INHERITANCE OF RESISTANCE TO BACTERIAL SPOT (*XANTHOMONAS CAMPESTRIS* PV. *VESICATORIA* (DOIDGE) DYE) IN PEPPERS (*CAPSICUM* SPP.)

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ABSTRACT

From 201 lines of pepper tested for resistance to bacterial spot, 17 P.I. lines which were introduced from India and 3 which were introduced from Central or South America showed resistance.

Seven isolates of Xanthomonas campestris pv. vesicatoria collected from various locations in Hawaii showed no differences in virulence. Pepper line 23-1-7 which has been reported by A. A. Cook to have resistance to race 2 of the pepper strain of X. campestris pv. vesicatoria in Florida was susceptible to all the isolates, which indicates that the strain that occurs in Hawaii is pepper strain race 1 and not race 2.

The inoculation method generally used was infiltration with an inoculum level of 10^8 cells/ml. Infiltration was accomplished by forcing the inoculum into the underside of leaves of 1 month old seedlings with a DeVilbiss air brush connected to a compressor set at 20 psi until an area about 5 mm in diameter appeared water-soaked. Disease was graded 1 week after inoculation on a scale of 1 (low disease) to 4 (high disease). The results obtained from this inoculation method were highly correlated with the field performance of lines with varying degrees of resistance and of individual plants of an F_2 population of the cross Keystone x line 112 (P.I. 308787).

Twelve resistant lines were crossed to the susceptible cultivar Keystone to study the inheritance of resistance. A hypersensitive reaction found in line 79 (P.I. 271322) was controlled by a single dominant gene. An "immune" response found in line 177 (P.I. 163192) was controlled by a single recessive gene. Both lines 79 and 177 also showed quantitatively inherited resistance as well. Lines 47 (P.I. 244670), 127 (P.I. 369994), 112 (P.I. 308787), 110 (P.I. 297495), 4 (P.I. 163192), 34 (P.I. 224451), 43 (P.I. 241670), 119 (P.I. 322719), 137 (P.I. 377688), and 131 (P.I. 369998) had only quantitatively inherited resistance. Lines 79, 177, 47, 127, and 112 were the most effective in transmitting resistance.

Crosses were made between line 79 (hypersensitivity), line 177 (immunity), and line 43 (quantitative factors). When line 79 and 177 were crossed, the hypersensitivity gene was masked by the immunity gene to give an F_2 ratio of 9 hypersensitive : 4 immune : 3 neither. The hypersensitive character segregated normally in the cross of lines 79 and 43, but the segregation of the immunity character was altered in the cross of lines 177 and 43.

The correlation coefficient between pungency and disease in the greenhouse of the F_2 population of the cross of Keystone (low pungency) x line 112 (P.I. 308787) (high

pungency) was not significant, but in the field it was significant (-0.216), suggesting that high pungency may contribute some field resistance to bacterial spot.

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INTRODUCTION

The Capsicum peppers, which are native to tropical America, include both hot types which are important spices and condiments, and sweet types (bell pepper) which are important as either raw or cooked vegetables. In Hawaii, peppers are grown on all the major islands. Most of them are sweet peppers, but hot ones are also grown. Bacterial spot, caused by Xanthomonas campestris pv. vesicatoria (Doidge) Dye, is often a major problem in pepper in Hawaii. Spots develop on the leaves and result in defoliation, thus reducing the effective photosynthetic area and the vigor of the plant. Spots which develop on the fruits reduce their marketability.

Seed treatment, antibiotics, and chemical sprays can control this disease to a certain extent, but resistant cultivars would be more dependable if they were available. Resistance to bacterial spot has been found in cultivars (Horsfall and McDonnell, 1940, Martin, 1948) and in plant introductions (Sowell, 1960, Sowell and Langford, 1963, Sowell and Dempsey, 1977), suggesting the possibility of genetic control of the disease.

Cook and Stall (1963) reported that they found a single dominant gene for resistance in P.I. 163192 and introduced it in a commercial cultivar in Florida (Cook, 1979). However, they later discovered a pathotype of X. campestris

pv. vesicatoria that can infect plants with this gene (Cook and Stall, 1969, 1982). Pepper lines with this gene for resistance were also susceptible in Hawaii (Sekioka, personal communication). Other sources of resistance have been reported but their inheritance has not been studied. Because of the potential breakdown of vertical or major gene resistance it would be desirable to discover horizontal or polygenic resistance to this disease as well.

The purpose of this study was 1) to develop an inoculation technique for testing resistance in the seedling stage in the greenhouse, 2) to investigate possible variability in X. campestris pv. vesicatoria in Hawaii, 3) to study the inheritance of resistance from various sources, and 4) to determine the relationships between the genes for resistance to permit breeding higher levels of resistance to this disease.

LITERATURE REVIEW

1. Early reports, including nomenclature

Bacterial spot of pepper was first reported in 1912 in Texas by Heald and Wolf. A detailed description of the symptoms and isolation of the pathogen was made in 1918 by Sherbakoff. Although Sherbakoff isolated the causal bacterium and proved its pathogenicity by inoculation, he did not name it. Higgins (1922) studied the disease extensively and reported that the causal organism of bacterial spot of pepper was very similar to the bacterial spot pathogen of tomato studied by Gardner and Kendrick (1921a), as well as the "tomato canker" pathogen studied and named by Doidge (1920) (The latter disease is not the tomato canker caused by Corynebacterium michiganense). Gardner and Kendrick (1923) concluded, after comparative studies, that the causal organisms of bacterial spot of pepper and tomato and of "tomato canker" are identical. The name agreed upon was Bacterium vesicatorium Doidge in accordance with priority rules.

The name Bacterium vesicatorium was revised in several steps to the current name, Xanthomonas campestris pv. vesicatoria (Doidge) Dye. Lehmann and Neumann (Dowson, 1943) divided the genus Bacterium into two subgenera, Bacterium proper for peritrichously flagellate bacteria and Pseudomonas for polarly flagellate bacteria. Thus,

Bacterium vesicatorium became Pseudomonas vesicatorium. Bergey (Dowson, 1943) gave a new name Phytomonas to the plant pathogenic group of polar flagellate bacteria (subgenus Pseudomonas of Lehmann and Neumann) making it Phytomonas vesicatorium (Doidge) Bergey. However, Phytomonas was rejected by Dowson (1941, 1943) for the reason that the name Phytomonas was created solely on the basis of pathogenicity, irrespective of other characters. Dowson returned to Pseudomonas, elevated it to the genus level, and split it into Pseudomonas which secretes fluorescence in certain media, and Xanthomonas which forms a characteristic yellow, abundant, slimy growth on solid media. Thus this bacterium became Xanthomonas vesicatoria (Doidge) Dowson. The genus Xanthomonas was again changed some more lately. Dye (1962, 1963) reported that the various Xanthomonas species that have been proposed could not be differentiated by any differential tests, but only by pathogenicity on particular hosts and suggested that the many Xanthomonas species should all be regarded as special forms of one species, but adapted to particular hosts. An amended list of those species and subspecies of Xanthomonas was reported by Dye and Lelliot (1974). Those species that are generally indistinguishable from Xanthomonas campestris or each other except by their host range, were proposed to be pathovars of Xanthomonas campestris (Young et al., 1978). This proposal was approved by the International Society for

Plant Pathology, Committee on Taxonomy of Phytopathogenic Bacteria, effective January 1, 1980 (Dye et al., 1980). Currently the accepted name of the causal organism of bacterial spot of pepper and tomato is Xanthomonas campestris pv. vesicatoria (Doidge) Dye.

2. Symptoms, including factors affecting infection.

The bacterial spot disease most often affects the leaves but stems and fruits are also affected. The symptoms on the leaves are spots which first appear as small, circular, pale green pimples. They are raised on the underside of the leaf and occasionally on both surfaces but usually there is a slight depression or concavity on the upper surface. After a few days the center of the spot dies and collapses. The spot continues to enlarge forming a circular or irregular pale yellow spot with a water-soaked-appearing border which is a little raised. Later the border turns dark brown. The size of the spots depends on the age of the spot, the nutritional condition of the leaf, and the degree of susceptibility of the plant. On the stems, irregular or oblong scars are formed, while, on the fruit, the spots are raised and warty (Higgins, 1921). Sometimes the lesions develop first on the margin of the leaves and spread toward the middle resulting in leaf scorch or blight (Cox et al., 1956).

There are some reports on factors which affect development of bacterial spot disease. Shekhawat and Chakravarti (1976) reported that maximum disease developed when there was high humidity, the temperature ranged between 22-34 C, and the plants were 43 to 50 days old. According to Diab et al. (1982), high relative humidity with free moisture on the leaves for long periods favored infection. However, X. campestris pv. vesicatoria had modest requirements for relative humidity in order to cause disease. When inoculated plants were exposed to high relative humidity (> 85 %) for a few hours during 1 or 2 days, the pathogen caused symptoms. Short periods of unfavorably low relative humidity after inoculation with the pathogen temporarily prevented disease development, but it continued later when high humidity was subsequently provided. Long periods at low relative humidity irreversibly prevented the pathogen from initiating disease, even if high relative humidity was provided.

Plants grown at high nutritional levels had a higher incidence of leaf spot but retained infected leaves for longer periods of time than plants grown at low nutritional levels (Townsley and Crossan, 1961, Nayadu and Walker, 1960, Jenkins and Horn, 1963). Woltz and Jones (1979) reported that the occurrence and severity of bacterial spot of tomato and pepper were consistently increased by increased amounts of magnesium and suggested avoiding luxurious plant

nutrition with Mg and excluding Mg from streptomycin spray applications for improved disease control.

Nayadu and Walker (1960) reported that the disease developed on tomato most rapidly at a continuous night temperature of 24 C while a night temperature of 16 C suppressed the disease regardless of day temperature. They also reported that older leaves are less susceptible to infection and that the disease is suppressed at extremely high levels of N and K.

3. Host range and survival of X. campestris pv. vesicatoria

This organism is pathogenic to tomato, pepper and other solanaceous species such as Solanum nigrum, S. dulcamara (bitter sweet), S. rostratum, Physalis minima, Datura stramonium var. tutula, Lycopersicon pimpinellifolium, Lycium halimifolium (matrimony vine), L. chinense, Hyoscyamus niger (henbane), H. aureus, and Nicotiana rustica (wild tobacco) (Doidge, 1920, Gardner and Kendrick,1921a, 1923). Kishun and Sohi (1979) reported Argemone mexicana L. and Tinospora cordifolia (Wild) Miers as new hosts for X. campestris pv. vesicatoria and suggested that these weeds may play an important role in the survival of the pathogen during seasons when there are no tomato or pepper crops in the field.

X. campestris pv. vesicatoria has been reported to survive in a number of ways. Seed transmission was noted

early by Higgins (1922) and Gardner and Kendrick (1923). The bacterium can remain alive on the seed for 10 years (Bashan et al., 1982). Contaminated seed was considered to be the most important off-season survival mechanism of the pathogen (Cox, 1982). It can survive in infected plant tissue (Krupka and Crossan, 1956, Lewis and Brown, 1961, Person, 1965, and Peterson, 1963). However, it did not survive after two weeks in non-sterile soil at 25 C in a laboratory, suggesting that it is a poor soil inhabitant (Peterson, 1963). Crossan and Morehart (1964) isolated X. campestris pv. vesicatoria from within vascular and cortical tissues of secondary roots, tap roots, lower and upper stems, peduncles, and ovarian tissue, as well as from seed. Epiphytic survival was also found (Leben, 1962).

4. Variability of X. campestris pv. vesicatoria

Burkholder and Li (1941) grouped X. campestris pv. vesicatoria into a tomato strain, which is starch-hydrolyzing, and a pepper strain, which is non-starch-hydrolyzing, although some strains infect both tomato and pepper. Lai and Watson (1973) reported that tomato isolates are virulent only to tomato, but not to pepper, but pepper isolates are virulent to both tomato and pepper. Matew and Patel (1977, 1979) studied physiological characters, protein, bacteriophage sensitivity, and pathogenicity and suggested that the different isolates from

tomato, chili, and Datura are different strains of the bacterium. They speculated that the pepper strain may have been the original one because pepper isolates infect both tomato and Datura, the tomato isolates are less virulent on chili plants, but still infect Datura, and the Datura isolate is the most specialized, infecting neither tomato nor pepper. They also reported high specificity of bacteriophages isolated from infected chili and Datura plants and suggested the possibility of using bacteriophages for differentiation of the pathogenic strains. O'Brien et al. (1967) reported that pepper and tomato isolates of X. campestris pv. vesicatoria can be distinguished serologically. Lovrekovich and Klement (1965) reported that pepper strains can be separated from tomato strains by the use of a gel-diffusion method and specific phages. All pepper isolates were non-starch-hydrolyzing. Tomato isolates were separated into starch-hydrolyzing and non-starch hydrolyzing ones. Additional evidence that pepper isolates are non-starch hydrolyzing is provided by Chun and Alvarez (1982). The selectivity of the SX medium (Schaad and White, 1974), the MS medium (Mulrean and Schroth, 1981), and Chun and Alvarez's SM medium (in press) is based on the ability of xanthomonads to hydrolyze starch for their carbon source. Chun and Alvarez (1982) have clearly shown that neither SX nor their SM medium supports growth of pepper isolates of X. campestris pv. vesicatoria.

Some authors do not agree that the strains differ. Dye et al. (1964) concluded, on the basis of bacteriological determinative tests, pathogenicity, and phage susceptibility, that xanthomonads isolated from tomato and pepper are identical in their pathological behavior. Charudattan et al. (1973) reported no correlation between serology and pathology of X. campestris pv. vesicatoria isolates, and also no correlation between the host origin of the isolates and their ability to hydrolyze starch.

Cook and Stall (1969) were able to distinguish pathological races of X. campestris pv. vesicatoria by their ability to develop hypersensitive or susceptible reactions on tomato and pepper cultivars. They reported finding a tomato strain to which all peppers are hypersensitive, a pepper strain race 1 to which all peppers are susceptible, and a pepper strain race 2 to which certain lines with the major gene for resistance from P.I. 163192 (Cook and Stall, 1963) are hypersensitive.

Recently Cook and Stall (1982) reported that pepper strain race 1 is distributed worldwide including Hawaii, but race 2 has been found only in Florida and Guadeloupe. Dahlbeck and Stall (1979) reported mutations from tomato race 1 to pepper race 2 and pepper race 2 to pepper race 1 in cultures suggesting high mutability of X. campestris pv. vesicatoria. Thayer and Stall (1961) reported variation in susceptibility to streptomycin, colony type, and physiology

among isolates and suggested that variation in the pathogen may be a factor contributing to inconsistencies in field control of bacterial spot with streptomycin.

5. Control

1) Non-genetic

a. Seed treatment

Seed treatment in mercuric chloride 1 to 3000 for 5 minutes (Gardner and Kendrick, 1923), or in a 1 to 3000 corrosive sublimate solution for 5 minutes (Gardner and Kendrick, 1921b) were recommended for disinfection of tomato and pepper seed. These mercuric compounds are now banned for seed treatment. Libman and Webb (1963) reported that seed exposure to 50 ppm hexachlorophene (2,2'-methylene bis(3,4,6-trichlorophenol)) and 100 ppm of its sodium salt for 3 to 4 days completely inhibited the bacteria on infested tomato seeds, but caused some injury to the emerging root tips.

Baker (1947) recommended hot water treatment of pepper, egg plant, or zinnia seed at 51.7 C for 30 minutes to remove Rhizoctonia and other fungi in and on the seed without significantly reducing germination. However, they did not test whether this treatment also kills X. campestris pv. vesicatoria. Several temperatures have been regarded as the thermal death point of the pathogen. Gardner and Kendrick

(1921a) reported 49 to 51 C. Doidge (1920), however, reported 10 minutes at 56 C.

Goode and Sasser (1980) recommended using disease-free seed produced in dry climates, or seed treated either in hot water at 56 C for 30 minutes or with sodium hypochlorite. They also recommended that cultural practices such as isolation, crop rotation, and sanitation practices that eliminate volunteer plants be used as part of an integrated control system. Cox (1982) reported obtaining 98% cleanup of infested tomato seed with a sodium hypochlorite treatment.

b. Spray chemicals and others

Sprays of streptomycin nitrate (Altman and Davis, 1955), Agrimycin, and copper A (Cox, 1957) were effective in controlling the disease. Absorption of streptomycin by the leaves was increased by adding glycerol (Gray, 1956). The effectiveness of streptomycin may be nullified by the occurrence of streptomycin-resistant strains of the pathogen (Thayer and Stall, 1961, Stall and Thayer, 1962).

Mixtures of tribasic copper sulfate and dodine (Cyprex) (Crossan et al., 1963) or captan, tribasic copper sulfate, and pentachloronitrobenzene (Borders, 1962) were also effective. Dougherty (1978) reported that the copper compounds that control the disease also reduce yield. Wiebel et al. (1967) evaluated fertility levels, bactericidal and non-bactericidal chemicals, and growth

retarding compounds in the field, but none of the treatments significantly reduced defoliation. Cox (1982) reported that the combination of seed treatment with sodium hypochlorite and a 90-minute copper-maneb premix, on a regular preventive spray schedule, provides outstanding control, and speculated that if the tomato industry would universally adopt this program, bacterial spot would no longer be a serious threat to tomato production.

2) Genetic

a. Resistance reported

Differences in susceptibility to bacterial spot between pepper cultivars were first reported by Horsfall and McDonnell (1940) and Martin (1948) but levels of resistance were not very high. Later, Sowell (1960) tested 659 P.I. lines in Georgia and reported that 15 were sufficiently resistant to be of value to plant breeders. Sowell and Langford (1963) then reported that of the 15 lines which were reported resistant previously, only P.I. 163184, 163189, 163192, 183441, and 244670 were consistently resistant in replicated trials. P.I. 271322 and 322729, as well as some introductions of Capsicum chinense and C. pendulum were added to the resistant list by Sowell and Dempsey (1977). The resistance in P.I. 163184, 163189, 163192, 183441, 271322, and 322719 was confirmed in Australia (Hibberd et al., 1979). Shekhawat and Chakravarti

(1979) in India reported that 4 out of 89 Indian peppers tested were moderately resistant. Sowell (1980) added one more resistant line, P.I. 369994, when he tested 490 new P.I. lines.

Although several people have reported finding resistance, there are very few breeding programs to introduce the resistance into commercial varieties. Only in Florida has there been such a breeding program (Dahlbeck et al., 1979, Cook, 1978, 1979).

b. Inheritance studies reported

Diseases caused by Xanthomonas spp. are known in many crops, and the inheritance of resistance to them has been studied in crops such as rice (Ezuka et al., 1975, Olufowote et al., 1977, Petpist et al., 1977, Sidhu et al., 1978, Ogawa et al., 1978), cowpea (Singh and Patel, 1977a), and mungbean (Singh and Patel, 1977b).

Horsfall and McDonnell (1940) reported that when resistant cultivars of pepper are crossed with susceptible ones, the progeny is resistant and suggested that resistance is probably dominant. Dempsey (1960), however, reported the resistance of the cultivar, Santanka, to be recessive. Cook and Stall (1963) reported a single dominant gene for resistance from P.I. 163192, while several other lines (P.I. 163184, 163189, 183922, 246331, and 244670) had multiple genes for resistance. Adamson and Sowell (1982) confirmed

the single dominant gene previously reported in P.I. 163192. They also reported a different single dominant gene in P.I. 322719 and 2 or more additive genes in P.I. 163189, at least one of which is linked with the dominant gene of P.I. 163192.

6. Pathogenesis and nature of resistance

1) Pathogenesis

Phytopathogenic bacteria are known to enter the plant through natural openings such as stomata, hydathodes, and nectaries, or wounds (Smith, 1911). Bacterial spot can develop following such wounds as epidermal abrasion, leaf hair breakage, and water congestion in the intercellular spaces (Vakili, 1967).

Bacteria causing leaf spot diseases multiply within the intercelluar spaces of the parenchymatous tissue of the leaf and not within the host cells per se. Fluid in the intercellular spaces contains an abundance of all the nutrients necessary for the bacteria (Klement, 1965, Klement and Goodman, 1967). Despite the presence of all necessary nutrients, phytopathogenic bacteria are able to multiply for only a short period of time in nonhosts and resistant plants. The question which arises is; how do bacteria infect plants and why are plant pathogenic bacteria limited to their own hosts? The question of host specificity remains basically unanswered. Although numerous degradative

enzymes such as pectinase and cellulase, and toxins such as tabtoxin or phaseotoxin are known to take part in the pathological process, it has not been established that they are responsible for pathogenic specificity (Klement and Goodman, 1967, Kelman, 1969). Some fungal toxins such as HV-toxin produced by Helminthosporium victoriae, the cause of a blight disease of oats, and HC-toxin of H. carbonum, the cause of ear rot of corn, are host specific and have been considered as primary determinants of host specificity. Most bacterial toxins are, however, not host specific. Both toxigenic and non-toxigenic mutants of Pseudomonas syringae pv. phaseolicola develop disease on beans upon inoculation but the non-toxigenic strain does not produce systemic chlorosis (Oguchi et al., 1981). Thus, bacterial toxins are not the primary determinant of host specificity. Patil (1974), however, had a different view. He proposed that nonspecificity of toxins of phytopathogenic bacteria in isolation does not preclude their having a role in host specificity. This is supported by the studies of Gantotti (1980) and Oguchi et al. (1981), who reported that an extracellular non-specific toxin produced by P. syringae pv. phaseolicola suppressed the hypersensitive resistance to this pathogen but not others in resistant bean cultivars. Translation inhibitors such as blasticidin S and cycloheximide, however, blocked the hypersensitive reactions of bean plants to not only P. syringae pv. phaseolicola but

to three other phytobacteria, pv. tabaci, pv. tomato, and pv. lachrymans, also. From these results they proposed that the most important function of the toxin is to breach the host defense; host specificity depends on the dynamic relationship between the ability of the host to express resistance and the ability of the pathogen to produce a toxin concentration above the critical level necessary for the suppression of resistance in host tissues.

El-Banoby and Rudolph (1979) reported that extracellular polysacharides (EPS) obtained from culture filtrates of Pseudomonas syringae pv. glycinea, pv. lachrymans, pv. phaseolicola, pv. pisi, and Xanthomonas campestris pv. malvacearum and pv. cerealis induced "persistant water-soaked spots" only in leaves of host species of the EPS-producing bacteria. Treatment of non-host or resistant plants did not result in "persistent water-soaking". The specificity of the extracellular polysaccharides is interesting because it is in contrast to the nonspecificity of the toxins produced by the same plant pathogenic bacteria.

2) Resistance and hypersensitivity (HR)

Kelman and Sequeira (1969) classified resistance in plants into two systems: constitutive or induced.

Constitutive systems may involve:(1) inhibition of bacteria by preformed compounds that are toxic per se or

which are converted rapidly to toxic products when cells are injured; or (2) a combination of adverse physiological factors that currently remain ill-defined. As an example of this system, extracts from healthy corn plants are significantly more inhibitory to the Erwinia species that do not attack corn than to a pathotype of Erwinia chrysanthemi which is infective to corn. A number of other bacterial pathogens of corn were less sensitive to the differential inhibitory fraction than other phytopathogenic and saprophytic bacteria.

