

# FINGER LAKES

## *Vineyard Notes*



Cornell University  
Cooperative Extension

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## GRAPE DISEASE CONTROL, 2013

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It's (past) time for the annual update and review on controlling fungal diseases of grapes in our eastern climate. As always, I'd like to acknowledge the outstanding team of grape pathologists here in Geneva, including faculty colleagues (David Gadoury and Bob Seem); research technicians (the phoenix-like Duane Riegel, Judy Burr, Dave Combs); and graduate students and post-docs too numerous to mention. It's the combined research efforts of all of these people that serve as the basis for most of the following.

### FUNGICIDE CHANGES & NEWS

**New products.** A slow year on this front. Vivando, Luna Experience (still no label for NY in 2013), and several downy mildew-specific products (Forum, Presidio, Ranman, Reason) were discussed in detail last year; anyone wanting that information can click on [www.fruit.cornell.edu/grape/pdfs/Wilcox-Grape%20Disease%20Control%202012.pdf](http://www.fruit.cornell.edu/grape/pdfs/Wilcox-Grape%20Disease%20Control%202012.pdf).

However, there a couple of new products worth noting:

**a. Torino** is a new powdery mildew-specific product that received EPA registration near the end of 2012. **Although legal to apply virtually everywhere else in the U.S., it is not yet registered in NY and is not likely to be for the current growing season.** Torino represents a new class of chemistry with a mode of action unrelated to any other product on the market. Thus, it is not cross-resistant to anything else out there and provides an effective new option for rotational spray programs designed to limit exposure to any one class of fungicides. Of course, this is the heart and soul of any resistance-management program.

Tprino is labeled for use on grapes at a rate of 3.4 fl oz/A. It's a pretty clean material, with just a 4 hr REI and a 3 day PHI. It is labeled for a maximum of two applications per year, so is obviously marketed for use within rotational spray programs.

We've worked with Torino in multiple field trials for nearly a decade across three different vineyards, in addition to greenhouse trials to investigate its protective and post-infection properties. The latter showed us that it has both, with post-infection properties equivalent to the DMIs in their heyday before resistance started appearing: good control when applied thoroughly at labeled rates 3 to 4 days after spores first land on the leaves.

The field trials have shown significant activity, sometimes in the same league as the top products, sometimes just a notch below. The 3.4 fl oz/A rate is not a "rich" one that you can cut safely. My bottom line: This is a good to very good product, but not a "big gun". It should have utility when used within a rotational program as the label intends, but it would not be my first choice during the bloom/early post-bloom period that's critical for cluster disease control. Its short PHI and powdery mildew specificity (as in, not active against yeasts) might make it attractive if/when sulfur use gets iffy and PM control is still desired (see below in the PM section).

**b. Botector** is a new biopesticide active against Botrytis, which has both federal (EPA) and NY registration. And it did a very good job in my trial last year (admittedly not a high pressure year for Botrytis), which is first year that I worked with it.

Botector is a living preparation of a yeast-like fungus (*Auerobasidium pullulans*), whose manufacturer claims that it works by competing with the *Botrytis* fungus for colonization sites on grape tissues. Which means that it needs to be applied in advance (how long?) of a potential infection event (recall that all of our standard *Botrytis* fungicides except Flint have at least 1 or 2 days worth of post-infection activity as well). It is labeled for use at a rate of 10 oz/A for up to three applications per year, with a 4-hr REI and a 0-day PHI. The current label conservatively specifies not to apply it with “other chemicals” (it is a live product, after all). However, the manufacturer has conducted some tests to determine some compatibilities and these are ongoing, see [http://www.bio-ferm.com/fileadmin/user\\_upload/content/produkte/botector/Miscibility\\_of\\_A\\_\\_pullulans\\_04\\_12\\_2012\\_.pdf](http://www.bio-ferm.com/fileadmin/user_upload/content/produkte/botector/Miscibility_of_A__pullulans_04_12_2012_.pdf) for details if interested (focus on active ingredient names rather than European product names).

## FUNGICIDE RESISTANCE

A number of fungicides that were once highly active have lost their efficacy against certain pathogens in some vineyards as the result of that fungus developing resistance to these materials. It is extremely likely that this phenomenon will continue to increase in importance into the future, as modern fungicides are almost always more prone to resistance development than the old traditional, multi-site products like mancozeb, captan, copper, sulfur, etc. Paying attention to basic resistance management principles and practices from the get-go will be essential to sustaining the utility of virtually any new product that we are likely to see and want to use.

Simply put, anything new that's going to get registered now and into the future has to be squeaky clean in the tests used to assess any possible effects against what are euphemistically termed “non-target organisms” (i.e., you, me, and other life forms that we don't wish to harm beyond disease-causing fungi). To get that ideal, rare mix of being deadly to target fungi and (nearly) benign to everything else, it generally comes to pass that the compound affects a single process in the fungal metabolism, and often one specific site of one specific fungal enzyme that's involved in this process. This is the so-called “lock and key” analogy, where the fungicide molecule “key” physically fits into the fungal enzyme “lock” and prevents it from functioning, thereby killing the pathogen. The upside to such activity is that these materials are often very effective at controlling disease yet very non-toxic to (most) non-target organisms. The downside is that the fungus only has to make a subtle change to that one lock so that the key no longer fits, making the fungicide ineffective. If this change does not significantly impair the functioning of the enzyme, the fungus survives treatment and

reproduces to form progeny that also have the altered “lock”. The result is that we end up with an altered population that now has resistance to the fungicide and all related materials that work by fitting into the same target site.

Within an agricultural context, fungicide resistance is a classic example of evolution, i.e., it is the result of the selection of specific individuals from within the entire pathogen population that are best able to survive and reproduce when exposed to that material. When the proportion of such individuals within the vineyard population increases to the point that the material no longer provides an acceptable level of disease control even when applied properly (time, coverage, rate), a condition termed “practical resistance” is reached. The risk of this occurring is a function of both the fungicide itself (the biochemical basis of the resistance response and its probability of occurring) and the individual disease involved.

Diseases at greatest risk for practical resistance development are those caused by pathogens with the potential for multiple generations per year (short incubation periods) and which also produce large number of spores that can be widely disseminated by air currents (spread the love!). Unfortunately for grape growers, the poster children for this phenomenon are powdery and downy mildews, with *Botrytis* just a half step behind. In contrast, diseases at least risk are those with a limited number of annual disease cycles, caused by pathogens with limited potentials for dispersal. *Phomopsis* cane and leaf spot is a prime example of this type, having but one disease cycle (fungal generation) per year and spores that are distributed only very short distances by splashing rain. Black rot lies somewhere in the middle, having a generation period that's several times longer than those of the mildews, a limited period of susceptibility for the host tissue that is likely to perpetuate the fungus between years (berries), and the type of spore that spreads the disease is distributed only a short distance by rain splash.

Resistance to a fungicide is termed qualitative when individuals within the pathogen population are either sensitive to the typical range of doses encountered in the field or are virtually immune to even 100 or 1,000 times those levels. In such cases, shifts in the makeup of the population can occur very quickly (“disruptive” shifts), with the resistant individuals becoming predominant within just a few years of use and control failures occurring suddenly and practically without warning.

Examples of qualitative resistance that has occurred among grape pathogens in the eastern US include (i) the benzimidazoles (e.g., Benlate, Topsin-M), with resistance among powdery mildew and *Botrytis* populations becoming common in many locations a human generation ago;

(ii) downy mildew resistance to the Group 11 strobilurin or QoI or fungicides (Abound, Flint, Sovran, one of the two Pristine components, Reason), which is common in the mid-Atlantic and southern states and appears to have shown up in at least some NY locations, although the extent of problems in NY is not well characterized; (iii) powdery mildew resistance to the QoI materials, a problem in some NY vineyards since 2002, noted in a PA about that time, and one that Anton Baudoin at Virginia Tech has been documenting with some regularity in the mid-Atlantic region as well; and (iv) the phenylamides (e.g., Ridomil), where downy mildew resistance is common around the world in regions where these materials have been used with some regularity, although limited use in eastern North America appears to have managed it effectively here, at least so far.

When resistance is quantitative, individuals that are poorly controlled by one dose (or rate, loosely speaking) of the material may be controlled by incremental increases in that dose or by substituting a similar dose of a related material that has greater intrinsic activity (i.e., 1 mg of Fungicide B provides more control than 1 mg of Fungicide A even when resistance is not an issue). In this case, the sensitivity distribution within the pathogen population shifts incrementally after repeated use of the same class of materials, with progressively greater proportions of the fungal individuals requiring progressively higher doses of the material for a given level of control ("displacement" shifts).

A well-characterized example of quantitative resistance is that to the Group 3 demethylation inhibiting (DMI) fungicides among populations of the powdery mildew fungus, which we have been discussing for many years now. A recent and very practical illustration of the importance of the effect of such displacement shifts, and how the concept of spray "activity" (determined by both the dose of a particular fungicide and its intrinsic activity) is provided by our experiences with two common DNMI fungicides, myclobutanil (Rally) and difenoconazole (a component of Revus Top, Inspire super, and Quadris Top), which will be reviewed below.

Given the preceding, basic resistance management strategies are predicated on two simple principles: (i) Limit the initial selection of resistant individuals (i.e., don't favor their survival over those of susceptible types); and (ii) Limit the reproduction of those resistant individuals that do preferentially survive treatment, so that they do not build up to damaging levels. Specifically:

- Limit the number of selection events, i.e., limit the number of applications of an at-risk fungicide and related products having the same biochemical mode of action. In recent years,

related materials have become much easier to identify and recognize by the Resistance Group number that is now on the front of each label of products that are considered to be at risk for resistance development.

- Limit the size of the pathogen population from which you may be selecting resistant individuals, thereby limiting the potential number of resistant survivors. Translation: try to avoid using a material at high risk of resistance development as a "rescue" treatment against a severe outbreak of the target disease. Of course, you might legitimately wonder about the wisdom of worrying about the future effectiveness of a particular fungicide if your business might not remain viable unless the disease is brought under control now, but look to see if there are other acceptable fungicide options before taking this plunge. You won't go to hell for doing it once, but it's not a sustainable practice.

- Limit the reproduction (buildup and spread) of resistant individuals that have survived exposure to the at-risk fungicide. This can be accomplished several ways:

- (i) Utilize appropriate cultural practices to limit disease development (pathogen reproduction).

- (ii) Rotate with effective, unrelated fungicides while also recognizing that the fewer sequential applications of an at-risk fungicide, the less opportunity for reproduction of resistant individuals before they are controlled by something else. A conservative recommendation is to never apply products in the same Resistance Group twice in a row, that is, always alternate with a different type of material. A more liberal approach would be never to apply them more than two times in a row before rotating. Some labels require that no more than two sequential applications be made, for this very reason.

- (iii) Apply at-risk materials in combination with another unrelated fungicide, either through tank mixing or the use of a pre-packaged product containing two or more unrelated ingredients active against the target organism. Be aware that resistance management efforts dependent upon rotation and/or combination with unrelated fungicides are only as effective as the companion materials themselves: a weak companion material or low use rate will have a limited effect on slowing the reproduction of resistant individuals that have survived exposure to the at-risk ingredient. Examples of this problem abound and are likely to become more common in the future given the current industry trend to launch products that contain more than one active ingredient. Last year's discussion of the new Luna Experience (referenced at the beginning of this tome) provides one of the more recent examples and will be repeated in detail if and when this product is ever cleared for use in NY.



- An additional strategy appropriate to fungicides subject to quantitative resistance (e.g., DMI materials) is to reduce the proportion of the pathogen population that is resistant to any given application of them. This can be done by increasing the activity of the application, either by increasing the rate of the product to a legal maximum or substituting a related fungicide having greater intrinsic activity.

A trial that we conducted on Chardonnay grapes a couple of years ago illustrates this concept vividly. In this particular trial, Rally (active ingredient = myclobutanil) provided virtually ZERO control of powdery mildew on clusters. This once was among the most effective PM fungicides that we had, before the pathogen population “shifted” to become dominated by individuals with far less sensitivity to DMI fungicides than most individuals in the “wild type” population that was present before these materials were introduced. In contrast, several different difenoconazole treatments (Revus Top, Inspire Super) provided 97-100% control of disease severity, even though the per-acre rates of the two DMI active ingredients were equivalent. Why? Our tests showed that difenoconazole is, on average, nearly 40 times more active than myclobutanil on an ounce-versus-ounce basis. Quite simply, the population of the PM fungus in this vineyard has shifted to the point that the maximum label rate of Rally does not provide a high enough dose of myclobutanil to control most individuals on clusters, yet these same fungal individuals are controlled by a similar dose of the more-active difenoconazole.

And don't forget, maximizing spray coverage will also maximize the dose of product encountered by fungal targets at any given rate of application. The fungus only responds to the dose of product on the part of the plant that it's trying to infect, it doesn't care how much you put into the spray tank and deposited somewhere else.

## POWDERY MILDEW (PM) NEWS AND REMINDERS

*Your annual quick review of PM biology with respect to management considerations.*

(i) In eastern North America, the fungus overwinters primarily or entirely (most regions) as minute fruiting bodies (cleistothecia) that form on leaves and clusters during late summer and autumn, then wash onto the bark of the trunk where they survive the winter. Spores are produced within them, and in New York, those of any consequence are discharged between bud break and bloom (more or less) to initiate the disease, after which it can spread rapidly via the millions of new spores produced from each of these

“primary” infections. Thus, the amount of fungus capable of starting disease this year is directly proportional to the amount of disease that developed by the end (almost) of last year. An important consequence of this is that disease pressure will be higher, and PM sprays during the first few weeks of shoot growth are likely to be far more important, in blocks where PM control lapsed last year than in blocks that remained “clean” into September. (In much of the Northeast, cleistothecia resulting from infections that occur after Labor Day are unlikely to mature before temperatures become limiting and/or frost kills the leaves and eliminates their food source.)

The annual illustration of what this means: A while back, we conducted an experiment in a Chardonnay vineyard where we either (a) sprayed up through Labor Day, maintaining a clean canopy throughout the entire season; (b) quit spraying other vines a month earlier, to represent a planting with moderate levels of foliar PM by the end of the season; or (c) quit spraying in early July, to represent a planting where PM control broke down for one reason or another. The next spring, the levels of cleistothecia (number per kilogram of bark) in these treatments were (a) 1,300; (b) 5,300; and (c) 28,700, respectively. Now, consider a hypothetical case where 20% of the overwintering spore load is discharged during the first couple of weeks after bud break (a reasonable scenario, based on published studies). But 20% of what? In the clean treatment (a), this number might be relatively inconsequential; in dirtier treatment (b), it's equal to the entire seasonal supply on the clean vines; and in treatment (c), it's four to five times greater than the entire seasonal supply on the clean vines.

Not surprisingly, these differences can affect the success of the control program (or its required intensity) in the forthcoming season. So what kind of an effect did they have in this particular vineyard? When we intentionally waited until the immediate prebloom period to apply a minimal spray program to these same vines the year after inducing our variable foliar disease levels, the resulting cluster disease severities were (a) 11%, (b) 22%, and (c) 48% of the cluster area infected, respectively, even though all were sprayed exactly the same during the second season. Conclusion: Higher disease in Year 1 = More primary infections to start off Year 2 = Many more new (“secondary”) spores by the time the fruit were formed and highly susceptible to infection = Much heavier disease pressure to “overwhelm” the fungicide spray program that was employed.

(ii) Powdery mildew functions as a “compound interest” type of disease, that is, the final “yield” (amount of disease at the end of the year) is a function of both (a) the initial “deposit” (amount of overwintering inoculum at the start of



the season, whose effect was just illustrated) and (b) the “interest rate”, i.e., how fast the fungus reproduces. This is affected by cultivar susceptibility, the degree to which we manage the disease through cultural and chemical methods, and the environment. On a susceptible cultivar, a few infections can snowball and build up to a whole lot in a very short period of time if conditions are favorable for reproduction of the fungus. The most important environmental factor that governs the rate of reproduction is temperature, with a new generation produced every 5 to 7 days at constant temps between the mid-60's and mid-80's Fahrenheit (more details are provided in the NY and PA Pest Management Guidelines for Grapes, and in an on-line fact sheet). Thus, days in the 80's and nights in the 60's and 70's provide ideal conditions for the fungus 24 hr a day. Conversely, a very cold night or two can seriously set the fungus back, as discussed a little farther below.

