Pierce's disease: Incidence, spread and control

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Introduction

Xylella fastidiosa is the bacterial agent of a number of xylemlimited diseases, which affect many crop, ornamental and native plant species. It is found throughout the warmer parts of North and South America and is most well known for causing Pierce's disease (PD) of grapevine and citrus variegated chlorosis (CVC). The bacterium is spread from infected native and cultivated plant species by xylem-feeding insects, most notably by leafhoppers (Homoptera:Cicdellidae) and spittlebugs (Homopter:Cercopidae). The sharpshooter group of leafhoppers are the most common vectors of X. fastidiosa in the United States. While the insect is feeding in host plant xylem, the bacterium is inoculated into the vessels. If the host plant is susceptible, the bacterium reproduces rapidly and moves both with and against the transpirational stream to colonize the xylem. It also is able to move laterally through xylem vessel pores in some manner that is not yet understood. Disease results when the vessels are occluded by bacterial growth and from induced gums and tyloses associated with PD infection (Fry and Milholland 1990; Hopkins et al. 1974). The symptoms appear related to water stress (Goodwin et al. 1988a; Goodwin et al. 1988b) with marginal leaf-burn, leaf blade defoliation ('matchsticks') and uneven bark maturation leading to 'green islands' on developing canes (Stevenson et al. 2005). Fruit clusters typically raisin, and are left un-harvested, and vine death occurs as the disease spreads downward into the trunk, usually within a few years (Hopkins and Purcell 2002). A complete listing of literature related to PD can be found at the following website (http://www.cnr.berkeley.edu/xylella/refs/).

Vectors

Purcell (1989) reviewed the insect vectors of *X. fastidiosa*. One of the earliest studies was by Hewitt et al. (1945) who examined 45 species of leafhoppers and found four species capable of vectoring PD including the green sharpshooter (*Draeculacephala minerva* Ball), the red-headed sharpshooter (*Carneocephala fulgida* Nott), and the blue-green sharpshooter (*Graphocephala atropunctata* Signoret). This study also found an association with alfalfa dwarf, now known to be caused by *X. fastidiosa*, when they observed PD affected vines in close proximity to alfalfa fields and disease symptoms and vectors on both crops. Mircetich et al. (1976) found that the red-headed sharpshooter could transmit *X. fastidiosa* from infected almond trees to healthy trees and to Carignane grapevines.

Freitag (1951) noted that the green sharpshooter, the redheaded sharpshooter, and the blue-green sharpshooter were able to vector *X. fastidiosa* to a wide range of plant species including johnson grass, bermuda grass, rye grass, timothy, canna, toyon, scotch broom, hubam clover, alsike clover, crimson clover, red clover, ladino clover, and species of *Artmenisia, Coprosoma, Godetia, Hedera, Lonicera, Oenothera, Sambucus,* and *Symphoricarpos.* Hopkins and Mollenhauer (1974b) used enzyme-linked immunosorbent assay (ELISA), fluorescence microscopy, and direct culturing to test for X. fastidiosa in Florida and found the bacterium in American elder, Virginia creeper, peppervine, American beautyberry, and blackberry. Raju et al. (1983) used ELISA to detect X. fastidiosa in wild strawberry, miner's lettuce, Himalayan blackberry, and periwinkle. Buzombo et al. (2006) tested 96 native and ornamental plants surrounding a Texas vineyard and detected X. fastidiosa in Yaupon holly, Vitis mustangensis (a resistant species), American sycamore, southern dewberry, frogfruit, Japanese honeysuckle and crape myrtle. In addition to previously known grape, almond and oleander reservoirs, Costa et al. (2004) detected X. fastidiosa in Spanish broom and Brassica species surrounding a Temecula (near San Diego, California) vineyard. Almond, Spanish broom and wild mustard isolates of X. fastidiosa clustered with strains known to be pathogenic to grape implicating them as PD hosts. Interestingly, these investigators did not find strains of X. fastidiosa virulent to grapevine in nearby citrus orchards; even though citrus is a favored feeding host of known PD vectors. Eucalyptus species have been found to be excellent hosts of the glassy-winged sharpshooter, but to date they have not been found to be hosts of X. fastidiosa (Costa et al. 2004). Given this evidence it is clear that the host ranges of both the vectors of PD and X. fastidiosa are very large and cross many plant families.

