GENETIC ANALYSIS OF BACTERIAL WILT RESISTANCE AND CERTAIN OTHER CHARACTERS IN A TOMATO CROSS, LYCOPERSICON ESCULENTUM MILL. X L. PIMPINELLIFOLIUM MILL.

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I. INTRODUCTION

One of the most important diseases limiting tomato production in tropical areas is bacterial wilt, caused by the soil-borne bacterium, Pseudomonas solanacearum E. F. S.

Breeding for resistance has proved to be the best way to control bacterial wilt in several crops. Similarly, it appears that successful commercial production of tomato in many parts of the tropics requires the development of tomato varieties resistant to the pathogen.

A voluminous literature, approaching 1,000 papers has been published on the subject of bacterial wilt. The genetics of resistance to the disease, however, has been investigated in only a few crops. Resistance is governed by multiple genetic factors in tobacco (Smith and Clayton, 1948) and is suspected to be similarly multifactorial in other species (Singh, 1961).

Many attempts have been made to control bacterial wilt by chemical and physical treatments of soil (Stevens, 1906, Garner et al., 1917; Smith, 1944; 1947; and Sequeira, 1958). With few exceptions, however, chemical means of reducing losses due to wilt have not been practical (Kelman, 1953) because of phytotoxicity or expense of application.

Tomato breeders have been unsuccessful in producing commercial varieties immune to bacterial wilt. A useful source of genetic resistance, however, is available in Lycopersicon pimpinellifolium Mill. The present study was based on this resistant source material.

The major objectives of the investigation were: to investigate the inheritance of resistance to bacterial wilt in tomato, to estimate the degree of environmental modification of resistance, and to determine whether resistance is linked with the sp^+ (indeterminate growth) and Mi^+ (nematode susceptibility) loci on chromosome 6.

II. REVIEW OF LITERATURE

1. Economic Importance

Bacterial wilt disease was reported to be world-wide at the beginning of the 20th century (Kelman, 1953). The disease appeared to be well established in most regions when scientific investigations were first initiated. Bacterial wilt has caused heavy losses and sometimes total loss, in many crops especially in the family Solanaceae. Smith (1914) reported that the disease had put an end to commercial tobacco production in certain sections of the United States. At one time, it threatened to annihilate the tobacco industry (Garner et al., 1917). In warm, humid areas of the world, the disease has also been devastating to peanuts and bananas.

In the Philippines, Welles and Baldan (1922) indicated that as many as 95% of the tomato plants were killed by bacterial wilt in certain fields of the College of Agriculture at Los Banos. Agati (1949) showed that there were instances of crop failure in some provinces in the Philippines as a result of the wilt. Dosado (1958) reported that the disease destroyed all susceptible tomato varieties and took a heavy toll of native Philippine lines.

According to Sherbakoff (1917) the succeeding susceptible crops in infested fields were increasingly attacked. Crop rotations of four to five years with immune crops reduced the incidence of the disease. However, Sherbakoff (loc. cit.) warned that infested fields remained infested indefinitely.

2. Symptomatology

Specific expression of bacterial wilt symptoms vary with the crops, and the rate of development is influenced by environmental conditions. Symptoms associated with infection by the bacterium on most host plants include

sudden wilting, stunting, and yellowing of foliage. Vascular discoloration is noticeable. If a section of stem is suspended in water, fine milky strands of bacteria stream out from the margins of the vascular tissue (Kelman, 1953). Since tomato plants contain no latex, this method is efficient in diagnosing the presence of the disease. Similarly, it helps in field diagnosis to distinguish bacterial wilt from vascular wilts caused by fungi.[1]

3. Effects of Environment on Disease Severity

Vaughan (1944) found that plants wilted at a soil temperature of 26.4°C but recovered turgor at 12.7°C. Disease development was optimum between 25 and 35°C, but inhibited at temperatures below 12°C. These findings were corroborated by Gallegly and Walker (1949). The wilt bacterium is sensitive to desiccation (Sequeira, 1958).

Walles and Roldan (1923), Gallegly and Walker (1949), and Kelman (1953) reported that the rate of development of wilt in tomato was maximized by wet, warm weather and moist soils. Smith (1943) found that wilt was more severe in wet than in dry areas of the field.

4. Variability of the Pathogen

Bacterial wilt-resistant peanuts developed in Indonesia and resistant tobaccos developed in the United States have retained full resistance for many years. However, the existence in the soil of strains of P. solanacearum is known and isolates of the organism have been made by various workers (Buddenhagen, 1960). Variations in the morphological and physiological characteristics of the strains have been noted (Kelman, 1954; Kelman and Person, 1953; Perlaza, 1960; Klement and Lozenovich, 1962).

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[1] Unpublished information from Dr. O. V. Holtzmann, Department of Plant Pathology, University of Hawaii.

Virulence of any strain of the bacterium varies from host to host and between locations on the same host (Kelman and Person, 1961). Sequeira (1958) found that newly arising mutant forms of the pathogen were pathogenic to a small fraction of varieties or species tested. At the same time, no species or variety was susceptible to all the mutants. Zafrira and Palti (1960) compared different strains of wilt on tomato, potato and eggplant from different geographical locations. American isolates and Israeli isolates differed in their host range and some of their morphological and biochemical traits.

Buddenhagen (1962) reported a strain which was widespread in banana soils that did not affect commercial triploid bananas. He suggested that "moko" disease of bananas in South America was caused by a specific strain of P. solanacearum.

P. solanacearum undergoes a relatively rapid loss of pathogenicity in culture (Hella, 1931). In the breeding program, virulent strains are needed and can be maintained by covering bacterial wilt cultures in solid medium with a layer of sterile mineral oil (Kelman and Jensen, 1951).

5. Breeding for Resistance

The control of bacterial wilt disease through breeding was suggested as early as 1903 (Stevens and Sackett, 1903). Many foreign and domestic tobacco varieties were tested in the early trials but none had sufficient resistance to be valuable in breeding programs.

The United States Department of Agriculture, in cooperation with the State of North Carolina, renewed efforts in 1934 to find wilt-resistant types of tobacco. Within four years, 1,304 collections were tested. Very few lines showed resistance. Clayton and Smith (1942) successfully selected two moderately resistant strains. One highly resistant strain was found,

but proved to be otherwise poor. Later, a line TI 448A was found to possess high resistance, without objectionable growth characteristics and quality. Crosses were made between TI 448A and a commercial variety. From this cross, 32,000 hybrid plants were tested by Clayton and Smith (1942) and five potentially valuable plants were selected. After five years of intensive and careful selection in the presence of disease epiphytotics and an environment favorable for bacterial growth, a high quality tobacco with high wilt resistance was obtained (BCARS, 1945a). This was released as "Oxford 26".

Several wilt-resistant varieties of peanuts have been found in Indonesia (Schwarz, 1926). In eggplant, resistant varieties have been released from a cross of Natals (resistant) and Javanese varieties (Winstead and Kelman, 1960).

In the case of bacterial wilt in tomato, the search of a resistant variety by many investigators has not been as successful as with other solanaceous crops. Currence (1934) ascribed this to the seemingly variable and mutable nature of the pathogen. With the testing of different varieties by various workers in many parts of the world, however, it has been ascertained that there are different levels of bacterial wilt susceptibility.

The importance of a resistant variety of tomato to bacterial wilt was recognised by Rolfs (1898) and Earle (1900). They detected different levels of susceptibility in commercial varieties but they did not find the resistance to be adequate. In Florida, Hume (1903) found that a plum-type tomato showed some resistance. Several years later, Sherbakoff (1919) tested 60 different varieties but found all to be susceptible. Early screening was also conducted in North Carolina but the results were unsuccessful (Massey, 1923). Stanford (1917) concluded that resistance in tomato to bacterial wilt cannot be augmented by seed selection from disease-free plants which remained healthy to maturity.

Efforts towards finding a wilt-resistant commercial tomato variety were renewed in the mid-thirties (Schmidt, 1937). The only variety that showed resistance was Louisiana Pink, and it was crossed with the commercial susceptible variety, Marglobe. Resistance among selections from this cross was not consistent in field trials. A number of other types were tested and a small-fruited currant tomato from South America showed high resistance (BCARS, 1942). In 1943, lines which showed definite resistance were selected from crosses of Louisiana Pink x T 414 from Puerto Rico and T 414 x Devon Surprise (BCARS, 1945b).

A more intensified program of finding resistant tomato lines combined with commercial fruit size and quality was later undertaken (BCARS, 1948). In 1950, 909 collections were evaluated. Of these, only 26 had an appreciable degree of resistance. One line did have good fruit size but the quality was not suitable (BCARS, 1952).

In Ohio and Indiana, Alexander et al. (1942) tested 448 lines from South and Central America and various foreign countries and they found no line with a satisfactory level of resistance. To facilitate the work, a Cooperative Screening Committee was formed and more than 100 wild species and strains were tested. Ellis and Barham at North Carolina used greenhouse tests whereas McGuire in Hawaii used a field test (Alexander, 1955).

Extensive screening of resistant varieties also has been done outside the United States. Labrousse (1932) found no high level resistance in 20 commercial varieties tested in France. Negative results were also obtained in field trials with different varieties in the Philippines (Mendiola and Ocfemia, 1926 and Rupis et al., 1962); Puerto Rico (Wells, 1931); Fiji (Simmonds and Barham, 1934); Ceylon (Park and Fernando, 1936); South Africa (Wager, 1944); Queensland (Aberdeen, 1946); and Malaya (Burnett, 1949).

Molla (1931) studied 23 tomato varieties in field and greenhouse trials in Puerto Rico. In distinction to previous findings, he suggested that the variety Marglobe, was partially resistant, and that Louisiana Pink was among the more susceptible varieties. None of the native Puerto Rican lines was resistant. As a result of the breeding work, a variety was developed which was more resistant than Marglobe and the local varieties.

Warmke and Granado (1949) made selections from 43 Mayaguez lines in Puerto Rico. The selections outyielded both the imported and local varieties in a test in infested fields.

Park and Fernando (1938) tested eight local and foreign varieties in Ceylon but found none of these that showed enough resistance to be useful for commercial planting. In Fiji, Simmonds and Parham (1934) noted that a small-fruited, cherry-type tomato was relatively resistant, but during the early period of growth a large number of plants died due to bacterial wilt.

To facilitate the development of wilt-resistant lines in the tomato breeding program, Deasis (1938) in the Philippines evaluated different breeding procedures. His experimental data favored backcrossing to the resistant parent to other breeding procedures.

At present there is no great problem in selecting plants highly resistant to bacterial wilt in Hawaii. The major problem is that of combining resistance with the necessary horticultural characters for commercial acceptability. Barham et al. (1956) reported that promising lines were selected from a progeny of large-fruited F_2 selections grown in North Carolina. Similarly, promising lines have been obtained at the Hawaii Agricultural Experiment Station (1956). However, no lines with fully satisfactory levels of resistance and with commercial fruit qualities have been found.

6. Inheritance Studies

Resistance to a number of pathogens of the tomato has been the subject of genetic studies. Schaible et al. (1951) cited the reports of several investigators, noting that resistance to each of the pathogens -- Septoria lycopersici Speg., Fusarium oxysporum f. lycopersici (Sacc.) Snyder and Hansen, Stemphylium solani Weber and the spotted wilt virus -- was based on a single dominant factor. Resistance to wilt caused by Verticillium albo-atrum Reinke and Berth, was shown by Schaible et al. (1951) to be inherited as a monofactorial dominant condition. Resistance to the root-knot nematode, Meloidogyne incognita (Kofoid and White) Chitwood is also governed by a single dominant gene (Gilbert and McGuire, 1956).

There has been little success in analysing the genetic control of resistance to bacterial wilt in tomato. McGuire (1956) noted that environmental factors altered the expression of resistance. He observed wide variations in disease readings of individual varieties in successive tests. The greatest obstacle encountered was the difficulty in differentiating various levels of resistance. Shifriss and Myers (1942) tried to specify a criterion for detecting various degrees of resistance of cucumbers to mosaic virus. The segregating populations were grouped and inoculated at different stages of growth of the plants. They suggested that the delay in appearance of the symptoms was due to genes in the host rather than the effects of environment. They emphasized that a weekly record was essential in obtaining reliable data on the total number of genes involved in the disease expression.

Smith and Clayton (1948) were the first to report the manner of inheritance of bacterial wilt in any solanaceous crop. They reported that resistance in tobacco is recessive, and governed by multiple factors.

McGuire (1956) indicated that resistance of tomatoes to bacterial wilt was heritable. Later, McGuire (1960) proposed that resistance derived from North Carolina lines was recessive, since F_1 hybrids were susceptible to wilt. A new type of resistance from a small-fruited wilt tomato (*L. pimpinellifolium*) appeared to be dominant. Singh (1961) investigated the inheritance of the North Carolina type of resistance in tomato, and found that resistance was recessive, proposing that three genes governed the resistance.

III. MATERIALS AND METHODS

1. Materials

Anahu (*L. esculentum*) is a large-fruited variety developed by Dr. J. C. Gilbert at the Hawaii Agricultural Experiment Station. Like other commercial varieties, it is susceptible to bacterial wilt but has resistance to four diseases, namely, common races of root-knot nematodes, Fusarium wilt, Stemphylium leaf spot and spotted wilt virus in Hawaii. It has a determinate habit of growth, uniform ripening, and yellow gel around the seed of the ripening fruit. The fruit weighs an average of about 150 grams.

An inbred line of *L. pimpinellifolium* designated as HES 3808-2 possessing a high degree of resistance to bacterial wilt was selected by Dr. D. C. McGuire. It has been maintained as source of resistance to some diseases at the Hawaii Agricultural Experiment Station. It is susceptible to root knot nematodes but has some tolerance to blights. It has an indeterminate habit of growth, with very small fruit weighing about 15 grams. The immature fruits have green shoulders with green gel around the seeds.

Yellow Plum was chosen as a susceptible check variety. In the field, plants of this yellow-fruited variety were alternated with each test plant.

2. Inoculation Procedures

A large area of the Poamoho sub-station of HARS was made available for tests. Field inoculation was made by hypodermically injecting bacterial wilt suspension near the soil line in the stem of established susceptible plants spaced closely in the field. A second planting of susceptible plants was made to assure a heavy and uniform infestation. Toothpicks were used in this inoculation in place of (often-broken) hypodermic needles. Two to three toothpicks soaked for 24 hours in a high concentration of bacterial wilt suspension were inserted in the stem after wounding (there) with a knife.

Suspensions were also poured into the irrigation water for both field inoculations.

Early reports indicated that under glasshouse conditions resistant varieties succumb to bacterial wilt. Recently, Winstead and Kelman (1952), Dowson (1958), and Zafrira and Palti (1960) succeeded in studying the disease under laboratory conditions using dilute bacterial suspensions. Modifications made of these techniques are discussed together with the results.

