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I am submitting herewith a thesis written by James Larry Jones entitled "The Inheritance of Resistance to Isolates of Fusarium oxysporum var. nicotianae in Burley Tobacco." I recommend that it be accepted for nine quarter hours of credit in partial fulfillment of the requirements for the degree of Master of Science, with a major in Agronomy.

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THE INHERITANCE OF RESISTANCE TO ISOLATES OF *FUSARIUM OXYSPORUM* VAR. *NICOTIANAE* IN BURLEY TOBACCO

A Thesis

Presented to

the Graduate Council of

The University of Tennessee

In Partial Fulfillment

of the Requirements for the Degree

Master of Science

---

by

James Larry Jones

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ABSTRACT

The Inheritance of Resistance to Isolates of Fusarium oxysporum var. nicotianae in Burley Tobacco. (Under the direction of Creighton Lee Gupton).

The objectives of this study were: (1) to determine the mode of inheritance of resistance in burley tobacco when using individual isolates of Fusarium oxysporum and (2) to estimate the genetic parameters conditioning resistance. Five burley tobacco varieties which range from highly resistant to susceptible were crossed in a diallel scheme to provide the genetic material for the study. Plants were subjectively evaluated for their disease reaction as six-week old seedlings in the greenhouse.

The population means of the P_1, P_2, F_1, F_2, P_1F_1 and P_2F_1 generations of each cross were subjected to a generation means analysis to determine the genetic effects involved in resistance to the Fusarium isolates. The F_1 means were also evaluated for their general and specific combining ability.

Although a statistical difference in virulence was found among the isolates, and at least two physiological races were represented among them, the mode of inheritance and genetic effects for resistance appeared to be very similar for each isolate.

Results indicated that resistance of burley tobacco to the Fusarium isolates used in this study was quantitatively inherited and that general combining ability and additive gene effects were of major importance.

Dominance and epistatic effects were also important in some population and isolate combinations.

It was concluded that the tobacco breeder can effectively use a mixture of Fusarium isolates when breeding for Fusarium wilt resistance. The breeding program should be designed to accumulate genes for resistance into pure lines since genetic effects are predominately additive.

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CHAPTER I

INTRODUCTION

Controlling plant diseases by using resistant varieties offers a double economic gain. Disease resistant varieties eliminate both the loss from the disease and the cost of sprays and other treatments. Breeding for resistance to diseases has been the major contribution of the tobacco breeder. However, the breeder must avoid sacrificing yield and quality while incorporating disease resistance or the variety will not be grown.

Fusarium oxysporum (Schlecht) Wr. var. nicotianae J. Johnson, the fungus which causes Fusarium wilt of tobacco, is one of the soil-borne pathogens for which host resistance is the most practical means of control. Although Fusarium wilt of tobacco is usually not of major importance it occassionally causes severe losses.

An understanding of the mode of inheritance and the genetic effects involved for a given trait is necessary to develop effecient breeding procedures. This knowledge enables the breeder to better estimate plant response.

Gritton et al. (1965), using a mixture of Fusarium isolates, found that resistance to Fusarium wilt in flue-cured tobacco was quantitatively inherited and that most of the genetic variability was additive. The present investigation was undertaken to determine the mode of inheritance

of resistance in burley tobacco when using individual isolates of Fusarium and to estimate the genetic parameters conditioning this resistance.

CHAPTER II

REVIEW OF LITERATURE

Occurrence and Importance of the Disease

Fusarium wilt of tobacco was first reported in the United States in 1916 in Maryland on broadleaf tobacco, and in 1919 in Ohio on burley tobacco (Johnson, 1921).

Although Fusarium wilt has been reported from many tobacco growing areas of the world, it is not considered to be a major tobacco disease in any country. In the United States, the disease is important in the flue-cured Border Belt of North Carolina, and in the burley area of western Kentucky (Lucas, 1965).

Symptoms

Disease symptoms may appear at any stage in the growth of the tobacco plant. The usual symptoms are a yellowing and drying of leaves on one side of the plant. The affected leaves are often distorted, and the midribs are curved owing to unequal growth of the leaf. The tip of the affected plant often turns yellow and is drawn over toward the diseased side (Figure 1).

Wilting is not noticeable on inoculated seedlings in the greenhouse. The leaves of the diseased plants bleach and turn yellow, but remain turgid for days before wilting. A dark brown discoloration of the xylem tissue can be seen in the cross section of a diseased plant.



Figure 1. Fusarium wilt diseased plant showing the characteristic distortion of leaves on one side of the plant.

Davis and Dimond (1954) attributed vascular browning in Fusarium infected tomato plants to melaninization of free phenols by fungal enzymes.

Mechanism of Wilting and the Nature of Resistance

Several theories have been proposed to explain the fundamental cause of wilting in Fusarium wilt infected plants. The restriction of translocation of water in the host by vascular plugging appears to be a major factor. It has been shown that vascular plugging and wilt can be caused by the action of cellulolytic and pectolytic enzymes produced by the fungus which breaks down cell wall constituents. The freed materials move into the vessels where they gel forming plugs that restrict the flow of water (Pierson et al., 1955; Gothoskar et al., 1955; Husain and Dimond, 1960). Waggoner and Dimond (1955) concluded that mycelium was a contributing factor in the reduction of water flow, but that it could not be the total cause of impeded water flow. Davis and Dimond (1954) stated that certain phenols may participate in producing both vascular discoloration and wilting. Fusaric acid, a metabolic product of Fusarium oxysporum, has been reported to play a role in wilting. The fusaric acid injures the semi-permeability of the plasma boundary layers in the plant and destroys turgor thus producing wilt (Gaumann, 1957, 1958).

The relative resistance or susceptibility of a plant to the Fusarium organism appears to depend upon its ability to suppress fungal enzyme activity and upon the amount and nature of its cell wall constituents (Deese and Stahmann, 1962a, 1962b, 1960; Edgington and Dimond, 1959; Edgington, et al., 1961). Beckman et al. (1962) concluded that resistance to Fusarium wilt in banana was initially owing to a highly sensitive

host response mechanism by which invaded vessels are rapidly sealed off in advance of the invading organism. Winstead and Walker (1954) suggested that metabolites of low molecular weight produced by the fungus have a specific effect on susceptible tomato varieties and may in some way aid in the establishment of infection by the pathogen.

Disease Variability

The Fusarium organism is quite variable and the reaction of an individual genotype to Fusarium wilt is influenced by many factors. Buxton (1959) reported that Fusarium oxysporum produced variants in three major ways; (a) heterokaryosis, (b) recombination through a parasexual mechanism, and (c) adaptation in response to changes in environment.

Nematodes have been reported to increase the incidence of Fusarium wilt (Powell, 1963; Porter and Powell, 1967; Holdeman, 1956; and Jenkins and Coursen, 1957). Porter and Powell (1967) found nematode-Fusarium wilt interactions in both resistant and susceptible varieties, and in some treatments where both fungus and nematodes were present 100 percent of plants became diseased, whereas no disease occurred when the fungus was used alone.

McClure (1949) showed that wounds involving the xylem are necessary for infection of sweet potato or tobacco with Fusarium oxysporum. However, more than mere mechanical wounding appears to be involved with the nematode-Fusarium wilt complex (Jenkins and Cousen, 1957; and Porter and Powell, 1967). Melendez and Powell (1965) concluded that a growth-

promoting substance may be present in tissues modified by root-knot nematode infections.

Fusarium wilt is considered a warm weather disease. Although it can occur within a wide range of temperatures, the optimum temperature for infection and growth of the fungus appears to be between 28° and 31° C. (Johnson, 1921).

The disease is unpredictable in occurrence. Lucas (1965) reports that Fusarium wilt may be severe in a field one year, but may not appear the following year even though susceptible tobacco is grown. Attempts to infest soil on the Experiment Station farm at Lexington, Kentucky, were unsuccessful. Only 1 or 2 percent mortality was obtained in highly susceptible burley plants when grown in soil that had been artificially contaminated over 15 years in succession with virulent strains of the fungus (Valleau et al., 1942).

