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INHERITANCE OF RESISTANCE TO FUSARIUM OXYSPORUM

F. SP. CEPAE IN CULTIVATED ONIONS

presented by

Jeffery W. Bacher

has been accepted towards fulfillment
of the requirements for

Masters degree in Horticulture

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INHERITANCE OF RESISTANCE TO *FUSARIUM OXYSPORUM* F. SP. *CEPAE* IN CULTIVATED ONIONS

by

Jeffery W. Bacher

A THESIS

Submitted to
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ABSTRACT

INHERITANCE OF RESISTANCE TO *FUSARIUM OXYSPORUM* F. SP. *CEPAE* IN CULTIVATED ONIONS

By

Jeffery W. Bacher

The inheritance of resistance to *Fusarium oxysporum* f. sp. *cepae* was studied to increase the effectiveness and speed of introduction of Fusarium resistant cultivars. The effect of inbreeding on the level of resistance was also investigated.

Two genes, *Foc1* (*A*) and *Foc2* (*B*), are proposed to govern resistance to *F. o. cepae*. Results indicated that both *A* and *B* genes were partially dominant (*AA* > *Aa* and *BB* > *Bb*) and that the interaction between loci was additive (*AABB* > *AABb* or *AaBB* > *AaBb*). Resistance was not epistatic; plants with genotypes AAbb, Aabb, aaBB and aaBb were susceptible. Three genotypes resulted in a resistant phenotype; *AABB*, *AABb*, and *AaBB*. Genotype *AaBb* was moderately resistant.

Three cycles of screening for resistance and selfing of line 6701 resulted in an 89% reduction in the level of resistance to *F. o. cepae*. The original level of resistance was not restored by sib mating of S_1 (selfed once) generation plants. Lethal genes did not appear to be the major cause of this decline.

ACKNOWLEDGEMENTS

I wish to express my sincere thanks and appreciation to my guidance committee; Dr. M. L. Lacy, Dr. Jim Hancock and Dr. Lowell Ewart. I would also like to thank Uma Gupta for taking the time to discuss my research and his helpful suggestions.

Mostly I wish to express my love and appreciation to my family... Susan, Aaron and Nora... they are my source of love and strength.

To Susan... for your patience and love... I dedicate my work and my life to you.

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INTRODUCTION

Thirty to thirty-five percent of world crop production is lost annually to diseases, pests, and weeds (22,44). About 10 to 12 percent of this loss is attributable to plant diseases (22,44,66). The USDA (86) estimated the average annual loss from all diseases of onions grown in the United States at 20 percent. Fusarium basal rot caused by Fusarium oxysporum f. sp. cepae (F. o. cepae) is responsible for 4 percent of this loss. The United States is the fourth largest onion producer in the world, with a crop valued at $428 million (87). A 4 percent yield reduction from F. o. cepae would represent a loss of $17 million annually. The State of Michigan ranks eighth in the country in production of onions, with a crop valued at $16.4 million (87). If 4 percent of the crop were lost to F. o. cepae that would be a loss of $0.66 million annually.

The need to control losses due to disease is obvious. Millions of dollars are spent annually on chemical applications to reduce or prevent these losses. This adds to the cost of production and is ultimately passed on to the consumer in terms of increased cost and pollution to our environment.

One of the most appealing solutions to losses from disease is the use of disease resistant cultivars. The advantages of resistance as a control strategy are primarily its effectiveness and relatively low cost. Resistant cultivars do not require the application of chemicals, or at least reduced levels of them, and are therefore environmentally sound. Often there may be no effective and economical chemical control available to growers, which has been the case with F. o. cepae.

The incorporation of disease resistance into onion breeding lines is one of the major objectives of the onion breeding program at Michigan State University. Fusarium oxysporum f. sp. cepae, a fungal disease which causes the onion bulb to rot in the field or in storage, is a potentially serious disease. Because of the length of time necessary to develop new cultivars, foresight must be used in development of resistant cultivars before wide-spread epidemics and serious losses occur. Knowledge of the inheritance of disease resistance to F. o. cepae would greatly increase effectiveness and speed of introduction of Fusarium resistant cultivars. Therefore, a study of the genetics of resistance to F. o. cepae was undertaken.

LITERATURE REVIEW

PATHOGEN: *FUSARIUM OXYSPORUM* F. SP. *CEPAE* (*F. o. cepae*)

Formae speciales of *Fusarium oxysporum* occur worldwide and are the most commonly found Fusaria in soils, accounting for 40 to 70 percent of the total Fusarium species present (32). Most of these species are beneficial saprophytes which decompose dead organic matter (82). *F. o. cepae* is a facultative saprophyte; it can parasitize a host plant or remain dormant in decaying plant material until conditions are favorable for germination (62). Dissemination can occur by wind, water, farm equipment or by use of infected onion sets (4,62). Katan (51) also, reported that *F. o. cepae* can be carried on onion seed.

The pathogen invades onion bulbs through the root tips or injuries caused by other organisms (2). The deterioration begins at the base of the bulb with a mealy decay of all tissues at the basal plate and progresses upward into the bulb center (26,55). This area may become covered with white to purple cottony mycelium (55). If the bulb loses water rapidly, it will become tough, leathery and mummified. Bulbs may be completely rotted by harvest

or slowly decay during transit and storage (26). Foliar symptoms of F. o. cepae begin with a chlorosis and dieback of leaf tips (18,45). Part or all of a leaf may become chlorotic and die from the tip to the base.

REDUCTION IN YIELD

Research on yields of onion cultivars on Midwestern organic soils artificially infested with F. o. cepae showed a 53 to 68 percent reduction in yield of the two cultivars most commonly grown in Michigan; Spartan Banner and Krummery Special (54). Both numbers of plants and weight of bulbs per hectare were significantly reduced in infested soils. There was a 60 percent mean reduction in yield in the F. o. cepae infested trials compared to the non-infested trials.

F. o. cepae resistance trials conducted in Michigan on naturally infested soil reported losses due to Fusarium basal rot in field plus normal storage conditions of 30 percent in Spartan Banner 80, 41 percent in Krummery Special and 37 to 54 percent in Sweet Sandwich (97). Yield reductions at harvest ranged from 0 to 23 percent (6.8% mean reduction) and from 3 to 67 percent loss in storage (16.2% mean loss). The combined average loss for both field and storage was 23 percent.

DISEASE MANAGEMENT WITH RESISTANCE

The traditional method of control of F. o. cepae has been through disease resistance (54). The advantages of

REDUCTION IN YIELD

Research on yields of [illegible] on Midwestern

resistance as a control strategy are: (1) that resistance is inherited and therefore is present in the plant, (2) it is not detrimental to the environment, (3) it is highly selective, (4) non-phytotoxic, (5) can be highly effective in disease control, and (6) requires low maintenance costs once cultivars are developed (31). On the other hand, breeding for disease resistance can take many years and is often only partially effective. In some cases resistance genes are not available in germplasm which will cross normally with Allium cepa. Because of the existence of different races of a pathogen a cultivar may be resistant in one growing area and not in others. A mutation to virulence in the pathogen can also result in a loss of resistance in a cultivar that was previously resistant to the disease.

INHERITANCE OF RESISTANCE TO FORMAE SPECIALES OF FUSARIUM OXYSPORUM

The inheritance of resistance in plants to many Fusarium oxysporum species has been studied. This knowledge of the nature of inheritance suggests a pattern with which to compare the onion host-pathogen system. A partial summary of genetic studies on inheritance of resistance to Fusarium oxysporum is given (Figure 1).

Resistance to plant diseases is generally inherited dominantly and virulence of the pathogen inherited recessively (27,79,90). Sidhu (79) reviewed 1042 papers on the inheritance of disease resistance and found 927 (89%)

resistance as a control strategy are: (1) that resistance is inherited and therefore is present in the plant, (2) it is not detrimental to the environment, (3) it is highly selective, (4) non-phytotoxic, (5) can be highly effective in disease control, and (6) requires low maintenance costs once cultivars are developed [illegible]. On the other hand, breeding for disease [illegible] wilt.

Figure 1. Summary of Genetic Studies on Inheritance of Resistance to Formae Speciales of Fusarium oxysporum.

