused to early sweet corn in 1939 in Illinois, but field corn was less severely affected than in 1938 (198). It was moderately severe in the same year on field corn in Oklahoma (78).

While the disease was, and is, primarily a disease of sweet corn, it became increasingly evident following the epidemics of 1932-1933 and 1937-1938 that the late-season leaf-blight phase was increasing on dent corn (114). The occurrence of the leaf-blight phase had been noted in Ohio since 1939, but, except

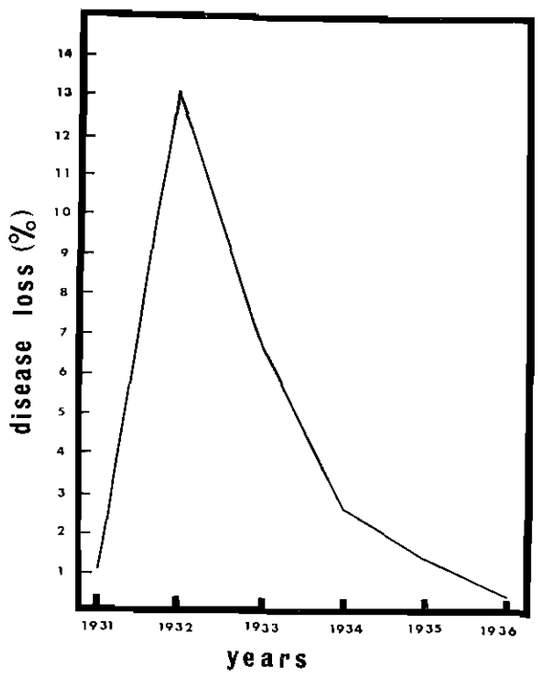


Figure 3. Disease losses in sweet corn due to wilt, 1931-1936 (124, 127, 364).

for a few isolated outbreaks, no severe disease losses occurred (105).

Losses from this disease have been inconsequential during the past 20 years except for a few, small, sporadic outbreaks. The greatest reduction in yield since the 1938 epidemics occurred in Illinois in 1953 (245, 339). New Jersey (15) and Ontario (85) also reported severe outbreaks of the disease in the same year. The disease was destructive on both sweet corn and dent corn in Kentucky in 1958 (189). No serious losses have been reported since that time.

Disease Symptoms

General discussions of symptoms of the disease have been presented (92, 108, 289, 373-375). Symptoms may vary with geographical location [e.g., Italy, United States (30, 184)].

The symptoms of the disease may appear on sweet corn at any stage of development. Some are killed in the seedling stage, while others may not succumb until tasseling or even later (Fig. 4). If the plant is infected at an early stage the initial symptoms usually are wilting, stunting, and linear watersoaked lesions with wavy or irregular margins (Figs. 5, 6). The lesions parallel the leaf veins and range from one to more than 10 mm in width, and may extend the length of the leaf (Fig. 6). As the disease progresses, the leaves yellow and present a necrotic "fired" appearance (Fig. 7), progressing upward from the bottom of the plant. The disease symptoms at this stage may be confused with drought damage, nutritional deficiency, or insect injury (Figs. 8, 9). Leaves may wilt, either before or after yellowing, depending

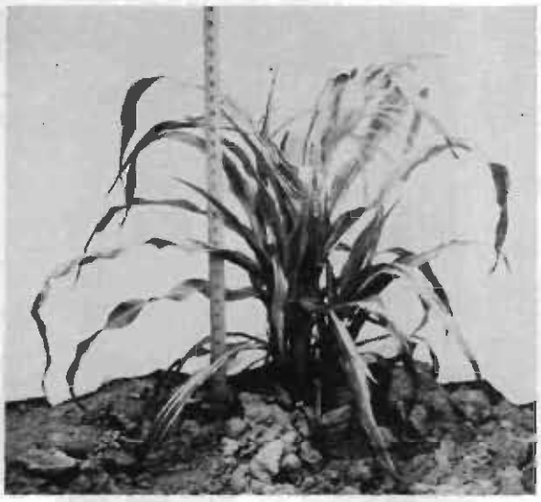


Figure 4. A susceptible sweet-corn plant heavily infected with the bacterium at tasseling stage. Photo courtesy Crops Research Division, Agricultural Research Service, U.S. Department of Agriculture.

Figure 5. A young susceptible sweet-corn plant infected with the wilt organism, showing wilting, stunting, and streaking of the upper leaves. Photo courtesy of Crops Research Division, Agricultural Research Service, U.S. Department of Agriculture.

upon the severity of infection, temperature, and available moisture. Plants infected with wilt may have premature, dwarfed, bleached, and dead tassels (211, 226). Several workers have reported a root and stalk rot associated with infection by *E. stewartii* (211, 259, 364). A stalk rot, characterized by cavities in the pith of the stem, is known to occur in severe cases of wilt (200, 375), as well as in other corn diseases. The identification of the causal organism in such cases may require other diagnostic techniques.

The best diagnostic procedure in the field is as follows: Cut across the lower portion of the stem of a wilted or dying sweet-corn plant, while the stem is still a normal green color. Small droplets of yellowish, bacterial exudate will appear at the cut vascular surface (Fig. 10). Threads of bacterial ooze $\frac{1}{16}$ to $\frac{1}{4}$ inch long may be drawn from the cut surface (226). The exudate may ooze through stomata of the inner husks in cases of severe infection (289). The surface of the enveloped kernels may then be covered with bacterial slime. The exudate occasionally causes leaves above the enclosed tassel to stick together, preventing tassel emergence and causing distortion of the stalk (13, 129, 156, 171, 201, 203, 273, 289). The bacteria have been found in most parts of infected plants, including roots, stems, leaf blades and sheaths, tassels, cobs, husks, and kernels (156, 200, 201, 289, 375). Infected kernels may be deformed, shrunken, and discolored (Fig. 11).

The symptoms of Stewart's bacterial wilt on field corn differ from those described above, especially in the case of the common leaf-infection phase (154, 288, 289, 370). Early infection on very susceptible dent corn produces symptoms similar to those found on infected sweet corn. The plants may succumb or remain stunted and unproductive. Leaf-blight infection of dent corn usually manifests itself at tasseling or later. Lesions commonly originate from flea beetles feeding on leaves. These feeding scars aid in distinguishing the leaf-blight phase from symptoms caused by other foliar diseases. The lesions at first resemble those found on infected sweet corn, then become necrotic. If numerous lesions are present, they may coalesce to involve large areas of the leaf surface (140). Leaf lesions on resistant dent varieties are smaller and less numerous, often forming small oval spots, 1-2 inches long. Bacteria will ooze from the cut ends of vascular bundles in thin, free-hand sections of leaf lesions mounted in water (Fig. 12), but not from the cut end of the stem, as in sweet corn (Fig. 10). Infection progresses upward in early-infected field and sweet corn. Corn plants infected later may show infection only on the upper leaves, while lower foliage remains uninfected. There is no evidence that the bacteria spread from one leaf to another, even on the same plant, except by the beetle vectors. The long lesions are always associated with the feeding wounds of corn flea beetles (375).

A number of leaf diseases are readily distinguishable from Stewart's bacterial wilt: Northern corn leaf blight (*Helminthosporium turcicum* Pass.), Southern corn leaf blight (*H. maydis* Nisik.), Helminthosporium leaf spot (*H. carbonum* Ullstrup) (Fig. 13A, B, D) (375). Three other bacterial pathogens cause leaf diseases of corn (173, 289): *Pseudomonas alboprecipitans* Rosen, causing Bacterial leaf blight (Fig. 13C) (187, 188, 375), *Ps. syringae* v. Hall (Holcus spot) (190, 375), and *Ps. andropogonis* (E. F. Smith) Stapp (Bacterial stripe)

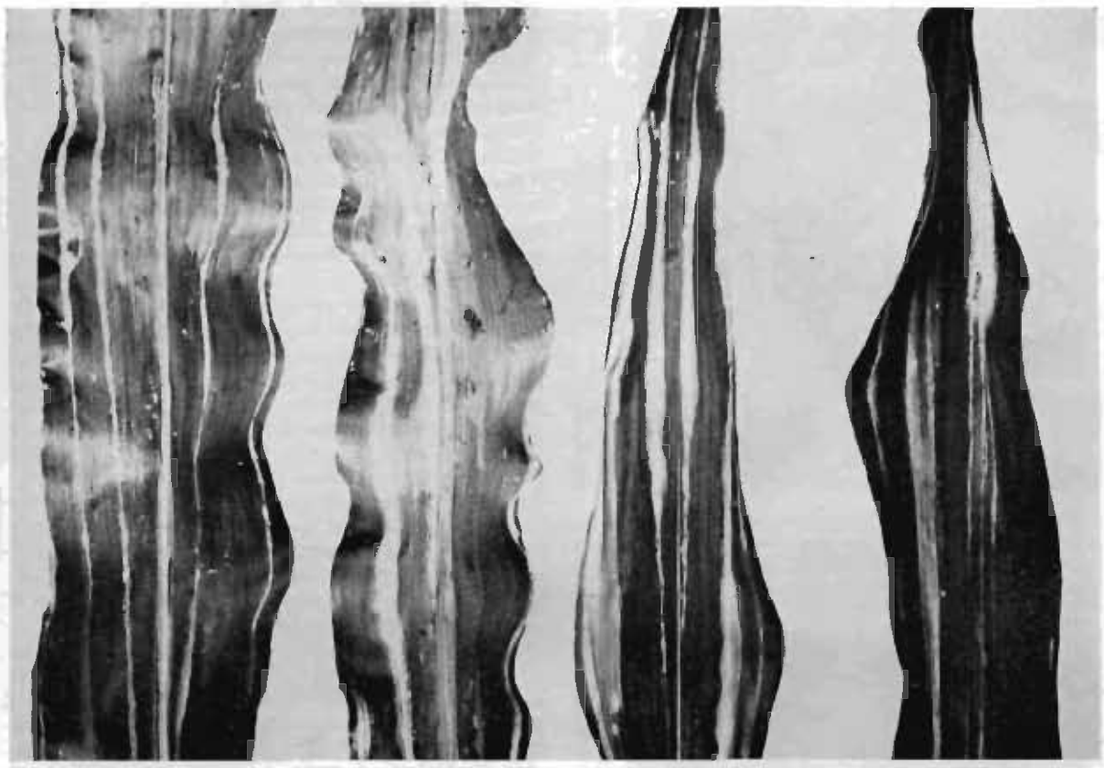


Figure 6. Sweet-corn leaves showing characteristic lesions of the disease. Photo courtesy of Crops Research Division, Agricultural Research Service, U.S. Department of Agriculture.

