Vineyard Notes



GRAPE DISEASE CONTROL, 2016 Wayne F. Wilcox, Department of Plant Pathology, Cornell University, NY State Agricultural Experiment Station, Geneva NY 14456 (wfw1@cornell.edu)

It's (past) time once again for the (almost) 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 nowretired Duane Riegel and Judy Burr); and graduate students and post-docs too numerous to mention here. Special recognition is also due to Rick Dunst, formerly with the Cornell program at the Lake Erie grape facilities, for his (and the crew's) invaluable input on the work conducted there. It truly is the combined efforts of all of these people that serve as the basis for most of the following.

SHAMELESS COMMERCE DIVISION (with apologies to the Magliozzi brothers)

For those who don't know but might care, be aware that a major revision of the former Compendium of Grape Diseases, first published over a quarter of a century (!) ago, finally hit the streets late last summer. It is now entitled Compendium of Grape Diseases, Disorders, and Pests 2nd Edition, to reflect the fact that it also includes information about non-infectious disorders and several important pests and pest groups. It provides an updated and more extensive treatment of most topics than the original, to reflect the considerable new knowledge gained since that volume was published; sections on "new" diseases such as red blotch (virus) and Botryosphaeria blight (wood canker) that weren't even recognized back then; and detailed new sections focused on disease management subjects important to practitioners, including the fungicides used on grapes and spray application technology. 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 support industry personnel who are not expected to have formal training in the underlying biological disciplines. 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, ³/₄ of them new.

Although the book is international in scope and has contributors from a dozen different countries, all of the chapters impacting on subject matter pertinent to eastern North America were written or co-authored by individuals from our region. These include Mike Ellis from Ohio State; Wendy McFadden-Smith from OMAFRA in Ontario, Canada; Amy Rossman from USDA-ARS in Beltaville, MD; Sead Sebanadovic from Mississippi State; Turner Sutton from North Carolina State; Tony Wolf from Virginia Tech; and from Cornell, Tom Burr, Marc Fuchs, David Gadoury, Martin Goffinet, Andrew Landers, Greg Loeb, Justine Vanden Heuvel, and yours truly, who also served as senior editor (with Doug Gubler and Jerry Uyemoto from UC Davis as co-editors).

The book is sold by our plant pathologists' professional society (American Phytopathological Society), which retains ALL proceeds since the authors and editors were too stupid to ask for royalties: <u>http://www.apsnet.org/apsstore/hopapspress/Pages/44792.aspx</u>. Amazon carries it, too, but it's usually a tad more expensive there.

FUNGICIDE CHANGES & NEWS

Several new products have come onto the market over the recent past, although most of them still aren't available to NY growers. Here's a rundown of the ones that I'm aware of, and an update on a couple of issues pertaining to them.

a. Luna Experience. No, it's still not registered in NY, but it's getting closer (just keep saying, "next year" and eventually you might be correct). However, for grape growers in the other 49 states, there are a couple of new developments to be aware of.

First, the product is now registered on all grapes, it is no longer limited strictly to "wine grapes" and the explicit restriction against use on dual- or multi-purpose cultivars such as Concord (and Thompson Seedless where that is grown) is now history. FYI, the wine grape limitation reflected an EPA requirement for additional positive data on health/environmental safety issues, which has now been met; crop safety was never a concern.

Second, there was some press near harvest time last year about one of the active ingredients (fluopyram, the newish Group 7 component of the product) being implicated in crop injury on some European wine grapes after several years of use, with resulting law suits. European use patterns and rates are different than those in the U.S. (e.g., allowable seasonal maxima are nearly three times higher in Europe than here), and although there have been no similar reports of damage in our country after 4 years of use everywhere but NY, the manufacturer (Bayer) is understandably being cautious.

Thus, although they have not changed the directions on the label, Bayer is currently recommending a more limited use pattern until this issue is resolved more clearly. Specifically, they are recommending a seasonal limit of 13.6 fl oz/A rather than 34 fl oz/A as allowed on the label, applying either one application of up to 8.6 fl oz/A through bloom OR two applications of up to 6.8 fl oz/A through bunch closure. Although the 6.8 fl oz rate is moderately lower than the rate recommended for Botrytis control, our trials suggest that it is adequate for this purpose during the early stages of Botrytis development from bloom through bunch closure; however it may be inadequate for black rot control unless tank-mixed with some other product that's effective against this disease (e.g., mancozeb). NOTE: When the *2016 NY and PA Pest Management Guidelines for Grapes* went to press last November, it was thought that the preceding recommendations would be on a new label for this growing season, which we erroneously reflected in the publication.

b. "Yes, but" on Zampro. The downy mildew-specific product, Zampro received US-EPA registration in September 2012, and finally received New York DEC registration just days before this past Christmas. Now for the bad news: The NY registration includes a prohibition against use, sale, and distribution on Long Island, and this must be stated on the label of product sold anywhere in NY. Unfortunately, the process of getting US-EPA approval of a new label that

includes this information, then attaching it as a replacement onto containers of product already in the commercial pipeline takes enough time that it just isn't going to happen in time for use this season. But as we Yankees fans are saying already, there's always next year.

For the rest of you living in states where US-EPA approval is considered definitive, recall that 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, i.e., 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. 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.

One good thing that could be said about the past two growing seasons in the Finger Lakes region is that they've provided excellent opportunities to test the efficacy of downy mildew fungicides. Zampro has provided excellent disease control in both years, as has Revus Top (along with a couple of biological products still under development, which is very unusual). Some specific data from these trials are provided in the Downy Mildew section later on.

c. Fracture. Fracture is a product whose active ingredient is a fragment of a naturally occurring plant protein, and which was registered for use on grapes last year. It purportedly acts by breaking down fungal cell walls, although there are indications of activity against certain bacterial diseases as well (which means either that it doesn't work the way it's purported to or that these "indications" of activity against bacteria are misleading). In our trials last year, it provided very good control of both Botrytis and, especially when combined with an insecticide, sour rot (see later section on new sour rot research). It also is labeled for powdery mildew control, although I have no experience with it against that disease. Fracture 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). No OMRI certification currently, although the company says it is being sought.

Over the years, I've worked with more than a couple of "biorational" products that have made similar claims of activity against grape diseases and most of them have not lived up to their hype. I was pleasantly surprised to see Fracture perform so well in two different trials last year, under pretty decent pressure. Nevertheless, I'm hesitant to figuratively "bet the farm" (or to encourage commercial growers to do so literally) on an unproven technology based solely upon 1 year's worth of data. We'll be running trials again this season on Botrytis, sour rot, and powdery mildew, and if the results are consistent with 2015, I'll believe we have something. Until then, I'd encourage growers to view this product as "promising" but still experimental, and if curious to see for themselves then try it on a limited basis, ideally with a standard material nearby for comparison.

d. 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. With the application rates now labeled for use, I'd rate it as a "middle of the pack" DMI for controlling powdery mildew, a bit better than Rally (and tebuconazole products?) and more or less equivalent to Mettle but not as good as

difenoconazole (the DMI component of Revus Top, Inspire Super, Quadris Top). It also has provided excellent activity against black rot in trials run elsewhere. This material is now labeled for use on grapes as two different products, one of which is a single-component product with the trade name of Rhyme, not yet registered in NY. Its popularity will likely be determined by the combination of its relative efficacy and price compared to other solo DMI products.

The second flutriafol product, registered in NY but not allowed on Long Island, is Topguard EQ, a combination of this active ingredient (a.i.) and azoxystrobin, the a.i. from Abound and other more recent products. For what it's worth, the amount of azoxystrobin provided by the rate of Topguard EQ labeled against most grape diseases is about 2/3 to ³/₄ of that provided by the lowest label rate of Abound. The evolving status of the Group 11 (strobie) fungicides such as azoxystrobin has been discussed over the years; suffice it to say that the azoxystrobin in this mix should provide some additional control of powdery beyond that provided by the flutriafol component; it's protective (forward) activity against black rot should significantly complement the post-infection activity of the flutriafol component, making the combination outstanding against this disease; and it should **not** be depended on for downy mildew control (of course, the flutriafol component has no activity against DM, so you're probably SOL with the product when it comes to 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 difference being that the DMI in Quadris Top is the more active one against powdery mildew; this latter product also provides somewhat more azoxystrobin at the rates labeled for powdery mildew and black rot control. Not sure how the two compare in price.

e. Generic azoxystrobins. Speaking of azoxystrobin, note that its patent has expired, hence it may be showing up as various new solo products in addition to finding its way into mixes such as Topguard EQ. One labeled (even in NY!) solo product that we've worked with is Azaka. In our 2014 trial (the only one we've run), it was somewhat less effective against powdery mildew than Abound when both were used at the same rate of active ingredient, indicating an effect of the products' different formulations. However, companies commonly tinker with their formulations of new products when necessary, so it's always possible that this is no longer an issue.

f. Aprovia. Aprovia (solatenol) is the latest entrant in the list of "next generation" SDHI or Group 7 fungicides. This all started around the turn of the millennium with boscalid (the non-strobie component of Pristine), which was the first modern member of this old fungicide class and was far more active against a range of diseases than its predecessors. Since then, most of the major fungicide companies have been working on the next generation of Group 7 materials, and these have started to hit the market, although not always for use on grapes. The first of the next-gen group to be registered in the U.S. on grapes was fluopyram, discussed above as Luna Experience.

The second one registered on U.S. grapes has the trade name Aprovia when formulated as this single active ingredient (for commercial reasons, apparently, a very attractive combination product involving this a.i. has not been developed for grapes in the U.S. although it is registered on a number of other crops). Aprovia is now available for the 2016 growing season, except in New York of course ("maybe next year"). It has provided excellent/outstanding control of

powdery mildew over several years in my trials on Chardonnay, but unlike the Group 7 component of Luna Experience and Pristine, it does not provide significant control of Botrytis.

