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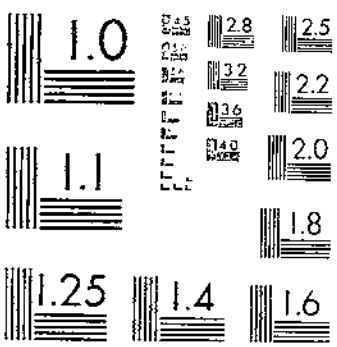
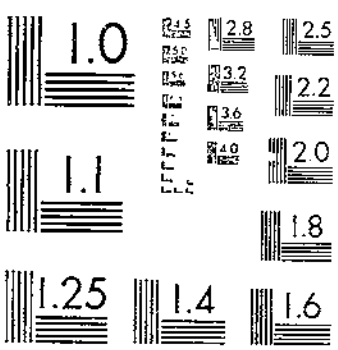


MICROCOPY RESOLUTION TEST CHART
NATIONAL BUREAU OF STANDARDS 1963-A



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BACTERIAL WILT OF CORN

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INTRODUCTION AND HISTORY

The first article dealing definitely with bacterial wilt or Stewart's disease of corn is that by Stewart (*19*),[3] published in December, 1897, and reprinted the following year. The author stated that he had had this disease under observation for three years in New York, where considerable injury (20 to 100 per cent) had been done to sweet corn as grown in the market gardens of Long Island. Stewart then detailed the external and internal signs of the wilt disease and described his isolation and inoculation tests. Because of the extreme prevalence of corn wilt and the resultant infection of many of the control plants, his inoculations, though giving contributory evidence, were not fully conclusive. However, the invariable presence of countless numbers of yellow bacteria occluding the vascular elements of diseased plants gave strong evidence of a causal relation. He believed the evidence good that dissemination is chiefly through the seed and suggested stable manure and farm implements as other probable carriers of infective material. In addition to reporting from Long Island,

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[2] The study herein reported was carried out under the general direction of the late Erwin F. Smith, formerly in charge of the Laboratory of Plant Pathology. The writers desire to acknowledge the cooperation of the West Virginia Agricultural Experiment Station, particularly for furnishing land at Morgantown for experimental purposes; the Maine Agricultural Experiment Station, particularly for supplying Maine-grown seed; H. W. Gerry and A. E. Hitchcock for assisting in taking many of the field notes near Washington, D. C., and James F. Brewer for preparing the illustrations.

[3] Italic numbers in parentheses refer to Literature Cited, p. 29.

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Stewart reported the disease from New Jersey and Iowa, in the latter case the evidence coming from New York-grown plants from Iowa seed. The germ itself he briefly described, but, without ready access to the literature, he did not name it.

In the early part of 1898, however, a culture was sent to Erwin F. Smith, of the United States Department of Agriculture, with the request that he work it over and name the organism. The early results of this cultural study were presented by Smith to the American Association for the Advancement of Science, in August, 1898, and published (*13*) in the annual report the following December. With some exceptions, he found the organism to possess the characters assigned to it by Stewart, and he added several others to distinguish it further. He described the organism, naming it *Pseudomonas stewarti*, gave its growth characters on various culture media, and added the further observation that he had seen the disease in two fields in the southwestern lake region of Michigan on flat land in dent corn sown late for fodder.

Halsted in 1899 reported (*3*) that among several varieties of corn grown together at the New Jersey Agricultural Experiment Station, only one, First-of-All, showed unmistakable signs of the bacterial disease caused by *Pseudomonas stewarti*.

Two years later Smith published a fuller account (*14*) of the cultural characters of the causal organism and in March, 1903, the "completed proof that *Pseudomonas stewarti* is the cause of the sweet-corn disease of Long Island" (*15*). In the summer of 1902 he had visited Long Island and obtained, first hand, pure cultures of the causal organism, with which he inoculated 500 sweet-corn seedlings, partly by way of the fluid oozing from water pores at the tips of the leaves and partly by spraying the plants with water suspensions of the bacteria. Both methods gave typical infections, more than 300 cases of the disease being obtained. This experiment, conducted in Washington, D. C., where corn wilt had not been previously found, gave the first fully conclusive proof of the pathogenicity of this organism and at the same time showed that wounds are not necessary for infection. The vascular system was shown to be the primary seat of the disease, though small cavities filled with the yellow bacteria finally appear in the parenchyma.

In 1909 Smith published (*16*) a brief account of experiments showing the dissemination of wilt through infected seed corn, and of tests of seed disinfection with mercuric chloride seeming to indicate benefit from the treatment. Attempts to isolate the organism from the surface of seed from the same lot were unsuccessful.

Smith's monographic account of corn wilt appeared in 1914 in his work, Bacteria in Relation to Plant Diseases (*17*, *v. 3, p. 89–147*). Here he gave the history and previous literature of the malady and recorded the large volume of information resulting from his own observation and experimentation over a long period. To this work and to Smith's textbook on bacterial diseases of plants (*18*, *p. 160–176*) the reader is referred for detailed descriptions of the parasite and its behavior in culture. Some other phases of his studies will be referred to in connection with the account of the present investigation.

Several other papers referring to corn wilt have also appeared. Garman (*2*) called attention to the disease in Kentucky as occurring in 1916 in Golden Bantam corn and suggested the probability of dissemination through seed, since corn had not been grown in that field for at least 15 years.

McCulloch (*4*) in 1918 gave an account of studies of 14 isolations obtained from several States and in each case tested by successful inoculation and reisolation. In no case was true motility found, and all of the various flagella stains tested gave negative results. The name of the organism was accordingly changed to *Aplanobacter stewarti* (E. F. Smith) McCulloch.[4] Two distinct types of colony were also reported, one with a smooth surface, the other with a definite central depression or crater. Each strain continued true to type even after reisolation; otherwise in morphology and biology the two types appeared to be identical.

During the winter of 1919–20 Reddy (*9*) conducted inoculation tests, using water suspensions of bacteria from pure cultures injected into the stem bases of potted seedlings of dent, flint, and sweet corn (varieties not stated). The two series of 200 plants each gave 90 per cent of infection, and under the conditions of the test the organism appeared to be about equally infective to the three hosts used. As to intensity of infection, flint seemed most susceptible and dent least. There were no cases of natural infection from soil which immediately before had produced an artificially infected crop.

In 1924 Thomas (*21*) reported wilt as particularly destructive in Ohio on early varieties grown for the roasting-ear market, but less so on the midseason or late sorts grown for canning purposes. He gives field and greenhouse evidence of transmission through seed but little if any of transmission through the soil. Considerable experimental data are included relative to varietal differences in susceptibility, and the statement is made that all samples of northern-grown seed tested have been free from infection.

In 1925 St. John-Brooks, Nain, and Rhodes (*12*) reported a serological study as showing that the wilt organism does not agglutinate with serum of seven other yellow-pigmented plant pathogenes, viz. *Bacterium campestre*, *Bact. malvacearum*, *Bact. pruni*, *Bact. gummisudans*, *Bact. phaseoli*, *Bact. phaseoli* var. *sojense*, and *Bact. pelargoni*, tests in all cases being made with 1:100 dilution of high titer serum.

In 1926 Reddy, Holbert, and Erwin (*11, p. 779*) reported that no practical control of bacterial blight of sweet corn was obtained by treating the seed. When nearly disease-free seed was used, the field stands and yields were affected but little by seed treatments with organic mercury compounds.

In 1928 Reddy and Holbert (*10*) reported experimental data as showing wide differences in reaction to inoculations with *Aplanobacter stewarti* in a number of inbred lines of yellow dent corn maturing in the same length of time. All progenies of some inbred lines were uniformly high in resistance, other inbred lines were less resistant, and still others were uniformly susceptible. These results

[4] Synonymy: *Pseudomonas stewarti* E. F. Smith; *Bacterium stewarti* E. F. Smith; *Phytomonas stewarti* (E. F. Smith) Bergey et al.

were believed to suggest the possibility of developing resistance to bacterial wilt in some of the popular wilt-susceptible varieties of sweet corn. No apparent correlation was found between wilt resistance and vegetative vigor or other important diseases of corn.

Preliminary reports of the present investigation (*6*, *7*, *8*), begun in 1918 and continued for seven years, have already been published. This series of studies was undertaken because of a lack of information in regard to the means of overwintering and summer dissemination of the disease, the relation of soil and weather conditions to its presence and spread, and the whole question of varietal differences in resistance and methods of control. This report summarizes the data obtained on these subjects.

GEOGRAPHIC DISTRIBUTION

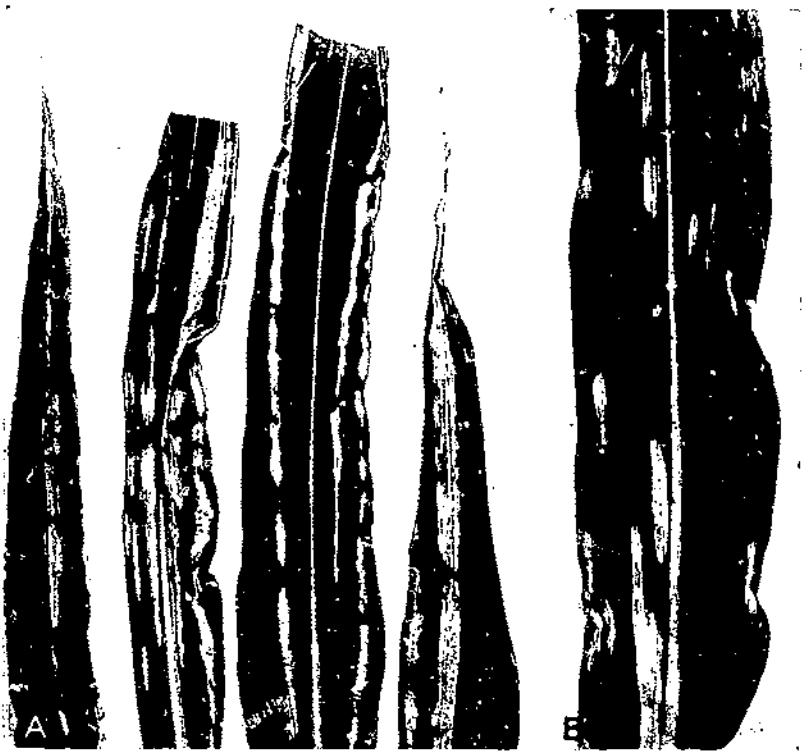
The writers have seen the disease and isolated the causal organism in specimens from the District of Columbia, Georgia, Illinois, Indiana, Kentucky, Maryland, New Jersey, New York (including Long Island), Ohio, Virginia, West Virginia, South Carolina, and Tennessee, and in addition they have observed the disease in Connecticut, Missouri, Iowa, and Pennsylvania. In all cases typical signs of the disease were seen, including always the sticky yellow bacterial slime oozing from the cut ends of vascular bundles.