Isolates and Race Specificity

Numerous strains and races of the fungus Fusarium oxysporum have been reported. Pathogenicity of a particular strain may or may not be restricted to the plant species from which it was isolated (Armstrong et al., 1942; Hendrix and Nielsen, 1958; Armstrong and Armstrong, 1958a, 1958b; and Armstrong, 1940). Armstrong et al. (1942) concluded that isolates from cotton, okra, coffee, weed, and tobacco were apparently the same physiological race. All caused wilting in the first three hosts and in a susceptible burley tobacco but not in a resistant burley or in a flue-cured variety. The tomato isolate failed

to cause wilting in cotton, okra, and watermelon and the watermelon isolate did not cause wilting in tomato and sweet potato. Smith and Shaw (1943) grouped 94 isolates of Fusarium oxysporum into three physiological races on the basis of results of inoculation on flue-cured tobacco, burley tobacco, sweet potato, and cotton. Strain 1 was pathogenic on burley tobacco and sweet potato but not on flue-cured tobacco or cotton; strain 2 was pathogenic on burley tobacco, flue-cured tobacco and sweet potato but not on cotton; and strain 3 was pathogenic on burley tobacco and cotton but not on flue-cured tobacco or sweet potato. Everette (1958) suggested that the isolates of Fusarium were not confined to three physiologic groups, and that the separation of the strains depends largely upon the use of a wide variety of test plants. The 22 isolates used by Everette appeared to represent at least 17 different strains of the organism.

Inheritance Studies

Fusarium wilt occurs on several crop plants. Studies on inheritance of resistance to Fusarium wilt in various host plants have shown that it varies from dominant, to intermediate, to recessive, and from polygenic to a single factor. The results of investigations on inheritance of resistance to Fusarium wilt in crops other than tobacco are summarized in Table I.

Johnson (1921) found that varieties of tobacco differed markedly in their resistance to Fusarium wilt. The F_1 generation of a cross between

TABLE I

INHERITANCE OF RESISTANCE TO FUSARIUM WILT IN CROPS OTHER THAN TOBACCO

Host Plant	Mode of Inheritance of Resistance	Author
flax	multiple factors	Tisdale (1917)
flax	several factors	Burnham (1932)
flax	several factors	Allison and Christensen (1938)
flax	two complementary genes	Knowles and Houston (1955)
flax	major gene	Knowles and Houston (1955)
cabbage	single dominant gene	Walker (1926, 1930)
brussels sprouts	single dominant gene	Black and Walker (1933)
Wisconsin All Seasons Cabbage	single dominant gene	Blank (1937)
Wisconsin All Seasons Cabbage	several factors	Blank (1937)
blackeye cowpeas	dominant	Mackie (1934)
cowpea	two major dominant genes	Hare (1957)
canning peas	single dominant gene	Wade (1929)

TABLE I (CONTINUED)

Host Plant	Mode of Inheritance of Resistance	Author
watermelons	several factors	Bennett (1936)
citron X watermelon	recessive	Walker (1941)
Indian cotton	dominant	Kulkarni (1934)
Indian cotton	recessive	Kulkarni (1934)
cotton	dominant	Jenkins et al. (1939)
Upland cotton	single factor	Smith and Ballard (1947)
Upland cotton	major dominant gene with modifiers	Smith and Dick (1960)
Sea Island cotton	two dominant genes with additive effects	Smith and Dick (1960)
sweet potato	multigenic	Hughes et al. (1963)
tomato	recessive	White (1924)
muskmelon	intermediate	Leach and Currence (1938)

resistant and susceptible burley seemed intermediate in resistance to Fusarium wilt. Valleau (1934) indicated that the inheritance of resistance to Fusarium wilt of tobacco was determined by a simple Mendelian recessive. One thousand and twenty-five F_2 hybrids between resistant Turkish tobacco and susceptible white burley segregated into 272 resistant and 735 susceptible plants when inoculated and set in the field. Later, in a review article, Valleau (1952) stated "In going over the records again and in the light of further experience it is difficult to say whether resistance is recessive or is sometimes dominant." One study by Valleau indicated that resistance in Turkish tobacco was at least partially dominant. In another study the F_2 of a Cuban X Ky. 5 (susceptible) cross produced 26 plants which showed symptoms of wilt, 52 healthy plants (29.4 percent), and 99 dead plants. Valleau pointed out that the 29.4 percent healthy plants was not far from an expected 25 percent if resistance were controlled by a single factor pair. The F_1 of the cross, McCuller's 27X Ky. 57 (susceptible), had 17 plants out of 60 with mild wilt symptoms. Resistance in this cross appeared to be partially dominant. Valleau (1952) concluded that resistance to Fusarium wilt in tobacco is controlled by a single factor pair that is partially dominant or recessive. Clayton (1953) reported that Fusarium resistance is inherited on a polygenic basis. Gritton et al. (1965) concluded that resistance to Fusarium wilt in flue-cured tobacco is inherited quantitatively and that most of the genetic variability is additive.

CHAPTER III

MATERIALS AND METHODS

Isolate Selection

Experiments were conducted during the summer of 1966 in an attempt to select isolates of Fusarium that differed in virulence to the burley tobacco varieties for use in the inheritance of host reaction. Twenty plants of each variety were inoculated with each of 13 different Fusarium oxysporum var. nicotianae isolates.

Isolates designated as Whiteville, 439, NC-2, and 435 were originally selected for the inheritance study. However, isolate 435 was replaced by isolate Greene after the first planting date because the former grew extremely slow in culture. The initial selection of isolates was based on morphological differences in culture, and on differences in host reaction to the isolates. The disease reaction indices of the burley varieties to the selected isolates of Fusarium are presented in Table II.

Inoculations were also made on cotton, sweet potato, and burley tobacco in an attempt to classify the selected isolates according to Smith and Shaw's (1943) findings. Plants of 'Stardel', a susceptible cotton variety, and slips of 'Porto Rico', a susceptible sweet potato variety, were inoculated when approximately six inches in height. Six-week old plants of 'Judy's Pride', and 'Ky. 16', both suscpetible

TABLE II

DISEASE INDICES OF FIVE BURLEY VARIETIES INOCULATED WITH FIVE *FUSARIUM OXYSPORUM* VAR. *NICOTIANAE* ISOLATES

Burley Variety	Isolate				
	Whiteville	435	439	Greene	NC-2
Ky. 12	1.23[a]	2.00	1.18	0.25	0.90
Ky. 10	1.95	2.53	3.15	1.00	2.15
Bu. 37	2.50	3.15	4.30	3.15	2.00
Bu. 21	3.60	4.17	4.72	3.45	4.10
J. Pride	4.32	4.65	4.92	4.30	4.10

[a]0 = no symtpoms, 5 = dead plant.

burley tobacco varieties were inoculated. The disease reaction of the cotton, sweet potato, and burley varieties to each isolate are given in Table III. Isolate Greene affected cotton and the burley varieties but not sweet potato and thus is strain 3 of Smith and Shaw's (1943) classification. The other isolates affected the burley varieties and sweet potato, but not cotton, and are either strain 1 or 2. These isolates could not be further differentiated since flue-cured tobacco was not included as a tester host.

Derivation of Genetic Stocks

The material for studying the inheritance of Fusarium wilt resistance in burley tobacco originated from a five-parent diallel of burley tobacco varieties. The parental varieties and their relative disease reaction to Fusarium wilt were as follows: 'Kentucky 12', high resistance; 'Kentucky 10', moderate resistance; 'Burley 37', low to moderate resistance; 'Burley 21', low resistance; and 'Judy's Pride', highly susceptible (Hoffbeck, 1966 and Table II).

Foundation seed of Kentucky 12 and Kentucky 10 was obtained from the Kentucky Agricultural Experiment Station, while the Burley 37, Burley 21, and Judy's Pride varieties were obtained from the foundation seed stocks at the Tobacco Experiment Station, Greeneville, Tennessee.

