



## Breeding and genetics of *Fusarium* basal rot resistance in onion

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### Summary

*Fusarium* basal plate rot (FBR), caused by *Fusarium oxysporum* f. sp. *cepae*, is an important soil-borne disease of onions worldwide. The causal organism infects the basal stem plate of the bulb and eventually kills the entire plant through degradation of the basal plate. *F. o. f. sp. cepae* infections in dormant bulbs during storage allow secondary infections to occur. The primary method of infection by *F. o. f. sp. cepae* is through direct penetration of the basal stem plate. Infection can also occur through wounded tissue particularly roots and basal portions of bulb scales. The most cost-effective methods of control are crop rotation and host plant resistance. Current research suggests that a single gene, two genes, or multiple genes govern resistance to FBR. Breeding programs have successfully used screening procedures to develop intermediate- and long-day, FBR-resistant cultivars.

### Introduction

Onions (*Allium cepa* L.) are an important crop in the United States and worldwide. In the United States during 1998, onions ranked second in yield per hectare and in total production, third in total value, and fourth in the number of hectares harvested of 25 principal vegetables grown for fresh market consumption (U.S. Dept. Agr., 1999). On a world-wide basis, onions ranked as one of the five most important fresh market vegetable crop for most production statistics (FAO, 1999). As with other vegetable crops, onions are susceptible to numerous foliar, bulb, and root fungal pathogens that reduce onion yield and quality. One of those diseases, *Fusarium* basal rot (FBR) [*Fusarium oxysporum* Schlechtend.: Fr. f. sp. *cepae* (H.N. Hans.) W.C. Snyder & H.N. Hans], is a root and bulb fungal disease of onions grown in temperate and subtropical regions (Brayford, 1996). In addition to onion, FBR affects other *Allium* species such as shallots (*A. cepa* L. var. *ascalonicum* Backer), Welsh onion (*A. fistulosum* L.), and chives (*A. schoenoprasum* L.) (Takakuwa et al., 1977; Stevenson & Heimann, 1981; Kodama, 1983; Havey, 1995). *F. o. f. sp. cepae* invades the plant through roots and the basal stem plate via the

soil. The disease progresses from slight discoloration of the basal plate to total necrosis, death of older leaves and the entire plant, and eventual rot of the internal bulb scales. In New Mexico, USA, FBR is the second most important soil-borne disease in terms of losses (N. Goldberg, pers. comm., 1998). Losses of 25% to 35% to FBR have been reported for the Midwestern USA. (Lacy & Roberts, 1982). The objective of this paper is to review the current knowledge of the symptoms, epidemiology, and control methods for FBR, genetics of host plant resistance, and breeding research used to develop FBR-resistant cultivars.

### Pathogen

*F. o. f. sp. cepae* is one of over a hundred formae speciales of *F. oxysporum* (Burgess et al., 1994). The pathogen produces chlamydospores, macroconidia, and microconidia. Chlamydospores are round, thick-walled, and formed abundantly in soil (Burgess et al., 1994). They are the primary source of inoculum under field conditions. Macroconidia are short to medium in length, falcate, thin-walled, slightly tapered at the ends, and usually 3-septate (Burgess et al., 1994). Mi-

croconidia are usually non-septate, oval to reniform in shape, and abundant in culture (Burgess et al., 1994). *F. o. f. sp. cepae* isolates from different fields and different countries differ in their virulence (Villeveille, 1996). Isolates obtained from fields in France were more virulent than those obtained from Wisconsin, USA (Villeveille, 1996). In addition, isolates from different fields in France differed in their virulence (Villeveille, 1996). Although isolates differ in their virulence, separate races have not been identified (Havey, 1995).

### Symptoms

The visual symptoms of FBR can be observed on plant leaves, roots, basal stem plate, and bulb scales of small seedlings, mature plants, and dormant bulbs. Symptoms on leaves of small seedlings are difficult to observe. If the environmental conditions are conducive to pathogen growth, *F. o. f. sp. cepae* will kill young seedlings before visual symptoms are observed (Tahvonen, 1981). In addition, FBR can cause delayed seedling emergence (Davis & Reddy, 1932), seedling damping off (Srivastava & Qadri, 1984), and stunted growth of seedlings (Entwistle, 1990). On mature plants, the first aboveground symptoms of FBR would be chlorosis of all leaves. This chlorosis leads to tip necrosis and eventually progresses to entire leaf necrosis and plant death (Wall et al., 1993; Havey, 1995; Brayford, 1996). The infection within the basal plate also causes root death and root abscission. A noticeable symptom of FBR is the separation of roots from the bulb at the stem plate during uprooting. Within the basal plate, *F. o. f. sp. cepae* causes a brown discoloration of the basal plate tissue. Once the entire basal plate is destroyed, the stem plate can be easily removed from the rest of the bulb. In severe cases, *F. o. f. sp. cepae* infects the basal portions of the bulb scales, and white mycelium can be observed on the basal portions of exterior bulb scales. In addition, FBR provides a mode of entry for secondary pathogens to infect the bulb scales.

