



The inheritance of resistance to *Xanthomonas campestris* pv. *translucens* (J.J.R.) Dowson in barley
by Gurbuz Mizrak

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in Plant
Pathology

Montana State University

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Abstract:

Inheritance of resistance to bacterial leaf streak was investigated in 23 barley cultivars. Parents, F1s and F2s of one complete diallel set with six varieties and those of 23 other crosses were tested with one Montana isolate (X-67) of *Xanthomonas campestris* pv. *translucens* in the field. An inoculation method is described which results in a uniform level of infection.

Barley cultivars, Herta, Summit, Oderbrucker, Betzes, Alpine, Luther, CI 12558, CI 12569, CI 12595, CI 12777, CI 12787, CI 12866, CI 13095, PI 382511, PI 382650, PI 382720, PI 382732, and PI 383077 were found to be promising sources for resistance to bacterial leaf streak. Resistance in these cultivars varied with regard to gene action and number. Some of them may have identical genes or different genes in the same chromosomes. Further study is needed to determine identical, linked, and independent genes. Also, a close relation, which suggests a linkage, was found between earliness and susceptibility.

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in

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Bozeman, Montana

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Signature Gürbüz Mızrak

Date March 5, 1985

In dedication to:

My daughters Tuğba and Gaye,
my wife, my father and mother

VITA

Gürbüz Mızrak, son of Hüseyin and Memduha, was born on February 2, 1949 in İnziloglu, Ankara, Turkey. He grew up in İnziloglu where his father farmed.

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ABSTRACT

Inheritance of resistance to bacterial leaf streak was investigated in 23 barley cultivars. Parents, F_1 s and F_2 s of one complete diallel set with six varieties and those of 23 other crosses were tested with one Montana isolate (X-67) of Xanthomonas campestris pv. translucens in the field. An inoculation method is described which result in a uniform level of infection.

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CHAPTER 1

INTRODUCTION

Bacterial leaf streak of cereals is a world wide disease caused by the bacterium, Xanthomonas campestris pv. translucens (J.J.R.) Dowson. In Montana, the disease has increased in irrigated cereals fields in the past few years, and reportedly decreases yields in barley (Hall and Sands, 1983). All commercial barley varieties grown in Montana are susceptible to this disease. Organic mercuric seed treatments apparently controlled the disease for a number of years. After the curtailment of these seed treatments, no effective method has been available to control seed transmission of the disease.

Breeding for resistance is another mechanism to control diseases. In order to implement a breeding program, breeders need resistant germplasm, and a reliable screening test. Bacteria, as with other pathogens, produce new strains which can overcome host resistance, especially if the resistance is determined by single genes in susceptible backgrounds (See Literature Review). Therefore, combining resistant genes from

different sources should be a breeding strategy.

Expression of resistance to bacterial leaf streak is influenced to a great extent by environment (See Literature Review). In many cases, the results of field and greenhouse tests are not the same (Kim, 1982). Plant cultivars resistant in the field may be susceptible in the greenhouse. It has been observed that the variation in reaction of a particular cultivar can be very large even within a small area. The difficulty in creating uniform infection has made the results of screening tests unreliable. For the above reasons, developing an appropriate screening method in the field is necessary.

The inheritance of resistance to bacterial leaf streak in barley is unknown. Previously some barley varieties and lines were screened for disease resistance and some of them were found to be promising as sources of resistance (Stewart, 1952; and Kim, 1982). The goals of this study were to determine the inheritance of resistance to bacterial leaf streak in barley by using reliable screening techniques, to find different sources of resistance, and to devise a breeding plan for developing resistant barley varieties to bacterial leaf streak. It is hoped that a long-lasting resistance to

bacterial leaf streak in barley might be developed by combining resistance from various sources.

CHAPTER 2

LITERATURE REVIEW

Bacterial leaf streak of grasses caused by the bacterium, Xanthomonas campestris pv. translucens (J.J.R.) Dawson, has been observed since the late 1800s (Mathre, 1982). The earliest report of this disease on barley came from Madison, Wisconsin in 1912 where it appeared as an unusual leaf blight in a plot of Montana two-row barley and subsequently spread to other varieties.

This disease was first described by Jones et al. (1917). They reported bacterial leaf streak of barley in Colorado, Iowa, Minnesota, Montana, North Dakota, South Dakota, Oregon, and Wisconsin. It was found in Nebraska (Pady, 1944), in Northern Europe and Asia (Jaczewski, 1935). Bacterial leaf streak is now known to occur world wide on barley, wheat, rye, triticale, and numerous grasses (Wallin, 1946; Koleva, 1981; Leyns et al., 1981; Egli and Schmidt, 1982; Mathre, 1982; and Miyajima, 1982). The disease has become prevalent in sprinkler irrigated barley and wheat fields of Montana within the past few years (Hall and Sands, 1983).

Early reports showed that local and general epiphytotics on susceptible cultivars reduced yield (Dickson, 1956). Commonly, bacterial leaf streak in barley reduces yield by 10-15% (Mathre, 1982; Hall and Sands, 1983). Yield reduction of severely infected wheat fields has been estimated to be as high as 50% (Hagborg, 1968).

Symptoms

The first symptoms on barley appear on the young green leaf blade and sheath as small, water-soaked lesions that later become light-brown and dark-brown regions. These lesions tend to develop as longitudinal streaks between the veins, then coalesce into narrow glossy-surfaced stripes extending the entire length of the leaf (Zilinsky, 1983). In the later stage of development, the lesions show a characteristic translucency (Dickson, 1956). A bacterial exudate is usually present on the lesions. Under humid conditions, this exudate may appear as numerous tiny cream-like to yellow droplets giving the leaf surface a shellacked appearance. The appearance of exudate is a key characteristic in distinguishing this disease from net blotch, barley stripe, and barley stripe mosaic (Mathre, 1982). Under dry conditions the exudate quickly hardens

into yellow granular beads or glistening scales easily detached and readily softened or dissolved in water (Jones et al., 1917).

Similar lesions develop on the floral bracts and are termed "black chaff". The entire head can turn light to very dark brown. Severe late infection usually results in retarded spike elongation and blighting of the spike and adjacent tissues. An infected head may be bent and distorted (Dickson, 1956). Bacterial growth can often be seen as shellac on the surface of the seeds and a portion of the grain may be blighted (Hall and Sands, 1983).

El-Banoby and Rudolph (1979) reported that persistent water-soaked leaf spots were induced in susceptible cultivars of barley and wheat by treatment with preparations of extracellular polysaccharides (ESP) from Xanthomonas campestris pv. translucens f.sp. cerealis.

The Bacterium

Xanthomonas campestris pv. translucens (J.J.R.) Dowson, the causal organism of bacterial leaf streak of barley, belongs to the genus Xanthomonas. According to Dye (1980), the genus Xanthomonas is defined as follows: Gram negative rods 0.4 x 1.0 um, single polar flagellum,

aerobic, catalase positive, H_2S positive, oxidase negative, Tween 80 hydrolysis, nitrate not produced, and indole not produced. All Xanthomonas species recognized at present are plant pathogens and, so far as known, are found only in association with plants or plant material. Most xanthomonads produce the extracellular polysaccharide slime "gum-xanthan" on media containing glucose. Colonies of these species on nutrient agar or YDC agar are mucoid, convex, and shining.

The five nomenspecies of the genus Xanthomonas are X. campestris, X. fragariae, X. albilineans, X. axanopodis, and X. ampelina. X. campestris can be differentiated from the others by its positive test for protein digestion. The bacteria in this nomenspecies can be identified by growth characteristics on SX agar and by pathogenicity tests (Dye, 1980). X. campestris pv. translucens can not grow on SX agar and its strains are pathogenic to barley, wheat, rye, triticales, oats, rice and some other grasses (Smith et al., 1919; Reddy et al., 1924; Hagborg, 1942; Fang et al., 1950; Patel and Shekhawat, 1971; Dye, 1980; Egli and Schmidt, 1982; and Leyns et al. 1984). Previously, eight form species of X.c. pv. translucens have been described according to their host specificity (Table 1).

Table 1. The form species of Xanthomonas campestris pv. translucens and their hosts.

Form species	Hosts	References
<u>Xct</u> f.sp. <u>undulosa</u>	wheat, spelt, barley and rye	Smith, Jones and Reddy, 1919.
<u>Xct</u> f.sp. <u>secalis</u>	rye	Reddy, Godkin and Johnson, 1924.
<u>Xct</u> f.sp. <u>hordei</u>	barley and rice	Hagborg, 1942; Patel and Shekhawat, 1971.
<u>Xct</u> f.sp. <u>hordei-</u> <u>avenae</u>	barley and oats	Hagborg, 1942.
<u>Xct</u> f.sp. <u>cerealis</u>	wheat, oats, barley, rye smooth brome grass, quack grass, Italian rye grass, perennial rye grass, and meadow fescue	Hagborg, 1942; Fang et al., 1950; Egli and Schmit, 1982.
<u>Xct</u> f.sp. <u>phlei</u>	<u>Phleum</u> sp.	Wallin and Reddy, 1945; Egli and Schmidt, 1982.
<u>Xct</u> f.sp. <u>poae</u>	<u>Poa</u> sp.	Egli and Schmidt, 1982.
<u>Xct</u> f.sp. <u>arrhenatheri</u>	<u>Arrhenatherum</u> sp.	Egli and Schmidt, 1982
<u>Xct</u> = <u>Xanthomonas campestris</u> pv. <u>translucens</u>		

Host Range

According to Schuster and Coyne (1974), bacterial pathogens with wide host ranges can survive between crop seasons on alternate hosts which provide a bridge during off-seasons. Such pathogens are not handicapped by discontinuous plant growth unlike the parasites that have narrow host ranges.

Boosalis (1952) investigated the overwintering of Xanthomonas campestris pv. translucens f. sp. cerealis on infected quack grass. It was pathogenic to wheat, oats, barley and rye. This example shows the importance of disease transmission from wild flora.

Previous studies show that the Xanthomonas campestris pv. translucens group has 23 different hosts in the Gramineae. These include, barley, wheat, rye, oats, triticale, rice, foxtail barley, spelt, einkorn, smooth brome, cheat grass brome, yellow bristle grass, quack grass, orchard grass, water foxtail, reed canary grass, Japanese millet, Phleum sp., Poa sp., Arrhenatherum sp., Italian rye-grass, perennial rye-grass and meadow fescue (Smith et al., 1919; Reddy et al., 1924; Hagborg, 1942; Wallin and Reddy, 1945; Fang et al., 1950; Boosalis, 1952; Patel and Shekhawat, 1971; Leyns et al., 1981; Cunfer and Scolari, 1982; Egli and Schmidt,

1982; Kim, 1982; and Leyns et al. 1984).

Barley seems to be susceptible to most of the form species of *X.c.* pv. translucens (Table 1). Hall et al. (1981) reported that the isolates from barley, wheat and rye in irrigated cereal fields of Montana were pathogenic on barley. Cunfer and Scolari (1982) found that strains of *X. c.* pv. translucens from triticales were equally virulent to triticales, wheat and rye, but much less to barley. They reported that strains from wheat and rye were usually most virulent on the host from which they were isolated, however, only one cultivar of each host species was tested. Strains from barley were restricted in host range, being pathogenic primarily to barley.

The form species hordei was originally reported to be pathogenic only to barley (Hagborg, 1942). However, Patel and Shekhawat (1971) showed that rice was susceptible to *X. c.* pv. t. f.sp. hordei at the 20 and 40 day old stages. According to Fang et al. (1950), *X. c.* pv. t. f.sp. cerealis occurs naturally on smooth brome grass, quack grass, and produces water-soaked streaking symptoms on barley following wound inoculation, but it is more virulent on grasses.

Bamberg (1936) found that strains of *X. c.* pv. translucens differ in pathogenicity. According to Hagborg

(1942), different isolates of the same form species of X. o. pv. translucens are different in pathogenic capabilities and he suggested the need for recognizing races within this special form.

