LAKE ERIE REGIONAL GRAPE PROGRAM-Vineyard Notes May 2015

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Grape Disease Control 2015

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Upcoming Events:

Wednesday, May 13, 2015- Coffee Pot meeting Philip Baideme Farm, 7935 Route 5, Westfield, NY 14787

Friday, June 26 & Saturday, June 27, 2015- Hops Conference at CLEREL *(see flyer and registration form)*

Sunday, July 26, 2015- ISHS Shaulis Symposium at SUNY Fredonia

Monday, July 27-Wednesday, July 29- ISHS Conference at SUNY Fredonia

Use the included forms, go to our web-site or stop in the office to register.

**Check the web-site for more upcoming events and meetings.











GRAPE DISEASE CONTROL, 2015

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After a 1-year hiatus, it's 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, Bob Seem, Lance-Cadle-Davidson); research technicians (including the phoenix-like Duane Riegel, Dave Combs, and Judy Burr); and graduate students and post-docs too numerous to mention here. It truly is the combined research efforts of all of these people that serve as the basis for most of the following.

THE WINTER FROM HELL

We won't dwell upon it, but there are going to be some consequences this coming season as a result of our recently-concluded (or so it's starting to appear) WFH. It's beyond the scope of this screed to discuss and sometimes lament many of the more obvious issues, but there are a couple of disease-related points that are worth covering briefly:

• *Phomopsis infection of new suckers*. This issue falls into the "observation/speculation" category rather than one that contains relatively well-established facts. That being said, the last time (2004) that upstate NY had winter temperatures that killed top wood in a significant number of locations, I (and others) noticed that sucker growth destined to become new trunks developed an awful lot of Phomopsis infection during the early growing season.

<u>Here's the speculation</u>: The Phomopsis fungus is something of a "necrotroph", i.e., it is particularly good at forming spores when it grows on dead stuff. (For example, note that when berry infections occur, the fungus doesn't form its spore bodies until it has spread throughout the entire berry and turned it brown). Thus, it is <u>possible</u> that Phomopsis infections already established within the canopy will be stimulated to produce additional spores if that wood is killed. Indeed, we found some evidence years ago that infected dead cane segments formed significantly more spores than did infected live segments. And it is <u>certain</u> that whatever Phomopsis spores are produced will be distributed by rain drops (that's how they work), and that those are subject to gravity. So new suckers developing beneath a source of Phomopsis spores couldn't be in a worse place.

<u>How big a problem is this?</u> How long is a piece of string? Obviously, it all depends: degree of winter injury, importance of suckers for trunk renewal, level of Phomopsis already present within the canopy (levels of disease development in previous years), the number and duration of rain events in up through bloom (Phomopsis spores are largely gone after that). This won't be an issue for many people but it might be for some. At the very least, be aware that if you have a significant amount of dead wood on the wires and you plan on training up new suckers, it would behoove you to get that wood off the wires (so that spores don't rain down on new growth) sooner rather than later. I have no data or experience as to the economics and/or advisability of

spraying to protect against such infections. If a combination of disease pressure and risk aversion compels you to consider this option, one approach would be to spot-spray individual groups of suckers as you would for post-emergent weeds (but first, think about what was in the sprayer the last time that you used it!), applying a low per-acre rate of captan or mancozeb in 100 gallons of water (e.g., 2 lb of mancozeb 75DF per 100 gallons).

• *Crown gall*. Recall that the crown gall bacterium is present within the tissues of just about every grapevine out there. It doesn't do any harm most of the time, but when a grapevine cell is injured (e.g., by freeze damage), the bacterium is able to insert a portion of its DNA into that of the injured plant cell and direct it to start producing tumors (galls). Which means that we'll probably be seeing more than average levels of crown gall this summer, once vines have had a chance to make some normal and abnormal growth. And of course, most galls are found on the vine trunk where woody tissue is injured but not killed outright.

There's not really a lot that you can do about this disease once it develops, other than prune out trunks that are diseased beyond the point of being productive even when they're babied, then replace them with renewals (see the Phomopsis/sucker discussion above). But consider this a heads-up.

• *Inoculum reduction*. You don't sell heating fuel but you still hoped that there might be a silver lining to our Siberian experience? Sorry, not on this account. The fungi that cause our common grape diseases have seen cold weather before—after all, downy mildew, powdery mildew, and black rot all evolved here. They're adapted to it. The amount of disease that you had in a vineyard block last year (or years, in the case of Phomopsis and wood canker diseases) will have almost infinitely more influence on the inoculum level that you start this year with than will our low winter temperatures.

• Wood canker diseases. Most of these, and all that have been documented in New York and locales to our south, appear to gain entry into the vine through pruning wounds. However, one fungus (*Cytospora*) that is known to infect winter-injured wood of various fruit trees in our region was also recovered from cankered vines in Quebec during a survey a few years ago. And the survey dealt lightly if at all with some of our NY regions most prone to winter injury. Just another heads-up that IF the occurrence of cankers caused by this fungus in Quebec was a result of the winter injury that can be common there, there MIGHT be the potential for an analogous scenario to develop here. Keep an eye out, that's all.

FUNGICIDE CHANGES & NEWS

New products. There have been no new blockbuster groups released recently or that are on the immediate horizon. However, there have been a few new single-disease chemistries targeted at downy or powdery mildew that have been released over the past few years, and the few major companies still standing are all coming out with their own new-and-improved "Group 7" products, the first modern one of which (boscalid, the non-strobie component of Pristine) was released over a decade ago. There have also been several new "biopesticides" (packagings of live organisms or natural chemical products produced by them in an industrial fermenter) that have received registration and hit the market. A quick rundown on some of the above.

a. *Relatively new downy mildew-specific product, Zampro.* Although Zampro received US-EPA registration in September 2012, it is still not registered in the sovereign state of New York. 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 are not absorbed well by plant tissues and consequently are strongest in a protective mode. Anecdotal observations suggest that ametoctradin has significant post-infection activity in addition to protective activity, although publicly-available data on such issues are limited.

One good thing that could be said about the 2014 growing season in the Finger Lakes region is that it provided an excellent opportunity to test the efficacy of downy mildew fungicides. We ran our trial in a block of Chardonnay vines, in which (to put pressure on the products and for convenience in scheduling with our other trials) we sprayed most treatments at 2-week intervals. Unsprayed vines were defoliated by September and were killed to the ground this winter (bummer). However, season-long sprays of both Zampro and Revus Top provided virtually complete control of downy mildew, with only single spots developing on one leaf out of every 20 to 30. No other products looked this good when applied similarly. (Note: We obviously do NOT recommend using any one product season long, but it's hard to evaluate the activity of individual products when we use them in a rotational program on the same vines).

Another part of this trial was an eye opener. One treatment consisted of a biopesticide product in rotation with two sprays of Zampro interspersed during the summer; these Zampro sprays were applied 23 days apart, with biopesticide applications before, after, and in between. And because we were a tad late in making the first spray of the biopesticide (imagine that!), we tank-mixed it with a phosphorous acid product to provide some kickback activity against any infections that might have occurred before we got our act together. In another treatment, we applied only phos acid in the first spray plus the two sprays of Zampro (June 17th and July 10th), so we could see what the non-biopesticide components of the program were contributing to the final results. Much to my surprise, vines that received only the initial spray of phos acid plus two of Zampro looked amazingly good: only one leaf in five had any infection, and on average <1% of the total leaf area was infected. By the end of the season, these vines were green and healthy, standing right next to vines that had lost their leaves after receiving various ineffective treatments. Such a use pattern most definitely falls within the "Don't try this at home" category, but these results dramatically show how active the product is. Maybe New York growers will get to use it some day—indeed, the manufacturer says they're hoping for next year.

b. *Relatively new downy mildew-specific product, Ranman.* Ranman also represents a new class of chemistry, with no other products in this group registered for use on grapes. Hence, you can rotate it with anything. It works primarily in a protective mode, and has seen some commercial use in NY, with reported success. It works primarily in a protective mode. In the abovementioned Chardonnay trial last year, a season-long program of Ranman provided 90% control of disease severity (% of the leaf area infected) versus >99% for Zampro and Revus Top. Not the best but pretty good, especially considering the pressure. Remember, we were spraying at 2-week intervals in a wet year, with unsprayed vines and others receiving ineffective

treatments scattered throughout the block, producing LOTS of inoculum to go with all of the rain.

c. *New-ish powdery mildew-specific products*. Both Vivando and Torino have been around for a few years, long enough to have just about become "standard" products (or not). To reiterate briefly: Each is a member of a new class of chemistry, so they are excellent for use in resistance-management programs and can be rotated with anything else within this context (no cross-resistance issues with other products).

<u>Vivando</u> has consistently been among the top performers in my trials for a number of years. It has protective and post-infection activity, and appears to provide meaningful vapor-phase activity (moves from treated to untreated tissues in a gaseous form).

