GRAPE DISEASE CONTROL, 2017

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It's (past) time once again for what has heretofore been a nearly annual update and review on controlling the fungal diseases that grape growers must regularly contend with in our eastern climate. As always, I'd like to acknowledge the outstanding team of grape pathologists here in Geneva, which includes bacteriologists (Tom Burr's program) and virologists (Marc Fuchs's program) in addition to those of us who work on fungal diseases: faculty colleagues and cooperators (David Gadoury, Lance-Cadle-Davidson); research technicians (Dave Combs and the now-retired Duane Riegel and Judy Burr); and graduate students and post-docs too numerous to mention now. Special recognition is also due to Rick Dunst, formerly with the Cornell program at the Lake Erie grape research facilities, for his (and the crew's) invaluable input on the work conducted there. I'd also be seriously remiss in failing to acknowledge the significant contributions of Cornell Cooperative Extension educators over the years, who often are the unsung heroes in facilitating on-farm field work and sampling, and who serve as absolutely essential "eyes and ears" (as do private-sector consultants) for university types who want to know what's really going on in the field rather than pretending that they do. It truly is the combined efforts of all of these people that serve as the basis for most of the following.

END OF THE LINE

As many of you know, I will be retiring from Cornell at the start of 2018. Without getting maudlin, I'd just like to say that this has been a dream job for me. And the best part of it by far (except for, perhaps, the "responsibility" of learning about industry products both on and off the clock) has been the opportunity to interact with so many growers, vineyard managers, private- and public sector consultants, and other industry support personnel over the years. (And some of my university colleagues, too). I thank you all for making it so enjoyable and rewarding. Although I do intend to renounce the less-pleasant aspects of the job as quickly as possible (even "dream" gigs have their share), I don't plan to disappear and will be looking for opportunities to maintain contact with the industry on a professional basis as well as a social one once I leave Cornell.

Our plant pathology unit has requested permission from Cornell administration to advertise for a new faculty member with general responsibilities similar to those that I have had (omitting reference to the industry products aspect, however). Few requests to search for new faculty hires are granted these days, but we are cautiously optimistic that we'll get the go-ahead to begin the long process this summer.

SHAMELESS COMMERCE DIVISION, REDUX

Just a reminder that the *Compendium of Grape Diseases, Disorders, and Pests 2nd Edition* was published shortly before the 2015 harvest began. It provides an updated and significantly more extensive treatment of most topics than the original (published in 1988), and includes detailed new sections focused on disease management topics such as fungicides and spray application

technology. In-depth treatments of grapevine anatomy and rootstocks are provided as well. This new edition is nearly twice the length of the original and contains 375 photos and illustrations, ¾ of them new.

Although technical jargon occasionally creeps in and is sometimes unavoidable, the book was written and edited with a focus on a primary readership of informed grape growers, their advisors, and industry support personnel who are not expected to have formal training in biology but are interested in gaining a deeper understanding of the subject matter than is available through the trade press or similar publications. The book is available through our plant pathologists' professional society (American Phytopathological Society) and Amazon. Just Google the title if interested.

FUNGICIDE CHANGES, NEWS, & REVIEWS

Several newish products are now or "soon" will be available to NY growers, with the latter group already in the mix for those outside our borders. Here's a rundown on them.

a. Luna Experience (LE). Five years after the federal government and all other affected states determined that LE is safe for use on grapes according to label directions, the NY regulatory authorities have finally come to the same conclusion and allowed its use in our state (except on Long Island). A quick review of some basics:

LE is a combination product consisting of two unrelated active ingredients, (i) tebuconazole, a traditional sterol-inhibitor or DMI (Group 3) fungicide, originally sold as Elite and now marketed as a generic under several different trade names; and (ii) fluopyram, one of several "next generation" SDHI (Group 7) fungicides. Just for reference, this group of fungicides dates back to a product from the 1960's, which had a very limited scope of activity and no relevance to grape growers. Near the turn of the millennium chemists made a significant change to the basic chemical structure of the group, greatly expanding the range of fungi affected and leading to the development of boscalid, the non-strobie component of Pristine. Since then, all of the major fungicide companies have been developing further spin-offs from the boscalid model, resulting in what I'm calling the "next generation" of these products. A number of them seem to be more active than boscalid against some specific diseases, although boscalid still has its place.

Luna Experience is labeled for powdery mildew control at a rate of 6.0–8.6 fl oz/A, and for Botrytis and black rot control at 8.0 – 8.6 fl oz/A (for the record, it's also labeled for control of Phomopsis at the higher rate, but is most likely to "control" this disease when there's very little or no rain). In our tests, we've looked at a rate of 6 fl oz/A for a number of years now, and have always obtained excellent control of powdery mildew with it. LE has typically been an excellent performer against Botrytis as well. Our results examining the rate response for Botrytis control have not been cut-and-dried, but if it were my vineyard, I'd be comfortable with the 6 fl oz rate from bloom through bunch closure, but would bump it up to at least 8 fl oz by veraison or later, especially if there was any pressure. You should also use the higher rate if relying upon this product for black rot control for the first few weeks following bloom when berries are highly susceptible.

Now for the fine print. The fluopyram component does the heavy lifting against powdery mildew and provides virtually all of the activity against Botrytis. However, it's the tebuconazole (DMI) component that provides most of the black rot activity. The 6.0-8.6 fl oz rate of LE provides a dose of tebuconazole equivalent to 2.8–4.0 oz of a 45DF (or 45 WSP) formulation of this DMI. For reference, 4.0 oz/A is the labeled rate of today's 45DF tebuconazole generics such as Tebustar and the original product, Elite, which always provided outstanding control of black rot in our trials. Hence the recommendation for the higher rate for dependable black rot control. Tebuconazole also once provided excellent control of powdery mildew at this higher rate, and still has very meaningful levels of activity against the disease; however, like all of the older DMI products it has been slipping for some years, and this slippage is even more pronounced when used at lower rates. Thus, if you are hoping that the tebuconazole component will control any powdery mildew individuals resistant to the Group 7 materials, you're better off with the higher rate for this purpose as well. Or theoretically, you could add 1.2 oz of a 45 WSP or 45DF generic tebuconazole product to the lower rate of Luna Experience and wind up with the higher rate of the tebuconazole component, most likely at a somewhat lower cost.

Regarding resistance: remember that Pristine and Endura (a boscalid-only product registered on grapes but not used extensively) have been on the market for nearly 15 years. The vast majority of growers have strictly limited their use, and whereas we haven't seen evidence of resistance in powdery mildew there have been a few reports from other regions of Botrytis resistance in grapes and many instances of Botrytis resistance in other crops such as strawberries. Thus, it is important to continue limiting the number of applications of ALL Group 7 products COMBINED (two per season is a conservative but pretty safe number, although labels allow more). There is strong evidence that all fungal individuals resistant to boscalid are not also cross-resistant to various next-generation Group 7 products, but some of them are. Which means that IF powdery mildew or Botrytis control seems to have been slipping in recent years with Pristine (or Endura), LE or another Group 7 product MIGHT provide better control if the cause was the increasing presence of some resistant fungal colonies in the vineyard population. (They MIGHT also provide better control IF they're simply more active compounds). That being said, all Group 7 products should be considered as a single entity in terms of rotations and limiting the total number of seasonal sprays, although there have been some marketing efforts to the contrary.

b. Zampro now available in NY (except Long Island). Now that (most) NY growers can now actually purchase and use this product, a final quick review for those to whom this was only a theoretical consideration before:

Zampro is a combination product that contains two active ingredients: (i) dimethomorph, a fungicide that is in the same group as the active ingredient of Revus (Group 40); and (ii) ametoctradin, which is new chemistry unrelated to any other fungicide now on the market. The Group 40 materials have some post-infection activity but generally are not absorbed well by plant tissues, causing them to be strongest in a protective mode--since for a fungicide to exert post-infection activity, it needs to get inside the plant tissue where the fungus is residing after infection. (Speculation: Post-infection activity of the Group 40 products might be improved through the addition of a penetrating surfactant, e.g., non-ionic or, especially, organosilicate). Anecdotal observations suggest that ametoctradin has significant post-infection activity in

addition to protective activity, although solid, publicly available data on this subject are very limited.

The 2014 and 2015 growing seasons were both excellent for testing downy mildew fungicides in the Finger Lakes due to very high disease pressure. Zampro provided excellent disease control in both years, as did Revus Top (and LifeGard, a biological product discussed below). Some specific data from these trials are provided in the Downy Mildew section later on.

c. Flutriafol products. Flutriafol is a DMI (a.k.a. sterol inhibitor, Group 3) fungicide that we've been looking at for a number of years. It was registered in most states last year and is now registered in NY (except Long Island), both as a "solo" (Rhyme) and "combination" (Topguard EQ) product. Rhyme (the solo product) is labeled for use at 4-5 fl oz/A. In our testing, we've found the 4 fl oz rate to be marginal versus powdery mildew, sometimes equivalent to higher rates but other times a bit weaker, whereas the 5 fl oz rate has provided consistent results: relative to other DMI products, I'd rate it as a bit better than Rally and tebuconazole products, more or less equivalent to Mettle, but not quite as good as difenoconazole (the DMI component of Revus Top, Inspire Super, Quadris Top). In fairness, these are sometimes "hair-splitting" differences that can be apparent in the high-pressure test situations that we employ (Chardonnay vines in a setting with high inoculum carryover from the previous year and unsprayed vines scattered throughout the plot to maintain increasing pressure throughout the season), but they are less likely to be meaningful in well-managed vineyards with much lower inoculum levels and attendant disease pressure. Like most of the other DMI fungicides, flutriafol products have also has provided excellent activity against black rot in trials run elsewhere.

The combination product is Topguard EQ, which also includes azoxystrobin, the active ingredient from Abound and other more recent generic products. The labeled rate is 5-6 fl oz/A for powdery mildew and black rot control; as with Rhyme, the higher rate should be a bit more consistent (see Table 1 to compare the actual amount of active ingredient provided by the different formulations). Up to 8 fl oz/A is allowed, but this higher rate appears designed primarily to provide additional azoxystrobin to target other diseases for which it shouldn't be relied upon anyway, i.e., downy mildew (significant resistance concerns), Phomopsis (weak), and Botrytis (weak).

Bottom line: the two contributions of the azoxystrobin in this mix are (i) it should provide some additional control of powdery beyond that provided by the flutriafol component, at least for that proportion of the pathogen population that is not resistant to the strobies; and (ii) its protective (forward) activity against black rot should significantly complement the post-infection activity of the flutriafol component, making the combination outstanding against this disease. Note that Topguard EQ and Quadris Top, a product released a few years ago, both contain a mixture of azoxystrobin and a DMI fungicide, the principal differences being that (a) the DMI in Quadris Top (difenoconazole) appears to be a bit more active against powdery mildew under high pressure conditions, and (b) Quadris Top also provides somewhat more azoxystrobin at the rates labeled for powdery mildew and black rot control (Table 1). Not sure how the two compare in price.

d. Merivon. This was once intended as a replacement product for Pristine, produced by the same company (BASF). It is now registered on wine (and raisin) grapes via a "supplemental label" through the end of 2018, even in NY (with the usual exception of Long Island). There are some major drawbacks to the use restrictions on the label and it's questionable whether many growers will want to use the product with these in place, but here's the scoop.

Merivon is a combination product containing pyraclostrobin--the strobie component of Pristine--plus a "next generation" Group 7 fungicide (fluxopyroxad), to replace Pristine's original Group 7 component (boscalid). When we began testing it a while back, Merivon provided somewhat better control of powdery mildew than Pristine and significantly better control of Botrytis, even though we were applying the same rate of pyraclostrobin. Clearly, the next-gen Group 7 component was more active than the older boscalid component, as discussed earlier.

Unfortunately, some unspecified phytotoxicity problems developed in other locations during the testing process, and the company stopped developing the product on grapes several years ago. Then, seemingly out of the blue, this limited-time supplemental label was submitted and approved just recently. Why the change of heart on the part of company? Answer: Powdery mildew resistance to the QoI (strobilurin) fungicides finally hit California hard last year. Thus, BASF apparently wanted to have their better Group 7 mixed with pyraclostrobin, since it's the Group 7 mixture component that will be carrying an increasingly large share of the load going forward. To address their fears of phytotoxicity (and associated lawsuits), they've limited the rate to 4.0-5.5 fl oz/A, which provides the same amount of pyraclostrobin as 8-11 oz/A of Pristine (Table 1) and has provided outstanding control of powdery mildew in the few trials that we've run in years past. Higher rates (8-11 fl oz/A) were tested against Botrytis, and even at 8 fl oz, we got 93% control of the disease in one trial versus 64 and 54% control with Pristine at 19 oz and 12.5 oz, respectively. Unfortunately we never did test Merivon at 5.5 fl oz, nor is Botrytis control or suppression claimed on the supplemental label.

So even at the current rate range, there appears to be some promise from an efficacy point of view. However, in a further attempt to avoid a possibility of plant injury, the company has also placed a restriction on the label that prohibits the use of Merivon in any sort of tank mix with other pesticides or with any surfactant additive (spreaders, spreader-stickers, etc.), to reduce uptake. The tank-mix restriction obviously makes it impractical to use in most sprays in our part of the world since we often have multiple diseases and pests to control with each spray application, but such is not necessarily true in California and other western states, where most of the grape acreage is.

e. Fracture. Fracture is a product whose active ingredient is a fragment of a naturally occurring plant protein, and which has been registered for use on grapes for a couple of years. It has a 4-hr REI and a 1-day PHI, and the residue of its active ingredient is exempt from tolerance by the US-EPA (i. e., it is considered safe enough to humans that there is no limit on the allowable residue level in/on food products). We've gotten good control of Botrytis in several trials and have seen some activity against sour rot as well. Although labeled for powdery mildew control, it wasn't that impressive in the one trial we've run with that disease, nor should it have activity against downy mildew. It's pricey.

f. Aprovia. Aprovia (solatenol) is another entrant in the list of "next generation" SDHI or Group 7 fungicides. Although labeled for use in most states before last season began, NY approval is still awaiting. Unlike the other three grape products containing Group 7 materials (Luna Experience, Pristine, and Merivon), Aprovia is a "solo" product, there is no second active ingredient mixed with the solatenol.

Aprovia has provided excellent/outstanding control of powdery mildew over several years in my trials on Chardonnay, but unlike the Group 7 components of the other three products listed above, it does not provide significant control of Botrytis. In addition to powdery mildew, the label does claim control of angular leaf scorch (ALS), anthracnose, black rot, and Phomopsis. I have no personal experience with the product versus these diseases, nor have I seen independent data for efficacy against ALS, anthracnose, or Phomopsis. However, there is a growing body of data from elsewhere concerning black rot control, and it does not look very good. For example, in a high-pressure trial run by Bryan Hed from Penn State, a rotational program that included Pristine in the two critical sprays at bloom plus 2 weeks later (followed by ziram and another Pristine) provided 96% control of the disease on clusters. In contrast, the very same program in which Aprovia was substituted for Pristine in the three relevant sprays averaged only 50% control in two different treatments utilizing different labeled rates of Aprovia. As the old saying goes, let the buyer beware.

g. LifeGard. LifeGard is a new biological product that recently received federal registration and is approved for use in most states, although we're still waiting for NY to come through (shock. It is the first biological that has provided good (not to mention, excellent) control of downy mildew in my trials. I only have results from two trials--in 2014 and 2015 (no rain = no disease in 2016)--and in one of them we mistakenly applied a very excessive rate. Nevertheless, in both of those years our unsprayed vines were defoliated from downy mildew and both years LifeGard provided control comparable to our best standard materials.

Another old saying goes, if it sounds too good to be true it probably is. My own experience with the product is very limited and I've seen results from other trials where it didn't look nearly this good, so I'm still cautious about it. Nevertheless, if this were a new conventional fungicide, I'd do just what I'm doing now: provide my results with the big caveat that they are very limited, they haven't been repeated by others (that I know of), and we could see something entirely different in 2017. I think this caution should hold doubly true with an unproven technology, which LifeGard is said to employ (see next paragraph). Nevertheless, for those who are interested in such materials (OMRI listed, 4 hr REI, 0 day PHI), it may be worth experimenting with.

LifeGard contains a bacterium that purportedly acts by triggering plants' natural defense mechanisms against pathogens. There have been numerous efforts for several decades to develop such a product that can provide meaningful levels of control of important diseases, on a consistent basis. Virtually all of them have come up short, especially on grapes, which seems to be a plant in which so-called "induced systemic resistance" (ISR) responses are difficult to achieve. Although an ISR should have an effect against all diseases, on grapes LifeGard is labeled only for control of downy mildew and in the one non-definitive trial where we looked at

it against powdery mildew, there didn't seem to be much effect. We'll be looking at it again this year on powdery mildew and Botrytis in addition to downy mildew, so stay tuned.

h. "Combination product" trend. As the previous run-down of newish products indicates, there is a trend within the pesticide industry to release new products that contain a combination of two (or even more) active ingredients. With fungicides, there are several different and sometime complimentary rationales for doing so: expand the range of diseases controlled, reduce the risk of developing resistance to one or more at-risk components, provide an opportunity to keep selling one of your active ingredients that is no longer under patent.

These mixtures often can be beneficial, but it also can be confusing as to which diseases are controlled by which component/s of a mixture, and what rates are provided by the individual components relative to other products that contain the same ingredient (such confusion and lack of transparency is not always accidental from a marketing perspective).

Clearly, the rate of each component in the mixture matters, especially when one is low against a disease of interest and its companion fungicide is ineffective against that same disease, either by nature or because a portion of the pathogen population is resistant to it. To shed a bit of light on the relative rates of the individual components provided by various combination products, I've prepared the following table, which contains minor updates to a similar table in the 2017 NY and PA Pest Management Guidelines for Grapes.

Table 1. Relative doses of individual active ingredients provided by "combination products" when applied at label rates

		Active ingredient, amount provided (oz/A)								
Product	Label rate (per acre)	azoxystrobin	copper hydroxide	cyprodinil	difenoconazole	fluxapyroxad	mancozeb	mandipropamid	pyraclostrobin	tebuconazole
Abound 2.08SC, Azaka 2.08SC	10.0-15.5 fl oz	2.56-4.0								
DithaneM45 80WP	1.5-4.0 lb						19.2-51.2			
Gavel 75DF*	2.0-2.5 lb						21.3-26.7			
Inspire Super	16-20 fl oz			4.18-5.23	1.46-1.83					
Kocide 2000	1.5-3.0 lb		12.9-25.8							
Luna Experience 3.3SC*	6.0-8.6 fl oz									1.25-1.80
Merivon	4.0-5.5 fl oz								1.03-1.41	
Pristine	8-23 oz								1.02-2.94	
Quadris Top 2.7SC	12-14 fl oz	2.51-2.92			1.58-1.83					
Revus Top 4SC	7 fl oz				1.82			1.82		
Revus 2.08SC	8 fl oz							2.08		
Rhyme 2.08SC	4-5 fl oz					1.04-1.30				
Ridomil Gold Copper*	2.5 lb		24.0							
Ridomil Gold MZ*	2.5 lb						25.6			
Switch 62.5WG*	11-14 oz			4.13-5.25						
Topguard EQ	5.0-8.0 fl oz	1.54-2.46				1.14-1.82				
Tebuconazole 45DF (generics)	4.0 oz									1.80
Vangard 75WF	10 oz			7.5						

*Only the active ingredient in common with one or more other registered products is listed

i. Recap from last year. Several other somewhat new materials were discussed in this section last year. Included were the powdery mildew-specific products, Vivando and Torino; the polyoxin-D products (Oso, Ph-D); Botector, a biocontrol product for Botrytis control; Regalia, a plant extract that's another purported "induced systemic resistance" elicitor, but which seems to be most active against powdery mildew, perhaps via direct physical contact; and Double Nickel, a natural fermentation product derived from a bacterial species related to those that produce Serenade and Sonata. Rather than repeat this information again for the nth time, I'll simply refer you to the 2016 version of the tome if interested. Pretty easy to find via Google, or try this link: http://www.fruit.cornell.edu/grape/pdfs/Wilcox-Grape%20Disease%20Control%202016.pdf

• *Biopesticides and disease pressure*. There's an increasing number of new biopesticides hitting the marketplace, in part because of their obvious appeals if they work and in part because (relatedly) they're easier to register than conventional materials. As discussed previously, the live organisms and natural products produced by them that are sold for disease control rarely have the same level of activity as the standard synthetic fungicides used in grape production. This becomes all the more apparent when we test various products under high disease pressure, which is intended to differentiate the stronger from the less-strong materials. However, anybody who plans to rely on biopesticides (or at least, anyone who plans to do so and remain in business) knows that they must to be viewed merely as one component within a much broader integrated system that stresses limiting inoculum, the use of cultural techniques to limit disease

development, and perhaps utilizing less-susceptible cultivars if such are available for the intended market.

Case in point: a couple of years ago we conducted two powdery mildew control trials in different settings. One was in a vineyard of highly susceptible 'Chardonnay' where inoculum was abundant and pressure was high; 99% of the surface area of unsprayed clusters was diseased by late summer. The other was in a vineyard of the moderately susceptible hybrid 'Rosette' where inoculum was less abundant and pressure was moderate; here, "only" 40% of the surface area of unsprayed clusters became diseased. Result: the biopesticide Double Nickel provided only 24% control relative to the unsprayed vines in the Chardonnay vineyard even when applied every 7 days, whereas it provided 92% control in the Rosette vineyard when applied every 14 days. Similarly, when the biopesticide Oso was alternated with JMS Stylet Oil at 2-week intervals (both vineyards), it provided only 57% control in the Chardonnay vineyard but 97% control in the Rosette vineyard.

• *Strobilurin resistance*. One last time: Do NOT depend on any of the strobies to provide control of powdery or downy mildew when used alone. There may be exceptions in blocks of at least moderately resistant cultivars (some natives and hybrids) with a limited history of using these products, but it's risky. Fortunately, we have a couple of competitive products (Pristine, Quadris Top, Topguard EQ) in which a strobie is combined with an unrelated fungicide that gives at least very good control of powdery mildew *at appropriate rates*. Thus, where a significant proportion of the pathogen populations is still susceptible to the strobies (no previous control failures), the additive effects of the two mixing partners generally adds up to excellent powdery mildew control.

Unfortunately, we do not have similar mixtures (with the possible exception of Tanos), price-competitive or otherwise, of a strobie with a partner fungicide that provides control of downy mildew. Therefore, most growers who may still wish to use Pristine or one of the other strobie combination products to control diseases other than DM should tank mix it with something that they know will control downy mildew if they want to be on the safe side, even if they have not had DM resistance problems before. The nature of strobilurin/Group 11 resistance is that it can hit like a ton of bricks without warning, e.g., see the photo below (Fig. 1), taken a couple of years ago in a Merlot vineyard of an excellent Finger Lakes grower who was not aware that two Pristine applications made during the critical period near bloom were no longer effective against downy mildew, even though they had been in previous years. For what it's worth, black rot resistance has not been reported from anywhere in the world where it occurs. As explained in another section, the danger for BR resistance is much lower than for DM, PM, or Botrytis, although it could develop at some point.

Figure 1. Pre-harvest downy mildew damage found in 2014 throughout a Finger Lakes Merlot planting treated twice with a strobilurin fungicide near bloom. Resistance had not been recognized in this planting previously.

SPEAKING OF FUNGICIDE RESISTANCE. Although this topic has been covered thoroughly in the past, it's important enough that it's worth one final review. As noted previously, it's a virtual certainty that the phenomenon of fungicide resistance will only continue to increase in importance into the future, since modern synthetic fungicides are almost invariably more prone to resistance development than the old traditional, "multi-site inhibitors" such as mancozeb, captan, ziram, sulfur, copper, etc. And as I try to stress every year, paying attention to basic resistance management principles and practices will be essential to sustain the utility of virtually any new highly-active product that we are likely to see and want to use, not to mention all but a handful of our current effective products.

Remember, anything new that's going to get registered into the foreseeable future has to be almost squeaky clean in the many tests that regulators use to assess possible effects against what are euphemistically called "non-target organisms " (you, me, multiple other life forms besides disease-causing fungi, etc.). It's not that hard to register a product that doesn't cause much harm to anything, including its supposed target organisms. (Be aware that in the U.S., product registration requires only that a product is shown to be safe; by law, "adequate" efficacy is defined by the marketplace and not by regulators, so just because a label says that a product controls a disease there is no legal reason that it has to). But unfortunately, it's very rare to find a compound that's deadly to target fungi at a certain use rate while still being (nearly) benign to most other life forms.

When such a compound is discovered, the molecule typically affects only a single process in the fungal metabolism, and often by disrupting just one specific site within one fungal enzyme that

governs that process. This is the so-called lock-and-key analogy, by which the fungicide molecule "key" physically fits into the fungal enzyme "lock" (i.e., it binds to that protein at a specific site, for you biology geeks), thereby preventing the enzyme--and the pathogen--from functioning. The upside to such specific activity is that low doses of these materials are often very effective at controlling disease yet are quite non-toxic to at least most non-target organisms. The downside is that only a subtle change (mutation) to that one enzyme "lock" may be all that's needed for the fungicide key to no longer fit and therefore to have no effect. If this happens, and the change does not significantly impair the functioning of the enzyme, the fungus survives treatment and reproduces to form lots of progeny that also have this altered "lock". And of course, these progeny also survive treatment with the fungicide and produce even more of their own offspring, etc. The end result is that we end up with resistance to the fungicide and all related materials that work by fitting into the same original "lock".

As just illustrated, fungicide resistance is a classic albeit rapid example of evolution ("evolution on steroids"), i.e., it is the result of the preferential survival and reproduction of individuals with a specific genetic characteristic (mutated target enzyme that doesn't bind to the fungicide) in response to the "selection pressure" provided by sprays of that material. Eventually, this process progresses to the point that such individuals dominate the pathogen population to an extent that the fungicide no longer provides acceptable disease control even when applied properly, and the ball game is over. The risk of this occurring is a product of two different factors: (i) the mode of action of the fungicide itself (i.e., what are the chances that a simple mutation to the target-site enzyme "lock" will occur which makes the fungicide unable to bind and exert activity); and (ii) the individual disease involved.