(iii) High humidity also increases disease severity, with optimum conditions for development about 85% RH. Although there is no practical threshold level necessary for the disease--PM develops to some extent over the entire range of humidities that we experience--research has shown that disease severity is twice as great at a relative humidity of 80% versus one of 40%. Vineyard sites and canopies subject to poor air circulation and increased microclimate humidity, and seasons with frequent rainfalls, provide a significantly greater risk for PM development than their drier counterparts. Thick canopies and frequent rainfall are also associated with limited sunlight exposure, which greatly increases the risk of disease development in its own right. Collectively these appear to be important environmental variables that distinguish between “easy” and “challenging” PM years (see below).

(iv) Berries are extremely susceptible to infections initiated between the immediate prebloom period and fruit set, then become highly resistant to immune about 2 weeks (Concord) to 4 weeks (*V. vinifera*) later. This is when you use the good stuff and don't cut corners in terms of application frequency and technique. Your annual reminder.

(v) Failure to control even inconspicuous PM infections on the berries can increase the severity of Botrytis and sour rot at harvest, and can promote the growth of wine-spoilage microorganisms (such as *Brettanomyces*) on the fruit. Another annual reminder. There are lots of other ways that these beasts get into grape berries, and providing excellent PM control from pre-bloom right through bunch closing does not guarantee their control on susceptible cultivars, but it's a relatively easy method to eliminate one potential pathway for their attack.

(vi) Powdery mildew is a unique disease in that the causal fungus colonizes its host almost entirely on the surface of the infected tissues, sending little “sinkers” (haustoria) just one cell deep to feed. This makes it subject to control by topical applications of any number of “alternative” spray materials (oils, bicarbonate and monopotassium phosphate salts, hydrogen peroxide, etc.), all of which have little to no effect on other disease-causing fungi, which live down inside the infected tissues where they are sheltered from such sprays. Recall that there are two primary limitations to the aforementioned group of products, which need to be considered if you want to use them effectively: (a) they work by direct physical contact with the fungus, and therefore can only be as efficient as the spray coverage that you provide; and (b) they work primarily in a post-infection/curative mode by killing the fungus right after they hit it, with modest (JMS Stylet Oil) to no (potassium salts) residual activity against any spores that land on the vine after these materials have been applied. This means that they need fairly frequent re-application, or should be tank-mixed with something that provides good protective (forward) activity in order to lengthen the effective spray intervals.

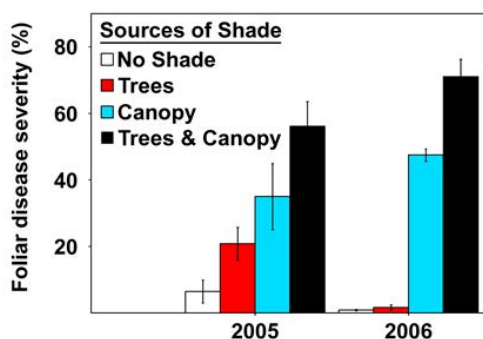
#### *Sort-of new research I: Effect of sunlight exposure*

As noted in previous missives, “it has long been known” that PM is most severe in shaded regions of the vineyard (canopy centers, near trees, etc.), but until recently there was very little work done to determine either the magnitude or cause(s) of this effect. Former graduate student, Craig Austin (now gainfully employed and paying taxes), completed a thorough study of the phenomenon a couple of years ago and showed just how profound this influence can be. To recap:

One Craig's first experiments was conducted in a Chardonnay vineyard near the Finger Lakes village of Dresden (NY), where a small portion of the easternmost row was bordered by a group of 50-foot tall pine trees. In previous years, we had seen PM completely destroy the clusters on the three panels of vines immediately next to the trees, despite a spray program that controlled the disease adequately on all other vines in the block. These panels were shaded during the morning and it wasn't until the sun crested over the treetops just before noon each day that the vines received their first direct exposure to sunlight.

So, we initiated a trial in which Craig inoculated leaves on either (a) the outer (exposed) or (b) inner (shaded) portions of vines, which were located either (i) immediately next to or (ii) 200 feet away from these trees, thereby providing a total of four levels of natural shade. The resulting disease severity increased substantially with each increasing level of shade, becoming 8 to 40 (!) times more severe on the most heavily

shaded leaves (interior of vines next to the trees) compared to the unshaded leaves on the exterior of vines away from the trees (Fig. 1). Although shading could potentially change air temperature or relative humidity within the vine canopy, our measurements did not show this. However, they did show that UV radiation levels and leaf temperatures were dramatically different among the different treatments. Within the shaded regions, UV levels were (as one would expect) a mere fraction of those in the sun, and temperatures of leaves in the sun were as much as 10° to even 30°F higher than those of leaves in the shade. As we later found out, both elevated leaf temperature and UV radiation are responsible for the inhibitory effects of sunlight on PM development.



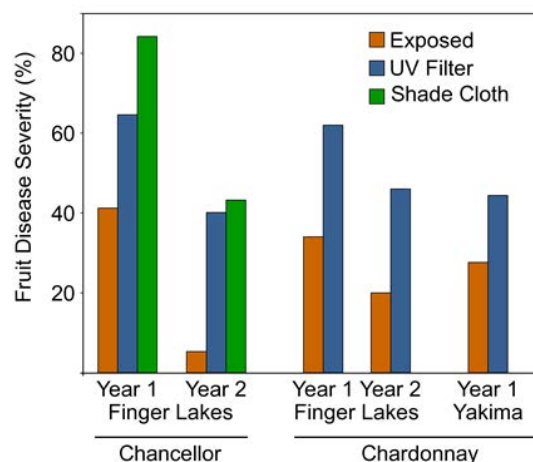
*Figure 1. Percent leaf area diseased on Chardonnay leaves receiving (i) full solar radiation, on the outer canopy edge of vines away from trees (No Shade); (ii) morning shade from a nearby group of pine trees but otherwise exposed to the sun, i.e., leaves on the outer canopy edge of these vines (Trees); (iii) shade provided only by the vine itself, i.e., leaves located within the center of the canopy of vines away from the trees (Canopy); or both tree and the internal canopy shading (Trees & Canopy).*

*Sunlight characteristics influencing powdery mildew development.* UV radiation from the sun can damage the cellular structure of virtually all forms of life. However, powdery mildew is uniquely vulnerable to such damage: as noted previously, the PM fungus lives primarily on the outside of infected tissues, whereas nearly all other pathogens live and grow within infected organs where they are protected from UV. On top of that, the PM fungus is white--it has no pigment ("suntan") to protect against this radiation.

Additionally, direct sunlight heats up exposed leaf and berry surfaces, as it does anything else it hits--as we all know from the difference between standing in the sun or taking two steps away into the shade. On warm days, this additional heat in and of itself can suppress or even kill PM colonies on sun-exposed tissues. Recall that powdery mildew grows best at temperatures near 80°F, but stops growing at temperatures above 90°F and will start to die at temperatures above 95°F, depending on how hot it is and for how long. On a hypothetical spring or summer day in the 80's, the

temperature of shaded leaves and clusters will remain near that of the air--i.e., at or near the optimum for PM development. However, nearby vines or portions thereof that are fully exposed to sunlight often have temperatures elevated to a point where PM growth will stop or even "go backwards".

*Surface Temperature and UV: Field Experiments.* In order to separate these two specific sunlight components, we suspended a Plexiglas "roof" over Chancellor and Chardonnay vines in Geneva, NY and Chardonnay vines in a vineyard at Washington State University's Irrigated Agriculture Research and Extension Center in Prosser, WA (grateful acknowledgement to Dr. Gary Grove and staff for their contributions to this trial). Plexiglas blocks UV radiation while allowing passage of the sun's rays (wavelengths) that elevate surface temperature. At the Chancellor vineyard in Geneva, we also suspended shade cloth over other vines, which shielded them from both UV radiation and the heating rays as well. Clusters were inoculated with PM spores at 75% capfall. As shown in Figure 2, we found that removing UV radiation (Plexiglas filter) increased disease severity on fruit by anywhere from 50% to fivefold (depending on the particular year and trial), for both varieties and locations. The Chancellor shade cloth treatment, which eliminated both the increase in surface temperature *and* UV radiation, further increased disease severity in one of the two experiments, showing that the elevated temperature of sunlight-exposed clusters helped reduce disease severity on them as well.



*Figure 2. Percent cluster disease severity on cv. 'Chancellor' and cv. 'Chardonnay' vines receiving: full solar radiation (Exposed); sunlight from which 95% of the UV radiation had been filtered (UV Filter); or sunlight reduced to 20% of ambient via neutral density shade cloth, (Shade Cloth),. Vineyards were located in Geneva, NY (Finger Lakes) or Prosser, WA (Yakima)*

*Sunlight Manipulation in the Vineyard.* Given that UV radiation and sun exposure reduce PM, how can we use this information to better manage the disease? We examined this question in a young Chardonnay vineyard in Geneva, NY by comparing two training systems, Vertical Shoot Positioning (VSP) and Umbrella-Kniffen (UK), and removing basal leaves around clusters to provide different levels of light exposure in the fruiting zone. UK provided more shoots per linear foot of row than VSP, hence more potential for canopy shading in the fruit zone. Within each training system, Craig removed basal leaves at two dates: 2 weeks post-bloom (fruit set) and 5 weeks post-bloom, taking either one leaf (“Light”) or two leaves (“Heavy”) from above and below the cluster. We inoculated clusters with PM spores at bloom and rated disease severity in each treatment.

We found that both factors affected PM severity (Figure 3). First, powdery mildew severity was lower in the VSP than in the UK training system, regardless of leaf pulling treatment. Second, leaf removal at fruit set significantly reduced the amount of disease in both training systems, but leaf removal 5 weeks after bloom had no effect.

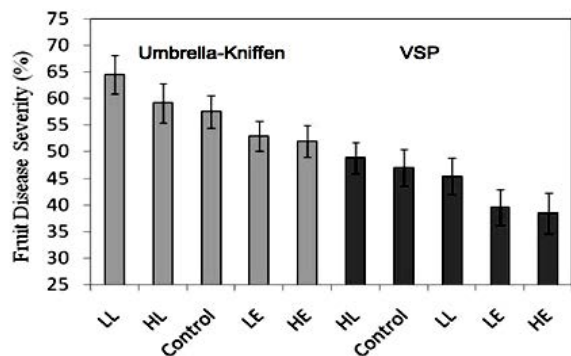


Figure 3. Powdery mildew severity on Chardonnay clusters subjected to five different leaf-removal treatments in each of two vine-training systems. Leaf-removal code: **First letter** is leaf removal severity, H = heavy, L = light (either two leaves or one leaf above and below each cluster, respectively); **Second letter** is leaf removal timing, E = early, L = late (2 and 5 wk post-bloom, respectively). Each data bar represents the mean for 30 clusters per treatment.

The benefits of the early (versus late) leaf removal once again illustrates the critical nature of those first few weeks following the start of bloom--this is when you want to hit the fungus not only with your best spray program but also with the cultural control tools you have available. *Bottom line: simply by utilizing a VSP training system and basal leaf removal at fruit set, we were able to reduce fruit disease severity by 35% relative to UK-trained vines with no leaf removal.* It should be noted that in 2009, a summer during which it sometimes seemed that there was no direct sunlight reaching the state of NY, we did not see the same effect of training system in this vineyard but did see the same effect of early leaf pulling.

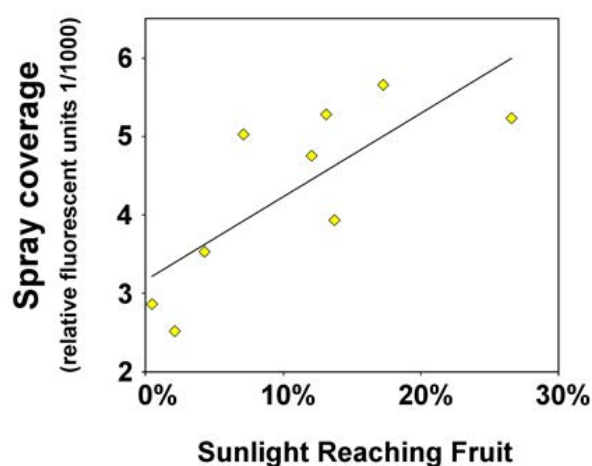


Figure 4. Effect of canopy density on deposition of sprays onto clusters of ‘Chardonnay’ vines treated in mid-July with a conventional airblast sprayer.



*Exposure of fruit to sunlight and pesticides.* It's common sense that canopy management practices that increase sunlight penetration into the fruiting zone should also increase the penetration of sprays applied to control pests and diseases. With the assistance of Dr. Andrew Landers, we were able to quantify the effect that canopy density can have on spray coverage. Vines in our 'Chardonnay' planting subjected to the above canopy manipulations were sprayed with a conventional Berthoud air blast unit and deposition on clusters from each vine was assessed in the lab. As expected, we found a direct relationship between the quantity of spray deposited on each cluster and the sunlight exposure level (Figure 4), with well-exposed clusters receiving approximately twice the deposition as those with poor exposure.

*Management Implications.* For all experiments at all locations, increasing sunlight exposure on leaves or fruit reduced the severity of powdery mildew on those tissues – independent of spray coverage. And when improved spray coverage is factored in, the benefit of canopy management for PM control is not only compounded but extends to other diseases as well. However, a central concept associated with quality viticulture is “balance”. Zero sunlight exposure might lead to diseased berries, but absolute maximum exposure can lead to sunburned berries instead. The optimum level of exposure will vary among vineyards, but the principle is the same. And it's all about balance.

#### *Almost-new research II: What's a bad PM year?*

Former Cornell graduate student Michelle Moyer, working in the lab of Drs. David Gadoury and Bob Seem, also completed her thesis research not too long ago (before becoming gainfully employed as well, as an extension viticulturist at Washington State University), in which she examined some other aspects of powdery mildew biology. Michelle focused on trying to define just what makes a “bad” PM year while it is occurring, so that growers might take action to prevent damage rather than conduct a post-mortem after it's too late.

A few highlights:

- Severe fruit infection is much more likely if the disease become well established on the foliage pre-bloom, providing abundant new spores to infect the adjacent fruit while they're highly susceptible. This is logical, and is consistent with the experiment described earlier concerning variable levels of the overwintering fruiting bodies of the PM fungus (cleistothecia) and their effects on cluster disease the following season. But Michelle nailed it down.
- Relatedly, after analyzing over 25 years worth of climate and disease severity data, Michelle showed a significant association

between severe disease one season and accumulated degree days the previous autumn. Again, this goes back to the same concept concerning the importance of overwintering cleistothecia; this time, it has to do with how many of them mature before leaf drop. Basically, a long, warm autumn allows more late-season PM infections (the ones that form after spray programs relax or stop) an opportunity to develop, viable overwintering spores than does a shorter and cooler fall period.

- We know that PM is favored by warm temperatures, cloudy weather (reduced UV), and high humidity, but is there an easy way to integrate these factors for measurement purposes? Yes. Michelle found a strong relationship between PM severity in any given year and the “pan evaporation” measurements during the critical pre-bloom through fruit set period that year. Pan evaporation is a figure reported by some weather stations that measures--surprise!--the depth of water that evaporates from an exposed pan over a given period of time (my kind of high-tech gadgetry!). Its main purpose is to help schedule irrigations but, conveniently, it also integrates the three major environmental variables that govern PM development--temperature, relative humidity, and solar radiation. A simple decision tree has been suggested for assessing PM severity risk, based upon a combination of post-veraison degree-day accumulation the previous year and pan evaporation data during the critical part of the current growing season.

We're still working on how to bring this all out of the general conceptual realm and into a format that growers and advisors can utilize as part of their disease management decision-making process, but here are a couple of specifics:

- Of the two factors (pan evaporation and heat units the previous fall), the more important is pan evap. Over the past quarter century, our worst years for PM development have been 1986, 1992, and 2003, with 46, 50 and 47% of the cluster area of unsprayed Rosette vines covered with mildew (Rosette is a moderately-susceptible hybrid, not to be confused with highly-susceptible *V. vinifera* cultivars such as Chardonnay). In those years, the average pan evap values were 5.2, 4.5, and 5.4 mm/day from June 1 to July 31. In contrast, two of the years with the least mildew were 1988 with <1% disease severity on unsprayed clusters (!) and 2001 with just 3%; corresponding pan evap values were 6.9 and 5.9 mm/day in these respective years. In 2011, another mild year for powdery mildew, the mean value during this period was 6.7 mm/day.