Etiology and Epidemiology

Bacterial leaf streak is a widely occurring disease affecting leaves, leaf sheaths, and glumes. The bacterium infects leaves through stomata and wounds, spreading intercellularly (Jones et al., 1917 and Bamberg, 1936). The bacterial invasion is confined to the thin-walled parenchyma possessing intercellular spaces, i.e., mesophyll of leaf, chlorenchyma and ground tissue parenchyma of the leaf sheath. In naturally infected leaves no vascular bundle elements were shown to be infected (Sheakhavet and Patel, 1978).

The seed-born nature of this disease was reported by Jones et al.(1916) and Smith et al.(1919). The bacterium penetrates the pericarp by causing infection of the plumula through wounds or stomata on the coleoptile. Spreading rapidly through these tissues, it finally reaches the enclosed foliage leaves. By elongation, the infected leaf develops the water-soaked streaks on the primary leaf (Wallen, 1946).

According to Boosalis (1952), barley plants from

infected seeds planted in sterilized soil showed a higher percentage of infection than those planted in nonsterilized soil. He found that seeds of barley, wheat and rye inoculated with X. translucens alone resulted in a few infected seedlings, whereas 25 to 40 percent of seedlings from seeds co-inoculated both with a root rot fungus (Helminthosporium sp.) and X. translucens succumbed to Xanthomonas streak. Other than seed transmission, this bacterium may persist from one season to the next on straw of barley, wheat, some other grasses, and on winter hosts.

Hedrick(1956) and Leach et al.(1957) thought that bacterial exudates behave as a hydrophilic colloid. Its high waterholding capacity may aid bacterial survival during unfavorable conditions and seasons. According to Schuster and Coyne (1974), maintenance under dry conditions commonly favors bacterial survival in plant residues. Residues on or near the soil surface are more favorable to bacterial survival than those incorporated in soil. The bacteria in dry, undecayed residues are protected from antagonistic microflora which may require moisture for movement. Probably for the above reason, X. c. pv. translucens is very tolerant to dry conditions. Jones et al.(1917) reported that the bacterium survived

for eight months on straw from blighted plants and two years on seeds. According to Boosalis (1952), X. c. pv. translucens was not able to survive long in soil kept at 27° C and in the field during the winter months unless the soil was sterile and asepsis was maintained. Bamberg (1936) reported that the bacterium survived 77 days in dry soil at St. Paul, Minnesota. He found that this organism can survive at extremely low temperatures ranging to -33° C, remaining viable in soil cultures for at least 124 days after December 15.

Bamberg (1936) found that low relative humidity retarded the growth of the bacterium in culture. The retarding effect was noticeable at 50 percent relative humidity and became greater as the R.H. decreased. Optimum temperature for the growth of this organism on agar was between $25-30^{\circ}$ C. The maximum temperature for sustained growth was approximately 40° C with the minimum being slightly below 10° C.

Severe epidemics occur in wet seasons with the optimum temperature around 20° C (Kim, 1982). According to Bamberg (1936), temperature had a marked effect on the length of the incubation period and disease development. At 20° C from two to seven days were necessary for incubation, while at 10° C from eight to 20 days were

required. Disease development was slowed even more at or below 10° C. Stewart (1952) reported that the highest infection on susceptible plants occurred at 15.5 to 18° C, moderate infection at 21° C, slight infection at 24° C, and no infection at 27° C, an optimum temperature for growth in culture. According to Kim (1982), symptom development was influenced by temperature. Symptoms did not appear under the 15.5° C day and 4.5° C night regime. Under the 21.1° C day and 12.8° C night, and 32° C day and 1.8° C night regimes, symptoms developed. Bamberg (1936) reported that for disease development, low relative humidity is more often a limiting factor than low temperature.

Effective transmission from plant to plant by aphids, wind, rain and by direct plant to plant contact, occurs only when the host is water-congested (Boosalis, 1952). Field epidemiology studies in Montana using a marked strain of *X. c. pv translucens* f. sp. *hordei* indicated that this bacterium is capable of spreading 28 square meters from a single infection locus within 39 days (Hall et al, 1981).

Selective Media

Selective media that have been developed for different xanthomonads are not efficient for isolating

X. campestris pv. translucens (Kim, 1982). Wilbrink's agar, not selective for the bacterium, is generally used for isolation and maintenance. Kim (1982) developed the KM-1 medium for isolation of X. campestris pv. translucens. This medium contains 10 g of lactose (Sigma Chemical Co., St. Louis, MO.), 4.0 g of D(+) trehalose (Sigma), 0.2 g of thiobarbituric acid (Sigma), 0.8 g of K_2HPO_4 and KH_2PO_4 (Sigma) respectively, 0.03 g of yeast extract (Difco Laboratories, Detroit), 1 g of NH_4Cl (Mallinckroft Chemical Works, St. Louis, MO.) and 15 g of Bacto agar per liter of double distilled water. Before adding agar, the ingredients are dissolved completely on a hot plate with stirring bar and pH is adjusted to 6.6 with 1 N NaOH solution. After autoclaving these ingredients, cycloheximide (Sigma), dissolved in ethanol, tobramycin (Sigma), dissolved in ethanol-water (1:1, v/v), and ampicillin (Sigma), dissolved in ethanol-water (1:1, v/v) with addition of 1 pellet of sodium hydroxide (Mallinckroft Inc., Paris, Kentucky) are added aseptically to final concentration of 100 $\mu g/ml$, 8 $\mu g/ml$ and 1 $\mu g/ml$, respectively. Kim (1982) noted that the medium provides a sensitive and reliable detection of the bacterium. Other Xanthomonas spp. grow on the KM-1 medium, but have not been found in cereal grains. He

also added that this medium had high selectivity against soil-born plant pathogenic bacteria.

Schaad and Forster (1984) reported that many strains of X. c. pv. translucens from Idaho grew poorly on KM-1 agar when used for isolation of the bacterium from seeds. They developed a semiselective medium, XTS agar, for isolation of X. c. pv. translucens from seeds. XTS agar contains 23 g of nutrient agar, 5 g of glucose, 200 µg of cycloheximide, 100 µg gentamycin, and 8 µg of cephalixin in one liter. They reported that 91% of the saprophytic bacteria associated with wheat and barley seeds did not grow on XTS agar.

Screening for Disease Resistance

Most of the early attempts to select plants for resistance to bacterial diseases were carried out in locations where natural epidemics occur. However, it is necessary to inoculate test plants artificially to obtain a uniform disease epidemic (Russell, 1978). This is necessary so that different breeding lines and genotypes can be accurately compared for disease reaction. Historically, the following techniques have been used to inoculate barley plants with X. c. pv. translucens:

1. Spraying bacterial suspension (Jones et al., 1917)

2. Partial vacuum technique (Boosalis, 1950).

3. Injection of bacterial suspension by hypodermic syringe (Bamberg, 1936; and Hagborg, 1936).

4. Seed inoculation by soaking seed in bacterial suspension for one hour (Wallin, 1946).

5. Seedling inoculation by poking the seedling with a sharp nichrome needle and flooding with bacterial suspension (Hagborg, 1936).

6. Mowing off the top of the barley plants in the evening, followed by immediate spraying with bacterial suspension, and sprinkler irrigation at a 3 day interval (Kim, 1982).

Kim (1982) suggested that the most effective methods were: Injecting the inoculum into the leaf of a seedling or into the leaf sheath of an older plant with a hypodermic syringe (#3), partial vacuum technique (#2), and his method (#6).

Previous studies revealed that there are resistant sources in barley to bacterial leaf streak. Jones et al. (1917) found that Chevalier, California, Summit (six-rowed and different from the cultivar recently released) and Oderbrucker (CI. 4666) were the most resistant cultivars of 53 tested barley cultivars. Stewart (1952) reported that the barley cultivars, Spartan, CI 6240, Velvon, and

some other lines showed high degrees of field resistance. Patel and Shekhawat (1971) tested 51 barley cultivars in the seedling stage for reaction to X. c. pv. translucens f. sp. hordei. They found no complete resistance, but variations in the degree of susceptibility were observed.

Kim (1982) screened 54 commercial varieties, and 722 lines from the World Barley Collection for resistance to bacterial leaf streak. Barley varieties, Herta, Summit(MT 729), Oderbrucker, Alpine, Luther, Betzes, Wabet, and Shabet were found to be resistant. He also found 21 resistant lines, mostly from Ethiopia in the USDA World Barley Collection.

Screening tests for bacterial diseases carried out under natural conditions generally reflect the effectiveness of field resistance more accurately than greenhouse or laboratory tests (Russell, 1978). Previous studies showed that there is usually no correlation between field and greenhouse tests for resistance to bacterial leaf streak on cereals. Bamberg (1936) reported that the wheat varieties, Marquis, Mindum and Kubanka appeared resistant in the field, yet were rather heavily infected when hypodermically inoculated. Stewart (1952) noted that some barley varieties were quite resistant in the field, but were susceptible in the greenhouse. Kim

(1982) did not find any significant correlation between results in the growth chamber and the field.

Mechanism of Resistance

The mechanism of resistance in barley to bacterial leaf streak is unknown. Generally, there are two types of resistance mechanisms to bacterial diseases: (i) Preformed resistance, and (ii) induced resistance. Preformed resistance involves the mechanisms which inhibit bacterial penetration into intercellular spaces of the plant (Klement, 1972). Pathogenic bacteria penetrate their host plants through natural openings (stomata or lenticels) or wounds. Russell (1978) suggested that the concentration, size and morphology of stomata or lenticels as well as their distribution on the surface of the host plant, which are genetically controlled characteristics, may influence the host susceptibility to bacterial infection.

According to Klement (1972), the fluid in intercellular spaces of plants contains, in abundance, all nutrients necessary for most pathogenic and saprophytic bacteria, when cultivated in vitro. But inside the living plant only the host specific bacteria are able to grow. Sasser et al. (1970) reported that even though bacteria enter the host plant, the osmotic

potential in the intercellular leaf fluid is a controlling factor in the development of disease. The dry outside air normally inhibits the population increase of pathogenic bacteria. A period of high relative humidity and rainfall reduce the osmotic potential and allow bacterial multiplication within the host plant.

Garber (1961) suggested that buffering capacity of plant fluids affects resistance to bacterial diseases. As a response to bacterial infection, the pH of plant sap may change and affects the formation, penetration and effectiveness of enzymes produced by bacterial pathogens. He also found that the nutrients necessary for the pathogen may be absent from the resistant host tissues. Some antibacterial substances in plant tissue may inhibit bacterial multiplication (Garber, 1961; Hildebrand and Sands, 1966; and Klement, 1972).

In contrast to preformed resistance, induced resistance includes defense mechanisms that start in plants as a result of infection. There are two types of induced defense reaction : (i) Preimmunity, and (ii) hypersensitivity. According to Klement and Goodman (1967), preimmunity is regarded as a nonspecific acquired immunity which manifests itself in plants pretreated (inoculated) with one bacterium that immunizes or

protects the plant from postinfection by another bacterial pathogen. Goodman (1980) noted that two different defense mechanisms are elicited for preimmunity. The first may reflect a short-lived release of antagonistic substances. The second resistance mechanism may be caused by host cell activity.

Klement (1972) defines the hypersensitive reaction as an incompatible reaction between particular host and bacterial genotypes. Following the penetration of pathogenic bacteria into the substomatal cavities, a few host cells die around the bacteria and this confines the infection so that the plant remains practically free from symptoms. Hypersensitivity seems to be associated with race specificity breakdown.

Mew et al. (1984) found that Xanthomonas campestris pv. oryzae on rice multiplied outside the water pores of the leaf and some bacteria entered the leaves through these pores. However, a strain that lost its virulence, did not multiply significantly on the leaf surface. They suggested that nonpathogenic bacteria were immobilized and inhibited from dividing by excretions from water pores.

Many factors are involved in the complicated mechanisms of resistance to bacterial diseases in plants

and much research is still needed to understand the mechanical and molecular processes involved. Bacteria have unique advantages compared to plants, such as, the ability to multiply very rapidly, to produce new virulent strains, and thus, to overcome host resistance.

Inheritance of Resistance

Inheritance of resistance in barley to bacterial leaf streak is obscure. A lengthy literature search failed to find a single citation or reference.