In the summer of 1965, an individual plant was selected from each variety and bagged to obtain selfed seed. Cuttings from the original

TABLE III

RESULTS OF INOCULATIONS WITH FUSARIUM ISOLATES ON COTTON, SWEET POTATO, AND BURLEY TOBACCO

Isolate	Cotton	Sweet Potato	Judy's Pride	Ky. 16
439	R.[a]	S.	S.	S.
Whiteville	R.	S.	S.	S.
Greene	S.[b]	R.	S.	S.
NC-2	R.	S.	S.	S.

[a]Resistant.

[b]Susceptible.

selections were rooted in the greenhouse to produce flowers for hybridization in the fall of 1965. Ten F_1 populations were produced by making crosses in all possible combinations, excluding reciprocals, among the five parents. P_1F_1, P_2F_1, and F_2 populations were produced in the greenhouse in the spring of 1966. The backcross generations resulted from crossing each F_1 population to each of its parents, P_1 and P_2. The F_2 seed were produced by selfing an F_1 plant of each cross.

Procedures For Producing Inoculum

The isolates of Fusarium oxysporum var. nicotianae were maintained as stock cultures on potato dextrose agar at approximately 3 °C. The organisms from the stock cultures were transferred to medium in petri dishes and allowed to grow for three or four days. To produce the inoculum, the pathogens from the petri dishes were cultured on potato dextrose broth.

The ingredients of the medium were placed in a 2000 ml. Erlenmeyer flask and heated for approximately thirty minutes. Fifty milliliters of the medium were placed into 100 ml. Erlenmeyer flasks and sterilized in an autoclave at 15 pounds pressure for 20 minutes.

The organisms were transferred under aseptic conditions from the petri dishes to flasks containing sterilized liquid medium. The organisms were incubated at room temperature for two weeks. The flasks were shaken occasionally to aerate the cultures.

The inoculum was prepared for use by mixing the culture in a Waring blender for 10 to 15 seconds, and diluting 1:1 with tap water.

A small amount of pyrophyllite was added to the inoculum as a thickner.

Method of Inoculating Plants

Uniform six-week old seedlings were pulled from the seed pots. The roots were washed, immersed in the inoculum, and rubbed gently between the thumb and forefinger. The plants were left in the inoculum for two to three minutes before transplanting to two and one-half inch pots and set in galvanized flats containing sand. The plants were watered when needed and liquid fertilizer (20-20-20) was applied weekly. Greenhouse temperatures were maintained between 70 and 75 degrees F. at night and between 80 and 90 degrees F. during the day. The plants were allowed to grow for approximately three weeks before symptom evaluations were made.

Experimental Design

Ten plants each of the P_1, P_2, F_1, P_1F_1, and P_2F_1 generations and 20 plants of the F_2 generation were inoculated with each Fusarium isolate in three planting dates. All generations derived from the 10 crosses among the five parental varieties were included in each greenhouse planting.

Parents, all generations, and Fusarium treatments were completely randomized within 4 greenhouse benches for each planting date. The three planting dates served as replications.

Procedures for Scoring Fusarium Wilt Symptoms

The plants were cut off at the soil surface and evaluated for their disease reaction according to the disease index given in Table IV. Figure 2 shows plants typical of the six disease classes. The disease index for a population was obtained by multiplying the number of plants in each disease class by the disease class number, summing, and dividing by the total number of plants tested.

Analysis of Data

A mean disease index was derived for each generation by averaging plot totals over replications. These means were used to compute the analyses of variance and the genetic analyses.

An analysis of variance was computed for individual planting dates and for the three planting dates combined. Each cross was analyzed for generation differences since the isolate X genotype effect was significant in the combined analysis. The analysis indicated that generation differences existed within each of the 10 crosses. Therefore, the generation means of each cross were subjected to the genetic analysis outlined by Gamble (1962). Because isolate differences were detected in the combined analysis, a genetic analysis was computed for each isolate.

Separation of Genetic Effects

Gamble's (1962) analysis was designed to separate gene effects affecting the genetic variation of a quantitative trait into six

TABLE IV

DISEASE INDEX FOR SCORING FUSARIUM WILT SYMPTOMS

Disease Class Number	Description of Symptoms
0	No visible symptoms
1	Slight vascular discoloration; no visible external symptoms
2	Vascular discoloration; slight yellowing and discoloration of leaves
3	Vascular discoloration; moderate leaf yellowing and distortion
4	Severe wilting and pronounced distortion of plant
5	Dead plant



Figure 2. Plants typical of the six disease classes. Left to right: class 0, class 1, class 2, class 3, class 4, and class 5.

parameters. These parameters are: the mean (m), additive (a), dominance (d), additive X additive (aa), additive X dominance (ad), and dominance X dominance (dd) effects.

Means of the P_1, P_2, F_1, F_2, P_1F_1, and P_2F_1 populations are required to estimate the six parameters mentioned above. These estimates are obtained as follows:

$$m = \overline{F_2}$$

$$a = \overline{P_1F_1} - \overline{P_2F_1}$$

$$d = -\tfrac{1}{2}\overline{P}_1 - \tfrac{1}{2}\overline{P}_2 + \overline{F}_1 - 4\overline{F}_2 + 2\overline{P_1F_1} + 2\,\overline{P_2F_1}$$

$$aa = -4F_2 + 2\overline{P_1F_1} + 2\overline{P_2F_1}$$

$$ad = -\tfrac{1}{2}\overline{P}_1 + \tfrac{1}{2}\overline{P}_2 + \overline{P_1F_1} - \overline{P_2F_1}$$

$$dd = \overline{P}_1 + \overline{P}_2 + 2\overline{F}_1 + 4\overline{F}_2 - 4\overline{P_1F_1} - 4\overline{P_2F_1}$$

Variances of the estimates of the gene effects were computed as follows:

$$\sigma^2 m = \sigma^2_{\overline{F_2}}$$

$$\sigma^2 a = \sigma^2_{\overline{P_1F_1}} + \sigma^2_{\overline{P_2F_1}}$$

$$\sigma^2 d = \tfrac{1}{4}\sigma^2_{\overline{P}_1} + \tfrac{1}{4}\sigma^2_{\overline{P}_2} + \sigma^2_{\overline{F}_1} + 16\sigma^2_{\overline{F}_2} + 4\sigma^2_{\overline{P_1F_1}} + 4\sigma^2_{\overline{P_2F_1}}$$

$$\sigma^2 aa = 16\sigma^2_{\overline{F}_2} + 4\sigma^2_{\overline{P_1F_1}} + 4\sigma^2_{\overline{P_2F_1}}$$

$$\sigma^2 ad = \tfrac{1}{4}\sigma^2_{\overline{P}_1} + \tfrac{1}{4}\sigma^2_{\overline{P}_2} + \sigma^2_{\overline{P_1F_1}} + \sigma^2_{\overline{P_2F_1}}$$

$$\sigma^2 dd = \sigma^2_{\overline{P}_1} + \sigma^2_{\overline{P}_2} + 4\sigma^2_{\overline{F}_1} + 16\sigma^2_{\overline{F}_2} + 16\sigma^2_{\overline{P_1F_1}} + 16\sigma^2_{\overline{P_2F_1}}$$

An approximate test of significance for a given genetic effect was computed by dividing the estimate of the genetic effect by its standard error

(obtained as the square root of the above variances). The standard errors were not directly presented in this paper, however, significance was estimated by assuming an infinite population and using the "t" values (>2.576, >1.96, and >1.645 for significance at the .01, .05, and .10 levels of probability, respectively).

Combining Ability Analysis

The parameters, excluding mean (m), estimated by Gambles (1962) procedure were dependent upon backcross generation means. Since a portion of the backcross data in this investigation did not conform to expectations, a combining ability analysis developed by Griffing (1956) was computed using only the F_1 means of the ten crosses.

The analysis of variance table and expectations of mean squares for Griffing's analysis (experimental method 4 Model II) are given in Table V.