So, what does this mean in practical terms? It appears that years with pan evap values in June and July > 6 mm/day are likely to be “light” PM years and those with values < 5 mm/

day will be killers. And where do find pan evap data, should you want it? Some weather networks provide this and some weather stations provide a value for a related parameter called “ETO” (potential evapotranspiration); should you want it, you can get pan evap by multiplying ETO x 1.25. Or, you can simply use the computer on top of your neck and factor these general principles (sunny and dry = good for you; cloudy and wet = good for mildew) into your disease control program, as you should be doing by now anyway.

- Another interesting fact: cold nights (below 40°F) throw PM for a loop. After as little as 2 hr at 36°F, portions of existing fungal colonies are killed, new infections take longer to form colonies and produce the secondary spores that spread the disease, and the colonies that do form are reduced in size (hence, less damage and even fewer new spores than if they were larger). Thus, cold nights during the period between early shoot growth and bloom (where we are right now!) have the potential to restrict the ability of the PM fungus to produce new spores capable of infecting the young, highly susceptible berries as they form. Or seen another way, a lack of such nights can give the disease a running start relative to a “normal” year. Note that prolonged cloudy conditions that otherwise favor PM by increasing humidity and limiting exposure to direct sunlight also keep us from getting those chilly spring evenings that typically accompany nice blue-sky days. Something to keep in mind should such conditions come to pass.

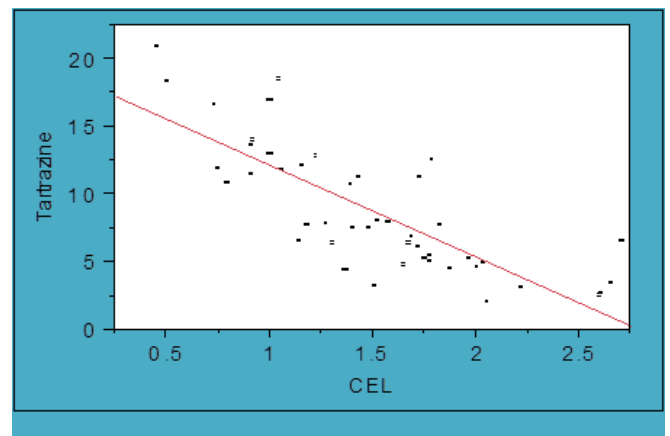
**A note to Concord growers, many of whom have the potential to set a large crop:** Remember that the value of mid-summer control on ConCORDs is very strongly influenced by the combination of crop level and ripening conditions (heat, sunlight), and that foliar PM can be a significant limitation on the vine's ability to photosynthesize and ripen the crop, particularly under otherwise-challenging conditions. When the vine's capacity to do so is not being pushed (moderate crop size, plenty of water and sunshine, few other stresses), research has shown that it can tolerate a lot of foliar PM without significant negative consequences. In contrast, this same research also has shown that at high cropping levels (there were a lot of buds left in most vineyards during pruning, and these are typically carrying two and three clusters each), good PM control can be essential to get the fruit to commercial levels of ripeness. And in cloudy, rainy years—those that give you a double whammy, lousy for ripening and ideal for mildew development—even moderate crops can be affected. Unfortunately, there is no simple formula to tell you how much control is cost effective, and every case is likely to be different, depending on disease pressure, weather conditions, vine vigor, fruit prices, etc. But keep this basic concept in mind.

A minimal two-spray Concord PM program of pre-bloom and 10-14 days later will keep the berries clean and can be good enough in “average” vineyards in a “typical” year, but it certainly is minimal. However, a second or even third post-bloom shot is probably warranted in blocks carrying a heavy crop, especially if ripening conditions are anything less than ideal. You don't get all of that extra fruit for free.

### ***New research III: Another update on spray deposition vs. canopy management.***

As a follow-up to a previously-discussed trial, over the last two summers, we (Andrew Landers, Nicole Landers, and yours truly) looked at the effect of canopy density on the deposition of sprays onto grape clusters in five different Finger Lakes vineyards (cvs. Chardonnay, Vignoles, Cabernet Franc, GR-7, and Rosette). These vineyards were subjected to different canopy management practices, and had different canopy densities. Canopy density was determined in each measured panel of each vineyard on the basis of Cluster Exposure Layer (CEL), the average number of objects (usually leaves) between each test cluster and the sprayer. Spray deposition was determined by measuring the amount of tartrazine (a food-grade dye) on a sample of clusters from the same panels, which were weighed to allow us to “standardize” the deposition among clusters of different sizes (amount of deposit per unit weight of cluster). Then, the average deposition in a panel was graphed as a function of its CEL value.

Results from a spray applied in early July of 2011 are shown in Figure 5, in which data from all five vineyards are combined.



*Figure 5. Effect of canopy density (cluster exposure layers = CEL) on deposition of a spray tracer (tartrazine) onto grape clusters in 5 Finger Lakes vineyards. Vines were treated in early July 2011 with a conventional airblast sprayer applying 50 gpa.*

Although individual data points show the typical variability around the “average” line indicated in red, the relationship between spray deposition and canopy density is clear. For example, clusters blocked from the sprayer by one layer of objects (leaves; CEL = 1.0) received approximately twice as much spray as those blocked by two layers (CEL = 2.0).

Results from 2012 are presented for two different vineyards, in which these trials were conducted both in the pre-bloom period and several weeks later, after young berries had formed and the canopies had filled out further.

Again, we see a huge effect of cluster exposure upon spray deposition, with the deposits sometime reduced by 75% when CEL values were raised from an average of 0.5 (a total of 5 leaves obscuring 10 measured clusters) to an average of 1.5 (a total of 15 leaves obscuring 10 measured clusters). In the GR7 vineyard, we also see that data points are clumped into two distinct groups, corresponding to the two sampling times: there were fewer leaves obscuring clusters in the first (pre-bloom) trial period than the second (postbloom) one, resulting in significantly more deposition pre-bloom.

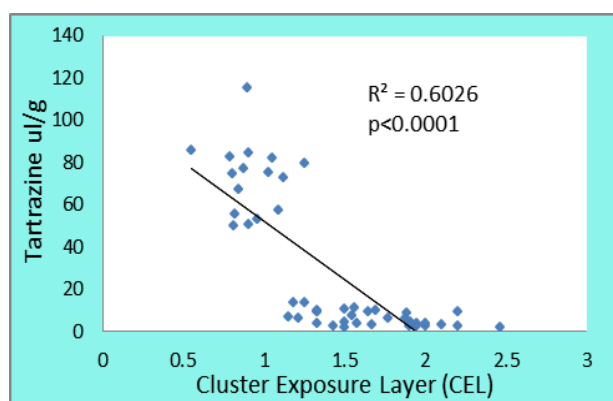


Figure 6. Effect of canopy density (cluster exposure layers = CEL) on deposition of a spray tracer (tartazine) onto grape clusters in vineyard of cv. GR7. Vines were treated *pre- and post-bloom* (June and July) 2012 with a conventional airblast sprayer applying 50 gpa.

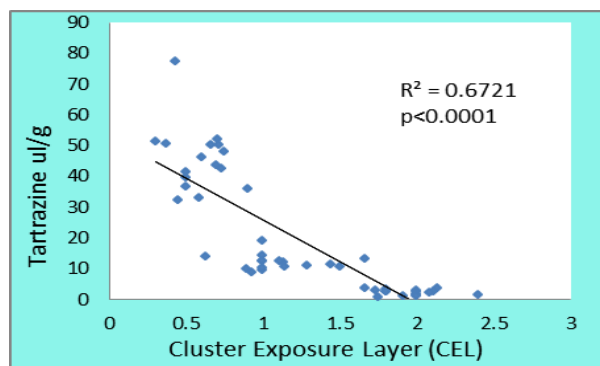


Figure 7. Effect of canopy density (cluster exposure layers = CEL) on deposition of a spray tracer (tartazine) onto grape clusters in vineyard of cv. Vignoles. Vines were treated *pre- and post-bloom* (June and July) 2012 with a conventional airblast sprayer applying 50 gpa.

Obviously this has implications for the management of all diseases and arthropod pests against which you spray, not just PM.

## Fungicides

**Sulfur.** Another summary of the major findings and conclusions from our studies on sulfur activities a few years back. Additional discussion is provided in last year’s version of the newsletter, referenced at the beginning of this one.

- We were unable to demonstrate any negative effects of low temperatures on either the protective or post-infection activities of sulfur (control was the same at 59°F as it was at 82°F when we sprayed with the equivalent of 5 lb/A of Microthiol).
- Sulfur provides very good protective activity on sprayed tissues, but not on new leaves that emerge after the last application. Duh.
- Sulfur provides excellent post-infection control when applied up through the time that young colonies start to become obvious. Although it does have some eradicant activity against raging infections, it's significantly stronger against the younger ones that you still can't see or those that are just starting to become visible. If things reach the “Omigod!” stage, the choice for an eradicant would be. That would be Stylet Oil (or even Oxidate if you're afraid of paying too much income tax after your expenses have been deducted). But don't expect miracles and don't waste your time and money if you can't thoroughly soak things.
- Rainfall of 1 to 2 inches decreases sulfur's protective activity significantly. This effect is more pronounced with generic “wetable” formulations than with so-called “micronized” formulations (e.g., Microthiol), which have smaller particle sizes so adhere better to tissue surfaces. Liquid formulations are probably similar to Microthiol). The negative effects of rainfall can be somewhat compensated for by adding a “spreader-sticker” adjuvant to the spray solution and/or increasing the application rate. See Table 1 on the next page for field data standardized across years to reflect % disease control relative to the unsprayed check vines in the relevant experiment.



Table 1. Powdery mildew control on Rosette (2004-06) and Chardonnay (2007-10) grapes as affected by sulfur rate and adjuvant (Geneva, NY)

Treatment, rate/A	04	'05	'06	'07	'08	'09	'10	2004	'05	'06	'07	'08	'09	'10
Microthiol, 5 lb.....	68	67	86	97	76	70	61	47	76	70	89	90	4	16
Microthiol, 5 lb +														
Cohere, 0.03% (vol)...	84	80	89	97	83	73	64	64	73	79	90	96	4	37
Microthiol, 10 lb.....	87	89	91	99	91	83	77	76	77	85	94	---	6	41
Microthiol, 10 lb +														
Cohere, 0.03% (vol)...	---	---	---	---	95	86	86	---	---	---	---	98	9	65

\* % reduction of the diseased area on leaves and clusters, relative to the unsprayed check treatment.

***“Sort of new” research: Effect of sulfur use patterns on residues at harvest and potentially stinky wines.*** Most winegrowers know that elemental sulfur (S)--the form of sulfur used for controlling PM--can result in the formation of stinky hydrogen sulfide or  $H_2S$  (the smell of rotten eggs or the stuff you blame the dog for after a meal) if residues in the must at the start of fermentation are “excessive”. Although other factors such as yeasts stressed out by poor nutrition can also cause this, high S residues on the harvested fruit are commonly considered the cause when things get stinky, and undoubtedly are some of the time.

The question that’s always asked by winegrowers is, “How late can I spray sulfur and get away with it?” And until recently, my answer always was, “Everyone has an opinion but nobody has any data.” This was largely due to the simple fact that whereas the consistent danger level in must was determined to be 10 parts per million (ppm) or as low as 1 ppm occasionally when researchers spiked clean juice with various concentrations of S before fermentation, there was no practical way of measuring S residues in must when grapes were subjected to different spray practices in the field and subsequently crushed. The work of former graduate student Misha Kwasniewski (recently given his sheep skin and hired as extension enologist at the Univ. of Missouri), who worked under the direction of Gavin Sacks while enduring my prodding, changed that.

Misha and Gavin have reported on the elegantly simple and cheap method that they developed for measuring S on intact clusters or in musts after pressing, and made it available to growers and wineries. Some of you have already used it but those who haven’t and are interested in doing so can contact one of the principles. But here are the take-home messages from what was learned by applying the technique to samples from the field studies that we conducted over a 3-year period:

- Must residues were affected by both the rate and formulation of the sulfur product used. Rates of 5 or 6 lb/A

yielded greater residues than comparable treatments of 2.5 or 3 lb/A (give that man a Ph.D.!). And at a given rate, a micronized formulation (we used Microthiol) yielded greater residues than a wettable powder formulation (we used Yellow Jacket). This also is not surprising, increased tenacity/longer performance is one reason that you pay more for these micronized formulations. But as you get closer to harvest, you might want to cut the rates and use a WP if residues are a concern.

- Regardless of rate and formulation, a cutoff of 5 weeks before harvest always yielded residue levels below the consistent danger threshold. Again, lower rates and the WP formulation sometimes allowed use closer to harvest while still remaining below threshold.

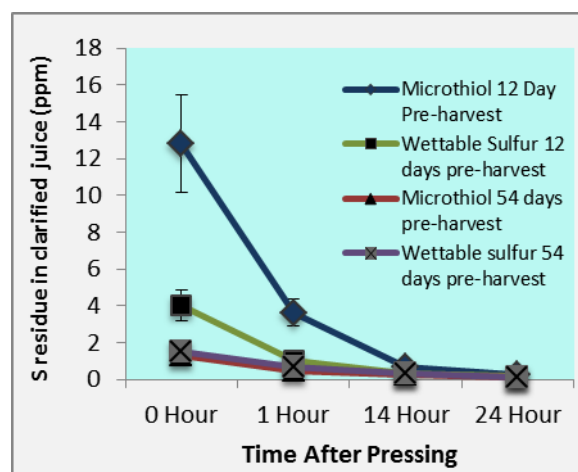


Figure 8. The effect of clarification through settling on elemental sulfur residue present in juice pressed from fruit that received sequential applications of two commercial sulfur formulations (5 lb/A formulated product) during the 2011 season, ceasing either 54 or 12 days before harvest. Samples were obtained from 12 inches below the juice surface, at the post-pressing time intervals indicated. Data for 38- and 25-day PHI treatments were intermediate between those for the 12- and 54-day extremes but are omitted for the sake of simplicity.

- Common white wine vinification practices—i.e., musts were clarified by allowing them to settle after crushing and fermentations were not conducted on the skins—yielded musts with S residues far below 1 ppm at the start of fermentation, even when residues exceeded the 10 ppm threshold immediately after crushing. That is, the S particulates settled out within 24 hr, after which they were found in the sediment rather than the juice. These results are consistent with an obscure 1980 German study that Misha ran across, and strongly suggest that typical white wines should not be stinky as a result of sulfur use in the vineyard. See Figure 8 for a graphic representation of this phenomenon.

**“Alternative” materials.** As noted many times in previous years, there are numerous “alternative” materials labeled (and not) for PM control. And as noted before, the effect of these materials is typically due to their topical activity against the PM fungus growing on the surface of the infected grape tissues, meaning (i) control is predicated entirely upon spray coverage; and (ii) these products seldom control diseases other than PM, and when they do they’re not as effective against those as they are PM, regardless of what the advertising might claim.

Some years back, we compared seven products registered by the EPA and classified as “biopesticides” (Elexa, Kaligreen, Nutrol, Oxidate, Prev-Am, Serenade, Sonata) for control of PM on Rosette vines under two different scenarios: (a) season long, to determine the extent of their activities without any help; and (b) using “standard” material to provide control into the early postbloom period, then switching to the alternative products to maintain disease control on the leaves and cluster stems after the berries had become relatively resistant to infection. Generally, sprays of the biopesticide were applied at 10-day intervals, and a “commercial standard” at the time (rotating a DMI, Pristine, and sulfur at 14-day intervals) was also used for comparison. The bottom lines were:

- When applied throughout the season at 10-day intervals, none of biopesticides were as effective as the standard program at 14-day intervals. However, using standard materials through 10 days postbloom followed by the alternatives provided control of berry infections equivalent to the standard. No surprise, since the prebloom through early post-bloom period is when you get (or don’t get) most all of your control of berry infections.
- There was a wide range in the performance of the biopesticides for keeping foliar disease down in the summer. A few (Nutrol, Kaligreen, and Prev-Am) were nearly as efficacious as the standard program, which relied on sulfur to

finish the season (albeit at 14- rather than 10-day intervals). These may interest some growers who are trying to avoid sulfur in late-season sprays.