Induced resistance systems include : (1) the protective response, and (2) the hypersensitive reaction. Protective reactions have been induced by three different means: (a) by treatment of the host with heat-killed bacterial cells, (b) by prior introduction of living cells of certain avirulent or saprophytic bacteria, and (c) by introduction of incompatible strains of the pathogen that do not cause a hypersensitive response.

A hypersensitive reaction (HR), which is the only system that has been studied in pepper for resistance to X. campestris pv. vesicatoria, is the defense reaction of plants against pathogens that occurs in an incompatible host-parasite relationship. Whenever a pathogen attacks an incompatible plant, the plant reacts by the formation of hypersensitive necrosis, which prevents further spread of

the pathogen. This leads, in turn, to the localization of the disease (Klement and Goodman, 1967).

Hypersensitivity of plants against phytopathogenic bacteria was established in the early 1960's by Klement (1963) and Klement et al. (1964). The hypersensitive reaction is induced only in incompatible host-bacteria combinations such as between a pathogenic bacteria and a nonhost plant, between a normally virulent bacteria and a resistant plant, or between an avirulent bacteria and a normally sensitive host plant. It does not occur when saprophytes interact with plants. This property of pathogens to induce HR has been used in differentiating phytopathogens from saprophytes (Klement and Goodman, 1967).

The most important characterestic that separates HR from the compatible response is rapidity. At the initial stage of infection, X. campestris pv. vesicatoria multiplied at about the same rate in both resistant and susceptible cultivars of pepper. The bacterial population, however, reached a peak earlier and at a lower level in resistant plants than in susceptible plants. Macroscopic HR lesions developed within 24 hr after inoculation on resistant leaves, which is in contrast to the 5 days necessary for lesion development on susceptible leaves injected with the same inoculum concentration. The affected tissue of the resistant plants faded from green through light tan to a chalky white color (Stall and Cook, 1966).

Both physiological and anatomical changes are known to occur in plant tissues undergoing HR. Phytopathogenic bacteria have been known (Klement and Goodman, 1967) to disrupt cell membranes and to consequently affect cell membrane permeability. Membrane disruption and a change in permeability were measured by electrolyte loss from the pepper leaves (Stall and Cook, 1968). When X. campestris pv. vesicatoria cells (10^8 cells/ml) were introduced into leaves of hypersensitively resistant and susceptible peppers, the maximum loss of electrolytes occurred earlier (12 hrs) in hypersensitive plants than in susceptible ones (60 hrs). Stall and Cook (1968), and Chen et al. (1969) reported an inhibition of X. campestris pv. vesicatoria by the extracts obtained from previously inoculated pepper leaves. When inoculum of 10^8 cells/ml was used, inhibition of the pathogen occurred in extracts from hypersensitive leaves after 8-hr incubation, but 84-hr incubation was necessary to obtain extracts having a similar degree of inhibition from susceptible leaves. The degree of inhibition in extracts from both hypersensitive and susceptible tissues corresponded closely with the amounts of electrolytes in diffusates from the two types of tissues. The concomittant release of both factors after invasion by a pathogen suggested that a change in the cell membrane system is necessary for release of the biostatic factor. Suppression of the electrolyte losses was obtained by adding

0.2 M calcium nitrate to the inoculum immediately prior to injection (Cook and Stall, 1970, 1971).

Bacteria, unlike most other plant pathogens, do not make direct contact with the living host protoplast following their introduction into plant tissue. They multiply in intercellular spaces or vascular elements without intracellular invasion (Kelman and Sequeira, 1972). For HR reaction to occur, bacterial contact with the plant cells is necessary (Stall and Cook, 1979). According to Goodman et al. (1976) and Sequeira et al. (1977), incompatible bacterial cells attach readily to the walls of plant mesophyll cells. Then fibrillar and granular material extruded from the host cell walls and bound by the outer wall layer envelop the attached bacteria. At the site of attachment, the host cell wall is eroded, the plasmalemma separates from the cell wall and becomes convoluted and numerous membrane-bound vesicles accumulate in the space between plasmalemma and the cell wall. As a result, the host cell collapses and a hypersensitive reaction develops within 6 to 12 hr after infiltration. In contrast, cells of a virulent pathogen are not attached and remain free to multiply in the intercellular fluid, causing no visible changes in organelle structure during the first 12 hrs after inoculation. Saprophytic bacteria are attached and enveloped, but do not cause visible HR. Prevention of bacterial cell contact with plant cells by continuous

water-soaking of inoculated leaf tissue resulted in suppression of HR (Cook and Stall, 1977).

Cook and Stall (1969) also reported that necrosis similar to the hypersensitive reaction was induced by volatile materials produced in vitro by X. campestris pv. vesicatoria and other plant pathogenic and saprophytic bacteria in both resistant and susceptible pepper leaves. Stall and Cook (1970) reported that ammonia was one of the volatiles produced and was associated with electrolyte leakage from pepper leaves.

MATERIALS AND METHODS

1. Pathogen Material

Pepper leaves infected with bacterial spot were collected from various locations on Oahu and Kauai, Hawaii (Table 1). *X. campestris* pv. *vesicatoria* was isolated from the pepper leaves on either yeast extract-dextrose-calcium carbonate agar (YDC) (Wilson et al., 1967) or tetrazolium chloride medium (TZC) (Kelman, 1954) plates. Pure cultures were obtained by 2 successive streakings of a typical *Xanthomonas* colony onto fresh medium. The bacterial isolates were then stored in sterile distilled water tubes in a cold room as described by Person (1969). Whenever a specimen of the original culture was needed, the storage tubes were shaken and the bacteria were removed with a flamed loop and streaked onto TZC plates. A colony exhibiting normal *Xanthomonas* morphology was restreaked onto YDC plates and used as inoculum.

The seven isolates were tested on 10 different lines of pepper to look for differences in virulence or host-parasite interactions. These hosts were inoculated in the greenhouse by the infiltration method described later. The experiment was repeated once. The repetition was considered as a replication and the results were analyzed as a split plot design.

Table 1. -- Sources of *Xanthomonas campestris* pv. *vesicatoria*.

Isolate	Host	Location
1	Pepper	East West Center Community garden, Oahu
2	Pepper	Manoa Community garden, Oahu
3	Pepper	Poamoho Experiment Station, Oahu
4	Pepper	Nakayama farm in Kahuku, Oahu
5	Pepper	Fukuyama farm in Kahuku, Oahu
6	Pepper	Kauai Branch Station, Kapaa, Kauai
7	Pepper	Kauai Producer's Limited, Kauai

2. Inoculation methods

1) Planting medium and growing conditions

Pepper seeds were planted in a mixture of 2 parts vermiculite and 1 part peat moss supplemented with Osmocote (14-14-14, 1 tablespoon per gallon of vermiculite) in Speedling trays (6 x 12 holes, 2 x 2 inches). Extra seeds were planted; the extra seedlings were used for filling missing holes and the remainder thinned to 1 plant per hole. Plants were grown in a greenhouse where temperatures ranged from a minimum of 20 C to a maximum of 40 C for all inheritance studies.

Some plants were transplanted to the Waimanalo Experiment Station, which is irrigated with an overhead sprinkler system. Fertilization, insecticide spraying, and weeding were done routinely by the farm crew.

2) Viable number of cells/ml and optical density

In order to facilitate adjusting the bacterial concentrations in routine inoculations, the relationship between the viable number of cells/ml and the optical density of bacterial suspensions was examined. Four bacterial suspensions with different concentrations were obtained by diluting a thick suspension of X. campestris pv. vesicatoria consecutively in 1 to 1 ratio with distilled sterile water. Optical densities of the four bacterial suspensions were measured in a spectrophotometer at 470 nm.

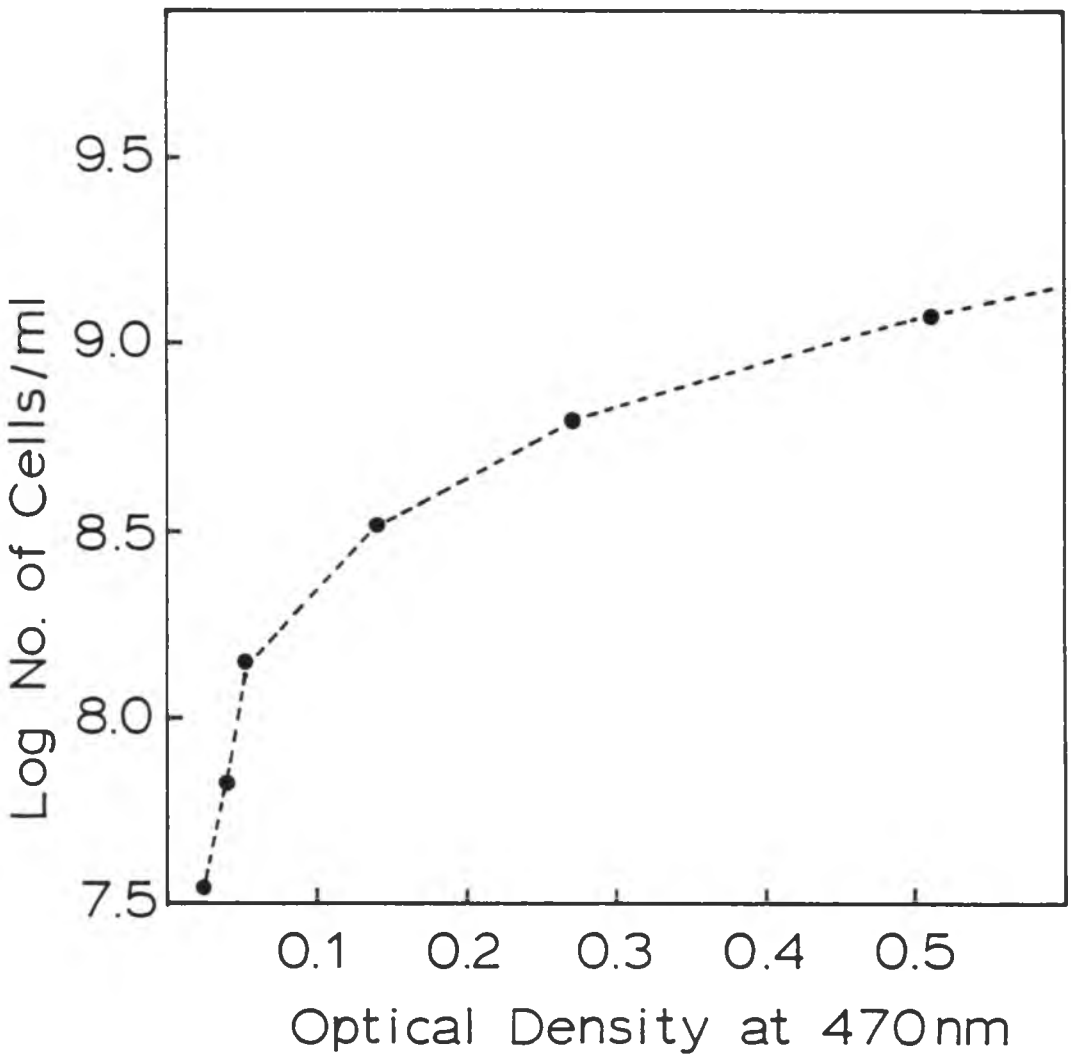


Figure 1. Relationship between the optical density at 470nm and viable number of cells/ml in distilled water.

The number of viable cells/ml in the four bacterial suspensions with the known optical densities were measured by the serial dilution and plate count technique (Brock and Brock, 1978). The tryptone-yeast extract-dextrose medium (tryptone 5 g, dextrose 5 g, yeast extract 3 g, K_2HPO_4 0.7 g, $MgSO_4.7H_2O$ 10 ml of 1 M solution, agar 20 g, per liter of distilled water) was used for plating the dilutions because the medium is clear and supports fast growth of X. campestris pv. vesicatoria. The relationship between the log number of cells/ml and optical density is presented in Figure 1.

3) Preparation of inoculum

Bacterial cells were obtained from a 48 hour old culture of the East-West Center isolate of X. campestris pv. vesicatoria on YDC which contained yeast extract, 10 g, $CaCO_3$ (Malincrodt Ultrafine), 20 g, Dextrose, 20 g, and Agar 20 g per liter of distilled water with a sterile cotton swab and dispersed in distilled water. The optical density of the bacterial suspension was initially adjusted to 0.5 at 470 nm using a spectrophotometer. This point is equivalent to about 10^9 cells per ml as determined by viable count (Figure 1). A further ten-fold dilution of the suspension was made to obtain a bacterial suspension of approximately 10^8 cells/ml. The bacterial concentration of 10^8 cells/ml

has been commonly used in inoculations (Cook and Stall, 1968, 1977, Stall and Cook, 1966, Sowell and Dempsey, 1977).

4) Application of inoculum and disease grading

a. Initial application method for preliminary study

The first preliminary test inoculations were done by spraying a bacterial suspension of approximately 10^8 cells/ml (estimated by McFarland turbidity tubes) mixed with some carborundum on one month old seedlings with a DeVilbiss atomizer connected to a pump with 15 to 20 psi until all the foliage was wet. Carborundum was used because the importance of wounds for infection was emphasized by Vakili (1967). The inoculated plants were then incubated for 2 days in a humidity chamber constructed of wood and black plastic film. Disease readings were taken 10 to 13 days after inoculation using a 1 to 5 scale: 1 = no visible spots, 2 = pin point spots, 3 = small round spots with a diameter around 2 mm or smaller, 4 = spots larger than type 3 and expanding, 5 = many type 4 spots coalesced or showing marginal scorch or blight.

b. Development of a precise method of inoculation

The initial inoculation method used had some drawbacks. The amount of disease developed in two preliminary inoculations differed, so repeatability was poor. It was

time-consuming to spray the entire foliage of a large number of seedlings with the small DeVilbiss air brush.

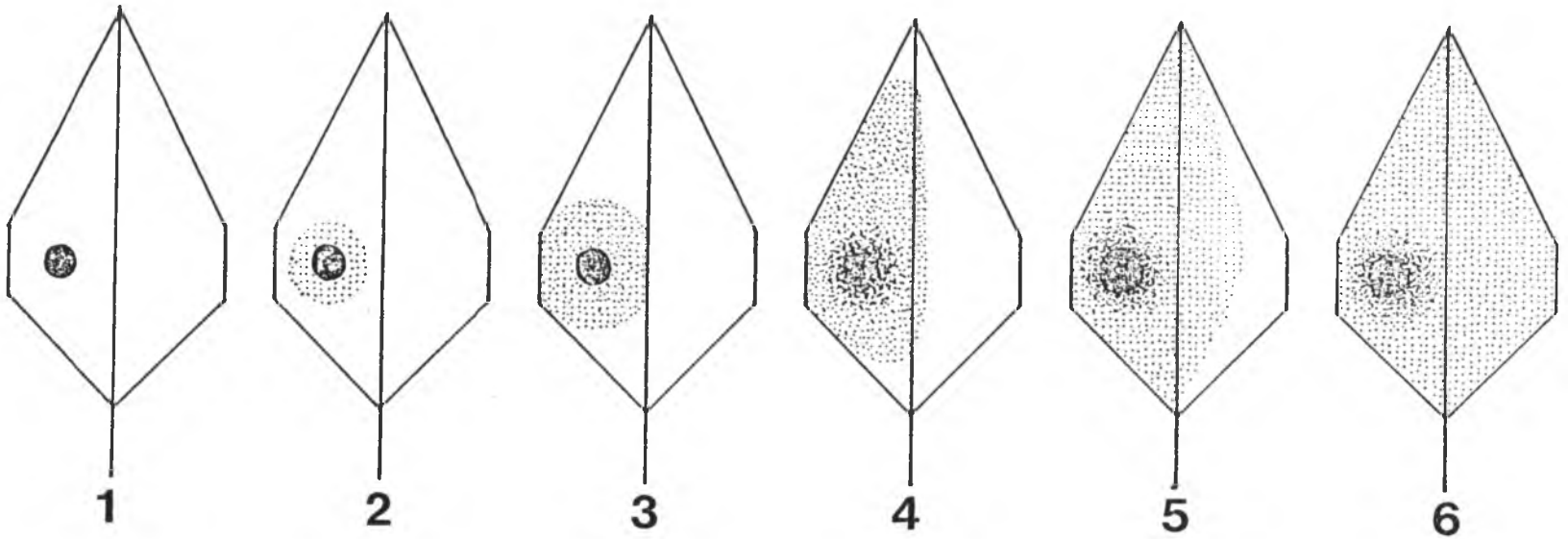
For more efficient and repeatable results, the following two more precise methods were tested. A split plot design with two replications was used for each application method. The main effects were two levels of inoculum: 10^8 cells/ml and 10^9 cells/ml. The subplots were 9 lines of pepper.

Infiltration

Infiltration was done by spraying inoculum closely onto the underside of the leaf with a DeVilbiss air brush connected to a 20 psi compressor until the leaf area appeared water-soaked (Figure 3). Sterile water infiltration was used as a control. An area about 5 mm in diameter was infiltrated on 2 leaves of each one-month-old pepper seedling. The inoculated plants were left on the greenhouse bench without incubation.

Disease was graded 7 and 10 days after inoculation. A grading scale modified from Webster's (1978) 0 to 5 scale for bean was based on the type and size of the resulting lesions. The 1 to 6 grading scale is shown diagramatically in Figure 2 and pictures of representative leaves are in Figures 4-8. Individual leaves were graded by comparing with the diagram. The disease grade of a plant is the mean of the disease grades of the two inoculated leaves.

Figure 2. Disease grading scale for the infiltration method of inoculation.



1. Disease is confined at the infiltration site with no necrotic water-soaked spots around the lesion.

2. There are some necrotic or water-soaked spots but they are limited to the edge of the infiltrated area; the total affected area no more than 10% of the total leaf area.

3. Water-soaked spots are extensive around the infiltration site; the total affected leaf area is about 25% of the total leaf area.

4. Necrotic and water-soaked spots are larger; the affected area is about 50% of the total leaf area.

5. Affected leaf area more than 50%, but not defoliated.

6. Leaves chlorotic, about to defoliate or already defoliated.

Figure 3. Infiltration of bacterial suspension into the leaf mesophyll tissue by DeVilbiss air brush connected to a compressor.

Figure 4. Disease grade 1. A leaf of line 43 (P.I. 241670)

Figure 5. Disease grade 2. A leaf of one of the F2 plants of the cross Keystone x line 79-3 (P.I. 271322)

Figure 6. Disease grade 3. A leaf of one of the F2 plants of the cross Keystone x line 79-3 (P.I. 271322)

Figure 7. Disease grade 4. A leaf of one of the F2 plants of the cross Keystone x line 79-3 (P.I. 271322)

Figure 8. Disease grade 5. A leaf of one of the F2 plants of the cross Keystone x line 79-3 (P.I. 271322)

Figure 9. Hypersensitive lesion on a leaf of an F2 plant of the cross Keystone x line 79-3 (P.I. 271322).

Figure 10. The "Immune" response found in line 177 (P.I. 163192)


3


4


5


6


7


8


9


10

Spray inoculation

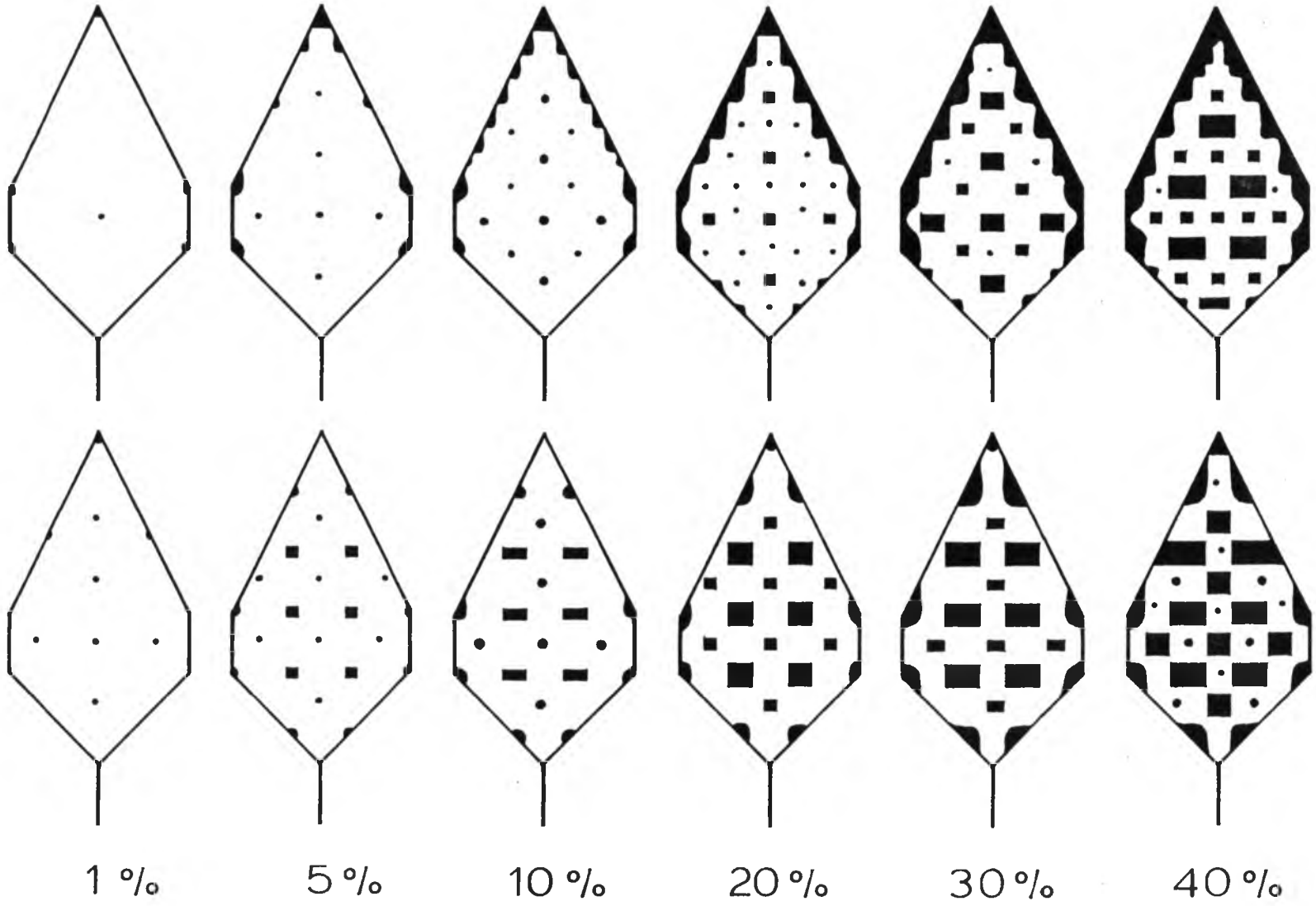
A hand-operated stainless steel spray tank with built-in pressure gauge and a cone-type nozzle was used. The bacterial inoculum was sprayed evenly on both sides of leaves of one month old seedlings. The nozzle pressure was maintained at 30 psi during the inoculation. The inoculated seedlings were incubated in a humidity chamber made of a wooden frame lined with black plastic film for 48 hours and were removed to the greenhouse bench. The diseased area of individual leaves was visually estimated 16 days after inoculation using a precalculated simulated spot diagram (Figure 11). The diseased leaf area of a single plant was estimated by taking the average of individual leaves. Plants sprayed with tap water served as a control.