Definite published reports by others on geographical distribution of wilt include those by Stewart (*19*) from Long Island; Halsted (*3*) from New Jersey; Smith (*17*) from the District of Columbia, Long Island, Maryland, southern Michigan, Ohio, Virginia, and West Virginia; Garman (*2*) from Kentucky; Thomas (*21*) from Ohio; Tehon (*20*) from Illinois; Tucker (*22*) from Puerto Rico; Gardner (*1*) from Indiana; and Martin (*5*) from New Jersey.

Reports of the disease to the Division of Mycology and Disease Survey of the Bureau of Plant Industry, Department of Agriculture, by many observers, from 1910 to 1932, include records from the following States: Arkansas, California, Delaware, Georgia, Illinois, Indiana, Iowa, Kansas, Kentucky, Maryland, Massachusetts, Michigan, Mississippi, Missouri, New Jersey, New Mexico, New York, North Dakota (rare), Ohio, Oklahoma, Pennsylvania, South Dakota (very minor), Tennessee, Texas, Virginia, West Virginia, and Wisconsin (very minor), and the Territory of Puerto Rico.

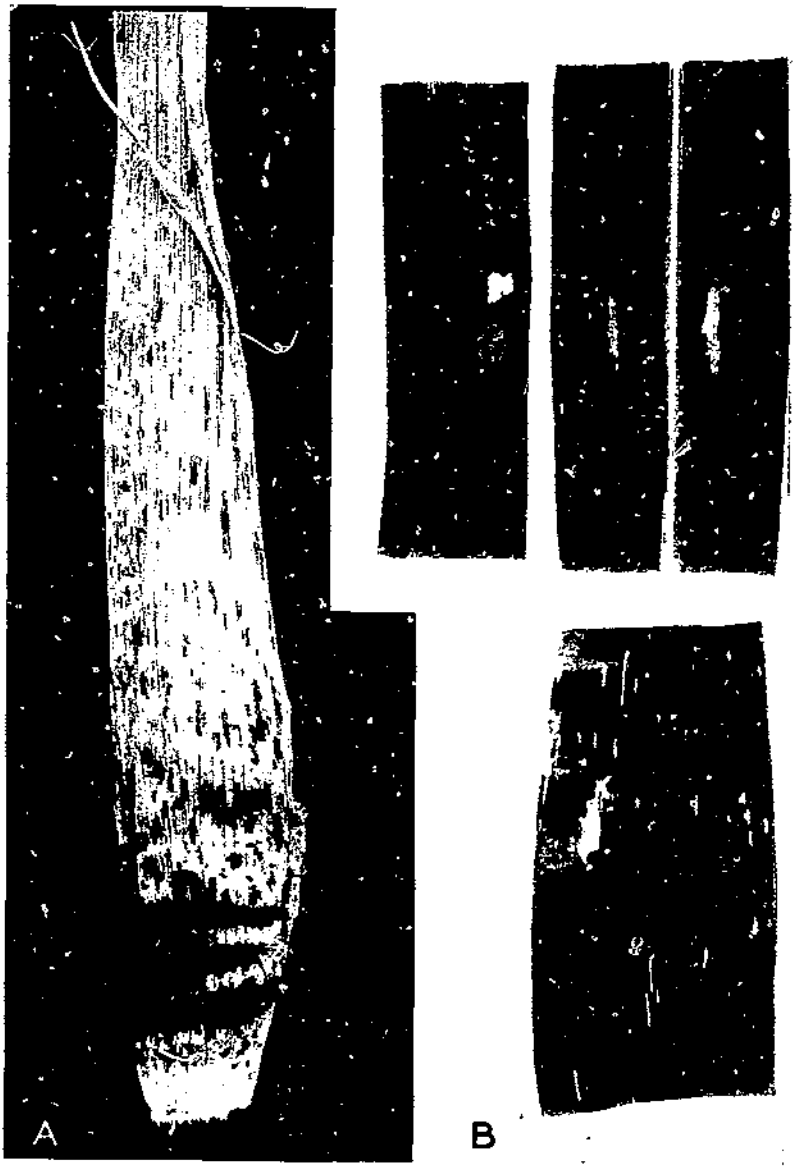
Further reports are as follows: Under date of July 17, 1918, E. H. Smith wrote as to California: "I have never found but one case of bacterial wilt of corn." In correspondence of July 17, 1918, Fred C. Werkenthin said: "Relative to the corn wilt caused by *Bacterium stewarti*, I wish to say that I have written to District Agent J. G. Hamilton of Albuquerque, N. Mex., in whose county I located this corn wilt last year."

For several years the present writers sought diligently for the disease in the Canadian border States of North Dakota, Minnesota, Wisconsin, Michigan, New York (northern portion), Vermont, New Hampshire, and Maine, but never found a single case, except a few affected Golden Bantam plants at Albany, N. Y., which marks the northernmost locality where they found this disease. The evidence at hand therefore seems to indicate that the disease is primarily



A, Typical light-green flabby streaks on corn leaves in early stages of bacterial wilt disease. B, Fungous leaf spot of corn; not to be confused with wilt disease

A, General diffuse type of wilt disease. B, Comparison of normal and wilt-diseased plants of the same age in adjoining hills. C, Normal and wilt-diseased corn plants from the same hill, at maturity



A, Inner husk from wilt-diseased corn plant, showing bacterial slime oozing from the stomata. B, Corn leaves showing incipient wilt disease originating at injuries by flea beetles

one of the Middle and Southern States and that it rarely causes appreciable damage in the northern tier of States.

SIGNS OF THE DISEASE; HOSTS; LOSSES

This specific infectious bacterial disease the writers have found under natural field conditions affecting varieties of the sweet, pop, and field corn types. It is, however, in the earlier sorts of sweet and flint corn that the inroads have been greatest, frequently amounting to the loss of practically the entire crop in seasons when, and in areas where, the disease is prevalent.

Usually the first signs to appear are long, wilted streaks of a lighter green than the adjoining healthy parts of the leaf blade, but without conspicuous yellowing. (Pl. 1, A.) On the other hand, if the progress of the disease is rapid, and especially if accompanied by dry weather, one or more leaves may turn lighter green and wilt as a whole. This diffuse type of wilting must not, however, be confused with the wilting of a tuft of the innermost leaves such as often results from insect attack at the heart of the bud; and, again, the wilted streaks should be distinguished from certain rather elongated fungous leaf spots (pl. 1, B) which frequently are first seen as spindle-shaped areas with a water-soaked appearance at the center. In bacterial wilt the external signs progress from the point of entry of the parasite, which is ordinarily the lower part of the plant when infection is from the seed or near the base of the stalk during the early stages of growth. The disease then spreads until the whole plant becomes wilted, withered, and finally dead. (Pl. 2.) Even in the dead plant some traces of the green leaf color usually remain; conspicuous yellowing never results. In some cases the tassels develop prematurely and become withered and dead before the plant as a whole has succumbed.

The most critical determining sign, however, consists in the oozing to the surface from freshly cut vascular elements of conspicuous yellowish beads of sticky bacterial slime. These bacterial masses not only occur in stems but often may be seen easily even at the cut ends of leaf veins. This slime has not been observed to ooze to the uncut surface of the plant except through the stomata in the epidermis of the inner husks of badly diseased plants. (Pl. 3, A.) In such advanced cases the vascular elements of stem and leaf are packed full of these yellow bacterial masses, which are often found also in the tissues toward the base of the kernel.

Corn wilt is primarily a disease of the vascular system, but in its later stages the parenchyma tissues are also invaded. In fact, the bacteria have been found in most parts of the plant, including roots and stems, leaf blades and sheaths, tassels, husks, cobs, and kernels.

TRANSMISSION THROUGH SEED

Many years ago the transmission of bacterial wilt of corn through the seed was shown by Smith (*17, p. 114–120*) to occur, on the basis both of strong circumstantial evidence from plantings of seed of suspected or known derivation from wilt-diseased plants, and of microscopic demonstration of the bacteria from the vascular system

of stem and cob up into the basal region of kernels from plants known to be diseased. Because of the copious oozing of the bacterial slime to both surfaces of the husks, he suspected much of the transmission through seed to be from the surface contamination of the kernels, but failed in all attempts at direct isolation of the organism from this source. He records no attempts at isolation from the interior of the kernels, but notes the probability that infection of seedlings may come from bacteria within as well as on the surface of the kernels. The present writers have also failed in repeated attempts to isolate the organism from the seed surface; but many times during the course of the investigation they have made successful isolations from the endosperm of surface-sterilized (1:1,000 mercuric chloride) kernels, and with these pure cultures they have obtained typical infection in corn seedlings, followed by reisolation of *Aplanobacter stewarti*. Some of these isolations were made from kernels on ears collected from diseased plants in the fall and held in a cornerib until early spring.

Besides the direct isolations from seed, a considerable number of plantings were made during four years, with seed collected by the writers from wilt-diseased and healthy plants, with seed from the open market, and with seed grown in Maine and Michigan localities where the disease had not been known to occur; and careful records were made of the progeny during growth and at maturity. Brief summaries of these tests follow.

In the greenhouse, where the factor of dissemination by insects did not enter, 23 progenies of seed from a badly diseased ear, sown during the following winter in pots, gave 3 wilt-infected plants (13 per cent). In a second test, 54 progenies of seed from several badly infected ears, similarly sown, yielded 1 infected plant (2 per cent). In a third greenhouse test, 2 quarts of the worst-looking seed selected from a peck of the same lot of seed used in a large commercial planting in Maryland, from which the preceding season's crop had been almost a total failure due to bacterial wilt, was sown in a large greenhouse bed. Among the progenies of the entire lot, only 2 per cent gave definite evidence of the wilt disease. Among hundreds of plants from open-market seed grown in the same greenhouse during these tests not a single case of wilt occurred except those resulting from direct inoculation by pure cultures of the causal organism.

In larger field plantings, near Washington, D. C., where the factor of dissemination by insects was not controlled, five tests with seed collected by the writers from healthy and from wilt-infected plants gave an average of 43 and 56 per cent of wilt-infected plants, respectively. In two tests, open-market seed gave an average of 3 per cent, and seed from diseased plants 6 per cent of wilt cases.

It was reasoned that if seed were an important source of field infections, the planting of seed grown in wilt-free localities or the use of seed 2 to 3 years old ought to reduce materially the number of wilt cases. Field sowings were accordingly made, also near Washington, D. C. In one test, open-market seed gave 72 per cent, seed from Maine and Michigan 83 per cent, and seed from infected plants 78 per cent of wilt cases. In three tests, open-market seed gave an average of 48 per cent, and Maine and Michigan seed 54 per cent of infected plants. In 10 further tests in which seed 1, 2, and 3

years old was planted at the same time under similar conditions as in all these tests, the averages for wilt-infected progenies were 17, 24, and 18 per cent, respectively.