- Here’s the annual reminder that potassium bicarbonate products such as Kaligreen, Armicarb, and Milstop have the same mode of action as Nutrol (monopotassium phosphate = dihydrogen potassium phosphate): They suck the water out of PM colonies that they contact, just like what happens if you pour salt on a slug (I was a perverse child and quite fascinated by that phenomenon). Which means that you have to treat an existing infection (even if it’s so small that you can’t see it), so claims of protective activity are “fanciful”, to be polite. And they don’t control other diseases, grow hair on bald men, or improve your love life in later years, regardless of some advertising claims to cure myriad other ills. Prices can vary significantly. Nutrol is not OMRI certified as “organic”, if that’s important to you philosophically or commercially. Also be aware that unlike the bicarbs, which are formulated with a surfactant, you’ll need to add one with Nutrol to get optimal coverage of the entire surface of the leaves and berries.

We’ve done a lot of work with various of these various products over the years, and the bottom line is that they all work the same and provide equivalent control of powdery mildew at label rates, use the cheapest one unless OMRI certification is an issue for you.

## BLACK ROT (BR) NEWS AND REMINDERS

*1. As fruit mature, they become increasingly resistant to infection, spray accordingly.* Another annual reminder. Remember that under NY conditions, berries are highly susceptible to black rot from cap fall until 3-4 weeks (Concord) or 4-5 weeks (Riesling, Chardonnay) later. Then, they begin to lose susceptibility, finally becoming highly resistant to immune after an additional 2 weeks. Note that this means that Concords can become infected up to 5 or 6 weeks after the last cap has fallen, and *V. vinifera* varieties up through 7 weeks post-bloom. In the mythical “average” year, most growers won’t need to be too concerned towards the end of these susceptible periods, since the berries have become quite resistant although not completely immune, the overwintering spore load is long gone by then, and nearly all leaves and berries on the vine are clean and so there is practically no “secondary” source of new spores to spread the disease. However, protection should continue throughout the entire period of susceptibility if infections got started in the vineyard for one reason or another, unless you either know or want to gamble that the weather is going to stay dry until the fruit become fully resistant.

Recall that in most vineyards, mummified berries are far and away the main (and oftentimes, only) overwintering source of

the BR fungus. Spores from mummies on the ground—which is where they should be unless somebody screwed up and didn't prune them off the vine during the dormant season (see below)—are typically depleted by a week or two after bloom. (Now for the CYA fine print: remember that these spores are liberated from the mummies during rains. So, if it doesn't rain from early postbloom until 2 or 3 weeks later, as occasionally happens, the last shot of them will just sit and wait until the rains finally do arrive). Thus, if the disease has been very well controlled by the time the overwintering spores are depleted, there should be no source for new infections even though fruit may still remain susceptible, and additional sprays are not likely to be necessary. In contrast, if new black rot infections are established (and producing spores right within the clusters), protection will need to continue so long as fruit retain any susceptibility.

As often noted, we've regularly obtained excellent control with sprays applied just before the start of bloom plus 2 and 4 weeks later. Such a program provides protection throughout the period of peak susceptibility and during most or all of the time remaining before berries become highly resistant. But as noted above, you get away with stopping sprays before berries are fully resistant when there are few to no active infections present and/or the weather is dry, but growers routinely get nailed when they quit too early and there are active infections present. Also, waiting until the immediate prebloom period is a lot safer in a vineyard that was clean last year than in one with more than a touch of disease and the relatively high overwintering spore load that this will entail. Recognize when you can cut corners and when you can't.

2. *Mummies retained in the canopy provide significantly more pressure for BR development than those dropped to the ground.* Mummies in the canopy produce **many** more spores than those on the ground (as in 10 to 20 times as many) and continue to produce them throughout the period of berry susceptibility, whereas spores from ground mummies are finished shortly after bloom. Furthermore, spores from mummies in the canopy are **much** more likely to land on and infect susceptible berries than are those produced from mummies on the ground, since they are released right next to the new clusters (most of these are dispersed by rain drops, which doesn't work so well for the fungus if the mummies are on the ground). As often noted, when I go into a vineyard and find a BR "hot spot", the first thing I do is look for last year's mummies still hanging in the trellis near the current zone of activity. I almost always find them.

3. *The incubation period for BR can be very long.* Under upstate NY conditions, we've found that clusters infected during the first few weeks after bloom show symptoms about

2 weeks later and that all diseased berries are apparent within 21 days after the infection event. However, clusters infected near the end of their susceptible period do not develop symptoms until 3 to 5 weeks after infection. (Note that since the fungus is responding to accumulated heat units rather than accumulated risings of the sun, these periods will be a tad shorter in significantly warmer climates). In New York vineyards in that mythical average year, black rot that begins to show up in mid- to late August is probably the result of infections that occurred in mid- to late July, depending on the cultivar. This fact should be considered when trying to determine "what went wrong" should such disease occur.

4. *The SI [DMI] fungicides are most effective in "reach-back" activity, whereas the strobilurins are most effective in "forward" activity.* Just a reminder of how these materials work (along with supporting data), and why mixing a DMI + protectant fungicide (mancozeb, ziram, strobie) gives such good BR control—reach-back activity from the DMI plus forward activity from the protectant.

Table 4. Protective and post-infection activities of a strobilurin (Abound) and sterol inhibitor (Nova = Rally) fungicide in control of black rot under field conditions

	% Disease control <sup>c</sup>	
	<u>Abound</u>	<u>Rally</u>
Protective (days) <sup>a</sup>		
5	90	65
8	93	39
11	66	0
Post-infection (days) <sup>b</sup>		
3	39	95
7	42	87
10	15	39

<sup>a</sup> Sprays were applied at label rates to Concord vines in the field at indicated number of days before infection with black rot spores.

<sup>b</sup> Sprays were applied at label rates to Concord vines in the field at indicated number of days after infection with black rot spores.

<sup>c</sup> Percent reduction in the number of diseased berries relative to unsprayed clusters.

5. *Fungicides.* Nova/Rally and Elite were always the kings in



our trials, which haven't been run since we lost our BR test vineyards a few years back. Elite is no longer marketed as such, although I'd assume that the generic tebuconazole products do the same thing if used at an equivalent rate to supply 1.8 oz/A of *active ingredient*. Trials run by colleagues in Ohio and PA show that Mettle (still not labeled in NY, although that may change next year) and the difenoconazole products have similar levels of activity (note that all four of the abovementioned fungicides belong to the same chemical family within the DMIs, the triazoles). In many of our trials, the strobies were right up there at a similar level of performance. Of course, the most important time to control black rot (bloom and early postbloom) is also the critical time for controlling PM on the clusters, and diminishing levels of PM control with most DMI fungicides make them potentially problematical at this critical part of the season in many vineyards. However, if BR is a greater concern than cluster PM (which could be true of many native and hybrid cultivars, and all cultivars in some production regions well to the south of NY), this may not matter so much. And it may be even less of a factor given the superiority of PM control provided to date by the difenoconazole products, although I'd still use them gingerly on Chardonnay and other highly susceptible *V. vinifera* cultivars during this period.

All of the strobies appear to be equivalent to one another and provide very good to excellent control, equal to mancozeb and ziram under moderate pressure and superior under very wet conditions, since they're more rainfast. Of course, rainy conditions are when superior performance against BR is most necessary. (FYI, the non-strobie component of Pristine provides virtually no control of black rot). Mancozeb and ziram are old standards and provide very good control under most commercial conditions. Captan is only fair, and likely to be inadequate if there's any pressure. Copper is discussed below. Sulfur is poor.

6. *Special considerations for "organic" growers.* Black rot is perhaps the "Achilles heel" for organic grape production in the East. In the only good trial that we've run with copper, it provided 40% disease control when applied at 2-week intervals, versus essentially 100% control with Rally/Nova. (I recently found an old report from a trial that Roger Pearson ran in the mid-1980's, where he got control similar to ours when using a copper product). That being said, towards the end of the wet 2006 season I visited an organic grower who had suffered severe losses from BR in several previous wet seasons, anticipating that I'd see more of the same. But I had to search to find a black rot berry. What had he done? He'd implemented a rigorous program to remove mummies during pruning, and sprayed copper once a week throughout much of the growing season. This was pretty hard on some of the

hybrid vines and runs counter to the thinking of many with a "sustainable" orientation (after all, copper is a metallic element that by definition doesn't break down into anything else, so it accumulates in the soil forever), but it did control the disease in a manner that conforms to the letter of the organic law.

Unfortunately, we don't know of any "magic bullets" for organic producers to spray, although there are several products out there that claim to be. Bryan Hed at Penn State has looked at a number of possibilities and we've followed up with a couple of the most promising, but the typical scenario is that things look good in the greenhouse and not so good in the field (most likely, they wash off, among other issues). Right now, it looks like nothing is as good as copper.

Therefore, the simple fact remains that sanitation and cultural practices form the **absolutely critical** first (and second and third....) line/s of defense against BR for growers who wish to produce grapes organically. So if that means you, you'll need to pay strict, bordering on religious, attention to limiting inoculum within the vineyard. Ideally, this would include removing or burying (tillage, mulch) all mummies that you might encounter at the site. The next best option is do this to as many of them as you can. At the very least, it is imperative that all mummified clusters be removed from the trellis during pruning. And if you're able to patrol the vineyard from 2 to 6 weeks after cap fall and prune out any affected clusters or portions thereof before they allow the disease to spread, even better. Note that spores for disease spread during the current season are dispersed by rain primarily within the canopy, so they should pose little risk of causing new infections if said clusters are simply dropped to the ground. (And if dropped this early, they should decompose before next season rolls around, but toss 'em into the between-row aisle where they're most likely to get mowed or buried during cultivation, just to make sure).

## DOWNY MILDEW (DM) NEWS AND REMINDERS

Recall that the DM organism persists in the soil as resting spores (oospores) that originate within infected leaves and berries. Hence, the more infection last year, the more oospores this year and vice versa. Last year was a pretty easy one for downy in many locations, which means that the DM "season" may start in earnest just a little later than it would have otherwise. But by the time you read this, it should just about be upon us.

The first "primary" infections, originating from overwintering oospores in the soil, require a minimum rainfall of approximately 0.1 inch and a temperature of 52°F or higher to "activate" them and splash their infectious progeny into the canopy or onto nearby sucker growth. Of course, heavier

rainfall and warmer temperatures above these minimum values will increase the probability and severity of primary infection.

Once primary infections occur, new "secondary" spores (sporangia) form in the white downy growth visible on infected clusters and, particularly, the underside of infected leaves. Several different weather factors must come together for sporangia to form and spread the disease, but this can occur rapidly when they do. Basically, what's required are humid nights to form the sporangia (warm and humid is even better) with rain following soon thereafter to allow germination and infection. Without rain, most of the ungerminated sporangia will die the next day if exposed to bright sunshine; however, they can survive for several days between rainfalls if conditions remain cloudy, which helps to keep the epidemic running.

Spread is most rapid with night and morning temps of 65-77° F, although it can occur down into the 50's. With an incubation period (generation time) of only 4 to 5 days under ideal conditions, disease levels can increase from negligible to overwhelming in very short order if the weather remains favorable for long stretches of time—repeated humid nights, frequent rains, and extended periods of cloudy weather. See: Summers of 2008 and 2009, August and September 2011.

The erratic development of DM coupled with its explosive and potentially devastating nature makes it an ideal candidate for scouting, especially after fruit have become resistant and the consequences of incomplete control are somewhat less. No need to spray for this when it isn't there, but you don't want to allow it to get rolling once it rears its ugly little head. Keep an eye on the vineyard to see which of these two scenarios you might be able to avoid. For additional guidance, my colleagues, Bob Seem and David Gadoury, have developed a computer model (DMCAST) that integrates a number of weather and crop development factors to advise when infections are likely to occur. This model can be accessed via the NYS IPM Program website ([www.nysipm.cornell.edu/newa/](http://www.nysipm.cornell.edu/newa/)).

*Fruit susceptibility.* Clusters of some varieties—including all *V. vinifera* cultivars—are highly susceptible to infection as soon as the fungus becomes active during the prebloom period (in Geneva, our first infections in high-inoculum vineyard sites typically occur about 2 to 3 weeks before the start of bloom). Recent research indicates that berries become highly resistant to direct infection within about 2 weeks after the start of bloom, although losses due to berry stem infections can occur under some poorly-defined conditions for at least 2 additional weeks after that.

When berry stem infections occur, the DM organism follows that pipeline into the fruit and causes the aptly-termed "leather berry" symptom (hard and dry berry, no DM spores produced upon it). There was a bit of that around a few years back (2008 and 2009), likely due to inadequate protection as people prematurely let their guard down a couple of weeks after bloom. The past three years have been relatively dry during that 2- to 4-wk postbloom period, which makes it easy to forget how these things can happen. But don't.

For many years, the standard fungicide test protocol on hyper-susceptible Chancellor vines in a high-inoculum site at Geneva has been to start spraying about 2+ weeks prebloom and continue through approximately 4 weeks postbloom. The best materials consistently provide virtually complete control of fruit and cluster stem infections using this schedule, even in bad years and on perhaps the worst possible variety for cluster infections, under abnormally high inoculum pressure. But remember that vines with susceptible foliage remain vulnerable to defoliation from DM right into the fall if disease-conducive weather persists, even long after the fruit have lost their susceptibility. Which is something we'd all rather avoid.

*Fungicides.* Ridomil remains the best downy mildew fungicide ever developed, but cost and lack of activity against other diseases have limited its use. But if you get to the point that you're ready to call in the big guns, this is the Howitzer. And as some people have pointed out, one effective application of Ridomil can ultimately be cheaper than two or three shots of something else that just never gets on top of things. Those in regions where potential ground water residues are an issue (that means you, Long Island) should also be aware that Ridomil is especially prone to leaching due to its unusually high solubility in water, and be prepared to address that issue. Although Ridomil is very highly prone to resistance development, this has never been detected on grapes in the U.S., probably due in large part to its somewhat limited use here. Nevertheless, it's a real concern, and all resistance-management precautions should be followed strictly in order to maintain the viability of this "nuclear option" against DM. Remember that the PHI on Ridomil Gold Copper is 42 days versus 66 days for Ridomil Gold MZ.

Note the discussion regarding DM resistance to the strobil and related materials at the beginning of this tome: use them with caution in regions where resistance has not yet become a problem, and think of use in regions where it already has developed as a disease-management form of Russian roulette: odds are that you'll get away with it for awhile, but you don't want to pull that trigger too often. Within this context and in vineyards without resistance, Abound (or Quadris Top) is very good, Pristine is even better, and over the past couple of

years the new product, Reason, has given excellent control in our trials—and it's cheap, although it doesn't control any other diseases. Sovran is marginal and Flint is poor. Copper, mancozeb, and captan are old standards because they work, but are prone to wash-off under heavy rains and may need to be reapplied more frequently in wet years. Ziram is much better than nothing, but it wouldn't be your first choice if good materials were an option. Presidio, Revus, and Ranman are relatively new DM-specific fungicides whose specific activities are not as well characterized yet as I'd like. All three have provided good to excellent control in our trials. From what I can tell, Ranman has primarily protective activity; Revus is strongest in the protective mode but does have some limited (how much? probably not great) post-infection activity; and Presidio provides significant post-infection and protective activity. It's also the most expensive of the three.

Which brings us to the phosphorous acid (also called phosphite and phosphonate) products once again. We've discussed these *ad nauseum* over the past few years, so will only review the main points this time around. Recall that these are excellent materials for anyone consciously seeking a "least toxic" or "sustainable" approach to growing grapes, due to their low toxicity (4 hr REI, exempt from US-EPA residue tolerances) and minimal environmental impact. They're also very good for anybody who wants a DM fungicide that's easy to use, price-competitive, and pretty effective. Although there are occasional reports and testimonials alluding to the ability of these materials to control other grape diseases, I have not found this to be so in several different trials that we've run. In general, these are very good and reliable fungicides against downy mildews and some other closely related diseases on a variety of crops (DM is the only one that's important on grapes), but control of anything else is erratic at best. If you do get control of another disease, think of it as an unanticipated bonus. I certainly wouldn't encourage you to literally bet the farm on it, unless you're the type of person who starts preparing to retire after you buy a lottery ticket.

You know by now that there are several phosphonate products labeled for control of DM, and a number of other "nutrient formulations" on the market that contain phosphorous acid but are not labeled for DM control. Which means that it's only legal to obtain disease control with these latter products if you don't do so on purpose. Whether this seems fully rational or not, remember that the law requires that any material you apply for a pesticidal purpose must be labeled for such. You can still be cited if there's evidence that you broke the law, regardless of what you think about it.

