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ORIGINAL PAPER

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Five independent loci each control monogenic resistance to gummy stem blight in melon (*Cucumis melo* L.)

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Abstract Inheritance and segregation analysis demonstrated that five independent genes in melon confer monogenic resistance to foliar infection by the fungal pathogen *Didymella bryoniae*, resulting in the disease known as gummy stem blight (GSB). In this study, two new monogenic sources of GSB resistance were characterized. Resistance in *Cucumis melo* PI 482398 was monogenic dominant based on segregation analysis of F_1, F_2 and backcross populations, while resistance in *C. melo* PI 482399 showed monogenic recessive inheritance. Four accessions, PI 482398, PI 157082, PI 511890, and PI 140471, each previously known to carry monogenic dominant resistance to GSB, were intercrossed to determine genetic relationships among these resistance sources. Recovery of susceptible individuals in F_2 populations confirmed that these accessions possess different resistance genes. Resistance loci were designated *Gsb-1* (formerly *Mc*, monogenic dominant resistance from PI 140471), *Gsb-2* (monogenic dominant resistance from PI 157082), *Gsb-3* (monogenic dominant resistance from PI 511890), *Gsb-4* (monogenic dominant resistance from PI 482398) and *gsb-5* (monogenic recessive resistance from PI 482399).

Introduction

Gummy stem blight (GSB) is a severely destructive foliar disease of cucurbits caused by the ascomycete fungus *Didymella bryoniae* (Auersw.) Rehm and its anamorph *Phoma cucurbitacearum* (Fr.:Fr.) Sacc. Agriculturally significant losses in melon, the focus of this study, occur regularly in the southern United States (Sherf and MacNab 1986) and in several other regions worldwide including Asia and Europe. In melon, symptoms of this disease include spreading necrotic lesions on leaves and water-soaking of both leaves and hypocotyls resulting in the formation of stem cankers in cortical tissue that produce a characteristic brown gummy exudate. In a susceptible interaction, these lesions continue to expand, eventually girdling the stem leading to wilting and death of the plant (Sitterly and Keinath 1996). The ineffectiveness of chemical control especially during periods of high rainfall and humidity (Norton and Cosper 1989), and the widespread resistance of *D. bryoniae* to commonly used systemic benzimidazole fungicides (Keinath and Zitter 1998) have generated strong interest in the possibility of breeding melon varieties resistant to GSB.

Because of the agricultural importance of this disease, early searches for genetic resistance were undertaken. Although several sources of resistance to GSB have been reported in the ensuing decades (Sowell Jr. et al. 1966; Sowell Jr. 1981; McGrath et al. 1993), GSB-resistant melon varieties and breeding lines released to date, (e.g., Norton 1971, 1972; Norton et al. 1985; Norton and Cosper 1989), all derive resistance from PI 140471 (a wild melon originating in Texas) and have failed to provide adequate levels of resistance (Sowell Jr. 1981; Sitterly and Keinath 1996; Zhang et al. 1997). While resistance is clearly apparent in this accession which is a dudaim type, the level of resistance derived from this source appears to be diminished when introgressed into large-fruited commercially acceptable genetic backgrounds (Zhang et al. 1997). In an effort to find new sources of GSB resistance, 798 *C. melo* plant introductions were screened in greenhouse and field tests for high levels of resistance to GSB, emphasizing accessions from the humid tropics and subtropics along with accessions previously known to have resistance to other foliar diseases (Zhang et al. 1997). This study identified several accessions with resistance equal to or greater than PI 140471. From that effort, four additional wild melon accessions (PI 157082 from China, PI 511890 from

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Mexico, PI 482398 and PI 482399 from Zimbabwe) were selected for further characterization. GSB resistance in both PI 157082 and PI 511890 was subsequently shown to be monogenic and dominant, and furthermore, the resistance in PI 157082 was shown to segregate independently from resistance attributed to the locus *Mc* for GSB resistance in PI 140471 (Zuniga et al. 1999). A preliminary inheritance study of resistance derived from PI 482399 indicated that this resistance was recessive (Zuniga et al. 1997).