3. Analysis of Data

The number of days until death from wilt was used as the measure of resistance. Populations were described by weighted survival means, expressed in number of days from planting until death from bacterial wilt. The weighted mean, T, was calculated as follows:

$$T = \frac{n_1 t_1 + n_2 t_2 + \dots n_i t_i}{n}$$

where: n_1 = survivors to t_1 only

n_2 = survivors between t_1 and t_2 only, etc.

t = number of days from planting until time of observation

t_i = number of days at last observation + 7

n = total number of plants in each population

Plants surviving at the last observation were arbitrarily given seven days more as resistance. It is recognized throughout the following discussions that the assumption of a 7-day increment is arbitrary, and means based on this assumption are interpreted cautiously. In reality, the results suggest that many plants surviving at the end of field test periods would not have succumbed to wilt at all under these conditions, i.e., t_i (and T) for these plants is equal to infinity.

IV. RESULTS AND DISCUSSION

1. Wilt Resistance Studies

Most of the genetically-segregating lines to be discussed were derived from a susceptible parent, Amahu (P_1) and a resistant inbred line of *L. pimpinellifolium*, HES 3808-2 (P_2) which were crossed in 1960. The F_1 plants were grown, self-pollinated and backcrossed to each of the parent lines. The seeds from crosses on different F_1 plants were bulked to make up the F_2, the BC to Amahu (BC_1) and the BC to HES 3808-2 (BC_2) populations. Check plants of the wilt-susceptible variety, Yellow Plum, were included in all tests. Plants of the following seven lines thus were included in all of the critical studies:

P_1 (Amahu) = wilt-susceptible, *L. esculentum*

P_2 (HES 3808-2) = wilt-resistant, *L. pimpinellifolium*

F_1 (Amahu x HES 3808-2)

F_2 (F_1 self-pollinated)

BC_1 (F_1 x P_1)

BC_2 (F_1 x P_2)

Check (Yellow Plum)

1a. Field. Four field experiments were conducted in order to observe segregations for resistance under different environmental conditions. The lines were planted in each of four seasons. Each trial consisted of about 100 plants of the F_1 and each parental line, and from 100 to 500 plants of the F_2 and each backcross population. Susceptible check plants were alternated with test plants in all trials.

The numbers of survivors were recorded weekly, starting from the third week after transplanting. Seedlings that died from causes other than bacterial wilt were excluded from all analyses. Instances were noted in which

plants wilted but recovered. Such cases may have been caused by high levels of bacterial inoculum in the field.

The first field test (summer) was conducted from July to November, 1961 (Figure 1). Data were recorded for 1,840 plants. The check and P_1 were similarly susceptible to wilt with the greatest number of dead plants occurring between the 3rd and 5th week. The survival curves of these populations were clearly exponential. The P_2 (NKS 3808-2) was fully resistant until the end of the experiment, in 17 weeks. The F_1 plants remained resistant until the 9th week, but many plants died in the 10th week. Plants in the F_2 continued to die in a steady fashion from the 3rd week after transplanting. Death rate of BC_1 was about intermediate between F_2 and P_2, and the BC_2 plants reacted much as the F_1 population.

The fall test was conducted from September, 1961 to February, 1962 (Fig. 2). Data were recorded for 1,086 plants. As in the summer test, most of the P_1 and check plants died between the 3rd and the 5th week. Some check plants survived until the end of the test, perhaps a consequence of the low levels of inoculum in some portions of the field. The F_1 showed resistance until the 14th week. The reactions of the other populations were the same as in the summer test except that there was a generally delayed death of the plants.

The breeding lines were re-planted in November, 1961, for a winter test, and the experiment terminated in April, 1962 (Fig. 3). Data were recorded for 1,973 plants. The check and susceptible parent again showed exponential survival patterns, with most of the plants dying between the 3rd and the 8th week. Wilt severity was less than in previous tests, in both segregating and non-segregating lines. The P_2 plants survived without symptoms of wilt until the end of the test. The F_1 resisted the organism until the 14th week and the BC_2 until the 15th week. The pattern of responses

of the other populations were the same as in the other tests.

Results from the spring test (April to August, 1962) were similar to the previous tests (Fig. 4). Data were recorded for 3,472 plants. The P_1 and check plants, died early in the season as in previous summer and fall tests. The P_2, BC_2 and F_1 overcame the disease in a similar fashion until the 14th week. Plants of the P_2 started to die at the 15th week, although from causes other than wilt, so far as could be determined. There was a delayed death of the plants in this test, possibly because of the cool months at the beginning of the test which inhibited the multiplication of the organism and delayed the expression of the disease.

Comparative studies of the data from the four plantings (Figs. 1, 2, 3, 4) revealed several interesting differences. Considering the period until 50% survival for susceptible checks, the winter test averaged over 6 weeks, while other tests ranged between 3 and 4½ weeks. The delayed symptoms of wilt in the winter test were less evident in F_1 and segregating populations. Plants from the susceptible parent survived longer, producing a few fruits, but of poor quality. The resistant parent showed high resistance in all the tests.

Few F_1 plants showed symptoms of wilt before the 14th week, except in the severely-infected summer trial (Fig. 1). The 50% survival periods in summer, fall, winter and spring tests, were 10, 16, 15 and 15 weeks. The low value in the summer is probably due to the severity of the disease in this season (reflected also in BC populations).

Plants in the F_2 and BC_1 populations continued to die from planting until the end of each test. Onset of symptoms followed by death occurred more rapidly in the BC_1 than in the F_2 population. The BC_2 population showed resistance until the 16th week in the winter and spring tests but in the summer and fall tests, a number of plants died early in the season.

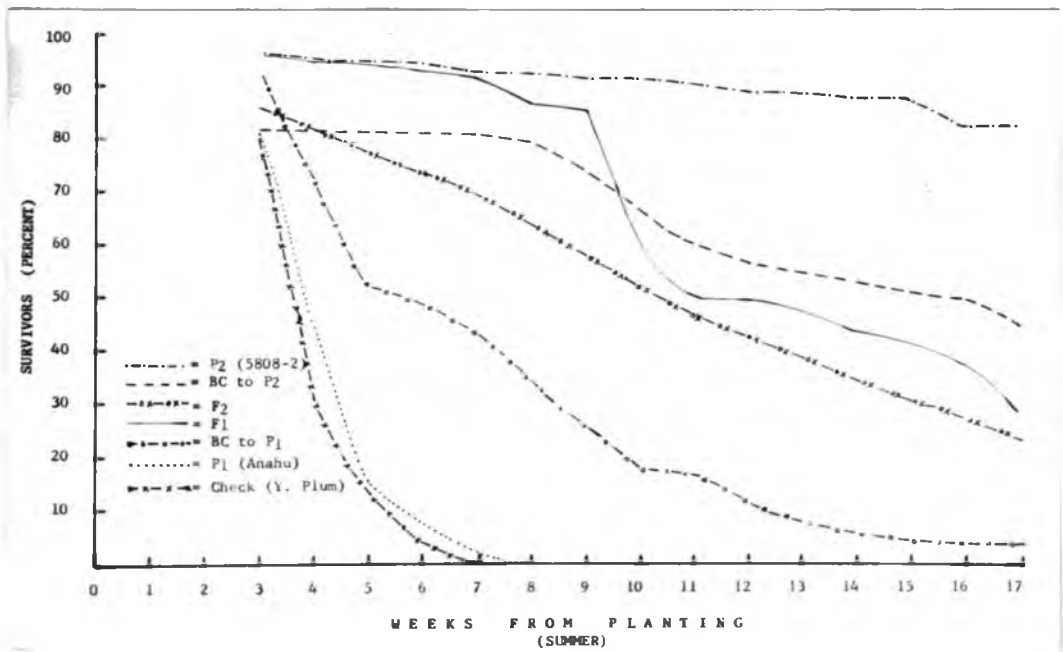


Figure 1. Weekly percentage of surviving plants in a bacterial wilt-infested field (summer test).

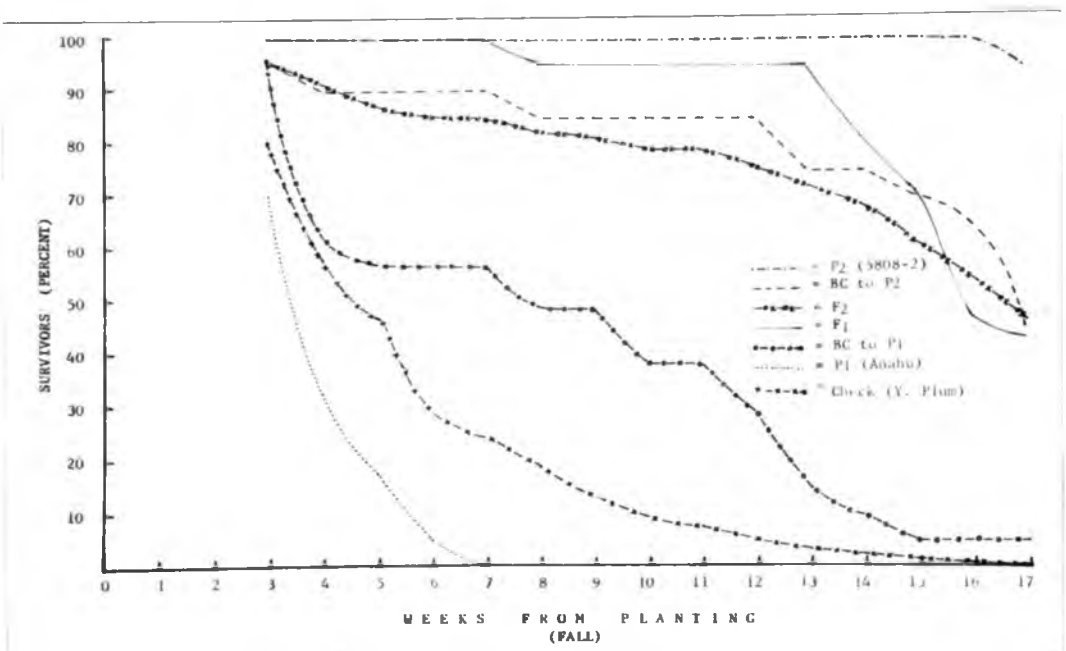


Figure 2. Weekly percentage of surviving plants in a bacterial wilt-infested field (fall test).

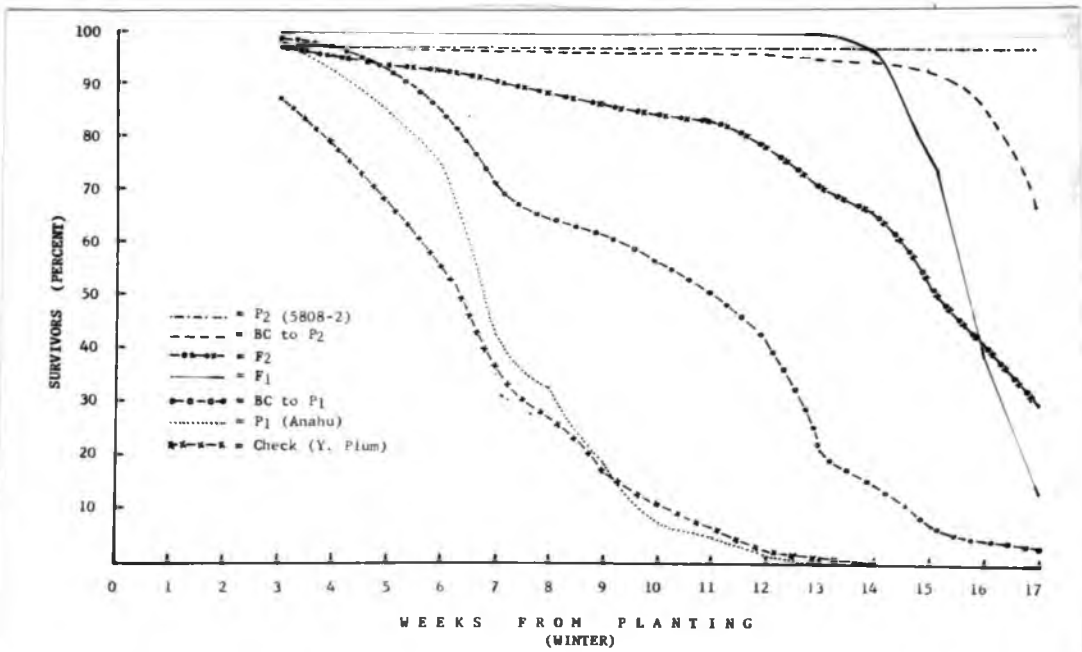


Figure 3. Weekly percentage of surviving plants in a bacterial wilt-infested field (winter test).

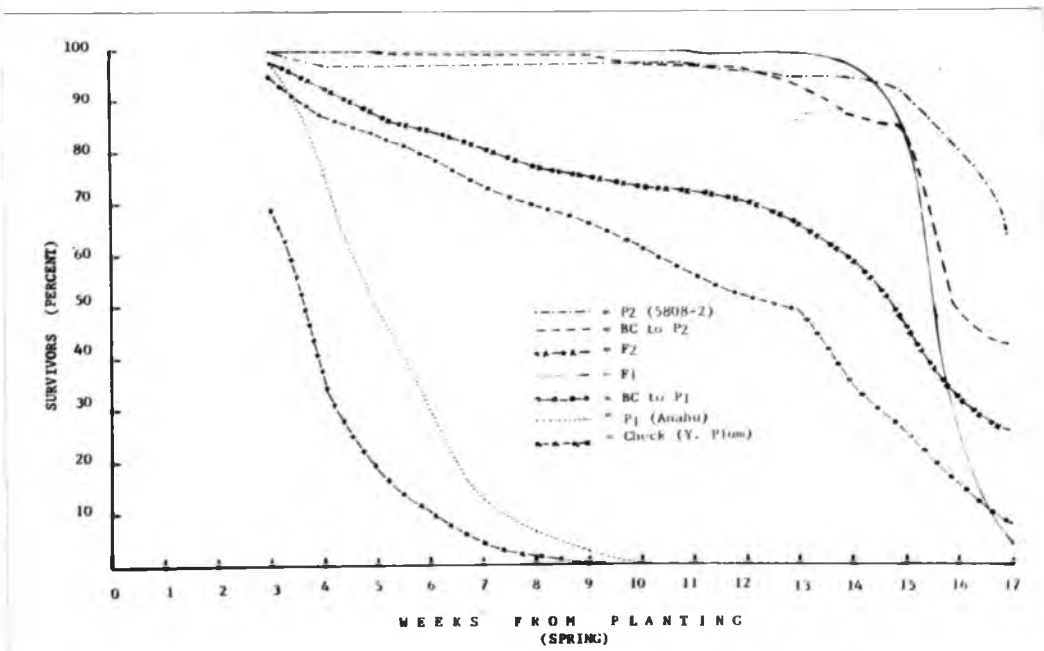


Figure 4. Weekly percentage of surviving plants in a bacterial wilt-infested field (spring test).

The rate of death in all experimental populations was faster in the summer test than in the other tests. The severity of wilt reaction among segregating plants of BC_1 in the summer is illustrated in Figure 5. Variations among segregating plants were particularly clear nine weeks after transplanting, at mature fruit stage, in this planting. The resistance of the resistant parent, NES 3008-2, was especially striking late in the winter season (Fig. 6).