For three consecutive years in the back-when, we ran a series of field experiments designed to determine the so-called

"physical modes of action" of phosphonates in control of downy mildew. These results and conclusions have been reported in detail in previous years, but a quick review of the major points:

- Phosphonates generally provided good but limited (3 to 8 days) protective activity, depending on the rate used, as well as the particular trial and which leaves were being evaluated. Protective activity in the older leaves sometimes declined significantly after 3 days, particularly at lower label rates, as phosphonates are highly mobile in the plant and tend to get "shipped" out of these older leaves.
- Phosphonates provided excellent "kick-back" activity against new infections. When applied 3 or 4 days after an infection period started, few lesions developed and spore production was greatly to totally inhibited. When applied 6 days after the start of an infection (small lesions just starting to become visible), the lesions continued to expand but production of spores was greatly inhibited. Extended post-infection control was better at the highest label rates than at the lowest, and was improved further when an initial application was repeated 5 days later (waiting for 7 would probably be OK). If you truly need some significant kick-back activity, don't go cheap and do keep an eye on things; if it looks like lesions are starting to come through, hit 'em again.
- Phosphonates did not eradicate well-established infections, but when applied to actively sporulating lesions, they limited further spore production by approximately 80%. Limiting the production of these spores will obviously limit the potential for disease spread.
- **CAUTION:** The phosphonate products have become very popular, for the good reasons cited above. But they're not miracle drugs, and a number of people have pushed them beyond their limits in terms of both spray intervals and rates. Furthermore, there appears to be a subconscious tendency among some folks to think that these aren't "real" fungicides, so don't need to be treated as such. Nevertheless, they are real fungicides when it comes to the DM organism, i.e., they're toxic to it. And just as with other real fungicides, the organism can develop resistance to these materials if given a chance.

And I've heard horror stories. For example, at the annual meeting this past winter in a state that will remain nameless to protect the guilty but where DM pressure is intense, more than one grower acknowledged tossing a phosphonate product into every spray tank all summer. This is sheer folly, I couldn't write a better scenario to assure that they won't last.



Although sudden and total resistance to the phosphonates is not likely to occur, there is good evidence that they can lose some of their effectiveness over time, similar to what we've seen with the DMI fungicides and their ability to control powdery mildew (or not). Don't burn these materials out! Rotate them with something else like you would any other fungicide with the potential for resistance development (i.e., no more than two sequential applications before switching to another product in a different resistance group) if you want to keep using them into the future.

## BOTRYTIS NEWS AND REMINDERS

Although there are a number of fungi that can cause bunch rots, especially in the lower midwest and southeast, Botrytis is still king where cooler or more moderate temperatures prevail when it rains and fruit are on the vine. A review of what makes it tick.

*1. Biology.* The Botrytis fungus thrives in high humidity and still air, hence the utility of cultural practices such as leaf pulling and canopy management to minimize these conditions within the fruit zone. It's a "weak" pathogen inasmuch as it primarily attacks highly succulent, dead, injured (e.g., grape berry moth, powdery mildew), or senescing (expiring) tissues such as wilting blossom parts and ripening fruit. Although the fungus does not grow well in berries until they start to ripen, it can gain initial entrance into young fruit through wilting blossom parts still attached to them, old blossom "trash" that gets trapped within clusters after it falls from the old flowers, and scars left on the young berries by the fallen caps. Such infections typically remain latent (dormant), but some may become active as the berries start to ripen (senesce), causing them to rot. Should this occur, disease can spread rapidly through the rest of that cluster or to others nearby, reducing both marketable yield and quality. Some recently-determined details regarding the above:

- Latent infections can be common following a wet bloom period, but the vast majority of them remain inactive through harvest and never rot the fruit. Factors that cause latent infections to activate (i.e., cause disease rather than remain invisible) are incompletely understood, but high humidity and high soil moisture (hence, lots of water in the plant tissues) are two environmental factors that promote this process. Note that for the preceding reasons, a wet bloom period (to establish latent infections) followed by a wet pre-harvest period (to activate them and provide conditions for further spread) is a perfect "recipe" for Botrytis. Berries with high nitrogen levels or subject to various mechanical injuries (nice work by Bryan Hed from Penn State on that last one) also are more prone to becoming diseased via the activation of

latent infections.

- Serious Botrytis losses are the result of rampant disease spread during the post-veraison/ pre-harvest period, after berries begin to ripen and become highly susceptible to rot by the fungus. Thus, latent infections established at bloom can be important if only a few of them become active and provide the initial "foot hold" from which subsequent spread can occur during ripening.

Because relatively few of these early infections typically do become active and turn into rot, controlling them at bloom provides only modest benefit if the post-veraison season is dry and doesn't support further disease spread. However, it can be critical in a year with a wet pre-harvest period (especially if the mid-summer was wet as well), which favors both the increased activation of latent infections and their rapid spread. So in one sense, bloom sprays are an insurance policy against the future unknown. Sometimes they pay huge dividends, sometimes not (data below). Considering your cultivar/clone, site, market, and experience, what's the potential risk (and your tolerance of it) of not being insured?

- The pronounced impact that cluster compaction has on Botrytis development appears to be due largely to its effect on the berry-to-berry spread that occurs at the point of their contact with one another. In one experiment with a tight-clustered Pinot Noir clone, a single diseased berry in a cluster, first showing symptoms 2.5 weeks after veraison, spread the disease to over 50 (!) berries in that same cluster by harvest. In contrast, spread was reduced by 90% (!) in the same group of vines where clusters had been loosened by removing some berries by hand right after set. Note that this single diseased berry per cluster (which we produced by inoculation) was meant to simulate the post-veraison activation of just one latent infection initiated at bloom, and vividly illustrates the particular importance of controlling blossom infections on tight-clustered cultivars and clones, since the risk of subsequent spread is so great should they become active.

In the above example, loosening clusters by hand thinning was possible on a small scale in a research trial to demonstrate a principle. Unfortunately however, there are few practical, foolproof ways of achieving the same effect on a commercial scale other than through clonal and varietal selection. The watchword here is foolproof. Over the years, several workers have experimented with prebloom sprays of gibberellic acid for this purpose, with some success. (Most recently, Bryan Hed and colleagues at Penn State have published an in-depth paper on their positive results with Chardonnay and Vignoles). And there are now some GA formulations (e.g., ProGibb 4%, which is even OMRI approved) that are labeled

for use on wine grapes. These labels contain warnings about possible yield reductions during the current and/or following years and a range of rates specific to different varieties. Nevertheless, some growers and investigators have been able to get the benefit of such treatments without noting negative effects. IMHO, loosening cluster compactness represents the “holy grail” for Botrytis management. And GA treatments just may have their place towards achieving this goal. But this technology is not foolproof, we do not have all the answers yet, and there are risks involved. I would caution anyone interested to still view it as technology in its infancy for wine grapes, to do their own experiments on a small scale for awhile before jumping in both feet first, and to keep their eyes and ears open with respect to the experiences of others.

- There is no single “correct” timing regimen for fungicide applications in a Botrytis management program. The standard “full” program used in fungicide trials, provided on many fungicide labels, and employed by some growers of highly susceptible and valuable cultivars consists of four sprays: at bloom, bunch closure, veraison, and 2-3 weeks pre-harvest. We have looked at the relative contributions of the two early sprays, the two late sprays, or all four in most years over the past 16 seasons, and a summary of these data is presented below in Figure 9. Note that in some years, the two early sprays provided better control than the later sprays. In an equivalent number of seasons, the opposite was true. In some years, two early sprays OR two late sprays provided the same control as all four; but in many (especially 2008!), applying all four did the best.

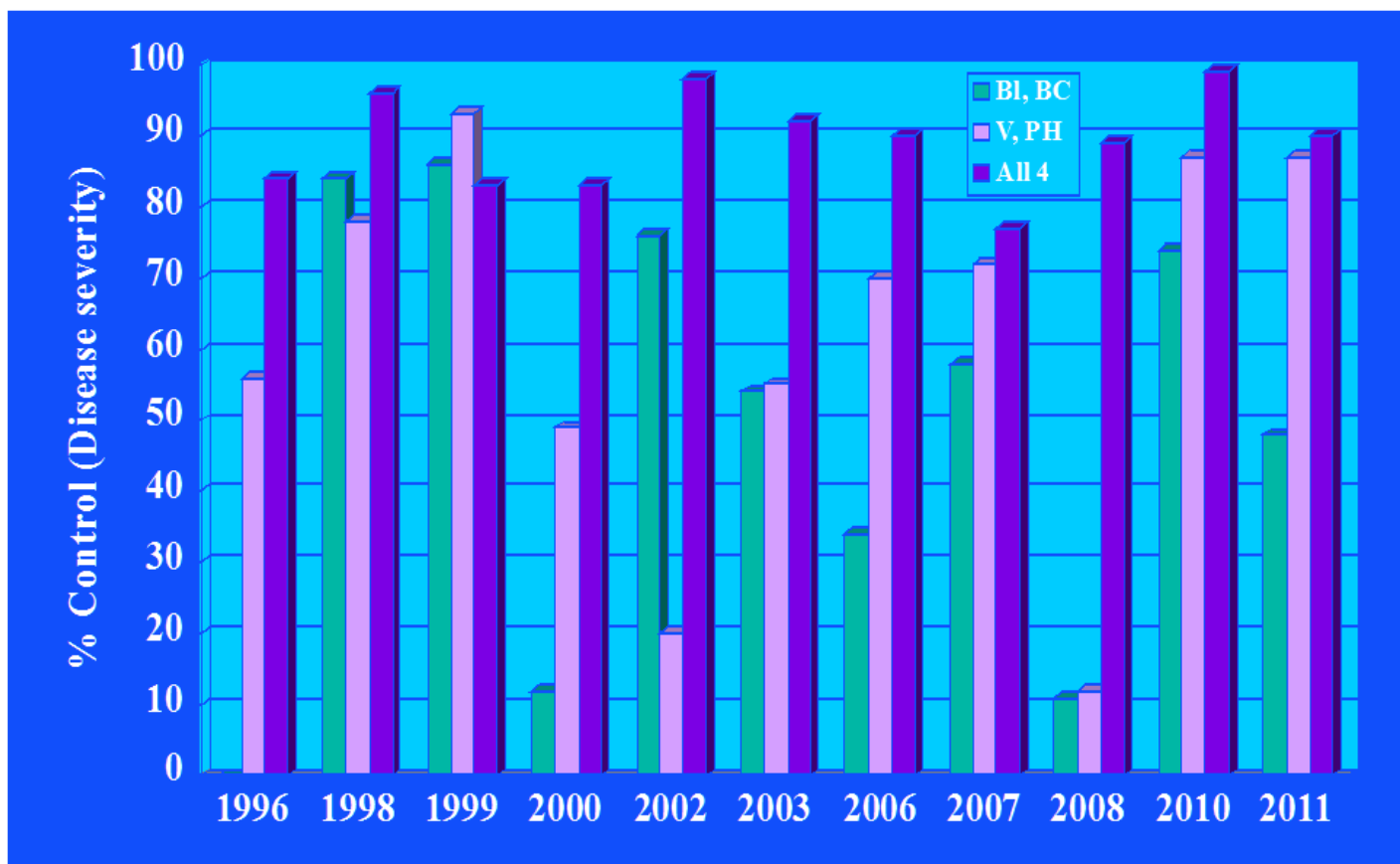


Figure 9. Influence of spray timing on the control of Botrytis bunch rot in Geneva, NY (cv. Aurore, 1996-2000; cv. Vignoles, 2002-2011). Sprays we applied at (i) Bloom + bunch closure (Bl, BC); (ii) Veraison and 2-3 wk later (Ve, PH); or (iii) at all four of these stages. Data are expressed as percent reduction of diseased berries relative to vines receiving no Botrytis fungicides.

The relative benefits of early versus late applications, and the total number necessary, will vary among years according to rainfall patterns and, quite likely, differences among cultivars and clones (e.g., cluster tightness). Think in general terms of early sprays as limiting the establishment of primary infections, and later sprays as limiting disease spread. But never forget: Botrytis is not a disease that you can just “spray your way out of”. These materials help, but they won’t do the job by themselves in a tough block and/or tough year unless you help them along with appropriate cultural practices (canopy management, leaf pulling, etc.).

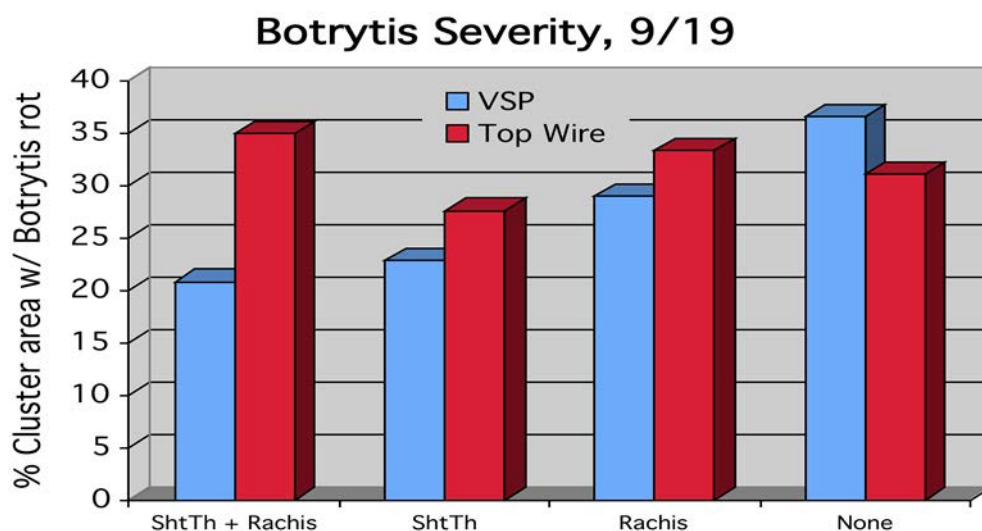
**New research: Effects of cultural control practices on Botrytis and sour rot control.** In 2011, I participated in a trial conducted in a commercial ‘Vignoles’ block in the Finger Lakes region, organized by Tim Martinson, Justine vanden Heuvel, and Hans Walter-Peterson. Although originally set up a couple of years ago to examine the effect of canopy management practices on fruit quality, it became obvious that these treatments were also affecting fruit rot, so we decided to give it a hard look in 2011. What a good year to do so!

