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PLANT DISEASE FEATURE

Rotting Grapes Don't Improve with Age: Cluster Rot Disease Complexes, Management, and Future Prospects

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Abstract

Cluster rots can be devastating to grape production around the world. There are several late-season rots that can affect grape berries, including Botrytis bunch rot, sour rot, black rot, Phomopsis fruit rot, bitter rot, and ripe rot. Tight-clustered varieties such as 'Pinot gris', 'Pinot noir', and 'Vignoles' are particularly susceptible to cluster rots. Symptoms or signs for these rots range from discolored berries or gray-brown sporulation in Botrytis bunch rot to sour rot, which smells distinctly of vinegar due to the presence of acetic acid bacteria. This review discusses the common symptoms and disease cycles of these different cluster rots. It also includes useful updates on disease diagnostics and management practices, including cultural practices in commercial vineyards and future prospects for disease management. By understanding what drives the development of different cluster rots, researchers will be able to identify new avenues for research to control these critical pathogens.

Keywords: bitter rot, black rot, *Botrytis cinerea*, bunch rots, cluster rots, grapevine, pests, *Phomopsis viticola*, ripe rot, sour rot

Grape cluster rots are significant diseases of grapevines that have plagued growers for centuries (Steel et al. 2013). In the 1880s, black rot disease swept across the European countryside, ravaging vineyards in multiple growing regions. In its wake, the disease left innumerable small, hard, shriveled, mummified berry husks and caused the near collapse of the continental wine industry (Barnes 1979; Wilcox et al. 2015b). Today, in the United States, these grape rots continue to cost millions of dollars in yield losses to growers each year and extensive input costs are needed to manage them (Madden et al. 2017). Although there is abundant knowledge about managing certain rots such as Phomopsis fruit rot, black rot, and Botrytis bunch rot, other cluster rot disease complexes are still mysterious and understudied. Specifically, there is limited information about the particular pathogens at play, the environmental conditions conducive for disease, and the underlying mechanisms of pathogenicity (Hall et al. 2018a). Many cluster rots need interactions between multiple pathogenic microbes, susceptible cultivars, vectoring pests, and local weather conditions, creating a complex relationship. Understanding the interactions between these factors, or grape cluster rot disease ecology, is critical for creating and implementing effective field management practices, especially during the critical infection period (Fig. 1). Growers and researchers alike are interested in learning more about these disease complexes and strive to develop best practices to manage them in the field. This review article highlights some of the most common grape cluster rot complexes found in the United States. We describe the rots, starting with emergent early-season diseases (black rot, Phomopsis fruit rot, and bitter rot) followed by late-season diseases such as Botrytis bunch rot, ripe rot, and sour rot. We describe important insect pests and those that vector microbial pathogens, discuss current molecular diagnostics, and outline best management practices. Finally, we underscore important field management practices and discuss future prospects for understanding and managing resistance to grape cluster rots.

Black rot. Black rot on grapevine is caused by the fungal pathogen *Guignardia bidwellii* (Ellis) Viala & Ravaz (anamorph: *Phyllosticta ampelicida* (Engelmann) van der Aa 1861). This grape pathogen is native to North America (Hoffman and Wilcox 2002) but has spread to Europe and South America since the end of the 19th century (Molitor and Beyer 2014). The disease is still expanding to new territories; for example, the first observation of black rot in China occurred in 2013 on Kyoho grape (*V. labruscana* hybrid) (Cui et al. 2015). As of 2015, *G. bidwellii* is considered a quarantine pathogen in Australia (Sosnowski et al. 2012). Crop losses between 5 and 80%, and up to 100%, can result without proper management of this disease (Molitor and Beyer 2014; Rinaldi et al. 2013; Wilcox et al. 2015b). In central Europe, disease severity of 0.8% was reported to be equivalent to the cost of the black rot fungicide spray regime (Molitor and Beyer 2014). Black rot is favored by warm weather but can be a significant threat in moderately temperate humid regions with late springs and summer rainfall (Spotts 1977). On berries, the first symptom appears as a cream-colored dot, which expands and develops an outer ring of light brown necrotic tissue; this brown necrotic area eventually overtakes

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the entire berry (Spotts 1980) (Fig. 2). Pycnidia can also be observed early in the infection; these small blue to black spots spread across the surface of the berry, sometimes in concentric rings. Once the entire berry has rotted, it darkens and shrivels into a hard, bluish-black mummy. *G. bidwellii* can overwinter in mummified berries, as well as in cane lesions, and produces both ascospores and conidia that continue the disease cycle (Becker and Pearson 1996; Hoffman and Wilcox 2002).

Although *G. bidwellii* (*P. ampelicida*) infection can result in symptoms similar to herbicide damage, fertilizer damage, or wilting and rotting caused by other fungi, the appearance of pycnidia on the shoot, leaf, or cluster lesions is characteristic of black rot (Molitor and Beyer 2014). However, because pycnidia are not always formed in warmer climates, this generalization can lead to incorrect field diagnosis (Molitor and Beyer 2014). Studies conducted in 2012 and 2013 have established a basis for molecular and proteomic (protein) identification for taxonomic purposes rather than detection (Molitor and Beyer 2014). Microsatellite markers have been used to detect and differentiate between *G. bidwellii* and *Plasmopara viticola*, the causal agent of downy mildew of grape, which can cooccur on grape (Molitor and Beyer 2014).

Berries infected near the end of their susceptible period may not dry out but growers may notice that they develop an unpleasant taste (Molitor et al. 2012). Fruit is most susceptible 3 to 5 weeks after bloom, and susceptibility decreases 6 to 7 weeks after bloom (Hoffman and Wilcox 2002). Although the pathogen can be controlled relatively easily with modern fungicides, it is a challenge in organic production when growing susceptible grape cultivars (Wilcox et al. 2015a). *V. vinifera* cultivars all appear to be highly susceptible to black rot (Wilcox et al. 2015a). Potential for black rot resistance was found in the hybrid cultivars 'Felicia', 'Merzling', and 'Villard blanc' in a 2012 study by F. Rex (Molitor and Beyer 2014).

Phomopsis fruit rot. Another cluster rot disease with a wide geographic range is Phomopsis fruit rot, caused by *Phomopsis viticola* (Sacc.) *Phomopsis* (Sacc.) Bubák (teleomorph: *Diaporthe* Nitschke). The pathogen has historically been characterized by its host affiliation, although more than one *Phomopsis* sp. is sometimes found on a given host. *Phomopsis* spp. can be best identified by molecular methods, and phylogenies are available to resolve taxonomic issues (Gomes et al. 2013; Higgins et al. 2021; Úrbez-Torres et al. 2013). Phomopsis cane and leaf spot and fruit rot symptoms are small black lesions or spots that can appear on leaves and shoots (Fig. 2). Phomopsis rots occur later in the season, often after black rot appears on fruit, and can be diagnosed when fruit turn brown and become soft and shriveled. Additionally, the fruit will develop small, black, pepper-like fungal fruiting bodies on shriveled fruit (Savocchia et al. 2007). Although this disease occurs across the globe, it causes the most economic damage in temperate grape-growing regions (Anco et al. 2013; Pearson and Goheen 1988). *P. viticola* overwinters in the diseased tissues of the grape cane and is also a known trunk disease pathogen in some growing regions (Úrbez-Torres et al. 2013). During harvest time, necrosis of the grape berries and pycnidia can

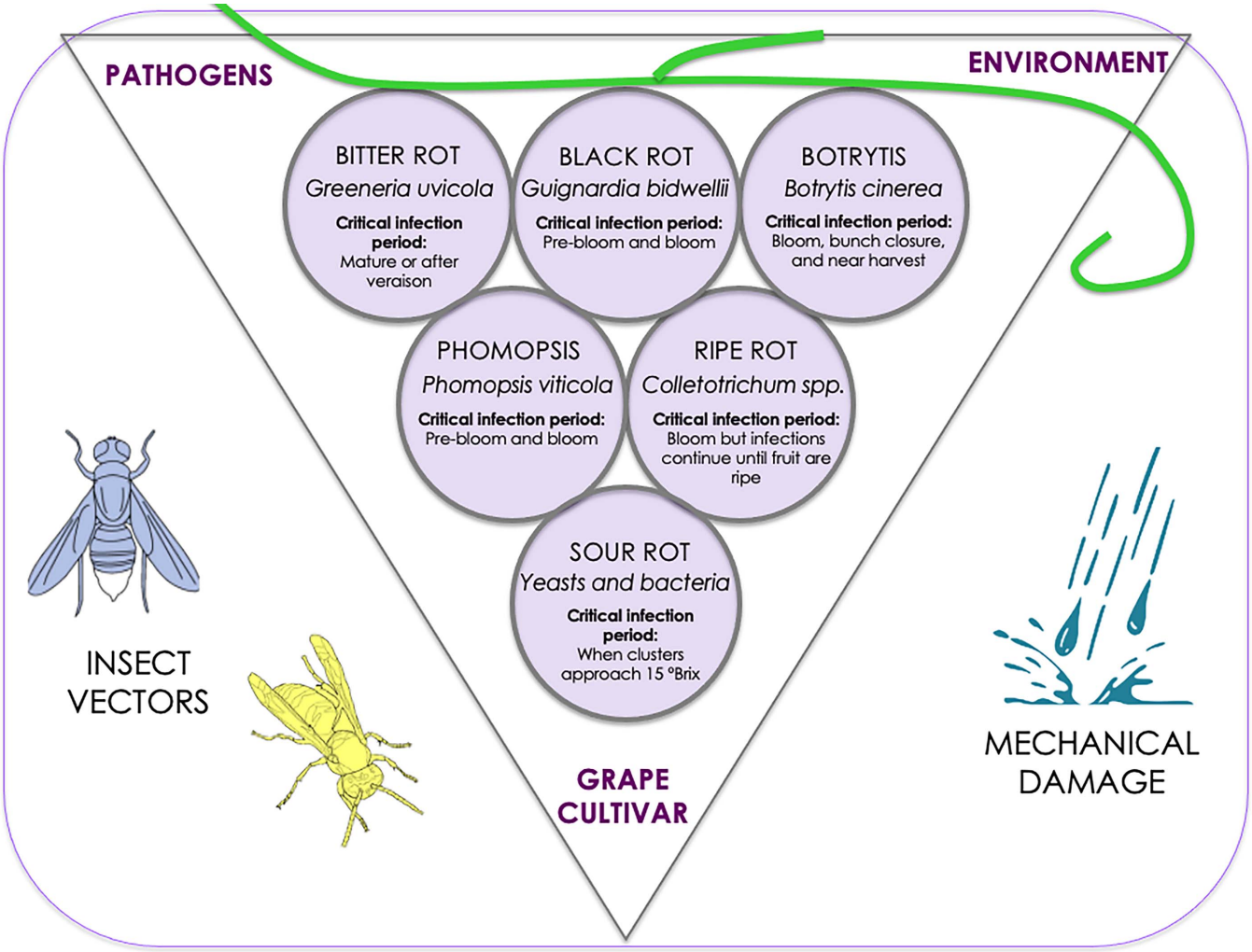

Fig. 1. Common grape cluster rots, the main pathogens involved, and critical infection periods for these diseases (circles). Other factors that affect disease development are additional complexes of pathogens, susceptible cultivars, and a conducive environment for pathogen growth. Insect vectors such as fruit flies and wasps as well as mechanical damage to the berries can induce cluster rot diseases.

also be seen when infections are severe (Savocchia et al. 2007). Once spring arrives, the pycnidia produce conidia that are rain dispersed to infect new host tissue. Initial production of conidia is robust but decreases later in the growing season. Early-season management strategies include dormant cane applications, in-season fungicide applications, and removal of diseased canes through pruning (Nita et al. 2007; Pearson and Goheen 1988). Grape rachis and berries are susceptible to Phomopsis fruit rot the entire growing season and inoculum is continually available if environmental conditions are favorable (Anco et al. 2013; Erincik et al. 2001).

The effects of temperature and wetness on infection of *P. viticola* have been studied extensively (Bugaret 1984; Erincik et al. 2003). Optimum temperature for infection is between 23 and 25°C, and at least 4 h of wetness is required for infection (Bugaret 1984). The minimum and maximum temperatures for infection are 5 and 35.5°C, respectively (Erincik et al. 2003). Predictive models are currently available which estimate sporulation on canes as a way to understand how much inoculum is present to infect fruit; these models serve as a warning system and allow us to understand the *P. viticola* disease cycle (Anco et al. 2013).