(375). None of these bacterial diseases is of major importance.

A number of bacteria have been isolated from wilted corn plants which differ physiologically from *E. stewartii* (129, 170, 181). Several *Corynebacterium* spp., resembling *E. stewartii* in culture, were capable of considerable growth in corn hosts (392), but were not shown to maintain themselves on corn under field conditions. A Russian report stated that corn bacteriosis may be caused by organisms other than *E. stewartii* (250).

Nutritional disorders or environmental injuries are mistaken occasionally for Stewart's bacterial wilt. Nitrogen or potash deficiencies may cause yellowing and scorching of the foliage, as well as stunting of corn (92). Chlorosis and necrosis of leaf tissue may also be caused by high temperatures and moisture deficiencies (92). The symptoms of Stewart's bacterial wilt differ in several ways from those produced by nutritional deficiencies or temperature-moisture stress: lesions are discrete and have a characteristic shape, they are always associated with corn-flea-beetle feeding scars, and they contain bacterial cells within the vascular tissue.

The Causal Organism

Erwinia stewartii (E. F. Smith) Dye, 1963
Syn.: *Pseudomonas stewarti* E. F. Smith, 1898
Bacterium stewarti E. F. Smith, 1914
Aplanobacter stewarti (E. F. Smith) McCulloch, 1918
Bacillus stewarti (E. F. Smith) Holland, 1920
Phytomonas stewarti (E. F. Smith) Bergey et al., 1923
Xanthomonas stewarti (sic) (E. F. Smith) Dowson, 1939
Xanthomonas stewartii (E. F. Smith) Dowson, 1939

Taxonomy.—Stewart (347) provided the original cultural description in 1897. The bacterium was originally described as motile with a single polar flagellum (299, 302, 305). Since no flagella or true motility were observed by McCulloch, the pathogen was transferred to the genus *Aplanobacter* (227). Holland placed the organism in the genus *Bacillus* (157), but it was changed to *Phytomonas* in the first edition of Bergey's manual (27).

The genus *Phytomonas* was shown (109) to be invalid because of prior use by protozoologists. Bacteriologists, therefore, used either *Pseudomonas* or *Bacterium* for polarly flagellate plant pathogens, or *Bacterium* or *Aplanobacter* for non-motile pathogens. Rahn pointed out in 1929 the "practical impossibility" of using plant pathogenicity as a basic taxonomic character (269).

Burkholder (63) placed the pathogen in the genus

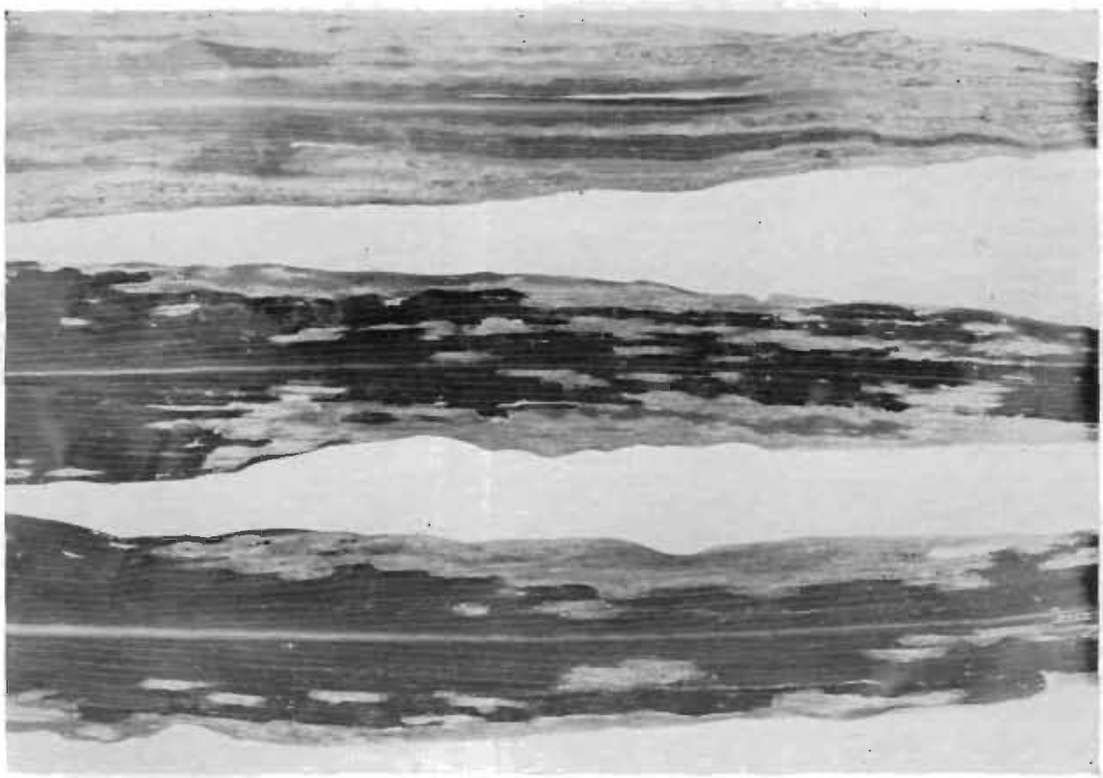


Figure 7. Dent-corn leaves exhibiting lesions resulting from late bacterial infection. The lesions are associated with flea-beetle feeding scars. Photo courtesy Crops Research Division, Agricultural Research Service, U.S. Department of Agriculture.

Bacterium. This was accepted by Elliott (116) but rejected by Dowson (93, 94), who placed the organism in the genus *Xanthomonas*. In the 1957 Bergey's Manual, Burkholder included the pathogen in *Xanthomonas* under *Species incerta sedis* (64).

Various investigators have attempted to clarify the chaotic position of bacterial plant pathogens (60-62, 94, 115, 387). The genus *Bacterium* is, at best, an artificial and heterogeneous group (63). The arguments advanced by Dowson for including the wilt organism in the genus *Xanthomonas* are only slightly more convincing (93, 94); in spite of the lack of motility and the presence of an atypical yellow pigment, he concluded that the pathogen was a *Xanthomonas* on the basis of "biochemical characters." More recently, Dowson's classification has been challenged by several workers on the basis of the unique physiological properties of the wilt bacterium. Dye (see THE CAUSAL ORGANISM, *Physiology*) recently suggested that *X. stewartii* was not closely related to other *Xanthomonas* species, and should be named *Erwinia stewartii* (E. F. Smith) Dye (96-98). Dye's suggestion echoes earlier suggestions (150, 382) that the bacterium appeared closely allied with *Erwinia*, and might be a degenerate member of the Enterobacteriaceae, a family that includes *Erwinia*.

It is certain that the pathogen does not belong to the *Xanthomonas* group because of its many anomalous physiological properties (Table 1). Current practice (83) follows Dye's suggestion for naming the bacterium.

Morphology.—Erwinia stewartii is a non-motile, non-flagellate, non-spore-forming, capsule-forming, Gram-negative rod, 0.4-0.8 μ by 0.9-2.2 μ. The bacterium is aerobic to facultatively anaerobic, and occurs singly or in short chains (63, 151, 180, 300). It frequently exhibits "barred" staining and evidence of "snapping" division (138).