The genetics of resistance to bacterial diseases follows no set pattern. Resistance to some bacterial diseases varies considerably according to location and environment. Different authors have reached conflicting conclusions about the genetic control of resistance to the same diseases. Some workers have reported that the resistance to Xanthomonas campestris pv. oryzae in rice is monogenically controlled while others indicated that it is polygenic. The resistance to the disease has also been reported by different workers to be either as a dominant or a recessive characteristic and race-specific or nonspecific (Coyne et al., 1971; Murty and Khush, 1971; Ou, 1973; Ezuka et al., 1975; Yamamoto et al., 1977; Panda and Chaudhary, 1978; Webster et al., 1983).

These results suggest that both pathogens and hosts

have great variability, and the genetics of resistance depends not only on the genotypes of the host and pathogen involving polygenic systems, but also on the environmental conditions in which these genotypes are grown (Russell, 1978).

Many cases have been found where reciprocal hybrids differ from one other and resemble their respective maternal parents in some phenotypic characteristics due to cytoplasmic inheritance. In the case of disease resistance, southern corn leaf blight disease is a good example for cytoplasmic factors. All corn hybrids containing the Texas cytoplasmic male sterility gene were heavily infected by the disease incited by Helminthosporium maydis in 1970 in the U.S.A. (Ullstrup, 1972). Therefore, reciprocal crosses might be necessary to detect cytoplasmic factors.

Vanderplank (1982) reported that the introduction of resistance to bacterial diseases into host plants results in a change toward more frequent virulence in the pathogen. According to Brinkerhoff (1970), the resistance conditioned by a few genes in cotton to bacterial blight was often unstable. In the United States, the resistance of cotton varieties with single B genes in a susceptible background was matched in a season by increased virulence

in Xanthomonas campestris pv. malvacearum. According to Vanderplank (1982), cotton breeders seem to have succeeded in synthesizing a high level of resistance that now appears to be stable.

Control

According to Jones et al. (1917), X. c. pv. translucens could be eliminated by treating barley seeds for two hours with formaldehyde solution. Nuncie (1938) reported that Ceresan at 1/2 ounce per bushel or seed, controlled the disease. The pathogen is very sensitive to mercury compounds; therefore seed treatment with organic mercury compounds has been used successfully in controlling the disease (Dickson, 1956). However, the use of organic mercury compounds has been prohibited due to safety considerations. Goodman and Henry (1947) found that subtilin diluted 1:1000 provided adequate control of the disease. Koleva (1981) reported that hot water (at 53° C for 30 min.), 0.1 phytobacteriomycin (2 liter/100 Kg seed), quinolate (200 gr/100 Kg seed) and Vitavax (200 gr/100 Kg seed) were found to be effective seed treatments.

Kim (1982) reported that the bactericide, cupric hydroxide, had been registered and recommended for seed treatment without evidence of complete control. According

to Mathre (1982), copper hydroxide could reduce seed-born inoculum.

The use of pathogen free seed produced under dryland conditions, crop rotation, the destruction of perennial weeds susceptible to the pathogen, and destroying infested plant debris are recommended as the best control measures (Jones et al., 1917; Boosalis, 1952; and Mathre, 1982).

Mathre (1982) reported that no resistant cultivars are known, although some are more susceptible than others.

CHAPTER 3

MATERIALS AND METHODS

Materials

Twenty-three barley cultivars were used as parents in the crossing program (Table 2). Of these cultivars CM 67, Kangbori, and Suweon 191 (SW 191) are very susceptible to the disease, and the remainder have some resistance (Kim, 1982).

In this study, three different groups or crosses were tested with one Montana barley isolate, X-67, in the field: (1) a complete diallel set, (2) other resistant x susceptible crosses, and (3) additional resistant x resistant crosses.

Crosses with six parents were made in a complete diallel mating design to fully investigate gene actions in these cultivars. The parents used in the diallel set were Herta, Summit, Oderbrucker, Betzes, CM 67 and SW 191 (Table 3). The first four cultivars have some resistance to bacterial leaf streak, and the last two are very susceptible to the disease (Kim, 1982). The cultivars selected as parents for the diallel set were chosen

Table 2. Barley cultivars used as parents in the crossing program

Cultivars	Number	Origin
Herta	CI 8097	Sweden
Summit	MT 729	England
Oderbrucker	CI 4666	Germany
Betzes	CI 6398	Poland
CM 67(*)	CI 13782	USA
Suweon 191 (*)		S. Korea
Kangbori (*)		"
Alpine	CI 9578	USA
Luther		"
	CI 12558	Ethiopia
	CI 12569	"
	CI 12577	"
	CI 12595	"
	CI 12776	Sweden
	CI 12777	"
	CI 12787	Ethiopia
	CI 12866	"
	CI 13095	"
	PI 382511	"
	PI 382650	"
	PI 382720	"
	PI 382732	"
	PI 383077	"

(*): Susceptible varieties

because they have good agronomic types with the exception of Oderbrucker. Oderbrucker has been reported to be resistant since 1917 (Jones et al., 1917; and Kim, 1982). All possible crosses among these cultivars were made and inheritance of the disease resistance was studied in detail.

Table 3. Crosses made for the diallel set and their parents, F₁s and F₂s which were evaluated

Cross	Parents	F ₁ s	F ₂ s
Herta x Summit	+	+	+
" x Oderbrucker	+	+	+
" x Betzes	+	+	+
" x CM 67	+	+	+
" x SW 191	+	-	+
Summit x Herta	+	+	+
" x Oderbrucker	+	+	+
" x Betzes	+	+	+
" x CM 67	+	+	+
" x SW 191	+	+	+
Oderbrucker x Herta	+	+	+
" x Summit	+	+	+
" x Betzes	+	+	+
" x CM 67	+	+	+
" x SW 191	+	+	+
Betzes x Herta	+	+	+
" x Summit	+	+	+
" x Oderbrucker	+	+	+
" x CM 67	+	+	+
" x SW 191	+	+	+
CM 67 x Herta	+	+	+
" x Summit	+	-	+
" x Oderbrucker	+	+	+
" x Betzes	+	-	+
" x SW 191	+	+	+
SW 191 x Herta	+	-	+
" x Summit	+	+	+
" x Oderbrucker	+	+	+
" x Betzes	+	+	+
" x CM 67	+	+	+

SW 191: Suweon 191

+: Evaluated

-: Not evaluated

In the second group, eighteen crosses were made between other promising resistant cultivars and one of

the susceptible varieties to detect whether the resistant cultivars had heritable resistance to the disease (Table 4).

In the third group, resistant cultivars from different origins were crossed with each other to determine if they had different genes for resistance (Table 4).

F₁s were grown in the greenhouse during the winter of 1983-84 and checked phenotypically to determine if they were true F₁s or selfed. Selfed plants were eliminated.

Parents, F₁s and F₂s were space planted at the Horticulture Farm near Bozeman, Montana on 21 May 1984. A serpentine planting design was used. Individual plants were spaced 15 cm apart in rows 3 m in length and 30 cm apart (20 seeds/row). Twenty seeds of each parent and F₁ were planted. For each F₂, 120 seeds were planted. One row of CM 67 was planted between every two rows as the spreader to enhance uniform infection (Figure 1).

Inoculation

Artificial inoculation was carried out with a strain (X-67) of *X. c.* pv. *translucens*, highly virulent to barley. This isolate was obtained from an irrigated barley field at Sidney, Montana, where the natural

Table 4. Crosses made in the resistant x susceptible and resistant x resistant categories and their parents, F₁s, and F₂s which were evaluated.

Cross	Parents	F ₁	F ₂
Alpine x Herta (#)	+	+	+
" x Oderbrucker (#)	+	+	+
" x Kangbori	+	+	+
Luther x Herta (#)	+	+	+
" x Summit (#)	+	-	+
" x Oderbrucker (#)	+	-	+
" x Kangbori	+	+	+
CI 12558 x CM67	+	+	+
CI 12569 x Kangbori	+	+	+
CI 12577 x "	+	+	+
CI 12595 x "	+	-	+
CI 12776 x "	+	+	+
CI 12777 x "	+	+	+
" x CM 67	+	+	+
CI 12787 x SW 191	+	+	+
CI 12866 x CM 67	+	-	+
" x Kangbori	+	+	+
CI 13095 x SW 191	+	-	+
PI 382511 x "	+	+	+
PI 382650 x "	+	+	+
PI 382720 x Kangbori	+	+	+
PI 382732 x SW 191	+	-	+
PI 383077 x "	+	-	+

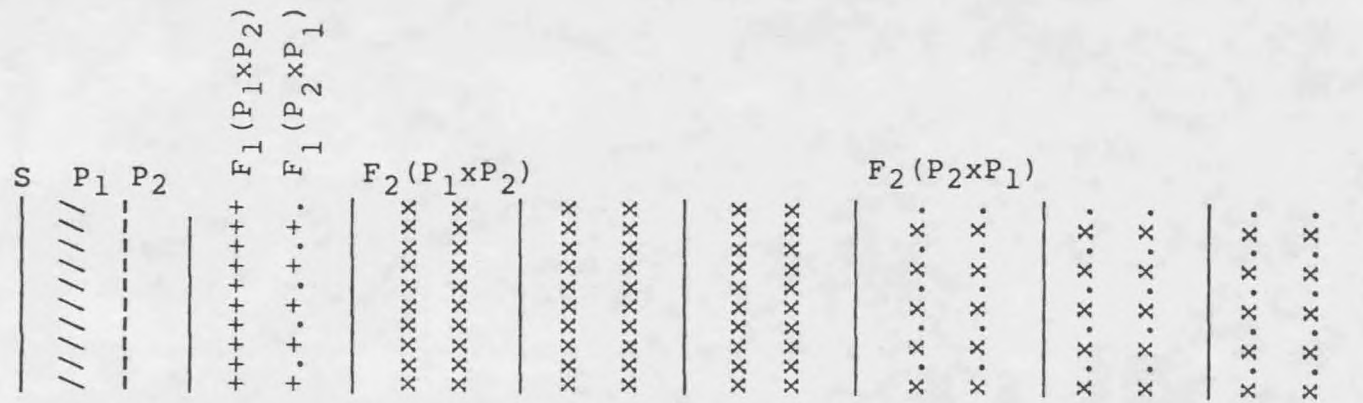
+: evaluated

-: not evaluated

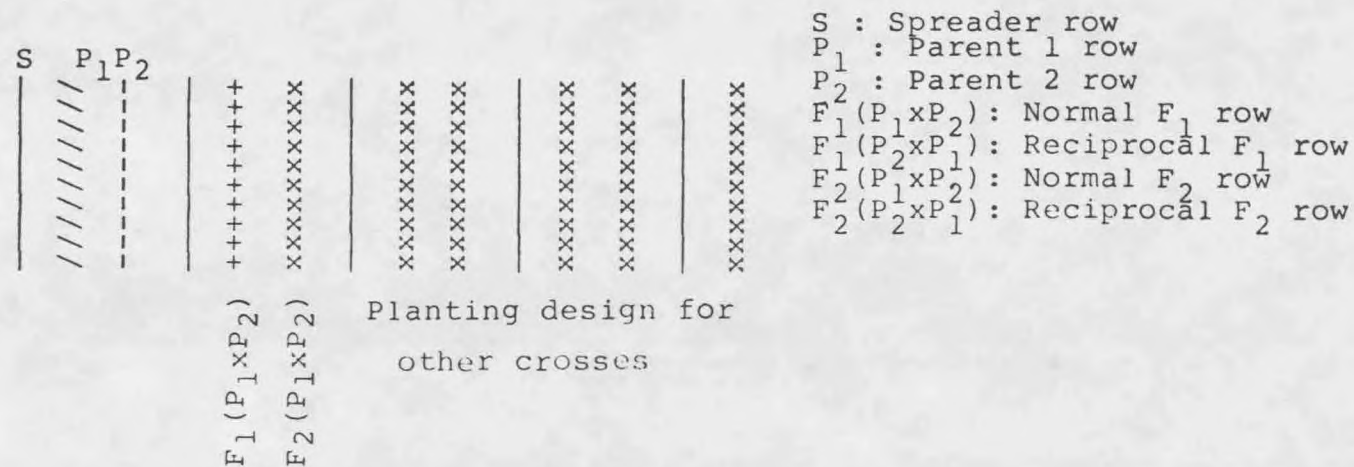
(#): resistant x resistant crosses

infection rate of bacterial leaf streak is usually very high.