Several studies have investigated whether fruit possess ontogenic resistance to *P. viticola* (Erincik et al. 2001; Gregory 1913; Lal and Arya 1982; Pscheidt and Pearson 1989). Although initial studies suggested that ontogenic resistance exists, follow-up studies showed that berry and rachis infections can occur at all growth stages between prebloom and véraison, with no evidence of decreasing susceptibility over time (Erincik et al. 2001).

Bitter rot. Bitter rot is caused by the fungus by *Greeneria uvicola* Punith. (syn. *Melanconium fuligineum*). This disease persists in grape-growing regions with warm summer rains such as in the southeastern United States, coastal Australia, Greece, and parts of South America and Asia (Greer et al. 2014; Kummuang et al. 1996). Environmental conditions that are most favorable for this disease are subtropical with high rainfall and warm conditions which occur during the final stages of berry ripening (Steel et al. 2012). *G. uvicola* is a serious problem for many grape growers, with crop losses ranging from 10 to 50% (Longland and Sutton 2008; Steel et al. 2012). Although this fungus can infect detached fruit of hosts other than grape, it is not known to cause problems on horticultural plants other than grape (Steel et al. 2012).

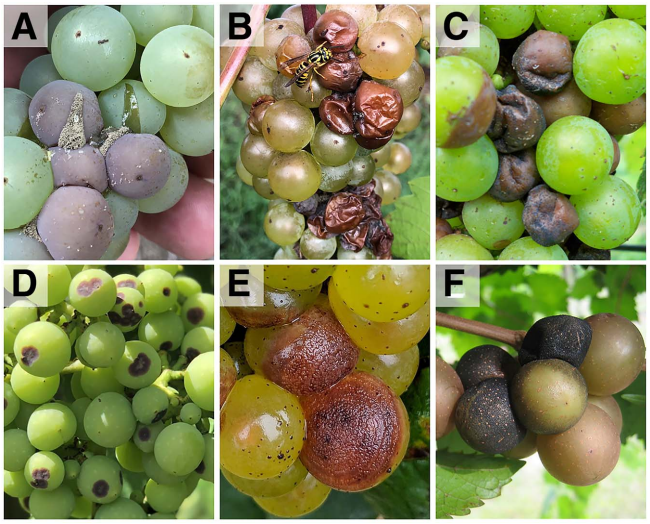

Fig. 2. Common cluster rot diseases of *Vitis vinifera*, *Vitis* interspecific hybrids, and *V. rotundifolia*. **A,** Botrytis bunch rot caused by *Botrytis cinerea* on 'Aurore'. **B,** Sour rot caused by yeasts and acetic acid bacteria on Aurore. **C,** Phomopsis fruit rot caused by *Phomopsis viticola* (photo courtesy of Justin Scheiner, Texas A&M University). **D,** Black rot caused by *Guignardia bidwellii* on 'Niagara'. **E,** Ripe rot caused by *Colletotrichum* spp. on 'Chardonnay' (photo courtesy of Charlotte Oliver, Washington State University). **F,** Bitter rot caused *Greeneria uvicola* (photo courtesy of Phillip Brennen, University of Georgia).

V. vinifera are most susceptible to *G. uvicola* between véraison and harvest (Longland and Sutton 2008). *G. uvicola* can overwinter and live saprophytically on fallen fruit and necrotic bark; with the onset of spring, the fungus exudes conidia (Longland and Sutton 2008). Rain spreads the conidia to new areas of the vine, including pedicels, and in wet, warm conditions infection can occur, optimally at 22.4 to 24.6°C with 6- to 12-h periods of wetness (Longland and Sutton 2008). *G. uvicola* primarily attacks mature berries but has also been found to infect grapevine flowers and pedicels, where it remains latent until the berries begin ripening (Greer et al. 2014; Steel et al. 2012). Rain splash can cause secondary infections by moving conidia from infected berries to other fruit. Damaged berries tend to be more prone to infection, and symptoms include sunken, circular lesions on the berry (spots) (Fig. 2). A few resistant cultivars of grape exist: *V. aestivalis* 'Cynthiana' (syn. 'Norton') is highly resistant; French-American hybrids of *V. vinifera* are more resistant than *V. vinifera* cultivars; and *V. vinifera* 'Merlot', 'Riesling', and 'Sauvignon Blanc' are moderately resistant (Reisch et al. 1979; Wilcox et al. 2015a).

Bitter rot can concurrently occur with ripe rot (*Colletotrichum* spp.) in a vineyard, and even on the same cluster (Greer et al. 2011; Steel et al. 2012). The two are often confused because they tend to be favored by the same weather conditions. However, bitter rot is characterized by masses of acervuli, which can leave hands black when clusters are handled while wet, whereas ripe rot infection results in a slimy mass or dried crust of pink to salmon-colored spores (Wilcox et al. 2015a). In wine production, bitter rot results in bitter taints or off-flavors, whereas wine made from ripe-rot-affected grape has a musty aroma, comparable with a burlap sack, and higher glycerol, gluconic acid, and volatile acidity levels (Meunier and Steel 2009; Steel et al. 2012).

Botrytis bunch rot. *Botrytis cinerea* Pers. (1794) is perhaps the most notorious of the rots and is found almost everywhere grapevines are grown. In grape, this necrotrophic fungal pathogen causes rot primarily in clusters and is a major issue in tight-clustered varieties. Signs and symptoms include sporulation and decomposition of berry tissue. *B. cinerea* has wide-scale economic importance, affecting over 200 host species, including grape, strawberry, tomato, and potato (Coley-Smith and Verhoeff 1980; Dean et al. 2012; Simionato et al. 2017). Interestingly, the majority of fungicides used to control this pathogen were developed specifically for grape disease management (Dean et al. 2012; Steiger 2007). Even though *B. cinerea* prefers wet conditions, it is fairly ubiquitous across the United States and the world (Karchani-Balma et al. 2008). *B. cinerea* has been found on plants on remote oceanic islands and even in microfungal communities collected from Antarctica (Azmi and Seppelt 1998; Garfinkel et al. 2019; Rodríguez et al. 2014).

Initial symptoms for grape are typically brown lesions on the berry skin and underlying mesocarp, causing discoloration on both tissue types (Fig. 2). As the disease progresses, masses of gray spores can be seen on the fruit (Oliveira et al. 2017; Sutton 1998). *B. cinerea* can infect grape directly through conidial germination and the grapevine flowers through mycelial penetration early in the growing season (Armijo et al. 2016; Viret et al. 2004). Often, the fungus remains dormant in the developing grape berry until the berries mature and soluble solids reach approximately 12 brix. Mature berries can be infected directly, typically through cracks or other wounds. Following infection, grape berries change metabolically and have increased production of gluconic acid and glycerol (Armijo et al. 2016). Abundant spore production and a polycyclic disease cycle make *B. cinerea* a challenging pathogen to manage (Fernández-Ortuño et al. 2012; Leroux et al. 2002). This is further compounded by a high risk of fungicide resistance development to commonly used fungicide classes. Management of Botrytis bunch rot relies on a single fungicide application at bloom, followed by leaf thinning around the cluster zone to improve airflow in hot and dry regions. In growing regions with high humidity, management may include additional sprays as clusters develop and ripen. Fungicide resistance is a primary factor in shaping *Botrytis* populations, and these populations can persist across years (DeLong et al. 2020; Fernández-Ortuño et al. 2015; Kozhar et al. 2020; Naegele et al. 2021). Hybrids of *V. labrusca* cultivars (e.g., 'Isabella') differ in their susceptibility to *B. cinerea* and cluster

architecture plays a role in Botrytis bunch rot management. A positive correlation has been shown between *B. cinerea* incidence and severity and the degree of compactness of grape clusters (Bondada et al. 2016; Hed and Centinari 2021; Hed et al. 2009; Tello and Ibáñez 2018).

Interestingly, although both *V. vinifera* and *Botrytis* spp. originated in Eurasia, most *V. vinifera* cultivars have little or no resistance to *Botrytis* spp., and table grape varieties, with their thin berry skin, are more susceptible than wine grape (Gabler et al. 2003; Naegele 2018). Some wine grape cultivars with reduced susceptibility have been reported but, often, this is due to a loose, open cluster architecture that promotes epicuticular wax deposition on berries (Gabler et al. 2003; Kocsis et al. 2018; Marois et al. 1986; Richter et al. 2020; Vail and Marois 1991). In the vineyard, cultivars such as Cabernet Sauvignon and Petit Verdot have low observed disease incidence compared with tight-clustered cultivars such as Pinot Gris and Riesling (Pañitrur-De La Fuente et al. 2018; Vail and Marois 1991). In the lab, however, these cultivars had similar levels of susceptibility to *B. cinerea*. Wild *Vitis* spp., including *V. cinerea*, *V. rotundifolia*, and *V. labrusca*, have some degree of resistance, and have been used to generate resistant interspecific hybrids (Eibach and Töpher 2003; Pedneault and Provost 2016; Reisch et al. 2014; Wan et al. 2015). Several resistant wine grape hybrid cultivars are widely grown, including Regent (Germany) and Frontenac (northern United States) (Atucha et al. 2018; Migicovsky et al. 2016). For most susceptible grape cultivars, chemical management can minimize disease severity. Increasing air flow in and around the clusters through manual leaf thinning or chemical applications of DL-β-amino-n-butyric acid can effectively reduce cluster rot incidence in the vineyard although, over time, these practices can lead to resistance (English et al. 1990; Hed and Centinari 2021; Kocsis et al. 2018). Timing and amount of leaf removal is critical because it can affect berry set and yield, in addition to cluster compactness and disease severity (Molitor et al. 2011, 2012; Mosetti et al. 2016; Würz et al. 2020; Zoecklein et al. 1992). When leaf removal occurs after véraison, little effect on disease severity is observed because infection typically takes place during bloom.

Ripe rot. Ripe rot occurs in subtropical viticulture regions worldwide, including Australia (Greer et al. 2014; Meunier and Steel 2009), India, Asia, (Steel et al. 2013), and Brazil (Echeverrigaray et al. 2020). This widespread disease was originally thought to be caused by *Colletotrichum gloeosporioides* sensu lato and *C. acutatum* sensu lato.

Identification of *Colletotrichum* spp. originally relied on conidia shape and size, colony morphology, host plant, and other characteristics and physiological traits. However, more recent molecular identification techniques have identified additional *Colletotrichum* spp. that are involved in the disease, including *C. fructicola, C. kahawae, C. limitticola, C. nymphaeae, C. karstii*, and *C. viniferum* (Echeverrigaray et al. 2020; Oo and Oh 2017). Different species within this pathogen complex displayed varying amounts of virulence on their grape hosts, and those with low virulence were found less frequently on the grape (Echeverrigaray et al. 2020). For the first time, a gene region in the grapevine genome was discovered to confer resistance to ripe rot, which will help breeders design host resistance genotypes in the future (Fu et al. 2019).

Ripe rot (*Colletotrichum* spp.) was first reported in the United States in 1891, and can be found primarily in warm and moist growing regions in the southeastern United States (Steel et al. 2013; Wilcox et al. 2015a). Although grape berries are susceptible to ripe rot from bloom to ripening, the disease appears most often during postvéraison and close to the point of berry maturity (Echeverrigaray et al. 2020). Symptoms begin with circular, reddish-brown spots of decay on the skin of affected berries; the spots eventually enlarge to include the entire berry (Fig. 2). As the berry further decays, salmon-colored masses of conidia form. Eventually, diseased berries shrivel and die; at this point, symptoms may resemble a number of other rots, including black rot, bitter rot, and Phomopsis fruit rot (Wilcox et al. 2015a). Ripe rot is not the same as anthracnose of grape (*Elsinoë ampelina*) but they are sometimes confused because diseases incited by *Colletotrichum* spp. are given the common name anthracnose on many other crops (Wilcox et al. 2015a).

Sour rot and summer bunch rot. Sour rot is a disease complex in temperate grape-growing regions involving acetic acid bacteria, various yeasts, *Drosophila* fruit flies, and the grape host. This disease complex is still poorly defined; however, it is known that yeast infection occurs first, converting sugars in the grape to ethanol. Following the yeast infection, acetic acid bacteria in the berry juices oxidize the alcohol into acetic acid, which gives the disease its distinctive vinegar-like smell along with browning of the berry skin (Fig. 2) (Hall et al. 2018a). As the berries rot, volatiles that attract insects are produced that facilitate disease progression and spread. *Drosophila* fruit flies, the main insects associated with the sour rot disease complex, carry the yeasts and acetic acid bacteria on their bodies and in their guts, and can transfer them to the berries as they feed or lay their eggs. Late in the growing season, cracks in the berry skin, caused by biotic and abiotic factors, create entry points for the microbes on the flies to disperse to the berry (Kenney and Hall 2021). Social wasps may also play a role in sour rot ecology (Madden et al. 2017). Disease management trials indicated that the most impactful way to manage sour rot was to use insecticides in tandem with antimicrobial applications in order to manage these two aspects of the disease complex (Hall et al. 2018a). In a more holistic approach to sour rot management, cultural controls such as canopy training systems can be utilized. Canopies that were trained to vertical shoot positioning also exhibited significantly less sour rot severity than those trained to a high wire cordon system (Hall et al. 2018b) (Fig. 3). Other approaches include using gibberellic acid (GA) to loosen clusters and, thereby, reduce disease in other bunch rots (Hed et al. 2009) (Fig. 4).