Pierce's disease

Pierce's disease was first discovered in southern California near where Disneyland now resides and where Newton B. Pierce (1892) described it as Anaheim Disease. Pierce was brought to California to study this epidemic, which quickly wiped out tens of thousands of acres of vineyard and was partly responsible for the movement of viticulture to the northern part of the State. The causal agent was thought to be a virus until 1973, when it was described as a rickettsia-like bacterium (Goheen et al. 1973, Hopkins and Mollenhauer 1973). It has since been described as a gram-negative bacteria related to Xanthomonas, but belonging a new genus and species Xylella fastidiosa (Wells et al. 1987). Strains of X. fastidiosa infect a wide range of cultivated plants. In Brazil, citrus variegated chlorosis (CVC) has caused millions of dollars in loses for the citrus industry (Chang et al. 1993). Coffee leaf scorch is also caused by X. fastidiosa and has been detected in Brazil and, more recently, in Costa Rica (Rodriguez et al. 2001). Other important examples include mulberry leaf scorch, almond leaf scorch (Davis et al. 1980), alfalfa dwarf (Goheen et al. 1973), and phony peach disease (Davis et al. 1981).

This tremendous diversity of susceptible native and cultivated host plants (Cooksey 2004) inspired research designed to evaluate genetic diversity and cross-virulence among different strains of the bacteria. Hendson et al. (2001) tested 46 strains isolated from seven different host species and found that isolates from grape formed a genetically similar group. Chen et al. (2002) suggested that the CVC and PD strains of *X. fastidiosa* diverged recently because the bacterium is native to the Americas and citrus was introduced from Asia. Although the CVC strains are genetically distinct from PD strains, the citrus strains have caused symptoms in grapes (Li et al. 2002). Strains of *X. fastidiosa* have not been compared among native grape hosts, but variation would be expected to be minimal compared to that across cultivated crop hosts or native plant hosts. Cross inoculation studies with strains from the genetically divergent *V. vinifera* and *Muscadinia rotundifolia* have successful infected both hosts (Hopkins 1984).

Pierce's disease is widely spread and severely impacts viticulture in the southeastern portion of the United States where V. vinifera cultivars are most successfully grown in screenhouses to prevent vectoring of X. fastidiosa by sharpshooter insects. Pierce's disease is also found across warmer regions of the western United States where its incidence is more sporadic and disease outbreaks are episodic and somehow related to the proper timing of environmental conditions (most often high rainfall to encourage native vegetation), vector population increases, and the expansion of bacterial populations in native host plants. There have been notable outbreaks of PD that have caused extensive damage in Texas and Arizona (Kamas et al. 2000, Buzoumbo et al. 2006), and most dramatically in California. The last large outbreak in California occurred in the late 1990s and destroyed thousands of acres in the Temecula Region, near San Diego, California, and it coincided with a large increase in PD incidence in Napa and Sonoma County vineyards in northern California, which also caused millions of dollars in damage. This California outbreak led to a State and Federal program, which coordinates control and research efforts on Pierce's disease (http:// www.cdfa.ca.gov/pdcp/).

The glassy-winged sharpshooter introduction and spread

The outbreak of PD in Temecula was linked to the introduction of a new vector, the glassy-winged sharpshooter (Homalodisca coagulata) that was originally found in the citrus orchards and ornamental plant nurseries of southern California (Blua et al. 1999) after introduction from the southeastern United States or northeastern Mexico (Hoddle 2004). The glassy-winged sharpshooter did not spread X. fastidiosa or cause appreciable damage from feeding in citrus or nursery plantings and built to very high levels in citrus where the term 'sharpshooter rain' was coined to describe their abundant extraction and excretion of xylem sap while feeding (http://ceventura.ucdavis.edu/IPM/ The_GWSS_A_serious_new_PD_vector_for_California_ Vineyards.htm). This sharpshooter spread in citrus orchards and multiplied in orchards adjacent to Temecula vineyards. The native plants of this high desert region do not support much X. fastidiosa, but eventually the glassy-winged sharpshooters found a source of X. fastidiosa and began spreading it into vineyards.

