A GENETIC STUDY OF BACTERIAL BLIGHT RESISTANCE IN FIVE LINES OF AMERICAN UPLAND COTTON

By

DOUGLAS FARRAR OWEN

Bachelor of Science
Texas Technological College
Lubbock, Texas
1951

Master of Science
Oklahoma State University
Stillwater, Oklahoma
1961

Submitted to the Faculty of the Graduate School of
the Oklahoma State University
in partial fulfillment of the requirements
for the degree of
DOCTOR OF PHILOSOPHY
May, 1965

A GENETIC STUDY OF BACTERIAL BLIGHT RESISTANCE

IN FIVE LINES OF AMERICAN UPLAND COTTON

Thesis Approved:

Jay C. Murray
Thesis Adviser

James S. Brooks

Lloyd A. Brinkerhoff

Robert M. Reed

Dean of the Graduate School

ACKNOWLEDGEMENTS

The author wishes to express sincere appreciation to Dr. Jay C. Murray for his guidance and encouragement throughout the course of this study and for his constructive criticism in the preparation of the dissertation. Gratitude is expressed to Dr. L. A. Brinkerhoff for furnishing the bacterial blight pathogen used in the study and for assistance in inoculating and grading the plants. The author would also like to thank the members of his advisory committee: Dr. Jay C. Murray, Dr. James S. Brooks, Dr. Robert Reed, and Dr. Lloyd A. Brinkerhoff for their valuable advice and assistance in writing this dissertation.

Gratitude is expressed to Jerome Simmons, Margaret Simmons, E. F. Young, Jr., Ed Oswalt, Jerry Baker, and Herman Waters for their able assistance in this study.

Grateful acknowledgement is extended to Dr. Franklin P. Gardner and the Oklahoma State University Department of Agronomy for providing assistantship funds and facilities for conducting the study.

Appreciation is also expressed to the National Cotton Council Foundation for Cotton Research and Education for the Graduate Fellowship from September, 1963 until September, 1964.

The author is forever grateful to his wife, Wanda, for typing the manuscript and for her faith, patience, and encouragement.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF TABLES, continued

CHAPTER I

INTRODUCTION

Bacterial blight of cotton is found in all countries where cotton is grown. In the United States, it is particularly severe in Oklahoma, Texas, and New Mexico.

The causal organism is Xanthomonas malvacearum (E. F. Sm.) Dows., and is capable of affecting all the above ground parts of the cotton plant. The bacteria may overwinter on the surface of the seed, within the seedcoat, and on diseased cotton stalks and bolls from the previous crop. Volunteer seedlings from infected bolls can be responsible for early appearance of the disease. The bacteria are spread from old crop residue and from infected leaves principally by wind-driven, splashing rain and by irrigation water.

Disease symptoms appear on the leaves as water-soaked angular lesions which turn brown or black when dry. On the bolls the lesions are round and water-soaked when fresh, but usually appear black and sunken when dry. The disease produces black elongated lesions on the stems and fruiting branches of very susceptible varieties. Because of the various symptoms the disease is commonly referred to as "angular leaf spot," "boll blight," and "blackarm." Blackarm does not occur on upland cottons in the United States as often as angular leaf spot and boll blight, but in other countries, and in the Sea Island and Egyptian cottons it may be severe.

Leaf infection maintains the disease in the field during the entire growing season and provides a source of inoculum for boll infection. In this study the degree of infection on the leaves was the criteria for studying the reaction of the parents and hybrids to the disease. Reaction to infection may vary from none (immune) to large angular watersoaked lesions (susceptible). Severe leaf infection may cause partial defoliation, reduction in photosynthetic activity and yield, but the boll blight phase of the disease probably causes the greatest economic loss. In this stage the bacteria penetrate the ovary wall and enter the locule. The bacterial slime stains the fibers, thus reducing the grade of the lint and the blight lesions provide a port of entry for many boll-rotting fungi which normally do not infect healthy bolls.

One of the most effective methods of controlling the disease is the use of resistant varieties. Varieties having some resistance to bacterial blight have been known for many years, and it has been shown that many varieties will produce individual plants having varying degrees of resistance to the disease. Plant breeders have used this variability to develop blight resistant varieties, but relatively few genetic studies have been conducted to determine the inheritance of resistance to bacterial blight. Therefore, the object of this study is to determine the genetic control of bacterial blight resistance in five lines of upland cotton.

CHAPTER II

LITERATURE REVIEW

Various literature reviews were presented by Bird and Blank (1951), Brinkerhoff et al. (1952), Smith (1953), Russell (1955), Green and Brinkerhoff (1956), Bird and Hadley (1958), and Brinkerhoff (1963).

Knight is the outstanding worker in the study of the genetic control of resistance to bacterial blight. The results of his experiments were published in a series of papers (1939, 1941, 1944, 1947, 1948, 1950, 1953, 1954, 1963). Knight and Clouston (1939) reported that two factors for resistance, designated B_1 and B_2, were found in Gossypium hirsutum L. variety Uganda B31. Knight (1948) suggested that factor B_2 was the standard factor controlling resistance in American upland cottons and that the highest degree of resistance was obtained with B_2 in conjunction with complexes of minor and modifying genes. In Gossypium arboreum L. Knight (1953) found a resistance-intensifying gene, B_{6m}, that confers no resistance when alone, but associated with B_2B_3 confers a resistance closely approaching immunity.

Brinkerhoff et al. (1952) reported the occurrence of blight resistant plants in 10 of 18 American upland varieties surveyed and tolerant plants in three additional varieties. Green and Brinkerhoff (1956) reported that three different single dominant genes controlled

the resistance in three lines developed from the varieties surveyed. The identification of three different genes does not confirm the suggestion by Knight (1948) that B_2 is the standard factor controlling resistance in American upland cotton. In all of Knight's studies the genes for resistance were transferred to a Sakel (Gossypium barbadense) background; therefore it may be questioned whether or not the results would be the same for the same genes in other (G. hirsutum) backgrounds. He found that B_1 and B_2 conferred greater resistance to Uganda B31 than to Sakel but he attributed this to the presence of modifying factors in Uganda B31 (1939).

In 1953 Knight reported that the resistance developed in Stoneville 20 by Simpson and Weindling (1946) was controlled by a single dominant gene which he designated B_7. Simpson had previously reported to Blank (1951) that his research indicated that resistance in Stoneville 20 was recessive. Bird and Blank (1951) also found that Stoneville 20 resistance appeared to be recessive and Green and Brinkerhoff (1956) reported that Stoneville 20 resistance in a cross with Acala 892 was controlled by a major recessive gene but that segregation was obscured by other genes with less effect. Bird and Hadley (1958) reported that conflicting data could be obtained when only F_1 and F_2 generations were studied in a single genetic background. They found that in the Stoneville 20 x Deltapine cross, resistance appeared to be dominant; whereas in the Stoneville 20 x Acala cross, resistance seemed to be recessive. In the Stoneville 20 x Stoneville 2B cross no dominance was indicated and when the data from all crosses were analyzed statistically there was no evidence of dominance.