The various fungicide groups have been assigned a rating by an industry consortium (the Fungicide Resistance Action Committee, FRAC) to reflect the relative risk of resistance developing to them. Some of these ratings are theoretical but most are based upon worldwide experience with the products, so ratings of newer groups can change over time as more experience is gained. Nevertheless, they tend to be pretty accurate and informative. Again, these are relative rankings, which does not mean that resistance is unlikely to develop to a group rated as low-to-medium risk if products in it are over-used. Rather, it means that for any given disease, resistance is likely to develop first and with less use for a high-risk group. Within this scheme, some of the major grape fungicide groups fall into the following categories:

- High risk: The QoI/strobilurins (Group 11); Ridomil products (Group 4); benzimidazoles (the long-defunct Benlate and current Topsin-M, Group 1).

- Medium-to-high risk: The SDHI (Group 7) fungicides, discussed previously; Rovral (Group 2); ametoctradin (one component of Zampro, Group 45); Ranman (Group 21)

- Medium risk: The DMI fungicides (Group 3); the AP (Group 9) fungicides (Vangard, Scala, one component of Switch); Quintec (Group 13); Vivando (Group U 08)

- Low-to-medium risk: The Group 40 fungicides (Revus/Revus Top, one component of Zampro); Elevate (Group 17); fludioxonil (one component of Switch, Group 12).

Note that although fungicides in these group are designated as low–to-medium risk, a case of practical resistance to the Group 40 component of Revus Top was confirmed by Anton Baudoin in Virginia last year, i.e., poor control of downy mildew in the examined vineyard was associated with a preponderance of resistant pathogen individuals within. "Low-to-medium" risk does NOT mean no risk.

• Low risk: Mancozeb, captan, ziram, sulfur, copper, oils, salts (e.g., bicarbonates)

With respect to the disease part of the equation, those at the greatest risk for resistance development are caused by pathogens which (i) can produce multiple generations per year (i.e., the few resistant individuals that might arise through mutation can become a few million rather quickly if the weather is favorable and there's nothing stopping their spread and multiplication); and (ii) also produce a large number of spores that can be widely dispersed by air currents, thereby spreading the resistant strain far and wide (share the love!). Among plant diseases that satisfy both of these criteria in spades, three of the most notorious are powdery mildews, downy mildews, and Botrytis. Grape growers are just "lucky" to hit this trifecta and have to deal with the whole lot.

In contrast, diseases at the least risk are those with a limited number of annual disease cycles, caused by pathogens with a limited potential for dispersal: Phomopsis cane and leaf spot is a prime example on both of these accounts, having but one disease cycle (fungal generation) per year and spores that are distributed only short distances by splashing rain. Black rot lies somewhere in between, having a generation period that's several times longer than those of the mildews, a limited period of susceptibility for the host tissue most likely to perpetuate the fungus between years (berries), and a spore type responsible for spreading the disease that is distributed only a short distance by rain splash.

Resistance to a fungicide is said to be qualitative (yes/no, black/white) when individuals within the pathogen population are either sensitive to the typical range of doses applied in the field or are virtually immune to even 100 or 1,000 times those levels. Such immune individuals are very rare before the fungicide is ever used (or else it wouldn't work from Day 1), but unless they are controlled in some other manner such as applying unrelated effective fungicides, the only thing checking their reproduction is the weather and whatever cultural techniques might be employed. Thus, in a year where the weather provides multiple infection (reproduction) events throughout the season, the pathogen population can quickly become dominated by the immune individuals and control failures occur suddenly if the resistant fungicide is the only thing really standing in their way. Which is just what happened in some NY vineyards where the strobies failed to control downy mildew in 2014 (years earlier in some states to our south) or powdery mildew way back in 2002. And of course, this will happen first and most spectacularly on extremely susceptible cultivars, where there's not even a smidgen of host resistance to slow the whole process down. Which is exactly what happened with Chardonnay for powdery mildew with the strobies way back when and many *V. vinifera* cultivars (especially Merlot) for downy more recently.

Examples of fungicides to which such qualitative resistance (immunity) among grape pathogens has either occurred within or threatens eastern US vineyards include (i) the strobilurin and other

QoI fungicides (Abound and other azoxystrobins, Flint, Sovran, Reason, one component of Pristine), where downy mildew resistance is common in many regions, resistance among powdery mildew populations seems to be growing outside its NY "epicenter" (significant problems hit California in 2016), and Botrytis resistance is being found by Anton Baudoin wherever he looks for it in Virginia (could well be in NY, too); (ii) the Ridomil and generic equivalents, where downy mildew resistance is common in regions throughout the world wherever these materials have been used more than sparingly (so let's keep using them sparingly here!); and (iii)

In contrast to the above model, resistance is said to be quantitative when individuals poorly controlled by one dose (or rate, loosely speaking) of the material may be controlled by either (i) incremental increases in that dose, or (ii) the substitution of a similar dose of a related material that has greater intrinsic activity—that is, 1 oz of the active ingredient in Fungicide A provides more control than 1 ounce of active ingredient in related Fungicide B. With quantitative resistance, repeated use of the same fungicide group results in a gradual "shift" in the overall sensitivity to that class within the pathogen population, with a progressively greater proportion of the fungal individuals requiring progressively higher doses of any one fungicide before a given level of control is obtained. In other words, you need to keep bumping up the rate over time just to stay even.

Note that unlike the yes/ no or white/ black type control scenario due to resistance as described above for the qualitative type, this quantitative type of resistance results in a yes/ kind of/ not really control scenario (or white/ light gray/ dark gray to stay with the same metaphor). A well-characterized example of quantitative resistance is that exhibited towards the DMI (Group 3) fungicides that we've been using for 30+ years now against powdery mildew, with varying rates of success and discussing for nearly as long. A very practical illustration of how this principle applies to both long-term resistance management and immediate disease control will be reviewed one last time below.

All of our resistance management strategies have at least one of two very basic and obvious goals: (1) Limit the preferential selection of resistant individuals in the first place; and (2) When resistant individuals are selected, limit their reproduction and spread. Thus, the basic resistance management strategies for all fungicide groups include:

• Limit the number of selection events, i.e., limit the number of sprays of any at-risk fungicide group. A no-brainer if there ever was one.

• Limit the size of the pathogen population from which you might be selecting resistant individuals, thereby limiting the potential number of resistant survivors. In English: Try to avoid using a material at significant risk of resistance development as a "rescue" treatment if a severe disease outbreak occurs. Of course, you might legitimately wonder about the wisdom of maintaining the future utility of a fungicide for a business that could have little future itself if a disease outbreak isn't brought under control immediately. But at least stop to think whether there might be other acceptable fungicide options before taking this plunge. There often are.

• Limit the reproduction of resistant individuals that have been selected, i.e., survived exposure to the at-risk fungicide. That is, don't let them build up and spread the disease. This can be accomplished several ways:

(i) Utilize appropriate cultural practices to limit disease development (pathogen reproduction). This usually won't do the trick all by itself, but it's always the first line of defense and more influential than many people realize in terms of delaying resistance development.

(ii) Rotate at-risk fungicides with effective, unrelated materials. Of course, this is part and parcel of the previous recommendation to limit the total number of sprays of an at-risk fungicide, since by doing so you'll apply something else unless you just quit spraying altogether (not recommended). But there's a conceptual difference for those who care to think of it this way: limiting the total number of sprays of any one fungicide group reduces the number of "selection events" that favor the survival of individuals resistant to that mode of action, whereas application of the rotational partner limits the potential for any such survivors to reproduce.

Within this context, a conservative (and safe) recommendation is never to apply products in the same Resistance Group twice in a row, i.e., always alternate with a different type of material. This minimizes the period of time during which resistant survivors might reproduce before you clobber them with something else. A more liberal approach would be not to apply them more than two times in a row before rotating. The greater the risk of resistance development (fungicide x disease combination, as discussed above), the more conservative you should be, especially once that group of products has been used for a while in the vineyard.

(iii) Apply at-risk materials in combination with another unrelated fungicide that's active against the target disease, either through tank mixing or use of a pre-packaged product containing two or more effective ingredients. Remember that resistance management efforts dependent upon rotation and/or combination with unrelated fungicides can only be as effective as the companion materials themselves: a weak companion material or a product that provides a low rate of the companion (unfortunately, a common problem with prepackaged mixtures) will have a limited effect on slowing the reproduction of resistant individuals that survive exposure to the at-risk ingredient.

• 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 such fungicides. 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 that has a greater intrinsic activity (1 oz of fungicide A provides more control than 1 oz of fungicide B).

Data presented in Table 2 below illustrate this concept vividly. Note that in this particular trial, Rally (active ingredient = myclobutanil) provided virtually ZERO control of powdery mildew on the Chardonnay clusters when used alone all season long. In contrast, the 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 lab tests showed that when averaged across the 26 powdery mildew colonies tested, 1

oz (or gram, etc.) of difenoconazole provided the same control as 26 oz (or grams, etc.) of myclobutanil. Quite simply, the population of the PM fungus in this vineyard has shifted to the point that the majority of individuals are no longer controlled by the maximum legal rate of Rally (which was fully adequate before the population became dominated by less-sensitive individuals), whereas it is still effectively controlled by a similar dose of difenoconazole, which intrinsically is far more active against the fungus.

And don't forget, maximizing spray coverage will also maximize the dose of fungicide that any fungal target is actually exposed to at any given rate of application coming out of the sprayer. The fungus only responds to the dose of product on the part of the plant it's trying to infect, it doesn't care how much you put into the spray tank and deposit somewhere else.

Table 2. Control of powdery mildew on Chardonnay grapes; Geneva, NY 2010

	Leaf infection		Cluster infection	
Treatment, rate/A*	% Leaves	% Lf area	% Clusters	% Clstr area
None	100	70.2	100	99.5
Revus Top, 7 fl oz**	64	1.7	27	3.2
Inspire Super, 16 fl oz	67	2.1	16	2.0
Inspire Super, 20 fl oz**	39	1.1	6	0.2
Rally, 5 oz	100	33.2	100	96.7
Vivando, 10 fl oz	12	0.3	12	0.4
Vivando, 15 fl oz	6	0.1	0	0.0

* Seven sprays applied at 14-day intervals.
** Inspire Super at 20 fl oz contains the same dose of difenoconazole as the Revus Top treatment.

RECAPPING FROM LAST YEAR: FUNGICIDE SENSITIVITIES OF NORTHERN GRAPE CULTIVARS. The development of a several new cold-hardy grape cultivars has expanded the geographical boundaries of commercial grape production, bringing new questions to the viticultural community at large, where experience with these cultivars is usually limited at best. In terms of disease management, there have been a few surprises (who knew Marquette was so susceptible to anthracnose until there were widespread plantings?) and some basic questions without clear answers. One of these is cultivar tolerance to several common fungicides (copper, sulfur, and the DMI difenoconazole [Revus Top, Quadris Top, Inspire Super]) that are used without problem on *V. vinifera* cultivars but which can cause varying levels of injury on some of our more common natives and hybrids.

Recently, my colleague Dr. Patty McManus at the University of Wisconsin published the results of a study that she initiated on this topic in 2012, in which she examined 10 of these cultivars in field trials where a few hybrids were included for comparison. The long version is a must-read for current and potential growers of these grapes and those who advise them, most of whom I hope are already aware of it from the February 2016 newsletter of the Northern Grapes Project: http://northerngrapesproject.org/wp-content/uploads/2016/02/NG-News-Vol5-I14-Feb2016.pdf For those who may have missed it and are interested, a very brief synopsis of the take-home conclusions:

• *Copper*. Brianna was deemed sensitive enough that it should not be treated with fungicides containing copper. It was recommended that copper use be restricted to one or two sprays per season on Frontenac, Frontenac gris, LaCrescent, Leon Millot, Maréchal Foch, Marquette, and St. Croix. There were no apparent problems on La Crosse, MN1220, Noiret, NY76, Petite Pearl, Valiant, or Vignoles.

• *Sulfur*. Patty and colleagues concluded that Brianna, Leon Millot and Maréchal Foch should not be treated with sulfur (the sensitivity of Maréchal Foch and its sibling Leon Millot was already known, so the injury that developed in these trials attests to the accuracy of the results in general). They also recommend that sulfur use be restricted to one or two sprays per season on LaCrescent and St. Croix. Frontenac, Frontenac gris, La Crosse, MN1220, Marquette, Noiret, NY76, Petite Pearl, Valiant, and Vignoles we OK.

• *Difenoconazole*. Only Noiret showed occasional injury, consistent with observations of minor injury on this cultivar in NY. All of the others were OK. (Note that in limited observations in NY after Revus Top was first released and this issue was just being recognized, damage also was observed on Brianna and minor damage was observed on St. Croix).

POWDERY MILDEW (PM) OVERVIEW AND REMINDERS

Another review of PM biology with respect to management considerations.

(i) The fungus overwinters as minute fruiting bodies (chasmothecia, which used to be called cleistotheia) 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, most such spores of any consequence are "usually" discharged between bud break and bloom (more or less) to initiate the disease. These first "primary" infections then produce millions of new "secondary" spores, which can spread the disease rapidly via multiple repeating cycles of infection throughout the rest of the season, so long as susceptible tissue is present and the environment is conductive. Now we come to the fine print.

Powdery mildew is a classic example of a so-called "compound interest" disease. In this illustrative analogy, the final "yield" (amount of disease at the end of the year) is a function of two variables (a) the initial "deposit" (how much primary inoculum you start the season with); and (b) the rate of return on that deposit, i.e., how fast the disease spreads after the initial primary infections occur (as affected by the pathogen's own reproductive rate, environmental conditions, cultivar susceptibility, and roadblocks that growers erect via cultural management and spray applications). Thus, for PM, the size of the "initial deposit" capable of starting disease this year (the number of chasmothecia present at bud break) is directly proportional to the amount of disease that developed last year. Which means that disease pressure will be higher, and PM sprays during the first few weeks of shoot growth are likely to be particularly critical, in blocks where PM control lapsed last year as compared to blocks that remained "clean" into September. (In much of the Northeast, chasmothecia initiating 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). Fortunately, 2016 was a pretty light year for PM throughout much of NY, and most blocks were pretty clean going into the fall.

The annual illustration of putting some specific numbers to this general concept: Some years ago, we conducted an experiment in a Chardonnay vineyard where we either (a) sprayed up through Labor Day, maintaining a clean canopy 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 a third group of vines in early July, to represent a planting where PM control broke down for one reason or another. The next spring, the relative levels of chasmothecia in these three treatments (number per kilogram of bark, to be specific) 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 some 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, this makes a difference. When we intentionally applied a weak spray program to these same vines the year *after* the variable foliar disease levels were allowed to develop, the resulting cluster disease severity (proportion of the cluster area infected) was (a) 11%, (b) 22%, and (c) 48% in these respective groups, even though all vines were sprayed exactly the same during the second season. Or using our banking analogy, the current-season rate of interest was the same across all three groups of vines, but the initial deposit was different.

The basic concept that the degree of control provided in one season can have a big effect on the success of the control program (or its required intensity) the following year is one that we're all aware of, but here's a concrete example of just how important it is to reduce inoculum levels in a vineyard as part of a total disease management program. Depending on the disease involved, inoculum reduction can sometimes be provided by sanitation procedures that remove diseased plant organs (e.g., cankered wood, black rot mummies, old Botrytis-infected cluster stems) before the season begins. But for many diseases, and PM in particular, the easiest way to minimize inoculum levels at the start of one season is to minimize disease development the previous year, by implementing good control programs then. This is a major reason that some blocks are almost always clean and some are almost always otherwise, i.e., it becomes either a virtuous or a vicious circle. "Emergencies" can sometimes be addressed via eradicative sprays--e.g., Stylet Oil or Oxidate to kill late summer colonies before they produce chasmothecia or delayed dormant sprays of lime sulfur to kill these overwintering structures on the vine before they initiate infection in the spring—but it's a lot more effective, and usually cheaper and easier, to keep these emergencies from developing in the first place.

(ii) A critical factor that governs the rate of disease spread is temperature, with a new generation of the PM fungus produced every 5 to 7 days at constant temps between the mid-60's and mid-80's (°F); 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 with nights in the 60's or 70's provide ideal conditions for the fungus 24 hr a day. Conversely, cooler temperatures prolong the generation time (e.g., 11 days at a constant temperature of 59°F) and a very cold night or two below 40°F or

so can seriously set the fungus back while it's trying to ramp up in the spring, as discussed a little farther on.

(iii) High humidity also increases disease severity, with optimum conditions for development being about 85% RH. Unlike all of our other fungal diseases, PM doesn't require rain for the disease to spread and it develops to some extent over the entire range of humidity that we experience during the growing season. However, research has shown that disease severity is twice as great at a relative humidity of 80% versus an RH of 40%. Thus, vineyard sites (and canopies) subject to poor air circulation and increased microclimate humidity, and seasons with frequent precipitation, provide a significantly greater risk for PM development than their drier counterparts. Of course, thick canopies and seasons with frequent rainfall are also associated with limited sunlight exposure, which by itself is an even greater driver of disease risk than high humidity. Collectively, the combined effects of RH and sunlight exposure 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 (when the fungus establishes on the tiny flower stem, from which it later expands onto the developing fruit) and fruit set. Berries of *V. vinifera* cultivars begin to lose some susceptibility after that, and become relatively resistant about 4 weeks after their individual flowers open, whereas Concord berries highly resistant to immune about 2 weeks after flowering. This is when you use the good stuff and don't even think about cutting corners in terms of spray frequency and application technique. Your annual reminder.

(v) Failure to control even inconspicuous PM infections on the berries can increase the severity of Botrytis at harvest, and can promote the growth of wine-spoilage microorganisms (such as *Brettanomyces*) on the fruit. Another annual reminder. Providing excellent PM control from pre-bloom right through bunch closing does not guarantee control of bunch rots and wine spoilage beasties, but it's a relatively easy way to eliminate one avenue for them to get started.

(vi) Powdery mildew is a unique disease in that the causal fungus lives almost entirely on the surface of infected tissues, sending little "sinkers" (haustoria) just one cell deep to feed. In other words, it's living right out in the open, which makes it subject to control by topical treatment with any number of "alternative" spray materials (oils, bicarbonate and monopotassium phosphate salts, hydrogen peroxide, various plant extracts and microbial fermentation products, etc.), all of which have little to no effect on other disease-causing fungi that do their dirty work down inside the infected tissues. (This same principle applies to the natural PM "fungicide", sunlight, as discussed a bit further on).

Recall that there are two primary limitations to the abovementioned 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, so can only be as effective 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 only modest (JMS Stylet Oil) to zero (potassium salts) residual protective 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

meaningful protective (forward) activity in order to lengthen effective spray intervals. It seems likely that materials such as the potassium salts, which exert all of their activity via a one-time "hit", are relatively unaffected by the application rate once a certain threshold value for this activity has been reached (if you hit a bug hard enough to squash it, you won't squash it any worse by hitting it harder). In contrast, for surface-active materials such as JMS Stylet Oil that also provide some residual protective activity, the interval between sprays can be extended to a modest extent by increasing the rate (unless it rains 1/4 of an inch or so, in which case they largely wash off).

One last time: The inhibition of PM development by sunlight

"Everybody knows" that PM is most severe in shaded regions of the vineyard (canopy centers, near wooded edges, etc.). Here's a final recap of the work of former graduate student, Craig Austin (now gainfully employed and paying taxes to help support us imminent retirees, bless him), who showed definitively just how profound this influence can be, and why.

One of 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 clump 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 same block. These panels were shaded by the trees during the morning and it wasn't until the sun crested over the treetops about noon each day that the vines received their first direct exposure to sunlight. So, we initiated a trial in which Craig inoculated leaves of individual vines that were located either (i) immediately next to or (ii) about 200 feet away from the trees, on either (a) the sun-exposed outer edge of the canopy, or (b) the inner portion of their self-shaded dense canopy, thereby providing treatments with four different levels of natural shading.

As shown below in Fig. 2, 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 canopy of vines next to the trees) compared to the no-shade leaves on the exterior of vines away from the trees.

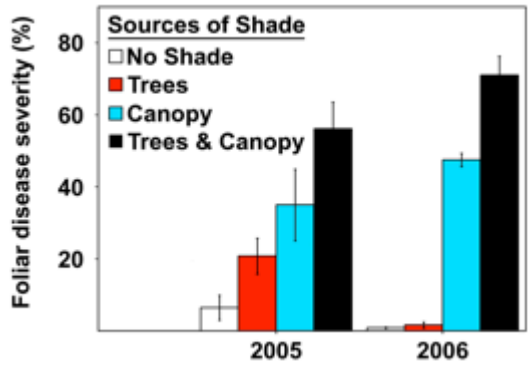


Figure 2. Percent area diseased on Chardonnay leaves receiving (i) full solar radiation, on the outer canopy edge of vines away from trees (No Shade); (ii) shade in the morning provided by an adjacent grouping of trees to the east, but full sun exposure for the rest of the day--i.e., leaves on the outer canopy edge of vines next to the trees (Trees);

(iii) shade provided throughout the day but only by the vine canopy itself, i.e., leaves were located within the canopy center of vines away from the trees (Canopy); or (iv) shade provided by both the trees and the vine itself, i.e., leaves located within the center of the canopy of vines next to the trees (Tree & Canopy).

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 averaged 12°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.

Sunlight characteristics influencing powdery mildew development. As noted above, direct sunlight heats up exposed leaf surfaces, as it does anything else it hits: we all know the difference between standing in the sun on a bright summer day or taking two steps away into the shade. On warm days, this additional heat from absorbed solar radiation can suppress or even kill PM colonies on sun-exposed leaves and berries. 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 much above 95°F, depending on how hot it is and for how long. On a hypothetical late spring or summer day in the low 80's, the temperature of shaded leaves and clusters will remain near that of the air, optimum for PM development. However, with an average increase of 12°F, the temperature of leaves and clusters that are fully exposed to sunlight will be elevated to a point where the PM fungus will stop growing or even start to die so long as these conditions persist.

UV radiation from the sun can damage the cellular structure of virtually all life forms. And as pointed out previously, powdery mildew is a disease that's uniquely vulnerable to such damage, since the PM fungus lives primarily on the outside of infected tissues where it's exposed to UV (versus nearly all other pathogens, which grow within infected organs and consequently are protected from it). On top of that, the PM fungus is white--it has no pigment ("suntan") to protect against this radiation. Lab experiments confirmed that UV doses typical for a summer afternoon in the Finger Lakes region (hardly a world beater when it comes to sunshine intensity!) are deleterious to pathogen development. Furthermore, the same tests showed that this inhibitory effect is even greater at a leaf temperature of 86°F (above optimum for the disease, closing in on the maximum value of 90) than at 77°F (optimum). That is, there's an interaction between the deleterious effects of higher temperatures and UV radiation such that the whole package is more harmful to the PM fungus than the sum of its two parts. And as just noted previously, sun-exposed leaves are hotter than those in the shade, so sunlight actually exerts a triple whammy against this disease through its effects on heating the exposed tissues into unfavorable territory and delivering harmful UV radiation, plus the interactive effect of these two components.

Surface temperature and UV: Field experiments. In order to separate the individual effects of the heat and UV provided by sunlight, Craig 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 collaboration with this part of the study). Plexiglas blocks UV radiation but permits passage of the sunlight wavelengths that elevate leaf

temperature. At the Chancellor vineyard in Geneva, we also suspended shade cloth over other vines to block 80% of the available sunlight, thereby shielding them not only from most UV radiation but also from most rays of the sun that cause heating. Clusters were inoculated with PM spores at 75% capfall. As shown in Figure 3, we found that removing UV radiation while still allowing exposed tissues to heat up (Plexiglas filter) increased disease severity on fruit by anywhere from 50% to fivefold across both varieties and locations. The Chancellor shade cloth treatment, which further eliminated the sunlight-induced increase in temperature in addition to blocking UV radiation, also increased disease severity beyond that simply due to UV filtering in one of the two experiments.

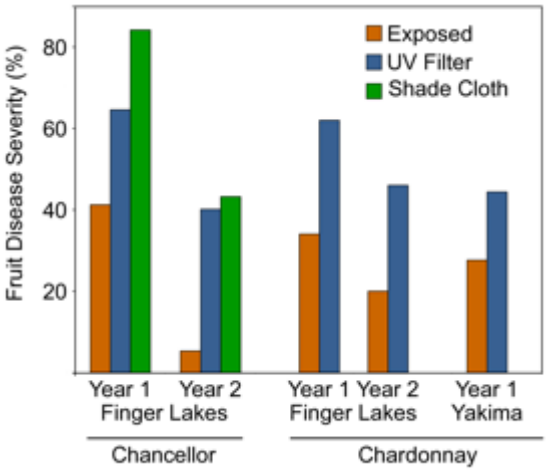


Figure 3. Percent cluster disease severity on cv. 'Chancellor' and cv. 'Chardonnay' vines receiving: (i) full solar radiation (Exposed); (ii) sunlight from which 95% of the UV radiation had been filtered (UV Filter); or (iii) sunlight reduced to 20% of ambient via neutral density shade cloth (Shade Cloth). Vineyards were located in Geneva, NY (Finger Lakes) or Prosser, WA (Yakima).

Manipulating sunlight exposure to manage PM. Given that sunlight exposure reduces PM, how can we use this information to better manage the disease culturally? We examined this question in a young Chardonnay vineyard in Geneva, NY by looking at the effects of both training system and leaf pulling. For training system, we compared Vertical Shoot Positioning (VSP) versus Umbrella-Kniffen (UK), which provided more shoots per linear foot of row than VSP and therefore more potential for canopy shading in the fruit zone. Within each training system, Craig removed either one or two leaves above and below each cluster at one of two timings: 2 weeks post-bloom (fruit set) or 5 weeks post-bloom. He inoculated clusters with PM spores at bloom and rated disease severity in each treatment late in the summer.

