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GERMPLASM

Registration of FC305 Multigerm Sugarbeet Germplasm Selected from a Cross to a Crop Wild Relative

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ABSTRACT

FC305 (Reg. No. GP-286, PI 671963) sugarbeet (*Beta vulgaris* L.) germplasm was developed and released by the USDA–ARS, at Fort Collins, CO, Salinas, CA, and East Lansing, MI, in cooperation with the Beet Sugar Development Foundation, Denver, CO. This germplasm is a diploid, sugarbeet population in normal cytoplasm, segregating for self-sterility (*Sf:SsSs*), multigermity (*M:mm*), and hypocotyl color (*R:rr*). FC305 has moderate resistance to cercospora leaf spot (CLS) (caused by *Cercospora beticola* Sacc.), aphanomyces root rot (caused by *Aphanomyces cochlioides* Drechsl.), and beet curly top (*Beet curly top virus*), as well as resistance to Fusarium yellows [caused by *Fusarium oxysporum* Schlecht. f. sp. *betae* (Stewart) Snyd. & Hans.]. This germplasm is segregating for resistance to rhizomania (*Beet necrotic yellow vein virus*) and contains both *Rz1* and *Rz2* genes for resistance. There was no resistance to root-rotting strains (AG-2–2) of *Rhizoctonia solani* Kühn. FC305 could provide an alternate source of resistance to CLS and Fusarium yellows in a diverse genetic background to enrich the cultivated sugarbeet germplasm base. FC305 provides a source from which to select disease-resistant, multigerm pollinator parents. Because monogerm and O-type is within its parentage, it should be possible to select monogerm, O-type, CMS-maintainer lines from FC305 as well.

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Fusarium yellows is important in sugarbeet (*Beta vulgaris* L.) production areas in the United States and is found throughout sugarbeet growing areas worldwide (Panella and Lewellen, 2005). The causal agent of Fusarium yellows is the fungal, soil-borne pathogen *Fusarium oxysporum Schlecht*. f. sp. *betae* (Stewart) Snyd. & Hans. (FOB). The severity of Fusarium yellows is influenced by temperature, inoculum dose, and presence of sugarbeet cyst nematode (*Heterodera schachtii* Schm.) (Gao et al., 2008; Hanson et al., 2009b; Harveson and Rush, 1998; Landa et al., 2001). When conditions favor its occurrence, yield losses can be devastating (Hanson et al., 2009b). Unfortunately FOB is highly variable in its morphology, pathogenicity, and genetic structure (Harveson and Rush, 1997; Hill et al., 2011; Ruppel, 1991). Other species of *Fusarium* also have been shown to cause yellowing-like symptoms on sugarbeet (Burlakoti et al., 2012; Hanson and Hill, 2004). Research to date has identified resistant commercial cultivars and a high degree of variability in virulence (Hanson et al., 2009b). Management of this disease is heavily dependent on the use of resistant hybrid cultivars (Hill et al., 2011).

Cercospora leaf spot (CLS), caused by *Cercospora beticola* Sacc., is a continual problem in sugarbeet growing areas where the summers are hot and humid (Jacobsen and Franc, 2009). It is an extremely damaging and, therefore, economically important

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Abbreviations: BCT, beet curly top; BNYVV, *Beet necrotic yellow vein virus*; BSDF, Beet Sugar Development Foundation; CLS, cercospora leaf spot; DI, disease index; FOB, *Fusarium oxysporum* f. sp. *betae*; KWS, Kleinwanzlebener Saatzucht; RB-BNYVV, resistance (*Rz1* mediated) breaking strains of BNYVV; spg, sprouts per gram.

foliar disease of sugarbeet worldwide (Panella and McGrath, 2010). Today, CLS is managed in most of the sugarbeet growing areas that are prone to CLS by a combination of CLS-resistant cultivars and timely fungicide applications, especially in areas under heavy disease pressure (Miller et al., 1994; Secor et al., 2010). The incidence of fungicide resistant *C. beticola* strains is increasing, which underscores the fact that the best management program is a combination of rotating chemical protectants, coupled with a strong level of disease resistance in the commercial cultivars (Bolton et al., 2012; Davidson et al., 2006; Hanson, 2010).