Common Name	Author	Monogenic Dominant	Monogenic Recessive	Partial Dominant	Polygenic
Alfalfa	Hijano(36)	x		x	
Bean[z]	Ribeiro(72)	race 1		race 2	
	Bravo(10)			x	x
Cabbage	Walker(92)	x			
Celery	Orton(67)	x			
Cotton	Netzer(64)	x			
	Kappleman (50)				x
Cowpea	Rigert(73)	race 1 race 2 race 3			
Cucumber	Netzer(63)	x			
Pea	Haglund(33)	x			
Chickpea	Sindhu(80)		x		
Musk-melon	Zink(99)	x			
Radish[z]	Peterson(70)				x
Sweet Potato	Collins(20)				x
Tomato	Circulli(19)	race 1 race 2			
Tulip[z]	Eijk(91)				x
Water-melon	Netzer(65)	x			
	TOTAL	14	1	3	5

[z] Host of root rot Fusaria (all others are hosts of vascular wilt Fusaria).

reported resistance due to dominant genes and 108 to recessive genes and that inheritance was usually monogenic. Sidhu (79) speculated that many reported cases of multigenic resistance might have been due to the use of pathogen populations composed of many races in the inheritance studies.

Most of the crops summarized in Figure 1 are hosts for vascular wilt Fusaria. Inheritance of resistance to these wilt Fusaria is almost exclusively monogenic dominant. Of the crops listed which are hosts for root rot Fusaria (bean, radish, tulip) inheritance was found to be polygenic. Bravo (10) found that resistance in beans (Phaseolus vulgaris) was largely but not fully dominant over susceptibility. Estimates of the number of genes controlling resistance ranged from 3 to 7. In a study on resistance in radish to Fusarium oxysporum f. conglutinans F1 progenies of susceptible x resistant plants expressed a level of resistance intermediate between the parents (70). The F2 progenies and backcrosses showed resistance of about 15% suggesting that resistance was polygenic in nature.

PERMANENCE OF GENETICALLY INHERITED RESISTANCE TO FUSARIA

Van der Plank (88) has classified all plant disease into two epidemiological categories depending on whether their epidemics are mathematically analogous to simple interest or compound interest in money. Fusarium is considered to be a typical example of simple interest diseases and is therefore predicted to spread slowly,

reported resistance due to dominant genes and 100 to recessive genes and that inheritance was usually monogenic. Sidhu (79) speculated that many reported cases of monogenic resistance might have been due to the use of pathogen populations composed of many races in the inheritance studies.

perhaps taking decades for a new race to become widespread. F. o. cepae is a soil-born disease with no known sexual cycle (60). Without a sexual cycle, recombination of genes to produce more virulent races is greatly reduced and mutation, a much slower process, provides the organisms main method of variation (60). In such cases monogenic resistance is often long lasting (74). Vertical resistance, usually involving resistance mechanisms whose inheritance is governed by single genes, is considered to be generally effective against formae speciales of Fusarium oxysporum (74,89,90). Robinson (74) states that Fusarium oxysporum can be completely controlled by vertical resistance provided that: the host is not an annual, that at least one 'strong' gene is known, and that rotation is practiced.

Crill (23) reports that monogenic resistance to race 1 was completely effective in preventing losses from Fusarium wilt for 11 years (from 1949 to 1960) in Florida's tomato crop. Race 2 was discovered in 1960 in Florida, but by the time it had become a major state wide problem varieties with monogenic resistance to race 1 and 2 were developed.

Monogenic dominant "Type A" resistance has successfully controlled cabbage yellows, caused by Fusarium oxysporum f. sp. conglutinans, for more than 50 years (9).

perhaps taking decades for a new race to become widespread. *F. o. cepae* is a soil-borne disease with no known sexual cycle (60). Without a sexual cycle, recombination of genes to produce more virulent races is greatly reduced and mutation, a much slower process, provides the organisms main method of variation (60). In such cases monogenic resistance is often [illegible] Vertical resistance [illegible]

[illegible]

MECHANISMS OF RESISTANCE

Onion lines varied in their response to infection by F. o. cepae by age (40). In general older plants showed the greatest resistance to infection, followed by young seedlings, then bulbs. It has been suggested that if different types of resistance operate during different stages of development then resistance may be polygenic in nature (40). Soki (81) hypothesized that testing for resistance at the bulb stage would be the most accurate measure of resistance.

Onion cultivars susceptible and resistant to F. o. cepae were found to be anatomically similar and equally susceptible to F. o. cepae in their root and stem plate tissue (1). This agrees with Beckman (7,8) who reported that wilt-producing Fusaria grow equally well in resistant and susceptible tissue. Resistance appears to depend primarily on a physical localization of the parasite (7). It was suggested that systemic distribution of the pathogen in a susceptible type interaction is dependent upon fungal products which rapidly degrade vascular gels and which inhibit respiratory or growth metabolism of host cells.

Vascular occlusion is common and has been considered a major factor in disease development and resistance to many wilt diseases caused by F. oxysporum (7,8). The blocked vessels reduce or eliminate the spread of the pathogen through the vascular system, thereby localizing the fungus. Beckman (7) reported that tolerant varieties exhibited more

and earlier vascular occlusion than susceptible varieties when infected with vascular wilt Fusaria. Tylose formation, occlusion of xylem vessels and hypertrophy were reported by Shalaby (75) in F. o. cepae infected onion tissues. Abawi (1) found that xylem vessels were clogged by tylose-like structures in both tolerant and susceptible varieties.

Pennypacker (69) reported that vascular wilt Fusaria and the root rot Fusaria use different mechanisms to attack their hosts. Root rot Fusaria are generally confined to the cortical areas of their host, only penetrating the vascular system late in the disease process. The two groups of Fusaria induce different anatomical responses in their hosts; gums, gels, and tyloses follow invasion by the vascular wilt Fusaria, whereas hypertrophied and hyperplastic tissue formation is generally found in response to infection by the cortical rot Fusaria.

Plant pathogens produce an array of enzymes capable of attacking plant cell components. It has been demonstrated that pectic enzymes can macerate and kill plant tissues in a manner similar to that occurring in soft-rot diseases and that fragments released from the cell wall can elicit plant defense reactions (21).

Collmer (21) reviewed the role of pectic enzymes in plant pathogenesis and outlined the following steps which occur during the interaction of a pectolytic pathogen and a potential host:

"...(1)the entering pathogen possesses structural genes

and earlier vascular occlusion than susceptible varieties when infected with vascular wilt Fusaria. Tylose formation, occlusion of xylem vessels and hypertrophy were reported by Sh[illegible] (75) in *F. o. cepae* infected onion tissues. Abawi (1) found that xylem vessels were clocked by tylose-like structures in both tolerant and susceptible varieties.

Pennypacker [illegible] vascular wilt Fusaria [illegible]

encoding pectic enzymes, (2)these genes are expressed in a characteristic manner in the infected tissue, (3)the enzymes are exported from the pathogen cytoplasm to the host tissue, (4) in some tissues the enzymes encounter inhibitors or protected substrates, (5) in other tissues the enzymes are active and cleave structural polymers in the primary cell wall and middle lamella, facilitating pathogen penetration and colonization."

F. o. cepae produces two types of pectic enzymes, exo-polygalacturonase (exo-PG) and endo pectin trans eliminase (endo-PTE) on a mineral medium supplemented with onion cell walls (41). These enzymes are also synthesized in vivo and have been extracted from infected onion tissue in which the onset of bulb rot was correlated with the presence of endo-PTE.

Endo-PTE is the main enzyme responsible for host tissue maceration and cell death (42). However, Endo-PTE showed little activity during the early stage of onion stem plate infection, even though in culture the stem plate cell walls induced extensive production of pectic enzymes. Low pH of stem plate tissue could suppress production or activity.

Similar patterns of fungal distribution and enzyme production have been found during the early stages of infection of both susceptible and tolerant plants (41). The fungus rapidly invaded stem plate tissue then spread more slowly to the outer bulb scales and eventually to base

endogenous acetic enzymes. Although genes are repressed in a characteristic manner in the infected tissue, (1)the enzymes are secreted from the [illegible] [illegible] host [illegible] (2) some tissues the enzymes encounter [illegible] substrates, (3) in other tissues [illegible] leave structural [illegible]

of the inner leaf sheaths. Different genotypes vary markedly in their ability to contain the pathogen to the stem plate, thereby delaying bulb decay. Susceptible plants restricted the pathogen to the stem plate from 2 to 3 months, whereas tolerant plants contained it for up to 9 months. This suggests that additional factors may operate in bulb scale and leaf sheath tissue of tolerant plants to interfere with pectic enzyme activity and thereby delay spread of pathogen.