This insect has many characteristics that make it a more robust vector of PD (Blua and Morgan 2003). The glassy-winged sharpshooter can vector PD from vine to vine and feeds both on succulent shoot tips and mature woody canes. The native sharpshooter vectors only feed on succulent shoot tips, which limits the season during which they can spread PD, and they rarely spread PD from vine to vine, instead spreading it from native hosts into vineyards. Thus, the glassy-winged sharpshooter spreads PD much more effectively and results in exponential increases of dying vines (Perring et al. 2001). Although PD caused major losses in the Temecula Valley in the late 1990s, the real threat created by the introduction and spread of the glassy-winged sharpshooter lies to the north, in the Central Valley with its extensive wine, table and raisin grape vineyards, and in the premium vineyards of the North Coast counties. While the spread of the glassy-winged sharpshooter has been slower than expected (due to intensive insecticide applications and the release of predatory wasps), there was a glassywinged sharpshooter related PD outbreak in Kern County in 2000 (Hopkins and Purcell 2002) indicating that the insect can expand its range in California.

Since its discovery in southern California citrus orchards, the glassy-winged sharpshooter has moved north into the Central Valley counties of Kern and Tulare, and smaller populations are established in Fresno, Santa Clara, Solano and Sacramento County (Hopkins and Purcell 2002). These populations present a large threat to the premium winegrowing counties of Santa Barbara, Santa Cruz, Napa, Sonoma and Mendocino where native sharpshooters currently vector PD. If the glassy-winged sharpshooter reaches these counties, PD will likely be spread much more widely and beyond what are localized 'hot spots'.

Climatic effects on PD

Pierce's disease pressure has historically been most severe in the southeastern United States. This is likely due a combination of high vector populations and a much longer growing season (Hopkins and Purcell 2002). The longer growing season also allows more time for X. fastidiosa populations to build up in vines which increases the chance of vine to vine transmission, and allows more time for X. fastidiosa to spread more thoroughly into host vines so that there is less chance that all of the infected tissue is removed with pruning. Winter temperatures in colder areas limit PD by severely impacting bacterial survival (Purcell 1980). Temperatures of 12°C and below negatively impact X. fastidiosa colony growth in vitro, and chilling host plants to 5°C can reduce bacterial concentrations in live tissue (Feil and Purcell 2001). Thus, PD is limited by cold winter temperatures that kill X. fastidiosa colonies residing in canes and trunks. This phenomenon is well-known and used in the southeastern United States where PD susceptible peaches and grapevines can be cultivated on colder hill tops, but not in warmer valleys. In California, PD is very rare north of southern Mendocino County, yet the vectors and host plants are found north through Oregon and Washington.

Climatic modeling, CLIMEX, has been used to predict where the glassy-winged sharpshooter could spread (Hoddle 2004). It predicted that this insect could survive in northern California, but not Oregon or Washington. This model was also used to verify that suitable conditions for the glassy-winged sharpshooter and *X. fastidiosa* exist in many of the areas where *V. vinifera* grapes are grown. It predicted that cold winter temperatures would prevent PD from establishing in much of France, Italy and northern and central Spain. However, it also predicted that *X. fastidiosa* would not survive in the Balkans, yet it has been detected in Kosovo (Berisha et al. 1998).

Control - limiting the vector

Efforts to eradicate the glassy-winged sharpshooter have utilized a range of conventional insecticides including pyrethroids, carbamates, organophosphates and neonicotinoids (Redak and Bethke 2003a, Redak and Bethke 2003b). The most effective of these, the systemic neonicotinoid imidacloprid is now the insecticide of choice and has demonstrated excellent control (Stone-Smith et al. 2005, Toscano and Byrne 2005, Toscano and Gispert 2005). Byrne and Toscano (2006) recommend imidacloprid application in early June when the glassy-winged sharpshooter is most likely to move into vineyards from nearby citrus groves or ornamental plantings.

