Physiology of Fusarium Foot Rot Of Squash

George A. Gries

CONNECTICUT AGRICULTURAL
EXPERIMENT STATION
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GEORGE A. GRIES

Squash foot rot flared up so suddenly in the northeastern United States in the late 'thirties that it was alarming to growers. Knowing that root rot diseases in general are difficult to combat, everyone feared that squash growing in the area might be depressed to an uneconomic level. Research by several laboratories soon demonstrated methods of control that caused the disease to decline in destructiveness just as dramatically as it had risen. Now squash foot rot seldom occurs in Connecticut.

The first characterization of squash foot rot was published by Doidge and Kresfelder (1932) in South Africa. It was first reported in the United States from California by Snyder (1938) who worked extensively, not only upon the disease, but upon the taxonomy and morphology of the causal organism.

Foot rot of squash appeared in Connecticut and New York State at approximately the same time. The first published report of the occurrence of the disease in the East (New York) was that of Chupp (1939). When foot rot first appeared in Connecticut, the immediate need of practical information for combating the disease prompted Dr. Florence E. McCormick to undertake studies on the life history of the causal organism and the control of the disease. This work was continued by her until her retirement in 1941 when it was subsequently taken over by the present author.

There were two basic reasons for taking up the research on foot rot. First, it was a serious local disease on which information was needed and, secondly, it served as a segment in a larger research aimed at discovering additional basic information on root diseases in general. Experimental work had indicated that potato scab (Gries, Horsfall and Jacobson, 1944, and Schroeder and Albrecht, 1942) and club root of cabbage were influenced by the ratio of potassium and calcium in the soil independently of soil acidity relations. Squash foot rot proved to be a similar case.

The role of decomposing organic materials as a causative in non-parasitic plant diseases and as an influencing factor in parasitic diseases was another phase of the general root disease problem under study. Since it had been shown (Gries, 1942) that the by-products of rotting organic residues influenced the physiological functions of many parasites, this approach was also followed in the studies on the survival of the squash foot rot organism in the soil.

[1] The author wishes to express his thanks to Dr. James G. Horsfall for his valuable suggestions during the course of these investigations and for his assistance in the preparation of the manuscript.

SYMPTOMS AND CAUSE OF THE DISEASE

Symptoms on the Lower Stem

The first symptom of the disease in the field is a wilting of the leaves of the host plant similar to the wilting due to the bacterial wilt disease or to that attending a heavy infestation of the vine borer. Within a very few days, and frequently within a matter of hours after the first wilting is noticed, the entire upper portion of the plant collapses. Upon close examination of the basal part of the stem, a soft, sometimes mushy, area may be distinguished even before there is any change in color. As the disease progresses, the infected area becomes first a light and then dark brown as the parenchymatous tissues become completely destroyed. About this stage it is not at all uncommon to see a whitish or pinkish mass of fruiting mycelium of the fungus both on the rotted regions of the stem and on the surrounding soil. As the rot progresses, undoubtedly aided in part by other saprophytic fungi and bacteria, the tissue becomes drier until finally only the fibrous anastomosing strands of the vascular system remain.

The symptoms are similar on plants of any age, except that when plants are attacked in the seedling stage, the vascular system is also frequently destroyed, since it has not as yet become heavily lignified. The fungus does not usually affect the root system nor does it often progress far up the stem. The level on the stem at which the rotting occurs appears to be governed largely by the atmospheric and soil moisture, possibly as these two factors affect the aeration of the fungus' microclimate. Under conditions of very wet soil and high atmospheric humidity, the rotting may occur two to three inches above the soil line. Under dry soil conditions, however, the rotting is commonly below the soil level at a depth where the soil is in good tilth. Only this lower stem region or "foot" is commonly attacked. Hence, the name "foot rot".

An advanced stage of wilting as it appears on a mature plant is shown in Figure 1, while various stages of the disease are shown on the potted seedlings in Figure 2. The growth of the fungus on the surface of the soil surrounding a diseased stem is clearly shown in the latter illustration.

Symptoms on the Fruit

The fruit-rotting stage of the disease is not easily distinguished from the rots caused by many other fungi which gain entrance to the fruit from the soil. The first symptom to appear is a soft water-soaked area which later assumes a light brown color. As the fruit surface begins to dry out, there appear the characteristic white to gray zonate or radiating areas that are common to many fruit-rotting Fusaria. As these areas become more numerous, they may coalesce until finally the entire surface of the fruit may be covered. Following these initial lesions, the entire fruit, except for the skeleton, undergoes disintegration as a very soft watery rot. After this stage, with the cutinized surface apparently lacking or cracked, the fruit desiccates until only the dry shell remains.