Hunter and Brinkerhoff (1963) reported that all of the known genes for resistance to bacterial blight are being transferred to an Acala (G. hirsutum) background. Subsequent tests for homology will be conducted to determine if the genes for resistance found in American varieties other than Stoneville 20 are the same as those reported by Knight.

Much of the differences in experimental results by different researchers may be attributed to variability of the pathogen. Brinkerhoff (1963) cited several instances where resistant strains developed by Knight may have been attacked when grown in areas other than the Sudan. Rose (1959) reported a breakdown of Knight's blight resistant cottons in the Sudan. Hunter and Blank (1954) described a new race that is widespread now in Texas and New Mexico where resistant varieties have been grown. Brinkerhoff described ten new races of X. malvacearum in addition to the two previously known races 1 and 2. Several of the new races were isolated from lesions that developed on previously resistant plants.

Environmental conditions also contribute a great deal to the differences in experimental results. Hutchinson (1959), referring to Knight's work, stated that the difference between major and minor genes controlling resistance depends more on environment than on the magnitude of the gene effect. In other words, in some environments a given gene may confer a high degree of resistance, whereas under a different environment it may have an entirely different reaction. Hutchinson (1959) also said that at locations in Africa other than the Sudan a failure of uniform incidence of the disease prohibited workers from following the segregation of the major genes described by Knight.

The differences Hutchinson assumed to be caused by environment could have been caused by different races of bacteria at the different locations. Brinkerhoff and Hunter [1] reported different disease reactions of some races and differentials when grown under different environments in Texas and Oklahoma. Some of the difference was due to different individuals doing the grading and to different grading systems used. The special committee for designating races of X. malvacearum reported to the Cotton Disease Council (1964) that the races are not stable and a simple and effective method of preserving cultures is needed. They also concluded that "other factors contributing to differing pathogenicity grades are soil fertility, weather conditions and individuals grading the tests." Brinkerhoff (1963) presented evidence that races of X. malvacearum and differential strains of upland cotton reacted differently at high and low temperatures and high and low relative humidities. In general, the differentials tend to become more resistant at high temperatures and low relative humidities. This evidence confirmed observations by Stroughton (1929) and Weindling (1948).

In addition to variability of the pathogen and environmental conditions, the age, nutrition and general growing conditions of the plants may influence the type of reaction obtained. Weindling (1949) found that younger leaves became more severely diseased than

---

[1] Cotton Disease Investigations in Oklahoma in 1962. The Cotton and Cordage Fibers Branch, Crops Research Division, ARS, USDA and Dept. of Botany and Plant Pathology, Oklahoma Experiment Station, Oklahoma State University.

the more mature leaves and that plants growing under moist conditions were more susceptible than plants growing under relatively dry conditions. Low nitrogen levels increase the severity of the disease symptoms. Bird and Smith (1961) included nitrogen fertilization as one of the control methods used in combating the disease. Bird and Joham (1959) found that 80 pounds of nitrogen per acre caused tolerant plants to become resistant. They reported that nitrate nitrogen was better in increasing resistance than ammonia nitrogen. Bird (1963) has found that potassium and phosphorus are involved in the bacterial blight resistance mechanism in that the P-K nutrition level may govern the influence of nitrogen, and that strains having different levels of resistance may respond differently to nutrition levels.

The lines carrying the $B_{L?}$ gene (Brinkerhoff, 1963) which were used in this study were developed from the varieties screened by Brinkerhoff et al. (1952). Preliminary genetic studies by Green and Brinkerhoff [2/] indicated that resistance was controlled by a single dominant gene, but chi-square values did not show a good fit of the data to the expected ratios and further study was proposed. In 1960 Murray et al. [3/] conducted a genetic study of an F_2 population resulting from a cross between the CR-4 line having the $B_{L?}$ gene

---

2/ Annual Report of the Cotton Breeding Research Program, Oklahoma Agricultural Experiment Station and Cotton and Cordage Fibers Research Branch USDA. 1956.

3/ Oklahoma Cotton Breeding and Genetics Program - 1960. Processed Series P-380. April, 1961.

and the susceptible Parrott variety. Again no clear cut ratios were obtained. There appeared to be a large environmental effect and perhaps incomplete dominance. In 1961 blight resistant strains having the $B_{L?}$ gene were studied for linkage. The F_2 populations of crosses between resistant lines and multiple dominant marker stock T-586 and multiple recessive marker stock T-582 were observed; however no new cases of linkage were found.4/

4/ Oklahoma Cotton Breeding and Genetics Program - 1961. Processed Series P-429. October, 1962.

CHAPTER III

MATERIALS AND METHODS

Resistant lines:

The five blight resistant lines used in this study were CR-4, 4-11, 17-3, PI 201626, and 2-B-4-B-4-1-1. CR-4 and 4-11 are sister strains developed from the Stormmaster variety, and 17-3 was developed from a resistant plant of the Lankart 57 variety. PI 201626 came from an Ethiopian introduction and 2-B-4-B-4-1-1 was developed from a resistant plant of an unknown variety.

The lines had been maintained by self-pollination and selection of resistant plants. Plants grading 3.0 and 4.0 were discarded. The seed from plants having the same grade were bulked and after several years of inbreeding the lines were grown in isolated blocks and maintained by mass selection of resistant plants.

Field procedures:

The method of inoculation used was that described by Brinkerhoff et al. (1952) and Brinkerhoff (1963). The inoculum was Race 1 of X. malvacearum provided by Dr. L. A. Brinkerhoff of the Oklahoma State University Botany and Plant Pathology Department. Hand operated single-nozzle guns from a power sprayer operated at about 400 psi produced visible water soaking of the leaves without serious mechanical damage. Disease symptoms appeared within 7 to 14 days. The grading system used was that described by Brinkerhoff et al. (1952) and Brinkerhoff (1963), and is summarized in Appendix Table XXIII. Grades 0.0, 0.1,

0.2, 1.0, 1.2 and 2.0 were considered resistant; grades 0.3, 1.3, 2.3, and 3.0 were intermediate; and grade 4.0 was considered fully susceptible. Brinkerhoff (personal communication) has data from 1963 tests indicating that grades 0.3 and 1.3 belong in the resistant class, but this would not change the results of this study since numbers in these two classes are relatively small and the resistant and intermediate classes were combined when ratios were calculated.