Much of the Cercospora-resistant germplasm currently in use came out of Munerati's program in Italy in the early 1900s, in which *B. vulgaris* spp. *maritima* was the source of resistance genes (reviewed in Lewellen, 1992). Since then, a major concern in the development of CLS-resistant sugarbeet has been the loss of vigor in those lines due to the continual inbreeding to fix the multiple genes that confer resistance (Coons et al., 1955; McFarlane, 1971; Panella, 1998; Panella and McGrath, 2010). This makes the production of seed on these lines difficult. The use of hybrid cultivars has ameliorated this problem to some extent. Nonetheless, inbreeding creates a continuing need to broaden the genetic base in our CLS-resistant germplasm for increased vigor and diversity among parental lines to maximize heterosis. In addition to broadening the genetic base of the commercial sugarbeet cultivars, novel genes for resistance to CLS may lead to transgression of the currently available resistance to CLS (Allard, 1960, p. 472; Rieseberg et al., 1999).

Methods

FC305 Parent Populations

FC305 (Reg. No.GP-286, PI 671963) was created by crossing a sugarbeet population enriched for sucrose production as the female and a sea beet (*Beta vulgaris* subsp. *maritima*) population as the male. The female parent in this cross was a population that had been developed to improve the sucrose content of the germplasm created at Fort Collins, CO. In synthesizing the population used as the female, genetic male sterile (*aa*) plants from 3859 (released as C859, PI 565285) from USDA–ARS at Salinas, CA, were used as females (Lewellen, 1995; Owen, 1942). The germplasm 3859 was used because it carried genetic variability for genetic male sterility (*aa*), resistance to rhizomania (*Rz1*), and high resistance to beet curly top (BCT) (Lewellen, 1995). Pollen was provided by three obsolete commercial hybrids, 'MonoHy T6' (17 plants), 'MonoHy A7' (35 plants), and 'MonoHy A4' (25 plants), and by a smooth root germplasm released from USDA–ARS at East Lansing, MI, with resistance to CLS and aphanomyces, 'SR 87' (PI 607899) (25 plants) (Saunders et al., 2000; Theurer, 1993). Forty-six plants of the female (male-sterile), 3859, were harvested to produce 19951011H2. 19951011H2 was bulk increased with 139 of 145 plants harvested to produce 19961005 (Fig. 1). There were two more cycles of intercrossing with only the male sterile plants harvested, which produced 19991024H2. The CMS trait of the MonoHy hybrids would have been purged because they were crossed into the normal cytoplasm of 3859. This population was the female parent in the cross that produced FC305.

Seed of the male parent in the cross that created FC305 was received as accession BGRC 45511 in 1994. A BGRC accession number signifies that the seed came from the genebank at Braunschweig, Germany; however, since then, seed has been transferred to the Federal ex situ Genebank for agricultural and horticultural crops at the Leibniz Institute of Plant Genetics and Crop Plant Research (IPK). The IPK genebank renumbered the accession as BETA 1800. Seed from this accession was collected by H. A. Cortessi with support of the International Board for Plant Genetic Resources (IBPGR; today, Bioversity International) east of Thessaloniki, Greece, in 1983 from a population that appeared to be a mixture of cultivated beet (*Beta vulgaris* subsp. *vulgaris*) and sea beet. It was labeled with collection number GR/83 003. Subsequent testing in Italy at San Stino in 1989 and in China (Hulan County) in 1996 by partner institutions of the BGRC indicated that the accession had strong resistance to CLS. In two independent experiments in 1989 conducted by Kleinwanzlebener Saatzucht (KWS) and Stichting voor Plantenveredeling, which became Plant Research International (Wageningen, the Netherlands), BGRC 45511 was screened for resistance to rhizomania (on a scale of 1 =healthy to 9 = dead); the results were 1 (18 accessions, range 1–7) and 7 (19 accessions, range 1–9), respectively, indicating some tolerance of rhizomania. When grown in the greenhouse at Fort Collins, CO, BGRC 45511 behaved as an annual.