In summarizing the foregoing, together with unpublished data from detailed notes, it may be said that *Aplanobacter stewarti* has been isolated directly from the interior of seed grown on wilt-diseased plants as long as five months after the seed was harvested. No tests for longer periods have been made. The planting of seed known to be wilt-diseased in the greenhouse has given 2 to 13 per cent of wilt cases when there was no wilt in the controls; in both these instances the factor of dissemination by insects was eliminated. Progenies of open-market seed and of seed from known healthy and wilt-diseased plants grown in the field in localities where wilt was prevalent have varied widely in individual cases in amount of wilt. However, where wilt-free and wilt-diseased seed were of the same strain and under conditions where dissemination by insects was not too great a factor, differences in percentages of wilt were sometimes very considerable. Even with the insect factor at its height, averages of considerable numbers of seed lots of the same varieties have usually given a higher percentage of wilt in the progeny of wilt-diseased than of healthy plants. Again, where the insect factor was not too prominent, the numbers of early cases and of bad cases of wilt at maturity were usually greater in progenies of wilt-diseased plants.

In spite of the clear demonstration of transmission of bacterial wilt of corn through the seed, in sections where wilt was prevalent the disease occurred in startling amounts regardless of that factor, given only that susceptible varieties were planted. For example, in field plantings in Maryland, progenies of single lots of Maryland-grown seed from healthy plants have given percentages of wilt as high as the following: Golden Bantam, 71 per cent; First-of-All, 59 per cent; Premo, 40 per cent; and Peep-o'-Day and Golden Cream, each 27 per cent. Under similar conditions, Golden Bantam seed from Maine or Michigan sections where wilt was unknown have given as high as 92 and 86 per cent, respectively, and other Maine-grown varieties have given as high as 96 per cent of wilt, when planted in Maryland. In fact, Maine and Michigan seed in general, when planted in a locality where wilt was prevalent, have given more of the disease than seed of the same varieties grown in more southern localities. This is interpreted to mean that when corn is grown in sections where wilt does not occur there is no elimination of the more susceptible strains by actual killing of the plants, such as occurs where the disease is prevalent.

Furthermore, in large field plantings of seed 1, 2, and 3 years old, the average percentages of wilt did not vary widely for progenies of the three seed groups when planted contiguously at the same time. However, individual lots of seed planted at different dates during the same season or in different seasons often showed considerable variations in amount of wilt.

Field plantings in Maryland of 470 lots of seed of two representative early and two late sorts of sweet corn obtained from seedsmen in nearly all sections of the United States gave an average of 48 per cent of wilt for the early and 5 per cent for the late sorts.

These facts substantiate the statement that transmission by seed is a serious factor, probably the only factor, in introducing bacterial wilt of corn into a new locality; and inroads from this source alone may be considerable. It is clear, however, that once the disease becomes prevalent in a locality, the planting of resistant or susceptible varieties or strains is far more important in determining the ultimate damage to the crop from wilt than is the origin of the seed from healthy or wilt-diseased plants. These facts do not in any way minimize the danger from transmission through seed, but they do serve to emphasize the greater danger, under certain conditions, from the use of susceptible varieties.

DISINFECTION OF SEED

At the beginning of the investigation, when it was believed that a considerable proportion of seed infection originated in surface smearing with bacteria from the copious exudations through the stomata of the inner husks, several series of experiments were run to test the effects of disinfectants and dry heat on sweet-corn seed sterilized for three hours in a dry oven at 150° C., cooled, and immersed in a suspension of a three to four day agar culture of *Aplanobacter stewarti* in sterile tap water, and held one to four days before the tests.

In this way it was found that treatment with copper sulphate (1:100) or with commercial formaldehyde solution (1:320) for 15 minutes, or with the latter for 3 minutes followed by 2 hours in a formaldehyde-saturated atmosphere, was effective in destroying all the bacteria adhering to the seed.

Several trials were made in which seed treated with the bacterial suspension were submitted to dry heat at 60° C. for one to three hours, both with and without being presoaked in sterile tap water. None of the seed thus treated gave any bacterial growth when placed on sterile agar. The inoculated controls gave 88 to 100 per cent of bacterial growth when placed on sterile agar.

Since under natural conditions infection through the seed seems to come largely if not wholly through bacteria carried within the seed, it appeared problematical how far externally applied disinfectants might influence infections from this source. However, it was thought possible that if seed could be heated at a temperature high enough to destroy the bacteria without seriously impeding germination, such treatment might prove a practicable method of controlling seed transmission of the disease. The thermal death point of *Aplanobacter stewarti* being around 53° C., preliminary germination tests were carried out with seed of different sweet-corn varieties heated for different periods at 60° to 120° C. As a result it was found that germination was not seriously lowered by three hours' dry heat at 60°, 70°, or sometimes even at 80°.

A large seed-disinfectant test was then carried out in the field, with the use of seed collected the preceding fall from healthy and from wilt-diseased plants grown in the same locality. The dry-heat applications were made for 3 hours at 70° C. and for 3 and 6 hours at 80°, and the seed was planted the following day. Other lots of seed were soaked for 3 minutes in a 1:320 formaldehyde solution,

subjected to the fumes of formaldehyde for 3 hours, and planted within 2 to 3 hours afterwards. In all, 175 lots of 100 seeds each were used in this experiment.

Careful study of the detailed results showed high variations in total percentages of wilt in comparable groups, sometimes higher in the untreated, sometimes higher in the treated, sometimes lower and sometimes higher in the healthy as compared with the wilt-diseased seed controls. Germination in these field plantings was seriously lowered by both treatments at 80° C.; that of the seeds subjected to other treatments was practically identical with that of the controls.

The wide fluctuations in percentages of wilt due to extraneous infections make it impossible to draw final conclusions. No great variations in average percentages of wilt in controls as compared with treated seed were evident. However, the heat treatments consistently gave slightly less wilt, this difference being greater the larger the number of seed lots on which the averages were based. Another field test with the use of 37 lots of 100 seeds each gave essentially similar results.

Smith (*17, p. 125–127*) carried out a test in the greenhouse using (1) 1:1,000 mercuric chloride solution for 15 minutes on hand-picked good-looking seed from a sack of seed strongly suspected of harboring the wilt organism, which gave 1.2 per cent of wilt in the progeny; (2) the same solution for 10 minutes on seed similarly selected, which gave 1.8 per cent of wilt; (3) unselected, untreated seed as it came from the sack, which gave 9 per cent of wilt; and (4) selected bad seed from the same sack, untreated, which gave 9.3 per cent of wilt. These results might seem to favor the treatment, were it not that the treated seed was the selected, best-looking seed from the sack, while the untreated seed was unselected or bad seed. Reddy, Holbert, and Erwin (*11*) found no evidence of control of bacterial wilt of corn by treating seed corn with 0.25 per cent solutions of chlorophol.

In correlating the results of the studies on disinfection of seed and on transmission through the seed, it appears that in wilt-prevalent localities transmission through seed is responsible for a much smaller proportion of the field infections than was formerly supposed, while dissemination during the growth period of the plants often reaches a percentage so high as to nearly or quite mask the effects of transmission through seed, even in the early cases. Because of the small amount of transmission through the seed and the more or less sporadic distribution of the preponderant summer dissemination, the effects of using treated rather than untreated wilt-diseased seed could scarcely become evident except where the results of large numbers of tests were averaged, or under conditions eliminating most of the secondary infections.

For practical purposes, then, seed disinfection would be of little value, even though effective, except for possible use in sterilizing seed to prevent the introduction of the disease into new localities; in such cases the treatment would need to be 100 per cent effective. Because the organisms are carried within the seed, externally applied disinfectants would probably be of little value for this purpose; the experimental results, so far as they go, bear out this idea. Heat treatment at temperatures well above the thermal death point

(53° C.) of *Aplanobacter stewarti*, reaching all parts of the seed without materially reducing germination or vigor of the plant, would seem to offer an effective method of eliminating the organism from the seed where it is deemed advisable to make the attempt. The writers' experiments offer distinct encouragement in this respect, but need further confirmation under many widely differing conditions and the adaptation of the method to a commercial scale.

TRANSMISSION THROUGH SOIL

Since there was no conclusive information in the literature concerning the possibility of transmission of bacterial wilt of corn through the soil, the problem was approached from a number of different angles, in both greenhouse and field.

Of the field tests, five (in which about 1,400 plants and at least an equal number of uninoculated controls were used) were carried out by inoculating the soil with cut pieces of heavily infected corn stems and planting the seed among them at once. The final averages for wilt cases were 41 per cent for the inoculated soil and 38 per cent for the uninoculated controls. In these as in succeeding tests, the inocula were tested simultaneously for virulence, by successful infection of other corn seedlings. The needle-puncture method was used, and soil that had not been in corn for at least two seasons. Two similar tests (involving about 1,600 plants, with adequate controls) were carried out by burying the infected material in the fall and planting the seed the following spring. The averages were 24 per cent for the inoculated and 26 per cent for the uninoculated soil. In two other tests (on 183 plants and an equal number of controls) the soil was inoculated with a heavy tap-water suspension of the bacteria, and the corn was planted at once; averages for wilt were 70 per cent for the inoculated and 62 per cent for the uninoculated soil. In one further test (on 80 plants and an adequate number of controls) the seed rather than the soil was soaked in the bacterial suspension before planting; the averages for wilt were 74 per cent for the inoculated and 64 per cent for the uninoculated seed.

Since in these experiments insects were not controlled, two field tests (on 17 plants) were made with cloth-covered cages. Either the soil or the seed was inoculated and the seed planted at once. No wilt resulted.

It was thought that a comparison of the relative numbers of slight, medium, and bad cases of wilt to healthy plants in individual hills might possibly shed some light on the mode of infection. Accordingly, 1,865 hills of sweet corn each containing 2 to 4 plants to a hill (average 3) in a field that had not been in corn for two years were used as a basis for counts at maturity of all the hill combinations of wilt-diseased and healthy plants. Since all the bad cases of wilt first appeared early in the season, most if not all of the seed infection (if any) would be included in this group. Furthermore, if more slight or medium cases occurred in hills with bad cases than with other combinations, it would tend to indicate some relation between the two. The counts showed the following to be the most frequent hill combinations, and with the same proportion of the total number of hills (about 20 per cent) represented in each:

Healthy alone, healthy and bad wilt, and healthy and slight wilt. Hills with bad wilt alone (10 per cent of total hills), and with healthy, slight, and bad, or with slight and bad wilt (8 per cent each), are the only other significant hill combinations. Apparently there is no clear-cut tendency for late cases to follow early cases in individual hills; and, furthermore, nearly half of 310 other hills containing only 1 plant to the hill had late-appearing cases of wilt. It seems very probable, therefore, from these data that most if not all of these late cases had become infected from sources extraneous to seed or soil.