<u>Torino</u> was registered by the US-EPA in 2012 but was not available in New York until last year. Controlled trials 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: excellent control when applied thoroughly at labeled rates 3 to 4 days after spores first land on the leaves.

Multiple field trials have shown significant activity, sometimes in the same league as the top products, sometimes a notch below. The 3.4 fl oz/A label rate is not a "rich" one that you can cut safely. This is a good to very good product, but not a "big gun". It should have utility when used within a rotational program as the label intends (maximum two applications per year), but it would not be my first choice during the bloom/early post-bloom period that's critical for cluster disease control. Its short PHI (3 days) and powdery mildew specificity (so no activity against yeasts) might make it attractive if/when sulfur use gets iffy late in the season and PM control is still desired (see below in the PM section).

d. *"Newest generation" Group 7 products.* The Group 7 (or SDHI) class of fungicides was already around way back when I was an undergraduate (yes, they had tractors and sprayers then). However, the available product had a very narrow spectrum of activity and didn't control any important grape disease. Around the turn of the millennium, boscalid (the non-strobie component of Pristine) came along, representing a new generation of this class with excellent activity against powdery mildew and Botrytis. Since then, other companies have developed their own compounds within this group. Currently, there are three newer (since boscalid) such fungicides sold in the U.S., but only one is registered on grapes: fluopyram, which provides one component of both Luna Experience (the other component is tebuconazole, the DMI once sold as Elite but now sold alone as a generic product) and Luna Tranquility (the other component is pyrimethanil, the active ingredient in Scala). Development of a fourth very active compound in this group has progressed to the stage that its manufacturer now has a commercial product name for it (a very good sign), but registration on grapes and other crops is still pending.

We've discussed Luna Experience before: it's excellent against powdery mildew and black rot (higher rate needed for BR), very good against Botrytis, and still <u>not registered in New York</u>. It also can't be used on grapes intended for any use other than wine (no residue tolerances established for these uses). Likewise, Luna Tranquility, which provides Botrytis control only, is still <u>not registered in NY</u> and can only be used on wine grapes. Rumor has it that the NY

registration issue (and restriction to wine grapes only?) might be resolved for both products before next season. Stay tuned.

e. "New" active ingredient, polyoxin-D (Oso/Tavano, 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, but in recent years been developed against certain fruit and foliage diseases also. It is registered for control of Botrytis and powdery mildew on grapevines, and is recognized as a "reduced-risk" biopesticide. 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 is now called <u>Oso</u>, but last year this same product was called <u>Tavano</u>; both may be found on the market this year, until the latter one is used up (got it?). The other formulation, sold by a different company, is called <u>Ph-D</u>. I worked with both last year. My experience with them is limited, so the following is a qualified "early returns" assessment. That being said, my impression is that they have activity but are not as strong as the best conventional standards. However, that may be good enough in some situations, as will be shown a bit below.

f. *Other new biopesticides.* As discussed in previous years, <u>Botector</u> 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 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 (e.g., mancozeb, captan, strobilurins). This is an issue that is still getting sorted out, although the company that manufacturers it provides some specific guidance on its website (do a Google search for current url, if interested). The product is OMRI certified.

<u>Regalia</u> is a preparation derived from a plant extract, which is purported to boost plant defense mechanisms. A non-toxic product that would do this well and on a regular basis against a range of pathogens is the holy grail for companies that develop and sell plant pharmaceuticals, and just as elusive. Anyone who came up with one that worked on grapes 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" defense responses, and products that claim to do this have not been blockbusters. There's a reason for that.

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 (absurdly high inoculum levels in a vineyard of super-susceptible Chardonnay) and good to very good control under more moderate conditions (reasonably high inoculum in a vineyard of the susceptible hybrid cultivar, Rosette). This is a good bottom-line result, but when it comes to the mechanism behind it, we all know that a number of materials control PM via direct contact action. Which is what I assume is happening here rather than defense induction, because we've also gotten poor control of downy mildew and Botrytis with this product. Which is OMRI certified.

<u>Double Nickel</u> is a naturally-derived fermentation product from a species of the soilborne bacterial genus *Bacillus* (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. It's fair to say that I've never worked with a live organism or a natural product produced by one that has the same level of activity as the standard synthetic fungicides used in grape production. And this becomes all the more apparent when we test various products under high disease pressure, which is basically intended to separate the stronger from the less-strong materials. But anybody who intends to use biopesticides (or at least, anyone who intends to use them and stay in business) knows that they need to be used as a component within an integrated system that stresses limiting inoculum, employs various cultural control techniques (such as good canopy management), and perhaps utilizes less-susceptible cultivars if such are available for the intended market.

Case in point: as alluded to above, last year we conducted two powdery mildew control trials: one in a 'Chardonnay' vineyard with extremely high disease pressure (99% of the surface area of the unsprayed clusters was diseased, i.e., they were complete toast); the other, in a 'Rosette' vineyard, had moderately high pressure (40% of the surface area of unsprayed clusters was diseased). As the simple 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, whereas it provided 92% control in the Rosette vineyard when applied every 14 days. Similarly, when the biopesticide Tavano was alternated with JMS Stylet Oil at 2-week intervals (both vineyards), it provided only 57% control in the Chardonnay vineyard but 97% control in the Rosette vineyard.

E	EFFECT OF	DISEASE PR	ESSURE ON
% (CONTROL (OF CLUSTER	PM SEVERITY
		<u>C1</u> 1	D

	Chardonnay	KUSCIIC
Untreated	(99)	(40)
Double Nickel	24*	92**
Tavano/JMS	57**	97**
*7-day intervals		

Strobilurin resistance and downy mildew. The strobies started failing to control downy mildew in the grape-growing regions of the mid-Atlantic and southern states nearly 10 years ago, and have pretty much run their course for this purpose in many to most of those production areas. In New York, we first lost crop due to <u>powdery</u> mildew resistance to the strobies in a number of high-value vineyards in 2002, but realized that we could still maintain control of that disease if we used these materials in mixture (e.g., Pristine), provided that we strictly limited the number of times that we sprayed them each year, with two being the recommendation. We also realized that we do not have any pre-mixed, price-competitive strobie products that contain a second ingredient active against <u>downy</u> mildew such as Pristine does for powdery, so held our breath and hoped that we could keep this class of chemistry alive against DM through limited use.

Which we largely did for another dozen years. However, I know of at least two commercial vineyards that lost crop last year due to a failure of the strobies to control downy, and they failed in my own trial vineyard as well. Some growers that I've spoken with still feel comfortable using these products in rotational programs to control downy. I don't any longer, at least not without a huge WARNING sign. In particular, I think that it's (perhaps unacceptably) risky to rely on these materials (i.e., no other DM fungicide present) for downy control during the immediate prebloom/early postbloom period when clusters are highly susceptible, especially if DM pressure is high then. And I would NEVER use them alone twice in a row during downy season, especially while clusters were susceptible.

Pristine, in particular, has been a great product for NY growers and I don't want to cry wolf about this. On the other hand I feel like we've been warned, so neither do I want to channel my boyhood hero, Alfred E. Neuman ("What, me worry?"), only to find out later that growers lost money when they could have avoided it. If you've been relying on Pristine or azoxystrobin (e.g., Quadris Top) for DM control, you have three choices: (i) keep rolling the dice and pay close attention if it looks like control is slipping; (ii) quit using them, at least while clusters are susceptible and an unexpected control failure will reduce the crop; or (iii) tank mix them with mancozeb, captan, or another DM product, at least while clusters are susceptible. Sorry to be the bearer of bad news.

SPEAKING OF FUNGICIDE RESISTANCE

As per the example above, a number of fungicides that once were highly active have lost their efficacy against certain pathogens in some vineyards as the result of those target organisms developing resistance to the materials in question. It is extremely likely that this phenomenon will continue to increase in importance into the future, as modern fungicides are almost invariably more prone to resistance development than the old traditional, multi-site products like mancozeb, captan, ziram, sulfur, copper, etc. As I try to stress every year, paying attention to basic resistance management principles and practices will be essential to sustaining the utility of virtually any new highly-active product that we are likely to see and want to use. Consider this your annual reminder.

Simply put, anything new that's going to get registered now and into the future has to be squeaky clean in the tests used to assess any possible effects against what are euphemistically termed "non-target organisms" (such as you, me, and other life forms that we don't wish to harm

beyond disease-causing fungi). For a compound to provide the ideal, extremely rare mix of being deadly to target fungi and (nearly) benign to everything else, it typically will affect only a single process in the fungal metabolism, and often just one single specific site in one fungal enzyme that's involved with it. This is the so-called "lock and key" analogy, where the fungicide molecule "key" physically fits into the fungal enzyme molecule "lock" and prevents it from functioning, thereby killing the pathogen or preventing its further development. The upside to such specific activity is that these materials are often very effective at controlling disease and quite non-toxic to (most) non-target organisms; the downside is that the fungus may only need to make a subtle change to that one enzyme "lock" so that the key no longer fits and the fungicide becomes ineffective; 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 the altered "lock". The result is that we end up with resistance to the fungicide and all related materials that also worked by fitting into this same original lock.

