Pmr1, a Gene for Resistance to Powdery Mildew in Sweet Cherry

James W. Olmstead and Gregory A. Lang¹

Department of Horticulture, Michigan State University, East Lansing, MI 48824-1325

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Abstract. Most sweet cherry (*Prunus avium* L.) cultivars grown commercially in the United States are susceptible to powdery mildew, caused by the fungus *Podosphaera clandestina* (Wall.:Fr.) Lev. Recently, hybrid populations segregating for resistance to powdery mildew were developed by crossing a mildew-resistant sweet cherry selection, PMR-1, with the susceptible cultivars Bing, Rainier, and Van. Although segregation within these populations indicated a single gene was responsible for the powdery mildew resistance conferred by PMR-1, the gene action could not be determined. Therefore, a reciprocal cross between 'Bing' and 'Van' was made to determine the allelic state of the susceptible parents used previously. All progeny (n = 286) from this cross were susceptible to powdery mildew. This information, combined with results from previous segregation data, indicate the powdery mildew resistance gene is inherited in a dominant manner and is present in PMR-1 in the heterozygous allelic state. We have named this gene *Pmr1*. Furthermore, in combination with known pedigree information, we have been able to predict the susceptibility of more than 60 additional commercial and recently released sweet cherry cultivars.

Many sweet cherry cultivars grown commercially are susceptible to powdery mildew, including all leading North American cultivars such as 'Bing', 'Van', 'Rainier', 'Lapins', and 'Sweetheart' (Olmstead et al., 2000, 2001a), as well as 'Stella', the progenitor of all current self-fertile cultivars worldwide. In the arid production regions of the western United States, powdery mildew is the primary disease requiring management in commercial cherry orchards. Multiple applications of fungicide from bloom until after harvest are required for chemical control (Grove and Boal, 1991). Repeated use of demethylation-inhibiting (DMI) fungicides has promoted site-specific development of *P. clandestina* resistance to these fungicides (Grove, 1997).

Toyama et al. (1993) identified a source of genetic resistance to cherry powdery mildew in the sweet cherry selection PMR-1, a self-incompatible (*S_aS_b*) (Hauck et al., 2001) selection from Washington State Univ. Resistance was conferred to progeny from PMR-1 x (susceptible parent) crosses through a single gene (Olmstead et al., 2001b); however, the gene action was not determined. Consequently, while PMR-1 conferred resistance to 50% of the progeny in crosses with 'Bing', 'Van', and 'Rainier', inheritance of resistance with other susceptible parents, like 'Stella' or 'Lapins', was unknown. The objective of this study was to determine the gene action model and allelic state for PMR-1. This information would be useful to suggest or infer the powdery mildew resistance status of several previously unscreened cultivars.

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¹To whom reprint requests should be addressed. E-mail address: langg@msu.edu

Materials and Methods

A gene action model for the resistance gene in PMR-1 could not be determined from the previous segregating progenies because the allelic state of the susceptible parents was not tested. Therefore, we developed further progeny populations from these parents. In Apr. 2000, reciprocal crosses were made at Washington State University's Roza Experimental Orchards (Prosser, Wash.) between two highly susceptible cultivars, 'Bing' and 'Van', used previously to develop segregating progeny populations with PMR-1 (Olmstead et al., 2001b). About 2 weeks prior to anthesis in the orchard, pollen was collected (Fogle, 1975) from cut shoots of each cultivar, forced in the laboratory, and stored at 0 °C. Flowers on orchard trees were emasculated by hand just prior to controlled pollination. To obtain segregating progenies of ~100 individuals for each reciprocal cross, ~2000 hand pollinations were made with each maternal parent. Hand-pollinated flowers were protected from insect visitation and bird damage by cages covered in cheesecloth, and the resulting fruits were harvested at full maturity (July 2000). Pits were extracted immediately, cleaned thoroughly, surface disinfested for 1 min in 0.5% sodium hypochlorite, and allowed to soak in sterile distilled water for 24 h. After soaking, the endocarps were cracked, and multiple groups of 50 seeds from each cross were placed in polyethylene bags containing ~700 g of moist, sterile sand mixed with 5 g of fungicide powder (Ferbam; FMC Corp., Philadelphia).

Bags of seed were stratified at Michigan State Univ. at 4 °C in the dark for 5 months (Galletta, 1983), by which time radical elongation had begun for most seeds. After stratification, seeds were planted individually in

10-cm³ (0.9 L) plastic pots containing a 75% peat : 25% vermiculite pasteurized greenhouse medium (Baccto; Michigan Peat Co., Houston) and germinated in a greenhouse maintained at 20 to 25 °C. Seedlings were fertilized once with 14N–14P–14K Osmocote slow-release fertilizer (Miracle Gro; The Scotts Co., Marysville, Ohio) and watered as needed. In Aug. 2001, the seedlings were screened for colonization by powdery mildew during a naturally occurring infection in the greenhouse. Visual observations were subsequently confirmed by viewing representative leaves from each of the progeny under a dissecting microscope (Wild Heerbruug, Heerbruug, Switzerland) at 20×.