In May 1961, the five blight-resistant lines and the susceptible Acala 44 variety were planted in the breeding nursery at Perkins, Oklahoma. When the plants were young, prior to flowering, they were inoculated with a race of X. malvacearum thought to be Race 1, but it was later found to be a mutant race. The resistant parents were immune and the Acala parent was susceptible. Crosses were made between the resistant lines and Acala 44. There was some indication in the F_1 and F_2 generations the following year that the use of the mutant race may have allowed some plants to be used as parents which normally would have been classified as susceptible or intermediate when inoculated with Race 1.

In the spring and summer of 1962, the F_1 generation of all the 1961 crosses and the S_1 generation of the resistant parents were grown, inoculated with Race 1, graded and selfed. Resistant plants of CR-4, 17-3, and 2-B-4-B-4-1-1 were crossed with Acala 44. Resistant plants of 17-3 and PI 201626 were crossed with resistant plants of CR-4. These crosses were harvested in October and the seed sent to Iguala, Mexico, to provide seed for an F_2 generation the following spring.

In September 1962 selected resistant F_1 and susceptible Acala parental plants were transplanted to the greenhouse in Chickasha. Backcrosses were made and the F_1 plants were selfed. These selfs and

backcrosses were harvested in March of 1963 and the F_2 and backcross populations were grown in the nursery at Perkins, Oklahoma. These plants were inoculated and graded and the F_2 population was self-pollinated. Supplemental irrigation was used on all populations.

Growth Room procedure:

Some of the backcross seed from the 1962 backcrosses in the greenhouse at Chickasha and F_3 seed from the F_2 plants in the 1963 nursery at Perkins were grown during the fall and winter of 1963-64 in growth rooms at an approximate temperature of 80° F. and approximately 60% relative humidity. The humidity could not be regulated exactly but no great fluctuations occurred.

Inoculum was obtained from susceptible cotyledons of Acala 44 seedlings that had been inoculated with a concentrated broth culture of Race 1 of X. malvacearum. Four or five severely infected cotyledons were macerated in a sterile mortar containing 10 ml of sterile water and the solution was filtered through several layers of cheesecloth to remove the plant tissue. The filtrate was added to 90 ml of sterile water to make up 100 ml of inoculum.

The backcross seedlings were inoculated by injecting the bacterial suspension into the cotyledon with a small hypodermic needle attached to a 5 ml glass syringe. The F_3 seedlings were inoculated by scratching the cotyledons with the hypodermic needle and syringe filled with inoculum.

The backcross seedlings were graded resistant, intermediate or susceptible but the F_3 seedlings were graded on a scale of 0 to 5. Grade 0 was given to seedlings having an immune reaction; grade 1 represents seedlings having a few very small round dry lesions; grade 2 describes seedlings having more numerous and slightly larger round or angular dry lesions; grade 3 seedlings had relatively few small single angular

wet lesions; grade 4 describes seedlings with wet angular lesions intermediate in size and number between grade 3 and 5; and grade 5 represents seedlings having large wet, coalesced lesions. The CR-4 checks graded 1 and 2 and the Acala 44 checks all graded 5. Therefore, those seedlings which were graded 2 and below were considered to be resistant, while those which were graded 3 or above were called susceptible.

CHAPTER IV

RESULTS AND DISCUSSION

The results and discussion part of this study is divided into five sections according to the data obtained from each one of the five resistant lines of cotton listed in Chapter III. Ths discussion of the CR-4 material is divided into two parts: field data and seedling data. Seedling data were not obtained from any of the other lines of resistance.

Section A: PI 201626

The S_1 data (Table I) and the grade distribution of the (Acala 44 x PI 201626)F_2 (Table II) indicate that resistance is controlled by a single gene in PI 201626. The F_1 plants were given susceptible classifications because they did not show as much resistance as the PI 201626 parent. The F_2 and S_1 data indicate that resistance in this material is not completely dominant; therefore, the F_1 plants probably should have been classified as intermediate between the PI 201626 and Acala 44 parents.

The grading distribution of the F_2 generations of a reciprocal cross of PI 201626 x CR-4 (Table III) is in agreement with the data presented in Tables I and II and shows that PI 201626 and CR-4 have different genes for resistance to bacterial blight. There are some differences in the grade distributions of the two populations but this could be due to slight differences in the parents used or different physiological responses to environmental conditions.

TABLE I

DISEASE REACTION OF (ACALA 44 x PI 201626)F_1 AND (PI 201626)S_1 PLANTS

Pedigree	Grade R	Grade R-	Grade S	X^2 for 3:1 ratio
(Acala 44 x PI 201626)F_1	0	0	15	- -
(PI 201626)S_1	1	16	2	2.12

TABLE II

GRADE DISTRIBUTION OF THE (ACALA 44 x PI 201626)F_2 POPULATION

Pedigree	Grade R				Grade I				Grade S		X^2 for 3:1 ratio
	0.1	0.2	1.2	2.0	0.3	1.3	2.3	3.0	4.0	tot.	
(Acala 44 x PI 201626)F_2	1	8	3	20	3	12	65	17	32	161	2.26

Data presented in Section E suggest that CR-4 has three genes for resistance. The (CR-4 x PI 201626)F_2 is segregating for two genes instead of three or more indicating that part of the PI 201626 and CR-4 resistance is the same; however, if the CR-4 parent used in the cross had two pair of recessive alleles, a 15:1 ratio could be obtained even though CR-4 had three genes for resistance.

TABLE III

GRADE DISTRIBUTION OF F_2 POPULATIONS OF RECIPROCAL CROSSES OF PI 201626 x CR-4

Pedigree	Grade												X^2 for 15:1 ratio
	R					I				S			
	0.0	0.1	0.2	1.2	2.0	0.3	1.3	2.3	3.0	4.0	tot.		
(PI 201626 x CR-4)F_2	29	0	10	0	0	7	1	2	1	1	51		1.6
(CR-4 x PI 201626)F_2	10	2	38	6	35	24	13	21	23	6	178		2.5

Section B: 17-3

The disease reaction of (Acala 44 x 17-3)F_1 and (17-3)S_1 plants (Table IV) indicates that resistance in 17-3 is controlled by a recessive gene and the S_1 data indicate that the 17-3 parent was segregating 3 susceptible to 1 resistant.

TABLE IV

DISEASE REACTION OF (ACALA 44 x 17-3)F_1 AND (17-3)S_1 PLANTS

Pedigree	Grade			X^2 for 1:3 ratio
	R	R-	S	
(Acala 44 x 17-3)F_1	0	0	16	- -
(17-3)S_1	1	4	15	0.0

The grade distribution of the (Acala 44 x 17-3)F_2 population (Table V) indicates that the F_2 population is segregating 15 resistant to 1 susceptible; thus indicating that resistance in the 17-3 material is dominant and controlled by two genes. These data do not agree with the F_1 and S_1 data (Table IV). One explanation might be the possibility that the F_1 and S_1 plants that were graded susceptible should have been classified as intermediate. On the other hand, another race of the pathogen capable of attacking plants resistant to Race 1 might have developed and caused the susceptible reaction.