Within an agricultural context, fungicide resistance is a classic example of evolution, i.e., it is the result of the selection of specific individuals from within the entire pathogen population that are best able to survive and reproduce when exposed to that material. When such individuals survive and reproduce preferentially and their proportion of the population increases to the point that the fungicide no longer provides an acceptable level of disease control even when it is applied at the proper time and at the recommended rate with good spray coverage, a condition termed "practical resistance" is reached. The risk of this occurring is a function of both the fungicide itself and its biochemical mode of action (i.e., how likely is it that a simple change to the target site "lock" might occur, which doesn't harm the fungus but makes the fungicide less active or ineffective) and 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 can become a few hundred thousand in short order if the weather is favorable and there's nothing providing control of them); and (ii) which also produce large number of spores that can be widely dispersed by air currents (spread the love!). Powdery and downy mildews are prime examples on both accounts, and Botrytis isn't too far behind. In contrast, diseases at 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, 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 that is likely to perpetuate the fungus between years (berries), and a spore type that spreads the disease that is distributed only a short distance by rain splash.

Resistance to a fungicide is said to be <u>qualitative</u> (yes or no, black and white) when individuals within the pathogen population are either sensitive to the typical range of doses used in the field or are virtually immune to even 100 or 1,000 times those levels. Unless they are controlled in some other manner (e.g., applying effective, unrelated fungicides) the only thing checking their reproduction is the weather and whatever cultural techniques might be employed; thus, the population can quickly become dominated by these resistant individual, with a resultant loss of disease control, in a year that is favorable for multiple generations to develop and spread.

Which is just what happened in some NY vineyards where the strobies failed to control downy mildew in 2014 (see above) or powdery mildew in 2002.

Examples of fungicides to which qualitative resistance among grape pathogens has either occurred in 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 QoI or strobilurin fungicides (e.g., Abound, Flint, Sovran, Reason, one component of Pristine), where downy mildew resistance is common in many regions and resistance among powdery mildew populations seems to be growing now outside its NY "epicenter"; and (iii) the phenylamides (e.g., the Ridomil products and generic metalaxyl), where downy mildew resistance is common in regions throughout the world where these materials have been used more than sparingly.

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 (1 oz of the active ingredient in Fungicide A provides more control than 1 ounce of active ingredient in the related Fungicide B). In this case, the sensitivity distribution within the pathogen population shifts incrementally after repeated use of the same class of materials, with a progressively greater proportion of the fungal individuals within the population requiring progressively higher doses of any one fungicide before a given level of control is obtained.

A well-characterized example of quantitative resistance is that to the demethylation inhibiting (DMI) fungicides ("sterol inhibitors") among populations of the powdery mildew fungus, which we have been discussing for years. A recent, very practical illustration of the importance of the concept of spray "activity" within this context (a function of both the dose of a particular fungicide and its intrinsic activity) is provided by our experiences with difenoconazole, which will be reviewed slightly below.

Given the preceding, basic resistance management strategies 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 different products that have the same biochemical mode of action by the Resistance Group number that is on the front of each label for those that are considered at risk and for which other related products are also on the market (e.g., Group 11 for the QoI fungicides, Group 3 for the DMIs).

• Limit the size of the pathogen population from which you might be selecting resistant individuals, thereby limiting the potential number of resistant survivors. Basically, try to avoid using a material at high risk of resistance development as a "rescue" from a severe outbreak of the target disease. Of course, you might wonder about the wisdom of maintaining the future utility of a fungicide for a vineyard operation that possibly won't stay in business 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.

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

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

(ii) Rotate with effective, unrelated fungicides: the fewer applications of an at-risk fungicide, the less opportunity for reproduction of resistant individuals before they are controlled by something else. 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 individuals might reproduce before exposure to other, effective fungicides. 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 want to be.

(iii) Apply at-risk materials in combination with another unrelated fungicide, either through tank mixing or use of a pre-packaged product containing two or more ingredients that are active against the target organism. 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 one with a low use rate (a common problem with some 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 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 40 times more active than myclobutanil on an ounce-by-ounce basis of active ingredient. Quite simply, the population of the PM fungus in this vineyard has shifted to the point that the maximum label rate of Rally does not provide a high enough dose of myclobutanil to control enough individuals to prevent disease from developing on clusters, yet these same fungal individuals are effectively controlled by a similar dose of the more-active difenoconazole.

And don't forget, maximizing spray coverage will also maximize the dose of product 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 deposited somewhere else.

1 2	, 0	1 .		
	Leaf in	fection	Cluster in	Ifection
Treatment, rate/A*	% Leaves	% Lf area	% Clusters	% Clstr area
None	100	70.2	100	99.5
Revus Top, 7 fl oz**	64	1.7	27	3.2
Inspire Super, 16 fl oz	67	2.1	16	2.0
Inspire Super, 20 fl oz**	39	1.1	6	0.2
Rally, 5 oz	100	33.2	100	96.7
Vivando, 10 fl oz	12	0.3	12	0.4
Vivando, 15 fl oz	6	0.1	0	0.0

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.

POWDERY MILDEW (PM) NEWS AND REMINDERS

Your (almost) annual brief 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 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 published studies). But 20% of what? In the "clean" treatment (a), this number might be relatively inconsequential; in dirtier treatment (b), it's equal to the entire seasonal supply on the clean vines; and in treatment (c), it's four to five times greater than the entire seasonal supply on the clean vines.

Not surprisingly, this makes a difference—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 the same vines the year after allowing our variable foliar disease levels to develop, the resulting cluster disease severities were (a) 11%, (b) 22%, and (c) 48% (proportion of the cluster area infected) 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 fruit were formed and highly susceptible to PM. Meaning that the higher the spore level was when berries were susceptible, the less effectively the fungicide program controlled it. 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 this can be accomplished in part by sanitation procedures that remove diseased plant organs before the season begins (e.g., cankered wood, black rot mummies). And in annual production systems, it can be greatly facilitated by crop rotation. But in a perennial crop like grapes, the very best way to minimize inoculum levels at the start of one season is by minimizing disease development the previous year, by implementing good control programs. This is a major reason that some blocks are almost always clean and some are almost always otherwise, i.e., it's a virtuous or 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 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 about 85% RH. Although there is no practical threshold level necessary for the disease--PM develops to some extent over the entire range of humidities that we experience 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 and fruit set, then become highly resistant to immune about 2 weeks (Concord) to 4 weeks

(*V. vinifera*) later. This is when you use the good stuff and don't 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 on susceptible wine grapes from pre-bloom right through bunch closing does not guarantee control of bunch rots and spoilage beasties, but it's a relatively easy way to eliminate one avenue for getting them started.

(vi) Powdery mildew is a unique disease in that the causal fungus lives almost entirely on the <u>surface</u> 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 <u>inside</u> 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 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 good protective (forward) activity in order to lengthen effective spray intervals.

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, 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 on either (a) the outer edge (exposed) or (b) inner (shaded by the vine's own canopy) portions of individual vines, which were located either (i) immediately next to or (ii) 200 feet away from these trees, thereby providing a total of four levels of natural shade. The resulting disease severity increased substantially with each increasing level of shade, becoming 8 to 40 times more severe on the most heavily shaded leaves (interior of vines next to the trees) compared to the unshaded leaves on the exterior of vines away from the trees (Fig. 1).



Figure 1. 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 next to the trees (Canopy); or (iv) shade provided by both the trees and the vine itself, i.e., leaves located within the center of the canopy of vines next to the trees (Tree & Canopy).

Although shading could potentially change air temperature or relative humidity within the vine canopy, our measurements did not show this. However, they did show that UV radiation levels and leaf temperatures were dramatically different among the different treatments. Within the shaded regions, UV levels were (as one would expect) a mere fraction of those in the sun, and temperatures of leaves in the sun 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. As noted above, 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 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 above 95°F, depending on how hot it is and for how long. On a hypothetical 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, nearby vines or portions thereof 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. However, as a disease powdery mildew is uniquely vulnerable to such damage: as noted previously, the PM fungus lives primarily on the <u>outside</u> of infected tissues, whereas nearly all other pathogens live and grow <u>within</u> 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 greater once leaf

temperatures get in to 80's and beyond. And we've just discussed the increase in leaf temperature that occurs when they are exposed to the sun.

Surface temperature and UV: Field experiments. In order to quantify the effects of each 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 2, we found that removing UV radiation while 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 eliminated both UV radiation *and* the increase in temperature caused by sun exposure, further increased disease severity in one of the two experiments.



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



Figure 4. Effect of canopy density on deposition of sprays onto clusters of 'Chardonnay' vines, provided by a conventional airblast sprayer in mid-July.