The bacterium was cultured on Wilbrink's agar in an incubation chamber at 28° C for 3 days and then



Planting design for a cross of the diallel set



Planting design for
other crosses

Figure 1. Planting design of field test, Horticulture Farm, Bozeman, 1984.

transferred to liquid Wilbrink's medium. The culture was shaken at room temperature for 36 hours. Before inoculation, the bacterial suspension was diluted 1:3 with double distilled water.

Plants were first inoculated at the tillering stage. Inoculation was carried out in the early evening. All entries were sprinkler irrigated prior to inoculation for one hour to obtain a humid condition. The plants of CM 67 in the spreader rows, were wounded by mowing to a height of 10 cm. All entries were then inoculated by spraying the bacterial suspension, using a hand pump tank sprayer. The second inoculation was performed ten days after the first one, without wounding the plants. One week after the second inoculation, the third one was performed, using a bacterial suspension prepared by cutting up infected leaves from the spreader rows and soaking for two hours in water. Plots were sprinkler irrigated at three day intervals to keep conditions favorable for disease development up to the time for disease scoring.

Disease Scoring

The disease reactions were rated when the spreader rows were highly infected. Fourteen F_2 populations having an early, susceptible parent and a late, resistant parent, were chosen to determine the relation between

earliness and susceptibility. In these populations, the plants heading at the same time as the early parents were marked and their disease reaction recorded.

The following index, partly based on Kim's scale (Kim, 1982), was used to classify the plants for disease reaction:

- 1: No distinguishable symptoms.
- 2: Short streak symptoms on bottom leaves.
- 3: Medium long streak symptoms on all leaves except flag leaves.
- 4: Streak symptom on flag leaves, but less than 10 of area infected.
- 5: 10 percent of flag leaves infected.
- 6: 30 percent of flag leaves infected.
- 7: 50 percent of flag leaves infected.
- 8: 70 percent of flag leaves infected.
- 9: 90 percent of flag leaves infected.

The most highly infected flag leaf of every individual plant was rated for disease.

Statistical Procedures

The following statistical procedures were used to evaluate data:

1. Parent and F_1 variations for disease reaction were considered as environmental variations. Each plant

in the parental and F_1 rows was read and standard deviations were calculated. In the ranges of the resistant and susceptible parent means $\pm$ twice standard deviations were assumed as the ranges of resistant and susceptible classes. The reaction range of the F_1 was used to detect the individuals having the F_1 genotype in F_2 populations.

2. For the crosses which had F_1 s, the degrees of dominance were calculated (Li, 1955).

3. Chi-square tests were used to determine if the observed classes fit a hypothetical genetic ratio.

4. Homogeneity tests were done to compare segregations in F_2 populations of the crosses with one common parent.

CHAPTER 4

RESULTS

Diallel Set

The complete diallel set with six parents, their F_1 s and F_2 s was studied in three different groups: (1) Resistant x resistant crosses, (2) susceptible x susceptible crosses, and (3) resistant x susceptible crosses.

Resistant x Resistant Crosses

Disease reaction of four resistant parents (Herta, Summit, Oderbrucker and Betzes) and that of their F_1 s and F_2 s are shown in Table 5 and 6. The four resistant parents have statistically the same level of resistance to isolate X-67. Their F_1 s and F_2 s, including reciprocals, were found to have statistically the same level of resistance as their parents; thus no segregation was detected.

Susceptible x Susceptible Crosses

Both susceptible parents (CM 67 and SW 191) statistically had the same level of susceptibility to bacterial leaf streak (Table 5). Their F_1 s and F_2 s,

Table 5. Disease reactions of parents in the diallel set.

Cultivar	disease reaction	Cultivar	diseaes reaction
Herta	2.7 $\pm$ 1.4	Betzes	3.0 $\pm$ 1.2
Summit	2.9 $\pm$ 1.3	CM 67	7.6 $\pm$ 1.4
Oderbrucker	2.9 $\pm$ 1.3	SW 191	7.7 $\pm$ 1.6

including reciprocals also were not statistically different from their parents. The F_2 populations apparently did not segregate (Table 6).

Resistant x Susceptible Crosses

These crosses, between four resistant parents (Herta, Summit, Oderbrucker and Betzes) and two susceptible ones (CM 67 and SW 191) were catagorized in two groups according to their susceptible parents: (1) CM 67 crosses and (2) SW 191 crosses (Table 7, 8, 9, and 10; Figure II).

CM 67 Crosses

Herta x CM 67

The F_1 disease reaction of this cross fell between an intermediate and susceptible reaction, and the degree of dominance was calculated as 0.47. This indicated that susceptibility is probably incompletely dominant. No significant reciprocal differences were found in F_1 s and F_2 s. The F_2 populations segregated continuously, appearing

Table 6. Disease reactions of F_1 s and F_2 s for the resistant x resistant and susceptible x susceptible crosses in the diallel set.

Gen.	Cross	Normal	Reciprocal	Both
F_1	Herta x Summit	3.4 $\pm$ 1.2	3.3 $\pm$ 1.2	3.3 $\pm$ 1.2
	Herta x Oderbrucker	2.3 $\pm$ 1.2	2.3 $\pm$ 1.5	2.3 $\pm$ 1.3
	Herta x Betzes	2.1 $\pm$ 1.4	2.1 $\pm$ 1.4	2.1 $\pm$ 1.4
	Summit x Oderbrucker	2.5 $\pm$ 1.5	2.6 $\pm$ 1.5	2.5 $\pm$ 1.4
	Summit x Betzes	2.9 $\pm$ 0.7	3.0 $\pm$ 1.0	2.9 $\pm$ 0.8
	Oderbrucker x Betzes	2.9 $\pm$ 0.6	2.7 $\pm$ 1.0	2.8 $\pm$ 0.7
	CM 67 x SW 191 (*)	7.7 $\pm$ 1.0	7.7 $\pm$ 1.2	7.7 $\pm$ 1.0
F_2	Herta x Summit	2.7 $\pm$ 1.2	2.9 $\pm$ 1.3	2.8 $\pm$ 1.3
	Herta x Oderbrucker	2.0 $\pm$ 1.2	1.8 $\pm$ 1.3	1.9 $\pm$ 1.3
	Herta x Betzes	2.0 $\pm$ 1.6	2.4 $\pm$ 1.4	2.2 $\pm$ 1.5
	Summit x Oderbrucker	2.0 $\pm$ 1.4	2.5 $\pm$ 1.3	2.3 $\pm$ 1.4
	Summit x Betzes	3.0 $\pm$ 1.0	2.9 $\pm$ 1.0	3.0 $\pm$ 1.0
	Oderbrucker x Betzes	3.1 $\pm$ 1.0	3.1 $\pm$ 1.1	3.1 $\pm$ 1.1
	CM 67 x SW 191 (*)	7.7 $\pm$ 1.2	7.7 $\pm$ 1.3	7.7 $\pm$ 1.2

(*): Susceptible x susceptible crosses

as normal curves slightly skewed to the susceptible side. From this result, it can be concluded that Herta probably has polygenic resistance to the disease.

Summit x CM 67

No significant reciprocal difference was found in F_2 s. The F_1 showed a susceptible reaction. The degree of dominance for this cross was estimated as 0.84 which indicates that susceptibility is very close to complete dominance. Since the F_1 value was partly in the susceptible parent class, it was not possible to differentiate individuals in the F_2 population having the F_1 genotype from those of the susceptible parent. For this reason, the individuals in the 6-8 classes were considered as one group and the individuals in the 2-4 classes, which were in the resistant parent class, were considered as another group.

Segregation in the F_2 population fit a ratio of 1:3 with a probability of 0.20-0.25. This indicated that Summit may have one recessive gene for resistance to the disease.

Oderbrucker x CM 67

In the F_1 s and F_2 s, no significant reciprocal differences were found. F_1 s of this cross had a susceptible reaction and the degree of the dominance was calculated as 0.84. Thus, susceptibility is probably almost completely dominant. Segregation in the F_2 fit the ratio of 1:3, 1:6:9 and 7:9 with the probabilities of

Table 7. Disease reactions of parents, F₁s and F₂s for Cm 67 crosses in the diallel set.

Cross	Parents disease reactions		Disease reactions of F ₁ s			Frequencies of F ₂ s on disease class bases								
	P ₁	P ₂	N	R	C	1	2	3	4	5	6	7	8	9 ^c
Herta x	2.8±0.9 ^a	7.5±1.1	5.9±0.9	5.7±1.0	5.9±1.0	N: 0	2	1	16	40	30	6	2	0
CM 67	2 to 3 ^b	7 to 8	5 to 7	5 to 6	5 to 7	R: 0	1	3	13	14	25	9	9	0
						C: 0	3	4	29	54	55	15	11	0
Summit x	2.7±1.3	8.0±1.3	7.3±1.9			0	0	9	18	12	21	27	10	0
CM 67	2 to 4	7 to 9	6 to 8			0	0	1	9	6	30	38	12	0
						0	0	10	27	18	51	65	22	0
Oderbrucker	2.8±0.8	7.9±1.6	7.1±1.6	7.5±1.6	7.2±1.5	0	0	3	14	6	26	26	21	0
x CM 67	2 to 3	7 to 9	6 to 8	7 to 8	6 to 8	0	0	7	18	15	23	16	5	0
						0	0	10	32	21	49	42	26	0
Betzes x	3.3±0.9	7.6±1.6	7.3±0.9			0	0	5	8	19	36	30	25	0
CM 67	3 to 4	7 to 9	7 to 8			0	0	0	2	7	13	18	24	0
						0	0	5	10	26	49	48	49	0

P₁: Parent 1
P₂: Parent 2
N: Normal cross

R: Reciprocal cross
C: Combined value
a: Estimated variation

b: Actual variation
c: Disease class (1 to 9)

Table 8. Probable F_1 genotypes and hypothetical segregations in F_2 s of CM 67 crosses in the diallel set.

Cross	Probable F_1 genotype	Combined classes to detect hypothetical segregation in F_2	Ratio	Probability
Herta x CM 67	-	Continous segregation	-	-
Summit x CM 67	Aa	(1+2+3+4+5) : (6+7+8+9)	1:3	0.20-0.25
Oderbrucker x CM 67	Aa	(1+2+3+4) : (5+6+7+8+9)	1:3	0.25-0.50
	AaBb	(1+2+3) : (4+5) : (6+7+8+9)	1:6:9	0.05-0.10
	AaBb	(1+2+3+4+5) : (6+7+8+9)	7:9	0.01-0.025
Betzes x CM 67	AaBb	(1+2+3+4) : (5+6) : (7+8+9)	1:6:9	0.90-0.95

0.25-0.50; 0.05-0.10 and 0.01-0.025. These results indicate that Oderbrucker may have either one or two recessive genes for resistance.

Betzes x CM 67

The F_1 was in the susceptible category, and the degree of dominance for this cross was found as 0.87, which indicates that susceptibility is almost completely dominant. The F_2 population fit the ratio of 1:6:9 with a high probability ($P = 0.90-0.95$). This shows that Betzes probably has two recessive genes for resistance.

SH 191 Crosses

Herta x SH 191

No F_1 plant was tested for this cross. If we assume that the class of F_2 which has the highest frequency represents the F_1 class, the degree of dominance is 0.59. This suggests that susceptibility is incompletely dominant. The F_2 population did not fit any ratio and segregated continuously. Herta probably has polygenic resistance to bacterial leaf streak. This conclusion corresponds to the result of the cross of Herta x CM 67.

Summit x SH 191

No significant reciprocal differences were found in the F_1 s and F_2 s. The degree of dominance was computed as 0.48 which shows that susceptibility is incompletely dominant.

Table 9. Disease reactions of parents, F_1 s and F_2 s for SW 191 crosses in the diallel set.