The factor of dissemination by insects was uncontrolled in most of the field tests just summarized. Accordingly, 20 more tests were run at various times in greenhouse pots and beds, on an aggregate of at least 2,500 plants subjected to inoculation of the soil or outside of the seed in the various ways outlined for the field tests, and in each case accompanied by adequate controls.

In all these inoculation experiments, under controlled conditions and with the insect factor precluded, no single case of wilt developed except in a few plants subjected to severe root pruning, followed by immediate inoculation of the soil in contact with the cut roots. In the latter cases inoculation was, of course, merely one form of direct inoculation of the plants.

In the field tests the influences of differing soil conditions and of dissemination by insects complicated the results. However, in the one test where two distinct soil types were definitely separated in the field and thus capable of correlation, the higher percentages of wilt clearly occurred in the good rather than in the poor soil type, instead of in the inoculated rather than in the control soil; and where inoculated and uninoculated soils were both good or both poor, the percentage of wilt was about the same in both. There were many variations among the different lots of seed in a test; sometimes there was more wilt in the inoculated plots, sometimes in the controls. However, the average percentages of wilt in 7 of the 10 tests were approximately the same in both inoculated and control plots.

A correlation of the results of all of these experiments, with due regard to the conditions under which they were carried out, surely gives no clear evidence of any true transmission of infection through the soil, but, on the other hand, does give much definite evidence against it. The data at hand are interpreted to mean that overwintering of *Aplanobacter stewarti* in the soil, while it may conceivably take place in rare instances, is not a contingency likely to occur or to be of practical significance.

VARIETAL DIFFERENCES IN SUSCEPTIBILITY

Fortunately, not all varieties of corn are susceptible to bacterial wilt. Extensive varietal tests during four seasons, comprising in each season several (3 to 10) acres of land, together with varietal inoculations in the greenhouse, have shown conclusively that there now exist a considerable number of varieties highly resistant to the disease.

In the writers' first experimental field planting of about 7,000 hills near Washington, D. C., only 10 cases of wilt were found throughout

the season. The field was separated from other cornfields by woods; the soil was a very light, sandy loam; the date of planting was preceded by several weeks without rain; and there were no commercial plantings of early sweet corn in the locality.

TABLE 1.—*Table varieties of corn arranged in the order of increasing percentage of wilt from natural infection in field plantings, 1919 and 1920*

Variety	Number of tests	Percentage of wilt	
		Range	Average
Zigzag Evergreen	2	0	0
Old Colony (1919 only)	2	0– 2	1
Stoke's Double-Barreled Best (1919 only)	2	0– 4	2
Narrow-Grained Evergreen	4	0– 8	2.3
Ideal Early	6	0– 14	3
Country Gentleman	10	0– 10	3.4
Maine Style Evergreen	2	2– 5	3.5
Stowell Evergreen	14	2– 19	4
Early Evergreen	10	0– 17	4.1
Adams' Extra Early (1919 only)	14	0– 10	4.2
Black Mexican	10	0– 25	4.6
Late Mammoth	9	0– 10	4.8
Ne Plus Ultra	4	0– 15	5.3
Perry's Hybrid (1919 only)	3	4– 7	6
Improved Giant	4	0– 10	6
Bantam Evergreen	8	0– 6	6.3
White Evergreen	2	6– 9	8
Nonesuch	3	0– 18	8.1
Long Island Beauty	3	3– 2	6.7
Early Sheffield (1919 only)	2	9– 11	10
Stabler's Early	4	3– 21	10.5
Black Sugar	2	0– 25	12.3
Bear's Foot	4	4– 34	3.6
Metropolitan	3	4– 43	14.8
Hickox	6	2– 24	15
New Early Malakoff	5	5– 33	15.5
Kendall's Early Giant	14	5– 49	16.9
Early Mammoth	5	0– 55	16.9
Improved Shoe Peg	2	8– 30	19
Golden Rod	4	12– 33	19.6
White Cory	4	4– 32	22
Howling Mob	8	3– 48	22.4
Colossal	6	4– 65	23
New Champion	3	19– 32	23.3
Moore's Early Concord	3	7– 41	25.8
Early Crosby	9	5– 70	26
Moore's Concord	4	18– 43	26.6
Premo	14	15– 59	27.3
Maule's XX	6	10– 52	29.3
Early Champion	12	22– 57	30
Early Minnesota	11	0– 53	30.1
Golden Cream	9	2– 59	30.1
Mammoth White Cory	12	4.5–100	30.2
New Extra Early White Cory	4	0.5– 68	33
Golden Bantam	28	14– 79	34.1
Early Dawn	5	22– 55	35.5
Cosmopolitan	3	32– 41	36.1
Peep-o'-Day	11	19–100	37
Mayflower	7	18– 63	40.2
Extra Early Red Cory	4	28– 65	46
Early Red Cory	3	36– 56	48
Potter's Excelsior	3	33– 60	50.5
Early Cory or First-of-All	8	30–100	57.3

Results of variety plantings of May and July during the following two years, in the same locality, are shown in Table 1. A similar variety planting of the fourth season gave percentages of wilt-diseased plants as shown in Table 2. A supplementary test of two varieties each of early and of late sweet corn, with 15 or more hills to each lot of seed, gave average percentages of wilted plants as follows: Golden Bantam (143 seed lots), 49 per cent; White Cory (36 seed lots), 48 per cent; Stowell Evergreen (142 seed lots), 4.5 per

cent; Country Gentleman (148 seed lots), 5.4 per cent. Seed of these varieties, furnished by the Division of Seed Investigations of the Bureau of Plant Industry, came from seed companies located in

TABLE 2.—*Table varieties of corn arranged in the order of increasing percentage of wilt from natural infection in field plantings, 1921*

Variety	Number of tests	Percentage of wilt	
		Range	Average
Zigzag Evergreen	1		0
Narrow-Grained Evergreen	1		0
Ideal Early	1		0
Country Gentleman	3		0
Stowell Evergreen	5		0
White Evergreen	1		0
Early Evergreen	3		0
Leadall Evergreen [1]	1		0
Improved Giant	1		0
Nonesuch	1		0
Black Sugar	1		0
Hickox	1		0
De Lue's Golden Giant [1]	1		0
New Early Malakoff	3		0
Improved Stowell Evergreen [1]	1		1
Adams' Extra Early	2	0– 1	1.5
Ne Plus Ultra	1		1.5
Moore's Concord [1]	2	0– 3	1.5
Late Mammoth	3	0– 4.6	1.53
Adams' Early [1]	1		1.8
Long Island Beauty	1		1.85
Stabler's Early	1		1.9
Golden Evergreen [1]	1		2
Bantam Evergreen	3	0– 3	2.3
Howling Mob	2	2– 3	2.5
Cosmopolitan	1		3.17
Golden Rod	1		3.7
Black Mexican	6	0– 6	3.8
Golden Cream	3	0– 9	3.86
Maule's XX	3	3– 7	5
Bear's Foot	1		5.5
Colossal	2	0–11	5.5
White Mexican [1]	1		6
Golden Sweet [1]	1		6.7
Metropolitan	3	5– 9	7.5
Mayflower	1		7
Early Minnesota	6	4–15	9.65
Early Champion	3	5–12	9.85
White Cob Cory	1		10
White Cory	1		11
Golden Giant [1]	1		11
Peep-o'-Day	2	7–16	12.5
Premo	5	4–20	13
Potter's Excelsior [1]	1		14.5
Kendall's Early Giant	4	5–48	18
Early June [1]	1		22
Mammoth White Cory	5	10–33	22
New Extra Early White Cory	1		23
Early Dawn	1		23.5
Golden Bantam	7	16–39	24.7
First-of-All	4	16– 3	25.5
Nuetta [1]	1		25
Improved Early Dakota [1]	1		32.4
Early Iowa [1]	1		37.6
Extra Early Red Cory	1		39
Early Mayflower [1]	2	40–19	41
Early Crosby	3	11–83	48

[1] Not in tests of 1919 and 1920; all others in those tests.

all sections of the United States. In all these field plantings wilt was the result of natural infection, and each test (seed lot), unless otherwise stated, represented at least 24 hills of corn.

In a further test in the greenhouse, seedlings of 24 varieties of sweet corn were inoculated by needle punctures in the stem with pure cultures of *Aplanobacter stewarti*. The average percentages

of wilted plants for the different varieties, though of course higher than for the spontaneous infections in the field plantings, showed in general the same relative differences in susceptibility, the earlier sorts giving nearly or quite 100 per cent of infection, the later sorts 0 to 30 per cent, and the midseason sorts percentages between these two extremes.

More limited data published by Smith in 1914 (*17, p. 117–118*) and by Thomas in 1924 (*21*) agree essentially with these tests by the writers as to the relative susceptibility of sweet-corn varieties to the wilt disease.

Similar tests of 14 pop varieties and of 46 field varieties of corn, carried out by the writers during three seasons in field and greenhouse, showed entirely comparable results as to varietal differences in susceptibility. Tests with pop corn were hardly extensive enough for full varietal comparisons, but the Black Beauty and Tom Thumb varieties proved notably susceptible. In the field-corn tests the dent varieties almost invariably exhibited high resistance, while many of the flint varieties were extremely susceptible.

Variations in amount of wilt for any given variety were to be expected in different plantings and of course were realized in these tests, on account of variations in temperature, rainfall, humidity, strains of seed used, dissemination by insects, soil texture, cultural practices, presence or absence of the causal organism in the seed, etc. In view of these facts, there was a surprising correlation in the different variety groups as tested from season to season. It was found as a result of these studies that an arrangement of the sweet-corn varieties according to the length of time required for maturing coincided with surprising exactitude with an arrangement according to the relative percentages of wilt cases occurring spontaneously or from inoculation in the varieties compared. The later varieties such as Zigzag Evergreen, Country Gentleman, and Stowell Evergreen consistently gave a very low percentage of the disease, while the earliest maturing sorts, such as First-of-All and other members of the Early Cory group, Early Mayflower, Golden Bantam, Early Crosby, etc., under the same conditions underwent extreme losses, even to the point of complete destruction of crops and plants. In general, wilt prevalence among midseason varieties was between these two extremes. The Evergreen group as a whole was affected but little; and it is interesting to note that Bantam Evergreen, a cross between Golden Bantam and Stowell Evergreen, appears to carry with it very little of the susceptibility of the Golden Bantam parent. Among the 46 varieties of field corn tested under field conditions, 24 at no time showed spontaneously occurring cases of wilt; a few of the dent varieties have given 5 per cent or less, but the earlier flint sorts proved most susceptible. For example, Jehu has given as high as 100 per cent of infected plants, Square Flint 40 per cent, and Longfellow and King Philip about 20 per cent each.