It has been shown that cell walls from different parts of onion bulbs and from bulbs of genotypes differing in resistance to F. o. cepae differed in the extent to which they induced the fungus to produce endo-PTE (42). These patterns of enzyme induction were correlated with resistance and susceptibility and the different patterns of host tissue colonization by the fungus.

Pectic enzymes secreted by plant pathogens in vivo are influenced by host sugar content (29,43). Sugar contents of equivalent parts of two onion genotypes susceptible and resistant to F. o. cepae were similar, but different parts showed marked differences. Sugars are present in bulb scales and leaf sheaths in sufficient concentrations to repress pectic enzyme synthesis by the fungus; whereas lower sugar concentrations, equivalent to those in mature bulbs and stem plate tissue, are too low to suppress endo-PTE synthesis. Different parts of the onion bulb differ in their susceptibility to bulb rot. Bulb scales and

leaf sheaths are resistant (high sugar levels), stem plates susceptible (low sugar levels). It was suggested that differences in sugar content could help to explain differences in susceptibility between different parts of the bulb.

Holz and Knox-Davis (42) hypothesized that the slow spread of the pathogen from the stem plate to the outer bulb sheaths was due in part to the low pectic enzyme inducing properties of the cell walls of these tissues. This delay in endo-PTE synthesis would minimize cell damage and enable host defense mechanisms to localize the fungus to the stem plate tissue. Suggested defense mechanisms of tolerant genotypes which could result in further localization of the pathogen to the stem plate tissue are: (1) phytoalexin production(52,53); antifungal substances are produced in inoculated onion bulb scales and leaf sheath tissue(1), (2) formation of structural barriers; tyloses and gum-like material are formed in uninvaded xylem of the leaf base adjacent to infected stem plate tissue (8,75), (3) enzyme inhibitors or protected substrates(21), (4) hypersensitive reaction or necrotic responses(27,75).

Changes in cell wall structure and resistance of plant tissue occur with age (98). Zink (98) reported a marked decrease in the calcium content of onion bulbs with age. In field studies on onion bulb rot it was found that tolerant genotypes lost their resistance in calcium deficient soils. Histological studies revealed that young bulb scale and

leaf sheaths are resistant (high sugar levels), stem plates susceptible (low sugar levels). It was suggested that differences in sugar content could help to explain differences in susceptibility between different parts of the bulb.

Holz and Knox-Davis [illegible] hypothesized [illegible] spread of [illegible] pathogen [illegible] bulb [illegible]

leaf sheath tissues from bulbs grown in these soils were readily invaded by the pathogen. In onion plants, calcium mobilization and the type of pectic substances formed in bulb scale and leaf sheath tissue could influence pectic enzyme induction by the host cell walls and therefore also the resistance and susceptibility of the tissue.

SOURCES OF RESISTANCE

There are many sources of partial resistance or tolerance to Fusarium basal rot that have been reported in the literature (1,40,81,96). However, since multiple races of the pathogen F. o. cepae may exist, cultivars screened in one growing area may not prove to be resistant in other areas. Also cultivars need to be adapted to local environmental conditions such as daylength, temperature, length of growing season, etc., and may not grow well in areas that differ greatly from those in which they were developed.

Lacy and Zandstra (54,97) evaluated onion cultivars in Michigan growing areas for yield response on soils naturally and artificially infested with F. o. cepae. Two of the cultivars most commonly grown in Michigan, Spartan Banner 80 (which showed a 53% reduction in yield in artificially infested fields) and Krummery Special (which showed a 68% reduction) were among the higher yielding cultivars tested. In naturally infested fields Spartan Banner 80 had a 30% reduction in yield, Krummery Special a 41% reduction and Sweet Sandwich 25%.

Another source of resistance to disease exists in related Allium species. Jones (47) found that Allium fistulosum carried a high degree of resistance not only to Fusarium basal rot, but also to pink root, downy mildew, smut, yellow dwarf virus and thrips. Abawi and Lorbeer (1) compared commercial onion cultivars with other Allium species and interspecfic hybrids and found that the cultivars possessing the highest resistance were Beltsville Bunching (A. fistulosum x A. cepa) and Japanese Bunching (A. fistulosum) which had 85 and 98 percent survival rates respectively.

Among the inbred lines developed by the USDA at University of Wisconsin, line 6701 has shown the highest level of resistance to F. o. cepae with 97% of seedlings screened surviving (68). Line 6701 was included in this inheritance study as a resistant parent.

MATERIALS AND METHODS

SCREENING FOR RESISTANCE TO *FUSARIUM OXYSPORUM* F. SP. *CEPAE*

Greenhouse screening for resistance to *F. o. cepae* was selected over field testing because of the need for increased control over environmental conditions, inoculation levels, race(s) of pathogen used, and the flexibility of conducting screening trials when desired. A high positive correlation between greenhouse and field testing for resistance to *F. o. cepae* (71) indicates that results obtained under greenhouse conditions will be applicable to field situations.

Disease development increases with increasing inoculum concentrations of *F. o. cepae* (3,58); therefore an inoculum concentration must be chosen which will cause serious disease in susceptible but not resistant lines. Inoculum concentrations of 20,000 to 40,000 microconidia per gram of sand, typically used for *F. o. cepae* trials by others (68), were found to be too extreme for this study.

Inoculum levels of 10,000 microconidia per gram of sand were used in this study. This level was selected based on inoculum concentration experiments (68) conducted on inbred lines 1849 and 6701 (the same lines used in this study) at

University of Wisconsin and on preliminary screening trials which showed that seedlings from susceptible line 1849 were all killed at concentrations as low as 10,000 microconidia per gram of sand.

SCREENING PROCEDURE FOR *FUSARIUM OXYSPROUM* F. SP. *CEPAE* RESISTANCE IN ONION.

The screening procedure was based primarily on the method developed by Mary Palmer and Paul Williams of the Department of Botany and Plant Pathology, University of Wisconsin (68). Single spore isolate F593-1, obtained from a *F. o. cepae* culture designated as PHW 593-19, was used in this study. The culture was originally isolated from a field in Wisconsin and was obtained from Dr. M. L. Lacy, Department of Botany and Plant Pathology, Michigan State University.

F. o. cepae cultures are maintained on sterilized muck soil at 4°C. Infested soil was transferred to potato dextrose agar plates and grown at 24°C. A 7 mm diameter piece of colonized agar was aseptically added to 50 ml of potato dextrose broth in a 125 ml flask then incubated on a shaker at 24 to 26°C for 3 to 6 days. The mycelia and broth were blended at low speed for 2 minutes, centrifuged for 10 minutes at 3400 rpm and the pellet was resuspended in distilled water. The microconidia were then counted with a hemacytometer to determine concentration.

An inoculation concentration of 10,000 microconidia per gram of sand was used. Twenty kilograms of white

silica sand was weighed out, placed in a 50 x 29 x 11 cm stainless steel pan and sterilized in an autoclave. The appropriate amount of spore suspension was added to distilled water to give 2000 ml total volume and provide 10,000 spores per gram of sand. The inoculum was mixed thoroughly with the sand. Grooves 15 mm apart and about 5-7 mm deep were made across the pan. One hundred seeds per row were planted and covered. The pan was covered with aluminum foil and placed in a controlled temperature water bath set at 24°C. under high intensity discharge lights for a 15-16 hour photoperiod. The sand temperature was maintained at 20-22°C. The foil was removed after the seedlings had emerged. When cotyledons were about 1 cm tall, the sand temperature was increased to 26-28°C (the optimum temperature range for the growth of F. o. cepae in culture is 24 to 27°C (3,26,93)). Plants were watered as needed and 15:16:17 fertilizer was applied weekly after two weeks at a rate of 200 ppm Nitrogen. The number of seedlings were counted 10 days after sowing and again 21 and 28 days later. On day 28 the seedlings were evaluated and counted as either healthy or diseased.

PATHOGENICITY TESTS

Pathogenicity tests were conducted with six isolates of F. o. cepae. The isolates used were obtained from Dr. M. L. Lacy, Department of Botany and Plant Pathology, Michigan State University. Three isolates originally obtained from Wisconsin were designated as F593, F156-A, and F156-B; two

were isolated from New York fields, F110-A and F110-B; one was isolated from an infested onion field in Michigan, FOC-1-1. An inoculation concentration of 20,000 microconidia per gram of sand was used. The isolates used were single spored to assure genetic homogeneity, otherwise the screening procedure described previously was followed. Sixty seedlings(each) from three resistant and two susceptible inbred lines selected from preliminary F. o. cepae screening trials were used.