Cross	Parents disease reactions		Disease reactions of F ₁ s			Frequencies of F ₂ on disease class bases									
	P ₁	P ₂	N	R	C ¹ s		1	2	3	4	5	6	7	8	9 ^c
Herta x SW 191	2.0 ⁺ 1.6 ^a	7.6 ⁺ 1.0 ^b	-	-	-	N:	0	4	3	7	19	27	20	13	0
	1 to 3	7 to 8				R:	0	2	6	10	11	35	27	11	0
						C:	0	6	9	17	30	62	47	24	0
Summit x SW 191	3.2 ⁺ 0.8	8.0 ⁺ 1.4	6.2 ⁺ 1.4	6.7 ⁺ 1.0	6.4 ⁺ 1.3		0	0	3	10	4	28	25	19	0
	3 to 4	7 to 9	5 to 7	6 to 7	5 to 7		0	0	10	7	12	33	13	6	2
							0	0	13	17	16	61	38	25	2
Oderbrucker x SW 191	2.8 ⁺ 0.8	7.3 ⁺ 1.4	6.2 ⁺ 1.1	6.4 ⁺ 1.8	6.2 ⁺ 1.2		0	1	15	14	10	11	18	7	0
	2 to 3	6 to 8	5 to 7	5 to 7	5 to 7		0	3	14	16	11	14	19	4	0
							0	4	29	30	21	25	37	11	0
Betzes x SW 191	3.1 ⁺ 1.6	7.7 ⁺ 1.6	7.0 ⁺ 1.1	7.5 ⁺ 1.4	7.1 ⁺ 1.1		0	2	3	5	8	7	25	6	0
	2 to 3	7 to 9	6 to 8	7 to 8	6 to 8		0	4	5	7	11	9	33	9	0
							0	6	8	12	19	16	58	15	0

P_1 : Parent 1

R: Reciprocal cross

b: Actual variation

P_2 : Parent 2

C : Combined value

c: Disease classes (1 to 9)

N: Normal cross

a: Estimated variation

Table 10. Probable F_1 genotypes and hypothetical segregations in F_2 s of SW 191 crosses in the diallel set.

Cross	Probable F_1 genotype	Combined classes to detect hypothetical segregation in F_2	Ratio	Probab.
Herta x SW 191	-	Continous segregation	-	-
Summit x SW 191	Aa	(1+2+3+4) : (5+6+7+8+9)	1:3	0.05-0.10
Oderbrucker x SW 191	Aa AaBb	(1+2+3) : (4+5+6+7+8+9) (1+2+3+4) : (5+6+7+8+9)	1:3 7:9	0.25-0.50 0.25-0.50
Betzes x SW 191	AaBb	(1+2+3) : (4+5+6) : (7+8+9)	1:6:9	0.10-0.20

Probab.: Probability

The F_2 population fit the ratio of 1:3 with a probability of 0.05-0.10. This result indicates that Summit probably has one recessive gene for resistance, which agrees with that of the cross of Summit x CM 67.

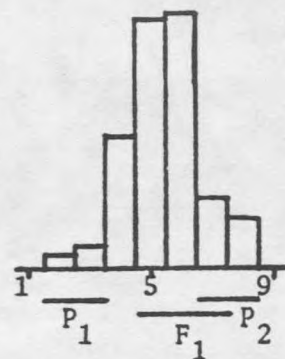
Oderbrucker x SW 191

The disease reactions of F_1 and F_2 resulting from reciprocal crosses were not significantly different from those of normal F_1 and F_2 . The F_1 showed a susceptible reaction, and the degree of dominance was estimated as 0.75, which might indicate almost complete dominance. The F_2 population fit the ratios of 7:9 and 1:3 with the same probability ($P = 0.25-0.50$). Thus, Oderbrucker may have one or two recessive resistance genes.

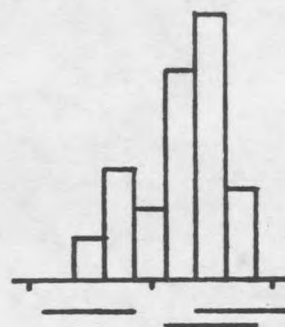
Betzes x SW 191

In the F_1 s and F_2 s, no significant reciprocal differences were found. The F_1 of this cross was almost in the susceptible parent class. The degree of dominance was calculated as 0.86. Susceptibility is probably almost completely dominant. The F_2 population fit the ratio of 1:6:9 with the probability of 0.10-0.20. Betzes probably has two recessive genes for resistance. The same conclusion was made for the cross of Betzes x CM 67.

Herta x CM 67



Summit x CM 67

Oderbrucker
x CM 67

Betzes x CM 67

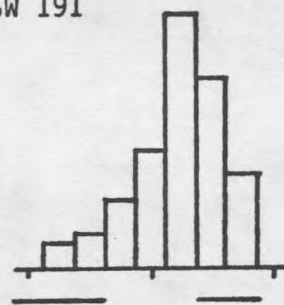
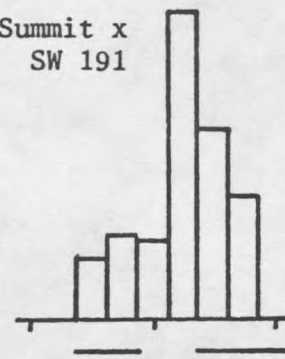
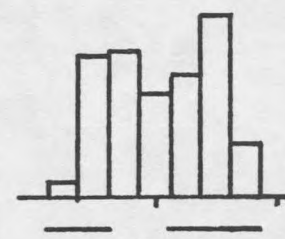
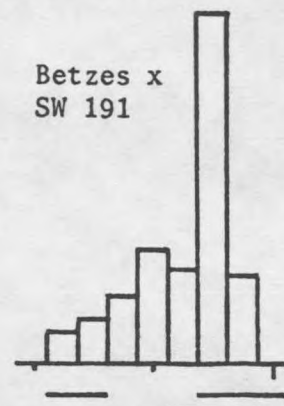
Herta x
SW 191Summit x
SW 191Oderbrucker
x SW 191Betzes x
SW 191

Figure II. F_2 distributions of CM 67 and SW 191 crosses in the diallel set. P_1 :Resistant parent's class. P_2 :Susceptible parent's class. F_1 : F_1 class. 1 to 9:Disease classes in F_2 .

Resistant x Susceptible Crosses

In this category, some promising barley cultivars resistant to bacterial leaf streak were crossed with three susceptible cultivars (CM 67, SW 191 and Kangbori). The results are shown Table 11, 12, 13, 14, 15, 16, 17 and 18. These crosses were studied in three groups according to their susceptible parents.

CM 67 Crosses

Three different crosses were made with CM 67, and according to homogeneity test, their F_2 populations segregated differently (Table 11 and 12; Figure III).

CI 12558 x CM 67

The degree of dominance was estimated as 0.13 which is close to zero, and indicates that resistance is intermediate. The F_2 population did not fit any ratio and segregated continuously. The result showed that CI 12558 may have polygenic intermediate resistance to bacterial leaf streak.

CI 12777 x CM 67

The F_1 of this cross showed almost a susceptible reaction. The degree of dominance was 0.70, which indicates that susceptibility is partially dominant. The F_2 population fit the ratios of 7:9 and 1:6:9 with the probabilities of 0.25-0.50 and 0.05-0.10. Thus, CI 12777

Table 11. Disease reactions of parents, F_1 s and F_2 s for CM 67 crosses in the resistant x susceptible cross category.

Cross	Parents disease reactions		F_1 disease reaction	Frequencies of F_2 on disease class bases								
	♀	♂		1	2	3	4	5	6	7	8	9 ^c
CI 12558 x	1.9 ⁺ 0.9 ^a	7.5 ⁺ 1.0	4.5 ⁺ 1.7	0	3	11	22	14	12	7	7	0
CM 67	1 to 3 ^b	7 to 8	3 to 6									
CI 12777 x	1.5 ⁺ 1.0	8.0 ⁺ 1.3	6.5 ⁺ 1.4	0	1	10	9	17	12	15	12	1
CM 67	1 to 2	7 to 9	6 to 7									
CI 12866 x	1.5 ⁺ 1.0	7.7 ⁺ 1.0	-	0	20	16	17	9	10	12	1	0
CM 67	1 to 2	7 to 8										

a: Estimated variation

c: Disease classes

b: Actual variation

Table 12. Probable F_1 genotypes and hypothetical segregations in F_2 s of CM 67 crosses in the resistant x susceptible category.

Cross	Probable F_1 genotype	Combined classes to detect hypothetical segregation in F_2	Ratio	Probability
CI 12558 x CM 67	-	Continous segregation	-	-
CI 12777 x CM 67	AaBb AaBb	(1+2+3+4+5) : (6+7+8+9) (1+2) : (3+4+5) : (6+7+8+9)	7:9 1:6:9	0.25-0.50 0.05-0.10
CI 12866 x CM 67	Aa AaBb	(1+2+3+4+5) : (6+7+8+9) (1+2+3+4) : (5+6+7+8+9)	3:1 9:7	0.50-0.75 0.25-0.50

may have two recessive genes for resistance.

CI 12866 x CM 67

In this cross, no F_1 plant was tested. The F_2 populations fit the ratios of 3:1 and 9:7 with the probabilities of 0.50-0.75 and 0.25-0.50. The segregation of the F_2 indicates that resistance is partially dominant and CI 12866 may have one or two genes for resistance.

SW 191 Crosses

Six resistant lines were crossed with SW 191. The homogeneity test showed that the F_2 populations of CI 12787, CI 13095, PI 382732 and PI 383077 segregated in the same manner (Table 13, 14 and 15; Figure III).

CI 12787 x SW 191

The F_1 of this cross reacted near the resistant parent class. The degree of dominance was computed as 0.61. This shows that resistance is partially dominant. The F_2 population fit the ratios of 3:1 and 12:3:1 with the probabilities of 0.50-0.75 and 0.25-0.50. CI 12787 may have one or two dominant resistant genes.

CI 13095 x SW 191

No F_1 plant was tested for this cross. The segregation in the F_2 suggests that resistance is dominant. With the same probability ($P = 0.50-0.75$), the F_2 population fit the ratios of 3:1 and 12:3:1. CI 13095

Table 13. Homogeneity test for SW 191 crosses in the resistant x susceptible cross category.

Cross	Phenotypes of F ₂ (12:3:1 expected)			D.F.	Chisquare	Probability
	R	I	S			
CI 12787 x	O: 75.00	18.00	10.00	2	2.12	0.25-0.50
SW 191	E: 77.25	19.31	6.44			
CI 13095 x	87.00	22.00	5.00	2	0.68	0.50-0.75
SW 191	85.50	21.38	7.12			
PI 382732 x	81.00	15.00	5.00	2	1.46	0.25-0.50
SW 191	75.75	18.94	6.31			
PI 383077 x	55.00	15.00	10.00	2	5.42	0.05-0.10
SW 191	60.00	15.00	5.00			
Total				8	9.68	
Summed data	298.00	70.00	30.00	2	1.35	0.20-0.25
	298.50	74.63	24.87			
Homogeneity χ^2				6	8.33	0.20-0.25

R :Resistant

S :Susceptible

O :Observed

I :Intermediate

D.F. :Degrees of freedom

E :Expected

Table 14. Disease reaction of parents, F₁s and F₂s for SW 191 crosses in the resistant x susceptible cross category.