The present study was undertaken to provide a comprehensive understanding of the resources in *C. melo* available for resistance to this disease and to determine which breeding strategies will be most likely to yield success. Despite the commercial importance of the Cucurbitaceae, this family has lagged behind some other families such as the Solanaceae and Cruciferae with respect to investments in genomic resources. Basic knowledge of the genetics of important traits in this family, particularly agriculturally important traits such as GSB resistance that are found in a number of cucurbit crop species, will be essential in order to extend the impact of genomic analysis in melon to melon-breeding programs. The first objective of this study was to determine the mode of inheritance of GSB resistance found in PI 482398 and PI 482399. The second objective was to determine the genetic relationship among resistance sources PI 140471, PI 157082, PI 511890, PI 482398, and PI 482399.

Materials and methods

Germplasm and population development

All plant introduction (PI) accessions were obtained from the USDA NPGS Ames, Iowa. The GSB-susceptible genotype used as a parent in this study was 'Cornell ZPPM 339' (ZM) (Cornell University, Ithaca, N.Y.), a cantaloupe breeding line with resistance to zucchini yellow mosaic virus, papaya ringspot virus and powdery mildew. The GSB susceptible check for disease screens was 'Honeydew Greenflesh' (HD) (Asgrow Seed Co., San Juan Bautista, Calif.). Controlled pollinations were carried out in the field and greenhouse as described previously (Zuniga et al. 1999). For inheritance studies, ZM was crossed with PI 482398 and PI 482399 to generate reciprocal F_1, F_2 and backcross populations, as described previously (Zuniga et al. 1999). To determine whether resistance genes segregated independently, the five resistant PI accessions, PI 140471 (471), PI 157082 (082), PI 511890 (890), PI 482398 (398) and PI 482399 (399), were intercrossed to generate F_1, F_2 and backcross populations.

Disease screening

Resistance evaluations were made in the greenhouse according to methods described in Zhang et al. (1997) using the highly virulent *D. bryoniae* isolate NY1 (Keinath et al. 1995). Two susceptible control plants and two resistant control plants (471) were planted in each seedling flat to monitor inoculation efficiency and to test severity of infection. Spore suspensions were applied to stem and leaf surfaces of 3- to 4-week-old seedlings at 5×10^5 spores/ml using a backpack sprayer, followed by a 72-h incubation period at 25°C in a mist chamber to promote uniform development of disease symptoms. Symptom severity was measured 21 days post-inoculation using a scale from 1 to 5 based on stem damage ratings (Zuniga et al. 1999) where 1=no damage; 2=a single lesion 1–10 mm long or coalesced lesions 1–20 mm long with no girdling of the stem; 3=lesions 21–80 mm and/or girdling of the stem; 4=withered stem; 5=dead seedling. Our previous studies have indicated that classification of segregating progenies is most reliably based on stem damage ratings where resistant individuals typically score 1 or 2 and susceptible individuals usually score 4 or 5 (Zhang et al. 1997; Zuniga et al. 1999). The number of individuals falling into resistant (stem damage ratings 1–2) and susceptible (stem damage ratings 3–5) categories was determined in each segregating population, and the resulting ratios were tested for goodness-of-fit using χ^2 analysis.

Results

Inheritance of resistance to GSB derived from PI 482398 (398)

Parental lines and (ZM×398) F_1, F_2 and backcross populations were scored for response to inoculation with *D. bryoniae*. Results are reported in Table 1. The susceptible parent (ZM) was uniformly susceptible with an average disease rating of 4.4. In contrast, the average disease rating for 398 was 1.19; one 398 plant out of 26 was rated a 3, possibly due to high inoculum pressure from an adjacent susceptible check plant 'Honeydew Greenflesh' (HD). HD typically supported severe infections, leading to death of the plant during GSB screening, and local increases in test severity were occasionally noted. Because of extreme difficulty making this cross, only a very small number of F_1 seed were available, but all F_1 plants were uniformly highly resistant to infection from GSB, consistent with dominant inheritance (Table 1). The F_2 population showed segregation consistent with a 3 resistant:1 susceptible ratio indicating monogenic dominant inheritance. The backcross population to the resistant parent 398 was completely resistant while the backcross population to the susceptible parent ZM showed segregation consistent with a 1 GSB resistant:1 GSB susceptible ratio. Taken together, these data indicate that