We found that both factors affected disease severity (Figure 4). First, PM was less severe 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 regardless of intensity, but leaf removal 5 weeks after bloom had no effect. 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 to combat the disease. Quite simply, it's when you either do or don't get control of PM on the berries.

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 more than one-third relative to UK-trained vines with no leaf removal. Of course this doesn't mean that canopy management techniques will allow you to stop spraying, but it's the essence of IPM: use all of the tools at your disposal to manage diseases, as good growers (by definition) typically do. This takes significant pressure off the fungicide component of the control program, which reduces the pressure for resistance development, improves control levels over the long haul, and gets the birds tweeting (well, maybe not that last bit). It should be noted that in a second year of this trial, 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 but did see the same benefit from early leaf pulling.

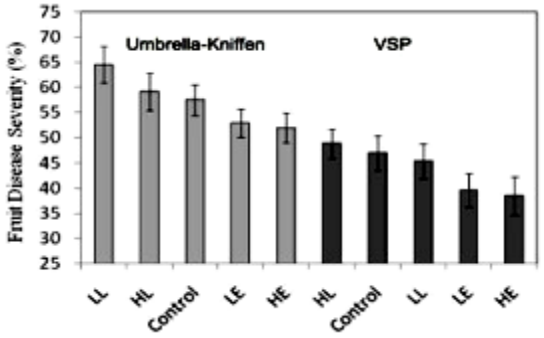


Figure 4. Powdery mildew severity on Chardonnay clusters subjected to five different leaf-removal treatments in each of two vine-training systems. Leaf-removal code: **First letter** is leaf removal severity, H = heavy, L = light (either two leaves or one leaf above and below each cluster, respectively); **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.

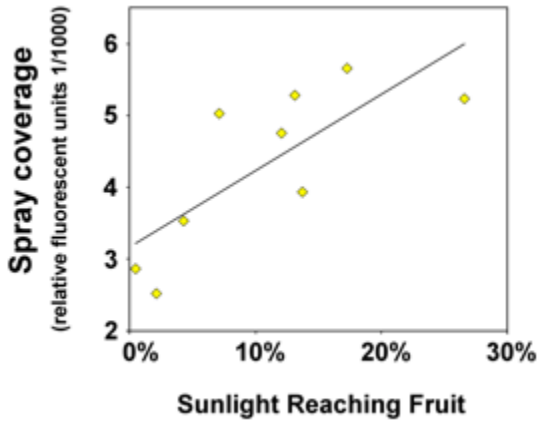


Figure 5. Effect of canopy density on deposition of sprays onto clusters of 'Chardonnay' vines, provided by a conventional airblast sprayer in mid-July.

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 crawly 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 the same 'Chardonnay' planting subjected to the above canopy manipulations were sprayed with a conventional 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 its sunlight exposure level (Figure 5), with well-exposed clusters receiving approximately twice the deposition as those with poor exposure.

Subsequently, Andrew Landers, Nicole Landers, and yours truly expanded this part of the study into four commercial Finger Lakes vineyards plus another experimental block in Geneva, which collectively represented a range of *V. vinifera* and hybrid cultivars (Cabernet Franc, Chardonnay, GR-7, Rosette, and Vignoles) and common industry canopy management practices as imposed by the different vineyard managers. Canopy density was determined for replicate test panels in each vineyard on the basis of Cluster Exposure Layer (CEL), the average number of objects (usually leaves) between clusters and the sprayer. The deposition of a dilute food-grade dye solution, applied with the same Berthoud airblast unit delivering 50 gal/A to all vineyards in early July (a critical time for controlling multiple diseases), was then determined in the lab by measuring the dye washed from a sample of clusters from each of the test panels used to determine CEL. Finally, the average deposition in each test panel was graphed as a function of its CEL value, yielding Figure 6 below.

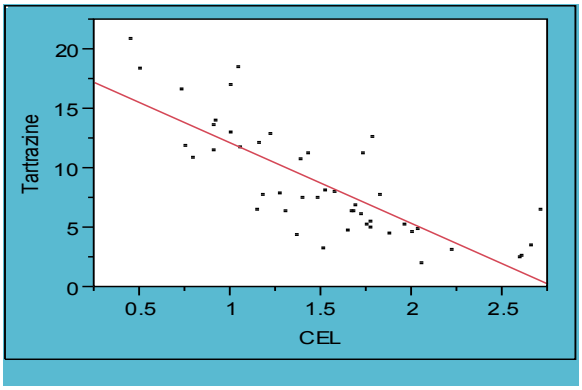


Figure 6. Effect of canopy density (cluster exposure layer = CEL) on deposition of a spray tracer dye (tartrazine) onto grape clusters in five Finger Lakes vineyards. Vines were treated in early July with a conventional airblast sprayer applying 50 gallons/acre.

Although individual data points show the typical field variability around the "average" line indicated in red, the relationship between spray deposition and canopy density is clear. For example, clusters separated from the sprayer by one layer of leaves (CEL = 1.0) received a bit more than twice as much spray as those separated by two layers (CEL = 2.0); or, put another way, at any point on the graph adding one extra leaf layer reduced spray deposition by a little more than half.

Obviously this has major implications for the management of ALL diseases and arthropod pests against which you spray. Over the years, I've been asked many times whether someone should use, say, 6 or 8 oz per acre of a particular product, but never within the context of whether the clusters needing protection are covered on average by ½ or 1 ½ leaves of canopy, which exerts a greater impact on the dose of fungicide deposited on the berries than does the typical tweaking of the rate that goes into the tank. I'm just sayin'.

Summary. In all vineyards, in all seasons, 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, we all know that a central concept associated with quality viticulture is "balance". Zero sunlight exposure might lead to diseased berries, but maximum exposure might lead to disease-free berries that are sunburned instead. It's all about understanding what's going on and attaining the appropriate balance.

Once again: What makes for a high-pressure ("bad") PM year?

Current Washington State University viticulturist Michelle Moyer examined some different aspects of powdery mildew biology while working as a Cornell graduate student in the lab of Drs. David Gadoury and Bob Seem a few years back. Michelle focused on trying to recognize what makes a "bad" PM year while it's still occurring, so that growers might take action to prevent damage rather than conduct a post-mortem later on. Or, conversely, avoid making more sprays that they need to.

To review a few highlights:

• Severe fruit infection was much more likely when Michelle inoculated young leaves to establish PM on the foliage pre-bloom, providing abundant new spores to infect the adjacent new berries during their period of extreme susceptibility. This is logical and consistent with the results discussed earlier concerning how the inoculum level carried over from one season affects the level of fruit disease that develops the next. (Multiple early infections from abundant overwintering inoculum = oodles of new spores produced just in time to infect the newly-formed berries).

• Relatedly, after analyzing 30 years worth of climate and disease severity data, Michelle showed a significant association between severe disease one season and accumulated heat units (degree days) the previous autumn. This also goes back to the earlier discussion concerning the formation and maturation of the PM fungus's overwintering fruiting bodies (chasmothecia) during the late summer and autumn and how that affects disease pressure the following year. Specifically, a long and warm autumn allows late-season infections (the kind that sneak in when PM sprays are relaxed after late summer) an opportunity to form mature chasmothecia with viable overwintering spores. In contrast, a short and cool fall period results in leaves senescing and dying before chasmothecia mature.

• 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, it turns out. Michelle found a very strong relationship between PM severity on clusters at the end of any given season and "pan evaporation" measurements during the critical pre-bloom through fruit set period earlier. 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 (don't you love 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.

Of the two environmental measures identified (pan evaporation and heat units the previous fall), pan evap was more important. Anyone wanting to delve into the details can get them in the Plant Disease journal article, available online (*M. Moyer, et. al. 2016. Weather during critical epidemiological periods and subsequent severity of powdery mildew on grape berries. Plant Dis. 100:116-124*). For everyone else, the basic take-home message is that over a 30-year time span, there was a consistent, strong relationship between PM severity and weather during the period between immediate pre-bloom and fruit set or a bit beyond: sunny and dry = good for you, cloudy and damp = good for mildew. As we would suspect from everything that we now know about this disease.

• Another interesting finding from Michelle's work: cold nights (below 40°F) throw PM for a serious loop. After as little as 2 hr at 36°F, portions of existing colonies are killed; new infections take longer to form colonies and the next generation of spores that spread the disease; and the colonies that do form are reduced in size (hence, their new spores not only arrive later but are fewer in number). Significance: cold nights during the weeks after bud break have the potential to restrict the ability of the PM fungus to establish itself on new shoot growth and produce spores capable of infecting highly susceptible young berries during their critical period of susceptibility.

High disease levels resulting from abundant spore production during this critical period has been discussed within various contexts above, so it's obvious that anything that limits spore production then is good. Or seen another way, a lack of cold nights during the first month-plus of shoot growth can give the disease a running start relative to a "normal" year, when we typically get a few of them during this period. Note that prolonged cloudy conditions that otherwise favor PM by increasing humidity and limiting exposure to direct sunlight during the day ALSO promote this disease by providing a thermal "blanket" above the land at night, limiting radiant cooling and keeping us from getting those really chilly spring evenings we'd otherwise have. Something to keep in mind should such conditions come to pass.

The annual reminder to Concord growers: Remember that the value and necessary level of mid-summer PM control on Concords in any given block or year is strongly dependent upon a combination of crop load in that block and favorability of the weather for ripening (heat + sunlight). That's because meaningful levels of foliar PM can impose a significant limitation on the vine's ability to photosynthesize and ripen the crop, particularly under otherwise-challenging conditions.

Research has shown that a Concord vine can tolerate a fair bit of foliar PM without significant negative consequences if it is not being pushed hard to ripen the crop: low to moderate yield, adequate water and sunshine, few other stresses. However, this same research also has shown that at high cropping levels, good PM control can be necessary to get the fruit to a commercial degree of ripeness. And in cloudy, rainy years—which present the old double whammy because they're both 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 the specific crop load, disease pressure,

growing conditions, vine vigor, fruit prices, etc. But keep the general concept in mind.

A minimal two-spray Concord PM program of immediate pre-bloom and 10-14 days later will keep the berries clean and may be good enough in vineyards with a “moderate” crop in a "typical" year, but it certainly is minimal. In contrast, blocks carrying the robust crops that are now necessary to make a go of this business may benefit from starting a couple of weeks before bloom (as influenced strongly by the weather factors discussed above) and continuing into the mid-summer in order to keep the canopy clean and firing on all cylinders. These “extra” sprays before and after the two critical ones for control on clusters don’t necessarily need to be “Cadillac” materials, just something that gives a reasonable bang for the buck.

PM Fungicides

Sulfur. An abbreviated summary of the major findings and conclusions from our studies on sulfur activities some years back:

• We were unable to demonstrate any negative effects of low temperatures on the degree of control provided by either protective or post-infection sulfur sprays. In a number of repeated tests, control was the same at 59°F as it was at 82°F when we inoculated leaves with PM spores at various times before or after spraying with the equivalent of 5 lb/A of Microthiol. Some Australian research conducted about the same time as ours also showed no difference in control at 59°, 68°, or 86°F when an equivalent rate was used, although there was marginally less control at 59°F with a low rate equivalent to 1.7 lb/A. It appears that the former “conventional wisdom” concerning the detrimental effect of low temperatures on sulfur efficacy was not all that wise (we can find no hard data to back up these assertions), particularly if you consider that the PM fungus itself is not that active at cooler temperatures. It is likely that higher temperatures “boost” sulfur activity by vaporizing some deposits and moving them to unsprayed tissues in this vapor phase. Nevertheless, sulfur activity should be adequate early in the season if spray coverage is reasonable. Note also that it’s typically easier to get first-rate control with sulfur on leaves than it is on clusters, and we’re only dealing with leaves early in the season.

• Sulfur provides excellent post-infection control when applied up through the time that young colonies start to become obvious. Although it does have some eradicative activity against raging infections (see below), it’s significantly stronger against very young colonies. Practically speaking, this means that when a PM spore lands on a new, unprotected leaf that developed after the most recent spray was applied and then begins the infection process, there's still time to control it with the next spray if that's put on thoroughly soon enough after infection begins. “Soon enough” meaning until about 1 week after infection is initiated if temps remain mostly in the upper 60’s and above, a few days longer if there are significant cooler periods mixed in.

• Post-infection sprays applied to heavily-diseased tissues are much less effective than those applied to incubating or very young colonies. Sulfur is not the material of choice as an eradicant if you reach the “Omigod!” stage. That would be JMS Stylet Oil or the similar PureSpray Green (or even Oxidate, but at a much higher cost). And remember that once the leaf or berry cells beneath a well-established mildew colony have been sucked dry by the fungus, nothing’s going to bring them back to life even if the mildew is eradicated. An eradicative spray can't raise the

dead, but it can keep things from getting worse. And for the 1,002nd time, the results you get with eradicative sprays will only be as good as the spray coverage you can provide. These materials work by contact, they simply won't have any effect on mildew colonies that they don't touch (such as the backs of mildewed clusters facing the center of the vine, which often remain diseased when eradicative sprays are applied and the coverage doesn't reach them).

• A number of different field and greenhouse trials designed to clarify the effects of rainfall on sulfur activity produced occasionally variable, but generally consistent results. To wit:

- Rainfall of 1 to 2 inches decreases sulfur's subsequent protective activity significantly. No kidding.
- Removal by rainfall is more pronounced with generic "wettable" formulations than with so-called "micronized" formulations (e.g., Microthiol), which have smaller particle sizes and so adhere better to tissue surfaces. (We didn't look at liquid formulations, but I would guess them to perform similarly to Microthiol). The micronized and liquid formulations cost more for a reason.
- The negative effects of rainfall can be compensated for somewhat by adding a "spreader-sticker" adjuvant to the spray solution and/or increasing the application rate (from 5 to 10 lb/A in our field trials and their equivalents in greenhouse experiments). Increasing the rate and adding the adjuvant each increases control, and these effects generally are additive. Table 3 below provides field data, standardized across years to reflect % disease control relative to unsprayed check vines in the same trial (100% is perfect) on cv. Chardonnay or the interspecific hybrid cv. Rosette, when sprays were applied at approximately 14-day intervals throughout the season.

Table 3. Percent control of powdery mildew severity on Rosette (2004-06, '12, '14) and Chardonnay (2007-10) grapes as affected by sulfur rate and adjuvant, when applied at 14-day intervals (Geneva, NY)

	Foliar disease control (%)*									**Cluster** disease control (%)*								
Treatment, rate/A	2004	'05	'06	'07	'08	'09	'10	'12	'14	2004	'05	'06	'07	'08	'09	'10	'12	'14
Microthiol, 5 lb…….	68	67	86	97	76	70	61	59	86	47	76	70	89	90	4	16	61	87
Microthiol, 5 lb + Cohere, 0.03% …..	84	80	89	97	83	73	64	87	92	64	73	79	90	96	4	37	92	95
Microthiol, 10 lb……	87	89	91	99	91	83	77	62	90	76	77	85	94	---	6	41	83	83
Microthiol, 10 lb + Cohere, 0.03%...	---	---	---	---	95	86	86	84	97	---	---	---	---	98	9	65	95	98

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

Reprise: Effects of sulfur use patterns on harvest residues 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 (H_2S) = "rotten egg gas" if residues in the must at the start of fermentation are "excessive". Although other factors can also cause this, such as yeasts stressed out by poor nutrition, S residues crushed fruit invariably get the blame when things get stinky.

The question that growers long have asked is, "How late can I spray sulfur and still be safe?" And until recently, my answer was, "Everybody has an opinion but there's practically no 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 even as low as 1 ppm in some cases, these

conclusions were reached after researchers spiked clean juice with various concentrations of S before fermentation and then saw what happened. But there was no practical way of measuring S residues on fruit subjected to different spray regimes in the field or in the resultant musts produced from crushing them. The work of former graduate student Misha Kwasnewski (now enologist at the University of Missouri), who worked under the direction of wine chemist Gavin Sacks while also enduring my prodding, changed that.

Misha and Gavin have reported on an elegantly simple, cheap, and effective method that they developed to measure S on grape berry surfaces and in musts after pressing, and have made it available to growers and wineries through various media. Here are the take-home messages from trials where we utilized this technique to measure S residues after applying various spray regimes to Chardonnay and Riesling vines over a 3-year field study period:

• S residues in the resulting musts were affected by both the rate and formulation of the sulfur product used. For a given product, rates of 5 or 6 lb/A yielded greater residues than when half those amounts were used under the same timing regimens, which is hardly surprising. We also found that a micronized formulation (Microthiol) yielded greater residues than a wettable powder formulation (Yellow Jacket) when applied at the same rate, which also is not surprising since increased tenacity (hence, longer residual activity) is one reason that growers are willing to pay more for the micronized formulations (the micronized formulations have smaller sulfur particles than the wettable powder forms, so they stick tighter to the leaves and berries). But whereas a longer period of residual activity is desirable in terms of controlling mildew, it might not be when it's time for harvest, so you might want to cut rates and/or use a WP formulation as you get closer to the end of the season if S residue levels are a concern (Fig. 7).

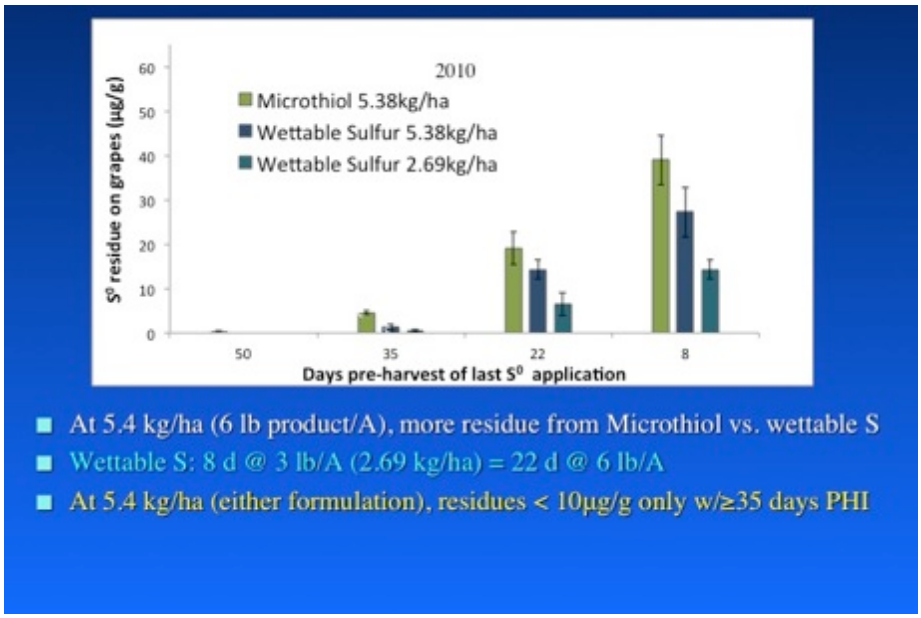


Figure 7. Effect sulfur formulation, rate, and pre-harvest interval on S residues of harvested fruit in one year of a 3-yr trial. Sequential sprays were applied at 2-week intervals, ceasing either 50, 35, 22, or 8 days before harvest in different plots.

• Regardless of rate and formulation, a cutoff of 5 weeks before harvest always yielded residue levels on fruit below the consistent danger level of 10 ppm. Again, lower rates and the WP formulation sometimes allowed use to within 3 weeks or even closer to harvest while still remaining below this threshold. A cutoff of 8 weeks before harvest was sometimes required in order to remain below the more conservative threshold of 1 ppm, depending on rate, formulation, and year.

• When common white wine vinification practices were followed—musts were clarified by allowing them to settle after crushing and fermentations were not conducted on the skins—musts at the start of fermentation had minimal S residues, far below 1 ppm even when residues exceeded 10 ppm right 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 those of 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, even when residues on harvested fruit are high (note that this is not the case for red or other wines fermented on their skins). See Figure 8 for a graphic representation of this phenomenon. (Anyone interested in all of the gory details from this study can find them in Am. J. Enol. Vitic. 65:453-462, which is available for free online; a Google search of "kwasniewski sulfur residue" brought me right to it).

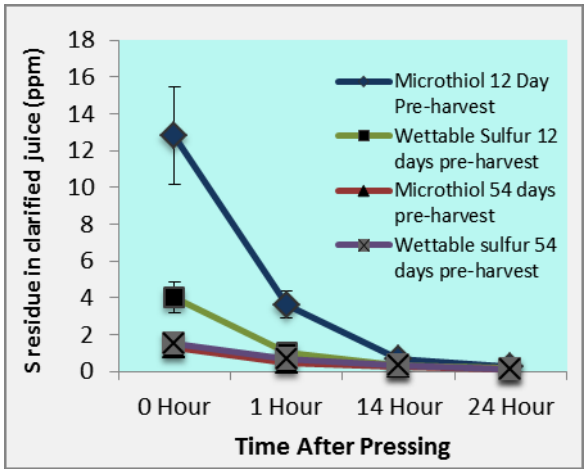


Figure 8. The effect of clarification through settling on elemental sulfur residues present in juice prior to the start of fermentation. Juice was pressed from fruit that received sequential applications at 2-week intervals of two commercial sulfur formulations (5 lb/A formulated product), ceasing either 54 or 12 days before harvest. Samples were obtained from 12 inches below the juice surface at the post-pressing times indicated. Data for 38- and 25-day PHI treatments were intermediate between those for the 54- and 12-day extremes, but are omitted for the sake of simplicity.

"Alternative" materials. There are numerous "alternative", "soft", "organic", etc. products labeled for PM control, many of which can be quite effective if used properly. Manufacturer claims to the contrary notwithstanding, most--if not all--of these "alternative", etc. products probably provide their control via simple contact with the nascent or established colonies of the PM fungus that are trying to grow on the surface of infected leaves and berries. This means that thorough spray coverage is ESSENTIAL for them to work, as discussed previously for oils and Oxidate. Products in this broad category that we've worked with and which have shown efficacy

are included in the NY and PA Pest Management Guidelines for Grapes. These include oils (JMS Stylet Oil, Purespray Green, Trilogy), other plant extracts (Regalia), fermentation products of various microorganisms (Serenade, Sonata, Double Nickel, Ph-D, Oso), and potassium salts (Armicarb, Kaligreen, Milstop, Nutrol).

Some years back, we did extensive work with Nutrol (monopotassium phosphate = dihydrogen potassium phosphate), both in the greenhouse and in the field. In greenhouse tests, we inoculated vines with PM spores at various times after or before spraying them with a 1% solution of the product (8 lb /100 gal water), in order to assess its protective and post-infection activities, respectively. What we found was that it provided absolutely no protective (residual) activity: just as much disease developed when spores were placed on leaves sprayed with Nutrol 1 to 10 day before inoculation as on leaves that were similarly sprayed with water. In contrast, the product provided substantial levels of control when it was sprayed up to 10 days AFTER spores were placed on the leaf.

Why? Well, where I grew up in coastal California we had creatures called banana slugs, succulent slimy slithering blobs a few inches long, nice and plumped up with fluid. And perverse children (I knew a few) were awed by what happened if you poured table salt on them: they shriveled up to almost nothing right before your very eyes as the salt sucked the water right out of the beasts. And that's exactly what happens to a PM colony when you spray an adequate solution of monpotassium phosphate, potassium bicarbonate, or probably any other salt onto it, most effectively when the colony is young and easy to wet. However, it's the salt solution that does the trick, specifically the fact that it's more concentrated than the dilute solution of dissolved nutrients inside the mildew colony, so water flows from the low concentration into the high one until the mildew colony has no water left in it. Thus, if the sprayed salt solution dries up without hitting a mildew colony (or slug!), it has lost its chance to do anything useful for us, and nothing will happen if a mildew spore subsequently lands on the leaf/berry amid dried salt crystals.

In addition to explaining why the salts have little to no effect on diseases other than PM—regardless of what some companies seeking your money might claim or individuals who want to be "green" might wishfully think--this tells us at least two things that have practical implications for disease management, which we've confirmed with field testing. First, if there is no residual protective activity and we are relying entirely on post-infection "knock down" from each spray, we need to spray often enough that the fungus does not have time to infect after one application and produce a new generation of spreading spores before we make the next knock-down application. And recall that the fungus needs only 5 to 7 days to complete this whole process if temperatures remain between the low 60's and mid-80's (°F). Which means that spray intervals should not exceed 7 days for salts and other products with little to no residual activity unless temperatures deviate from this range for significant periods of time. Indeed, we obtained MUCH better control when we applied Nutrol in a 0.5% solution every 7 days than when we applied it in a 1% solution every 14 days.

Second, if the activity from various salt products is due to them sucking the water out of the PM colonies (so-called "osmotic shock"), it shouldn't really matter what salt is used, so long as the concentration is high enough to do the job and it doesn't harm the plant. Note that potassium

bicarbonate products (Armicarb, Kaligreen, Milstop) are sold for this purpose rather than sodium bicarbonate—everyday baking soda—not because the former salt is more effective against PM but because too much sodium causes plant injury. And in multiple field trials, we've seen absolutely no difference in the control provided by any of the different potassium salt products if used at their labeled rates, even though there can sometimes be significant differences in the prices that are charged for them.

DOWNY MILDEW (DM) REVIEW 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. Last year was a very light DM year throughout most of NY, so oospore levels should be lower than average this spring. However, these resting spores are a bit like weed seeds: their viability goes down over time, but at least some of them can persist for many years. Thus, although the oospore "crop" from 2016 should be fairly low, there should be some holdover from the more DM-intense years of 2014 and 2015. Bottom line: early season inoculum pressure won't be as intense as in the last couple of years, but it won't be absent either.