Summer bunch rot is caused by a collection of microbes, resulting in symptoms similar to those of sour rot. However, it is not understood if the microbial community composition of summer bunch rot disease complex is similar to that of the sour rot disease complex (UC IPM 2017) (Table 1). Summer bunch rot is often described as synonymous with sour rot (Pisani et al. 2015; Rooney-Latham et al. 2008) or, sometimes, sour rot is referred to as a principal component of the summer bunch rot complex (Duncan et al. 1995; Stapleton 1992). An older definition describes summer bunch rot as a specific type of sour rot caused by initial infection of *Diplodia natalensis* Pole-Evans (syn, *Lasiodiplodia theobromae* (Pat.) Griffon & Maubl and *Botryodiplodia theobromae* Pat., teleomorph, *Botryosphaeria rhodina* (Cooke) Arx); however, this is primarily based on a summer bunch rot complex that affected 'Thompson Seedless' grape in the San Joaquin and Coachella

Fig. 3. A, Late-season cluster rot management of 'Reisling'; notice evidence of leaf removal and clear canopy and fruit zones as the result of training. **B,** High wire training of 'Niagara', common in juice grape due to a lack of cluster rot issues in *Vitis* × *labruscana*. **C,** V-style trellis and **D,** three-dimensional "T" trellis commonly used in table grape production systems to open the canopy.

Valleys of California in the 1960s (Hewitt 1974, 1979). Later observations in the same area did not recover any *D. natalensis* (Duncan et al. 1995) but, instead, found *Aspergillus niger* and *A. carbonarius* to be the primary pathogens associated with affected berries (Rooney-Latham et al. 2008). Both *A. niger* and *A. carbonarius* produce the mycotoxin ochratoxin A (OTA), which is carcinogenic, immunosuppressive, teratogenic, and nephrotoxic in animals (Pfliegler et al. 2020; Zhang et al. 2017). Due to the toxicity of OTA, the allowable concentration is limited to 2.0 µg/liter in juice, musk, and wine production and <10 µg/kg in dried fruit (Zhang et al. 2017). Volatile organic compounds (VOCs), including ketones, transnerolidol, and C-8 alcohols, are often associated with OTA synthesis in *A. carbonarius* (Zhang et al. 2017). VOC profiles or patterns can be used to predict OTA accumulation (Zhang et al. 2017) or even to differentiate between different bunch rot pathogens in grape juice production (Steel et al. 2013).

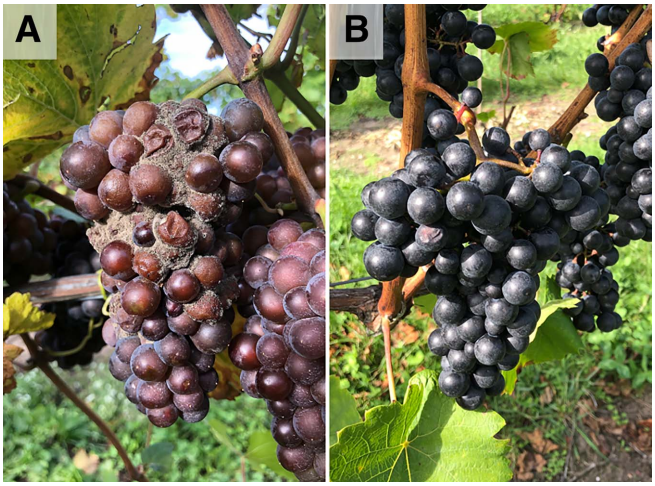

Fig. 4. Tight-clustered varieties are particularly susceptible to cluster rot diseases such as Botrytis bunch rot. **A,** Photo of *Vitis vinifera* 'Pinot Gris'. Berries with more loose clusters tend to be slightly susceptible but are less likely to have significant cluster rot problems, **B,** Photo of *V. vinifera* 'Blaufränkisch' (or commonly 'Lemberger') in the United States.

Molecular diagnostics. Traditional diagnosis of cluster rots is typically done visually by symptoms alone (Figs. 1 and 2). However, a variety of molecular diagnostic techniques are available for cluster rot diseases of grape and can be useful for early disease detection. Although there are several primary pathogens, grape berries are a complex substrate and there are several known secondary colonizers such as *Cladosporium cladosporioides*, *C. herbarum*, *Penicillium* spp., *Rhizopus arrhizus*, and *Alternaria* spp. For the primary pathogens, most of these techniques are based on PCR and utilize a variety of unique loci (Table 2). *Botrytis cinerea* has the most extensive suite of marker systems and the majority of markers target various transposons (Martinez et al. 2008) (Table 2). Other marker systems target the internal transcribed spacer gene region, β-tubulin, or calmodulin genes (Samuelian et al. 2011). Sour rot is more complex and no known marker systems exist for the causal agents; an amplicon sequencing approach is required for both the yeast and the bacterial portions of the disease complex (Hall et al. 2018a).

Grape berries are a complex and ephemeral substrate for pathogens to colonize and on which to reproduce. The particular pathogen that can predominate on the fruit varies depending on cluster architecture and other ecological factors (moisture levels, temperature, and solar radiation). The major challenges in applying molecular diagnostic tools are generally getting clean DNA in adequate amounts to detect the pathogen and determining the best way to collect samples. The presence of PCR inhibitors in fruit (e.g., polyphenols and sugars) can complicate pathogen detection by reducing amplification efficiency or preventing amplification entirely (Salzman et al. 1999). These tools find utility in early prevention or detection of latent infections but may become most important when conducting spore-trapping experiments. In vineyards, a number of spore-trapping studies have been performed to monitor powdery mildew (Thiessen et al. 2016); in time, this technology could be utilized for cluster rot pathogens.

Best management practices for disease. One of the main goals for managing cluster rot diseases is to minimize injuries to berries that enable pathogens to establish. Reducing the number of berries per cluster on tight-clustered varieties (Fig. 4) can be done mechanically but may be labor intensive. In addition, effective management

Table 1. Common fungi and bacteria associated in the grape sour rot disease complex[a]

Scientific name	Microbe	Source	Ecological role or function	Reference
Aspergillus niger	Fungus	Inoculum	Most abundant type of *A. niger* associated with sour rot; produces ochratoxin A	Hall et al. 2018a; Tjamos et al. 2004
Hanseniaspora uvarum	Fungus	Inoculum	Ascomycete involved in fermentation	Barata et al. 2012b; Hall et al. 2018a
Pichia kluyveri	Fungus	Inoculum	Weakly fermentative yeast	Barata et al. 2008; Hall et al. 2018a
Saccharomyces cerevisiae	Fungus	Inoculum	Fermentative species that increases in abundance when grape skin is damaged	Barata et al. 2008; Hall et al. 2018a
Saccharomyces sp.	Fungus	Sequencing	Fermentative species that increases in abundance when grape skin is damaged	Barata et al. 2008; Hall et al. 2018a
Metschnikowia sp.	Fungus	Sequencing	Present as an endophyte in healthy grape berries	Hall and Wilcox 2019; Hall et al. 2018a
Gluconobacter sp.	Bacterium	Inoculum and sequencing	Present as an endophyte in healthy grape berries	Hall and Wilcox 2019; Hall et al. 2018a
Acetobacter aceti	Bacterium	Inoculum	Acetic acid bacteria present in sour rotten grape	Barata et al. 2012a; Hall et. al 2018a
Hafnia sp.	Bacterium	Sequencing	Bacteria associated with grapevines, especially the rhizosphere	Hall et al. 2018a; Karagöz et al. 2012
Rahnella sp.	Bacterium	Sequencing	Endophyte on grape	Hall et al. 2018a; Pacifico et al. 2019

[a] Microbes labeled as "Inoculum" were used in the Hall et al. (2018a) article as sources of inoculum in pathogenicity experiments and those labeled as the same authors identified microbes through "Sequencing" by using amplicon-based next-generation genetic data taken from field samples.

of insects (e.g., grape berry moth, vespids, and other insects), birds, and powdery mildew will greatly reduce the risk of cluster rot in many vineyards. Promoting air circulation within the grapevine canopy also reduces the risk of cluster rots (Alzohairy and Miles 2020). Canopy management methods aimed at improving air circulation and reducing humidity include leaf removal in the fruit zone, shoot positioning, shoot thinning, and hedging. Limiting excessive vegetative growth by balance pruning and avoiding excess nitrogen fertilization can reduce disease; however, this may not always stave off cluster rot onset. Other in-season practices that limit canopy growth include promoting an optimal balance of light and shade and avoiding excessive shade that can encourage growth of fungal pathogens such as *B. cinerea* (Gubler et al. 1987; Smart et al. 1990).

Despite all of these cultural tools, chemical options are still critical to control cluster rot diseases. Multisite fungicides can be effective but are generally only moderate in their efficacy; thus, vineyard managers are forced to use site-specific products. Site-specific fungicides in Fungicide Resistance Action Committee (FRAC) codes 2, 7, 9, 11, 12, and 17 are effective on some cluster rot pathogens such as *B. cinerea* (Table 3). For early-season rots such as black rot and Phomopsis fruit rot, fungicides in FRAC codes 3 and 11, as well as ethylene bisdithiocarbamates, are the most effective. Generally, these management strategies are not effective for sour rot control; sterilants such as hydrogen peroxide and peroxyacetic acid are used commercially (Van Timmeren et al. 2019) and are applied in addition to insecticides (Gillett et al. 2018). Unfortunately, fungicide resistance

Table 2. Molecular diagnostic markers to detect cluster rot pathogens in vineyards

Cluster rot disease, pathogens	Type of assay	Target locus[a]	Primers	Sequences (5′ to 3′)	References
Botrytis bunch rot, *Botrytis cinerea*	PCR	Flipper transposase gene	FLIP and FLIP 2	GGACCACCCCTCTTTTGGAC and CGGTTGTGTAAAGTGGTGCG	Duan et al. 2014; Martinez et al. 2008; Si Ammour et al. 2019; Suarez et al. 2005
	–	Insertion site of Flipper	FLIP2 and FABR	CGGTTGTGTAAAGTGGTGCG and GTGCCACCTAAGTTGAGTACCCC	–
	–	Boty transposon	BOT1 and BOT2	AGCCAAGGGCTCAAGATGA and TACGCTCGTTGTGGTGAAGT	–
	–	β-Tubulin gene	Bc1F and Bc1R	GTTACTTGACATGCTCTGCCATT and CACGGCTACAGAAA GTTAGTTTCTACAA	–
	–	SCAR marker	Bc2F and Bc2R	TTCGTGATTATCACCTGGGTTG and GCTCCTAGAACGTACGACCACA	–
	–	GS spacer	Bc3 and Bc3R	FGCTGTAATTTCAATGTGCAGAATCCI and GGAGCAACAATTAATCGCATTTC	–
	–	Cutinase-A gene	CG11[d] and CG12[d]	AGCCTTATGTCCCTTCCCTTG and GAAGAGAAATGGAAAATGGTGAG	–
	–	Initiate LAMP reaction	F3 and B3	CTACACAACGACCACAGT and CCACCAGGTAGTTTCAATCC	–
	–	*Bcos5* gene	P1 and P2	GATACCCCTCAACAAAAGCCT and CCAGGTTGTCTTCCTACTTGC	–
	–	IGS	Bc3F and Bc3R	GCTGTAATTTCAATGTGCAGAATCC and GGAGCAACAATTAATCGCATTTC	–
Sour rot, acetic acid bacteria; yeasts	Amplicon sequencing	V4 region 16s rRNA	F515 and R806	GTGTGCCAGCMGCCGCGGTAA and GGACTACHVGGGTWTCTAAT	Bokulich et al. 2014; Hall et al. 2018a
	–	ITS 1	BITS and B58S3	CTACCTGCGGARGGATCA and GAGATCCRTTGYTRAAAGTT	–
Black rot, *Guignardia bidwellii*	PCR	ITS1-ITS2 region	ITS 4 and ITS 5	TCCTCCGCTTATTGATATGC and GGAAGTAAAAGTCGTAACAAGG	Donaldson 1995; Glass and O'Donnell and Whiteley 1999; Rinaldi et al. 2017; White et al. 1990
	–	β-Tubulin gene	Bt2a and Bt2b	GGTAACCAAATCGGTGCTGCTTTC and ACCCTCAGTGTAGTGACCCTTGGC	–
	–	Calmodulin gene	CL1 and CL2A	GA(GA)T(AT)CAAGGAGGCCTTCTC and TTTTTGCATCATGAGTTGGAC	–
Bitter rot, *Greeneria uvicola*	Real-time PCR	ITS region	GuF2b and GuR2	TCTGAACGTATCTCTTCTGAG and TAAGTCAACCTAAGCGAGAAG	Samuelian et al. 2011
Ripe rot, *Colletotrichum* spp.	Real-time PCR	ITS region	CaITS_F701 and CaITS_R699	GGATCATTACTGAGTTACCGC and GCCCGCGAGAGGCTTC	Debode et al. 2009; Samuelian et al. 2011
Phomopsis fruit rot, *Phomopsis viticola*	PCR	EF1-α gene	EF1-728F and EF1-986R	CATCGAGAAGTTCGAGAAGG and TACTTGAAGGAACCCTTACC	Schilder et al. 2005
	–	Calmodulin gene	CAL-228F and CAL-737R	GAGTTCAAGGAGGCCTTCTCCC and CATCTTTCTGGCCATCATGG	–