TABLE V

GRADE DISTRIBUTION OF THE (ACALA 44 x 17-3)F_2 POPULATION

Pedigree	Grade										X^2 for 15:1 ratio
	R				I				S		
	0.0	0.2	1.2	2.0	0.3	1.3	2.3	3.0	4.0	tot.	
(Acala 44 x 17-3)F_2	2	104	1	26	40	2	50	31	13	269	0.92

The grade distribution of the (PI 201626 x 17-3)F_2 population (Table VI) is a good fit for a 63:1 ratio, although no susceptible plants were recovered. Since evidence has been presented to show that PI 201626 has a single gene for resistance and that 17-3 has two genes for resistance, it is reasonable to assume that the (PI 201626 x 17-3)F_2 population is segregating for three genes for resistance.

TABLE VI

GRADE DISTRIBUTION OF THE (PI 201626 x 17-3)F_2 POPULATION

Pedigree	Grade											X^2 for 63:1 ratio
	R					I				S		
	0.0	0.1	0.2	1.2	2.0	0.3	1.3	2.3	3.0	4.0	tot.	
(PI 201626 x 17-3)F_2	5	3	14	6	6	20	10	29	0	0	93	1.68

Section C: 2-B-4-B-4-1-1

The disease reaction of the (2-B-4-B-4-1-1)S_1 and (Acala 44 x 2-B-4-B-4-1-1)F_1 is given in Table VII. The F_1 data has a 3.125 chi-square value for a 1:1 ratio indicating that the resistant parent had one gene for resistance. The S_1 data indicate that the resistant parent was heterozygous and was segregating for a single gene for resistance to bacterial blight.

TABLE VII

DISEASE REACTION OF (ACALA 44 x 2-B-4-B-4-1-1)F_1 AND (2-B-4-B-4-1-1)S_1 PLANTS

Pedigree	Grade			Ratio	X^2
	R	R-	S		
(Acala 44 x 2-B-4-B-4-1-1)F_1	0	21	11	1:1	3.125
(2-B-4-B-4-1-1)S_1	5	16	4	3:1	1.09

The (Acala 44 x 2-B-4-B-4-1-1)F_2 data (Table VIII) support the data presented in Table VII and show in addition that resistance is not completely dominant.

No data were obtained which would distinguish between 2-B-4-B-4-1-1 and the other four blight resistant lines studied, but the data presented in other sections indirectly suggest that the 2-B-4-B-4-1-1 line is not genetically the same as the 17-3, CR-4 and 4-11 lines.

TABLE VIII

GRADE DISTRIBUTION OF THE (ACALA 44 x 2-B-4-B-4-1-1)F_2 POPULATION

Pedigree	Grade									X^2 for 3:1 ratio
	R			I				S		
	0.2	1.2	2.0	0.3	1.3	2.3	3.0	4.0	tot.	
(Acala 44 x 2-B-4-B-4-1-1)F_2	6	9	109	3	5	99	62	80	373	2.51

Section D: 4-11

The disease reaction of the (Acala 44 x 4-11)F_1 population (Table IX) indicates that resistance is dominant in this material and that the population is segregating 1:1. Unfortunately, no selfed seed were obtained from the 4-11 parent plants; consequently, the S_1 generation is missing from these data.

The F_2 and backcross populations (Table X) do not have enough susceptible plants to fit 3:1 and 1:1 ratios respectively. This might be due to classifying too many grade 4.0 plants in the grade 3.0 classification.

TABLE IX

DISEASE REACTION OF THE (ACALA 44 x 4-11)F_1 POPULATION

Pedigree	Grade R	R-	S	X^2 for 1:1 ratio
(Acala 44 x 4-11)F_1	0	14	9	1.09

TABLE X

GRADE DISTRIBUTION OF THE (ACALA 44 x 4-11)F_2 AND (ACALA 44 x 4-11)F_1 x ACALA 44 POPULATIONS

Pedigree	Grade R			I				S			
	0.2	1.2	2.0	0.3	1.3	2.3	3.0	4.0	tot.	ratio	X^2
(Acala 44 x 4-11)F_2	59	2	43	3	0	71	28	39	245	3:1	10.8
(Acala 44 x 4-11)F_1 x Acala 44)	31	0	0	6	3	9	17	41	107	1:1	5.84

Section E: CR-4

Field data:

The disease reaction of the (Acala 44 x CR-4)F_1 and (CR-4)S_1 populations (Table XI) indicates that resistance is dominant in this material and that the CR-4 parents used in the crosses were not homozygous for resistance. A comparison of the grade distributions of the progenies arising from the susceptible and the resistant F_1 plants substantiates this conclusion (Table XIII).

TABLE XI

DISEASE REACTION OF THE (ACALA 44 x CR-4)F_1 AND (CR-4)S_1 POPULATIONS

Pedigree	Grade			Ratio	X^2
	R	R-	S		
(Acala 44 x CR-4)F_1	1	27	29	1:1	0.02
(CR-4)S_1	6	10	7	3:1	0.36

The (Acala 44 x CR-4)F_2 had too many susceptible plants for a good fit to a 3:1 ratio, but when adjusted according to the disease reaction of F_3 seedlings there was a satisfactory fit to a 3:1 ratio (Table XII). The (CR-4 x PI 201626)F_2 gave a chi-square value of 2.5 for a 15:1 ratio indicating that the two lines have different genes for resistance (Table XII). The (CR-4 x 17-3)F_2 seemed to be segregating for three genes for resistance (Table XII). This agrees with data presented in the 17-3 section showing that resistance in 17-3 is controlled by two genes. The

backcross data (Table XII) gave a satisfactory fit to a 1:1 ratio, but only four of the 101 plants graded had as much resistance as the resistant parents. A possible explanation for this is presented later in connection with the seedling data.

The data presented in Tables XI and XII lend support to the hypothesis that resistance in the CR-4 line is simply inherited. However, the (Acala 44 x CR-4)F_2 data came from both resistant and susceptible F_1 plants and if these F_1 plants are genetically different (Table XI) the validity of the F_2 data is questionable. With the F_1 plants segregating 1:1 and a single gene conferring resistance, the F_2 should be 62.5% susceptible. The fact that the F_2 was only 28.46% susceptible and the adjusted F_2 was only 26.97% susceptible is strong evidence that CR-4 has several pairs of genes for resistance to bacterial blight.