In the spring, these dormant oospores "wake up" and produce the pathogen's infectious "swimming" spores, which cause the season's first ("primary") infections. This process requires a temperature of 52°F or higher and a minimum rainfall of approximately 0.1 inch, which provides the swimming spores an opportunity to be splashed up into the canopy or onto nearby sucker growth and keeps the tissues wet long enough thereafter to allow infection. Of course, even heavier rainfall and warmer temperatures increase the probability that primary infections will occur.

Once primary infections develop, new "secondary" spores (sporangia) form in the white downy growth that's visible on infected young clusters and, particularly, the underside of infected leaves (Fig. 9). 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 very humid nights to form the sporangia (warm and very humid is even better) with rain following soon thereafter to facilitate their dispersal and promote germination and infection. Without rain, most of the ungerminated sporangia will stay in place and die the next day if exposed to bright sunshine (microbial vampires!); however, they can survive for several days between rainfalls if conditions remain cloudy, which helps to keep an epidemic running.

Fig. 9. Typical white "downy" appearance on the underside of a DM-infected leaf, consisting of masses of the pathogen's reproductive structures (sporangia).

Spread is most rapid with night and morning temps of 65-77°F, although new infections 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 protection is lacking and the weather remains favorable for DM—conducive temperatures, repeated humid nights, frequent rains, and extended periods of cloudy weather--for long stretches of time. See: Summers of 2008, 2009, 2011, 2013, 2014, and 2015 in much of upstate NY.

The erratic development of DM coupled with its explosive and potentially devastating nature strongly encourages scouting for it, especially after fruit have become resistant and the consequences of less-than-perfect control are lessened. No need to spray for the disease when it isn't a threat, but you don't want to allow it to start rolling should it become active. Keep an eye on the vineyard to see whether either of these possibilities is something that you might be able to avoid. For additional guidance, my colleagues, Drs. David Gadoury and Bob Seem, have developed a computer model (DMCAST) that integrates a number of weather and crop development factors to provide guidance as to when infections are likely to occur. An interactive version of this model, developed and posted by the NYS IPM program, can be accessed online via the NEWA site at http://newa.cornell.edu/index.php?page=grape-downy-mildew

Cluster/berry susceptible period. Clusters of some varieties—including all *V. vinifera* cultivars--are highly susceptible to infection as soon as they're visible and the DM organism becomes active. In Geneva, our first infections on highly susceptible cultivars exposed to plenty of overwintering inoculum typically are initiated about 3 weeks before the start of bloom (unless there's no rain until later). Research indicates that berries become highly resistant to direct infection within about 2 weeks after the start of bloom, resulting in classic DM spore production from the diseased tissue. However, the DM organism can also infect individual berry stems (pedicels) for a couple of additional weeks thereafter and follow this pipeline into the fruit, causing the aptly-termed "leather berry" symptom--a hard and dry berry with no DM spores produced upon it (Fig. 10). Which all means that unprotected berries can get infected one way or another for about a month after capfall, perhaps a tad longer depending on cultivar and weather.

Fig. 10. "Leather berry" symptom of downy mildew, resulting from infection through the berry stem after fruit become resistant to direct infection; note lack of typical DM spores present. Such fruit often fall to the ground.

For many years, the standard fungicide test protocol on hyper-susceptible Chancellor vines at Geneva has been to start spraying about 2 to 3 weeks pre-bloom and continue through approximately 4 weeks post-bloom. The best materials have consistently provided virtually complete control of fruit and cluster stem infections using this schedule, even in bad years in a vineyard with high inoculum pressure and perhaps the worst possible variety for susceptibility to cluster infections (the interspecific hybrid cv. Chancellor). But remember that vines with susceptible foliage remain vulnerable to defoliation from DM right into the fall if disease-conducive weather persists, long after the fruit have lost their susceptibility (Fig. 11).

Fig. 11. Progression of DM foliar symptoms in late August on cv. Chardonnay, from younger to older leaves.

Fungicides. Ridomil remains the best downy mildew fungicide ever developed: excellent protective and post-infection activity, some apparent eradicative activity, and strong activity in the vapor phase, which helps to move biologically meaningful levels of the product from sprayed tissues to other tissues close by that might have been missed by the spray application (e.g., hidden due to "shingling" by an overhanding leaf). Although cost and lack of activity against other diseases have limited its use in the U.S., this has its upside, because the material still works here. And if you get to the point that you're ready to call in the big guns, this is the Howitzer. Growers in regions where the potential for leaching into ground water is an issue (e.g., Long Island) should also be aware that Ridomil is especially prone to this problem due to its unusually high solubility in water, and be prepared to address the issue. Ridomil is HIGHLY prone to resistance development--indeed, it's no longer effective in many, if not most, parts of the viticultural world where DM is a recurrent problem--and although resistance has never been detected on grapes in the North America, this is probably due largely to its relatively limited use as noted above. Since resistance development is a MAJOR concern, in an ideal world <u>all</u> resistance-management precautions would be followed in order to keep this fungicide a viable part of our arsenal against DM. In the real world, Ridomil is often used to put out the fire if DM threatens to get out of hand, contrary to the recommendation to avoid use of at-risk fungicides as a "rescue" treatment once an epidemic has broken out. Which means that doing so is risky and using it more than once per season under such circumstances is just asking for trouble. If you do happen to use Ridomil as a rescue treatment, using it a second time during the same season is strongly <u>not</u> recommended. Remember that the PHI on Ridomil Gold Copper is 42 days versus 66 days for Ridomil Gold MZ.

Note the discussion at the beginning of this tome regarding some specifics of several newer DM fungicides. Zampro and Revus/Revus Top have been excellent in our trials for a number of years now. So has Presidio (not discussed previously) in a more limited number of tests, although cost seems to have limited its adoption in the grape market. Ranman is quite good, but hasn't held up quite as strongly as the preceding products under intense pressure and extended (14-day) spray intervals. And as noted in the very beginning section of Fungicide Changes, News, and Reviews, very limited initial trials with LifeGard have been encouraging, but there are good reasons to be cautious about jumping in feet first with this otherwise-unproven product until we (and others) have more results to confirm the encouraging ones we've seen so far.

Figs. 12 and 13 below provide data from spray trials that we conducted on cv. Chardonnay in 2014 (horrendous DM pressure) and 2015 (really bad DM pressure), which illustrate some of the statements above. As noted on the figures, sprays began about 2 weeks prebloom and were applied at 2-week intervals thereafter until late August (7-day intervals for LifeGard in 2104). Also note that we mistakenly applied an extremely excessive rate of LifeGard in 2014 (oops!), so those results must be taken with a cube of salt.

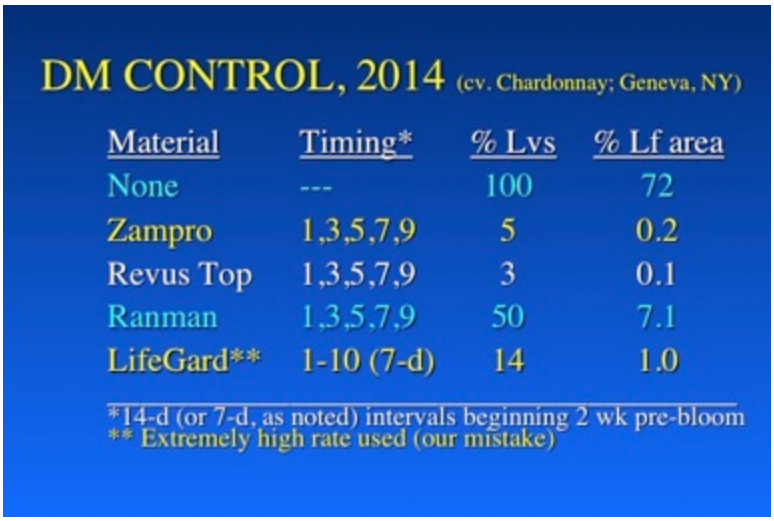


Fig. 12. Control of DM incidence (% leaves diseased) and severity (% leaf area diseased) on Chardonnay vines treated with different products in 2014.

Fig. 13. Control of DM incidence (% leaves diseased) and severity (% leaf area diseased) on Chardonnay vines treated with different products or rotational programs in 2015.

Copper, mancozeb, and captan are old standards because they work. These are protective fungicides restricted to the surface of sprayed tissues, and although resistance development is not a danger, wash-off under heavy rains is. Thus, they may need to be reapplied more frequently in wet years—which, of course, is when you need them the most. Ziram is much better than nothing, especially on cultivars like Concord that aren't highly susceptible to DM, but it wouldn't be your first choice if one of these other materials (or one of the newer ones) were an option.

Which brings us, once again, to the phosphorous acid products (also called phosphites and phosphonates). We've discussed these *ad nauseum* for over 10 years now, so will only review the main points briefly. Recall that they are excellent materials for anyone who wants a product that works but also is consciously seeking a "least toxic" or "sustainable" approach to growing

grapes: 4 hr REI, exempt from US-EPA residue tolerances, and minimal environmental impact. Although there are occasional reports and testimonials alluding to the ability of these materials to control other grape diseases (allegedly by inducing natural defense responses in the plant), I have not found this to be so in several different trials that we've run. In general, the phosphonates are very good and reliable fungicides against downy mildews plus a few other closely related diseases that occur on crops other than grapes, but that's because they are toxic to this one narrow group of pathogens; however, the materials are not toxic to the "true" fungi (the terminology is a long story, but basically all of the other disease-causing organisms that grape growers spray for), and control of the diseases that they cause is erratic at best and usually absent altogether. If you do get control of another disease with a phosphonate spray, think of it as an unanticipated bonus. I certainly wouldn't encourage you to even hope for it, much less bet the farm on it (literally), unless you're the type of person who starts shopping for a new car 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 phosphonate but are not labeled for DM control. Which means that you can spray these latter products but it's only legal to obtain disease control with them if you didn't intend for that to happen. Whether this seems fully rational or not, remember that the law requires any material applied for a pesticidal purpose to be labeled for such, and you can still be cited for breaking a law regardless of your opinion if that's what an enforcement officer has cause to think you have done. Also be aware that it's sometimes difficult to discern both the phosphonate concentration and the quality of these non-labeled products.

From 2003-05, 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 significant but limited protective activity (at least 3 days, sometimes up to 8), depending on the rate used, as well as the particular trial (weather, cultivar) 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 "shipped out" of them to the younger leaves and roots.

• Phosphonates provided excellent "kick-back" activity against new infections. When they were applied 3 or 4 days after leaves were inoculated, few lesions developed at either the low or high labeled rate and spore production from these few lesions was greatly to totally inhibited. When applied 6 days after inoculation, the small lesions that were just starting to become visible at that time continued to expand but production of spores from the expanded lesions was greatly inhibited. Control was better when higher label rates were used and when an initial application was repeated 5 days later (waiting for 7 days to make the second application would probably be OK, too). 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 become active, hit 'em again. But tank-mix with a protective fungicide, at least in the repeat application, both to improve efficacy and to help guard against the proliferation of less-sensitive/resistant strains of the DM organism (see below).

• Phosphonates did not eradicate well-established infections when applied to actively sporulating lesions, but they did limit further spore production by approximately 80%. Limiting the production of these spores will obviously limit the potential for disease spread, but it also increases the opportunity for selecting resistant strains of the DM organism.

• **CAUTION**: The phosphonate products have become very popular over the years, for the good reasons cited above. But they're not miracle drugs, and some people like to push them past their limits in terms of both increased spray intervals and reduced rates. Furthermore, there appears to be a subconscious tendency for some people to think that these aren't "real" fungicides, perhaps for reasons having more to do with marketing and avoidance of registration costs rather than science (nutrient formulations!). However, these are real fungicides when it comes to the DM organism, i.e., they're toxic to it. And just as with other real systemic fungicides, the pathogen can develop resistance to these materials if given a chance.

Although sudden and total resistance to the phosphonates has not occurred after more than a dozen years of widespread use, there is evidence that they can lose some of their effectiveness over time, similar to what we've seen with the DMI fungicides versus powdery mildew: progressively higher use rates being needed in order to obtain progressively lower levels of control. Unfortunately, there are real limits to the rates that we can use, not only for legal and economic reasons but also due to the potential for plant injury at rates higher than those already labeled. And because all phosphonate products are made up of the same basic active ingredient, there is no chance that a "new and improved" phosphonate with greater intrinsic activity will come along to save us if we burn them out, which is the only thing that has kept the DMIs alive for so long (they'd be useless against PM by now if nothing stronger than Bayleton—remember that one?—had been developed). If the phosphonate products we currently have quit working, that class of chemistry is gone for good.

So DON'T burn these materials out by relying on them exclusively throughout the summer. DO consciously rotate/alternate them with something else: never apply more than two sequential applications before using a different DM fungicide, and not applying them even twice in a row is better yet if you don't need to (e.g., extended kick-back activity required, as discussed above). Treat them just like you would any other fungicide with a potential for resistance development, to make sure that you can keep using them into the future.

BLACK ROT (BR) REVIEW AND REMINDERS

1. As fruit mature, they become increasingly resistant to infection. 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 through 5-6 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 overly concerned for the last couple of weeks of these susceptible periods, since by then the overwintering spore load is long gone and nearly all leaves and berries (the potential sources of "repeating" spores) are clean in the vast majority of commercial vineyards.

Recall that in unless a vineyard has been hammered by BR in the recent past, mummified berries are by far the major, if not the 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. 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, so additional sprays are not likely to be necessary. In contrast, if new black rot infections are established on leaves and/or young berries, and are consequently producing new infectious spores right next to or within the clusters, protection will need to continue so long as fruit retain any susceptibility. Ditto if someone screwed up and left a passel of mummies from last year hanging in the vines when they were pruned. CAVEAT: Vines located within 50 to 100 yards of wood edges are potentially at risk from a limited number of air-borne spores ("ascospores") of the BR fungus from wild vines within. And unless someone prunes those vines during the winter, they can potentially harbor BR mummies that produce ascospores into the summer (see below).

As often noted, we've regularly obtained excellent control with sprays applied right at the start of bloom plus 2 and 4 weeks later. Such a program protects the fruit throughout their period of peak susceptibility and during most or all of the time remaining before they become highly resistant. As noted above, you can get away with stopping sprays before berries are fully resistant if there are few to no active infections present, but growers routinely get nailed if they quit too early and there are diseased leaves or berries (or last year's mummies) on the vine. At the other end of this time scale, waiting until the immediate pre-bloom period is a lot safer in a vineyard that was clean last year than in one that had more than a touch of disease, due to the relatively high overwintering spore load that this latter scenario will entail. Recognize when minimal programs are likely to work and when they are not. The drought that most parts of NY experienced throughout the period of berry susceptibility last year resulted in very little BR area wide, which should make control this year easier than "normal". But that doesn't mean that you can just forget about it.

2. Mummies retained in the canopy provide significantly more pressure for BR development than those dropped to the ground. Another reminder: mummies in the canopy produce many more spores than those on the ground (as in 10 to 20 times as many over the course of the season) and continue to produce them throughout the period of berry susceptibility, whereas spores from ground mummies are depleted by or 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. This is especially true for the splash-dispersed conidiospores, which are produced in greater quantity than the air-born ascospores. 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.

Fig. 14. Production of air-borne "ascospores" from black rot mummies (previous year's fruit infections) overwintered in the trellis or on the ground. The yellow arrow ("Bl") indicates the approximate time of bloom.

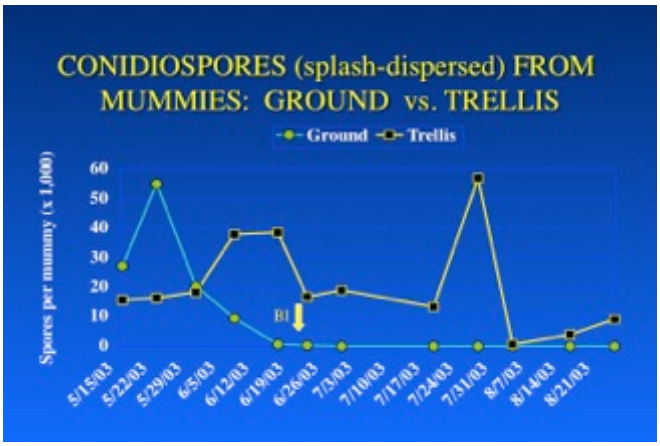


Fig. 15. Production of splash-dispersed "conidiospores" from black rot mummies (previous year's fruit infections) overwintered in the trellis or on the ground. The yellow arrow ("Bl") indicates the approximate time of bloom.

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 by about 2 weeks later and that all diseased berries are apparent within 21 days after the start of the infection event. However, clusters infected near the end of their susceptible period do not even begin to develop symptoms until 3 to 5 weeks after an infection event starts (Fig. 16). (Note that since the fungus is responding to accumulated heat units rather than accumulated risings of the sun, these periods will be a bit shorter in significantly warmer climates). In New York vineyards, black rot that begins to show up in mid- to late August is probably the result of infections that occurred in mid-July, depending on the cultivar. This fact should be considered when trying to determine "what went wrong" should such late-summer disease develop.

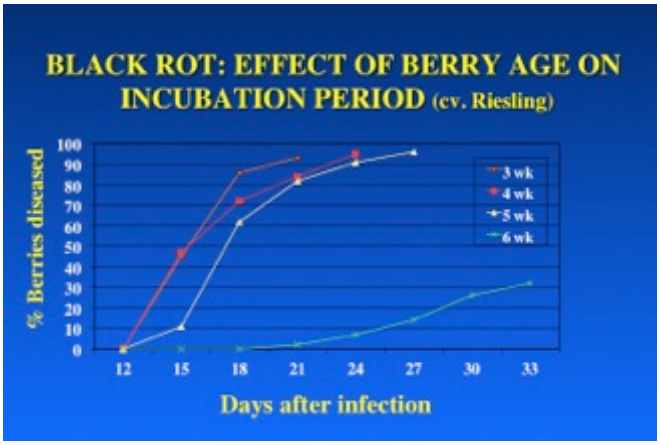


Fig. 16. Effect of 'Riesling' berry age on the incubation period for black rot (similar for 'Chardonnay' berries, data not shown). Berries were inoculated in the vineyard with BR spores either 3, 4, 5, or 6 weeks after cap fall and the percentage showing symptoms was determined every 3 days thereafter.

4. The DMI [SI] 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 DMI (sterol inhibitor) fungicide (Rally) in control of black rot under field conditions

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

[a] For protective treatments, sprays were applied at label rates to Concord vines in the field at the indicated number of days before infection with black rot spores.

[b] For post-infection treatments, sprays were applied at label rates to Concord vines in the field at the indicated number of days after infection with black rot spores.

[c] Percent reduction in the number of diseased berries relative to unsprayed clusters.

5. Fungicides. DMI (Group 3) fungicides: Rally and Elite were always the kings in our evaluation trials, which haven't been run since we lost our BR test vineyards some years back. Elite is no longer marketed as such, although generic tebuconazole products should do the same thing if used at an equivalent rate of 1.8 oz of *active ingredient* per acre (e.g., 4 oz/A of a 45DF formulation). Trials run by Mike Ellis in Ohio, Bryan Hed in PA, and Mizuho Nita in VA have all shown that Mettle and the difenoconazole products also provide similar levels of activity, and

I would expect Rhyme and the DMI component of Topguard EQ to do the same thing. (Vitacure or Procure [triflumizol], an old DMI in a subgroup different from that of the previous materials, appears to be less effective). In many of our trials, the strobies were right at the top as well. Of course, the most important time to control black rot (bloom and early postbloom) is also a critical time for controlling PM and DM, and use of strobie and DMI products during the critical period for these diseases is often complicated by the various resistance issues discussed previously. But they're still great for BR.

Mancozeb and ziram are old standards and provide very good control under most commercial conditions. 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 the strobies are more rainfast. Of course, rainy conditions are when superior performance against BR is most necessary. 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 can be the "Achilles heel" for organic grape production in the East. Unlike PM and DM, we don't have any good OMRI-approved products for BR control. Copper is the best that we have, and it's not known as a BR fungicide: in the only good trial that we've run with copper, it provided 40% disease control under moderately high pressure when applied at 2-week intervals versus essentially 100% control with Rally. A report from a trial that Roger Pearson ran in the mid-1980's shows that he got a similarly modest level of BR control with season-long applications of a copper product under high pressure conditions.

That being said, towards the end of a very wet season a few years ago, 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 berry with black rot. What had he done? *He'd implemented a rigorous sanitation program to get rid of mummies* 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 conformed to the letter of the organic law.

All things considered, sanitation and cultural practices form the <u>absolutely critical</u> first (and second and third....) line(s) of defense against BR for growers who wish to produce grapes organically. So if this includes you, you'll need to pay <u>strict</u>, bordering on obsessive, attention to maintaining limited levels of 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 possible. At the very least, it is <u>imperative</u> that <u>all</u> mummified clusters be removed from the trellis during pruning. And if you're able to patrol the vineyard regularly from 2 to 6 weeks after bloom 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 <u>within</u> 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 buried during cultivation practices or covered with mulch, if appropriate. Inoculum produced from overwintering cane lesions--which are rare unless the vineyard had serious black rot previously—can be minimized with a late dormant application of lime sulfur (expensive and unpleasant, not something you want to do unless necessary but which you may want to do if it is).

BOTRYTIS BUNCH ROT (BBR): THE FULL MONTY

The following is a lightly-edited version of an article that's appeared in a couple of online sources since last fall, discussing the biology and general management programs for BBR. A review of Botrytis fungicides follows it, if you just want to cut to that chase at the end.

Botrytis a "weak" pathogen that primarily attacks highly succulent, dead, or injured tissues, or those that are senescing (slowly breaking down). Feeding sites of grape berry moth larvae, powdery mildew scarring of the grape skin, and pre-harvest splitting caused by overcrowiding within tight clusters and/or excessive rain are common berry injury sites attacked by Botrytis. Withering blossom parts, aborted fruitlets, and ripening berries as they near maturity are important senescing tissues with respect to BBR development (Fig. 17).

Fig. 17. Common entry points of the Botrytis fungus into newly-forming berries: dead/dying blossom parts and cap scars (arrows).

The Botrytis fungus thrives in high humidity and still air, hence the well-known value of cultural practices such as leaf pulling and canopy management to minimize these conditions within the fruit zone. Although the fungus does not grow well in berries until they start to ripen, it can gain entrance into young fruit through senescing blossom parts, old blossom "trash" sticking to berries within the cluster, and scars left by the fallen caps. Such infections remain latent (dormant) and unseen while berries are green. However, some of them can resume activity and rot the berries as they start to ripen (senesce) if the conditions are "right", after which further spread can occur as new infections expand from these sites into additional ripening berries. This begs the question, when are damaging infections most likely to occur? And relatedly, when are sprays directed at this disease most important and valuable?

We (the royal "we", graduate student Stella Zitter did all of the work) began investigating this question some years ago in a block of different Pinot noir clones in cooperation with the late Dr.

Robert Pool and his technician, Steve Lerch. Because it is well known that BBR is more severe in cultivars and clones with compacted fruit clusters, we chose to work with tight-clustered clone 29 (PN29) and the loose-clustered Mariafeld clone, which commonly develops lower levels of this disease than most other clones. We added a third "clone", PN29 vines whose clusters were thinned by hand (individual berries were removed) after fruit set so that their architecture resembled that of Mariafeld. This was to help determine whether Mariafeld's relative resistance in the vineyard is due to some chemical or physiological factor specific to the clone or simply to the fact that its clusters are looser than most other Pinot noir clones.

For two consecutive years, clusters of the three clonal treatments were inoculated with spores of the Botrytis fungus and kept wet overnight to promote infection, at four different growth stages: (i) late bloom; (ii) pea-sized berries; (iii) bunch closure; and (iv) veraison. Selected clusters were taken to the lab 10 days later in order to determine the percentage of berries with invisible latent infections, whereas the remainder were allowed to mature on the vine and were rated at harvest to determine the percentage of the berries that had become rotten by Botrytis. (An interesting side note: latent infections are determined by killing the berries—e.g., by freezing or treatment with certain herbicides—after which the fungus colonizes the dead berry and forms spores, as if it were growing on inert agar in a petri dish. This indicates that the fungus typically is held in a latent state on the vine through some active process provided by the living berries until they begin to senesce = ripen).

The results from these trials are presented in Figures 18 through 21. There was no consistent effect of inoculation timing on the establishment of latent infections, although a greater percentage of berries did become infected from the late bloom inoculation in Year 2. Similarly, there was no effect of the clonal treatment on latent infection establishment in either of the two years (Figs. 18 and 20). In contrast, both the time of inoculation and clonal treatment had a pronounced effect on the percentage of berries that actually became diseased after they matured. That is, the highest levels of disease resulted from inoculations at veraison, consistent with the preference of the Botrytis fungus to colonize senescing tissues. Also, the greatest number of rotten berries always developed in the naturally compacted clusters of PN29, whereas there were significantly fewer in the naturally looser clusters of the Mariafeld clone or in clusters of PN29 that had been thinned to resemble those of Mariafeld (Figs. 19 and 21). Furthermore, it was clear that latent infections often failed to become active and cause berry rot, particularly in the clusters with less compaction. In Year 2 for example, 64 and 76% of the berries developed latent infections when clusters were inoculated at late bloom in the PN29/thinned and Mariafeld treatments, respectively, yet only 2 and 1% of the berries in those same respective treatments actually became diseased by harvest (Fig. 21).

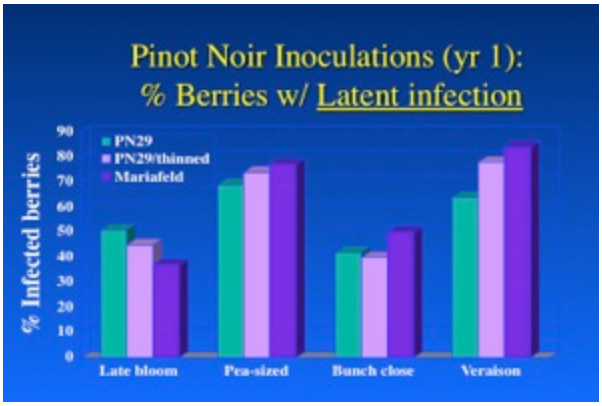


Fig. 18. Effect of clonal treatment and inoculation timing on the frequency of Pinot noir berries with invisible latent infections in Year 1 of the study.