At this point, it is worth noting that 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, I have seen data from elsewhere concerning black rot control, and it was not very impressive; indeed, although Aprovia did provide some activity, it was not equivalent to standard products and I would not rely on it to control black rot if there was any significant pressure at all and berries were in a susceptible stage. All the more's the pity that Syngenta is not registering the combo product on grapes, as it would provide outstanding black rot control. But that's not an issue where most U.S. grapes are grown.

g. A recap from last year. A condensed version from this section of last year's tome, for those who may have missed it, forgotten, or are having trouble getting to sleep:

• *New-ish powdery mildew-specific products*. A final recap about Vivando and Torino: Each is a member of a new class of chemistry, so they are excellent for use in resistance-management programs as they can be rotated with all other products for this purpose. <u>Vivando has</u> consistently been one of the top performers in my trials since we started working with it a number of years ago. It has both protective and post-infection activities, and appears to provide meaningful activity in the vapor phase (moves from treated to untreated tissues in a gaseous form and then does its thing). Controlled trials with <u>Torino</u> have shown us that it has both protective plus post-infection activities, with the latter equivalent to the older DMIs in their heyday before resistance started appearing, i.e., it provides excellent control when applied thoroughly at labeled rates 3 to 4 days after spores first land on the leaves. Multiple field trials have shown that it is a good to very good product but not a "big gun", and that rates should not be cut. Its potential "fit" is as a rotational material outside the critical bloom/early postbloom period, and its 3-day PHI and powdery mildew specificity (no effect of residues on fermentative yeasts) might increase its attractiveness later in the season.

• "New" active ingredient, polyoxin-D (Oso, Ph-D). Polyoxin-D was developed some years ago in Japan as a naturally-derived fermentation product from soil-inhabiting microorganisms (*Streptomyces* species) for use in the control of particular soilborne diseases. In recent years it also has been developed against certain fruit and foliage diseases. It is registered for control of Botrytis and powdery mildew on grapevines, and is recognized as a "reduced-risk" biopesticide although it is not OMRI certified. Polyoxin-D works by inhibiting the synthesis of a component (chitin) in the cell walls of "true" fungi, so it has no activity against the downy mildew organism, which does not synthesize this compound. It is absorbed by treated tissues and therefore provides both protective and limited post-infection activity.

Two different companies sell their own polyoxin-D formulations in the grape market. One has the trade name <u>Oso</u>, the other <u>Ph-D</u>. I've worked with both, but more for Botrytis than powdery mildew control. Generally, they've been "good" against Botrytis under moderate disease pressure, and weak to very good against powdery mildew under moderate and high pressure, respectively, as shown below.

• *Botector* is a living preparation of a yeast-like fungus (*Auerobasidium pullulans*), which is purported to work by competing with the *Botrytis* fungus for colonization sites on susceptible grape tissues. Which means that it needs to be applied before (how long?) a potential infection event occurs. It is labeled for use at a rate of 6 oz/A in up to 50 gal/A of water or 8 oz/A in 51-100 GPA; I haven't seen consistent differences between 5 oz/A and higher rates in my limited testing, which has shown "good" activity under moderate pressure. One significant limitation to use of this product--even if subsequent testing and experience supports early indications of efficacy--is the fact that any activity relies upon the growth of a living fungus. And it can be killed by some broad-spectrum fungicides that might be applied to control other diseases (e.g., mancozeb, captan, strobilurins, some SDHI [Group 7] and DMI [Group 3] products). Bio-ferm, the German company that developed the material, has produced a long list of conventional pesticides that can and cannot be used with it, which is available through the company's website; if interested see: http://www.bio-

ferm.com/fileadmin/user_upload/content/produkte/Mischbarkeitslisten/160425_Compatibility_of __A._pullulans.pdf

Note that these pesticides are listed by their European trade names, which often are different from the U.S. trade name for the same active ingredient; however, the a.i. is also provided on the list for cross-referencing. Botector is OMRI certified.

• *Regalia* is a preparation derived from a plant extract, which is purported to act by boosting plant defense mechanisms. A non-toxic product that controls grape diseases by boosting the plant's natural defense system is something of a Holy Grail for companies that develop and sell plant pharmaceuticals. And just as elusive. Anyone who came up with one that really worked would make a fortune, and plenty of people have tried. But unfortunately, grapes seem to be a very difficult crop in which to "turn on" natural defense responses, and products that claim to do this have not been blockbusters, to say the least. For good reason.

Regalia is registered on grapes for the control of powdery mildew (PM), downy mildew, and Botrytis. In several trials, I've obtained fair to good control of PM under very high disease pressure and good to very good control under more moderate conditions. This is a good bottomline result, but when it comes to the mechanism behind it, we all know that a large variety of materials can control PM via direct contact action. Which is what I assume is happening here rather than a general defense induction, because we've also gotten poor control of downy mildew and Botrytis with Regalia. It is OMRI certified.

• *Double Nickel* is a natural fermentation product derived from a species of the soil-dwelling bacterial genus *Bacillus* (two different *Bacillus* species have been used to produce the biopesticides Serenade and Sonata). It's registered to control most grape diseases. It's given us poor to fair control of PM under very high disease pressure but good to very good control under more moderate pressure; poor control of downy mildew under high pressure; and fair to good control of Botrytis under moderate pressure. The product is OMRI certified.

• *Biopesticides and disease pressure.* 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 separate the stronger from the

less-strong materials. However, anybody who plans to use biopesticides (or at least, anyone who plans to use them and remain in business) knows that they must to be viewed as a mere component within a much broader integrated system that stresses limiting inoculum, employs various cultural techniques to limit disease development (e.g., good canopy management), and perhaps utilizes less-susceptible cultivars if such are available for the intended market.

Case in point: as alluded to above, in 2014 we conducted two powdery mildew control trials: one in a 'Chardonnay' vineyard with extremely high disease pressure (99% of the surface area of unsprayed clusters was diseased, they were complete toast); the other, in a 'Rosette' vineyard, had moderate pressure (40% of the unsprayed surface area was diseased). As the table below shows, the biopesticide Double Nickel provided only 24% control relative to the unsprayed vines in the Chardonnay vineyard even when applied every 7 days, but 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 Rosette vineyard.

	Chardonnay	Rosette
Untreated	(99)	(40)
Double Nickel	24*	92**
lso/JMS	57**	97**

• *Strobilurin resistance*. We don't need to keep flogging this dead horse much longer. Suffice it to say that in general I would no longer count on any of the strobies to provide control of powdery or downy mildew when used alone, although there may be exceptions in blocks of at least moderately resistant cultivars (e.g., Concord and a number of hybrids) with a limited history of using these products. Fortunately, we have a couple of competitive products (Pristine, Quadris Top, possibly/probably Topguard EQ) in which a strobie is combined with an unrelated fungicide that gives at least very good control of powdery mildew. Thus, considering that even in "strobie-resistant" vineyards a significant proportion of the pathogen populations is still susceptible to these fungicides, the partial control that they contribute on top of the heavy lifting provided by the mixing partner generally adds up to excellent powdery mildew control when these combo products are used.

Unfortunately, we do not have similar mixtures, 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 in a Merlot vineyard of a very good Finger Lakes grower who was not aware that two Pristine applications made during the critical period near bloom were no longer effective against this disease, even though they had been in previous years. For

what it's worth, black rot resistance has not been reported from anywhere in North America or Europe that the disease occurs. As explained in another section, the danger for BR resistance is much lower than for DM and PM, although it could happen eventually.



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 a 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.

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, life forms other than diseasecausing fungi, etc.). It's not that hard to register a product that doesn't cause much harm to anything, including its intended target organisms (in the U.S., product registration requires only that a product is shown to be safe; by law, adequate efficacy is determined by the marketplace rather than the regulators). But unfortunately, it's very rare to find a compound that's (nearly) benign to most life forms yet deadly to target fungi.

When such a compound is discovered, the molecule typically affects only a single process in the fungal metabolism, and often 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 these materials are often very effective at controlling disease yet 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". 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", which is no longer present within a significant proportion of the developing pathogen population.

As just illustrated, fungicide resistance is a classic albeit rapid example of evolution, 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. When this process results in these individuals dominating the pathogen population to a point that the fungicide no longer provides acceptable disease control even when applied properly, a condition termed "practical resistance" is reached. The risk of this occurring is a function of both (i) the mode of action of the fungicide itself (i.e., what is the target-site enzyme "lock", what are the chances that a simple mutation will occur that doesn't harm the fungus but makes the fungicide unable to bind); and (ii) the individual disease involved.

Diseases at the greatest risk for practical resistance development are those caused by pathogens (i) with the potential for producing multiple generations per year (a few resistant individuals that might arise through mutation can become a few hundred thousand in short order if the weather is favorable and there's nothing providing control of them, i.e., preventing their multiplication); and (ii) which also produce large number of spores that can be widely dispersed by air currents, thereby spreading the resistant strain far and wide (spread the love!). Powdery and downy mildews are textbook examples on both accounts, and Botrytis isn't too far behind; indeed, grape producers are "lucky" to have three of the pathogen groups most notorious for fungicide resistance development cause three of their most important diseases.

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 <u>qualitative</u> (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.

Examples of fungicides to which such qualitative resistance (immunity) among grape pathogens has either occurred within or threatens eastern US vineyards include (i) the benzimidazoles (e.g., the former Benlate, Topsin-M), with resistance among powdery mildew and Botrytis populations common in many locations; (ii) the strobilurin and other QoI fungicides (Abound and other azoxystrobins, Flint, Sovran and other kresoxim-methyls, 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", and Botrytis resistance is being found commonly by Anton Baudoin when he looks for it in Virginia (could well be in NY, too); and (iii) the phenylamides (e.g., the Ridomil products and generic metalaxyl), 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!).

In contrast to the above model, resistance is said to be <u>quantitative</u> 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. In this case, repeated use of the same class of materials 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 of resistance described above for some other fungicide groups, this quantitative type represents a yes/kind of/not really or white/light gray/dark gray scenario. A well-characterized example of quantitative resistance is that exhibited towards the DMI ("sterol inhibitors", Group 3) fungicides within populations of the powdery mildew fungus, which we have been discussing for years. A very practical illustration of how this principle applies to both long-term resistance management and immediate disease control will be reviewed briefly below.

Given the preceding, basic resistance management strategies for all fungicide groups include:

• Limit the number of selection events, i.e., limit the number of applications of an at-risk fungicide and related products having the same biochemical mode of action. It is now very easy to recognize whether different products have the same or different biochemical modes of action, simply by looking at the Resistance Group number that is on the front of each label for most

products that are considered at risk of resistance (e.g., Group 11 for the QoI fungicides, Group 3 for the DMIs, Group 7 for the SDHIs).