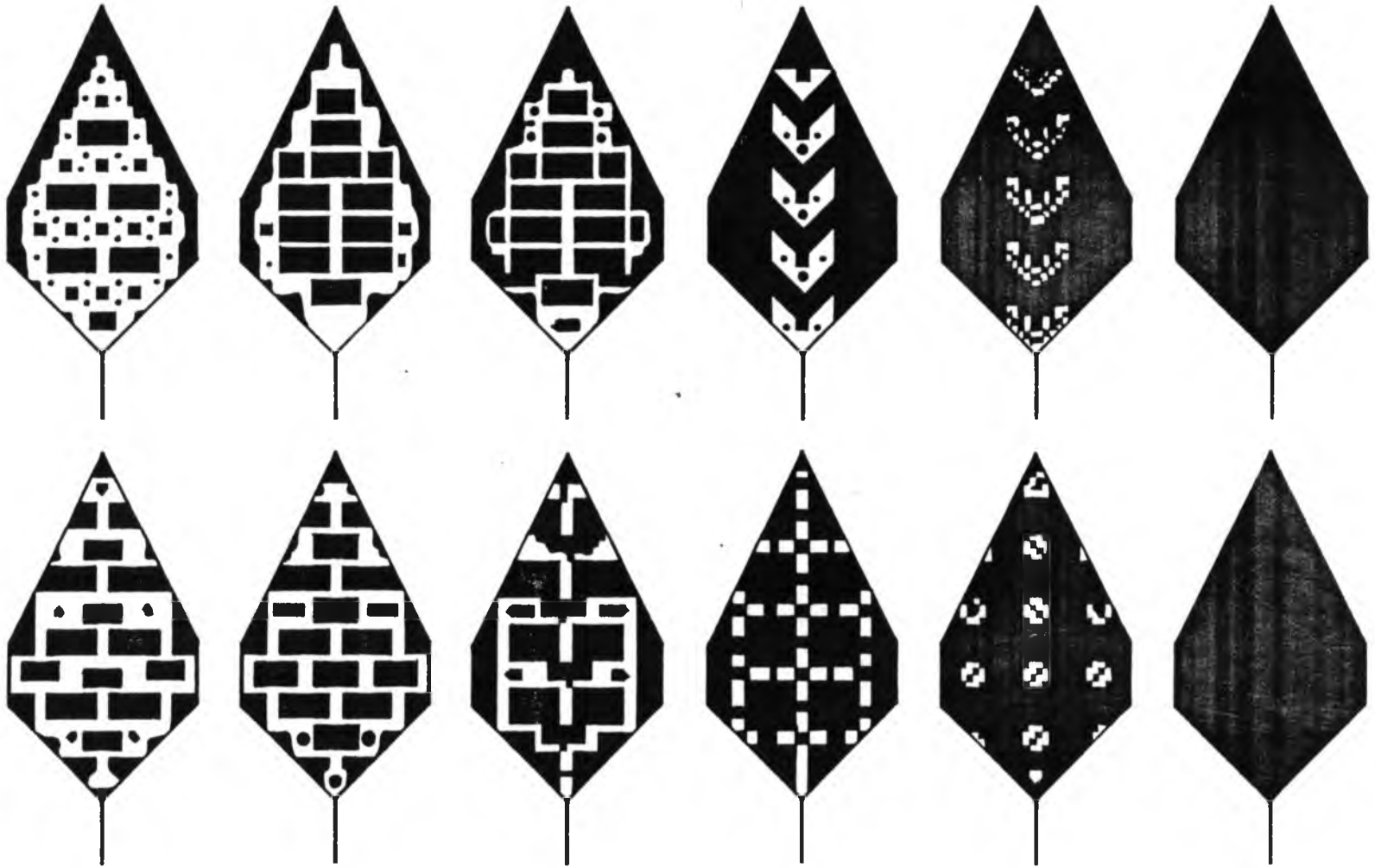
Data taken in percentage were arcsine transformed for analysis.

c. Injection by syringe for testing hypersensitivity of line 79 (P.I. 271322)

Three isolates of bacteria were used in the hypersensitivity test: An EWC isolate of X. campestris pv. vesicatoria, Pseudomonas syringae pv. phaseolicola HB-36 which is a toxin-producer, and pv. phaseolicola G50 Tox^- which is a non-toxin-producing mutant. The Pseudomonas isolates were received from A. M. Alvarez in Plant Pathology. X. campestris pv. vesicatoria was cultured on

Figure 11. Simulated percent diseased leaf area.


1 %
5 %
10 %
20 %
30 %
40 %


50 %
60 %
70 %
80 %
90 %
100 %

YDC plates and the Pseudomonas isolates on King's medium B (KMB) (King et al., 1954). Cell suspensions of the isolates were obtained by dispersing the bacterial mass taken from a 48 hour old culture in distilled water. Cell concentration was adjusted to approximately 10^8 cells/ml by use of McFarland turbidity tubes. Two plants each of Keystone and line 79 (P.I. 271322) were used. Four leaves of about the same age on a plant were chosen, and the bacterial suspensions and distilled water were separately injected into the intercellular space of the mesophyll tissue of the leaves. Observations were made 24 and 48 hours after inoculation.

d. Correlation of greenhouse infiltration results and field performance

Parental lines

Eighteen pepper lines with various degrees of resistance to X. campestris pv. vesicatoria were inoculated in the greenhouse on February 16, 1982, and then transplanted to a Waimanalo field on April 12, 1982. The transplanting was planned for early March but delayed about one month by rainy weather in March (151.38 mm precipitation) and early April. Water and fertilizer were withheld to reduce seedling growth while the plants were being held in the greenhouse. A duplicate uninoculated set was also transplanted to the same field. Spacing was 3 feet

between rows and 1.5 feet between plants in the row. A row of inoculated Keystone plants was planted between every two test rows. A split plot design with two replications was used. The main effects were inoculation versus no inoculation and the subplots were the pepper lines with six plants each.

It took 1 month for the plants to recover from transplanting shock because the seedlings were under stress in the greenhouse during the period before transplanting. Disease development was poor throughout May due to dry weather (Figure 12) although the field was irrigated twice a week by a sprinkler system. Therefore, the plants in the inoculation plots were reinoculated on June 3, 1982. A bacterial suspension of approximately 10^8 cells/ml, prepared by washing 2 plates of a 48 hour-old culture on YDC medium into a gallon spray tank with tap water, was sprayed onto the plants evenly until both sides of all the leaves were wet. Abundant disease then developed due to the reinoculation and frequent rain thereafter. Disease was graded twice, June 22 and July 12, 1982. The disease rating evaluates for the total diseased leaf area per plant. The diseased leaf area is the spotted area on intact leaves plus a value of 100% for defoliated leaves. The diseased leaf area was visually estimated and recorded using a 0 to 10 pretransformed scale (Little and Hill, 1980, p 161): 0 = No spots, 1 = trace to 2.5%, 2 = 2.6 - 10%, 3 = 11 - 21%, 4 =

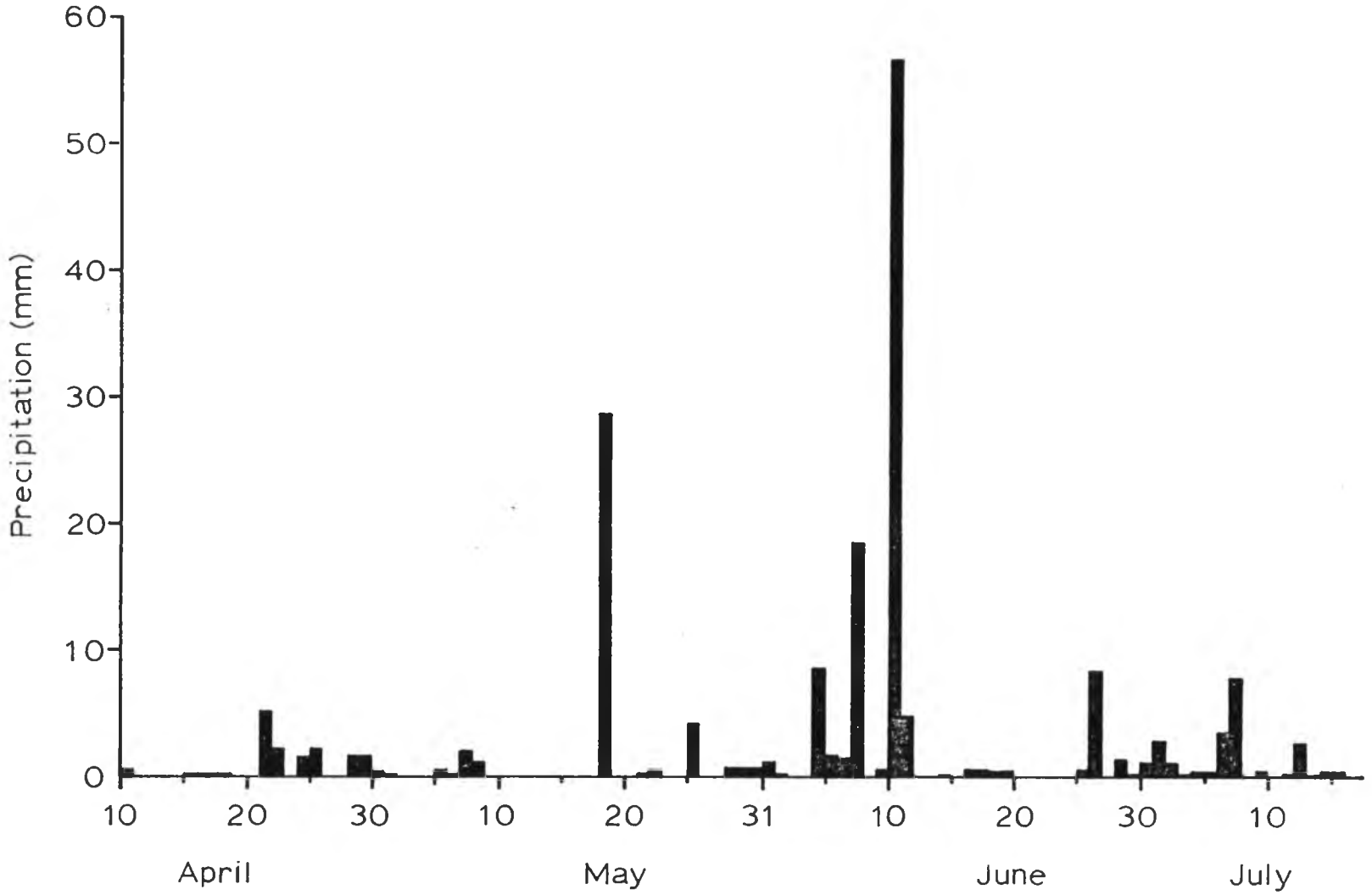


Figure 12. Daily precipitation during the period of field experiment in 1982 at Waimanalo Experiment Station.

22 - 35%, 5 = 36 - 50%, 6 = 51 - 65%, 7 = 66 - 79%, 8 = 80 - 90%, 9 = 91 - 97.5%, and 10 = 97.5 - 100%.

Since results of the two evaluations were not significantly different, the data taken on July 12, 1980 were used for analysis.

Individual F_2 plants

F_2 plants of the cross of Keystone x Line 112 which were planted on January 19, 1982 and inoculated on February 18, 1982 in the greenhouse by the infiltration method, were also transplanted to Waimanalo on April 12, 1982. To facilitate identification of individual plants which had been rated in the greenhouse, the seedlings were planted in the same sequence as found in the Speedling trays in the greenhouse. Spacing was 3 feet between rows and 1.5 feet between plants within the row. These plants were also reinoculated on June 1, 1982, in the same way as the parental lines. Disease was graded twice, on June 22 and July 8, 1982.

3. Plant material

1) Germplasm collection

One hundred sixty eight P.I. lines of Capsicum annuum, C. chinense, C. chacoense, and C. pendulum were obtained from the Southern Regional Plant Introduction Station in

Georgia. The lines were chosen for one of the following three reasons:

(1) previously reported to be resistant (Sowell, 1960, Sowell and Dempsey, 1977)

(2) introduced from Central or South America, the center of diversity of pepper

(3) introduced from India but not yet tested, since many lines previously reported to be resistant had come from India (Sowell, 1960).

Six breeding lines were received from T. T. Sekioka on Kauai. In addition, 24 Korean cultivars were received from the Horticultural Experiment Station in Suweon and the Hung-Nong Seed Company in Korea. The complete list of plant materials tested is in Tables 33 and 34 in the Appendix.

2) Preliminary evaluation for resistance

Eighty one lines of pepper including Keystone and Yolo Wonder as susceptible checks were planted in pots in the greenhouse on October 4, 1979. Twenty-four-day-old seedlings were inoculated. Disease readings were taken 10 days after inoculation. Thirty lines which appeared to have resistance in the greenhouse plus the susceptible Keystone and Yolo Wonder were transplanted to the Waimanalo Experiment Station on December 3, 1979. Spacing was 4 feet between rows and 1.5 feet between plants in a row. An overhead irrigation system was used to increase disease

development. A row of the susceptible cultivar Keystone was planted between every two rows of test plants as a check and source of inoculum. On December 29, 1979, the plants were evaluated for defoliation: 0 = no visible spots or defoliation, 1 = trace to 20% defoliation, 2 = over 20 to 40%, 3 = over 40 to 60%, 4 = over 60 to 80%, and 5 = over 80 to 100% defoliation.

One hundred twenty additional pepper lines were planted in Speedling trays in the greenhouse on January 28, 1980 and inoculated on March 9, 1980 in the same way as the first planting. Disease ratings were taken 13 days after inoculation. Fifty apparently resistant lines, 6 susceptible P.I. lines, and Keystone and Yolo Wonder were transplanted to Waimanalo on March 23, 1980. The Korean lines had arrived too late to be included in the greenhouse inoculation, but were tested by natural infestation in the field. Field ratings were taken on June 26, 1980.

On the basis of their disease reaction in the greenhouse and field, their general vigor, and previous work, the following 16 lines were chosen for further study: 4, 30, 34, 43, 47, 79, 104, 110, 112, 114, 115, 127, 131, 137, 174, and 177.

3) Development of parent lines

One or two representative plants without any disease were chosen from each of the apparently resistant lines and

transplanted to the Magoon tile beds for making cross and self-pollinations.

Crosses were unsuccessful with lines 30, 104, 114, 115, and 174. Line 174 (P.I. 260435) is Capsicum chacoense which is poorly compatible with C. annuum. The other lines were attacked by melon flies or the buds dropped.

The remaining lines (Table 2) were included in the inheritance study. Individual plants were selfed and crossed to the susceptible cultivar, Keystone and to each other. The selfed progeny were inoculated and tested for uniformity of resistance. If resistance was not uniform, another round of selfing and crossing was done so all parents used could be considered homozygous. Fruit size was recorded to confirm the hybrid nature of crosses.

Lines 34, 43, 47, 110, 127, 131, and 137 were uniform in the first test (Table 2). Crosses that had been made at the same time as the selfs were used for the inheritance study.

Lines 79 and 112 were both variable but one generation of selfing identified homozygous plants which were then used as parents.

Lines 4 and 177 were both from P.I. 163192, although from different sources (Tables 33 and 34 in the Apendix) and both were variable. The two lines differed in several characters such as fruit size and pungency, which suggests

Table 2. -- Selection of parental lines

Line	P.I. number	Frequency of progeny at dis. grade[z]				Dry fruits	
		0.5	1.0	2.0	3.0	Length (cm)	Width (cm)
4A[y]	163192		2	8			
4AB			12			5.0	2.0
177A	163192		5	1	4		
177AB		12	4				
177ABC		12				6.1	1.8
34A	224451			12		1.6	1.0
43A	241670		12			9.3	1.2
47A	244670		12			10.3	1.3
79	271322		5	3			
79A			12 (HR)			8.6	1.4
110A	297495		12			6.3	1.2
112A	308787		2	8			
112AB			12			6.9	1.2
119A	322719			12		5.9	4.6
127A	369994		12			6.0	1.2
131A	369998		12			6.0	1.8
137A	377688			12		6.5	1.5
Keystone						9.0	7.5

[z]Scale runs from 1 (low disease) to 5 (high disease), 0.5 is a special kind of reaction in line 177.

[y]A = after 1 round of selfing,
B = after 2 rounds of selfing,
C = after 3 rounds of selfing.

that one of them may have been selected or crossed with other germplasm.

4. Methods for inheritance study

1) Crossing technique and pollination control

Although pepper is usually considered to be a predominantly self-pollinating crop, as much as 36.8% natural cross pollination has been reported (Odland, 1941). Therefore, the following steps were taken to prevent natural cross pollination. Flowers on plants to be used as male parents or to be selfed were covered with a small glassine envelope one day before the flower would open. Flower buds on plants to be used as female parents were emasculated before the anthers split, and pollen from an isolated flower from the male parent immediately applied on the stigma. The pollinated bud was then covered. Flowers which had been crossed or selfed were left covered until fruit set was evident and the fruit started to swell. All fruits were tagged and records were kept on an individual plant basis. Crossing and selfing were done in the greenhouse or in outside beds.

2) Modification of data

Although data were taken on a 1 to 6 scale, disease grades of 4.0 and over were consolidated because the susceptible check, Keystone, ranged from 3 to 6, with no

difference in resistance. Thus, grades over 4 were not considered meaningful and would only bias the data structure in the inheritance study. Observations over 4.0 were all counted as 4.0 in the analyses.

3) Quantitative analysis

a. Generation mean analysis

Crosses between Keystone and 10 resistant lines showed continuous variation in segregating generations. Biometrical analysis was applied to those crosses. Adequacy of an additive-dominance model was tested by the individual scaling test outlined by Mather and Jinks (1971, 1977). The additive-dominance model formulates generation means in terms of three parameters: the mid-parent (m), additive component (a), and dominance component (d). Three relations are tested for adequacy of the additive-dominance model when P_1, P_2, F_1, F_2, B_1, and B_2 results are available:

$$A = 2\bar{B}_1 - \bar{P}_1 - \bar{F}_1 \quad \text{with} \quad V_A = 4V_{\bar{B}1} + V_{\bar{P}1} + V_{\bar{F}1}$$

$$B = 2\bar{B}_2 - \bar{P}_2 - \bar{F}_1 \quad \text{with} \quad V_B = 4V_{\bar{B}2} + V_{\bar{P}2} + V_{\bar{F}1}$$

$$C = 4\bar{F}_2 - 2\bar{F}_1 - \bar{P}_1 - \bar{P}_2 \quad \text{with} \quad V_C = 16V_{\bar{F}2} + 4V_{\bar{F}1} + V_{\bar{P}1} + V_{\bar{P}2}$$

where $V_{\bar{B}1}$, the variance of $\bar{B}_1$, $= V_{B1}/n$, where n is the number of individuals observed in B_1 and used in calculating $\bar{B}_1$.

The expected values of A, B, and C are all 0. A test

whether the above relation A holds good is, therefore, done by finding $A/\sqrt{V_A}$ and looking up its probability in a table of normal deviates (Cumulative Normal Frequency Distribution in Snedecor and Cochran, 1976, p 548). If the absolute value of $A/\sqrt{V_A}$ is greater than 1.96, it is significant at the 0.05 probability level, and if it is greater than 2.58, then it is significant at the 0.01 probability level.

When the scaling test fails and the additive-dominance model is inadequate, the six-parameter model is applied as outlined by Mather and Jinks (1971, 1977) with Gamble's (1951) notation. The six parameters are mean effect (m), additive effects (a), dominance effects (d), additive x additive interaction (aa), additive x dominance interaction (ad), and dominance x dominance interaction (dd). Notations, m, a, d, aa, ad, and dd correspond m , [d], [h] [i], [j], and [l], in Mather and Jinks (1971, and 1977), respectively. Equations for estimating these parameters are found on p 90 of Biometrical Genetics by Mather and Jinks (1971). For example, the dominance effect (d) is estimated with an equation $d = 6\bar{B}_1 + 6\bar{B}_2 - 8\bar{F}_2 - \bar{F}_1 - 3/2(\bar{P}_1) - 3/2(\bar{P}_2)$. The statistical significance of d may be tested by using the following equations for variance and degrees of freedom (M. J. Bassett, personal communication).

$$V_d = 36V_{\bar{B}1} + 36V_{\bar{B}2} + 64V_{\bar{F}2} + V_{\bar{F}1} + 9/4(V_{\bar{P}1}) + 9/4(V_{\bar{P}2})$$

$$Df = Df_{VB1} + Df_{VB2} + Df_{VF2} + Df_{VF1} + Df_{VP1} + Df_{VP2}$$

The t test for the significance of parameter d is

$$t_{Dfd} = d / \sqrt{V_{d.}}$$

b. Estimation of Heritability

The total phenotypic variance (V_P) observed in a population is the sum of three factors: the environmental variance (V_E), the genetic variance (V_G), and a variance due to genetic and environmental interactions (V_{GE}). This can be expressed in a formula:

$$V_P = V_G + V_E + V_{GE}$$

The V_{GE} component is generally assumed to be so small in comparison to V_G and V_E that it can be made equal to zero. V_G can be further partitioned into three subcomponents: additive effects (V_A), dominance effects (V_D), and epistatic effects (V_I). The most important of all these components is V_A.

Heritability is a measure of the degree to which a phenotype is genetically determined and the degree to which it can be changed by selection (Jenkins, 1979). Two estimates are commonly used, broad sense heritability and narrow sense heritability.

Broad sense heritability is defined as:

$$h^2 (B) = \frac{V_G}{V_P} = \frac{V_G}{V_G + V_E} = \frac{V_A + V_D + V_I}{V_A + V_D + V_I + V_E}$$

While narrow sense heritability is defined as:

$$h^2 (N) = \frac{V_A}{V_P} = \frac{V_A}{V_A + V_D + V_I + V_E}$$

If V_I is assumed to be insignificant, V_G, V_D, and V_E can be estimated from experimental data as described by Simmonds (1979).

If $V_{F2} = V_A + V_D + V_E$,

$$V_{BC1} + V_{BC2} = V_A + 2 V_D + 2 V_E,$$

and $V_E = \frac{V_{P1} + V_{P2} + V_{F1}}{3}$ (P_1, P_2, and F_1 are non-segregating populations with only environmental variance)

Then $V_G = V_{F2} - (V_{P1} + V_{P2} + V_{F1})/3$

and $V_A = 2 V_{F2} - (V_{BC1} + V_{BC2})$.

The heritabilities can then be estimated with the following formulas:

$$\text{Broad sense heritability} = \frac{V_{F2} - (V_{P1} + V_{P2} + V_{F1}) / 3}{V_{F2}}$$

$$\text{Narrow sense heritability} = \frac{2 V_{F2} - (V_{BC1} + V_{BC2})}{V_{F2}}$$

c. Estimation of number of segregating genes

The number of segregating genes was estimated for crosses which were segregating quantitatively. Two methods

of calculation were used: Castle (1921) and Wright (Burton, 1951).