Differences in colony characteristics have been observed by several workers. Ivanoff et al. (180) grouped 22 *E. stewartii* isolates into three main groups, based on growth and colony characteristics on nutrient glucose agar. The colonies ranged in size from 3 to 12 mm, and differed in color, consistency, surface, elevation, and form and amount of growth (Table 2). Lincoln reported that colony diameters of *E. stewartii* ranged from 1.8 to 11.0 mm when grown on nutrient glucose agar (208). He reported generation times of from 1.8 to 3.2 hours; other colony characteristics were similar to those reported by Ivanoff et al. McCulloch (227) observed smooth flat-surfaced colonies, or colonies with definite central depressions on beef-peptone agar. A correlation between colony appearance and virulence was observed by Wellhausen (390), Lindstrom (212), and Ivanoff

TABLE 1. Comparative physiology of *Erwinia stewartii* and typical *Xanthomonas* species.[a]

Character	Typical *Xanthomonas* spp.	*E. stewartii*	Literature citations
Motility	Yes	No	227
Flagellation	Polar monotrichous	None	98
O_2 requirement	Strict aerobes	Facultative anaerobe	98, 150
Temperature relations:			
minimum	—	8-9°C.	98, 116
maximum	—	c. 39°C.	98, 116
optimum	—	30°C.	98, 116
thermal death point	—	53°C.	98, 116
Growth habits on:			
Uschinsky's solution	Scanty to heavy	Long, copious growth	116
Cohn's solution	Mostly no growth	No growth	116
Fermi's solution	Scanty or none	Feeble growth	116
nutrient broth	Usually turbid, with or without pellicle or ring	Feeble growth, whitish ring, yellow precipitate	64, 116
Salt (NaCl) tolerance	2-3%	5-7%	65
Triphenyltetrazolium chloride (TTC) tolerance	Low	High	219
Action on milk	Variable	No curd, no clearing, slightly acid	64, 116
Indole production	Rare or none	Slight or none	64, 116
H_2S from cysteine	Yes	Yes, no	28, 150
Urease	No	No	150
Voges-Proskauer reaction	Negative	Negative, positive	64, 116
Lipolysis	Positive	Negative	97, 293
Gelatin liquefaction	Usually	Slight or none	64, 116
Organic nitrogen	Assimilated	Assimilated; essential for avirulent strains	231
Inorganic nitrogen ($-NO_3$ & $-NH_4$)	Variable	Assimilated, nitrate reduction only by most virulent strains	150, 231, 315
Nitrate destruction	None	None	150, 315
Asparagine as sole source of C & N	No	Yes	98, 319
Hydrolysis of:			
soluble starch	Yes	No	150
casein	Yes	No	150
aesculin	Yes	No	98
pectin: liquefaction of sodium pectate gel, pectin methylesterase, pectin polygalacturonase, and protopectinase	Variable—usually some pectinase activity		95, 98
Utilization of:			
gluconate	No	Yes	98
propionate	Yes	No	98
tartrate	No	Yes	98
Metabolism of:			
glucose	Oxidative	Fermentative	98
sucrose	Oxidative	Fermentative	98
lactose	Oxidative	Fermentative	98
salicin	None	None	98, 180
inositol	None	Oxidative, acid produced	98

TABLE 1. (*Continued*)

Character	Typical *Xanthomonas* spp.	*E. stewartii*	Literature citations
galactose	Variable	Acid, no gas	231
mannose	Variable	Acid, no gas	231
arabinose	Variable	Acid, no gas	231
xylose	Variable	Acid, no gas	231
mannitol	Variable	Acid, no gas	231
maltose	Variable	No acid	231
rhamnose	Variable	Alkaline	231
dulcitol	Variable	Alkaline	231
fructose	Variable	Acid, no gas	231
glycerol	Variable	Acid, no gas	231
Producing "*Xanthomonas*[b] carotenoid"	Yes	No	317, 318
Producing "*Xanthomonas*[c] polysaccharide"	Yes	No	133
Pathogenesis	Plant necrosis	Plant wilt and necrosis	64

[a] Where conflicting reports occur, both authorities are listed. In some cases, different isolates have given variable results.

[b] A "*Xanthomonas* carotenoid" is defined as a carotenoid "alcohol" with absorption maxima at 418, 437, and 463 mμ in petroleum ether (317, 318).

[c] A "*Xanthomonas* polysaccharide" yields aldobiouronic acid, glucose, and mannose on hydrolysis (133).

(180). Virulence was associated with large, watery, smooth, spreading, mucoid colonies; avirulent strains were smaller, rough, raised, and non-mucoid. The color of the colony is typically buff-yellow, but may range in hue from creamy to orange (180). Single-cell isolates of *E. stewartii* sector on potato-dextrose agar plates (but not on beef-peptone agar), to produce white isolates which retain their white appearance upon subsequent transfers (120). The white variants were no more virulent than the parent culture. Two white variants reported by McNew (228, 229) were virulent, but did not remain viable on nutrient-dextrose agar slants, as did typical yellow colonies.

Serology has failed to indicate the relationship of *E. stewartii* to other bacteria. St. John-Brooks et al. were unable to find such a relationship between *E. stewartii* and various other plant-pathogenic bacteria, including several *Xanthomonas, Erwinia,* and *Corynebacterium* species (292). This study, as well as those which attempted to correlate serological properties of *E. stewartii* with specific colony characteristics, physiological attributes, or degree of virulence (54, 234), used agglutination, but not the newer serological techniques [e.g., Ouchterlony gel-diffusion test (252)]. Frampton and Hildebrand (126) were unable to relate electrophoretic mobility to virulence, but found that mobility-pH curves at constant ionic strength were distinctive for *E. stewartii.*

Physiology.—The physiology of *E. stewartii* is summarized in Table 1. These physiological characters further emphasize the difficulties in the taxonomy of the bacterium. Hollis has suggested that wilt bacteria, including *E. stewartii,* resemble rhizosphere

TABLE 2. Growth characteristics of three main groups of *E. stewartii* on nutrient-glucose agar, after 14 days incubation at 24°C[a]

Characteristics	Type A	Type B	Type C
	I. On agar plates		
Color	Orange-yellow	Lemon-yellow	Cream-yellow
Colony diameter	10-12 mm	8-10 mm	3-5 mm
Consistency	Butyrous to butyrous-viscid	Viscid	Somewhat membranous
Surface	Smooth or crateriform and slightly rough	Smooth or concentrically ringed	Smooth
Elevation of growth	Convex	Raised	Flat
	II. On agar slants		
Amount of growth	Abundant	Abundant	Slight
Form of growth	Spreading	Mucoid	Filiform

[a] After Ivanoff *et al.* (180).

Figure 8. Typical flea-beetle feeding scars on corn leaves. Bacterial infection is not apparent. Photo courtesy Crops Research Division, Agricultural Research Service, U.S. Department of Agriculture.

forms in their general level of nutritional competence (158).

Gorin and Spencer (133) found that the pathogen produced a unique extracellular polysaccharide when grown on a dialyzed yeast extract medium. The polysaccharide yielded galactose, *inter alia*, upon hydrolysis, instead of mannose, as was the case with *Xanthomonas* spp. Whether this difference in the chemical composition of the polysaccharide is related to the pathogenic properties of the bacterium is unknown. There is evidence that virulence of the organism is correlated with colony viscosity (see THE CAUSAL ORGANISM, *Genetics*). Corey and Starr found that morphology, colony size, and symptoms were directly related to differences in polysaccharides produced by different isolates of *X. phaseoli* (86). Hodgson et al. have also investigated the effects of polysaccharides on wilt induction in tomato plants (152). Similar studies on the nature of polysaccharides of *E. stewartii* and their effects on host metabolism might be equally valuable.

Dye (95) found that the pectolytic activity of 38 species of *Xanthomonas* was variable, and concluded that the presence or absence of the various pectolytic enzymes was not a taxonomic criterion. *Erwinia stewartii* did not liquefy sodium pectate gel, and did not produce pectin methyl esterase, pectin polygalacturonase, and protopectinase. The pathogen also does not possess lipolytic activity (95, 316).

Starr and Stephens (317, 318) showed that xanthomonads produced a typical "*Xanthomonas*-carotenoid" with absorption maxima in petroleum ether at 418, 437, and 463 mμ. The wilt bacterium does not produce this pigment and therefore should not be placed in *Xanthomonas*.

A number of other physiological characteristics also exclude *E. stewartii* from other xanthomonads. *Xanthomonas* species typically fail to grow in nutrient broth amended with 5% NaCl, whereas this bacterium generally grows in salt concentrations of 6-7% (65, 98, 150). Lovrekovich and Klement (219) showed that *E. stewartii* possessed a tolerance to triphenyl tetrazolium chloride greatly in excess of 20 typical *Xanthomonas* species. Smith (304) reported that *E. stewartii* was able to grow on media amended with gentian violet, 1:100,000. Ark (21) found that vitamin C (1:200 to 1:1,000,000), cysteine (1:1,000), glutathione (1:1,000), pyrogallol (1:1,000), resorcinol (1:10,000), and tannic acid (1:100 to 1:10,000) prolonged the life of *E. stewartii* and several other phytopathogens on solid culture media. According to Starr and Weiss (319), *E. stewartii* grew slowly in a medium containing asparagine as the sole source of carbon and nitrogen, whereas no *Xanthomonas* species grew in this synthetic medium. Several authors have indicated that *E. stewartii* grows as a facultative anaerobe (98, 150); they include in *Xanthomonas* only those organisms that oxidatively metabolize glucose and other sugars. Since *E. stewartii* shows fermentative anaerogenic metabolism of glucose, sucrose, and lactose, and an oxidative anaerogenic metabolism of inositol, it should be excluded from *Xanthomonas*.

Ivanoff developed a selective medium with which *E. stewartii* could easily be isolated from contaminated plant material or from soil (169, 171, 176). The selective action of the medium was attributed to the presence of sodium taurocholate and a high NaCl concentration.