Data showing the grade distribution of (Acala 44 x CR-4)F_2 plants from resistant and susceptible F_1 parents (Table XIII) indicate that inheritance of resistance is not simple but may be rather complex. The progeny of the R and R- F_1 parents would be expected to segregate. However, if the inheritance of resistance were relatively simple, the progeny of the S parents should be homozygous and no segregation would be expected. The data show that the progeny of the R and R- parents did segregate as expected but, contrary to expectations, the progeny of the S parents also segregated. Data from F_3 seedlings (Table XXII) show that the progeny of susceptible F_2 plants also segregate. This is more evidence that resistance to blight in the CR-4 material is not simply inherited, but is controlled by several pairs of genes. An attempt to reconcile the apparent conflict in data will be made in the following discussion of the seedling data.

TABLE XII

GRADE DISTRIBUTION OF THE (ACALA 44 x CR-4)F_2, ADJUSTED (ACALA 44 x CR-4)F_2, (CR-4 x PI 201626)F_2, (CR-4 x 17-3)F_2, and (ACALA 44 x CR-4)F_1 x ACALA 44 BACKCROSS POPULATIONS

Pedigree	Grade												
	R					I				S			
	0.0	0.1	0.2	1.2	2.0	0.3	1.3	2.3	3.0	4.0	tot.	ratio	X^2
(Acala 44 x CR-4)F_2	1	3	100	19	114	21	5	80	137	191	671	3:1	4.29
Adjusted by F_3	1	3	100	19	114	21	3	75	154	181	671	3:1	1.39
(CR-4 x PI 201626)F_2	10	2	38	6	35	24	13	21	23	6	178	15:1	2.50
(CR-4 x 17-3)F_2	2	1	21	0	4	17	9	19	0	0	73	63:1	1.16
(Acala 44 x CR-4)F_1 x Acala 44	0	0	1	0	3	0	0	32	8	57	101	1:1	1.67

TABLE XIII

GRADE DISTRIBUTION OF (ACALA 44 x CR-4)F_2 PLANTS SEPARATED ACCORDING TO THE GRADE OF THE F_1 PARENTS

F_1 Grade	No. of F_1 Plants	F_2 Grade										
		R					I				S	
		0.0	0.1	0.2	1.2	2.0	0.3	1.3	2.3	3.0	4.0	total
R & R-	18	1	3	74	13	84	16	2	36	44	38	310
S	25	0	0	8	2	22	0	0	37	78	138	285

Seedling data:

The disease reaction of seedlings resulting from a backcross of resistant (Acala 44 x CR-4)F_1's to Acala 44 (Table XIV) fit a three gene hypothesis very well.

TABLE XIV

DISEASE REACTION OF THE SEEDLINGS OF THE BACKCROSS (ACALA 44 x CR-4)F_1 x ACALA 44

Pedigree	Grade R	I	S	Total	X^2 for 7:1 ratio
(Acala 44 x CR-4)F_1 x Acala 44	162	184	51	397	0.0435

Although this data conflicts with the data of the field grown testcrosses presented in Table XII, the three gene hypothesis better explains the behavior of CR-4 than a one or two gene hypothesis. If a single major gene and two minor genes were postulated, this could explain why Green and Brinkerhoff (unpublished data) obtained too many resistant plants to fit a single gene hypothesis. If CR-4 is heterozygous for three genes this could explain why the progeny of plants graded 0.2, 1.2, 2.0, 2.3, 3.0, and sometimes 4.0 segregate instead of breeding true. In an (Acala 44 x CR-4)F_1 x Acala 44 testcross progeny the following grade and genotype relationship could be postulated.

Genotype	Grade	Genotype	Grade
AaBbCc	2.0	aaBbCc	3.0
AaBbcc	2.3	aaBbcc	3.0
AabbCc	2.3	aabbCc	3.0
Aabbcc	2.3	aabbcc	4.0

This hypothesis provides an explanation of the excess intermediate and susceptible grades resulting from the backcross to Acala 44 (Table XII), especially if the major gene (gene A) and one minor gene are homozygous recessive. Under certain conditions the minor genes might contribute very little resistance and the plant could easily be classified incorrectly as grade 4.0. This could explain why some of the F_2 hybrids that graded 4.0 produced segregating progeny (Appendix Table XXII). The situation is further complicated by environmental interactions causing plants grown under favorable conditions to appear more resistant than when grown under less favorable conditions. A favorable environment might also permit new races to build up that would attack some of the plants and cause susceptible reactions.

It is also easy to see that a 1:1 ratio could be obtained in this testcross. If the parent CR-4 had the AaBbCc genotype, and gene B conferred more resistance than gene C, then an explanation of the 1:1 ratio (Table XII) can be worked out. If the aabbCc and aabbcc genotypes were phenotypically indistinguishable and both were classified as grade 4.0, the expected number of resistant and susceptible plants in a backcross population of 101 individuals would be 44.3 and 56.7 respectively. This compares favorably to the 44 resistant and 57 susceptible actually observed.

The segregation of the F_1 plants (Table XI) can be explained by this hypothesis. If the resistant parents were heterozygous for the major gene, they could have been graded from immune to 2.0 depending upon environmental effects and whether the minor genes were homozygous or heterozygous. Upon being crossed to Acala 44 the major gene would segregate 1:1 in the F_1 and the minor genes would confer little or no resistance with the major gene in the homozygous recessive condition. The

same premise could be used to explain the 3:1 ratio (Table XII). A resistant parent with a homozygous major gene for resistance crossed with Acala 44 would segregate 3:1 in the F_2 generation, but the segregation would be modified according to the number of homozygous or heterozygous minor genes for resistance present and the genotypic-environmental interactions involved.

Appendix Tables XV through XXII present the grade distribution data of the F_3 generation of (Acala 44 x CR-4)F_2 plants that produced mature, self-pollinated bolls. Due to late selfing and adverse weather conditions only 119 plants of the 671 in the F_2 generation produced selfed bolls, and of these only 113 produced F_3 seedlings.

The F_2 individuals that graded 4.0 should have produced only grade 5 progeny since the Acala checks all graded 5. None of the F_2 plants produced seedlings that were all graded 5 and only 23 of the 49 grade 4.0 families had seedlings that were all graded 3 or above (Table XXII). In other words the susceptible parental type was not recovered in the F_2 or F_3 generation. This is further evidence for the presence of a number of genes for resistance. This evidence is in harmony with that of Knight (1953), Green and Brinkerhoff (1956) and Bird and Hadley (1958) and Brinkerhoff (1963). It also agrees with the three genes postulated in the discussion of Table XIV. Since only 113 F_2 plants produced F_3 families the probability of recovering either of the parental types was not very great.