The sum of squares for general and specific combining ability were obtained as follows:

$$Sg = \frac{1}{p-2}\sum_i Xi.^2 - \frac{4}{p(p-2)}X..^2,$$

$$Ss = \sum_i \sum_j x_{ij}^2 - \frac{1}{p-2}\sum X_{i.}^2 + \frac{2}{(p-1)(p-2)}X..^2$$

Mean squares were computed in the usual manner and a variance ratio to test for significance of effects was computed as follows:

(1) g.c.a. effects . . . . $F\left[(p-1), \overline{m}\right] = M_g/M_e'$

(2) s.c.a. effects . . . . $F\left[p(p-3)/2, \overline{m}\right] = M_s/M_e'$

TABLE V

ANALYSIS OF VARIANCE TABLE AND EXPECTATIONS OF MEAN SQUARES FOR THE COMBINING ABILITY ANALYSIS

Source	D. F.	Sum of Squares	Mean Squares	Expectation of Mean Squares
General combining ability	p-1	Sg	Mg	$\sigma^2 + \sigma^2_s + (p-2)\sigma g^2$
Specific combining ability	p(p-3)/2	Ss	Ms	$\sigma^2 + \sigma^2_s$
Error	m	Se	Me'	σ^2

CHAPTER IV

RESULTS AND DISCUSSION

A continuous distribution in disease reaction was displayed by the tobacco populations for each of the individual Fusarium oxysporum var. nicotianae isolates which suggests that multigenic inheritance is involved. The results of this study compare favorably with the findings of Gritton et al. (1965) in flue-cured tobacco and the statement by Clayton (1953) that Fusarium wilt resistance in tobacco is quantitatively inherited.

The mean Fusarium wilt disease reaction of five burley tobacco varieties and all possible F_1, F_2, P_1F_1, and P_2F_1 crosses to four isolates of Fusarium is given in Tables VI, VII, VIII, and IX. The means represent 10 individual plant indices from the P_1, P_2, F_1, P_1F_1, and P_2F_1 populations, and 20 individual plant indices from the F_2 population averaged over replications.

The analysis of variance (Table X) calculated from the disease index means for each planting date revealed a significant difference among genotypes at each date, while differences among isolates were significant only at planting dates 1 and 2. The disease indices by genotypes, isolates, and planting dates are presented in the Appendix (Table XVII, page 53).

A summary of the combined analyses of variance for three Fusarium isolates and three planting dates is presented in Table XI. The Greene

TABLE VI

MEAN DISEASE INDEX OF PARENTS, MIDPARENT, AND VARIOUS GENERATIONS AVERAGED OVER THREE REPLICATIONS FOR ISOLATE 439

Population	Generation						
	P_1	P_2	F_1	F_2	P_1F_1	P_2F_1	MP[a]
Ky. 12 X Ky. 10	0.63	1.64	1.44	1.77	1.40	1.47	1.14
Ky. 12 X Bu. 37	0.63	3.00	1.73	1.68	1.37	2.53	1.82
Ky. 12 X Bu. 21	0.63	3.60	2.00	2.32	1.47	2.57	2.12
Ky. 12 X Judy P.	0.63	4.77	2.47	2.72	2.33	4.07	2.70
Ky. 10 X Bu. 37	1.64	3.00	2.23	1.98	2.70	2.47	2.32
Ky. 10 X Bu. 21	1.64	3.60	2.63	2.48	3.20	3.27	2.62
Ky. 10 X Judy P.	1.64	4.77	3.20	3.63	3.57	3.30	3.21
Bu. 37 X Bu. 21	3.00	3.60	3.13	3.28	3.43	3.43	3.30
Bu. 37 X Judy P.	3.00	4.77	3.60	3.15	3.23	4.53	3.89
Bu. 21 X Judy P.	3.60	4.77	4.07	4.25	4.03	4.67	4.19

[a]MP = Midparent value.

TABLE VII

MEAN DISEASE INDEX OF PARENTS, MIDPARENT, AND VARIOUS GENERATIONS AVERAGED OVER THREE REPLICATIONS FOR ISOLATE WHITEVILLE

Population	Generation						
	P_1	P_2	F_1	F_2	P_1F_1	P_2F_1	MP[a]
Ky. 12 X Ky. 10	0.43	1.30	1.20	1.20	1.13	1.17	0.87
Ky. 12 X Bu. 37	0.43	2.40	1.47	1.53	1.33	1.40	1.42
Ky. 12 X Bu. 21	0.43	3.57	1.83	1.20	1.47	2.23	2.00
Ky. 12 X Judy P.	0.43	4.10	2.17	2.92	1.87	3.13	2.27
Ky. 10 X Bu. 37	1.30	2.40	2.07	2.00	2.03	2.30	1.85
Ky. 10 X Bu. 21	1.30	3.57	2.30	2.15	2.37	2.63	2.44
Ky. 10 X Judy P.	1.30	4.10	3.43	2.65	2.33	2.80	2.70
Bu. 37 X Bu. 21	2.40	3.57	2.80	2.25	2.33	3.07	2.99
Bu. 37 X Judy P.	2.40	4.10	3.53	2.67	2.57	3.43	3.25
Bu. 21 X Judy P.	3.57	4.10	3.67	3.50	3.57	4.37	3.84

[a]MP = Midparent value.

TABLE VIII

MEAN DISEASE INDEX OF PARENTS, MIDPARENT, AND VARIOUS GENERATIONS AVERAGED OVER THREE REPLICATIONS FOR ISOLATE NC-2

Population	Generation						
	P_1	P_2	F_1	F_2	P_1F_1	P_2F_1	MP[a]
Ky. 12 X Ky. 10	0.40	1.57	1.30	1.33	1.50	1.50	0.99
Ky. 12 X Bu. 37	0.40	2.33	1.43	1.18	1.77	1.83	1.37
Ky. 12 X Bu. 21	0.40	2.77	1.60	1.73	1.47	2.33	1.59
Ky. 12 X Judy P.	0.40	4.43	2.33	2.37	2.23	3.57	2.42
Ky. 10 X Bu. 37	1.57	2.33	2.00	1.93	1.50	2.27	1.95
Ky. 10 X Bu. 21	1.57	2.77	2.27	2.17	2.77	2.33	2.17
Ky. 10 X Judy P.	1.57	4.43	2.60	2.43	2.57	3.70	3.00
Bu. 37 X Bu. 21	2.33	2.77	2.40	1.77	2.40	1.97	2.55
Bu. 37 X Judy P.	2.33	4.43	3.30	3.18	2.70	3.80	3.38
Bu. 21 X Judy P.	2.77	4.43	4.07	3.15	3.37	3.87	3.60

[2]MP = Midparent value.

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TABLE IX

MEAN DISEASE INDEX OF PARENTS, MIDPARENT, AND VARIOUS GENERATIONS AVERAGED OVER TWO REPLICATIONS FOR ISOLATE GREENE

Population	Generation						
	P_1	P_2	F_1	F_2	P_1F_1	P_2F_1	MP[a]
Ky. 12 X Ky. 10	0.15	1.35	0.85	1.33	0.20	1.81	0.75
Ky. 12 X Bu. 37	0.15	3.20	1.60	1.53	1.10	2.05	1.68
Ky. 12 X Bu. 21	0.15	3.90	1.80	2.23	1.60	2.30	2.03
Ky. 12 X Judy P.	0.15	4.55	2.30	2.48	1.45	3.15	2.35
Ky. 10 X Bu. 37	1.35	3.20	2.35	2.28	1.40	2.25	2.28
Ky. 10 X Bu. 21	1.35	3.90	2.80	2.03	1.80	2.95	2.63
Ky. 10 X Judy P.	1.35	4.55	2.80	2.20	2.90	3.75	2.95
Bu. 37 X Bu. 21	3.20	3.90	3.45	3.55	3.95	3.20	3.55
Bu. 37 X Judy P.	3.20	4.55	3.15	3.63	3.50	4.15	3.88
Bu. 21 X Judy P.	3.90	4.55	4.35	4.08	4.30	4.45	4.23

[a]MP = Midparent value.