Genetics.—Intensive investigations on the genetics of *E. stewartii* have illuminated the problem of virulence in this organism, and perhaps in other phytopathogenic bacteria as well. The effect of successive passages of the bacterium through susceptible and resistant hosts, on the virulence of the pathogen was investigated by Wellhausen (390). Continued passages through a highly resistant corn host increased virulence, while those through a susceptible host decreased virulence. In either case, a limit was reached at which no further change took place. It would seem that, at this limit, an equilibrium was established between the host and the pathogen, after which further passages produced no effect. Similar passages through susceptible varieties of teosinte produced the same phenomenon on both corn and teosinte; whereas passages through unrelated and highly resistant grass hosts rendered the organism less virulent for corn, but more pathogenic for the grasses through which it was passed. Wellhausen's work was confirmed and amplified by Lincoln (207)

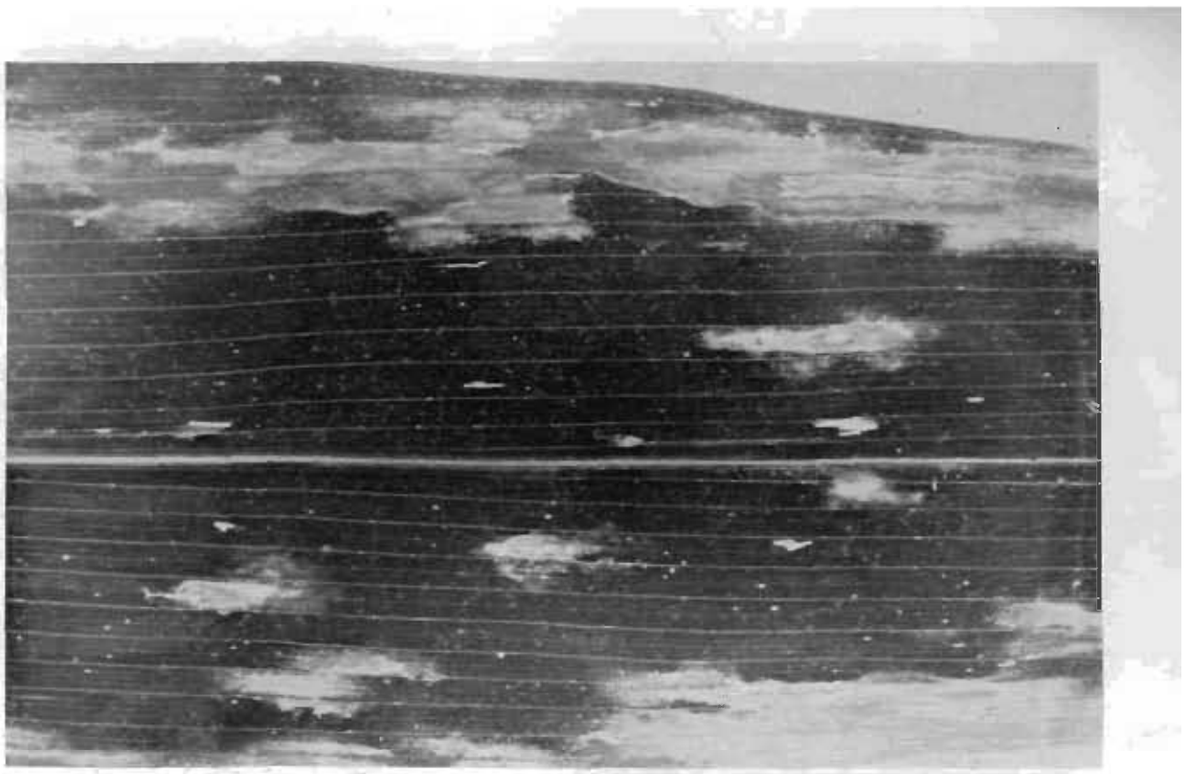


Figure 9. A corn leaf showing bacterial lesions associated with flea-beetle feeding sites. Photo courtesy Crops Research Division, Agricultural Research Service, U.S. Department of Agriculture.

and by Lincoln and Lindstrom (211), who passed known mixtures of virulent and avirulent wilt bacteria through resistant and susceptible hosts. Virulent types were selected out by resistant hosts, and avirulent types by susceptible hosts. Lincoln concluded that the intensity and direction of the selection process was dependent upon host resistance. The work of Wellhausen and Lincoln clarified the old concept that virulence is maintained or enhanced by continued host passage, as opposed to repeated transfers or *in vitro* cultivation. Lincoln (208) and Wellhausen (390) also observed that changes in virulence during host passage were correlated with changes in colony morphology. Cultures of low virulence produced firm colonies on nutrient-glucose agar, whereas those of high virulence produced spreading, watery, and viscid colonies. The virulence of attenuated isolates could be increased by passage through appropriate host plants. Virulent isolates could also be attenuated by similar passages. Lincoln showed in these studies that the change in virulence obtained by passage through the host was due to a shift in the ratio of virulent to less virulent forms (208). The virulence index of mixtures that contained different proportions of virulent and avirulent *E. stewartii* tested on susceptible corn was compared with the index of pure strains. Virulence bore a direct relationship to the ratio of virulent to avirulent bacteria (Figs. 14, 15). Thus, the proportion of virulent and avirulent types in the inoculum determined the virulence of the culture. The mutation rate for color and morphology of the colonies of three bacterial strains was reported at 1 in 20,000 to 1 in 80,000. Bacterial mutants showed both increases and decreases in virulence, and many such variants were as stable as the parent type. Lincoln found no evidence for sexual fusion of white and yellow strains of the bacterium in the living host.

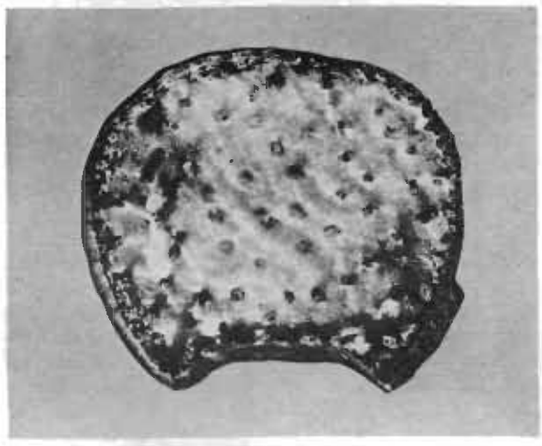


Figure 10. Cross-section of a sweet-corn stem infected with the wilt organism. Bacterial ooze is exuding in small droplets from the vascular bundles of the stem. Photo courtesy Crops Research Division, Agricultural Research Service, U.S. Department of Agriculture.

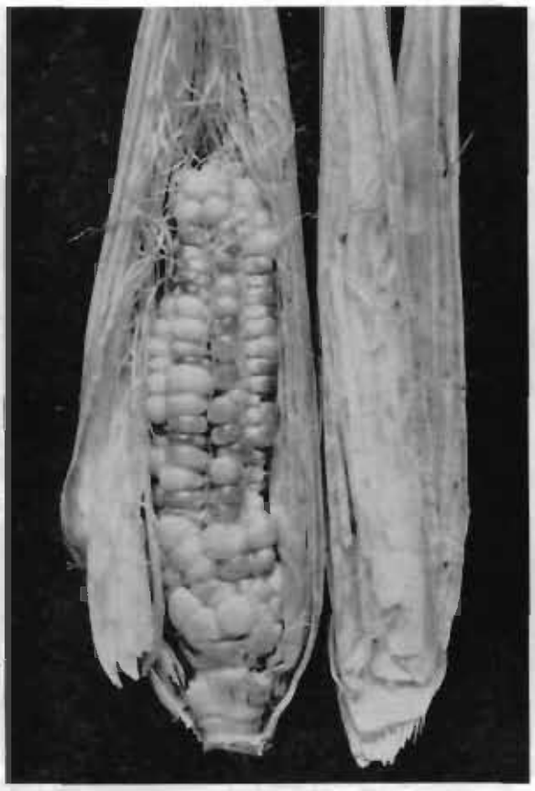


Figure 11. Ear of sweet corn infected by the bacterium, showing misshapen, shrunken, and discolored kernels. Photo courtesy Crops Research Division, Agricultural Research Service, U.S. Department of Agriculture.

Figure 12. Photomicrograph of the bacteria streaming from a vascular bundle in an excised infected corn leaf. Photo courtesy Crops Research Division, Agricultural Research Service, U.S. Department of Agriculture.