[a] SCAR = sequence-characterized amplified region, LAMP = loop-mediated isothermal PCR, IGS = integrated genome sizing, ITS = internal transcribed spacer, and EF1 = elongation factor 1.

is a worsening problem for controlling cluster rot diseases, particularly Botrytis bunch rot. For example, a recent study found that *B. cinerea* isolates recovered from Michigan vineyards were resistant to six of the eight major FRAC codes used to control *B. cinerea* (Alzohairy et al. 2021).

Insect pest management for late season cluster rots. The development of cluster rot often involves the interplay of multiple organisms, and insects play a critical role. Insects can serve as pathogen vectors, facilitating the colonization of microorganisms by injuring or damaging the berries, or may contribute to the microbial community found in diseased clusters (Barata et al. 2012b; Entling and Hoffmann 2019; Fermaud and Le Menn 1992; Hall et al. 2018a; Ioriatti et al. 2018). Vineyard insects appear to have a close association with the development of sour rot and the transmission of Botrytis bunch rot (Fermaud and Le Menn 1992; Hall et al. 2018a). Grape sour rot does not develop typical symptoms without *Drosophila* fruit flies (Hall et al. 2018a). These flies play a critical role as vectors and contribute significantly to the development of the disease through their feeding activity (Entling and Hoffmann 2019; Hall et al. 2018a). The microbiome of *Drosophila melanogaster* contains both bacteria and yeast that contribute to sour-rot infection; however, these microorganisms seem to be unable to reproduce sour-rot symptoms individually. In fact, the microbial agents associated with sour rot seem to live as endophytes and epiphytes on the surface of healthy berries (Hall et al. 2019). Full development of the disease seems to require the presence of *Drosophila* flies (Hall et al. 2018a). Berry injury by feeding larvae and possible insect salivary or digestive enzymes is critical for the development of symptoms (Hall et al. 2018a). However, *D. melanogaster* oviposition on grape is limited to overripe or physically damaged berries (Rombaut et al. 2017). *Drosophila* infestations are often seen in berries previously wounded by other animals or cracked by changes in moisture (Entling and Hoffmann 2019). In contrast, the invasive species *D. suzukii* has the ability to infest fresh, healthy berries due to the presence of a special serrated ovipositor (Atallah et al. 2014). In field conditions, both *Drosophila* spp. are commonly present in sour rot-infected grape (Rombaut et al. 2017). Both *D. melanogaster* and *D. suzukii* harbor microbes associated with sour rot but only *D. suzukii* is able to wound unripe berries, whereas *D. melanogaster* appears late in the season in overripe wounded berries. It is speculated that initial infestations of *D. suzukii* may enhance sour rot infections by providing oviposition substrates for *D. melanogaster* later in the growing season (Rombaut et al. 2017). Entling and Hoffmann (2019) investigated the individual and joint effects of *D. suzukii* and *D. melanogaster* infestation on sour rot development. The authors concluded that both fly species contribute to the development of the disease but no synergistic effect was found (Entling and Hoffmann 2019). Similar to *Drosophila*, it appears that paper wasps harbor some

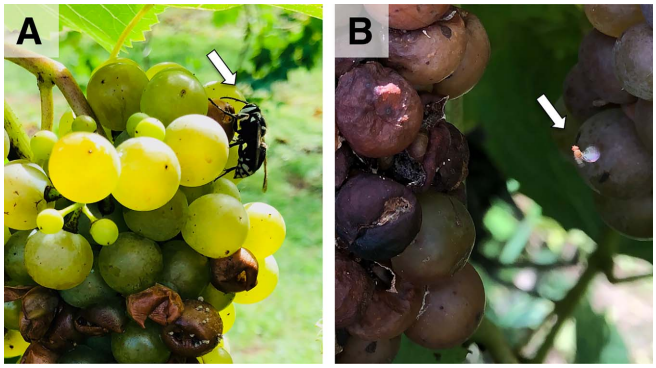

Fig. 5. Insects associated with dispersal of sour rot on grapevine include **A,** bald-faced hornet *Dolichovespula maculata* and **B,** *Drosophila* fruit flies. Arrows denote the associated insect vector.

Table 3. Efficacious site-specific fungicides commonly used to control *Botrytis cinerea* in vineyards and resistance mechanisms that have been characterized

Active ingredient	Target site of action	FRAC code[a]	Resistance reported?	Mechanism of resistance	References
Thiophanate-methyl	β-tubulin assembly in mitosis	1	Yes	Amino acid substitutions in *β-tubulin* gene at locations 198 and 200	Fernández-Ortuño et al. 2015
Iprodione	NADH cytochrome c reductase in lipid peroxidation	2	Yes	Amino acid substitutions of *Bos1* gene at locations 365 and 369, and other point mutations and rearrangements a part of multiple drug resistance and potentially unknown mechanisms	Fernández-Ortuño et al. 2015; Grabke et al. 2014
Boscalid, fluopyram, fluxapyroxad, isofetamid, penthiopyrad, pydiflumetofen, pyraziflumid	Complex II: succinate-dehydrogenase	7	Yes	Amino acid substitutions succinate dehydrogenase subunit B at locations 225, 230, and 272	Alzohairy et al. 2021; Samaras et al. 2016
Cyprodinil	Methionine biosynthesis	9	Yes	Mutations of *Bos1* gene and other point mutations and rearrangements a part of multiple drug resistance	Alzohairy et al. 2021; Kretschmer et al. 2009; Saito et al. 2019
Azoxystrobin, kresoxim-methyl, mandestrobin, pyraclostrobin, trifloxystrobin	Complex III: cytochrome bc1	11	Yes	Amino acid substitution in *cytb* gene known as G143A complicated by presence of an intron	Alzohairy et al. 2021; Saito et al. 2016; Samuel et al. 2011
Fludioxonil	MAP/histidine-kinase in osmotic signal transduction	12	Yes	Mutations of *Bos1* gene and other point mutations and rearrangements a part of multiple drug resistance	Fillinger et al. 2012; Kretschmer et al. 2009; Ren et al. 2016
Fenhexamid	3-Keto reductase, C4-de-methylation	17	Yes	Amino acid substitutions in *erg27* gene at locations 63 and 412	Alzohairy et al. 2021; Fernández-Ortuño et al. 2015

[a] Fungicide Resistance Action Committee.

of the sour-rot-associated microbes; the wasps are not able to physically injure the berries but can vector these microorganisms during their foraging behavior (Madden et al. 2017). The underlying mechanisms of sour rot association with drosophilid insects and paper wasps is not well understood. It is likely that the chemical composition of diseased clusters recruits fruit flies that are attracted to acetic acid and ethanol odors (Barata et al. 2012b; Giang et al. 2017) (Fig. 5) Additionally, sour-rot-infected grape may be a more nutritious food source for *Drosophila* than healthy berries. Free-living yeasts constitute a rich protein source for many species of *Drosophila*, and their concentration affects the survivorship of the larvae (Lewis and Hamby 2019). Rombaut et al. (2017) reported an increase in oviposition of *D. melanogaster* in sour-rot-infested grape compared with healthy ones. Therefore, the close association of *Drosophila* fruit flies with sour rot may represent a mutualistic relationship in which the flies contribute to the dispersion of the microbes which, in turn, enhance the survival of the flies (Barata et al. 2012b).

Control of *Drosophila* fruit flies is key in sour rot management programs because insect pests play a large role in disease development (Hall et al. 2018a). *Drosophila* flies are a problem late in the growing season and are controlled mainly with insecticides. Some insecticide sprays targeting *Drosophila* flies reduce sour rot severity; in a recent study, weekly applications of insecticides combined with antimicrobials reduced sour rot severity by 64% compared with untreated vines (Hall et al. 2018b). Spray programs should rotate products with different modes of action to avoid or delay the development of resistance. *D. melanogaster* has developed resistance to pyrethroid, organophosphate, and neonicotinoid insecticides (Sun et al. 2019). Cultural practices such as rigorous vineyard sanitation, mass trapping, and exclusion netting seem to help reduce populations of *Drosophila* (Ebbenga et al. 2019; Haye et al. 2016). Berry skin thickness affects the capability of *D. suzukii* to oviposit in grape berries; there seems to be a negative correlation between the number of eggs laid and berry skin resistance in different grape cultivars (Entling et al. 2019).

Various species of Lepidoptera moths from the family Tortricidae also appear to be associated with cluster rot diseases. Tortricid moths lay their eggs on grape berries or flowers; after hatching, the larvae feed on developing fruit (Gilligan et al. 2011). Developing larvae often move within the cluster, inflicting wounds on several berries. The physical damage facilitates pathogen infection, and the movement of the larvae enhances the possibility of transporting pathogens from diseased berries to healthy ones. Studies with the European grapevine moth (*Lobesia botrana*) demonstrated colonization by *B. cinerea* of larval wounds and at the entrance of larval galleries (Fermaud and Le Menn 1992). Larvae of *L. botrana* artificially contaminated with *B. cinerea* carried the pathogen and inoculated the walls of larval galleries (Fermaud and Le Menn 1992). These results suggest that cluster infestations with tortricid larvae may increase the incidence and severity of Botrytis bunch rot.

Future prospects. Although cluster rots have plagued growers for centuries, some strategies for improving their management have emerged. Here, we highlight some promising opportunities for grape cluster rot management: (i) focused development on new and better cultural controls, (ii) refining and implementing detection technologies such as remote sensing and spore traps in vineyards for rapid and early disease detection, (iii) harnessing the power of gene editing and microbiome datasets and, finally, (iv) using additional ecological information to formulate targeted disease control interventions.

First, there is an urgent need to develop new and better cultural controls to combat grape diseases, especially for cluster rot management. Given that high moisture and warm temperatures play a role in disease progression, encouraging airflow can help manage disease, especially in tight-clustered varietals (Hed et al. 2009). Leaf canopy pruning promotes better air circulation and allows more sunlight, which can help reduce disease pressure for fungal and oomycete diseases powdery mildews and downy mildews, including cluster rots (Hickey and Hatch 2018). It is also possible to manipulate the grape cluster architecture to promote more airflow and dryness. Plant hormonal sprays induce cluster openness by changing the overall shape and number of grape per cluster (Hed et al. 2011). For instance, the plant hormone GA can modulate grape cluster compactness and reduce incidence and severity of cluster rots. A common method to induce GA production naturally, especially in table grape, is physical girdling of the trunk, which allows for cluster elongation. Girdling can result in cluster openness (Abu-Zahra and Salameh 2012) and can improve berry weight and sugar content by increasing plant stomatal conductance (Pereira et al. 2020); however, it could cause problems with trunk infections. In a study by Hed et al. (2011) and Hed and Centinari (2021), GA sprays were added to 'Vignoles' and 'Chardonnay' wine grape in vineyards in the eastern United States. When this hormonal spray was used, disease incidence was reduced by 40 to 50% for Botrytis bunch rot compared with clusters that were not sprayed. Spraying the correct amount of hormone at the right time (typically prebloom) is critical because it can negatively affect grape yield; this method still needs more research, especially in different microclimates. Moreover, spraying GA and other plant hormones that change the cluster phenotype is still not approved for most cultivars, and the brands that are approved for use vary between states and growing regions (B. Hed, personal communication). There is a tradeoff between the amount of spray used and the negative effects on vine and cluster growth and structure if used improperly. Still, growers are interested in these hormonal sprays as a promising strategy to loosen cluster architecture and suppress cluster rot development (Hed et al. 2011). Selection for cultivars with thicker cuticles appears to contribute some degree of resistance against infections from *B. cinerea* and seems to enhance resistance against sour rot (Entling and Hoffmann 2019; Gabler et al. 2003). Moreover, it has been reported that grapevines sprayed with silicon had less severe powdery mildew (Bowen et al. 1992; Reynolds et al. 1996) but there is no report of silicon increasing plant resistance to grape rots.

