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ANY PLANT DISEASE that is capable of destroying 90 per cent or more of a crop is a serious matter to the grower of the crop. Fusarium wilt of muskmelons is such a disease. Although it is not widely distributed in the state, it is slowly spreading and in time probably will be found wherever muskmelons are grown. The disease is relatively new and is described in detail for the first time in this bulletin.

Although muskmelons are not a major crop in Minnesota, they are grown rather extensively in the southern half of the state, especially in the vicinity of St. Paul and Minneapolis. Muskmelons are grown primarily for local consumption, although much of the crop is hauled by truck to the northern markets of Minnesota and adjoining states. Where muskmelons are grown in Minnesota, they constitute an important cash crop and are rather intensively cultivated. They are grown chiefly on light sandy loam soils that warm up quickly in the spring and maintain a realtively high soil temperature throughout the summer.

In recent years, because of the prolonged deficiency of rainfall, a general practice of irrigating muskmelons has developed. The overhead system of irrigation is the most common type in use. In very dry seasons the water is applied at the rate of about one inch per week. Barnyard manure is used extensively and is usually supplemented with commercial fertilizers applied at the rate of about 500 pounds per acre of a 4-8-6 formula.

These practices make the cost of production relatively high, but good yields of good-quality melons are usually obtained. Since these are placed on the market at a time when the southern and western supplies are limited, they generally demand a fair price. No accurate figures on the total value of the muskmelon crop in Minnesota are available, but the crop is a very important source of income for the truck farmers of the state.

The most commonly grown variety is the Golden Osage, which is probably identical with Bender's Surprise. A slightly earlier strain of this variety is grown under the name of Golden Sunrise. The variety Sugar Rock is also grown rather extensively.

Diseases have not been very important as limiting factors in muskmelon culture in Minnesota. Perhaps the most destructive disease has been anthracnose (*Colletotrichum lagenarium*), but it is prevalent only

[1] Completion of this project was made possible by workers supplied on Project No. 4841, Minnesota Works Progress Administration. Sponsor: University of Minnesota.

when rainfall in late summer is unusually abundant. In most years anthracnose appears too late in the season to cause much loss and often is entirely absent; therefore it has not been practical to adopt a consistent control program. Occasional plants are affected with bacterial wilt (*Erwinia tracheiphila* E.F.S.), and mosaic is usually present but causes relatively little loss.

FUSARIUM WILT

In 1931 a very destructive Fusarium wilt of muskmelons was found in Minnesota. More extensive observations were made in 1932, and the disease was briefly described the following year (9). It was found only in two local areas in Hennepin and Ramsey counties, and, although the extent of these affected areas apparently has increased considerably, the disease has not yet been found in any other part of the state.

Chupp (4, 5) in 1930 and 1931 reported a Fusarium wilt of muskmelons causing a trace to one per cent loss in several fields in New York. At first it was thought that this disease and the one found in Minnesota were different, but more recent comparisons indicate that they may be identical.

The disease causes much loss wherever it occurs. Some fields are so heavily infested that it is impossible to grow muskmelon plants to maturity. The soil is "muskmelon-sick." Several farmers in one locality have discontinued growing melons entirely because of the disease. Although muskmelons have been cultivated extensively for many years in many parts of the country, this disease was not reported until 1930. It seems hardly possible that so destructive a disease could have escaped observation for so long a time if it had been present. There had been several reports in the literature of Fusaria pathogenic to muskmelons, but none of the diseases appears to be identical with this one. All evidence points to the conclusion that this is a new disease or it is one that has been introduced into the United States in recent years from some unknown source.

In 1898 Sturgis (16) observed a Fusarium fruiting on the stem of a wilted muskmelon in a field where bacterial wilt was prevalent, and, although no inoculation experiments were made, he suggested that "the wilt disease which has been so prevalent lately in our muskmelon fields may be due in part to the attacks of a species of Fusarium possibly the same as that associated with the wilt of watermelons."

The same year, Selby (14) reported a wilt of garden cucumbers, Japanese climbing cucumber, and muskmelon, and stated that it appeared "to be due to the same or to a similar fungus to that causing the southern watermelon wilt described by E. F. Smith." Here also no inoculations were made. In 1920 Taubenhaus (17) published the results of his investigations on the wilts of watermelons and related crops. In numerous field trials on "watermelon-sick" soil he showed that 100 per cent of the

cantaloupes remained healthy, while 69 to 98 per cent of the watermelons were killed. Taubenhaus made no mention of a wilt of cantaloupes in this study.

Stone (15) in 1911 mentioned a Fusarium stem rot of cucumbers and muskmelons in the greenhouse but made no inoculations and did not identify the pathogen. It is not probable that he was dealing with the wilt in question, because greenhouse tests and field observations show that cucumbers are not affected by the wilt of muskmelons occurring in Minnesota.

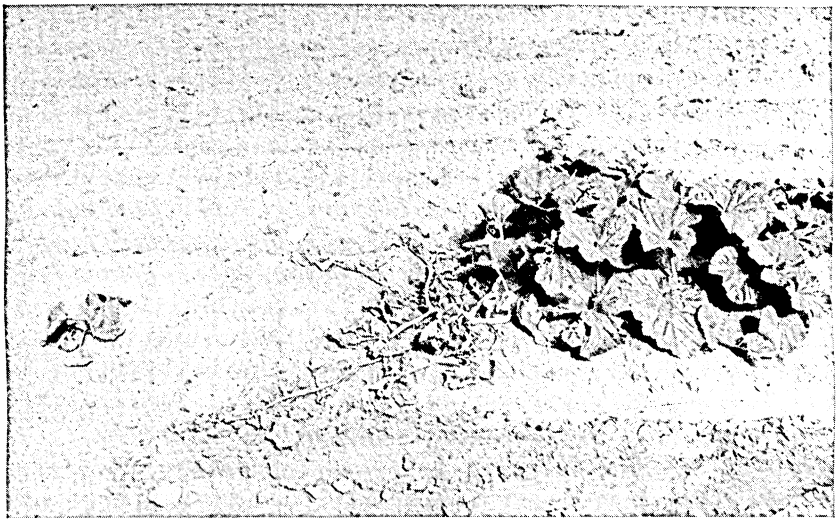

FIG. 1. MUSKMELON WILT

The small plant on the left is a replant, the original plants in the hill having been killed by wilt previously. One of the older plants in the hill on the right is wilted, and the other is still healthy although it died a few weeks later.

Cook (6) in 1923 reported a greenhouse disease of melons from which he isolated a Fusarium "very similar to *Fusarium vasinfectum.*" No inoculations were made to determine the pathogenicity of the fungus, and the disease is not described in sufficient detail to justify conclusions as to its identity.

Wollenweber (22) isolated a Fusarium from muskmelon fruits in Canada, but no wilt of the plants was observed.

According to Wollenweber and Reinking (22), Dufrenoy and Rodigin have referred to a foot rot of melons in Europe caused by Fusaria (*Fusarium oxysporum* var. *aurantiacum* and *F. reticulatum*).

From the data available for comparison, it seems unlikely that any of these diseases are the same as the disease in question.

In the summer of 1937 an attempt was made to obtain more information about the occurrence of muskmelon wilt in the United States by

means of a questionnaire sent to the various state experiment stations, but the results were somewhat unsatisfactory because of the lack of positive identification in many cases of its suspected occurrence. There are good reasons for believing that the disease occurs in Minnesota, New York, Michigan, and Arizona. Less positive reports were received from Delaware, Florida, Louisiana, Montana, Rhode Island, Texas, Virginia, and West Virginia. In addition, letters have been received indicating its occurrence in Ontario and Vancouver Island, Canada.

The records of the Division of Mycology and Plant Disease Survey of the United States Department of Agriculture contain reports of wilt on muskmelon attributed to *Fusarium* sp., *Fusarium niveum,* or *F. vasinfectum* from the following states: Alabama, Arizona, Arkansas, California, Connecticut, Delaware, Florida, Georgia, Illinois, Indiana, Louisiana, Mississippi, Missouri, New Jersey, New Mexico, New York, North Carolina, Ohio, Oregon, South Carolina, Texas, and West Virginia. It is, of course, impossible to determine whether these reports refer to the muskmelon wilt discussed in this bulletin or to some other less destructive infection.

THE ECONOMIC IMPORTANCE OF THE DISEASE

Because of the limited distribution of Fusarium wilt, actual losses are not very great. However, as previously reported by Leach (11), the pathogen can easily be carried long distances on or in the seed, and in time such distribution is very likely to occur. Potentially, therefore, the disease is of great economic importance. In Minnesota the losses from the disease have not been much greater in each of the last five years than in the year it was first discovered. This is not because the disease is less virulent but because the growers have learned the nature of the trouble (previously attributed to many unrelated factors) and have rotated crops so as to avoid planting melons on infested soil. In this way losses have been temporarily reduced, but as more and more soil becomes "muskmelon-sick" it is becoming more difficult to avoid the losses, and many farmers probably will be unable to grow melons profitably unless some method of control is devised. Furthermore, the disease will likely increase in severity in regions of intensive culture, as has been true for all similar Fusarium wilts, and unless suitable means of control are devised greater losses may be expected in future years.

SYMPTOMS

The symptoms of Fusarium wilt of muskmelons are very similar to those of the better-known watermelon wilt. Plants may be affected in any stage of development (Figs. 1, 2, 3, and 4). In heavily infested soils and at low temperatures, seedlings may be destroyed before they emerge from the soil. The symptoms on very young plants may be

confused with those of the damping-off caused by other fungi. On older seedlings, especially in cool soils, the disease acts as a root rot, the parts below ground being completely necrotic. In many plants infection appears to take place through the "peg" and often causes a hypocotyl rot, the roots apparently remaining healthy until the death of the plant. In other cases small local lesions are formed on the hypocotyl. In still others seedlings may wilt without any evidence of local necrosis (Fig. 2).