• 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 high risk of resistance development as a "rescue" treatment if a severe disease outbreak occurs. Of course, you might wonder about the wisdom of maintaining the future utility of a fungicide for a business that might have no future itself if the disease 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 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 often isn't enough in and of itself, but it's always the first line of defense and more influential than many people give it credit for 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 need to use 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), the more conservative you should be, especially once there's a history of using that group of products.

(iii) Apply at-risk materials in combination with another unrelated fungicide active against the target disease, either through tank mixing or use of a pre-packaged product containing two or more effective ingredients. Be aware 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 use rate of the companion (unfortunately, a not-uncommon problem with prepackaged mixtures) will have a limited effect on slowing the reproduction of any resistant individuals that might 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 materials. 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.

Data presented in Table 1 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 tests showed that difenoconazole is, on average, nearly 26 times more active than myclobutanil on an ounce-by-ounce basis of active ingredient (not 40x more active as incorrectly reported before, but still...). Quite simply, the population of the PM fungus in this vineyard has shifted to the point that it is no longer controlled by the myclobutanil dose provided in the maximum label rate of Rally (which was more than 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 fungal targets are actually exposed to at any given rate of application. 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.

	Leaf ir	fection	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**		1.1	6	0.2			
Rally, 5 oz		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			

Table 1. Control of powdery mildew on Chardonnay grapes; Geneva, NY 2010

* Seven sprays applied at 14-day intervals.

** Inspire Super at 20 fl oz contains the same dose of difenoconazole as the Revus Top treatment.

MORE NEWS FROM 2015—ANGULAR LEAF SCORCH

Angular leaf scorch (ALS) is a disease that occurs very sporadically when we have wet springs, especially if we have two in a row as we just did in the Finger Lakes. How much damage it really does is not clear, but like any disease, that depends on how severe it gets. It does not affect fruit directly but can cause premature loss of severely diseased leaves if it gets out of hand in a bad year. It also can cause a lot of head scratching and some concern among growers when it shows up from time to time and they wonder how bad it might get and what to do about it. Several photos below (Figs. 2-5) show various symptoms seen last year on a native, *V. vinifera*,

and hybrid cultivars, provided as a diagnostic FYI for those who might have been in the head-scratching contingent or find themselves there in the future.

The ALS fungus overwinters in infected leaves on the vineyard floor, hence the double whammy of two consecutive wet springs: the first one provides an above-average number of diseased leaves to produce an above-average amount of inoculum the following year, the second one goes to town with that. Infection from this source reportedly occurs during rainy periods from the period of early shoot growth through bloom or so, with a possibility for additional spread from current-season infections should prolonged wet periods occur subsequently (they did).

Although there are few specific labels for the control of ALS, mancozeb products applied to control Phomopsis, black rot, downy mildew, and anthracnose also appear to provide good control of ALS. In Europe, the strobilurin fungicides have provided good control of a closely related disease named *rotbrenner*. The DMI fungicide, difenoconazole, is labeled for control of *rotbrenner* and should provide some post-infection control of ALS in addition to moderate protective activity (excellent protective activity for Quadris Top due to the azoxystrobin component).



Fig 2. Symptoms of angular leaf scorch on cv. Riesling (courtesy C. Daum). Riesling was once described as only slightly susceptible, but recent inoculation experiments showed it to be among the most susceptible cultivar tested.



Fig. 3. Symptoms of angular leaf scorch on cv. Noiret. (courtesy C. Daum)



Fig. 4. Symptoms of angular leaf scorch on cv. Concord. Concord was once described as only slightly susceptible, but recent inoculation experiments showed it to be moderately to highly susceptible.



Fig. 5. Symptoms of angular leaf scorch on cv. Concord, different vineyard than Fig. 4. (courtesy C. Daum)

NEW FINDINGS: 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 and brought new questions to the associated viticultural community at large, where experience with these cultivars is generally 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 a few basic questions without clear answers. One of these is cultivar tolerance to several common fungicides that are used without problem on *V. vinifera* cultivars but which can cause varying levels of injury on certain natives and hybrids. Of particular interest are copper, sulfur, and the DMI difenoconazole (Revus Top, Quadris Top, Inspire Super), pertaining to some largely obvious issue that include resistance management, organic acceptability, efficacy, and cost.

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: <u>http://northerngrapesproject.org/wp-content/uploads/2016/02/NG-News-Vol5-I14-Feb2016.pdf</u> 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 hadn't yet been recognized, damage also was observed on Brianna and minor damage was observed on St. Croix).

POWDERY MILDEW (PM) REMINDERS

Your annual 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, after which it can spread rapidly via the millions of new spores produced from each of these "primary" infections. Thus, the amount of fungus capable of starting disease this year is directly proportional to the amount of disease that developed last year. An important consequence of this is that disease pressure will be higher, and PM sprays during the first few weeks of shoot growth are likely to be far more important, in blocks where PM control lapsed last year 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.)

The annual illustration of what this means: 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 in early July, to represent a planting where PM control broke down for one reason or another. The next spring, the levels of chasmothecia (number per kilogram of bark) in these treatments were (a) 1,300; (b) 5,300; and (c) 28,700, respectively. Now, consider a hypothetical case where 20% of the overwintering spore load is discharged during the first couple of weeks after bud break (a

reasonable scenario, based on 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 <u>entire seasonal supply</u> on the clean vines; and in treatment (c), it's <u>four to five times greater</u> than the entire seasonal supply on the clean vines.

Not surprisingly, this makes a difference—that is, the degree of control provided in one season can affect the success of the control program (or its required intensity) the following year. When we intentionally waited until the immediate prebloom period to apply a minimal spray program to these same vines the year *after* the variable foliar disease levels were allowed to develop, the resulting cluster disease severities (proportion of the cluster area infected) were (a) 11%, (b) 22%, and (c) 48% in these respective groups, even though all vines were sprayed exactly the same during the second season.

WHY: Higher disease in Year 1 = More inoculum to cause primary infections early in Year 2 = Many more new ("secondary") spores produced from these early infections by the time new berries were forming and highly susceptible to PM. And the more secondary spores that were present when these new berries started forming, the less effective was the fungicide program in controlling the disease. This is a basic concept 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 disease management program. Depending on the disease involved, sometimes inoculum reduction can 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. And in annual production systems, it can be greatly facilitated by crop rotation. But for a perennial crop like grapes, the very best way to minimize inoculum levels at the start of one season is often 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.

(ii) Powdery mildew functions as a "compound interest" type of disease, that is, a few infections can snowball and build up to many in a short period of time if conditions are favorable for reproduction of the fungus (a high "interest rate"). The most important environmental factor that governs the rate of reproduction is temperature, with a new generation produced every 5 to 7 days at constant temps between the mid-60's and mid-80's (°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 and nights in the 60's and 70's provide ideal conditions for the fungus 24 hr a day. Conversely, a very cold night or two can seriously set the fungus back while it's trying to ramp up in the spring, as discussed a little farther below.

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

(iv) Berries are extremely susceptible to infections initiated between the immediate prebloom period (when the fungus establishes on the tiny flower stem, from which it later expands onto the developing fruit) and fruit set, after which they become highly resistant to immune about 2 weeks (Concord) to 4 weeks (*V. vinifera*) later. This is when you use the good stuff and don't 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 <u>excellent</u> 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. This makes it subject to control by topical treatment with any number of "alternative" spray materials (oils, bicarbonate and monopotassium phosphate salts, hydrogen peroxide, plant extracts, etc.), all of which have little to no effect on other disease-causing fungi that do their dirty work down inside the infected tissues. Recall that there are two primary limitations to the aforementioned group of products, which need to be considered if you want to use them effectively: (a) they work by direct physical contact with the fungus, 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 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 (via vapors?), the interval between sprays can be extended to a modest extent by increasing the rate (unless it rains that is, in which case they largely wash off).

Once-new research I: Effect of sunlight exposure

"Everybody knows" that PM is most severe in shaded regions of the vineyard (canopy centers, near trees, etc.). Here's another recap of the work of former graduate student, Craig Austin (now gainfully employed and paying taxes to help support us future 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 group of

50-foot tall pine trees. In previous years, we had seen PM completely destroy the clusters on the three panels of vines immediately next to the trees, despite a spray program that controlled the disease adequately on all other vines in the block. These panels were shaded by the trees during the morning and it wasn't until the sun crested over the treetops just before noon each day that the vines received their first direct exposure to sunlight. So, we initiated a trial in which Craig inoculated leaves of individual vines on either (a) their sun-exposed outer edge or (b) the inner portion of their self-shaded canopy, when vines were located either (i) immediately next to or (ii) 200 feet away from the trees, thereby providing a total of four levels of natural shade. The resulting disease severity increased substantially with each increasing level of shade, becoming 8 to 40 times more severe on the most heavily shaded leaves (interior canopy of vines next to the trees) compared to the no-shade leaves on the exterior of vines away from the trees (Fig. 6).

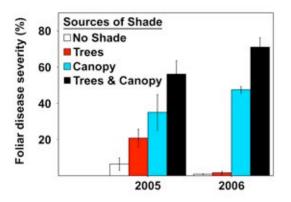


Figure 6. Percent area diseased on Chardonnay leaves receiving (i) full solar radiation, on the outer canopy edge of vines away from trees (No Shade); (ii) morning shade from an adjacent grouping of trees, but which were fully exposed to the sun for the rest of the day--i.e., leaves on the outer canopy edge of vines next to the trees (Trees); (iii) shade provided throughout the day by the vine canopy itself, i.e., leaves located within the center of the canopy 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 <u>leaf</u> temperatures were dramatically different among the different treatments. Within the shaded regions, UV levels were (as one would expect) a mere fraction of those in the sun, and temperatures of leaves in the sun were as much as 10° to occasionally even 30°F higher than those of leaves in the shade. As we later found out, both elevated leaf temperature and UV radiation are responsible for the inhibitory effects of sunlight on PM development.

Sunlight characteristics influencing powdery mildew development. Direct sunlight heats up exposed leaf surfaces, as it does anything else it hits--as we all know from the difference between standing in the sun on a bright day or taking two steps away into the shade. On warm days, this additional heat 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 80's, temperatures of shaded leaves and clusters will remain near that of the air—which is at or near the optimum for PM development. However, leaves and clusters that are fully exposed to

sunlight often have temperatures elevated to a point where the PM fungus will stop growing or even start to die.