CHAPTER V

SUMMARY AND CONCLUSIONS

The data do not support the hypothesis that the five lines of cotton studied all have the same single gene for resistance to bacterial blight. Data show that PI 201626 and CR-4 have different genes for resistance, but since CR-4 may be segregating for three genes (Table XIV) the 15:1 ratio (Table III) indicates that part of the resistance in CR-4 and PI 201626 might be the same. On the other hand, if the CR-4 parent used in the cross with PI 201626 had two pair of recessive alleles then a 15:1 ratio would have been obtained even though CR-4 material in general had more than one gene for resistance.

Data presented indicate that 17-3 has two genes for resistance to bacterial blight (Table V) and that 17-3 crossed with PI 201626 and CR-4 segregates in the F_2 generation for more than two genes (Tables VI and XII). This shows that resistance is not controlled by a single gene and that these three lines have different genes for resistance.

Field data (Table XII) indicate that CR-4 has a single gene for resistance, but evidence is presented to show that resistance in CR-4 is controlled by several pairs of genes (Tables XIII, XIV, and XXII). A hypothesis of one major gene and two minor genes is proposed to reconcile the seemingly conflicting data.

The 2-B-4-B-4-1-1 data (Tables VII and VIII) indicate that it has a single gene for resistance, but in view of the conflicting field and

seedling data on CR-4 it might be that 2-B-4-B-4-1-1 behaves similarly and the F_2 data could be misleading. No evidence was found that would distinguish this line from the other four lines.

Even though the testcross data (Table XIV) and the F_3 data (Tables XV through XXII) are good evidence that blight resistance in the CR-4 line is not simply inherited, the question is not resolved and additional research should be undertaken. One of the factors contributing to the difficulty in making a genetic analysis of the material is the human error involved in the grading system. Methods should be developed which would reduce human error to a minimum. Assuming that such error were random and normally distributed it could be reduced by using large populations and replicated tests. Another means of reducing error in grading would be the development of techniques for uniform inoculation and the use of mechanical instruments for measuring the ratio of infected area to a total leaf area. The cost of such equipment might be prohibitive, but if such equipment were available it would greatly reduce human error in grading and it would permit a much larger number of grade classifications to be used. This in turn would facilitate the use of statistical and quantitative methods of analysis to determine the approximate numbers of genes involved, heritabilities, type of gene action and other information useful to geneticists and plant breeders.

Populations of 200 to 400 plants of the parental, F_1, F_2 backcross, and F_3 generations could be grown in replicated tests and Powers' (1950) partitioning method used to separate the various genotypes and to test for gene interaction and the relative magnitude of the gene effects. Known and tested races of the pathogen should be used and plants inoculated under optimum conditions for infection. The age and physiology

of the leaves at the time of inoculation determines to a great extent the type of reaction produced.

The proposal by Hunter and Brinkerhoff (1963) to incorporate all sources of blight resistance into homozygous Acala lines should be carried out and tests for homology conducted as soon as the genes for resistance are homozygous. Allard (1956) stated: "Thus most studies have involved parents at the extremes of the distributional range of the character under consideration. In such hybrids the genetic situation would be expected to be complex. But in homozygous lines derived from the original cross by inbreeding, the genetic situation must be of reduced complexity, and the chances of successful analysis should be correspondingly increased. Studies of such derived lines have been rare." He further says that studies of "subqualitative" genes governing metrical characters are important because the knowledge of their prevalence is inadequate and "knowledge of the properties of genes governing quantitative characters, e.g., their stability in different environments and their interactions with other genes governing quantitative characters, is almost nonexistent."

Since the time Knight first reported that genes for resistance to bacterial blight in cotton were accumulative in their effects, cotton breeders have been striving to find combinations of those genes that give the greatest degree of resistance. One method of determining these would be the use of diallel analysis after the various parents were homozygous. The best lines of general and specific combining ability could be determined for disease resistance, yield, earliness and other characters all from one set of diallel crosses.

The results of this study indicate that caution is necessary when

studying different sources of resistance to bacterial blight. The failure of a cross between two resistant lines to produce susceptible plants in the F_2 generation is not prcof that the two lines have the same genetic composition. This is especially true when resistance ranges from immunity to tolerance. If inheritance of resistance is quantitative and gene action is additive in conferring increased resistance, then susceptible plants would not be expected unless large populations were grown.

The results also indicate that if these lines are to be used as sources of resistance for improved cotton varieties, progeny testing and large populations will be necessary to select the most resistant segregants and to insure against loss of resistance in subsequent generations.

LITERATURE CITED

Allard, R. W. 1956. Biometrical approach to plant breeding. Brookhaven Symp. in Biol. 9:69-85.

Bird, L. S. 1963. Report of the Bacterial Blight Committee. Proc. Cotton Disease Council 23:3-9. Natl. Cotton Council. Memphis, Tenn.

_____ and L. M. Blank. 1951. Breeding Strains of cotton resistant to bacterial blight. Texas Ag. Expt. Sta. Bull. 736,25p.

_____ and H. H. Hadley. 1958. A statistical study of the inheritance of Stoneville 20 resistance to the bacterial blight disease of cotton in the presence of Xanthomonas malvacearum races 1 and 2. Genetics 43:750-767.

_____ and H. E. Joham. 1959. Influence of nitrogen source and carbohydrate change by debudding and girdling on bacterial blight resistance caused by B_7 gene in cotton. Plant Disease Reporter 43:86-89.

_____ and H. E. Smith. 1961. Bacterial blight of cotton. Texas Agr. Expt. Sta. Misc. Publ. MP-534.

Brinkerhoff, L. A. 1963. Variability of Xanthomonas malvacearum, the cotton bacterial blight pathogen. Oklahoma State Univ. Tech. Bull. T-98, 95 p.

_____, J. M. Green, R. Hunter and G. Fink. 1952. Frequency of bacterial blight-resistant plants in twenty cotton varieties. Phytopathology 42:98-100.

Green, J. M. and L. A. Brinkerhoff. 1956. Inheritance for three genes for bacterial blight resistance in upland cotton. Agron. J. 48:481-484.

Hunter, R. E. and L. M. Blank. 1954. Pathogenicity differences of Xanthomonas malvacearum isolates. Phythopathology 44:332.

_____ and L. A. Brinkerhoff. 1963. Report of the bacterial blight committee. Proc. Cotton Disease Council 23:6 Natl. Cotton Council. Memphis, Tenn.

Hutchinson, J. 1959. The application of genetics to cotton improvement. Cambridge Univ. Press, London. 87 p.

Knight, R. L. 1944. The genetics of blackarm resistance IV. Gossypium punctatum Sch. and Thon. J. Genetics 46:1-27.

_____ 1947. The genetics of blackarm resistance V. Dwarf-bunched and its relationship to B_1. J. Genetics 48:43-50.

_____ 1948a. The genetics of blackarm resistance VI. Transference of resistance from Gossypium Arboreum to G. barbadense. J. Genetics 48:359-369.