Data from the four trials have been pooled in Figure 7. The exponential survival patterns of genetically uniform populations (check, P_2 and F_1) are particularly clear in this summary. The susceptible check and the susceptible parent, Asahi, were almost identical in weekly death response. The F_1 population showed little death from wilt until the 13th week, after which a rapid decline appeared. The F_2 population segregated many plants which were killed by the disease in early weeks after planting, giving a nearly linear survival pattern. Similarly, death of BC_1 plants due to wilt was nearly linear with time, about 6% of the plants expiring each week. However, a disproportionate number (30%) of the BC_1 plants were killed by wilt between the 2nd and 4th week after transplanting.

In Figure 8, the wilt survival data from the four seasons have been summarized in another way. The average numbers of days until death have been calculated, on assumptions outlined in the Materials and Methods. Differences among the seasons are again evident, with the most severe reactions in the summer test and least severe in the winter test.

In the fall and winter tests, data were recorded weekly until all plants died (Table 1). In both tests, over 95% of the resistant parent (NES 3008-2) were surviving at 19 weeks, well beyond the end of normal fruit-bearing season. All plants in the BC_1 and F_1 died by the 20th week. At this time, only 11% of the P_2, 15% of BC_2, and 30% of P_2 plants were surviving.

Figure 5. Wilt reaction of tomato plants in summer trial, nine weeks after planting. A = P_2; B = BC_1; C = F_1 (see text).

Figure 6. The resistant parent, HRS 3808-2 and surviving F_2 plants in background, 20 weeks from planting in the winter test.

Contributing to the wilt-induced death by this time were several other causes. Early blight affected many plants, and necrotic stems were common. Death from normal causes ("reproductive stress") is common by this period among commercial varieties grown in Hawaii. It was largely for this reason that wilt resistance data were interpreted critically only until the 17th week (Fig. 7).

The relationship of soil temperatures to disease severity was tested by use of a soil thermograph. Soil temperature readings were taken only during two of the four field tests, as well as in tile bed and advanced progeny tests. The bulb of the thermograph was buried about 15 cm into the soil in the root zones.

Weekly soil temperatures during the fall (advanced progeny) and winter tests were lower by about 4°C than those for spring and summer (tile bed) test (Fig. 9). The percentages of surviving plants in the different populations were generally higher in the winter test than in any of the other tests. In the winter season, it is evident that the average soil temperature is not high enough for optimum bacterial activity. These results are in agreement with those of Vaughan (1944) and Gallegly and Walker (1949). The optimum temperature for bacterial development comes during the summer season.

Comparison of the temperature readings with the corresponding percentages of dead plants in each week did not show a direct relationship. It is probable that a certain period of time elapsed before the disease was expressed in the plants.

1b. Greenhouse. Two tests were conducted to test the performance of the breeding lines in flats. The last observations were made when the plants were showing a decline in vigor. The crowded growing conditions in the seed flats hastened senescence of the experimental plants.

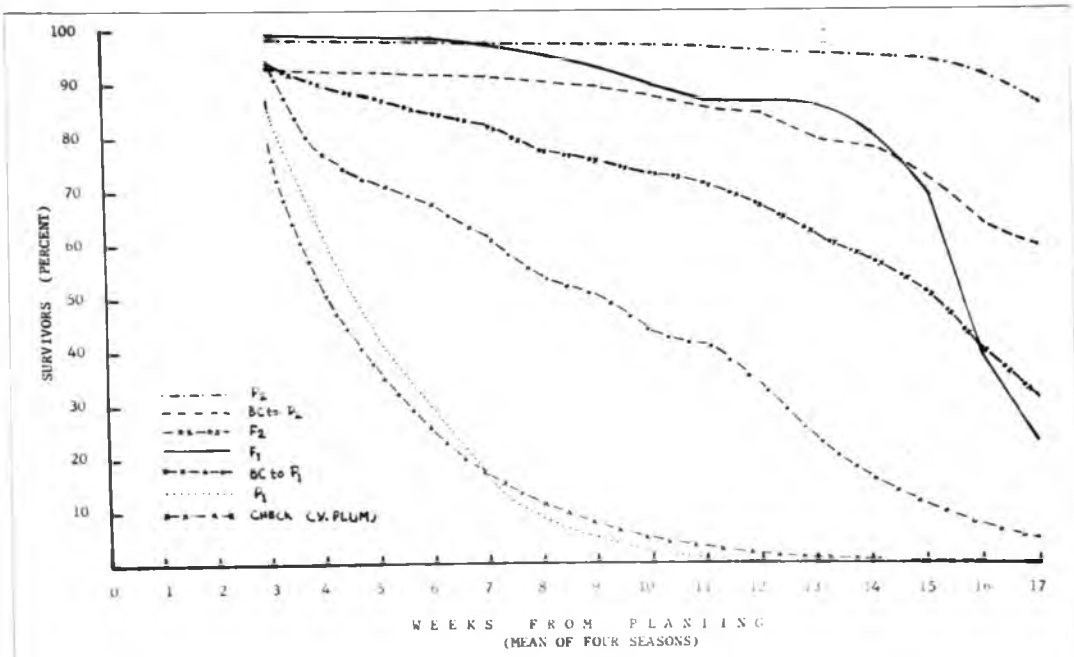


Figure 7. Weekly percentage of surviving plants in bacterial wilt-infested fields (Means of four seasons).

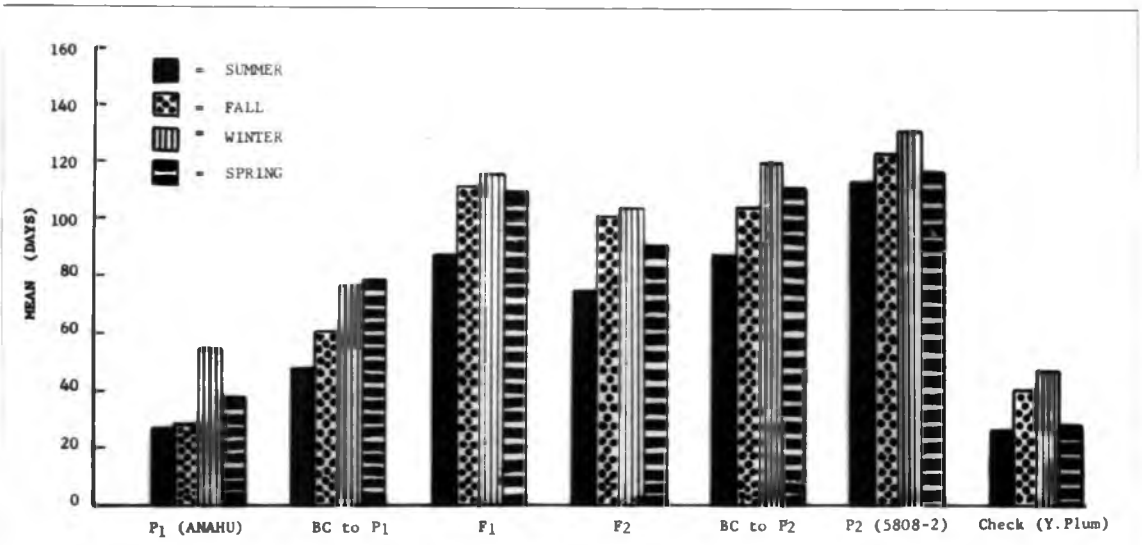


Figure 8. Weighted survival means in numbers of days from planting until death of tomato plants in four field tests.

Table 1. Weekly percentages of surviving plants from seventeen weeks until last observation in bacterial wilt-infested fields.

POPULATION	WEEKS					
	17	18	19	20	21	22
Fall Test:						
P1 (Anahu)	--	--	--	--	--	--
BC1	4.8	4.8	4.8	--	--	--
F1	42.9	38.1	9.6	--	--	--
F2	46.4	35.4	22.6	10.9	--	--
BC2	45.0	40.0	30.0	15.0	--	--
P2 (5808-2)	95.0	95.0	95.0	30.0	--	--
Winter Test:						
P1 (Anahu)	--	--	--	--	--	--
BC1	2.6	--	--	--	--	--
F1	13.4	0.8	--	--	--	--
F2	30.2	23.0	20.5	16.2	6.5	--
BC2	67.5	49.2	45.8	36.7	18.4	0.8
P2 (5808-2)	97.5	97.5	96.6	89.9	50.6	9.2

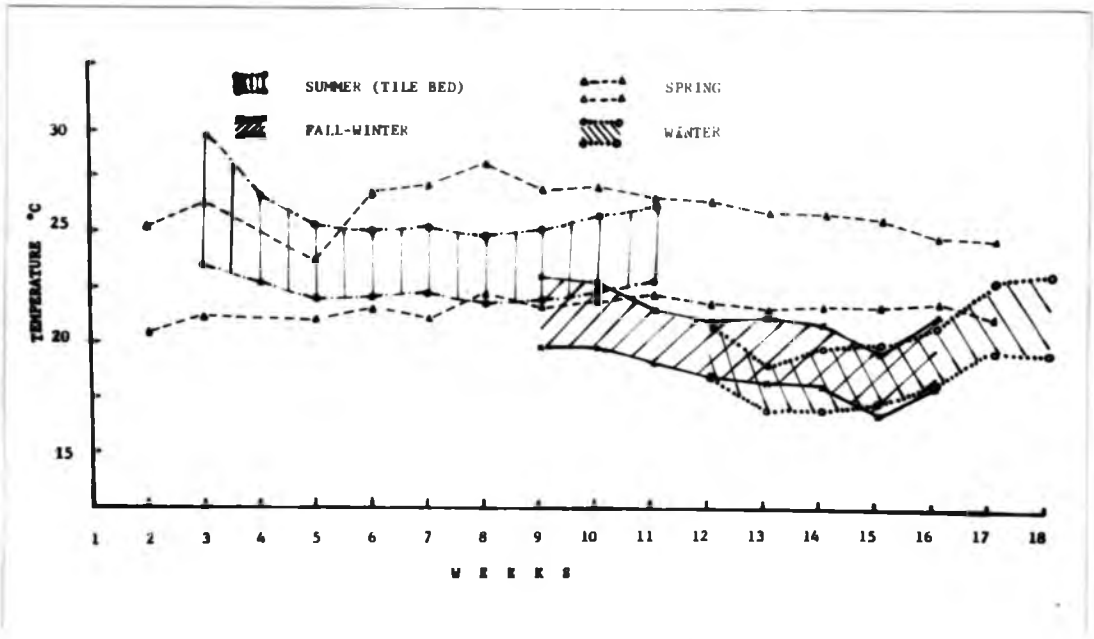


Figure 9. Weekly maxima and minima soil temperatures.

A preliminary test was conducted in seed flats with sterilized soil from July to September, 1961. The 284 seedlings tested represented the seven populations also grown in field tests. The 30-day old seedlings were inoculated through the root system. The inoculum was prepared by dissolving one loop (about 0.01 ml) of ooze from an infected stem in 5 ml of water. A cut was made on the roots about 3 cm from the stem of each seedling, and three drops of the suspension applied. Since this test was conducted to gain preliminary information on the reactions of the progenies under greenhouse conditions, only one observation was made at 67 days following planting (Fig. 10). The following percentages of survivors were obtained:

Check = 0%

P_1 = 0%

BC_1 = 0%

F_2 = 37.5%

F_1 = 47.9%

BC_2 = 62.5%

P_2 = 62.5%

The results of this preliminary test encouraged further studies with controlled inoculations, for results paralleled closely those of the field tests (Fig. 7).

In a second greenhouse test, the sterilized soil was inoculated 18 days before planting. The inoculum was prepared by allowing infected tomato cutting to ooze into a 50-ml beaker of distilled water for 12 hours. Ten milliliters of the fresh suspension were pipetted to each of five Erlenmeyer flasks containing 250 ml of nutrient broth. These were incubated for 24 hours for rapid multiplication of the bacterium. The bacterial wilt suspension was diluted to seven liters with water, and 300 ml of the inoculum were

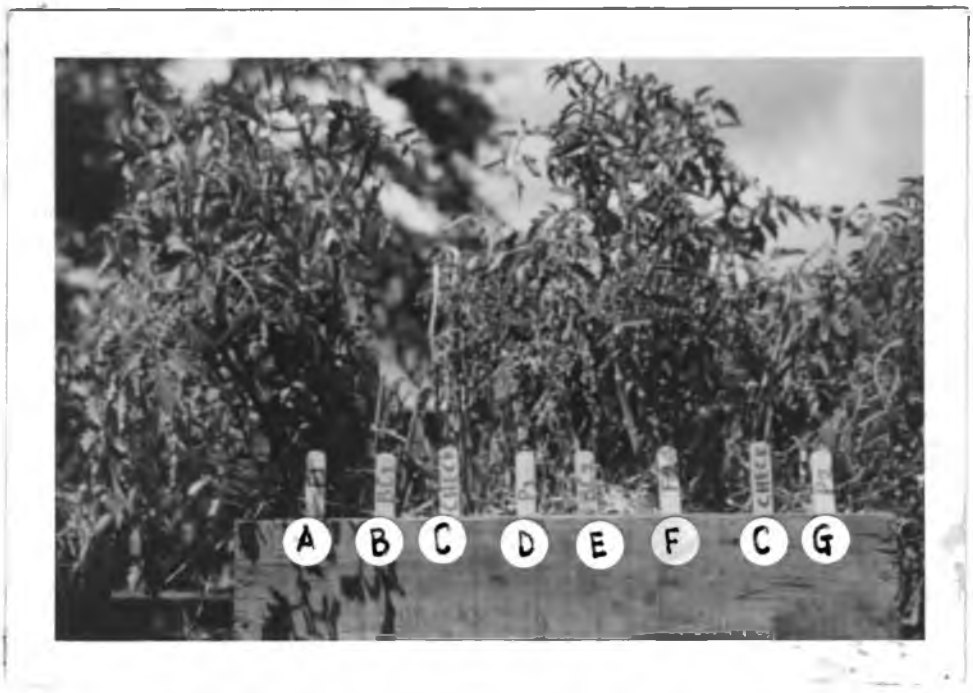


Figure 10. The breeding lines, A = F_1; B = BC_1; C = Check; D = P_1; E = BC_2; F = F_2; G = P_2. (Flat, Preliminary study)

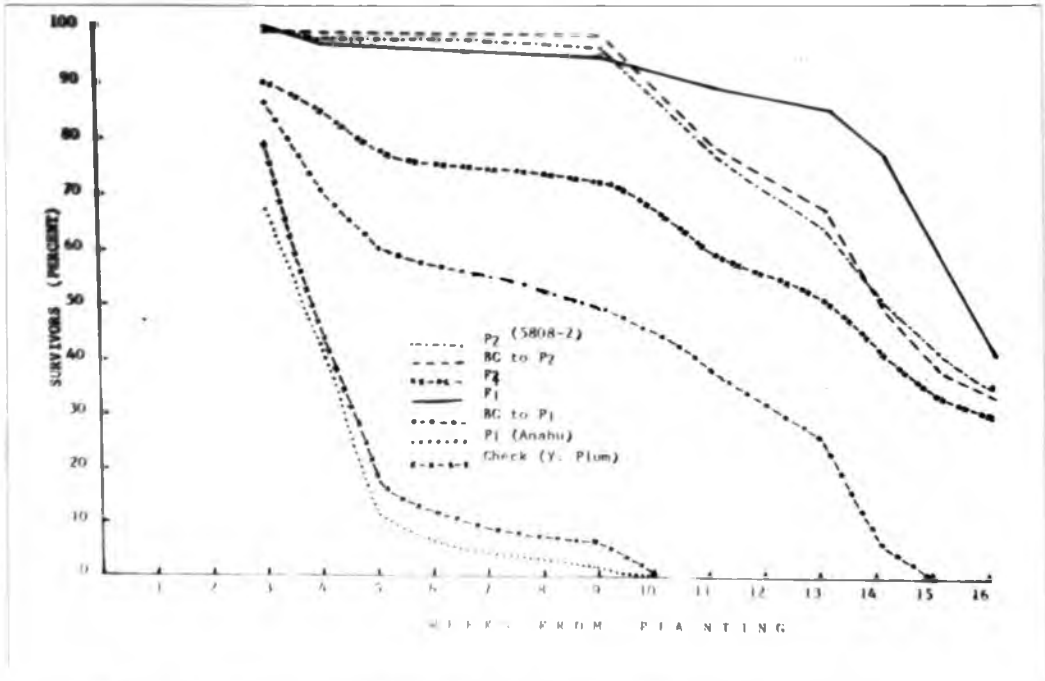


Figure 11. Weekly percentage of surviving plants in a bacterial wilt-infested soil (Flat test No. 2).

poured evenly in each of the 22 flats. To avoid desiccation of the bacteria, the soil was moistened every other day until planting time. Between March and August, 1962, data were recorded on 706 seedlings in this test.