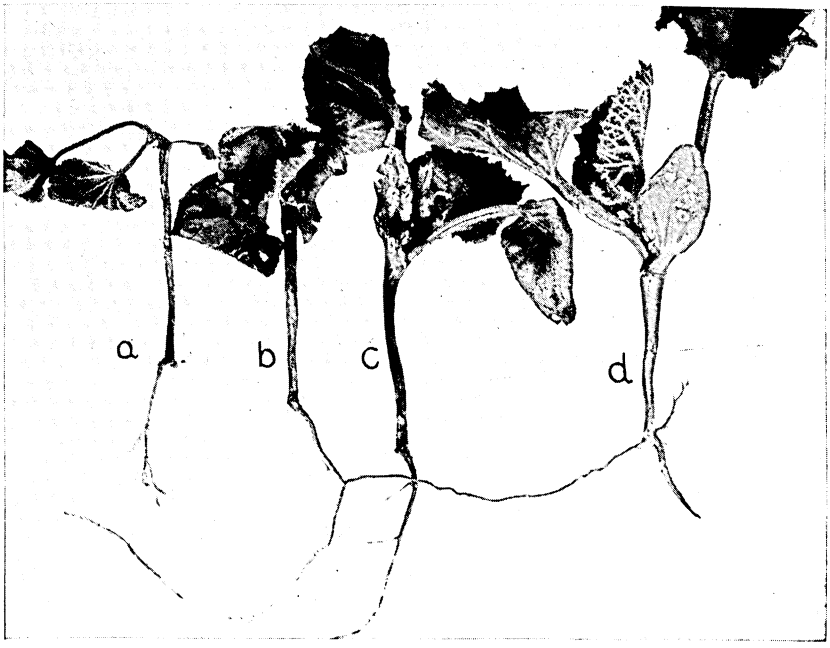

FIG. 2. FOUR MUSKMELON SEEDLINGS AFFECTED WITH WILT

Seedlings *a* and *b* wilted without local necrotic lesions. Seedling *c* has a large necrotic lesion on the lower part of the hypocotyl just above the peg. The pathogen was fruiting in salmon-pink sporodochia on this lesion. The lower part of the main root of seedling *d* is necrotic while the hypocotyl is uninjured externally.

The first symptom on older plants is nearly always a wilting of one or more runners. Before the plant is completely wilted, necrotic lesions may appear on the stem near the ground line. These lesions may involve only one side of the stem, extending for some distance as a long, narrow, brown streak (Fig. 3), but sooner or later the plant wilts completely and dries up. The necrotic lesions on old stems first appear as dull, dark green, collapsed tissue which rapidly turns brown. If moisture conditions are favorable, necrotic lesions on plants of all ages become covered with salmon-pink masses of conidia of the pathogen.

When plants escape infection until large fruits have been formed, the fungus often invades the stem end of the partly ripe fruits and causes a fruit rot (Fig. 4). Spores are produced in great abundance in salmon-pink waxy masses on the surface of such fruits.

FIG. 3. A NECROTIC STEM LESION ON A WILTED PLANT

The lower part of the stem of a muskmelon plant affected with Fusarium wilt showing the necrotic streak extending along one side of the stem from the base of the hypocotyl to the sixth leaf. This is typical of plants that do not succumb to the disease until after they have made considerable growth.

ETIOLOGY

As previously reported (9), muskmelon wilt is caused by a species of Fusarium closely resembling the pathogen of watermelon wilt (*Fusarium bulbigenum* Cke. and Mass. var. *niveum* Wr.). The fungus is easily isolated from freshly wilted plants, as it usually occurs in such abundance that secondary organisms are not troublesome. Numerous isolations have demonstrated the constant association of the same fungus with typically wilted plants.

The pathogenicity of the fungus has been repeatedly demonstrated by greenhouse inoculations. In these experiments, seeds of the Golden Osage variety were planted in previously sterilized soil in six-inch pots,

and the soil was then watered with a suspension of spores of the fungus. Pots treated in the same way, except that they were watered with clean tap water, were used as checks. Pots of naturally infested soil were included so that the normal symptoms could be compared with those resulting from artificially infested soil. Because of the similarity of the assumed pathogen to the pathogen of watermelon wilt, seeds of the Kleckley Sweet watermelon were planted in some of the pots, and some of the pots were inoculated with spores of the watermelon wilt pathogen. The results of some of these tests are given in Table 1.

Table 1. Results of Comparative Tests for Pathogenicity of Fusaria Isolated from Wilted Muskmelons and Wilted Watermelons

Pot No.	Inoculum	Seeds planted	Plants emerged	Plants wilted after 35 days
1	Fusarium isolated from muskmelon	G.O.*—10	8	8
		K.S.†—10	9	0
2	" " " "	K.S.—10	9	0
3	" " " "	G.O.—15	15	12
		K.S.—15	13	0
4	Fusarium isolated from watermelon	G.O.—15	14	0
		K.S.—15	12	11
5	" " " "	G.O.—10	6	1
6	" " " "	G.O.—15	14	0
		K.S.—15	12	10
7	Muskmelon-sick soil	K.S.—15	15	0
8	" " "	G.O.—10	6	6
9	" " "	K.S.—10	7	0
10	" " "	G.O.—15	13	13
		K.S.—15	13	0
11	No inoculation	G.O.—10	10	0
12	" "	K.S.—10	10	0
13	" "	G.O.—15	14	0
14	" "	K.S.—15	13	0

* G.O. = the Golden Osage variety of muskmelon.
† K.S. = the Kleckley Sweet variety of watermelon.

It is quite obvious from the results obtained that the cultures isolated from wilted muskmelon plants were pathogenic to muskmelons but not to watermelons and that the culture of the watermelon wilt pathogen used was not pathogenic to muskmelons although it caused a wilt of the watermelon seedlings (see Fig. 5).

The specific pathogenicity of these fungi for their respective hosts is shown also by field reactions. Although watermelons also are grown extensively in the locality where muskmelon wilt is most prevalent, no wilt of watermelon plants has ever been found. During the six-year period from 1932 to 1937, inclusive, both watermelons and muskmelons have been planted on a plot of "muskmelon-sick" soil. The soil is so

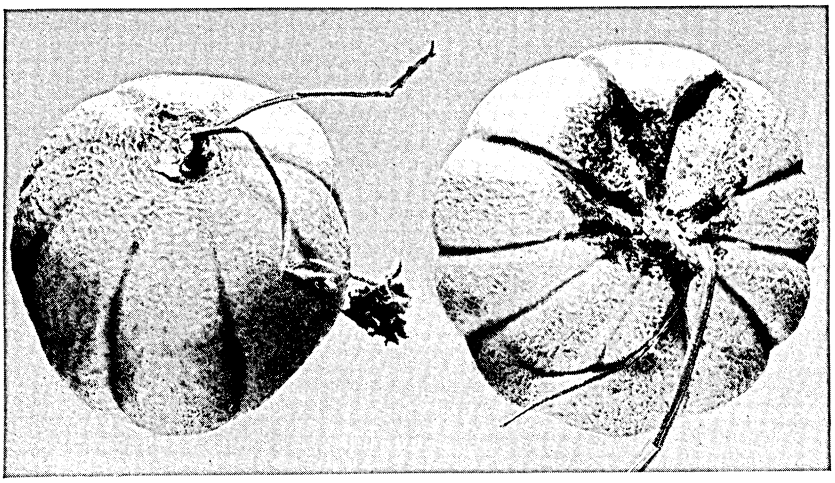

FIG. 4. MUSKMELON FRUITS PARTLY DECAYED BY THE WILT FUNGUS

Two almost mature muskmelons from a plant killed by Fusarium wilt. Note that the pathogen has infected the fruits through the stem end. The pathogen fruits in great abundance in salmon-pink sporodochia or pionnotes. The pathogen also invades the fruit and infects the seed, the fungus being found under the seed coat but not in the embryo.

heavily infested that practically 100 per cent of susceptible varieties of muskmelons are killed by wilt, but during this period not one wilted watermelon plant has been found. In other sections of the state where watermelon wilt is present, muskmelons growing in the same field were not affected.

IDENTITY OF THE MUSKMELON WILT PATHOGEN

The pathogen of muskmelon wilt is very similar morphologically and in cultural characteristics to *Fusarium bulbigenum* var. *niveum,* the cause of watermelon wilt. However, they differ strikingly in pathogenicity. As stated above, cross inoculations and field tests show that the muskmelon pathogen will not infect watermelons and the watermelon pathogen will not attack muskmelons. According to Wollenweber and Reinking (22) who examined a culture of the muskmelon pathogen, it should be classified as a "form" of *F. bulbigenum* var. *niveum.*

Several tested isolates of the fungus were compared on six different media with three cultures of *Fusarium bulbigenum* var. *niveum* from watermelon, and although there were slight differences in growth characters, there was greater variation between different isolations of each pathogen than there was between the two groups of pathogens. In general, the pigmentation of the watermelon pathogen tended to be slightly more intense than that of the muskmelon pathogen, but it was not consistently so on all media.

The size of the spores varied only slightly, the macrospores of the watermelon pathogen being slightly longer than those of the muskmelon fungus measured in comparison at the same time.

In view of the similarity of the two fungi, it is thought best to consider it a new "form" [2] of *Fusarium bulbigenum* var. *niveum*. A brief technical description is given below, and representative spore forms are illustrated in Figure 6.

Fusarium bulbigenum Cke. and Mass. var. *niveum* Wr. f.2.

Differs very slightly from the type form in minor cultural characters, and the observed differences may not be significant or constant. Mycelium is white at first, often later becoming vinaceous purple, or some closely related color, on most media. Color varies from bright red on very acid media to blue on alkaline media. Sclerotia are formed in old cultures, usually blue.