As to field corn, wilt usually presents no problem, since the more susceptible flint varieties are grown for the most part in northern sections where the disease is rarely in evidence.

It is unfortunate that among the earliest table varieties the only highly resistant sorts found were in the Adams' Early and Ideal Early groups, neither of which are true sweet varieties. There is,

however, an abundance of resistant strains among the later maturing varieties. It seems clear, then, that aside from the possible development of resistant early sorts, the only insurance against serious inroads in a locality favorable to this disease appears to lie in the planting of the already existing later varieties that have proved resistant. The compartively light occurrence of wilt usually experienced in the Middle States is to be explained in part by the fact that resistant varieties such as strains of the Evergreen or Country Gentleman types have been the ones most generally planted. On the other hand, so far as this disease is concerned, the susceptible varieties may be planted along the Canadian border with a reasonable degree of assurance. The writers have been unable to find the disease there, in either garden or commercial plantings, and only rarely has it been reported by others. Large variety tests with identical lots of seed planted during the same season in Maryland and at Orono, Me., with abundance of wilt in Maryland and none at all in Maine, surely tend to confirm the writers' extended observations.

ENVIRONMENTAL RELATIONS

TEMPERATURE AND RAINFALL

In the endeavor to elucidate the relations between weather conditions and incidence and prevalence of wilt, a considerable number of experiments and records were made, as detailed below.

The first experimental plantings were in an isolated field not planted to any crop for many years. The seed used comprised 286 lots of 149 varieties of sweet, pop, and field corn, obtained either from the congressional seed distribution or directly from dealers in 15 States, and planted in approximately 7,000 hills near Washington, D. C. In this as in succeeding tests there were not less than 24 hills to a lot of seed and not less than 3 to 4 plants to a hill. Throughout the season only 10 plants with distinct external signs of bacterial wilt were found. Varieties planted by commercial growers in this vicinity had been entirely of field corn and of late-maturing varieties of sweet corn, and the experimental field was surrounded by shrubbery and woods. The soil in this planting was a very light sandy loam; and reference to Figure 1, A, will show that dry soil conditions prevailed throughout the first part of the 1918 season; in fact, there had been no rain for several weeks prior to planting, at which time the moisture content of the surface soil was very low. Furthermore, the air-temperature readings, based on weekly averages of maximum and minimum temperatures, were comparatively low for the season and locality, and high temperatures did not prevail until after mid-July.

The following season 89 lots of 68 varieties of sweet and pop corn were planted in the same locality in three fields that had not been in corn for many years. In the first planting (May 16), out of a total of 8,716 plants, 17.5 per cent became spontaneously infected. In the second planting (July 14), out of a total of 2,414 plants, 33 per cent were attacked. In the final planting (September 3), out of a total of about 2,500 plants, only 0.2 per cent (5 cases) contracted wilt. The soil used in the first test was a moderately heavy sandy loam. At the time of the first planting the temperature was

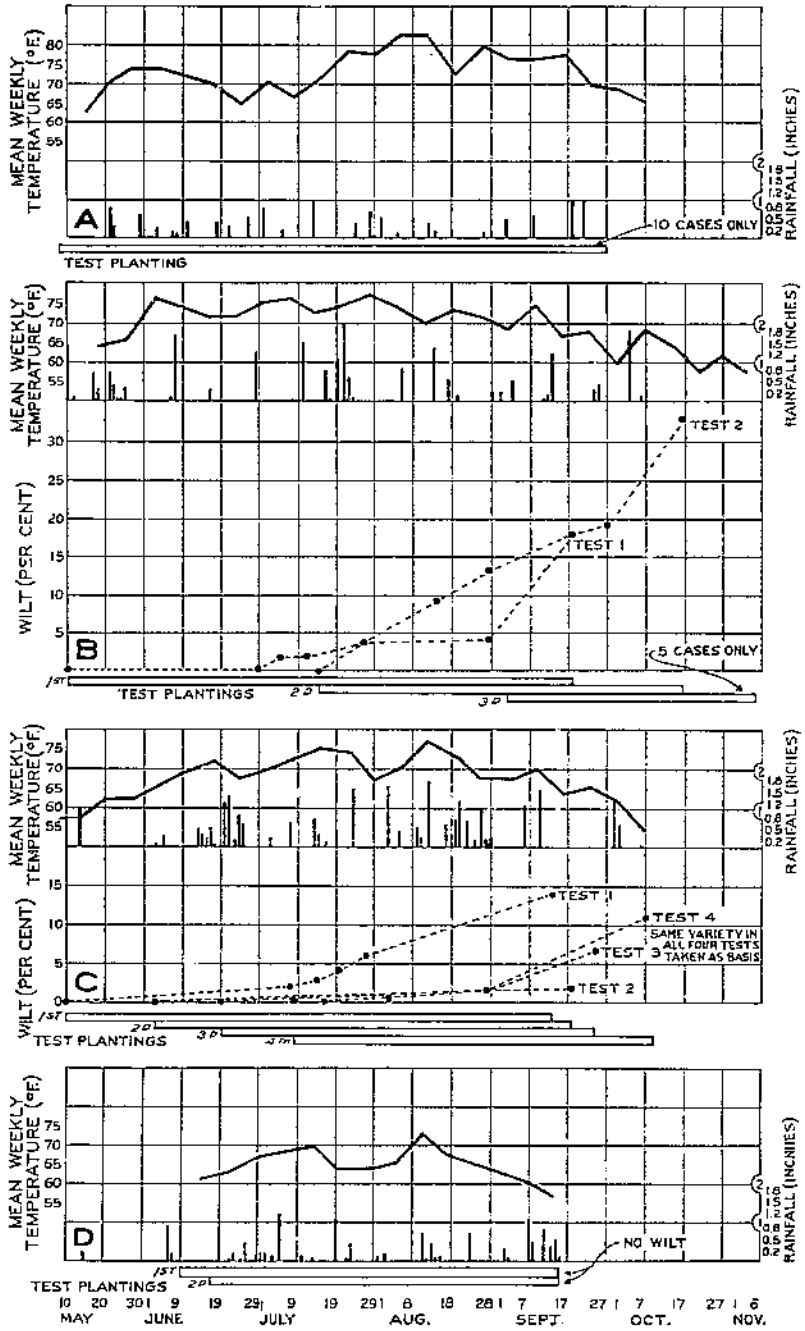


Figure 1.—Rainfall, temperature, and percentage of bacterial wilt in field plantings: A, Tuxedo, Md., 1918; B, Tuxedo, Md., 1919; C, Tuxedo, Md., 1920; D, Orono, Me., 1920

rather cool (fig. 1, B), with a period of rainfall immediately preceding, and with very moist soil conditions prevailing. Here the curve of wilt incidence was very gradual at the start and became steep only during the last three weeks of the growing season.

The second planting was made during a period of heavy rainfall, in similar soil, with very moist soil conditions, and with high temperatures prevailing throughout the first two-thirds of the growing season. In this case the wilt curve was much higher and steep from the first. Furthermore, from the insect relations subsequently discovered, it seems probable that the increased infective momentum of the insect disseminators, attained during the growing period of the second planting, undoubtedly had a part in causing the more rapid rise of the second wilt curve.

The third planting was made in poorer soil, during a period of lighter rainfall and lower soil moisture, cooler nights and gradually lowering mean temperature, with consequent slower growth of the plants, and with insect prevalence lessened. The amount of wilt there was almost negligible.

During the third season the same lots of 42 varieties of sweet and pop corn were planted on May 10, June 2, June 19, and July 8, and gave, respectively, 14.3 per cent (1,541 plants), 2.1 per cent (3,859 plants), 6.5 per cent (2,359 plants), and 11.2 per cent (1,430 plants) of wilt. (Fig. 1, C.)

The first planting was made in fairly good soil with abundant soil moisture, preceded by plentiful rainfall and followed in a few days by a heavy rain, then by several weeks of drought terminated by high rainfall fairly well distributed throughout the remainder of the growing season. The temperature and wilt curves were low at first, but rose fairly rapidly later in the season.

The second planting, made in distinctly poorer soil, came in the midst of several weeks of drought. Temperatures were still rather low at first, but soon rose, and there was plentiful rainfall later on. Here the wilt curve was very low throughout.

The third planting was made in rather better soil after several days of moderate rainfall followed by heavy rains, and with decreasing, followed by rising, temperature. The wilt curve was also very low at first but later rose.

The last planting (on July 8), in fairly good soil and under favorable soil-moisture conditions, followed by unusually abundant rainfall and the highest temperatures of the season, gave a wilt curve approaching that of the first planting.

During the same season two duplicate variety tests were run at Orono, Me., comprising 139 lots of sweet and pop corn seed identical with those planted near Washington, D. C., and including also 54 lots of seed collected from wilt-infected plants the preceding season and planted June 9 and 17, respectively. Neither externally nor internally were there any signs even remotely resembling bacterial wilt in any of the plants at any time during the season. Reference to Figure 1, D (Orono, 1920), shows that there was little rainfall at or near planting time in both tests and that temperatures were comparatively low throughout the season. Furthermore, the seedlings made very slow growth during the early part of the season, seeming

to stand still for weeks, and none of the insects later shown to be instrumental in disseminating the disease were found there.

One more large variety test near Washington, D. C., comprising 117 lots of seed of 57 varieties of sweet corn planted May 10, 1921, gave an average of 9.7 per cent of wilt cases. As shown by Figure 2, A (wilt curve not shown), abundant rainfall occurred about planting time, followed later, however, by many weeks of scant rainfall or drought. Temperatures were comparatively low during the first third of the growing season.

During several succeeding seasons date-of-planting tests were carried out at intervals throughout the growing season. In the first series, with 20 weekly plantings from April 7 to August 25, the maximum wilt prevalence occurred in the plantings of late June and early July, with less disease at the beginning and at the end of the season. There was a 6-week drought at midseason accompanying a part of this higher prevalence of wilt. However, the field notes during the growth period showed that most of the disease had come from infections contracted after the cessation of the drought. The field was a level, sandy loam soil, and all of the eight varieties used were susceptible to wilt. The disease percentages were in general low in the planting of April 7. In plantings made during the remainder of April and throughout May the wilt percentages were variable, with, however, a general tendency toward increasing wilt prevalence. The plantings of June and July were rather uniformly high in wilt prevalence, and this was the period of most uniformly high temperature of the season. The rainfall was scant in June and more plentiful in July, and the highest wilt percentages occurred in plantings made during the period of most copious rainfall and highest temperatures. In the August plantings there was a rapid falling off in wilt prevalence, reaching zero for the planting of August 25. Wilt percentages for each planting (in this and in succeeding date-of-planting tests the wilt curves are plotted on planting dates) and data on temperature and rainfall are shown in Figure 2, A.