Most of the P_1 and check plants died between the 3rd and 5th week, and all the plants died by the 10th week (Fig. 11). The P_2 and BC_2 showed resistance until the 9th week, then death of the plants occurred at a faster rate than the F_1. The results obtained in these populations deviated from those obtained in field tests. A possible explanation for these discrepancies is the fact that the F_1 showed more general vigor than the other populations. Under the crowded conditions of seedling growth in the flats, competition among the plants was greater than in the field. Some plants of the P_2 and BC_2 might have been weakened and thus succumbed to the wilt. These results showed convincingly that no true immunity to the wilt occurred in the lines tested. The ability of P_2 and related lines to resist wilt in the field was not sufficient to stem bacterial growth in the crowded flats. The F_2 and BC_1 populations, however, performed in inoculated flats almost exactly as they had in the field.

1e. Test of three bacterial strains. Strains of *P. solanacearum* have been isolated by Quimsan and Aragaki (1963) from bird of paradise (*Strelitzia reginae* Banks) and edible ginger (*Zingiber officinale* Roscoe). These strains were compared with the strain from tomato for their pathogenicity on the parental lines and the hybrids. The test was conducted from July to October, 1962 in tile beds.

The tile beds were sterilized with chloropicrin three weeks before planting. Inoculum was prepared in the same way as in previous tests (Test No. 2). At planting, the roots of the seedlings were dipped in bacterial wilt inoculum. After covering the roots with soil, 15 ml of the inoculum were poured at the base of each seedling. About 350 seedlings representing

the six populations were tested in each strain. In every case, more plants were tested in the segregating populations. Check plants were not included in this test, since susceptibility of the P_1 and check plants was similar in previous tests. In each tile bed, three rows of P_1 were planted to serve as checks. Since it was expected that the P_1 plants would die early, they were alternated with F_2 plants to fill up the anticipated vacant space and at the same time increase the number of F_2 plants. The reaction of tomato seedlings in this test is illustrated in Figure 12.

The weekly survival patterns of the breeding lines infected with tomato strain of wilt (Fig. 13) were very similar to those obtained in flat and field tests. All the P_1 died by the 5th week, with the highest number of plants dying in the 2nd and 3rd week. The P_2 remained highly resistant and F_1 plants withstood the disease well only until the 9th week. Survival curves in this test were essentially identical to those obtained in the four field tests (Fig. 7).

Weekly survival data were also taken from the six populations, following infection with wilt strain obtained from bird of paradise and edible ginger plants. The responses of the breeding lines to both bird of paradise and ginger strains were essentially identical (Figs. 14, 15). During the 2nd and 3rd week, a large proportion of the P_1 died. However, percentages of surviving plants were much greater at all times with bird of paradise and ginger strains than with the tomato strain (Fig. 13). While all the P_1 had died by the 5th week following infection with the tomato strain, almost 30% were surviving in tests with both the bird of paradise and ginger strains. Although at the end of these tests (12 weeks) all the P_1 died, the death rate was very slow compared to that in test of tomato strain. The evidence that the tomato strain was more virulent than the other two strains was confirmed by survival patterns of P_2, F_1 and segregating

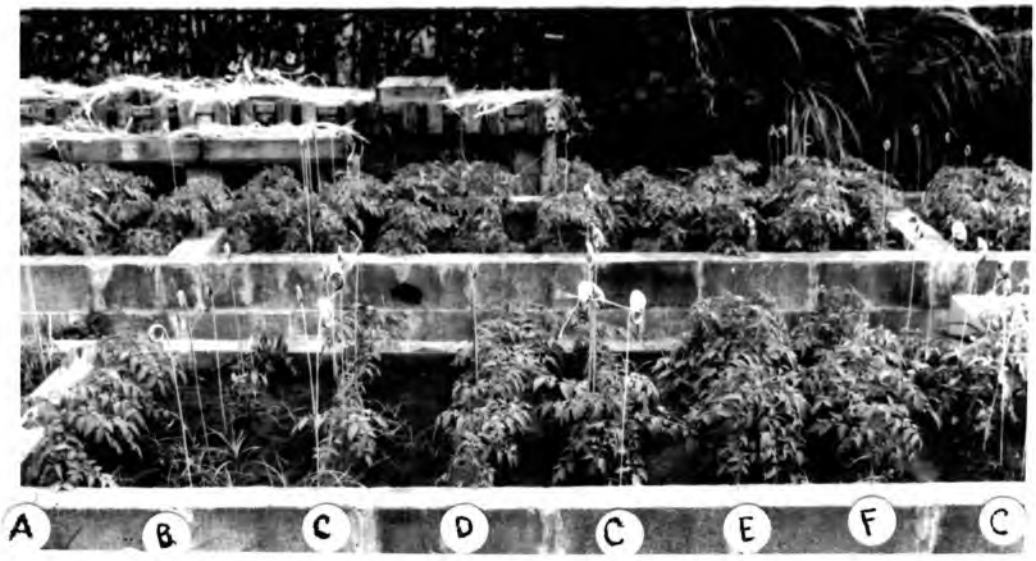


Figure 12. The breeding lines tested against tomato strain. A = BC_2; B = BC_1; C = P_1 alternated with P_2; D = P_2; E = F_1 and F = P_2.

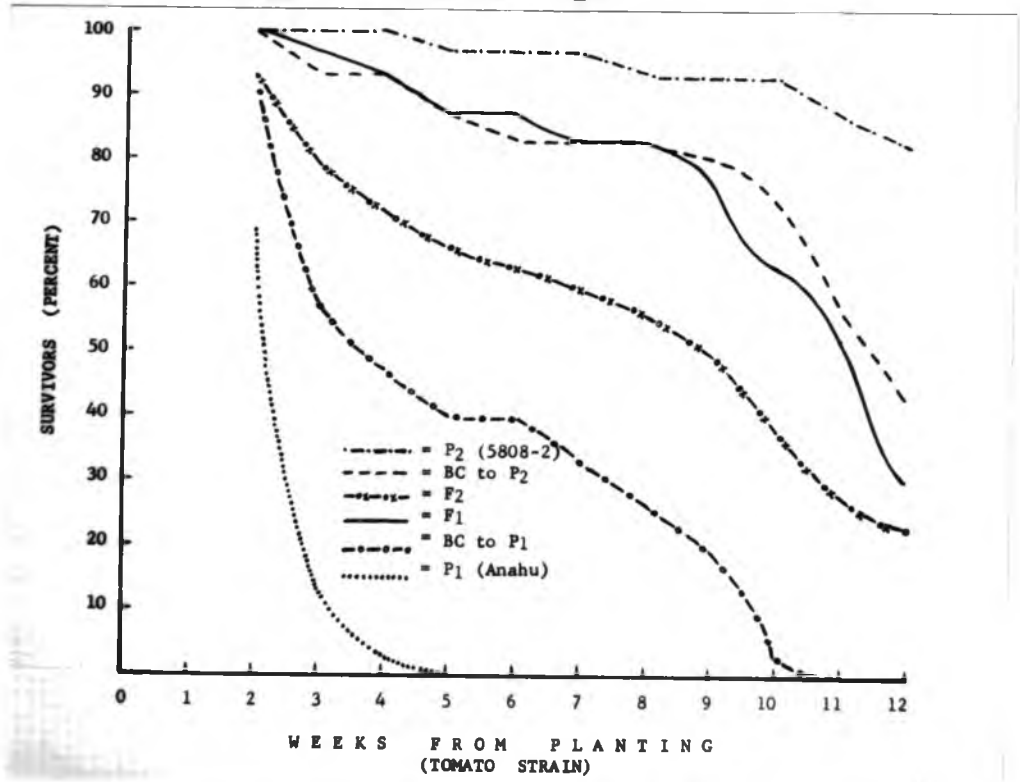


Figure 13. Weekly percentage of surviving plants in a wilt-infested soil (strain from tomato).

populations. Figure 16 shows the greater number of dead plants in the tomato strain than in both bird of paradise and ginger strains.

The F_2 showed resistance to all the strains until the 8th week. The survival rates from the 8th week to the end of the experiment were higher in tests with both the bird of paradise and ginger strains than with the tomato strain. The F_1 survival curves were essentially similar for the three strains, although plants infected with the tomato strain began to succumb in an exponential fashion by the 9th week. At last observation, 76% and 93% of the plants survived in tests with bird of paradise and ginger strains, respectively, whereas only 30% of plants infected with the tomato strain survived. In the F_2, death rate of the plants in both bird of paradise and ginger strains was similar. The F_2 survival curve in the tomato strain was the same as in previous tests. The BC_1 survival curves in all the strains were lower than the F_2. The BC_2 showed resistance similar to the F_2 in tests of bird of paradise and ginger strains. In the tomato strain, resistance was shown although about 20% of the plants died by the 9th week.

The weighted survival means (Fig. 17) of plants in the different populations also showed that death of plants in the tomato strain occurred earlier than in tests with the bird of paradise and ginger strains.

1d. Advanced progeny tests. Individual plants with differing levels of resistance were selected from the segregating F_2 and backcross lines for progeny tests. One progeny test was conducted in the spring, from March to August, 1962, with a second test in the winter, from August, 1962 to January, 1963. All the progenies tested were obtained by self-pollination. Twenty F_2 lines randomly sampled from individual F_1 parents grown in a wilt-free field were also progeny-tested in infested soil. The general field aspect of these tests is illustrated in Figure 18. The raw data from which survival

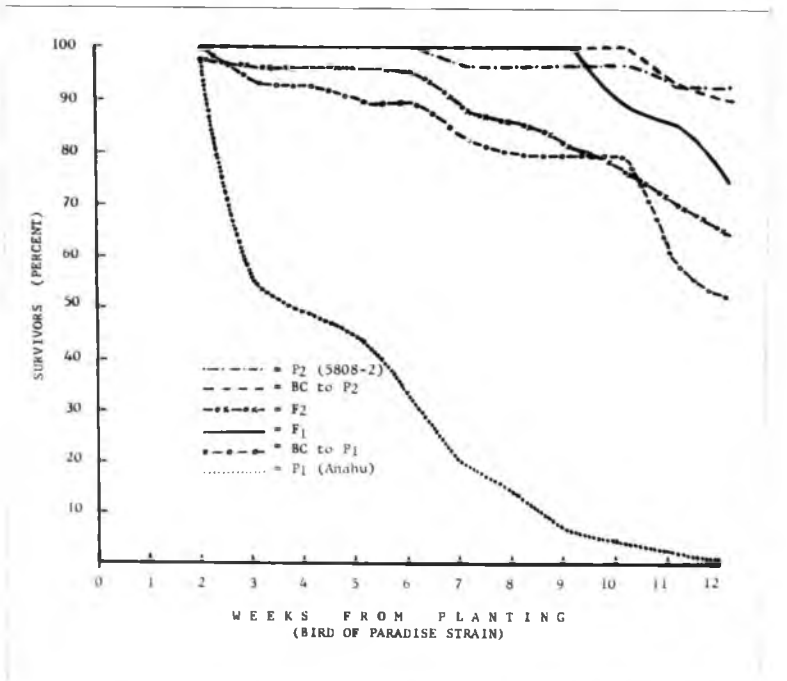


Figure 14. Weekly percentage of surviving plants in a wilt-infested soil (Bird of Paradise Strain).

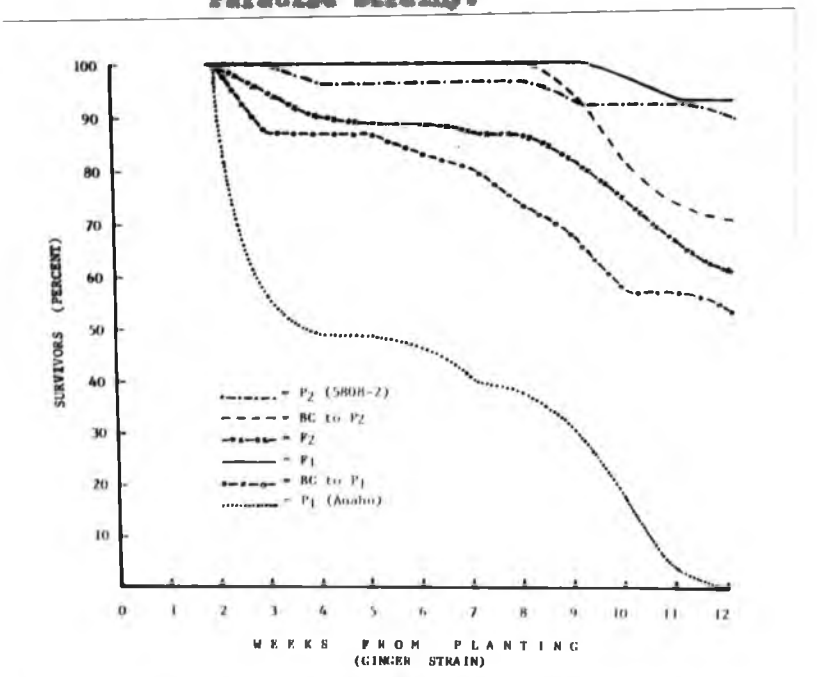


Figure 15. Weekly percentage of surviving plants in a wilt-infested soil (Edible Ginger Strain).

Figure 16. The breeding lines tested against three strains of P. solanacearum. Strains from (A) edible ginger, (B) tomato and (C) bird of paradise. Pegs with white tags indicate dead test plants, those without tags indicate dead F_1 plants.