Both micro- and macroconidia are formed, the latter produced in ochraceous salmon pionnotes. In some cultures only microconidia are present. In nature macrospores are often produced in abundance in light ochraceous or salmon-pink sporodochia on affected tissues. Microconidia are one-celled or two-celled, ellipsoid or slightly curved. Macroconidia are two- to five-septate, mostly three-septate, sickle-shaped, pointed at top and pedicellate.

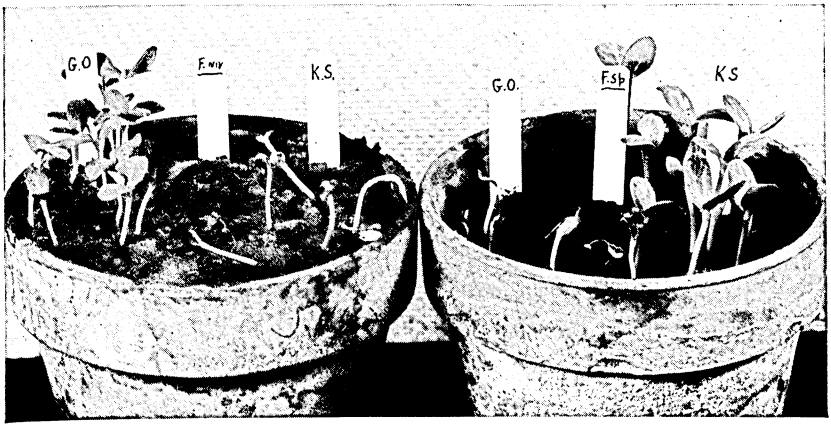

FIG. 5. CROSS INOCULATIONS WITH MUSKMELON WILT AND WATERMELON WILT

A comparative test of the pathogenicity of the Fusarium isolated from wilted muskmelons and the watermelon wilt pathogen. Left, muskmelon seedlings (G.O.) and watermelon seedlings (K.S.) growing in steamed soil infested with a culture of the watermelon wilt pathogen. Right, the same as the pot on the left except that the soil is infested with the muskmelon wilt pathogen. Note the distinct host specificity of the two fungi.

[2] Inasmuch as the two fungi differ essentially only in pathogenicity, they perhaps should be referred to as races, but in order to avoid confusion the terminology of Wollenweber and Reinking is followed.

Typical spherical or oval chlamydospores formed either terminally or intercalary, 5 to 15 microns in diameter.

In what appeared to be a "normal" culture on potato dextrose agar the macrospores with the different numbers of septations occurred in the following frequencies: 2-septate 24.5 per cent, 3-septate 63.5 per cent, 4-septate 9.5 per cent, and 5-septate 2.5 per cent.

The approximate ranges in size in microns of the macrospores on potato dextrose agar are:

2-septate, 20.2 — 40.0 × 2.7 — 4.8, mean 30.7 × 3.9
3-septate, 24.8 — 57.25 × 3.8 — 4.9, mean 42.4 × 4.3
4-septate, 38.0 — 62.6 × 3.8 — 5.4, mean 53.7 × 4.5
5-septate, 45.4 — 77.5 × 4.0 — 5.1, mean 56.6 × 4.6

Three-septate macroconidia produced in nature on melons similar to those shown in Figure 4 were somewhat narrower than those produced on potato dextrose agar, the mean dimensions being 33.8 × 4.3 microns. In a second series of measurements of 3-septate macroconidia produced on potato dextrose agar the mean dimensions were 34.2 × 4.5 microns. General observations and a series of measurements of relatively small numbers of spores from several different sources indicated that there may be considerable variation in length of spores where they are produced on artificial media. The spores appear to be more uniform when produced on affected plants in the field. Representative spores as taken from a "normal" culture on potato dextrose agar are illustrated in Figure 6.

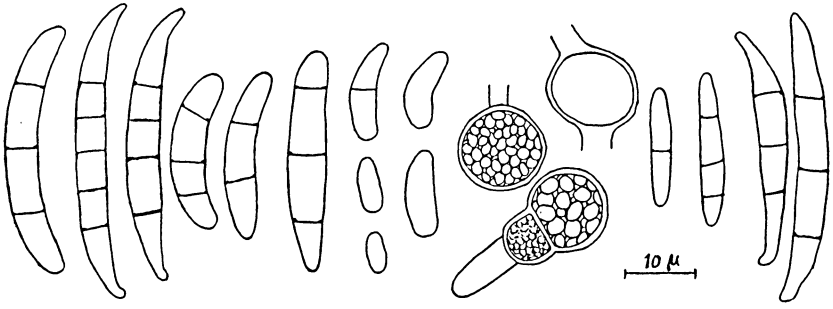

FIG. 6. SPORES OF THE MUSKMELON-WILT FUNGUS

Representative spore forms of *Fusarium bulbigenum* var. *niveum* f.2, the pathogen of muskmelon wilt. A camera lucida sketch.

FACTORS INFLUENCING THE DEVELOPMENT OF MUSKMELON WILT

Muskmelon wilt was first observed in Minnesota in 1931 and 1932, years characterized by hot, dry summers during which Fusarium wilts of many plants were unusually destructive. Since practically all of the diseases of this nature that have been studied are favored by high soil temperatures, it was natural to assume that muskmelon wilt would be

influenced in the same way. It was thought possible that it would prove destructive only in very warm, dry years with high soil temperatures and that with the return of normal weather it would be of little significance. However, in subsequent years much seedling wilt was observed early in the season in cool weather and in soils at relatively low temperatures. Therefore, it seemed desirable to learn more about the influence of soil temperature on the development of the disease.

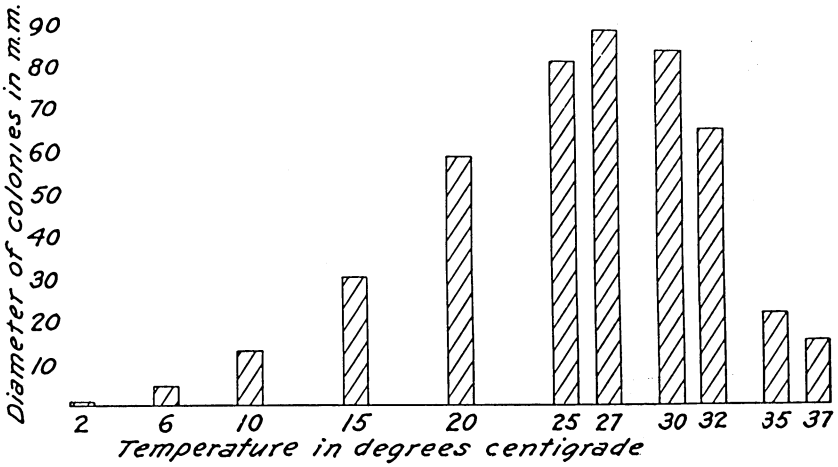

FIG. 7. THE INFLUENCE OF TEMPERATURE ON THE GROWTH OF THE FUNGUS

Diagrammatic representation of the influence of temperature on the growth of the muskmelon wilt pathogen on potato dextrose agar in petri dishes.

Before undertaking the study of the influence of soil temperature, the influence of temperature on the growth of the fungus on agar was determined. Petri dishes, each poured with 20 cc. of potato dextrose agar, were inoculated in the center with the organism and incubated for 18 hours at room temperature, after which they were distributed to incubators maintained at the following temperatures: 6°, 10°, 15°, 20°, 25°, 27°, 30°, 32°, 35°, 37° C. The temperature of the various incubators varied less than one degree from the desired temperature during the course of the experiment. Four petri dishes were used at each temperature, and each set of dishes was enclosed in a moist chamber in order to eliminate insofar as possible the influence of desiccation at the higher temperatures. After six days the growth at 27° C. had practically covered the plate. At this point the colonies were measured, and the average colony diameter at each temperature was determined. The data are presented graphically in Figure 7, and a photograph of a representative set of the plates is shown in Figure 8.

It will be noted that the fungus grew best at 27°. Very little growth occurred at 6° or 37°. The growth curve indicates that the optimum temperature lies close to 27°, the minimum slightly below 6° and the maximum not far above 37°.

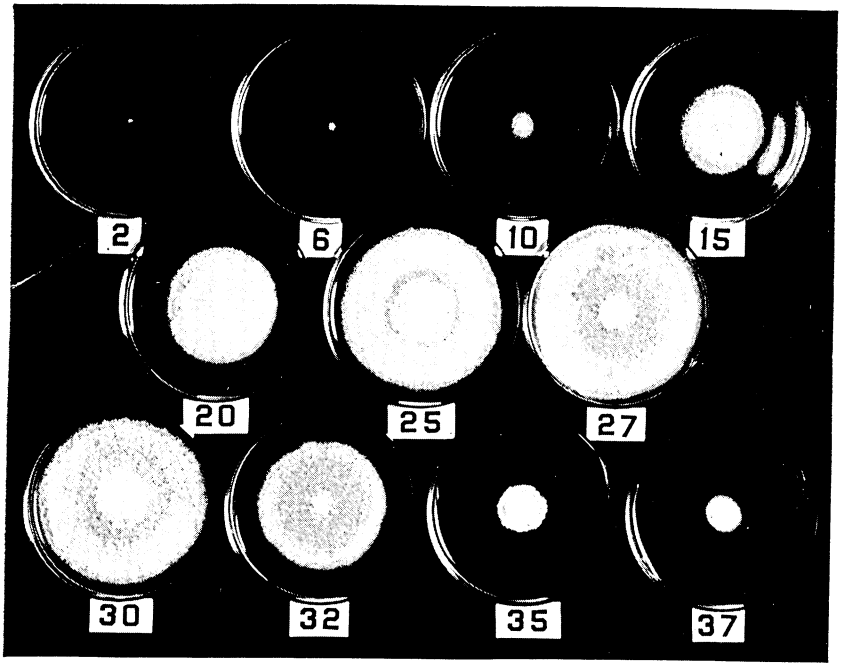

FIG. 8. GROWTH OF THE FUNGUS AT DIFFERENT TEMPERATURES

A photograph of cultures of the muskmelon wilt pathogen growing on potato dextrose agar at different temperatures as indicated.