Exposure of fruit to sunlight and pesticides. It's common sense that canopy management practices that increase sunlight penetration into the fruiting zone should also increase the

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

Subsequently, Andrew Landers, Nicole Landers, and yours truly expanded this part of the study into four commercial Finger Lakes vineyards plus another experimental block that represented a range of cultivars (Chardonnay, Vignoles, Cabernet Franc, GR-7, and Rosette) and which were subjected to various canopy management practices. 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 foodgrade dye solution, applied with a Berthoud airblast unit 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 5 below.



Figure 5. Effect of canopy density (cluster exposure layers = CEL) on deposition of a spray tracer dye onto grape clusters in 5 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 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 more than twice as much spray as those separated by two layers (CEL = 2.0).

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

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, a central concept associated with quality viticulture is "balance". Zero sunlight exposure might lead to diseased berries, but absolute maximum exposure can lead to sunburned berries instead. Again, 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 is occurring, so that growers might take action to prevent damage rather than conduct a post-mortem after it's too late; or, conversely, avoid making more sprays that they need to.

To review a few highlights:

• Severe fruit infection is much more likely if the disease becomes well established on the foliage pre-bloom, providing abundant new spores to infect the adjacent fruit while they're highly susceptible. This is logical and consistent with our results showing the effects of carry-over inoculum from the year before (discussed above), but she demonstrated it very convincingly.

• Relatedly, after analyzing over 25 years worth of climate and disease severity data, Michelle showed a significant association between severe disease one season and accumulated degree days the previous autumn. This also goes back to the earlier discussion concerning formation and maturation of the overwintering fruiting bodies of the PM fungus (chasmothecia) during late summer and autumn of one growing season and disease pressure the following year. That is, a long, warm autumn allows more late-season infections (the kind that can sneak in when PM sprays are relaxed in late summer/early autumn) an opportunity to form mature chasmothecia with viable overwintering spores than does a shorter and cooler fall period, which may lead 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. Michelle found a strong relationship between PM severity in any given year and "pan evaporation" measurements during the critical prebloom through fruit set period that year. Pan evaporation is a figure reported by some weather stations that measures--surprise!--the depth of water that evaporates from an exposed pan over a given period of time (I 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.

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

• Of the two factors (pan evaporation and heat units the previous fall), the more important is pan evap. Over the quarter century of data that Michlle analyzed, our worst years for PM

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

So, what does this mean in practical terms? It appears that years (or critical periods within them, e.g., immediate prebloom through early postbloom) during which pan evap values are >6 mm/day are likely to be "light" PM years and those with values < 5.5 mm/day are likely to be killers. But where do you find pan evap data, should you want it? Some weather networks provide this and some weather stations provide a value for a related parameter called "ETO" (potential evapotranspiration); you can calculate pan evap by multiplying ETO x 1.25. Or, you can simply use the computer on top of your neck and factor these general principles (sunny and dry = good for you; cloudy and wet = good for mildew) into your disease control program.

• Another interesting finding: cold nights (below 40°F) throw PM for a loop. After as little as 2 hr at 36°F, portions of existing colonies are killed, new infections take longer to form colonies and their secondary spores that spread the disease, and the colonies that do form are reduced in size (hence, their new spores not only arrive later but are fewer in number). 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 the young, highly susceptible berries. Or seen another way, the lack of such nights 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 the disease by providing a "blanket" over the land at night, thereby keeping us from getting those really chilly spring evenings by limiting radiant cooling. 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 and the weather conditions that affect ripening (heat and 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 vine can tolerate a lot of foliar PM without significant negative consequences when 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 a double whammy because they're not only lousy for ripening in their own right but are also 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.

The minimal two-spray Concord PM program of 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. However, this is not going to be a typical year for some growers in portions of the Lake Erie region, and a minimal program may make economic sense if you're carrying a light crop. In contrast, growers who carry the robust crops that are increasingly necessary to make a go of this business know that they don't get all of that fruit for free, and it often can make sense to start a couple of weeks before bloom and continue into the mid-summer in order to keep a clean canopy. However, 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. Another summary of the major findings and conclusions from our studies on sulfur activities a few years back:

• We were unable to demonstrate any negative effects of low temperatures on either the protective or post-infection activities of sulfur. 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 before or after spraying them 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, although there was a slight decrease in activity at 59°F when the rate was reduced to the equivalent of 1.7 lb/A. It appears that the potential detrimental effect of low temperature on sulfur efficacy was over-emphasized in years past, particularly if you consider that the PM fungus itself is not all that active at cooler temperatures.

It should be recognized that sulfur does become more volatile as temperatures increase, after which it can move to unsprayed tissues through its vapor phase. This should "broaden the reach" of sulfur at higher temperatures, although just how important this is has not been determined objectively. The question is not whether sulfur might be more active at higher temperatures but whether it is sufficiently active at lower temperatures to provide PM control under those conditions. And the answer to the latter question is yes.

• <u>Sulfur provides very good protective activity on sprayed tissues, but not on new leaves that</u> <u>emerge after the last application</u>. Stop the presses. Bet you're glad you have guys from the University to figure that out for you.

On a less sarcastic note, there is probably <u>some</u> protective activity that is provided by sulfur vapors moving from sprayed leaves to newly-developed unsprayed leaves, as described above. But in our tests, which involved potted vines in a greenhouse at temperatures generally in the low 70's to upper 80's (°F), there was not enough vapor activity to protect unsprayed leaves from the inoculum level we were using. The situation might be different in a vineyard with <u>lots</u> of sprayed leaves contributing sulfur vapors to their unsprayed neighbors, particularly if the inoculum level was relatively low. But nobody has done the work (yet) to determine this.

• <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 was produced after the last spray was applied and begins the infection process, there's still time to control it with the next spray (applied in a post-infection mode) if that's put on soon enough. 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.

• <u>Post-infection sprays applied to heavily-diseased tissues are much less effective than those applied to incubating or very young colonies</u>. 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 1,001st 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.

• 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 protective activity significantly.

- 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 somewhat compensated for by adding a "spreader-sticker" adjuvant to the spray solution and/or increasing the application rate (from 5 to 10 lb/A in our field trials and their equivalents in greenhouse experiments). Both increasing the rate and adding the adjuvant produce an effect, and these effects generally are additive. See Table 2 below for field data, standardized across years to reflect % disease control on vines of cv. Chardonnay or the interspecific hybrid cv. Rosette relative to the unsprayed check vines in the same trial, when sprays were applied at approximately 14-day intervals throughout the season.

Table 2. Powdery mildew control 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)

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

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

"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 some other factors can cause this, such as yeasts stressed out by poor nutrition, S residues from the harvested fruit invariably get the blame when things get stinky, probably deservedly so in some cases.

The question that's commonly asked by growers is, "How late can I spray sulfur and still be safe?" And until recently, my answer was, "Everybody has an opinion but nobody has any data". This was largely due to the simple fact that whereas the consistent danger level in must was determined to be 10 parts per million (ppm) or 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 or in the resultant musts produced from them when grapes were subjected to different spray regimes in the field. 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 for measuring S on grape berry surfaces and in musts after pressing, and have made it available to growers and wineries through various media. Anyone interested who was not previously aware of this can contact one of them, but here are the take-home messages from what we learned by applying their technique to samples collected from different field treatments that we imposed on Chardonnay and Riesling vines over a 3-year study period:

• Must residues were affected by both the rate and formulation of the sulfur product used. Rates of 5 or 6 lb/A yielded greater residues than 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), which also is not surprising since increased tenacity/longer performance is one reason that growers are willing to pay more for the micronized formulations. 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 less before 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 immediately after crushing. That is, the S particulates settled out within 24 hr, after which they were found in the sediment rather than the juice. These results are consistent with an obscure 1980 German study that Misha ran across, and strongly suggest that typical

white wines should not be stinky as a result of sulfur use in the vineyard, even when residues on harvested fruit are high (this is <u>not</u> the case for red or other wines fermented on their skins). See Figure 6 for a graphic representation of this phenomenon. (Anyone interested in all of the gory details can find them in Am. J. Enol. Vitic. 65:453-462, which is available for free on the American Society of Enology and Viticulture website).



Figure 6. The effect of clarification through settling on elemental sulfur residues present in juice pressed from fruit that received sequential applications of two commercial sulfur formulations (5 lb/A formulated product) during the 2011 season, ceasing either 54 or 12 days before harvest. Samples were obtained from 12 inches below the juice surface at the post-pressing 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/Tavano), 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 earlier as on leaves that were sprayed only 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 and slimy creepy-crawlies a couple of inches long plus nice and plump. And perverse children (I knew a few) were awed by what happened when you poured table salt on them: it sucked the water right out of the beasts and they shriveled up to almost nothing right before your very eyes. And that's just what happens to a PM colony when you spray an adequate solution of monpotassium phosphate, potassium bicarbonate, sodium bicarbonate, etc. onto it (preferably, when it's young and easy to wet). However, the solution itself dries up and does nothing if there's no mildew colony (or slug!) present when the spray is applied, nor does anything happen if a 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 labels might claim or those who want to be "green" might like to pretend--this tells us at least two things that have practical implications, which we 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 apply them often enough that the fungus does not have time infect after one application and subsequently mature to make a new crop of spores before we make the next application that might knock it down. And recall that the fungus needs only 5 to 7 days to do this 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 it does not harm the plant. Note that potassium bicarbonate products (Armicarb, Kaligreen, Milstop) are sold for this purpose rather than sodium bicarbonate —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 have seen no difference between any of the different potassium salt products when they've been used at their labeled rates, even though there can sometimes be significant differences in the prices charged for them.