The symptoms of the fruit-rotting stage of this disease as it occurs in the field may vary considerably from the above description since the over-all appearance of the rotting fruit depends not only on

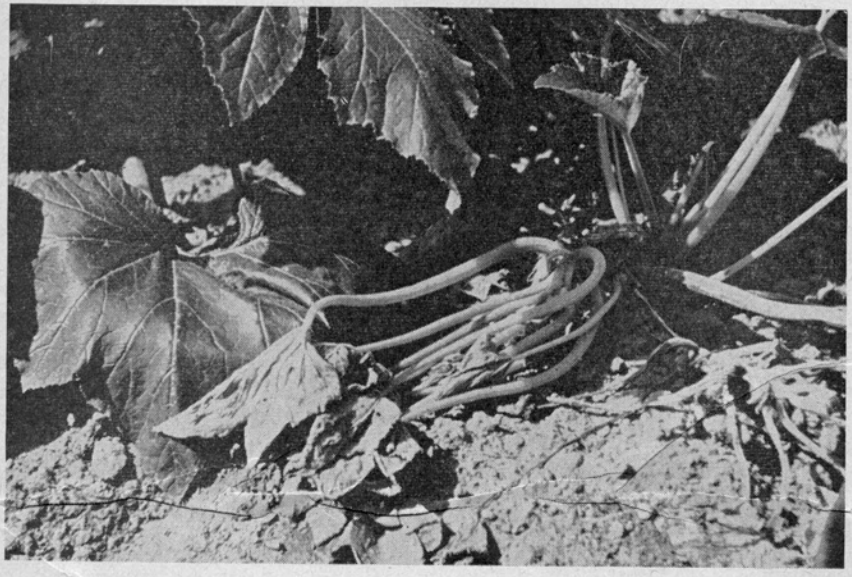


Figure 1. Typical field symptoms produced by the foot rot Fusarium on a mature Early Prolific summer squash plant.

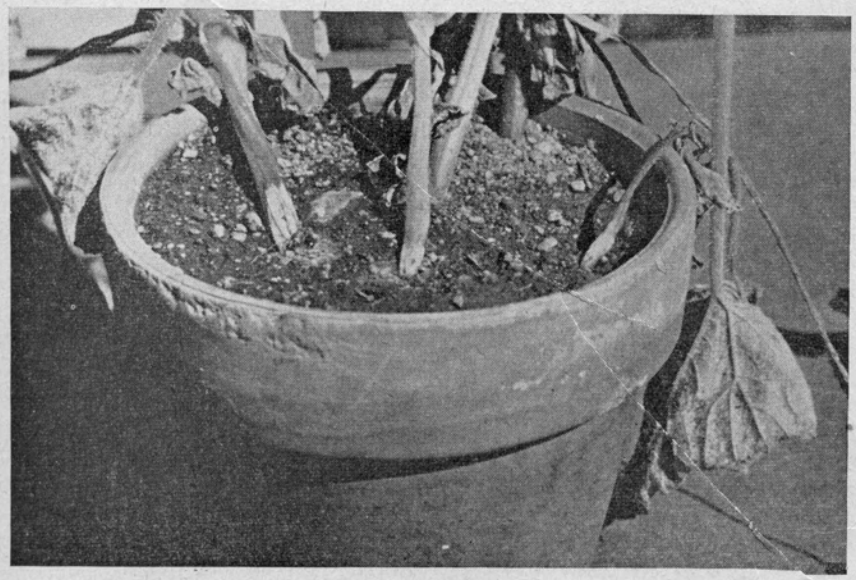


Figure 2. Foot rot symptoms on Early Prolific summer squash seedlings. Note the growth of the fungus on the soil surface around the base of the stems.

the foot rot fungus, but on the multitude of other fruit-rotting fungi and bacteria which are associated with the rot as it occurs in the field. Often in rotting fruits a yellowish vile-smelling slime develops as a result of the activity of certain common bacterial saprophytes.

The Causal Organism

In an effort to be consistent with the bulk of the literature published on this disease and its causal organism in the United States, the name *Fusarium solani*, f. *cucurbitae* has been used throughout this paper. This is the name proposed by Snyder and Hansen (1941).

THE HOST RANGE

Snyder (1938) reported the disease on *Cucurbita pepo*, *C. moschata*, and *C. maxima* and certain varieties of these species. He further noted that the Italian Zucchini squash is particularly susceptible. In Connecticut, the disease has been almost entirely limited to the yellow summer squash varieties (*C. pepo*), and to progeny of crosses involving this as one of the parents. A limited number of greenhouse inoculations have shown its host range to coincide with that listed by Snyder. No evidence of any resistance in the varieties of common summer squash has been noted.

TRANSMISSION AND INOCULATION

Seed Transmission

Almost as soon as the disease was observed in 1939, it was obvious that the fungus had entered the State on contaminated seed. It appeared throughout Connecticut and in nearby states the same year and attacked plants from certain seed lots and not from others. In some fields the diseased and healthy seed lots were side by side.

Examination of seeds soon showed the fungus to be present either as spores on the outside or as mycelium in the seed coat. In a few cases, we have observed it in the embryo of viable seeds, usually as mycelium, although occasionally the fungus when thus found is producing abundant spores. From these sources the fungus parasitizes the young plant growing from the infected or infested seed, girdling and rotting the young stem close to the soil surface. The fungus, once established in the soil from diseased seedlings, or from diseased non-viable seeds, may be spread in the field either mechanically through cultivation or by the growth of the fungus on organic debris in the soil. The spores of the fungus may also be spread by insects and by surface water.

Mechanism of Seed Contamination

The problem to be solved, of course, was the mechanism by which the seeds became contaminated. Inspection of stems on diseased

plants soon showed that the discoloration extends only a very short distance up the stem—never into the fruits. Clearly, then, the seeds do not become contaminated systemically through the pathway of the vascular elements. The evidence all points to the rotting fruit as the source of contamination. The seeds in rotted fruit may be actually penetrated by the mycelium or the seed may simply be coated with spores.