FC305 Population Development

BGRC 45511 was bulk increased in the greenhouse (104 plants) to produce 19981001H. One hundred plants from 19981001H were crossed with genetic male sterile plants (*aa*)

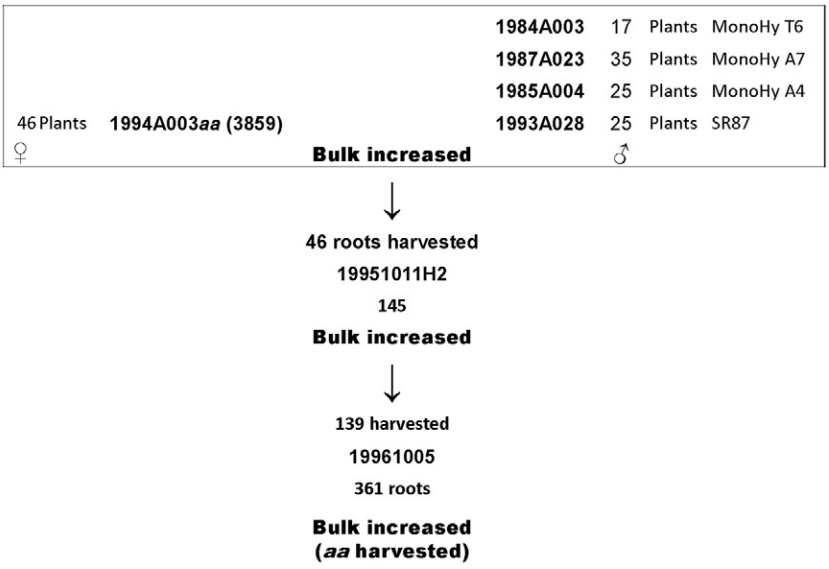

Fig. 1. In synthesizing the population used as the female to cross with the wild sea beet, genetic male sterile (*aa*) plants from 3859 were used as females. Pollen was provided by three obsolete commercial hybrids, 'MonoHy T6', 'MonoHy A7', and 'MonoHy A4', and a smooth root germplasm, SR 87, in a polycross design. Forty-six plants of 3859 were harvested to produce 19951011H2. 19951011H2 was bulk increased with 139 of 145 plants harvested to produce 19961005.

of 19991024H2 (100 plants), and 56 of the female plants (*aa*) of 19991024H2 were harvested to produce 20011046H2.

A bulk increase of 272 biennial plants of 20011046H2 produced 20021036bb, which was increased (96 plants) to produce 20051006. Seventy-nine half-sib families were produced in the greenhouse from a mix of 20051006 (59 plants) and 20021036bb (43 plants). The 57 families with the most seed were screened in the field at Frankenmuth, MI, in an artificially inoculated CLS epiphytotic. Remnant seed from the best-performing four families was increased (396 plants) in the greenhouse to produce 20091029MS (seed from male sterile plants) and 20091029PF (pollen fertile plants). Two bulk increases in the greenhouse of 20091029PF produced 20111029 (62 plants) and 20141014 (147 plants). Seed from 170 plants of 20111029 was increased in isolation in the field in 2013 as seed production 20131006. FC305 will be released from seed productions 20111029, 20131006, and 20141014. It has been tested as either 20091029PF or 20111029.

Characteristics

Agronomic and Morphological Description

FC305 has fertile cytoplasm and is predominately multigerm but segregates for the monogerm seed ball trait—20091029PF (5%), 20111029 (16% monogerm), 20131006 (13% monogerm), and 20141014 (19%). Segregation may occur for O-type (maintainer of CMS equivalents) from the 3859 parent, but testing has not been done to determine if plants that express restorer genes are present (Owen, 1945). FC305 segregated for genetic male sterility (*aa*) and self-sterility (S^s) because they were introduced through Salinas germplasm 3859. 20091029PF was 29% male sterile, 20131006 was 30% male sterile, and 20141014 was 20% male sterile. FC305 is expected to be segregating for the smooth root trait introduced through the East Lansing germplasm SR87 (Saunders et al., 2000). FC305 also segregated for hypocotyl color, with the percentage of green hypocotyls in the populations as follows: 20091029PF (42%), 20111029 (32%), 20131006 (45%), 20141014PF (22%), and 20141014MS (45%). When tested for germination, sprouts per gram (spg) of seedballs were as follows: 20091029PF had 134 spg, 20111029 had 167 spg, 20131006 had 28 spg, 20141014PF had 129 spg, and 20141014MS had 66 spg.