### Epidemiology

Fusarium basal rot is a worldwide disease that is commonly found wherever onions are grown (Walker & Tims, 1924; Link & Bailey, 1926; Palo, 1928; Davis & Henderson, 1937; Shalaby & Struckmeyer,

1966; Holz & Knox-Davies, 1974; Sokhi et al., 1974; Ashour et al., 1980). *F. o. f. sp. cepae* can be spread by infected debris, infected soil (Abawi & Lorbeer, 1971a), irrigation water, farm equipment (Everts et al., 1985), onion seed (Kodama, 1983; Koycu & Ozer, 1997), and onion transplants (Kodama, 1983; Everts et al., 1985). The pathogen can also persist in the soil for several years due to its production of chlamydospores that are long-term survival structures (Brayford, 1996). Other *Allium* species such as chives, garlic (*A. sativum* L.), and shallot are mentioned as suffering losses from FBR (Havey, 1995). Welsh onion has been reported both as susceptible (Takakuwa et al., 1977; Kodama, 1983; Shinmura et al., 1998) and resistant (Havey, 1995) to FBR. *F. o. f. sp. cepae* also has the ability to infect weed species such as oxalis (*Oxalis corniculata* L.) (Abawi & Lorbeer, 1972), and pigweed (*Chenopodium album* L.) (Tsutsui, 1991) as well as many crop and vegetable species such as maize (*Zea mays* L.), wheat (*Triticum aestivum* L.), rice (*Oryza sativa* L.), soybean (*Glycine max* Merr.), pea (*Pisum sativum* L.), cucumber (*Cucumis sativus* L.), and squash (*Cucurbita pepo* L.) (Tsutsui, 1991).

Losses to FBR can occur in the field and/or during storage. Losses in the field to FBR are reported to range from 3% (N. Goldberg, pers. comm., 1998) to 35% (Lacy & Roberts, 1982) depending upon the time of year, environmental conditions, cultivar, and level of inoculum. In southern New Mexico, USA, the incidence of FBR for fall-planted cultivars ranged from 0.6% to 40.3% while the incidence for spring-planted cultivars ranged from 2.9% to 29.2% (Table 1) (Cramer et al., 2000). In Bangalore, India, the incidence of FBR ranged from 20% to 80% of bulbs infected using a bulb infection method (Somkuwar et al., 1996). For onion cultivars grown in Brazil, the incidence of FBR ranged from 12% to 75% of bulbs inoculated with *F. o. f. sp. cepae* as seedlings (Stadnick & Dhingra, 1996). FBR reduces the number of marketable bulbs in a field as well as causes a reduction in the weight of bulbs at harvest (Lacy & Roberts, 1982). Most of the damage from FBR is observed during storage (Stadnik & Dhingra, 1996). Losses to FBR during storage were greater than losses observed in the field. Minor infections of FBR in the basal plate can go undetected during harvest (Brayford, 1996). Pathogen growth continues during storage until the entire bulb becomes unmarketable. FBR provides a mode of entry for secondary pathogens that may spread to adjacent bulbs. In the field, seedcorn maggot, *Delia platura* (Meigen) is attracted to infected bulbs (Havey, 1995).

*F. o. f. sp. cepae* can infect onion bulbs in many different ways. The main method of infection is by direct penetration of the basal plate (Havey, 1995). In addition, *F. o. f. sp. cepae* can invade through roots or basal portions of older leaves (Shalaby & Struckmeyer, 1966). Infection is facilitated by wounding of the plant tissue caused by other organisms or mechanical damage (Brayford, 1996). For example, pink root disease, caused by *Phoma terrestris* E.M. Hans. (syn. *Pyrenochaeta terrestris* (E.M. Hans.) Gorenz, J.C. Walker, & R.H. Larson), can provide a mode of entry for *F. o. f. sp. cepae* into plant roots.

The mode of action of *F. o. f. sp. cepae* is enzymatic. The fungus releases pectic enzymes including exo-polygalacturonase (exo-PG) and endo-pectin-trans-eliminase (endo-PTE) that work to break down pectin in the cell wall of the onion (Holz & Knox-Davies, 1985a). Exo-PG activity is quite high during the initial infection of the bulb and consequently involved in maceration of the basal plate tissue during later infection (Holz & Knox-Davies, 1985a). Endo-PTE has been retrieved from infected basal plate tissue two weeks after initial infection of the onion basal plate and during the period of FBR decay (Holz & Knox-Davies, 1985a). This decay results in the release of apoplast sugar from the bulb tissue that feeds the fungus and ensures its continued growth and reproduction (Holz & Knox-Davies, 1986).