The influence of soil temperature on the development of the disease was studied in the greenhouse in constant-temperature tanks which have been described elsewhere (3). Two experiments were made, one in February and March, 1935, and one in May and June, 1936. Only five tanks were available for the test in 1935. These were adjusted to the following temperatures: 15°, 20°, 25°, 30°, and 35° C. Temperature readings were made three times daily. The 15° temperature proved to be too low for germination of muskmelon seeds and was not maintained throughout the experiment. The actual mean temperatures maintained for the four remaining tanks were 21.1 ± 0.11°, 25.2 ± 0.07°, 29.8 ± 0.06°, and 35.2 ± 0.08° C. The air temperature in the greenhouse fluctuated from 20° to 28° C. The soil used was naturally infested soil from a "melon-sick" field with a check of similar soil from a nearby field where no wilt had been found. Sterilized soil was not used because of the injury that often occurs on cucurbit seedlings grown in steamed soil. Five pots of infested soil and one pot of clean soil were used at each temperature. Fifteen seeds of Golden Osage muskmelon were planted in each pot, and on emergence these were thinned to 10 plants in each pot. The moisture content of the soil was not accurately controlled, but a fairly uniform and comparable moisture content was maintained by frequent watering with both subsurface and surface irrigation.

Data recorded for each pot included the number of days required for emergence, for the appearance of the first wilt, and for 50 per cent wilt. The total amount of wilt after 35 days, when the experiment was concluded, was also recorded. The means of these data for each temperature are given in Table 2 and illustrated graphically in Figure 9.

Table 2. The Influence of Soil Temperature on the Development of Muskmelon Wilt in the 1935 Experiment

Soil temperature	Days required for emergence	Days required for 50 per cent wilt	Total wilt in per cent
21.1° C.	8.5	14	86
25.2° C.	6.0	9	84
29.8° C.	5.0	16	52
35.2° C.	4.0	22	54

In the 1936 experiment six tanks were adjusted to the following temperatures: 19°, 23°, 28°, 33°, 38°, and 40° C. Forty degrees proved too hot for satisfactory growth of muskmelons. Only a few seeds germinated at this temperature, and data for this temperature therefore are not included. The actual mean temperatures maintained

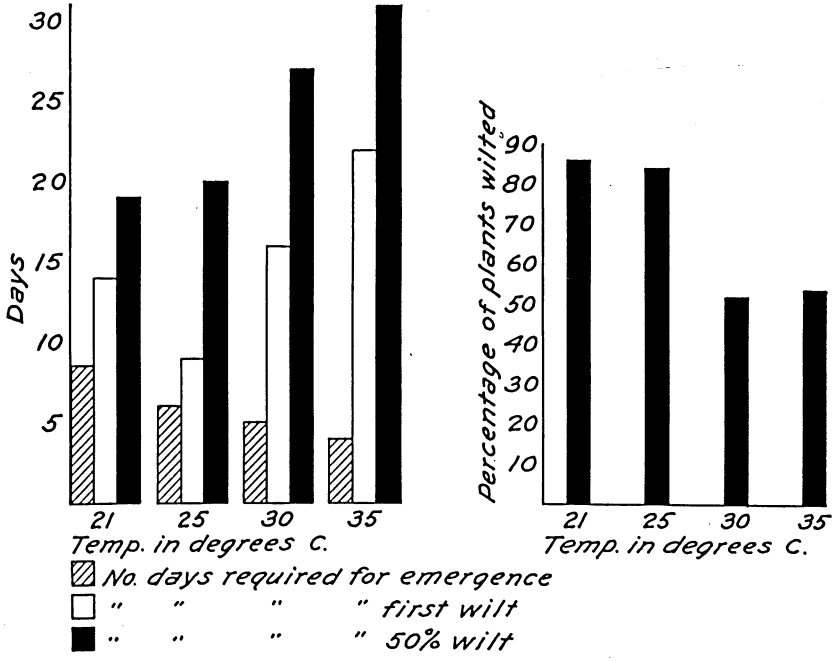

FIG. 9. THE INFLUENCE OF TEMPERATURE ON EMERGENCE AND WILT DEVELOPMENT, 1935

Diagrammatic representation of the influence of soil temperature on the emergence of muskmelon seedlings and on the development of wilt in the 1935 experiment.

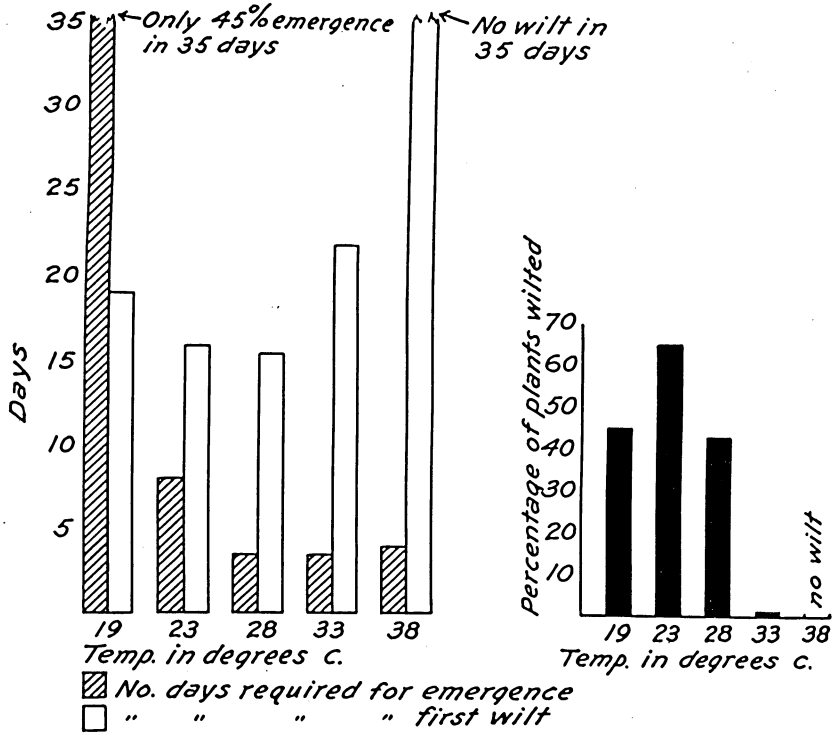

FIG. 10. THE INFLUENCE OF TEMPERATURE ON EMERGENCE AND WILT DEVELOPMENT, 1936

Diagrammatic representation of the influence of soil temperature on the emergence of muskmelon seedlings and on the development of wilt in the 1936 experiment.

in the soils were 19.2 ± .09°, 22.9 ± .07°, 27.7 ± .01°, 32.8 ± .10°, and 37.7 ± .01° C. Naturally infested soil collected from the field in midwinter was used. It proved to be less heavily infested than the soil used the previous year. The check soil was made by mixing a silt loam and sand. It was less fertile than the infected soil, and, although the plants grew well, they were less vigorous in the early stages of growth. Six pots of infested soil and two of clean soil were included at each temperature. The pots were planted and watered in the same way as in 1935, and similar data were recorded. Because infection was less severe, the mean total of wilted plants reached 50 per cent in only one tank. For this reason the time required for 50 per cent wilt was not available. The other data are given in Table 3 and Figure 10. Because of the differences in soil and other environmental factors, the data from the two experiments are not directly comparable and cannot be interpolated, but the conclusions to be drawn from the two experiments are essentially the same. The most important facts demonstrated by the

experiments are that the severity of the disease is not favored by high temperatures and that there is no critical temperature below which the host will grow and escape infection in infested soil. This is in striking contrast to most other Fusarium wilts whose reactions to soil temperatures have been studied. For example, the minimum temperature for the development of tomato wilt is 19° C. (8), for cabbage yellows 17° C. (8), and for flax wilt 14° C. (8). At these minimum temperatures the respective hosts will grow fairly well and escape serious infection. Muskmelon wilt, in contrast, develops extremely well at temperatures too low for good germination of muskmelon seed and growth of the plants. Muskmelon wilt, then, is not merely a hot-weather disease, but severe wilt may be expected in infested soil even though it may be barely warm enough for muskmelons to grow.

Of equal interest is the fact that there is a definite decrease in severity of the wilt at soil temperatures above 30° C., although there is no maximum temperature above which muskmelon plants thrive without becoming infected. This was more striking in the second experiment. In the heavily infested soil of the first experiment there was not significant difference in the final amount of wilt at 30° C. and at 35° C., but it developed earlier and more typically at 30° C. In fact, the symptoms were decidedly atypical on many of the plants recorded as wilted at 35° C., and perhaps they should not have been recorded as wilted.

Table 3. The Influence of Soil Temperature on the Development of Muskmelon Wilt in the 1936 Experiment

Soil temperature	Days required for emergence	Days required for first wilt	Total wilt in per cent
19.2° C.	Less than 50% emergence	19	45*
22.9° C.	8.0	16	65
27.7° C.	3.5	15	43
32.8° C.	3.5	22	16
37.7° C.	4.0	No wilt	No wilt

* Percentage based on number of plants emerged. Much of poor germination at this temperature probably was caused by wilt and if so counted would make this percentage much higher.