Second, plant disease detection technologies such as remote sensing and new and improved spore traps can be used in vineyards to rapidly find disease, especially when time and resources are limited. Remote sensing has traditionally been used to aerially map changes in Earth's vegetation; however, more recently, it is being applied to the field of plant pathology to detect abiotic and biotic plant stress (Mahlein 2016). Images taken from airplanes or drones allow researchers to detect "hot spots" in a vineyard or locations where changes in foliage functional chemistry and reflectance indicate disease. The map that is generated can inform growers where to target on-the-ground scouting efforts for finding disease and where to sample vines to verify disease using molecular DNA identification (Zarco-Tejada et al. 2018). Although spectroscopy imaging technology for plant disease detection

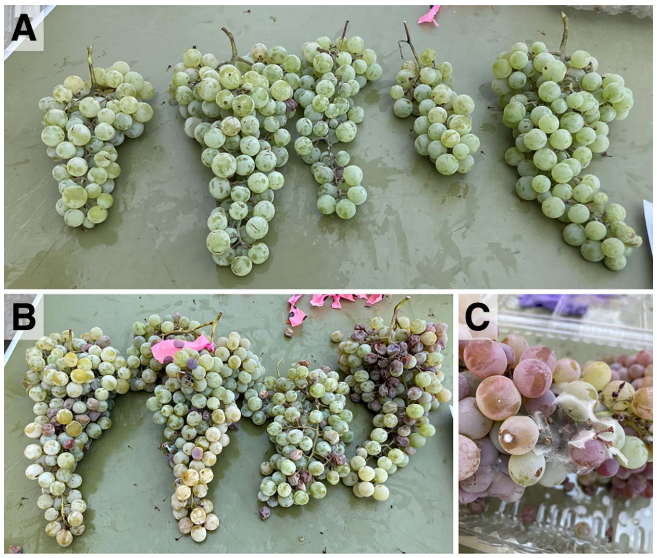

Fig. 6. Breeding for *Botrytis*-resistant grape cultivars could reduce postharvest losses, which are a problem for table grape. **A,** *Botrytis*-resistant breeding line 8 weeks postharvest and **B,** *Botrytis*-susceptible breeding line 8 weeks postharvest. **C,** Close-up of postharvest fungal-induced rot.

is still a burgeoning field, it has been used for rapid detection of *Xylella fastidiosa* in olive trees (Zarco-Tejada et al. 2018) and holds promise for diseases of grape.

Spore traps are another technology that has evolved to be more efficient and specific for disease detection. Many spore traps can stay in the field longer with better batteries and solar cells and they have increased their efficiency to collect airborne inoculum (Thiessen et al. 2016). These traps have the potential to improve inoculum models and refine disease forecasting systems. Furthermore, these technological advancements allow spore traps to collect plant pathogens with better accuracy and precision for microscopy analysis of spore identification and counts, as well as for downstream DNA amplification via PCR and high-throughput sequencing to assess aerial microbiota to detect potential pathogens (Crandall et al. 2020) or detect fungicide resistance (Miles et al. 2021).

Third, genetic tools can be used for cluster rot management and to determine indicators and biocontrol agents. As countries enact stricter fungicide restrictions, the need for cluster-rot-resistant grape cultivars will grow. Marker-assisted selection for grape breeding has been underway for certain fungal pathogens such as powder mildews (Dalbó et al. 2001) and other recent technologies such as embryo rescue are leading to new disease-resistant cultivars (Li et al. 2020). CRISPR gene editing is being developed for grape and could be a promising alternative or supplement to traditional breeding methods (Maher et al. 2019) (Figs. 6 and 7). Historically, traditional breeding of new disease-resistant grape varieties using wild ancestors has met resistance from industry and consumers alike, because of reported poor wine quality and "off flavors" (González-Centeno et al. 2019). The problems may be less likely in a CRISPR-edited grape as compared with wild material, because they would not be introgressed as in traditional breeding approaches. However, as consumer acceptance of blended bulk wines increases and the quality of hybrid wines continues to improve, new hybrid disease-resistant cultivars are becoming available (González-Centeno et al. 2019). This is evident in the release and widespread interest in powdery-mildew-resistant grape cultivars (Fuller et al. 2014; González-Centeno et al. 2019). Their widespread use will likely be dependent on disease pressure for a given region, and the cost and availability of cultural or chemical controls to manage disease (Sambucci et al. 2019).

Fourth, because cluster rots are complexes of pathogens, other microbes, and insect vectors that attack the grape berry, a better understanding of cluster rot ecology—specifically, the composition, structure, and function of the microbial communities that cause disease—is essential for developing targeted fungicides and management. Currently, pesticides are applied in two phases: early season (1 to 4 weeks after bloom) and late season (bloom to harvest). In the case of Phomopsis cane and leaf spot, azoxystrobin and captan are typically applied. For black rot, azoxystrobin, tebuconazole, and trifloxystrobin are applied (Todaro and Miles 2018). Finally, some pioneering microbiome studies have sought to identify the surface microbiome of healthy grape berries (Zhu et al. 2021) and the players in cluster rot interactions (Carmichael et al. 2019; Hall et al. 2019). Nevertheless, there remains enormous potential to screen the fruit microbiome for beneficial microbes that suppress disease on the grape berry surface and to understand the environmental cues that influence the epidemiology of these disease complexes (Hall et al. 2018a).

Fig. 7. Variability in symptoms and severity in **A,** a moderately susceptible *Vitis vinifera* table grape and **B,** a moderately resistant interspecific hybrid wine grape after inoculation with water (far left) or one of five different isolates of *Botrytis*.

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Literature Cited

Abu-Zahra, T., and Salameh, N. 2012. Influence of gibberellic acid and cane girdling on berry size of black magic grape cultivar. Middle East J. Sci. Res. 11: 718-722.

Alzohairy, S., and Miles, T. 2020. Michigan Grape Facts: Managing Botrytis Bunch Rot. https://www.canr.msu.edu/grapes/uploads/files/BotrytisBunchRot-final.pdf

Alzohairy, S. A., Gillett, J., Saito, S., Naegele, R., Xiao, C. L., and Miles, T. D. 2021. Fungicide resistance profiles of *Botrytis cinerea* isolates from Michigan vineyards and development of a TaqMan assay for detection of fenhexamid resistance. Plant Dis. 105:285-294.

Anco, D. J., Madden, L. V., and Ellis, M. A. 2013. Effects of temperature and wetness duration on the sporulation rate of *Phomopsis viticola* on infected grape canes. Plant Dis. 97:579-589.

Armijo, G., Schlechter, R., Agurto, M., Muñoz, D., Nuñez, C., and Arce-Johnson, P. 2016. Grapevine pathogenic microorganisms: Understanding infection strategies and host response scenarios. Front. Plant Sci. 7:382.

Atallah, J., Teixeira, L., Salazar, R., Zaragoza, G., and Kopp, A. 2014. The making of a pest: The evolution of a fruit-penetrating ovipositor in *Drosophila suzukii* and related species. Proc. R. Soc. B 281:20132840.

Atucha, A., Hedtcke, J., and Workmaster, B. 2018. Evaluation of cold-climate interspecific hybrid wine grape cultivars for the upper Midwest. J. Am. Pomol. Soc. 72:80-93.

Azmi, O. R., and Seppelt, R. D. 1998. The broad-scale distribution of microfungi in the Windmill Islands region, continental Antarctica. Polar Biol. 19: 92-100.

Barata, A., González, S., Malfeito-Ferreira, M., Querol, A., and Loureiro, V. 2008. Sour rot-damaged grapes are sources of wine spoilage yeasts. FEMS Yeast Res. 8:1008-1017.

Barata, A., Malfeito-Ferreira, M., and Loureiro, V. 2012a. Changes in sour rotten grape berry microbiota during ripening and wine fermentation. Int. J. Food Microbiol. 154:152-161.

Barata, A., Santos, S. C., Malfeito-Ferreira, M., and Loureiro, V. 2012b. New insights into the ecological interaction between grape berry microorganisms and *Drosophila* flies during the development of sour rot. Microb. Ecol. 64: 416-430.

Barnes, E. H. 1979. Black rot of grape. Pages 209-213 in: Atlas and Manual of Plant Pathology. Springer, Boston, MA, U.S.A.

Becker, C. M., and Pearson, R. C. 1996. Black rot lesions on overwintered canes of *Euvitis* supply conidia of *Guignardia bidwellii* for primary inoculum in spring. Plant Dis. 80:24-27.

Bokulich, N. A., Thorngate, J. H., Richardson, P. M., and Mills, D. A. 2014. Microbial biogeography of wine grapes is conditioned by cultivar, vintage, and climate. Proc. Natl. Acad. Sci. U.S.A. 111:E139-E148.

Bondada, B., Covarrubias, J. I., Tessarin, P., Boliani, A. C., Marodin, G., Marodin, G., and Rombola, A. D. 2016. Postveraison shoot trimming reduces cluster compactness without compromising fruit quality attributes in organically grown Sangiovese grapevines. J. Enol. Vitic. 67:206-211.

Bowen, P., Menzies, J., Ehret, D., Samuels, L., and Glass, A. D. M. 1992. Soluble silicon sprays inhibit powdery mildew development on grape leaves. J. Am. Soc. Hortic. Sci. 117:906-912.

Bugaret, Y. 1984. L'Excoriose de la vigne: Recherches sur le *Phomopsis viticola* Sacc., nouvelles possibilités de lutte. Ph.D. thesis, Université de Bordeaux, Bordeaux, France.

Carmichael, P. C., Siyam, N., Chidamba, L., and Korsten, L. 2019. Exploring the microbial communities associated with *Botrytis cinerea* during berry development in table grape with emphasis on potential biocontrol yeasts. Eur. J. Plant Pathol. 154:919-930.

Coley-Smith, J. R., Jarvis, W. R., and Verhoeff, K. 1980. The Biology of Botrytis. Academic Press, London, U.K.

Cornell University. French-American and Other Interspecific Varieties. http://www.hort.cornell.edu/reisch/grapegenetics/bulletin/wine/winetext4.html

Crandall, S. G., Saarman, N., and Gilbert, G. 2020. Fungal spore diversity, community structure, and traits across a vegetation mosaic. Fungal Ecol. 45:100920.

Cui, C., Jiang, J., Ouyang, H., Li, C., Liu, D., and Huang, T. 2015. First report of *Greeneria uvicola* causing bitter rot of grape in China. J. Phytopathol. 163:780-782.

Dalbó, M., Ye, G. N., Weeden, N. F., Wilcox, W. F., and Reisch, B. I. 2001. Marker-assisted selection for powdery mildew resistance in grapes. J. Am. Soc. Hortic. Sci. 126:83-89.

Dean, R., Kan, J. L. V., Pretorius, Z. A., Hammond-Kosack, K. E., Pietro, A. D., Spanu, P. D., Rudd, J. J., Dickman, M., Kahmann, R., Ellis, J., and Foster, G. D. 2012. The Top 10 fungal pathogens in molecular plant pathology. Mol. Plant Pathol. 13:414-430.

Debode, J., Van Hemelrijck, W., Baeyen, S., Creemers, P., Heungens, K., and Maes, M. 2009. Quantitative detection and monitoring of *Colletotrichum acutatum* in strawberry leaves using real-time PCR. Plant Pathol. 58:504-514.

DeLong, J. A., Saito, S., Xiao, C., and Naegele, R. P. 2020. Population genetics and fungicide resistance of *Botrytis cinerea* on *Vitis* and *Prunus* spp. in California. Phytopathology 110:694-702.

Duan, Y. B., Ge, C. Y., Zhang, J. X., and Zhou, M. G. 2014. Development and evaluation of a novel and rapid detection assay for *Botrytis cinerea* based on loop-mediated isothermal amplification. PLoS One 9:e111094.