DOWNY MILDEW (DM) NEWS AND REMINDERS

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

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

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

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--repeated humid nights, frequent rains, and extended periods of cloudy weather--for long stretches of time. See: Summers of 2008, 2009, 2011, 2013, and 2014 in the Finger Lakes and Lake Erie regions.

The erratic development of DM coupled with its explosive and potentially devastating nature makes it an ideal candidate for scouting, especially after fruit have become resistant and the consequences of incomplete control are diminished. No need to spray for it when it isn't there, but you don't want to allow it to start rolling if it's 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. Bob Seem and David Gadoury, have developed a computer model (DMCAST) that integrates a number of weather and crop development factors to advise when infections are likely to occur. 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 fungus becomes active during the prebloom period (in Geneva, our first infections on highly susceptible cultivars typically occur about 3 weeks before the start of bloom, or 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, although the DM organism can also infect individual berry stems (pedicels) for a couple of additional weeks and follow that pipeline into the fruit to cause the aptly-termed "leather berry" symptom (hard and dry berry, but no DM spores produced upon it). 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.

For many years, the standard fungicide test protocol on hyper-susceptible Chancellor vines at Geneva has been to start spraying about 2+ weeks prebloom and continue through approximately 4 weeks postbloom. 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, even 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. But if you get to the point that you're ready to call in the big guns, this is the Howitzer. Growers in regions where potential ground water residues are an issue (that means you, Long Island) should also be aware that Ridomil is especially prone to this problem due to its unusually high solubility in water, and be prepared to address the issue. It's highly prone to resistance development (indeed, it's no longer effective in many other DM-prone parts of the world), and although resistance has never been detected on grapes in the U.S., this is probably due in large part to relatively limited use. Resistance development is a real 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 once an epidemic has broken out). Which means that using it more than once per season under such circumstances is just asking for trouble and is specifically <u>not</u> recommended. Remember that the PHI on Ridomil Gold Copper is 42 days versus 66 days for Ridomil Gold MZ.

Note the discussion at the beginning of this tome regarding some specifics of several newer DM fungicides. Zampro (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 nearly 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 organisms; however, these materials are not toxic to other ("true") fungi, and control of other diseases 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 what you think about it.

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 good but limited (3 to 8 days) protective activity, <u>depending</u> <u>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 spores were put onto wet leaves, 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 infection began, 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 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 last in the repeat application, both to improve efficacy and help guard against the proliferation of less-sensitive/resistant strains of the DM organism (see below).

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

• <u>CAUTION</u>: The phosphonate products have become very popular, 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 spray intervals and 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 than with science. However, they <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 is not likely to occur, 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 needed to obtain progressively lower levels of control. Unfortunately, there are real limits to the rates that we can use (not only for legal and cost reasons but also due to the potential for plant injury at higher rates) and there is no chance that a "new and improved" phosphonate with greater intrinsic activity will come along (all phosphonate products are made up of the same basic active ingredient).

DON'T burn these materials out by relying on them exclusively throughout the summer. DO rotate them with something else (i.e., no more than two sequential applications), like you would any other fungicide with the potential for resistance development, to make sure that you can keep using them into the future.

BLACK ROT (BR) NEWS AND REMINDERS

1. As fruit mature, they become increasingly resistant to infection. Another annual reminder. Remember that under NY conditions, berries are <u>highly</u> 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 <u>additional</u> 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 are clean in the vast majority of commercial vineyards.

Recall that in most vineyards, mummified berries are by far the major (and oftentimes, only) overwintering source of the BR fungus. Spores from mummies on the ground--which is where they should be unless somebody screwed up and didn't prune them off the vine during the dormant season (see below)--are typically depleted by a week or two after bloom. (Now for the CYA fine print: remember that these spores are liberated from the mummies during rains. So, if it doesn't rain from bloom until 3 weeks later, as occasionally happens, the last shot of them won't occur until then). Thus, if the disease has been very well controlled by the time the overwintering spores are depleted, there should be no source for new infections even though fruit may still remain susceptible, and additional sprays are not likely to be necessary. In contrast, if new black rot 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 at the start of bloom plus 2 and 4 weeks later. Such a program provides protection throughout the period of peak susceptibility and during most or all of the time remaining before berries become highly resistant. But as noted above, you can get away with stopping sprays before berries are fully resistant if there are few to no active infections present and/or the weather is dry, but growers routinely get nailed when they quit too early if there are active infections present. Also, waiting until the immediate prebloom period is a lot safer in a vineyard that was clean last year than in one that had more than a touch of disease, due to the relatively high overwintering spore load that this latter scenario will entail. Recognize when you can cut corners and when you can't.

2. Mummies retained in the canopy provide significantly more pressure for BR development than those dropped to the ground. Another reminder: mummies in the canopy produce <u>many</u> more spores than those on the ground (as in 10 to 20 times as many) and continue to produce

them <u>throughout</u> the period of berry susceptibility, whereas spores from ground mummies are depleted shortly after bloom. Furthermore, spores from mummies in the canopy are <u>much</u> 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.

3. The incubation period for BR can be very long. Under upstate NY conditions, we've found that clusters infected during the first few weeks after bloom show symptoms by about 2 weeks later and that all diseased berries are apparent within 21 days after the infection event. However, clusters infected near the end of their susceptible period do not even start to develop symptoms until 3 to 5 weeks after an infection event begins. (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- to late July, depending on the cultivar. This fact should be considered when trying to determine "what went wrong" should such late-summer disease develop.

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 (Nova = Rally) fungicide in control of black rot under field conditions

	% Diseas	e 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*. Nova/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 to supply 1.8 oz/A of active ingredient. Trials run by Mike Ellis in Ohio and Bryan Hed in PA show that Mettle and the difenoconazole products also have similar levels of

activity. (Vitacure, an old DMI in a different subgroup of this class, appears to be less effective). In many of our trials, the strobies were right up there at a similar top level. 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 can be important for Botrytis control as well. However, use of these products during the critical period is complicated by the diminished activity of at least some DMI products against PM in many vineyards, and the threat or occurrence of DM resistance to the strobies.

All of the strobies appear to be equivalent to one another and provide very good to excellent control, equal to mancozeb and ziram under moderate pressure and superior under very wet conditions, since they're more rainfast. Of course, rainy conditions are when superior performance against BR is most necessary. 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.

There's no single "correct" answer about what to use, as this will be influenced by the relative importance of the diseases to be controlled, the current resistance status and former fungicide use patterns within each vineyard, current weather conditions, etc. Understand the principles and react accordingly.

6. Special considerations for "organic" growers. Black rot is perhaps the "Achilles heel" for organic grape production in the East. It is not known as a BR fungicide, and in the only good trial that we've run with copper, it provided 40% disease control when applied at 2-week intervals versus essentially 100% control with Rally/Nova. (I recently found an old report from a trial that Roger Pearson ran in the mid-1980's, where he got 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 program to remove mummies during pruning, 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.

Unfortunately, we don't know of any "magic bullets" for organic producers to spray, although there are several products out there that almost claim to be. Bryan Hed at Penn State has looked at a number of possibilities and we've followed up with a couple of the most promising, but the typical scenario has been that things that have looked good in the greenhouse haven't held up in the field (most likely, they wash off with rain, among other things). Right now, it looks like nothing is as good as copper in terms of an organically-acceptable fungicide against BR.

Therefore, 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 means 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 possiblen. 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 from 2 to 6 weeks after cap fall and prune out any affected clusters or portions thereof before they allow the disease to spread, even better. Note that spores for disease spread during the current season are dispersed by rain primarily <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. Inoculum produced in overwintering cane lesions--which are rare unless the vineyard had severe 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) NEWS AND REMINDERS

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

1. Biology. The Botrytis fungus thrives in high humidity and still air, hence the utility of cultural practices such as leaf pulling and canopy management to minimize these conditions within the fruit zone. It's a "weak" pathogen inasmuch as it primarily attacks highly succulent, dead, injured (e.g., grape berry moth, powdery mildew, rain-cracked), or senescing (expiring) tissues such as wilting blossom parts and <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. 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. Some recently-determined details regarding the above:

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

(Note that this scenario of latent infections becoming established during the initial stages of berry development and remaining inactive through harvest can be a nightmare for table grape producers, who sometimes harvest apparently-healthy fruit only to have latent infections become active in transit or storage as the berries continue to ripen/senesce. Professional table grape

producers have developed programs to contend with this, but anyone considering getting into the production of grapes for fresh fruit needs to pay close attention to controlling Botrytis EARLY).