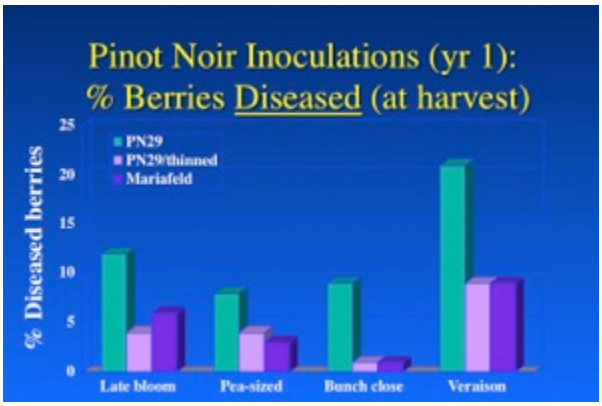


Fig. 19. Effect of clonal treatment and inoculation timing on the frequency of Pinot noir berries with symptoms of Botrytis bunch rot at harvest in Year 1 of the study.

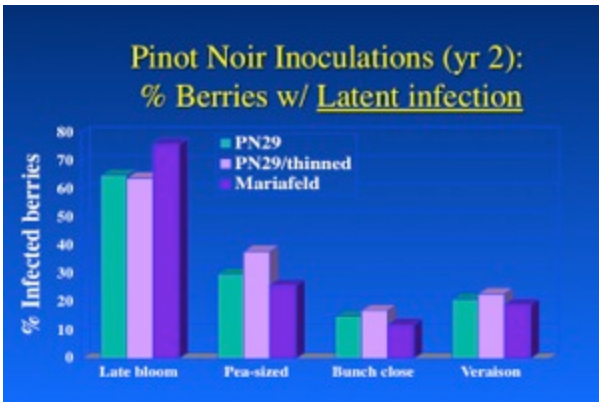


Fig. 20. Effect of clonal treatment and inoculation timing on the frequency of Pinot noir berries with invisible latent infections in Year 2 of the study.

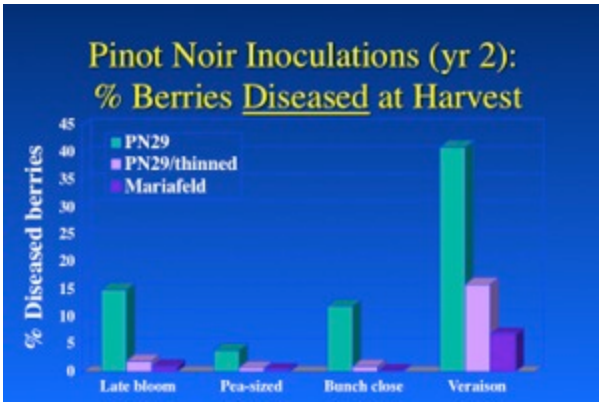


Fig. 21. Effect of clonal treatment and inoculation timing on the frequency of Pinot noir berries with symptoms of Botrytis bunch rot at harvest in Year 2 of the study.

Collectively, these results led us to hypothesize that the higher levels of disease occurring in the tight-clustered PN29 clone resulted from a relative few latent infections becoming active during the post-veraison period and then spreading to a much greater degree than when such clusters were thinned, or in the similarly-loose Mariafeld clusters. To examine this possibility, 10 days after veraison we inoculated either 1, 3, or 5 berries on various PN29 clusters, which were either naturally compacted or had been thinned by hand at fruit set as before. To do so, Stella used a hypodermic needle to inject the designated berries with Botrytis spores, thereby producing individual rotten berries within clusters about 1 week later. These served as initial "point sources" of the disease from which it could spread, and were meant to simulate the occasional post-veraison activation of latent infections.

As Fig. 22 shows, the disease was able to spread extensively throughout the natural, unthinned PN29 clusters: from a single rotten berry that first developed 2.5 weeks after veraison, the disease subsequently spread to an average of 50 additional berries by harvest (it was an excellent year for Botrytis development!). In contrast, disease spread was minimal within the thinned clusters in which a single berry was inoculated and only modestly greater when three or five berries were inoculated.

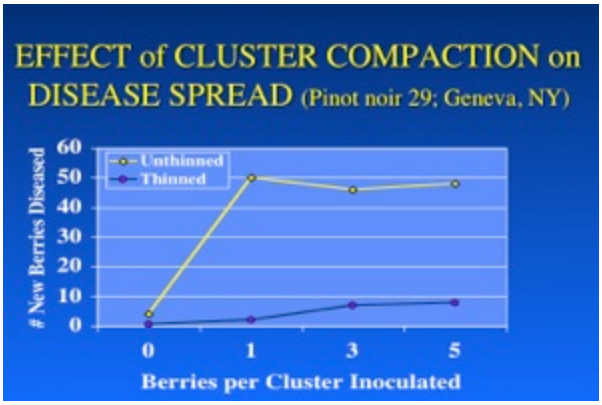


Fig. 22. Effect of cluster tightness on disease spread. Selected clusters on vines of Pinot noir clone 29 were hand-thinned after fruit set to approximate the looseness of those of the Mariafeld clone. Either 0, 1, 3, or 5 berries per

cluster were inoculated at veraison and disease was present on those initial "point sources" 1 wk later. Data reflect the number of additional berries to which the disease had spread by harvest.

In a related experiment the following year, bunches of a tight-clustered Chardonnay clone were similarly thinned (or not) and inoculated. Additionally, based upon a phenomenon we had observed years ago with Botrytis infections of strawberries, some vines received four weekly sprays of urea (8 lb/A) starting at veraison, to see if high berry N content would affect disease spread. (Note that due to its late application, this treatment increased assimilable N in the must without increasing canopy growth.) Once again, little disease spread occurred in the thinned clusters regardless of nitrogen treatment, whereas significant spread did occur in the naturally compacted clusters. Furthermore, elevated berry N also increased spread within these clusters when the system was not "saturated" with the maximum number of inoculated berries. For example, when three berries per cluster were inoculated, the disease spread to three additional berries in the thinned clusters with or without post-veraison N sprays; in contrast, it spread to 31 and 11 additional berries in the compacted clusters on vines that did or did not receive the N applications, respectively (Fig. 23).

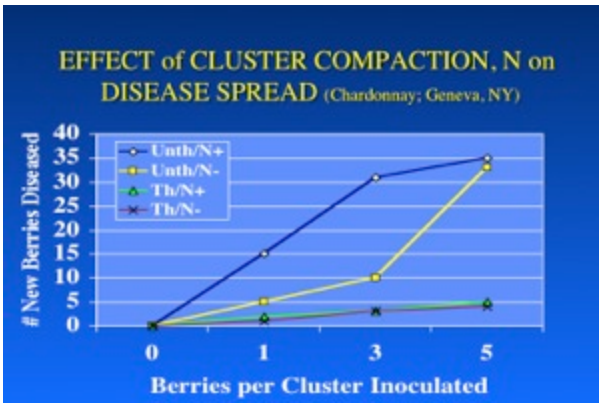


Fig. 23. Preharvest spread of Botrytis on Chardonnay berries as affected by the number of initial infection sites, cluster architecture, and berry nitrogen status. Selected clusters were hand-thinned after fruit set to minimize contact among berries as they matured (Th) whereas others were allowed to retain their natural tightness (Unth). Selected vines received four weekly foliar sprays of urea (8 lb/A) starting at veraison (N+) whereas others did not (N-). Either 0, 1, 3, or 5 berries per cluster were inoculated at veraison, providing individual diseased berries 1 wk later, from which spread could occur. Data reflect the number of additional berries diseased at harvest.

Thus, it appears that latent infections that occur during the bloom and post-bloom period probably result in relatively few rotten berries in and of themselves but instead can serve the role of "primary" infections, providing a foothold for the pathogen from which damaging levels of secondary spread can occur if a few of these latent infections become active and conditions are favorable for further disease development pre-harvest. Such conditions include not only climatic factors but various vine factors, including high berry nitrogen levels and compacted clusters as shown above. Cluster compaction appears to be extremely influential (as any grower with tight-clustered cultivars and clones already knows), since the fungus can spread through tight clusters from just a single initial rotten fruit, via berry-to-berry contact (Fig. 24).

Fig. 24. Spread of Botrytis via berry-to-berry contact within a compacted cluster of Chardonnay grapes.

IMHO, loosening cluster compactness represents the "Holy Grail" for Botrytis management, and although there have been a few sightings reported, I don't think that it's been found yet. Over the years, several workers have experimented with prebloom sprays of gibberellic acid for this purpose, with some success. For example, Bryan Hed and colleagues at Penn State published an in-depth paper on their positive results with GA on Chardonnay and Vignoles a couple of years ago. And there are some GA formulations (e.g., ProGibb 4%, which is even OMRI approved) that are now labeled for use on wine grapes. But note that these labels contain warnings about possible yield reductions during the current and/or following years and a very wide range of rates specific to individual varieties. Using gib or any growth regulator is not a trivial undertaking and you need to go about it carefully. Nevertheless, some growers and investigators have been able to get the benefit of such treatments without noting negative effects. Others have been somewhat less successful. But the less successful instances are also less interesting, so they get less press.

Several researchers worldwide, including Bryan, have had some success with leaf removal around clusters just as bloom is beginning (concept: starve the developing clusters for food and they will set fewer fruit). However, this technique still has its own bugs to work out, e.g., determining variety-specific responses, adjusting bud numbers to compensate for lower yield per cluster, developing techniques to accomplish the feat on a very time-sensitive basis across a commercial-scale operation, etc. Nevertheless, the potential payoffs should an effective technology be (reasonably) perfected are major, particularly in regular Botrytis "problem" blocks. I would caution anyone interested to view those techniques just mentioned as experimental ones with significant promise, and to do their own experiments on a small scale for awhile to get a feel for things while keeping their eyes and ears open with respect to the experiences of others.

Because most latent infections initiated during and after bloom do not become active and rot berries before harvest, it would be helpful to predict when pre-harvest activation might occur, which could potentially start an epidemic. Although the factors that stimulate activation are not well understood, we have identified three that appear to be involved: high berry nitrogen content, high atmospheric relative humidity (RH), and high plant water content.

Since we had already determined that increasing berry nitrogen levels could increase secondary spread of the disease, we decided to see whether it might also promote the activation of latent

infections. Chardonnay vines were inoculated with Botrytis spores at bloom to initiate latent infections, some were sprayed with urea (8 lb/A) five times at weekly intervals beginning 1 week before veraison, and the effect was evaluated at harvest in two different ways. In the first, Stella determined the percentage of the inoculated clusters that had at least one diseased berry, presumably the result of a latent infection initiated at bloom that had become active (while doing so, she separately evaluated the neighboring clusters that were subject only to natural infection). For both the inoculated and uninoculated clusters, the incidence of disease was nearly half-again as great on vines receiving the urea sprays versus those that did not (Fig. 25). Stella also determined the percentage of the cluster area that was diseased on these bunches (essentially, the percentage of diseased berries), which integrates the effect of N on both the activation of latent infections and their subsequent spread through the affected bunches. This measure of disease severity was doubled and tripled for the inoculated and uninoculated clusters, respectively, on vines treated with urea versus those that were not (Fig. 26).

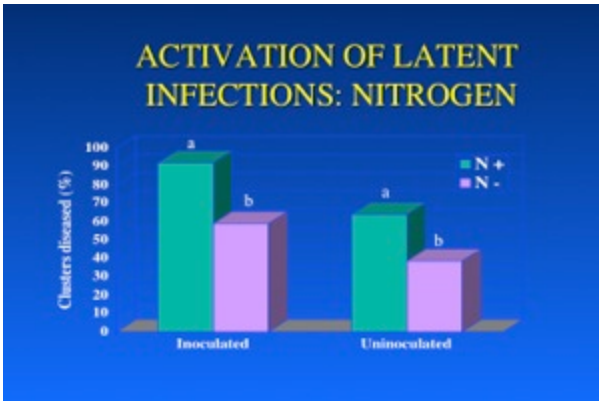


Fig. 25. Effect of nitrogen on latent infection activation. Selected clusters on field-grown Chardonnay vines were inoculated with Botrytis spores at bloom to initiate latent infections, and some vines were sprayed with urea (8 lb/A) five times at weekly intervals beginning 1 week before veraison (N+) whereas others were not (N-). Data show the percentage of clusters with at least one diseased berry at harvest, on both the inoculated clusters and uninoculated neighboring clusters flagged at bloom for later comparison.

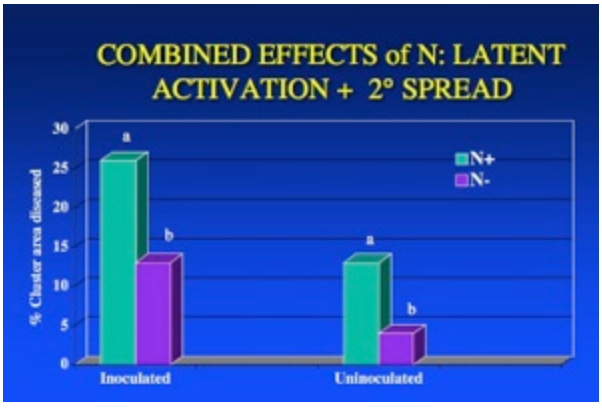


Fig. 26. Combined effects of nitrogen on latent infection activation and secondary spread. Selected clusters on field-grown Chardonnay vines were inoculated with Botrytis spores at bloom to initiate latent infections, and some vines were sprayed with urea (8 lb/A) five times at weekly intervals beginning 1 week before veraison (N+) whereas others were not (N-). Data show the percentage of the cluster area diseased at harvest, on both the inoculated clusters and uninoculated neighboring clusters flagged at bloom for comparison.

To examine the effects of high RH, we utilized potted Chardonnay vines. These were inoculated with Botrytis spores at bloom in order to initiate latent infections and then maintained in a covered screenhouse, where they were subject to ambient environmental conditions while being protected from rain. At either veraison or 10 days pre-harvest, 25 pots were moved to a large humid chamber (95% RH), and five of these were removed either 1, 3, 5, 7, or 9 days later and returned to the screenhouse. At harvest, we then determined the percentage of all clusters that had at least one diseased berry, presumably the result of a latent infection initiated at bloom that had become active. As shown in Fig. 27, imposing high RH for as long as nine consecutive days had no effect on latent infection activation if the treatment began at veraison. However, prolonged humid conditions during the pre-harvest period markedly increased the frequency of clusters with active infections by harvest, from 10% with 0 or 1 day of exposure to 30 and 80% after 3 and 9 days of highly humid conditions.

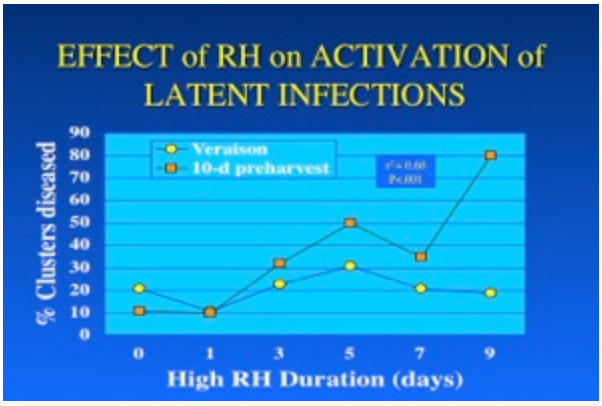


Fig. 27. Effect of relative humidity on the activation of latent infections. Potted Chardonnay vines were inoculated with Botrytis spores at bloom in order to initiate latent infections and maintained in an outdoor screenhouse, protected from rain. At either veraison or 10 days pre-harvest, selected pots were moved to a large humid chamber (95% RH) and returned to the screenhouse either 1, 3, 5, 7, or 9 days later. Data show the percentage of clusters that had at least one diseased berry at harvest.

Although latent infections usually do not become active until after veraison, we occasionally see peas-sized berries with Botrytis symptoms when extensive rainfall occurs during the post-bloom period. Again, based upon what is known about the interaction of the Botrytis fungus with other crop plants such as strawberries, it seemed possible that this might be due in part to berries becoming more susceptible to colonization by the pathogen (latent infections becoming active) when vines are provided unrestricted access to water in the soil. To examine this possibility, we again inoculated potted Chardonnay vines with Botrytis spores at bloom and maintained them in a covered screenhouse. The vines were watered regularly until veraison, then the pots were split into two groups, which were both watered with a hose to keep the clusters dry, but on different schedules: (i) almost daily, in order to keep the soil wet (WET); or (ii) only when the shoot tips began to wilt (DRY). The percentage of clusters with at least one diseased berry (presumably the result of an activated latent infection originally established at bloom) was determined at harvest, after which the harvested clusters were incubated at 95% RH for an additional 4 days to see whether additional latent infections might become active.

As shown in Fig. 28, latent infections had become active by harvest in approximately three times as many clusters in the WET treatment as in the DRY, although the only difference between the two was the amount of water added directly to the soil (the foliage and berries did not get wet in either). And when the harvested clusters were then incubated under high RH conditions, the percentage of diseased clusters more than doubled in the DRY treatment, whereas it was virtually unchanged in the WET (Fig. 28). These results suggest that in the DRY treatment, a significant number of viable latent infections had failed to become active by harvest but then did so once conditions became more favorable during the subsequent high-RH incubation. In contrast, the pre-harvest conditions were much more favorable for latent infection activation when vines were constantly provided high amounts of water, so subsequent incubation under high RH conditions had little additional effect.

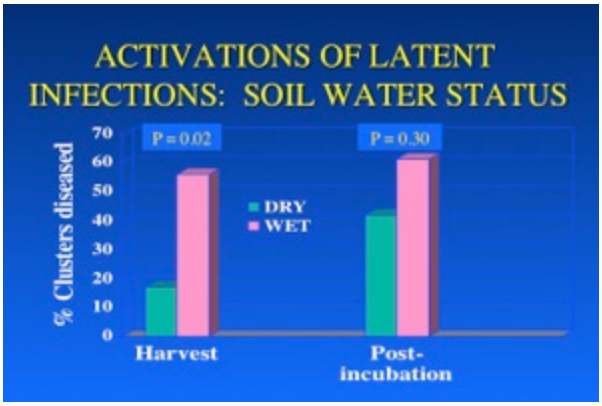


Fig. 28. Effect of soil water content on the activation of latent infections. Potted Chardonnay vines were inoculated with Botrytis spores at bloom in order to initiate latent infections, then were maintained in a covered outdoor screenhouse and watered regularly until veraison. Subsequently, vines were watered: (i) daily, to keep the soil wet (WET); or (ii) only when the shoot tips began to wilt (DRY). The percentage of clusters with at least one diseased berry was determined at harvest and again after the harvested clusters were incubated at 95% RH for an additional 4 days.

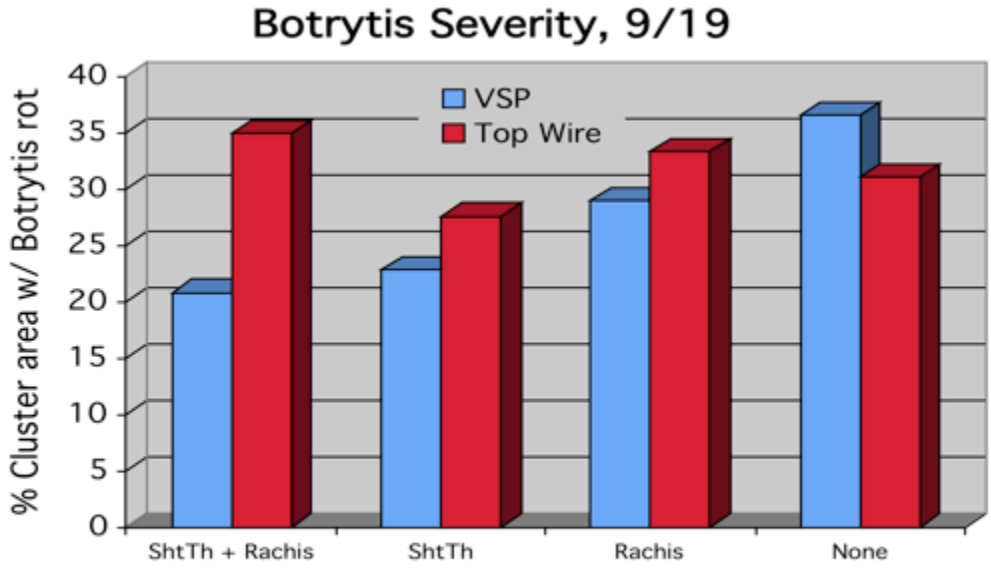
Management, cultural. Cultural practices to improve airflow around the clusters, such as canopy management and leaf pulling, are well known and widely practiced. Removal or destruction of vineyard debris, particularly old cluster stems which serve as a major source of overwintering inoculum, is useful as well and worth employing to whatever extent is practical. Minimizing cluster compaction through cultivar and clone selection at planting, and perhaps by utilizing some experimental techniques such as gibberellic acid application and trace bloom leaf removal, can have a major positive impact. Excessive levels of nitrogen application (and pre-harvest irrigation, where that is practiced) should be avoided if BBR is a consideration.

Illustrative of the effects of cultural practices on Botrytis development are results from a 2011 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 designed a couple of years previously to examine the effect of canopy management practices on fruit quality, it became obvious that these treatments also significantly affected fruit rot (talk about effects on quality!), so we decided to give it a hard look in 2011 (what a year to do so!).

The treatments involved were:

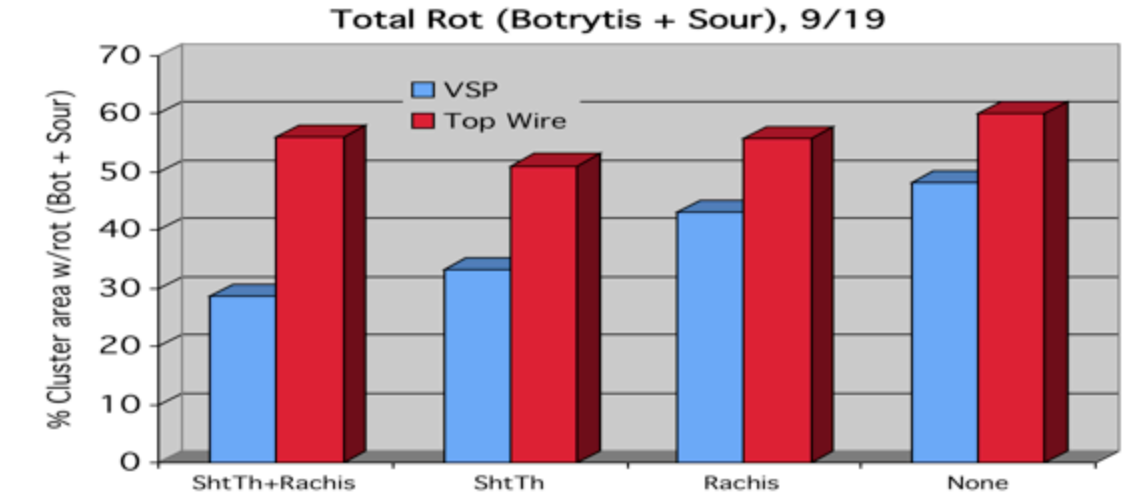
• Training system (Top Wire Cordon and VSP)
• Shoot thinning (thinned to 5 shoots per linear foot of row, versus unthinned = approximately 7 per foot of row)
• Removal of old rachises (important source of overwintering Botrytis inoculum) at the time of thinning, versus no removal

The grower managed the vineyard via his standard practices, which included a Botrytis spray regimen. We rated the plots for both Botrytis and sour rot levels at harvest on September 19; the VSP treatment also was rated 10 days pre-harvest. A few sets of data and interpretations/notations are provided in Figs. 29-31 below.



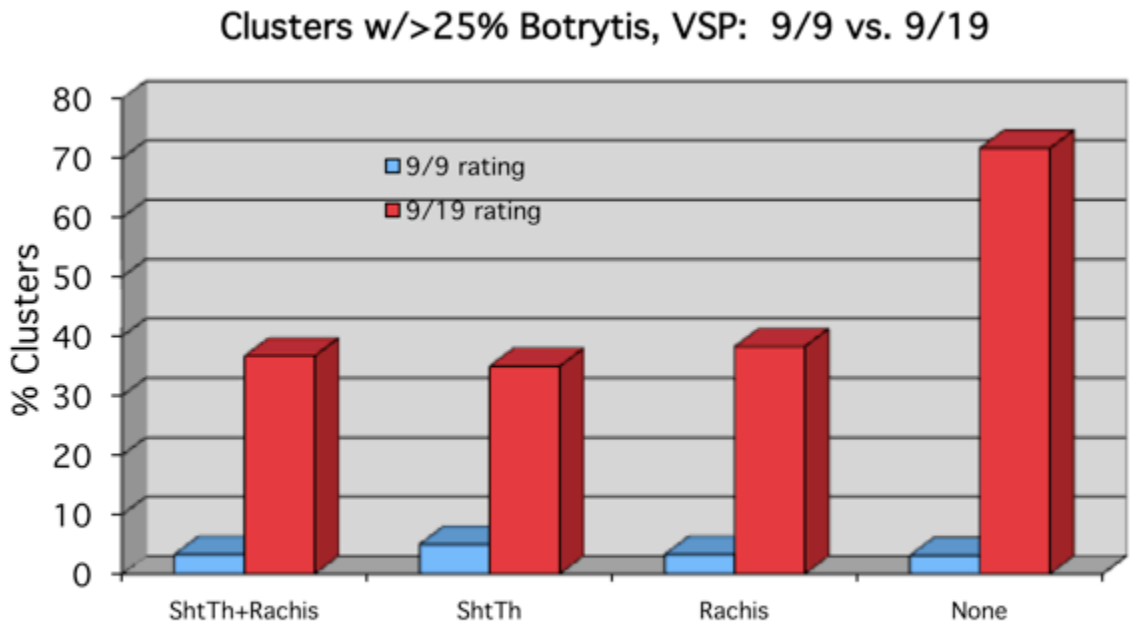
• Positive effect of canopy manipulation treatments in VSP, not in TW
• In VSP, Shoot Thin + Rachis Removal was best, 43% reduction versus check treatment

Fig. 29. Average severity of Botrytis at harvest on Vignoles clusters from vines trained to either a VSP or top-wire cordon system and subjected to three different early-season canopy management treatments (or none).