UV radiation from the sun can damage the cellular structure of virtually all forms of life. And powdery mildew is a disease that's uniquely vulnerable to such damage, since the PM fungus lives primarily on the outside of infected tissues whereas nearly all other pathogens live and grow within infected organs, where they are protected from UV radiation. On top of that, the PM fungus is white--it has no pigment ("suntan") to protect against this radiation. Lab experiments confirmed that doses of UV typically measured during a summer afternoon in the Finger Lakes region (hardly a world beater when it comes to sunshine intensity!) are deleterious to pathogen development and that this effect is even more pronounced (interactive effect) once leaf temperatures get in to 80's and beyond. And as noted above, sun-exposed leaves are hotter than those in the shade, so sunlight actually exerts a triple whammy against this disease (heat, UV, and their interaction).

Surface temperature and UV: Field experiments. In order to quantify the individual effects of these two specific sunlight components, 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 7, we found that removing UV radiation while still allowing exposed tissues to heat (Plexiglas filter) increased disease severity on fruit by anywhere from 50% to fivefold, for 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 further increased disease severity in one of the two experiments.

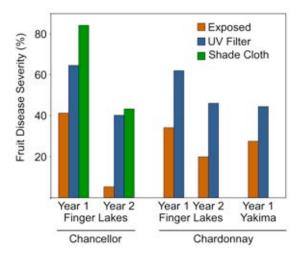


Figure 7. Percent cluster disease severity on cv. 'Chancellor' and cv. 'Chardonnay' vines receiving: (i) full solar radiation (Exposed); (ii) sunlight from which 95% of the UV radiation had been filtered (UV Filter); or (iii) sunlight

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

We found that both factors affected PM severity (Figure 8). First, powdery mildew severity was lower in the VSP than in the UK training system, regardless of leaf pulling treatment. Second, leaf removal <u>at fruit set</u> significantly reduced the amount of disease in both training systems, 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. Quite simply, it's when you either do or don't get control of the disease on 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 <u>all</u> of the tools at your disposal to manage diseases, as good growers typically do, by definition. This takes the pressure off the fungicide component, which reduces the pressure for resistance development, improves control levels over the long haul, and gets the sun shining and the birds tweeting (well, maybe not that last bit). It should be noted that in 2009, a summer during which it sometimes seemed that there was no direct sunlight reaching the state of NY, we did not see the same effect of training system in this vineyard but did see the same effect of early leaf pulling.

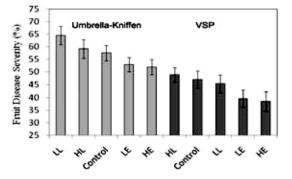


Figure 8. Powdery mildew severity on Chardonnay clusters subjected to five different leaf-removal treatments in each of two vine-training systems. Leaf-removal code: **First letter** is leaf removal severity, H = heavy, L = light (either two leaves or one leaf above and below each cluster, respectively); **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.

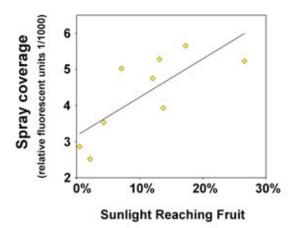


Figure 9. Effect of canopy density on deposition of sprays onto clusters of 'Chardonnay' vines, provided by a conventional airblast sprayer in mid-July.

Exposure of fruit to sunlight <u>and pesticides</u>. 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 9), 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, 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 their different 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 a Berthoud airblast unit delivering 50 gal/A in early July (a critical time for controlling multiple diseases on clusters), was then determined in the lab by measuring the dye washed from a sample of clusters from each of these same test panels. Finally, the average deposition in each test panel was graphed as a function of its CEL value, yielding Figure 10 below.

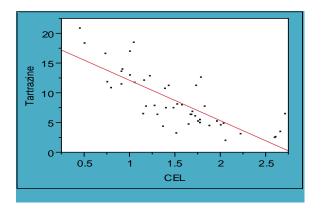


Figure 10. Effect of canopy density (cluster exposure layers = CEL) on deposition of a spray tracer dye (tartrazine) onto grape clusters in five Finger Lakes vineyards. Vines were treated in early July with a conventional airblast sprayer applying 50 gallons/acre.

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 $\frac{1}{2}$ or 1 $\frac{1}{2}$ leaves of canopy, which exerts a greater impact on the dose of fungicide deposited on the berries than does that rate range going 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 can lead to disease-free berries that are sunburned instead. It's all about balance.

Once-new research II: What's a 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 define just 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 she inoculated young leaves to establish PM on the foliage pre-bloom, providing abundant new spores to infect the adjacent fruit while they were first developing and highly susceptible. This is logical and consistent with the results discussed earlier concerning how the inoculum level carried over from one year affects the level of fruit disease that develops the next.

• 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, whereas a short and cool fall period leads to 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 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 period, there was a consistent relationship between weather from the immediate pre-bloom period through fruit set or a bit beyond: sunny and dry = good for you, cloudy and damp = good for mildew.

• 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 to 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). Thus, cold nights during the period between early shoot growth and bloom have the potential to restrict the ability of the PM fungus to produce new spores capable of infecting highly susceptible new 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 that time is good. Or seen another way, a lack of cold nights during the first several weeks 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 get things ripe: low to moderate crop size, plenty of 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 have become 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. Workers from Australia also reported no differences in control at 59°, 68°, or 86°F when used at this rate (there was a slight decrease in control at 59°F with a low rate equivalent to 1.7 lb/A). It appears

that the potential detrimental effect of low temperatures on sulfur efficacy has been overemphasized in years past, particularly if you consider that the PM fungus itself is not all 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 top-rate control with sulfur on leaves than it is on clusters, and we're only dealing with leaves early in the season.

• <u>Sulfur provides excellent post-infection control when applied up through the time that young colonies start to become obvious.</u> 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 <u>thoroughly</u> soon enough after infection begins. Which is up through about 1 week after infection is initiated if temps remain mostly in the 70's and 80's, 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 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 (as in the backs of mildewed clusters, which often remain diseased when such sprays are applied by commercial growers and the coverage doesn't reach them). It might be worth at least thinking about spraying both sides of the vine if you've gotten into a big enough pickle that you're putting on an eradicant spray.

• 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 further protective activity significantly. Shock.

- This effect 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 "spreadersticker" 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 2 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.

(2007-10) grapes as affected by suffur rate and adjuvant, when applied at 14-day intervals (Geneva, NY)							1)											
	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

Table 2. 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)

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

"Sort-of new" sulfur research: Effects of use patterns on sulfur 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 from the harvested fruit invariably get the blame when things get stinky.

The question that growers commonly ask is, "How late can I spray sulfur and still be safe?" And until recently, my answer was, "Everybody has an opinion 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), 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. 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 applied this technique after imposing various sulfur treatments 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/longer performance is one reason that growers are willing to pay more for the micronized formulations (smaller particle size = sticks tighter to the fruit). But if residues are a concern, you might want to cut rates and use a WP formulation as you get closer to harvest.

• 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 <u>not</u> the case for red or other wines fermented on their skins). See Figure 11 for a graphic representation of this phenomenon. (Anyone interested in all the gory details can find them in Am. J. Enol. Vitic. 65:453-462, which is available for free through the American Society of Enology and Viticulture website).

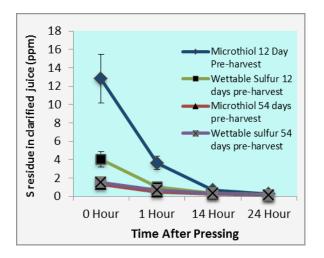


Figure 11. The effect of clarification through settling on elemental sulfur residues present in juice prior to the start of fermentation. Juice was pressed from fruit that received sequential applications at 14-day intervals of two commercial sulfur formulations (5 lb/A formulated product), ceasing either 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 product/100 gal water, in order to assess the product's 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 another salt onto it, most effectively when the colony is young and easy to wet. However, it's the salt <u>solution</u> 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 <u>do not affect diseases other than PM</u>—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) REMINDERS

Recall that the DM organism persists in the soil as resting spores (oospores) that originate within infected leaves and berries. Hence, the more infection last year, the more oospores this year. And last year was another one that produced above-average levels of DM by the end of the season in many regions, including much of upstate NY.

The season's first ("primary") infections, originating from dormant oospores overwintering in the soil, require a minimum rainfall of approximately 0.1 inch and a temperature of 52°F or higher to "wake up" these oospores and splash their infectious progeny up into the canopy or onto nearby sucker growth. 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. 12). 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. 12. Typical white "downy" appearance on the underside of a DM-infected leaf, consisting of masses of reproductive structures of the causal pathogen.

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 it--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 imperfect 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 if it does get active. Keep an eye on the vineyard to see which of these possibilities 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 at http://newa.cornell.edu/index.php?page=grape-downy-mildew

Fruit susceptibility. Clusters of some varieties—including all *V. vinifera* cultivars--are highly susceptible to infection as soon as the DM organism becomes active during the prebloom period (in Geneva, our first infections on highly susceptible cultivars exposed to plenty of inoculum typically occur about 3 weeks before the start of bloom, later if it doesn't rain until then). Research indicates that berries become highly resistant to <u>direct</u> infection within 2 weeks after the start of bloom, resulting in classic DM spore production from diseased fruit. 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. 13). 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. 13. "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+ 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. 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.

Fungicides. Ridomil remains the best downy mildew fungicide ever developed, although cost and lack of activity against other diseases have limited its use in the U.S. Which 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 VERY prone to resistance development--indeed, it's no longer effective in many of the DM-prone parts of the world--and although resistance has never been detected on grapes in the North America, this is probably due in large part to relatively limited use as noted above. Resistance development is a MAJOR concern, and in an ideal world all resistance-management precautions should 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 (so much for the recommendation to avoid the 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 and is strongly not 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 (unless you live in NY) and Revus/Revus Top are very effective. So is Presidio (not discussed previously), 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.