If this were the only source of contamination, the percentage of diseased seeds in commercial stocks would be small, because normally seeds would be saved only accidentally from diseased fruits. Probably, the greatest of all sources of contamination is the fermentation vat. Squash seeds are normally freed of pulp by a fermentation process. A few diseased fruits may supply sufficient inoculum to contaminate the entire vat of seeds.

The role of insects in the dissemination of the disease has been studied in detail only partially. Dr. Beard of this station showed that the squash bug (*Anasa tristis*) probably does not act as a vector of squash foot rot (Beard—unpublished data). Two other insects, the squash borer (*Melittia satyriniformis*) and the striped cucumber beetle (*Diabrotica vittata*) may possibly act as disseminating agents and should be studied from this angle. The injuries made on the vines by these two pests undoubtedly serve as infection courts for the fungus.

Efficacy of Seed Treatment

Some success has been attained in experiments in which the seeds have been treated with hot water to reduce the amount of infection. During the winter of 1943-44, samples were taken from a stock of heavily contaminated Early Prolific yellow straight-neck summer squash seed. These samples were hot water-treated over a wide range of time-temperature combinations from temperatures of 50°C. to 65°C. and at time intervals from five to 20 minutes at each temperature. The seeds were planted in uncontaminated soil in small flower pots to minimize the effect of the spread of the organism through the soil.

In Table 1 the results for three of the most effective time-temperature combinations and for the check are shown.

TABLE 1. EFFECT OF HOT WATER SEED TREATMENT ON THE EMERGENCE AND SURVIVAL OF SEEDLINGS OF EARLY PROLIFIC SUMMER SQUASH

Treatment	No. of seeds treated	No. of seedlings emerging	No. of diseased plants		Per cent survival of emerging seedlings	
			After 4 weeks	After 6 weeks	After 4 weeks	After 6 weeks
No treatment	200	111	111	111	0.00	0.00
55°C.–15 min.	200	42	7	9	83.33	78.57
60°C.– 5 min.	200	87	34	45	60.92	48.28
60°C.–10 min.	200	60	28	28	53.33	53.33

From the above table it is apparent that it is possible to reduce substantially the percentage of infection of the emerging seeds by

using the proper time-temperature values. This cannot be done, however, without sacrificing a relatively high percentage of viability. The percentage of infected viable seeds is probably lower than could be discerned from the data on emergence since many seedlings are frequently killed by the fungus between the time of actual germination of the seeds and the time they emerge through the soil.

Although the design of the experiment discussed above does not permit a distinction between surface-borne and imbedded contamination, other tests seem to indicate that surface-borne infection can be eliminated without a great reduction in the germination of the seeds. On the other hand, when surface-sterilized seed is used, the degree of reduction in the number of viable seeds closely parallels the number of seeds showing true seed infection in the seed coat or embryo. Thus, it would seem that the lethal time-temperature constants are very similar for the embryo of infected seeds and for the imbedded fungus tissue, if indeed it is not lower for the embryo.

Sterilization of the seed in 1-1000 bichloride of mercury for 15 minutes followed by thorough rinsing in water gave satisfactory results only when the seed infection was primarily of the surface-borne type as indicated by microscopic examination. It would thus seem that when it is absolutely essential to reduce the infection in a quantity of seed, the hot water treatment is preferable to a corrosive sublimate treatment or to a combination of the two.

The use of copper or organic seed protectants in combating surface-borne spores has not been studied in detail, but none of the materials tried has been effective in reducing penetration of healthy seedlings by the fungus in heavily contaminated soil.

Effect of Age of Seed

As is the case with most seed-borne plant diseases, squash foot rot is most easily and efficiently controlled by the use of disease-free seed from fields in which the disease has not been known. Since it is not possible to distinguish diseased seed without microscopic examination or a culturing procedure, seedsmen have found another method for insuring relatively clean seed. They have learned through experience that seed two or more years old when planted does not produced diseased seedlings. One explanation forwarded for this "cleaning-up" of the seed is that the fungus infection dies out. This hypothesis seems to be tenable for the surface-borne type of contamination, which probably does desiccate and lose its viability. A more probable explanation of the "self-sterilization" of those seeds in which the mycelium, alone or with spores, is in the seed coat or embryo is that the fungus itself destroys the germinating power of the seed, probably through the digestion of the embryo tissue. We have in several cases succeeded in culturing the fungus from samples of old seed which when planted did not give seedling infection. Apparently, the fungus-containing seeds do not germinate and the fungus present does not spread rapidly in the soil.

EFFECT OF TIME AND MANNER OF INOCULATION IN THE FIELD

Experiments have been conducted on the effect of time and position of inoculation on the amount of seed contamination. Inoculations were made by injecting one cubic centimeter of a spore suspension of the fungus (containing approximately 50,000 spores) into three parts of the fruit: pedicel, basal fleshy portion, and seed cavity. Inoculations were made at all positions at approximately five-day intervals between the date of flowering and the date of maturity. In nearly all experiments the fruits were left on the vine until they were mature (five to six weeks).