The treatments involved were:

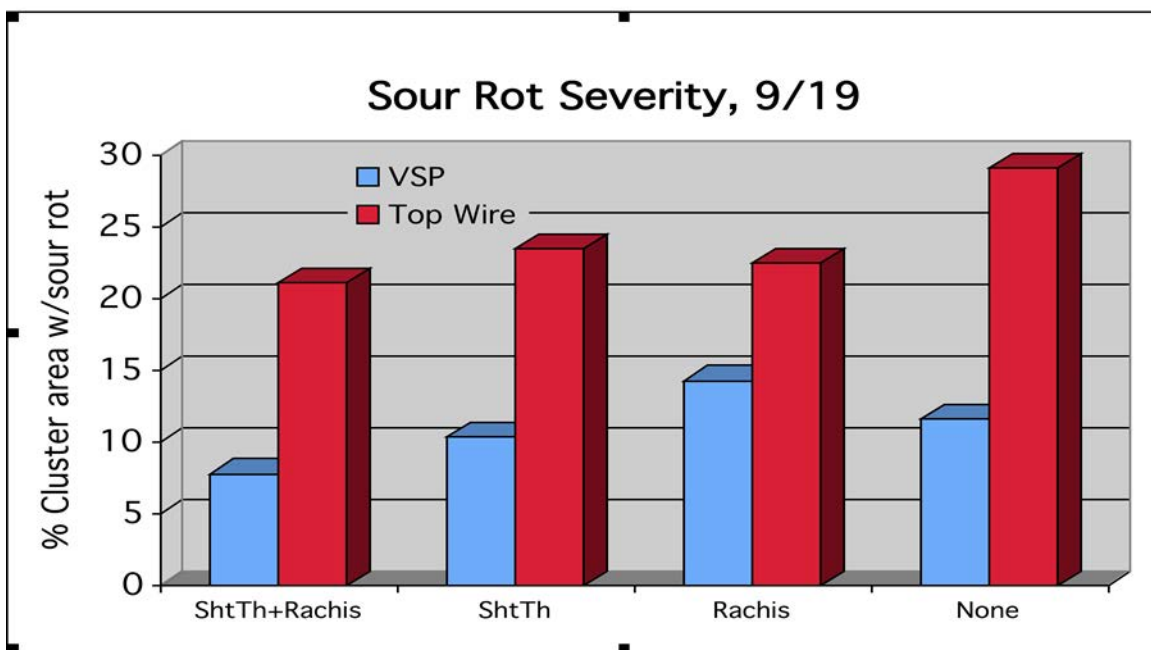
- Training system (Top Wire Cordon vs. a VSP variant)
- Shoot thinning (thinned to 5 shoots per linear foot of row vs. unthinned = approximately 7 to 8 per foot of row)

The grower maintained his crop via standard practices, which included a commercial Botrytis spray regimen. We rated the plots for incidence and severity of both Botrytis and sour rot at harvest on September 19; the VSP treatment was also rated 10 days pre-harvest. Some data and interpretations:

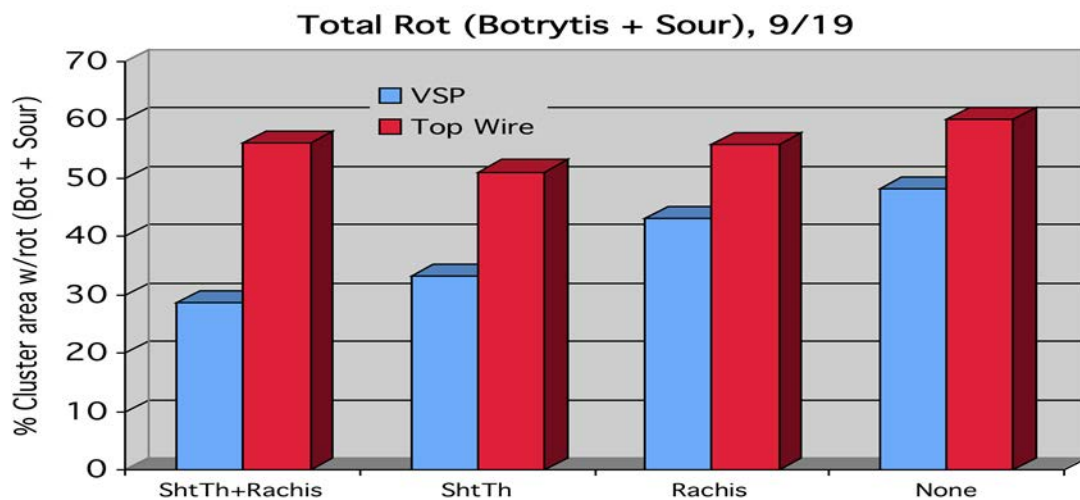
- Removal of old rachises (source of Botrytis inoculum) at the time of thinning versus no removal. The grower maintained his crop via standard practices, which included a commercial Botrytis spray regimen. We rated the plots for incidence and severity of both Botrytis and sour rot at harvest on September 19; the VSP treatment was also rated 10 days pre-harvest. Some data and interpretations:



- Positive effect of canopy manipulation treatments in VSP, not in TW
- In VSP, Shoot Thin + Rachis Removal was best, 43% reduction versus check treatment



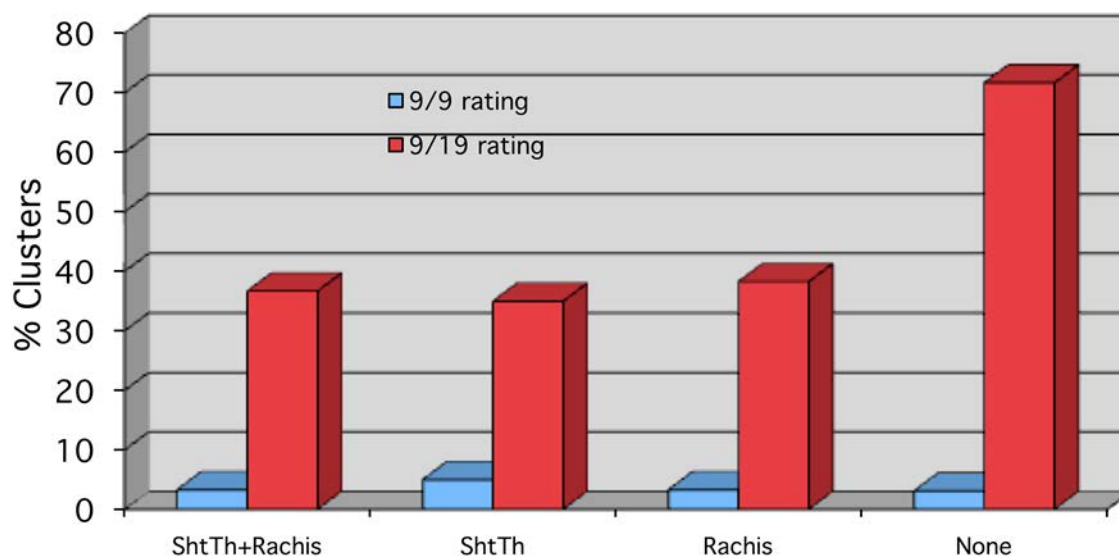
- Effect of training system was greater than that of canopy manipulation: across all four treatments, average of 11.0% cluster area w/sour rot for VSP, 22.2% for Top Wire.
- Effects of training system and canopy manipulation were additive: best treatment = Shoot Thin + Rachis Removal/VSP (7.8%), worst treatment = Check/Top Wire (29.1%)



- Effects of training system and canopy manipulation were additive:
  - With no canopy manipulation (check), effect of going from TW to VSP was modest: 20% reduction in average % rot.
  - Within VSP, thinning shoots and removing rachises reduced rot by 40% relative to the check
  - Going from TW to VSP and thinning shoots + removing rachises reduced rot by 52% relative to the TW check

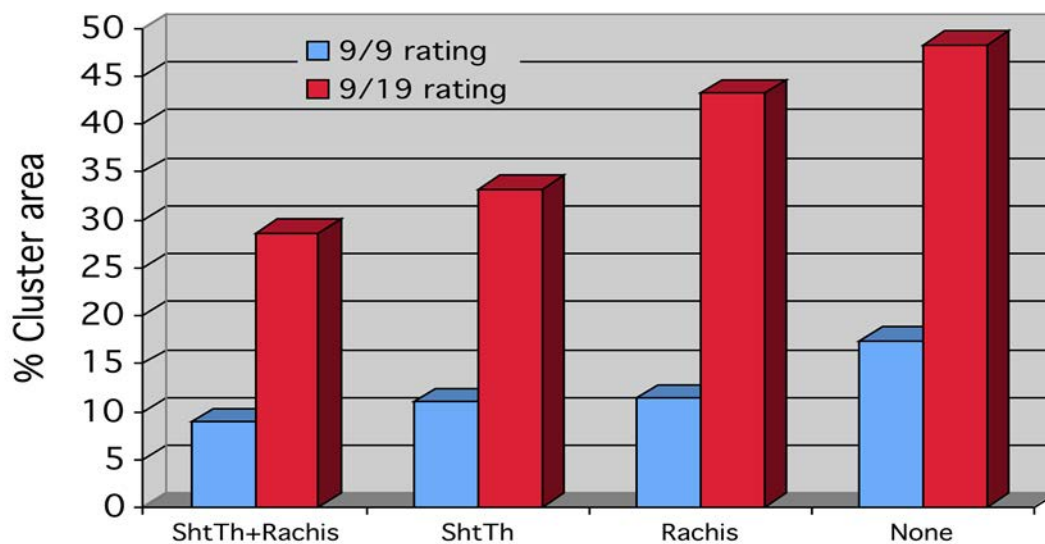


### Clusters w/>25% Botrytis, VSP: 9/9 vs. 9/19



- Major jump in percentage of clusters with heavy Botrytis over last 10 days preharvest in all categories, but nearly twice as bad when no canopy manipulation

### Total rot (Bot + Sour), VSP: 9/9 vs. 9/19



- Modest differences among treatments in amounts of total rot became greatly amplified the final 10 days before harvest.

2. *Fungicides, physical modes of action.* Over several years, we looked at the various “physical modes of action” of the available Botrytis fungicides, to get a better idea of some of their specific characteristics and differences. Following is a repeat of previous summaries of the major findings and conclusions for this project:

- In one set of tests, we examined the ability of the fungicides to protect the **internal** berry tissue against infection from spores that might be deposited inside them following mechanical damage, such as that from rain cracking, berry moth feeding, etc. Chardonnay clusters were sprayed at pea-sized berries, bunch closure and veraison, then a hypodermic needle was used to inject berries with Botrytis spores 2 weeks after the last spray. Scala, Vanguard, and Elevate provided excellent control, and Rovral was close. Pristine (19 oz/A) was comparable in preventing rot, but was less effective in limiting spore production from the limited number of berries that did develop symptoms. Flint and Endura provided the least protection of the internal berry tissues. However, all fungicides completely prevented spread to the neighboring berries when inoculated berries became diseased; in contrast, such spread occurred in two-thirds of the unsprayed clusters.

- In a more direct test for residual protective activity on the berry surface, clusters on a second set of Chardonnay vines were sprayed on the same dates as above and Botrytis spores were applied to the surface of the unwounded berries 2 weeks after the final application. As we would hope, all fungicides provided virtually complete control.

- In another test, Pinot Noir clusters were inoculated with Botrytis spores at late bloom but weren't sprayed with Botrytis fungicides until veraison. The purpose of this test was to see whether the fungicides could eradicate or suppress latent (dormant) infections long after their initiation, so long as the materials were applied before such infections became active. (Recall from above that preharvest activation of bloom-initiated latent infections is often the kick-start to a Botrytis outbreak). Under the conditions of this test (individual clusters sprayed by hand, absolutely thorough spray coverage to an extent not likely in commercial production), a single application of Scala or Vanguard at veraison provided almost complete control of latent infections established at bloom, 60 days earlier.

- Take home-messages and cautions:

- All of the current “standard” fungicides registered for Botrytis control provided excellent protective activity on the surface of the berries. That's why they got developed and marketed in the first place.

- The so-called AP fungicides (Vanguard and Scala) and

Elevate also provided very good protective activity within the berries, a pretty useful trait when injuries occur after spraying. This was anticipated for the AP's, since such fungicides are known to be absorbed by plant tissues, but Elevate was long sold as a surface protectant. However, this appears to have more to do with the company's marketing strategy than with science, as the recently-determined biochemical mode of action virtually requires that the fungicide be absorbed by fruit in order for it to work.

- Similarly, the same three materials provided very good curative activity against latent infections initiated at bloom, even when applied 2 months after infection. Nevertheless, as shown in Figure 8, we often get better control in our field trials when these fungicides are sprayed at bloom and bunch closure in addition to veraison and 2 weeks later. This suggests that the level of curative effect from the later sprays doesn't replace the need for earlier applications when conditions favor infection at bloom, although it probably contributes to the overall level of control that they provide.

## SOUR ROT

"Sour rot" is a term often used colloquially to describe a collection of late-season berry rots associated with a variety of fungi and bacteria other than *Botrytis* and a few additional well-defined causes of decay such. However, detailed work conducted over the past 5 years on the Niagara Peninsula of southern Ontario by Wendy McFadden-Smith and associates has shown that "true" sour rot--characterized by a pronounced smell of vinegar emanating from the decaying berries--is caused almost entirely by two genera of acetic acid-forming bacteria (*Acetobacter* and *Gluconbacter*). Once these organisms initiate infection, the wild yeast *Hanseniaspora* (*Kloeckera*) often follows, producing ethyl acetate (smells like nail polish remover) as one of its metabolites. Diseased berries severely reduce the quality of wines made from them, and once the disease gets rolling, growers are often faced with the choice of harvesting the affected blocks prematurely or watching the berries rot on the vine. Note the dramatic increase in rot severity (Botrytis + sour rot) over the last 10 days before harvest documented in the graph within the Botrytis section above.

Sour rot was a major regional problem during the wet harvest season of 2011, but was also a significant problem across Long Island in 2012 and was reported in a number of Finger Lakes vineyards as well. It has been a persistent and growing problem on the Niagara Peninsula of Ontario for the past 5 years and seems likely to be an even more widespread issue than is often discussed, in part because it has been poorly

defined and in part because there have been no specific control programs to employ against it, so people like me haven't wanted to talk about it much. Essentially, the only advice has been to use good canopy management practices, control other diseases, hope for the best, and deal with the worst. So hats off to Wendy for tackling this black box and starting to make some sense out of it.

The sour rot microorganisms (SRM) are incapable of breaching intact berry skins, so must rely upon wounds to gain entrance. Wounds caused by the feeding of birds and grape berry moth larvae are effective and conspicuous, but typically affect only a small percentage of the total berries in a vineyard. Far more common are wounds resulting from vineyard-wide weather or developmental factors, such as rain cracking or pulling away of the berries from their pedicels (individual berry stems) in tight bunches as they swell and compress against one another. Wendy's group has also shown that these infections don't occur until the berries reach a sugar content of 15°Brix, and that they're far more likely to become severe under warm conditions. The disease develops optimally at temperatures in the mid-70's (°F), and whereas it will also function at lower temps, "too" frigid (e.g., the 2009 harvest season) will stop it cold, as it were.

Although vinegar flies (*Drosophila* spp.) have long been associated with sour rot, it generally has been thought that these are secondary organisms drawn to the smell of fermenting berries only after the disease has become problematic. However, new research from Europe, the arrival of the invasive species spotted wing drosophila (SWD) to the region, and some preliminary work Greg Loeb's lab in Geneva last season collectively suggest that insects within this group may actually be an important causal component of sour rot. For example, Portuguese researchers recently isolated the causal agents of sour rot from *Drosophila* flies in local vineyards, but no sour rot developed even when berries were injured if grape clusters were protected from the flies using fine netting. This suggests that vinegar flies might actually serve as vectors, initiating the disease by delivering the causal bacteria to the grapes. Recent work by Wendy's group in Ontario also failed to detect the sour rot bacteria on grapes until after a disease epidemic had begun, lending additional support to this proposed scenario.

Most vinegar fly species lay their eggs near the junction of the berry to the pedicel. This summer, we (Greg and his crew plus new graduate student Megan Hall working with yours truly, in coordination with Wendy and her gang) plan to explore the hypothesis that the larvae that hatch from those eggs can pass into the berry flesh via microscopic fissures created when the berry pulls away from the pedicel as it swells (particularly in compacted clusters and following heavy rains

in the pre-harvest period), bringing the sour rot bacteria with them and thereby setting the whole epidemic in motion.

If true, this scenario suggests several potential control tactics, which we also will be investigating and plan to report upon next year. For the record, these involve three different actionable targets:

- Susceptibility of the cluster to injury and initial infection by the sour rot microorganisms. Canopy management and cluster loosening, in a nutshell.
- Bacterial populations on berries before they have an opportunity to infect. As in, reduce them.
- *Drosophila* flies in the vineyard. As in, control them.

Stay tuned.

## SUMMER ROTS

A term sometimes used for two similar diseases (ripe rot and bitter rot) common in more southern, humid (and quite warm) production regions. Those beneath the Mason-Dixon line and in the lower Midwest deal with these diseases on a regular basis and they occur sporadically to the north. Bitter rot, in particular, seems to pop up with some regularity on Long Island, particularly on Chardonnay. Those of us to the north should probably start being more aware of these diseases, especially in wet years and if they keep getting a little bit warmer than we've grown accustomed to. It's not a disease threat to be over-emphasized in our region just yet, but neither is it one to be flat out ignored.

Bitter rot appears to be the more likely threat in our "marginal" northern areas, as it doesn't have quite the need for heat that ripe rot does. Usually, symptoms first occur after veraison, as the bitter rot fungus moves into the berry from the berry stem and turns the diseased portion brown (on white varieties) or a dull purple. Once the berry is completely rotted, it becomes absolutely covered with numerous prominent, raised black pustules (the fungal fruiting bodies). You can't miss 'em. More details on the appearance of symptoms and how to distinguish them from Phomopsis and BR symptoms can be found in the 2013 NY and PA Pest Management Guidelines for Grapes (hard copy and on-line).