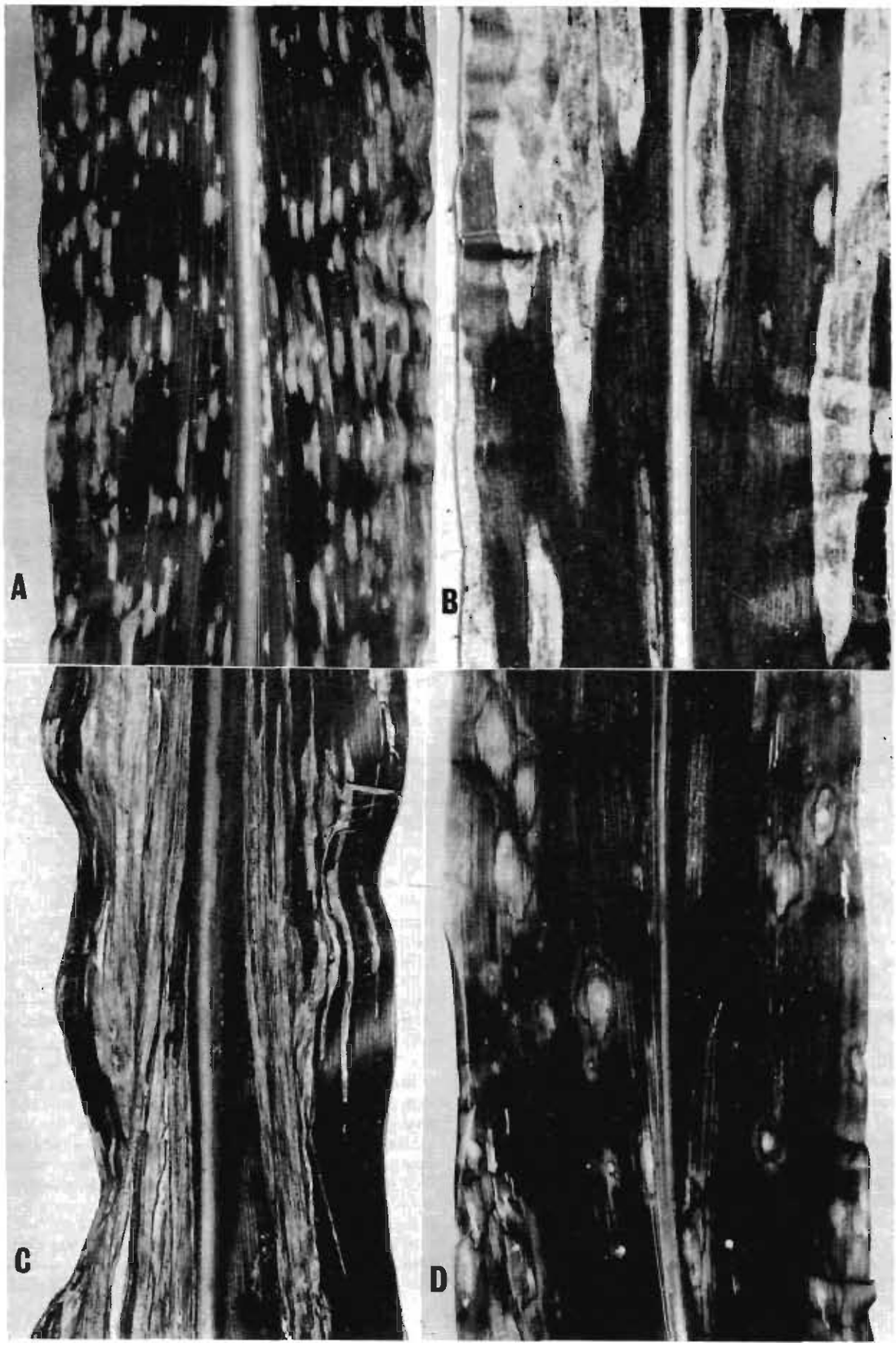
It has been shown that natural mutations in *E. stewartii*, tobacco mosaic virus, and *Drosophila melanogaster* Meigen are low in incidence, but can be increased by exposure to comparable X-ray doses (134). Since the rate at which mutants appeared was of the same order of magnitude, Gowen postulated a common basic structure for inheritance in all three organisms (134). X-ray radiation of low quantum energy increases the frequency of mutation in *E. stewartii*, but the type of mutants do not differ from natural mutants, according to Lincoln and Gowen (210). These workers suggested that the terms "mutant," "variant," "saltant," and "dissociate" were synonymous with reference to bacteria, and were the results of gene mutations. Similar genetic studies were conducted by Lindstrom (212, 213) on the effects of temperature, nutrition, and host-passage. He found that mutation rates of the bacterium were increased by elevated temperatures (Fig. 16) (213).

Lincoln observed mutations in colony color, surface appearance, and size (209). The mutation rate at 36°C was approximately 10 times greater than the rate at 12°C, with intermediate mutation rates observed at intermediate temperatures. He also found that the proportions of bacterial types changed, indicating that selection might be a strong force in changing the composition of bacterial populations. "It is apparent that some change in the genic balance has occurred because of the mutation of the dark yellow locus to pale yellow, resulting in greater genetic stability. It is probable that each mutation affects the genic balance and usually has pleiotropic effects on the organism" (209).

A total of 64 bacterial isolates, including 55 mutants and six parental strains, showed highly significant differences in degree of virulence when tested against a susceptible corn inbred (369). While it was shown that most mutants emerge with lower virulence than their parents, three mutants possessed a higher level of virulence. These genetic investigations have been reviewed by Gowen (135).

Ivanoff (180) and McNew (229, 232) studied the variability of *E. stewartii* in culture, and the effects of such variability on virulence. Highly virulent cultures of the bacterium became less virulent in a step-wise sequence following repeated transfers. The use of certain culture media promoted attenuation. McNew also reported that incubation at 36°C favored the increase of avirulent forms, as did maintenance of

Figure 13. Leaf blights of corn: A. Southern corn leaf blight, B. Northern corn leaf blight, C. Bacterial leaf blight, D. Helminthosporium leaf spot. Photo courtesy Crops Research Division, Agricultural Research Service, U.S. Department of Agriculture.


A
B
C
D

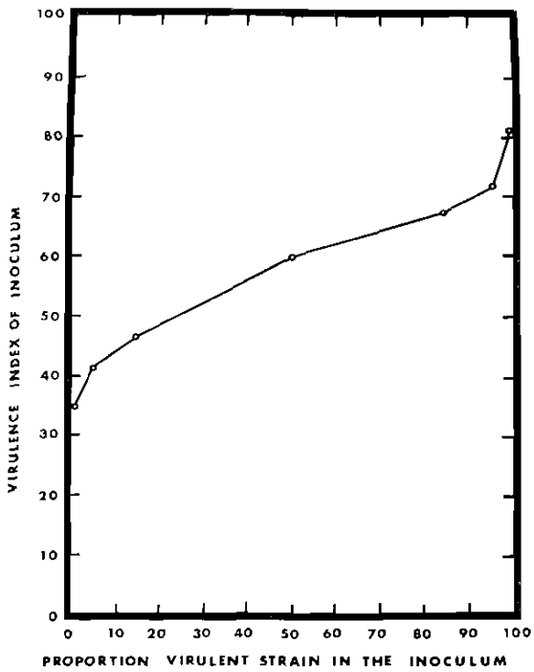


Figure 14. Change in virulence index of inoculum composed of various proportions of virulent and avirulent *E. stewartii* strains. From Lincoln (208).

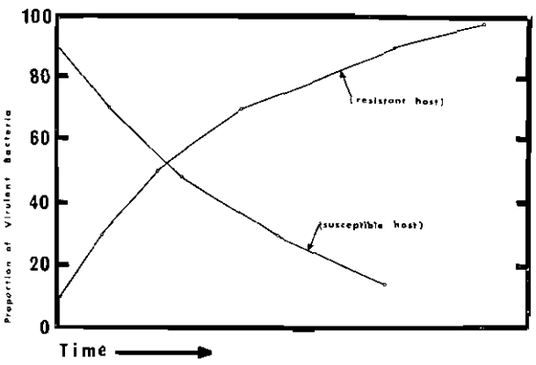


Figure 15. The effect of host passage on the proportion of virulent to avirulent bacteria in mixed culture inoculations. From Lincoln (208).

cultures without transferring to fresh media (232). He also found a correlation between virulence and the ability to utilize inorganic nitrogen (231). Most virulent strains used ammonium nitrogen in preference to the nitrate form, and were capable of reducing nitrates to nitrites. The production of nitrites, however, was not the sole factor involved in the wilting of host plants.

Bacteriophage.—Thomas described a bacteriophage in 1935 capable of lysing *E. stewartii* (356). The phage was most prevalent in diseased kernels,

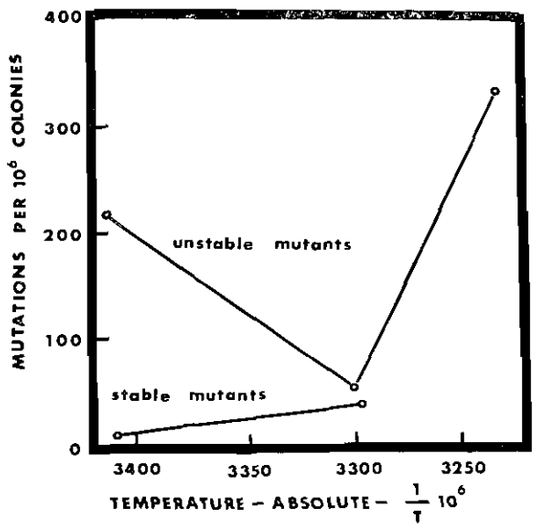


Figure 16. The effect of varying temperatures on the mutation rates of stable and unstable mutants of *E. stewartii.* From Lindstrom (213).

lower nodes, and roots. The phages could generally be recovered from field corn, but not sweet corn except from infected seed (357). They could always be recovered from infected plants (360). The phage causes a change or loss of color, a change in viscosity, and a reduction or loss of virulence in the bacterium (356). Although the serological, immunological, and thermal inactivation properties have not been investigated, the phage appears to be relatively stable, as the lysing ability of one preparation was unchanged after 12 years of storage in a sealed tube at room temperature (361). Cross-infection studies also have not been made, but tests of phages which attack other xanthomonads have indicated that none lyse *E. stewartii* (195, 348). This is further evidence that *E. stewartii* does not belong taxonomically in the genus *Xanthomonas.*

The use of phage in the control of the disease has been attempted by Thomas. When seed corn was steeped in a phage preparation, dried, and planted, a reduction in wilt infection was obtained, and it was postulated that this effect might be important in reducing disease loss (356, 359). Thomas also presented data which he believed indicated that phages might be a factor in making corn varieties resistant to corn wilt (360).

Miscellaneous Investigations.—McNew (230) found the poured-plate technique a satisfactory method for obtaining single-cell cultures of the wilt bacterium. More than 99% of the loci examined were occupied by single cells, and 80-94% of these cells multiplied, depending on the nutrient content of the medium employed.

Techniques for inoculating corn plants with *E. stewartii* have been described by several investigators. Ivanoff (172) devised a modified hypodermic syringe for rapid inoculation of large numbers of plants.

Inoculation and rating methods for the disease were described by Lockwood and Williams (217). Bacterial suspensions were applied to the cut ends of clipped seedlings and disease ratings were made on the basis of severity, area of the lesions, and wilting. Similar methods were presented by McNew (229) and Wellhausen (390).