_____ 1948b. The genetics of blackarm resistance VII. Gossypium arboreum. J. Genetics 49:109-116.

_____ 1950. The genetics of blackarm resistance VIII. Gossypium barbadense. J. Genetics 50:67-76.

_____ 1953a. The genetics of blackarm resistance IX. The gene B_{6m} from Gossypium arboreum. J. Genetics 51:270-275.

_____ 1953b. The genetics of blackarm resistance X. The gene B_7 from Stoneville 20. J. Genetics 51:515-519.

_____ 1954. The genetics of blackarm resistance XI. Gossypium anomalum. J. Genetics 52:466-472.

_____ 1963. The genetics of blackarm resistance XII. Transference of resistance from Gossypium herbaceum to G. barbadense. J. Genetics 58:328-346.

_____ and T. W. Clouston. 1939. The genetics of blackarm resistance I. Factors B_1 and B_2. J. Genetics 38:133-159.

_____ 1941. The genetics of blackarm resistance II and III. J. Genetics 41:391-409.

Powers, L. R., L. F. Locke, and J. C. Garrett. 1950. Partitioning methods of genetic analysis applied to quantitative characters of tomato crosses. U. S. Dept. Agr. Tech. Bull. 998.

Rose, M. F. 1959. Cotton Research and development of the commercial crop in the Sudan. Empire Cotton Gr. Rev. 36:252-262.

Russell, K. M. 1955. Studies on Xanthomonas malvacearum, the cause of bacterial blight of cotton. Ph.D. Dissertation 2723 Newnham College, Univ. of Cambridge.

Simpson, D. M. and R. Weindling. 1946. Bacterial blight resistance in a strain of Stoneville cotton. Agron. J. 38:630-635.

Smith, A. L. 1953. Plant Diseases, The Yearbook of Agriculture. p. 307-311.

Stroughton, R. H. 1929. The relation of environmental conditions to the angular leaf spot disease of cotton. Ann. Appl. Biol. 16:188.

Weindling, R. 1948. Bacterial blight of cotton under conditions of artificial inoculation. U. S. Dept. Agr. Tech. Bull. No. 956. 59p.

Appendix

TABLE XV

DISTRIBUTION OF GRADES OF (ACALA 44 x CR-4)F_3 SEEDLINGS WHOSE F_2 PARENTS WERE GRADED 0.2

F_2 Plant	F_1 Grade	Grade of F_3 Seedling							
		Resistant			Susceptible				%
		0	1	2	3	4	5	Total	Resistant
H30-1	S		14	6	1			21	95.5
H31-8	R-			11	4	12		27	40.8
H31-20	R-			8	4	9		21	38.0
H39-8	R-			7	2	8		17	41.2
H54-12	- -	11		2	1	2	1	17	76.5
total		11	14	34	12	31	1	103	56.2

TABLE XVI

DISTRIBUTION OF GRADES OF (ACALA 44 x CR-4)F_3 SEEDLINGS WHOSE F_2 PARENTS WERE GRADED 0.3

F_2 Plant	F_1 Grade	Grade of F_3 Seedling							
		Resistant			Susceptible				%
		0	1	2	3	4	5	Total	Resistant
H51-8	R-			16	14			30	53.5
H54-14	- -		2	1	8	15		26	11.5
total			2	17	22	15		56	34.0

TABLE XVII

DISTRIBUTION OF GRADES OF (ACALA 44 x CR-4)F_3 SEEDLINGS WHOSE F_2 PARENTS WERE GRADED 1.2

F_2 Plant	F_1 Grade	Grade of F_3 Seedling							
		Resistant			Susceptible			Total	% Resistant
		0	1	2	3	4	5		
G9-10	S		1		3			4	25.0
H55-17	- -			11	6	2		19	58.0
total			1	11	9	2		23	52.2

TABLE XVIII

DISTRIBUTION OF GRADES OF (ACALA 44 x CR-4)F_3 SEEDLINGS WHOSE F_2 PARENTS WERE GRADED 1.3

F_2 Plant	F_1 Grade	Grade of F_3 Seedling							
		Resistant			Susceptible			Total	% Resistant
		0	1	2	3	4	5		
G9-1	S			3	2	3		8	37.5
G-9-8	S				5	1	1	7	0.0
H54-18	- -		31	3				34	100.0
total			31	6	7	4	1	49	75.5

TABLE XIX

DISTRIBUTION OF GRADES OF (ACALA 44 x CR-4)F_3 SEEDLINGS WHOSE F_2 PARENTS WERE GRADED 2.0

F_2 Plant	F_1 Grade	Grade of F_3 Seedling							
		Resistant			Susceptible				%
		0	1	2	3	4	5	Total	Resistant
H12-6	R-		4	1	2			7	71.5
H16-5	R-			5		5		10	50.0
H28-8	S	1	4	3	8			16	50.0
H30-8	S			3	8			11	27.2
H31-1	R-		14		2			16	87.5
H31-3	R-		19	5	2			26	92.5
H31-9	R-			14	2	2		18	78.0
H34-5	R-		6	1	2	3	3	15	46.6
H37-7	S		10		4	1		15	66.6
H38-2	S			12		5		17	70.6
H38-4	S			11		4		15	73.4
H39-3	R-			2		4		6	33.3
H41-2	R-	10		2	1	1	2	16	75.0
H43-2	R-	14		10	3	8	4	39	61.5
H45-1	S	17	6	2	4	4	1	34	73.6
H46-2	R-	3						3	100.0
H46-9	R-	3	1					4	100.0
H50-3	R-	15	7	1		2		25	92.0
H51-1	R-	28						28	100.0
H52-2	R-	6	12			1		19	94.6
		97	83	72	38	40	10	340	74.25

TABLE XX
DISTRIBUTION OF GRADES OF (ACALA 44 x CR-4)F_3
SEEDLINGS WHOSE F_2 PARENTS WERE GRADED 2.3

F_2 Plant	F_1 Grade	Grade of F_3 Seedling							
		Resistant			Susceptible				%
		0	1	2	3	4	5	Total	Resistant
G9-5	S			2	2			4	50.0
G9-9	S				6			6	0.0
H16-6	R-		3	2	7			12	41.6
H28-13	S			11	11			22	50.0
H30-17	S			2			19	21	9.5
H31-7	R-			2	5	16		23	8.7
H38-22	R-			10		8		18	55.5
H39-9	R-	14			4	5	1	24	58.4
H40-13	S			7	9	11	4	31	22.6
H44-6	S		5	5	5			15	66.6
H44-7	S		3	1	3	1		8	50.0
H48-6	S		2	5	2		1	10	70.0
H50-6	R-	12	2		7	1		22	63.6
H53-16	R-			12	4	8		24	50.0
H55-1	- -			19	7	13		39	48.7
H56-3	- -			9		6		15	60.0
H56-28	- -			2	3	26		31	6.5
total		26	15	99	75	95	25	335	41.8