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, 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 group of pathogens; however, the materials are not toxic to other ("true") fungi, and control of the diseases that they cause is erratic at best. If you do get control of another disease, think of it as an unanticipated bonus. I certainly wouldn't encourage you to even hope for it, 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 it's only legal to obtain disease control with these latter products if you don't do so on purpose. Whether this seems fully rational or not, remember that the law requires 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.

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), <u>depending on the rate used</u>, 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 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.

• <u>CAUTION</u>: 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 is 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 <u>are</u> 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 a dozen years of common 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 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. 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) 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 <u>can</u> 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 too concerned towards the end 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 most vineyards, 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 <u>very</u> 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 infectious 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.

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 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.

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 (Fig. 14). 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. 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.

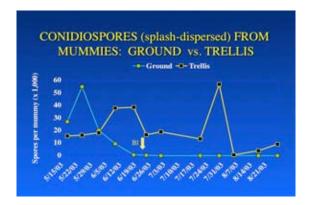


Fig. 14. Production of splash-dispersed "conidiospores" from black rot mummies (previous year's fruit infections) overwintered in the trellis or on the ground. The yellow arrow ("Bl") indicates the approximate time of bloom. Note that the BR fungus also produces a smaller quantity of a second type of spore (airborne "ascospores"). These spores are similarly produced throughout the summer from mummies in the trellis, whereas mummies on the ground continue limited production until 1 to 2 weeks after bloom (data not shown).

3. The incubation period for BR can be <u>very</u> 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. 15). (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.

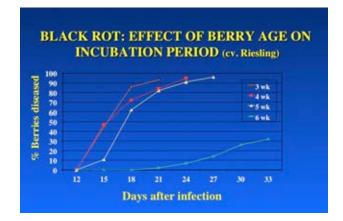


Fig. 15. Effect of 'Riesling' berry age on the incubation period for black rot (similar for 'Chardonnay' berries, data not shown). Berries were inoculated in the vineyard with BR spores either 3, 4, 5, or 6 weeks after cap fall and the percentage showing symptoms was determined every 3 days thereafter.

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

	% Disease control ^c			
Protective (days) ^a	Abound	<u>Rally</u>		
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 <u>before</u> 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 <u>after</u> 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 Topgaurd 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 such 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.

All of the strobies appear to be equivalent to one another and provide very good to excellent control, equal to mancozeb and ziram (and ferbam, ugh) 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. Mancozeb and ziram are old standards and provide very good control under most commercial conditions. Captan is only fair, and likely to be inadequate if there's any pressure. Copper is discussed below. Sulfur is poor.

6. Special considerations for organic growers. Black rot 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 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.

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 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).

BOTRYTIS (Bot) REMINDERS

Although there are a number of fungi that can cause bunch rots, especially in hotter climates, Botrytis is still king throughout most of the world where pre-harvest temperatures tend to be on the moderate side. A review of what makes it tick.

1. Biology. The Botrytis fungus thrives in high humidity and still air, hence the utility of cultural practices such as leaf pulling and canopy management to minimize these conditions within the fruit zone. It's a "weak" pathogen inasmuch as it primarily attacks tissues that are highly succulent/juvenile, dead, injured (grape berry moth, powdery mildew, rain-cracked), or senescing = expiring (wilting blossom parts, <u>ripening fruit</u>). Although the fungus does not grow well in berries until they start to ripen, it can gain initial entrance into young fruit through wilting blossom parts, old blossom "trash" sticking to berries, and scars left by the fallen caps (Fig. 16). Such infections typically remain latent ("dormant"), but some may become active as the berries start to ripen (senesce), causing affected berries to rot. Should this occur, disease can spread rapidly through the rest of the cluster or to others nearby, reducing both marketable yield and quality. (Latent infections can also become active if the berry is killed naturally or artificially, Fig. 17).



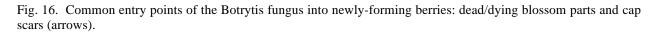




Fig. 17. Young grape berry killed by placing it into the freezer, allowing latent Botrytis infection to become active and colonize it.

Some details regarding the above, gleaned a while ago now:

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

• Serious Botrytis losses result from rampant disease spread during the post-veraison/ pre-harvest period, after berries begin to ripen and become highly susceptible to rot by the fungus. Thus, latent infections established at bloom can be important not so much due to the direct losses that they might cause (relatively few of them become active), but because only a few becoming active post-veraison can provide an initial "foot hold" from which substantial disease spread can occur during ripening.

Because relatively few early infections typically do become active and turn into rot, controlling them at bloom provides only modest benefit if the post-veraison season is dry and doesn't support further disease spread. However, early control can be critical in a year with a wet preharvest period, which favors both the increased activation of early-season latent infections and their rapid spread subsequently. So in one sense, bloom sprays are an insurance policy against the unknown weather 2 to 3 months later. Sometimes they pay large dividends, sometimes not (data below). What's your risk (and tolerance of it) from not being insured? How good is your crystal ball?

• The pronounced impact that cluster compaction has on Botrytis development appears to be due largely to its effect on the berry-to-berry spread that occurs at the point of their contact with one another (Fig. 18). In one experiment with a tight-clustered Pinot noir clone, a single diseased berry in a cluster, produced (by inoculation) to first show symptoms 2.5 weeks after veraison, spread the disease to over 50 (!) berries in that same cluster by harvest. In contrast, spread was reduced by 90% (!!) in the same panel of vines where a single diseased berry was produced in clusters that had been loosened to look like a bunch of table grapes, by removing some berries by hand immediately after set (Fig. 19). Note that this single diseased berry per cluster was produced in order to simulate the post-veraison activation of just one latent infection initiated at bloom, and vividly illustrates the particular importance of controlling blossom infections on tight-clustered cultivars and clones, so that they can't serve as a source of pre-harvest spread.



Fig. 18. Berry-to-berry spread of Botrytis via contact within a tight cluster of grapes.

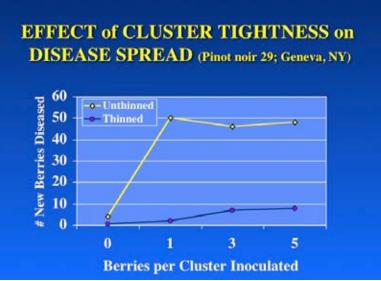


Fig. 19. Spread of Botrytis through natural tight clusters of Pinot noir clone 29, versus spread through clusters that had been thinned via selective berry removal after set. In both treatments, either 0, 1, 3, or 5 berries per cluster were inoculated with Botrytis 10 days after veraison, producing individual disease "sources" 1 week after that. Data show the number of additional berries to which the disease spread from these initial sources by harvest.

Loosening clusters by hand thinning was possible on a small scale in a research trial to demonstrate a principle, but unfortunately there are few practical, foolproof ways of achieving the same effect on a commercial scale other than through clonal and varietal selection. The watchword here is foolproof. Over the years, several workers have experimented with prebloom sprays of gibberellic acid for this purpose, with some success. (Most recently, Bryan Hed and colleagues at Penn State have published an in-depth paper on their positive results using this technique with Chardonnay and Vignoles). And there are now some GA formulations (e.g., ProGibb 4%, which is even OMRI approved) that are labeled for use on wine grapes. These labels contain warnings about possible yield reductions during the current and/or following years and a range of rates specific to different varieties. It's 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. Those instances are less interesting and get less press.

IMHO, loosening cluster compactness represents the "Holy Grail" for Botrytis management, and although there have been sightings, I don't think that it's been found yet. GA treatments very well may have their place but this technology is not foolproof, as we do not have all the answers and there are risks involved. Several researchers, including Bryan, have had some success with pre-bloom leaf removal around clusters (concept: starve them for photosynthates and the clusters 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 still view those just mentioned as experimental techniques 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.

• There is no single "correct" timing regimen for fungicide applications in a Botrytis management program. The standard "full" program used in fungicide trials, provided on many fungicide labels, and employed by some growers of highly susceptible and valuable cultivars consists of four sprays: at bloom (to limit the initiation of latent infections), bunch closure (last chance to cover the entire berry surfaces with spray), veraison (start of increasing susceptibility), and 2-3 weeks pre-harvest (start of the period of maximum susceptibility, with a consequent potential for rapid spread).

We have looked at the relative contributions of the two early sprays, the two late sprays, or all four in most years since the mid-1990's; a summary of these results is presented in Figure 20, where data have been standardized to show the percent control provided each year compared to the unsprayed vines in that same experiment. Note that in some years, the two early sprays provided better control than the two later sprays. In an equivalent number of seasons, the opposite was true. In some years, two early sprays OR two late sprays provided the same control as all four; but in some (especially 2008!), applying all four provided the best results.

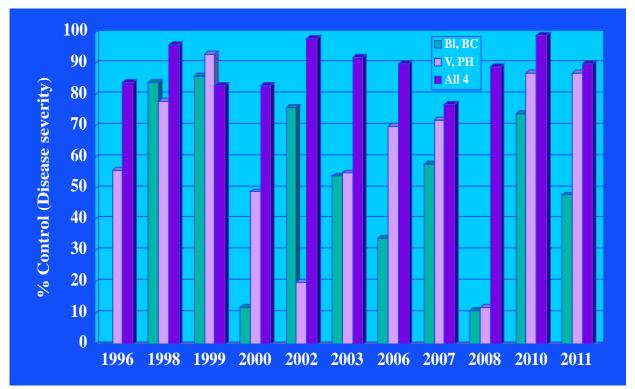


Fig. 20. Influence of spray timing on the control of Botrytis bunch rot in Geneva, NY (cv. Aurore, 1996-2000; cv. Vignoles, 2002-2011). Sprays were applied at (i) Bloom + bunch closure (Bl, BC); (ii) Veraison and 2-3 wk later (Ve, PH); or (iii) at all four of these stages. Data are expressed as percent reduction of diseased berries relative to vines receiving no Botrytis fungicides.

The relative benefits of early versus late applications, and the total number necessary, will vary among years according to rainfall patterns and, quite likely, differences between cultivars and clones (e.g., cluster tightness). Think in general terms as early sprays limiting the establishment of primary infections and later sprays as limiting disease spread.