Zahl and his co-workers (409) investigated the induction of tumor-hemorrhage in mice by suspensions of killed bacteria (408). They suggested that Gram-negative bacteria, including *E. stewartii*, possessed a factor which induced vascular toxicity. This toxicity was demonstrated by the induction of hemorrhage in implanted mouse tumors when bacterial suspensions were introduced parenterally into the test animals.

Mai (222) studied the toxicity of fungus and bacterial culture filtrates to encysted nematode larvae (*Heterodera rostochiensis* Wollenweber, 1923), and found that filtrates from *E. stewartii* were not lethal.

Host-Parasite Relations

The occurrence of the pathogen in the host plant was studied by Smith (302) and Ivanoff (169, 171). Bacteria were found in xylem vessels, intercellular spaces, cavities adjacent to disrupted vessels, and between cells in corn leaves. Invaded tissues of leaves and husks were discolored and dead. Bacteria were observed in vessels and pith tissue of the stem, shank, and cob, and in some cases on the shank surface. Bacteria were found in the vascular tissue of the rachises, glumes, rachillas, in the vessels of filaments, and in anthers and pollen. Bacteria were present in diseased kernels in the vascular tissue of the chalazal region, the aleurone layer, and between endosperm cells, but not in the embryo.

Wellhausen (389) discovered marked histological differences in the reaction of vascular bundles of various inbred corn lines to bacterial invasion. Rapid plugging of the vessels, and deterioration of the adjacent parenchyma, occurred in very susceptible corn lines, and sometimes the entire vascular bundle was destroyed. Infection of the protoxylem stimulated cell division and lignification of the neighboring parenchyma tissue of moderately susceptible sweet and white flint corn. The conducting tissue gradually became plugged and contributed to the slow death of the plant. The morphology of the vascular bundles did not change in highly resistant corn lines, although in a few cases slight plugging of the vessels occurred.

Warren (386) showed, by means of radioactive P^{32}, that the rate of bacterial movement in the host was correlated with the transpiration rate of the plant. The rate and extent of movement in the host did not greatly differ between resistant or susceptible inbreds. Transpiration and fluometric data obtained by Harris (139) indicated that wilting of infected sweet corn plants was caused primarily by mechanical plugging of the xylem tissue.

Several workers have reported that plants attacked by *E. stewartii* were rendered more susceptible to stalk and ear rots caused by *Diplodia zeae* (Schw.) Lev. and *D. macrospora* Earle (155, 192, 384). Although Smith stated that wounding is not necessary for invasion by *E. stewartii* (301, 302), he and other investigators later showed that the bacterium is almost exclusively a wound invader (151). Bhide reported that infection occurred through hydathodes, but not lenticels (28).

Disease Cycle

Seed Transmission.—More than 20 years elapsed after the discovery of Stewart's bacterial wilt of corn before overwintering of the causal organism was understood. Stewart thought that the bacterium was present in the soil and in infected seed, and that infection took place in the underground plant parts (347). Later work by Smith (303) corroborated the evidence for the presence of bacteria within the seed. Evidence from culturing and planting infected seed showed that the pathogen survives within the seed (129). Infected seeds are important in transporting the bacteria to different areas, but probably are unimportant in overwintering. Only about 2% of the plants from infected seed develop the disease under controlled conditions that minimize secondary spread or disease development from other sources (107, 273, 290). It is likely that infected seed provides an infection source for spread of the pathogen by insect vectors (see below).

Soil Transmission.—Overwintering of *E. stewartii* in the soil and in plant debris has not been shown (271, 277, 352). The planting of seed with infested debris, or in soil inoculated with a bacterial suspension, did not significantly increase the percentage of wilt over the control in field tests (107).

Insect Vectors.—The discovery of insect vectors of the pathogen indicated a potential overwintering site (117, 270). The corn or brassy flea beetle, *Chaetocnema pulicaria* Melsh. (Fig. 17), overwinters in the adult stage and feeds on young corn plants after emerging from hibernation. Surface-disinfected and triturated adult beetles, collected and cultured in April, yielded large numbers of *E. stewartii* in almost pure cultures (117). Corn inoculated with these cultures by wounding or insect feeding developed characteristic wilt symptoms. Elliott and Poos also found that *C. pulicaria* was the only one of 40 species examined that carried the bacterium (263): 75% of the flea beetles carried the pathogen in this test and 19% in another (264). These workers showed that *C. pulicaria* was the only insect of importance in overwintering (117, 118, 261). Although *Chaetocnema denticulata* Ill., the toothed flea beetle, carried small quantities of the bacteria, it was doubtful whether overwintering occurred in that species. It is now thought that the corn flea beetle, *C. pulicaria*, is the most important means of overwintering. The bacterium may also overwinter in soil, plant debris, and infected seed. *Chaetocnema pulicaria* was found (107, 271-273) to transmit the bacterium to 37-100% of the corn plants in 7 tests in cages. No infection occurred in the controls. Similar tests with *C. denticulata* gave 83% transmission; no infection occurred in the controls.

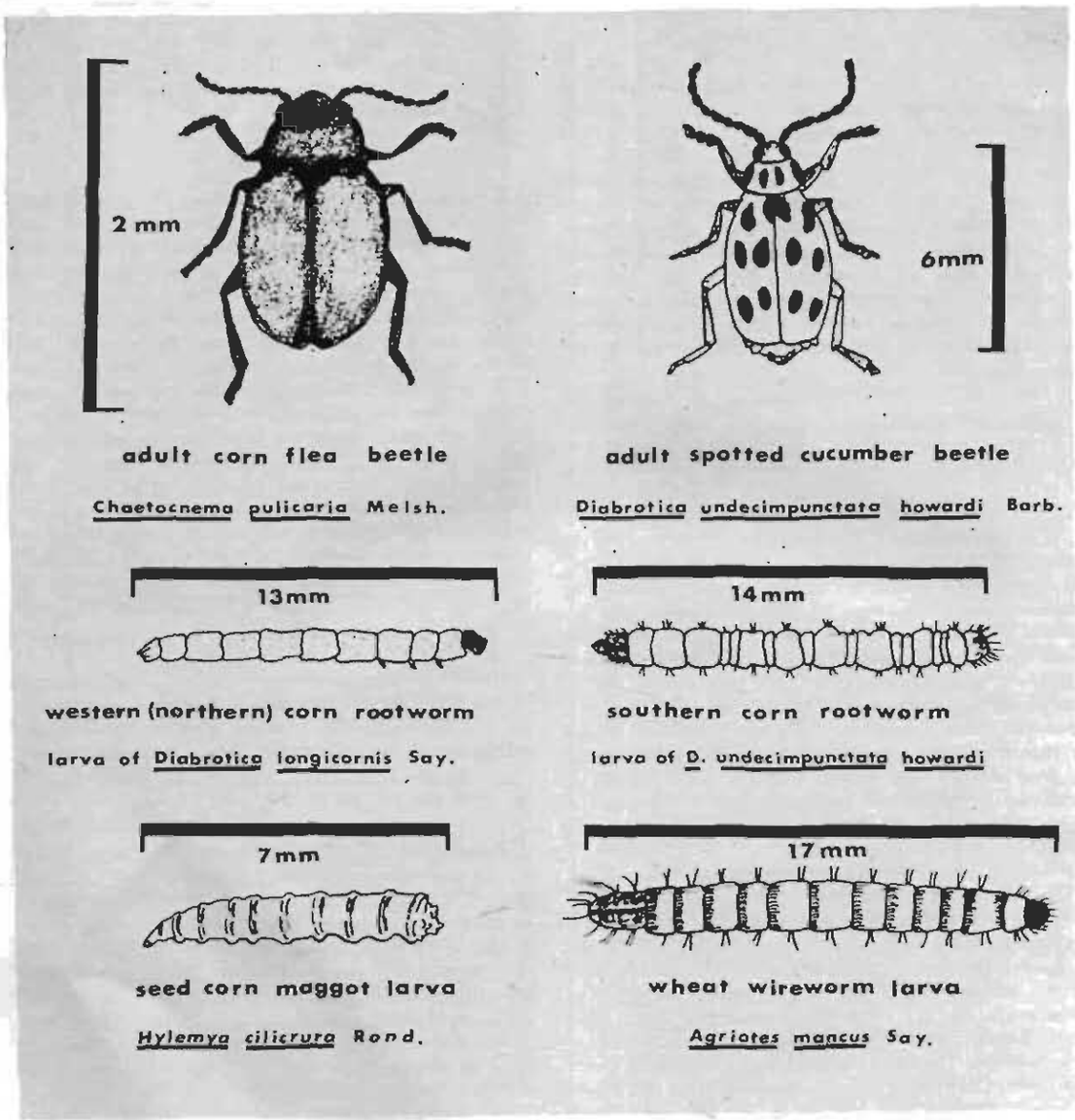


Figure 17. Insect vectors of *E. stewartii*.

The larval stage, or Southern corn rootworm, and adult form of the 12-spotted cucumber beetle, *Diabrotica undecimpunctata howarti* Barb. (formerly *D. duodecimpunctata* Oliv.), have also been shown to spread *E. stewartii* in the field (263, 264, 272, 273). The larval and adult forms of the 12-spotted cucumber beetle harbored the bacterium for prolonged periods in the alimentary tract, but were not efficient vectors. Ivanoff (169, 171) found that the bacteria in soil, infected corn roots through wounds made by white grubs, the larval stage of *Phyllophaga* sp. (May beetles). He also found the Northern corn rootworm, *Diabrotica longicornis* Say (170, 171, 181), and the Western corn rootworm, *D. virgifera* Lec., to be vectors.

Frutchey (129) found 2 additional insect vectors: larvae of the seed corn maggot, *Hylemya cilicrura* Rond., and the common wheat wireworm, *Agriotes mancus* Say. Adults of *H. cilicrura* infected 72% of the plants on which they were caged.