Cross	Parents disease reactions		F ₁ disease reaction	Frequencies of F ₂ on disease class bases									
	♀	♂		1	2	3	4	5	6	7	8	9 ^c	
CI 12787 x SW 191	1.4 ⁺ -1.1 ^a 1 to 2 ^b	8.6 ⁺ -1.5 7 to 9	3.4 ⁺ -1.1 3 to 4	19	27	14	15	8	10	5	5	0	
CI 13095 x SW 191	2.1 ⁺ -0.7 2 to 3	7.5 ⁺ -1.4 7 to 9	-	0	58	29	5	7	10	4	1	0	
PI 382511 x SW 191	1.5 ⁺ -1.1 1 to 2	6.9 ⁺ -1.5 6 to 8	3.6 ⁺ -1.0 3 to 4	12	26	19	10	1	2	3	3	0	
PI 382650 x SW 191	1.5 ⁺ -1.0 1 to 2	6.9 ⁺ -1.7 6 to 8	3.0 ⁺ -1.6 2 to 4	2	20	13	12	7	13	7	7	0	
PI 382732 x SW 191	1.3 ⁺ -1.0 1 to 2	7.8 ⁺ -1.3 7 to 9	-	28	19	22	12	8	7	4	1	0	
PI 383077 x SW 191	1.2 ⁺ -0.9 1 to 2	7.7 ⁺ -1.5 7 to 9	-	14	13	17	11	7	8	8	2	0	

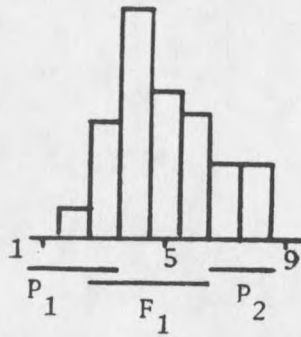
a: Estimated variation c: Disease classes

b: Actual variation

Table 15. Probable F_1 genotypes and hypothetical segregations in F_2 s of SW 191 crosses in the resistant x susceptible category.

Cross	Probable F_1 genotype	Combined classes to detect hypothetical segregation in F_2	Ratio	Probability
CI 12787 x	Aa	(1+2+3+4) : (5+6+7+8+9)	3:1	0.50-0.75
SW 191	AaBb	(1+2+3+4) : (5+6) : (7+8+9)	12:3:1	0.25-0.50
CI 13095 x	Aa	(1+2+3) : (4+5+6+7+8+9)	3:1	0.50-0.75
SW 191	AaBb	(1+2+3) : (4+5+6) : (7+8+9)	12:3:1	0.50-0.75
PI 382511 x	AaBb	(1+2) : (3+4+5) : (6+7+8+9)	9:6:1	0.20-0.25
SW 191 x	AaBb	(1+2+3+4+5) : (6+7+8+9)	15:1	0.10-0.20
PI 382650 x	AaBb	(1+2+3+4) : (5+6+7+8+9)	9:7	0.50-0.75
SW 191				
PI 382732 x	AaBb	(1+2+3+4) : (5+6) : (7+8+9)	12:3:1	0.25-0.50
SW 191 x	Aa	(1+2+3+4) : (5+6+7+8+9)	3:1	0.20-0.25
PI 383077 x	Aa	(1+2+3+4) : (5+6+7+8+9)	3:1	0.10-0.20
SW 191 x	AaBb	(1+2+3+4) : (5+6) : (7+8+9)	12:3:1	0.05-0.10

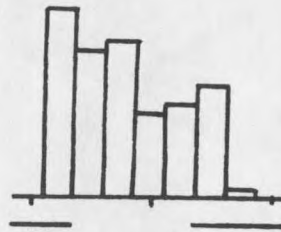
CI 12558 x CM 67



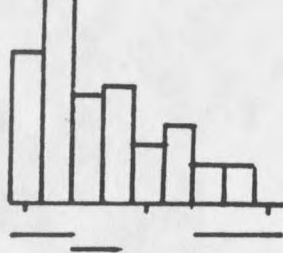
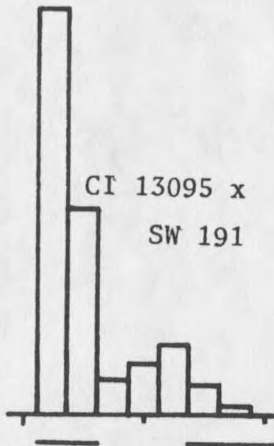
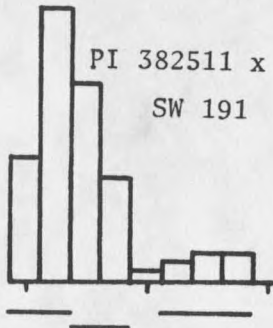
CI 12777 x CM 67



CI 12866 x CM 67



CI 12787 x SW 191

CI 13095 x
SW 191PI 382511 x
SW 191

PI 382650 x SW 191

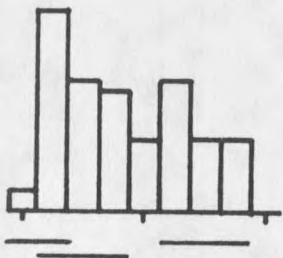
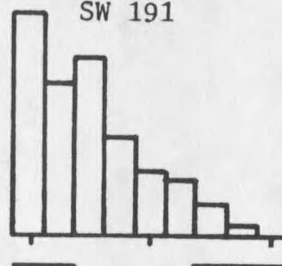
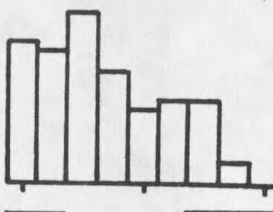
PI 382732 x
SW 191PI 383077 x
SW 191

Figure III. F_2 distributions of CM 67 and SW 191 crosses in the resistant x susceptible category. P_1 :Resistant parent's class. P_2 :Susceptible parent's class. F_1 : F_1 class.

may have either one dominant resistant gene with some modifying genes or two dominant resistant genes with one of the dominant genes being epistatic to the other.

PI 382511 x SH 191

The F_1 of this cross showed a moderately resistant reaction. The degree of dominance was estimated as 0.39 which shows that resistance might be either incompletely dominant or intermediate. The F_2 population fit the ratios 9:6:1 and 15:1 with the probabilities of 0.20-0.25 and 0.10-0.20. PI 382511 may have two incompletely dominant genes for resistance. In the first case, both dominant genes interact with each other.

PI 382650 x SH 191

The disease reaction of the F_1 was close to the resistant parent class. The degree of dominance was calculated as 0.61, which indicates that resistance is partially dominant. With a high probability ($P = 0.50-0.75$), the F_2 population fit the ratio or 9:7. PI 382650 may have two incompletely dominant genes for resistance.

PI 382732 x SH 191

Parents and F_2 were tested for this cross. The F_2 population fit the ratios of 12:3:1 and 3:1 with the probabilities of 0.25-0.50 and 0.20-0.25. The results show that this barley line may have one or two dominant

genes for resistance. If PI 382732 has one gene, the gene is incompletely dominant.

PI 383077 x SH 191

For this cross, no F_1 plant was tested. With the probabilities of 0.10-0.20 and 0.05-0.10, the F_2 population fit the ratios of 3:1 and 12:3:1. According to the F_2 segregation, PI 383077 might have one or two dominant genes. If it has one resistant dominant gene, dominance is incomplete or there may be some other modifying genes.

Kangbori Crosses

Susceptible cultivar Kangbori was crossed with two resistant varieties and seven lines (Table 17 and 18, Figure IV). The F_2 populations of Alpine, Luther, and CI 12777 with Kangbori fit the same ratio (9:6:1). A homogeneity test indicated that the F_2 of these crosses segregated in the same manner (Table 16, 17 and 18; Figure IV).

Alpine x Kangbori

The reaction of the F_1 was found in the resistant parent class. The degree of dominance was estimated as 0.82, which indicates that resistance is almost completely dominant. The F_2 population fit the ratio of 9:6:1 with the probability of 0.20-0.25. This indicates that Alpine

Table 16. Homogeneity test for Kangbori crosses in the resistant x susceptible category.

Cross	Phenotypes of F ₂ (9:6:1 expected)			D.F.	Chisquare	Probability
	R	I	S			
Alpine x K	O: 50.00	23.00	7.00	2	2.99	0.20-0.25
	E: 45.00	30.00	5.00			
Luther x K	46.00	41.00	7.00	2	2.05	0.25-0.50
	52.88	32.25	5.88			
CI 12777 x K	40.00	37.00	7.00	2	2.66	0.25-0.50
	47.25	31.50	5.25			
Total				6	7.70	
Summed data	136.13	101.00	21.00	2	2.23	0.25-0.50
Homogeneity χ^2	145.13	96.75	16.13	4	5.47	0.20-0.25
(9:7 expected)						
CI 12777 x K	54.00		30.00	1	2.18	0.10-0.20
	47.25		36.75			
CI 12866 x K	53.00		39.00	1	0.07	0.75-0.90
	51.75		40.25			
Total				2	2.25	
Summed data	107.00		69.00	1	1.46	
Homogeneity χ^2	99.00		77.00	1	0.79	0.25-0.50

may have two dominant genes for resistance.

Luther x Kangbori

The F_1 showed a resistant reaction to the disease. The degree of dominance was computed as 0.99. This suggests that resistance is completely dominant. With the probability of 0.25-0.50, segregation of the F_2 fit a 9:6:1 ratio. Therefore, like Alpine, Luther may have two dominant genes for resistance.

CI 12569 x Kangbori

Dominance deviation was calculated as 0.25, which indicates that resistance is probably intermediate. The F_2 population fit a 1:2:1 ratio with the probability of 0.05-0.10. Probably, CI 12569 has one gene for resistance and its effect is intermediate.

CI 12577 x Kangbori

The F_1 reaction to bacterial leaf streak was found between the susceptible and resistant parent classes. The degree of dominance was estimated as 0.13 which shows that resistance is intermediate. The F_2 population did not fit a 1:2:1 ratio. The probability was computed as 0.025-0.05 which is lower than an acceptable level. Further study is needed to determine inheritance in this parent.

Table 17. Disease reactions of parents, F_1 s and F_2 s for Kangbori crosses in the resistant x susceptible cross category.

Cross	Parents disease reactions		F_1 disease reaction	Frequencies of F_2 on disease class bases									
	♀	♂		1	2	3	4	5	6	7	8	9 ^c	
Alpine x Kangbori	1.9 ⁺ -1.3 ^a 1 to 3 ^b	7.9 ⁺ -1.3 7 yo 9	2.8 ⁺ -1.3 2 to 3	9	20	21	8	8	7	7	0	0	
Luther x Kangbori	2.8 ⁺ -0.5 2 to 3	7.7 ⁺ -1.3 7 to 9	2.8 ⁺ -0.4 2 to 3	0	3	43	16	16	9	7	0	0	
CI 12569 x Kangbori	1.2 ⁺ -0.9 1 to 2	7.4 ⁺ -1.3 6 to 8	3.9 ⁺ -1.6 3 to 5	0	10	23	10	4	10	10	3	0	
CI 12577 x Kangbori	1.5 ⁺ -1.4 1 to 3	6.8 ⁺ -1.1 6 to 8	4.3 ⁺ -1.4 3 to 5	2	21	22	12	15	7	4	0	0	
CI 12595 x Kangbori	2.0 ⁺ -1.1 1 to 3	7.7 ⁺ -1.0 7 to 8	-	5	9	26	24	12	13	2	2	0	
CI 12776 x Kangbori	2.4 ⁺ -1.1 2 to 3	7.8 ⁺ -0.8 7 to 8	6.2 ⁺ -1.5 5 to 7	0	8	25	17	9	13	13	4	0	
CI 12777 x Kangbori	1.7 ⁺ -1.0 1 to 2	7.6 ⁺ -1.0 7 to 8	2.5 ⁺ -1.1 5 to 7	4	13	23	14	10	13	7	0	0	
CI 12866 x Kangbori	1.5 ⁺ -1.0 1 to 2	7.5 ⁺ -1.0 7 to 8	-	4	18	17	14	11	17	10	1	0	
PI 382720 x Kangbori	1.4 ⁺ -1.3	7.3 ⁺ -1.6	3.8 ⁺ -1.7	18	25	18	15	6	8	6	0	0	

a: Estimated variation

c: Disease classes

b: Actual variation

Table 18. Probable F_1 genotypes and hypothetical segregations in F_2 s of Kangbori crosses in the resistant x susceptible category.