- 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

Fig. 30. Total rot severity (Botrytis + sour rot) at harvest on Vignoles clusters from vines trained to either a VSP or top-wire cordon system and subjected to three different early-season canopy management treatments (or none).



- 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

Fig. 31. Increase in the percentage of clusters with major Botrytis damage (>25% berries diseased) during the last 10 days before harvest on Vignoles clusters from vines trained to either a VSP or top-wire cordon system and subjected to three different early-season canopy management treatments (or none).

Management with fungicides, when. Fungicide sprays targeted specifically at BBR also can be beneficial on susceptible cultivars and/or clones, particularly in a wet year. However, it's important to remember that unlike some of our other common fungal diseases, it is very difficult to control Botrytis primarily through a good spray program. Integrating cultural control practices with fungicide applications is a necessity when management of this disease is required.

The fundamental questions regarding fungicides are which materials and when? Traditional BBR spray programs call for possible applications at bloom (or late bloom); just as bunches are closing; veraison; and pre-harvest. The earlier timings are designed to prevent the initial establishment of infections through susceptible blossom parts and blossom trash, whereas the later sprays are designed to prevent not only initial infections through injured ripening berries but probably more important, the berry-to-berry spread of active infections throughout the ripening clusters. Despite some pronouncements to the contrary, none of these timings are necessarily better than the others since either, both, or neither ends of the seasonal spectrum can be important, depending on the infection pressure at that particular time.

This concept is nicely illustrated by data that we have gathered over 12 different seasons since 1996. Figure 32 shows the control provided by two Botrytis sprays applied early (late bloom plus bunch closure), late (veraison plus 2 weeks pre-harvest), or at all four of those stages, expressed as a percent reduction in disease severity relative to vines in the same trial that received no Botrytis sprays. Note that in some years (e.g., 1998, 1999, 2007, 2015), either two early sprays or two late sprays provided as much or nearly as much control as all four. In 2002, the two early sprays alone provided most of the control provided by the full program whereas the two late sprays alone provided very little. In contrast, the two late sprays were as effective as the full program in 2011, whereas the two early sprays provided only half as much control. And in the remaining years, the full program was superior to one confined to either the first or last two applications, with the relative contributions of the early and late timings varying among years.

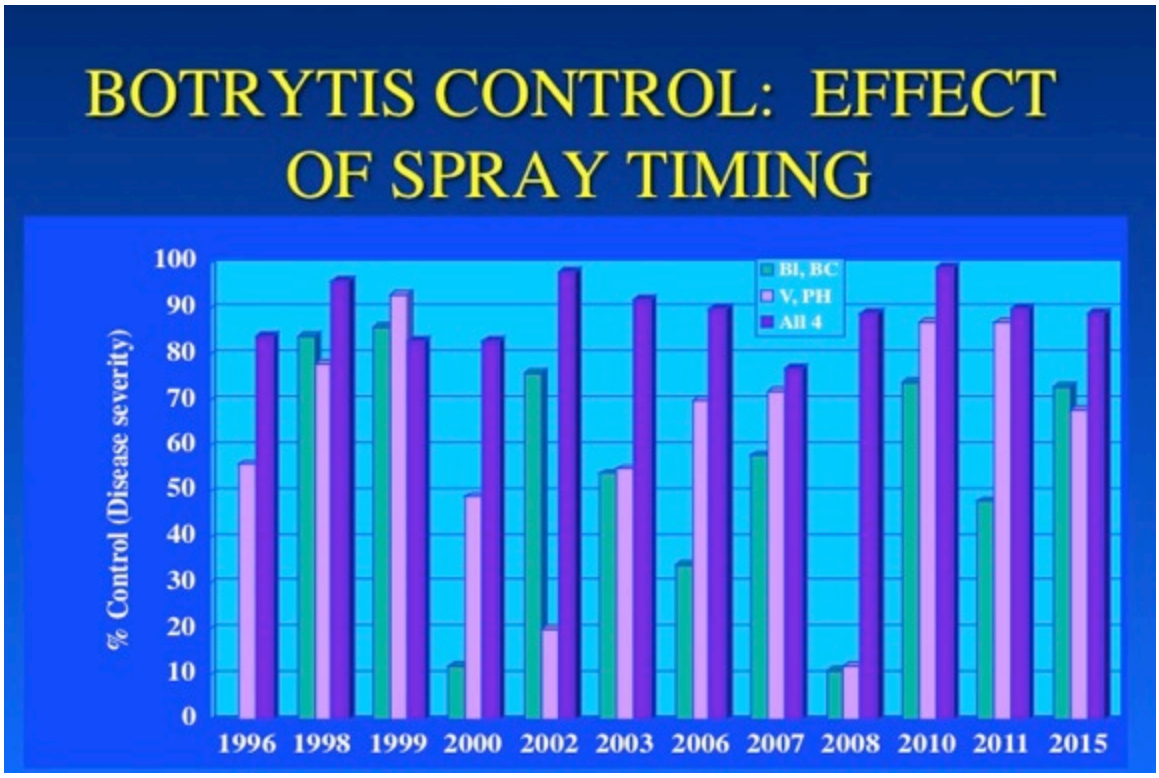


Fig. 32. Influence of spray timing on the control of Botrytis bunch rot in the Finger Lakes region of NY (cv. Aurore, 1996-2000; cv. Vignoles, 2002-2015). Sprays were applied at (i) Bloom + bunch closure (Bl, BC); (ii) Veraison + 2 to 3 wk later, i.e., pre-harvest (Ve, PH); or (iii) at all four of these stages. Data are expressed as percent reduction of diseased berries relative to vines in the same trial that received no Botrytis fungicides.

Fungicides, physical modes of action. Over a several-year period a decade or so back, we looked at the various "physical modes of action" of the Botrytis fungicides available then, 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 from this project:

• In one set of tests, we examined the ability of the fungicides to protect the **internal** tissue of sprayed berries against infection from spores that might be deposited inside them following mechanical damage (rain cracking, berry moth larva feeding, etc.). Chardonnay clusters were sprayed the pea-sized berry stage, bunch closure and veraison, then a hypodermic needle was used to inject berries with Botrytis spores 2 weeks after the last spray. Scala, Vangard, 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 (the non-strobie component of Pristine) provided the least protection of the internal berry tissues. However, all fungicides completely prevented spread to the neighboring berries when inoculated fruit 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 unwounded berries 2 weeks later. 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 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 were sprayed by hand, providing complete spray coverage to an extent not likely to be obtained in commercial production), a single spray of Scala or Vangard applied at veraison provided almost complete control of latent infections that were established at bloom, 60 days earlier. Elevate and Rovral were almost as good. When another group of clusters inoculated at bloom was sprayed at veraison plus 15 days later, Scala, Vangard, and Elevate provided complete control; Rovral reduced infection by about three-fourths; whereas Flint, Pristine, and Endura provided 55-60% control.

• Take home-messages and cautions:

  • All of the "standard" fungicides registered for Botrytis control provided excellent protective activity on the surface of the berries. That's why they got developed and sold in the first place.

  • The so-called AP or Group 9 fungicides (Vangard, Scala) and Elevate also provided very good protective activity within the berries. This was anticipated for the AP's since such fungicides are known to be absorbed by plant tissues, but Elevate was long promoted strictly as a surface protectant. However, this turns out to be a function of marketing strategy rather than fact. Also note that we did not test Switch, since it was not registered for use on grapes at the time this work was done. Although Switch contains the active ingredient in Vangard (cyprodinil), it provides only 70% as much of it as Vangard at their respective label rates. I don't know how this lower rate compares with the higher, nor what within-berry control that the second Switch component might provide.

  • Similarly, the same three materials provided very good curative activity (or suppression) against latent infections initiated at bloom, even when applied 2 months after infection. Nevertheless, as shown previously in Figure 32, 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 activity provided by the two later sprays under field conditions doesn't replace the need for earlier applications when conditions favor infection at bloom, although it probably contributes to the overall level of control obtained.

SOUR ROT: THE BASICS AND CURRENT RESEARCH

SOUR ROT is often used as an imprecise catch-all term to describe the "snork" that can take over injured clusters near harvest if the weather becomes wet. Unfortunately, this means that different people (and fungicide labels) sometimes use this same name to refer to a general condition that has different causes. For the rest of this discussion, I'll be referring to what I call

"true" sour rot--a syndrome that involves pre-harvest cluster decay accompanied by the smell of vinegar (after all, there's a reason they call it sour rot). This disease was pretty much a "black box" until my colleague, Wendy McFadden-Smith, working with the provincial government of Ontario (OMAFRA) in Canada started delving into it in some detail a few years back. Inspired by and building upon Wendy's pioneering work, we began our own studies in 2013, spearheaded by almost-finished graduate student Megan Hall and with the invaluable cooperation of entomologist Greg Loeb. The following is based almost entirely upon the contributions of the aforementioned individuals.

The characteristic visual symptom of sour rot is a tan to occasionally reddish discoloration of the rotting berries, which eventually lose their integrity and begin to decompose; no moldy growth needs to be present (Figs. 33 and 34). Whereas various molds, including Botrytis, are sometimes found on sour-rotted clusters (Fig. 35), these organisms are not necessary for sour rot to develop. Indeed, although some potential role for them cannot be dismissed entirely in specific cases, such molds usually occur coincidentally with the yeasts and bacteria that cause sour rot, since all of these organisms utilize the same food source and are favored by the same environmental conditions, yet yeasts and bacteria are not visible to the naked eye. One additional group of organisms characteristically associated with sour-rotted clusters, which are highly visible and appear to be an important if not essential component of the disease, are *Drosophila* "fruit flies" or "vinegar flies" (Fig. 36), as discussed later.

Fig. 33. Pre-harvest sour rot on cv. Riesling. Note almost complete lack of mold growth on the diseased berries (photo courtesy of Megan Hall).

Fig. 34. Pre-harvest sour rot on cv. Riesling. Note complete lack of mold growth and the breakdown of diseased berries (photo courtesy of Megan Hall).

Fig. 35. Pre-harvest sour rot on cv. Riesling. Note coincidental presence of additional black, secondary mold fungi (photo courtesy of Megan Hall).

Fig. 36. Pre-harvest sour rot on cv. Riesling. Note lack of mold growth and presence of numerous *Drosophila* fruit flies (arrows) (photo courtesy of Megan Hall).

Wendy McFadden-Smith has shown that the measure of volatile acidity in crushed grapes harvested from different vineyards is strongly correlated with the pre-harvest severity of sour rot (as defined above) that's present in these same vineyards. It's generally accepted that the vinegar in such clusters is produced by certain acetic acid-forming bacteria (species of *Acetobacter* and *Gluconobacter* are most often implicated), and that various wounds are necessary for infection to occur and disease to develop subsequently. Occasionally, these bacterial infections are accompanied or followed by infections by several wild yeasts that produce ethyl acetate (which smells like nail polish remover or varnish), although this does not occur typically.

In our work over the past 4 years, we've discovered an equally important contribution of yeast species to sour rot development. When Megan began sampling multiple sour rotted clusters from vineyards throughout the Finger Lakes region of New York and elsewhere, she always detected high populations of the standard wine yeast, *Saccharomyces cerevisiae*, in the rotten fruit along with significant concentrations of ethanol in addition to acetic acid. For example, in one series of sour rot samples from 16 different affected vineyards, she found an average acetic acid content of 2.4 g/L in juice of the affected clusters and an average ethanol content of 0.23% (v/v), with some samples as high as 4.6 g/L and 0.48%, respectively. In comparisons among the individual simples she often found an inverse relationship between the two products, i.e., as the amount of acetic acid increased there was less ethanol and vice versa. In retrospect, this isn't

surprising, since ethanol is the base product that the abovementioned bacteria convert to acetic acid.

So, sour rot appears to be the culmination in a step-wise process that begins with injury to the berries, which allows entry of both the yeasts that convert the grape's juice to ethanol and the bacteria that subsequently convert this into acetic acid (yeasts and bacteria each require wounds or natural openings to gain entrance into plant organs). We have reproduced both the visual and accompanying olfactory (smell) symptoms of sour rot in the lab by wounding ripe berries and co-inoculating them with *S. cerevisiae* and *Gluconobaceter oxidans* or *Acetobacter aceti*. However, to reliably produce typical sour rot symptoms, including acetic acid production within diseased fruit, we also have found that we must simultaneously expose the inoculated clusters to *Drosophila* flies. Both the "everyday" species, *D. melanogaste*r, and the so-called "spotted wing" Drosophila, *D. suzukii*, are equally effective in this regard although *D. melanogaster* is MUCH more common in affected vineyards.

The role of Drosophila *fruit flies*. Many people have observed the association of *Drosophila* flies with sour-rotted clusters. Because these insects are attracted to the smell of both ethanol and acetic acid, it has been thought that they are secondary colonizers of rotten berries attracted to an abundant food source—a good place to lay their eggs--and that they might help to spread the disease passively by moving the responsible microbes on their bodies as they travel within the vineyard. However, a study from Portugal published shortly before we began our project suggested that the flies might actually play a direct role in the development of the disease, leading us to examine this possibility.

The results from one illustrative experiment are shown in Figs. 37 and 38. In this experiment, Megan inoculated berries in different petri dishes with (i) the standard wine yeast, *S. cerevisiae*, and an acetic acid bacterium (*A. aceti*), or (ii) water; and simultaneously (a) introduced *D. melanogaster* fruit flies into the dishes, or (b) omitted insects from them. She then measured ethanol and acetic acid accumulation on each of the next 5 days. As shown in Fig. 37, ethanol began to accumulate significantly by Day 4 in the inoculated berries, with or without flies. One day later (Day 5), ethanol accumulation doubled in the inoculated treatment when flies were not present, whereas there was little additional accumulation in the inoculated treatment that included flies (arrow). Why? As shown in Fig. 38, ethanol was not being converted to acetic acid when the flies were not also present, whereas this did occur when flies were present (arrow). The two most likely explanations for such a phenomenon are (i) the flies are introducing microbes from their gut, which are also involved in the process of oxidizing ethanol to acetic acid; and (b) the insects are catalyzing this process through some non-microbial (e.g., enzymatic) mechanism. Of course, it is possible that both mechanisms are involved, and we have experimental evidence for each of them.

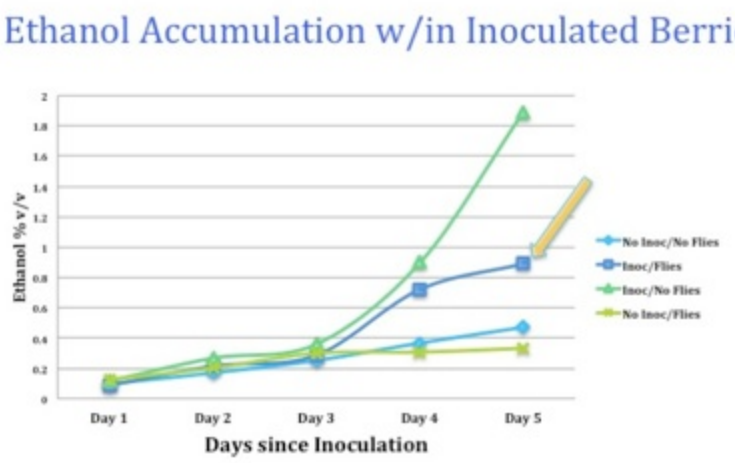


Fig. 37. Ethanol accumulation over the course of 5 days after grape berries were inoculated in the lab with a combination of *S. cerevisiae* and *A. aceti* and exposed or not to *D. melanogaster* fruit flies.

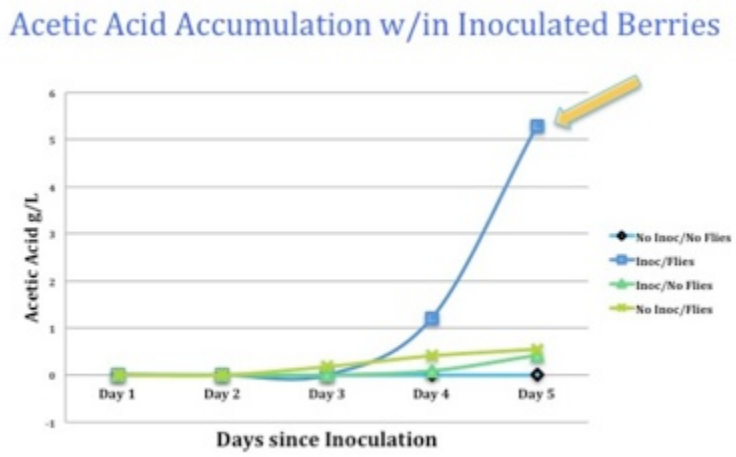


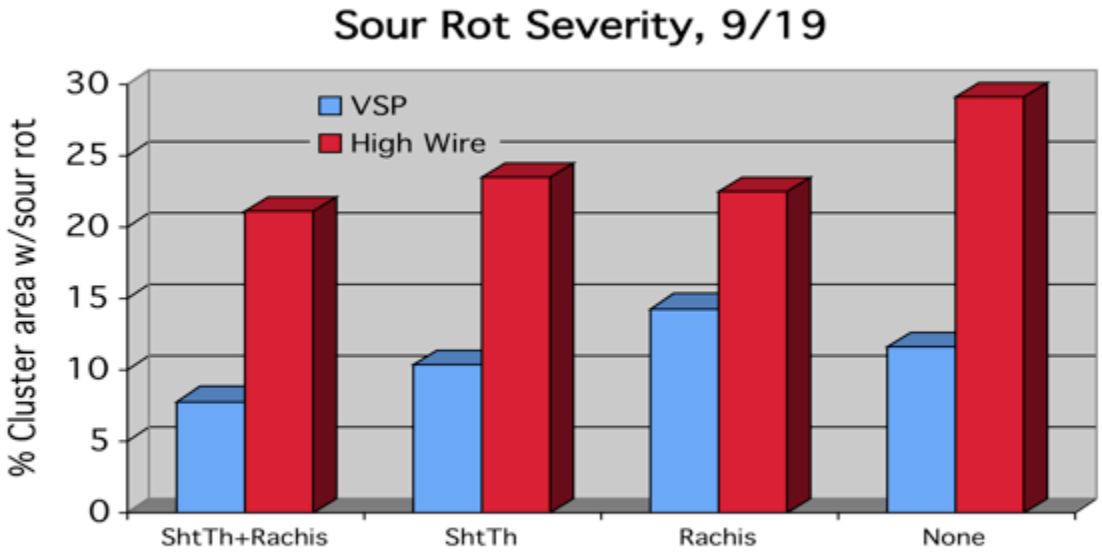
Fig. 38. Acetic acid accumulation over the course of 5 days after grape berries were inoculated in the lab with a combination of *S. cerevisiae* and *A. aceti* and exposed or not to *D. melanogaster* fruit flies.

To my mind, two of the more important things that Wendy and her Ontario group have determined insofar as understanding the development of sour rot are: (1) Berries of Pinot Noir and Riesling (the primary cultivars they've worked with) do not become worrisomely susceptible to infection until they mature to a point of about 15°Brix (minor levels of disease developed from inoculations at 13°Brix in their tests, nothing at 10°Brix); and (2) The disease develops rapidly and severely at temperatures between 68 and 77°F; much more moderately at 59 to 68°F; and just barely chugs along at temperatures in the 50's. They've also done a nice job of documenting that sour rot doesn't get started in *V. vinifera* vineyards until rain occurs after berries have reached 15°Brix and temperatures are at least in the 60's. Rain probably plays a few different roles in disease development, but two of the more important are that (i) it moves the

causal organisms around and into open wounds, plus (ii) it can help cause the injuries necessary for infection to occur in the first place (e.g., cracking that results as berries swell rapidly and/or become excessively compacted in tight clusters). All of this is certainly consistent with local observations and anecdotal reports from elsewhere that sour rot is worse under relatively warm conditions (not to mention the need for rain), but now we have some concrete numbers to go by in terms of initiating potential spray programs.

Disease management. Based on what we know about its biology, an integrated management program for sour rot might include: (1) Provide a berry microclimate within the canopy that's less conducive to pathogen growth; (2) Minimize berry injuries; (3) Minimize populations of the responsible microbial pathogens; and (4) Minimize populations of the responsible *Drosophila* flies.

Canopy microclimate. Before starting this present study, there was the opportunity to measure the effect of canopy management on sour rot in a field trial on the interspecific hybrid 'Vignoles', a tight-clustered variety that is very susceptible to pre-harvest rots. The experiment, described previously in the Botrytis section of this tome, was designed to study how fruit composition and Botrytis development are affected by different canopy management practices, and these treatments were imposed on adjacent rows of vines trained to either a High Wire Cordon or Vertical Shoot Position (VSP) system. The pre-harvest weather conditions were very wet and favorable for sour rot development, and the effects of these management factors on disease levels at harvest (Sept. 19) were significant, as shown in Fig. 39 below.



• 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%)

Fig. 39. Average sour rot severity at harvest in Vignoles clusters from vines trained to either a VSP or high-wire cordon system and subjected to three different early-season canopy management treatments (or none).

In September 2014, Megan returned to this vineyard to assess sour severity in a different season. No variable canopy management treatments were imposed, but the effect of training system was significant once again, with twice as much disease in the High Wire vines versus VSP. (Likely reason: with the High Wire system, the vigorous shoots grow down almost to the ground, essentially enclosing the clusters within a "tent" of leaves). The data are presented below in Fig. 40 below.

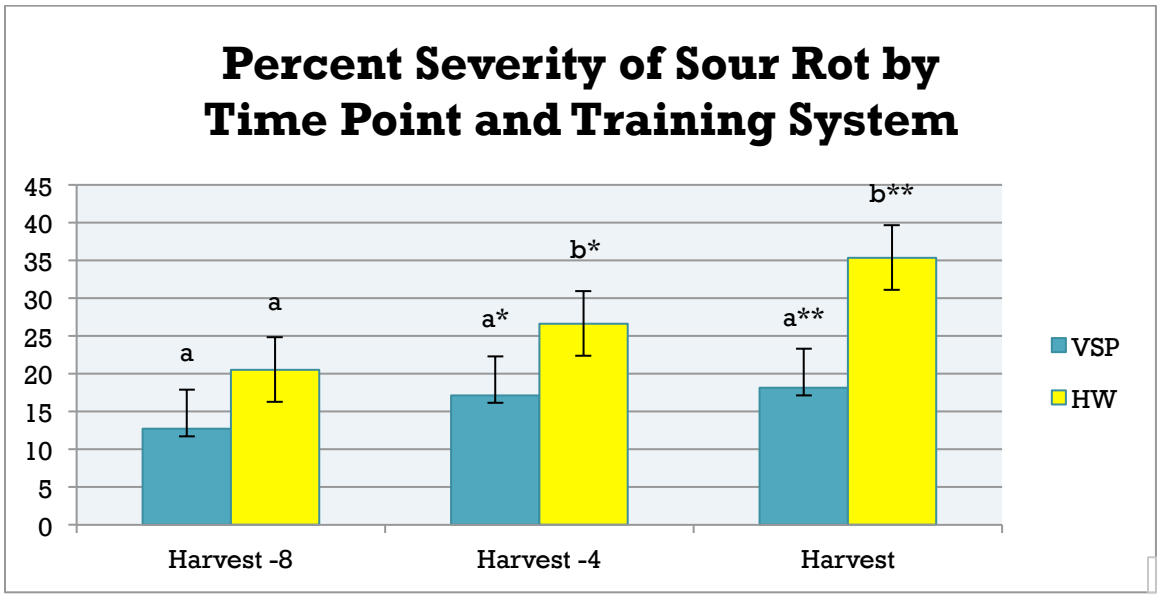


Figure 40. Effect of training system (VSP and High Wire [HW]) on the development of sour rot in a commercial vineyard of cv. Vignoles, Finger Lakes NY, 2014. Disease severity represents the average percent of the cluster area affected with sour rot, assessed on the day of harvest plus 4 and 8 days before.

Megan returned to this vineyard again in 2015 and found the same effect for a third time. As shown in Fig. 41, by 8 days pre-harvest (14 Sep) 29% of the berries in the high-wire system had sour rot whereas 16% were diseased in VSP vines in the row next to them. At that point, the grower sprayed a labeled insecticide active against fruit flies (Mustang Maxx, zeta-cypermethrin) and a labeled antimicrobial, Oxidate (dilute hydrogen peroxide), after which the disease essentially stopped progressing.

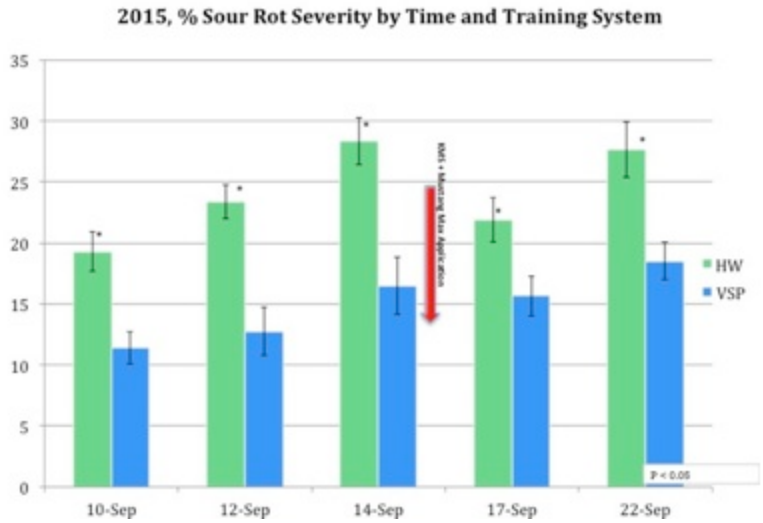


Figure 41. Effect of training system (VSP and High Wire [HW]) on the development of sour rot in a commercial vineyard of cv. Vignoles, 2015. Disease severity represents the average percent of the cluster area affected with sour rot, assessed across a 12-day period before harvest on 22 September. Note the effect on disease progression after the grower applied an insecticide and antimicrobial after the 14 Sep assessment.