Resistance to Disease

Cercospora Leaf Spot

Screening of half-sib families in 2008 for CLS was done at the Saginaw Valley Bean and Beet Research Farm (B&B) in Michigan under an artificial epiphytotic (Hanson et al., 2009a; Ruppel and Gaskill, 1971). Because seed was limited, a single two-row plot was planted with internal controls (a susceptible check, 19941027, and a resistant check, 19821051H2). The nursery was inoculated on 8 July with a liquid spore suspension. A disease index (DI) based on visual observations of the plot on a scale from 0 to 10 (where 0 = no apparent infection and 10 = complete defoliation) was used to evaluate resistance to CLS (Ruppel and Gaskill, 1971). Selection of four families based on their DI was made on 28 August, the height of the epiphytotic, and remnant seed of those families was planted in the greenhouse (Hanson et al., 2009a). The average DI of the four families was 1.75; the resistant control was 2.3, and the susceptible control was 5.8.

FC305 was evaluated by Betaseed, Inc., in a field nursery for resistance to CLS at Rosemount or Randolph, MN, during development of the germplasm (Table 1). All plots were two rows, 3 m long with 56 cm row spacing. The seed was treated with Allegiance (dimethylphenyl methoxyacedtyl, Bayer CropScience), Thiram (tetramethylthiuram disulfide, Bayer CropScience), and Tachigaren (hymexazol, Mitsui Chemicals Agro Inc.). Trials were planted in early May and thinned to a uniform stand of 17 cm between plants. The nursery was inoculated during the first 2 wk of July with *C. beticola* infected leaves at a rate of 5.0 kg ha^{-1}. Solid set sprinklers (2011) or a linear overhead sprinkler (2012, 2013) provided adequate moisture for initial infection and then as needed to maintain conditions favorable for CLS development (Panella et al., 2008). The KWS rating scale (Kleinwanzlebener Saatzucht, 1970; Panella et al., 2008) was used to evaluate leaf spot infection. A DI was based on visual ratings from 1 (absence of leaf spots) to 9 (all leaves on the plant are entirely necrotic). Ratings were taken each week during the infection period. The experimental design was a randomized complete block with three replicates (P. O'Boyle, personal communication, 2014). In 3 yr of testing, FC305 had an intermediate level of resistance; the mean DI for all of the ratings was better (i.e., lower) than the USDA–ARS susceptible check and higher than the USDA–ARS resistant check (Table 1). However, at the last reading in the severe epidemic in 2013, FC305 was not significantly different from the susceptible check (Table 1).

Table 1. FC305 (20091029PF and 201110129) evaluated in the Betaseed, Inc., cercospora leaf spot nursery at Rosemount, MN (2011) and Randolph, MN (2012 and 2013).

Entry	Description	2011		2012		2013	
		Last reading†	Mean	Last reading	Mean	Last reading	Mean
		1–9‡					
FC305	20091029PF	5.5	3.0	4.3	2.7		
FC305	201110129			3.7	2.5	8.0	4.3
USDA–ARS tolerant check	19821051H2	2.9	2.0	1.9	1.4	4.3	2.1
USDA–ARS susceptible check	19941027	5.8	3.2	6.8	4.0	8.7	5.4
$LSD_{0.05}$		2.25	0.92	1.4	0.7	1.2	0.63
CV		24.9	16.4	21.8	15.7	11.9	10.7

† The last reading is usually the most severe of the epiphytotic.

‡ The disease index is based on the Kleinwanzlebener Saatzucht visual rating system, where 1 = the absence of leaf spot spots and 9 = all leaves entirely necrotic.

Fusarium Yellows

A Fusarium screening nursery was planted by Betaseed, Inc., near Moorhead, MN, in 2013. It was a completely randomized complete blocked design with three replicates. Reaction to Fusarium was scored on the basis of stand persistence and foliar yellowing of plots. The disease index used to evaluate the lines was based on a visual rating from 1 (completely healthy) to 9 (all dead or missing) (P. O'Boyle, personal communication, 2014). Pressure was lighter than desired (susceptible control at a DI of 4.3 instead of 7 to 9) (Table 2). Nonetheless, there were significant differences among entries. FC305 had a DI of 1.0, indicating resistance to Fusarium yellows (Table 2)