Cross	Probable F_1 genotype	Combined classes to detect hypothetical segregation in F_2	Ratio	Probability
Alpine x Kangbori	AaBb	(1+2+3) : (4+5+6) : (7+8+9)	9:6:1	0.20-0.25
Luther x Kangbori	AaBb	(1+2+3) : (4+5+6) : (7+8+9)	9:6:1	0.25-0.50
CI 12569 x Kangbory	A_1A_2	(1+2) : (3+4+5) : (6+7+8+9)	1:2:1	0.05 -0.10
CI 12577 x Kangbori	A_1A_2	(1+2) : (3+4+5) : (6+7+8+9)	1:2:1	0.025-0.05
CI 12595 x Kangbori	AaBb	(1+2+3+4) : (5+6) : (7+8+9)	12:3:1	0.10-0.25
CI 12776 x Kangbori	Aa Unknown	(1+2+3+4) : (5+6+7+8+9) —	3:1 —	0.10-0.20 —
CI 12777 x Kangbori	AaBb	(1+2+3) : (4+5+6) : (7+8+9)	9:6:1	0.25-0.50
PI 382720 x Kangbori	AaBb	(1+2+3) : (4+5+6+7+8+9)	9:7	0.10-0.20
	Aa	(1+2+3+4) : (5+6+7+8+9)	3:1	0.25-0.50
CI 12866 x Kangbori	AaBb	(1+2+3+4) : (5+6+7+8+9)	9:7	0.75-0.90

CI 12595 x Kangbori

No F_1 plant was tested for this cross. With similar probabilities ($P = 0.10-0.20$), the F_2 population fit the ratios of 3:1 and 12:3:1. The above results indicate that CI 12595 may have one or two incompletely dominant genes for resistance.

CI 12776 x Kangbori

The F_1 reaction of this cross was found closer to the susceptible parent class. The degree of dominance was calculated as 0.54 which indicates that susceptibility is incompletely dominant. Although the F_1 reaction was closer to the susceptible parent class, most of the individuals in the F_2 population showed a resistant reaction. The F_2 population did not fit any ratio.

CI 12777 x Kangbori

The F_1 of this cross showed a resistant reaction. The degree of dominance was computed as 0.84. These results suggest that resistance to bacterial leaf streak is almost completely dominant. The F_2 population fit the ratios of 9:6:1 and 9:7 with the probabilities of 0.25-0.50 and 0.10-0.20. This indicates that CI 12777 may have two dominant genes for resistance to the disease.

CI 12866 x Kangbori

Parents and F_2 were tested for this cross. With a

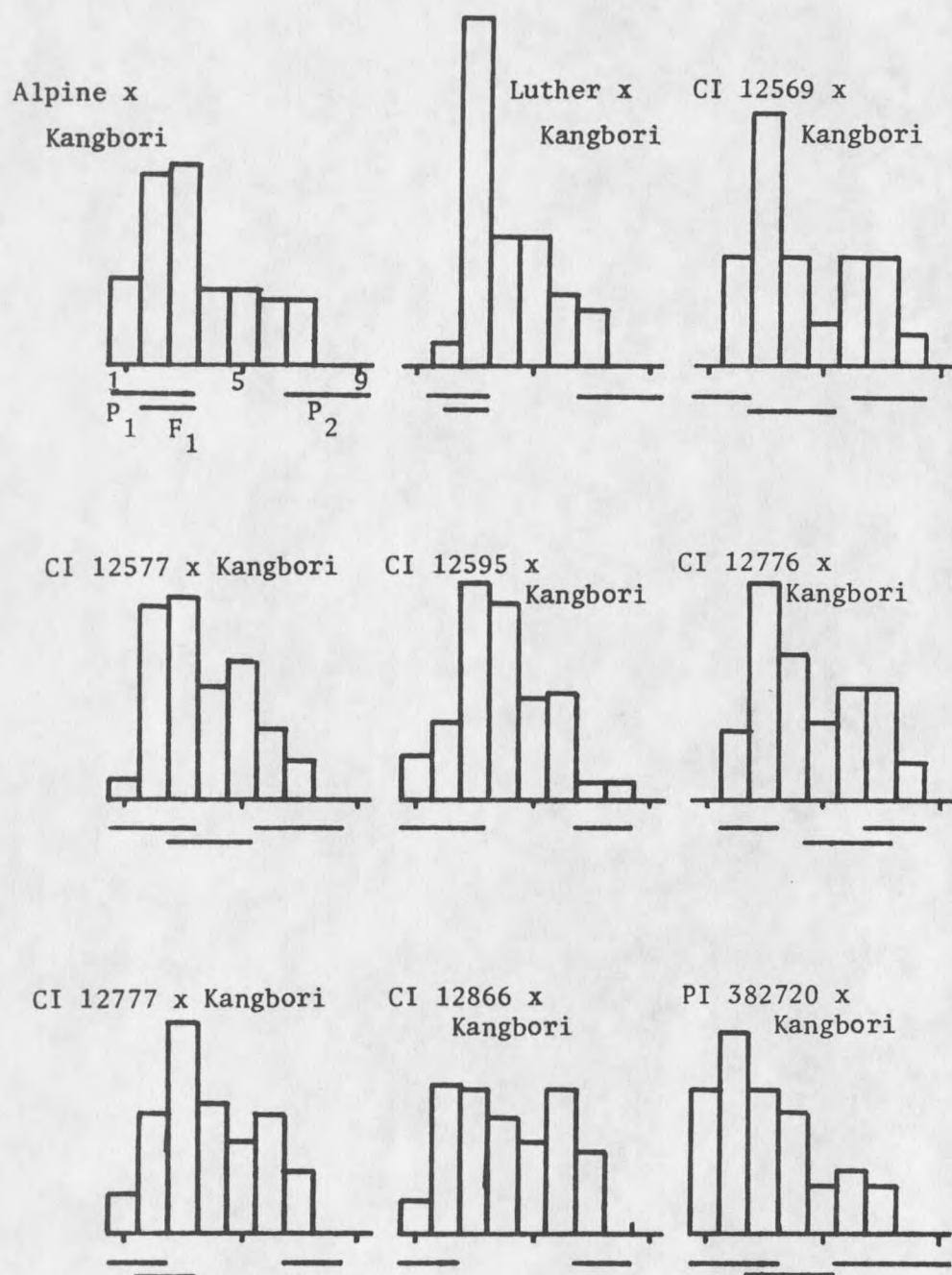


Figure IV. F_2 distribution of Kangbori crosses in the resistant x susceptible category. P_1 : Resistant parent's class. P_2 : Susceptible parent's class. F_1 : F_1 class.

high probability ($P = 0.75-0.90$), the F_2 population fit a 9:7 ratio. This leads to the conclusion that CI 12866 might have two dominant genes for resistance and either recessive homozygote masks the effect of the other gene.

PI 382720 x Kangbori

The F_1 reaction to the disease was found to be near that of the resistant parent class. The degree of dominance was estimated as 0.31 which indicates that resistance is probably incompletely dominant. The F_2 population fit the ratio of 3:1 with the probability of 0.25-0.50. The results suggest that PI 382750 may have one dominant gene for resistance.

Resistant x Resistant Crosses

In this category, Alpine and Luther were crossed with Herta, Summit and Oderbrucker, which were used as resistant parents in the diallel set. Statistically, all parents had the same level of resistance to the disease (Table 17).

The reaction of F_1 s and F_2 s to the strain (X-67) did not significantly differ from each other and from that of the cultivars. The results suggest that the parent cultivars used in this category may have at least one common gene or closely linked genes for resistance. Another possibility is that the size of F_2 populations

Table 19. Disease reactions of parents, F₁s and F₂s for the crosses in the resistant x resistant category.

Cross	----- Parents -----		F ₁	F ₂
Alpine x Herta	1.7 ± 1.0 1 to 2	2.8 ± 0.9 2 to 3	2.8 ± 1.0 2 to 3	2.3 ± 1.4 1 to 3
Alpine x Oderbr.	1.9 ± 1.4 1 to 3	2.4 ± 1.5 1 to 3	1.7 ± 1.0 1 to 2	1.7 ± 1.5 1 to 3
Luther x Herta	1.9 ± 1.4 1 to 3	2.0 ± 1.2 1 to 3	2.3 ± 1.0 2 to 3	1.4 ± 1.3 1 to 3
Luther x Summit	1.6 ± 1.1 1 to 2	2.3 ± 1.6 1 to 3	-	2.0 ± 1.8 1 to 4
Luther x Oderbr.	1.5 ± 1.0 1 to 2	2.0 ± 1.4 1 to 3	-	1.7 ± 1.5 1 to 3

Oderbr.: Oderbrucker

as not large enough to detect segregations.

Relation between Earliness and Susceptibility

The disease reactions of early plants were recorded in 14 F₂ populations which had a late, resistant parent and an early, susceptible parent. Their percentages in each disease class were computed (Table 20). In all F₂ populations, early plant percentages were found to be higher in the susceptible classes and lower or zero in the resistant classes. Thus, earliness was mostly associated with susceptibility. However, some early and resistant plants were observed in the F₂ populations of CI 12595, CI 12787, PI 382511, and PI 383077.

Table 20. Disease class frequencies and percentages of early plants in 14 F₂ populations which have a late, resistant parent and an early, susceptible parent.

C r o s s e s															
D.C.	A	B	C	D	E	F	G	H	I	J	K	L	M	N	$\bar{X}$
1	-	19(0)	-	28(0)	14(0)	12(0)	2(0)	9(0)	-	2(0)	-	5(0)	4(0)	18(0)	11(0)
2	^a ₂₀₍₀₎ ^b	27(7)	58(7)	19(5)	13(23)	26(12)	20(0)	20(5)	3(0)	21(0)	10(0)	9(0)	18(0)	25(0)	21(1)
3	16(6)	14(14)	29(0)	22(5)	17(24)	19(16)	13(8)	21(5)	43(0)	22(9)	23(0)	26(19)	17(0)	18(11)	21(8)
4	17(35)	15(33)	5(20)	12(0)	11(55)	10(40)	12(50)	8(25)	16(13)	12(25)	10(20)	24(38)	14(14)	15(33)	13(29)
5	9(44)	8(75)	7(71)	8(38)	7(71)	1(100)	7(100)	8(63)	16(44)	15(73)	4(0)	12(58)	11(0)	6(86)	9(59)
6	10(90)	10(90)	10(70)	7(29)	8(63)	2(100)	13(46)	7(57)	9(78)	7(71)	10(70)	13(75)	17(76)	8(43)	9(68)
7	12(83)	5(60)	4(75)	4(50)	8(63)	3(100)	7(86)	7(100)	7(57)	4(50)	10(70)	2(50)	10(70)	6(83)	6(71)
8	1(100)	5(40)	1(100)	1(100)	2(100)	3(100)	7(100)	-	-	-	3(67)	2(50)	1(100)	-	3(86)
9	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

D.C. :Disease classes	E :PI 383077 x SW 191	J :CI 12577 x Kangbori	$\bar{X}$:Mean
A :CI 12866 x CM 67 ^c	F :PI 382511 x "	K :CI 12569 x "	a :Actual frequencies
B :CI 12787 x SW 191 ^c	G :PI 382650 x "	L :CI 12595 x "	b :Early plant percentages in each class
C :CI 13095 x "	H :Alpine x Kangbori ^c	M :CI 12866 x "	c :Early, susceptible parents
D :PI 382732 x "	I :Luther x "	N :PI 382720 x "	

CHAPTER 5

DISCUSSION

Disease Development and Planting Design

As Boosalis (1952) reported, one of the ways to disseminate bacterial leaf streak of barley is by contact of a diseased plant with a healthy one when both are water-congested. For this reason one very susceptible cultivar (CM 67) row was planted between every two experimental rows. Early artificial inoculation caused high infection of these spreader rows and provided adequate inoculum for the adjacent rows through the growing season. Hall et al. (1981) reported that Xanthomonas campestris pv. translucens was capable of spreading 28 square meters from a single infection locus within 39 days. Bockelman (1984, personal communication) observed that the disease uniformly spread only from the inoculated row to the adjacent row. His observation supported the planting of a spreader row between every two rows to create uniform infection. Sprinkler irrigation promoted the infection process in two ways: (i) By splashing the bacterial cells from infected plant

rows to the others, and (ii) by creating dew on the plant surface thus enabling bacterial movement and invasion. The factors mentioned above helped create a uniform infection and eliminated escapes.