In the second series nine plantings of the First-of-All variety were made between April 29 and July 17 in the same field. Each planting was divided into four lots, which were cut at four different dates during the last month of the growing period, the last cutting being at maturity, and therefore taken as giving the total wilt of the season. In each planting there was a general tendency toward increase in number of wilt cases at each successive cutting, even though the first cutting took place about 50 days after the date of planting of the first 5 plantings and about 40 days after planting of the last 4. This would indicate that progressive infections occurred during each growth period; and data regarding wilt cases showing yellow ooze in the vascular elements of the leaves only (incipient wilt), all of which were recent cases contracted from dissemination by insects, would show the largest percentages of infections to have taken place during midsummer, after the insect disseminators had attained their maximum infective momentum for the season. The mean weekly temperature at the first planting (fig. 2, B) was 65° F., but rapidly became higher. There had been no rain for 15 days preceding this planting, but the soil was still very moist, and five days

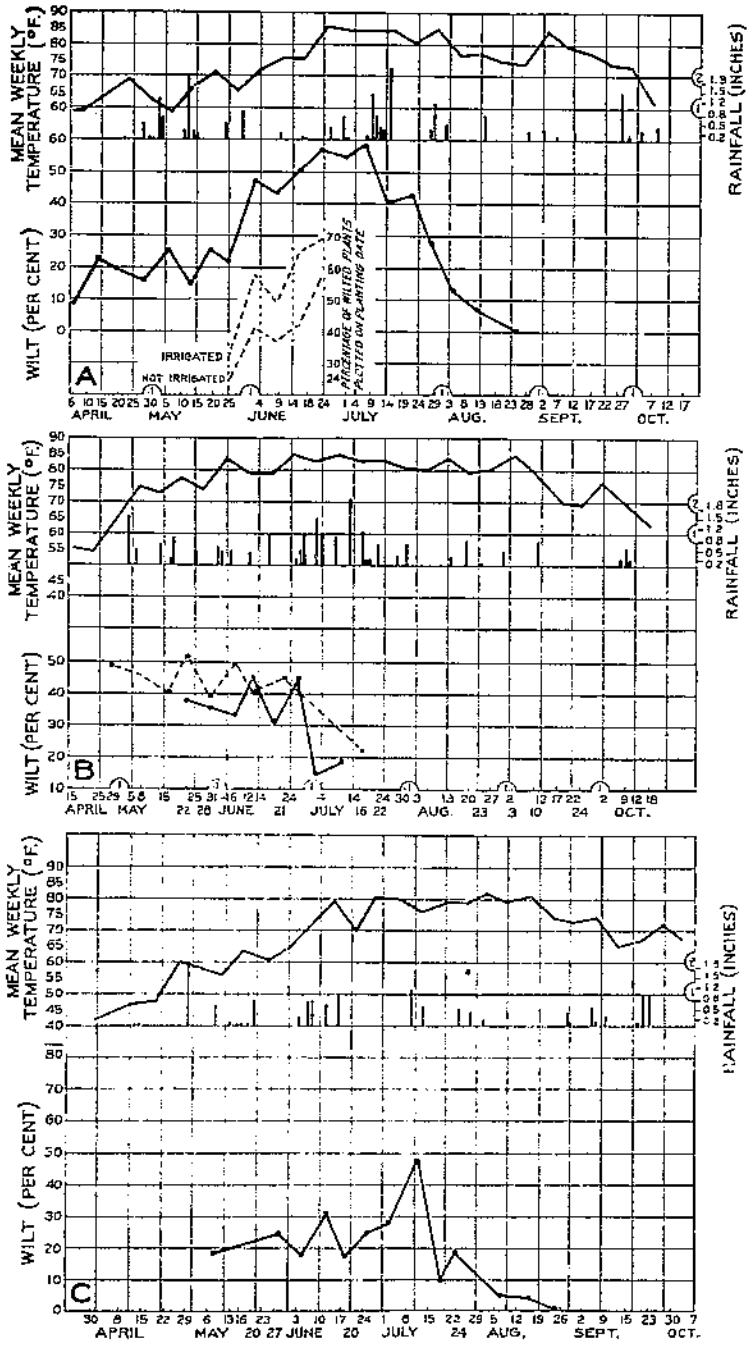


FIGURE 2.—Rainfall, temperature, and percentages of bacterial wilt in field plantings of sweet corn at Tuxedo, Md.: A, 1921; B, 1922; C, 1923. All wilt curves are plotted on planting dates and indicate the total percentage of wilt cases for each planting

later there was a heavy fall of rain, followed by a rather uniform distribution of plentiful rainfall throughout the remainder of the season and high temperatures until September. The total number of wilt cases for the nine plantings, based on data for the presence of yellow ooze in the stems at the last cutting, varied considerably, but was large in all plantings except the last.

During the same season, in another field, the Golden Bantam variety was planted on 8 successive dates from May 22 to July 10, when the mean weekly temperatures were about 75° to 80° F. at the time of the first 2, and about 80 to 85° F. at the time of the other 6 plantings. On the basis of the data on the presence of yellow ooze in the stems, there were from 30 to 47 per cent of wilt cases in the first 6 plantings and a falling off in wilt prevalence in the last 2. The plantings maturing in August showed by far the highest percentages of incipient cases, which fell off to zero in the mature plants of the final planting.

Except for the first and last weeks of the growing periods of the last two series of plantings, temperatures were high and rainfall was plentiful and well distributed throughout the season. The disease likewise was rather uniformly prevalent except for a falling off toward autumn, as is usually the case. The maximum dissemination in both tests took place during midsummer.

During the next season 13 plantings were made between May 8 and August 25. The disease here was highly fluctuating but with some relation to weather conditions, as shown in Figure 2, C. Wilt was moderate in prevalence for the first two plantings, made during the spring period of moist soil conditions, but with comparatively low temperatures. For some unknown reason wilt dropped somewhat in the third planting in spite of rising temperature and two weeks of plentiful rainfall; but the fourth planting, made eight days later during a rainy period at a mean weekly temperature of 80° F., rose distinctly in wilt percentage. Plantings 5 to 7 fell in wilt prevalence with a period of drought covering the planting dates, the drop being greatest in the fifth, which coincided with a week of cooler temperatures. The highest percentage of wilt of the season occurred in the eighth planting, made on July 9, between two heavy rains and followed by continued high temperatures. In the ninth planting, made during a dry period, wilt infections decreased in spite of high temperatures. The tenth planting, followed immediately by several moderate rains and by high temperatures for about a month, again rose somewhat in wilt percentage. In the last three plantings, made during a long period without rains and followed soon by the decreasing temperatures of autumn, wilt infections decreased rapidly, reaching zero in the final planting.

In this connection, field inoculations in young plants of susceptible varieties, made under late-fall conditions, have usually given negative results, though observations were in some cases possible for several weeks before frost killed the plants. The following experiment was also performed: 172 potted plants of four of the most susceptible varieties of sweet corn grown in the same greenhouse were inoculated by needle punctures with pure cultures of *Aplanobacter stewarti*. The plants were then separated into two lots, each the same as to age and variety, one lot being held at 75° to 85° F., the other at 55° to

65°. About one month later, when the final notes were taken, 83 per cent of those in the tropical greenhouse and 33 per cent of those in the cold greenhouse were found to have contracted the wilt disease.

Results of these general and date-of-planting field tests and of temperature tests in the greenhouse appear to show a definite relation between wilt prevalence and temperature and rainfall, though it is clear that other influences have also been operative. That dissemination by insects is the principal remaining factor will be shown later.

SOIL TYPE

One experiment relative to the effect of soil type on wilt development has already been discussed briefly under "Transmission Through Soil" (p. 10). During the same season rows of identical varieties were planted directly across an area comprising three types of soil which may be roughly described as (1) very light sandy soil, (2) slightly heavier sandy loam, and (3) fertile sandy loam, the whole receiving identical treatment. There were approximately the same total numbers of plants in the three portions of the plot. Thirty-eight cases of wilt developed in the light sandy third, 345 cases in the medium third, and 500 cases in the most fertile third of the plot.

In a third similar test, with about 8,000 plants on two types of soil, one poor and one fairly good sandy loam, 19 per cent of the plants on the poor soil and 28 per cent of those on the good soil were wilt infected.

In a fourth test over three soil types, the part on very poor sandy soil gave 21 per cent of wilt, that on a medium rich clay loam 41 per cent, and that on the transitional part between the other two 27 per cent.

In a fifth test the same seed was sown in two parts of a plot of newly cleared land part of which had been under cultivation during the preceding season and part of which consisted of freshly cleared soil not yet settled down. Of about 900 plants in this plot, 84 per cent in the cultivated part, and 64 per cent in the freshly cleared part contracted wilt.

In these tests the general tendency, other factors being equal, has been for a greater increase of wilt in the heavier and richer types of soil than in very light sandy soils. These results confirm the findings of earlier studies by Smith, who states (*17, p. 124*) that an abundance of moisture stimulating rapid growth is very favorable both to primary infection during the seedling stage and to the general distribution of the bacteria through the stem of the plant later on.

STUNTING

In inoculations of very thickly sown plants in pots and in similar plants subject to natural infection only, Smith (*17, p. 110, 218*) attributed the unusually low infection and retarded course of wilt disease to the very slow growth and hardened, stunted condition of the plants. The few tests of this kind which the writers have made have tended in general to confirm this conclusion. For instance, in one test with seed sown very thickly both inside and outside 18-mesh wire cages, 54 per cent of the plants inside and 80 per cent outside of the cages developed wilt from spontaneous infections.

Plants of the same variety, three or four to a hill, but otherwise under the same conditions in contiguous rows, gave 90 per cent of wilt. Here the smaller amounts of wilt paralleled the thicker sowing, but the wire netting, which kept out a considerable number of insects, caused a much greater difference in the amount than that shown by stunted as compared with normal plants.

LOCALITY

One test as to the effect of locality has been discussed already (p. 17), in which 139 identical lots of seed planted near Washington, D. C., and at Orono, Me., gave a high percentage of wilt in Maryland and none at all in Maine.