Beet Curly Top

Beet curly top (BCT) disease is caused by *Beet curly top virus*, which is transmitted by the beet leafhopper (*Circulifer tenellus*) (Bennett, 1971; Strausbaugh et al., 2008). Although one of the parental components from Salinas, CA (3859, PI 565285) had moderate resistance to BCT, no selection was made for resistance to BCT during the development of FC305 (Lewellen, 1995). FC305 was tested at the joint USDA–ARS Beet Sugar Development Foundation (BSDF) curly top nursery at Kimberly, ID, in 2011, 2012, and, 2013 as previously described (Panella et al., 2008; Panella and Strausbaugh, 2012; Panella and Strausbaugh, 2013; Panella and Strausbaugh, 2014). The plots were visually evaluated and rated on a DI scale of 0 (no symptoms) to 9 (dead). The most important rating is the final rating, in which the disease expression is at its peak (Mumford, 1974). Only in the milder infection in 2011 was FC305 significantly more resistant than the susceptible control and not significantly different from the resistant controls (Table 2). This germplasm may have some potential for selection of higher resistance to BCT.

Aphanomyces Damping Off and Root Rot (Aphanomyces Black Root)

FC305 was evaluated for resistance to aphanomyces root rot (caused by *Aphanomyces cochlioides* Drechsl.) in field nurseries near Shakopee, MN, by Betaseed, Inc. (Panella et al., 2008). A DI based on a visual 1 to 9 rating scale of stand persistence and plant health was used to evaluate aphanomyces root rot damage, where a rating of 1 is a complete stand of healthy beets and a rating of 9 has no surviving plants. Ratings were taken one to three times during the growing season. Experimental design was a randomized complete block with three replicates (P. O'Boyle, personal communication, 2014).

Table 2. FC305 (20111029 and 20091029PF) evaluated in 2011 and 2012 in the Betaseed, Inc., aphanomyces root rot nurseries at Shakopee, MN, and in 2013 for response to Fusarium yellows in Moorhead, MN. This germplasm also was evaluated in 2011, 2012, and 2013 in the USDA–ARS Beet Sugar Development Foundation beet curly top evaluation nursery in Kimberly, ID.

Entry	Source	Aphanomyces root rot rating		
		2012	2011	2011
		Foliar†	Foliar	Root‡
		1–9		1–9
FC305	20111029	3.3		
FC305	20091029PF	5.8	5.5	7.0
Resistant control	Betaseed, Inc.	3.0	3.0	5.0
Susceptible control	Betaseed, Inc.	7.9	7.2	7.9
$LSD_{0.05}$		1.9	1.3	0.9
CV		30.9	14.2	8.2
		Beet curly top rating§		
		2013	2012	2011
			1–9	
FC305	20091029PF		5.5	4.3
FC305	20111029	5.7		
Resistant control	HM PM90	4.2	4.2	3.1
Resistant control	Beta G6040	4.4	5.0	4.2
Susceptible control	Monohikari	6.3	6.1	5.8
$LSD_{0.05}$		1.0	0.7	1.3
		Fusarium yellows rating§		
		2013		
		1–9		
FC305	20111029	1.0		
Resistant control	Betaseed, Inc.	1.3		
Susceptible control	Betaseed, Inc.	4.3		
$LSD_{0.05}$		0.79		
CV		27.6		

† A disease index (DI) based on a visual 1 to 9 rating scale of stand and plant health was used to evaluate aphanomyces root rot damage, where 1 = no symptoms and 9 = dead plants.

‡ Roots were lifted, and a DI based on a visual 1 to 9 rating (per plot) was used to evaluate root damage, where 1 = no rot and 9 = dead plants, totally rotted.

§ Plots were visually observed and rated on a scale of 1 (no symptoms) to 9 (dead) to develop a DI to evaluate resistance. Ratings are from the final rating or most severe rating, which is the most important indicator of the resistance of the germplasm being screened.

In the aphanomyces evaluations of 2011 and 2012, FC305 was significantly less resistant than the resistant control and significantly more resistant than the susceptible control (Table 2). However, in the 2012 nursery, the 20111029 source of FC305 was significantly more resistant than 20091029PF and not significantly different from the resistant control (Table 2). FC305 showed an intermediate resistance and may have some potential for selection of higher resistance to *A. cochlioides*.