• 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 if only a few of them become active and provide the initial "foot hold" from which subsequent spread can occur during ripening.

Because relatively few of these early infections typically do become active and turn into rot, controlling them at bloom provides only modest benefit if the post-veraison season is dry and doesn't support further disease spread. However, it can be critical in a year with a wet preharvest period (especially if the mid-summer was wet as well), which favors both the increased activation of latent infections and their rapid spread. So in one sense, bloom sprays are an insurance policy against the future unknown. Sometimes they pay huge dividends, sometimes not (data below). 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. 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 right after set. 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.

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 practice 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.

IMHO, loosening cluster compactness represents the "holy grail" for Botrytis management, and it's just about as elusive. GA treatments very well may have their place but this technology is not foolproof, we do not have all the answers yet and there are risks involved. However, the potential payoffs are large, particularly in regular "problem" blocks. I would caution anyone interested to still view it as an experimental technique with a lot of promise, to do their own experiments on a small scale for awhile to get a feel for things, and to keep their eyes and ears open with respect to the experiences of others.

• There is no single "correct" timing regimen for fungicide applications in a Botrytis management program. The standard "full" program used in fungicide trials, provided on many fungicide labels, and employed by some growers of highly susceptible and valuable cultivars consists of four sprays: at bloom (to limit the initiation of latent infections), bunch closure (last chance to cover entire berry surfaces), veraison (start of increasing susceptibility), and 2-3 weeks pre-harvest (start of period of greatest susceptibility with 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 7, 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 many (especially 2008!), applying all four provided the best results.



Figure 7. Influence of spray timing on the control of Botrytis bunch rot in Geneva, NY (cv. Aurore, 1996-2000; cv. Vignoles, 2002-2011). Sprays we applied at (i) Bloom + bunch closure (Bl, BC); (ii) Veraison and 2-3 wk later (Ve, PH); or (iii) at all four of these stages. Data are expressed as percent reduction of diseased berries relative to vines receiving no Botrytis fungicides.

The relative benefits of early versus late applications, and the total number necessary, will vary among years according to rainfall patterns and, quite likely, differences between cultivars and clones (e.g., cluster tightness). Think in general terms of early sprays as limiting the establishment of primary infections, and later sprays as limiting disease spread. But remember that Botrytis is not a disease that you can just "spray your way out of". These materials 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 cultural practices (canopy management, leaf pulling, etc.).

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

The treatments involved were:

- Training system (Top Wire Cordon 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 maintained his crop via standard practices, which included a Botrytis spray regimen. We rated the plots for incidence and severity of both Botrytis and sour rot at harvest on September 19 (harvest); the VSP treatment was also rated 10 days pre-harvest. A few sets of data and interpretations/notations are provided below.



Botrytis Severity, 9/19

• Positive effect of canopy manipulation treatments in VSP, not in TW

• In VSP, Shoot Thin + Rachis Removal was best, 43% reduction versus check treatment



• Effects of training system and canopy manipulation were additive:

• With no canopy manipulation (check), effect of going from TW to VSP was modest: 20% reduction in average % rot.

Within VSP, thinning shoots and removing rachises reduced rot by 40% relative to the check
Going from TW to VSP and thinning shoots + removing rachises reduced rot by 52% relative to the TW check



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

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



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

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

2. *Fungicides, physical modes of action*. Over several years, we looked at the various "physical modes of action" of the available Botrytis fungicides, to get a better idea of some of their specific characteristics and differences. Following is a repeat of previous summaries of the major findings and conclusions 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 berries became diseased; in contrast, such spread occurred in two-thirds of the unsprayed clusters.

• In a more direct test for residual protective activity on the berry **surface**, clusters on a second set of Chardonnay vines were sprayed on the same dates as above and Botrytis spores were applied to the surface of 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). <u>Under the conditions of this test</u> (individual clusters sprayed by hand, providing <u>complete</u> 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 current "standard" fungicides registered for Botrytis control provided excellent protective activity on the surface of the berries. That's why they got developed and marketed in the first place.

• The so-called AP fungicides (Vangard and 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 sold as a surface protectant. However, this was a function of marketing strategy rather than science.

• 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 7, 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 the 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

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) can 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 (hence the name, duh).

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, who has been in the forefront of sour rot research for more than 5 years now, has shown that the measure of VA in grapes harvested from different vineyards is strongly associated with the pre-harvest level 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*), 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. There appears to be a progression of steps involved in this whole process, which probably begins with the production of ethanol by "good" yeasts as the injured berries start leaking grape juice (ethanol is the substrate that the abovementioned bacteria convert to acetic acid, and we've found a lot of *Saccharomyces* yeasts associated with sour-rotted berries in the field). Indeed, Megan Hall, a graduate student now working on this disease in Geneva, has measured both ethanol and acetic acid in every one of a large sample of sour-rotted clusters, with their relative concentrations being inversely proportional (as ethanol concentrations go down, acetic acid concentration go up and *vice versa*) which would be consistent with such a scenario. A lot of the details are still rather murky, but we know a lot more than we did when Wendy started working on this a few years ago.

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°); 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. These data probably make sense to Finger Lakes Pinot Noir growers who remember September 2013—very warm and wet after Labor Day as clusters of this cultivar were nearing harvest and accumulating sugars rapidly, with nasty sour rot ensuing soon thereafter.

The Ontario contingent has also done a nice job of documenting that sour rot doesn't get started in the 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 bacteria 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).

Another piece of the puzzle that we're beginning to understand is the increasingly apparent role that fruit flies (*Drosophila* spp.) play in the cause and spread of this disease. Clusters with sour rot are typically swarming with fruit flies. A prominent line of thinking over the years has been that these insects 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, suggests that the flies may actually play a direct role in the initiation and/or spread of the disease. Which caught our interest, see below.

Thus, in terms of managing sour rot, it seems that the likely strategies are: (1) Provide a berry microclimate in the canopy that's less conducive to pathogen growth; (2) Minimize berry injuries; (3) Minimize populations of the microbial pathogens; and (4) Control the fruit flies if they are, indeed, a factor.

In 2013, we (graduate student Megan Hall, entomologist Greg Loeb and his technician Steve Hessler, along with yours truly and technician Dave Combs) began a multi-year project to better understand sour rot and how we might improve our management of it. We're still in the relatively early stages, but here's what we've found so far:

Canopy microclimate. Prior to 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 previously in the Botrytis section. The effects were pretty dramatic, as shown 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%)

In September 2014, Megan returned to this vineyard to assess sour severity in a different season. No variable canopy management treatments had been imposed, but the effect of training system was pronounced once again, with twice as much disease with Top Wire training versus VSP. The data are presented below.



Figure 8. 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.

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.

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 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). It must be noted that whereas KMS is used widely in wineries both to sanitize equipment and as an 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. Also, it is nasty stuff if you get it in your eyes or breathe in the dust.

Control fruit flies. Although some growers have tried this approach, I'm not aware of any experimental data evaluating its efficacy prior to our trials the past 2 years. Our 2014 trial was snakebit (most clusters destroyed by hail, very little rain at the experimental site) and we didn't learn much from it, but in technical jargon the 2013 trial was a doozy.

2013 trial results. We looked at a combination of insecticide and antimicrobial sprays. Alternate rows in a 'Vignoles' vineyard were sprayed with the insecticide Delegate (weekly, beginning at 15° Brix), with the adjacent row receiving no insecticide. Then, within these insecticide-plus or -minus rows, we applied various antimicrobial treatments, also on a weekly schedule: (i) 0.5% KMS, beginning at 15° Brix; (ii) 1.0% KMS, beginning at 15° Brix; (iii)

Kocide at 2 lb/A (registered!), beginning at 15° Brix; (iv) 1.0% KMS, beginning at first appearance of disease symptoms; (v) none (check). The results are presented below.



Figure 8. 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.

Bottom line: Antimicrobials with insecticide provided an average of 50% control (vs. check); antimicrobials without insecticide provided an average of 9% control (vs. check); and insecticide without antimicrobials provided 15% control.

A few comments:

- These are data from a single experiment and I'll feel even better once we're able to repeat the results in another bad sour rot year. However, both our results and those from Ontario indicate that some antimicrobial sprays can reduce sour rot. 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 fungicides to provide much benefit in our region or those with similar climates, other than perhaps reducing the number of certain injury sites (e.g., pre-harvest Botrytis infections). In warmer climates such as California, Texas, and South Australia, 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 not clear.
- We have many other reasons to believe that fruit flies are important players in this disease complex. For example, when Megan tries to reproduce sour rot symptoms on berries in the lab, the only combination of factors that gives us symptoms consistent with what we see in the field is wounding followed by inoculation with *Saccharomyces* yeast (to produce ethanol from the juice) plus acetic acid bacteria (to oxidize the ethanol to acetic acid) **plus fruit flies**. We have a couple of ideas about why this might be and are working to see if

they're correct or not—stay tuned. Also, it should be noted that whereas the spotted wing Drosophila is getting a lot of attention these days and may be a component in the mix, it doesn't seem to be a major player, as grapes are not a preferred host; the "garden variety" species—D. melanogaster, which has always been around—seems to be far more important from what we can tell so far.