The optimum soil temperature for development is between 28C and 32C; however, the disease can occur at a soil temperature range of 15C to 32C (Walker & Tims, 1924; Kehr et al., 1962; Abawi & Lorbeer, 1972; Kodama, 1983). Soil temperatures of 12C or less resulted in little or no disease development (Walker & Tims, 1924; Kehr et al., 1962; Abawi & Lorbeer, 1972; Kodama, 1983). Under growing conditions in southern New Mexico, USA, early-maturing, overwintered, fall-planted cultivars exhibit a lower incidence of FBR than later maturing cultivars partially due to low soil temperatures that are nonconducive for disease development (Table 1) (Cramer et al., 2000). The optimum pH for growth is 6.6 but growth can occur at a pH range of 2.2 to 8.4 (Walker & Tims, 1924).

## Control

*F. o. f. sp. cepae* can be controlled through host plant resistance, crop rotation, solarization, biological control, and fungicide application. Numerous

Table 1. Percentage of Fusarium basal rot incidence from onion variety field trials at the Fabian Garcia Agricultural Science Center, Las Cruces, NM, USA in 1998–1999<sup>z</sup>

Entry <sup>z</sup>	Seed source	Maturity date	FBR incidence (%)
<i>Seeded 15 Sept. 1998</i>			
Buffalo	Shamrock	23 May	30.8
Cardinal	Shamrock	1 June	21.5
Caribou	Shamrock	2 June	31.0
Chula Vista	Petoseed	1 June	1.3
Daybreak	Shamrock	25 May	7.4
Don Victor	Rio Colorado	22 May	5.4
Excalibur	Rio Colorado	24 May	2.4
Ibex	Shamrock	24 May	1.6
Lexus	Petoseed	2 June	5.2
Linda Vista	Petoseed	1 June	12.9
NuMex BR1	NMSU	25 May	0.6
NuMex Crispy	NMSU	4 June	15.0
NuMex Dulce	NMSU	10 June	21.0
NuMex Luna	NMSU	25 June	40.3
NuMex Mesa	NMSU	25 May	2.1
NuMex Starlite	NMSU	3 June	16.8
NuMex Sunlite	NMSU	31 May	4.5
NuMex Sweetpak	NMSU	23 May	5.1
NuMex Vado	NMSU	8 June	17.3
Texas Early White	Petoseed	30 May	4.2
<i>Seeded 28 Jan. 1999</i>			
Alabaster	Sunseeds	21 July	14.5
Aspen	Petoseed	26 July	17.7
Candy	Petoseed	2 Aug.	17.1
Cimarron	Sunseeds	16 July	5.4
Dawn	Shamrock	28 July	6.9
Frosty	Duane Palmer	14 Aug.	13.3
Impala	Shamrock	1 Aug.	9.2
La Nina	Rio Colorado	25 July	5.5
Mesquite	Duane Palmer	16 Aug.	11.6
Navigator	Rio Colorado	23 July	9.4
NuMex Bolo	NMSU	16 July	6.2
NuMex Casper	NMSU	27 July	8.9
NuMex Centric	NMSU	2 Aug.	8.9
NuMex Jose Fernandez	NMSU	21 July	2.9
Rio Gigante	Rio Colorado	13 July	10.4
Riviera	Asgrow	2 Aug.	8.2
Rumba	Sunseeds	4 Aug.	29.2
Spano	Sunseeds	24 July	19.1
Tara	Rio Colorado	27 July	19.4
Utopia	Asgrow	27 July	6.6

<sup>z</sup> Adapted from Cramer et al. (2000).

intermediate- and long-day onion hybrids possess moderate levels of resistance to FBR. Although this resistance is not absolute, losses to FBR can be significantly reduced through the use of FBR-resistant cultivars. Currently, resistant cultivars are available for intermediate and long-day onions but not available for short-day onions. Under field conditions in southern New Mexico, USA, two fall-planted cultivars, 'NuMex Dulce' and 'NuMex Vado' exhibited moderate resistance to FBR (Table 1) (Cramer, 2000). Among spring-planted cultivars, 'Dawn', 'Impala', 'La Nina', 'Navigator', 'NuMex Casper', 'NuMex Centric', 'Riviera', and 'Utopia' showed high levels of FBR resistance while 'Aspen' and 'Frosty' showed moderate levels of resistance when grown in fields infested with *F. o. f. sp. cepae* (Table 1). Studies conducted at the Indian Institute of Horticulture Research determined that three onion lines, IHR-141, IHR-506, and Sel 13-1-1 were resistant to FBR in both laboratory and field screenings replicated over years (Ganeshan et al., 1998). In addition, Indian breeding lines, 'Hybrid-1', 'IHR Yellow', and 'Sel. 29' were resistant to FBR from seed and bulb infection (Somkuwar et al., 1996). These three lines were considered resistant based upon 20% of total bulbs infected with FBR. In Brazil, cultivars, 'Bola Precoce', 'Roxa do Barreiro', 'Cebola de Verao', 'Crioula', 'Monte Alegre', 'Pera IPA 3', 'Roxa IPA 3', and 'Texas Grano 502' were considered resistant to FBR at harvest after inoculation of transplants (Stadnik & Dhingra, 1996). After bulbs were stored for 90 days, only 'Cebola de Verao' was considered resistant to FBR. Latent infections of bulbs in the other cultivars reduced bulb yield during storage.