Table 1 Response of resistant (PI 482398) and susceptible (Cornell ZPPM 339) *Cucumis melo* genotypes and derived populations to inoculation with *Didymella bryoniae*. Symptom severity was measured 21 days post-inoculation using a 1–5 scale based on stem damage ratings where 1= no damage; 2= a single lesion 1–10 mm long or coalesced lesions 1–20 mm long with no girdling of the stem; 3= lesions 21–80 mm and/or girdling of the stem; 4= withered stem; 5= dead seedling

Genotype	Number of plants		Expected	
	R[a]	S[b]	Ratio (R:S)	*P* value
ZM	0	28	0:1	–
398	25	1	1:0	–
(ZM×398) F_1	3	0	1:0	–
(ZM×398) F_2	72	33	3:1	0.13
(ZM×398)×ZM	22	33	1:1	0.14
(ZM×398)×398	56	0	1:0	–

[a] Resistant (stem damage rating 1–2)
[b] Susceptible (stem damage rating 3–5)

Table 2 Response of resistant (PI 482399) and susceptible (Cornell ZPPM 339) *C. melo* genotypes and derived populations to inoculation with *D. bryoniae.* Symptom severity was measured 21 days post-inoculation using a 1–5 scale based on stem damage ratings where 1= no damage; 2= a single lesion 1–10 mm long or coalesced lesions 1–20 mm long with no girdling of the stem; 3= lesions 2–80 mm and/or girdling of the stem; 4= withered stem; 5= dead seedling

Genotype	Number of plants		Expected	
	R[a]	S[b]	Ratio (R:S)	*P* value
ZM	0	28	0:1	–
399	25	1	1:0	–
(ZM×399) F_1	0	28	0:1	–
(ZM×399) F_2	32	77	1:3	0.29
(ZM×399)×ZM	0	54	0:1	–
(ZM×399)×399	34	22	1:1	0.11

[a] Resistant (stem damage rating 1–2)
[b] Susceptible (stem damage rating 3–5)

resistance to GSB in PI 482398 is conferred by a single dominant gene.

Inheritance of resistance to GSB derived from PI 482399 (399)

The reactions of parental lines, (ZM×399) F_1, F_2 and backcross populations to GSB inoculation are indicated in Table 2. The average disease rating for 26 399 plants was 1.5. As observed with the accession 398, one 399 plant also located adjacent to a susceptible check plant was rated 3. In contrast to results obtained for 398, the F_1 population derived from the cross between 399 and ZM was uniformly susceptible to infection from GSB, consistent with recessive inheritance (Table 2). Segregation observed in the F_2 population was consistent with a 1 resistant:3 susceptible ratio, suggesting monogenic recessive inheritance. The backcross population to the susceptible parent ZM was uniformly susceptible, and the backcross population from the resistant parent 399 segregated in a manner consistent with a 1 resistant:1 susceptible ratio. Together, these data demonstrated that GSB resistance from PI 482399 is controlled by a single recessive gene.

Gsb-1 (formerly *Mc*) is distinct from other loci controlling monogenic dominant GSB resistance

The only gene designated to date for resistance to GSB in melon is *Mc*, a locus symbol that reflects the pathogen synonym, *Mycosphaerella citrullina* (Prasad and Norton 1967). Based upon results reported above and the fact that *Mc* is not consistent with current rules governing gene nomenclature in melon, we submitted a proposal to the *Cucumis* Gene Nomenclature Committee for revision of *Mc* derived from PI 140471 to *Gsb-1*. This proposal has been accepted (J. McCreight, personal communication).

Table 3 *Gsb-1* from *C. melo* PI 140471 is genetically distinct from monogenic dominant resistance to gummy stem blight in *C. melo* PIs 157082, 511890, and 492398. Response of resistant (R) parents, (R×R) F_1, and (R×R) F_2 populations to inoculation with *D. bryoniae.* Symptom severity was measured 21 days post-inoculation using a 1–5 scale based on stem damage ratings where 1= no damage; 2= a single lesion 1–10 mm long or coalesced lesions 1–20 mm long with no girdling of the stem; 3= lesions 21–80 mm and/or girdling of the stem; 4= withered stem; 5= dead seedling