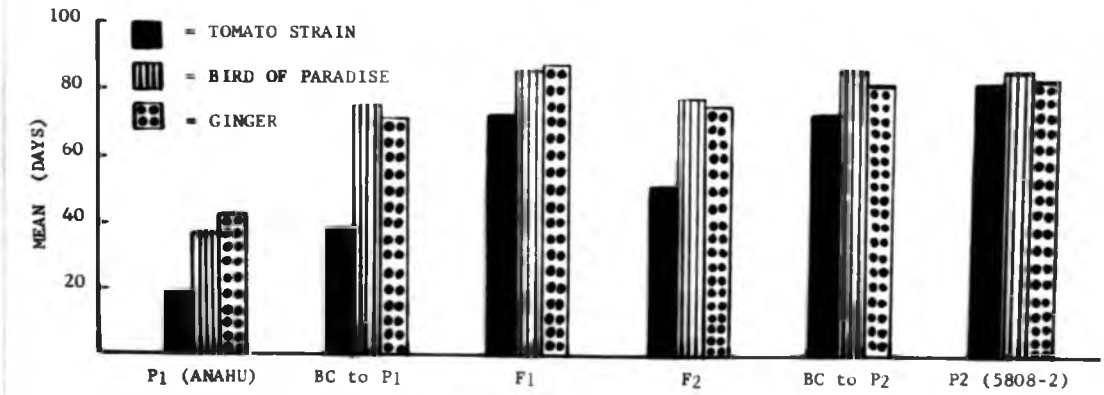


Figure 17. Weighted survival means expressed in number of days from planting until death from three strains of P. solanacearum.

means (Table 2) were calculated are presented in Appendix Table 1.

The stages of growth and durations in weeks in the normal life of a tomato crop under the conditions of these experiments were: seedling, 4 weeks; blooming, 4 weeks; maturity, 3 weeks; early bearing, 4 weeks; late bearing, 4 weeks; and senescence. These stages were used as class intervals in the classification of the parental wilt scores (Table 2). Any plant dying from wilt prior to maturity (9 weeks) was considered susceptible, since the highest percentage of dead P_1 and check plants occurred prior to maturity. The few susceptible plants that survived until maturity were not able to produce marketable fruits.

Since the susceptible parent died within 9 weeks and the resistant parent survived beyond 17 weeks, the interval of 9 to 17 weeks (early bearing and late bearing stages) was designated as intermediate (I). In this group, plants that died from 9 to 13 weeks were classed as partially susceptible (P. S.) and those that died from 13 to 17 weeks are classed as partially resistant (P. R.). The surviving plants at 17 weeks were considered resistant (R).

The advanced progenies segregated widely, a fact evidenced by the large standard errors obtained for progeny means (Table 2). Most F_3 families included a few highly susceptible plants which died from wilt in 3 - 5 weeks. In general, there was a fair correspondence between progeny means and the level of resistance of the parent.

Six partially susceptible (P. S.) F_2 parents produced progenies with survival means ranging from 63 to 81 days. Partially resistant parents produced lines ranging from 39 to 112 days, indicating that a wide range of parental genotypes were classed as partially resistant.

In general, plants classified as resistant produced progenies of intermediate response, although at least 7 of the 30 lines tested were as

Table 2. Survival means of advanced selfed progenies in number of days from planting until death from bacterial wilt. (P.S.= Partially Susceptible; P.R.= Partially Resistant).

Parent Line	Parent Wilt Score	Season	Number of Plants	Survival Means in Days	Reference Number (App. Table 1)
F_2	P. S.	Spring	268	63 ± 4.6	1
				66 ± 5.3	2
				70 ± 4.9	3
				73 ± 5.3	4
				74 ± 4.9	5
				81 ± 4.4	6
F_2	P. R.	Spring	168	78 ± 5.0	7
				92 ± 4.2	8
				94 ± 2.6	9
				112 ± 2.4	10
F_3	P. R.	Winter	83	39 ± 6.7	11
				59 ± 9.2	12
				72 ±11.6	13
				75 ± 9.5	14
F_2	P. R.	Winter	47	94 ± 4.6	15
BC_2	P. R.	Winter	57	48 ± 9.4	16
				70 ±10.6	17
				70 ±11.5	18
F_2	Res.	Spring	357	77 ± 6.3	19
				85 ± 4.9	20
				86 ± 5.2	21
				95 ± 6.7	22
				98 ± 4.7	23
				104 ± 4.9	24
				108 ± 5.0	25
				112 ± 3.5	26
F_2	Res.	Winter	87	115 ± 2.2	27
				122 ± 3.0	28
F_3	Res.	Winter	85	63 ±10.1	29
				101 ±10.2	30
				112 ± 7.9	31
				112 ± 6.6	32
BC_1	Res	Spring	55	77 ± 7.2	33
				102 ± 5.6	34

Table 2 (Continued)

Parent Line	Parent Wilt Score	Season	Number of Plants	Survival Means in Days	Reference Number (App. Table 1)
BC_1	Res.	Winter	81	49 ± 10.9	35
				63 ± 9.9	36
				77 ± 8.5	37
				95 ± 10.7	38
BC_2	Res.	Winter	50	53 ± 10.3	39
				91 ± 11.2	40
				94 ± 11.5	41
BC_1 (S_1)*	Res	Winter	144	40 ± 7.6	42
				44 ± 8.3	43
				64 ± 12.2	44
				66 ± 11.7	45
				69 ± 12.9	46
				82 ± 9.8	47
				115 ± 3.7	48
F_2	Unknown	Winter	380	23 ± 2.2	49
				29 ± 5.8	50
				40 ± 13.6	51
				56 ± 6.6	52
				57 ± 10.9	53
				60 ± 10.6	54
				70 ± 8.7	55
				71 ± 7.7	56
				75 ± 8.8	57
				77 ± 12.4	58
				78 ± 11.3	59
				80 ± 11.2	60
				80 ± 9.9	61
				81 ± 8.2	62
				82 ± 6.8	63
				87 ± 11.0	64
				89 ± 10.8	65
				102 ± 11.6	66
				105 ± 6.8	67
				116 ± 7.5	68

* BC_1 selfed twice.

resistant as the original F_1 lines ($P_1 \times P_2$). The means of two F_3 lines obtained from resistant parents (scored at 115 and 122 days) fell into the highly resistant group. Progenies from resistant BC_1 parents were classed as partially susceptible (ranging from 49 to 77 days) and partially resistant (93 and 102 days). Four F_4 lines, derived from resistant F_3 (and, in turn, resistant F_2) parents were studied. Three of the four F_4 lines were highly resistant, averaging from 101 to 112 days. The means of seven BC_1 (selfed twice) from resistant parents were highly variable in performance, ranging from 40 to 115 days.

The survival means of selfed progenies derived from 20 F_2 parents in a wilt-free field varied widely from high susceptibility to resistance comparable to that of P_2 (Table 2). Among the 380 F_3 plants (Table 3), 92 plants (or 25%) survived until the end of the test (17 weeks), at which time most of these appeared wilt-free. On the other hand, 40% of the F_3 plants were highly susceptible, succumbing to wilt within 3 weeks of planting. Between these two extremes, an almost linear increase in mortality occurred.

Table 3. Number of F_3 plants dying each week from bacterial wilt (Progenies of F_2 plants grown in a wilt-free field).

Weeks	1	2	3	4	5	6	7	8	9	10
Number of Plants	22	57	33	13	25	6	10	8	7	9

Weeks	11	12	13	14	15	16	17	Survivors
Number of Plants	8	7	9	22	26	13	11	92

Among the 20 F_3 families (Table 2), three averaged in the highly-susceptible group ranging from 23 to 40 days. The remaining family means were distributed throughout the range, with some concentration around 80 days. The weighted mean survival of this F_3 population was 72 days, compared to 93 days for the F_2 population preceding it.

Smith and Clayton (1948) progeny-tested wilt-resistant F_3 lines of tobacco. Seven of the 166 selections were highly resistant. The other 159 lines segregated widely. The proportion of highly resistant lines recovered was about the same as that for the tomato populations here.

Generally, susceptible plants were not able to produce fruits, and seeds could not be collected. It was, therefore, impossible to progeny-test plants with susceptible scores. The distributions of the survival means as a whole (Table 2) suggest that the phenotypes of the progenies represented to some extent the parental genotype, but there were gradations within all classes. There was little evidence to suggest that the season when the progeny test was conducted had a significant effect on the data collected. However, it is probable that selections of highly resistant plants for advanced tests and breeding work should be made under the most severe natural conditions of infection.

1a. Test of North Carolina tomato lines. Five tomato inbred lines obtained from Dr. H. H. Winstead of North Carolina were field-tested for wilt resistance in the spring season, March to August, 1962 (Table 4).

The survival means of the North Carolina lines ranged from 77 to 96 days. Under Hawaii conditions, therefore, the North Carolina lines were intermediate in resistance between P_2 (NHS 3808-2) and P_1 (Anahu), which averaged around 30 and 120 days, respectively.

Figure 18. Advanced progeny test for wilt resistance. Lines derived by self-pollination of (A) partially-resistant, (B) resistant, and (C) partially susceptible parents.

Early in the growing season, the North Carolina lines were vigorous, whereas the check and F_1 plants had died (Fig. 19). Later in the season, the vigor of North Carolina lines was lost and only very few fruits were formed. The plants were stunted (Fig. 20) and death occurred much earlier than in PI 3008-2 (P_2).

Plants from two North Carolina lines (NC 61-35 and NC 61-6-1) were crossed to hybrids of Anahu x 3008-2. Two North Carolina lines (NC 61-35-CF and NC 61-6-36) that produced fruits approaching commercial size were crossed with Anahu to facilitate selection of a line with high quality and wilt resistance. Hybrids were included in the winter test from August, 1962 to January, 1963 (Table 4).

The survival means of crosses of the North Carolina lines with Anahu were classed in the susceptible group. This F_1 was much more susceptible than the F_1 of North Carolina x (Anahu x PI 3008-2). These results conform with the report of McGuire (1960). The hybrids between North Carolina lines and the (highly-resistant) F_1 of Anahu x PI 3008-2 survived much later in bearing season (70 and 102 days), survival means comparing favorably with those of the F_1 of Anahu x PI 3008-2 (Fig. 13).

2. Genetic Interpretation of Data

Genetic interpretation of the wilt resistance data presented here depends greatly on the interpretation of the resistance or immunity (under experimental conditions) of the resistant parent, P_2 (PI 3008-2). Insofar as the studies permit such a conclusion, P_2 plants rarely died of wilt in the field during the period in which survival data were taken. Field death of plants in this "resistant" line occurred as a combination of environmental and pathological conditions, among which wilt may have played a minor part. However, P_2 cannot be considered immune to wilt; when careful

Table 4. Wilt reactions of North Carolina lines and their hybrids.

Lines Tested		Number of Plants	Average Number of days to death from wilt
Parent Lines:	NC 61-55	52	77 ± 3.4
	NC 61-55-OP	53	82 ± 3.0
	NC 61-S-36	20	93 ± 3.3
	NC 61-S-6	26	95 ± 8.7
	NC 61-S-1	33	96 ± 2.9
Hybrids:	NC 61-55 OP x Anahu	25	34 ± 4.5
	NC 61-S-36 x Anahu	24	42 ± 3.9
	NC 61-55 x F_1 (Anahu x 5808-2)	50	70 ± 7.1
	NC 61-S-1 x F_1 (Anahu x 5808-2)	26	102 ± 6.1

Figure 19. (A) North Carolina wilt-resistant line (NC 61-55-CP), (B) test plant, and (C) check plants on both sides, died early in the season.

Figure 20. North Carolina line at bearing stage.

inoculations are made with massive doses of the pathogen, it also succumbed to the bacterium (Fig. 13).

It is held by most students of bacterial resistance that, in both animals and plants, true immunity does not exist. The immunological resistance and genetic resistance of rats to typhoid bacteria, for example, reflects the unusual ability of the resistant animals to slow down bacterial multiplication, while immunity as such does not occur. The same conclusion appears valid for the resistance of tomato lines tested here to wilt-inducing bacteria.

On this important assumption -- that F_2 was essentially field-immune to wilt under conditions of the experiments conducted here -- rests the majority of the genetic interpretations which follow.

As a second major fact in interpreting these data, it is clear that F_1 plants were never immune, but succumbed to the disease ultimately in all tests. Resistance, if it may be called that, of the F_1 plants was simply the ability to suppress the bacterium and to grow for a much longer period than the susceptible parent. It should be noted that this resistance was sufficiently great to carry most F_1 tomatoes through the heavy-bearing season. Thus, while the F_1 plants must be considered susceptible rather than immune, they had a practical level of resistance of great value to the plant breeder.

A primary conclusion derived from a comparison of the F_1 and its parents must be simply that the genes of neither parent are fully dominant. Earlier interpretations of similar data by Singh (1961) were made on the early flowering and fruiting stages. At these stages, the F_1 plants are apparently healthy. One can conclude, as Singh did, that genes for resistance are "dominant" at this stage. In practice, this is a useful way to consider the data, although it infers that one must consider the same (or other) genes

for resistance to have been recessive at later stages, when all the F_1 plants have proved susceptible to the disease. Each of these considerations, while useful to the breeder, are invalid for correct genetic interpretation of the data. The genes do not change in their dominance, nor do we need to suggest that some genes act at early stage (dominant alleles confering resistance) while other genes act at later stage (their dominant alleles conferring susceptibility). Rather, in line with the interpretation of other bacterial and viral resistance data, it must be considered that the genes conferring resistance are largely additive in their effect, conferring on the F_1 the ability to suppress the growth and symptoms of the bacterial invasion until much later in a tomato's development. On this basis, most of the following interpretations have been made.

2a. Exponential survival curve. The weekly percentages of surviving plants were plotted on a semi-logarithmic scale in an attempt to clarify the relationships of death rates among populations (Fig. 21). One of the major factors influencing bacterial disease development is the differential rate of growth of the pathogen in different host plants. Sadasivan (1961) reported that there was generally a direct relationship between wilt development and numbers of resident pathogenic bacteria. Severe injury to the root system at transplanting provides an avenue of entrance of the organism into the host; as Sadasivan (1961) stated, wilt-susceptible plants produce greater amounts of root exudates than do those of resistant plants. The wounds in the roots may have induced a greater release of the exudates (amino acids and sugars) thus creating a condition conducive for the growth of the organism.