The Castle formula:

$$n = \frac{(\bar{P}_2 - \bar{P}_1)^2}{8\,(V_{F2} - V_{F1})}$$

The Wright formula:

$$n = \frac{0.25\,(0.75 - h + h^2)\,D^2}{V_{F2} - V_{F1}}$$

where D is $\bar{P}_1 - \bar{P}_2$ and $h = (\bar{F}_1 - \bar{P}_2) / (\bar{P}_1 - \bar{P}_2)$ This formula for estimating the number of genes depends on the following assumptions:

(a) there is no non-allelic interaction,

(b) the gene differences are of equal effect,

(c) one parent supplies only plus factors and the other only minus factors among those in which they differ,

(d) the degree of dominance of all plus factors is the same for all,

(e) there is no linkage of the genes.

5. Pungency test

Mature fruits were dried 4-5 days at 65-70 C until the fruits were breakable by hand. Whole fruits including seeds were finely ground with a coffee grinder. Pungency was tested following Ting and Barron's (1942) method.

A level teaspoonful of ground powder was put in the 125 ml flask and 10 ml of diethyl ether was added. The flask was stoppered with a cork, shaken well, and allowed to settle for 5 minutes. These ether extracts varied from yellow through various shades of orange. About 5 ml of the extract was decanted into a dry test tube and a 1% solution of Vanadium oxytrichloride in carbon tetrachloride was added until no further color change took place. Extracts of non-pungent peppers changed from orange red to yellow upon addition of the indicator. Extracts of hot peppers turned light to dark green, the intensity of color increasing with increasing pungency. Extremely hot extracts developed a dark blue color. Pungency was visually graded on a 1 to 10 scale by comparing the color in the tubes with standards prepared by mixing solutions of potassium dichromate and copper nitrate in different proportions as described by Van Blarcom and Martin (1947). Pungency grade 1 is sweet and 10 is extremely hot.

RESULTS AND DISCUSSION

1. Preliminary evaluation of germplasm

The results of the preliminary evaluation of germplasm in the plantings of October, 1979 and January, 1980 are given in Tables 33 and 34 in the Appendix. Both tests showed a continuous range of disease resistance. The second planting in general developed a little less disease than the first planting.

Based on these tests, the following lines seemed to be resistant: 2 (PI 163184), 3 (PI 163189), 4 (PI 163192), 11 (PI 183441), 30 (PI 224435), 34 (PI 224451), 43 (PI 241670), 47 (244670), 79 (PI 271322), 96 (PI 288304), 102 (PI 297487), 103 (PI 297488), 110 (PI 297495), 112 (PI 308787), 114 (PI 308789), 115 (PI 308790), 119 (PI 322719), 127 (PI 369994), 131 (PI 369998), 137 (PI 377688), and 177 (PI 163192, received from A. H. Dempsey). All except lines 30 from Cuba, 34 from Guatemala, 43 from Ecuador, and 137 from Malaysia originated from India. It is interesting that many lines from India show resistance, while only a few from Central or South America, the center of genetic diversity of Capsicum peppers, do.

Twelve lines (4, 34, 43, 47, 79, 110, 112, 119, 127, 131, 137, 177) were chosen for further study on the inheritance of resistance (Table 2).

Line 170 (T. T. Sekioka's cross of Shepherd x Jessore) and line 14 (P.I. 201234) also appeared to be resistant, but they were not retained because very few seed were available. Spots developed on most Korean lines by natural infection in the field, although they were not inoculated in the greenhouse as the other lines were. Two lines did not show any spots, but line 200 (Bulam House) is a commercial hybrid, so it is not appropriate for an inheritance study, and line 196 was weak in vigor.

The disease grades of individual plants within some P.I. lines were variable as was also observed by Cook and Stall (1963). P.I. 163192, on which Cook has done much work, was one of the most variable in disease reaction and also morphological features. According to Sowell (personal communication), only one row of tomatoes separates each row of peppers to reduce outcrossing when pepper seeds are increased in the Plant Introduction nursery in Georgia. If there are many active bees in the field, some crossing would not be surprising. Thus, a few rounds of single plant selection were done to insure homogeneity of the material if the accession was not uniform.

2. Variability of Hawaiian isolates of *Xanthomonas campestris* pv. *vesicatoria*

Seven isolates of *X. campestris* pv. *vesicatoria* from various locations in Hawaii were inoculated onto 10 pepper

lines representing a range of resistance to test for possible differences in virulence between isolates (Figure 13). The results were analyzed statistically using a value of 0.5 for the reaction in line 177 and 1.0 for the reaction in line 79 (Table 3). It can be seen that only the differences between lines were significant. Neither the differences between isolates nor the interaction were significant, indicating that all the isolates were the same.

Line 23-1-7 was included in testing the pathogen isolates because this has been reported in Florida to be resistant to race 2 of the pepper strain of X. campestris pv. vesicatoria, but not race 1 (Cook and Stall, 1969a). Clearly, line 23-1-7 is not resistant to the strain in these tests, suggesting that the strain in Hawaii is race 1. These results confirm the report of Cook and Stall (1982) that race 1 is present in Hawaii and elsewhere, but race 2 is found only in Florida and Guadeloupe. The hypersensitivity found in line 79 has also been tested with both race 1 and 2 in Florida by A. A. Cook (personal communication) and found to be effective only against race 1. Apparently, only race 1 exists in Hawaii at this time.

3. Comparison of inoculation techniques

The two inoculation techniques are compared in Figures 14 and 15 and Tables 4 and 5. The results were similar for the two methods, with clear distinctions possible between

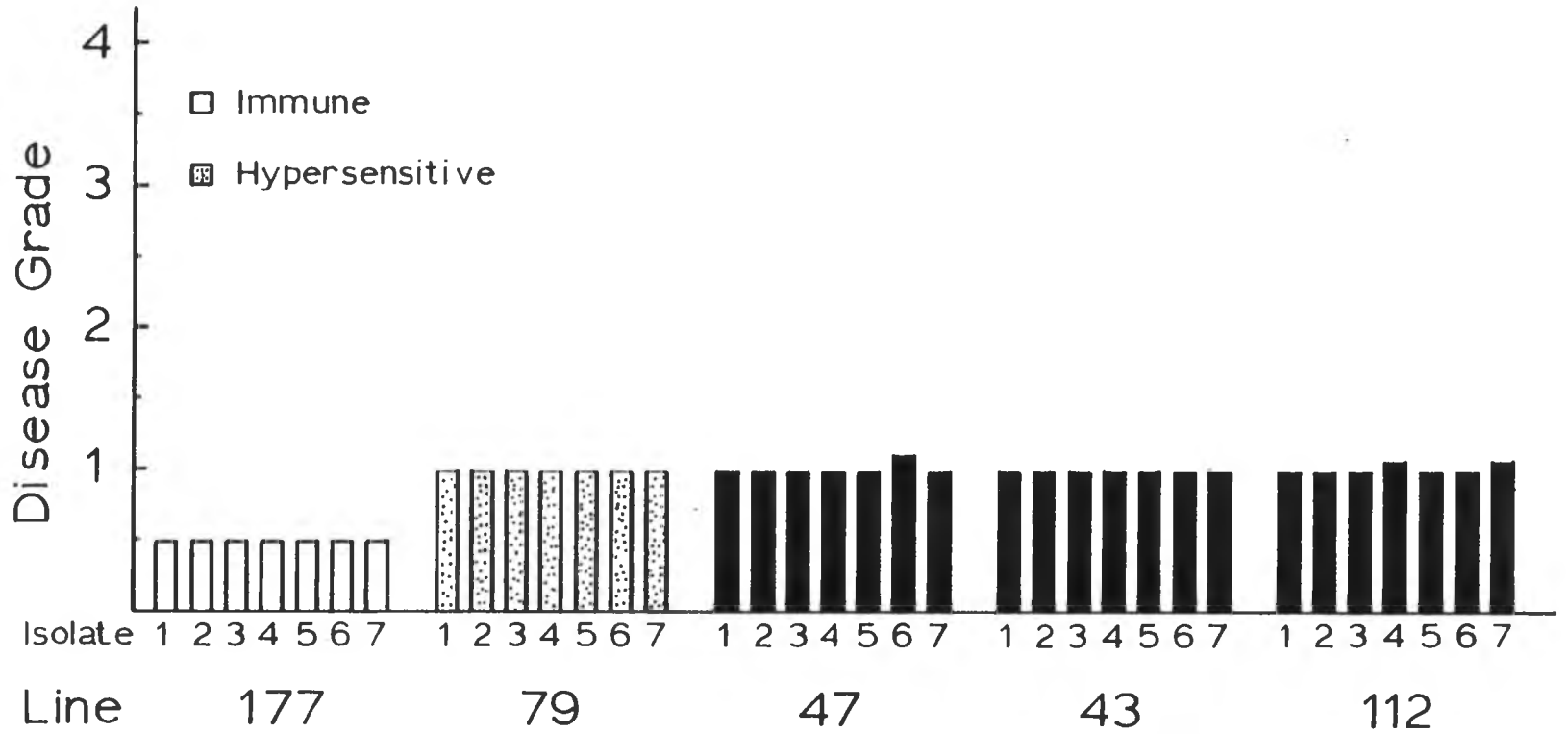


Figure 13. Disease grades of selected lines of pepper 7 days after inoculation with 7 Hawaiian isolates of Xanthomonas campestris pv. vesicatoria.

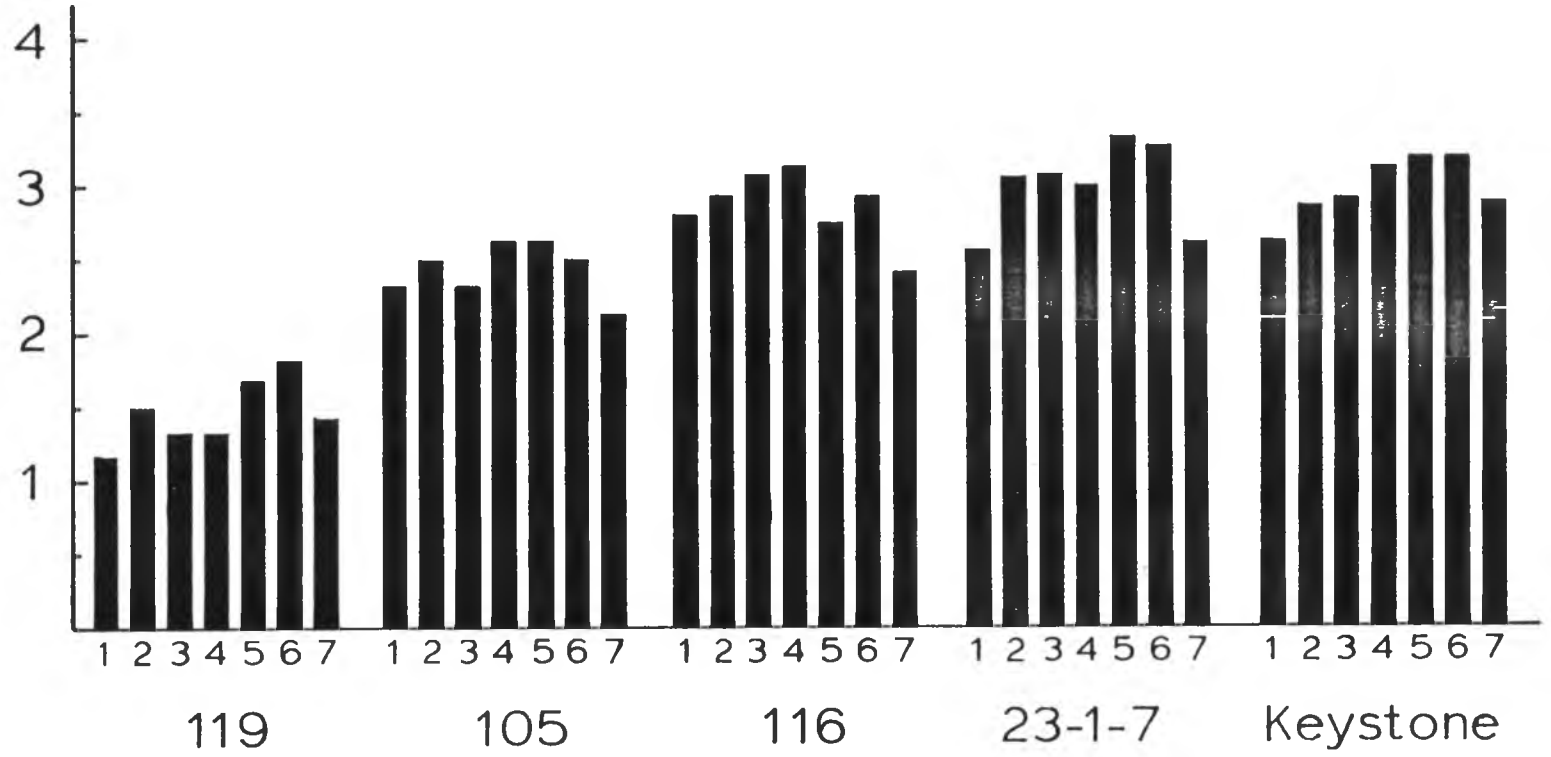


Figure 13 (continued). Disease grades of selected lines of pepper 7 days after inoculation with 7 Hawaiian isolates of Xanthomonas campestris pv. vesicatoria

Table 3. -- Analysis of variance for testing 7 Hawaiian isolates of Xanthomonas campestris pv. vesicatoria on 10 lines of pepper.

Source	df	Sum of Squares	Mean Square	F
Replication	1	0.56	0.56	1.37^{ns}
Isolates	6	4.06	0.68	1.66^{ns}
Error (a)	6	2.44	0.41	
Lines	9	477.48	53.05	353.67^{**}
Isol. x Lines	54	8.68	0.16	1.07^{ns}
Error (b)	63	9.51	0.15	
S. E.	419	32.29	0.08	

** P < 0.01, highly significant

ns P > 0.05, non-significant.

resistant and susceptible lines, although there are some obvious differences.

The difference between the two inoculum levels used was not significant for either method (Table 4 and 5). Only line 124 in the spray inoculation method showed a substantial difference for inoculum levels (Figure 15) but the interaction was not significant. It is, therefore, concluded that a bacterial suspension of 10^8 cells/ml is adequate.

A comparison of the symptoms developed on different lines by the two inoculation methods is examined in detail. When line 124, line 105, Yolo Wonder, and Keystone, which were all susceptible, were inoculated by infiltration, the infiltrated area turned water-soaked within 3 days after inoculation, and secondary water-soaked spots started to appear outside the infiltrated area a day later. The lesions expanded and chlorosis started to develop by five days after inoculation. The tissue then turned thin and light brown with time. The edge of the lesions appeared oil-soaked and shiny. Usually chlorotic yellowing surrounded the lesions. Leaves started to abscise 7 to 10 days after inoculation. These lines were clearly susceptible.

When the same lines were inoculated by spraying, some areas around the leaf margin already appeared water-soaked when the seedlings were removed from the incubation chamber

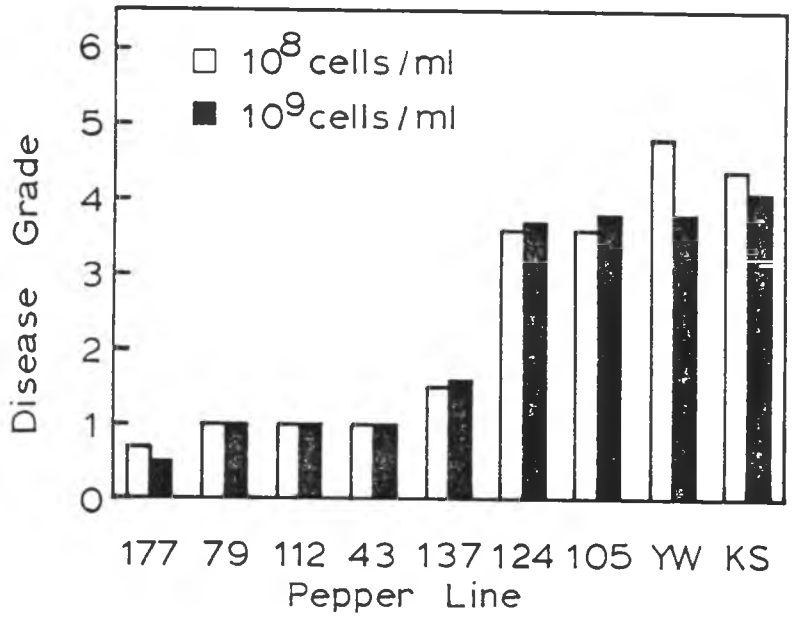


Figure 14. Disease grades of selected lines of pepper 10 days after inoculation with 10^8 and 10^9 cells/ml of X. campestris pv. vesicatoria.

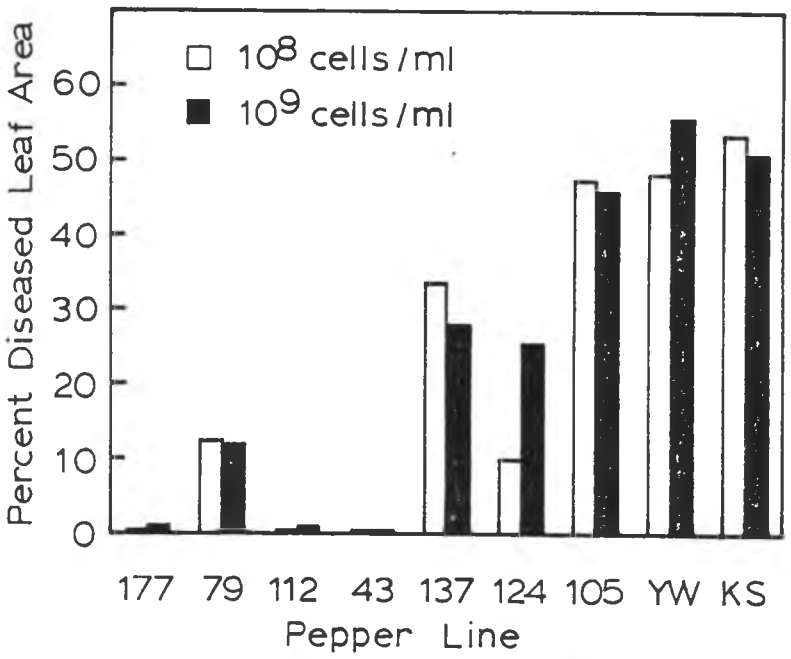


Figure 15. Percent diseased leaf area taken 16 days after spray inoculation with 10^8 and 10^9 cells/ml of X. campestris pv. vesicatoria.

Table 4. -- Analysis of variance for disease grades of pepper seedlings inoculated by infiltration with two levels inoculum, 10^8 cells/ml and 10^9 cells/ml

Source	df	Sum of Squares	Mean Square	F
Inoculum (I)	1	0.34	0.34	0.49^{ns}
Error (a)	2	1.37	0.69	
Lines (L)	8	333.22	41.65	67.18^{**}
Interaction (I x L)	8	2.10	0.26	0.42^{ns}
Error (b)	16	9.94	0.62	
Sampling error	108	37.25	0.34	

** P < 0.01, Highly significant.
ns P > 0.05, non-significant.

Table 5. -- Analysis of variance for diseased leaf area of pepper seedlings inoculated by spraying with two levels of inoculum, 10^8 cells/ml and 10^9 cells/ml

Source	df	Sum of Squares	Mean Square	F
Inoculum (I)	1	95.07	95.07	2.46^{ns}
Error (a)	2	77.17	38.59	
Line (L)	8	41445.42	5180.68	138.71^{**}
Interaction (I x L)	8	634.69	79.34	2.12^{ns}
Error (b)	16	597.52	37.35	
Sampling error	108	3563.54	33.00	

** P < 0.01, highly significant.
ns P > 0.05, non-significant.

after 2 days with the foliage still wet from the spraying. Spots soon developed along the leaf margin and on the underside of the leaves in a mosaic pattern. As the spot development advanced, the lower leaves defoliated. The spots were similar to those which developed after infiltration, ie. water-soaked at the beginning, then chlorotic, then oil-soaked and shiny, and finally becoming light brown and warty.

Lines 43 and 112, which were resistant, reacted similarly to each other. When inoculated by infiltration, the infiltrated area appeared slightly glossier 2 days after inoculation and then started to discolor after 1 more day. The area then became lightly sunken. The lesions were usually confined to the infiltrated area (Figure 4). Secondary spots were rare, but if any did develop, they were pin-point sized, black, and dry. The inoculated leaves stayed attached on the plants until the end of the study period. There was no chlorosis. All plants were classified as disease class 1, which is resistant. Lines 43 and 112 also appeared resistant when they were inoculated by spraying. Only a few small spots developed and there was no defoliation. The maximum infected leaf area was only about 1 % of the total leaf area.

Line 79, another resistant line, reacted very differently from lines 43 and 112. When line 79 was inoculated by infiltration, the infiltrated area turned dark

violet 2 days after inoculation, and then changed to purple and to brown with time (Figure 9), appeared necrotic, and finally became white and crusty. This reaction was later shown to be hypersensitivity (HR). When inoculated by spraying, however, line 79 developed some necrotic spots around the leaf margin and some defoliation occurred. The mean diseased leaf area was about 12 % of the total leaf area which is only partially resistant. No hypersensitivity was observed. This is likely a result of the continuous water-soaking produced under this method. The leaves would become partially water-soaked as a result of the spraying and then would be placed in a completely dark incubation chamber for 48 hours. They would still be wet when they were removed. Cook and Stall (1977) have reported that such continuous water-soaking can prevent a hypersensitive reaction from developing. Stall and Cook (1979) have also suggested that the reason the hypersensitive reaction is prevented is that contact between the bacteria and the plant cell is necessary for the reaction to develop and this contact is prevented by the continuous water-soaking. Light has also been reported to be important for the development of a hypersensitive reaction (Kelman and Sequeira, 1972).

The resistant line 177 also reacted differently to the two inoculation methods. When it was inoculated by infiltration, the infiltrated area on some plants showed hardly any reaction and was recognizable only by a slightly

glossy appearance (Figure 10). Since these plants never develop any lesions after infiltration, I will use the term "immune" for this reaction. For statistical analysis, a disease grade of 0.5 was used. The terms "immune" and "immunity" have also been used in cotton to describe a high level resistance, indicated by no visible symptoms, to X. campestris pv. malvacearum (Knight and Hutchison, 1950). Other plants of line 177 reacted like lines 43 and 112. When line 177 was inoculated by spraying, all the plants reacted similarly to lines 43 and 112.

Lines 137 and 124 appeared intermediate under both inoculation methods, with line 137 showing more resistance when inoculated by infiltration, and line 124 when inoculated by spraying. In the field (Table 34 in the Appendix), line 137 was more resistant than line 124, so for these lines, the infiltration method gave results more similar to the field.

The infiltration method was more uniform, as is shown by the size of the sampling error which was only about half of the error (b) variance for the infiltration method (Table 4), but about the same as the error (b) variance for the spraying method (Table 5).

Thus, the infiltration method of inoculation was chosen because only it detected the hypersensitive reaction (HR) in line 79 and the immune reaction in line 177, it generally gave better correlation with the field results, it was more

uniform, it did not require incubation, and it was easier to evaluate 2 leaves per plant instead of all the leaves.

Inoculation by infiltration was first suggested by Schuster (1955) and has been used in beans (Walker and Patel, 1964, Coyne and Schuster, 1974, Webster, 1978, and Webster, 1980), in cucumber (Chand and Walker, 1964a, 1964b), and in tobacco (Stokes, 1960).