These plants did not collapse completely but were recorded as wilted because necrotic symptoms were observed at the ground line when they were pulled up for examination. Further examination of these plants and those remaining at the close of the experiment revealed that many of them were infected in the cortex of the hypocotyl only. Careful microscopic examination of these plants showed that the vascular bundles were not injured, even though most of the cortex of the hypocotyl was destroyed. At the higher temperatures the fungus apparently is unable to penetrate the vascular elements. In many plants the entire cortex and pith had been destroyed, leaving the four vascular bundles separated but each with its own periderm and effectively protected against invasion by the pathogen. The roots of such plants, although not so well

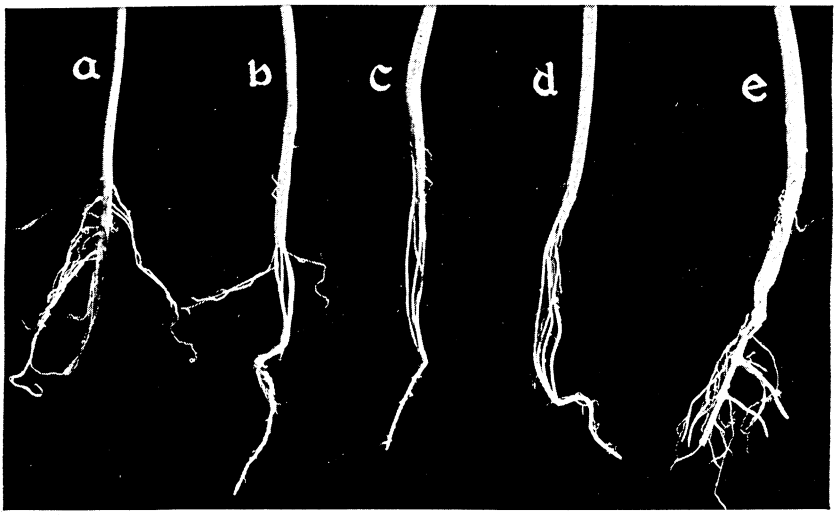

FIG. 11. THE EFFECT OF WILT ON THE ROOTS AND HYPOCOTYL AT DIFFERENT SOIL TEMPERATURES

The hypocotyls and roots of four plants representative of those surviving after growing in the soil temperature tanks for 35 days. *a*, A plant from the 25° tank. Note that the lower part of the root is necrotic although the plant had not yet succumbed to the disease. *b*, *c*, and *d*, Three plants that survived in the 35° tank. Note that the cortex of the hypocotyl has been destroyed but that the four vascular bundles are intact, each having formed its own periderm that has effectively prevented the invasion of the bundle by the fungus. *e*, A plant grown in noninfested soil at 35° C.

developed as those of plants in clean soil, showed no signs of infection. At the higher temperatures the periderm apparently is formed in the roots and around the vascular bundles of the hypocotyl quickly enough to prevent entry of the pathogen. It will be noted from the growth curve of the pathogen that it grows very slowly at 35° C. In Figure 11 representative plants with this type of infection are shown in comparison with wilted plants at lower temperatures and with a healthy plant. In Figure 12 is shown a photomicrograph of a section through the vascular bundles of a plant grown in infested soil at 35° C., and in Figure 13, sections through two bundles, one from a plant grown in infested soil at 35° C. and one from a plant grown in infested soil at 25° C. When these sections were stained with Sudan IV it was found that a separate and distinct periderm had been formed around each vascular bundle in the plants grown at 35° C., while none was found in those grown at 25° C. No mycelium was found in the vascular elements of the former but was abundant in the latter.

In 1937 seeds of a susceptible variety were planted in the field in heavily infested soil. The first planting was made in May and was followed by a period of relatively cool weather. The second planting was made in June and was followed by a period of very hot weather. In the

first planting more than 90 per cent of the plants wilted in the seedling stage, and 100 per cent of the plants died before maturity. In the second planting very few plants died in the seedling stage, and only 57 per cent had wilted when they were killed by frost. When the surviving plants were pulled up and examined, many had the same type of cortical necrosis as those in the higher soil temperatures in the controlled greenhouse experiments. Representative specimens of these plants are shown in Figure 14. During the course of the summer the temperature of the soil at three and four inches below the surface often was above 35° C. In Table 4 are recorded the temperatures of the soil in the melon field as recorded at different hours of the day in the latter part of July, 1937, while these experiments were in progress.

The most probable explanation of the difference in response to soil temperature shown by muskmelon wilt, in comparison with similar diseases of other plants, lies in the higher minimum and optimum temperature for the growth of muskmelons. Muskmelon seed germinated very poorly at temperatures below 20° C. and scarcely at all below 18° C. It will be seen that the pathogen grew fairly well at 15° C. and 20° C. The optimum temperature for the growth of muskmelons was not determined very accurately, but in the 1935 experiment the seed germinated more quickly, and the plants grew best at 35° C. In 1936 germination

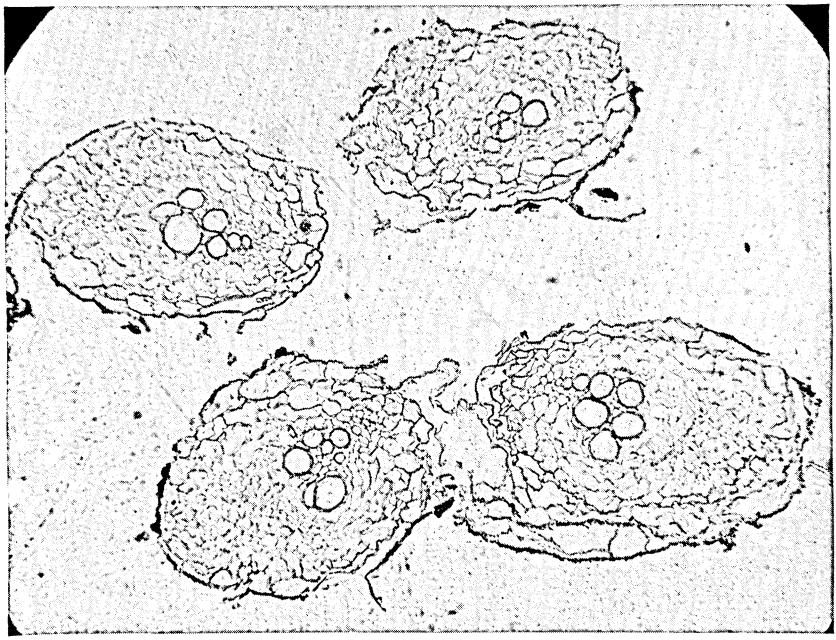

FIG. 12. DESTRUCTION OF HYPOCOTYL CORTEX AT 35° C. SOIL TEMPERATURE

A photomicrograph of a cross section through the hypocotyl of a plant grown in infested soil at 35° C. showing the four isolated vascular bundles, X 70.

was most rapid at 27.7° and 32.8° C. In the early stages the plants grew best at 32.8° and at 37.7° C., but the best prolonged growth was at 32.8°. The optimum temperature probably lies between 33° and 37° C. Thus it will be seen that as the soil temperature rises above 27° it is becoming progressively more unfavorable for the growth of the pathogen and more favorable for the growth of the suscept. This continues until the optimum for the growth of the suscept is reached, at which temperature conditions are very unfavorable for growth of the pathogen. Consequently, the severity of the wilt decreases as the soil temperature rises above 27° C.

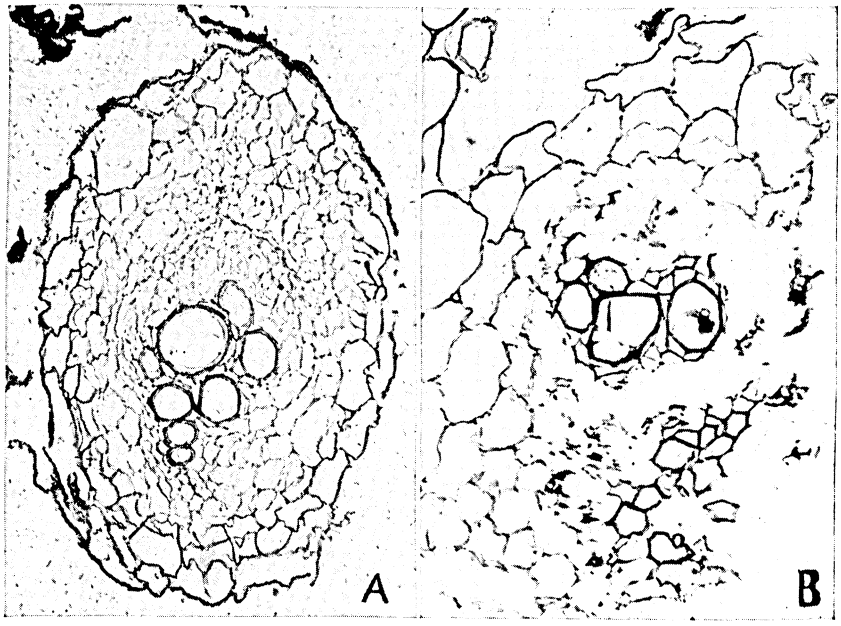

FIG. 13. EFFECT OF WILT ON VASCULAR BUNDLES AT 35° AND 25° C.

Cross sections of vascular bundles in the hypocotyls of: *A*, a plant surviving at 35° C., and *B*, a plant wilting at 25° C. The large cells forming the periphery of the section *A* are suberized as shown by their affinity for Sudan IV stain. In section *B* there were no suberized cells, and the vascular tissue was partly disintegrated. Note the mycelium in the large vessel on the right.

On the other hand, as the soil temperature decreases below 27° C. it becomes progressively more unfavorable for the growth of the suscept until growth is greatly retarded at 20° C. and practically ceases at 15° C. At these temperatures the pathogen will still grow fairly well. Thus wilt is severe at any temperature below 27° C. at which muskmelons will grow.