Redak and Bethke (2003a) tested a range of low-impact or

organic pesticides to control glassy-winged sharpshooter, but none of the treatments achieved the level of success found with imidacloprid. Blua et al. (2005) evaluated five-meter-high screen barriers to prevent the movement of the glassy-winged sharpshooter into vineyards. These screens seemed to deter or prevent the sharpshooters from entering ornamental plant nurseries but were judged to be too expensive for general vineyard use.

The culmination of efforts to develop effective parasitoids of glassy-winged sharpshooters led to the release of mymarid parasitoid wasps in southern California in a joint effort between the University of California and the California Department of Food and Agriculture. These species included *Gonatocerus ashmeadi* (native to SEUS and northeastern MX), *G. triguttatus* (MX and southeast Texas), *G. morrilli* (southern CA) and *G. fasciatus* (southeast and midwest United States). These wasps have established in southern California and are actively parasitizing glassy-winged sharpshooter eggs, but concern remains that effectiveness of parasitoids will not be great enough to prevent further movement of the glassy-winged sharpshooter (Pilkington et al. 2006).

Control - limiting X. fastidiosa

There have also been efforts to limit PD by reducing or eliminating *X. fastidiosa* with antibiotics including tetracyclines and aminoglycosides (Kuzina et al. 2006). However, introducing these compounds into xylem vessels on a commercial scale is impractical and maintaining effective xylem titers would require repeated inoculations. Hopkins (2005) discovered a strain of *X. fastidiosa* (EB92-1) from elderberry that seems to induce systemic acquired resistance in Cabernet Sauvignon over eight years of field testing and natural infection in Florida. Trials with this strain will soon be established in California vineyards to test its efficacy and commercial potential.

Control – breeding for PD resistance

If the glassy-winged sharpshooter does spread more widely in California due to climatic change, disregard or collapse of quarantine restrictions, failure of predatory species, or the development of resistance to imidacloprid and neonicotinoid insecticides, the utilization of PD resistant cultivars would allow vineyards to be productive in the expanded 'hot-spot' areas. Resistant cultivars would also be greatly beneficial to growers in other regions were PD limits viticulture.

When European settlers first invaded North America in the 1500s they noticed the abundance of native grape species and concluded that vineyards would thrive in both the northern and southern parts of the 'New World'. Efforts to establish *V. vinifera* failed due to a combination of fungal diseases (powdery and downy mildew, and black rot) and grape phylloxera that now plague viticulture around the world. These establishment efforts also failed because of cold winter temperatures in the northeast and PD in the southeast. These biotic and abiotic problems led to grape breeding efforts that produced hundreds of hybrids of *V. vinifera* and native American species adapted to the diseases and climatic conditions of the United States. These breeding efforts have continued to the present with the goal of combining the wine and fruit quality of *V. vinifera* with the resistances and tolerances of the American grape species.

Grape species native to the southeastern United States have always been considered the most resistant to PD. These species thrive in areas of high PD pressure where *V. vinifera* plantings fail. Longterm field trial data from Mississippi highlighted the PD resistance of some grape species (Loomis 1958). Species from the southeastern United States such as *Muscadinia rotundifolia* (including ssp. *popenoii* and *munsoniana*) and *V. cinerea* forms persisted for many years. Selections of *V. arizonica*, native to the southwestern United States and northern Mexico where there is strong PD pressure, also appear resistant (Krivanek and Walker 2005, Krivanek et al. 2005, 2006, et al. Riaz et al. 2008). Species from PD-free areas such as the northeastern United States (*V. labrusca*) or Eurasia (*V. vinifera*) die quickly.