4. Confirmation of hypersensitive reaction found in line 79

The reaction in line 79 that appeared to be hypersensitivity was tested by syringe inoculation. Tissue collapse was observed beginning within 24 hours after injection of a bacterial suspension of 10^8 cells/ml and was complete after 48 hours (Table 6). Tested the same way, Keystone did not show any response within 24 hours and developed only water-soaked lesions 48 hours after inoculation. The reaction observed on line 79 was identical to that produced by Pseudomonas syringae pv. phaseolicola on both pepper lines. This kind of reaction between a phytopathogenic bacterium and a non-host is defined as hypersensitivity (Klement et al., 1964). A hypersensitive reaction in pepper to P. syringae pv. phaseolicola was previously reported by Crosthwaite et al. (1979).

Table 6. -- Responses of line 79 (P.I. 271322) and Keystone to injection with two strains of Pseudomonas syringae pv. phaseolicola and one strain of X. campestris pv. vesicatoria[z]

Line	Time	P. syringae pv. phaseolicola[y]	X. campestris pv. vesicatoria	Distilled water
79	24 h	+	+	-
	48 h	++	++	-
Keystone	24 h	+	-	-
	48h	++	WS	-

[z]- no response
+ minor tissue collapse
++ complete tissue collapse
WS water-soaked lesion.

[y]Two strains were used.

5. Correlation between disease grades in the greenhouse and performance in the field

1) Parental lines

The comparisons of the disease ratings for the parental lines in the greenhouse after inoculation and in the field with and without inoculation are given in Table 7. The analysis of the results in the field only are given in Table 8. This analysis shows that there was no difference between inoculation and no inoculation, the differences between lines were highly significant, and there was also a highly significant interaction between lines and inoculation.

In Table 7 it can be seen that all the lines that were resistant in the greenhouse were also resistant in the field, whether inoculated or not. Lines 177 (immune), 79 (hypersensitive), 127, and 131 developed no disease in the field, even when inoculated. Lines 43, 110, 112, 47, 4, and 137 developed a small amount of disease when inoculated, but not without inoculation. Lines 119, 102, 34, and 105 showed some disease even when not inoculated, but seem to have some resistance. Line 116 and Yolo Wonder were highly diseased in the greenhouse and in the field when inoculated, but not so when not inoculated. Keystone and line 23-1-7 were highly diseased even when not inoculated. The difference between inoculated and not inoculated for line 116 and Yolo Wonder seems to be the main source of the significant interaction effect in the analysis of variance.

Table 7. -- A comparison of bacterial spot disease development on pepper lines in the greenhouse after inoculation by infiltration and in the field with and without inoculation.

Line	P.I. number	Greenhouse[z]	Diseased leaf area index in the field	
			Inoculated	Natural
177	163192	0.5a[y] (Immune)	0.0a	0.0a
79	271322	1.0b (H. R.)	0.0a	0.0a
43	241670	1.0b	0.1a	0.0a
110	297495	1.0b	0.1a	0.0a
112	308787	1.0b	0.2a	0.0a
127	369994	1.1bc	0.0a	0.0a
47	244670	1.2bc	0.1a	0.0a
4	163192	1.5c	0.1a	0.0a
119	322719	1.9d	1.1b	0.4b
102	297487	2.0d	0.9b	0.1ab
131	369998	2.1d	0.0a	0.0a
137	377688	2.2d	0.3a	0.0a
34	224451	2.6e	1.0b	0.1ab
105	297490	4.1f	2.6c	0.9c
Yolo Wonder		4.3fg	5.7ef	1.9d
Keystone		4.7gh	4.9de	4.7f
23-1-7		5.0h	6.6f	3.5e
116	308791	5.0h	4.6d	1.4cd

[z]Disease grades taken 7 days after infiltration. 1 = lesion confined at the infiltrated area, 2 = lesions with secondary spots around and total affected area no more than 10% of the total leaf area, 3 = area of spots and chlorosis about 25% of total leaf area, 4 = area of spots and chlorosis about 50% of total leaf area, 5 = area of spots and chlorosis more than 50% or defoliated.

[y]Mean separation within columns by Baysian L. S. D., 5% level.

Table 8. -- Analysis of variance for bacterial spot disease ratings of pepper lines in the field.

Source	df	Sum of squares	Mean Squares	F
Block	1	0.02	0.02	0.18^{ns}
Inoculation	1	7.33	7.33	52.36^{ns}
Error (a)	1	0.14	0.14	
Lines	17	124.58	7.33	122.16^{**}
Inoc. x Lines	17	8.31	0.49	8.17^{**}
Error (b)	34	2.03	0.06	
S. E.	332	6.41	0.02	

** P < 0.01, highly significant.

ns P > 0.05, non-significant.

The correlation between the results for individual lines in the greenhouse, in the field with inoculation, and in the field without inoculation was generally very good, except for the two susceptible lines which developed significantly less disease under natural infection. Therefore, field performance can generally be accurately predicted by the results of inoculation by infiltration in the greenhouse.

The diseased leaf area index used for field evaluation is an improvement over the methods used by Sowell (1960). Sowell used the percentage of infected plants, a defoliation index, and a scale based on the average number of spots per leaf. All these criteria have disadvantages. The percentage of infected plants does not quantify the severity of the disease on infected plants. The defoliation index does not quantify spots on the leaves still attached on the plants. A scale based on the average number of spots per leaf may result in inaccurate distinctions between plants with a large number of small-sized spots which indicate some resistance, and plants with a small number of large spots like marginal scorch or blight, which indicate high susceptibility. The diseased leaf area index I used considers the number and size of the spots on intact leaves as well as defoliation, and was satisfactory in ease of measurement and in the precision of the data taken.

2) Individual F_2 plants

The correlation coefficients between disease grades for the same plant on 3 dates in the greenhouse and on 2 dates in the field for F_2 plants of the cross between Keystone and line 112 are given in Table 9.

The highest correlation between a rating in the greenhouse and one in the field (0.44) was between the rating taken 7 days after inoculation (February, 25) in the greenhouse and the field rating on July 8, although all the other correlations were very similar (0.38 to 0.43). Thus, disease ratings taken 7 days after inoculation were generally used. These correlations are low because there was a wide range in the field ratings for a given greenhouse class (Table 10, Figure 16). Many plants were rated lower in the field than in the greenhouse while only a few were rated higher, which indicates more susceptible plants appeared disease-free in the field. The mean field disease index of plants in each greenhouse grade increased linearly (Figure 16), but the standard deviation also increased, which indicates that plants that were resistant in the greenhouse performed more predictably in the field than plants that were more susceptible. It appears a large number of "escapes" by susceptible plants in the field produced the low correlation figures.

Table 9. -- Correlation between disease grades taken on 3 dates in the greenhouse and diseased leaf area indexes taken on 2 dates in the field of F2 plants of a cross between Keystone and line 112 (P.I. 308787)

		Greenhouse			Field	
		2/25	2/28	3/5	6/22	7/8
Greenhouse	2/25	1.00	0.86**	0.78**	0.43**	0.44**
	2/28		1.00	0.86**	0.39**	0.38**
	3/5			1.00	0.41**	0.39**
Field	6/22				1.00	0.81**
	7/8					1.00

** significant at 0.01 probability level.

Table 10. -- Frequency at diseased leaf area index in the field on July 8, 1982 of F2 plants from each greenhouse disease grade on February 28, 1982

Grades in the green-house	No. of trans-plants	Frequency at diseased leaf area index in the field							Mean ± S.D.
		0	1	2	3	4	5	6	
1.0	27	16	10	1					0.44 ± 0.58
1.5	65	27	27	8	3				0.80 ± 0.83
2.0	88	20	46	10	7	1	2		1.17 ± 1.06
2.5	55	9	24	10	5	7			1.58 ± 1.24
3.0	43	7	17	7	6	4	2		1.74 ± 1.40
3.5	37	4	14	9	4	5		1	1.89 ± 1.39
4.0	19	1	5	5	2	5	1		2.42 ± 1.43
4.5	9		2	3		1	2	1	3.11 ± 1.90

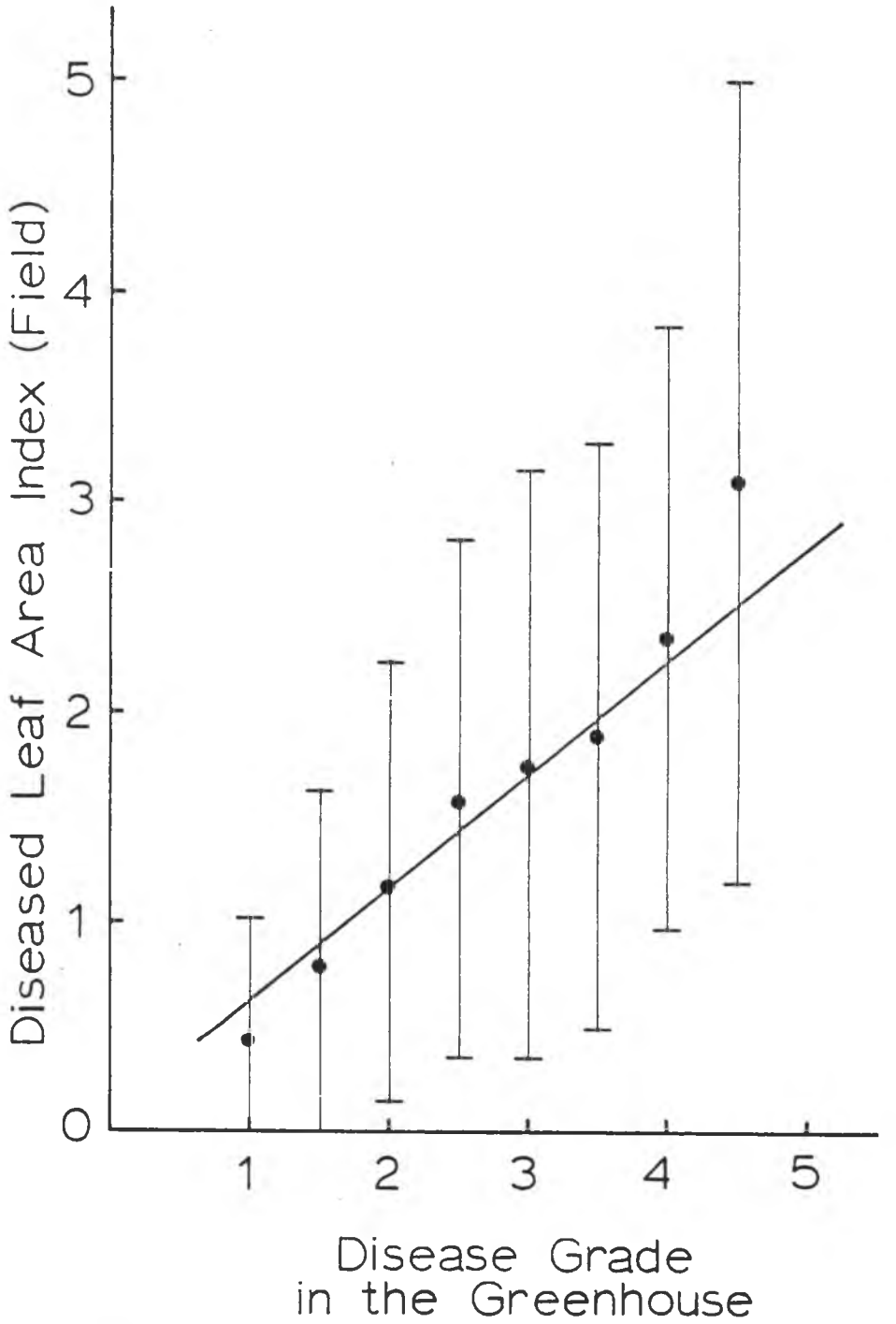


Figure 16. Mean and standard deviation of diseased leaf area index in the field of plants previously graded in the greenhouse.

It can thus be concluded that inoculation by infiltration in the greenhouse is reliable in identifying resistance that will be effective in the field.

6. Inheritance of resistance to bacterial spot

1) Inheritance of hypersensitivity in line 79 (P.I. 271322)

Plant No. 3 of line 79 barely survived when transplanted from the field to the crossing bed and died before any selfed seed could be obtained, so it was not possible to test it for homozygosity like the other parents (Table 2). But pollen taken from this plant and used to pollinate flowers on Keystone produced a good set and much seed. When 12 F_1 plants were inoculated, 6 plants showed a hypersensitive reaction, while 6 did not. F_2 seeds were saved from both hypersensitive and non-hypersensitive plants. One backcross was made between a hypersensitive F_1 plant and Keystone. F_3 seeds were saved from 3 hypersensitive F_2 plants and 2 non-hypersensitive F_2 plants with different disease degrees. The results of testing these populations for their bacterial spot reactions are shown in Table 11.

The F_1 plants in this second test included 25 plants which were hypersensitive and 26 which were non-hypersensitive with a range of disease reactions. This suggests that the original 79-3 plant was heterozygous for

Table 11. -- Bacterial spot reactions in parents and progeny of a cross of Keystone x plant 79-3 of P.I. 271322

Population	Hyper-sensitive	Non-hypersensitive								
		Frequency at disease grade of							Total plants	Mean
		1.0	1.5	2.0	2.5	3.0	3.5	4.0		
P_1 (Keystone)						12	4	49	65	3.78
F_1 (P_1 x 79-3)	25	5		7	8	5	1		26	2.21
F_2 from HR[z] F_1	544	6	5	103	21	18	8	13	174	2.33
F_2 from NH[y] F_1		17	13	109	23	28	5	9	204	2.20
		20	8	94	35	32	7	9	205	2.26
BC (P_1 x HR F_1)	58		1	2	9	11	7	54	84	3.59
F_3 from HR F_2	102		1	4	5	13	7	10	40	3.14
	102	1	10	14	5	7	4		41	2.23
	22		3	2	2			1	8	2.19
Pooled F_3	226	1	14	20	13	20	11	10	89	2.62
F_3 from NH F_2 G1[x]		36	34	42	20	8	2	1	143	1.79
F_3 from NH F_2 G3[x]		2		15	25	58	18	26	144	3.02

[z]HR = hypersensitive
[y]NH = non-hypersensitive.
[x]G1, G2 = disease grade 1, 3.

hypersensitivity and also carried other genes for resistance. The F_2 populations derived from hypersensitive F_1 plants segregated into 544 hypersensitive to 174 non-hypersensitive plants, with a range of disease grades in the non-hypersensitive plants. The F_2 population derived from non-hypersensitive F_1 plants, however, had only non-hypersensitive plants with a continuous range of disease grades.

The three F_3 populations obtained from hypersensitive F_2 plants all segregated into hypersensitive and non-hypersensitive plants. No non-segregating homozygous lines were found. The two F_3 populations derived from non-hypersensitive F_2 plants had no hypersensitive plants but both showed a range of distribution in disease grades. The population from an F_2 plant with a disease grade of 1.0 had a more resistant distribution than the F_3 population from the F_2 plant with a disease grade of 3.0. This indicates that different F_2 plants had different genes for resistance.

The segregation for hypersensitivity vs. non-hypersensitivity was tested for a fit to a 1 dominant gene ratio (Table 12). The F_2 population derived from hypersensitive F_1 plants fits a 3 to 1 ratio very well, which suggests that hypersensitivity is controlled by a single dominant gene. The F_3 populations from

Table 12. -- Segregation for hypersensitivity to Xanthomonas campestris pv. vesicatoria in progenies of a cross between Keystone and plant No. 3 of line 79 (P.I. 271322)

Population	Observed No.		Expected	X^2	Probability range for X^2
	HR[z]	NH	ratio		
F_1	25	26			
F_2 from HR F1	544	174	3:1	0.225	0.5-0.75
F_2 from NH F1	0	204	0:1		
	0	205	0:1		
BC_1 (Keystone x HR F1)	58	84	1:1	4.760	0.025-0.05
F_3 from HR F2	102	40	3:1	0.760	0.25-0.5
	102	41	3:1	1.028	0.25-0.5
	22	8	3:1	0.044	0.75-0.9
Pooled	226	89	3:1	1.801	0.1-0.25
F_3 from NH F2	0	148	0:1		
	0	106	0:1		
	0	254	0:1		

[z]HR Hypersensitive NH Non-hypersensitive.

hypersensitive F_2 plants also fit a 3:1 ratio. Only the backcross to Keystone differed significantly from the ratio expected with a single dominant gene.

Since plant 79-3 died before selfed seed was obtained, more seeds of line 79 were grown and another hypersensitive plant, 79-4, was found. The progeny of plant 79-4 were all hypersensitive (Table 2), so it was presumably homozygous for the hypersensitivity gene. Crosses were made between Keystone and line 79-4, and the F_1 was backcrossed to both parents and selfed to produce F_2 seed. The results of testing these progenies are given in Table 13.

All F_1 plants were hypersensitive. There were no non-hypersensitive plants as in the cross with plant 79-3. The F_2 segregated into 556 hypersensitive and 191 non-hypersensitive plants which showed a complete range of disease reactions. The backcross to plant 79-4 was all hypersensitive, while the backcross to Keystone segregated into 167 hypersensitive to 120 non-hypersensitive plants with a complete range of disease grades.

These data were tested for their fit to the ratios expected with 1 dominant gene (Table 14). All segregations agree with the hypothesis except the backcross to Keystone, which differed significantly from the expected 1:1 ratio. The previous backcross had also deviated significantly (Table 12), but in the opposite direction. If the two backcrosses are combined, the total becomes 225

Table 13. -- Bacterial spot reactions in parents and progeny of the cross between Keystone and plant 79-4 (P. I. 271322)

Population	Hyper-sensitive	Non-hypersensitive								
		Frequency at disease grade of							Total plants	Mean
		1.0	1.5	2.0	2.5	3.0	3.5	4.0		
Keystone (P_1)						27	8	1	36	3.1
Plant 79-4 (P_2)	60									
F_1	96									
BC_1 (F_1 x P_1)	167	5	13	52	29	14	4	3	120	2.2
BC_2 (F_1 x P_2)	491									
F_2	556	61	50	42	21	9	2	6	191	1.7

Table 14. -- Segregation for hypersensitivity in crosses between Keystone and plant 79-4 (P. I. 271322)

Population	Observed No.		Expected ratio	X^2	Probability range for X^2
	HR[z]	NH			
Keystone (P_1)	0	36			
Plant 79-4 (P_2)	60	0			
F_1 (P_1 x P_2)	96	0			
BC_1 (F_1 x P_1)	167	120	1:1	7.697	0.01-0.005
BC_2 (F_1 x P_2)	491	0	1:0		
F_2	556	191	3:1	0.129	0.5-0.75

[z]HR Hypersensitive NH Non-hypersensitive.

hypersensitive and 204 non-hypersensitive plants, which fits the expected 1:1 ratio (X^2 = 1.0279 and probability range = 0.25-0.5).

It is concluded that the hypersensitivity observed in P.I. 271322 is controlled by a single dominant gene. Although Sowell and Dempsey (1977) and Hibberd et al. (1979) have previously reported resistance in P.I. 271322, they did not report it to be hypersensitivity, nor did they study the inheritance.

In addition to the dominant gene controlling hypersensitivity, there are other quantitative genes as well.

2) Inheritance of the Immune response observed in line 177 (P.I. 163192)

The type of response observed in line 177 has been designated "immune". The reactions of a cross between this line and Keystone are given in Table 15.

The F_1 plants were neither immune like line 177 nor susceptible like Keystone but developed lesions which were graded from 1.0 to 1.5, the same as in other resistant progeny. When the F_1 was backcrossed to Keystone, no plants were immune but the disease grades ranged all the way from 1.0 to 4.0. The backcross to line 177, however, segregated into 144 immune plants and 144 plants that were not immune,

Table 15. -- Reactions to bacterial spot in parents and progeny of a cross between line 177 (P.I. 163192) and Keystone

Population	Immune plants	Non-immune plants									
		Distribution in disease grades							Total	Mean	Variance
		1.0	1.5	2.0	2.5	3.0	3.5	4.0			
Keystone (P_1)						1	3	32	36	3.9	0.045
Line 177 (P_2)	36								0		
F_1 (P_1 x P_2)		15	9						24	1.2	0.061
BC_1 (F_1 x P_1)		16	32	41	39	43	39	75	285	2.8	0.930
BC_2 (F_1 x P_2)	144	119	15	10					144	1.1	0.081
F_2	120	178	34	20	19	15	14	27	307	1.7	1.033

but resistant with a disease range of 1.0 to 2.0. The F_2 segregated into 120 immune plants and 307 which were not immune, but showed the full range of disease grades from 1.0 to 4.0.

The data for immunity versus non-immunity were tested for their fit to a single recessive gene ratio (Table 16). The fit of the observed numbers to the expected 1:3 ratio in the F_2, 1:1 ratio in the backcross to line 177, and 0:1 ratio in the backcross to Keystone, was good in all cases. This shows that the immunity character found in line 177 is controlled by a single recessive gene.

The range of disease grades of the non-immune plants indicates that line 177 must also carry other genes for resistance to bacterial spot. If we consider the non-immune plants only and assume a disease grade for the parental line of 1.0, a minimum number of genes can be estimated by the Castle and Wright (1921) and Wright (Burton, 1951) formulas. The estimates are 1.08 and 1.48 respectively. The high resistance of the F_1 and the skewness toward susceptibility in the F_2 suggests that the genes for resistance have a high degree of dominance. Therefore, the estimate of minimum number of genes by the formula of Wright (Burton, 1951) should be more realistic because that formula counts dominance effects.

In summary, therefore, line 177 carries a recessive gene which confers the immune reaction, as well as

Table 16. -- Segregation for immunity to bacterial spot in parents, and progeny of a cross between Keystone and line 177 (P.I. 163192)

Population	Observed No.		Expected ratio	X^2	Probability range for X^2
	Immune	Non-immune			
Keystone (P_1)	0	36			
Line 177 (P_2)	36	0			
F_1 (P_1 x P_2)		24			
BC_1 (F_1 x P_1)		285	0:1		
BC_2 (F_1 x P_2)	144	144	1:1	0.0	> 0.9
F_2	120	307	1:3	2.19	0.1-0.25

additional genes which act quantitatively, but with considerable dominance effect.

Cook and Stall (1963) had also worked with P.I. 163192 and reported a single dominant gene for resistance which they incorporated into line 23-1-7 (Dahlbeck and Stall, 1979). However, line 23-1-7 is resistant only to race 2 of X. campestris pv. vesicatoria, which is not found in Hawaii, and not to race 1, which seems to be the race in Hawaii. The gene for immunity in line 177, therefore, is different from the gene reported by Cook and Stall (1963). Adamson and Sowell (1982) also reported that P.I. 163192 carries a single dominant gene. It would be interesting to test their material also for its reaction in Hawaii.

3) Quantitatively inherited resistance

In addition to the continuous distributions observed in parts of the crosses of Keystone and 79-4 (Table 13) and 177 (Table 15), 10 other crosses showed only continuous distributions in the segregating generations (Table 17). These crosses will be referred to in the following by their resistant parent.