The influence of soil temperature on muskmelon wilt is similar to that on watermelon as found by Walker and reported briefly by Tis-

dale (18). Walker states, "The results showed that the most favorable temperature for development of wilt lies in the neighborhood of 27° C., that injury, though varying in type and degree, was abundant at all temperatures down to 18° C., where germination of the watermelon seed was very definitely retarded. Above 30° C. practically no wilt occurred, and this temperature was the lower limit for most rapid growth of the watermelon plant itself."

Table 4. The Temperatures of the Soil in the Muskmelon Wilt Plot as Recorded at Different Hours on 10 Days in July 1937

Hour	Depth	Temperature in degrees Centigrade									
		7/13	7/14	7/15	7/16	7/19	7/20	7/21	7/28	7/29	7/30
7:00 a.m.	Surface	33°	29°		23°	33°	29°		26°		28°
	1 in.	30°	29°		23°+	27°	29°		23°		25°+
	2 in.	29°	29°		23°+	24°	27°		22°		24°
	3 in.	28°	28°		23°	22°	25°		21°		23°
	4 in.	28°	28°		23°	21°	24°		21°		22°+
9:00 a.m.	Surface	45°	31°	26°	25°	45°	46°	51°	34°	40°	33°
	1 in.	41°	29°	25°	27°	41°	41°	46°	33°	36°	32°
	2 in.	36°	29°	25°	26°	37°	37°	37°	31°	32°	28°
	3 in.	33°	29°	25°	25°	35°	35°	34°	30°	29°	27°
	4 in.	31°	29°	25°+	25°	32°	31°	31°	28°	27°	25°
11:00 a.m.	Surface	47°	36°	28°	25°	54°	54°	54°	37°	45°	38°
	1 in.	43°	34°	28°	25°	49°	49°	50°	35°	39°	37°
	2 in.	40°	32°	27°	25°	44°	42°	46°	34°	32°	33°
	3 in.	37°	31°	28°	24°	39°	38°	41°	33°	29°	31°
	4 in.	35°	30°+	28°+	23°	34°	32°	35°	32°	27°	29°
1:00 p.m.	Surface					56°	53°		45°	45°	43°
	1 in.					53°	54°		41°	43°	43°
	2 in.					49°	51°		39°	41°	40°
	3 in.					47°	42°		35°	38°	37°
	4 in.						42°		34°	35°	35°
3:00 p.m.	Surface	54°									
	1 in.	51°									
	2 in.	50°									
	3 in.	46°									
	4 in.	42°									

PATHOLOGICAL HISTOLOGY

The muskmelon wilt pathogen, like that of watermelon wilt, is not a strict vascular parasite. It develops readily and spreads rapidly in the vascular tissues, but very soon invades the cortex adjacent to the infected bundles, causing the typical elongated necrotic streaks. The extent of secondary cortex infection varies greatly in individual plants. Some plants will wilt completely before any cortex necrosis is evident. Others will show extensive necrosis before wilting is observed. Humidity and soil moisture seem to influence the rapidity of wilting. In all cases the cortex is rapidly invaded by the fungus as soon as wilting is complete.

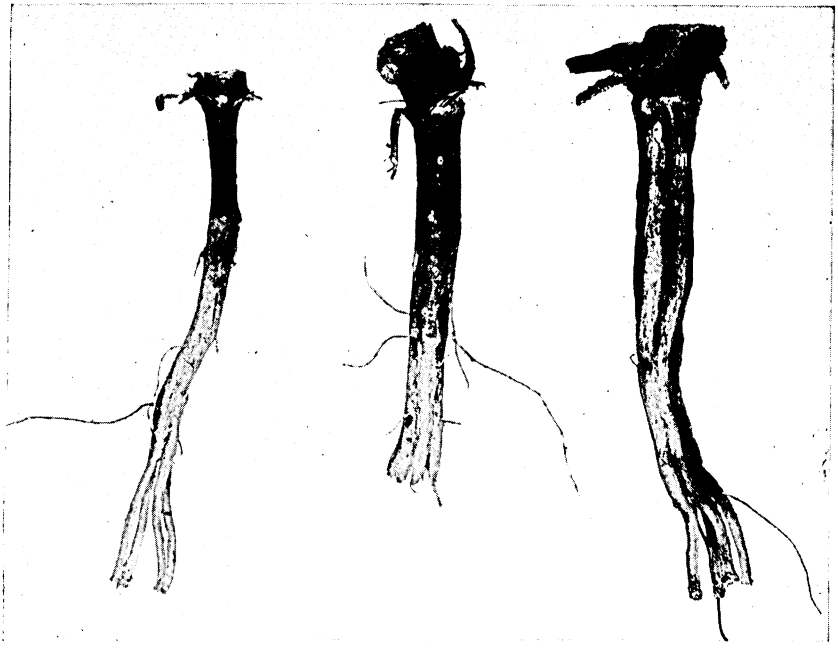

FIG. 14. DESTRUCTION OF HYPOCOTYL CORTEX IN WILT-INFESTED FIELD AT HIGH SOIL TEMPERATURES

The hypocotyls of three muskmelon plants of a susceptible variety planted late and growing in the field in wilt-infested soil at relatively high soil temperatures. The roots have been cut off to show the vascular bundles isolated by the destruction of the cortex tissue. The plants were fairly vigorous and showed no signs of wilt.

The exact method of infection is difficult to determine. Observations on a large number of seedlings indicate that infection may occur through both roots and hypocotyl. The "peg" appears to be a frequent infection point. In some seedlings a necrosis of the tips of the roots is the first histological symptom observed. In others the hypocotyl will be necrotic, the roots showing no external indication of infection (Fig. 2). Still other seedlings may wilt and show no indication of the point of infection, but microscopic examination will show the fungus in the vascular elements of the root or hypocotyl or both. Figure 15 is a camera lucida sketch of the fungus in the vascular tissues of such a plant.

METHODS OF DISSEMINATION

Since the disease appears to be rather limited in its distribution, it is obvious that much loss may be avoided by preventing or retarding its spread. If this is to be done, it is essential that all methods of dissemination be known.

The fungus persists in the soil and infects the underground parts of the plants. Some spores may be produced on the parts of the plant above ground, but this is not the rule. For these reasons the rate of spread would not be expected to be very rapid. Moreover, observations of the writers and the experiences of growers indicate that the pathogen must have time to increase in the soil before it becomes very destructive. This building-up process may require several years of muskmelon culture. Neverthless, wind-borne spores can not be overlooked as a means of dissemination. Because of the obvious difficulties in detecting such spread, little definite information is available.

Dissemination of the fungus in soil adhering to cultivation implements and the feet of animals and man is also strongly probable. Here again a small amount of inoculum transported to a clean field would require several years to build up to destructive proportions, making it difficult to detect the source of infestation. This method of dissemina-

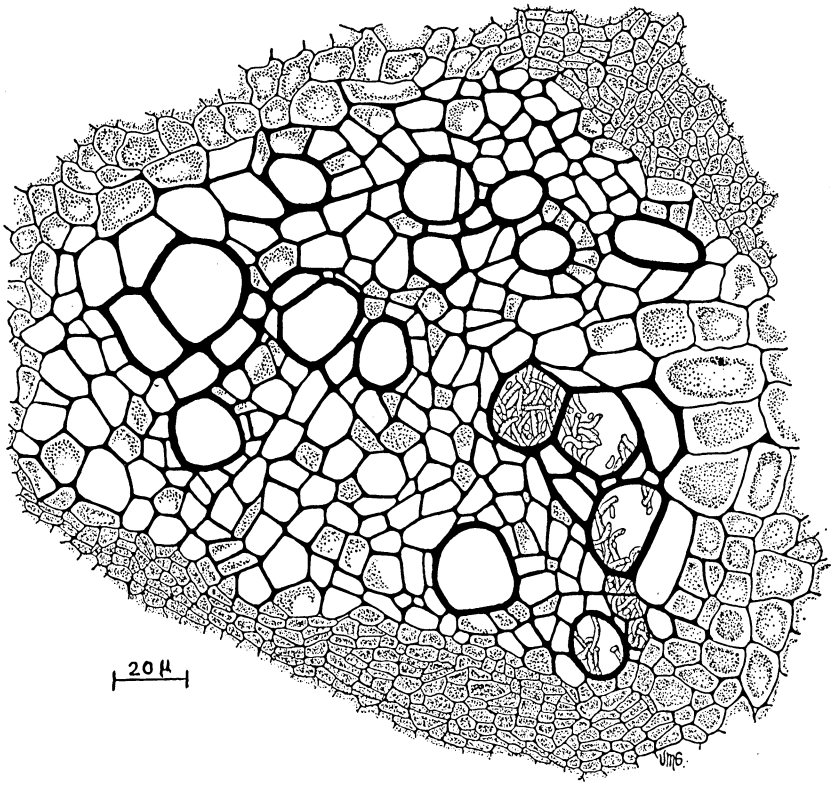

FIG. 15. MYCELIUM OF WILT FUNGUS IN VASCULAR BUNDLE OF MUSKMELON STEM

A camera lucida drawing of a cross section of a vascular bundle of a muskmelon plant in the early stages of wilt. Note the mycelium in the vessels. From a paraffin section stained with thionin and Orange G.

tion, together with wind-blown spores, probably accounts for most of the spread to fields in the immediate vicinity of centers of infestation. Spread in this way would be relatively local, and many years would be required for the disease to become widespread. However, there is always the possibility of new centers of infestation arising in widely separated regions. It would be through such long-distance dissemination that the most extensive spread would be expected.

Dissemination by means of infected seed is the most common means of long-distance dissemination of diseases of this type. For this reason the possibility of dissemination through infected seed was investigated. It was observed that in lightly infested fields plants sometimes escaped infection until they had formed fair-sized fruits. In these plants the fungus frequently invaded the stems and penetrated into the fruits through the peduncle. Two fruits infected in this manner are shown in Figure 4. These fruits had mature seeds, and it was not unreasonable to expect that the fungus might have infected some of them. The fruits were therefore harvested and examined. It was found that the fungus had invaded a considerable portion of the interior of the fruit in each case, and the mycelium was observed in close contact with the seeds. Seeds from the infected portion of the fruits were saved for further study. They were washed free of the placenta, and some were dried thoroughly and stored in a paper bag until ready for further examination. Others were killed in Dietrich's solution and imbedded in paraffin for histological study.