Different cultivars of *V. vinifera* are reported to vary in their susceptibility to PD, however all eventually succumb to the disease. Raju and Goheen (1981) inoculated 25 *V. vinifera* cultivars and ranked their susceptibility based on ELISA quantified *X. fastidiosa* levels. They stated that Colombard and Chardonnay were among the most susceptible and that Chenin blanc and Silvaner were the least affected. However, none of these cultivars survives after inoculation under vineyard conditions. Varieties do succumb over varying time lengths of time, but the planting of 'less' susceptible *V. vinifera* varieties has not proven to be a viable control strategy. Scattered vines in an affected vineyard may also survive for long periods of time, but once they are effectively fed upon and inoculated they also succumb.

The use of genetic engineering to produce PD resistant cultivars is limited by the availability of genes and the limited public acceptance of this approach to plant improvement. However, several efforts have been attempted. Aguero et al. (2005) transformed susceptible V. vinifera cultivars with a polygalacturonase-inhibiting protein from pear (pPGIP) with the intent of limiting X. fastidiosa's cellwall degrading capability. Pierce's disease expression was delayed and reduced with lower bacterial levels in transformed grapevines and pPGIP was found in untransformed scions grafted on to transgenic rootstock. This effect was promising but much more work is needed to make genetically modified PD resistant grapes a reality. Efforts to locate and utilize PD resistance genes in grape species may be a more effective strategy for genetic engineering. These efforts may also help overcome public suspicion and opposition to genetic engineering in grape since the classical breeding with these genes is also possible and genetic engineering would represent a more targeted means of breeding PD resistance into a susceptible cultivar. However, these genes need to be isolated first and proven to be effective.

Breeders have been developing PD resistant hybrids for many years based on resistance from a number of Vitis and Muscadinia species. Resistant cultivars have been developed in public (Dunstan 1965, Loomis 1958, Mortensen 1977, 1983a, 1983b, Olmo 1986, Overcash 1981, 1982, among others) and private (Barrett, Bloodworth and others) breeding programs across the United States. These cultivars have high PD resistance, but relatively low fruit and wine quality relative to V. vinifera varieties. In the southeastern United States, grapes must also resist downy and powdery mildew, black rot and anthracnose, which have as great an impact on viticulture as PD does. These diseases are not found in California, which allows breeders to incorporate a greater percentage of high quality, but extremely disease susceptible V. vinifera into their breeding efforts and enables the production of much higher quality PD resistant cultivars in a shorter time span. The Walker lab has characterized and employed a wide range of PD resistant selections from breeders in the southeastern U.S.; from forms of V. arizonica collected in northern Mexico by H.P. Olmo in the 1960s, and from several V. vinifera × M. rotundifolia hybrid winegrape types with limited fertility. These breeding efforts are producing selections with high fruit quality and excellent PD resistance.

Breeding efforts to combat PD are dependent upon germplasm resources and the nature of their resistance. Resistance to PD would

be expected to be strongest and most common in its native range, and the species of the southeastern United States have been most commonly used in PD resistance programs (Hopkins et al. 1974a, Mortensen et al. 1977). These species include accessions of *M. rotundifolia, V. aestivalis, V. champinii, V. candicans, V. cordifolia, V. shuttleworthii, V. simpsonii* and *V. smalliana* (Hopkins et al. 1974, Mortensen et al. 1977, Krivanek and Walker 2005, Ruel and Walker 2006). The resistance of *M. rotundifolia* cultivars and accessions varies based on their geographic origin (Hopkins et al. 1974, Ruel and Walker 2006) and resistance was correlated with mean annual minimum temperature with selections from colder sites having less resistance. However, all *M. rotundifolia* selections were much more resistant than the tested *V. vinifera* cultivars (Ruel and Walker 2006). Such studies have not been carried out with other species that range across PD's acknowledged latitudinal boundary.