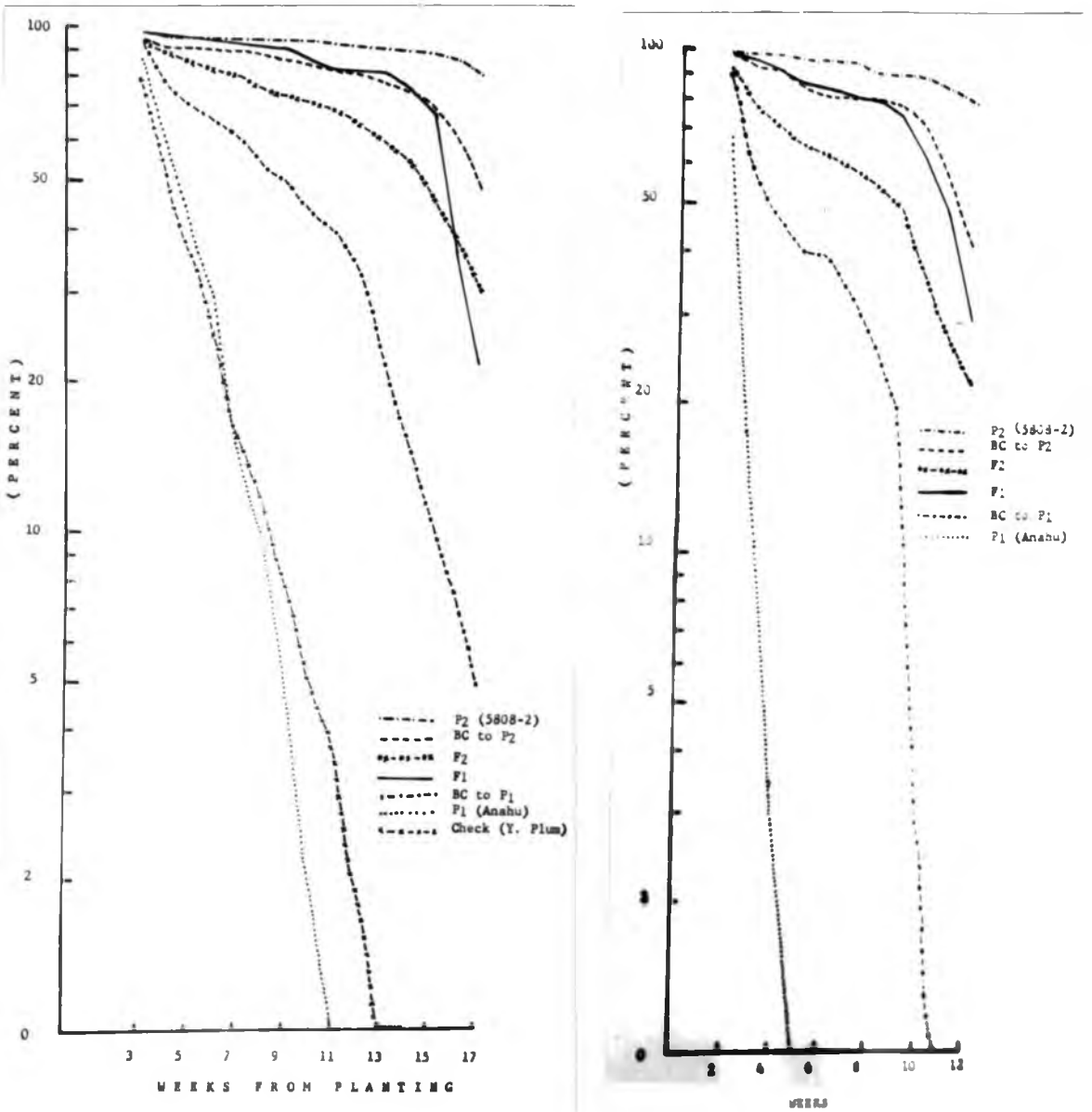
On the semi-logarithmic scale, the F_1 and check plants demonstrated perfectly linear death rates. These exponential survival patterns mimic those of other living organisms under stress, when no resistance or

protection is available. Sax (1962) referred to such stress as a condition arising particularly from pathological alteration of host tissues and deterioration of cell colloids. It may be assumed that susceptible plants have essentially no genetic or physiological ability to prevent or delay this deterioration. In contrast, the genes for resistance in F_2 presumably stopped multiplication or activity of the organism in field tests. Under tile bed conditions, the F_2 showed some death from wilt, only in the last few weeks of growth.

The results from the genetically-uniform F_1 population are less clearly interpretable, referring to the log-transformed data (Fig. 21). Under field conditions, F_1 plants withstood the bacterial attack about 15 weeks and then started to succumb, again in exponential fashion. In the heavily-infested tile beds, however, F_1 plants appeared to include two groups, about 20% dying between the 2nd and 6th week, the remainder passing out exponentially from the 10th week on. It is inferred that some segregation occurred among the F_1 plants.

The F_2 populations (Fig. 21) regressed toward the susceptible parent, and present the multitangential curve expected for polygenic segregation. If one gene governed resistance, one would expect the F_2 curve to dip sharply in the first few weeks, level out, then dip again. This did not occur. In the BC_1 (F_1 x susceptible parent), however, the superimposed exponential patterns became evident. Many BC plants died in the first few weeks, to produce an initial dip in the survival curve. Of more interest is the fact that the most resistant BC plants (about 30%) survived until about the time exponential drop appeared in the F_1.

Exponential portions of the curves for F_1, BC_1 and F_1 were parallel, insofar as could be determined (Fig. 21). It is inferred that levels of resistance to the bacterium reflect retardation of its attack or multiplication, an hypothesis called for by the parallelism of survival curves.



Mean of four seasons

Tile bed test

Figure 21. Weekly percentage of surviving plants in bacterial wilt-infested soils (Semi-logarithmic scale).

2b. Number of genes governing resistance. In order to discuss the results of these studies from a genetic standpoint, the distributions of the populations were considered (Table 5), using the groupings discussed previously. Data were summarized from the four field plantings (Fig. 7). When the populations of F_2 plants were classified as susceptible (S), intermediate (I), or resistant (R), a large proportion (32%) of resistant plants was recovered. This result suggests that a small number of genes may be involved in determining wilt resistance. Assuming no dominance or geometric effects, each allele has an equal and cumulative effect, and an F_2 distribution of 1(S): 2(I): 1(R) based on one gene pair could be calculated. The summarized F_2 data (Table 5) were close to this distribution, although individual seasons varied considerably about it. The observed values of S:I:R in the two backcross populations were close to unity.

Chi-square tests for monogenic ratios were made on the F_2 data for nine weeks (susceptible group) and at 17 weeks (resistant group). Chi-squares were also calculated for BC_2 ratios so as to confirm the inferences drawn from the F_2 ratios (Tables 6, 7). No dominance was assumed, the assumption being only that (S) = one homozygote, (R) = other homozygote, (I) = heterozygote.

At nine weeks, the F_2 chi-square values during the summer, fall and winter tests showed poor fits for the 1:3 ratio. The pooled chi-square value (3.4), however, fitted the expected values (Table 6). In the BC_1, the chi-square value in the fall test showed a good fit for 1:1 ratio. However, the pooled chi-square value (4.2) was significantly large ($P < .05$).

The F_2 values at 17 weeks (Table 7) in the fall and winter tests showed wide deviations from the expected ratio. Likewise, the pooled chi-square value was significantly large ($P < .01$). In the BC_2, values obtained in the spring test and pooled values deviated widely from the expected.

Table 5. Percentage of dead plants from bacterial wilt in the susceptible, intermediate and resistant groups.

Population	Season	Susceptible (1-9 weeks)	Intermediate (10-17 weeks)	Resistant Survivors	Total Plants
	Summer	100.0	--	--	
P_1	Fall	100.0	--	--	371
(Anahu)	Winter	81.7	18.3	--	
	Spring	97.8	2.2	--	
	Average	94.9	5.1	--	
	Summer	74.0	22.5	3.5	
	Fall	52.4	42.8	4.8	
BC_1	Winter	37.8	59.7	2.5	544
	Spring	33.2	59.2	7.6	
	Average	49.3	46.1	4.6	
	Summer	22.5	48.3	29.2	
	Fall	4.8	52.3	42.9	
F_1	Winter	0	86.6	13.3	340
	Spring	0	96.2	3.8	
	Average	6.8	70.9	22.3	
	Summer	41.3	34.2	24.5	
	Fall	19.2	34.5	46.4	
F_2	Winter	12.5	57.3	30.2	1,482
	Spring	24.1	50.9	25.0	
	Average	24.3	41.2	31.5	
	Summer	24.7	30.7	44.6	
	Fall	15.0	40.0	45.0	
BC_2	Winter	3.3	29.2	67.5	538
	Spring	0.8	57.2	42.0	
	Average	11.0	39.2	49.8	
	Summer	8.3	8.3	83.4	
P_2	Fall	0	5.0	95.0	
(5808-2)	Winter	2.5	0	97.5	335
	Spring	2.6	32.9	64.5	
	Average	3.4	11.6	85.1	
	Summer	100.0	--	--	
Check	Fall	86.4	13.6	--	
(Y. Plum)	Winter	83.0	17.0	--	4,768
	Spring	99.0	1.0	--	
	Average	92.2	7.8	--	
F_3	Winter	47.9	26.9	24.2	380

The F_3 population (Table 5) would be expected, on a monogenic hypothesis, to segregate 3S:2I:3R. The obtained ratio (40%:27%:25%) was clearly discrepant from this expectation. Non-random sampling of F_2 might have contributed to the bias here.

The data from greenhouse tests were also grouped into arbitrary S, I and R groups, and tested on the monogenic hypothesis with nodominance (Table 8). In test number one, the F_2 chi-square value at 47 days from planting (maturity stage) was 4.0. In the BC_2, the values fitted the expected 1:1 ratio. In test number two, the observed values in the F_2 and BC_2 were close to the expected. In laboratory test number three (tile bed), the F_2 chi-square value of 0.4 reflected a good fit to 3:1 ratio, and that from BC_2 a significant discrepancy from expectation.

While some of the distributions satisfy a monofactorial hypothesis for resistance, others departed significantly. It was apparent also that the classification of S:I:R on which this simple assumption was based was only arbitrary.

The alternative explanation of these data is that of a multifactorial basis for resistance. This hypothesis is more in keeping with results from studies of other bacterial pathogens of plants and animals. Several facts appear to favor the multifactorial explanation. It is evident that any multifactorial distribution could be arbitrarily divided to give a 1S : 2I : 1R ratio in F_2, with 1:1 BC ratios. Therefore, the designation of dividing points in time between S, I and R plants may have produced pseudo-monofactorial segregations. Critical evidence on the number of governing genes requires extensive advanced progenies. The advanced progenies grown here (Table 2) permit some conclusions, however. In the first instance, the distribution of F_3 plants derived from F_2 plants of unknown resistance deviated from the 3S: 2I: 3R expected on a monofactorial hypothesis (Table 3).

Table 6. Numbers of dead and surviving plants in bacterial wilt-infested fields at maturity (9 weeks from planting) with numbers expected under the monogenic hypothesis.

Season	Observed		Expected		Chi-square	P value
	Dead	Survivors	Dead	Survivors		
P_2 (1:3)						
Summer	98	139	59.3	177.7	33.8	0.01
Fall	79	333	103.0	309.0	74.5	0.01
Winter	40	281	80.2	240.8	26.9	0.01
Spring	123	389	128.0	384.0	0.3	0.60
Pooled	340	1,142	370.5	1,111.5	3.4	0.07
BC1 (1:1)						
Summer	105	37	71.0	71.0	32.6	0.01
Fall	11	10	10.5	10.5	0.1	0.82
Winter	45	74	59.5	59.5	7.0	0.01
Spring	87	175	131.0	131.0	29.6	0.01
Pooled	248	296	272.0	272.0	4.2	0.03

Table 7. Numbers of dead and surviving plants in bacterial wilt-infested fields at seventeen weeks from planting with numbers expected under the monogenic hypothesis.

Season	Observed		Expected		Chi-square	P value
	Dead	Survivors	Dead	Survivors		
F2 (3:1)						
Summer	179	58	177.7	59.3	0.1	0.85
Fall	221	191	309.0	103.0	100.2	0.01
Winter	224	97	240.8	80.2	4.7	0.03
Spring	384	128	384.0	128.0	0.0	1.00
Pooled	1,088	474	1,111.5	370.5	39.2	0.01
BC2 (1:1)						
Summer	83	67	75	75	1.7	0.18
Fall	11	9	10	10	0.2	0.60
Winter	61	59	60	60	0.3	0.55
Spring	164	104	124	124	6.4	0.01
Pooled	299	239	269	269	6.7	0.01

Table 8. Numbers of dead and surviving F_2 (3:1) and BC_2 (1:1) in bacterial wilt-infested soils under monogenic hypothesis.

Population	Observed		Expected		Chi-square	P value
	Dead	Survivors	Dead	Survivors		
Test No.1 (Sandflat)						
F_2	30	18	36	12	4.0	0.04
BC_2	28	20	24	24	1.3	0.25
Test No. 2 (Sandflat)						
F_2	239	97	252	84	2.7	0.10
BC_2	39	19	29	29	6.9	0.01
Test No. 3 (Tilahed)						
F_2	102	30	99	33	0.4	0.55
BC_2	17	13	15	15	5.3	0.02

The mean survival dates of F_3 families were distributed throughout the weeks studied, with, however, some evidence of trimodal dispersion (68:111 : 38 families). The survival patterns of BC_1 plants (Fig. 21) are critical to interpretation of the data. If this cross segregates monofactorially, about 50% of the plants should die in the first 9 weeks, followed by a plateau with little death occurring until about the 15th week (when exponential loss of F_1 plants occurred). A sharply bimodal death pattern of BC_1 plants seems not to have occurred, suggesting multifactorial patterns. Similarly, the distribution of deaths among F_3 plants (Appendix Table 1) was more or less continuous, without the expected trimodality of a monofactorial distribution. Several F_2 plants classified as resistant showed intermediate and susceptible F_3 segregants. This also suggests that the R plants are not homozygous for a single resistant allele.

Since it is of practical importance for resistance to hold up until mid-bearing season, the data for wilt resistance were also grouped in class intervals according to the stages of growth of the plants (Table 9). Based on these stages, frequency curves of the different populations were constructed (Fig. 22). These distributions similarly indicate the probability of a multifactorial basis for resistance.

Since the death of P_2 at the end of the season can be attributed to many factors, other than bacterial wilt, interpretation centers on the relationships among P_1, BC_1 and F_1 populations. Both P_1 and F_1 populations were unimodal, and more or less normally distributed when the pooled data are considered. However, the log-transformed data presented earlier (Fig. 21) clearly show these to be exponential curves, departing significantly from normality. In other words, a certain fraction of survivors died each week during the exponential killing periods (4 - 13 weeks for P_1, 13 - 19

Table 9. Percentage of plants dying in wilt-infested fields classified by stages of growth.

Population	Season	SUSCEPTIBLE		INTERMEDIATE		RESISTANT
		Blooming 1-4 wks.	Maturity 5-9 wks.	Bearing Life 10-13 wks.	14-17 wks.	Survivors at 17 wks.
P_1 (Anahu)	Summer	55.0	45.0	--	--	--
	Fall	67.3	32.5	--	--	--
	Winter	6.7	75.0	18.3	--	--
	Spring	24.2	73.6	2.2	--	--
	Average	38.3	56.5	5.2	--	--
BC_1	Summer	39.5	34.5	18.3	4.2	3.5
	Fall	38.1	14.3	33.3	9.5	4.8
	Winter	2.5	35.3	41.2	18.5	2.5
	Spring	13.0	20.2	17.6	41.6	7.6
	Average	23.3	26.1	27.6	18.5	4.6
F_1	Summer	5.0	17.5	30.0	18.3	29.2
	Fall	0	4.8	0	52.4	42.9
	Winter	0	0	0	86.7	13.3
	Spring	0	0	1.2	95.0	3.8
	Average	1.2	5.6	7.8	63.1	22.3
F_2	Summer	19.4	21.9	20.7	13.5	24.5
	Fall	9.2	10.0	9.0	25.5	26.4
	Winter	4.1	8.4	16.5	40.8	30.2
	Spring	7.8	16.2	9.8	41.2	25.0
	Average	10.1	14.1	14.0	30.3	31.5
BC_2	Summer	19.3	5.3	20.7	10.0	44.7
	Fall	10.0	5.0	10.0	30.0	45.0
	Winter	2.5	0.8	0.8	28.3	67.5
	Spring	0	0.8	6.0	51.2	42.0
	Average	8.0	3.0	9.4	29.9	49.8
P_3 (5808-2)	Summer	5.0	3.3	2.5	5.8	83.3
	Fall	0	0	0	5.0	95.0
	Winter	2.5	0	0	0	97.5
	Spring	2.6	0	2.6	30.3	64.5
	Average	2.5	0.8	1.3	10.3	85.1
Check (Y.Plum)	Summer	67.8	32.2	--	--	--
	Fall	41.8	44.6	10.0	3.6	--
	Winter	20.5	62.4	16.1	1.0	--
	Spring	63.9	35.6	0.5	--	--
	Average	48.5	43.7	6.6	1.2	--

weeks for F_1). The data obtained until 17 weeks reflect an incomplete picture of genetic resistance. Analysis of the data gathered beyond 17 weeks in the fall and winter tests reveal a continuous distribution of time of wilt-induced death of plants formerly classified as resistant (Table 1). Death patterns in the winter test (Fig. 23) have been plotted for the entire 21 weeks studied. One striking observation, alluded to previously, is that the BC_1 did not show a bimodal distribution (expected on monofactorial basis), but rather a continuous distribution.