Pedigree	Number of plants		Expected	
	R[a]	S[b]	Ratio (R:S)	*P* value
HD[c]	0	28	0:1	–
471	28	0	1:0	–
082[d]	28	0	1:0	–
890	28	0	1:0	–
398	28	0	1:0	–
(471×890) F_1	28	0	1:0	–
(471×890) F_2	186	9	15:1	0.35
(471×398) F_1	25	0	1:0	–
(471×398) F_2	181	15	15:1	0.42

[a] Resistant (stem damage rating 1–2)
[b] Susceptible (stem damage rating 3–5)
[c] Susceptibility check
[d] Segregation of (R×R) F_1 and (R×R) F_2 populations have previously established the independence of monogenic dominant resistance derived from 471 and 082 (Zuniga et al. 1999)

A previous study established that *Gsb-1* segregated independently from monogenic dominant resistance in PI 157082 (Zuniga et al. 1999). Based upon our approved proposal for nomenclature revision and this previously published result, we propose the designation *Gsb-2* for the locus that controls monogenic dominant resistance to GSB derived from PI 157082.

The same study also reported monogenic dominant resistance derived from PI 511890 (890) but again, no locus designation was given. In order to determine whether this resistance was controlled by a locus distinct from *Gsb-1*, 471 was crossed with 890 to generate F_1 and F_2 populations. If *Gsb-1* and the dominant GSB resistance gene in 890 occur at distinct loci, the (471×890) F_1 population should be uniformly resistant and the F_2 population should show segregation consistent with a 15 resistant :1 susceptible ratio. The data obtained from this experiment summarized in Table 3 confirm this hypothesis. Based upon this result, we propose the locus designation *Gsb-3* for the resistance allele derived from 890.

If *Gsb-1* and the newly identified monogenic dominant resistance in 398 occur at independent, unlinked loci, the same pattern of segregation should also be observed from F_1 and F_2 populations derived from the cross (471×398). Results shown in Table 3 are consistent with independence of these two loci. Based upon this result and the segregation data presented in Table 1, we propose the locus designation of *Gsb-4* for monogenic dominant resistance to GSB derived from 398. Taken together, these data confirm that *Gsb-1* is distinct from the loci we have designated *Gsb-2*, *Gsb-3*, and *Gsb-4*.

Table 4 *Gsb-2* from *C. melo* PI 157082 is genetically distinct from monogenic dominant resistance to gummy stem blight in *C. melo* PI 511890 and PI 492398. Response of resistant (R) parents, (R××R) F_1 and (R××R) F_2 populations to inoculation with *D. bryoniae.* Symptom severity was measured 21 days post-inoculation using a 1–5 scale based on stem damage ratings where 1= no damage; 2= a single lesion 1–10 mm long or coalesced lesions 1–20 mm long with no girdling of the stem; 3= lesions 21–80 mm and/or girdling of the stem; 4= withered stem; 5= dead seedling

Pedigree	Number of plants		Expected	
	R[a]	S[b]	Ratio (R:S)	*P* value
HD[c]	0	28	0:1	–
082	28	0	1:0	–
890	28	0	1:0	–
398	28	0	1:0	–
(890×082) F_1	28	0	1:0	–
(890×082) F_2	184	10	15:1	0.53
(082×398) F_1	9	0	1:0	–
(082×398) F_2	95	12	15:1	0.03

[a] Resistant (stem damage rating 1–2)
[b] Susceptible (stem damage rating 3–5)
[c] Susceptibility check

Gsb-2 segregated independently from *Gsb-3* and *Gsb-4*

To confirm directly that *Gsb-2* is independent of *Gsb-3* and *Gsb-4*, 082 was crossed with 890 and 398 as above to generate F_1 and F_2 populations. The resulting populations were screened with GSB and results are reported in Table 4. In both crosses, susceptible segregants were recovered in the F_2 populations, confirming the designation of these loci as distinct. In the case of the (082×398) F_2, more susceptible individuals were recovered than expected according to a 15 resistant:1 susceptible ratio. This result still favors the hypothesis of independent loci, rather than allelism or linkage, and may be due to environmental effects that increased phenotypic screen severity. These data demonstrate that *Gsb-2* is distinct from *Gsb-3* and *Gsb-4*.