The seeds were fermented inside each of the inoculated fruits, washed, dried and stored over winter. The following summer, they were planted in disease-free soil. Three kinds of data were collected (Table 2): (1) number of fruits producing at least 5 per cent of normal seeds, (2) number of seedlings that failed to emerge as a result of the disease or other factors and, (3) number of emerged seedlings showing disease.

Assuming that the disease killed the seedlings that did not emerge, the total of the last two will give the total infection of the apparently normal seeds. Unfortunately, the total number of seeds set was not determined. From that number could have been determined the effect on seeds while they were still within the fruit.

In any event, two marked trends show in the data in Table 2. The percentage of disease in the seeds decreased with the age of the seeds and with the distance between the point of inoculation and the cavity where the seeds were borne.

The rate for these trends is interesting. The quantitative effect of distance is difficult to appraise because the errors are large. The distance from one side of a seed cavity to the other may be as great as the distance to the pedicel.

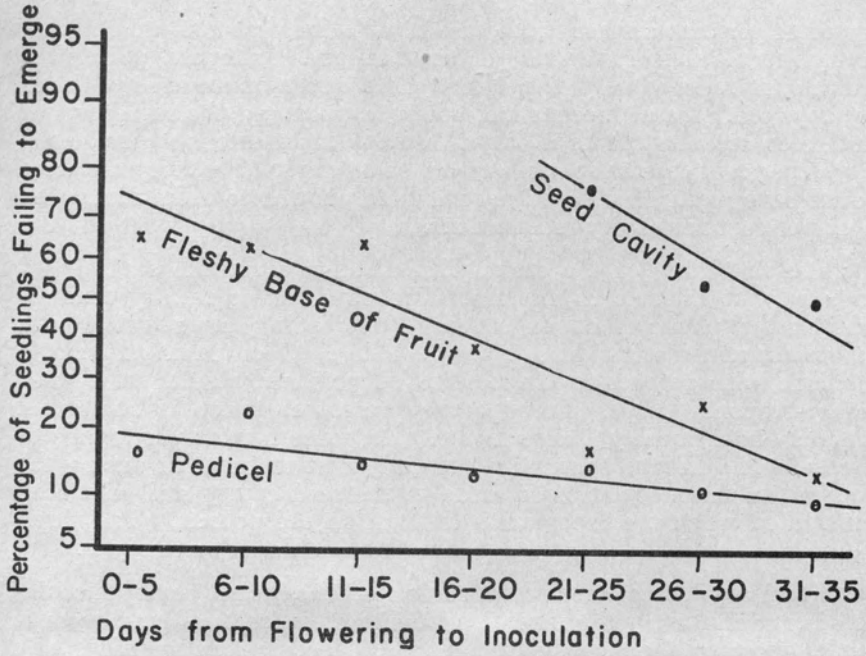
That error does not occur in the data on time, however. A graphic exploration of the data very soon reveals that the best straight line for the time response may be had with arithmetic probability paper using percentage of disease on the probability axis.

The available data, therefore, were plotted (Figure 3). Although variability about the line is understandably large, there seems to be no suggestion of other than linearity for any line except that for the data on emerged seedlings from fruit inoculated at the base.

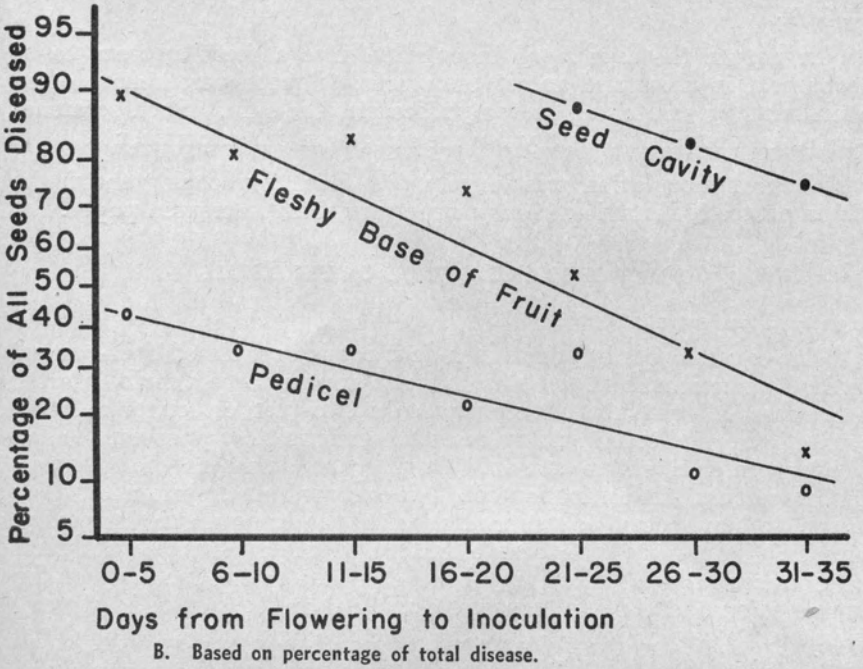
Two results are obvious: First, the curves are displaced progressively to the left and flattened as the distance from the seed cavity increases. The displacement may be viewed in two lights (1) that the percentage of contaminated seeds decreases with distance or (2) that a longer time is required as distance increases to give the same amount of disease. To take the case of total disease when inoculations were made 26 to 30 days after blooming, pedicel inoculations gave an aver-