- This trial was 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. Once we're convinced that they can, we'll start working on finding out how much less we can spray to get an economically acceptable result.
- KMS is not a legal treatment and Kocide has potential copper residue issues that, although legal, might cause problems with fermentation in the winery. We're also looking at Oxidate, which is expensive but legal and without potential fermentation issues. Wendy did not get benefit from it in her earlier trials, but some Finger Lakes growers tried this product as a "rescue" treatment in 2013 and are convinced that it helped (of course, such observations are seldom based on comparisons with an unsprayed row or rows). There's another new product out there with purported efficacy, which we're also taking a look at. Finally, we'll also be using Mustang Maxx as our insecticide in future trials, as Greg thinks it will have more residual efficacy. (Note that it is labeled for use on grapes with a 1-day PHI, although fruit flies are not a listed target pest). Again, stay tuned.

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 perceived risk and philosophy for addressing it. For now, I'd keep these concepts in mind: Disease can be initiated once rains occur after berries reach approximately 15° Brix; warm temperatures (extended periods 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 early than if you wait until things start blowing up in your face. Just how to do this economically and practically is the \$64,000 question (a term that was 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 go cheap and remain legal, I might concentrate on the fruit flies. If it was consistently warm and wet and I'd had a problem in that block before, I might start antimicrobials at 15° Brix before seeing symptoms and back off if the weather turned more favorable and/or disease development stayed in check. 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.

"OTHER" ROTS

SUMMER ROTS is a term sometimes used for two similar diseases (ripe rot and bitter rot) common in more southern, humid (and quite warm) production regions. Those beneath the Mason-Dixon line and in the lower Midwest deal with these diseases on a regular basis and they occur sporadically to the north. Bitter rot, in particular, seems to pop up with some regularity on Long Island, particularly on Chardonnay, and reports from southern PA suggest that it's no stranger there in some years. Those of us to the north should probably start being more aware of these diseases, especially in very wet years. They're not a threat to be over-emphasized in the more northern regions, but neither is it one to be flat out ignored. Particularly if our summers do continue to heat up.

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 2015 Pest Management Guildelines.

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, and serve to spread the disease to healthy fruit if rains continue. Infected fruit shrivel and mummify, and may either remain attached or fall to the ground. No foliar symptoms are produced.

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

Cultural practices such as pruning out dead spurs, removing overwintered mummies, and removing weak or dead cordons are important to help reduce the inoculum in the vineyard. Turner Sutton (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 very 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 and, therefore, are not consciously managed.

Sprays targeted against bitter rot and/or ripe rot may be needed in the late season if the weather is warm and wet, especially if the diseases are observed in the vineyard or have occurred there in the past. In southerly regions where they are consistent problems, it is typically necessary to apply protectant fungicides on a 2-week schedule from bloom until harvest, except during periods of drought. Because fruit are especially vulnerable in their final stages of ripening, pre-harvest sprays can be particularly useful when these diseases are active, to avoid rapid secondary spread. This potential utility must be balanced against wine makers' concerns about the effects of such sprays on fermentation (of course, they're also not thrilled about fruit with bitter rot, 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 these commercial blocks for several reasons, that does not have to be true. 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 (as clusters 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 preharvest drop of fruit onto the ground for both cultivars but the fungus also seems to move readily from the pedicel (berry stem) into the fruit of Niagaras (of course, downy mildew can kick Niagaras pretty hard in some years as well).

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

2. Early sprays also provide significant control of berry infections. In a trial conducted several years ago 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 infection. 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 variety. 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. Niagai	ra; Fredonia, 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. In both years, the single spray ("1x") was applied 2 weeks after the first spray that was applied in a comparison treatment at 1- to 3-in shoots. This comparison treatment received three applications in total ("3x"), i.e., 1- to 3-in shoots plus two additional sprays at 2-week intervals.

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

4. Little fungal inoculum, if any, is available by mid-summer. We monitored the release of Ph spores in several Lake Erie and Finger Lakes sites over 3 consecutive years. And in each year, we detected few if any infectious spores beyond early- to mid-July, with the vast majority released between bud break and bloom. A similar study conducted by Annemiek Schilder at Michigan State University produced generally similar results. These data suggest that even though berries may remain susceptible throughout the season, as shown by work from Mike Ellis and students at Ohio State, the risk of infection is probably very low once berries become peasized, 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 apparently a decent Ph material in California (where it doesn't rain during most of the growing season) has done practically nothing in our trials.

6. Spray application technique. Many growers like to spray alternate rows in the early season-the critical time for controlling Ph--assuming that sufficient spray will blow through the target row and impact on vines in the "middle" row. For 3 consecutive years, Andrew Landers helped us examine 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 benefits of alternate-row spraying are 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 also is obvious.

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) can be problematic as well. Concord, Catawba, and Leon Millot sometimes have problems with this disease in the Midwest, although it has not been an issue on them in NY. Here, it's the cold-hardy grapes that have put anthracnose on the map.

Although they are far from immune, most of these cold-hardy cultivars have significant albeit variable levels of resistance to powdery and downy mildew and black rot. However, it is very likely that (limited) resistance to these diseases is related to their 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. Downy mildew, powdery mildew, and black rot 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; apparently, *V. riparia* is particularly susceptible.

Anthracnose can cause nasty lesions on leaves and young shoots, typically on the first few internodes near their base. These lesions resemble the internode lesions typical of Phomopsis but they usually are more aggressive, expanding farther along the shoot and deeper into its center. Infected berries develop spots approximately ¹/₄-inch in diameter, with whitish-gray centers surrounded by reddish brown to black margins; supposedly, this has caused some people to call the disease "bird's-eye rot", but I've never heard anyone use that term.

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 in the lower Midwest than it is in NY; however, infection <u>can</u> occur at cooler temperatures if things stay wet for 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 new infection and additional 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 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. A "delayed dormant" application of lime sulfur can be very useful in vineyards where the disease has become established and problematic to control and/or in "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 works 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 and former students 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 controlled. 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 Dr. Philippe Rolshausen--a 10-year veteran of Doug Gubler's lab at UCD, with a wealth of experience in this field--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, upon 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, to boot.

These diseases are particularly common in older plantings such as those that predominate our juice grape industry, and although its leadership is not interested in addressing the problem, it's costing them money nevertheless and individual growers should at least make sure that the canker diseases are on their radar. Many wine 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 slowly-expanding 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 where systematic trunk renewal is not the norm, canker diseases will become increasingly important. One well-known international consultant has suggested that canker diseases might be the greatest threat to the industry since phylloxera, and although this seems like hyperbole, I also think that he is rightly trying to draw attention to a problem that is too often ignored.

Unfortunately, it's a good bit easier to recognize this problem than to manage it effectively. 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. 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 already is dead and 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/gallon of water) to freshly-made pruning wounds to protect against such diseases. This is not practical for routine pruning cuts, but may very well be worthwhile where larger cuts are being made for retraining purposes or to remove cankered arms and replace them with new growth (hint: there's obviously inoculum around that needs to be protected against, in such cases). In Australia, they've developed a small hand applicator gadget to do this quickly—basically, a plastic bottle filled with concentrated fungicide solution, which has a bristle brush on the end to "paint" the pruning wound with a rapid swipe or two (also, the 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 and have been reasonably successful in doing so (the Californians actually 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 worthwhile under some eastern conditions. Finding answers to these and related questions will require a long-term 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.

PUTTING IT ALL TOGETHER

As I preface this section every year, we all know that there are as many good disease control programs out there as there are good growers and advisors. The following are <u>some</u> considerations among the many possible alternatives. But as always, 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, 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 leading to significant inoculum retention) or block history suggests high risk. Ditto for blocks subject to **anthracnose**, especially if the weather has gotten warm now. <u>Option A</u>: Nothing. <u>Option B</u>: Captan, mancozeb, or ziram. The best one is whichever is cheapest and most convenient.

3- to 5-INCH SHOOT GROWTH. A critical time to control **Ph** rachis infections, 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 dry and you can cover up before the next rain. Getting in a bit late after rains have occurred 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.

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 late 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. Blocks susceptible to **Anth** need protection now.