Gsb-3 segregated independently from *Gsb-4*

To determine directly whether *Gsb-3* segregated independently from *Gsb-4*, 890 was crossed with 398 as above to generate F_1 and F_2 populations and screened for response to the pathogen. Results are reported in Table 5. Again, susceptible segregants were recovered in the F_2 population, confirming the designation of these loci as distinct. Taken together, this series of experiments supports the designation of four unlinked loci in melon, *Gsb-1-4*, each one of which has an allele which conferred monogenic dominant resistance to GSB.

Gsb-2 segregates independently from *gsb-5*

In addition to testing the allelic relationships between the monogenic dominant sources of resistance in this study, we also wanted to confirm the recessive nature of resistance from PI 482399 (399), designated *gsb-5*, and to evaluate its independence from dominant sources of resistance. Independent segregation of one monogenic recessive and one monogenic dominant gene in an F_2 population results in an expected ratio of 13 resistant individuals:3 susceptible individuals. When 195 F_2 individuals from the cross of (399×082) were screened for resistance to GSB, 160 individuals were resistant and 35 were susceptible, consistent with the expected 13:3 ratio (P=0.77) (data not shown). Similar tests are underway to confirm independence from the other dominant sources of resistance.

Table 5 *Gsb-3* from *C. melo* PI 511890 is genetically distinct from monogenic dominant resistance in *C. melo* PI 482398. Response of resistant (R) parents, (RxR) F_1 and (RxR) F_2 populations to inoculation with *D. bryoniae*

Pedigree	Number of plants		Expected	
	R[a]	S[b]	Ratio (R:S)	*P* value
HD[c]	0	28	0:1	–
890	28	0	1:0	–
398	28	0	1:0	–
(890×398) F_1	28	0	1:0	–
(890×398) F_2	179	17	15:1	0.16

[a] Resistant (stem damage rating 1–2)
[b] Susceptible (stem damage rating 3–5)
[c] Susceptibility check

Discussion

This study defines four genetically distinct loci, each of which has an allele that confers monogenic dominant resistance to GSB, and a fifth locus that controls monogenic recessive resistance. These loci are designated *Gsb-1* (formerly *Mc*) for monogenic dominant resistance from PI 140471, *Gsb-2* for monogenic dominant resistance derived from PI 157082, *Gsb-3* for monogenic dominant resistance derived from PI 511890, *Gsb-4* for monogenic dominant resistance derived from PI 482398, and *gsb-5* for monogenic recessive resistance derived from PI 482399.

The sources of GSB resistance described in our study have been determined in relationship to the highly virulent NY1 isolate of *D. bryoniae*, which was identified and isolated from infected melon tissue in New York (Keinath et al. 1995). While our studies were conducted only with this isolate, a range of *D. bryoniae* isolates collected from watermelon and melon from South Carolina to New York that included NY1 were evaluated on both watermelon and cantaloupe and showed no host-by-isolate interaction (Keinath et al. 1995). No studies to date have investigated the possibility of race-specific GSB resistance in melon, although a study using six American and two European *D. bryoniae* isolates found that GSB resistance in cucumber was not race-specific (St. Amand and Wehner 1995). This result may or may not be relevant to melon because the GSB resistance sources in cucumber

show polygenic inheritance (St. Amand and Wehner 2001). Since our original publication, the accessions used in this study have been included in trials under heavy disease pressure around the world with no evidence of strain-specificity, suggesting that the resistance attributed to these loci may be stable across broadly different environments. Clearly, our results should be confirmed by controlled studies using well-defined fungal isolates typical of target environments before these genes are deployed in resistant varieties for use in those production environments.

The redundancy for resistance to GSB revealed by this study in melon has not been reported for other cucurbits infected by this pathogen; however, multiple dominant race-specific resistance genes exist in melon for two other agriculturally important fungal diseases, powdery mildew and Fusarium wilt. Six dominant genes (*Pm-1, Pm-2, Pm-3, Pm-4, Pm-5, Pm-6*) are known for race-specific resistance to powdery mildew in melon (McCreight et al. 1993), while three unlinked dominant resistance genes in melon (*Fom-1, Fom-2,* and *Fom-3)* have been reported for race-specific resistance to Fusarium wilt (Risser et al. 1976; Zink and Gubler 1985, 1986). Although not yet isolated at the molecular level, both *Fom-1* and *Fom-2* are tightly linked to sequences bearing NBS-LRR motifs (Brotman et al. 2002; Wang et al. 2002).