TABLE X

MEAN SQUARES FROM THE ANALYSES OF VARIANCE OF DISEASE REACTION FOR THREE PLANTING DATES

Source of Variation	Degrees of Freedom	Date 1	Date 2	Date 3
Entries	44	2.5613**	2.7200**	2.3964**
Isolates	2	3.0182**	22.1774**	.5227
Gen. X Isolates	88	.2887	.3898	.2350

**-Significant at .01 level of probability.

TABLE XI

MEAN SQUARES FROM THE COMBINED ANALYSIS OF VARIANCE OF DISEASE INDICES FOR THREE ISOLATES AT THREE PLANTING DATES

Source of Variation	Degrees of Freedom	Mean Square
Dates	2	13.8856**
Isolates	2	9.4562**
Dates X Isolates	4	8.1310**
Entries	44	6.8377**
Dates X Entries	88	.4200**
Isolates X Entries	88	.3874*
Dates X Isolates X Entries	176	.2564

**-Significant at .01 level of probability.

*-Significant at .05 level of probability.

isolate was not included in the combined analysis because it was used at only two planting dates. Mean squares for dates, isolates, and tobacco entries were highly significant. The highly significant dates by isolates interaction was not surprising since significant differences among isolates occurred at two planting dates and not at the other. The mean squares for the dates by entries and isolates by entries interactions were quite small relative to the mean squares for dates, isolates, dates by isolates and entries. However, the dates X entries interaction was highly significant, and the isolates by entries interaction was significant at the .05 level of probability.

Since the combined analysis, which included all crosses, indicated that genetic differences were present among entries, and since the isolates X entries effect was significant, an analysis was computed for all generations from each cross using all isolates. This analysis revealed significant generation differences for each of the 10 crosses.

Although the burley parents responded generally as expected to the Fusarium organism, some plant to plant variability occurred which could have produced a blurring effect on the genetic situation. Not all plants of the susceptible varieties were diseased, and not all plants of the more resistant varieties were completely disease free. The plant to plant variability is not obvious from the data presented because it was masked by plot averages and planting date averages. Variability within the parents and F_1 populations should be environmental since the study was based on products of crosses between single plants assumed to be homozygous. Walker (1966) pointed out that in some cases single-gene

resistance might appear like multiple-gene resistance because of variation caused by environmental variation.

In this study, the highly resistant parent, Ky. 12, received a consistently low disease index for each isolate. Ky. 10 had a high to intermediate resistance reaction to the Fusarium isolates. Isolates 439 and NC-2 were more virulent on Ky. 10 than were isolates Whiteville and Greene. Bu. 37 and Bu. 21 showed a similar reaction to the 439, NC-2, and Greene isolates, however, Bu. 37 received a lower disease index for the Whiteville isolate than did Bu. 21. Judy's Pride, the susceptible parent, was given a relatively high Fusarium wilt index for each isolate. The relative performance of the parent varieties in the genetic study conform closely with the results of preliminary investigations (Table II, page 13). The F_1 disease indices fell within range of the parents in all crosses by isolates, and were usually near the mid-parent value. Generally, the F_2 disease indices were similar to the F_1 and midparent values, however, in a few instances the F_2 indices exceeded the parental limits which indicated transgressive segregation. The disease indices of the backcross generation fell between the F_1 and the recurrent parent indices approximately 50 per cent of the time. No definite pattern could be established for the backcross generation indices that were outside the range of the F_1 and recurrent parent. Since only 10 plants of the segregating backcross populations were tested at each planting date, the observed results may have occurred because of the small sample size. Examples of disease reactions of selected populations

to the four isolates of Fusarium are shown diagrammatically in Figures 3 and 4.

It has been pointed out (Everette, 1958) that a variety of tobacco may be resistant to one isolate of Fusarium oxysporum and susceptible to another. One objective of this study was to investigate the mode of reaction of burley tobacco to a set of Fusarium isolates. The isolates were selected on the basis of preliminary investigations previously described. The results showed a statistical difference in virulence among the selected isolates (Table XI, page 30), however, differences appeared to be only in severity of symptoms produced.

Separation of Genetic Effects

Since a significant statistical difference was detected among isolates and among generations for each cross, the generation mean analyses described by Gamble (1962) were computed separately for each cross and isolate. The procedure was designed to separate the gene effects affecting genetic variation of a quantitative trait into six parameters. Estimates of the six parameters representing mean effects, additive and dominance gene effects, and the three types of digenic epistatic effects are presented in Tables XII, XIII, XIV, and XV. The standard errors of the estimates are not included in the tables, instead, significant estimates are indicated by the usual symbols, and in addition, estimates larger than their standard error are indicated by $\pm\pm$.

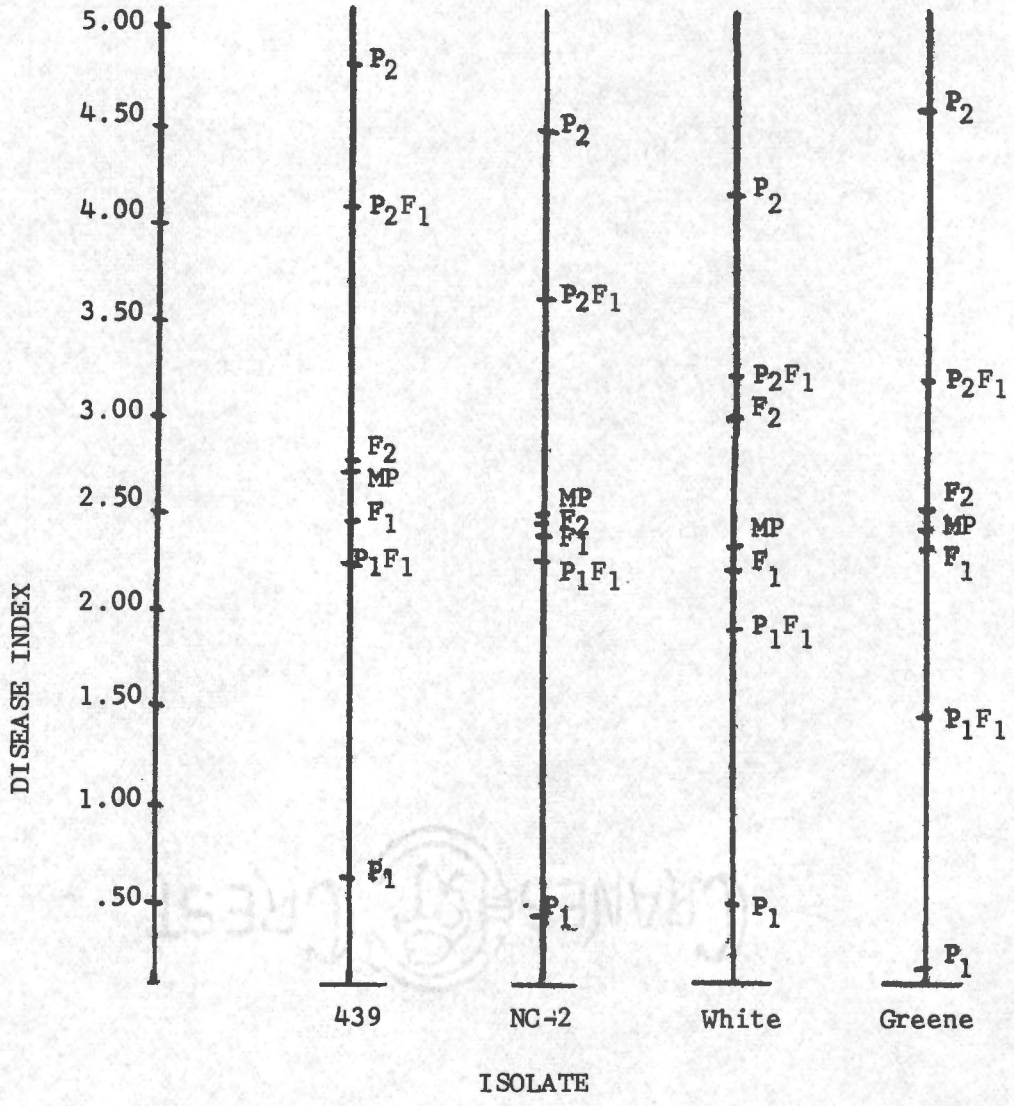


Figure 3. Relative Fusarium wilt reaction of six populations of the cross Ky. 12 (P_1) x Judy's Pride (P_2) to four isolates of Fusarium oxysporum var. nicotianae.