In another test identical lots of Golden Bantam seed from Maine and from Michigan were planted near Washington, D. C., where wilt had been prevalent in experimental plots for several seasons; at Westminster, Md., where commercial plantings of Golden Bantam had been seriously damaged by wilt in preceding seasons; at Wolfville and Mountain Lake Park, Md., both mountain farming communities; at Morgantown, W. Va., on the State experiment station farm; and in two city lots in Washington, D. C. Except for a small part of the plots near Washington, D. C., all plantings were made in May and June. Wilt prevalence from spontaneous infections was highest in the May and early June plantings near Washington and at Westminster and Wolfville (northeast of Frederick), Md., and lowest in the late plantings (July and August) near Washington, followed in ascending order by plantings in the city plots, at Mountain Lake Park (extreme western border of Maryland), and at Morgantown. Records obtained near Washington, D. C., showed almost no flea beetles present July 20 to 27; they began to appear again in the fields during the first week of August, and then fell off gradually in numbers toward the close of the season. Here the decreasing wilt prevalence in general followed the decreasing prevalence of flea beetles, low rainfall, and the gradually decreasing temperatures of autumn. Both flea beetles and 12-spotted cucumber beetles were present in all the localities, but no records were kept as to relative prevalence in the other sections. Since all the seed used had been grown in Maine and Michigan localities where the disease was not known to occur, and in each case was planted in land not in corn the preceding season, it seems clear (in connection with the studies on insect relations, discussed later) that the wilt infections were due to dissemination by insects and that, the seed being the same in all localities compared, the differences in wilt prevalence were due to differences related to the insect disseminators and the concomitant climatic conditions.

During the same season plantings of Golden Bantam seed from a badly wilt-infected commercial planting of the preceding season in Maryland were made in four localities. This seed gave the highest percentages of wilted plants near Washington and at Westminster, somewhat less at Wolfville, and least at Morgantown.

Identical lots of Golden Bantam and First-of-All seed selected from plants known to be wilted were also sown in different localities. The wilt was most prevalent near Washington, followed by Wolfville, Westminster, Morgantown, and Mountain Lake Park.

In plantings of over 60 identical lots of Golden Bantam seed sampled from the open market by the Division of Seed Investigations, Bureau of Plant Industry, wilt was found to be most prevalent near Washington, followed by Morgantown and Mountain Lake Park.

The significant results from these plantings of identical lots of seed in different localities, in all of which flea beetles were present, seem to be that (1) there was no more wilt in progeny of diseased than of healthy plants; (2) insects were responsible for most if not all of the dissemination; and (3) there was a tendency to follow flea-beetle prevalence, where known, and a secondary tendency to follow climatic conditions.

IRRIGATION

An irrigation test was carried out on a level field of very uniform sandy soil, with plantings of 30 hills each of four susceptible varieties of sweet corn to each irrigated and to each control nonirrigated plot. This test was repeated five times at different planting dates, and treated plots were irrigated uniformly twice a week, beginning at the date of planting. Averaging the results of the plots of all five planting dates, there was a little less than three-fourths as much wilt in the nonirrigated (40 per cent) as in the irrigated (55 per cent) portions. Reference to Figure 2, A, shows the general trend of wilt in these tests to be similar to that in the large general date-of-planting tests in this same field. Moderate rainfall occurred about the time of the first planting, but practically no more until some time after the last planting; this period was therefore particularly favorable for such a test.

An irrigation test carried out two years later, comprising approximately 800 plants of two susceptible varieties, gave approximately three-fourths as much wilt in the nonirrigated (23.5 per cent) as in the irrigated (31 per cent) plots.

It seems clear from these tests that soil moisture, with its concomitant more vigorous growth of plants, bears some relation, direct or indirect, to prevalence of this disease.

DISSEMINATION BY INSECTS

CIRCUMSTANTIAL EVIDENCE

After thoroughly studying the field and laboratory notes of their preliminary studies, it became very apparent to the writers that transmission through seed and soil, and climatic and soil environmental factors, were entirely inadequate to explain the observed facts relative to the origin and prevalence of wilt, but that most if not all moot points would become quite clear should insects prove to be implicated. At this stage of the investigation circumstantial evidence favoring insect dissemination was as follows:

In a general way the geographical distribution of corn wilt corresponds to that of the 12-spotted cucumber beetle, *Diabrotica duodecimpunctata* Fab., and of the flea beetles, *Chaetocnema pulicaria* Melsh. and *C. denticulata* Ill. For example, these insects are extremely common in Maryland and the District of Columbia, where wilt is particularly prevalent and destructive, and rare or absent in Maine, where the northern corn billbug and the common stalk borer are the prevailing corn insects and where wilt has been unknown.

The 12-spotted cucumber beetle is reported to be the most injurious in wet seasons and on lowlands. The experience of the writers with bacterial wilt has been similar; and furthermore, during two dry seasons there has been a higher percentage of wilt in irrigated than in contiguous unirrigated plots otherwise similarly treated.

The sporadic appearance and spread of wilt in rows from thoroughly mixed lots of seed and in blocks planted either with the same or with different lots of seed of the same varieties suggested dissemination by insects in both primary and secondary cases.

In experimental fields in five localities in Washington, D. C., Maryland, and West Virginia, a high percentage (62 to 100 per cent) of primary bacterial wilt cases in sweet-corn seedlings showed small larval channels at the base of the stem. Plants without bacterial wilt, pulled at random in the thinning-out process in these same fields, gave a uniformly low percentage (0 to 17 per cent) of these larval channels.

Many large planting tests were made with seed from plants known to have had bacterial wilt and from plants known to have been healthy, with seed purchased in the open market, and with seed from Maine and Michigan sections where the disease was not known to occur. When these various lots of seed were planted together in a locality where wilt was prevalent the ultimate amount that developed in the progeny tended to follow the susceptibility of the variety rather than any previous history of seed or soil. In fact, under the usual field conditions in Maryland and West Virginia, seed from Maryland-grown healthy plants was as likely to give a high percentage of wilt as was that collected from badly diseased plants from the same original seed and plot. Also, the percentage of early cases appeared to be as great from wilt-free seed as from open-market or from wilt-infected seed of the same varieties.

The amount of wilt has always dwindled nearly or quite to zero in late summer plantings, even when weather conditions have been apparently as favorable as in the case of early plantings developing abundant wilt. This decrease or absence of wilt has coincided with striking decreases in the number of flea beetles and with the well-known postponement of egg laying by the last or hibernating brood of Diabrotica until spring.

In large variety tests in fresh soil, 1, 2, and 3 year old seed of the same varieties planted at the same time all gave approximately the same amount of wilt when cut at maturity. On the other hand, these same seed lots when planted at different times during the same season often gave highly variable amounts of the disease. It is extremely doubtful that approximately the same percentage of wilt could have come from seed differing so greatly in age but planted at the same time, on the one hand, or that the highly varying percentages of wilt from different times of planting could have been entirely due to differing weather conditions, on the other hand.

It was noted during several seasons' observations that a considerable proportion of cases appeared to start from the upper part of the plant; and at the end of the season cases were common in which the tip was badly wilted and gave abundant yellow ooze from the cut stem, while the basal portion of the plant showed no external or internal signs of the disease whatever. In other instances the

middle portion was diseased, while the tip and base were normal. For example, in a field of sweet and pop corn varieties aggregating 156 rows of at least 80 hills each, there were at maturity 72 plants with distinct signs of wilt and ooze near the tip only. There were absolutely no signs of the disease, either externally or through occlusion of the vascular system (as observed under a hand lens) in the lower half of the plant, and in a large proportion of the cases external and internal signs appeared only very near the tip of the plant. Observations such as these might be multiplied. Clearly, such infections with this vascular bacterial disease could scarcely have come from either soil or seed.

In all probability, every infection arising from the seed would develop external or internal signs during the early part of the growth period. For several seasons portions of single plantings were cut, respectively, at the early tassel, late silk, premilk, and roasting-ear stages and showed a progressive increase in number of cases of the disease the longer the cutting was deferred. If all infection had come from soil or seed it is extremely improbable that a high progressive increase in number of wilt cases would have occurred at these succeeding periods of growth, since there is little doubt that practically all seed infections are evident by the tassel stage or at the latest by the silk stage.

A small block of Maine and Michigan Golden Bantam seed was planted in the middle of a large block of the wilt-resistant Stowell Evergreen variety which itself showed only traces of wilt throughout the season; the Golden Bantam seed here gave 50 per cent of wilt cases. A similar Golden Bantam block, in which the same seed lots and similar soil were used but which was planted in a field of cucurbits and near a large field of susceptible corn varieties, gave 72 per cent of wilt cases.

In a large number of cases observed in the general plantings, leaf wilt was seen to have originated at small, linear leaf injuries inflicted by flea beetles.

PRELIMINARY INSECT-EXCLUSION TESTS

In view of these various lines of circumstantial evidence, it became increasingly apparent that the facts would be explained if it could be shown that the early seasonal incidence of the disease (scattered primary cases apparently originating from the base of the plant) was due largely to introduction of the bacterial parasite by adult or larval insects working at the roots or base of the stem and that the midseason spread (secondary cases) was due to transfer of infection by insects from diseased to healthy plants. Like straws showing which way the wind blows, these facts at any rate all pointed more or less clearly to the probability of dissemination by insects.

Accordingly, a large isolated cage, 42 feet on a side, 6 feet high, and covered with 18-mesh wire netting, was used. The first season an early and a late planting of open-market seed representing a considerable number of susceptible varieties were sown in this cage, but at no time during the season did wilt appear in either planting. The disease was prevalent in plants from the same seed lots in other parts of the same locality, but there were no cornfields in the immediate vicinity of the cage.

The following season a partition was put through the center of this cage, each side of which was then (May 23) planted to identical seed lots of susceptible varieties of sweet corn. Into one compartment considerable numbers of 12-spotted cucumber beetles previously fed upon wilt-diseased plants were introduced at frequent intervals; no insects were introduced into the other compartment. Of the approximately 250 stalks of corn developing in each of these compartments, 10 per cent developed wilt in the insect side and 12 per cent in the "insect-free" side. Some of the smaller insects could, however, gain access through the 18-mesh netting covering the cage. Therefore, in a midseason planting, with the same seed lots, no insects were introduced, but a section of the netting 2 feet wide and midway between the top and bottom of the cage was cut away from one side of one of the compartments facing a contiguous field of sweet corn where wilt was prevalent. These conditions gave free access to all insects to this compartment, but left the completely closed compartment impervious to the entrance of the larger insects and much less easily accessible to the smaller insects, but at the same time gave approximately identical conditions as to light and air movements in the two sides. At harvesting time the closed compartment gave 13 per cent of wilt cases, while the open part gave 33 per cent, which was very nearly the average for these varieties in the open field near by.

Early in the same season, in another field, each of 50 hills of the First-of-All variety was covered at planting time with 18-mesh wire-netting cages, and 36 hills were left uncovered. Early in August 40 cages were removed. Outside the cages 79 per cent of the plants developed wilt; in those covered during the first part of the season only 65 per cent and in those under the cages throughout the season 34 per cent developed wilt.

In another level field of very uniform sandy loam, comprising 54 hills of the same variety, 6 hills were covered with the wire-netting cages at each of six successive intervals of time, the first lot on the date of planting, and 18 hills were left uncovered throughout, as controls. The season's wilt record showed a progressive increase in wilt prevalence the longer the seedlings were left uncaged after planting, except for a falling off in disease percentage in two of the middle lots in which cloth cages were used.