Duncan, R. A., Stapleton, J. J., and Leavitt, G. M. 1995. Population dynamics of epiphytic mycoflora and occurrence of bunch rots of wine grapes as influenced by leaf removal. Plant Pathol. 44:956-965.

Ebbenga, D. N., Burkness, E. C., and Hutchison, W. D. 2019. Evaluation of exclusion netting for spotted-wing *Drosophila* (Diptera: Drosophilidae) management in Minnesota wine grapes. J. Econ. Entomol. 112:2287-2294.

Echeverrigaray, S., Scariot, F. J., Fontanella, G., Favaron, F., Sella, L., Santos, M. C., Schwambach, J., Pedrotti, C., and Delamare, A. P. L. 2020. *Colletotrichum* species causing grape ripe rot disease in *Vitis labrusca* and *V. Vinifera* varieties in the highlands of southern Brazil. Plant Pathol. 69:1504-1512.

Eibach, R., and Töpher, R. 2003. Success in resistance breeding: "Regent" and its steps into the market. Acta Hortic. 603:687-691.

English, J. T., Bledsoe, A. M., and Marois, J. J. 1990. Influence of leaf removal from the fruit cluster zone on the components of evaporative potential within grapevine canopies. Agric. Ecosyst. Environ. 31:49-61.

Entling, W., Anslinger, S., Jarausch, B., Michl, G., and Hoffmann, C. 2019. Berry skin resistance explains oviposition preferences of *Drosophila suzukii* at the level of grape cultivars and single berries. J. Pest Sci. 92:477-484.

Entling, W., and Hoffmann, C. 2019. Single and combined effects of *Drosophila suzukii* and *Drosophila melanogaster* on sour rot development in viticulture. J. Appl. Entomol. 144:153-160.

Erincik, O., Madden, L. V., Ferree, D. C., and Ellis, M. A. 2001. Effect of growth stage on susceptibility of grape berry and rachis tissues to infection by *Phomopsis viticola.* Plant Dis. 85:517-520.

Erincik, O., Madden, L. V., Ferree, D. C., and Ellis, M. A. 2003. Temperature and wetness-duration requirements for grape leaf and cane infection by *Phomopsis viticola.* Plant Dis. 87:832-840.

Fermaud, M., and Le Menn, R. 1992. Transmission of *Botrytis cinerea* to grapes by grape berry moth larvae. Phytopathology 82:1393-1398.

Fernández-Ortuño, D., Chen, F., and Schnabel, G. 2012. Resistance to Pyraclostrobin and Boscalid in *Botrytis cinerea* isolates from strawberry fields in the Carolinas. Plant Dis. 96:1198-1203.

Fernández-Ortuño, D., Grabke, A., Li, X., and Schnabel, G. 2015. Independent emergence of resistance to seven chemical classes of fungicides in *Botrytis cinerea.* Phytopathology 105:424-432.

Fillinger, S., Ajouz, S., Nicot, P. C., Leroux, P., and Bardin, M. 2012. Functional and structural comparison of pyrrolnitrin- and iprodione-induced modifications in the class III histidine-kinase *Bos1* of *Botrytis cinerea*. PLoS One 7:e42520.

Fu, P., Tian, Q., Lai, G., Li, R., Song, S., and Lu, J. 2019. *Cgr1*, a ripe rot resistance QTL in *Vitis amurensis* 'Shuang Hong' grapevine. Hortic. Res. 6: 67.

Fuller, K. B., Alston, J. M., and Sambucci, O. S. 2014. The value of powdery mildew resistance in grapes: Evidence from California. Wine Econ. Policy 3:90-107.

Gabler, F. M., Smilanick, J. L., Mansour, M., Ramming, D. W., and Mackey, B. E. 2003. Correlations of morphological, anatomical, and chemical features of grape berries with resistance to *Botrytis cinerea.* Phytopathology 93:1263-1273.

Garfinkel, A. R., Coats, K. P., Sherry, D. L., and Chastagner, G. A. 2019. Genetic analysis reveals unprecedented diversity of a globally-important plant pathogenic genus. Sci. Rep. 9:6671.

Giang, T., He, J., Belaidi, S., and Scholz, H. 2017. Key odorants regulate food attraction in *Drosophila melanogaster.* Front. Behav. Neurosci. 11:160.

Gillett, J. M., Sysak, R. W., and Miles, T. D. 2018. Evaluation of fungicide programs for control of sour rot in grapes, 2017. Plant Dis. Manage. Rep. 12: PF060.

Gilligan, T. M., Epstein, M. E., Passoa, S. C., Powell, J. A., Sage, O. C., and Brown, J. W. 2011. Discovery of *Lobesia botrana* (Denis & Schiffermuller) in California: An invasive species new to North America (Lepidoptera: Tortricidae). Proc. Entomol. Soc. Wash. 113:14-30.

Glass, N. L., and Donaldson, G. C. 1995. Development of primer sets designed for use with the PCR to amplify conserved genes from filamentous ascomycetes. Appl. Environ. Microbiol. 61:1323-1330.

Gomes, R. R., Glienke, C., Videira, S. I. R., Lombard, L., Groenewald, J. Z., and Crous, P. W. 2013. *Diaporthe*: A genus of endophytic, saprobic and plant pathogenic fungi. Persoonia 31:1-41.

González-Centeno, M. R., Chira, K., Miramont, C., Escudier, J. L., Samson, A., Salmon, J. M., Ojeda, H., and Teissedre, P. L. 2019. Disease resistant bouquet vine varieties: Assessment of the phenolic, aromatic, and sensory potential of their wines. Biomolecules 9:793.

Grabke, A., Fernández-Ortuño, D., Amiri, A., Li, X., Peres, N. A., Smith, P., and Schnabel, G. 2014. Characterization of iprodione resistance in *Botrytis cinerea* from strawberry and blackberry. Phytopathology 104:396-402.

Greer, L. A., Harper, J. D. I., Savocchia, S., Samuelian, S. K., and Steel, C. C. 2011. Ripe rot of south-eastern Australian wine grapes is caused by two species of *Colletotrichum: C. acutatum* and *C. gloeosporioides* with differences in infection and fungicide sensitivity. Aust. J. Grape Wine Res. 17:123-128.

Greer, L. A., Harper, J. D. I., and Steel, C. C. 2014. Infection of *Vitis vinifera* (cv Chardonnay) Inflorescences by *Colletotrichum acutatum* and *Greeneria uvicola.* J. Phytopathol. 162:407-410.

Gregory, C. T. 1913. A rot of grapes caused by *Cryptosporella viticola*. Phytopathology 3:20-23.

Gubler, W., Marois, J., Bledsoe, A., and Bettiga, L. 1987. Control of Botrytis bunch rot of grapes with canopy management. Plant Dis. 71:599-601.

Hall, M. E., Loeb, G. M., Cadle-Davidson, L., Evans, K. J., and Wilcox, W. F. 2018a. Grape sour rot: A four-way interaction involving the host, yeast, acetic acid bacteria, and insects. Phytopathology 108:1429-1442.

Hall, M. E., Loeb, G. M., and Wilcox, W. F. 2018b. Control of sour rot using chemical and canopy management techniques. Am. J. Enol. Vitic. 69:342-350.

Hall, M. E., O'Bryon, I., Wilcox, W. F., Osier, M. V., and Cadle-Davidson, L. 2019. The epiphytic microbiota of sour rot-affected grapes differs minimally from that of healthy grapes, indicating causal organisms are already present on healthy berries. PLoS One 14:e0211378.

Hall, M. E., and Wilcox, W. F. 2019. Identification and frequencies of endophytic microbes within healthy grape berries. Am. J. Enol. Vitic. 70:212-219.

Haye, T., Girod, P., Cuthbertson, A. G. S., Wang, X. G., Daane, K. M., Hoelmer, K. A., Baroffio, C., Zhang, J. P., and Desneux, N. 2016. Current SWD IPM tactics and their practical implementation in fruit crops across different regions around the world. J. Pest Sci. 89:643-651.

Hed, B., and Centinari, M. 2021. Gibberellin application improved bunch rot control of vignoles grape, but response to mechanical defoliation varied between training systems. Plant Dis. 105:339-345.

Hed, B., Ngugi, H. K., and Travis, J. W. 2009. Relationship between cluster compactness and bunch rot in Vignoles grapes. Plant Dis. 93:1195-1201.

Hed, B., Ngugi, H. K., and Travis, J. W. 2011. Use of gibberellic acid for management of bunch rot on Chardonnay and Vignoles grape. Plant Dis. 95: 269-278.

Hewitt, W. B. 1974. Rot and bunch rots of grapes. Calif. Exp. Stn. Bull. 868.

Hewitt, W. B. 1979. Conceptualizing in plant pathology. Annu. Rev. Phytopathol. 17:1-14.

Hickey, C., and Hatch, T. 2018. Dormant spur and cane pruning bunch grapevines. Univ. Ga. Coop. Ext. Bull. 1505.

Higgins, D., Hatlen, R. J., Byrne, J., Sakalidis, M. L., Miles, T. D., and Hausbeck, M. 2021. Etiology of halo blight in Michigan hopyards. Plant Dis. 105:859-872.

Hoffman, L. E., and Wilcox, W. F. 2002. Utilizing epidemiological investigations to optimize management of grape black rot. Phytopathology 92:676-680.

Ioriatti, C., Guzzon, R., Anfora, G., Ghidoni, F., Mazzoni, V., Villegas, T. R., Dalton, D. T., and Walton, V. M. 2018. *Drosophila suzukii* (Diptera: Drosophilidae) contributes to the development of sour rot in grape. J. Econ. Entomol. 111:283-292.

Karagöz, K., Ateş, F., Karagöz, H., Kotan, R., and Çakmakçı, R. 2012. Characterization of plant growth-promoting traits of bacteria isolated from the rhizosphere of grapevine grown in alkaline and acidic soils. Eur. J. Soil Biol. 50:144-150.

Karchani-Balma, S., Gautier, A., Raies, A., and Fournier, E. 2008. Geography, plants, and growing systems shape the genetic structure of Tunisian *Botrytis cinerea* populations. Phytopathology 98:1271-1279.

Kenney, P., and Hall, M. 2021. Reducing sour rot spray applications initiated after symptom development does not impact disease control. Catalyst 5:22-28.

Kocsis, M., Csikász-Krizsics, A., Szata, B. É., Kovács, S., Nagy, Á., Mátai, A., and Jakab, G. 2018. Regulation of cluster compactness and resistance to *Botrytis cinerea* with β-aminobutyric acid treatment in field-grown grapevine. Vitis 57:35-40.

Kozhar, O., Larsen, M. M., Grunwald, N. J., and Peever, T. L. 2020. Fungal evolution in anthropogenic environments: *Botrytis cinerea* populations infecting small fruit hosts in the Pacific Northwest rapidly adapt to human-induced selection pressures. Appl. Environ. Microbiol. 86:e02908-e02919.

Kretschmer, M., Leroch, M., Mosbach, A., Walker, A. S., Fillinger, S., Mernke, D., Schoonbeek, H.-J., Pradier, J.-M., Leroux, P., De Waard, M. A., and Hahn, M. 2009. Fungicide-driven evolution and molecular basis of multidrug resistance in field populations of the grey mould fungus *Botrytis cinerea.* PLoS Pathog 5:e1000696.

Kummuang, N., Smith, B. J., Diehl, S. V., and Graves, C. H., Jr. 1996. Muscadine grape berry rot diseases in Mississippi: Disease identification and incidence. Plant Dis. 80:238-243.

Lal, B., and Arya, A. 1982. A soft rot of grape caused by *Phomopsis viticola.* Indian Phytopathol. 35:261-264.

Leroux, P., Fritz, R., Debieu, D., Albertini, C., Lanen, C., Bach, J., Gredt, M., and Chapeland, F. 2002. Mechanisms of resistance to fungicides in field strains of *Botrytis cinerea.* Pest Manage. Sci. 58:876-888.

Lewis, M. T., and Hamby, K. A. 2019. Differential impacts of yeasts on feeding behavior and development in larval *Drosophila suzukii* (Diptera: Drosophilidae). Sci. Rep. 9:13370.

Li, S., Li, Z., Zhao, Y., Zhao, J., Luo, Q., and Wang, Y. 2020. New disease-resistant, seedless grapes are developed using embryo rescue and molecular markers. 3 Biotech 10:4.

Longland, J. M., and Sutton, T. B. 2008. Factors affecting the infection of fruit of V*itis vinifera* by the bitter rot pathogen *Greeneria uvicola.* Phytopathology 98:580-584.