In Table 17, the crosses are arranged in the order of the means of the resistant parents. The F_2 means generally followed the same order as the resistant parent means. All the F_1 and F_2 means were between the parental means, and the backcross means were all between the means of the particular

F_1 and parent. The F_1 means of all crosses except three (43, 119, and 137) were lower (more resistant) than the mid-parent value. The F_2 means were equal or higher than the F_1 except for the same three crosses.

Although each cross was tested at a different time, experimental conditions were standardized, so Duncan's multiple range test could be used to separate the F_2 means. The most resistant F_2's (177, 79, 47) were significantly more resistant than 127, which was significantly more resistant than 112. The remainder of the crosses were significantly less resistant than 112 and were not significantly different from each other. This agrees with my general impression that 177, 79, 47, 127, and 112 were good parents, but the rest were not.

The individual scaling test outlined by Mather and Jinks (1971, 1977) was applied to test the adequacy of an additive-dominance model for the 8 crosses for which adequate data were available (Table 17). All 8 crosses had at least one significant value of A, B, or C, which means that all of the crosses show some deviation from the additive-dominance model. In other words, generation means cannot be completely accounted for by the mean, additive and dominance effects parameters. This suggests that epistasis or linkage are present. Therefore, the six parameters suggested by Mather and Jinks (1971, 1977) were calculated

Table 17. -- Generation means and scaling test results for crosses showing continuous variation.

Resistant Parents[z]	Generation Means							Scaling Test		
	P1	P2	F1	MP	F2[y]	BC1	BC2	A	B	C
177 (Part only)	3.9	-	1.2	-	1.7a	2.9	1.1	-	-	-
79 (Part only)	3.1	-	-	-	1.7a	2.2	-	-	-	-
47	3.8	1.0	1.5	2.4	1.8a	2.3	1.3	-0.7**	0.1	-0.6**
127	3.4	1.1	2.1	2.3	2.1b	3.2	1.6	0.9**	0.0	-0.3
112	3.9	1.1	2.0	2.5	2.4c	3.0	1.7	0.1	0.3**	0.6**
110	3.9	1.1	2.4	2.5	2.7d	3.6	2.0	0.9**	0.5**	1.0**
43	4.0	1.0	3.4	2.5	2.9de	3.6	2.4	-0.2	1.4**	-0.2
131	4.0	1.2	-	-	3.2e	-	-	-	-	-
4	4.0	1.5	2.2	2.8	2.8d	-	-	-	-	-
119	3.9	1.7	3.3	2.8	2.8d	3.6	-	0.0	-	-1.0**
34	4.0	1.8	2.0	2.9	2.9de	3.3	2.2	0.6**	0.6*	1.9**
137	3.3	2.2	3.2	2.8	2.8d	3.3	-	0.1	-	-0.7**

[z]All crossed with Keystone (susceptible P_1)

[y]Mean separation by Duncan's Multiple Range Test, at 0.05 probability level.

for the 6 crosses with all 6 generations: P_1, P_2, F_1, F_2 and both backcrosses (Table 18). The six parameters are the mean (m), the additive effect (a), the dominance effect (d), the additive x additive interaction (aa), the additive x dominance interaction (ad), and the dominance x dominance interaction (dd).

The mean effects and additive effects were significant for all the crosses (Table 18). Significant dominance and interaction effects were detected in crosses 47, 127, 110 and 43, but not in crosses 112 and 34. These results disagree with those of the scaling test (Table 17), which indicated all these crosses had epistasis. Likewise, the F_1's of crosses 112 and 34 seemed to show dominance. The reason for these discrepancies is not known. A similar situation was encountered in a bean pod length study by Bassett (1978).

The dominance effect was negative in cross 47 but positive in crosses 127, 110, and 43. A negative dominance effect means that the heterozygotes are more resistant (the disease is reduced). A positive dominance effect means that heterozygotes are more susceptibile (the disease is increased). The observed results agree with the calculated ones for crosses 47 and 43 because the F_1 in cross 47 was resistant, but in cross 43 it was susceptible. In cross 110, the F_1 mean was close to the mid-parent, but the F_2 mean was more susceptible, so the positive dominance value

Table 18. -- Estimates of genetic parameters calculated according to Mather and Jinks (1971) for bacterial spot resistance in 6 crosses segregating continuously

Resistant Parent	m[z]	a	d	aa	ad	dd
47	2.4**	1.4**	-1.5**	0.0	-0.8**	0.6*
127	1.1**	1.2**	3.2**	1.2**	0.9**	-2.1**
112	2.7**	1.4**	-0.5	-0.2	-0.2	-0.2
110	2.1**	1.4**	2.1**	0.4*	0.4**	-1.8**
34	3.5**	1.0**	-0.9	-0.6	0.0	-0.6
43	1.1**	1.5**	4.9**	1.4**	-1.6**	-2.6**

* significant at 0.05 probability level

** significant at 0.01 probability level

[z] m = mean effect

a = additive effect

d = dominant effect

aa = additive x additive interaction

ad = additive x dominance interaction

dd = dominance x dominance interaction.

agrees with the results. But, in cross 127 the F_1 and F_2 results do not agree with the calculated positive dominance value. According to the formula for calculation of the d value, the positive value resulted because the Keystone parent developed little disease but the backcross had many susceptible plants.

The signs of d and dd were opposite in all four crosses in which they were significant. Mather and Jinks (1977, p93) stated that when d and dd have the same sign, the interaction is complementary but when d and dd have opposite signs, the interaction is the duplicate type. Thus, the interactions here are all the duplicate type.

The additive x additive interaction was significant in crosses 127, 110, and 43, but not in cross 47, while the additive x dominance interaction was significant in all 4 crosses. The signs of aa or ad do not mean anything.

Narrow and broad sense heritabilities were estimated as well as the minimum number of genes conferring resistance (Table 19).

Broad sense heritabilities ranged from 67% to 92%, while narrow sense heritabilities ranged from 21% also to 92%. The components of variance used to estimate the heritabilities were calculated on the assumption that there are no epistatic effects. Since there probably were epistatic effects, the components of variance and heritabilities are most likely biased to some extent. Both

Table 19. -- Estimates of broad and narrow sense heritabilities and minimum number of genes controlling resistance in crosses between 10 resistant lines and Keystone

Resistant	Heritability		Minimum No. of genes	
parent	Broad	Narrow	Castle[z]	Wright[y]
47	0.82	0.78	3.14	3.79
127	0.67	0.21	3.32	3.35
112	0.92	0.92	1.41	1.50
110	0.84	0.73	1.97	2.31
4	0.86	-	1.25	1.37
34	0.90	0.83	0.72	0.95
43	0.83	0.71	2.60	3.07
119	0.79	-	1.57	1.73
137	0.73	-	0.54	0.73
131	0.91	-	-	-

[z]Castle (1921)

[y]Wright (Burton, 1951).

the broad and narrow sense heritabilities for resistance to bacterial spot were relatively high. These high heritabilities are probably due to the uniform experimental conditions and the careful inoculation of each individual plant.

The estimates of the minimum number of genes ranged from less than 1 to 3 or 4. In general, a higher level of resistance was related with a larger minimum number of genes.

The F_1 and F_2 means, scaling test, and the estimates of six parameters, heritabilities, and minimum number of genes indicate that all the crosses are different from each other in some way. Crosses could, however, be grouped into three groups based on the position between the parents of the F_1 and F_2 means: Group 1, in which both the F_1 and F_2 means are on the resistant side of the midparent (Figure 17), includes crosses 177, 79, 47, 127, and 112; Group 2, in which the F_1 means are on the resistant side but the F_2 means are either the same or more susceptible than the midparent (Figure 18), includes crosses 110, 4, and 34; and Group 3, in which the F_1 means are on the susceptible side of the midparent (Figure 19), includes crosses 43, 119, and 137. The inheritance of resistance is discussed by groups as follows:

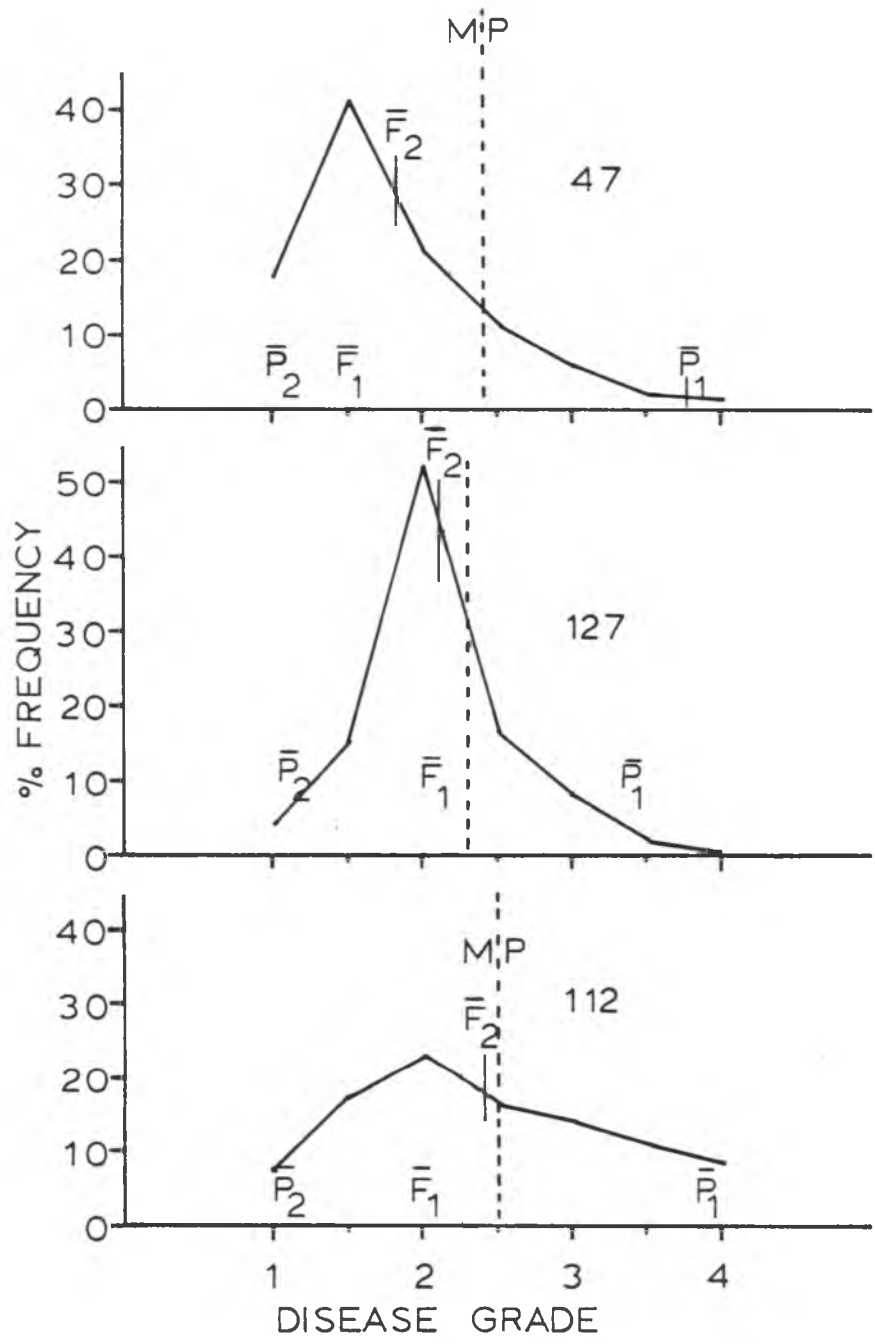


Figure 17. Percent frequency distribution of the F_2 population and location of the F_1 and F_2 means between parents of the crosses in group 1.

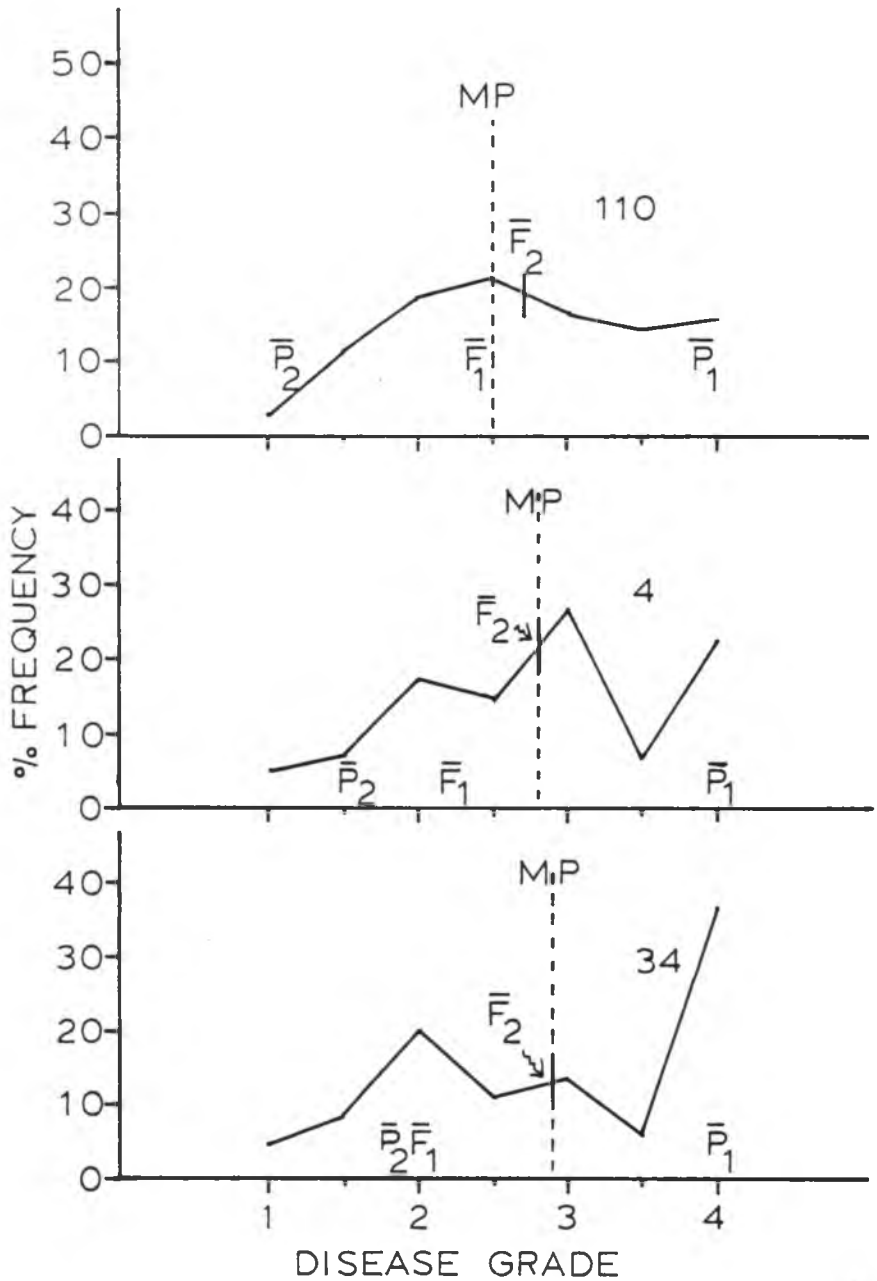


Figure 18. Percent frequency distribution of the F_2 population and location of the F_1 and F_2 means between parents of the crosses in group 2.

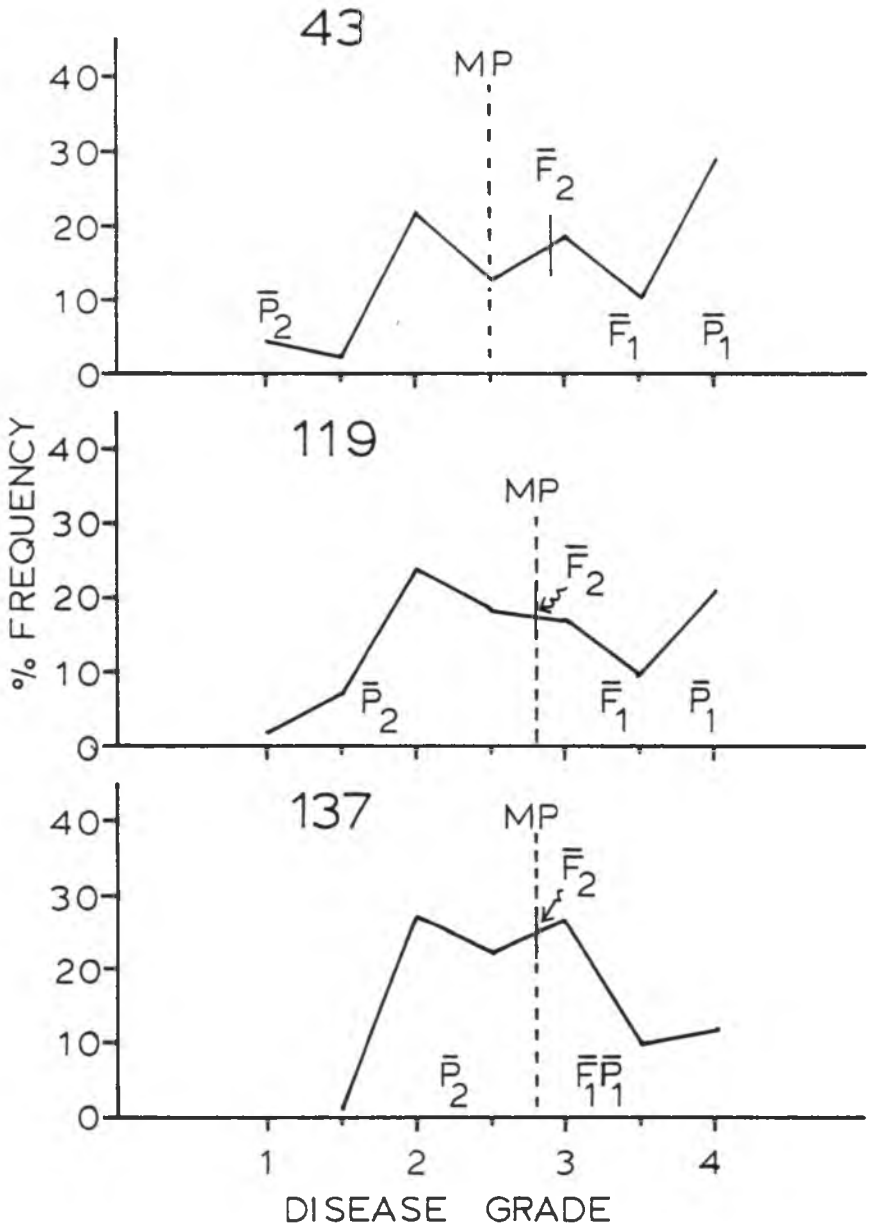


Figure 19. Percent frequency distribution of the F_2 population and location of the F_1 and F_2 means between parents of the crosses in group 3.

Crosses 79 and 177 (Group 1)

The F_2 means of the continuously distributed portions of crosses 79 and 177 were not significantly different from each other, nor from cross 47. It is, therefore, possible that the quantitatively inherited genes in line 79 and line 177 are the same as those in line 47.

Cross 47 (P.I. 244670) (Group 1)

Cross 47 was the most resistant of all the F_2 populations with only quantitative effects. The significant negative dominance effect and the location of the F_1, F_2, and both backcross means (Figure 17, Table 20) on the resistant side of the midpoint indicate that resistance in this line is partially dominant. Epistasis is also present and there are at least 3 pairs of genes involved. However, it is not possible to fit a specific segregation due to the continuous variation and interactions.

Resistance in P.I. 244670 was previously reported by Sowell (1960) and Sowell and Langford (1963). Cook and Stall (1963) reported that F_1 plants of a cross between P.I. 244670 and a susceptible cultivar were intermediate in resistance between the parents and they considered P.I. 244670 to have multigenic resistance. My results confirm the multigenic nature of the resistance in P.I. 244670, but also show the presence of some dominance effects.

Table 20. -- Frequency distribution for resistance to bacterial spot in parents and progeny of a cross between Keystone and line 47 (P.I. 244670)

Population	Frequency at disease grade of							Total No. of plants	Mean	Variance
	1.0	1.5	2.0	2.5	3.0	3.5	4.0			
Keystone (P_1)					5	9	39	53	3.8	0.107
Line 47 (P_2)	71	1						72	1.0	0.004
F_1 (P_1 x P_2)	10	51	7	4				72	1.5	0.115
BC_1 (F_1 x P_1)		22	41	47	12	9	3	134	2.3	0.354
BC_2 (F_1 x P_2)	134	58	16	6		1		215	1.3	0.165
F_2	98	225	116	62	31	10	9	551	1.8	0.427

Cross 127 (P.I. 369994) (Group 1)

The F_2 (mean 2.1) of cross 127 was significantly less resistant than cross 47 (1.8). The mode of the F_2 is 2.0 compared to 1.5 in cross 47 (Table 21). The frequency distribution of the F_2 was closer to a binomial distribution (Figure 17), but with a very slight skewness toward susceptibility. Unlike cross 47, d (dominance) was positive. This should mean that resistance is recessive, which does not agree with the F_1 and F_2 means. The reason is that the Keystone seed germinated fast because it was fresh with the result that the seedlings were older and the leaf tissue tougher than usual when inoculated, resulting in lower than normal disease grades for Keystone. Also, in the backcross to Keystone many plants were as susceptible as Keystone and the mean was close to Keystone, which would imply resistance was recessive.

Like cross 47, cross 127 had significant epistasis and involved a minimum of 3 to 4 genes.

The narrow sense heritability for this cross was unusually low. This was due to the small variance in the F_2 population. The reason for the small variance is the high frequency of the mode but it is not known why this happened. Gilbert (1961, 1973) pointed out that estimation of genetic variance and its components suffers from statistical weakness and that Warner (1952) method of estimating heritability does not permit the recognition of

Table 21. -- Frequency distribution for resistance to bacterial spot in parents and progeny of cross Keystone x line 127 (P.I. 369994)

Population	Frequency at disease grade of							Total No. of plants	Mean	Variance
	1.0	1.5	2.0	2.5	3.0	3.5	4.0			
Keystone (P_1)					12	8	7	27	3.4	0.174
Line 127 (P_2)	32	4						36	1.1	0.025
F_1 (P_1 x P_2)		2	40	14	2			58	2.1	0.086
BC_1 (F_1 x P_1)			15	35	78	57	66	251	3.2	0.355
BC_2 (F_1 x P_2)	48	135	83	9	2			277	1.6	0.156
F_2	16	59	195	63	32	9	3	377	2.1	0.285

interactions. Many statistical estimates such as means are 'robust' in the sense of being little affected by departure from normality; variances, however, are unrobust and therefore unreliably estimated. Gilbert (1973) suggested using parent-offspring regression from F_3 families for a more accurate and realistic estimate of heritability.

Resistance in P.I. 369994 was previously reported by Sowell (1980), he did not report on the inheritance of the resistance in this line.

Cross 112 (P.I. 308787)(Group 1)

The cross 112 F_2 was significantly less resistant than that of cross 127. The F_2 distribution of cross 112 was even closer to a normal binomial distribution (Figure 17, Table 22). Although the mean of the F_1 was more resistant than the parental mid-point, the dominance effect calculated was not significant (Table 18). Likewise, for the first time, there was no epistasis detected. Heritability, however, was still high and the number of genes involved was between 1 and 2, about half as many as indicated in the previous crosses. Since the F_2 distribution is roughly binomial with a mean almost exactly midway between the parents, and the number of genes estimated is about 2, it is possible that there are two pairs of genes segregating here with completely additive action.