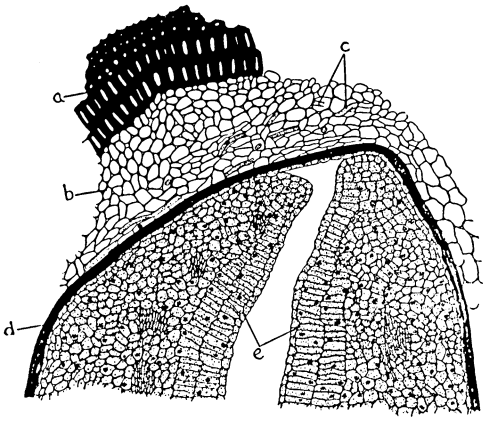

FIG. 16. MYCELIUM OF WILT FUNGUS UNDER SEED COAT

A semidiagrammatic sketch made from serial cross sections of a muskmelon seed internally infected with the wilt fungus. The seed was taken from one of the melons shown in Figure 4. *a*, Schlerenchyma cells of outer seed coat. *b*, Parenchyma of the spermoderm. *c*, Mycelium of the wilt pathogen. *d*, Densely compressed cells of the spermoderm beyond which the mycelium does not penetrate. *e*, Cotyledons of the embryo.

After several weeks, 30 of the seeds were surface-sterilized and planted on potato dextrose agar in petri dishes. Five of the seeds were surface-contaminated, and two others failed to germinate. Of the remaining 23 seeds, 20 remained free of fungus growth while three yielded pure cultures of pathogen. The pathogen did not appear on the agar until after the seeds had germinated, indicating that the infection was

probably internal. This fact was verified by histological examination of the seeds imbedded in paraffin. These were cut in serial sections, beginning at the point of attachment of the seed to the placenta. Fungus mycelium was found in the placenta and could be traced into the seed at the point of attachment. The mycelium in the seed was confined to the parenchyma of the spermoderm and did not infect the embryo. The dense layer formed by the compressed cells of the perisperm apparently forms an effective barrier that protects the embryo from infection. Figure 16 is a composite drawing made from serial sections showing a cross section of the seed with all the layers in place. Note the mycelium in the parenchyma of the spermoderm adjacent to the perisperm. In Figure 17 is shown a photomicrograph of one of the sections used in constructing Figure 16. The mycelium may be seen in the parenchyma at the upper part of the picture. Although the fungus does not penetrate the embryo, it is in a strategic position for infection of the germinating seedling.

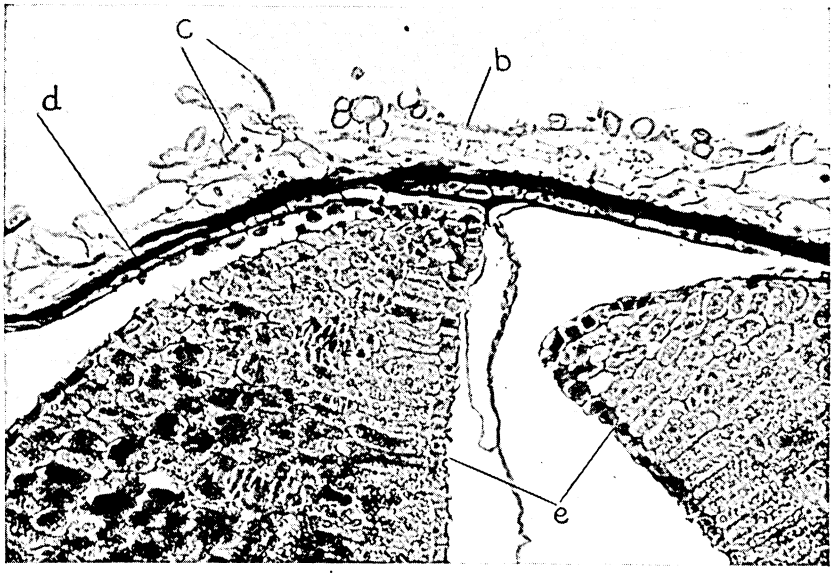

FIG. 17. MYCELIUM OF WILT FUNGUS INSIDE MUSKMELON SEED

A photomicrograph of a portion of the cross section of the muskmelon seed illustrated in Figure 16. The hard outer seed coat has been torn away in this section. *a*, Schlerenchyma cells of outer seed coat. *b*, Parenchyma of the spermoderm. *c*, Mycelium of the wilt pathogen. *d*, Densely compressed cells of the spermoderm beyond which the mycelium does not penetrate. *e*, Cotyledons of the embryo. Approximately X 300.

Further evidence of the internal infection of the seeds was obtained by planting some of the surface-sterilized seed in steam-sterilized soil. Twenty-three out of 30 seedlings were killed by the wilt in the early seedling stage. The mycelium under the seed coat is in a location con-

ducive to early infection of the young plants, and it is not likely that many plants from infected seeds escape infection.

Mycelium on the surface of the seed would probably be only slightly less effective in infecting the young plants. Therefore, it is evident that seed from infected fruits may be both surface-contaminated and internally infected, thus providing an important means of long-distance dissemination of the pathogen. If the disease should become established in the seed-producing regions, there would be a great danger of spreading the disease throughout the entire country.

CONTROL OF MUSKMELON WILT

Muskmelon wilt, like other diseases of similar nature, presents a real problem in control in heavily infested soil, but because of the limited distribution of muskmelon wilt, control in many cases will consist in preventing its spread into new areas. All possible precautions should be taken to prevent the spread of the disease to clean fields on cultivators, horses, or any other agency capable of transporting infested soil. In the infested areas in Minnesota, losses are being reduced temporarily by rotation of crops to avoid planting melons on soil known to be infested. But it will not always be possible to escape losses in this way. Land suitable for muskmelon culture in Minnesota is limited, and eventually more and more acreage will become thoroughly infested until very little clean land will be available. When this condition prevails, the disease will be the limiting factor in muskmelon culture.

During the course of this work, Weindling (20, 21), Bisby, James, and Timonen (2), and Allen and Haenseler (1) reported the pathogenicity of the fungus *Trichoderma lignorum* (Tode) Harz. for other soil fungi and suggested a possible role of this fungus in the control of certain plant diseases caused by soil fungi. It was thought desirable to test the effectiveness of this fungus in the control of muskmelon wilt. A culture of *Trichoderma lignorum* was secured from Dr. G. R. Bisby of the University of Manitoba. The inoculum was increased on oat hulls in one-quart mason jars. In the spring of 1934 four rows of Golden Osage muskmelons were planted in wilt-infested soil. Each row was two rods in length, and the seeds were planted about one inch apart, about 400 seeds per row. Before the seeds were covered, one quart of inoculum of *Trichoderma lignorum* was sprinkled over the seed in two rows. The other two rows were not inoculated. The seed germinated normally in all four rows, but wilt began to appear in all four rows almost as soon as the seedlings were fully emerged. Wilt continued to develop throughout the season, and there was no significant difference in the amount of wilt in any of the rows. None of the plants in any of the rows survived long enough to produce ripe fruit. It was evident from this experiment that the use of this fungus in the control of melon wilt offered little promise, and no further experiments were made with it.

The most satisfactory control measure for wilt diseases of this type has been in the production and use of wilt-resistant varieties. This method has been successful in the control of such diseases as Fusarium wilt of tomato, flax, cabbage, celery, and other crops. In these crops resistant varieties were obtained by selecting resistant biotypes from commercial varieties when grown on heavily infested soil. In the case of watermelon wilt, a disease closely resembling muskmelon wilt and caused by a closely related fungus, resistant varieties have been produced by crossing the resistant but nonedible citron with edible nonresistant varieties. The variety Conqueror was developed by Orton (12) in this way. Resistant varieties of good quality have also been obtained by selecting resistant biotypes within the edible variety Kleckley Sweet. The variety Pride of Muscatine, introduced by Porter and Melhus (13), and the variety Leesburg, introduced by M. N. Walker (19), were derived from resistant biotypes selected from the Kleckley Sweet. Porter and Melhus (13) also produced new resistant varieties (Iowa Belle and Iowa King) by crossing the variety Conqueror with susceptible varieties with desirable commercial qualities.

During the growing season of 1932 the writers initiated a plant-breeding program for the purpose of isolating a strain of muskmelon that might be resistant to the fungus. A preliminary report on the first three years of the program has been made elsewhere (7,10). It was noted that the Persian, Honeydew, Honeyball, and Casaba varieties of melons were relatively resistant. These varieties, however, are not suited to commercial production because of either late maturity or poor quality or both. Such varieties as Bender's Surprise, Emerald Gem, Pollock, and Sugar Rock were highly susceptible. A population of plants recorded as 73-33 that came from a chance hybrid, Honeydew x Bender's Surprise, was found to be intermediate for resistance and other characters. Data obtained on the percentage of plants wilted of the parental varieties and the hybrid strain when grown in comparable rows were: Honeydew, 35 per cent; Bender's Surprise, 96 per cent; and 73-33, 56 per cent. A large number of commercial strains and varieties belonging to the *reticulatus* subspecies have been tested without finding any significant resistance in any of them.

The relative susceptibility of certain varieties representative of those tested in 1934 and previous years is shown in Table 5. It will be noted that the selection 73-33 was less than 50 per cent resistant in this test. By continued selection of selfed and open-pollinated fruits, biotypes have been obtained that are much more highly resistant than the original selection.