In addition to host plant resistance, crop rotation with a crop like maize or spring wheat will reduce soil inoculum levels and onion bulb loss to FBR in the following year (Higashida et al., 1982). A crop rotation of four years with a nonsusceptible host is recommended before planting another onion crop in that field (Entwistle, 1990; Havey, 1995). Field solarization can decrease the incidence of Fusarium-caused diseases (Katan et al., 1980). Biological control using fungal and bacterial antagonists has been suggested as a possible control method for FBR. Under in vitro conditions, fungal antagonists, *Trichoderma viride*, *T. harzianum*, *T. hamatum*, *T. koningii*, and *T. pseudokoningii*, and bacterial antagonists, *Pseudomonas fluorescens* and *Bacillus subtilis* were effective against *F. o. f. sp. cepae* (Rajendran & Ranganathan, 1996). A combination of *T. viride* and *P. fluorescens*

were most effective for reducing FBR incidence under pot and field conditions.

Numerous chemical methods exist for the control of FBR. Soil fumigation with methyl bromide or metam sodium has proven effective for control of FBR (Jaworski et al., 1978). The cost of soil fumigation may be prohibitive for some growers but fumigation can provide other benefits like pink root and weed control. Sets and transplants can be treated with a fungicide such as benomyl and losses to FBR can be reduced (Koriem et al., 1991; Koycu & Ozer, 1997; Ozer & Koycu, 1998). In addition, seeds have been treated with benomyl (Barnoczkin-Stoilova, 1988; Abd-El-Razik et al., 1990; Koriem et al., 1991; Koycu & Ozer, 1997; Ozer & Koycu, 1998), carbedazim (Barnoczkin-Stoilova, 1988; Abd-El-Razik et al., 1990), carboxin hydroxyquinoline (Barnoczkin-Stoilova, 1988; Abd-El-Razik et al., 1990), iprodione (Barnoczkin-Stoilova, 1988; Abd-El-Razik et al., 1990), menab (Roberti et al., 1989), methoxyethyl mercury chloride (Barnoczkin-Stoilova, 1988; Abd-El-Razik et al., 1990), prochloraz (Ozer & Koycu, 1998), tebuconazole (Ozer & Koycu, 1998), and thiram (Gupta et al., 1987, Ozer & Koycu, 1998), with reduction in FBR infection. When seeds were infested with *F. oxysporum*, applications of thiram (1.35 and 4.05 g ai/kg seed), prochloraz (0.45 cc ai/kg seed) and a benomyl+thiram (1.50 and 0.45 g ai/kg seed) mixture stimulated the rate of onion seed germination (Ozer & Koycu, 1998). In addition, applications of benomyl+thiram (1.50 and 0.45 g ai/kg seed) inhibited the growth of *F. oxysporum*, and reduced the post-emergence damping-off of onion seedlings. Prochloraz (1.35 cc ai/kg seed) was the most effective seed treatment for controlling *F. oxysporum*-induced damping-off in infested soil.

### Genetics of host plant resistance

Several studies have been conducted to determine the genetics involved in onion resistance to FBR (Table 2). Bacher (1989) and Bacher and co-workers (1989), in a cross between a FBR-resistant and a FBR-susceptible long-day inbred, reported that resistance to *F. o. f. sp. cepae* was controlled by two partially dominant genes. They designated these genes as *Foc1* and *Foc2*. They hypothesized that the interaction between loci appeared to be additive. Dominant alleles must be present at both loci in order for plants to be resistant to FBR. Plants that were heterozygous at both loci

Table 2. Proposed genetic control of FBR resistance in onion

Mode of inheritance	Genetic material	Reference
Single dominant gene	Long-day inbreds	Tsutsui (1991)
Cytoplasmic genes	Long-day inbreds	Krueger (1986), Krueger & Gabelman (1989), Tsutsui (1991)
Two partially dominant genes	Long-day inbreds	Bacher (1989), Bacher et al. (1989)
Multiple genes	Short-day open-pollinated population	Villanueva-Mosqueda (1996)
	Long-day populations	Kehr et al. (1962); Lorbeer & Stone (1965); Holz & Knox-Davies (1974)

were intermediate in their resistance at 21 days after planting. If these plants were rated at 28 days, they would be rated as susceptible. After three cycles of self-pollination of the resistant parent, the level of resistance was significantly reduced as compared to the original resistant parent. This probably resulted from lack of vigor in the inbred lines. Sib-mating between plants within this line could not restore the level of resistance. They proposed that lethal genes were not involved in the susceptibility of these plants to FBR.