Table 22. -- Frequency distribution for resistance to bacterial spot in parents and progeny of a cross between Keystone and line 112 (P. I. 308787)

Population	Frequency at disease grade of							Total No. of plants	Mean	Variance
	1.0	1.5	2.0	2.5	3.0	3.5	4.0			
Keystone (P_1)					2	7	45	54	3.9	0.059
Line 112 (P_2)	61	10						71	1.1	0.059
F_1 (P_1 x P_2)		3	33	7				43	2.0	0.057
BC_1 (F_1 x P_1)		7	12	21	38	35	18	131	3.0	0.459
BC_2 (F_1 x P_2)	32	51	30	21	8	1		144	1.7	0.357
F_2	40	90	119	84	72	56	45	506	2.4	0.753

There are no previous reports of resistance in P.I. 308787. The resistance is, however, effective enough to be used in a breeding project and may be useful due to its additive manner of inheritance and quantitative (generalized) nature.

Cross 110 (P.I. 297495) (Group 2)

Cross 110 had the next most resistant F_2 population. The mean of this F_2 (2.7) was significantly different from the more resistant cross 112, but was not different from any of the rest of the F_2's (Table 17). Cross 110 differed from the previous because the F_1 and F_2 means were just about midway between the parents. The mode of the cross 110 F_2 is 2.5 compared to the 2.0 of cross 112 (Figure 18, Table 23). The dominance effect calculated (Table 18) was positive, which should indicate that resistance is recessive. Some indication of this can be seen in the more susceptible reactions of the F_2 and both backcrosses as compared to the midpoint of the two parents involved.

Although heritability was again high, there are estimated to be a minimum of 2 pairs of genes involved. Thus, the lower level of resistance in cross 110 than in cross 112 may be due to a slight recessive nature of the resistance. Resistance has not been reported previously for P.I. 297495. However, the previously discussed lines would make better resistant parents than this one.

Table 23. -- Frequency distribution for resistance to bacterial spot in parents and progeny of a cross between Keystone and line 110 (P.I. 297495)

Population	Frequency at disease grade of							Total No. of plants	Mean	Variance
	1.0	1.5	2.0	2.5	3.0	3.5	4.0			
Keystone (P_1)					3	1	40	44	3.9	0.069
Line 110 (P_2)	36	3	2					41	1.1	0.061
F_1 (P_1 x P_2)			5	5	1	1		12	2.4	0.220
BC_1 (F_1 x P_1)		1	7	26	26	44	168	272	3.6	0.333
BC_2 (F_1 x P_2)	41	77	70	49	28	11	10	286	2.0	0.579
F_2	14	55	90	103	82	71	78	493	2.7	0.716

Cross 4 (P.I. 163192) (Group 2)

Cross 4 fell within the group of less resistant F_2 populations. No backcrosses were made, so only a few tests could be made. It can be seen (Figure 18, Table 24) that, although the F_1 mean was more resistant than the parental midpoint, the F_2 was less resistant, suggesting that resistance is also partially recessive in this cross. Heritability was again high and the number of genes involved was calculated as slightly over 1.

Cross 4 was included in the inheritance study for comparison with cross 177 (P.I. 163192) in which the gene for an immune reponse was found, as well as some quantitatively inherited genes. Although both line 4 and line 177 were received as P.I. 163192, they obviously carry different genes for bacterial spot resistance. Not only did cross 4 not segregate for the immune gene, but the quantitatively inherited genes also seem different.

Cross 34 (P.I. 297495) (Group 2)

Cross 34, like crosses 110 and 4, had an F_2 population with a mean very close to the midpoint of the parents. The dominance calculation also showed that dominance was significant (Table 18), which agrees with the F_2 mean. However, the calculation that only 1 pair of genes are involved does not seem to agree with the observed F_2 distribution (Figure 18, Table 25), which does not seem at

Table 24. -- Frequency distribution for resistance to bacterial spot in parents and progeny of a cross between Keystone and line 4 (P.I. 163192)

Population	Frequency at disease grade of							Total No. of plants	Mean	Variance
	1.0	1.5	2.0	2.5	3.0	3.5	4.0			
Keystone (P_1)					1	1	31	33	4.0	0.037
Line 4 (P_2)	7	16	9					32	1.5	0.128
F_1 (P_1 x P_2)	1	3	47	10	9			70	2.2	0.165
F_2	12	16	38	32	58	15	49	220	2.8	0.792

Table 25. -- Frequency distribution for resistance to bacterial spot in parents and progeny of a cross between Keystone and line 34 (P.I. 224451)

Population	Frequency at disease grade of							Total No. of plants	Mean	Variance
	1.0	1.5	2.0	2.5	3.0	3.5	4.0			
Keystone (P_1)					2		69	71	4.0	0.028
Line 34 (P_2)		3	3					6	1.8	0.075
F_1 (P_1 x P_2)	2	7	20	4	2			35	2.0	0.197
BC_1 (F_1 x P_1)			8	4	12	4	26	54	3.3	0.566
BC_2 (F_1 x P_2)	4	7	22	4	5	1	3	46	2.2	0.554
F_2	14	27	66	36	46	20	124	333	2.9	0.963

all like a single gene distribution. Some interesting observations in cross 34 are that, although the F_2 mean was equal to the parental midpoint, the F_1 was equal to the resistant parent, and both the F_1 and the F_2 actually had some individuals which were more resistant than the resistant parent. Since it is unlikely that Keystone carries any genes that might cause transgressive segregation, it is possible that there are some inaccuracies in the figures reported for the parental line 34.

The level of resistance in line 34 is relatively low and this line bears small fruits (Table 2). Line 34 is, therefore, even less useful as breeding material than the previous ones. There is no previous report for resistance in line P.I. 224451.

Cross 43 (P.I. 241670) (Group 3)

Unlike in all the crosses already discussed, in cross 43 both the F_1 and F_2 populations were less resistant than the parental midpoint (Table 17). The recessiveness of resistance in this cross was confirmed by the positive d value calculated (Table 18). In addition to the dominance of susceptibility, epistasis was also significant but heritability was still high, and there was a minimum of 3 pairs of genes involved. The recessive nature of resistance in this cross is apparent in the backcross results (Table 26). The backcross to Keystone gave mainly susceptible

Table 26. -- Frequency distribution for resistance to bacterial spot in parents and progeny of a cross between Keystone and line 43 (P.I. 241670)

Population	Frequency at disease grade of							Total No. of plants	Mean	Variance
	1.0	1.5	2.0	2.5	3.0	3.5	4.0			
Keystone (P_1)						2	25	27	4.0	0.018
Line 43 (P_2)	65	4	1					70	1.0	0.027
F_1 (P_1 x P_2)			3	8	19	12	28	70	3.4	0.371
BC_1 (F_1 x P_1)			2	1	8	20	32	70	3.6	0.234
BC_1 (F_1 x P_2)	26	20	55	31	36	21	21	210	2.4	0.805
F_2	22	12	103	61	88	51	136	473	2.9	0.804

progenies, while the backcross to line 43 gave all classes of progeny, as would be expected if resistance is recessive. Resistance has not been previously reported in P.I. 241670. Line 43 (P.I. 241670) should be of value as a source for resistance to bacterial spot because, even though it is recessive, the resistance is effective.

Cross 119 (P.I. 322719) (Group 3)

Like cross 43, resistance in cross 119 appeared to be recessive. The F_1 mean was much more susceptible than the parental midpoint, and although it was more resistant than the F_1, the F_2 was also more susceptible than the midpoint (Figure 19, Table 27). The backcross to the susceptible parent gave a highly susceptible population, agreeing with the conclusion that resistance is recessive. No backcross to the resistant parent was obtained due to poor fruit set on both the F_1 and line 119. It was also observed that F_1 plants which were moved outdoors to produce F_2 seed developed spots and appeared susceptible.

Heritability was again high and there were a minimum of 1 - 2 pairs of genes involved. The F_2 showed a continuous distribution with a high number of individuals in all classes except the most resistant one.

Resistance in P.I. 322719 has been reported previously by Sowell and Dempsey (1977). This resistance was also reported to be controlled by a single dominant gene (Adamson

Table 27. -- Frequency distribution for resistance to bacterial spot in parents and progeny of a cross between Keystone and line 119 (P.I. 322719)

Population	Frequency at disease grade of							Total No. of plants	Mean	Variance
	1.0	1.5	2.0	2.5	3.0	3.5	4.0			
Keystone (P_1)					3	2	55	60	3.9	0.055
Line 119 (P_2)	7	23	1					31	1.7	0.057
F_1 (P_1 x P_2)			1	5	11	8	11	36	3.3	0.331
BC (P_1 x F_1)			1	6	8	23	34	72	3.6	0.258
F_2	8	31	104	77	72	41	88	421	2.8	0.717

and Sowell, 1982). My results do not agree with theirs. My results show a continuous variation in the F_2 with the resistance more recessive than dominant.

Cross 137 (P.I. 377688) (Group 3)

Like cross 119, cross 137 appeared to be segregating for resistance that was recessive. The F_1 mean was quite susceptible (Figure 19, Table 28) and the F_2 mean was less so. The backcross to the susceptible parent was also mostly susceptible. No backcross was obtained to the resistant parent because, again, both the F_1 and the resistant parent showed poor set. However, the heritability was high and the minimum number of genes involved is apparently quite low. Cross 137 is similar in almost all respects to cross 119.

Resistance in P.I. 377688 has not been previously reported. This line is not recommended for breeding for bacterial spot resistance because of its low level of resistance and poor fruit set.

Cross 131 (P.I. 369998)

A cross was made between Keystone and line 131. Although the F_1 seeds germinated and some F_2 seeds were obtained, the F_1 seed lot lost viability soon, so it could not be tested later, nor could backcrosses be made.

The F_2 population shows a continuous distribution skewed toward the resistant parent (Table 28). The resistance seem

Table 28. -- Frequency distribution for resistance to bacterial spot in parents and progeny of a cross between Keystone and line 137 (P.I. 377688)

Population	Frequency at disease grade of							Total No. of plants	Mean	Variance
	1.0	1.5	2.0	2.5	3.0	3.5	4.0			
Keystone (P_1)					10	8	3	21	3.3	0.134
Line 137 (P_2)			16	8				24	2.2	0.058
F_1[z]			1	8	34	22	6	71	3.2	0.178
(P_2 x P_1) x P_1			4	14	10	18	18	64	3.3	0.413
F_2[a]		10	170	139	167	63	75	624	2.8	0.454

[z]Reciprocal combined.

Table 29. -- Frequency distribution for resistance to bacterial spot in parents and progeny of a cross between Keystone x line 131 (P.I. 369998)

Population	Frequency at disease grade of							Total No. of plants	Mean	Variance
	1.0	1.5	2.0	2.5	3.0	3.5	4.0			
Keystone (P_1)					1	1	31	33	4.0	0.037
Line 131 (P_2)	25	6	3					34	1.2	0.104
F_2	5	9	44	38	43	23	145	307	3.2	0.747

to be recessive.

7. Crosses between resistant parents

1) Cross between line 79 (with the gene for hypersensitivity) and line 177 (with the gene for immunity)

Line 79 carries a single dominant gene for hypersensitivity, while line 177 carries a single recessive gene for immunity. Both lines also have additional genes which show quantitative inheritance. A cross was made between these 2 lines to test the relation between these genes for resistance. The reactions of the parents, F_1, and F_2 populations are given in Table 30.

All the F_1 plants were hypersensitive as expected since the gene for hypersensitivity is dominant, but the gene for immunity is recessive. The F_2 population segregated into 293 hypersensitive plants, 134 immune plants, and 76 plants which were neither. The plants which were neither hypersensitive nor immune were graded either 1.0 or 1.5. Susceptible plants (3.0 or over) were not found, although Keystone was infected as usual.

The observed numbers were tested for their fit to the expected ratios. When hypersensitivity only was considered, the observed numbers, 293 hypersensitive and 205 non-hypersensitive, do not fit a 3:1 ratio (x^2=69.19, $P < 0.005$) but do fit a 9:7 ratio (x^2=1.35 P=0.1-0.25). When immunity only was considered, the observed numbers, 134

Table 30. -- Segregation for resistance to Xanthomonas campestris pv. vesicatoria in a cross between line 177 (P.I. 163192) and line 79 (P.I. 271322)

Population	Hyper-sensitive	Immune	Others								Total plants Tested
			1.0	1.5	2.0	2.5	3.0	3.5	4.0	Mean	
Keystone							1	1	31	4.0	33
Line 177 (P_1)		28									28
Line 79 (P_2)	36										36
F_1 (P_1 x P_2)	32										32
F_2	293	134	71	5						1.0	503

X^2 = 69.19 ($P < 0.005$) for 3 hypersensitive to 1 non-hypersensitive in F_2

X^2 = 1.35 ($P = 0.1 - 0.25$) for 9 hypersensitive to 7 non-hypersensitive

X^2 = 0.97 ($P = 0.25 - 0.50$) for 1 immune to 3 non-immune

X^2 = 6.68 ($P = 0.025 - 0.05$) for 9 hypersensitive, 4 immune, and 3 others.

immune plants and 364 others including hypersensitive ones, fit the 1:3 ratio expected for a single recessive gene (x^2 = 0.97, P = 0.25-0.5).

These results suggest that there was an interaction between the hypersensitivity gene and the immunity gene. Since the segregation ratio for the immunity reaction was not changed but that for hypersensitivity was, it is possible to infer that the immune reaction (which prevents tissue collapse) masks the hypersensitive reaction (quick necrotic tissue collapse). Then the expected ratio becomes 9 hypersensitve, 4 immune, and 3 others. The observed Chi-square (6.68, P = 0.05-0.025) was significant at the 0.05 probability level but not at the 0.01 level. It is, therefore, concluded that the effect of the hypersensitivity gene from line 79 (P.I. 271322) is masked by the immunity gene from line 177 (P.I. 163192) when they are together.

Since susceptible plants were not recovered, the quantitative genes in line 177 and line 79 may be the same. These quantitative factors in the 2 lines also behaved similarly in the previous crosses. The distributions of disease grades of the non-hypersensitive plants in the F_2 population of the cross Keystone x line 79-4 (Table 13) and the non-immune plants in the F_2 population of the cross Keystone x line 177 (Table 15) were similarly skewed toward the susceptible parent and the means were not significantly different (Table 17). These results support the possibility

that the genes for quantitative resistance in both lines may be the same at least in part.

2) Cross between line 43 (with quantitative genes) and line 79 (with hypersensitivity gene)

It has been shown that resistance in line 43 (P.I. 241670) is controlled by multiple genes with recessive effects. A cross between line 43 and line 79 was made to compare the genes for quantitative resistance in the 2 parents. The results for the parents, F_1, and F_2 populations are presented in Table 31.

The F_1 plants were all hypersensitive as expected. The F_2 plants segregated into 381 hypersensitive and 117 non-hypersensitive plants, which fit the expected 3:1 ratio (X^2 = 0.62, P = 0.25-0.5). It is, therefore, concluded that the inheritance of the hypersensitivity gene was not affected by the quantitative background genes in line 43.

The non-hypersensitive plants in the F_2 population ranged from 1.0 to 3.0 in disease grade. Plants with disease grade 2.0 or over may be considered susceptible. The number recovered was small. The scarcity of susceptible plants is considered to be due to the effects of quantitative genes from both line 43 and line 79. Since some susceptible plants were recovered, the quantitative genes in line 79 and those in line 43 are considered to be

Table 31. -- Segregation for resistance to Xanthomonas campestris pv. vesicatoria in a cross between line 43 (P.I. 241670) and line 79-4 (P.I. 271322)

Population	Hyper-sensitive	Non-hypersensitive at disease grade								Total plants tested
		1.0	1.5	2.0	2.5	3.0	3.5	4.0	Mean	
Keystone						1	1	31	1.0	33
Line 43 (P_1)		28	7						1.1	35
Line 79 (P_2)	36									36
F_1 (P_1 x P_2)	72									72
F_2	381	96	16	3	1	1			1.1	498

X^2 = 0.602 (P = 0.25-0.5) for 3 hypersensitive to 1 non-hypersensitive ratio.

different. This is also suggested by the fact that the resistance in line 43 was somewhat recessive.

3) Cross between line 43 (quantitative genes) and line 177 (immunity gene)

Line 43 was also crossed with line 177 to examine the behavior of the immunity gene in the quantitative gene background of line 43. The results for the parents, F_1, and F_2 progenies are given in Table 32. The F_1 plants developed lesions at the infiltrated area but the lesions were very light and appeared intermediate between the immune response and disease grade 1.0. The F_2 plants ranged continuously from completely immune to intermediate to disease grade 2.5. One hundred thirty eight plants showed lesions between the immune type and 1.0. The observed numbers of immune type plants, intermediate, and non-immune with disease grade 1.0 or over were 227, 138, and 50, respectively. More immune type plants were recovered than expected for a single recessive gene. This suggests that the effect of the single recessive gene for immunity was modified by genes in line 43. Only one plant over 2.0 in disease grade was observed, suggesting no segregation for the quantitative genes.

Modification of the effect of major genes by minor genes is not rare. Bird and Hadley (1958) have shown that a major gene for resistance to bacterial blight resistance in cotton was continuously inherited in a tolerant polygenic

Table 32. -- Segregation for resistance to Xanthomonas campestris pv. vesicatoria in a cross between line 43 (P.I. 241670) and line 177 (P.I. 163192)

Population	Immune	Inter-mediate	Others								Total plants Tested
			1.0	1.5	2.0	2.5	3.0	3.5	4.0	Mean	
Keystone							22	1		3.0	23
Line 43 (P_1)			24							1.0	24
Line 177 (P_2)	24										24
F_1 (P_1 x P_2)		23	1								24
F_2	227	138	48	1		1				1.0	415

background. Innes and Brown (1969), and Green and Brinkerhoff (1956) also had difficulty in following segregation of major genes in somewhat tolerant genotypes.

It is, therefore, concluded that the single recessive gene for immunity in line 177 is influenced by the genes in line 43 and segregation of this major gene in F_2 was obscured.

8. Comparison of resistance to Xanthomonas diseases in pepper, cotton, and rice.

Interesting phenomena discovered in this study are the diversity of the resistance genes, the coexistence of major qualitative genes and minor, quantitative genes in the same line, and the high incidence of resistance in lines which originated in India.

At least three different kinds of resistance were found. A dominant gene conferring hypersensitivity was found in line 79 (P.I. 271322), a recessive gene conferring an "immune" response was found in line 177 (P.I. 163192), and quantitative genes were found in these lines and 12 other resistant lines. Many similarities are found in resistance to bacterial blight (Xanthomonas campestris pv. malvacearum) in cotton (Brinkerhoff, 1970). At least 11 genes for resistance (B1 to B11) have been reported. All but one (B7) are dominant or partially dominant but the level of resistance each gene confers is different. The

most effective gene (B4) confers immunity. The highest levels of resistance were found in the diploid cottons (Gossypium arboreum and G. herbaceum) of India and surrounding areas, with the level of resistance tending to be lower in materials from the peripheral areas. Resistance was also found in a New World cotton (G. hirsutum var. punctatum) acclimatized in the Old World (India and Africa), but only a low level of tolerance was found in New World cotton cultivars from the U. S. cotton belt. Based on this distribution, Knight and Hutchison (1950) postulated that bacterial blight of cotton originated in the Old World, and was introduced to the New World fairly recently. They stated that the high levels of resistance developed in India by the occurrence first of a major gene (B4) and then the accumulation of minor genes around it. It was frequently found that the less effective minor genes were associated with the major genes (Knight, 1948). Although the value of the minor genes is limited, whenever there has been prolonged selection under epidemic attack, the main gene has became fortified by lesser genes (Knight and Hutchison, 1950). Effective resistance can, in fact, only be built around a main gene (Knight and Hutchison, 1950, Parlevliet, 1980).

A number of genes (Xal to Xa8) have also been reported to control resistance to bacterial leaf blight (X. campestris pv. oryzae) in rice (Sidhu et al., 1978). All

except 2 are dominant genes. Like in pepper and cotton, many of the sources of resistance are from the Indian subcontinent.

Thus, in all 3 bacterial diseases, on pepper, cotton, and rice, there seem to be many genes for resistance which have varying degrees of effectiveness and a high frequency of occurrence in India.

9. Relationship between resistance and pungency

It is often suggested that resistance to bacterial spot might be related to pungency because many sources of resistance to bacterial spot are from hot pepper and some think strong substances produced by plants are as offensive to diseases and insects as they are to humans. An example of this feeling is the report by Weber (Sowell, 1960), who reported considerably less disease among "hot" varieties than among mild or sweet varieties.

Mature red fruits were harvested from F_2 plants of the cross Keystone x line 112 (P.I. 308787) and tested for a possible correlation between bacterial spot resistance and pungency. Line 112 is one of the hottest lines rating 10 on the Van Blarcom and Martin (1947) scale. Keystone has no pungency and is rated 1 (Table 35 in Appendix).

The correlation coefficient between pungency and disease grades taken 7 days after inoculation in the greenhouse was -0.09 (Figure 20) and was not significantly

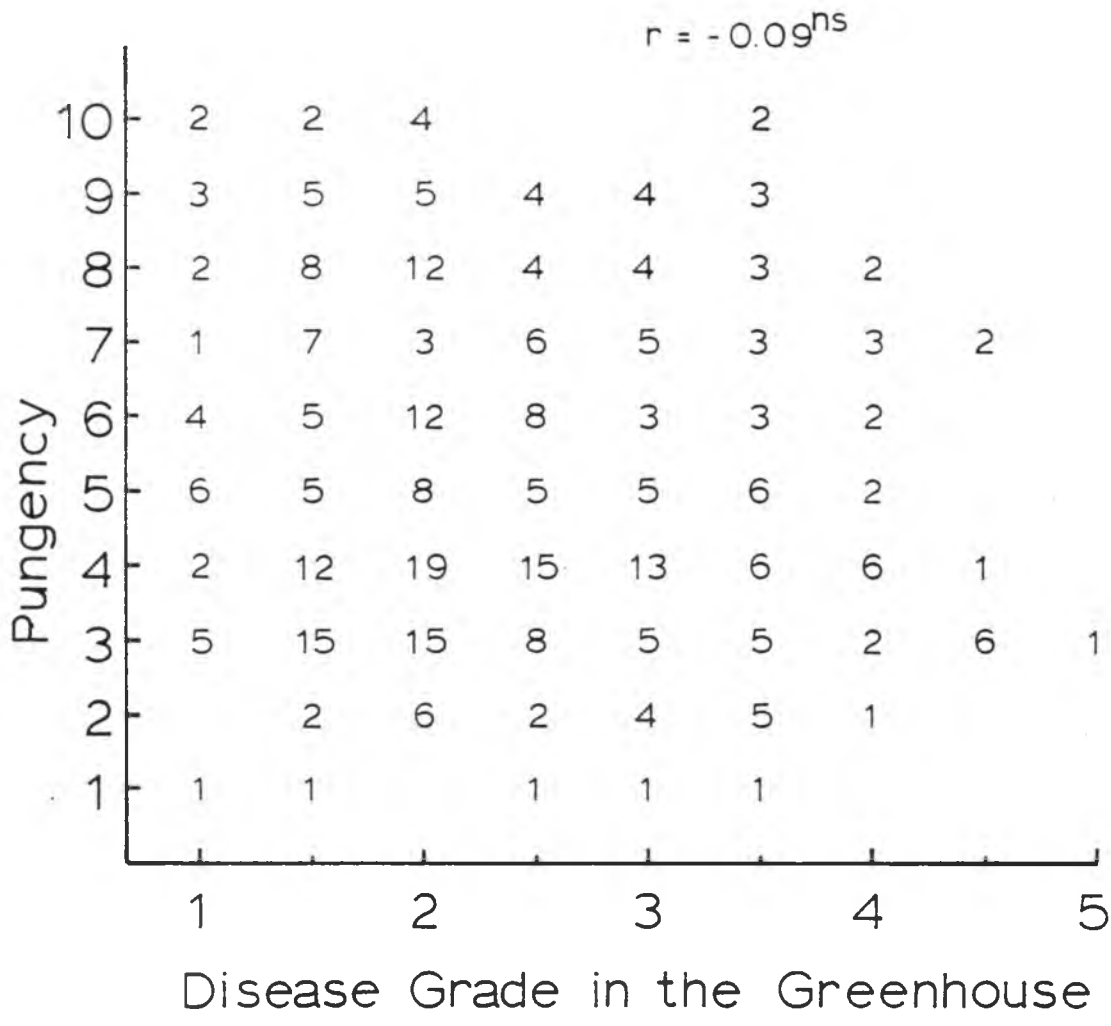


Figure 20. Pungency of pepper fruits on the F_2 plants from each greenhouse disease grade of the cross of Keystone x line 112 (P.I. 308787).