Results and Discussion

All progeny individuals from the 'Bing' x 'Van' and 'Van' x 'Bing' (94 and 192 progeny, respectively) reciprocal crosses were susceptible to powdery mildew. This result and those of our previous segregating progenies indicate that powdery mildew resistance is conferred following a dominant gene action model, and is heterozygous in PMR-1. We named this gene *Pmr1*, and present the allelic state in the three cultivars from our previous direct crosses (Table 1). Because mildew resistance conferred by *Pmr1* is dominant and PMR-1 is heterozygous for *Pmr1*, rather than homozygous recessive, PMR-1 as a parent should yield 50% or more mildew resistant progeny, regardless of cross-parent allelic state.

If the simple *Pmr1* model for genetic powdery mildew resistance is valid for other sweet cherry genotypes, breeding performed with two susceptible parents will yield susceptible progeny, as we observed (Olmstead et al., 2000, 2001a). In addition to the 12 cultivars screened previously for powdery mildew susceptibility (Olmstead et al., 2000), 35 cultivar accessions in the National Research Support Project #5 (NRSP5, Prosser, Wash.) germplasm collection were rated as susceptible to powdery mildew colonization in 1998 and 1999 (unpublished data) (Table 2), indicating a homozygous recessive allelic state for *Pmr1*. By using known pedigree information, we also are able to propose that several other breeding lines and commercial or recently released cultivars are homozygous recessive for *Pmr1* and therefore susceptible to powdery mildew (Table 2). Of particular interest in this list are JI 2420 and JI 2434, which are the source of nearly all self-fertile sweet cherry cultivars. As self-fertility has been a major breeding priority in most sweet cherry breeding programs worldwide (Brozik, 1996;

Table 1. The allelic state of *Pmr1* in the sweet cherry breeding selection PMR-1 and in several commercially important cultivars, determined by segregating progeny populations from direct crosses.

Cultivar	Allelic state	<i>Pmr1</i> allelic state	Mildew reaction
PMR-1	<i>Rr</i>	<i>PMR1pmr1</i>	Resistant
Bing	<i>rr</i>	<i>pmr1pmr1</i>	Susceptible
Van	<i>rr</i>	<i>pmr1pmr1</i>	Susceptible
Rainier	<i>rr</i>	<i>pmr1pmr1</i>	Susceptible

Table 2. Method of determination of proposed *pmr1pmr1* allelic state in 68 sweet cherry cultivars known or predicted to be susceptible to powdery mildew.

Cultivar	Field ^z obs.	Hybrid ^y cross	Pedigree ^x analysis	Cultivar	Field obs.	Hybrid cross	Pedigree analysis
Angela	X			Santina			X
Bing	X	X	X	Saylor	X		
Black Republican	X			Schmidt	X		
Black Tartarian	X			Schn. Spate Knorpel.	X		
Black Giant	X			Seneca	X		
Sumpaca Celeste			X	Skeena			X
Chinook	X		X	Somerset			X
Columbia	X			Sumleta Sonata			X
Corum	X			Sparkle	X		
Sumnue Cristalina			X	Spalding	X		
Ebony	X		X	Star	X		
Emperor Francis	X			Stella	X		X
Gil Peck	X			Sue			X
JI 2420			X	Summit	X		X
JI 2434			X	Sunburst	X		X
Kristin	X		X	Sweet Ann	X		
LaLa Star			X	Sweet September	X		
Lambert	X			Sweetheart			X
Lamida	X			Sylvia	X		X
Lapins	X		X	Symphony			X
Larian	X			Tehravivee			X
Liberty Bell			X	Tieton	X		
Lyons	X			Ulster	X		X
Merton Biggareau	X			Utah Giant	X		
Merton Heart	X			Valera	X		X
Napoleon	X			Van	X	X	
Newstar			X	Vandalay			X
Olympus			X	Vega	X		
Rainier	X	X	X	Velvet	X		
Regina	X			Vic	X		X
Salmo	X		X	Vista	X		
Sam	X			White Gold			X
Sumste Samba			X	Windsor	X		
Sandra Rose			X	Yellow Glass	X		

^zField observations of powdery mildew susceptibility at the NRSP5 germplasm collection (Prosser, Wash., 1998, 1999).

^yOlmstead et al., 2001b.

^xSusceptibility determined by parents either confirmed homozygous recessive for *Pmr1* by field observations or hybrid crosses.

Granger, 1998; Kappel and Lane, 1998; Lang et al., 1998; Sansavini et al., 1998; Saunier, 1996), most new cultivar releases or those currently in selection are likely to be susceptible to powdery mildew. This also may be of interest to the small but growing number of cherry growers attempting to use organic production methods, particularly those in the arid regions of the western United States where powdery mildew is the primary disease needing control.

In summary, PMR-1 is valuable for devel-

oping powdery mildew resistant sweet cherry cultivars. We have not identified any homozygous resistant genotypes that would confer resistance to all progeny. However, further genetic characterization of other recently identified resistant genotypes (Olmstead et al., 2000) is continuing, as well as attempts to develop selections that would be putatively homozygous dominant for *Pmr1*, selections that combine self-fertility with a dominant *Pmr1* allele, and resistant selections with fruit quality suitable for commercial orchard pro-

duction. We have recently made seven elite selections having commercial fruit quality potential from our powdery mildew resistant breeding populations, which will undergo wider testing and evaluation in 2003.

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