Although strong resistance to PD exists in American species, crosses to V. vinifera produce few resistant offspring due to the quantitative inheritance and the complicated genetics of resistance to PD (Walker and Tenscher 2005). Mortensen (1968) hypothesized that resistance in progeny of V. simpsonii, V. smalliana, and V. shuttleworthii is controlled by dominant resistance alleles at three different loci in these backgrounds. Progeny with a dominant allele at each locus would be resistant, thus few resistant progeny are produced in these crosses where the loci segregate independently. Breeding for PD resistance with a resistance source possessing a single dominant resistant gene would greatly accelerate breeding efforts and allow the backcrossing to elite V. vinifera cultivars with the expectation of 50% resistant progeny in each generation. In 1960, H.P. Olmo collected Vitis species across northern and central Mexico (Riaz et al. 2007). Many of these accessions were forms of V. arizonica that had been introgressed with several other southern and southwestern species, and many were later found to be highly resistant to PD (Krivanek et al. 2005a, 2005b, Riaz et al. 2007). Several of these accessions have been used to map resistance to PD and to produce PD resistant wine grapes in the Walker lab at the University of California, Davis, and table and raisin grapes in a collaborative breeding program between David Ramming at the United States Department of Food and Agriculture - Agricultural Research Service in Parlier, California.

Breeding efficiency can be greatly improved with the use of DNA markers closely associated with traits of interested. This process depends on well-characterized seedling populations with precise phenotypic data and the availability of accurate and reproducible genetic markers. Microsatellite or simple sequence repeat (SSR) markers are ideal for genetic mapping efforts because they are polymorphic, have a high level of heterozygosity, and are reliable and reproducible. They have been used to create genetic maps of *V. vinifera* (Adam-Blondon et al. 2004, Riaz et al. 2004) and for the mapping and study of PD resistance in populations derived from *V. arizonica* (Riaz et al. 2006).

Genetic mapping efforts in the Walker lab began with the evaluation of the 9621 population, a cross of two half-sibling *V. rupestris* \times *V. arizonica* type hybrids, D8909-15 and F8909-17 (Riaz et al. 2007). D8909-15 is the offspring of *V. rupestris* A. de Serres \times b42-26 *V. arizonica/girdiana*. This mapping population segregates for resistance to PD and the dagger nematode, *Xiphinema index*, and both traits are inherited as single dominant genes. The first genetic map was created by Doucleff et al. (2004) and was based primarily on AFLP (amplified fragment length polymorphism) markers. This map was followed by a map based on SSR markers, which located the PD resistance locus on chromosome 14 and named it *PdR1* (Krivanek et al. 2006). These mapping efforts were

further refined by Riaz et al. (2006) who located *PdR1* between the SSR markers VMCNg3h8 and VVIN64, which are 4.34cM and 2.78 cM from the resistance locus.

Fine scale mapping efforts found that the PdRI resistance allele in F8909-17 (PdRIa), the male parent of the 9621 population, represents only one of two alleles from the homozygous resistant V. arizonica/candicans b43-17 grandparent. The other PdRIallele (PdR1b) is present in F8909-08, a full-sibling of F8909-17 generated from the original V. rupestris A. de Serres × b43-17 V. arizonica/candicans cross (Riaz et al. 2008). Efforts are now underway to physically position the alleles of PdR1 on bacterial artificial chromosome libraries that have been developed from b43-17, and to determine whether PdR1a and PdR1b confer equivalent PD resistance to progeny.

Wine grape breeding efforts in the Walker lab have a number of objectives that are being pursued at the same time. The first objective is to utilize the unique source of PD resistance found in the V. arizonica selection b43-17 and fully understand the mode of its inheritance as the single dominant resistance gene, PdR1 (Krivanek et al. 2005a, 2005b, Krivanek et al. 2006, Riaz et al. 2006, 2007, 2008). A number of other resistance sources from the southeastern United States are also being used. Screening and evaluation of these resistance sources confirms past studies (Mortensen et al. 1968), and indicates that multiple genes control resistance in these resistance sources and that inheritance is complex and quantitative. It might be possible to locate these quantitative trait loci (QTLs) on genetic maps and finally use markers that are tightly linked to the QTLs in marker-assisted selection (MAS) to introduce multiple levels of PD resistance, broadening the base of resistance to prevent its breakdown. Such efforts are underway with David Ramming identify these QTLs and introgress these resistance sources into high quality seedless table grapes.