FLOWER AND SEED PRODUCTION

Culture

The onion plants were raised in growth chambers and grown under environmental conditions designed to decrease generation time by bypassing the bulbing stage (Figure 2). This method was based on preliminary research and work done by Shishido (76-78), Brewster (11-15) and others (5,6,34,35,37,38,49,57,83,84,95). Fungicide (benomyl) was applied as a spray and as a drench to reduce F. o. cepae in the non-inoculated control plants.

Pollination

The pollination procedure used was similar to that reported by Jones and Emsweller (47,48). To optimize seed set two aspects of flower development were considered: the flowering period and stigma receptivity.

Flowering period: the inflorescence is a roughly spherical umbel bearing 50 to more than 1,000 flowers on

Figure 2. Cultural Procedure to Decrease Generation Time in Onions Grown From Seed.

I.	Growth Environment (growth chambers)	
	A. Temperature	17°C (63°F)
	B. Light	200 micromoles $m^{-2} s^{-1}$
	C. Photoperiod	24 hrs cool-white fluorescent
	D. Nutrition	100 ppm 15:16:17
	E. Time Period	90 days
	F. Container	18 cell flats
	G. Media	Artificial peat-based media
II.	Flower Initiation (vernalization)	
	A. Temperature	9°C (49°F)
	B. Light	200 micromoles $m^{-2} s^{-1}$
	C. Photoperiod	24 hrs cool-white fluorescent
	D. Nutrition	No fertilizer
	E. Time Period	30-40 days
III.	Flower Bud Development (move to greenhouse)	
	A. Temperature	17°C (63°F) optimal
	B. Light	Natural daylight
	C. Photoperiod	14+ hrs natural daylight
	D. Nutrition	125 ppm 15:16:17
	E. Time to Bloom	50-60 days
	F. Container	6" clay pots
	G. Media	Artificial peat-based media

B.	Temperature	17°C (63°F)
C.	Light	200 microeinsteins m²/s
D.	Photoperiod	
E.		

an aggregate of cymes consisting of from 5 to 10 perfect flowers (47). The flowering of a single umbel may extend over 3 to 4 weeks (25,47). Flowering follows a general pattern where at first the number of flowers opening per day is small, but this rapidly increases, giving a peak which lasts for a average of 4 to 10 days (25). During this peak 50 or more flowers may open in a single day depending upon the temperature and the hours of light that day and the previous day (59).

Stigma Receptivity: Currah and Ockendon (25) found that the onset of stigma receptivity was closely connected with the production of stigmatic exudate. Pollen tubes grew in the stigma before presence of exudate, but few adhered to the stigmatic surface. They suggested the major role of the stigmatic exudate was pollen capture and that it did not have an essential function in pollen germination or tube growth. Moll (61) found that the stigma was most receptive on the 3rd, 4th, or 5th day after opening. The duration of stigma receptivity is strongly influenced by temperature (16,17).

Pollination Technique and timing

The umbels of the plants to be crossed were bagged as soon as the first flower opened. Plants destined to be pollen parents were allowed to flower. Open flowers on plants used as seed parents were removed until 20 or more flowers opened on a single day; thereafter, the anthers were removed from any open flower. Emasculation was

continued each morning and afternoon for a period of 3 to 5 days. At this point all the remaining unopened flowers were removed and a flower head of the pollen parent was tied with the emasculated head, and both are enclosed under the same bag. One-half to one teaspoon of fly pupae were added and within a few days adults emerged and pollinated the female flowers. The heads were bagged separately when pollination was completed, usually 5 to 7 days after flies had hatched. Plants to be self pollinated were bagged individually as soon as the first flower opened and flies were added as pollinators.

Cleaning and Storage of Seed

Once the seed was thoroughly dried it was separated from the receptacle through gentle grinding on a ribbed rubber pad. Separation of light seed and chaff was accomplished with forced airscreen cleaning. This not only removed fragments of receptacle, but also had the effect of removing the poorest quality seed.

Storage of onion seeds in normal temperature and humidity results in fairly rapid deterioration in viability, preceded by a rise in the incidence of cytological damage (24). To prevent deterioration the onion seed was stored at 2°C and 40% relative humidity .

SELECTION OF RESISTANT AND SUSCEPTIBLE PARENTS

Based on preliminary screening trials resistant line 6701 and susceptible line 1849 were chosen as parents for

this inheritance study. Flowering plants from each of the lines were self pollinated (selfed) and progeny tested to determine level of resistance to F. o. cepae. The resistant parent chosen (6701-1) was a single plant selection showing the highest level of resistance of those tested. Inbred lines 6701 and 1849 originated from the University of Wisconsin's breeding program. Line 6701 had been screened several generations for resistance to F. o. cepae and line 1849 has been used as a susceptible check in screening trials.

NOMENCLATURE

Selfed progeny are designated by line number followed by a hyphenated number which represents the n^{th} plant to be selfed (i.e. 6701-1). This nomenclature is extended for 2nd and 3rd generations of selfing (i.e. 6701-1-4-7). Resistant parent 6701-1 is also designated as 6701-1(RES) or RES. Susceptible line 1849 is designated as 1849(SUS) or SUS.

F1, F2, BACKCROSSES, AND SELFS USED IN INHERITANCE STUDY

To study the inheritance of resistance to F. o. cepae crosses between resistant line 6701-1 and susceptible line 1849 were made using pollination techniques previously described. F2's, backcrosses to the resistant and susceptible parents and selfing of parents were also performed.

EFFECT OF INBREEDING ON RESISTANCE

Allium cepae is naturally a cross pollinated species. Inbreeding or selfing of onions results in a decrease of vigor and changes in other traits (47,48). To determine if inbreeding had an effect on the level of resistance, plants from line 6701 were first screened then selfed. This cycle was repeated for three generations, in each case those plant(s) showing the highest level of resistance were selected as parents for the next generation. Progeny from each generation were screened for resistance to *F. o. cepae*.

STATISTICAL ANALYSIS

Chi Square analysis was used to test the hypothesized segregation ratios of F1, F2 and backcross families for goodness of fit to the observed data. Yates correction for continuity was used to calculate chi-square values (adjusted $X^2 = \Sigma$ ($|$observed - expected$|$ - 0.5)^2 ÷ expected).

Heterogeneity tests were also performed on all families to determine population heterozygosity (heterogeneity X^2 = total X^2 - pooled X^2).

A completely randomized experimental design was used in screening trials with a varying number of repetitions depending upon availability of seed. Analysis of variance was performed to test null hypotheses of no differences in the level of resistance to *F. o. cepae* between F1, F2 and backcross families. Because data were in the form of percents, arc-sin transformation was performed to assure homogeneity of variance.

RESULTS AND DISCUSSION

PATHOGENICITY OF SINGLE SPORE ISOLATES OF FUSARIUM OXYSPORUM F. SP. CEPAE

The purpose of the pathogenicity test was to determine: (1) which F. o. cepae isolates were the most virulent, (2) in which isolate there was the greatest range between the most resistant and the most susceptible lines and (3) which isolate gave results approaching the ideal situation (resistant line having 100% survival rate and susceptible line 0% survival rate).

Isolate 593 was chosen for further screening because, while not being the most virulent strain tested, it showed the greatest range between the most resistant (67% in 6701) and most susceptible (0% in 1849) lines (Table 1). Also lines 6701 and 1849 had been selected for resistance and susceptibility to F. o. cepae using isolate 593.

SCREENING RESULTS FOR RESISTANCE TO FUSARIUM OXYSPORUM F. SP. CEPAE

Parent lines, F1, F2 and backcross families were rated for resistance based on the percent survival of those seedlings emerging (Table 3).

The analysis of variance test found a significant difference between the level of resistance to F. o. cepae in the parents , F1, F2 and BC families (Table 2).

RESULTS AND DISCUSSION

[illegible] OF SIMPLE SPOKE ISOLATES OF [illegible]

The purpose of [illegible]

(1) whic[illegible]

Table 1. Pathogenicity of Six Single Spore Isolates of *Fusarium oxysporum f. sp. cepae* on 5 onion lines.