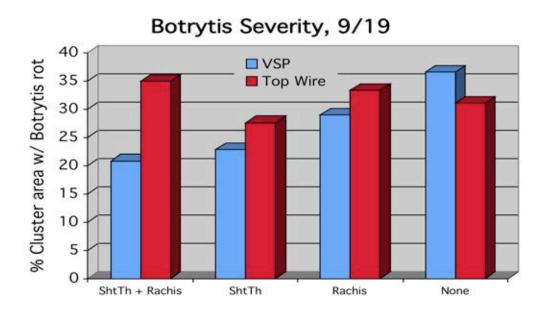
But remember: Botrytis is not a disease that you can just "spray your way out of". Botrytis fungicides can help, but they won't do the job by themselves in a tough block and/or tough year if you don't give them a hand with appropriate cultural practices (canopy management, leaf pulling, etc.).

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

The treatments involved were:

- Training system (Top Wire Cordon 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 Botrytis inoculum) at the time of thinning versus no removal

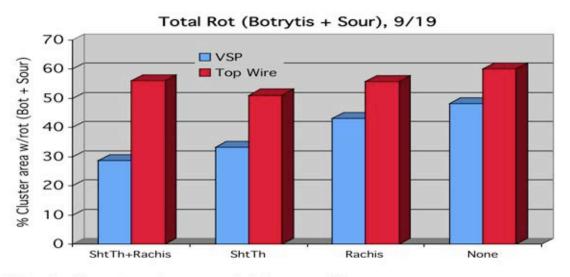
The grower managed the vineyard via his standard practices, which included a Botrytis spray regimen. We rated the plots for both Botrytis and sour rot levels at harvest on September 19; the VSP treatment also was rated 10 days pre-harvest. A few sets of data and interpretations/notations are provided in Figs. 21-24 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

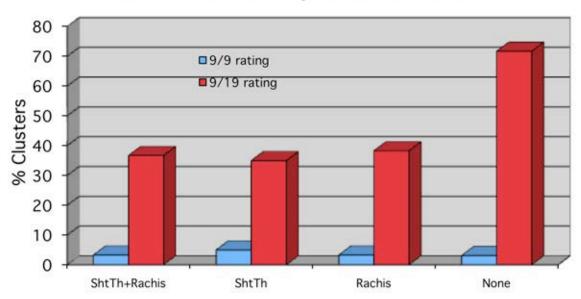
Fig. 21. Average severity of Botrytis at harvest on Vignoles clusters from vines trained to either a VSP or top-wire cordon system and subjected to three different early-season canopy management treatments (or none).



· 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 <u>and</u> thinning shoots + removing rachises reduced rot by 52% relative to
 the TW check

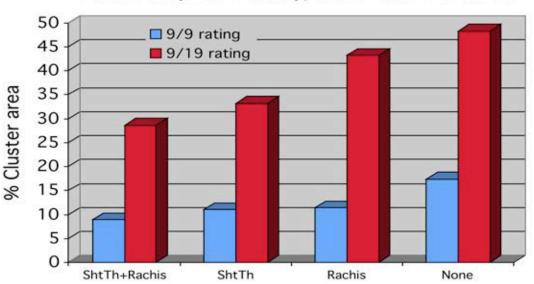
Fig. 22. Total rot severity (Botrytis + sour rot) at harvest on Vignoles clusters from vines trained to either a VSP or top-wire cordon system and subjected to three different early-season canopy management treatments (or none).



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

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

Fig. 23. 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).



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

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

Fig. 24. Increase in the total rot severity (Botrytis + sour rot) 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).

2. *Fungicides, physical modes of action.* Over a several-year period, 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** berry tissue against infection from spores that might be deposited inside them following mechanical damage, such as that from rain cracking, berry moth larva feeding, etc. Chardonnay clusters were sprayed at pea-sized berries, bunch closure and veraison, then a hypodermic needle was used to inject berries with Botrytis spores 2 weeks after the last spray. Scala, 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 <u>within</u> 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 as a surface protectant. However, this turns out to be a function of marketing strategy rather than (contrary to) fact.

• Similarly, the same three materials provided very good curative activity against latent infections initiated at bloom, even when applied 2 months after infection. Nevertheless, as shown in Figure 20, 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 curative.

SOUR ROT: THE BASICS AND NEW 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 (um, there's a reason they call it <u>sour</u> rot). The characteristic visual symptom is a tan to occasionally reddish discoloration of the rotted berries, which eventually break down, and no moldy growth need be apparent (Figs. 25 and 26). Although various molds, including Botrytis, are sometimes found on sour-rotted clusters (Fig. 27), these organisms are not necessary for sour rot to develop. Although some potential role for them in specific cases cannot yet be ruled out, they often appear to be "hitchhikers" or competitors for the same food source (and favored by the same wet conditions) as the sour rot yeasts and bacteria, which basically are not visible to the naked eye. One additional group of organisms characteristically associated with sour-rotted clusters, both highly visible and apparently an important if not essential causal element of the disease, are *Drosophila* fruit flies (Fig. 28), as discussed later.

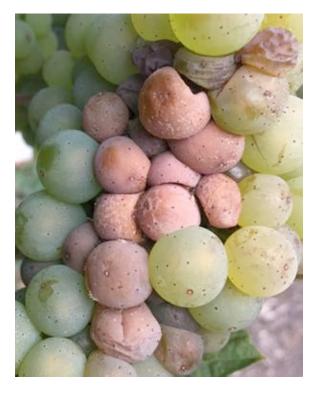


Fig. 25. Pre-harvest sour rot on cv. Riesling. Note almost complete lack of mold growth. (Courtesy M. Hall)



Fig. 26. Pre-harvest sour rot on cv. Riesling. Note complete lack of mold growth and breakdown of berries. (Courtesy M. Hall)



Fig. 27. Pre-harvest sour rot on cv. Riesling. Note coincidental presence of additional "hitchhiker" or secondary mold fungi. (Courtesy M. Hall)



Fig. 28. Pre-harvest sour rot on cv. Riesling. Note lack of mold growth and presence of numerous *Drosophila* fruit flies (arrows). (Courtesy M. Hall)

Winemakers often refer to and measure the cause of this vinegar smell (acetic acid) as volatile acidity (VA). Dr. Wendy McFadden-Smith at OMAFRA on Ontario's Niagara peninsula--whose work over close to the last decade took sour rot out of the "black box" category and started giving us a handle on what makes the disease tick--has shown that the measure of VA in grapes harvested from different vineyards is strongly associated with the pre-harvest proportion of clusters with sour rot symptoms. 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 wounds (birds, rain cracking, berry moth, compression in tight bunches, powdery mildew damage, etc.) are necessary to get the whole process started.

Sometimes these bacterial infections are accompanied or followed by infections by several wild "bad" yeasts, which can produce ethyl acetate (smells like nail polish remover or varnish), although this symptom does not seem to be typical. A far more important contribution of yeasts, broadly speaking, has been shown recently by Cornell grad student Megan Hall, still laboring under the yoke of entomologist Greg Loeb and yours truly. Specifically, after collecting numerous samples of sour rotted clusters from vineyards throughout the Finger Lakes and elsewhere, Megan always found large populations of "good" *Saccharomyces* yeasts associated with them, and significant concentrations of ethanol as well as acetic acid. Which isn't so surprising in retrospect, since ethanol is the substrate that the abovementioned bacteria convert to acetic acid, i.e., it's the precursor. 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

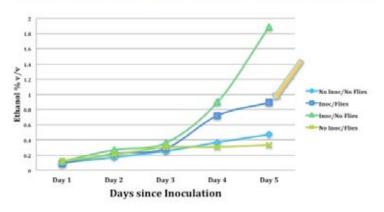
the grape's juice to ethanol and the bacteria that subsequently convert this into acetic acid (yeasts and bacteria each require wounds or natural openings to gain entrance into plant organs).

To my mind, two of the more important things that Wendy and her 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. This is certainly consistent with local observations and anecdotal reports from elsewhere that sour rot is worse under relatively warm conditions, but now we have some concrete numbers to go by.

The Ontario contingent has 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).

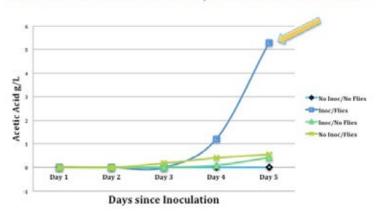
The role of Drosophila *fruit flies*. Clusters with sour rot usually are swarming with fruit flies (note that these insects are also called "vinegar flies" sometimes). A prominent line of thinking over the years has been that these flies are opportunists coming to feed on a convenient food source; indeed, they are attracted to the smell of both acetic acid and ethanol. However, a study from Portugal published in 2012, while far from conclusive, suggested that the flies may actually play a direct role in the initiation and/or spread of the disease. Thus, when Megan began her study the next year, she incorporated fruit flies into it. She quickly found that the actual species of fruit fly didn't really matter, i.e., the Spotted Wing Drosophila and the far more common "everyday" fruit fly (*D. melogaster*) have the same effect, and that effect is huge. Indeed, when Megan has tried to reproduce sour rot on grape berries in the lab, she only gets the whole range of field symptoms when she includes fruit flies with her inoculations.

The results from one illustrative experiment are shown in the two figures below. 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 (iii) introduced *D. melanogaster* fruit flies into the dishes at the same time, or (iv) omitted fruit flies from them. Then, she measured ethanol and acetic acid accumulation on each of the next 5 days. As shown in Fig. 29, ethanol began to accumulate significantly by Day 4 in the inoculated berries, with or without flies. After another 24 hr (Day 5), ethanol accumulation doubled in the inoculated treatment without flies but there was little additional accumulation in the inoculated treatment with flies (arrow). Why? As shown in Fig. 30, ethanol was not being converted to acetic acid if the flies were not also present, whereas it was in the presence of flies (arrow). We have a couple of theories as to the reason(s) behind this and are trying to test them, but the bottom-line effect is clear: fruit flies are a cause, not just a result, of sour rot development, so controlling them might help to control the disease.



Ethanol Accumulation w/in Inoculated Berries

Fig. 29. 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.

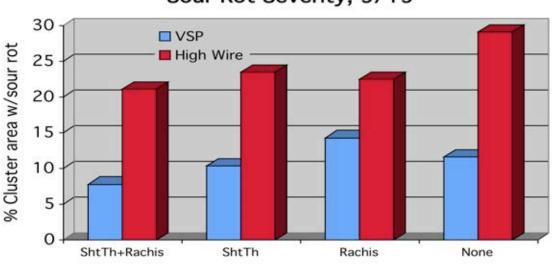


Acetic Acid Accumulation w/in Inoculated Berries

Fig. 30. 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.