Madden, A. A., Boyden, S. D., Soriano, J. A. N., Corey, T. B., Leff, J. W., Fierer, N., and Starks, P. T. 2017. The emerging contribution of social wasps to grape rot disease ecology. PeerJ 5:e3223.

Maher, M. F., Nasti, R. A., Volbrecht, M., Starker, C. G., Clark, M. D., and Voytas, D. F. 2019. Plant gene editing through de novo induction of meristems. Nat. Biotechnol. 38:84-89.

Mahlein, A. K. 2016. Plant disease detection by imaging sensors—Parallels and specific demands for precision agriculture and plant phenotyping. Plant Dis. 100:241-251.

Marois, J. J., Nelson, J. K., Morrison, J. C., Lile, L. S., and Bledsoe, A. M. 1986. The influence of berry contact within grape clusters on the development of *Botrytis cinerea* and epicuticular wax. Am. J. Enol. Vitic. 37:293-296.

Martinez, F., Corio-Costet, M. F., Levis, C., Coarer, M., and Fermaud, M. 2008. New PCR primers applied to characterize distribution of *Botrytis cinerea* populations in French vineyards. Vitis 47:217-226.

Meunier, M., and Steel, C. C. 2009. Effect of *Colletotrichum acutatum* ripe rot on the composition and sensory attributes of Cabernet Sauvignon grapes and wine. Aust. J. Grape Wine Res. 15:223-227.

Migicovsky, Z., Sawler, J., Money, D., Eibach, R., Miller, A. J., Luby, J. J., Jamieson, A. R., Velasco, D., von Kintzel, S., Warner, J., Wührer, W., Brown, P. J., and Myles, S. 2016. Genomic ancestry estimation quantifies use of wild species in grape breeding. BMC Genomics 17:478.

Miles, T. D., Neill, T. M., Colle, M., Warneke, B., Robinson, G., Stergiopoulos, I., Mahaffee, W. F. 2021. Allele-specific detection methods for QoI fungicide resistant *Erysiphe necator* in vineyards. Plant Dis. 105:175-182.

Molitor, D., Behr, M., Fischer, S., Hoffmann, L., and Evers, D. 2011. Timing of cluster-zone leaf removal and its impact on canopy morphology, cluster structure and bunch rot susceptibility of grapes. OENO One 45:149-159.

Molitor, D., Behr, M., Hoffmann, L., and Evers, D. 2012. Impact of grape cluster division on cluster morphology and bunch rot epidemic. Am. J. Enol. Vitic. 63:508-514.

Molitor, D., and Beyer, M. 2014. Epidemiology, identification and disease management of grape black rot and potentially useful metabolites of black rot pathogens for industrial applications—A review. Ann. Appl. Biol. 165:305-317.

Mosetti, D., Herrera, J., Sabbatini, P., Green, A., Alberti, G., Peterlunger, E., Lisjak, K., and Castellarin, S. D. 2016. Impact of leaf removal after berry set on fruit composition and bunch rot in 'Sauvignon blanc'. Vitis 55:57-64.

Naegele, R. P. 2018. Evaluation of host resistance to botrytis bunch rot in *Vitis* spp. and its correlation with botrytis leaf spot. HortScience 53:204-207.

Naegele, R. P., Delong, J., Abdelsamad, N., Saito, S., Alzohairy, S. A., and Miles, T. D. 2021. Population genetic analyses of *Botrytis cinerea* isolates from Michigan vineyards using a high-throughput marker system approach. Front. Microbiol. 12:705.

Nita, M., Ellis, M. A., Wilson, L. L., and Madden, L. V. 2007. Evaluations of new and current management strategies to control Phomopsis cane and leaf spot of grape. Plant Health Prog. 8.

O'Donnell, A. G., and Whiteley, A. S. 1999. Fluorescent *in situ* hybridization and the analysis of the single cell. Pages 221-235 in: Environmental Monitoring of Bacteria. C. Edwards, ed. Methods in Biotechnology, vol. 1. Humana Press, Totowa, NJ, U.S.A.

Oliveira, M. S., Amiri, A., Zuniga, A. I., and Peres, N. A. 2017. Sources of primary inoculum of *Botrytis cinerea* and their impact on fungicide resistance development in commercial strawberry fields. Plant Dis. 101:1761-1768.

Oo, M. M., and Oh, S.-K. 2017. Identification and characterization of new record of grape ripe rot disease caused by *Colletotrichum viniferum* in Korea. Mycobiology 45:421-425.

Pacifico, D., Squartini, A., Crucitti, D., Barizza, E., Lo Schiavo, F., Muresu, R., Carimi, F., and Zottini, M. 2019. The role of the endophytic microbiome in the grapevine response to environmental triggers. Front. Plant Sci. 10:1256.

Pañitrur-De La Fuente, C., Valdés-Gómez, H., Roudet, J., Acevedo-Opazo, C., Verdugo-Vásquez, N., Araya-Alman, M., Lolas, M., Moreno, Y., and Fermaud, M. 2018. Classification of winegrape cultivars in Chile and France according to their susceptibility to *Botrytis cinerea* related to fruit maturity. Aust. J. Grape Wine Res. 24:145-157.

Pearson, R. C., and Goheen, A. C., eds. 1988. Compendium of Grape Diseases. American Phytopathological Society, St. Paul, MN, U.S.A.

Pedneault, K., and Provost, C. 2016. Fungus resistant grape varieties as a suitable alternative for organic wine production: Benefits, limits, and challenges. Sci. Hortic. (Amsterdam) 208:57-77.

Pereira, G. E., Padhi, E. M. T., Girardello, R. C., Medina-Plaza, C., Tseng, D., Bruce, R. C., Erdmann, J. N., Kurtural, S. K., Slupsky, C. M., and Oberholster, A. 2020. Trunk girdling increased stomatal conductance in Cabernet Sauvignon grapevines, reduced glutamine, and increased malvidin-3-glucoside and quercetin-3-glucoside concentrations in skins and pulp at harvest. Front. Plant Sci. 11:707.

Pfliegler, W. P., Pócsi, I., Győri, Z., and Pusztahelyi, T. 2020. The *Aspergilli* and their mycotoxins: Metabolic interactions with plants and the soil biota. Front. Microbiol. 10:2921.

Pisani, C., Nguyen, T. T., and Gubler, W. D. 2015. A novel fungal fruiting structure formed by *Aspergillus niger* and *Aspergillus carbonarius* in grape berries. Fungal Biol. 119:784-790.

Pscheidt, J. W., and Pearson, R. C. 1989. Effect of grapevine training systems and pruning practices on occurrence of Phomopsis cane and leaf spot. Plant Dis. 73:825-828.

Reisch, B. I., Luce, R. S., and Mansfield, A. K. 2014. 'Arandell'—A disease-resistant red wine grape. HortScience 49:503-505.

Reisch, B. I., Pool, R. M., Peterson, D. V., Martens, M., and Henick-Kling, T. 1979. French-American and other interspecific varieties. In: Wine and Juice Grape Varieties for Cool Climates, Bulletin 223, Cornell Cooperative Extension. http://www.hort.cornell.edu/reisch/grapegenetics/bulletin/wine/winetext4.html

Ren, W., Shao, W., Han, X., and Chen, C. 2016. Molecular and biochemical characterization of laboratory and field mutants of *Botrytis cinerea* resistant to fludioxonil. Plant Dis. 100:1414-1423.

Reynolds, A. G., Veto, L. J., Sholberg, P. L., Wardle, D. A., and Haag, P. 1996. Use of potassium silicate for the control of powdery mildew (*Uncinula necator*) in *Vitis vinifera* L. cultivar Bacchus. Am. J. Enol. Vitic. 47:421-428.

Richter, R., Rossmann, S., Gabriel, D., Töpfer, R., Theres, K., and Zyprian, E. 2020. Differential expression of transcription factor- and further growth-related genes correlates with contrasting cluster architecture in *Vitis vinifera* 'Pinot Noir' and *Vitis* spp. genotypes. Theor. Appl. Genet. 133:3249-3272.

Rinaldi, P. A., Broggini, G. A. L., Gessler, C., Molitor, D., Sofia, J., and Mugnai, L. 2013. *Guignardia bidwellii*, the agent of grape black rot of grapevine, is spreading in European vineyards. Acta Phytopathol. Sin. 43:11.

Rinaldi, P. A., Paffetti, D., Comparini, C., Broggini, G. A. L., Gessler, C., and Mugnai, L. 2017. Genetic variability of *Phyllosticta ampelicida*, the agent of black rot disease of grapevine. Phytopathology 107:1406-1416.

Rodríguez, A., Acosta, A., and Rodríguez, C. 2014. Fungicide resistance of *Botrytis cinerea* in tomato greenhouses in the Canary Islands and effectiveness of non-chemical treatments against gray mold. World J. Microbiol. Biotechnol. 30:2397-2406.

Rombaut, A., Guilhot, R., Xuéreb, A., Benoit, L., Chapuis, M. P., Gibert, P., and Fellous, S. 2017. Invasive *Drosophila suzukii* facilitates *Drosophila melanogaster* infestation and sour rot outbreaks in the vineyards. R. Soc. Open Sci. 4:170117.

Rooney-Latham, S., Janousek, C. N., Eskalen, A., and Gubler, W. D. 2008. First report of *Aspergillus carbonarius* causing sour rot of table grapes (*Vitis vinifera*) in California. Plant Dis. 92:651.

Saito, S., Michailides, T. J., and Xiao, C. L. 2016. Fungicide resistance profiling in *Botrytis cinerea* populations from blueberry in California and Washington and their impact on control of gray mold. Plant Dis. 100:2087-2093.

Saito, S., Michailides, T. J., and Xiao, C. L. 2019. Fungicide-resistant phenotypes in *Botrytis cinerea* populations and their impact on control of gray mold on stored table grapes in California. Eur. J. Plant Pathol. 154:203-213.

Salzman, R. A., Fujita, T., Zhu-Salzman, K., Hasegawa, P. M., and Bressan, R. A. 1999. An improved RNA isolation method for plant tissues containing high levels of phenolic compounds or carbohydrates. Plant Mol. Biol. Rep. 17:11-17.

Samaras, A., Madesis, P., and Karaoglanidis, G. S. 2016. Detection of *sdh*B gene mutations in SDHI-resistant isolates of *Botrytis cinerea* using high resolution melting (HRM) analysis. Front. Microbiol. 7:1815.

Sambucci, O., Alston, J. M., Fuller, K. B., and Lusk, J. 2019. The pecuniary and nonpecuniary costs of powdery mildew and the potential value of resistant grape varieties in California. Am. J. Enol. Vitic. 70:177-187.

Samuel, S., Papayiannis, L. C., Leroch, M., Veloukas, T., Hahn, M., and Karaoglanidis, G. S. 2011. Evaluation of the incidence of the G143A mutation and *cytb* intron presence in the *cytochrome bc-1* gene conferring QoI resistance in *Botrytis cinerea* populations from several hosts. Pest Manage. Sci. 67:1029-1036.

Samuelian, S. K., Greer, L. A., Savocchia, S., and Steel, C. C. 2011. Detection and monitoring of *Greeneria uvicola* and *Colletotrichum acutatum* development on grapevines by real-time PCR. Plant Dis. 95:298-303.

Savocchia, S., Greer, L. A., and Steel, C. C. 2007. First report of *Phomopsis viticola* causing bunch rot of grapes in Australia. Plant Pathol. 56:725.

Schilder, A. M. C., Erincik, O., Castlebury, L., Rossman, A., and Ellis, M. A. 2005. Characterization of *Phomopsis* spp. infecting grapevines in the Great Lakes region of North America. Plant Dis. 89:755-762.

Si Ammour, M., Fedele, G., Morcia, C., Terzi, V., and Rossi, V. 2019. Quantification of *Botrytis cinerea* in grapevine bunch trash by real-time PCR. Phytopathology 109:1312-1319.

Simionato, A. S., Navarro, M. O. P., de Jesus, M. L. A., Barazetti, A. R., da Silva, C. S., Simões, G. C., Balbi-Peña, M. I., de Mello, J. C. P., Panagio, L. A., de Almeida, R. S. C., Andrade, G., and de Oliveira, A. G. 2017. The effect of phenazine-1-carboxylic acid on mycelial growth of *Botrytis cinerea* produced by *Pseudomonas aeruginosa* LV Strain. Front. Microbiol. 8:1102.

Smart, R. E., Dick, J. K., Gravett, I. M., and Fisher, B. M. 1990. Canopy management to improve grape yield and wine quality principles and practices. S. Afr. J. Enol. Vitic. 11:1-17.