Pedigree	%Germ	Percent Surviving*					
		593	156-A	156-B	110-A	110-B	FOC-1-1
6701	95	67	32	19	14	19	37
6693	70	55	50	52	43	38	69
2399	60	28	17	8	8	3	83
1849	80	0	0	0	0	0	8
611-1	70	0	0	0	0	0	12

* % of emerging seedlings that survived 21 days.

Table 2. Analysis of Variance of F1, F2 and BC Families.

	DF	Sum of Squares	Error Mean Square	F-value	Prob.
Between	36	35469.70	985.27	10.77**	0.000
Within	179	16382.29	91.52		
Total	215	51851.98			

** Highly significant (α=0.5)

Table 3. Summary Statistics of Screening Data for Resistance to *Fusarium oxysporum* f. sp. *cepae*.

CROSS	# PLANTS TESTED[z]	%SURVIVE CONTROL[y]	MEAN %R D21[x]	MEAN %R D28	STD ERR[w]	VARIANCE %R D28[v]
1849(SUS)	126	94	0.00	0.00e	3.91	0.00
SUS(selfed)	223	83	0.48	0.00e	3.62	0.00
6701	136	97	74.70	52.97a	3.91	23.94
RES-1(6701-1)	76	80	82.89	--	--	--
F1	53	96	37.02	10.03bcd	5.52	173.95
F2	331	96	35.79	15.40b	4.28	35.34
F1xF1	175	98	20.11	9.15bcd	4.28	162.78
BC-RES	255	96	65.59	37.46a	4.78	43.32
BC-SUS	202	100	8.57	1.22de	4.28	41.03
F1 (recip)	30	100	37.00	--	--	--
F2 (recip)	309	94	19.96	8.74bc	4.28	38.67
F1xF1(recip)	134	--	13.26	2.72cde	5.52	50.86
BC-RES (recip)	114	98	68.59	37.17a	5.52	99.59
BC-SUS1 (recip)	82	98	2.51	0.00e	4.78	0.00
RES-1xRES-1[u]	72	100	30.44	6.25	5.40	225.00
RES(self 1x)[u]	1088	91	39.52	17.77	2.01	131.29
RES(self 2x)[u]	932	80	27.54	7.08	1.94	108.72
RES(self 3x)[u]	395	89	24.35	6.05	2.48	115.53

[z] Number of seedlings emerged.
[y] % of emerging seedlings surviving 28 days in the control.
[x] % resistance on day 21.
[w] Standard error for transformed data from day 28.
[v] Variance of arc-sin transformed data from day 28.
[u] % resistance corrected for lowered survival rate due to lethal genes by following formula:
(% resistance) x (1 + (1 - frequency of survival in control)).

- BC-SUS is backcross susceptible, BC-RES is backcross resistant. Recip is reciprocal cross 6701-1x1849.
- Any two means with the same letter (abcde) are not significantly different by LSD means separation test (α=0.05).

The resistance values for cross 1849 x 6701-1 and reciprocal cross 6701-1 x 1849 were not significantly different as determined by LSD (α=0.5) test (Table 3). Therefore, it was concluded that resistance to F. o. cepae was not maternally inherited in the lines used in this study.

PROGENY TESTS OF PARENTAL LINES 6701-1(RES) and 1849(SUS)

Segregation of progeny for resistance to F. o. cepae indicated that the parental line 6701-1 was heterozygous at resistant loci and that line 1849 was homozygous susceptible. Progeny tested from selfed 6701-1 plants segregated for resistance and ranged in values from 0% to 42% surviving with a mean of 6%. The mean value for the resistant parent was probably low because of inbreeding depression and will be discussed later. Line 1849 appeared to be homozygous with all progeny testing susceptible.

USE OF GENOTYPE FREQUENCIES TO CALCULATE RATIO OF RESISTANT TO SUSCEPTIBLE OFFSPRING

Because of the heterozygosity of the parental line 6701-1 the ratio of resistant to susceptible offspring for individual F1, F2 and backcross families was expected to vary depending upon genotypes of the parents involved. Therefore, the approach taken was to start with hypothetical parental genotypes then calculate expected genotype frequencies for the different families. These were compared to the observed frequency of plants in each class (i.e. number of plants surviving on day 10, 21 and 28). Mean

percent resistance data for a F1, F2 or backcross families were compared to expected frequencies to test different hypotheses of gene action using chi-square analysis. Parental genotypes AABb for 6701-1 and aabb for 1849 gave the best fit to the observed data.

GENE ACTION OF PROPOSED RESISTANCE GENES Foc1 (A), Foc2 (B) AND LETHAL 1 GENE

Two genes; Foc1 (A) and Foc2 (B), are proposed to govern disease resistance to Fusarium oxysporum f. sp. cepae. Several models are proposed for their gene action:

Model I. (see Table 4 for expected gene frequencies). Both A and B genes are partially dominant (AA >> Aa and BB >> Bb) and the interaction between loci is additive (AABB > AABb or AaBB > AaBb). Resistance is not epistatic; both resistant alleles A and B must be present for the plant to remain healthy in screening trials past 28 days. Plants with genotypes AABB, AABb and AaBB are considered resistant. Plants heterozygous at both loci (AaBb) are intermediate in resistance and remain healthy for 21 days, then become diseased and may die. Genotype aabb is completely susceptible with all plants screened dying around day 10.

Model II. (Table 5). A gene is partially dominant and B gene is dominant. The interaction between loci is additive. This gene action results in only two resistant genotypes, AABB and AABb.

Model III. (Table 6). Both A gene and B genes are partially dominant. The interaction between loci is additive. In addition, a lethal recessive gene l results in death of seedlings after emergence. Gene l occurs only in the resistant parent.

Model IV. (Table 7). Both A and B genes are partially dominant. The interaction between loci is additive. In addition, recessive lethal gene l occurs in both resistant and susceptible parents in heterozygous state (Ll).

Table 4. Expected Genotype Frequencies of Parents, F1, F2 and Backcross Families for Cross 1849(P1) x 6701-1(P2). Model I. A and B Genes are Both Partially Dominant. Resistant Genotypes (3) are AABB, AABb, and AaBB.

TYPE	P1	FREQ	P2	FREQ	CROSS	FREQ	FREQUENCY OBS[z]	FREQUENCY EXP[y]
F1	aabb	1.00	AABb	1.0	AaBb	0.500	0.094	0.000
					Aabb	0.500		
F2	AaBb	0.50			AABB	0.031	0.151	0.156
	Aabb	0.50			AABb	0.063		
					AAbb	0.156		
					AaBB	0.063		
					AaBb	0.125		
					Aabb	0.313		
					aaBB	0.031		
					aaBb	0.063		
					aabb	0.156		
BC-R	AaBb	0.50	AABb	1.0	AABB	0.063	0.396	0.375
	Aabb	0.50			AABb	0.250		
					AAbb	0.188		
					AaBB	0.063		
					AaBb	0.250		
					Aabb	0.188		
BC-S	AaBb	0.50	aabb	1.0	AaBb	0.125	0.015	0.000
	Aabb	0.50			Aabb	0.375		
					aaBb	0.125		
					aabb	0.375		
RES[x]	AABb	1.00			AABB	0.250	0.820	0.750
					AABb	0.500		(D21)[w]
					AAbb	0.250		

z Observed (OBS) is the observed frequency of resistant seedlings after 28 days of screening.

y Expected (EXP) frequency of resistance = freq of resistant genotypes (underlined) X (1 - frequency of lethals(11).

x Resistant parent line 6701-1.

w Day 21 data used because day 28 data not available.

- FREQ = frequency, BC-S AND BC-R = backcross susceptible and resistant.
- Resistant genotypes and their frequencies are underlined.
- Parental genotypes: 1849 = aabb, 6701-1 = AABb.

Table 5. Expected Genotype Frequencies of Parents, F1, F2 and Backcross Families for Cross 1849(P1) x 6701-1 (P2). Model II. A Gene is Partially Dominant and B Gene is Dominant. Resistant Genotypes (2) are AABB and AABb.