The European cornborer, *Pyrausta nubilalis* Hbn. was implicated as a vector in Russia (250). A later paper attributed the "virtual absence" of Stewart's bacterial wilt in the USSR to the absence of the primary vector, *C. pulicaria* (381).

The known vectors of the wilt organism are discussed by Leach (205, 206), Metcalf, Flint, and Metcalf (240), Peairs and Davidson (256), and Carter (75), and are shown in Figure 17. It should be emphasized that only the corn flea beetle, *C. puli-*

caria, is important in the overwintering and dissemination of the bacterium under natural conditions.

Environmental Factors.—Rand and Cash suggested that anything that retarded seed germination and early development of the seedling reduced infection from diseased seed (271, 273). This observation has been confirmed in the United States (308) and Italy (254).

The mineral nutrition of corn may profoundly influence its reaction to infection by *E. stewartii.* Susceptibility increased with an increase in nitrogen, and especially ammonium nitrogen as opposed to nitrate forms (212). High phosphorus levels also increased susceptibility, but an increase in resistance was obtained at high calcium and potassium levels. Spencer and McNew (313) found that seedlings stunted by high concentrations of nitrogen, phosphorus, and potassium were attacked more severely than were those receiving adequate quantities to produce rapid plant growth. A deficiency of potassium increased infection more than a deficiency of nitrogen or phosphorus. A decline or an increase in potassium concentration (below or above 40 mg per 100 ml) favored wilting of Golden Bantam seedlings. The addition of nitrogen alone at high levels greatly increased the severity of bacterial wilt infection (212, 235, 236, 313).

McNew and Spencer (235) reported that weakly and highly virulent bacterial strains were approximately equal in invading nitrogen deficient seedlings, while in seedlings grown at high nitrogen levels, the highly virulent isolates were much more invasive. Virulent bacterial strains were equally invasive in young seedlings and mature plants, whereas less virulent strains were most invasive in plants that were more than 14 days old (233). Since the weakly virulent strains were dependent on organic nitrogen, they suggested that such organic compounds appeared in the vascular tissue only after the plant had begun synthesizing its own organic materials, i.e., after about 14 days (236). They also suggested that virulent strains of the bacterium could multiply rapidly in xylem vessels of young plants high in inorganic nitrogen, thus competing with the host for available nitrogen (236). Weakly virulent bacterial strains would consequently be less able to establish because of their inability to utilize inorganic nitrogen.

Some of the factors involved in the effects of mineral nutrition on pathogenicity are discussed by Shear and Wingard (296). They suggested that increased disease severity in potassium-starved seedlings might result from an increased nitrate-nitrogen level within the vascular tissue. Excess nitrogen may increase disease severity in several ways, e.g., by increasing succulence of tissues, by providing certain essential and complex nitrogenous compounds, or by providing nitrate-nitrogen in forms that may be used preferentially by certain bacterial strains (296).

An increase in soil moisture has been correlated with an increase in wilt severity (212, 271, 273). The effect of increased soil moisture is apparently manifested in earlier, more vigorous and succulent growth. High temperatures and abundant rainfall seem to accentuate disease severity (273).

Summary.—The disease cycle may be summarized as follows: The pathogen overwinters in the alimentary tract of hibernating adult corn flea beetles (*C. pulicaria*). The beetles emerge from hibernation in early spring and feed on the seedlings of early planted corn, infecting them. The beetles lay eggs on the plants soon after their emergence. New broods develop and adult forms from both the first and second generations may ingest bacteria from primary infection sites. They then spread infection to other plants in the field. The bacterium is carried by the flea beetle for the rest of its life. While only about 10 to 20% of the beetles emerging from hibernation carry *E. stewartii,* up to 75% of the beetles feeding on corn in midsummer may be carriers (289). Leaf infections on dent corn appear at this time, as a result of secondary spread. Later in the season the percentage of bacteria-carrying beetles decreases again. Although other insect vectors are known, they are of little importance compared with the corn flea beetle. Soil transmission of the pathogen is rare or non-existent. The pathogen may be seed-borne, establishing new foci of infection, serving as a means of long-distance dissemination, and providing inoculum for seasonal buildup. Disease severity is increased by conditions which promote rapid growth, e.g., rich soils, high nitrogen levels, low potassium levels, elevated soil moisture, and high temperatures during the growing season (Fig. 18).

Control Measures

While some of the suggested control measures have had merit in isolated circumstances, the use of resistant corn varieties is the only really effective measure at the present time. Some of these measures (e.g., seed selection and vector control) might prove useful in the future. Stevens (332) pointed out in 1942 that intensive selection of the indigenous corn

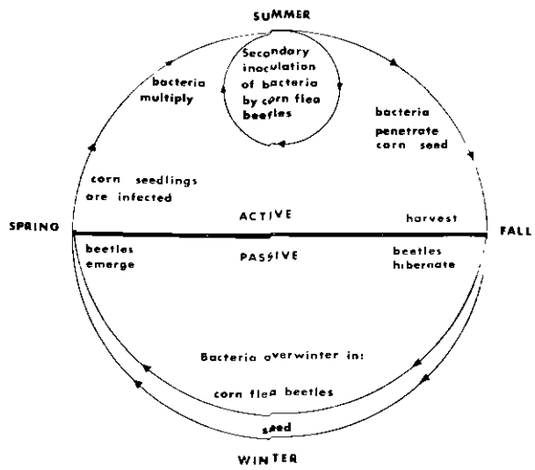


Figure 18. Disease cycle of Stewart's bacterial wilt of corn.

has gradually converted the crop from a freely cross-pollinated species, with almost unlimited capacities for adjusting to new pathogenic races, to one lacking the ability to adjust to new parasites. The extent of this intensive selection is indicated by the increase of hybrid corn acreage from 1.1 million acres in 1935 to 20.6 million acres in 1939, and to 78.4 million acres in 1960 (17). Hybrid seed corn accounted for 96% of all corn acreage in the United States in 1961. There have, however, been no recent devastating epidemics in the corn crop to parallel the losses in wheat caused by Race 15-B of *Puccinia graminis* f. sp. *tritici* Eriks. & E. Henn.

Seed Selection and Treatment.—The use of disease-free seed was and is widely recommended (352). It was generally recommended about 1920 that growers use only seed produced in northern areas in which the disease rarely occurred (271). The use of disease-free seed is not effective in areas in which *E. stewartii* is indigenous (289, 375). The exclusion of infected seed from areas now free from the disease is desirable (237), as for example, Mauritius (16). A Polish publication similarly recommended strict quarantine, as well as the use of bacteria-free seed, resistant varieties, and vector control (291). This would be important if the insect vectors were present in the area, since without the vector, no spread occurs after initial infection from diseased seed (255, 381). Some writers have suggested, however, that other, as yet unknown, insect vectors may be responsible for the spread of the disease in Italy and the USSR, where *C. pulicaria* does not occur (297). Cheremisinov (77) has recommended that grain be selected from the top portion of the cob, in addition to hybridization and regular fertilization with potassium and phosphorus, to control the disease. These recommendations are of doubtful value.

Numerous workers since Smith (303) have recommended assorted chemical and physical seed treatments to lessen transmission. Results have generally been inconclusive. For example, control was obtained with mercuric chloride by Eddins (99), Elliott (116), and Smith (303), and with other chemicals by Bowman (45), Somers (311), and Ullstrup (371). Control was not obtained with bactericides by Frutchey (129) and Reddy (278), nor with dry heat by Frutchey (129). Rich tested a number of antibiotics, growth regulators, and other chemotherapeutic compounds as possible seed treatments (280). Several of these treatments reduced wilt symptoms significantly in developing seedlings, but no evidence was presented for control beyond the seedling stage. Antibiotics, when applied as seed treatments at rates capable of reducing wilt incidence, were phytotoxic (248, 397). Sprays containing streptomycin or terramycin reduced the incidence and severity of wilt, but yields were not increased in experiments reported by Lockwood and Williams (216). These workers found that terramycin and cycloheximide in low concentrations reduced symptoms on inoculated plants in the greenhouse, as did certain surface-active agents (124, 398). Natti found that *E. stewartii* was inhibited by streptomycin and terramycin in a filter-disc assay (247). When streptomycin was applied to sweet-corn seedlings in field trials, no therapeutic effect was obtained; however, streptomycin did reduce field spread of the bacterium. Mehta (238) and Rangaswami (275) suggested that Vancomycin and mycothricin might prove effective. Thomas studied the phenol coefficient of a number of bactericidal materials (353).

Seed treatments are generally recommended regardless of whether vectors are present.

Control of Insect Vectors.—Chemical control of the flea-beetle vector is of value in reducing the spread of the disease, especially when used in conjunction with seed treatment. Poos obtained good control of Stewart's bacterial wilt in sweet corn by applying DDT in the seedling stage to reduce flea-beetle feeding (262). Adams and Chupp (1) also recommended vector control. Boewe reported that dieldrin effectively controlled the corn flea beetle (39, 246). Insecticides are still impractical as a control measure in many cases because of labor and material cost and the hazard of residues. Early spraying or dusting with insecticides is, nevertheless, a practical method of reducing spread of *E. stewartii* in early-infected fields (374).