Sosnowski, M., Emmett, R. W., Wilcox, W., and Wicks, T. J. 2012. Eradication of black rot (*Guignardia bidwellii*) from grapevine by drastic pruning. Plant Pathol. 61:1093-1102.

Spotts, R. A. 1977. Effect of leaf wetness duration and temperature on the infectivity of *Guignardia bidwellii* on grape leaves. Phytopathology 67: 1378-1381.
Spotts, R. A. 1980. Infection of grape by *Guignardia bidwellii*-factors affecting lesion development, conidia dispersal and conidial populations on leaves. Phytopathology 70:252-255.
Stapleton, J. J. 1992. Leaf removal for nonchemical control of the summer bunch rot complex of wine grapes in the San Joaquin Valley. Plant Dis. 76:205-207.
Steel, C. C., Blackman, J. W., and Schmidtke, L. M. 2013. Grapevine bunch rots: Impacts on wine composition, quality, and potential procedures for the removal of wine faults. J. Agric. Food Chem. 61:5189-5206.
Steel, C. C., Greer, L. A., and Savocchia, S. 2012. Grapevine inflorescences are susceptible to the bunch rot pathogens, *Greeneria uvicola* (bitter rot) and *Colletotrichum acutatum* (ripe rot). Eur. J. Plant Pathol. 133:773-778.
Steiger, D. 2007. Global economic importance of Botrytis protection. Pages 21-26 in: 14th Int. Botrytis Symp. Cape Town, South Africa.
Suarez, M. B., Walsh, K., Boonham, N., and O'Neill, T. 2005. Development of real-time PCR (TaqMan) assays for the detection and quantification of *Botrytis cinerea* in planta. Plant Physiol. Biochem. 43:890-899.
Sun, H., Loeb, G., Walter-Peterson, H., Martinson, T., and Scott, J. G. 2019. Insecticide resistance in *Drosophila melanogaster* (Diptera: Drosophilidae) is associated with field control failure of sour rot disease in a New York vineyard. J. Econ. Entomol. 112:1498-1501.
Sutton, J. C. 1998. Botrytis fruit rot (gray mold) and blossom blight. Pages 28-31 in: Compendium of Strawberry Diseases, 2nd Ed. J. L. Maas, ed. American Phytopathological Society, St. Paul, MN, U.S.A.
Tello, J., and Ibáñez, J. 2018. What do we know about grapevine bunch compactness? A state-of-the-art review. Aust. J. Grape Wine Res. 24:6-23.
Thiessen, L. D., Keune, J. A., Neill, T. M., Turechek, W. W., Grove, G. G., and Mahaffee, W. F. 2016. Development of a grower-conducted inoculum detection assay for management of grape powdery mildew. Plant Pathol. 65: 238-249.
Tjamos, S. E., Antoniou, P. P., Kazantzidou, A., Antonopoulos, D. F., Papageorgiou, I., and Tjamos, E. C. 2004. *Aspergillus niger* and *Aspergillus carbonarius* in Corinth Raisin and wine-producing vineyards in Greece: Population composition, Ochratoxin A production and chemical control. J. Phytopathol. 152:250-255.
Todaro, T., and Miles, T. D. 2018. Early season vineyard disease management. Michigan State University Extension and Department of Plant, Soil and Microbial Sciences. https://www.canr.msu.edu/news/early_season_vineyard_disease_management
Tomlinson, J. A., Dickinson, M. J., and Boonham, N. 2010. Detection of *Botrytis cinerea* by loop-mediated isothermal amplification. Lett. Appl. Microbiol. 51:650-657.
UC IPM. 2017. Summer Bunch Rot (Sour Rot). University of California Agriculture and Natural Resources, Statewide Integrated Pest Management Program. https://www2.ipm.ucanr.edu/agriculture/grape/summer-bunch-rot-sour-rot/
Úrbez-Torres, J. R., Peduto, F., Smith, R. J., and Gubler, W. D. 2013. Phomopsis dieback: A grapevine trunk disease caused by *Phomopsis viticola* in California. Plant Dis. 97:1571-1579.
Vail, M. E., and Marois, J. J. 1991. Grape cluster architecture and the susceptibility of berries to *Botrytis cinerea.* Phytopathology 81:188-191.
Van Timmeren, S., Fanning, P. D., Schöneberg, T., Hamby, K., Lee, J., and Isaacs, R. 2019. Exploring the efficacy and mechanisms of a crop sterilant for reducing infestation by spotted-wing *Drosophila* (Diptera: Drosophilidae). J. Econ. Entomol. 113:288-298.
Viret, O., Keller, M., Jaudzems, V. G., and Cole, F. M. 2004. *Botrytis cinerea* infection of grape flowers: Light and electron microscopical studies of infection sites. Phytopathology 94:850-857.
Wan, R., Hou, X., Wang, X., Qu, J., Singer, S. D., Wang, Y., and Wang, X. 2015. Resistance evaluation of Chinese wild *Vitis* genotypes against *Botrytis cinerea* and different responses of resistant and susceptible hosts to the infection. Front. Plant Sci. 6:854.
White, T. J., Bruns, T., Lee, S., and Taylor, J. 1990. Amplification and direct sequencing of fungal ribosomal RNA genes for phylogenetics. Pages 315-322 in: PCR Protocols: A Guide to Methods and Applications. M. A. Innis, D. H. Gelfand, J. J. Sninsky, and T. J. White, eds. Academic Press, San Diego, CA, U.S.A.
Wilcox, W. F., Gubler, W. D., and Uyemoto, J. K. 2015a. Compendium of Grape Diseases, Disorders, and Pests, 2nd Ed. American Phytopathological Society, St. Paul, MN, U.S.A.
Wilcox, W. F., Gubler, W. D., and Uyemoto, J. K. 2015b. Part IV. Grapevine Disease Management. Pages 177-198 in: Compendium of Grape Diseases, Disorders, and Pests, 2nd Ed. American Phytopathological Society, St. Paul, MN, U.S.A.
Würz, D. A., Rufato, L., Bogo, A., Allebrandt, R., Pereira de Bem, B., Marcon Filho, J. L., Brighenti, A. F., and Bonin, B. F. 2020. Effects of leaf removal on grape cluster architecture and control of botrytis bunch rot in Sauvignon Blanc grapevines in Southern Brazil. Crop Prot. 131:105079.
Zarco-Tejada, P., Camino, C., Beck, P., Calderon, R., Hornero, A., Hernandez-Clemente, R., Kattenborn, T., Montes-Borrego, M., Susca, L., Morelli, M., Gonzalez-Dugo, V., North, P. R. J., Landa, B. B., Boscia, D., Saponari, M., and Navas-Cortes, J. A. 2018. Previsual symptoms of *Xylella fastidiosa* infection revealed in spectral plant-trait alterations. Nat. Plants 4:432-439.
Zhang, X., Cheng, Z., Ma, L., and Li, J. 2017. A study on accumulation of volatile organic compounds during ochratoxin a biosynthesis and characterization of the correlation in Aspergillus carbonarius isolated from grape and dried vine fruit. Food Chem. 227:55-63.
Zhu, L., Li, T., Xu, X., Shi, X., and Wang, B. 2021. Succession of fungal communities at different developmental stages of Cabernet Sauvignon grapes from an organic vineyard in Xinjiang. Front. Microbiol. 12:718261.
Zoecklein, B. W., Wolf, T. K., Duncan, N. W., Judge, J. M., and Cook, M. K. 1992. Effects of fruit zone leaf removal on yield, fruit composition, and fruit rot incidence of Chardonnay and White Riesling (*Vitis vinifera* L.). Grapes. Am. J. Enol. Vitic. 43:139-148.

Sharifa Crandall, Ph.D.

Dr. Crandall is an Assistant Professor in the Department of Plant Pathology and Environmental Microbiology at Penn State University. Her research program centers on soilborne fungal and oomycete dynamics and asks how microbial communities assemble and interact with plant pathogens across ecological scales. Her lab uses a range of molecular tools to identify and quantify microbiomes and their function, from high-throughput sequencing and metagenomics to comparative genomics. She received her B.A. in Integrative Biology from the University of California, Berkeley, an M.S. degree in Forest Science from Yale University, and her Ph.D. from the University of California, Santa Cruz in Fungal Ecology and Management. Prior to joining Penn State, she held post-doctoral positions at California State University Monterey Bay (Seaside, CA) where she researched the synergistic effect of avocado root rot, soil salinity, and microbiomes and at the United States Department of Agriculture–Agricultural Research Service (Salinas, CA), where she developed assays for molecular diagnostics of downy mildew on cucurbit and hop; she also maintained a pathogen and endophyte DNA sequence database for the *Fusarium oxysporum* complex. When not at work, you can find Dr. Crandall foraging for mushrooms with family and friends.

Jamie Spychalla

Jamie Spychalla is a Ph.D. student in the Department of Plant Pathology and Environmental Microbiology at Penn State University. She is researching the ecology and management of sour rot disease as a graduate student in Dr. Sharifa Crandall's lab. Jamie is also a recipient of the American Society for Enology and Viticulture scholarship. She completed her B.S. degree in Molecular Biology and Spanish at the University of Wisconsin-Madison.

Uma Crouch

Uma Crouch is a Master's student in the Department of Plant Pathology and Environmental Microbiology at Penn State University and is part of the Crandall Lab. She received her B.Sc. in Ecology, Evolution, and Organismal Biology at California State University, Monterey Bay and then worked as a biological aid at the United States Department of Agriculture–Agricultural Research Service (Salinas, CA) until starting graduate school. Her current research focuses on developing intraspecies-specific molecular detection tools for the major fungal pathogen causing Verticillium wilt on potatoes, *Verticillium dahliae*, as well as exploring the pathogen's role as an endophyte in asymptomatic weed hosts.

Flor Acevedo, Ph.D.

Dr. Acevedo is an Assistant Professor of Entomology at Penn State University. Her research centers on understanding basic mechanisms mediating plant–insect interactions with the goal of enhancing host plant resistance. Currently, her research program uses insect pests of grapevines as a model system for various projects that include the identification of herbivore offense mechanisms in native and invasive pests of grape, the characterization of plant resistance traits to insect herbivores, the use of biostimulants in plant protection, the identification and rearing of natural enemies from native herbivore pests, and the use of technological tools to improve pest monitoring and tracking. Dr. Acevedo obtained her bachelor's degree in Agronomy from the University of Caldas (Colombia), and her Ph.D. in Entomology from Penn State University. During her 16 years of research experience, she has been involved in basic and applied research, making significant contributions to insect–plant interactions, chemical ecology, integrated pest management, insect biological control, and the general biology of insects. In addition to research, Dr. Acevedo is involved in extension and teaching.

Rachel Naegele, Ph.D.

Dr. Naegele is a Research Geneticist with the United States Department of Agriculture–Agricultural Research Service (USDA-ARS) in East Lansing, MI. Her research program focuses on abiotic and biotic stress resistance in plants, integrating plant pathology, entomology, breeding, population genetics, and genomics of plants and pathogens. Her love of plants and how they respond to external stresses has been a constant theme in her work. She received her Ph.D. from Michigan State University (MSU) in Plant Breeding, Genetics and Biotechnology working on *Phytophthora capsici* resistance in pepper and eggplant, followed by postdoctoral research experiences working with potato and vegetables (MSU, East Lansing, MI) and watermelon (North Carolina State University, Raleigh, NC) before taking a position with the USDA-ARS in Parlier, CA, where she worked on grape and grape pathogens starting in 2015. She currently works with sugarbeet genetics and the host response to pathogens and abiotic stress.

Timothy Miles, Ph.D.

Dr. Miles is an Assistant Professor in the Department of Plant, Soil and Microbial Sciences at Michigan State University. His research program focuses on fungal and oomycete pathogens on small fruit and hops covering a broad range of topics such as fungal genomics, fungicide resistance, metagenomics, postharvest diseases, and molecular diagnostics. He received his B.Sc. degree in Biology at Western Michigan University in Kalamazoo, MI and completed a Ph.D. from Michigan State University (MSU) in 2011 on anthracnose fruit rot of blueberry. Afterward, he held postdoctoral positions at the University of Idaho (Aberdeen, ID) and the United States Department of Agriculture–Agricultural Research Service (Salinas, CA), both focusing on molecular diagnostics of various plant pathogens (primarily *Phytophthora, Pythium*, and *Rhizoctonia* spp.). He then was an Assistant Professor at California State University–Monterey Bay, where he worked with undergraduate students on various molecular diagnostic projects for plant pathogens. He returned to MSU in 2018 and, currently, his research and extension interests are focused on developing and implementing the best disease management practices.