TYPE	P1	FREQ	P2	FREQ	CROSS	FREQ	FREQUENCY OBS[z]	FREQUENCY EXP[y]
F1	aabb	1.00	AABb	1.0	AaBb	0.500	0.094	0.000
					Aabb	0.500		
F2	AaBb	0.50			AABB	0.031	0.151	0.094
	Aabb	0.50			AABb	0.063		
					AAbb	0.156		
					AaBB	0.063		
					AaBb	0.125		
					Aabb	0.313		
					aaBB	0.031		
					aaBb	0.063		
					aabb	0.156		
BC-R	AaBb	0.50	AABb	1.0	AABB	0.063	0.396	0.313
	Aabb	0.50			AABb	0.250		
					AAbb	0.188		
					AaBB	0.063		
					AaBb	0.250		
					Aabb	0.188		
BC-S	AaBb	0.50	aabb	1.0	AaBb	0.125	0.015	0.000
	Aabb	0.50			Aabb	0.375		
					aaBb	0.125		
					aabb	0.375		
RES[x]	AABb	1.00			AABB	0.250	0.820	0.750
					AABb	0.500		(D21)[w]
					AAbb	0.250		

z Observed (OBS) is the observed frequency of resistant seedlings after 28 days of screening.

y Expected (EXP) frequency of resistance = freq of resistant genotypes (underlined) X (1 - frequency of lethals(11).

x Resistant parent line 6701-1.

w Day 21 data used because day 28 data not available.

- FREQ = frequency, BC-S AND BC-R = backcross susceptible and resistant.
- Resistant genotypes and their frequencies are underlined.
- Parental genotypes: 1849 = aabb, 6701-1 = AABb.

Table 6. Expected Genotype Frequencies of Parents, F1, F2 and Backcross Families for Cross 1849(P1) x 6701-1(P2). Model III. Both A and B Genes are Partially Dominant and Resistant Parent 6701-1 is Heterozygous for Lethal Gene l.

TYPE	P1	FREQ	P2	FREQ	CROSS	FREQ	FREQENCY OBS [z]	FREQENCY EXP [y]
F1	aabbLL	1.00	AABbL1	1.0	AaBbLL	0.330	0.094	0.000
					AaBbL1	0.170		
					AabbLL	0.330		
					AabbL1	0.170		
F2	AaBbLL	0.33			AABB--	0.033	0.151	0.143
	AaBbL1	0.17			AABb--	0.063		
	AabbLL	0.33			AAbb--	0.156		
	AabbL1	0.17			AaBB--	0.063		
					AaBb--	0.125		
					Aabb--	0.313		
					aaBB--	0.031		
					aaBb--	0.063		
					aabb--	0.156		
					(----11	0.083)		
BC-R	AaBbLL	0.33	AABbL1	1.0	AABB--	0.063	0.396	0.354
	AaBbL1	0.17			AABb--	0.250		
	AabbLL	0.33			AAbb--	0.188		
	AabbL1	0.17			AaBB--	0.063		
					AaBb--	0.250		
					Aabb--	0.188		
					(----11	0.055)		
BC-S	AaBbLL	0.33	aabbLL	1.0	AaBbL-	0.125	0.015	0.000
	AaBbL1	0.17			AabbL-	0.375		
	AabbLL	0.33			aaBbL-	0.125		
	AabbL1	0.17			aabbL-	0.375		

z Observed (OBS) is the observed frequency of resistant seedlings after 28 days of screening.

y Expected (EXP) frequency of resistance = freq of resistant genotypes (underlined) X (1 - frequency of lethals(11).

▪ Resistant genotypes and their frequencies are underlined.

▪ Parental genotypes: 1849 = aabb, 6701-1 = AABb.

Table 7. Expected Genotype Frequencies of Parents, F1, F2 and Backcross Families for Cross **1849**(P1) x **6701-1**(P2). **Model IV**. Both A and B Genes are Partially Dominant and Both Resistant Parent (6701-1) and Susceptible Parent (1849) are Heterozygous for Lethal Gene.

TYPE	P1	FREQ	P2	FREQ	CROSS	FREQ	FREQENCY OBS [z]	FREQENCY EXP [y]
F1	aabbL1	1.00	AABbL1	1.0	AaBbL-	0.500	0.094	0.000
					AabbL-	0.500		
					(----11	0.25)		
F2	AaBbL-	0.50			AABB--	0.033	0.151	0.137
	AabbL-	0.50			AABb--	0.063		
					AAbb--	0.156		
					AaBB--	0.063		
					AaBb--	0.125		
					Aabb--	0.313		
					aaBB--	0.031		
					aaBb--	0.063		
					aabb--	0.156		
					(----11	0.125)		
BC-R	AaBbL-	0.50	AABbL1	1.0	AABB--	0.063	0.396	0.328
	AabbL-	0.50			AABb--	0.250		
					AAbb--	0.188		
					AaBB--	0.063		
					AaBb--	0.250		
					Aabb--	0.188		
					(----11	0.125)		
BC-S	AaBbL-	0.50	aabbL1	1.0	AaBbL-	0.125	0.015	0.000
	AabbL-	0.50			AabbL-	0.375		
					aaBbL-	0.125		
					aabbL-	0.375		
					(----11	0.125)		

z Observed (OBS) is the observed frequency of resistant seedlings after 28 days of screening.

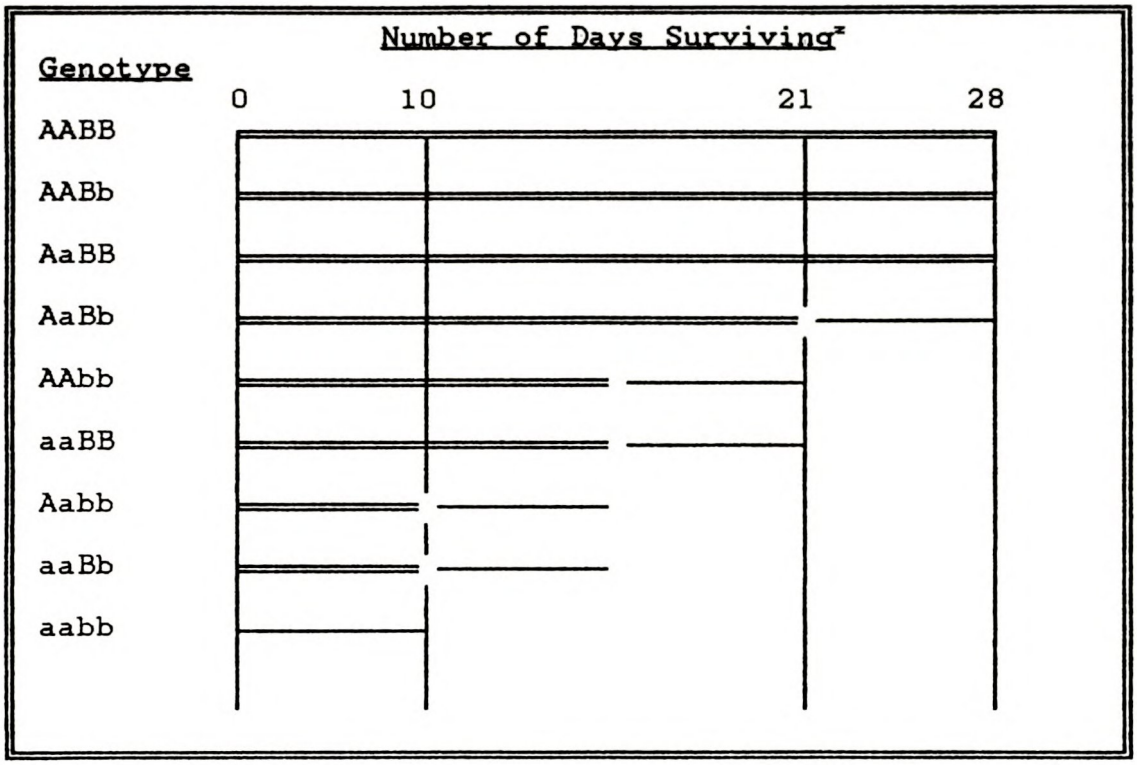
y Expected (EXP) frequency of resistance = freq of resistant genotypes (underlined) X (1 - frequency of lethals(11).

• Parental genotypes: 1849 = aabb, 6701-1 = AABb.

By matching the expected genotype frequencies for the various families with the observed frequencies of resistance on days 10, 21 and 28, the length of time a plant with a specific genotype survived during screening was estimated (Figure 3 for models I, III and IV and Figure 4 for model II). This time probably varied with differences in environmental conditions and also with the genetic background of the plant.