Numerous selections made from selection 73-33 have been inbred and reselected each year since the beginning of the work. The selection procedure has had three primary objects: (1) To obtain selections with as much resistance to wilt as possible; (2) to combine resistance with other desirable characters, particularly early maturity, and (3) to purify

the selections so that they would breed true for the desired characters. The general methods used in the breeding work may be of some interest and are outlined as follows:

Year 1932 Seed was obtained from all plants that fruited in a six-acre field of wilt-infested soil, as many as possible of the plants being self-pollinated.

1933 Replicated plantings on diseased soil were made of all selections and numerous commercial strains. Number 73-33 was found to be somewhat resistant, and a number of selections (mostly open-pollinated) were made from this population.

1934 Replicated plantings were again made, and self-pollinations were made on as many plants as possible. Seeds from 150 self-pollinated plants were saved. Very little wilt appeared in the selections made for resistance.

1935 Due to difficulties in making successful pollinations, relatively few plants were self-pollinated (53 in all). All of this seed was kept in addition to approximately 40 open-pollinated selections. Very little wilt appeared in any of the selected strains.

1936 Plantings were made of the 1935 selections and selfings again were difficult, approximately 30 being successful. In order not to lose any of the more desirable germ plasm, a large number of open-pollinated selections were made. Four of the most promising lines were isolated in such a way that cross-pollination, except within the line, was prevented.

1937 Essentially the same procedure as was used in 1936 was followed. Some of the more promising selections were tested on a larger scale for commercial qualities by local growers.

Considerable difficulty has been encountered in making hand-pollinations of the muskmelon flower. The method which is probably used most generally is to cover both pistillate and staminate flowers before they open. When the flowers open, the pollen is applied, and the pistillate flower is again covered until it closes. In the greenhouse this method gives satisfactory results, but outdoors it has not been satisfactory due to the low percentage of the flowers that set fruit.

Another method that has produced fairly good results in self-pollinating has been utilized to considerable extent. It consists of using a cage made by covering a frame of light boards with ordinary window screen. All open flowers are removed from the plant to be pollinated, and the cage is set over the plant. Somewhat later, usually the following morning, a few honey bees are released under the cage. The supply of bees needs to be renewed each day until a fruit has set. Houseflies have been

used as a substitute for bees and have also given good results, but they are not usually as readily available as honey bees. A supply of bees can be kept available for a considerable time by enclosing them in a shipping container and supplying them with sugar solutions. The cages that have been found satisfactory are 2′ x 4′ x 0.5′ and are shown in Figure 18.

The pollination records show that 13.7 per cent of the attempts with the cages were successful as compared to 3.6 per cent for the hand-pollinations. Although the percentage set is still low, the increased success when cages were used is great enough to justify their use in making pollinations.

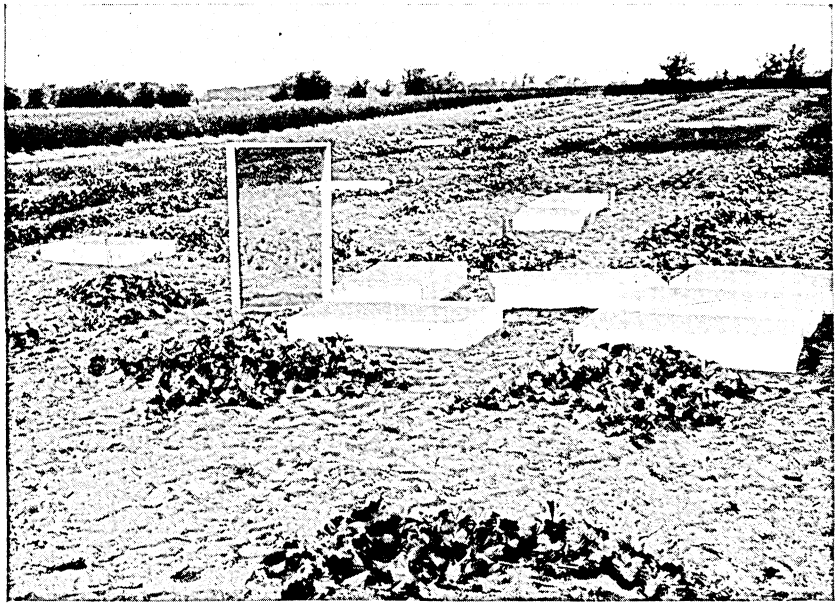

FIG. 18. WILT-RESISTANT SELECTIONS GROWING ON HEAVILY INFESTED SOIL

A view of the wilt-infested plot on which selections for wilt resistance are being made, showing the cages covered with screen wire used in pollination work.

For isolating rows for line-pollinating, a cage is constructed over the entire row, and a supply of bees is kept under the cage until the desired number of fruits have set. The type of cage found least expensive is made by putting aster cloth over wires and securing the wires to a light wood strip that is fastened to the ground. This type of pollination serves to increase the supply of seed of a strain when it has attained the desired uniformity. It is thought from observations made during the course of this breeding work that uniformity can not be safely overemphasized because the plants appear to lose vigor when inbred for several successive generations. However, a more extensive study would be needed to determine definitely the effects of inbreeding.

A brief description of four of the most promising selections is given below:

Selection 29-36: This strain is uniformly resistent to the disease. The immature fruit is gray-green and becomes yellow when ripe. It is distinctly ribbed and slightly netted. The fruit size is about average, and the shape is just slightly longer than thick. The flesh color is bright orange, and most individuals observed had a seed cavity of medium size. Average refractometer reading for six mature melons was 11.5. The plant is vigorous in growth and somewhat coarse in appearance.

Selection 37-36: This is a densely netted, dark green melon that is highly resistant to Fusarium. The fruit is large in size and somewhat variable in shape, from flat to distinctly oval. The size of the seed cavity appears to vary somewhat with different plants. Melons with both green and yellow flesh occurred in this strain in 1936. Refractometer readings for three melons were made and averaged 9.6 for the three.

Selection 43-36: The fruit on this strain is of a light green color similar to the color of a honeydew melon. The size of the fruit is medium, and the shape is oval although some fruits approached being round. The fruit has crisp green flesh and a small seed cavity. The fruit is slightly ribbed but generally is not netted. The vine is coarse and vigorous with deeply serrated foliage.

Selection 81-36: This is a rather small fruited type that has a distinctly oval-shaped fruit. The fruit color is a light gray when immature and changes to yellow when ripe. Smooth to heavily netted fruits were found in the row, and all fruits were distinctly ribbed. The flesh color was yellow with a medium-sized seed cavity. The vine was rather small with large leaves having only slight serrations. Refractometer readings for five melons averaged 12.6.

Growing conditions were not suitable for testing the earliness of these strains, but 81-36 appeared to be somewhat earlier than the other three. None of these selections is yet ready for general distribution.

SUMMARY

1. A destructive Fusarium wilt of muskmelons was observed in Minnesota in 1931. It is a limiting factor in melon production in certain local areas in the vicinity of St. Paul and Minneapolis. It appears to be spreading slowly from the original centers of infection.

2. The disease is very similar to the watermelon wilt and is caused by a fungus that is closely related to the watermelon wilt pathogen but which differs strikingly in pathogenicity. The fungus causing muskmelon wilt will not cause wilt of watermelons, and the fungus causing watermelon wilt will not cause muskmelon wilt. The fungus is described and designated as *Fusarium bulbigenum* Cke. and Mass. var. *niveum* Wr. f.2.

3. Muskmelon wilt apparently occurs also in New York, Michigan, and Arizona and perhaps in a number of other localities in the United States as well as in Ontario and Vancouver Island in Canada. In all cases it is relatively localized in its distribution and has been observed for a relatively short time only. The disease appears to be a new one or one of recent introduction from some unknown source.

4. Muskmelon wilt may affect the plant at any stage of development, causing a seedling blight or a more typical wilt of older plants. It is most destructive in the cooler soils and in early-planted melons. Muskmelons are relatively resistant in soils maintained at temperatures of 30° C. The disease is destructive in infested soils at any temperature below 27° C. at which muskmelons will grow. The pathogen grows best at approximately 27° C. and grows very little below 6° C. and above 37° C. Muskmelons grow best in soils maintained at 30° to 35° C. The seed will not germinate well at temperatures below 18° C.

5. The resistance at the higher soil temperatures appears to result from the rapid formation of a periderm about the vascular elements so that the fungus is not able to penetrate the stele. The cortex of the hypocotyl of plants grown in soil at 30° C., or above, may be completely destroyed by the fungus, but no fungus can be found in the vascular bundles.

6. Infection apparently may occur through any part of the plant underground not adequately protected by a periderm. The "peg" of the germinating seedling appears to be a common point of entrance in young plants.

7. When the plants wilt after fruit has been formed, the fungus often extends into the melon and infects the seed internally. The fungus penetrates the seed through the placenta and is found under the seed coat but not in the embryo. Such infected seed may be disseminated long distances and account for new centers of infection.

8. Control of wilt at the present time depends upon growing melons on noninfested soil and the prevention of its introduction into new areas. The most promising method of ultimate control is the production and use of wilt-resistant varieties.

9. The varieties of melons grown locally in Minnesota (Golden Osage, Bender's Surprise, Emerald Gem, etc.) appear to have little or no resistance to wilt. The Honeydew, Honeyball, Persian, and Casaba varieties appear to be relatively resistant. The latter varieties are not satisfactory for commercial production in Minnesota because of late maturity or poor quality or both. Promising new varieties of early maturity and good quality and highly resistant to wilt have been obtained by selecting from the progeny of a hybrid between the Honeydew and Golden Osage varieties. Among these are selections with green flesh of the Honeydew type and some with yellow flesh resembling the Golden Osage variety and many intergrading types. Further selection and inbreeding for purity is necessary before the new wilt-resistant varieties will be ready for distribution.

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