Minimize injury. In addition to standard practices designed to reduce damage to clusters from birds, insects, powdery mildew, etc., loosening the density of berries within clusters is likely to reduce mechanical injuries that often occur from compaction in tight-clustered cultivars and clones. Practices to reduce cluster compaction such as leaf removal at the start of bloom and application of specific growth regulators will reduce sour rot in the same way that they reduce Botrytis, although there are potential problems with these approaches, as discussed previously in the Botrytis section.

*Minimize the pathogen population***.** In several field trials, we have obtained significant control of sour rot with two general antimicrobials: Oxidate (the dilute formulation of hydrogen peroxide mentioned above) and a 0.5 to 1.0% (4 to 8 lb/100 gallons) solution of potassium metabisulfite (KMS), applied weekly once rains begin and berries reach 15° Brix. Wendy's research group in Ontario also has obtained significant control with KMS. Although KMS is used widely in wineries both to sanitize equipment and as a food-grade additive to musts and wines to kill wild microorganisms and prevent oxidation, it is NOT registered for spraying onto vines to control diseases, either in the US or Canada. Nevertheless, these results validate the concept of utilizing general antimicrobials to help control sour rot. Furthermore, in our trials, the antimicrobial treatments are always more effective when combined with an insecticide treatment effective against *Drosophila* flies, as discussed next.

Field trial results. We looked at a combination of insecticide and antimicrobial sprays in a research 'Vignoles' vineyard near Geneva, NY in 2013, '15, and '16. Alternate rows were sprayed with the insecticide Mustang Maxx (zeta-cypermethrin) weekly beginning at 15° Brix, with the remaining rows receiving no insecticide. Then, within the "insecticide +" or "insecticide –" rows, we applied various antimicrobial treatments, also on a weekly schedule. These treatments included KMS (0.5% or 1.0%), copper hydroxide (2013 only), Oxidate, and the

new biorational product, Fracture. Most antimicrobial treatments began at 15° Brix, before symptoms were present, but a few were not applied until symptoms were visible.

In 2013, the antimicrobial treatments applied **with** insecticide provided an average of 50% control relative to the untreated check; antimicrobials **without** insecticide provided an average of 9% control; and insecticide without antimicrobials provided 15% control (*data not shown*).

In 2015, the insecticide application itself had a major effect: across the seven individual antimicrobial treatments, there was an average of 43% fewer diseased berries when insecticide was applied relative to the same treatment that did not receive an insecticide application, and a 50% reduction in disease severity resulting from insecticide application when no antimicrobial was applied. When combined with insecticide sprays, the three antimicrobial products provided additional control if begun at 15°Brix, before symptoms were present, with approximately 70 to 80% fewer diseased berries relative to vines that received no insecticide or antimicrobial spray. Antimicrobial sprays that did not begin until disease symptoms were present provided no significant additional control beyond that provided by the insecticide (Fig. 42). Due to the logistics of the experimental design, we could not include a treatment where an insecticide plus antimicrobial was applied only after symptoms first appeared, which appeared to be effective in the commercial vineyard discussed above and shown in Fig. 41.

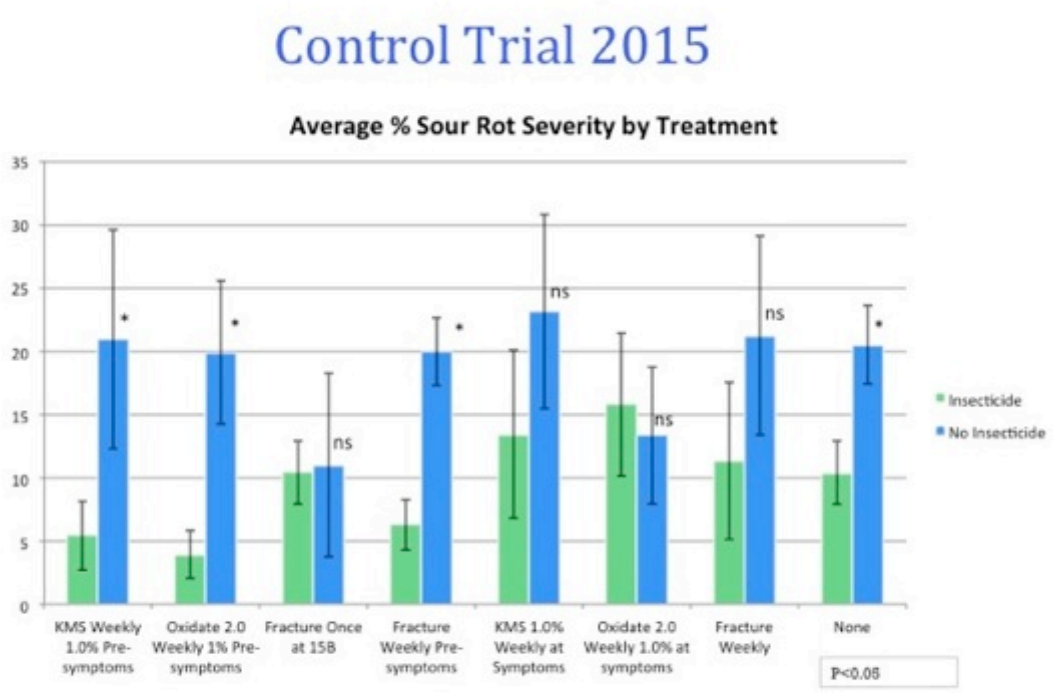


Figure 42. Degree of sour rot control provided by antimicrobial and insecticide (Mustang Maxx) sprays in an experimental 'Vignoles' vineyard; Geneva, NY 2015. Potassium metabilsulfite (KMS) in a 1.0% solution or labeled rates of Oxidate (hydrogen peroxide) or Fracture (BLAD peptide) were applied at weekly intervals beginning either at 15° Brix (Pre-symptoms) or after symptoms first appeared; Fracture was also applied once at 15° Brix. Insecticide was applied at weekly intervals beginning at 15° Brix.

A few parting thoughts (careful how you say that):

- The old adage that "the devil is in the details" is as appropriate here as it is in most cases, and we're still missing a number of details. That being said, I believe that we've gotten a decent handle on this disease and some general concepts about its management beyond hoping for the best and harvesting as quickly as possible if that doesn't come to pass. Because bacteria are a critical part of the complex and we haven't seen any consistent association with non-yeast fungi, I wouldn't expect typical fungicides to provide meaningful benefit against this disease in our region or those with similar climates, unless they happened to do so by controlling *Saccharomyces* yeasts, which would be a mixed blessing (talk to your wine maker. In warmer climates such as California, Texas, and South Australia, species of the *Aspergillus* fungus often are associated with sour rot, but whether they play a causal role in the whole complex or are mere "hitch hikers" is not entirely clear, although Megan has not been able to demonstrate a causal role in her lab studies.

- Our spray trials have been designed as a "proof of concept"—we nuked the hell out some vines in order to see whether insecticide plus antimicrobial sprays <u>can</u> have an effect. They seem to, provided that we start spraying about 15°Brix, as berries become susceptible but before the disease becomes visible, then keep on going. Of course, most growers would rather not spray that much, especially for a disease that might or might not appear otherwise. And unfortunately, the "rescue" treatments that we've tried (not spraying until disease symptoms are visible) have been less effective.

  However, it's very important to realize that in our trials we're treating a relative handful of rows embedded within a 1.5-acre solid block of Vignoles, and none of the other rows receive these sour rot treatments although they almost always get the disease. Which means that our treated rows are surrounded by nearly 1.5 acres worth of clusters that eventually are swarming with flies and full of sour rot microorganisms as the epidemic continues to build. Of course, this would not be the case in a commercial vineyard where the entire block was treated, and one spray (or two?) might be enough to stop disease progression if there wasn't a constant influx of flies and microorganisms from untreated vines all around. Indeed, this is exactly what we saw in the commercial block monitored in 2015, as shown in Fig. 41 above. We've also received anecdotal reports from other growers about stopping disease spread by spraying with a *Drosophila*-effective insecticide (Greg recommends Mustang Maxx for that purpose) and antimicrobial as soon as they see sour rot starting to develop. But of course they don't leave an unsprayed section, so it's impossible to tell whether it would have failed to spread even if they didn't spray. Time will tell.

- What does this all mean for now? Sour rot occurs sporadically and the "state of the art" with respect to understanding and controlling this disease is still a lot more sketchy than it is for most of our other important ones. Individual growers will approach managing it differently depending on their own individual risk as they perceive it and their philosophy for addressing this. For now, I'd keep these concepts in mind: Disease can be initiated once rains occur after berries reach approximately 15° Brix; warm temperatures (significant

periods of time in the upper 60's and above) are much more problematic than cooler temperatures; lots of rain can mean lots of disease; good canopy management will keep things from getting worse than they would otherwise; and it's much easier to keep things down to a dull roar if you address a disease outbreak as soon as you see it rather than waiting until things blow up in your face. Just how to do this most economically and practically is still a big question.

Knowing what we do at this point, if it was my vineyard and I had a few thousand dollars per acre of crop threatening to go south in a hurry, I'd probably keep a very close eye on my vineyards and the weather, and be ready to put something on to help control the fruit flies and responsible microbes as soon as I saw this disease get started, especially if the weather looked conducive for its spread. If I wanted to stay both cheap and legal I might concentrate just on the fruit flies, since we've obtained more consistent effects from insecticide sprays than antimicrobials (although the combination of the two is often best). If the weather was warm and wet and looking to stay that way for a bit, and I'd had a problem in that block before, I might start antimicrobials plus insecticide at 15° Brix even before seeing symptoms and back off if the weather turned more favorable (for me) and/or disease didn't get started.

Insofar as registered antimicrobials go, we've had anecdotal commercial success with Oxidate for sour rot control in addition to the experimental results obtained in the 2015 and 2016 trials (2016 data not shown), and its basic mode of action (surface sterilant) is one that I'd expect to be effective against this disease if it's applied often enough. However, it's not cheap. Fracture has looked good in our limited experience, but as noted at the very beginning of this tome many pages ago, but it's expensive and one of its purported modes of action should preclude activity against bacteria, so I'd like to remain a little cautious until I get more experience. Copper should work (and did in conjunction with insecticide in 2013), but residues persisting from pre-harvest applications may be problematic from a wine-making standpoint. Wendy has reported good results with bicarbonate products in Ontario and I can see how these might work since the target microbes are resident to some extent on the berry surface (that is, the ones that aren't brought in by flies, see previous comment about the relative contribution from insecticides), so we'll be taking a look at the salts this season as well.

"OTHER" ROTS

SUMMER ROTS is a term sometimes used for two similar diseases (ripe rot and bitter rot) common in more southern (warmer), humid production regions. Growers beneath the Mason-Dixon line and in the lower Midwest deal with these diseases on a regular basis and they occur sporadically in wet years farther to the north. Bitter rot, in particular, seems to pop up with some regularity on Long Island, particularly on Chardonnay, and reports from southern PA suggest that it's no stranger there in some years. Those of us to the north should at least start to become a little more aware of these diseases, considering the potential for evolving pest complexes as a result of climate change. They're not a threat to be over-emphasized in the more northern regions, but neither should they be flat out ignored.

Bitter rot is the more likely threat in our "marginal" northern areas, as it doesn't have the need for quite as much heat as 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 2016 Pest Management Guidelines. And there are copyrighted photos in the *Compendium*, which I can't reproduce here.

Ripe rot tends to predominate as you keep moving south, although it has been reported as far north as New England. But it likes things hot. Symptoms do not develop until after veraison but really get going as you get closer 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; these 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. I don't have my own photo of bitter rot to share but I do have one of ripe rot, see Fig. 43 below.

Figure 36. Symptoms of pre-harvest ripe rot. Note shriveled berries and spore masses on some berries, such as one in the center.

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 (recently retired from NC State), who probably has done more work with these diseases than anyone, nicely demonstrated the influence of retained inoculum by showing that rot tended to be worse on spur-pruned vines, where sections of old previous fruiting wood are systematically 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, Vangard, Luna products outside NY) provide little control of bitter rot or ripe rot, and relying on Botrytis-specific products for "rot" control during wet preharvest seasons can lead to outbreaks of bitter and/or ripe rot in regions where these latter diseases are present but not routine or consciously managed.

Sprays targeted against bitter 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 unless it stops raining for awhile. 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, winemakers also are understandably not thrilled about fruit with bitter rot either, as it's another aptly named disease. Obviously, legal preharvest restrictions on fungicide labels must be followed.

PHOMOPSIS (Ph) REVIEWS

Over the years, I believe I've seen Phomopsis cause more pronounced economic loss on Concord and (especially) Niagara grapes than any other disease. Most hybrid and *V. vinifera* cultivars are susceptible as well, and whereas they tend to be less problematic in the vast majority of such vineyards for several reasons, that's largely because these vineyards are sprayed and otherwise managed more intensively than are those of native cultivars. A brief review:

1. Early sprays are the most important for control of rachis (and shoot) 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--making sure that clusters are protected as soon as they first become visible, about 3 inches of shoot growth or so--are the most important for controlling disease on the rachises. Rachis infection by the Phomopsis fungus is ***the*** most consistent cause of economic loss that I see from this disease on Concord grapes and is even worse on Niagaras: it not only causes girdling of the rachises and consequent shriveling or pre-harvest drop of fruit from them for both cultivars (Fig. 44), but the fungus also seems to move readily from the pedicel (berry stem) into individual fruit as they ripen, especially on Niagaras (Fig. 45).

Figure 44. Girdling of the rachis by Phomopsis on cv. Niagara. Such infections typically are initiated soon after clusters emerge, during the first few weeks of shoot growth.

Figure 45. Phomopsis infection that has progressed from the pedicel (berry stem) into the berry on cv. Niagara. Such infections typically are initiated soon after clusters emerge, during the first few weeks of shoot growth.

Note that early sprays also provide the greatest control of young shoot infections, which then serve as sources of Ph spores for years to come if they are retained as infected canes, spurs, or pruning stubs (Fig. 46). Without shoot infections to begin with, there would be no spores to cause rachis and fruit infections in subsequent years.

Figure 46. Young shoot infected with Phomopsis. The pruning stub immediately above it was the likely source of Phomopsis spores that caused these infections. If the basal portion of a shoot such as this is retained as a pruning stub itself, it will similarly provide inoculum for new infections in years to come.

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

In a subsequent trial in a different high-inoculum Niagara vineyard, we 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 (Fig. 47). The quotes are to stress that this timing is approximate; the point is to get something on the young clusters soon after they emerge, ideally before the next rain but if not, then before the next one after the rain you just missed.

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.

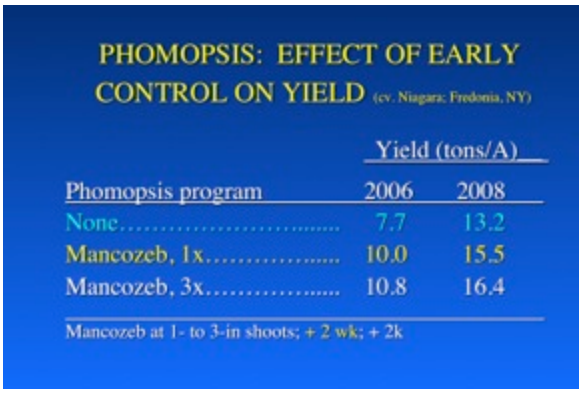


Fig. 47. Effect of a single well-timed Phomopsis spray on yield under high-yield, high disease pressure conditions. In both years, the single spray ("1x") was applied when shoots were approximately 3 to 5 inches long. In a comparison treatment ("3x"), sprays were applied at this same date plus 2 wk earlier (1- to 3-in shoots) and 2 wk later (about 10-in shoots).

3. 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 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 (Fig. 46). The Ph fungus can remain active in such wood for at least several years, so a "dirty" block is going to stay that way for a long time unless you prune the stuff out. Sanitation is especially important for anyone trying to grow grapes organically, since there are no organically-approved fungicides with good activity against Phomopsis (aside from dormant sprays of lime sulfur, that is).

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 (thanks also to Tim Weigle for considerable help with this). 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, but there's no reason to use them early if you're using them at all. Sulfur, although purportedly a decent Ph material in California (where it doesn't rain during most of the growing season) has done practically nothing in our trials. Although some additional products claim Phomopsis control on their labels, I'm skeptical, at least under the disease pressure conditions we often encounter in the east. However, even here they might do OK in blocks that are historically clean of Phomopsis and consequently have relatively little inoculum. It's amazing how well mediocre products can work if they're not really put to the test (or until they are).

6. Spray application technique. Many growers like to spray alternate rows in the early season--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 and I examined this issue in a commercial Niagara vineyard. Consistently, vines in the middle row received less spray per vine than those subjected to every-row spraying, and perhaps more importantly, the coverage on them was much more variable. The attraction of alternate-row spraying is obvious and I'm 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 really are rather than how you want them to be, so if you've had trouble controlling Ph while using alternate-row spraying, the suggested remedy is just as obvious as the benefits are otherwise.

ANTHRACNOSE

In NY and surrounding regions, most outbreaks of anthracnose historically occurred on Vidal Blanc and a few seedless table grape varieties, especially Reliance. In recent years, however, there have been regular outbreaks on some of the newer cold-hardy cultivars that are gaining in popularity and have expanded the geographical range of grape production in the Northeast and Upper Midwest. Marquette appears to be particularly susceptible, although Frontenac and La Crescent also have been affected. Some older cold-hardy cultivars (Edelweiss, Esprit, Brianna, St. Pepin, and Swenson White, Swenson Red) can be problematic as well. Concord, Catawba, and Leon Millot sometimes have problems with this disease in the Midwest, and I've received reports of problems on Steuben, DeChaunac, and Chelois from southern PA. But in NY, it's basically the cold-hardy grapes that have put anthracnose on the map beyond what we've historically experienced on occasion with Vidal and Reliance.

Although they are far from immune, most of these cold-hardy cultivars have significant albeit variable levels of resistance to powdery mildew, downy mildew, and black rot. However, it is very likely that such (limited) resistance to these diseases is related to the cultivars' relatively high susceptibility to anthracnose. Simply put, the cold hardiness of these cultivars comes largely from *Vitis riparia*, the wild grape common to the Finger Lakes region and found throughout much of the U.S. east of the Rocky Mountains. PM, DM, and BR are endemic to eastern North America, and the native *Vitis* species evolved with some resistance to them whereas *V. vinifera*, a species native to the Old World, did not. In contrast, the anthracnose organism is native to Europe, and North American species did not evolve with resistance to it; it would appear that *V. riparia* is particularly susceptible. A number of new growers, particularly of Marquette, have gotten into trouble because they correctly assumed that they could omit early sprays targeting PM, DM, and BR while not realizing that this left them vulnerable to anthracnose.

Anthracnose can cause nasty lesions on berries, leaves and young shoots, often near their base. Leaf lesions start as spots but often run together, causing large dead areas that sometimes fall out, leaving a tattered appearance (Fig. 48). Shoot lesions are somewhat similar to those caused by Phomopsis but they usually are more aggressive, expanding farther along the shoot and deeper into its center (Fig. 49). Infected berries develop spots approximately ¼-inch in diameter, often with whitish-gray centers surrounded by reddish brown to black margins (Fig. 50); old

books say that this has caused some people to call the disease “bird’s-eye rot”, but I’ve never heard anyone use that term.

Fig. 48. Anthracnose lesions on leaves.

Fig. 49. Anthracnose lesions on a young shoot.

Fig. 50. Anthracnose lesions on berries.

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 and are dispersed by splashing raindrops to young, susceptible tissues (this will be much more likely to happen from fruiting bodies that have overwintered on retained canes than from any diseased berries that might have fallen to the ground or been dropped there during dormant pruning operations). Temperatures in the mid-70's to low 80's (°F) are optimal, which is why the disease is more common on susceptible cultivars in the lower Midwest and mid-Atlantic region than it is in NY; however, infection can occur at cooler temperatures if things stay wet long enough. Additional spores, which also are splash-dispersed, are produced upon new infections and these can spread the disease rapidly via multiple repeating cycles of additional new infections and further spore production when it rains. 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; for example, berries are reported to 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 with soil through cultivation or, if practical, with mulch, although it is questionable how many of them will splash up into the canopy. Early-season sprays of mancozeb, captan, or ziram targeting Phomopsis also provide significant control of anthracnose, although this latter disease is not listed as a target on most labels (it's a disease that's not on many companies' radar due to a pretty low market potential, based on the acreage of the cultivars affected). DMI fungicides tend to have good anthracnose activity and several are

specifically labeled for anthracnose control, including Revus Top, Quadris Top, Inspire Super, Rally, and Mettle. Again, it seems likely that some other fungicides in this group also have activity against the disease, although it's very possible that the registrants don't have any data to demonstrate that fact to themselves before committing their reputation to a claim they can't substantiate (I don't know of anyone in the U.S. who runs spray trials that examine fungicide efficacy against anthracnose). In regions like the lower Midwest where anthracnose can be relatively common, I'm told that a "delayed dormant" application of lime sulfur can be very useful in vineyards where the disease has become established and problematic to control. Presumably, this would also apply to organic vineyards where traditional fungicides are not used. This treatment limits the production of infectious spores from overwintered cankers but does not protect new growth from any spores that do get produced. It's neither cheap nor fun to apply, but it's beneficial if you need it.

WOOD CANKERS

Eutypa dieback has been known to eastern grape growers for many years; in fact, not too long ago it was 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, a considerable body of work conducted across four continents since the turn of the millennium has greatly increased our understanding of the wood canker diseases. One of the leading international groups in this field has been the program of Dr. Doug Gubler at the University of California, Davis, and these studies are being expanded even further by his Davis colleagues (Kendra Baumgartner and crew) and former students (e.g., Jose Úrbez-Torres and Philippe Rolshausen) as they take positions elsewhere in the U.S. and Canada. We now know that there are a number of different fungi that cause canker diseases on grapevines throughout the world, each with its own specific biology and, potentially, appropriate management program. But there are some basic commonalities that apply to them all.

In the east, 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 managed. Nevertheless, we also have canker diseases and although these are less flamboyant than our usual rots and mildews, the perennial adolescent in me likes to refer to them as "silent but deadly" robbers of production and profit in our region, resulting from missing arms, blind wood that should be producing canes and fruit but isn't, weakened fruiting canes, etc.

A few years ago, we were very fortunate to have the above-cited Dr. Philippe Rolshausen working on the problem in eastern vineyards while he was employed temporarily at the University of Connecticut. After which he continued investigating canker diseases in our region while cooperating with other eastern grape pathologists from his base at UC Riverside after taking a position there. Philippe sampled cankered tissues from multiple eastern vineyards, determined the identities of the fungi associated with them, and confirmed their ability to cause disease in field trials in a Chardonnay vineyard at Geneva and a Concord vineyard in Portland, NY. In addition to Eutypa, he found many other organisms that are well-known causes of cankers in other parts of the world, including those responsible for a disease now known as Botryosphaeria dieback plus others responsible for the esca/black goo/measles syndrome. And a couple of new ones apparently unique to our region as well.

Canker diseases (sometimes called "trunk diseases") are particularly common in older plantings such as those that predominate our juice grape and "bulk" wine industries, and are almost certainly costing these industries as a whole more money (probably a good deal more) than many care to admit. It's a problem that needs to get onto the radar in our part of the world at some point and be consciously addressed more vigorously than it is now by most people. Most *vinifera* growers in northern climes and some hybrid grape growers provide a measure of control simply through the common practice of systematic trunk renewal, which consciously or otherwise replaces infected wood on a regular basis and in the process limits the girdling effect of slowly-expanding cankers and the loss of production associated with it. But as some of our newer high-value vineyards continue to age, particularly in regions or individual vineyards where systematic trunk renewal is not the norm (perhaps it should be?), canker diseases will become increasingly important. One famous international consultant has even suggested that canker diseases might be the greatest threat to viticulture since phylloxera. Although I believe that there's an element of hyperbole involved, I suspect that he is rightly trying to draw attention to a problem that is too often ignored or just "lived with", and which truly is an important problem worldwide wherever vineyards start to age. Interestingly, he's recently been advocating a revolutionary (to some) approach to managing it: trunk renewal (!).

Unfortunately, it's a good bit easier to recognize this problem than to manage it effectively beyond regularly renewing trunks. At the very least, we should be much more religious than many people are about getting <u>all</u> dead wood (especially dead, older wood) out of the vines and the vineyard itself, ideally putting it to the torch before the fungi within make spores to infect new pruning wounds and spread the disease. Alternatively, shredding the wood and letting it decompose on the ground is still a whole lot better than just letting it stay on or beneath the vine or in a permanent pile at the edge of the vineyard. And remember, when an arm is just sputtering along because it's largely girdled by a canker, most of that wood in the cankered region is already dead and likely producing spores. Prune it out by cutting at least 6 to 8 inches below any visible portion of the expanding canker (dead wedge exposed by the pruning cut) and train a new shoot to take its place.