Disease management. Given all of the above, logical management strategies for sour rot are: (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 fruit flies.

Canopy microclimate. Before starting our new sour rot study, there was the opportunity to measure the effect of canopy management on this disease as part of the 2011 Vignoles study discussed above in the Botrytis section. The effects were pretty dramatic, as shown in Fig. 31 below.



Sour Rot Severity, 9/19

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: <u>best treatment</u> = Shoot Thin + Rachis Removal/VSP (7.8%), <u>worst treatment</u> = Check/Top Wire (29.1%)

Fig. 31. Average sour rot severity at harvest in 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).

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 pronounced once again, with twice as much disease with Top Wire training versus VSP. (Likely reason: with the Top Wire system, the vigorous shoots droop almost to the ground, essentially enclosing the clusters within a "tent" of leaves). The data are presented below in Fig. 32.

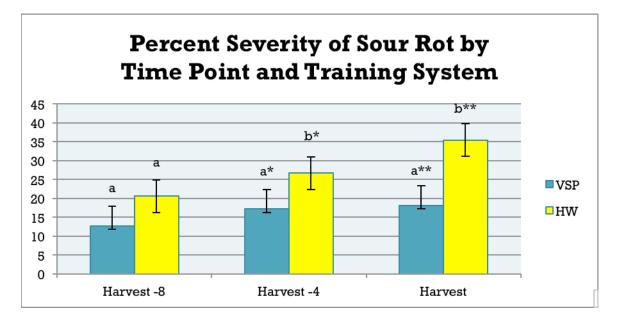
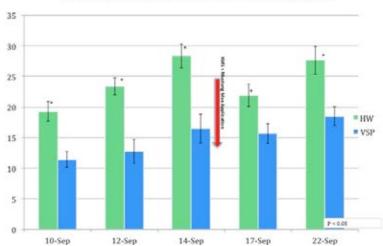


Figure 32. Effect of training system (VSP and Top Wire [HW]) on the development of sour rot in a commercial vineyard of cv. Vignoles, Finger Lakes NY, 2014. Disease severity represents the average percent of the cluster area affected with sour rot, assessed on the day of harvest plus 4 and 8 days before.

Megan went back to this vineyard last year and found the same effect a third time running. As shown in Fig. 33, by 8 days pre-harvest (14 Sep) nearly 30% of the berries in the top-wire system had sour rot whereas a little more than half that many were diseased in the VSP vines right next to them. At that point, the grower sprayed a labeled insecticide active against fruit flies (Mustang Maxx) and a labeled antimicrobial, Oxidate, after which further disease progression essentially stopped.



2015, % Sour Rot Severity by Time and Training System

Figure 33. Effect of training system (VSP and Top 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 over 12 days before harvest on 22 September. Note the effect on disease progression after the grower applied an insecticide (Mustang Maxx) and antimicrobial (Oxidate) after the 14 Sep assessment.

Minimize injury. Beyond the obvious (do what you can to reduce damage from birds, berry moth, powdery mildew, etc.), loosening clusters is likely to reduce mechanical injuries due to compaction, and will also go a long way toward reducing sour rot just as it does for Botrytis. Refer to the discussion on cluster loosening in the Botrytis section above, if so inclined. Calcium sprays to "toughen" the grape skins haven't reduced sour rot development when tried by Wendy et al., nor have Raingard or calcium chloride sprays applied as anti-cracking treatments. She's had pretty good results with a plant growth regulator that's used on apples, but we'll never see it on grapes in the U.S.

Minimize the pathogen population. A number of antimicrobial sprays tried in Ontario did not have any effect on sour rot development: Serenade, Pristine, vermicompost, potassium bicarbonate (e.g., Milstop, Armicarb). But what did reduce sour rot on a regular basis was potassium metabisulfite ("KMS", in shorthand), applied weekly at a rate of either 0.5 or 1.0% (4 or 8 lb per 100 gallons of water, respectively). We've also seen a benefit from KMS in two field trials, and from Oxidate and Fracture (both labeled) in the one year we've looked at those materials. It must be stressed that whereas 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. Which means that it's illegal to apply it for that purpose. Also, it is nasty stuff if you get it in your eyes or inhale it.

Control fruit flies. Our lab results keep showing that fruit flies are a critical component of the complex that causes sour rot, and the two good field trials we've run both showed significantly less disease when we controlled the flies. And whereas we did not have an unsprayed comparison in the commercial Vignoles block shown in Fig. 33 above, it sure looks like that spray accomplished something. Nobody wants to spray an insecticide close to harvest unnecessarily nor to recommend that others do so, but I know what I'd do if I saw this year's crop starting to melt before my eyes from sour rot and I wasn't ready to harvest immediately.

2013 and 2015 trial results ('14 was a bust, wrecked by hail). We looked at a combination of insecticide and antimicrobial sprays in a Vignoles vineyard at Geneva. Alternate rows were sprayed with the insecticide Delegate (2013) or Mustang Maxx (2015) weekly beginning at 15° Brix, with the remaining rows receiving no insecticide. Then, within these insecticide-plus or - minus rows, we applied various antimicrobial treatments, also on a weekly schedule. These included KMS (0.5% or 1.0%), Kocide at 2 lb/A (2013 only), Oxidate (2015 only), and Fracture (2015 only). Most antimicrobial treatments began at 15° Brix, before symptoms were present, but a few were either "rescue" treatments applied as soon as symptoms were seen or one-time shots.

As shown in Fig. 34 below, in 2013 the antimicrobial treatments applied **with** insecticide provided an average of 50% control (vs. untreated check); antimicrobials **without** insecticide provided an average of 9% control; and insecticide without antimicrobials provided 15% control.

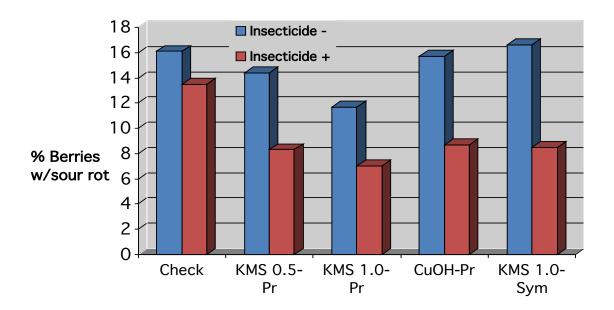
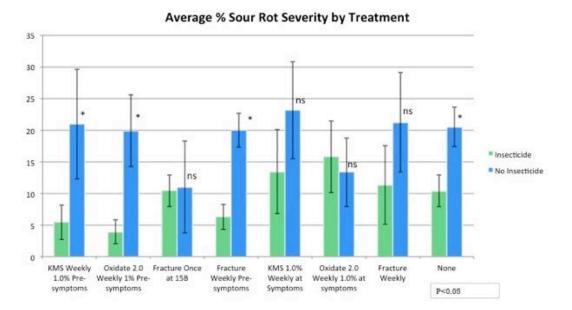


Figure 34. The effect of antimicrobial and insecticide (Delegate) sprays on sour rot control in an experimental 'Vignoles' vineyard; Geneva, NY 2013. Kocide (CuOH) at 2 lb/A or potassium metabilsulfite (KMS) in 0.5% or

1.0% solutions were applied at weekly intervals either preventatively (Pr) beginning at 15° Brix or after symptoms first appeared (Sym) = 1 week after 15° Brix. Delegate was applied at weekly intervals beginning at 15° Brix.

In 2015, the insecticide application itself has a pronounced 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. 35). Due to the logistics of the experimental set-up, we unfortunately did not have a treatment where insecticide + antimicrobial was not applied until symptoms first appeared.



Control Trial 2015

Figure 35. The effect of antimicrobial and insecticide (Mustang Maxx) sprays on sour rot control in an experimental 'Vignoles' vineyard; Geneva, NY 2015. Potassium metabilsulfite (KMS) in a 1.0% solution or labeled rates of Oxidate or Fracture 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. Mustang Maxx was applied at weekly intervals beginning at 15° Brix.

A few parting thoughts:

• The old saying that "the devil is in the details" is as appropriate here as it is in most cases, and we're still missing a lot of details. That being said, I believe that we're getting a handle on this disease and some general concepts about its management beyond hoping for

the bet and harvesting as quickly as possible once it takes off. Because bacteria are a critical part of the complex and we haven't seen any consistent association with "filamentous" (non-yeast) fungi, I wouldn't expect most fungicides to provide much benefit in our region or those with similar climates other than perhaps reducing the number of certain injury sites, although materials that significantly reduce yeast populations could possibly have an impact on sour rot development. In warmer climates such as California, Texas, and South Australia (and the mid-Atlantic, as new Univ. of Maryland pathologist Dr. Cass Swett is starting to find out), species of the *Aspergillus* fungus often are associated with sour rot, but what causal role they may or may not play in the whole complex is still not entirely clear.

• Our two field trials were 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, before the disease becomes visible, and 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. 33 above. 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 it is still a lot more sketchy than for most of our other important diseases. Individual growers will approach managing it differently depending on their own individual risk as they understand it, based to some extent on previous experience, 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; 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 economically and practically is the \$64,000 question (a term originally coined in 1950's currency!).

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 put something on to help control the

fruit flies and responsible microbes. If I wanted to stay both cheap and legal, I might concentrate just on the fruit flies. If the weather was warm and wet and looking to stay that way for a bit, especially if 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. Otherwise, I'd probably keep a very close eye on my vineyards and the weather, and be ready to jump in if I saw the disease starting and the weather looked conducive for its spread.

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 last year's trial, 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 looked good in our one trial last year, but as noted at the very beginning of this tome many pages ago, it's purported mode of action should preclude activity against bacteria, so I'd like to remain a little cautious until I see it work again. 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.

"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 <u>covered</u> with <u>numerous</u> 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 Guildelines. And there are photos in the Compendium.

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. 36.



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 <u>must</u> be followed.

PHOMOPSIS (Ph) NEWS AND REMINDERS

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. 37), but the fungus also seems to move readily from the pedicel (berry stem) into individual fruit as they ripen, especially on Niagaras (Fig. 38).