A hallmark of many dominant resistance genes, including those of the NBS-LRR type, is the hypersensitive response (HR). GSB resistance sources described in this study do not exhibit a detectable HR when challenged with *D. bryoniae* under the conditions employed in this study nor has this response been observed in any of the environments in which materials carrying these genes have been assessed. During the severe controlled environment screens described in this study, inoculated seedling leaves generally became infected in both susceptible and resistant individuals; however, the extent of infection and its consequences varies clearly. The resistance mechanism appears to operate mainly at the junction of leaves and stem. In susceptible plants, the infection quickly spread to the stem, causing lesions and girdling, while in resistant plants foliar lesions expanded less quickly and stem lesion formation appeared to be greatly reduced or inhibited.

The results from the study clearly indicated that in one case, resistance was recessive. Recessive fungal resistance is likely to represent a mechanism distinct from that controlled by dominant resistance genes. Recessive resistance in crop plants to fungal pathogens has been characterized at the molecular level only in the case of the *mlo* gene, which represents a defective negative regulator of constitutive defense (Buschges et al. 1997). Barley plants homozygous for *mlo* exhibit a lesion mimic phenotype, typical of a constitutive defense response. None of the parental, F_2 or backcross individuals homozygous for *gsb-5* exhibited a detectable lesion mimic phenotype. It is possible that *gsb-5* may represent an important target for identification at the molecular level because the wild type allele at this locus may encode a gene product that is important for pathogenesis. If this is the case, then this gene may serve as a useful target for engineering of resistance via gene silencing approaches. Recessive GSB resistance controlled by the *db* gene (Norton 1979) has already been used to breed resistant watermelon cultivars (Norton et al. 1986, 1995) and recessive resistance in melon to powdery mildew has also been reported (McCreight 2001). Efforts are underway to set up mapping populations to locate *gsb-5* and the other *Gsb* loci on the genetic map in melon, although a significant impediment to these studies is a relative lack of DNA polymorphism in the species.

It has been proposed that effects of genetic background and physiological stress may explain why *Gsb-1* alone when used in melon varieties does not confer a high level of resistance (Zhang et al. 1997). All of the resistant PIs in this study have relatively small fruit with thin or nearly absent flesh. A major objective of our current breeding efforts is to combine these GSB resistance genes to recover higher resistance levels in horticulturally acceptable lines with large sweet fruit. There is an indication in our breeding program that resistance gene combinations give significantly higher levels of GSB resistance than single genes alone (J.D. Frantz and M.M. Jahn, unpublished results). An important difficulty that arises as we pyramid these genes is the discrimination among lines that carry several *Gsb* genes. Because resistance has not been identified as race-specific, progeny tests or test crosses are required to confirm segregation of multiple genes and even then it is essentially impossible to identify which of the four dominant genes are segregating. In addition to setting up the populations necessary to identify linked molecular markers, we are currently pursuing parallel introgression programs to produce inbred lines carrying one or two of the *Gsb* genes. The dominant genes can then be brought together in the form of an F_1 hybrid, effectively pyramiding up to all four of the dominant genes. Hybrid breeding programs utilizing recessive *gsb-5* will require introgression into both parents of the F_1 with progeny testing along the way. Marker-assisted selection of these loci would greatly facilitate the creation of resistance gene pyramids by allowing the breeder to discriminate the homozygous allelic state from the heterozygous state and to confirm which resistance genes are present.

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References

Brotman Y, Silberstein L, Kovalski I, Perin C, Dogimont C, Pitrat M, Klingler J, Thompson GA, Perl TR (2002) Resistance gene homologues in melon are linked to genetic loci conferring disease and pest resistance. Theor Appl Genet 104:1055–1063

Buschges R, Hollricher K, Panstruga R, Simons G, Wolter M, Frijters A, van Daelen R, van der Lee T, Diergaarde P, Groenendijk J, Topsch S, Vos P, Salamini F, Schulze-Lefert P (1997) The barley *Mlo* gene: a novel control element of plant pathogen resistance. Cell 88:695–705

Keinath AP, Zitter TA (1998) Resistance to benomyl and thiophanate-methyl in *Didymella bryoniae* from South Carolina and New York. Plant Dis 82:479–484