TABLE 2. THE INFLUENCE OF THE AGE OF THE FRUIT AND THE PLACE OF INOCULATION WITH SPORES OF THE FOOT ROT FUNGUS ON THE VIABILITY AND DEGREE OF CONTAMINATION OF EARLY PROLIFIC SUMMER SQUASH SEEDS

Days from flowering to inoculation	Location of inoculation	Number fruits inoculated	Per cent fruits producing 95 per cent diseased seeds	Number normal seeds planted	Per cent seedlings failing to emerge	Per cent post-emergence disease	Per cent all seeds diseased
0 – 5	Pedicel	17	11.8	750	15.6	31.4	42.1
	Fleshy Base	13	84.6	200	65.5	68.1	89.0
	Seed Cavity	10	100.0	...	...	...	...
6 – 10	Pedicel	11	0.0	550	23.1	11.8	32.2
	Fleshy Base	11	63.6	200	61.5	46.8	79.5
	Seed Cavity	8	87.5	100	100.0	...	100.0
11 – 15	Pedicel	15	6.7	700	13.6	22.0	32.6
	Fleshy Base	15	73.3	200	64.0	51.4	82.5
	Seed Cavity	9	88.9	100	95.0	100.0	100.0
16 – 20	Pedicel	4	0.0	200	13.0	10.3	22.0
	Fleshy Base	4	50.0	200	37.0	56.3	72.5
	Seed Cavity	6	100.0	...	...	...	100.0
21 – 25	Pedicel	5	0.0	250	13.5	4.6	34.0
	Fleshy Base	6	0.0	300	16.0	17.2	53.7
	Seed Cavity	7	57.1	150	76.5	61.7	88.0
26 – 30	Pedicel	2	0.0	100	11.0	0.0	11.0
	Fleshy Base	5	0.0	250	23.6	12.6	33.2
	Seed Cavity	5	20.0	200	52.0	63.5	82.5
31 – 35	Pedicel	5	0.0	250	9.2	0.4	9.6
	Fleshy Base	6	0.0	300	12.3	1.1	13.3
	Seed Cavity	6	0.0	300	50.7	47.3	74.0



A. Based on percentage of seedlings failing to emerge.



B. Based on percentage of total disease.

Figure 3. Effect of age of fruit and location of inoculation on percentage of disease in seeds.

age of 11 per cent disease, fruit base inoculations gave 33 per cent and cavity inoculations gave 82.5 per cent disease. Likewise, pedicel inoculations produced only 42 per cent disease even on seeds as young as 2½ days old. Basal inoculation, however, produced the same level of disease on seeds as old as 25 days. Cavity inoculations were producing 74 per cent disease in seeds as old as 33 days. Probably the seeds would have matured before they were old enough to reduce infection to 42 per cent for cavity inoculations.

The flattening of the curve with distance from the seeds is intriguing, but difficult to explain. The situation is faintly reminiscent of the phenomenon of coverage now being investigated with fungicides and insecticides (Horsfall, 1945). Good distribution of a pesticide with respect to the pest to be poisoned results in a steep dosage-response curve. Perhaps the present situation is analogous. Time is dimensionally a dose factor. Response here is percentage of disease. The fungus spores are certainly best distributed among the seeds when the inoculation is made in the seed cavity and least well distributed when the inoculation is made in the pedicel. The slope, therefore, should be flattest for pedicel infection as it certainly is.

From a practical viewpoint, these results say that cavity inoculation is far and away the most drastic site of inoculation and from it results most of the seed contamination. Inoculations in the fleshy base were much less effective and inoculations in the pedicel were least effective.

In nature the avenue of inoculation into a fruit appears to be mechanical injuries and insect punctures. Squash researchers often label fruits by scratching hieroglyphics into the skin. Such scratches were often the point of entry for the fungus in our experiments.

Natural inoculation was largely prevented by coating the fruits with a protective fungicide or by wrapping them in treated paper.

EFFECT OF SOIL CONDITIONS ON THE DISEASE

Longevity of the Fungus in the Soil

The causal organism of squash foot rot is capable of over-wintering in Connecticut soils, but this does not appear to be a serious factor in commercial squash production when a two-year or longer rotation is practiced. Indeed, in many cases the fungus has apparently failed to over-winter a single season. During the summers of 1943 and 1944, no natural field infection could be obtained on experimental plots known to have been contaminated with the fungus the preceding summer. This may have been a failure of the fungus to withstand the Connecticut winters or may have been due to the inability of the fungus to spread through the soil during those two exceedingly dry summers. The possibility that the fungus could not withstand the association effects of other soil organisms is tendered considerable credence by an experiment conducted simultaneously.

Effect of Organic Matter

In the spring of 1943 naturally contaminated fine sandy loam, low in organic matter, was collected and divided into three portions. One portion was mixed with an equal volume of sand, one with an equal amount of well-composted squash vines, and one received no amendment. These three soil types were placed in screen-bottomed boxes 10″ x 3″ x 8″ and buried to the surface level of the soil. Each box was given a "booster" inoculation of Fusarium spores. At intervals during the next two years, representative boxes were removed and taken to the greenhouse. There the soil was potted and planted with uncontaminated squash seed. Disease readings on the plants grown in these soils were indicative of the longevity of the fungus in the three soil mixtures. In Table 3 is shown the presence of *Fusarium solani* f. *cucurbitae* in the field soil, humus-amended soil and sand-amended soil as determined by the incidence of foot rot over a period of two years following preparation of the soil types.