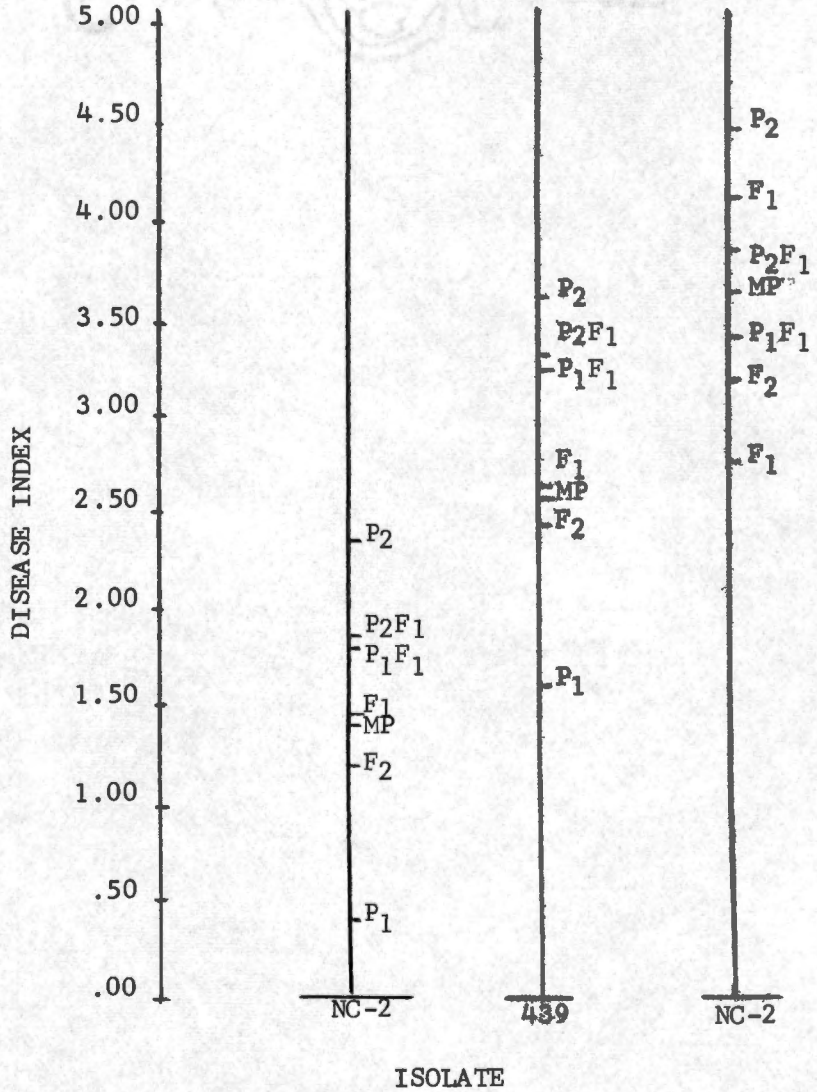


Figure 4. Examples of erratic disease index means obtained from some crosses by isolate combinations. Crosses from left to right: Ky. 12 (P_1) x Bu. 37 (P_2), Ky. 10 (P_1) x Bu. 21 (P_2), and Bu. 21 (P_1) x Judy's Pride (P_2).

TABLE XII

MEAN ESTIMATES OF GENETIC EFFECTS FOR THE 10 CROSSES OVER THREE REPLICATIONS FOR ISOLATE 439

Population	Gene Effect					
	m	a	d	aa	ad	dd
Ky. 12 X Ky. 10	1.77**	- .07	-1.04	-1.34	.43	.75
Ky. 12 X Bu. 37	1.68**	-1.16	.99	1.08	.02	-1.79
Ky. 12 X Bu. 21	2.32**	-1.10	-1.32	-1.20	.38	1.35
Ky. 12 X Judy P.	2.72**	-1.74**	1.68	1.92	.33	-4.38
Ky. 10 X Bu. 37	1.98**	.23	2.33	2.42	.91	-3.66
Ky. 10 X Bu. 21	2.48**	- .07	3.03	3.02	.91	-5.46
Ky. 10 X Judy P.	3.63**	.27	- .79	- .78	1.84*	- .15
Bu. 37 X Bu. 21	3.28**	.00	.43	.60	.30	-1.46
Bu. 37 X Judy P.	3.15**	-1.30*	2.63	2.92	- .41	-3.47
Bu. 21 X Judy P.	4.25**	- .64	2.08	.40	- .05	-1.29

**-Significant at .01 probability level.

*-Significant at .05 probability level.

†-Significant at .10 probability level.

‖ -Estimate larger than its standard error.

TABLE XIII

MEAN ESTIMATES OF GENETIC EFFECTS FOR THE 10 ACROSS OVER THREE REPLICATIONS FOR ISOLATE WHITEVILLE

Population	Gene Effect					
	m	a	d	aa	ad	dd
Ky. 12 X Ky. 10	1.20**	- .04	.13	- .20	.35	- .27
Ky. 12 X Bu. 37	1.53**	- .07	-1.11	- .66	1.41**	1.97
Ky. 12 X Bu. 21	1.20**	- .76 $\frac{1}{1}$	2.42*	2.60**	.81 $\frac{1}{1}$	-2.34 $\frac{1}{1}$ $\frac{1}{1}$
Ky. 12 X Judy P.	2.92**	-1.26 $\frac{1}{1}$	-1.78	-1.68	.57	.55
Ky. 10 X Bu. 37	2.00**	- .27	.88	.66	.28	-1.48
Ky. 10 X Bu. 21	2.15**	- .26	1.26	1.40	.88 $\frac{1}{1}$ $\frac{1}{1}$	-1.93
Ky. 10 X Judy P.	2.65**	- .47	.39	- .34	.93 $\frac{1}{1}$ $\frac{1}{1}$	2.34
Bu. 37 X Bu. 21	2.25**	- .74*	1.61	1.80 $\frac{1}{1}$ $\frac{1}{1}$	- .15	-1.03
Bu. 37 X Judy P.	2.67**	- .86 $\frac{1}{1}$ $\frac{1}{1}$	1.60	1.32	- .01	.24
Bu. 21 X Judy P.	3.50**	- .80 $\frac{1}{1}$	1.71	1.88	- .54 $\frac{1}{1}$ $\frac{1}{1}$	-2.75

**-Significant at .01 probability level.

*-Significant at .05 probability level.

$\frac{1}{1}$ -Significant at .10 probability level.

$\frac{1}{1}$ $\frac{1}{1}$ -Estimate larger than its standard error.

TABLE XIV

MEAN ESTIMATES OF GENETIC EFFECTS FOR THE 10 CROSSES OVER THREE REPLICATIONS FOR ISOLATE NC-2

Population	Gene Effect					
	m	a	d	aa	ad	dd
Ky. 12 X Ky. 10	1.33**	.00	1.00	.68	.58$\frac{1}{1}$	-2.11$\frac{1}{11}$
Ky. 12 X Bu. 37	1.18**	- .06	2.54*	2.48*	.91*	-4.09*
Ky. 12 X Bu. 21	1.73*	- .86$\frac{1}{11}$	.69	.68	.33	-1.91
Ky. 12 X Judy P.	2.37**	-1.34*	2.03	2.12$\frac{11}{11}$	.68	-4.23$\frac{1}{11}$
Ky. 10 X Bu. 37	1.93**	- .77$\frac{11}{11}$	- .14	- .18	- .39	.54
Ky. 10 X Bu. 21	2.17**	.44$\frac{11}{11}$	1.61$\frac{11}{11}$	1.52	1.04**	-2.84$\frac{1}{1}$
Ky. 10 X Judy P.	2.43**	-1.13$\frac{1}{1}$	22.41$\frac{1}{1}$	2.82	.30	- .06
Bu. 37 X Bu. 21	1.77**	.43	1.50	1.66$\frac{11}{11}$	.65$\frac{11}{11}$	- .50
Bu. 37 X Judy P.	3.18**	-1.10$\frac{11}{11}$	.19	.28	- .05	.08
Bu. 21 X Judy P.	3.15**	- .50	2.34$\frac{11}{11}$	1.88	.33	-1.02

**-Significant at .01 probability level.

*-Significant at .05 probability level.