The existence of a post-emergence lethal gene was indicated by the lower than expected survival rates in the uninoculated control plants. It is hypothesized that this post-emergence recessive lethal gene resulted in the death of seedlings after emergence when in the ll condition. The survival rates of uninoculated selfed progeny from line 6701-1 (80%) and 1849 (83%) were close to that expected from plants contained a heterozygous lethal gene (Ll) segregating 3:1 (75%) for normal and lethal phenotypes. This lethality was observed in uninoculated selfed plants but, not in the crosses (F1, F2 and BC families) where the average survival rate in the control was 97.7%. Therefore, it is likely that the two parents contained different lethal genes which remained concealed in a heterozygous state in the crosses and only segregated homozygous recessive in progeny of selfed plants.

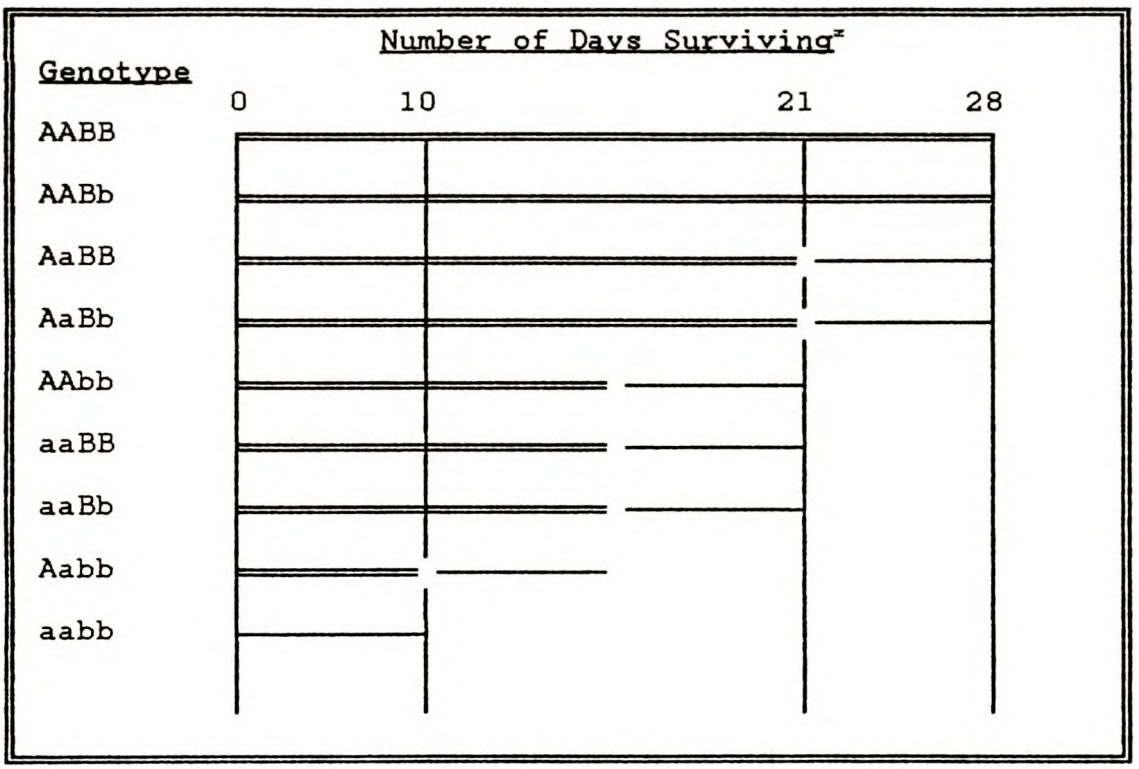
Figure 3. The Effect of Genotype on Resistance to Fusarium oxysporum f. sp. cepae. Model I. Both A and B Genes are Partially Dominant.



- ═══ healthy
- ——— diseased
- No line = death

z Number of days seedlings survive in F. o. cepae inoculated sand from emergence to death.

Figure 4. The Effect of Genotype on Resistance to Fusarium oxysporum f. sp. cepae. Model II. A Gene is Partially Dominant and B is Dominant.



- ═══ healthy
- ——— diseased
- No line = death

z Number of days seedlings survive in F. o. cepae inoculated sand from emergence to death.

CHI-SQUARE TESTS FOR F1, F2 AND BACKCROSS FAMILIES

The chi-square values for F1, F2, BC and SUS selfed families for all models (Table 8) along with a more complete table for Model I (Table 9) are given. The model that generated the best fit to the observed data was model I (both A and B gene partially dominant and no lethal genes). The chi-square values in all cases were not significant. This supports the hypothesis of two genes governing resistance with gene action being partially dominant at both loci.

Table 8. Chi-Square Values of Genetic Models for Resistance to Fusarium oxysporum f. sp. cepae.

Family	Model I	Model II	Model lII	Model IV
F1	0.38	0.38	0.38	0.38
F2	0.03	12.20*	0.12	0.52
BC-R	0.40	7.65*	1.79	5.08*
BC-S	0.03	0.03	0.03	0.03
SUS	0.00	0.00	0.00	0.00

- Model I : both A and B genes incompletely dominant.
- Model II : A gene incompletely dominant and B gene dominant.
- Model III : same as model I. with lethal gene in resistant line.
- Model IV : same as model I. with lethal genes in both parents.

* $X^2 > 3.841$ is significant at the 5% level (df=1).

Table 9. Chi-Square Test For F1, F2, Backcross Resistant and Susceptible Progenies Resulting from Cross 1849 x 6701-1[z]. Model I. Both A and B Genes Partially Dominant.

PARENTS	CROSSES	PROB RES.	OBSERVED SUS	OBSERVED RES	SUM	DF	X^2	PROB
SUS	SUS-20x	0.000	39	0	39	1	0.00	1.00
PARENT	SUS-21x		32	0	32	1	0.00	1.00
(selfed)	SUS-24x		29	0	29	1	0.00	1.00
	SUS-31x		51	0	51	1	0.00	1.00
	SUS-42x		33	0	33	1	0.00	1.00
	SUS-46x		21	0	21	1	0.00	1.00
	SUS-48x		18	0	18	1	0.00	1.00
	TOTAL		223	0	223			
	EXPECTED		223	0	223	1	0.00	1.00
RES		0.750						
PARENT	TOTAL		17	59	76			
(6701-1)	EXPECTED		19	57	76	1	0.16	0.70
F1	RES-1-55xSUS-27	0.000	16	3	19	1	0.33	0.60
	RES-1-55xSUS-27		15	0	15	1	0.00	1.00
	RES-1-55xSUS-27		12	2	14	1	0.16	0.70
	TOTAL		43	5	48			
	EXPECTED		48	0	48	1	0.38	0.60
F2	F1-4x	0.156	62	4	66	1	3.87	0.05
	F1-8x		48	15	63	1	2.63	0.10
	F1-9x		60	8	68	1	0.50	0.50
	F1-10x		57	15	72	1	1.13	0.30
	F1-14x		54	8	62	1	0.17	0.70
	TOTAL		281	50	331			
	EXPECTED		279	52	331	1	0.03	0.90
BC-RES	F1-1xRES-1-54	0.375	43	28	71	1	0.05	0.85
	F1-7xRES-1-36		46	27	73	1	0.01	0.95
	F1-29xRES-1-27		36	37	73	1	4.87	0.03
	F1-30xRES-1-26		29	9	38	1	2.53	0.10
	TOTAL		154	101	255			
	EXPECTED		159	96	255	1	0.40	0.50
BC-SUS	F1-22xSUS-42	0.000	40	0	40	1	0.00	1.00
	F1-33xSUS-46		67	0	67	1	0.00	1.00
	F1-34xSUS-43		21	0	21	1	0.00	1.00
	F1-37xSUS-48		25	0	25	1	0.00	1.00
	F1-41xSUS-10		46	3	49	1	0.13	0.75
	TOTAL		199	3	202			
	EXPECTED		202	0	202	1	0.03	0.90

z Proposed genotype = aabb x AABb.

* X^2 > 3.841 is significant at the 5% level (df=1). The chi-square values in the F2 and BC-RES families

increased as the number of parents containing lethal genes increased from 0 to 2 (in models I, III, and IV) resulting in a progressively poorer fit (Table 8). This indicates that the lethal genes were not expressed and, therefore, did not effect the survival rates. This supports the earlier statement that the lethal genes in the two parents were different genes.

Other models of inheritance were tested; single gene dominant, single gene recessive, two genes completely dominant and three genes completely dominant resulted in a poor fit between observed and expected values (Table 10).