TABLE 3. LONGEVITY OF *F. solani* f. *cucurbitae* IN THREE SOIL TYPES AS INDICATED BY THE ABILITY OF THE SOIL TO PRODUCE DISEASE ON HEALTHY SEEDLINGS
Two samples were used for each test

Date of test	Months from test initiation	Field soil	Sand-amended soil	Humus-amended soil
November, 1943	6	++	++	++
March, 1944	10	++	++	++
October, 1944	17	++	Not tested	00
April, 1945	23	++	++	00
August, 1945	27	0+	++	00

It is especially interesting to note that the fungus did not persist as long in the humus-amended soil as it did in either of the other two soil types. Either of two theories might explain this behavior. First, the increased microbiological activity of saprophytic organisms might usurp the nitrogen supply and effectively starve the pathogene for this essential element. This is known to occur in the case of certain other soil-borne parasites. Soil analyses run on the various samples, however, refute this theory since they showed a higher available nitrogen content in the humus-amended soil than in either of the other two.

The remaining theory advanced here to explain the relatively short existence of the foot rot Fusarium in the soil with a high organic matter content is that of antibiosis. Waksman and Horning (1943) and other authors have pointed out the omnipresence of soil organisms which produce antibiotic substances. It is quite probable that in the organic-amended soils, such organisms thrived and produced substances toxic to *F. solani* f. *cucurbitae*. In the field soil and sand-amended soil, the biological activity of such organisms must necessarily have been lower because of the limited nutritive conditions prevailing there.

The results of this experiment coincide with several observations made on the survival of the foot rot fungus in soil and in organic compost. In one case, year-old compost originally containing diseased squash fruit and vines failed to serve as inoculum when transferred to the greenhouse for experimental purposes. This observation lends support to the antibiosis theory since this compost had been prepared after established commercial procedures and thus always should have contained a sufficient supply of available nitrogen.

In both of the cases mentioned above, when no natural infection occurred the next season after a heavy infestation of foot rot the preceding year, a heavy crop of rye had been turned under in the spring. This additional organic matter may have been responsible for the failure of the disease to develop.

Effect of Other Microorganisms

In all cases of squash foot rot found at our Mount Carmel experimental farm during the summers of 1942 and 1943, an unidentified species of bacterium seemed invariably to be associated with the Fusarium in the rotting portions of the stem. It was thought advisable to test the pathogenicity of this organism to squash, both alone and in combination with the fungus. To attain this end, four groups of 10 mature plants each were inoculated on June 22, 1943, by means of injection with a two-cubic centimeter laboratory syringe into the stem base with the following inocula.

Each plant from one group of ten was inoculated with two cc. of a suspension of *Fusarium solani* f. *cucurbitae* spores at the rate of 1,000 spores per cc. The plants in the second group each received two cc. of a suspension of cells of *Bacterium* sp. at approximately 5,000 cells per cc. A third group of plants were inoculated with one cc. of a Fusarium spore suspension containing 2,000 spores, and with one cc. of a suspension of bacterial cells containing 10,000 cells. The plants in the fourth series were injected with two cc. of sterile distilled water.

Disease readings were made on these plants at intervals for five weeks, and the severity of the disease scored into four groups: (S) plants showing a softening around the point of inoculation; (R) plants showing a visible rotting of the basal portions of the stem; (W) those definitely wilting in addition to rotting; and (D) those plants which had collapsed and could be considered dead. These groups are artificial to be sure and do not represent equal time increments in the development of the disease after inoculation. The wilting stage especially is misleading since this symptom is associated so closely with climatic and edaphic factors. The progress of disease in the different groups of plants is shown in Table 5.

From the presence of *Fusarium solani* f. *cucurbitae* in such a high percentage of the plants dead in the bacterial-inoculated and

TABLE 5. INFLUENCE OF A SPECIES OF BACTERIUM UPON THE PROGRESS OF THE FOOT ROT DISEASE AFTER INOCULATION OF MATURE HEALTHY PLANTS
No. of plants affected[1]

Inoculum	Stage	Days after inoculation							Recovery of Fusarium
		8	14	16	21	26	30	35	
Fusarium	S	3	4	2	..	..	..	..	
	R	1	2	3	1	..	..	..	
	W	1	..	2	3	..	..	..	
	D	..	3	3	6	10	10	10	10
Bacterium	S	..	..	..	6	3	..	..	
sp.	R	..	..	..	4	3	5	..	
	W	..	..	..	..	..	1	..	
	D	..	..	..	..	4	4	10	8
Fusarium	S	..	2	5	..	..	..	..	
and	R	1	..	3	6	3	2	2	2
Bacterium	W	..	1	..	..	..	1	..	
	D	..	..	1	4	7	7	8	8
Sterile	S	..	..	..	9	9	7	5	2
water	R	..	..	..	1	1	2	..	
check	W	..	..	..	..	..	1	..	
	D	..	..	..	..	..	..	5	5

[1] 10 plants inoculated.

check series at the end of five weeks, it appears that the experiment was considerably confounded by the secondary spread of the fungus either by insects, wind or some other agent.