In NY, we've long had a Section 24(c) "Special Local Needs" (SLN) registration that allows us to apply a concentrated solution of Topsin-M 70 WSB (3.2 oz per gallon of water) to freshly-made pruning wounds to protect against such diseases. This is not practical for routine pruning cuts, but may very well be worthwhile where larger cuts are being made for retraining purposes or to remove cankered arms (or the entire trunk) in order to replace them with new growth (hint: if they're that badly cankered, there's obviously plenty of inoculum around that needs to be protected against). In Australia, they've developed a small hand applicator gadget to do this quickly—basically, a plastic bottle filled with concentrated fungicide solution, which has a bristle brush on the end to "paint" the pruning wound with a rapid swipe or two (also, the fungicide solution is tinted bright green, so that you can see which wounds have and have not received the material).

The Aussies and Californians also have been experimenting with applying fungicide sprays after normal dormant pruning operations to limit the number of new infections and have been reasonably successful in doing so. Mettle, a DMI fungicide that's been labeled on grapes for a few years now, is labeled in all states for spray application in 25 to 50 gpa within 24 hr after

pruning, with a 12-hr REI. There are still some biological and engineering questions to answer before we know whether this approach might be a worthwhile option under certain eastern conditions, although the Aussies have shown some pretty good results with a closely related product when they use application equipment that directs the sprays onto the cut surfaces. Finding answers to these and related questions will require a long-term research project that someone in the east will need to undertake eventually, but that won't happen without support from the industry. If we're serious about being "world class" and moving the industry forward, it needs to be done.

SPRAY PROGRAMS: PUTTING IT ALL TOGETHER

As I preface this section every year, we all know that there are as many good spray programs out there as there are good growers and advisors. The following is offered as an attempt to provide some considerations for the multiple possible alternatives that are out there. But as I always like to qualify what's next, just because it isn't listed here doesn't mean it's a bad idea.

"DELAYED DORMANT" (JUST BEFORE BUDS BREAK). An application of lime sulfur (calcium polysulfide) may be warranted in blocks with a persistent history of (i) anthracnose, or (ii) black rot and/or serious Phomopsis where "organic" practices are being followed. This is an expensive and unpleasant material to apply, but if you need it, you need it. Otherwise, as the Brooklyn vineyard managers like to say, fuhgeddaboudit.

1-INCH SHOOT GROWTH. A **Ph** spray may be warranted if wet weather is forecast, particularly if the pruning/training system (e.g., hedged vineyard resulting in significant inoculum retention) or block history suggests high risk. Ditto for blocks subject to **anthracnose**, especially if the weather has gotten warm already. If you're running the sprayer anyway and have a cultivar that's highly susceptible to PM, you might consider tossing in something cheap with a bit of forward activity (yes, sulfur, unless you want to come back in next time with Stylet Oil).

3- to 5-INCH SHOOT GROWTH. A critical time to control **Ph** rachis infections, especially in blocks with any history of the disease. Or those in which you don't want to develop one. Earlier is better than later if it looks like some rain is likely to settle in, later is fine if it's looking dry and you can cover up before it gets wet. Getting in a bit late after rains first occur with young clusters exposed is still much better than doing nothing, if those are the only two options. This spray can provide significant benefit against Ph fruit infections as well, since many of them originate from movement of the fungus 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 and persist if infected canes, spurs, or pruning stubs are retained into the future. **Although several products containing Group 3, Group 7, and Group 11 fungicides are labeled for control of Phomopsis, these are weaker than mancozeb, captan, and ziram and should not be depended upon at this critical time if Ph control is important**. But at least they tend to be more expensive than the better options for this disease.

Blocks susceptible to **Anth** need protection now unless it won't rain until the next spray application.

Now is the time to start thinking seriously about control of **PM** on *vinifera* varieties if temperatures remain above 50°F for long stretches of the day. This spray is much more likely to be important in vineyards that had significant foliar PM last year than in those that were "clean" into September; however, it may be beneficial even in relatively clean blocks of highly susceptible cultivars, particularly in cloudy, wet years when temperatures aren't severely limiting. And if you're already spraying for Ph, it makes sense include something for PM on highly susceptible (and valuable) varieties while you're at it.

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 such early sprays can become, although I have my doubts that they're very useful unless BR was a significant problem in that block last year. A low rate of something pretty effective (mancozeb, ziram) or even not so (captan) might make you feel better, especially if applying it for Ph or Anth anyway. It's still too early for **DM** in NY and similar climes.

Options (mix and match). A: Nothing. B: Mancozeb or ziram (BR, Ph, Anth). C: Captan (Ph, Anth, some BR). Easier on predator mites than mancozeb or ziram, probably good enough against BR this early, but there's the 3-day REI issue. Plus the issue with oil if you want to use that now or a bit later. D: Sulfur (PM). As discussed in the PM section, historical pronouncements concerning reduced activity of sulfur at temps below 65°F appear to have been significantly exaggerated. Sulfur is sufficiently active if the temp is warm enough for PM to be active, and is a cheap insurance option. With thorough coverage, sulfur sprays can eradicate incipient infections initiated during the previous week (+/-, depending on temps since infection). E: JMS Stylet Oil (PM). Should eradicate young infections that may have occurred already IF thorough coverage is provided (there isn't an easier time than now to obtain that), and can provide a few days of limited forward activity as well, 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 F: 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 product. Can mix with captan as well as mancozeb and ziram. Option G: Rally, tebuconazole generics, Mettle, Rhyme (PM, BR, Anth); or Revus Top or Topguard EQ (PM, DM, BR, Anth). Remember, we want to limit the use of all of these DMI (Group 3) products combined to a total of three applications per season, so budget them out time-wise accordingly. The DM protection provided by Revus Top or Topguard EQ (if no Group 11 resistance present) is not likely to be necessary yet. H: Serenade, Sonata, Regalia, Double Nickel, Oso, or Ph-D (PM) if you want to experiment with biopesticide products while disease pressure is low. I: One of the PM products plus mancozeb, ziram, or captan for Ph, BR, DM, and Anth.

10-INCH SHOOT GROWTH. **Do not** wait any later than now to start controlling **PM** on *V. vinifera* cultivars or highly susceptible hybrids. On Concord and other "moderately susceptible" cultivars (or "moderately resistant", depending on your perspective), we often recommend waiting until immediate prebloom, especially if you're after a minimal-input program. However, there has been the occasional season where we started seeing PM on Concords around the 10-in

shoot growth stage, and these uncontrolled early infections then spread to the clusters and started an epidemic rolling, causing real problems later in the season. And I've had excellent Concord growers tell me that when they wait until prebloom, they see a little PM already established, which can put them behind the 8-ball right from the start. So, get out in the vineyard to see what's happening, and pay attention to the weather. No need to spray before you need to, but if you already see PM, then you need to. Or if the coming weather conditions are forecast to particularly favor PM for a while (moderately warm temps, cloudy skies, no cold nights), you might need to very soon. Remember, as crop load goes up on this cultivar, 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 (Group 3) product, and a possible time to experiment with "alternative" materials if you're so inclined.

This is one of the best times to use JMS and other oils, or other eradicant material against young "primary" infections that might just be getting started, particularly if the PM program up until now has been marginal or absent. Keeping leaves virtually free of PM going into the prebloom period helps to assure that there will be minimal inoculum to infect new fruitlets during the immediate prebloom through early post-bloom period, when they're extremely susceptible to this disease.

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.

We once recommend not waiting any later than this to control **BR**. Significant experimentation and continued experience tells us that we can get away with withholding a BR spray at this time under most commercial conditions in NY unless inoculum levels are high because this disease was a problem last year (pretty uncommon in 2016) and the weather is wet. Although if you're targeting other diseases now, there's no harm in picking up this one along the way.

Rachis and fruit infections by **Ph** are still a danger in wet years, particularly in blocks with some history of the disease. **Anth** is in season and should be controlled by growers for whom this is a concern.

Options (mix and match. A: Mancozeb (BR, Ph, DM, Anth). An effective, reasonably economical choice for everything except PM; tank mix with a PM material to complete the picture if necessary. Excessive use (like pounding it in every spray during the early season) can lead to mite problems by suppressing their predators, although two applications per year didn't have that effect when we looked at this issue with Greg Loeb some years ago. You can substitute ziram if necessary or desired, although it's likely to have the same effect on predatory mites and you'll give up some DM control in the process (that being said, it's probably good enough against DM for another week or two on Concord and other moderately susceptible cultivars). B: Captan (Ph, DM, Anth, 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 pretty well last year (limited inoculum) and good BR materials will be used in the next three sprays. Include something for PM where needed. D: Revus Top (PM, BR, DM, Anth). Superior PM control relative to anything else recommended at this stage of the season other than Quintec or Vivando, plus it gets everything else except Ph and at a competitive price. But

remember, we want a maximum of three applications per year of all DMI (Group 3) products combined; so if you want to use this product during the season, decide when a limited number of sprays might be most beneficial, considering the other diseases that it also controls. Similarly, be aware that poor DM control caused by resistance to the Group 40 component of Revus Top was confirmed for the first time last year in one Virginia vineyard, emphasizing the need to limit all products containing Group 40 fungicides (which includes Zampro) to a maximum of 2 to 3 applications per year. Not for use on Concord and a few other native and hybrid cultivars (listed in the NY and PA Pest Management Guidelines for Grapes), which may become injured by it. E: Quadris Top or Topguard EQ (PM, BR, Anth). Have slight edge over Revus Top for BR and possibly PM at the higher ends of the rate ranges, but no dependable DM control due to widespread resistance to the strobie component of each mix. F: Quintec or Vivando (PM). Both are Cadillac PM materials, and each one should be limited to two applications per season for resistance management purposes (they are unrelated to one another, so both can be used twice should you want to). 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 options. G: Torino (PM). One logical time for plugging this into the program if you're interested in it. Protective plus post-infection activity and unrelated to any other fungicide on the market. So no concerns about cross-resistance and allows you to save other PM materials for use later in the season as pressure increases. H: Rally, tebuconazole generics, Mettle, Rhyme (PM, BR, Anth). All are Group 3 (DMI) fungicides equivalent to Revus Top against BR and presumably Anth (although not all are labeled against this disease), modestly (Mettle, Rhyme) to notably (Rally) less effective against PM. And no DM, of course. I: Sulfur (PM). Historical concern about reduced activity during cool weather is way down as we look at experimental data showing this to be a minor issue, plus temps should be going up anyway at this point of the year and beyond. Sulfur's post-infection activity may be useful against any newly-developing "primary" infections, before they have a chance to form spores and spread to young clusters. I: JMS Stylet Oil (PM). If (and only *IF*) coverage is thorough**,** this spray should eradicate early PM colonies that may have started, especially if previous PM sprays have been omitted or incompletely applied. There are excellent reasons to head into the prebloom period virtually free of any PM colonies on the foliage, but don't waste your money if you can't cover thoroughly. Also may help with mites. Will provide a few days protective activity going forward in addition to the eradicative action, although much of that residual activity will disappear after a rain. Mix with something offering forward protective activity against PM if your next spray will be more than a week from now. The petroleum-based PureSpray Green should have similar effects (doesn't seem to be widely distributed in the east), whereas the botanically-based oils (e.g., Trilogy) have been a bit less effective in limited testing. Don't mix any of these with captan. Option J: Nutrol, Armicarb, Oxidate, Kaligreen. (PM). Should eradicate young infections IF thorough coverage is provided, but no forward activity. J: Serenade, Sonata, Regalia, or Double Nickel (PM) if you want to experiment with OMRI-certified biopesticide products before entering the critical period for disease control. Ditto for the biopesticides Oso and Ph-D (PM), although they're not OMRI certified. K: A PM-specific product plus mancozeb, ziram, or captan (no captan + oil!) to pick up DM, BR, Ph, and Anth as necessary.

IMMEDIATE PREBLOOM TO EARLY BLOOM. **A critical time to control PM, BR, and DM on the fruit! Also Ph, and Anth in vineyards where those are potential issues. Just starting to enter Bot season as well. This and the first postbloom application are the most**

critical sprays of the entire season—USE EFFECTIVE MATERIALS AND DON'T CHEAT ON RATES, SPRAY INTERVALS, OR COVERAGE!!

Options (mix and match). A: Vivando or Quintec for excellent PM control, plus mancozeb for BR, DM, and Ph, and Anth. No current resistance concerns with Vivando, but we want to keep it that way by avoiding over-use. There are some reports of diminished control with Quintec in Europe and just a couple of rumors and suspicious instances locally and regionally, but by and large it's been an excellent performer in our trials and in commercial usage. Let's keep usage down to a maximum of 2 applications per year for each of these materials so that they remain at the top of the heap. B: Luna Experience also provides excellent PM control (another top-of-the-heaper) plus potentially excellent control of BR and Bot (and probably Anth), depending on rate. It's excellent against PM at 6 fl oz/A and against Botrytis at the recommended rate of 8.0-8.6 fl oz/A, although we have some evidence that the lower rate is also adequate for Botrytis at this early stage. However, 6 fl oz/A only provides about 70% as much tebuconazole (the mixture component that's active against BR and probably Anth) as do the labeled rates of various generic tebuconazole products (e.g., Tebustar), whereas the Luna Experience rate of 8.0-8.6 fl oz/A recommended for BR control provides 93-100% of the tebuconazole provided by these other products. Bottom line: If using Luna Experience this time of year, you'll need to use the higher rate to provide reliable black rot control (or add 1.25 oz/A of a 45 WSP product such as Tebustar to bring it up to full strength) and mix it with something to control DM (and Ph if this disease is an issue). Remember that we want to limit the use of all products containing Group 7 compounds (which includes Pristine and Aprovia in addition to Luna Experience) to a maximum of 2 to 3 applications per year, so budget their seasonal use accordingly. C: Revus Top (PM, BR, DM, Anth). I can't overemphasize the fact that the very good PM control we've seen with the difenoconazole component of this mix is due to its high "intrinsic" activity, but that this is rate dependent. Which means that you'll start losing activity--especially on the clusters!--if you get spotty spray coverage and only deliver a partial rate to your spray target. Inspire Super (PM, BR, Bot, Anth) and Quadris Top (PM, BR, Anth, some Ph) also include difenoconazole as part of their mix, but they need to be used at the top end of their rate ranges to deliver as much of this component as Revus Top (see Table 1 way back at the start of the treatise). Furthermore, Inspire Super doesn't provide DM control and Quadris Top shouldn't be relied upon to do so because of strobie (Group 11) resistance concerns. I wouldn't use either one of these products without adding something like mancozeb for DM. And remember that we want to limit all products containing Group 40 fungicides (which includes Zampro) to a maximum of 2 to 3 applications per year.

D. Pristine (PM, BR, anth, some Ph, Bot at higher rates). **It is risky to depend on Pristine for DM control any longer and I would not do so under most circumstances, especially while clusters are so vulnerable, as per the earlier discussion of DM resistance to Group 11 fungicides way back near the very beginning of this entire treatise**. If using Pristine, add something for DM control unless you have reason to think that your risk of DM resistance (and its consequences) is low enough that you're willing to take that gamble. We need to keep Pristine and other Group 11 fungicide application numbers down to 2 per season, to maintain their activity against diseases that are still controlled reliably. In this regard, some managers may prefer waiting until later in order to target late-season rots, particularly in regions where these are a somewhat regular concern. The 12.5-oz rate of Pristine will also provide significant

protection against Botrytis, 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 about it. E: Quadris Top or Topguard EQ (PM, BR, anth). These have an edge over Revus Top for BR under high pressure conditions (since they are strong in both forward and backward activity) and possibly PM, but no dependable DM control, so add mancozeb or another DM fungicide. F: Rally, tebuconazole generics, Mettle, or Rhyme (PM, BR, anth) PLUS mancozeb (DM, BR, Ph, Anth) or captan (DM, Ph, Anth). Remember the importance of rate for PM control with the DMI fungicides and that not all of these materials are created equal (Rally seems to be notably weaker than the others under pressure). One of the new DM-specific fungicides such as Zampro or Ranman 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. Add sulfur on *vinifera* and PM-susceptible hybrids (unless "sulfur shy") for additional PM control and resistance management. Like the difenoconazole products, these other DMI materials (Rally, Rhyme, tebuconazole generics, and Mettle) provide excellent postinfection activity against BR, which can make them especially valuable if unprotected infection periods occurred over the past week or 10 days. And remember that the two products that combine a DMI with a strobie (Topguard EQ and Quadris top) will be the best of the whole lot due to excellent kick-back activity from the DMI and the excellent protective activity from the strobie component (as per Table 4 way back in the BR section). If wet, mancozeb or ziram (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).

G: Mancozeb + sulfur (PM, BR, Ph, DM, Anth). Relatively economical and effective, particularly if used at shorter spray intervals and/or young vines with little to no fruit. Neither material is as rainfast as the new fungicides that are absorbed by leaves and fruit, so shorter spray intervals can be both necessary and difficult in wet years. Potential mite problems, as this mixture is hard on mite predators if used regularly. Option H: Zampro or Ranman to control DM, plus something else from above to control other diseases that threaten your particular varieties. This is just to remind you that these DM-specific materials can be part of the mix, although they might fit better in a few more weeks after BR and Ph are out of the picture.

BLOOM. The potential importance of Botrytis infections during bloom is discussed at length in the section on this disease a few pages back. Vangard (or Inspire Super), Switch, Scala, Elevate, Pristine, Rovral/Meteor/iprodione generic, and Luna Experience applied around the bloom period often provide beneficial control of this disease on susceptible varieties, particularly in wet years. The 3-oz rate of Flint was once effective, but the extent of compromise due to resistance is a concern and relying on it for Botrytis control is risky anymore (as it is for PM control, of course). It's certainly easier to use or include one of these materials for Botrytis control in the immediate prebloom/early bloom or the subsequent first postbloom spray when other diseases also are being targeted, rather than make a separate Botrytis application in between. And from what we know of these materials' activities, they should be effective when applied at one of the standard timings just before or after "full bloom" rather than separately in between. However, one problem with tank-mixing Botrytis-specific materials like the AP fungicides and Elevate with materials targeted at other diseases is that you'll be distributing them throughout the entire canopy, whereas the only place they're really doing anything useful is on the clusters. If this is a concern, refer to some of the work that Dr. Andrew Landers had presented to address the issue.

Also, if sulfur was the only PM material in the most recent (immediate pre-bloom/early bloom) spray, it's best to reapply about now on highly susceptible *viniferas* rather than wait until bloom has finished and berries have begun to set. That is, keep the spray interval very short if relying on sulfur at this time of year, especially if it's been raining since your last application or will be soon.

FIRST POSTBLOOM (10-14 days after immediate prebloom/early bloom spray). **Still in the critical period for controlling PM, BR, DM, Ph, (and Anth, for those affected) 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 or quality of the PM material on highly susceptible varieties if weather is warm and cloudy. For Botrytis-sensitive cultivars/blocks, make sure that this application has something in it with Bot activity if the weather is favorable for this disease and you haven't used anything for it yet. Same considerations and options as detailed under IMMEDIATE PREBLOOM/EARLY BLOOM. Juice grape growers can substitute Ziram (very good BR and Ph, only fair DM) for mancozeb or captan if necessary. Captan, mancozeb, or the strobies will protect against bitter rot and ripe rot, if/where those are concerns.

SECOND POSTBLOOM. **BR** control is still a good insurance policy under wet conditions and it should be considered critical if infections are evident on the vine, unless you're lucky enough to have a few weeks of rain-free weather in front of you; however, BR sprays can often be skipped from here on out on natives and hybrids if the vineyard's clean, especially if it's not pouring. And although the same is true for *V. vinifera* blocks that are SQUEAKY clean, their longer period of susceptibility and higher value makes continued BR control a good bet for another couple of weeks even if things look good right now. 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 which may follow upon the "diffuse" PM infections that can develop on berries during this period of their transition to a resistant state. 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 (refer to the discussion/data on this topic in the earlier PM section). Concords can withstand a reasonable bit of foliar PM unless the crop is large and/or ripening conditions are marginal. Thus, minimal programs can often stop now on this cultivar if crop size/ripening conditions don't warrant additional control, although at least one more PM spray is often justified. Try to avoid applications of fungicides at risk of resistance development if there's enough PM present in the vineyard that it's easy to spot without even trying. **Ph** danger is basically over unless very wet and a problem block; even then, it's way down and nearly over since most of the season's inoculum is gone by now, so further sprays are unlikely to be cost effective unless conditions are extreme.

Foliar **DM** will remain a potential threat throughout the rest of the season, depending on the weather, and can quickly turn into an epidemic on unprotected susceptible cultivars if we get into a period of regular rains and thundershowers. It's a whole lot easier to keep this disease under control later if you don't allow it to get started now. Clusters are still susceptible to DM and

those on susceptible varieties need to be protected for a couple of more weeks as weather dictates to avoid infection, particularly if disease already is established in the vineyard (take a look and see). Which disease(s) to focus upon most heavily will depend to a great extent on cultivar and weather. Bunch closure is a time for sprays to control **Bot** on susceptible cultivars, especially if it's wet. Berries of susceptible cultivars are still susceptible to **Anth** and bitter/ripe rot.

Options (mix and match). A: Revus Top (PM, DM, BR, Anth). Excellent versus DM and BR (and Anth), very good against PM. Remember, maximum of three DMI (Group 3) fungicide applications per season and 2 to 3 applications of products containing Group 10 materials. Can cause injury on Concords and some other natives and hybrids. B: Quintec or Vivando or Luna Experience (or Aprovia outside NY) for excellent PM control + an appropriate material for DM, BR, anth, bitter/ripe rot, and/or Bot as necessary. Note that Luna Experience also provides excellent control of Bot and BR, depending on rate; see preceding text under IMMEDIATE PREBLOOM for further discussion of these materials. Remember, Quintec and Vivando shouldn't be applied more than two times per season each, and ideally neither one should be applied twice in a row. We'd also like to limit all Group 7 materials (which includes Luna Experience, Aprovia, and Pristine) to two (maybe three) applications per season in total, again avoiding sequential applications of members of this group. Admittedly, it's getting harder to limit the applications of any one group as more and more products are now containing active ingredients from multiple groups, but that's still the best objective. However, it's also fair to assume that when a given "combo" product contains two active ingredients that are both effective against a particular target disease (e.g., Pristine vs. PM or Zampro vs. DM), one application of the combo product does not present as much danger as one application of another "solo" product that contains one of the component groups.

Logical BR options to complement Quintec or Vivando include mancozeb (if still within the 66-day PHI limit), ziram, or one of the strobies. The DMI (Group 3) materials (Rally, Mettle, Rhyme, tebuconazole generics, various difenoconazole products) also provide excellent control of BR (plus Anth), but using one of them in addition to top-shelf PM products seems like overkill with respect to this latter disease unless you really need it and the price is right. We've already discussed not counting on the strobie products (Abound, Pristine, Quadris Top, Sovran, Topguard EQ) for DM but they still appear to be excellent against BR in addition to anth and bitter/ripe rot (as are mancozeb and ziram).

DM options include mancozeb (ziram is only fair), captan, Zampro, Presidio (get a 2nd mortgage), Ranman, the phosphonates, and copper, all of which are discussed in the DM review section a ways back in the text.

C: Torino for very good PM control + an appropriate material for DM, BR, and/or Bot as necessary. Provides protective plus post-infection activity and is unrelated to any other fungicide on the market, so a good rotational partner. D: Sulfur for 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. E: Pristine, Abound, Sovran, Quadris Top, Topguard EQ, or Flint. All should work well against BR, anth, and bitter/ripe rot, and some against PM (Pristine, Quadris Top, probably Topguard EQ). Some might also work against DM, but don't count on it. You can tank mix mancozeb with this group of products if reliable DM control is needed or pay

your money and take your chances if it isn't and/or you're feeling lucky. Pristine also can provide good Botrytis control at appropriate rates (as mentioned before, Flint is considered risky anymore, due to widespread documented resistance in the only eastern locations where it's been looked for, i.e., Virginia). F: Rally, tebuconazole generics, Mettle, or Rhyme for fair to very good PM (depending on rate, product, resistance status of vineyard, cultivar) and excellent BR (and anth) + add something from above for DM. G: Copper + lime for DM, some PM. Good DM and good enough PM from here on out on Concord and other moderately susceptible 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 PM off the foliage into September to make things easier next year). Also **Botrytis** on susceptible cultivars, at bunch closure, veraison and pre-harvest, according to weather and other circumstances. 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. For problem vineyards, refer to the earlier extended section on sour rot development and possible control options after berries reach 15° Brix.

On *vinifera* and other cultivars requiring continued **PM** control, sulfur is an excellent and economical choice, which is why it's so popular. Refer to the earlier section on sulfur residues on treated fruit and their resultant musts for a discussion of this issue. 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, particularly if looking for a premium material to provide an extended period of protection in the final spray. Similarly, Luna Experience will also provide excellent activity against bot PM and Bot, so might be an attractive option near bunch closure or veraison, if Bot control is needed then. Torino is another PM-specific option that can fit into rotational programs this time of year, particularly if you're trying to take the pressure off other materials since it's not related to anything else. Pristine or one of the other strobie combo products (Quadris Top, Topguard EQ) should provide good control of bitter/ripe rot in addition to good PM control, but you'll need something for downy (higher rates of Pristine also provide good Botrytis control). Copper + lime can be used on Concords, but mid- to late summer sprays for PM on this variety are probably worth the expense only under high crop and/or poor ripening conditions, although copper may be desired for DM control as well. Alternative materials for PM such as Nutrol, Kaligreen, Armicarb, Regalia, Oxidate, Serenade, Sonata, Double Nickel. Oso, and Ph-D can have their place during this period, especially if you're trying to avoid sulfur, although they generally need to be sprayed more frequently than other non-sulfur products and most of them are not cheap. The well-documented ability of oils to decrease photosynthesis and consequently decrease Brix accumulation makes me 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 at the end of the SECOND POSTBLOOM section above. **Summer rots** are controlled with largely with captan and strobies (or mancozeb

early); a peak period of susceptibility appears to be near veraison. Strongly consider an "insurance" application against **Botrytis** on susceptible cultivars/clones/blocks 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.

Best wishes for the year that's now upon us. And as Wood Guthrie once sang, So long, it's been good to know ya.