Table 10. Expected Frequency of Resistant Phenotypes for Genetic Models of Complete Dominace and/or Recessiveness.

FAMILY	OBS FREQ*	Expected Frequency of Resistance				
		1 DOM	1 REC	2 DOM	1 DOM 1 REC	3 DOM
RES	0.830	0.750	100.0	0.750	0.750	0.750
F1	0.094	0.500*	0.000	0.500*	0.000	0.500*
F2	0.151	0.375*	0.250*	0.282*	0.094*	0.220*
BC-R	0.396	0.625*	0.500*	0.626*	0.313*	0.625*
BC-S	0.015	0.250*	0.000	0.125*	0.000	0.025*

* Observed frequency of resistant plants after 28 days of screening for resistance to F. o. cepae.

• Parental genotypes for models: monogenic dominace (1 DOM) = Aa x aa, monogenic recessive (1 REC) = aa x AA, 2 genes dominant = AABb x aabb, 1 gene dominant + 1 gene recessive = aaBb x AAbb, and 3 genes dominant = AABBCc x aabbbcc.

* An asterisk indicates that the expected frequency of resistant phenotypes predicted by a given model was significantly different than the observed frequency of resistance (by chi-square analysis).

Heterogeneity tests supported the hypothesis that parental line 6701-1 was heterozygous for resistance genes and line 1849 homozygous susceptible. Heterogeneity chi-square values were significant for F2 and backcross resistant families.

EFFECT OF INBREEDING ON RESISTANCE TO *FUSARIUM OXYSPORUM* F. SP. *CEPAE*

A cycle of screening and selfing of line 6701 was repeated for three generations. Those plants showing the highest resistance were selected as parents for the next cycle.

The level of resistance to *F. o. cepae* in selfed progeny decreased dramatically over the three generations from that of the original parent line. Analysis of variance test (Table 11) showed a significant difference between the percent resistance in the progeny of the selfed families. The greatest reduction occurred in the 1st(S_1) and 2nd(S_2) generations of selfing (Table 12) The difference between the 2nd (S_2) and 3rd (S_3) generations was not significantly different at the α=0.5 level as determined by LSD test. This loss of resistance, in part, was probably due to expression of recessive lethal genes in the homozygous state.

The trend of decreasing resistance in selfed progeny is still apparent after separating the effect of any post-emergence lethal gene (Table 12). The values given have been corrected to reflect a "true" (effect of lethal genes removed) level of resistance. Selfing of line 6701 for

Table 11. Analysis of Variance of S_0, S_1, S_2, and S_3 Generations.

	DF	Sum of Squares	Error Mean Square	F-value	Prob
Between	4	13273.16	3318.29	25.10**	.000
Within	84	11103.58	132.19		
Total	88	24376.74			

** Highly significant (α=0.01).

Table 12. The Effect of Selfing on the Level of Resistance on Resistant Line 6701.

	S_0	S_1	$S_1 x S_1$	S_2	S_3
No. plants selfed	--	29	5	32	19
No. seeds screened	136	1088	72	932	395
No. plants selected for next generation	--	1	--	6	--
% surviving[z] in control	97	91	100	80	89
% resistance D 21[x]	74.7	39.5	30.4	27.5	24.4
% resistance D 28[x]	53.0a	17.8b	6.3c	7.1c	6.1c

z % of emerging seedlings that survive for 28 days in non-inoculated conditions.

x (% resistance) x (1 + (1 - frequency of survival in control)).

▪ a,b, and c are significantly different (LSD α=0.5).

three generations resulted in a 89% decrease in the number of plants resistant to F. o. cepae. Since the reduction in survival rate due to lethal genes was removed this decrease in resistance was attributed solely to the effect of inbreeding.

S_1 plants were sib mated to see if this would restore some resistance that might have been lost due to other effects of inbreeding depression besides recessive lethals. The mean % resistance value was not significantly different between S_1 x S_1 progeny and S_2 progeny (Table 12). This lack of effect from crossing sister plants to restore vigor and/or resistance was also observed in the F2 generations (Table 3) where F1 sib families were mated and compared to F1 selfed families. This effect would be expected if a high degree of homozygosity between sister plants existed.

The possibility of a lethal gene linked to one or more of the resistance genes was considered, but this would have resulted in a much lower level of apparent resistances in BC-RES families. Also an increase in the level of lethals in the control in BC-RES families was not observed (96 to 98% of the BC-RES plants survived in the control).

SUMMARY AND RECOMMENDATIONS

SUMMARY OF INHERITANCE OF RESISTANCE TO *FUSARIUM OXYSPORUM* F. SP. *CEPAE*

Two genes, *Foc1* (*A*) and *Foc2* (*B*), are proposed to govern resistance to *F. o. cepae*. The *A* and *B* genes are partially dominant resulting in the homozygous genotypes *AA* and *BB* being more resistant than the heterozygous genotypes *Aa* and *Bb*, respectively. There appears to be an additive effect between loci with the order of resistance as follows: *AABB* > *AABb* or *AaBB* > *AaBb* > *AAbb* or *aaBB* > *Aabb* or *aaBb* > *aabb*.

Resistance is not epistatic; at least one resistant allele (*A* and *B*) at both loci must be present for a resistant phenotype to occur. Plants with genotypes AAbb and aaBB are susceptible.

Three genotypes result in a resistant phenotype: *AABB*, *AABb*, and *AaBB* (plants survived free of symptoms of *F. o. cepae* for 28 day in screening trials). Plants heterozygous at both loci (*AaBb*) appear to have some resistance, surviving around 21 days in screening trials, thereafter, developing symptoms of *F. o. cepae*.

The percent of plants resistant from cross 1849 x 6701-1 and reciprocal cross 6701-1 x 1849 was not

significantly different. Therefore, it was concluded that resistance to F. o. cepae was not maternally inherited.

The existence of recessive lethal genes in both of the parents was indicated by the lower than expected survival rates in the uninoculated control plants. Survival rates in the uninoculated selfed progeny from line 6701-1 and 1849 were close to a 3:1 (normal:lethal) segregation ratio, or 75% survival rate, expected from segregating plants containing a heterozygous recessive lethal gene (Ll). This lethality occurred in selfed plants but, not in crosses where the survival rate in the control was approximately 98%. It was concluded that the two parents contained different lethal genes and, therefore, they did not effect the survival rates of the F1, F2 and BC families screened for resistance to F. o. cepae.

RECOMMENDATIONS FOR BREEDING ONIONS FOR INCREASED RESISTANCE TO FUSARIUM OXYSPORUM F. SP. CEPAE

Assuming the proposed genetic model for resistance to F. o. cepae is true, it would be beneficial to increase the screening time from 21 days to 28 (or more) to eliminate the genotypes heterozygous for resistant genes. Genotypes AABB, AABb, AaBB, AaBb, AAbb and aaBB survive for 21 days in screening trials (see Figure 3.) while only genotypes AABB, AABb and AaBB survive for 28 days. Therefore, increasing the screening time would eliminate those plants with genotypes AaBb and AAbb, decreasing the frequency of recessive susceptible genes in the population. Considering

the partial dominant gene action proposed it is likely that genotypes AABb and AaBB might also be reduced or eliminated if screening time were extended.

Another effective way to increase selection pressure for resistance genes is to increase inoculation concentration (3,58,68,70). A combination of increased screening time and inoculum concentrations is perhaps an even better solution. Optimal screening time and inoculum concentrations for selection of resistance genes could be determined experimentally.

The effect of inbreeding on level of resistance should be considered when selecting for resistance to F. o. cepae in onions. Development of inbred lines requires a certain amount of selfing to increase homozygosity and homogeneity in the population. The effect of inbreeding can confound attempts to select for increased resistance. Some inbreeding depression, however, can be tolerated and evidence from the selfing study on line 6701 indicates that the decline in resistance will taper off after the 2nd generation of selfing with selection. Massing of selected plants and cross pollination will usually restore vigor (45,46); whether the level of resistance will also be restored will require further study. The use of large populations (1000-2000 seeds) for screening should allow heavy selection while retaining enough surviving plants to recombine to restore heterozygosity and vigor.

The pattern of resistance observed in this study and the proposed genetic model suggest that a hybrid with a relatively high level of resistance to F. o. cepae can be produced by crossing a resistant parent with either another resistant or intermediate level resistant parent. Crossing a resistant parent with a susceptible parent should result in a hybrid with little or no resistance since the trait is not completely dominant.

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