Also in a cross between five long-day inbreds, Tsutsui (1991) reported that resistance to *F. o. f. sp. cepae* was controlled by a single dominant gene. However, he proposed some variable expression in this gene and the possibility of additional genes for FBR resistance. Tsutsui (1991) also observed reciprocal differences in resistance between F<sub>1</sub> families. Krueger (1986) and Krueger and Gabelman (1989) observed reciprocal differences in FBR resistance when W404, a resistant inbred line, was used as a parent in hybrid crosses. These reciprocal differences suggest that either nuclear or cytoplasmic genes control resistance. Conversely, Bacher and co-workers (1989) did not observe reciprocal effects in the FBR resistance for their cross. Each of the studies involved a limited number of long-day inbreds and the results from those studies are only applicable to those particular inbreds.

In addition to single major gene inheritance, polygenic inheritance of FBR resistance has been suggested (Kehr et al., 1962; Lorbeer & Stone, 1965; Holz & Knox-Davies, 1974) but few studies have been conducted to determine this inheritance pattern for FBR resistance. For a short-day, open-pollinated onion population, narrow-sense heritability of FBR resistance was high (0.80) and highly correlated ( $r = 0.9$ ) with resistance to pink root disease (Villanueva-Mosqueda, 1996).

The mechanism of resistance to FBR is unknown. Both resistant and susceptible cultivars were similar anatomically and showed similar infection rates of the roots and basal plates by *F. o. f. sp. cepae* (Abawi & Lorbeer, 1971b; Tsutsui, 1991; Havey, 1995). In addition, fungal distribution and enzyme production during the early stages of infection were similar between susceptible and resistant cultivars (Holz & Knox-Davies, 1985a). However, fungal growth was slower in resistant than in susceptible cultivars (Abawi & Lorbeer, 1971b; Kodama, 1983). Low levels of pectic enzyme production were correlated with slow pathogen growth in bulbs (Holz & Knox-Davies, 1985a; 1985b). Cultivars also differed in their ability to restrict fungal growth to the stem plate and to prevent growth in the basal portions of the bulb scales. Resistant cultivars restricted pathogen growth to the stem plate for 9 months, while susceptible cultivars restricted growth for only 2 to 3 months (Holz & Knox-Davies, 1985a).

### Breeding for resistance

In order to develop lines that are resistant to FBR, numerous screening methods have been developed that involve field or greenhouse screening of seedlings, mature plants, or dormant bulbs (Retig et al., 1970; Holz & Knox-Davies, 1974; Bacher et al., 1989; Krueger et al., 1989; Tsutsui, 1991; Somkuwar et al., 1996; Stadnik & Dhingra, 1996; Ganeshan et al., 1998). Mature plants show the greatest resistance to FBR followed by seedlings and dormant bulbs (Holz & Knox-Davies, 1974). Some of this resistance may be attributed to plant vigor and the ability of the plant to withstand infection. Plant vigor would be the greatest for mature plants. In a study involving *F. o. f. sp. cepae* inoculation of 'Baia Periforme'

transplants of different ages, older transplants showed no signs of disease symptoms whereas the youngest transplants exhibited disease symptoms even though the fungus was isolated from inoculated transplants at each growth stage (Stadnik & Dhingra, 1997). Onions that are screened for *F. o. f. sp. cepae* resistance should be screened both during field growth and during the post harvest period. The seeds or bulbs are inoculated with *F. o. f. sp. cepae* that was grown in culture, and then the percent of germinated seeds or the percent of infected bulbs are recorded (Somkuwar et al., 1996). A positive correlation of results has been shown between bulbs screened in the greenhouse and bulbs screened in the field (Krueger et al., 1989). In some cases, the inoculated onions are grown until leaf drop, and plants are screened for infection at harvest (Retig et al., 1970). The harvested bulbs are then screened for up to forty days following the date of harvest (Retig et al., 1970). Screening methods and selection have been shown to improve the keeping time and slow the infection by *F. o. f. sp. cepae* in long day onion varieties (Retig et al., 1970).