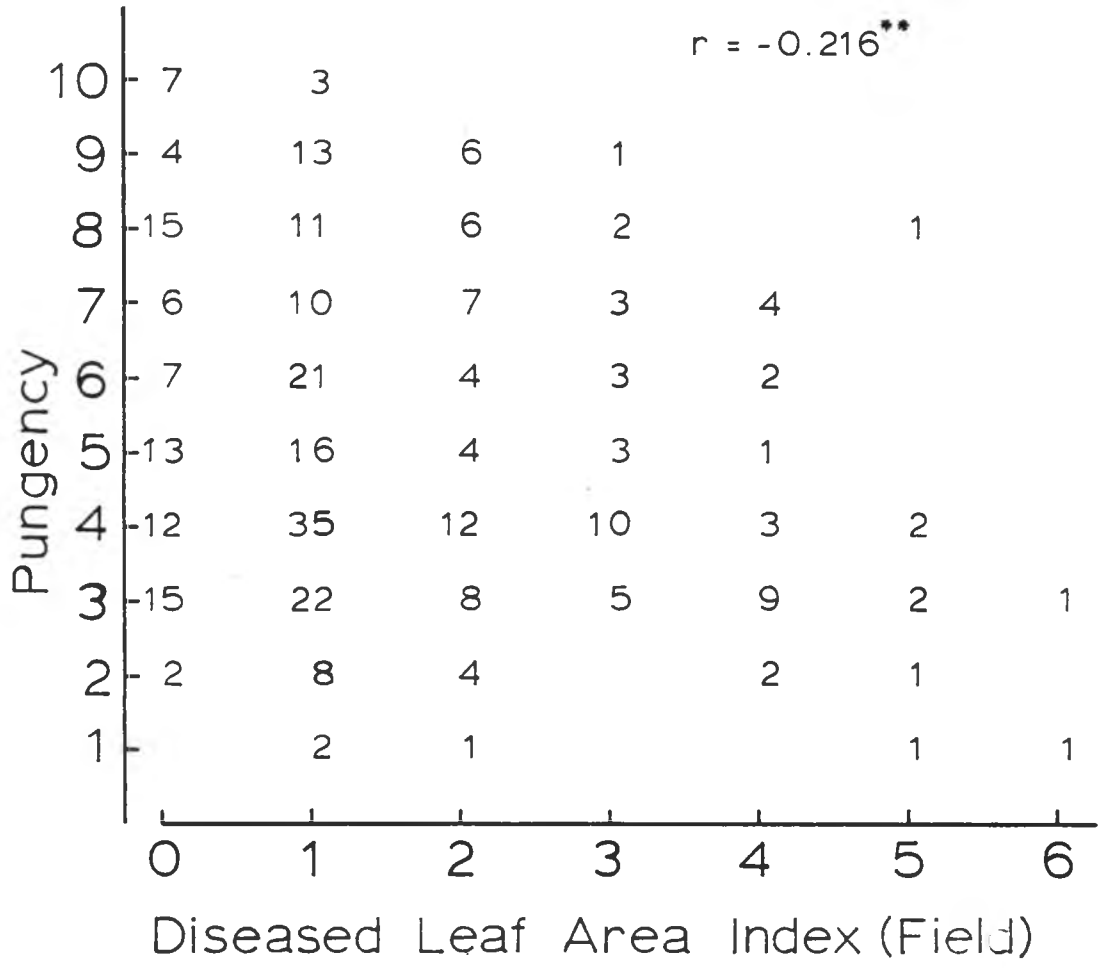


Figure 21. Relationship between pungency of pepper fruits and diseased leaf area index taken 37 days after inoculation in the field.

different from zero. However, the correlation coefficient between pungency and disease ratings in the field 37 days after inoculation, -0.216 (Figure 21), was highly significant. This shows that high pungency is associated with less disease and suggests that pungency may contribute to field resistance. However, the correlation coefficient is quite low and there are both mild and hot peppers in almost every disease grade in both the greenhouse and field. This indicates that selection of sweet peppers resistant to bacterial spot will not be difficult. This agrees with an experienced pepper breeder's opinion (Tanaka, personal communication).

Also there were some resistant parent lines, 4, 110, and 119 that were mild (Table 35 in Appendix).

SUMMARY AND CONCLUSION

One hundred sixty eight P.I. lines, 6 Hawaiian breeding lines from T. T. Sekioka, and 24 Korean cultivars were evaluated for resistance to bacterial spot. Several P.I. lines which originated in India as well as a few which originated in Central or South America showed resistance.

Seven isolates of Xanthomonas campestris pv. vesicatoria were collected from various locations on the islands of Oahu and Kauai and inoculated on 10 selected lines of pepper to test for any differences in virulence and host-specificity in Hawaii. There were no differences in virulence between isolates. Line 23-1-7 received from A. A. Cook in Florida with a gene for resistance to race 2 of the pepper strain of X. campestris pv. vesicatoria was susceptible, which shows that the strain present in Hawaii is not race 2. When I sent line 79 (P.I. 271322) with the hypersensitivity gene to Cook in Florida, he reported that it was hypersensitively resistant to the Florida race 1 but susceptible to the Florida race 2. Line 79, therefore, could also be used as an indicator for race identification like 23-1-7. The strain of the bacterial spot organism that occurs in Hawaii, therefore, appears to be pepper strain race 1.

Two inoculation methods, infiltration and spraying, were evaluated for their accuracy and efficiency in testing

for resistance. Both methods separated resistant lines from susceptible ones accurately, but the infiltration method had some adavantages: 1) The hypersensitive response in line 79 (P.I. 271322) and the "immune" response in line 177 (P.I. 163192) were easily observed, 2) The results were more similar to the field results, and 3) Incubation after inoculation was not required and evaluation of disease was easier. Two levels of inoculum, 10^8 and 10^9 cells/ml were also tested. There was no difference between the two levels of inoculum in disease development. Thus the method of inoculation used was infiltration with 10^8 cells/ml.

Selected lines of pepper were inoculated in the greenhouse, evaluated, and then transplanted to the field along with an uninoculated set of plants to compare the effect of inoculation versus no inoculation and disease development in the greenhouse and the field. No lines which were resistant in the greenhouse later developed disease in the field. Some partially resistant lines in the greenhouse were resistant in the field. Susceptible lines were diseased in the field whether they were inoculated or not.

A correlation was made between the disease grade in the greenhouse and the diseased leaf area index in the field for individual plants in the F_2 of the cross Keystone x line 112 (P.I. 308787). The correlation coefficient between the disease grade in the greenhouse 7 days after inoculation and the diseased leaf area index in the field 37 days after

inoculation in the field was 0.44. Although the correlation coefficient was relatively low, plants graded resistant in the greenhouse were consistently resistant in the field. The deviations came from plants graded susceptible in the greenhouse which developed less disease in the field. This infiltration technique and method of grading seemed quite reliable for detecting resistant individuals.

Twelve resistant lines were selected and crossed to the susceptible cultivar Keystone to study the inheritance of resistance. In line 79 (P.I. 271322), a single dominant gene that confers hypersensitivity was found as well as additional quantitatively inherited genes. In line 177 (P.I. 163192) a single recessive gene that confers a type of response called "immune" was found plus, again, quantitatively inherited genes. Lines 47 (P.I. 244670), 127 (P.I. 369994), 112 (P.I. 308787), 110 (P.I. 297495), 4 (P.I. 163192), 34 (P.I. 224451), 43 (P.I. 241670), 119 (P.I. 322719), 137 (P.I. 377688), and 131 (P.I. 369998) had only quantitatively inherited genes. Lines 47, 127, and 112 transmitted the most effective resistance. Lines 110, 4, and 34 were less effective. The resistance in lines 43, 119, and 137 appeared to be recessive, instead of dominant as in the other crosses.

Crosses were made between line 79 (hypersensitivity), line 177 (immunity), and line 43 (quantitative, but recessive). When lines 79 and 177 were crossed, the

hypersensitivity gene was masked by the immunity gene to give an F_2 ratio of 9 hypersensitive : 4 immune : 3 others. The quantitative genes did not show any segregation. The hypersensitive character segregated normally in the cross of lines 79 and 43, but the immunity character did not in the cross of lines 177 and 43. Instead of a discontinuous immune - not immune segregation, there was a continuous distribution between the two types.

The correlation coefficient between pungency and disease in the greenhouse was not significant, but in the field it was significant (-0.216). This suggests that pungency may contribute to field resistance in some way. However, the correlation coefficient is low and many mild individuals were found with low disease incidence. Therefore, selection of sweet, bacterial spot resistant lines should not be difficult.

APPENDIX

Table 33. -- Results of preliminary evaluation of October, 1979 planting of germplasm inoculated in the greenhouse and later transplanted to the field[z]

No.	P.I. No.	Origin	No. plants tested	Disease grade[y] (greenhouse)	Defoliation index[x] (field)	Remarks[w]
1	123469	India	14	3.8	2.9	
2	163184	India	17	3.0	0.4	R (P)
3	163189	India	19	3.1	0.9	R (P)
4	163192	India	11	3.0	0.5	R (M)
5	164471	India	24	3.0	1.4	R
6	164677	India	4	2.0	1.3	R
7	173877	India	16	3.4	0.9	R
8	182646	Guat.	18	3.7	1.5	R
9	183439	India	20	3.5	1.1	R
10	183440	India	7	3.1		R
11	183441	India	17	2.2	0.9	R
12	183922	India	18	3.4	1.1	R (P)
13	201232	Mex	16	3.6		
14	201234	Mex	2	1.5	0.5	
15	224414	Co Ri	20	3.7		
16	224415	Co Ri	17	4.2		
17	224417	Nicar	23	4.2		
18	224418	Nicar	23	3.3		
19	224419	Nicar	24	3.6		
20	224420	Nicar	19	4.2		
21	224421	Nicar	20	4.3		

Table 33. -- (continued) Results of preliminary evaluation of October, 1979 planting of germplasm inoculated in the greenhouse and later transplanted to the field[z]

No.	P.I. No.	Origin	No. plants tested	Disease grade[y] (greenhouse)	Defoliation index[x] (field)	Remarks[w]
22	224422	Nicar	21	4.2		
23	224423	Co Ri	22	4.8		
24	224425	Co Ri	21	4.5		
25	224429	Guat	21	3.8		
26	224430	Guat	10	2.7	1.6	
27	224432	Guat	11	4.0		
28	224433	Guat	18	4.8		
29	224434	Guat	21	4.1		
30	224435	Cuba	21	2.3	1.1	
31	224439	Nicar	19	4.1		
32	224442	Nicar	20	3.4		
33	224450	Nicar	21	2.9	1.6	
34	224451	Guat	20	2.3	1.1	
35	224452	Guat	18	3.7		
36	234249	Guat	22	3.5		
37	234250	Guat	14	4.2		
38	238054	Co Ri	20	4.3		
39	238058	Guat	19	4.0		
40	241641	Colom	18	4.9		
41	241644	Colom	8	3.9		

Table 33. -- (continued) Results of preliminary evaluation of October, 1979 planting of germplasm inoculated in the greenhouse and later transplanted to the field[z]

No.	P.I. No.	Origin	No. plants tested	Disease grade[y] (greenhouse)	Defoliation index[x] (field)	Remarks[w]
42	241655	Peru	12	3.0	0.9	
43	241670	Ecua	16	2.4	0.6	
44	241677	Peru	18	4.6		
45	244668	India	21	3.3		
46	244669	India	21	3.5	1.4	
47	244670	India	21	2.3	1.3	R (P)
48	246331	Ceyl	6	3.3	0.4	R (P)
49	249634	India	22	3.6		
50	249635	India	19	3.5		
51	257044	Colom	19	4.2		
52	257047	Colom	21	4.0		
53	257048	Colom	13	4.7		
54	257049	Colom	14	4.4		
55	257052	Colom	19	4.6		
56	257053	Colom	18	4.2		
57	257054	Colom	17	3.9		
58	257055	Colom	22	4.5		
59	257078	Colom	21	3.4		
60	257087	Colom	19	2.5	1.8	
61	257098	Colom	16	3.2		

Table 33. -- (continued) Results of preliminary evaluation of October, 1979 planting of germplasm inoculated in the greenhouse and later transplanted to the field[z]

No.	P.I. No.	Origin	No. plants tested	Disease grade[y] (greenhouse)	Defoliation index[x] (field)	Remarks[w]
62	257099	Colom	21	2.9	1.1	
63	257102	Colom	18	3.2		
64	257118	Colom	20	3.4		
65	257119	Colom	9	3.4		
66	257144	Peru	21	2.8	1.1	
67	257178	Peru	12	2.8	2.1	
68	257182	Colo	19	3.7		
69	257187	Colo	23	3.0	1.0	
70	260436	Boli	20	4.5		
71	260588	Boli	10	2.9		
79	271322	India	15	1.9	1.0	R
119	322719	India	19	2.4	1.0	R
169	Jessore x F_3(Emerald x Cook)		7	2.3	3.0	From Sekioka
170	Shepherd x Jessore		1	1.0	1.0	From Sekioka
172	Keystone	USA	42	4.4	2.5	
173	Yolo Wonder		18	3.6	2.5	

[z]Underlined lines were used in making crosses for inheritance study.

[y]Disease grade, 1 = No visible spots, 2 = Pin point spots, 3 = Small round spots with a diameter around 2mm or smaller, 4 = Spots larger than 3 and expanding, 5 = Many

type 4 spots coalesced or marginal scorch or blight accompanying defoliation.

[x]Defoliation index, 0 = No visible spots, 1 = trace to 20 % defoliation, 5 = 80 - 100 % defoliation.

[w]reported to be resistant by Sowell (1960), or Sowell and Dempsey (1977). M or P in parentheses indicates monogenic or polygenic inheritance according to Cook and Stall (1963).

Table 34. -- Results of preliminary evaluation of the January, 1980 planting of germplasm inoculated in the greenhouse and later transplanted to the field[z]

No.	P.I. No.	Origin	No. plants tested	Disease grade[y] (greenhouse)	Defoliation index[x] (field)	Remarks[w]
4	163192	India	5	2.2	0.6	R (M)
177	163192	India	11	2.3	0.2	
43	241670	Ecua	9	1.7	0.0	
72	267727	Guat	12	3.1		
73	267732	Pu Ri	11	3.5		
74	267736	Guat	11	3.5		
75	267737	Guat	9	3.1		
76	267738	Guat	5	3.0		
77	271043	India	11	2.5	0.5	
78	271321	India	6	2.7	0.5	
79	271322	India	10	1.6	0.0	R
80	271460	India	9	2.9	0.3	
81	271461	India	7	3.1		
82	271462	India	10	2.0	0.3	
85	281301	Boli	12	2.6		
87	281318	Chile	7	3.7		
89	281327	Colom	8	3.4		
90	281330	Co Ri	12	3.6	1.7	
91	281341	Elsal	4	5.0		
92	281400	Nicar	6	4.0		
93	281413	Peru	12	3.8	1.0	

Table 34. -- (continued) Results of preliminary evaluation of January, 1980 planting of germplasm inoculated in the greenhouse and later transplanted to the field[z]

No.	P.I. No.	Origin	No. plants tested	Disease grade[y] (greenhouse)	Defoliation index[x] (field)	Remarks[w]
94	281416	Phili	12	3.6		
95	288303	India	8	2.6	0.3	
96	288304	India	12	2.1	0.0	
97	288305	India	7	3.0	0.7	
98	297482	India	9	2.1	0.1	
99	297483	India	12	2.5	0.9	
100	297484	India	12	1.9	0.3	
101	297486	India	12	3.0	0.8	
102	297487	India	12	2.1	0.0	
103	297488	India	12	2.3	0.0	
104	297489	India	12	1.7	0.2	
105	297490	India	12	3.3	1.0	
106	297491	India	12	2.3	0.5	
107	297492	India	12	2.9		
108	297493	India	12	1.8	0.1	
109	297494	India	12	2.0	0.4	
110	297495	India	12	1.8	0.0	
111	297496	India	12	2.1	0.2	
112	308787	India	12	1.6	0.0	
113	308788	India	12	2.0	0.2	

Table 34. -- (continued) Results of preliminary evaluation of January, 1980 planting of germplasm inoculated in the greenhouse and later transplanted to the field[z]

No.	P.I. No.	Origin	No. plants tested	Disease grade[y] (greenhouse)	Defoliation index[x] (field)	Remarks[w]
114	308789	India	12	2.1	0.0	
115	308790	India	12	2.3	0.0	
116	308791	India	12	4.2	2.9	
117	315007	Peru	12	5.0		
118	322718	India	12	2.8	0.5	
119	322719	India	11	3.3	0.2	
120	322720	India	12	2.6	0.9	
121	322726	India	12	2.9	1.0	
122	322727	India	12	2.4	0.6	
123	322728	India	12	2.6	1.0	
124	322730	India	12	3.0	1.0	
125	323314	India	12	3.3		
126	358812	Maly	11	2.3	0.5	
127	369994	India	12	1.5	0.3	
128	369995	India	12	2.4	0.3	
129	369996	India	12	3.3		
130	369997	India	12	3.0	0.4	
131	369998	India	12	1.6	0.1	
132	369999	India	12	2.3	0.2	
133	370000	India	12	3.2		

Table 34. -- (continued) Results of preliminary evaluation of January, 1980 planting of germplasm inoculated in the greenhouse and later transplanted to the field[z]

No.	P.I. No.	Origin	No. plants tested	Disease grade[y] (greenhouse)	Defoliation index[x] (field)	Remarks[w]
134	370001	India	12	4.4	3.3	
135	370002	India	12	3.3		
136	370003	India	12	2.8	0.7	
137	377688	Mala	12	2.1	0.3	
138	378647	India	12	4.3		
139	390436	Ecua	12	4.1		
140	390437	Ecua	12	4.4		
141	390611	Peru	12	3.8		
142	390612	Peru	12	4.3		
143	406723	Co Ri	12	4.3		
144	406724	Co Ri	12	2.6	0.8	
145	406725	India	12	2.9	0.4	
146	406847	Hond	12	3.4		
147	406987	Pana	12	2.3	0.3	
148	410407	Braz	4	4.0		
151	200729	Guat	11	4.0		
153	215731	Peru	12	3.9		
154	215732	Peru	12	3.8		
155	215733	Peru	12	3.8		
156	215734	Peru	9	3.1		

Table 34. -- (continued) Results of preliminary evaluation of January, 1980 planting of germplasm inoculated in the greenhouse and later transplanted to the field[z]

No.	P.I. No.	Origin	No. plants tested	Disease grade[y] (greenhouse)	Defoliation index[x] (field)	Remarks[w]
157	215737	Chile	12	3.7		
158	215738	Peru	11	3.6		
159	224412	Boli	12	2.8	1.4	
160	224424	Co Ri	12	3.2	1.0	
161	224428	Co Ri	12	3.0		
162	224444	Boli	12	3.5		
163	224445	Boli	12	3.4		
164	224446	Co Ri	12	2.8	3.7	
165	224447	Co Ri	12	4.9	1.2	
166	224448	Co Ri	12	2.4	1.0	
172	Keystone	USA	25	4.4	3.8	
173	Yolo	USA	33	3.8	2.2	
174	260435	Boli	7	1.4	0.8	
175	281423	Pu Ri	5	2.4	1.0	
176	260569	Boli	12	3.2		
178	Gwangju	Korea	8		1.4	HES[v]
179	Namji	Korea	12		1.0	HES
180	Danyang	Korea	11		0.9	HES
181	Masan	Korea	11		1.1	HES
182	Munbong	Korea	8		1.3	HES

Table 34. -- (continued) Results of preliminary evaluation of January, 1980 planting of germplasm inoculated in the greenhouse and later transplanted to the field[z]

No.	P.I. No.	Origin	No. plants tested	Disease grade[y] (greenhouse)	Defoliation index[x] (field)	Remarks[w]
183	Boryung	Korea	11		1.0	HES
184	Sacheon	Korea	8		1.1	HES
185	Choson-gochu	Korea	9		0.9	HES
186	Jochiwon	Korea	9		0.8	HES
187	Jungweon	Korea	12		0.9	HES
188	Jeju	Korea	9		0.8	HES
189	Cheonan	Korea	11		0.5	HES
190	Cheongyong	Korea	11		0.5	HES
191	Pang-ie cho	Korea	10		0.6	HES
192	Hagok No.1	Korea	12		1.1	HES
193	Hwengyeri	Korea	9		1.0	HES
194	Gosung	Korea	2		1.0	HNSC[u]
195	Seodong	Korea	11		1.4	HNSC
196	Anjilbangi	Korea	12		0.0	HNSC
197	China	Korea	10		1.1	HNSC
198	Cheonan	Korea	6		1.0	HNSC
199	Cheongyong	Korea	9		1.3	HNSC
200	Bulam House	Korea	11		0.0	HNSC
201	Serona green	Korea	11		1.1	HNSC

z, y, x, w The same as Table 33.

[v]HES Seeds from Horticultural Experiment Station in Suweon, Korea.

[u]HNSC Seeds from Hung Nong Seed Company in Korea.

Table 35. -- Pungency of selected lines of pepper

Line	P.I. Number	Pungency[z]
4	163192	3
34	224451	10
43	241670	10
47	244670	10
79	271322	10
105	297490	9
110	297495	3
112	308787	10
116	308791	10
119	322719	4
127	369994	10
131	369998	10
137	377688	10
177	163192	10
Yolo Wonder		1
Keystone		1
23-1-7		1

[z]Determined by Ting and Barrons' (1942) chemical test and Van Blarcom and Martin's (1947) pungency scale.

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