The most significant conclusion to be drawn from the table is that the presence of the bacterium does not synergize the development of disease, but actually seems to decrease the virulence of the fungus pathogene. The mode of this action is not clear, but may easily be a case of mild antibiosis.

The presence of Fusarium in all five of the check plants which died and in eight out of 10 of the bacterium-inoculated plants indicates that the bacterium is probably purely saprophytic. The failure to find the fungus in two cases out of 10 when the plants had been inoculated with the bacterium alone may have been due to technique or may indicate that this normally saprophytic bacterium can under certain conditions kill the living tissue by extracellular enzymatic action and can actually cause a diseased condition of the plant. That this bacterium is parasitic is extremely doubtful since no cases have been found in Connecticut of a diseased squash containing this organism alone, although it or a similar species is ubiquitous on wounded squash tissue and attacks such tissue with equal facility whether it be stem, root or leaf. Experiments were run in the greenhouse during the winter of 1943 in which the bacterium was surface-inoculated on plants. It was conclusively shown that the bacterium lacked any ability to penetrate uninjured stem tissue.

During the summer of 1943, two Fusaria not identical with our strain of *Fusarium solani* f. *cucurbitae* were isolated from diseased

plants along with the known pathogene. These were included along with the known pathogene in pathogenicity trials the following summer and both proved to be purely saprophytic in nature.

EFFECT OF CALCIUM-POTASSIUM BALANCE

It seemed worthwhile to investigate the effect of calcium and potassium on the development of the disease. A companion study on potato scab was already underway. A simple test was made the first year (1942). Calcium hydroxide and sulfur were added to infested soil and plants were grown. Although all treated plants finally succumbed to the disease despite treatment, it seemed significant that treatment did affect the time for appearance of symptoms. The period between planting the healthy seeds in the infested soil and the development of symptoms was shortened considerably for the lime plots as compared with the check plots. The lime seemed to have encouraged the disease. These data are in keeping with the known preference of the parasitic Fusaria for alkaline conditions.

This initial result did not distinguish the effect of the Ca– from the –OH in the $Ca(OH)_2$ molecule. Indirectly, the –OH was suggested as the cause by the fact that the addition of sulfur in the same experiment delayed the development of symptoms on healthy seedlings.

The experiments were extended during the next two summers. In 1943, another variable was introduced. If the calcium ion, perchance, and not the –OH ion, had been responsible, then potassium should antidote the effect of soil calcium and reduce the disease. Therefore, calcium hydroxide and sulfur as before, were compared with potassium chloride at three doses each (Table 4).

Despite some heterogeneity in the results, it is clear that the calcium hydroxide again increased the disease. As before, it accelerated the rate of involvement of the plants as well. Although sulfur reduced the –OH ion in the soil, it did not seriously affect the percentage of disease until the pH became very low. Potassium reduced the total disease and the rate of involvement without reducing the pH greatly.

TABLE 4. INCIDENCE OF SQUASH FOOT ROT IN INORGANIC AMENDMENT PLOTS IN 1943

Amendment	Rate per acre in pounds	Soil reaction	Number of plants	Per cent diseased
Calcium hydroxide	250	pH 5.7	74	28.4
	500	6.4	72	43.2
	1,000	6.8	74	39.2
Potassium chloride	250	5.4	76	19.8
	500	5.7	78	25.7
	1,000	5.5	68	19.2
Sulfur	250	5.4	74	27.0
	500	5.0	63	25.4
	1,000	4.2	71	21.1
Check	...	5.7	74	25.7

A series of laboratory studies were made on the effect of the ratio between calcium and potassium on the fungus and on the reaction of squash plants to the disease. In nutritional studies with the fungus, no effect of calcium, or of calcium-potassium ratios could be found until the absolute amount of potassium present in the medium was reduced so as to be actually limiting. High Ca/K nutrient solutions did not appreciably affect fungus growth so long as there was enough total potassium for the nutrition of the fungus.

To test the effect of the calcium and potassium nutrition of summer squash on its reaction to the disease, healthy plants were started and grown in sterile sand cultures supplied with three different nutrient solutions, varying only in the ratios of calcium and potassium which they contained. After two weeks, no difference could be noted in the growth characteristics of the plants grown in the various calcium-potassium ratios. At this time all plant cultures were inoculated with the foot rot organism. The progress of the disease as it occurred under these three different calcium-potassium ratios is charted in Figure 4.

It may be seen that, here again, when the total amount of disease at the end of the growing season is considered, there is no difference between the results obtained between plants growing under the different nutritional conditions. All plants eventually became infected and died in every case. Only when the progress of the disease among the entire population is observed are the differences in disease reaction noticeable.

In keeping with the earlier appearance and higher incidence of the disease on the calcium plots in the field, the amount of disease at any given time in the sand cultures was higher when the Ca/K has a high than when it has a low value. The progress of the disease on the medium and low Ca/K ratios constantly lagged behind the disease development on the plants grown with the very high Ca/K ratio.

As a result of these experiments, it seems safe to say that the development of squash foot rot is affected by the calcium and/or potassium nutrition of the host plant. Apparently, high calcium and low potassium encourages the disease and high potassium and low calcium discourages it. The mechanism of this action is obscure. One is inclined to say that potassium lends resistance to this Fusarium disease as it does to the Fusarium wilt of cotton. On the basis of the data one could say also that it reduces the effect of calcium in encouraging the disease.