Keinath AP, Farnham MW, Zitter TA (1995) Morphological, pathological, and genetic differentiation of *Didymella bryoniae* and *Phoma ssp.* isolated from cucurbits. Phytopathology 85:364–369

McCreight JD (2001) Resistance to powdery mildew in melon PI 313970. Hortscience 36:455

McCreight JD, Nerson H, Grumet G (1993) Melon. In: Kalloo G, Bergh BO (eds) Genetic improvement of vegetable crops. Pergamon, New York, pp 267–294

McGrath DJ, Vawdrey L, Walker IO (1993) Resistance to gummy stem blight in muskmelon. Hortscience 28:930–931

Norton JD (1971) Gulfcoast-a sweet cantaloupe for the produce chain store market. Ala Agric Exp Stn Leafl 82

Norton JD (1972) Chilton-a high quality fruit for the commercial market. Ala Agric Exp Stn Leafl 84

Norton JD (1979) Inheritance of resistance to gummy stem blight in watermelon. Hortscience 14:630

Norton JD, Cosper RD (1989) Ac-70-154, a gummy stem blight-resistant muskmelon breeding line. Hortscience 24:709-711

Norton JD, Cosper RD, Smith DA, Rymal KS (1985) AUrora-a high quality disease resistant cantaloupe. Ala Agric Exp Stn Circ 278

Norton JD, Cosper RD, Smith DA, Rymal KS (1986) 'AU-Jubilant' and 'AU-Producer' watermelons. Hortscience 21:1460–1461

Norton JD, Boyhan GE, Smith DA, Abrahams BR (1995) 'AU-Sweet Scarlet' watermelon. Hortscience 30:393–394

Prasad K, Norton JD (1967) Inheritance of resistance to *Mycosphaerella citrullina* in muskmelon. J Am Soc Hortic Sci 91:396–400

Risser G, Banihashemi Z, Davis D (1976) A proposed nomenclature of *Fusarium oxysporum* f. sp. *melonis* races and resistance genes in *Cucumis melo*. Phytopathology 66:1105–1106

Sherf AF, MacNab AA (1986) Vegetable diseases and their control. Wiley-Interscience, New York

Sitterly RW, Keinath AP (1996) Gummy stem blight. In: Zitter TA, Hopkins DL, Thomas C (eds) Compendium of cucurbit diseases. APS Press, St. Paul, Minn., pp 27–28

Sowell G Jr. (1981) Additional sources of resistance to gummy stem blight of muskmelon. Plant Dis 65:253–254

Sowell G Jr., Prasad K, Norton JD (1966) Resistance of *Cucumis melo* introductions to *Mycosphaerella citrullina.* Plant Dis Rep 50:661–663

St. Amand PC, Wehner TC (1995) Eight isolates of *Didymella bryoniae* from geographically diverse areas exhibit variation in virulence but no isolate by cultivar interaction on *Cucumis sativus*. Plant Dis 79:1136–1139

St. Amand PC, Wehner TC (2001) Generation means analysis of leaf and stem resistance to gummy stem blight in cucumber. J Am Soc Hortic Sci 126:95–99

Wang YH, Choi W, Thomas CE, Dean RA (2002) Cloning of disease-resistance homologues in end sequences of BAC clones linked to *Fom-2*, a gene conferring resistance to *Fusarium* wilt in melon (*Cucumis melo* L.). Genome 45:473–480

Zhang Y, Kyle M, Anagnostou K, Zitter TA (1997) Screening melon (*Cucumis melo*) for resistance to gummy stem blight in the greenhouse and field. Hortscience 32:117–121

Zink FW, Gubler WD (1985) Inheritance of resistance in muskmelon *Cucumis melo* to *Fusarium* wilt. J Am Soc Hortic Sci 110:600–604

Zink FW, Gubler WD (1986) Inheritance of resistance to races 0 and 2 of *Fusarium-oxysporum*-f-sp-*melonis* in a gynoecious muskmelon *Cucumis melo*. Plant Dis 70:676–678

Zuniga TL, Zitter TA, Kyle MM (1997) Resistance to gummy stem blight in melon conferred by a single recessive gene. Phytopathology 87:S110

Zuniga TL, Jantz JP, Zitter TA, Jahn MM (1999) Monogenic dominant resistance to gummy stem blight in two melon (*Cucumis melo*) accessions. Plant Dis 83:1105–1107