$\frac{1}{1}$-Significant at .10 probability level.

$\frac{11}{11}$ -Estimate larger than its standard error.

TABLE XV

MEAN ESTIMATES OF GENETIC EFFECTS FOR THE 10 CROSSES OVER TWO REPLICATIONS FOR ISOLATE GREENE

Population	Gene Effect					
	m	a	d	aa	ad	dd
Ky. 12 X Ky. 10	1.33 $\frac{1}{1}\frac{1}{1}$	-1.61*	-1.21	-1.30	-1.01 $\frac{1}{1}\frac{1}{1}$	.48
Ky. 12 X Bu. 37	1.53**	- .95	.10	.18	.57	.07
Ky. 12 X Bu. 21	2.23**	- .70	-1.35	-1.12	1.17 $\frac{1}{1}\frac{1}{1}$	.97
Ky. 12 X Judy P.	2.48**	-1.70 $\frac{1}{1}$	- .78	- .72	.50	.82
Ky. 10 X Bu. 37	2.28**	- .85	-1.75	-1.82	.07	3.77
Ky. 10 X Bu. 21	2.03**	-1.15**	1.55 $\frac{1}{1}\frac{1}{1}$	1.38 $\frac{1}{1}\frac{1}{1}$	.12	- .03
Ky. 10 X Judy P.	2.20**	- .85 $\frac{1}{1}$	4.34**	4.50**	.75 $\frac{1}{1}\frac{1}{1}$	-6.30**
Bu. 37 X Bu. 21	3.55**	.75	.00	.10	1.10	- .40
Bu. 37 X Judy P.	3.63**	-1.35 $\frac{1}{1}$	1.25	.98	- .67	- .43
Bu. 21 X Judy P.	4.08**	- .15	1.30	1.18	.18	-1.53

**-Significant at .01 probability level.

*-Significant at .05 probability level.

$\frac{1}{1}$-Significant at .10 probability level.

$\frac{1}{1}\frac{1}{1}$-Estimate larger than its standard error.

Additive gene effects made a major contribution to the inheritance of resistance of burley tobacco to the Fusarium isolates. Thirteen of 40 of the crosses by isolates exhibited significant additive gene effects at the 10 per cent level of probability or greater, and in seven other crosses by isolates the estimates were greater than their standard errors. Additive genetic effects were more pronounced, and larger in magnitude relative to parameter m in the cross involving the highly resistant parent, Ky. 12, and the most susceptible parent, Judy's Pride. Additive gene effects were equally important in the inheritance of resistance to each isolate involved in the study.

The estimates of parameter d, dominance gene effects, were large in magnitude relative to parameter m in a few crosses by isolates which indicated the presence of at least partial dominance for resistance to Fusarium wilt. Significant dominance effects appeared in four crosses by isolates, and five of the estimates of dominance were larger than their standard error.

The data also indicated that one or more of the three types of epistasis is important in the inheritance of Fusarium wilt resistance. Relative magnitudes of the dominance X dominance gene effects were sometimes quite large and occasionally were greater than parameter m. The relative magnitude of the additive X dominance effects were quite small and appeared to be of minor importance.

The lack of significance of genetic effects with certain parental combinations apparent in the tables of genetic estimates (page 36, 37, 38, and 39) is not too surprising. Although the burley parent varieties

employed in the study range from highly resistant to very susceptible in their reaction to Fusarium wilt, some crosses involve parents of similar reactions to Fusarium wilt. For example, Ky. 10 and Bu. 37 are reported to possess intermediate and intermediate to low resistance respectively to the disease. Very little genetic variance can be expected from a cross of this type. Crosses involving more diverse genetic material should produce greater genetic effects.

Combining Ability Analysis

Since the variable disease ratings obtained for the backcross generations may have affected the estimates of genetic effects, the genetic analysis outlined by Griffing (1956) was computed to test for general and specific combining ability. Only the means of the F_1 generations were involved in this analysis.

The null hypothesis that there were no differences among the F_1 genotypes was rejected since highly significant F ratios were obtained for F_1 genotypes from the analysis of variance. These significant results justified continuing with the combining ability analysis. Mean squares for the combining ability analysis are given in Table XVI. General combining ability effects were highly significant and specific combining ability effects were negligible. These results are further evidence that additive gene action is involved in the inheritance of resistance to Fusarium wilt in burley tobacco.

Within the limits of this study, the mode of inheritance and genetic effects involved in conditioning resistance to individual Fusarium

TABLE XVI

MEAN SQUARES FROM THE COMBINING ABILITY ANALYSIS

Source	D. f.	Mean Square
General combining ability	4	1.4786**
Specific combining ability	5	.0386
Error	18	.0296

isolates were very similar. However, the genetic patterns displayed in genetic studies with individual isolates of an organism may vary depending upon the selection of specific tobacco genotypes and biotypes of the organism. Even though this investigation revealed similarity in the mode of inheritance of resistance to individual isolates of Fusarium, under a controlled environment and rigid selection of both host and pathogen a simple inheritance pattern for certain genotype and isolate combinations may be possible. A refined procedure of this type would be of little practical value to the breeding program since the plant breeder is interested in incorporating resistance to a mixture of Fusarium isolates in a naturally infested area.

Since the results of this investigation, and some earlier work indicate that the inheritance of resistance to Fusarium wilt is multigenic, and that additive gene effects and general combining ability are predominant, breeding programs should be designed to accumulate favorable genes for resistance. This accumulation of resistant genes can be accomplished through a hybridization method with resistant varieties and the recurrent selection of superior resistant progeny from the segregating populations.

CHAPTER V

SUMMARY

A study was conducted to investigate the mode of inheritance of resistance to individual isolates of Fusarium oxysporum var. nicotianae in burley tobacco. Five burley tobacco varieties, Ky. 12, Ky. 10, Bu. 37, Bu. 21 and Judy's Pride, which ranged from highly resistant to highly susceptible were used in a diallel crossing scheme to provide segregating material for the study. The plants were inoculated in the greenhouse at approximately six weeks of age and were subjectively evaluated for their disease reaction.

The population means obtained were analysed by Gamble's (1962) procedure to estimate additive, dominance, additive X additive, additive X dominance, and dominance X dominance gene affects involved in resistance to the Fusarium isolates. The F_1 means were subjected to a combining ability analysis outlined by Griffing (1956).

Although a statistical difference in virulence was found among the Fusarium isolates, and preliminary investigations with differential host plants revealed that at least two and possibly more physiological races were present among them, the mode of inheritance and genetic effects for resistance appeared to be very similar for each isolate.

Results indicated that resistance to the Fusarium oxysporum isolates of burley tobacco genotypes used in this study was inherited

quantitatively and that additive genetic effects, and general combining ability effects made a major contribution. Dominance and epistatic genetic effects were also important in some populations.

These results suggest that the tobacco breeder can effectively use a mixture of Fusarium isolates when screening breeding lines possessing Nicotianae tabacum type resistance to Fusarium wilt. The breeding program should also be designed to accumulate genes for resistance into pure lines since general combining ability and additive genetic effects are of major importance.