The modes of the F_1 and F_2 populations were essentially the same, although the mean of F_2 was much smaller. The BC_1 mean was about intermediate between P_1 and F_1.

2e. Dominance. It has been considered throughout previous discussions that dominance was lacking. It is convenient to think of the observations in terms of "phenotypic dominance". To the plant breeder, the resistance expressed by the F_1 at the critical fruit-bearing age is conveniently called dominance. However, the F_1 plants ultimately died from wilt, and might, therefore, lead to the argument that susceptibility was phenotypically dominating. From the standpoint of the genes involved, however, little can be said about dominance. The F_1 was more resistant than one parent, more susceptible than the other. These suggest largely additive effects of the alleles involved, a conclusion similar to that of Donado (1958). One index of dominance variance would be the regression of F_2 mean toward one or other parent. Such a regression appears to have occurred (Table 10), with the F_1 mean = 107, and F_2 = 93 days. This could be interpreted to indicate partial dominance of genes for resistance. Since the survival curves are not normally distributed, however, this conclusion should relate to log-transformed means, and the difference is less on such a scale. Any comparison of such means is invalid, however, since the means are based on the

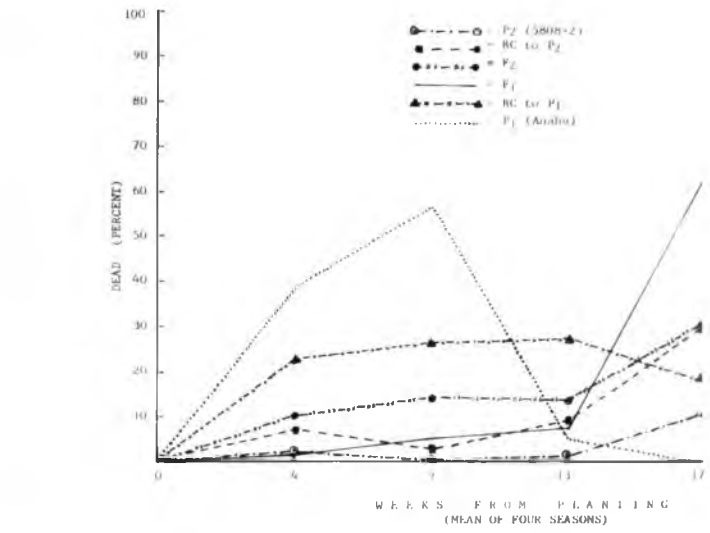


Figure 22. Percentage of dead plants in bacterial wilt-infested fields occurring in consecutive stages of growth (Means of four seasons).

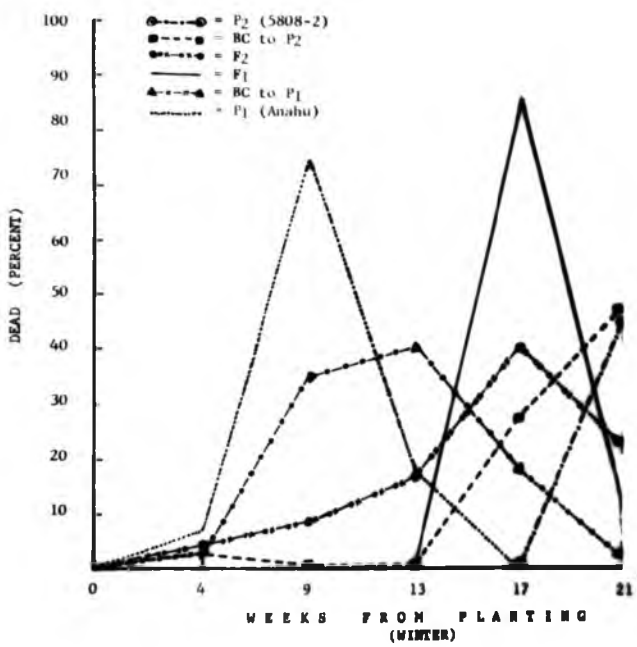


Figure 23. Percentage of dead plants in a bacterial wilt-infested field occurring in consecutive stages of growth (winter test).

Table 10. Weighted means expressed in number of days from planting until death from bacterial wilt (Duration of experiment = 17 weeks)

Population	Season	Weighted Means in Days	Total Number of Plants
	Summer	30 ± 0.7	120
F_1	Fall	30 ± 1.3	40
(Amahu)	Winter	55 ± 1.4	120
	Spring	38 ± 0.4	91
	Average	38 ± 0.9	
	Summer	49 ± 2.4	142
	Fall	61 ± 7.1	21
BC_1	Winter	77 ± 2.4	119
	Spring	79 ± 2.1	262
	Average	67 ± 3.5	
	Summer	89 ± 2.9	120
	Fall	113 ± 3.6	21
F_1	Winter	117 ± 2.1	119
	Spring	111 ± 0.7	80
	Average	107 ± 2.3	
	Summer	75 ± 2.6	237
	Fall	101 ± 1.7	412
F_2	Winter	105 ± 1.1	321
	Spring	92 ± 1.5	512
	Average	93 ± 1.7	
	Summer	88 ± 3.4	150
	Fall	106 ± 7.3	20
BC_2	Winter	122 ± 1.8	120
	Spring	112 ± 0.9	248
	Average	107 ± 3.4	
	Summer	114 ± 2.4	120
P_2	Fall	126 ± 0.4	20
(5808-2)	Winter	133 ± 1.6	120
	Spring	117 ± 2.0	76
	Average	123 ± 1.6	
	Summer	28 ± 0.2	951
Check	Fall	42 ± 0.9	552
(Y. PLUM)	Winter	50 ± 0.6	1,057
	Spring	29 ± 0.2	2,208
	Average	37 ± 0.5	

assumption that plants surviving at 17 weeks would have died of wilt within an average of 7 days from that time. In effect, many of these resistant plants may not have succumbed at all to wilt.

Another way to examine possible dominance effects is that of calculating means, on the log scale (Fig. 21), for F_1, BC_1, and P_1. At LD_{37} (37% killing), these were approximately 4.5, 12 and 16 weeks, respectively. The BC_1 average exceeds the mid-parental point. On multifactorial basis, dominance variance of BC_1 (and F_2) should equal one-half that of F_1, and BC_1 should equal midparent irrespective of dominance. In effect, these considerations indicate that the time scale chosen does not permit direct measure of dominance contribution in any multifactorial analysis. The time of onset of exponential killing in any genetically-uniform population of tomatoes appears to reflect the physiological-limitation of bacterial growth rates. These times appear not to be normally distributed, but additive only upon log transformation. This does not suggest geometric (multiplicative) gene action, but probably reflects only non-additive growth of the pathogen (as do survival curves).

It must be concluded that dominance is lacking or, alternatively, that the data do not permit its recognition.

The intermediate resistance of North Carolina lines and performance of their hybrids (Table 4) also support a multifactorial interpretation. Assuming the North Carolina lines carrying fewer genes for wilt resistance than P_2, progenies of North Carolina x Anahu would be expected to be more susceptible than the progenies of NC plants x F_1 (Anahu x 5808-2). Table 4 shows that this was, in fact, true.

Smith and Clayton (1948) also reported a cumulative effect of genes for wilt resistance in tobacco. They attempted to accumulate genes for high resistance (immunity) but the final result was negative. This result

was attributed in part to the irregular occurrence of severe wilt infestations in the field.

Because of the extreme variability of results and the many assumptions made in the analysis of the data, the results at hand do not give specific information as to the number of genes involved in wilt-resistance nor their degree of dominance. It seems probable that resistance is controlled multigenically and probably it involves major genes with minor modifying factors. It is suspected, however, that a low number of genes are involved; if many genes governed resistance, F_2 segregants with all or most of the "plus" or "minus" genes would rarely appear. Disease expression is altered by differences in pathogenicity of strains, by environmental growing conditions, and by the kinetics of pathogenic populations in the host.

3. Other Studies

In Root knot nematode reactions. Plants sampled from various levels of wilt resistance were tested for nematode response in seedling tests following the method of Gilbert and McGuire (1936). Since the wilt-resistant parent (P_2) is susceptible to nematods, this test was undertaken to determine the possible association of these characters. The parents as well as F_1 and F_2 families were included in the tests.

In the greenhouse, the seeds were planted in gallon cans filled with sterilized soil. Approximately 80 grams of fresh, heavily galled roots were distributed in a layer of about 3 cm, just below the seeds.

Five classes of root knot susceptibility were used in the readings. Class 1 plants showed no visible galls of any size. Class 2 plants had one or few tiny galls. Class 3 plants had greater number of small galls, but no larger galls. Class 4 plants had wide distribution of small galls larger than those of class 3 plants, and class 5 plants had heavy galls. The gall index was calculated by using the following formula:

$$\frac{\text{Class value x number of plants in the class}}{\text{Total number of plants}}$$

The wilt-infested fields used in this study were infested with natural populations of root knot nematodes. During the winter test, the response of the wilt-survivors was determined by digging the plants. The numbers of plants with and without galls were recorded.

Table 11 shows the reactions of the progenies to galling. In test number one, the gall index of the P_1 was 1.4, and the P_2 was 3.7. The index of F_1 was the same as P_1. This result conforms with the known dominant monofactorial condition of nematode resistance.

If wilt resistance is associated with nematode susceptibility, the progenies of the wilt-resistant P_2 parents might be more susceptible to nematode. The indices of eight lines from wilt-resistant parents varied from 1.8 to 2.8. These indices were intermediate to the parents, perhaps closer to the gall index of P_1 than P_2.

Among the three indices of progenies from wilt-partially susceptible parents, one was approaching the P_2. The other two lines showed substantial resistance to nematode.

In test number two (Table 11), the P_1 plants were also rated as resistant. One out of the four wilt-resistant parents showed resistance similar to the P_1. The intermediate gall indices approached the gall index of the P_2.

Among three lines from partially wilt-resistant parents, one line was completely susceptible. The gall index was the same as the susceptible parent (P_2).

These results demonstrate that wilt resistance was not associated with the Mi^+ locus (root knot nematode susceptibility) on chromosome 6. This finding is supported by the results obtained from the field test. The data

Table 11. Response of the parents and hybrids to root knot nematode.

Population	Test No. 1		Test No. 2	
	Number of Plants	Gall Index	Number of Plants	Gall Index
P_1 (Annbu)	144	1.4	26	1.0
F1	82	1.4	--	--
F_2	231	1.8	84	2.2
F_2	119	3.7	17	4.0
F_2 (partially wilt-susceptible)	82	1.7	28	1.4
	72	1.8	29	1.4
	108	3.0	28	2.1
F_2 (partially wilt-resistant)	--	--	24	1.7
	--	--	37	1.7
	--	--	23	4.0
F_2 (wilt-resistant)	146	1.8	29	1.6
	101	1.9	44	2.0
	101	2.2	9	2.4
	121	2.2	37	2.9
	102	2.4	--	--
	97	2.6	--	--
	130	2.6	--	--
	178	2.8	--	--

Table 12. Response of wilt-survivors in a bacterial wilt-infested field to root knot nematode.

Parent Line (Selfed)	Parent Wilt Score	Number of Lines	Number of Plants	Number of Plants	
				Nematode-Resistant	Nematode-Susceptible
F_2	Unknown	3	7	--	7
BC_1 (S_1)*	Resistant	1	5	--	5
F_2	Unknown	4	33	23	10
F_3	Resistant	3	35	28	7
BC_1	Resistant	1	8	4	4
BC_1 (S_1)*	Resistant	2	16	12	4
BC_2	Par. Sus.	1	3	1	2
BC_2	Resistant	2	15	12	3
F_2	Unknown	9	52	58	--
F_3	Resistant	1	14	14	--
BC_1	Resistant	3	11	11	--
BC_1 (S_1)*	Resistant	3	14	14	--
BC_2	Par. Res.	1	3	3	--
BC_2	Resistant	1	6	6	--
Total		35	222	180	42

* Selfed twice

in Table 12 indicate that regardless of whether the parental plant was resistant to wilt or not, the progenies were either root knot nematode-susceptible or resistant. Of the 222 surviving plants in a wilt-infested field, 180 plants had clean roots and 42 were heavily galled. A chi-square test based on 3:1 ratio gave a value of 4.4 ($P = 0.04$). Although the chi-square value showed a poor fit, the observed numbers approximated the expected values.

3b. [illegible] on growth habit. In the four field tests, segregation for plant form of either determinate (spsp) and indeterminate (sp^+) was observed among surviving plants (considered resistant to wilt) at 17 weeks. Indeterminate growth is controlled by a single dominant pair of genes, and the F_2 ratio would have been 3:1 for this trait if there was no association between this character and wilt resistance.

The observed numbers of surviving plants with the sp^+ and spsp phenotypes deviated widely from the expected values (Table 13). Out of 474 F_2 plants, only 14 were determinate. The data indicate that at least part of the genes for wilt resistance are linked with the sp^+ locus on chromosome 6, or that there is some generalised functional association between the traits.

No resistant plant with commercial fruit size was recovered in the F_2 (Fig. 24). The question arises as to whether resistance is associated with small fruit size. No experimental procedure was undertaken to answer this question, but there was slight indication that such an association existed. Again this could be explained by assuming that part of the genes controlling wilt resistance are linked with the genes governing fruit size, or that other associations exist. The result also demonstrates that where disease resistance is polygenically controlled, the transfer of resistance to a crop variety with complex quality characters is difficult to attain.

Some promising plants in advanced generations were selected with wilt tolerance and improved fruit size (Fig. 25). It remains to be seen whether or not all the genes of *L. pimpinellifolium* concerned with wilt resistance can be divorced from their parental genome and transferred to a larger fruited type.