Ripe rot tends to predominate as you keep moving south, although it has been documented as far north as New England. Symptoms do not develop until after veraison and become increasingly prevalent the closer you get to harvest (whoda thunk it with a name like that?). Infected fruit initially develop circular, reddish brown lesions on their skin, which eventually expand to affect the entire berry. Under humid conditions, small "dots" of slimy, salmon-colored

spores may develop across the rotten berry as the lesions become depressed, and serve to spread the disease to healthy fruit if rains continue. Infected fruit shrivel and mummify, and may either remain attached or fall to the ground. No foliar symptoms are produced.

Both diseases are favored by abundant, warm rains (77° to 86°F is optimum) between fruit set and harvest. Infections occurring before veraison typically remain “dormant” until fruit begin to ripen. Captan and the strobilurin fungicides are the go-to materials for control of these diseases in regions where they occur regularly (as is mancozeb, within its PHI restriction).

Cultural practices, such as pruning out dead spurs, removing overwintered mummies, and removing weak or dead cordons, are important to help reduce the inoculum in the vineyard. Turner Sutton at NC State, who has done more work with these diseases than anyone, has demonstrated this nicely by showing that they even tend to be worse in spur-pruned blocks, where some old (previous) fruiting wood is always retained. Both diseases are frequently controlled in the early- to mid-summer by sprays containing mancozeb, captan, or a strobie product directed against other diseases. However, with the exception of Flint and Pristine, fungicides used for Botrytis management (Elevate, Scala, Rovral, Vanguard) provide very little control of bitter rot or ripe rot, and their exclusive use during wet preharvest seasons can lead to outbreaks of these diseases in regions where they are not routine and, therefore, are not consciously managed.

Sprays targeted against bitter rot and/or ripe rot may be needed in the late season if the weather is warm and wet, especially if the diseases are observed in the vineyard or have occurred there in the past. In southerly regions where they are consistent problems, it is typically necessary to apply protectant fungicides on a 2-week schedule from bloom until harvest, except during periods of drought. Because fruit are especially vulnerable in their final stages of ripening, pre-harvest sprays can be particularly useful when these diseases are active, to avoid rapid secondary spread. This potential utility must be balanced against wine makers’ concerns about the effects of such sprays on fermentation (of course, they also love fruit with bitter rot, another aptly named disease). That legal preharvest restrictions on fungicide labels must be followed is a given.

## PHOMOPSIS (Ph) NEWS AND REMINDERS

At this point of the year we’re right in the heart of Phomopsis season. But for the record:

*1. Early sprays are the most important for control of rachis infections.* Your annual reminder that in multiple spray timing trials over a number of years, we found that

applications during the early shoot growth period (as clusters first become visible, starting about 3 inches of shoot growth and continuing until the second and sometime third clusters emerge) are the most important for controlling disease on the rachises. Rachis infection by the Phomopsis fungus is among the most common causes, if not *the* most consistent cause, of economic disease loss that I see on Concord grapes. It’s even worse on Niagaras since it seems to move more readily into the fruit of this cultivar (not to say that DM can’t kick Niagaras pretty hard in some years as well, but this generally happens later). Note that early sprays also provide the greatest control of shoot infections, which serve as sources of Ph spores in subsequent years if retained as infected canes, spurs, or pruning stubs. As said at the beginning, they’re important where this disease is a threat.

*2. Early sprays also provide significant control of berry infections.* In a trial conducted several years ago in a commercial problem block of Niagaras, we were surprised to find that sprays applied before and just after cluster emergence (the important sprays for controlling rachis infections) also provided nearly 70% control of berry infection. In retrospect, this shouldn’t have been too surprising, since it’s common to see rachis infections expand into the berry stem and then into the berry itself, especially on this variety. But it was an eye opener nevertheless.

In a subsequent trial in a different high-inoculum Niagara vineyard at the old Fredonia lab, we (thank you Rick, Mike, Kelly, and gang) documented a gain of over 2 tons/A in two particularly bad Phomopsis years, simply as a result of applying a single mancozeb spray during the early “3- to 5-inch” shoot growth stage. The quotes are to stress that this timing is approximate; the point is to get something on the young clusters soon after they emerge.

Thus, a minimal Ph spray program should include at least one application during this period. Research has repeatedly shown that waiting until the immediate prebloom spray is far too late if there is any significant disease pressure going on (inoculum in the vineyard + rain). Commercial experience has consistently shown the same thing.

PHOMOPSIS: EFFECT OF EARLY CONTROL ON YIELD <small>(cv. Niagara; Fredonia, NY)</small>		
Phomopsis program	Yield (tons/A)	
	2006	2008
None.....	7.7	13.2
Mancozeb, 1x.....	10.0	15.5
Mancozeb, 3x.....	10.8	16.4
Mancozeb at 1- to 3-in shoots; + 2 wk; + 2k		

*Table 5. Effect of a single well-timed Phomopsis spray on yield. In both years, the single spray (“1x”) was applied 2 weeks after the first spray (1- to 3-in shoots) in a comparison treatment that received three applications in total (“3x”).*



3. *Old wood, particularly dead wood and canes, may be particularly important sources of Ph spores.* The Ph fungus is especially prolific in dead tissues, including dead wood. The obvious practical implication of this observation is that removing old and dead wood during pruning operations is an important component of a Ph management program. This includes not only obvious sources such as dead canes and arms, but also less-obvious ones such as old pruning stubs and even apparently-healthy spurs. For years, Alice Wise on Long Island has pointed out that Phomopsis is worse on spur-pruned cultivars than on those that are cane-pruned, and this observation was confirmed to me recently by a commercial grower who is trying to avoid conventional fungicides and saw a big difference in Phomopsis development when she converted her *V. vinifera* blocks from spur- to cane pruning. Makes sense.

*Note that the Ph fungus can remain active in infected wood for at least several years, so a "dirty" block is going to stay that way until you prune the stuff out.*

4. *Little fungal inoculum, if any, is available by mid-summer.* We monitored the release of Ph spores in several Lake Erie and Finger Lakes sites over 3 consecutive years. And in each year, we detected few if any infectious spores beyond early- to mid-July, with the vast majority released between bud break and bloom. A similar study conducted by Annemiek Schilder at Michigan State University produced generally similar results. These data suggest that even though berries may remain susceptible throughout the season, as shown by work from Mike Ellis and students at Ohio State, the risk of infection is probably very low once berries become pea-sized, since inoculum is scarce beyond that time.

5. *Fungicides.* Mancozeb, captan, and ziram have all provided very good to excellent control of basal shoot and rachis infections in our fungicide trials. Experience with the strobies has been mixed. Fortunately, they've looked better against fruit (and maybe rachis) infections than they have against basal shoot infections. We've seen no difference between the efficacy of Abound versus Ziram for controlling fruit infections when mancozeb was used prebloom and these materials were compared in subsequent postbloom sprays. In a trial on Niagaras a couple of years ago, four sprays of Pristine were as effective as four of mancozeb, with some indication that the non-strobie component of the product was making a significant contribution. Sulfur, although touted as a Ph material in some regions, including California (where it doesn't rain during most of the growing season), did practically nothing in this same trial. In fact, NY recommendations going back to before I knew the difference between a spore and a sponge gave little credence to sulfur as a Ph fungicide. It doesn't appear that much about the fungus

or the element has changed since then.

6. *Spray application technique.* Many growers like to spray alternate rows in the early season when it's the critical time for controlling Ph, assuming that sufficient spray will blow through the target row and impact on vines in the "middle" row. For 3 consecutive years, Andrew Landers helped us examine this issue in a commercial Niagara vineyard. Consistently, vines in the middle row received less spray than vines subjected to every-row spraying, and perhaps more importantly, the coverage on them was much more variable. I recognize that the obvious benefits of alternate-row spraying and am a firm believer that there's no reason to fix things if they ain't broke. However, I'm also a firm believer in seeing things how they are rather than how you want them to be, so if you've had trouble in controlling Ph while using alternate-row spraying, the suggested remedy also is obvious.

## WOOD CANKERS

Eutypa dieback has been on the radar of eastern grape growers for many years; in fact, it is standard practice to cut through a piece of cankered trunk or cordon, see a wedge-shaped area of dead tissue, and diagnose it as Eutypa. However, work conducted for more than a decade now at the University of California, primarily in the lab of Dr. Doug Gubler at UC Davis, has shown that there are a number of different fungi that cause canker diseases in the west, each with its own specific biology and, potentially, appropriate management program. Similar research in Australia, South Africa, Chile, Italy, and other European countries over much of this same period is producing similar results.

In eastern North America, we (understandably) tend to preoccupy ourselves with the whole panoply of fruit and foliar diseases found in humid climates, which can destroy a crop in a single season if not adequately controlled. Nevertheless, we also have canker diseases, and although less flamboyant than our usual rots and mildews, they are "silent but deadly" robbers of production and profit here as well. We continue to see signs that they will become increasingly visible and important as our newer and higher-value vineyards continue to age. And informal surveys of traditional native plantings suggest that these diseases are a lot more important than they've been given credit for in the past, as one would expect given the age of many of such vines. I firmly believe that if we're serious about kicking our viticulture up a notch, we're going to need to start addressing these diseases. Pretending that they're not that important here isn't going to make them so, unless we really believe that things in our neck of the woods are basically different than they are in the rest of the world where grapes are grown.

As noted in previous issues of this volume, were very

fortunate to have Dr. Philippe Rolshausen--a 10-year veteran of Doug Gubler's lab at UCD and someone possessing a wealth of experience on this topic--working on cankers of eastern grapevines while he was at the University of Connecticut on a temporary appointment a few years back. And he continued to work with us from his base at UC Riverside after returning to the west.

The bottom line from Philippe's work is that although we might have a few unique organisms involved here and there, many of the fungi that he found in cankers throughout NY and other eastern regions are also well-known causes of disease in other parts of the world (including a number of fungi responsible for *Botryosphaeria* dieback and the esca and "black goo" syndromes). Recognizing this, and at the very least doing a better job of removing dead wood from vineyards and torching it before the fungi within start spreading disease to new pruning wounds, is the first order of business. There's also some exciting and more intensive canker management work going on in Australia right now that could have application here, should the industry show an interest in getting behind it.

## ANTHRACNOSE

In NY and neighboring states, anthracnose outbreaks historically occurred on Vidal blanc and a few seedless table grape varieties, especially Reliance. We'd hear about it from warmer areas in the Midwest, but it was more a curiosity than anything else for the vast majority of growers and advisors. In recent years, however, there have been regular outbreaks on some of the newer cold-hardy cultivars that are gaining in popularity and expanding the geographical range of grape production. Marquette appears to be particularly susceptible, although Frontenac and La Crescent also have been affected. Certain older cold-hardy cultivars from Elmer Swenson's program also can be problematic, including Edelweiss, Esprit, Brianna, St. Pepin, and Swenson White. In some Midwestern states, Concord, Catawba, and Leon Millot have been reported as encountering problems, although such occurrences are rare here.

Among the more conspicuous symptoms are dark, noticeably sunken lesions that develop on infected shoots, typically on the first few internodes near the base of the new shoot. These lesions resemble the internode lesions associated with Phomopsis but they are more aggressive, expanding farther along the shoot and deeper into its center than those caused by Phomopsis. Berry lesions, consisting of whitish-gray spots about 0.25-in across and surrounded by reddish brown to black margins, also are characteristic.

The fungus overwinters primarily on infected canes, although

the previous year's berries can also be a source. In spring, spores are produced from overwintering fungal structures on these tissues and are dispersed by splashing raindrops to young, susceptible tissues. Temperatures in the mid-70's to low 80's (°F) are optimal, although infection can occur across a wide range. Additional splash-dispersed spores are produced upon new infections, and these can spread the disease through multiple repeating cycles of new infection and further spore production. Hence, outbreaks occur most frequently in years with multiple rain events early and mid-season. Young tissues are most susceptible, becoming resistant as they are mature. Berries become relatively resistant by about 7 weeks post-bloom.

Diseased canes should be pruned during the dormant season and removed from the vineyard or destroyed. If numerous infected berries remain on the vineyard floor, most spores originating from them can be neutralized by covering the berries through cultivation or, if practical, covering them with mulch. Early-season sprays of mancozeb, captan, or ziram targeted against Phomopsis also provide significant control of anthracnose, although this latter disease is not listed as a target on most labels. Some DMI fungicides, e.g., difenoconazole (Revus Top, Quadris Top, and Inspire Super), and myclobutanil (Rally) are specifically labeled for anthracnose control. A "delayed dormant" application of lime sulfur can be useful in vineyards where the disease has become established and problematic to control and/or in "organic" vineyards where traditional fungicides cannot be used. This treatment limits the production of infectious spores from overwintered cankers.

## PUTTING IT ALL TOGETHER

As I preface this section every year, we all know that there are as many good disease control programs as there are good growers and advisors. The following are some considerations among the many possible alternatives. As always, just because it isn't listed here doesn't mean it's a bad idea. And as always, don't make this any harder than you need to.

**1-INCH SHOOT GROWTH.** This horse is out of the barn by now. but for the record: A **Ph** spray may be warranted if wet weather is forecast, particularly if the pruning/training system (significant inoculum retention) or block history suggests high risk. Option A: Nothing. Option B: Captan, mancozeb, or ziram. The best one is whichever is cheapest and most convenient.

**3- to 5-INCH SHOOT GROWTH.** A critical time to control **Ph** rachis infections if it's raining or likely to be soon, especially in blocks with any history of the disease. Early is better than late if it looks like some rain is setting in. Late is much better than nothing if those are the only two options,

i.e., you're past this stage, haven't gotten anything on, and wonder whether it's too late. This spray can provide significant benefit against fruit infections as well, since many of them originate from movement into the berries from infected rachises and berry stems. Also an important time to control basal shoot infections, since this is where the fungus will establish itself for the future if infected tissue is retained in canes, spurs, or pruning stubs.

Now is the time to start thinking about control of **PM** on *vinifera* varieties if temperatures remain above 50°F for long stretches of the day (they are, they are!). This spray is much more likely to be important in vineyards that had significant PM last year (we're talking late season foliar disease more than fruit infections here) than in those that were "clean" into the fall; however, it may be beneficial even in relatively clean blocks of highly susceptible cultivars, which tend to be relatively valuable as well. It's particularly true in cloudy, wet years unless low temperatures are severely limiting (hasn't been the case this year). If already spraying for Ph, most growers of on highly susceptible (and valuable) varieties include something for PM while they're at it. I would too.

In NY, spending extra money for **BR** control is almost never justified this early unless you're trying to clean up a severe problem block AND weather is wet and reasonably warm. In general, the farther south you go, the more important early sprays can become. Still too early for **DM**.

Option A: Nothing. Option B: Mancozeb or ziram (BR, Ph). Option C: Captan (Ph, some BR). Easier on predator mites than mancozeb or ziram, probably good enough against BR this early, but 3-day REI issue. Option D: Sulfur (PM). As discussed above, historical pronouncements concerning reduced activity of sulfur at temps below 65°F appear to have been significantly exaggerated. It should be good enough, and is a cheap insurance option. With thorough coverage, sulfur sprays can eradicate incipient infections initiated during the previous week or 10 days (depending on temps since then). Option E: The DMI products are an option here, but I don't really like them now unless you're also trying to control **anthracnose**. The include Rally, tebuconazole generics, Mettle [except NY] (PM, BR) and the difenoconazole (DFZ) products--Revus Top (PM, BR, DM), Inspire Super (PM, BR, Bot), and Quadris Top (PM, DM, BR). The problem is, we're trying to limit the number of DMI applications, and you're using a material with activities against diseases that you don't need to control right now (BR, DM) while burning one of your available applications of these materials. And did I already mention the critical nature of dosage with the DMI products, how dosage is a function of spray coverage, and the coverage problems with alternate row spraying? Option F: JMS Stylet Oil (PM). Should

eradicate young infections that have already occurred IF spray coverage is thorough. Also can provide a few additional days of limited forward activity, although much of this protective capability washes away with less than ½-inch of rain. Can use with mancozeb or ziram, but not with or near captan or sulfur (plant injury). Option G: Nutrol, Armicarb, Oxidate, Kaligreen. (PM). Should eradicate young infections. IF thorough coverage is provided, but no forward activity. If choosing this option so early in the year, go with the low end of the label rate and use the cheapest one. Option H: Serenade, Sonata, or Regalia if you want to experiment with these "biocontrol" products while disease pressure is low (PM). Option J: One of the above PM products plus mancozeb, ziram, or captan (no captan with oil) for Ph.