Several factors, like inoculum concentration, media temperature, seed age, preseedling incubation treatment of seeds, and timing of rating influence the seedling screening procedure (Tsutsui, 1991). The ideal inoculum concentration for screening was  $1.0 \times 10^4$  spores per gram of sand (Tsutsui, 1991). A sand temperature of 27°C was determined to be most favorable for *F. o. f. sp. cepae* infection (Tsutsui, 1991). Seedling vigor confounded seedling screening results. Less vigorous or old seeds of resistant lines appeared susceptible in FBR screening tests (Tsutsui, 1991). Seeds should not be incubated prior to planting as this treatment made FBR-susceptible lines appear more resistant to FBR (Tsutsui, 1991). Bacher and co-workers (1989) suggested increasing the time for rating from 21 to 28 days after sowing. This way, plants that would be rated as intermediate in their resistance at 21 days would be rated as susceptible by 28 days.

The onion breeding program at the University of Wisconsin-Madison led by Dr Warren Gabelman was successful in developing a number of long-day male-sterile, maintainer, and pollinator lines that possessed moderate, high, and very high levels of resistance to FBR (Table 3) (Goldman, 1996). The resistance has been since incorporated into intermediate- and long-day commercial hybrids. However, FBR resistance is currently lacking in short-day onion cultivars.

Table 3. Onion breeding lines resistant to FBR released by W.H. Gabelman, Univ. of Wisc., Madison<sup>z</sup>

Breeding line	Level of resistance	Year of release
W202A, W202B	High	1967
W205A, W205B	Moderate	1967
W206C	Moderate	1967
W207C	Very high	1967
W404A, W404B	Moderate	1972
Fusario 12	High	1972
Fusario 24	High	1972
Fusario 245	High	1972
Fusario 142	High	1972
W419A, W419B	High	1983
W420A, W420B	High	1983
W417A, W417B	High	1990
W434A, W434B	Very high	1990
W435A, W435B	Very high	1990
W439A, W439B	Moderate	1990
W440A, W440B	Very high	1990
W441A, W441B	High	1990
W446A, W446B	Very high	1990
W447A, W447B	Very high	1990
W449C	High	1990
W457A, W457B	Moderate	1993
W458A, W458B	Moderate	1993
W459A, W459B	Moderate	1993
W460A, W460B	Very high	1993
W461A, W461B	Moderate	1993

<sup>z</sup> Adapted from Goldman (1996).

## Conclusion

Fusarium basal rot is a devastating soil-borne disease of onions that can affect seedlings, mature plants, and dormant bulbs in most onion-growing regions of the world. Disease resistant cultivars are the best method of control. FBR-resistant, intermediate- and long-day cultivars have been developed, while resistant short-day cultivars are lacking. Improvements in screening procedures and a further understanding of the genetics involved in resistance will aid in the development of FBR-resistant cultivars.

## References

- Abawi, G.S. & J.W. Lorbeer, 1971a. Pathological histology of four onion cultivars infected by *Fusarium oxysporum* f. sp. *cepae*. *Phytopathology* 61: 1164–1169.