Rhizomania

Two major dominant resistance genes are incorporated into commercial sugarbeet germplasm to manage rhizomania caused by *Beet necrotic yellow vein virus* (BNYVV), *Rz1* and *Rz2* (Biancardi et al., 2010; De Biaggi et al., 2011). In some locations, BNYVV has been able to overcome the resistance of *Rz1* (resistance breaking BNYVV—RB-BNYVV), the first resistance gene deployed (Biancardi et al., 2002). In these areas, to provide sugarbeet with tolerance to RB-BNYVV strains, *Rz1* and *Rz2* are stacked in the commercial hybrids. FC305 was evaluated at the USDA–ARS BSDF rhizomania nurseries in Kimberly, ID, in 2012 (Strausbaugh and Panella, 2013) and 2013 (Strausbaugh and Panella, 2014). FC305 was significantly more resistant than the rhizomania-susceptible control (*rz1rz1rz2rz2*) in the early rating in 2012 but not significantly different from the rhizomania-susceptible control for the later rating in 2012 or the rating in 2013 (Table 3). Single-nucleotide polymorphism (SNP) markers linked to *Rz1* and *Rz2* were used to genotype a sample of 96 haplotypes, which consisted of 48 of the 127 plants from 20091029PF (Stevanato et al., 2012; Stevanato et al., 2014). Based on the SNP data, *Rz1* was present in the sample with an allele frequency of 70% and *Rz2* with an allele frequency of 40%.

Other Diseases

Fungal growth on harvested roots during storage before processing was determined (storage rot) (Toda et al., 2012) (Table 3). Although it was not significantly different from the rhizomania-susceptible check for fungal growth in storage in 2012, it also did not have more fungal growth than either of the rhizomania-resistant controls carrying the *Rz1* or *Rz2* resistance gene (Table 3). Only the rhizomania control with stacked resistance (both resistance genes, *Rz1rz1Rz2rz2*) had significantly less fungal growth in storage. In 2013, FC305 had significantly less fungal growth than the rhizomania-susceptible control and was not significantly different from the Rz1 control. It did show significantly more fungal growth in storage than did the *Rz1Rz2* control and the *Rz2* control.

FC305 was tested for resistance to rhizoctonia root and crown rot (caused by *Rhizoctonia solani* Kühn, AG-2–2, Isolate R-9) at Fort Collins, CO, in 2012 and 2013 as previously described (Panella et al., 2008). FC305 was not significantly more resistant than the susceptible control in either year and showed significantly less resistance than the resistant control in both years (data not shown). FC305 has not been tested for resistance to sugarbeet root aphid (*Pemphigus* sp.) but based on the parentage, it may be possible to find root aphid resistance in FC305.

Availability

Breeder seed of FC305 is maintained by the USDA–ARS and will be provided in quantities sufficient for reproduction on written request to Sugarbeet Research, USDA–ARS, Crops Research Laboratory, 1701 Center Ave., Fort Collins, CO 80526-2083. Seed of FC305 has been deposited in the National Plant Germplasm System, where it will be available for research purposes, including development and commercialization of new cultivars. We request that appropriate recognition be made of the source when this germplasm contributes to a new cultivar. U.S. Plant Variety Protection will not be requested for FC305.

Table 3. FC305 screened at the USDA–ARS Beet Sugar Development Foundation nursery in Kimberly, ID, for resistance to *Beet necrotic yellow vein virus* (the causal agent of rhizomania) and storage rot. The plots were one row 3 m long with 0.56 m row spacing and arranged in a randomized complete block design with six replicates.

Entry	Source	Fungal growth in storage†	Rhizomania foliar rating (% susceptible plants)	
			13 July	17 Sept.
		%	%	
		2012		
FC305	20091029PF	60	58	68
Susceptible check	*rz1rz1rz2rz2*‡	71	95	75
Moderately resistant check	*rz1rz1Rz2rz2*	46	3	0
Resistant check	*Rz1rz1*	46	0	0
Very resistant check	*Rz1z1Rz2z2*	32	0	0
Overall mean		30	36	31
$LSD_{0.05}$		20	20	17
		2013		
FC305	20111029	46	62	
Susceptible check	*rz1rz1rz2rz2*	70	72	
Moderately resistant check	*rz1rz1Rz2rz2*	28	32	
Resistant check	*Rz1rz1rz2rz2*	43	2	
Very resistant check	*Rz1rz1Rz2rz2*	26	0	
Overall mean		20	31	
$LSD_{0.05}$		14	21	

† Fungal growth in storage = the percentage of root surface area covered by fungal growth

‡ Rhizomania is managed through the use of plant resistance. Currently, two single genes in commercial cultivars are used for resistance, *Rz1* and *Rz2*.

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