SSR-based markers linked to *PdR1* are being actively used in the Walker breeding program to introgress PD resistance from b43-17 into high quality wine grapes. Many thousands of seedlings have been screened for PdR1 using MAS over the last three years and the results used to greatly accelerate the breeding process. The Walker lab has been able to germinate seed in the late Fall and plant early in the following Spring. Aggressive training practices then result in about a 2-meter shoot that is attached to a 1.25 m fruiting wire as a short cane in the dormant season. More than 50% of these shoots flower the next Spring and the MAS results are used to identify parents for the next generation of crosses. The goal of this effort is to achieve seedling populations with 94 to 97% high quality V. vinifera parentage and PD resistance from PdR1 (Walker and Tenscher 2007). The following cultivars have been used: Alicante Bouschet, Aligote, Barbera, Cabernet Franc, Cabernet Sauvignon, Carignane, Chardonnay, Durif, Grenache, Listan, Merlot, Monbadon, Pinot Noir, Sauvignon blanc, Semillon, Symphony, Teinturier du Cher, Tempranillo, and Zinfandel. In the next phase, resistance from powdery mildew from a number of backgrounds including M. rotundifolia, French hybrids, Chinese species and American species will be incorporated into PD resistant selections, with MAS.

The Walker lab also made wines at the 87.5% *V. vinifera* level with fruit from three vine replicates of two-year-old vines in 2007 to demonstrate that *V. vinifera* quality was being obtained at this relatively early stage of back-crossing. This 'micro'-vinification was compared to wines made at the same scale from Cabernet Sauvignon, Pinot noir and two standard PD resistant cultivars from the southeastern United States – Lenoir and Midsouth, all harvested from the UC Davis vineyards. The results of enological analysis and a tasting panel indicate good progress is being made (Tables 1 and 2).

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 Table 1. Analytical evaluation of reference varieties and advanced selections with the PdR1 resistance source. Diglucoside anthocyanins were detected in Midsouth and Lenoir. The 501 selections are 50% Syrah and the 502 selections are 50% Chardonnay. Juice analysis courtesy of ETS Laboratories, St. Helena, CA.

Genotype	L-malic acid (g/L)	°Brix	potassium (mg/L)	рН	TA (g/100 mL)	YAN (mg/L (as N)	catechin (mg/L)	tannin (mg/L)	Total antho- cyanins (mg/L)
Cab. Sauvignon	2.19	24.9	2460	3.65	0.62	227	59	250	404
Pinot Noir	2.43	26.5	2190	3.83	0.49	279	321	842	568
0501-12	4.20	29.4	2900	3.87	0.68	420	88	802	979
0502-01	2.90	25.9	2530	3.77	0.61	301	91	564	380
0502-10	4.92	23.7	2220	3.48	0.85	301	87	588	845
Lenoir	4.32	26.9	2920	3.67	0.75	164	195	341	1801
Midsouth	4.60	18.2	2220	3.49	0.81	278	32	230	971

Table 2. Tasting results of 2007 vintage wine rated on a 1 = poor to 5 = very good scale. There were nine tasters from the faculty and staff at UCD.

Variety/ Selection	Group Total	Low Score	High Score	Descriptors: color; aroma; flavor and texture
0501-12	33.5	2	4.5	dark purple; grapy, smoky, blueberry; warm, chocolate, rich, balanced, structured
0502-07	32	2.5	4	dark purple; grapy, earthy-smoky; rich, good structure & balance
Cabernet Sauvignon	27	2	5	red with hint of brown; herbal, weedy, bell pepper; warm, flat, with good tannin structure
0502-10	27	1	5	dark purple; bright red fruit, odd herbal-plastic note; non-vinifera flavor, okay structure
Lenoir	26	2	4.5	dark purple-black; blackberry, dried fig, slightly weedy, odd non-vinifera herbal character; lacks structure
0502-01	24	1.5	4	light-medium red with hint of brown; candy, red fruit; moderate body, slightly earthy, oxidized
Pinot Noir	20	1	3.5	light pink-red; simple red fruits; odd vegetal, cherry, light
Midsouth	18.5	1	3	red with slight brown edge; vegetal, oxidized, simple

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