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LITERATURE CITED

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APPENDIX

TABLE XVII

MEAN DISEASE INDEX OF 10 TOBACCO POPULATIONS INOCULATED WITH FOUR ISOLATES OF FUSARIUM OXYSPORUM VAR. NICOTIANAE

	439			NC-2			Whiteville			Greene	
	Planting Date			Planting Date			Planting Date			Planting Date	
Generation	1	2	3	1	2	3	1	2	3	2	3
Ky. 12 (P_1)	.20	.90	.80	.20	.00	1.00	.10	.80	.40	.30	.00
Ky. 10 (P_2)	1.10	2.22[a]	1.60	1.40	1.50	1.80	.80	1.30	1.80	1.10	1.60
F_1	1.22	2.00	1.10	1.30	.90	1.70	1.20	1.20	1.20	1.00	.70
F_2	1.35	1.80	2.15	1.45	.80	1.75	1.35	1.15	1.10	2.40	.25
P_1F_1	1.20	1.00	2.00	1.10	1.80	1.60	1.00	2.00	.40	.20	.20
P_2F_1	.90	2.10	1.40	1.40	1.80	1.30	.60	1.60	1.30	2.01*	1.60
Ky. 12 (P_1)	.20	.90	.80	.20	.00	1.00	.10	.80	.40	.30	.00
Bu. 37 (P_2)	1.90	4.60	2.50	1.90	2.10	3.00	1.90	2.40	2.90	3.00	3.40
F_1	.88	2.40	2.00	2.00	1.90	1.50	1.20	1.50	1.70	1.90	1.30
F_2	.90	1.85	2.10	1.30	.70	1.75	1.05	1.70	1.40	2.20	1.85
P_1F_1	1.30	1.90	1.90	2.00	1.90	1.40	.70	1.10	2.20	1.50	.70
P_2F_1	1.00	4.40	2.20	1.80	1.50	2.20	1.20	1.40	1.60	3.00	1.10
Ky. 12 (P_1)	.20	.90	.80	.20	.00	1.00	.10	.80	.40	.30	.00
Bu. 21 (P_2)	3.40	4.40	3.00	2.30	2.40	3.60	3.20	3.40	4.10	4.00	3.80
F_1	.90	3.50	1.60	1.70	1.40	1.70	1.40	2.50	1.60	2.20	1.40
F_2	1.20	3.65	2.10	1.35	2.10	1.75	1.15	1.05	1.40	2.60	1.85
P_1F_1	1.50	1.70	1.20	1.50	1.40	1.50	.70	1.80	1.90	1.20	2.00
P_2F_1	1.20	3.50	3.00	1.80	1.70	3.50	1.90	2.30	2.50	3.00	1.60

TABLE XVII (CONTINUED)

Generation	439			NC-2			Whiteville			Greene	
	Planting Date			Planting Date			Planting Date			Planting Date	
	1	2	3	1	2	3	1	2	3	2	3
Ky. 12 (P_1)	.20	.90	.80	.20	.00	1.00	.10	.80	.40	.30	.00
Judy (P_2)	4.90	4.80	4.60	4.30	4.50	4.50	3.70	4.50	4.10	4.70	3.80
F_1	2.70	2.60	2.10	2.50	2.10	2.40	2.30	2.10	2.10	2.10	2.50
F_2	2.45	3.62[a]	2.10	2.30	2.20	2.60	2.00	3.15	3.60	2.75	2.20
P_1F_1	1.70	3.60	1.70	2.30	1.60	2.80	1.40	3.10	1.10	1.40	1.50
P_2F_1	4.50	4.52[a]	3.20	3.90	2.30	4.50	2.50	3.90	3.00	4.60	1.70
Ky. 10 (P_1)	1.10	2.22	1.60	1.40	1.50	1.80	.80	1.30	1.80	1.10	1.60
Bu. 37 (P_2)	1.90	4.60	2.50	1.90	2.10	3.00	1.90	2.40	2.90	3.00	3.40
F_1	1.40	3.20	2.10	2.30	1.80	1.90	1.50	2.30	2.40	3.20	1.50
F_2	1.75	2.75	1.45	2.50	1.20	2.10	1.55	1.85	2.60	2.55	2.00
P_1F_1	1.40	4.00	2.70	2.20	.90	1.40	1.30	3.10	1.70	2.00	.80
P_2F_1	2.00	3.10	2.30	1.80	1.50	3.50	2.20	2.10	2.60	3.00	1.50
Ky. 10 (P_1)	1.10	2.22	1.60	1.40	1.50	1.80	.80	1.30	1.80	1.10	1.60
Bu. 21 (P_2)	3.40	4.40	3.00	2.30	2.40	3.60	3.20	3.40	4.10	4.00	3.80
F_1	3.30	2.90	1.70	1.80	2.10	2.90	1.80	2.60	2.50	3.20	2.40
F_2	2.05	3.60	1.80	1.95	1.95	2.60	2.15	2.60	1.70	2.30	1.75
P_1F_1	2.30	4.20	3.10	2.30	3.10	2.90	1.30	3.20	2.60	1.60	2.00
P_2F_1	3.40	4.10	2.30	2.10	2.40	2.50	2.30	3.40	2.20	3.10	2.80
Ky. 10 (P_1)	1.10	2.22	1.60	1.40	1.50	1.80	.80	1.30	1.80	1.10	1.60
Judy (P_2)	4.90	4.80	4.60	4.30	4.50	4.50	3.70	4.50	4.10	4.70	4.40
F_1	2.50	4.70	2.40	2.30	2.80	2.70	2.70	3.60	4.00	3.20	2.40
F_2	3.00	4.65	3.25	2.15	2.45	2.70	1.65	3.05	3.25	2.10	2.30
P_1F_1	2.90	4.90	2.90	1.60	2.70	3.40	2.90	1.50	2.60	3.40	2.40
P_2F_1	2.70	3.70	3.50	3.70	3.10	4.30	2.10	3.70	2.60	3.70	3.80

TABLE XVII (CONTINUED)

Generation	439			NC-2			Whiteville			Greene	
	Planting Date			Planting Date			Planting Date			Planting Date	
	1	2	3	1	2	3	1	2	3	2	3
Bu. 37 (P_1)	1.90	4.60	2.50	1.90	2.10	3.00	1.90	2.40	2.90	3.00	3.40
Bu. 21 (P_2)	3.40	4.40	3.00	2.30	2.40	3.60	3.20	3.40	4.10	4.00	3.80
F_1	2.30	4.60	2.50	2.10	2.50	2.60	1.10	3.70	3.60	4.10	2.80
F_2	2.90	3.95	3.00	1.60	1.90	1.80	1.65	2.60	2.50	4.45	2.65
P_1F_1	2.10	5.00	3.20	2.10	2.80	2.30	1.80	2.90	2.30	4.90	3.00
P_2F_1	2.50	4.30	3.50	1.90	1.10	2.90	2.80	3.30	3.10	4.00	2.40
Bu. 37 (P_1)	1.90	4.60	2.50	1.90	2.10	3.00	1.90	2.40	2.90	3.00	3.40
Judy (P_2)	4.90	4.80	4.60	4.30	4.50	4.50	3.70	4.50	4.10	4.70	4.40
F_1	3.20	4.80	2.80	3.80	2.90	3.20	2.70	4.60	3.30	4.20	4.10
F_2	3.10	3.90	2.45	2.80	2.75	4.00	1.75	3.55	2.70	4.10	3.15
P_1F_1	2.50	4.50	2.70	4.00	1.30	2.80	1.40	4.00	2.30	4.30	2.70
P_2F_1	5.00	4.50	4.10	3.90	3.10	4.40	2.70	3.70	3.90	4.20	4.10
Bu. 21 (P_1)	3.40	4.40	3.00	2.30	2.40	3.60	3.20	3.40	4.10	4.00	3.80
Judy (P_2)	4.90	4.80	4.60	4.30	4.50	4.50	3.70	4.50	4.10	4.70	4.40
F_1	4.40	4.10	3.70	3.80	4.30	4.10	2.90	4.20	3.90	4.80	3.90
F_2	4.10	4.65	4.00	2.55	2.95	3.95	2.10	4.50	3.90	4.60	3.55
P_1F_1	4.40	3.40	4.30	3.30	3.10	3.70	4.20	3.60	2.90	4.60	4.00
P_2F_1	4.50	4.90	4.60	2.80	4.20	4.60	4.00	4.80	4.30	4.80	4.10

[a]Missing plot estimate.

VITA

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