TABLE XXI

DISTRIBUTION OF GRADES OF (ACALA 44 x CR-4)F_3 SEEDLINGS WHOSE PARENTS WERE GRADED 3.0

F_2 Plant	F_1 Grade	Grade of F_3 Seedling							
		Resistant			Susceptible				%
		0	1	2	3	4	5	Total	Resistant
G9-4	S				2		4	6	0.0
H16-3	R-				4	9		13	0.0
H16-7	R-		3	5	4	8		20	40.0
H22-9	S				4	19		23	0.0
H30-5	S				2	15	2	19	0.0
H36-2	S			2	1	5		8	25.0
H37-1	S			2	3	10		15	13.3
H43-3	R-		9	6	4	11	4	34	44.2
H44-10	S		2	2	7			11	36.4
H49-10	S				4			4	0.0
H50-1	R-		8	7	4	3	2	24	62.5
H50-2	R-			1	1	4		6	16.7
H53-25	R-				2			2	0.0
H54-8	- -				4			4	0.0
H55-5	- -				2	19		21	0.0
total			22	25	48	103	12	210	22.4

TABLE XXII

DISTRIBUTION OF GRADES OF (ACALA 44 x CR-4)F_3 SEEDLINGS WHOSE PARENTS WERE GRADED 4.0

F_2 Plant	F_1 Grade	Grade of F_3 Seedling							
		Resistant			Susceptible				%
		0	1	2	3	4	5	Total	Resistant
H12-11	R-					9		9	0.0
H13-2	S				2	7		9	0.0
H13-3	S				9	5		14	0.0
H16-2	R-					9		9	0.0
H17-1	S			2	1	8	5	16	12.5
H17-4	S			1	6	8		15	6.7
H17-8	S		1		6	11	10	28	3.6
H17-9	S				5	2		7	0.0
H19-1	S			1	2	5	12	20	5.0
H20-1	S		1	3	5	13	4	26	15.4
H21-2	S			18	13			31	58.0
H21-6	S		2	4	5	3		14	42.8
H21-10	S			5		13		18	38.5
H21-15	S			2		4	6	12	16.7
H22-1	S			3	17			20	15.0
H22-3	S		1		6	5	8	20	5.0
H22-4	S					7	13	20	0.0
H22-5	S			2		11		13	15.4
H22-8	S					13		13	0.0
H22-11	S					11	6	17	0.0
H22-12	S				1	7	8	16	0.0
H22-13	S				1	28		29	0.0
H22-14	S			1		21		22	4.6
H27-1	S					18		18	0.0
H27-6	S			1	1	12		14	7.2
H27-14	S				1	10		11	0.0
H27-18	S					4	18	22	0.0
H27-22	S			1	3	15	9	28	3.6
H29-1	S			2		16		18	11.1
H30-1	S			1	5			6	16.7
H35-1	S				2	6		8	0.0
H36-3	S				2	13		15	0.0
H37-2	S				3	4		7	0.0
H37-4	S					15		15	0.0
H37-5	S					11		11	0.0
H38-3	R-				3	2		5	0.0
H40-23	S		3	7	6	13		29	34.5
H41-28	R-	5	3		5	11	5	29	27.6

Table XXII continued

F_2 Plant	F_1 Grade	Grade of F_3 Seedling							
		Resistant			Susceptible				%
		0	1	2	3	4	5	Total	Resistant
H42-1	S		2		7	10	12	31	6.5
H44-4	S		1			1	1	3	33.3
H46-19	R-				8	11		19	0.0
H48-1	S				1	6	11	18	0.0
H49-1	S		3	7	9	10		29	34.5
H49-13	S			1	2	7	12	22	4.6
H50-5	R-				6	10	10	26	0.0
H51-25	R-					14		14	0.0
H53-23	R-					20		20	0.0
H53-27	R-				2	26		28	0.0
H54-1	- -					11		11	0.0
total		5	17	62	145	476	150	855	9.8

TABLE XXIII

A SUMMARY OF LEAF GRADES USED IN CLASSIFYING BLIGHT INFECTION IN THE FIELD [1]

Grade	Host Reaction	Description of infection type or types
0	Immune	No visible lesions
1	Resistant	Dry pin-point to small round lesions
2	Resistant	Dry small angular lesions between veins; sometimes dry vein lesions
3	Mildly susceptible	Small to intermediate, angular, wet lesions between veins; intermediate to water-soaked vein lesions
4	Susceptible	Large, water-soaked, angular lesions that turn black on drying; large water-soaked vein lesions
Mesothetic reactions		
0.1, 0.2 & 0.3	Resistant	Predominantly immune; with a few lesions of infection types 1, 2, 3; or, more than one type may be present
1.2 & 1.3	Resistant	Predominantly type 1; with type 2, or types 2 and 3 lesions present
2.3	Intermediate	Predominantly type 2, but with type 3 lesions also present; type 1 lesions may also be present especially if the environment does not favor disease expression

[1] From original table. Brinkerhoff, L. A. 1963. Variability of Xanthomonas malvacearum, the cotton bacterial blight pathogen. Oklahoma State Univ. Tech. Bull. T-98, 95p.

VITA

Douglas Farrar Owen

Candidate for the Degree of

Doctor of Philosophy

Thesis: A GENETIC STUDY OF BACTERIAL BLIGHT RESISTANCE IN FIVE LINES OF AMERICAN UPLAND COTTON

Major Field: Genetics and Plant Breeding

Biographical:

Personal data: Born February 21, 1930 at Olton, Texas, the son of Charles M. and Leila Ellen Owen.

Education: Attended elementary school at Olton, Texas, graduated from Olton High School in 1947, received a Bachelor of Science degree from Texas Technological College with a major in Agricultural Education, in 1951. Received Master of Science Degree in Agronomy from Oklahoma State University, 1961.

Professional experience: Born in rural community and worked on the farm through high school and college years. Employed by Western Cottonoil Co., Littlefield, Texas, 1951-1955. Entered U. S. Army in January, 1955, and served at the Army Intelligence Center, Counter Intelligence Corps, Fort Holabird, Baltimore, Maryland, until January, 1957. Employed as an agent for the National Farm Life Insurance Co., Fort Worth, Texas, 1957. Served as Assistant Agronomist for the High Plains Research Foundation, Halfway, Texas, 1957-1959. Employed as Graduate Research Assistant in the Agronomy Department, Oklahoma State University, 1959-1963. National Cotton Council Graduate Fellowship, 1963-1964.

Member of: Alpha Zeta, Sigma Xi, American Society of Agronomy, and Crop Science Society of America.

Date of final examination: June, 1964.