Resistant Varieties and Host Genetics.—As early as 1899, Halsted (138) reported that a small early variety, "First of All," was severely attacked by the disease. According to Valleau (186), corn varieties producing larger plants were generally more resistant to the disease than shorter types. Although the varietal tests conducted by Reddy demonstrated no marked wilt resistance, flint corn appeared to be most susceptible while dent varieties were least affected (276). Rand and Cash found that in 53 sweet-corn varieties, the time of maturity correlated almost exactly with the percentage of wilt development (271). The late varieties consistently gave a low percentage of infection (10% or less), but early varieties were seriously affected (25 to 50% or more). Thomas also reported that susceptibility was correlated with earliness in sweet-corn varieties (352).

These early observations initiated a series of investigations on the nature of resistance to the disease. Reddy and Holbert (277) showed that all progenies of certain lines of concurrently-maturing inbreds uniformly showed high wilt resistance, whereas those of other lines were regularly susceptible. This study demonstrated the feasibility of incorporating wilt resistance into popular but wilt-susceptible sweet-corn varieties. They found no correlation between resistance and vegetative vigor, contrary to other observations (e.g., 186). Mahoney and Muncie (221) questioned the heritability of wilt resistance, and asserted that certain hybrid characters such as growth rate and hybrid vigor might enable a "resistant" plant to outgrow the disease rather than to resist or tolerate infection. Ivanoff and co-workers reported a correlation between resistance and both vigor and lateness in inbred strains of Golden Bantam sweet corn (177-179, 182, 183). Crosses between susceptible inbreds generally gave susceptible hybrids;

crosses between resistant and susceptible inbreds gave resistant hybrids. Three types of resistance were recognized: (i) vigor-correlated, where vigor was measured by height; (ii) lateness-correlated; (iii) "true" resistance. Dent corn varieties of a given lateness and height were found by Ivanoff to be no more resistant than sweet and flint varieties of the same lateness and height (175). He concluded that the resistance of open-pollinated dent corn was similar in both type and degree to that in open-pollinated flint and sweet corn.

Wellhausen (388) studied the genetics of 56 inbred lines of dent, flint, and sweet corn, and 14 crosses derived from these lines. He found that all gradations of resistance, from highly resistant to highly susceptible, were present in the inbred lines. The majority of the field-corn inbreds were resistant, the Evergreen group intermediate, and the early sweet-corn varieties susceptible. Wellhausen stated that resistance was dominant in all F_1 material tested and, that in a few cases, the F_1 material was more resistant than either parent. Results from testing backcrosses and late-generation progenies indicated that two major dominant complementary genes, with perhaps a third modifying gene, were involved in wilt resistance (214, 388-391). A study by Ivanoff and Riker (179) indicated that resistance in corn hybrids generally appeared to be dominant, and that tall and late hybrids tended to be more resistant than short and early hybrids. Hybrid sweet corn produced from highly resistant inbreds generally showed high resistance, regardless of its degree of earliness or lateness. Elliott (113), on the other hand, reported that some dent-corn inbreds, resistant to the leaf-blight phase, were more susceptible in later stages of growth. She emphasized the importance of later inoculations with the pathogen to obtain more accurate assessments of "true" leaf-blight resistance. It was suggested that resistance to *E. stewartii* might be correlated with resistance to *Helminthosporium turcicum* Pass. (290). Further investigations supported this suggestion with field reports of resistance to *E. stewartii* in corn bred for resistance to *H. turcicum* (257). Koehler (199) stated, "concurrent resistance to two entirely unrelated diseases would be something unusual, but appears to exist for [*E.*] *stewartii* and *H. turcicum* among corn belt inbred lines and crosses on which data are available. . . . The progress already made in obtaining resistance to both diseases while working for northern leaf blight will no doubt be of value for the corn belt, as a good method for obtaining resistance to Stewart's disease independently has not yet been devised."

Although the mechanism of the inheritance of resistance to *E. stewartii* is not fully understood, the systemic phase seems to be controlled by two major and one minor gene (346). Whitney and Mortimore (395) suggested that the compound, 6-methoxybenzoxazolinone, plays a role in sweet-corn resistance to the bacterium. *In vitro* growth was inhibited by the compound, which was also shown to occur in the roots and stalks of resistant sweet-corn hybrids.

As already stated, the use of resistant corn varieties and hybrids is presently the only practical method of controlling Stewart's bacterial wilt and leaf blight (153, 161). No commercial dent-corn hybrids are known to be immune to the leaf-blight phase of the disease. Peterson and Anderson (258) found that planting time had no effect on the leaf-blight phase, and that the field-corn inbreds tested varied widely in disease expression. Many sweet-corn hybrids with good resistance, developed during the past 30 years, have almost completely replaced the older susceptible varieties (6, 310). The early yellow sweet-corn varieties were grown extensively for the canning industry prior to the early 1930's. These varieties were prized for their early maturity and high quality, but were highly susceptible to wilt. The early yellow sweet-corn varieties, including Golden Bantam, Improved Golden Bantam, Extra Early Bantam, and Golden Early Bantam were, therefore, replaced by the later-maturing white varieties, Country Gentleman, Stowell Evergreen, and Vanguard (289). The improvement in wilt resistance was, unfortunately, offset by the imperfections of later maturity and poorer quality in these varieties. The sweet-corn hybrid, Golden Cross Bantam (P39 × P51), was first tested in 1931 by Smith, and released in the corn belt during the wilt epidemics of 1932 and 1933, before some of the basic knowledge about the disease was at hand (306, 308). This hybrid had shown high resistance in areas where open-pollinated varieties were highly susceptible to the disease. Smith's observations were subsequently confirmed, and the ready acceptance of Golden Cross Bantam by the canning trade and local markets provided the impetus for the rapid development of other wilt-resistant sweet-corn hybrids, including Marcross, Spancross, Whipcross, Tendergold, Indigold, Purgold, and many others (17). With the replacement of older wilt-susceptible varieties by newer early-maturing yellow hybrids, the threat of damaging epidemics had essentially disappeared by 1954 (Fig. 19). All corn-improvement programs take Stewart's bacterial wilt into consideration, and germplasm is now surveyed and tested for resistance in most sweet-corn seed-production schemes. Since new hybrids with resistance to *E. stewartii* are being released each year (44, 87), the reader is referred to local Experiment Station and Government publications for varietal listings and recommendations.

Disease Forecasting.—Forecasting of severe wilt outbreaks is based on mean winter temperatures preceding the growing season (241). Since the winter of 1932-33 was unusually mild in the northeastern United States, Stevens (323) predicted that "interesting developments" might occur in the summer of 1933 if there was actually a correlation between winter temperatures and wilt severity. The prediction was realized—1933 saw a repetition of the 1932 epidemic. In 1934, Stevens reviewed the evidence linking winter temperatures and wilt severity (325), and outlined an hypothesis for predicting the spread and severity of the disease. The hypothesis stated that the disease would be generally "absent in the northeastern United States following a winter with an index below 90, and present in destructive amounts following a winter with an index above

Figure 19. Sweet corn field plots showing varieties susceptible (left) and resistant (right) to the pathogen. Photo courtesy Crops Research Division, Agricultural Research Service, U.S. Department of Agriculture.

100° F." The winter index was the sum of the mean temperatures for December, January, and February; it was empirically derived from observations that populations of the primary vector, *C. pulicaria*, were reduced by severe winter temperatures. One severe winter might thus eliminate this disease in the following summer in northern states, and in southern areas a series of cold winters might be necessary to reduce disease incidence (160, 192, 244). Planting of susceptible varieties was determined by the disease forecast.

Based on Steven's hypothesis, a series of experimental forecasts was begun in the winter of 1934-1935, and the results were published annually in the Plant Disease Reporter (327, 329, 331, 334-336). The annual forecasts for the years 1935-1940 were critically evaluated in a final paper by Stevens and Haenseler (337). Stevens emphasized the validity of the "forecasting hypothesis," in a paper covering the year, 1942-1943 (333). Additional supporting evidence was provided by Haenseler (137), who found that the correlation between winter temperatures and wilt severity was valid in New Jersey from 1910 to 1936.

The forecasting experiment was a complete success; in fact it was terminated for that very reason. As Stevens stated, "By the year 1940 many had lost interest in the project. It had proven too easy" (345). The rapid increase in the use of wilt-resistant hybrids after 1932 also eliminated the need for such forecasting.

Boewe presented data in 1949 which indicated that the late-infection leaf-blight phase of this disease might develop to dangerous levels following winter indexes that were lower than those required for severe outbreaks of the early or wilt phase of the disease (32). While an index of 100 would eliminate the possibility of a severe wilt epidemic, an index of 85 was required for dent corn to escape severe attacks of leaf blight. Boewe published remarkably accurate annual forecasts for leaf-blight infection in Illinois (33-41). There is, then, ample evidence to show that forecasting of severity of the wilt and leaf-blight phases is a simple and accurate warning procedure. Local forecasts are of value in predicting infection levels following flea-beetle emergence in spring, enabling the grower to apply suitable vector-control measures. Factors other than winter temperatures may influence the severity of the disease in some areas. Biraghi reported that, in four seasons (1945-1949), the winter indices in Italy exceeded 100 at two locations, but that the disease was severe only in 1946 and 1947 (31). Since the primary vector, *C. pulicaria*, is not known to occur in Italy, other

vectors may harbor *E. stewartii*, and these vectors may respond to winter temperatures in a dissimilar way.

Stewart's bacterial wilt is still an important and destructive disease of susceptible sweet-corn varieties and hybrids, although such attacks are sporadic and isolated. The leaf-blight phase of the disease is a serious problem in field corn, since many susceptible dent-corn hybrids are grown in the United States and abroad. Genetic investigations of the host and the pathogen have led to spectacular successes in controlling wilt by selecting and breeding resistant sweet-corn hybrids. Studies of the insect vectors and experimental disease forecasting have also made significant contributions to our knowledge of the disease and its control.

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