**10-INCH SHOOT GROWTH.** We once recommend not waiting any later than this to control **BR**. Continued experience tells us that we can get away with withholding a BR spray at this time under most commercial conditions in NY unless this disease was a problem last year (inoculum levels are high) and weather is wet and warm. **DO NOT** wait any later than now to control **PM** on susceptible varieties. On Concord and other "moderately susceptible" cultivars, we normally recommend waiting until immediate prebloom. However, there have been seasons where we started seeing PM on Concorde around the 10-in shoot growth stage, and uncontrolled early infections spread to the point where people never did get control of them (including on the clusters) once they started spraying. And I've had excellent Concord growers tell me that when they wait until prebloom, they see a little PM already established, which puts them behind the 8-ball right from the start. So, get out in the vineyard and see what's happening. No need to spray before you need to, but if you already see PM, or you have experience with early disease development and weather conditions are forecast to favor PM, it might be a good idea. Remember, as crop load goes up, so does the need for good PM control and the ability to pay for it. Now is one of the best times to use a DMI, and a possible time to experiment with "alternative" materials if you're so inclined. It's also one of the best times to use an oil or other eradicator material against young "primary" infections that might just be getting started, particularly if the PM program up until now has been marginal or absent. **DM** control should be provided on highly susceptible varieties, especially if disease was prevalent the last year or two and rains of at least 0.1 inches at temps >52°F are anticipated or have occurred recently. Rachis and fruit infections by **Ph** are still a danger in wet years, particularly in blocks with some history of the disease.

Option A: Mancozeb (BR, Ph, DM). A broad spectrum, reasonably economical choice for everything except PM; tank



mix with a PM material to complete the picture if necessary. Excessive use can lead to mite problems by suppressing their predators. You can substitute ziram if necessary or desired but will give up some DM control in the process, although that might not be too significant this early. Option B: Captan (Ph, DM, some BR). An alternative to mancozeb if you're trying or are forced to avoid it. The limited BR activity should still be sufficient if the disease was controlled well last year (limited inoculum) and good BR materials will be used in the next three sprays. Toss in something for PM where needed. Option C: Sulfur (PM). Historical concern about reduced activity during cool weather is going down as we look at experimental data, and temps should be going up now anyway as we look at the calendar going forward. Post-infection activity may be useful against new "primary" infections before they have a chance to form new spores and spread to developing clusters. Option D: Revus Top (PM, BR, DM). Superior PM control relative to anything else recommended at this stage of the season other than Quintec or Vivando. Plus BR and DM control (and anthracnose for those needing it), all at a highly competitive price. A combination that's pretty hard to beat if that's what you're looking for. Except on Concord and a few other cultivars likely to become injured by it. Option E: Quintec or Vivando (PM). Both are Cadillac PM material that should be limited to two applications per season each (they are unrelated to one another) for resistance management purposes. You'll get even more bang for your buck with a Cadillac PM material in another week or two, but if you feel that you need or want to start throwing the kitchen sink at it now, these are viable options. Option F: Rally, tebuconazole generics, Mettle [outside NY] (PM, BR). Option G: JMS Stylet Oil (PM). If (and only \*IF\*) coverage is thorough, this spray should eradicate early PM colonies that may have started, should previous PM sprays have been omitted or incompletely applied. But don't waste your money if you can't cover thoroughly. Also may help with mites. Will provide a few days protectant activity going forward in addition to the eradicant action, although much of that residual activity will disappear after a rain (along with the oil, one would guess). Mix with something offering forward protective activity if your next spray is going to be more than a week from now. The petroleum-based PureSpray Green should have similar effects if you can find it, whereas the botanically-based oils are generally less effective. Option H: Torino (PM only) [not in NY, yet]. A good PM material unrelated to anything else out there. Limited to two sprays per year on label, probably not the best time to use up one of them except perhaps on highly susceptible cultivars. Option I: Nutrol, Armicarb, Oxidate, Kaligreen. (PM). Should eradicate young infections IF thorough coverage is

provided, but no forward activity. Option J: Serenade, Sonata or Regalia, if you want to experiment with OMRI-certified "biobased" products before entering the critical period for disease control. Option K: Mix and match among the aforementioned to control the diseases that you need to.

**IMMEDIATE PREBLOOM TO EARLY BLOOM. A critical time to control PM, BR, DM, and Ph on the fruit! Just starting to enter Bot season, too. This and the first postbloom spray are the most critical sprays of the entire season--DON'T CHEAT ON MATERIALS, RATES, SPRAY INTERVALS, OR COVERAGE!!**

Option A: Quintec or Vivando for PM control, plus mancozeb (for BR, DM, and Ph). Effective and no known resistance problems in the real world, but let's keep it that way by avoiding over-use (no more than 2 applications per year of each one). Option B: Pristine (PM, DM, BR, Bot at higher rates, some Ph). We'd like to keep this one down to 2 applications per season, too, especially with the increasing risk of DM resistance the longer that we keep using it. The 12.5-oz rate of Pristine will also provide significant protection against Bot, I wouldn't spend the extra money on the higher "Botrytis control" rate (18.5-23 oz/A) this early unless Botrytis pressure was really high and/or I was really worried. On highly susceptible cultivars, where DMI resistance is usually an issue to at least some extent and strobile resistance has occurred or is deemed risky, Quintec, Vivando, or Pristine would be the materials of choice for PM, but don't forget about DM and BR. With Pristine especially, I'd toss in some sulfur, particularly in blocks where PM has already developed strobile resistance, just for additional insurance at this critical time. Option C: Luna Experience (PM, Bot, variable BR depending on rate) + mancozeb (DM, BR, Ph). Refer to discussion in last year's tome for specifics regarding Luna Experience, including rotational considerations if using Pristine, as both contain "Group 7" materials. Option D: Revus Top (PM, BR, DM), Inspire Super (PM, BR, Bot), or Quadris Top (PM, BR, DM). Discussed in detail under "New fungicides" in last year's volume. Worth reviewing, especially if you're considering use at this time. If using Inspire Super, you'll need to add something for DM. I can't overemphasize the fact that the excellent PM control we've seen with difenoconazole is due to its high "intrinsic" activity, and that this is rate dependent so you'll start losing it --especially on the clusters!--if you get spotty spray coverage (i.e., only put a partial rate on your spray target). Option E: Abound or Sovran [plus sulfur, on cultivars where it can be used] (PM, BR, DM [only moderate DM for Sovran]). Still an effective option in some plantings, particularly on native and certain hybrid cultivars that have seen limited use over the years, although the scuttlebutt is that they're slipping in



some of these vineyards, too. As with most rumors, recognize this one for what it is and then apply your own experience in determining how much credence to give it. Nevertheless, I think it's fair to say that these materials' best days are behind them, although they're not dead yet (sounds uncomfortably familiar on a personal level). Refer to the discussion on strobilurin resistance in the "Fungicide Changes and News" section at the beginning of this epistle. **Option F:** Flint plus sulfur (PM, BR, Botrytis at the 3-oz rate) plus one of the many options for DM. **Option G:** Rally, tebuconazole generics, or Mettle [no Mettle in NY] (PM, BR) PLUS mancozeb (DM, BR, Ph) or captan (DM, Ph). IMHO, you'd choose this option only if you couldn't use difenoconazole as a DMI. One of the new DM-specific fungicides could also be used for DM control, but they may give more bang for the buck after bloom unless there's heavy DM pressure early (clusters are highly susceptible now, after all). Add sulfur on *vinifera* and PM-susceptible hybrids (unless "sulfur shy"). Like the difenoconazole products, Rally, the tebuconazoles, and Mettle provide excellent postinfection activity against BR, which can make them especially valuable if significant unprotected infection periods occurred over the last week or 10 days. If wet, mancozeb (or captan) should be included for control of Ph fruit infections in blocks where this has been a historical problem (note some processor restrictions and poor BR control with captan). **Option H:** Torino (PM only) [not yet registered in NY]. Good but not the best, right now you want the best. **Option I:** Mancozeb + sulfur (PM, BR, Ph, DM). Used to be cheap and effective, particularly if used at shorter spray intervals; it's no less effective than before, but not the best option for control of PM on highly susceptible and valuable cultivars at this critical time. Neither material is as rainfast as the strobies or SI fungicides, so shorter spray intervals can be both necessary and difficult in wet years. Of course, this is precisely when their activity is needed the most. Potential mite problems, as this mixture is hard on mite predators.

80% (+/-) CAP FALL. Vanguard (or Inspire Super), Switch, Scala, Elevate, Flint (3 oz rate), Endura, Pristine, or Luna Experience [not yet labeled in NY] for Botrytis control will probably be beneficial sometime around now on susceptible varieties, particularly in wet years. It's certainly easier to use or include one of these materials for Botrytis purposes in the "immediate prebloom/early bloom" or "first postbloom" spray applied to control other diseases, and from what we know of these materials' activities, they should be effective when applied then, although we've never directly compared these timings with one at 80% cap fall (results would likely be different from year to year anyway, depending on if and when rains fall throughout the pre- to post-bloom period). One problem with tank-mixing Botrytis-specific materials like the

AP's and Elevate is that you'll be distributing them throughout the entire canopy, whereas the only place they're effective is on the clusters.

Also, if sulfur was the only PM material in the previous (immediate pre-bloom) spray, reapply about now on highly susceptible *viniferas*, especially if it's been raining since the last application or will soon.

**FIRST POSTBLOOM** (10-14 days after immediate prebloom/early bloom spray). **Still in the critical period for controlling PM, BR, DM, and Ph on the fruit. And we're well into the start of Bot season. This and the immediate prebloom/early bloom spray are the most critical applications of the entire season--DON'T CHEAT ON MATERIALS, RATES, SPRAY INTERVALS, OR COVERAGE!!** Shorten the spray interval and/or jack up the rate and/or quality of the PM material on PM-susceptible varieties if weather is warm and cloudy. For Botrytis-sensitive cultivars/blocks/seasons, make sure that this application has some Bot activity if you haven't used anything for it yet. Same considerations and options as detailed under IMMEDIATE PREBLOOM. Juice grape growers can substitute Ziram (very good BR and Ph, only fair DM) for mancozeb or captan if necessary, or just go with Abound or Sovran for everything if they're still working. Captan, mancozeb, or the strobies will protect against bitter rot and ripe rot, if/where those are concerns.

**SECOND POSTBLOOM.** **BR** control is still advisable under wet conditions and should be considered critical if infections are evident on the vine unless you're willing to bet part of your crop that it's not going to rain within the next few weeks; however, BR sprays can often be skipped from here on out on natives and hybrids if the vineyard's clean. Fruit are less susceptible to **PM** now, but those of *vinifera* varieties (and susceptible hybrids?) still need good PM protection, particularly to guard against later bunch rots and colonization by wine-spoilage microorganisms. Of course, new foliage remains highly susceptible to PM throughout the season, and it behooves you to keep it clean for purposes of leaf function in addition to reducing primary inoculum for next year. Concord can withstand a fair amount of foliar PM unless the crop is very large and/or ripening conditions are marginal. Minimal programs on this cultivar can stop now if the preceding crop/ripening conditions don't apply, although one more PM spray now is often justified. Try to avoid DMI and, particularly, strobie fungicides if PM is easy to see now without trying very hard. **Ph** danger is basically over unless very wet and a problem block. Clusters are still susceptible to **DM** and should be protected on susceptible varieties if weather is wet, especially if disease already is established (take a look and see). Foliar DM is starting to

crank up and will remain a potential threat throughout the rest of the season, depending on the weather. It can quickly turn into an epidemic on susceptible cultivars if we get into a prolonged set of summer rains or thundershowers, if you let it get started now you may be fighting it the rest of the year. Option A: Pristine, Abound, Sovran, or Flint. See previous discussions on all of these. They provide good residual control of the listed diseases if used now, but strictly limit their use to a maximum of two sprays per year of ANY of these Group 11 materials, in order to maintain viability. And if you think they might not be working against DM, don't wait for somebody from the university to confirm that before you switch to something else. Pristine and Flint will provide good Botrytis control when used at the appropriate rate as a pre-bunch closure spray. Option B: Quintec, Vivando, or Torino [not yet labeled in NY] for PM control + captan (DM, Ph) or mancozeb (BR, DM, PH, but 66-day preharvest restriction and mite issues) as needed for these other diseases. If DM is the only other issue, Ridomil (in a bad year), a phosponate, copper, or one of the new DM-specific materials are additional options. Quintec, Vivando, and Pristine shouldn't be applied in more than two consecutive sprays. You may want to save one of your two Pristine shots for veraison or later, to pick up Botrytis and other rots. Option C: Revus Top (PM, BR, DM), Inspire Super (PM, BR, Bot), or Quadris Top (PM, BR, DM). Inspire Super will provide Bot control when applied pre-bunch closure, the low cyprodinil (Vanguard) rate that it provides might or might not be adequate, depending on pressure. If using this, you'll need to add something for DM on susceptible cultivars. Option D: Luna Experience [not yet labeled in NY] for excellent PM + Botrytis control + add something for DM control. See Luna Experience comments under Immediate Prebloom section. Option E: Rally, tebuconazole generics, or Mettle [no Mettle in NY] (PM, BR) PLUS mancozeb if still within the 66-day PHI limit (DM, BR) or one of the many DM options (captan, phosphites, new DM-specific materials discussed previously). Like the difenoconazole products, all of these DMI products provide excellent postinfection activity against BR, although they're not as effective against PM. Option F: Sulfur (PM) + the options listed above for BR and DM. In most years, lessening PM pressure makes this economical option increasingly practical as the season progresses. Option G: Copper + lime (DM, some PM). A reasonable PM option at this time for Concord and other native varieties in blocks where a spray is justified, generally not good enough for *vinifera* and susceptible hybrid cultivars.

**ADDITIONAL SUMMER SPRAYS.** Check the vineyard regularly to see what's needed, the main issues will be **PM** and **DM** on the foliage (remember, you'd like to keep foliage clean of PM into September). Also **Botrytis** on susceptible

cultivars, from veraison through pre-harvest. And the “**summer rot**” diseases (bitter rot, ripe rot) are potential threats in wet years, particularly in blocks or regions where they've occurred before.

On *vinifera* and other cultivars requiring continued **PM** control, use sulfur as an economical choice. See previous discussion in the PM section regarding sulfur residue issues.

DMIs, particularly the difenoconazole products, also are options; Revus Top is particularly attractive for the combined reasons of PM/BR/DM efficacy and cost (except on Concords, of course). But pay attention to previously-discussed maximum number of applications for all of these materials. Quintec or Vivando will certainly provide outstanding control if you need/want it and haven't used up your seasonal allotment yet. Ditto for Pristine and Luna Experience [no LE in NY, etc.] (save for later against Bot and perhaps other rots in the case of Pristine). And this can be an excellent time for the new Torino product [not yet registered in NY]. All of these materials provide the advantage of longer residual activity than sulfur in addition to the lack of concern about potential off-aromas. Copper + lime can be used on Concords, but mid-summer sprays for PM on this variety are probably worth the expense only under high crop and/or poor ripening conditions. Alternative materials such as Nutrol, Kaligreen, Armicarb, Regalia, Oxidate, Serenade, and Sonata can have their place during this period, especially if you're trying to avoid sulfur, although they generally need to be sprayed more frequently and most of them are not cheap. The well-documented ability of oils to decrease photosynthesis and consequently decrease Brix accumulation makes me a bit wary of recommending these products once the crop nears veraison, although a single application should be OK. For **DM**, there's the whole raft of products discussed previously. **Summer rots** are controlled with mancozeb, captan, and strobies; the peak period of susceptibility appears to be near veraison. Strongly consider an “insurance” application against **Botrytis** at or soon after veraison (depending on the weather), then determine the need for a subsequent pre-harvest spray based on weather and the need to limit spread of the disease, should it be revealed by scouting. **BR** should not be an issue after the second postbloom spray, except in very unusual circumstances (disease is established in the clusters of *vinifera* varieties, wet weather is forecast, and it's possible to direct sprays onto the clusters). **Ph** should not be an issue, period.



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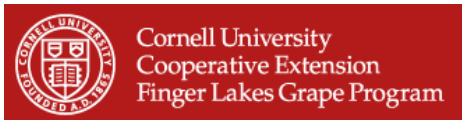
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