From these preliminary tests it appeared probable that small insects were acting as disseminators and that some of them were able to pass through the fine wire netting, which, however, served as a barrier to the majority.

DIRECT TESTS WITH DIABROTICA DUODECIMPUNCTATA

During three seasons numerous direct dissemination tests were made with *Diabrotica duodecimpunctata* fed on wilt-infected plants and introduced into hill cages with susceptible varieties of sweet-corn seedlings covered from date of planting.

In the five series of tests with wire-netting cages the data were of little value so far as this insect is concerned, owing to the entrance of many small insects. However, three series of tests were run, aggregating 129 cloth-covered hill cages into which wilt-fed beetles

were introduced and 149 such cages, used as controls, into which insects were not introduced. The full records showed, respectively, 3 and 1.2 per cent of wilt cases in the first and second insect tests (June and July) and no wilt in the third insect test (August). No wilt occurred in the 149 control cages except for five cases in cages from which the cloth covering had become torn and all clearly started from insect injuries.

Direct inoculations with intestinal contents of adults of *Diabrotica duodecimpunctata* gave a much higher proportion of infections. Three such tests were carried out in the field, with cloth-covered hill cages in which about 85 plants were inoculated with intestinal contents of wilt-fed beetles. Several of these inoculated plants developed within the first week a soft rot at the base of the stem and were therefore thrown out of the test. Out of 74 plants left, 21 per cent developed typical wilt. Three such tests, with inoculations at 1, 2, and 3 days after the wilt feeding, carried out in the greenhouse, gave very similar results.

Again, during September, 36 adult beetles, collected at random in the vicinity of several of the experimental fields where wilt was prevalent, were fed in the greenhouse upon healthy corn seedlings for three weeks. Similar inoculations were made with the intestinal contents of the 26 beetles still alive at the end of this period. Nineteen per cent of the seedlings thus inoculated finally developed wilt, with the typical ooze in the vascular bundles. A similar positive result was also obtained with the use of beetles fed for five days on seedlings with bacterial wilt from pure-culture inoculation. In this case inoculations from 16 beetles gave 25 per cent of plants with wilt and ooze. In each one of these tests one or more wilted plants taken at random were further tested by successful reinoculation into other corn seedlings.

Summing up the results of direct tests with adults of *Diabrotica duodecimpunctata*, those with wire-netting cages were inconclusive because of the entrance of other insects. However, in general, the percentage of infection was greatest in the plants outside of the cages, least in the control cages, and intermediate in plants caged with wilt-fed beetles. In the case of the cloth-covered control cages, barring accidents allowing entrance of other insects, there was no wilt in over 400 control plants. In the "insect" cages, however, there were six cases of wilt among a total of 389 plants tested with wilt-fed beetles in June and July; and no wilt among 182 plants tested during the latter three-quarters of August. Either in the direct tests or in the fields at large it was only rarely that wilt was observed to have started apparently from points gnawed by these beetles. On the other hand, many of the tests were run long enough for development of larval stages of another generation, and small channels in the base of the stem were frequently observed in the "insect" cages. These facts, taken in connection with the clear-cut results from inoculations with intestinal contents, lead to the supposition that very little of the secondary leaf infection comes from direct transfer by the mouth parts of these beetles, but that some, and possibly a considerable part, of the primary basal infection may come from their larval borings at the crown.

DIRECT TESTS WITH FLEA BEETLES

Since flea beetles were very prevalent, and since in the general field observations numerous cases of secondary leaf infection were observed to have started apparently from the typical linear injuries made by these beetles, direct tests with these insects were planned.

As a preliminary test, 20 wilt-fed flea beetles (without regard to species determination) collected from corn were introduced at midseason into one of the wire-netting cages covering a hill of half-grown First-of-All sweet corn. Later, wilt was directly observed starting from a considerable number of their injuries on the leaves. From some of these incipient leaf cases *Aplanobacter stewarti* was isolated in pure culture and tested by successful reinoculation in the greenhouse. There were no control cages in this test, but results seemed sufficiently clear to warrant further investigation.

During the following summer seven tests were carried out, in which was used the brassy flea beetle, *Chaetocnema pulicaria* Melsh. (determination by the Bureau of Entomology), fed 24 to 48 hours on corn plants with bacterial wilt and then introduced into cloth cages covering hills of susceptible sweet-corn varieties. In 4 of these 7 tests carried on with 16 cages having at least 4 plants to a cage, there was 100 per cent infection, and in the other 3 tests, 57, 60, and 87 per cent infection respectively. In control cages of similar type all plants remained free of wilt.

Similar experiments during a third season abundantly confirmed these results and added the toothed flea beetle, *Chaetocnema denticulata* Ill. (also determined by the Bureau of Entomology), to the list of direct disseminators. Briefly, in several field tests carried on with 16 cloth-covered cages, into each of which had been introduced 12 to 50 wilt-fed flea beetles, the records showed a total of 8 healthy plants and 40 with bacterial wilt. In other words, 83 per cent of the plants in these "insect" cages contracted the disease. The controls were the same as for the simultaneous test with *Diabrotica duodecimpunctata*, namely, out of 441 caged control plants no wilt at all appeared except in 5 plants in damaged cages which insects had entered.

Thus, the field observations and direct tests show clearly that at least in the Maryland region near Washington, D. C., where the experiments were carried out, the great bulk of the secondary infection—late spring and summer spread of the disease—is brought about by direct transfer of infectious material by flea beetles.

CONCLUSIONS

Bacterial wilt of corn, a vascular disease caused by *Aplanobacter stewarti*, occurs primarily in the Middle and Southern States; it is nonexistent or rare in the northern tier of States.

Transmission through the seed is a serious factor, probably the only factor, in introducing the disease into new localities, and inroads from this source alone may be considerable.

Because of the preponderant secondary summer dissemination of infection in a wilt-prevalent locality, the disinfection of seed under such conditions is of doubtful value. It would be of little value, even though effective, except for possible use in sterilizing seed to prevent

the introduction of the disease into new localities, and in such cases the treatment would need to be 100 per cent effective. Since the bacterial parasite is carried within the seed, externally applied disinfectants offer little hope of such a degree of success; but dry-heat sterilization at temperatures well above the thermal death point of the bacterial organism have killed all bacteria in laboratory tests without materially reducing the rate of germination or vigor of the plants.

Experimental data show that while overwintering of *Aplanobacter stewarti* in the soil may conceivably occur, it is not likely to take place or to be of practical significance.

Wide differences in resistance to the disease were found among commercial varieties of corn. In general, the degree of resistance was roughly proportional to the length of time required for the maturing of the variety; that is, in sweet corn, the earlier the variety the more susceptible it proved: in field corn, the early flint sorts were found to be more susceptible than the dent types. Therefore, in wilt-prevalent localities, the use of well-selected resistant varieties will reduce to a minimum inroads from the disease.

Weather conditions, such as temperature, rainfall, and soil moisture, and the growth condition of the plant, have considerable influence on infection and on subsequent development of the disease, but these factors are often deeply overshadowed by the factor of dissemination by insects.

The dissemination of the wilt organism by means of the mouth parts of the adults of the 12-spotted cucumber beetle does not appear to be an important factor in the spread of the disease. This insect, however, is capable of harboring the organism in its alimentary tract over considerable periods of time.

At least for the Maryland section, flea beetles are definitely shown to be responsible for the main summer spread of bacterial wilt of corn, through secondary leaf infection by direct transfer of the wilt organism.

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ADDENDA

(1937)

While not attempting any exhaustive survey of subsequent work (viz, since 1924) by others, the writers deem it highly advisable to refer to the following important lines of recent progress in the study of bacterial wilt of corn.

Not only has the direct summer dissemination of *Aplanobacter stewarti* by *Chaetocnema pulicaria*, *C. denticulata*, and *Diabrotica duodecimpunctata* been confirmed by Elliott and Poos (24), Elliott (25), and Poos and Elliott (27), but these authors have also demonstrated the overwintering of the organism in adults of *C. pulicaria*, which appear to be responsible for most of the early infections in

the field each year. Furthermore, Ivanoff (*25*) has studied the wilt organism with special reference to its life history and pathogenesis, including the demonstration that a corn rootworm, larva of *D. longicornis*, is also able to transmit the disease from infected to healthy plants.

The natural host range of *Aplanobacter stewarti* has been extended by Elliott (*23*) to include teosinte; and successful inoculations are reported by Poos and Elliott (*27*) on teosinte and jobs-tears (*Coix* sp.), and by Ivanoff (*26*) on sorghum, Sudan grass, yellow foxtail grass, German foxtail millet, and common millet. The latter author also reports similar symptoms as induced by the organism of Cobb's disease of sugarcane on corn and sorghum in the greenhouse.

In 1934 Stevens [5] stated that—

> the outstanding disease phenomenon among cultivated plants during the past few years is undoubtedly the destruction caused by bacterial wilt of corn * * * which reached a climax in 1932 and 1933 * * *. In the years indicated above, the disease was very abundant not only within its usual range but over a large area farther north, where susceptible varieties of sweet corn had long been grown successfully. The unprecedented losses of 1932 and 1933 were followed by a sudden drop to something like normal incidence and range of the disease in 1934.

Through an exhaustive study of available records, Stevens was able to reconstruct a fairly satisfactory picture of the behavior of bacterial wilt during the preceding 35-year period in relation to winter temperatures. A careful correlation of these data impelled him to present the hypothesis that increased incidence and severity of the disease and extension of its range northward are in some way related to unusually high winter temperatures. In general, the data accumulated in subsequent years have confirmed this hypothesis.[6] Moreover, Poos and Elliott (*27, p. 607*) state that "the relationship of winter temperatures to the abundance and distribution of the insects which overwinter and disseminate *Aplanobacter stewarti* may be very significant in predicting the appearance of bacterial wilt of corn in destructive abundance"; and Haenseler,[7] while confirming Stevens' general hypothesis, states that "there seems to be a certain time factor or lag period involved which must be studied further and evaluated before a quantitative prediction of wilt incidence can be made." It appeared that after a period of severe wilt, a single cold winter is not enough to reduce the parasite or its carriers sufficiently to give the expected disease control. Likewise, after a period with but little wilt, the first warm winter may not allow a sufficient increase in the parasite or its carriers to give a serious wilt outbreak the following summer.

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Since losses from wilt had been shown to be greatest in the early maturing commercial varieties of sweet corn, much attention has recently been paid to the selection and breeding of resistant strains and varieties. Notable in the results along these lines is the work of Smith (*28*), who presented Golden Cross Bantam, a new single cross hybrid yellow sweet corn developed in breeding investigations by the Bureau of Plant Industry in cooperation with the Indiana State Experiment Station. These studies have been continued, and seed is now available in quantity. Important progress in such work has also been made by many other experiment stations in the regions where sweet corn is commercially grown.

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