In the case of potato scab which has been studied much more extensively, the effect of Ca/K is multimodal (Gries, Horsfall and Jacobson, 1944). The high point in the middle dosage range for calcium and potassium in Table 4, suggests that a similar relation may hold here also.

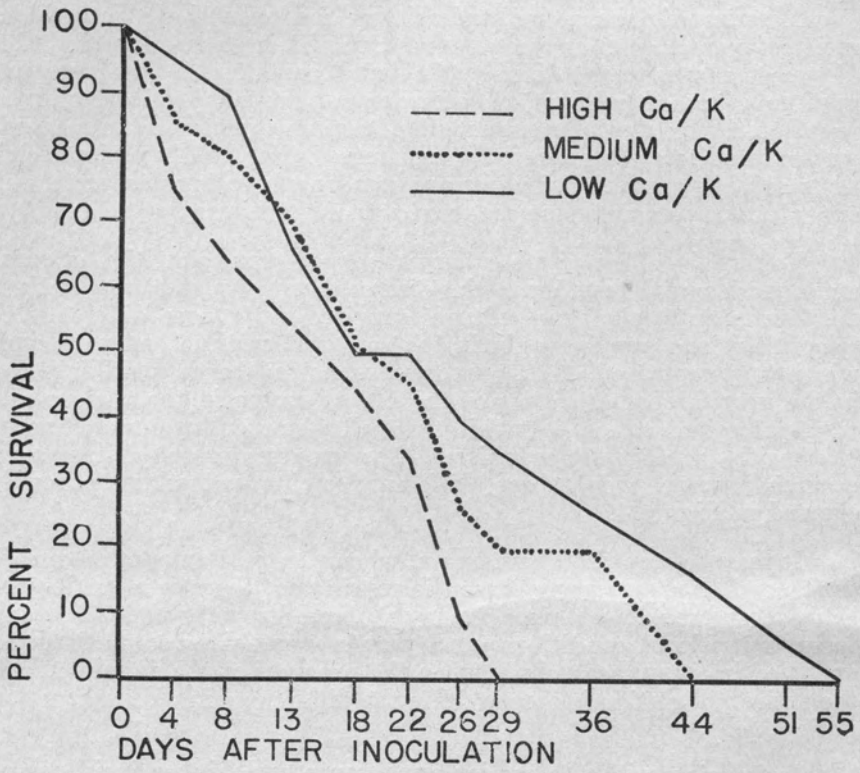


Figure 4. Survival of Early Prolific squash plants grown in sand culture in three nutrient solutions varying only with the ratio of calcium to potassium.

The experiments discussed above are disappointing, perhaps, to the practical man because no ratio of calcium and potassium were discovered which would even approach commercial control under conditions of severe disease. They do suggest, however, that the use of potassium and the avoidance of lime might be good practice.

SUMMARY

1. Foot rot is a disease of squash caused by the fungus *Fusarium solani* f. *cucurbitae* (*F. javanicum* v. *theobromae*). The most noticeable symptoms of the disease are the wilting and dying of the host plant following the rotting of the stem tissue near the soil level. The fungus also causes a fruit rot, although the importance of this stage is largely of concern to the seed producer. This species of *Fusarium* is capable of causing the disease of *Cucurbita pepo*, *C. moschata* and *C. maxima*. In Connecticut it has been most destructive to *C. pepo* and to the progeny of crosses involving this species as one of the parents.

2. Foot rot is a seed-borne disease. The fungus is carried as spores on the surface of the seed or as mycelium in the seed coat. Occasionally, it is found as sporulating mycelium in the embryo of the seed. True seed infection occurs only in fruits affected with the fruit-rotting stage of the disease, although surface-borne contamination is often effected through the fermentation process in the recovery of the seeds. The fungus seems to die out of the seed after two years of storage.

3. Hot water therapy has been successful in sterilizing seeds infected with the mycelium in the seed coat. This procedure is attended by an appreciable reduction in germination, however. Surface contamination can be controlled by a 15-minute immersion of the seeds in a 1:1000 corrosive sublimate solution. No success has been had in limited attempts to protect healthy seedlings in infested soils with chemical seed treatments.

4. The fungus causing foot rot may overwinter in the soil although, under Connecticut conditions, this is not a serious factor in the production of squash. The fungus is relatively short-lived in soils amended with organic matter. This is believed to be the result of the antibiotic effects of other microorganisms, rather than being a matter of competition for nutrients. In no case has the fungus been known to remain active in commercial fields for more than two winters.

5. Soil amendments of sulfur and potassium chloride delay the development of disease symptoms in the field by periods up to two weeks, while hydrated lime hastens the development of the disease. It is suggested that this may be a function of the calcium and/or potassium nutrition of the host plant, although it may be effectively duplicated by changing the soil reaction. Thus, the disease develops more rapidly in alkaline than acidic soils. It is suggested that lime be used sparingly where the fungus causing this disease is suspected of being present in the soil.

6. While no insects have been proved to be vectors of the disease, a thorough program of insect control should be practiced wherever this disease is known to be prevalent.

7. A species of bacterium commonly associated with the disease in Connecticut has been shown to have no role in its etiology. In inoculation studies this bacterium has actually been shown to slow down the development of disease symptoms rather than to synergize their appearance.

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