- Abawi, G.S. & J.W. Lorbeer, 1971b. Reaction of selected onion varieties to infection by *Fusarium oxysporum* f.sp. *cepae*. Plant Dis Rep 55: 1000–1004.
- Abawi, G.S. & J.W. Lorbeer, 1972. Several aspects of the ecology and the pathology of *Fusarium oxysporum* f. sp. *cepae*. Phytopathology 62: 870–876.
- Abd-El-Razik, A.A., F.G. Fahmy, A.M. Amein & A.I. El-Amein, 1990. Role of onion seeds in transmission of damping of causal fungi and chemical control of the disease. Seed Path Microbiol 1993: 247.
- Ashour, W.A., I.S. Elewa, A.A. Ali & T. Dabash, 1980. The role of some systemic and nonsystemic fungicides and fertilization on the enzyme activity and the control of *Fusarium oxysporum* f. sp. *cepae*, the cause of basal rot disease in onion. Agr Res Rev 58: 145–161.
- Bacher, J.W., 1989. Inheritance of resistance to *Fusarium oxysporum* f. sp. *cepae* in cultivated onions. MS Thesis, Michigan State Univ., East Lansing.
- Bacher, J.W., S. Pan & L. Ewart, 1989. Inheritance of resistance to *Fusarium oxysporum* f.sp. *cepae* in cultivated onions. In: L. Jensen (Ed.), Proc. 1989 Natl. Onion Res. Conf. Boise, pp. 85–91.
- Barnoczkiné-Stoilova, E., 1988. Selection of effective seed dressing fungicides to control *Fusarium* spp. on onion seeds. Seed Path Microbiol 1990: 198.
- Brayford, D., 1996. *Fusarium oxysporum* f. sp. *cepae*. Mycopathologia 133: 39–40.
- Burgess, L.W., B.A. Summerell, S. Bullock, K.P. Gott & D. Backhouse, 1994. Laboratory Manual for Fusarium Research, 3rd Ed. Univ of Sydney, Sydney, Australia, 133 pp.
- Cramer, C.S., J.N. Corgan, J.L. Mendoza & M.M. Wall, 2000. 1998–1999 Onion variety trials at New Mexico State University. New Mexico Agr Exp Stn Res Rpt 739.
- Davis, G. & W.J. Henderson, 1937. The interaction of the pathogenicity of a *Phoma* and *Fusarium* on onions. Phytopathology 27: 763–772.
- Davis, G.N. & C.S. Reddy, 1932. A seedling blight stage of onion bulb rot. Phytopathology 22: 8.
- Entwistle, A.R., 1990. Root diseases. In: H.D. Rabinowitch & J.L. Brewster (Eds.), Onion and Allied Crops. Vol. II, pp. 103–154. CRC Press, Boca Raton, Fla.
- Everts, K.L., Schwartz, H.F., Epsky, N.D. & J.L. Capinera, 1985. Effects of maggots and wounding on occurrence of *Fusarium* basal rot of onions in Colorado. Plant Dis 69: 878–882.
- Food and Agricultural Organization, 1999. FAOSTAT agriculture data <http://apps.fao.org/lim500/nph=wrapp.pl?Production.Crops.Primary&Domain=SUA&servlet=1>
- Ganeshan, G., C.S. Pathak & B. Veere Gowda, 1998. Reaction of onion lines to basal rot disease caused by *Fusarium oxysporum* f. sp. *cepae*. P K V Res J 22: 53–54.
- Goldman, I.L., 1996. A list of germplasm releases from the University of Wisconsin onion breeding program, 1957–1993. HortScience 31: 878–879.
- Gupta, R.P., P.K. Srivastava & U.B. Pandey, 1987. Control of damping-off in kharif onion nursery. Rev. Plant Pathol 67: 5298.
- Havey, M.J., 1995. *Fusarium* basal plate rot. In: H.F. Schwartz & S.K. Mohan (Eds.), Compendium of Onion and Garlic Diseases, pp. 10–11. APS Press, St. Paul, Minn.
- Higashida, S., I. Oshaki & Y. Narita, 1982. Effects of crop rotation on onion yields and its microbial factors. Bull Hokkaido Pref Agr Expt Stn 48: 1–9.
- Holz, G. & P.S. Knox-Davies, 1974. Resistance of onion selections to *Fusarium oxysporum* f. sp. *cepae*. Phytophylactica 6: 153–156.
- Holz, G. & P.S. Knox-Davies, 1985a. Production of pectic enzymes by *Fusarium oxysporum* f. sp. *cepae* and its involvement in onion bulb rot. Phytopath Zeit 112: 69–80.
- Holz, G. & P.S. Knox-Davies, 1985b. Production of pectic enzymes by *Fusarium oxysporum* f. sp. *cepae*: induction by cell walls from different parts of onion bulbs at different growth stages. Phytopath Zeit 112: 81–92.
- Holz, G. & P.S. Knox-Davies, 1986. Relation between endo-pectin-trans-eliminase and apoplast-symplast sugars in *Fusarium* bulb rot of onions. Physiol Mol Plant Pathol 28: 411–421.
- Jaworski, C.A., S.M. McCarter, A.W. Johnson & R.E. Williamson, 1978. Response of onions grown for transplants to soil fumigation. J Amer Soc Hort Sci 103: 385–388.
- Katan, J., L. Rotem, Y. Finkel & J. Daniel, 1980. Solar heating of the soil for the control of pink root and other soilborne diseases in onions. Phytoparasitica 8: 39–50.
- Kehr, A.E., M.J. O'Brien & E.W. Davis, 1962. Pathogenicity of *Fusarium oxysporum* f.sp. *cepae* and its interaction with *Pyrenochaeta terrestris* on onion. Euphytica 11: 197–208.
- Kodama, F., 1983. Studies on basal rot of onion caused by *Fusarium oxysporum* f. sp. *cepae* and its control. Rep Hokkaido Agr Expt Stn 39: 1–65.
- Koriem, S.O., F.N. Hussein & A.H. Metwally, 1991. Chemical control of pink root, basal rot, and neck rot diseases of onion produced by sets. Assiut J Agr Sci 22: 81–96.
- Koycu, N.D. & N. Ozer, 1997. Determination of seed-borne fungi in onion and their transmission to onion seeds. Phytoparasitica 25: 25–31.
- Krueger, S.K., 1986. Resistance to *Fusarium* basal rot in onions (*Allium cepa* L.). MS Thesis. Univ. Wisconsin, Madison.
- Krueger, S.K., A.A. Weinman & W.H. Gabelman, 1989. Combining ability among inbred onions for resistance to fusarium basal rot. HortScience 24: 1021–1023.
- Lacy, M.L. & D.L. Roberts, 1982. Yields of onion cultivars in Mid-western organic soils infested with *Fusarium oxysporum* f. sp. *cepae* and *Pyrenochaeta terrestris*. Plant Dis 66: 1003–1006.
- Link, G.K.K. & A.A. Bailey, 1926. *Fusaria* causing bulb rot of onions. J Agr Res 33: 929–952.
- Lorbeer, J.W. & K.W. Stone, 1965. Reaction of onion to *Fusarium* basal rot. Plant Dis Rep 49: 522–526.
- Ozer, N. & N.D. Koycu, 1998. Evaluation of seed treatments for controlling *Aspergillus niger* and *Fusarium oxysporum* on onion seed. Phytopath Medit 37: 33–40.
- Palo, M.A., 1928. A *Fusarium* causing bulb rot of onion in the Philippines. Philippine Agr 17: 301–316.
- Rajendran, K. & K. Ranganathan, 1996. Biological control of onion basal rot (*Fusarium oxysporum* f. sp. *cepae*.) by combined application of fungal and bacterial antagonists. J Biol Control 10: 97–102.
- Retig, N., A.F. Kust & W.H. Gabelman, 1970. Greenhouse and field tests for determining the resistance of onion lines to fusarium basal rot. J Amer Soc Hort Sci 95: 422–424.
- Roberti, R., P. Flori, V. Brandolini & L. Ghisellini, 1989. Effetti della concia di bulbi e semi di cipolla contro *Fusarium oxysporum* f. sp. *cepae* Sn. Et Hans. Difesa Delle Piante 12: 11–22.
- Shalaby, G.I. & E. Struckmeyer, 1966. The mode of entrance of the fusarium rot fungus into the bulbs of onions. J Amer Soc Hort Sci 89: 438–442.
- Shinmura, A., H. Sakamoto, T. Hayashi, H. Hoshi & A. Tanii, 1998. Occurrence of *Fusarium* basal rot of Welsh onion caused by *F. oxysporum*. Bull. Hokkaido Pref Agr Expt Stn 74: 35–41.