Gardner (1961) suggested that field stratification in small blocks and evaluation of individuals within blocks reduces error due to environmental influence. Thus, parents, F_1 and F_2 of one cross were planted in a small area (Figure 1). A serpentine planting design provided a uniform environment by localizing individuals which belonged to the same cross yet were in the different blocks.

Earliness and Susceptibility

Early plant percentages in the susceptible disease classes were found to be higher in the 14 F_2 populations that had an early, susceptible parent and a late resistant parent. This showed that one of the parental phenotypes (the early and susceptible one) was in a higher frequency in the F_2 population indicating that there might be a genetic linkage between earliness and susceptibility. However, there are other possible explanations:

- 1) In early summer, because temperature and relative humidity are adequate for bacterial infection, disease

development is rapid. As the season progresses, perhaps conditions worsen for the bacterium and that may slow down or stop disease development. As a result, early plants may become highly infected and late ones might escape from the disease. The above speculation may not be true because sprinkler irrigation was carried out every three days to keep conditions favorable for disease development until late in the season.

2) Levitt (1980) reported that high growth rate of early plants is directly related to a high photosynthetic rate. The method available to an early plant for maintaining high photosynthetic and growth rates, is to keep its stomata open longer. This will provide more opportunities for bacteria to enter the plant. Campbell et al. (1980) supported the above conclusion. They reviewed instances in which resistance and susceptibility to some pathogens had been related to the time of opening and closing of stomata.

3) Weller et al. (1980) found that under field conditions, disease symptoms always were detected after X. c. pv. phaseoli populations reached 5×10^6 cells per navy bean leaflet. If we assume that population sizes of bacteria are a function of time, bacteria will have more opportunity to reach a sufficient quantity on leaves of

early heading plants, since they are exposed to infection earlier and longer.

All or some of the factors above may have resulted in a close relation between earliness and susceptibility. If there is a genetic linkage between these characteristics, barley cultivars CI 12595, CI 12787, PI 382511, and PI 383077 may be promising for breaking this linkage, since some early resistant plants were found in their crosses.

Inheritance of Resistance

The results obtained from the CM 67 and SW 191 crosses with the four resistant parents in the diallel set suggest that resistance in Herta, Summit, Oderbrucker, and Betzes is the same with regard to gene action (resistance recessive), but may be different in regard to gene numbers (Table 7, 8, 9 and 10). The crosses of Herta with CM 67 and SW 191 showed continuous segregation in the F_2 populations. According to Vanderplank (1982), five reasons for continuous segregation are: (1) Resistance may be polygenic; (2) the characteristic could be determined by three genes, one of these having three times, and the other two times, the effect of the third gene; (3) in three different chromosomes, the characteristic may be determined by a

block of three, and two linked genes and a simple gene, and the gene effects are the same, and additive; (4) presence of a large environmental effect; monogenic resistance without complete dominance can underlie continuous variation; (5) some or all factors mentioned above can take place together in the same population. As explained before, the experiment was performed in a relatively uniform environment. For this reason we may discount the 4th assumption.

The Summit crosses with CM 67 and SW 191 showed that Summit may have one recessive gene for resistance to bacterial leaf streak. The crosses of Oderbrucker with CM 67 and SW 191 indicated that Oderbrucker may have either one or two recessive genes for resistance. The F_2 segregation of Oderbrucker x CM 67 fit both 1:3 and 1:6:9; ratios (Table 8), that of Oderbrucker x SW 191 fit 1:3 and 7:9 (Table 10). Although 1:3 was common in both crosses, the latter were found to be different. This may have been caused by different susceptible genetic backgrounds. The Betzes crosses with CM 67 and SW 191 gave the same results with their F_2 populations fitting the ratio 1:6:9. This suggests that Betzes may have two recessive genes for resistance to the disease.

Although results from the resistant x susceptible

crosses in the diallel set showed inheritance of resistance to be different in four parents, the crosses among these resistant parents did not significantly segregate in their F_2 populations. This may indicate that the resistant parents have at least one common resistance gene or different linked resistant genes on the same chromosome. Another possibility is that the F_2 populations were not large enough to detect all segregations. This is especially true for the Herta crosses, since Herta may have polygenic resistance. The susceptible x susceptible cross in the diallel set also did not segregate. In addition, CM 67 and SW 191 crosses with four resistant parents gave the same or similar segregations in the F_2 populations. Both of these results suggest that the two susceptibles (CM 67 and SW 191) probably do not contain any resistance genes.

CM 67 crosses in the resistant x susceptible cross category gave a variety of results. The F_2 population or CI 12558 segregated continuously, that of CI 12777 fit the ratios 7:9 and 1:6:9, and that of CI 12866 fit 3:1 and 9:7 (Table 12). As explained before, the continuous segregation in the F_2 population of CI 12558 might be caused either by polygenes or other factors. These results showed that CI 12777 probably has two recessive

genes, and CI 12866 may have either one or two dominant genes for resistance to bacterial leaf streak. If the resistance genes in CI 12777, and CI 12866 are located at separate or loosely linked loci, then, these cultivars may be useful parents for combining the genes.

The crosses of SW 191 in the resistant x susceptible category gave some similar and some different results. The F_2 populations of CI 12787, CI 13095, PI 382732, and PI 383077 fit the same ratios (3:1 and 12:3:1). For these crosses, a homogeneity test was done and they were found to be homogeneous. The results suggest that these lines could have either the same genes or different genes with the same effect for resistance. If it is assumed that they have the same resistant genes to the disease, in a breeding program the one which has the best agronomic type and other characteristics should be used. The F_2 populations of PI 382511 and PI 382650 fit different ratios. This shows that PI 382511 and PI 382650 are different from each other and from the first four lines. Unless the resistance genes of these lines are closely linked or at the same locus, they can be used in a breeding program to combine the resistance genes.

Kangbori crosses in the resistant x susceptible category segregated either similarly or differently.

Alpine, Luther, and CI 12777 crosses fit the ratio of 9:6:1. The result of a homogeneity test suggested that they may have the same genes for resistance. Since Luther is a mutant of Alpine, they are likely to have the same resistance genes to bacterial leaf streak. The F_2 of CI 12777 x Kangbori cross fit either a 9:6:1 or a 9:7 ratio, the latter being the same as that of CI 12866 x Kangbori cross. Although the homogeneity test showed that the F_2 population of CI 12777 and CI 12866 crosses segregated in a similar manner, gene effects in CI 12777 were found to be recessive in CI 12777 x CM 67 cross and those of CI 12866 were dominant in the CI 12866 x CM 67 cross (Table 12). This indicates that CI 12777 and CI 12866 have different genes for resistance to bacterial leaf streak. The F_2 segregation in the CI 12866 cross was found to be different than those of Alpine and Luther crosses. The other Kangbori crosses were found to be different from the above crosses and from each other in regard to disease resistance. If it is assumed that the resistance genes are located at separate or loosely linked loci, then, Alpine or Luther, CI 12866, CI 12569, CI 12577, CI 12595 and, PI 382720 seem the best parents to combine the various resistance genes to bacterial leaf streak.

In conclusion, there are diverse resistance sources

for bacterial leaf streak of barley. This study showed that some of these sources may have identical genes or different genes on the same chromosomes. Further study is needed to determine identical linked and independent genes.

The Use of Resistance Sources

Barley cultivars, Herta, Summit, Oderbrucker, Betzes, Alpine, Luther, CI 12558, CI 12569, CI 12595, CI 12777, CI 12787, CI 12866, CI 13095, PI 382511, PI 382650, PI 382720, and PI 383077, were found to be promising sources for resistance to bacterial leaf streak. Herta and CI 12558 may have polygenic resistance, and the remainder appear to have oligogenic resistance.

To develop resistant cultivars in a short time, the cultivars with oligogenic resistances can be screened for resistance where the disease is a problem. Then, the most resistant ones with desired agronomic characters should be used to transfer the resistance to commercial varieties by backcrossing.

The following procedure can be applied to develop germplasm for long-lasting disease resistance. Although no differential strains of the bacterium are known, it may be possible to identify major resistance genes in a polygenic background by screening plants in an

environment such as Sidney, Montana, where the disease severity is very high. Herta and CI 12558, that may have polygenic resistances, can be used as recurrent parents. By back-crossing, major genes in the cultivars identified as possibly having different loci for resistance can be carried into the polygenic background of Herta and CI 12558. Major genes in the polygenic background of Herta can be combined in later steps by inter-crossing the Herta-background lines with different major genes without losing polygenes. The same type of crosses can be made using CI 12558. Then, resistant lines with combined major genes in Herta- and CI 12558-background can be developed as resistant sources.

CHAPTER 6

SUMMARY

Inheritance of resistance to bacterial leaf streak was investigated in 23 barley cultivars. In one complete diallel set with six parents and 23 other resistant x susceptible and resistant x resistant crosses, parents, F_1 s and F_2 s were inoculated with one Montana barley isolate (X-67) of Xanthomonas campestris pv. translucens in the field.

Parents, F_1 s and F_2 s of each cross were grown together in a small area. One very susceptible cultivar (CM 67) was planted as the spreader in every third row. Before the inoculation, all entries were sprinkler irrigated; and only the plants in the spreader rows were wounded. Inoculation was carried out by spraying bacterial suspension. Plots were sprinkler irrigated at three day intervals. Disease reaction was rated when the infection severity of the spreader rows was very high. A 1 to 9 scale was used to classify plants in the case of disease reactions (1: completely resistant; 9: very susceptible).

In the ranges of resistant and susceptible parents reaction means $\pm$ twice standard deviations were assumed as the ranges of resistant and susceptible classes; that of F_1 was used to detect the individuals which have F_1 genotype in F_2 populations. The degree of dominance was estimated by calculating dominance deviation values. Chi-square and homogeneity tests were used to determine gene numbers and common genes of resistant cultivars.

Early artificial inoculation caused high infection of spreader rows and provided sustained inoculum for the adjacent rows through the growing season. Sprinkler irrigation promoted the infection process by splashing the bacterial cells from infected plant rows to the others, and by creating dew on the plant surface thus helping bacterial movement and invasion. All factors mentioned above resulted in a uniform infection. The experimental error was reduced by planting of parents, F_1 s and F_2 s of each cross together and comparing their disease reaction in a small area.

Most of the early plants in the 14 F_2 populations were found to be susceptible. This relation was strongly attributed to the linkage between these two characteristics. Beside linkage, stomatal opening and population size of the pathogen could be involved. In

order to develop early and resistant varieties to bacterial leaf streak, Barley cultivars, CI 12595, CI 12787, PI 382511, and PI 383077, that gave early and resistant plants in their F_2 populations, should be used.

The results obtained from the diallel set suggested that resistance in Herta, Summit, Oderbrucker and Betzes is the same with regard to gene action (recessive), however, may be different in regard to gene numbers. Resistance is probably polygenic in Herta, and conditioned by one recessive gene in Summit, one or two recessive genes in Oderbrucker and two recessive genes in Betzes. The crosses among these cultivars indicated that they have at least one common resistance gene or different linked resistance genes.

The crosses with a common susceptible parent (CM 67) showed that CI 12558, CI 12777 and CI 12866 differ in reaction to bacterial leaf streak. Resistance is probably conditioned by polygenes in CI 12558, by two recessive genes in CI 12777 and by two dominant genes in CI 12866. More study is needed to determine the possibility of linkage.

Results of resistant line crossed with the susceptible cultivar, SW 191, suggest that CI 12787, CI 13095, PI 382732 and PI 383077 may have one or two

dominant resistance genes in common or have different genes conditioning like effects. PI 382511 and PI 382650 may have two dominant resistance genes with different gene interactions.

Result of Kangbori (one of the susceptible cultivars) crosses with some resistant cultivars indicated that Alpine, Luther and CI 12777 may have two dominant genes which might be the same or different with like effects. Progeny from other Kangbori crosses suggested that resistance may be determined by two dominant genes in CI 12866, by one intermediate gene in CI 12569, and one or two dominance genes in PI 382730.

This study showed that there are diverse resistance sources of barley for bacterial leaf streak. Some of these sources may have identical or different genes in the same chromosomes. Further study is needed to determine if these genes are identical, linked, or independent.

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