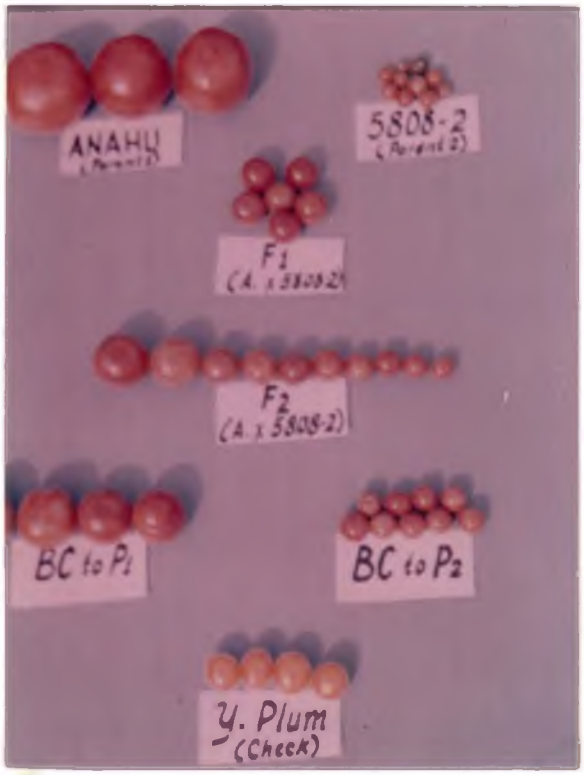


Figure 24. Representative fruits of the parents and hybrids (Anahu x HRS 5808-2).

Table 13. Number of surviving (wilt-resistant) F_2 plants in bacterial wilt infested fields with determinate and indeterminate growth.

Character	Total Number of Plants	Season			
		Summer	Fall	Winter	Spring
Indeterminate	460	58	187	95	120
Determinate	14	--	4	2	8
Total	474	58	191	97	128

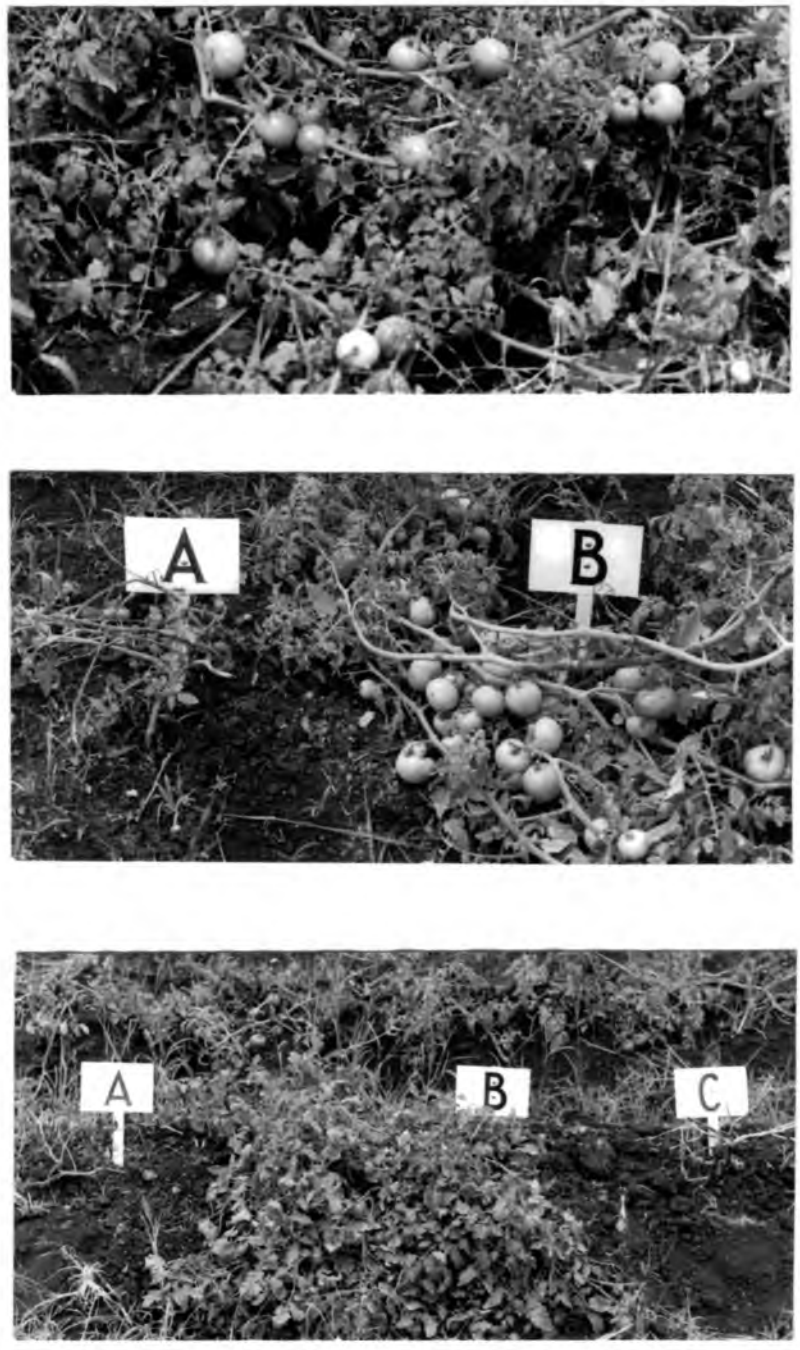


Figure 25. A wilt-tolerant progeny from a cross of North Carolina line x F_1 (Anahu x 5808-2) with improved fruit size (top) and F_5 selection from Anahu x 5808-2 (center). Note stunted test plant (center A). A wilt-tolerant determinate hybrid (bottom B) with dead test plants on both sides (A and C).

V. SUMMARY AND CONCLUSIONS

Weekly survival data were recorded for almost 13,000 tomato plants grown under conditions of severe bacterial wilt (*Pseudomonas solanacearum* E. F. S.). The progenies scored were derived from crosses of a susceptible commercial tomato, var. Asahu, and a wilt-resistant inbred from the species, *Lycopersicon pimpinellifolium* Mill. Results obtained from field trials in six seasons were corroborated by tests in wilt-inoculated flats and tile beds. The severity of the disease varied seasonally, with the most severe expression in the summer months, at times of highest soil temperatures.

Survival curves for the susceptible parent and (genetically-uniform) F_1 were exponential with time, 50% lethality occurring at about 4.5 and 16 weeks, respectively, after transplanting to the field. The resistant parent rarely succumbed to wilt in the field, and most plants were surviving at 17 weeks (end of good fruit-bearing) when experiments were concluded. Backcrosses of F_1 and the susceptible parent segregated plants varying widely in time of death, with exponential killing starting in about the 12th week. The other backcross was similarly intermediate to its parents, and F_2 and F_3 families segregated widely. A relatively high proportion of F_2 and F_3 segregants were classified as equally susceptible to the susceptible parent.

The data from segregating families were interpreted multifactorially, in the absence of convincing evidence of one or a few major genes for resistance. Partial dominance of genes conferring resistance could be suggested from the data, but with the caution that another choice of scale of measurement might change this conclusion. Otherwise, gene action must be held to be entirely additive. When the data were arbitrarily grouped as susceptible (dying within 9 weeks of planting), resistant (surviving at late-bearing stage, 17 weeks after planting), and intermediate, the following

ratios were approximated: BC_1 = 1S : 1I, BC_2 = 1I : 1R, F_2 = 1S : 2I : 1R, and F_3 = 2S : 1I : 1R. These ratios suggested that resistance could be dealt with in the breeding program much as if homozygosity for one or a few genes conferred resistance.

Several North Carolina inbred lines, which had been bred for wilt resistance, proved to be intermediate in wilt-susceptibility under Hawaiian conditions. Their progenies segregated as if the N. C. lines carried many, but not all, the additive factors for resistance of the L. pimpinellifolium line.

Survival rates among tomatoes of all lines were higher when the inoculum was obtained from infected bird of paradise (Strelitzia reginae Banks) or edible ginger (Zingiber officinale Roscoe) than when inoculum was taken from susceptible tomato plants.

There appeared to be no association of wilt resistance with root knot nematode susceptibility (mi^+). However, indeterminate growth (sp^+) was associated with bacterial wilt resistance, suggesting a linkage between sp^+ and resistance genes.

Resistant plants with commercial fruit size were not recovered. Crosses of the F_1 (Anahu x NES 5008-2) with large-fruited North Carolina lines having intermediate resistance gave promising, wilt-tolerant selections with improved fruit size.

APPENDIX

Table 1. Number of plants dying each week in the progenies of parents with different levels of wilt resistance (See Table 2 in text)

Reference Number	Weeks 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	Survivors	Total Plants
1	0	1	3	7	3	3	4	5	2	3	3	2	4	3	1	3	2	2	51
2	0	0	3	6	3	2	1	0	0	3	4	1	6	3	2	2	1	1	38
3	0	0	7	3	4	0	3	8	0	4	0	3	1	6	0	6	5	3	53
4	0	0	1	3	3	2	1	0	2	2	2	2	5	5	1	4	1	-	34
5	0	0	3	5	4	0	3	0	1	5	4	0	3	5	3	7	4	1	48
6	0	0	1	0	4	3	1	0	4	4	1	2	3	9	1	7	2	2	44
7	0	1	1	1	4	2	2	1	1	4	1	2	5	4	2	6	4	1	42
8	0	0	0	0	0	2	2	3	2	1	0	0	3	6	3	9	4	3	38
9	0	0	1	2	1	2	0	2	0	2	0	0	1	6	7	10	6	5	45
10	0	0	1	0	0	0	0	0	0	0	0	0	1	0	2	20	8	11	43
11	0	10	2	1	1	1	0	2	0	0	0	1	1	2	-	-	-	-	21
12	0	2	6	1	0	0	0	1	0	0	0	0	1	5	2	-	-	-	18
13	0	5	3	0	0	1	0	0	0	0	1	0	0	0	2	1	0	6	19
14	0	6	2	1	0	0	1	0	1	0	1	1	0	1	2	2	1	6	25
15	0	2	1	0	2	2	2	0	0	1	2	0	2	7	15	4	3	4	47
16	4	5	2	0	1	0	1	0	0	2	0	0	0	1	1	2	1	-	20
17	0	6	2	0	1	0	0	0	0	1	0	0	0	3	1	2	1	3	20
18	1	5	0	0	0	0	0	0	2	1	1	0	0	1	1	1	1	3	17
19	0	1	3	1	3	2	2	1	4	0	0	1	1	4	2	3	2	7	37
20	0	0	2	4	1	2	2	1	0	0	1	4	2	6	6	7	5	4	48

Continued (Table 1)

Reference Number	WEEKS 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	Survivors	Total Plants
21	0	1	4	4	3	3	0	2	0	0	0	0	0	6	9	6	9	8	55
22	0	0	0	1	2	1	3	2	1	1	0	2	1	2	2	13	3	10	44
23	0	0	0	3	1	2	0	2	1	0	0	1	0	0	6	10	13	5	44
24	0	0	3	0	1	2	1	1	0	0	0	0	1	1	1	6	4	26	47
25	0	0	1	1	0	0	1	2	1	1	1	0	0	1	0	0	3	29	41
26	0	0	0	0	2	0	1	0	0	0	0	0	0	1	0	7	14	16	41
27	0	0	0	0	0	0	0	0	0	0	0	2	0	5	5	6	5	9	32
28	0	0	0	2	1	0	0	0	0	0	0	0	0	0	3	0	6	43	55
29	1	7	2	1	1	0	0	3	0	0	0	0	0	0	2	0	0	7	24
30	0	2	3	0	0	0	0	0	0	0	0	0	0	1	0	0	0	15	21
31	0	1	0	1	0	0	0	0	0	0	0	0	0	1	2	0	0	13	18
32	0	0	0	1	0	1	1	0	0	1	0	0	0	0	0	2	2	14	22
33	0	2	2	3	2	3	1	1	0	1	1	0	0	0	4	6	2	6	34
34	0	0	0	1	0	0	0	2	0	0	0	0	0	2	2	7	3	4	21
35	5	1	1	2	0	0	1	1	0	0	1	0	0	0	2	1	0	1	16
36	2	4	2	0	0	0	0	0	1	1	1	1	0	2	3	1	0	1	19
37	2	1	3	2	2	1	0	0	0	1	1	0	3	2	2	0	0	8	28
38	1	2	0	0	0	2	0	0	0	0	0	0	1	1	0	1	1	9	18
39	1	2	4	2	1	1	0	0	0	1	0	1	0	0	1	1	0	2	17
40	0	1	1	0	1	1	0	0	0	0	0	1	1	2	0	0	0	6	16
41	0	3	2	0	1	0	0	0	0	0	0	0	0	0	0	0	0	13	19
42	3	7	5	1	0	0	0	1	0	1	0	1	1	0	2	1	-	-	23
43	0	8	2	2	0	0	0	1	2	0	1	0	0	1	1	0	0	1	19
44	1	5	3	2	0	0	0	0	0	0	0	0	0	0	1	0	2	5	19
45	1	6	4	0	0	0	0	0	1	0	0	0	0	0	1	0	0	8	21
46	3	5	0	0	1	0	0	0	0	0	0	0	0	0	1	1	2	5	18
47	4	2	2	0	1	0	0	0	0	0	1	0	2	0	2	2	2	8	26
48	0	0	0	0	0	0	0	0	0	1	0	0	1	2	1	5	0	8	18

Continued (Table 1)

Reference Number	WEEKS																	Survivors	Total Plants
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17		
49	2	8	3	4	2	1	-	-	-	-	-	-	-	-	-	-	-	-	20
50	2	9	0	0	3	0	1	0	0	1	0	0	1	-	-	-	-	-	17
51	8	4	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	4	17
52	0	3	1	4	4	1	2	0	3	3	1	0	0	1	0	0	1	2	26
53	1	6	4	1	1	0	0	0	0	0	1	0	1	0	0	0	0	6	21
54	4	5	0	0	2	1	0	1	1	0	1	0	0	0	0	0	1	6	22
55	2	1	2	0	1	1	1	0	0	0	2	2	2	1	3	1	0	1	20
56	0	1	4	0	1	1	2	0	0	1	0	1	0	2	1	3	1	1	19
57	1	1	3	0	0	0	0	1	0	1	0	0	1	6	4	-	-	-	18
58	0	5	2	0	0	0	0	0	0	0	0	0	1	1	1	0	1	6	17
59	1	2	3	1	2	0	0	0	0	0	0	0	0	1	2	0	0	8	20
60	0	3	2	1	2	0	0	1	1	0	0	0	0	0	0	0	1	9	20
61	0	2	2	1	1	0	1	0	1	0	0	0	0	2	3	2	1	3	19
62	0	1	1	1	2	0	1	1	0	2	0	2	1	3	1	0	1	4	21
63	0	1	0	1	0	0	2	2	0	0	3	1	0	4	2	2	1	-	19
64	0	2	2	1	1	1	0	0	0	0	0	0	1	0	1	1	0	9	19
65	0	1	2	0	0	0	0	2	1	1	0	0	0	0	2	0	0	7	16
66	0	2	2	0	0	0	0	0	0	0	00	0	0	0	0	0	0	13	17
67	1	0	0	0	1	0	0	0	0	0	0	1	1	1	4	3	3	5	20
68	0	0	0	0	1	0	0	0	0	0	0	0	0	0	2	1	0	8	12

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