- Sokhi, S.S., D.P. Singh & M.C. Joshi, 1974. Sources of resistance to basal rot of onions caused by *Fusarium oxysporum*. Indian J Mycol Plant Pathol 4: 214–215.
- Somkuwar, R.G., R. Veere Gowda., T.H. Singh & C.S. Pathak, 1996. Screening of onion for resistance to onion basal rot. Madras Agr J 83: 273–275.
- Srivastava, K.J. & S.M.H. Qadri, 1984. Some studies of damping-off disease of onion (*Allium cepa* L.). Indian Bot Rep 3: 147–148.
- Stadnik, M.J. & O.D. Dhingra, 1996. Response of onion genotypes to *Fusarium oxysporum* f.sp. *cepae* during the growth phase and in storage. Fitopatol Bras 21: 431–435.
- Stadnik, M.J. & O.D. Dhingra, 1997. Root infection by *Fusarium oxysporum* f.sp. *cepae* at different growth stages and its relation to the development of onion basal rot. Phytopath Medit 36(1): 8–11.
- Stevenson, T.R. & M.F. Heimann, 1981. Onion (*Allium cepa*) disorder: Fusarium basal rot. Univ Wisc Ext Bull A3114.
- Tahvonen, R., 1981. Storage fungi of onion and their control. J Sci Agr Soc Finland 53: 27–41.
- Takakuwa, M., N. Ishizaka, F. Kodama & I. Saito, 1977. Host range of *Fusarium oxysporum* f. sp. *cepae*, causal fungus of Fusarium basal rot of onion. Ann Phytopathological Soc Jap 43: 479–481.
- Tsutsui, K., 1991. Inheritance of resistance to *Fusarium oxysporum* in onion. MS Thesis. Univ. Wisconsin, Madison.
- U.S. Dept. Agr, 1999. VG 1–2(99). U.S. Govt. Printing Office, Washington, D.C.
- Villanueva-Mosqueda, E., 1996. Onion heritability for pink root resistance, *Fusarium* basal rot resistance, and bolting traits. MS Thesis. New Mexico State Univ., Las Cruces.
- Villevieille, M., 1996. Mise au point d'un test de selection pour *Fusarium oxysporum* Schlect. emend. Snyder & Hansen f. sp. *cepae*. Acta Bot. Gallica 143: 109–115.
- Walker, J.C. & E.C. Tims, 1924. A fusarium bulb rot of onion and the relation of environment to its development. J Agr Res 28: 683–693.
- Wall, M., E. Shannon & J. Corgan, 1993. Onion diseases in New Mexico. New Mexico State Univ. Coop. Ext. Serv. Circ. 538, 10 pp.