Figure 37. 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 38. 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. 39). Without shoot infections to begin with, there would be no spores to cause rachis and fruit infections later on.



Figure 39. 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 some years ago now in a problem block of Niagaras, 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 (Table 4). 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.

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.

CONTROL ON YIELI	D (cv. Ninga	rad Fredenia, N
	Yield (tons/A)	
Phomopsis program	2006	2008
None		13.2
Mancozeb, 1x	10.0	15.5
Mancozeb, 3x	10.8	16.4

Table 4. 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 2 weeks 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.

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. 39). 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.

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 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 seasonthe 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. 40). 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. 41). Infected berries develop spots approximately ¼-inch in diameter, often with whitish-gray centers surrounded by reddish brown to black margins (Fig. 42); 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. 40. Anthracnose lesions on leaves.



Fig. 41. Anthracnose lesions on a young shoot.



Fig. 42. 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. 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 than it is in NY; however, infection <u>can</u> 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 rapidly spread the disease through multiple repeating cycles of additional new infections and 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. Early-season sprays of mancozeb, captan, or ziram targeted against Phomopsis also provide significant control of anthracnose, although this latter disease is not listed as a target on most labels. 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. 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 on the radar of eastern grape growers for many years; in fact, it has been 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 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. Most vinifera and some hybrid grape growers provide a measure of control simply through the common practice of systematic trunk renewal, replacing diseased wood on a regular basis in the process and thereby limiting the girdling effect of slowlyexpanding cankers and the attendant 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 [infamous?;)] 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 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 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 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 freshlymade 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 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 quicklybasically, 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 (the Californians have a SLN label to use Rally for this purpose), but there are biological and engineering questions to answer-not to mention labeling issues to attend to-before we know whether this approach might be a worthwhile options under certain eastern conditions. Finding answers to these and related questions will require a longterm research project that someone younger than I will need to undertake, 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 are <u>some</u> considerations among the many possible alternatives. 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) <u>may</u> 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. <u>Option A</u>: Nothing. <u>Option B</u>: Captan, mancozeb, or ziram. The best one of these is whichever is cheapest and most convenient.

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.

Blocks susceptible to Anth need protection now.

Now is the time to start thinking about control of **PM** on *vinifera* varieties if temperatures remain above 50°F for long stretches of the day. 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 early sprays <u>can</u> become. It's still too early for **DM** in NY and similar climes.

Option A: Nothing. Option B: Mancozeb or ziram (BR, Ph, Anth). Option 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. Option 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 7+ days (depending on temps since then). Option 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 ¹/₂-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 one. Can mix with captan as well as mancozeb and ziram. Option G: Rally, tebuconazole generics, Mettle, Rhyme [not in NY] (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 timewise accordingly. The DM protection provided by Revus Top is not likely to be necessary yet. Option H: Serenade, Sonata, Regalia, Double Nickel, Oso, or Ph-D (PM) if you want to experiment with biopesticide products while disease pressure is low. Option I: One of the PM products plus mancozeb, ziram, or captan for Ph, BR, DM, and Anth.

10-INCH SHOOT GROWTH. We once recommend not waiting any later than this to control **BR**. Continued experience tells us that we can get way with withholding a BR spray at this time under most commercial conditions in NY <u>unless</u> this disease was a problem last year (inoculum levels are high) and weather is wet. Although if you're targeting other diseases now, there's no harm in picking up this one along the way. DO NOT wait any later than now to control **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. 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 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, you need to. Or if weather conditions are forecast to particularly favor PM for a while (moderately warm temps, cloudy skies, no cold nights), you might need to. 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. It's also one of the best times to use an oil 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. DM control should be provided on highly susceptible varieties, especially if disease was prevalent the last year or two and rains of at least 0.1 inches at temps >52°F are anticipated or have occurred recently. Rachis and fruit infections by **Ph** are still a danger in wet years, particularly in blocks with some history of the disease. Anth is in season and should be controlled by growers for whom this is a concern.

Option 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). Option 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 well last year (limited inoculum) and good BR materials will be used in the next three sprays. Include something for PM where needed. Option C: 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. Option 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. 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. Option E: Quadris Top or Topguard EQ (PM, BR, Anth). Have slight edge over Revus Top for BR and possibly PM (esp. Quadris Top), but no dependable DM control. Option 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. Option 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. Option H: Rally. tebuconazole generics, Mettle, Rhyme [not NY] (PM, BR, Anth). All are Group 3 (DMI) fungicides equivalent to Revus Top against BR and Anth, somewhat less effective against PM. And no DM, of course. Option I: JMS Stylet Oil (PM). If (and only *IF*) coverage is thorough, this spray should eradicate early PM colonies that may have started, especially previous PM sprays have been omitted or incompletely applied. But don't waste your money if you can't cover thoroughly. Also may help with mites. Will provide a few days 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) generally are a bit less effective. 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. Option J: Serenade, Sonata, Regalia, or Double Nickel (PM) if you want to experiment with OMRIcertified 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. Option K: A PMspecific 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, DM, Ph, and Anth on the fruit! Just starting to enter Bot season, also. This and the first postbloom spray are the most critical sprays of the entire season-USE EFFECTIVE MATERIALS AND DON'T CHEAT ON RATES, SPRAY INTERVALS, OR COVERAGE!! Option A: Vivando or Quintec (or Luna Experience or Aprovia outside NY) for PM control, plus mancozeb for BR, DM, and Ph, and Anth. Note that Luna Experience also provides control of Bot and BR, depending on rate. (For reference, the 6 fl oz/A rate of Luna Experience provides about 70% as much tebuconazole--the component in this mix that's active against BR--compared to the labeled rates of the various generic tebuconazole products or the former product, Elite; 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, respectively). And as noted previously, Aprovia is labeled for BR control but it's not as effective as the standard BR products and I wouldn't count on it under pressure. Also as previously mentioned, Vivando and Quintec, two completely unrelated fungicides, are both Cadillac materials for PM control. And now's the time that you want such materials on highly susceptible cultivars. (Luna Experience and Aprovia are right up there, too). 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 a few rumors elsewhere, but it's been an excellent performer in our trials and in commercial usage by and large. Let's keep usage down to a maximum of 2 applications per year for each of these so that they remain effective. Option B: Revus Top (PM, BR, DM, Anth). I can't overemphasize the fact that the very good to excellent 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 it--<u>especially on the clusters!</u>--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 Inspire Super doesn't provide DM control and Quadris Top shouldn't be relied upon to do so due to strobie resistance concerns. I wouldn't use either one of these without adding something like mancozeb for DM.

Option C. 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, 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. Option D: Quadris Top or Topguard EQ (PM, BR, anth). These have a slight edge over Revus Top for BR and possibly PM (esp. Quadris Top), but no dependable DM control so add mancozeb or another DM fungicide. Option E: Rally, tebuconazole generics, Mettle, or Rhyme [not in NY] (PM, BR, anth) PLUS mancozeb (DM, BR, Ph, Anth) or captan (DM, Ph, Anth). Personally, I'd choose this option only if I couldn't use difenoconazole as a DMI. One of the new DM-specific fungicides such as Zampro (outside NY) 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, 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. 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).

<u>Option F:</u> Mancozeb + sulfur (PM, BR, Ph, DM, Anth). Relatively economical and effective, particularly if used at shorter spray intervals. 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. <u>Option G:</u> Zampro (except in NY) 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 when 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), Scala, Elevate, Flint (3 oz rate was once effective, the extent of compromise due to resistance is a concern), Endura,

Pristine, Rovral/Meteor/iprodione generic, or Luna Experience [not yet in NY] applied around the bloom period often provide beneficial control of this disease on susceptible varieties, particularly in wet years. It's certainly easier to use or include one of them for Botrytis purposes in the immediate prebloom/early bloom or subsequent first postbloom spray when other diseases are being targeted as well 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 these timings rather than separately at "full bloom"; however, we've never directly compared these two approaches. One problem with tank-mixing Botrytis-specific materials like the AP's 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.

<u>Also</u>, if sulfur was the only PM material in the most recent (immediate pre-bloom/early bloom) spray, reapply about now on highly susceptible *viniferas*. That is, keep the spray interval 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 that may follow upon the "diffuse" PM infections that can develop on berries during this period of 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 such crop size/ripening conditions don't apply, 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. 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 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 to avoid infection as weather dictates, 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.

Option 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. Can cause injury on Concords and some other natives and hybrids. Option 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 control of Bot and BR, depending on rate; see text above 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 both Luna Experience and Aprovia) to two 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). Logical BR options to complement PMspecific fungicides include mancozeb (if still within the 66-day PHI limit), ziram, or one of the strobies. 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 zirma). The DMI (Group 3) materials (Rally, Mettle, tebuconazole generics, various difenoconazole products) also provide excellent control of BR (plus Anth), but using these in addition to top-shelf PM products seems like overkill with respect to this disease unless you really need it and the price is right. DM options include mancozeb (ziram is only fair), captan, Zampro (but not in NY), Presidio (get a 2nd mortgage), Ranman, the phosphonates, and copper. Option C: Torino for very good PM control + an appropriate material for DM, BR, and/or Bot as necessary. Provides protective plus postinfection activity and is unrelated to any other fungicide on the market, so a good rotational partner. Option 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. Option E: Pristine, Abound, Sovran, Quadris Top, Topguard EO, or Flint. Should work well against BR and PM (Pristine, Quadris Top, probably Topguard EQ), anth, and bitter/ripe rot, might work against DM but don't count on it. Pay your money and take your chances; tank-mix with something for DM control unless it isn't an issue. Pristine and Flint also can provide good Botrytis control at appropriate rates (Flint/strobie resistance becoming a problem?). Option F: Rally, tebuconazole generics, Mettle, or Rhyme [not in NY] for fair to

very good PM (depending on rate, resistance status of vineyard, cultivar) and excellent BR (and anth) + something for DM. <u>Option G</u>: Copper + lime for DM, some PM. 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 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.

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. 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. 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 such as Nutrol, Kaligreen, Armicarb, Regalia, Oxidate, Serenade, Sonata, Double Nicket. 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. consider "insurance" application against **Botrytis** Strongly an 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!



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