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 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 ½-inch of rain. Can use with mancozeb or ziram, but not with or near captan or sulfur (plant injury). Option F: Nutrol, Armicarb, Oxidate, Kaligreen. (PM). Should eradicate young infections IF thorough coverage is provided, but no forward activity. If choosing this option so early in the year, go with the low end of the label rate and use the cheapest one. Can mix with captan as well as mancozeb and ziram. Option G: Rally, tebuconazole generics, or Mettle (PM, BR, Anth); or Revus Top (PM, DM, BR, Anth). Remember, we want to limit the use of all of these DMI (Group 3) products combined to a total of three applications per season, so budget them out time-wise accordingly. The DM protection provided by Revus Top is not likely to be necessary yet. Option H: Serenade, Sonata, Regalia, Double Nickel, Tavano/Oso, or Ph-D (PM) if you want to experiment with OMRI-certified biopesticide products while disease pressure is low (Tavano/Oso and Ph-D are not OMRI certified). 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 unless this disease was a problem last year (inoculum levels are high) and weather is wet and warm. DO NOT wait any later than now to control PM on V. vinifera cultivars or highly susceptible hybrids. On Concord and other "moderately susceptible" cultivars, 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 uncontrolled early infections then spread to the clusters and basically got an epidemic started, 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 have experience with early disease development, and /or weather conditions are forecast to particularly favor PM (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 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 $>52^{\circ}F$ 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 for growers to 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 a couple of 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 (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. Toss in 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: Quintec or Vivando (PM). Both are Cadillac PM materials, and each should be limited to two applications per season for resistance management purposes (they are unrelated to one another). 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 F: Torino (PM). A 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 crossresistance and allows you to save other PM materials for use later in the season as pressure increases. Option G: Rally, tebuconazole generics, Mettle (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 H: JMS Stylet Oil (PM). If (and only *IF*) coverage is thorough, this spray should eradicate early PM colonies that may have started, should previous PM sprays have been omitted or incompletely applied. But don't waste your money if you can't cover thoroughly. Also may help with mites. Will provide a few days protectant activity going forward in addition to the 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 much 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 less effective. Don't mix any of these with Option I: Nutrol, Armicarb, Oxidate, Kaligreen. (PM). Should eradicate young captan. 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 OMRI-certified biopesticide products before entering the critical period for disease control. Ditto for the biopesticides Tavano/Oso and Ph-D (PM), although they're not OMRI certified. <u>Option K:</u> A PM-specific product plus mancozeb, ziram, or captan (no captan + oil!) to pick up DM, BR, Ph, and Anth as necessary.

IMMEDIATE PREBLOOM TO EARLY BLOOM. A critical time to control PM, BR, DM, and Ph on the fruit (Anth, too)! Just starting to enter Bot season, also. This and the first postbloom spray are the most critical sprays of the entire season--DON'T CHEAT ON MATERIALS, RATES, SPRAY INTERVALS, OR COVERAGE!! Option A: Vivando or Quintec for PM control, plus mancozeb for BR, DM, and Ph, and Anth. As mentioned before, 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. 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 one so that they remain effective. Option B. Pristine (PM, DM [?], BR, some Bot and Ph). We need to keep application numbers for this one down to 2 per season, also, especially with the increasing risk of DM resistance and its apparent development in some NY vineyards after 17 years of strobie use in the region. Some people may not want to rely on Pristine or other strobies (e.g., Quadris Top) for DM control while fruit are highly susceptible, and they probably shouldn't since a control failure now could be costly. Please refer to the discussion of DM resistance to Group 11 fungicides way back near the very beginning of this entire treatise. 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. On cultivars highly susceptible to PM, where DMI resistance is usually an issue to at least some extent and strobie resistance has occurred or is deemed risky, Quintec, Vivando, Pristine, or one of the Luna products (for those outside NY) would be the materials of choice for PM, but don't forget about DM and BR. With Pristine especially, I might toss in some sulfur, particularly in blocks where PM has already developed strobie resistance, just for additional insurance at this critical time. And something Option C: Revus Top (PM, BR, DM, Anth). (e.g., mancozeb) for DM, too. 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--especially on the clusters!--if you get spotty spray coverage and only deliver a partial rate to your spray target. Inspire Super (PM, BR, Bot, Anth) and Quadris Top (PM, BR, Anth, some Ph; DM if no resistance) also include difenoconazole as part of their mix, but Inspire Super doesn't provide DM control and Quadris Top relies upon a strobie to do so. I wouldn't use either one of these without adding something like mancozeb for DM. Option D: Luna Experience [not yet registered in NY!] (PM, Bot, variable BR depending on rate) or Luna Tranquility (PM, Bot). Add mancozeb to pick up DM and improve or provide activity against BR. For reference, the 6 fl oz/A rate of Luna Experience provides only 40% as much tebuconazole (the component in this mix that's active against BR) compared to the labeled rate of Elite or various generic tebuconazole products; the rate of 8.0-8.6 fl oz/A rate recommended for BR control provides 78-83% as much as these other teb products,

respectively. The pyrimethanil component of Luna Tranquility will provide extra punch against Botrytis, but that's probably not necessary now and the trade-off is a loss of BR and (modest? depends on rate) PM control from the teb component in Experience. If you want to and can use these products, I'd go with Experience at this time of year. Option E: Abound or Sovran. You know the story: When they were new, these were the cat's meow against most of our major diseases (OK, Sovran wasn't great on DM). But resistance is common or risky enough that I wouldn't rely on them against either PM or DM except in mixture or plantings where strobie use has been very limited and/or cultivar susceptibility is not too high. Option G: Rally, tebuconazole generics, or Mettle (PM, BR) PLUS mancozeb (DM, BR, Ph, Anth) or captan (DM, Ph, Anth). IMHO, you'd choose this option only if you 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. 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). Option H: 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. Option I: 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), Endura, Pristine, or one of the Luna products (Experience, Tranquility) [not yet labeled in NY] applied around the bloom period often provides 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 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 effective 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* (i.e., keep the spray interval short with sulfur at this time of year), especially if it's been raining since then or will 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 advisable under wet conditions and it should be considered critical if infections are evident on the vine, unless these are present and you're willing to bet part of your crop that you won't get an infection period before the fruit become resistant in a few more weeks; however, BR sprays can often be skipped from here on out on natives and hybrids if the vineyard's clean. And although the same is true for V. vinifera blocks that are CLEAN clean, their greater 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 winespoilage microorganisms that may follow upon the "diffuse" PM infections that can develop on berries as they transition to a resistant state near this point in their development. 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. Concords can withstand a good bit of foliar PM unless the crop is large and/or ripening conditions are marginal. Minimal programs can 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. Clusters are still susceptible 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. 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 for excellent PM control + an appropriate material for DM, BR, and/or Bot as necessary. 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. Logical BR options include mancozeb (if still within the 66-day PHI limit), ziram, or one of the strobies; BR resistance to the strobies has not been a problem and is unlikely to become one. 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 Quintec or Vivando seems like overkill versus PM. DM options include mancozeb (ziram is only fair), captan, Zampro (but not in NY), Presidio (get a 2nd mortgage), Ranman, the phosphonates, and copper. The potential efficacy and resistance danger for the strobie products (Abound, Pristine, Quadris Top, Sovran) has been discussed already. Option C: Torino for very good PM control + an appropriate material for DM BR, and/or Bot as necessary. Provides protective plus post-infection activity and is unrelated to any other fungicide on the market, so a good rotational partner. 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, or Flint. Should work well against BR and PM (Pristine), might work against DM. Pay your money and take your chances, or tank-mix with something for DM control if you're not feeling lucky. Pristine and Flint also can provide good Botrytis control at appropriate rates. Option F: Luna Experience [still not labeled in NY] for excellent PM and Bot control + something for DM and/or BR as necessary. See comments regarding Luna Experience and Luna Tranquility under IMMEDIATE PREBLOOM/EARLY BLOOM section. Option G: Rally, tebuconazole generics, or Mettle for fair to very good PM (depending on rate, resistance status of vineyard, cultivar) and excellent BR (and Anth) + something for DM. Option H: 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 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, haven't used up your seasonal allotment yet, and want a premium material (could make sense for an extended period of protection in the final spray). Same principle applies for Pristine and (if you can use them) Luna Experience and Luna Tranquility, which will provide even better Bot control than LE; remember, neither Luna product is registered in NY. 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. 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. Tavano/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 previously. **Summer rots** are controlled with mancozeb, captan, and strobies; a peak period of susceptibility appears to be near veraison. Strongly consider an "insurance" application against **Botrytis** on susceptible cultivars/clones/blocks at or soon after veraison (depending on the weather), then determine the need for a subsequent pre-harvest spray based on weather and the need to limit spread of the disease, should it be revealed by scouting. **BR** should not be an issue after the 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 coming year!







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2015 Coffee Pot Meeting Schedule

- May 6- 10:00am-Dan Sprague- 12435 Versailles Rd. Irving NY 14081
- May 13- 10:00am- Phillip Baideme- 7935 Route 5, Westfield NY 14787
- May 20- 10:00am- CLEREL, 6592 West Main Rd. Portland NY 14769
- May 27- 10:00am-Nick Mobilia- Arrowhead Winery 12073 East Main Rd. North East PA 3:00pm-Evan Schiedel/Roy Orton- 10646 West Main Rd. Ripley NY 14775
- June 3- 10:00am- Bob & Dawn Betts- 7365 East Route 20, Westfield NY 14787 3:00pm- North East Lab-662 N Cemetery Rd. North East PA 16428
- June 10- 10:00am- Peter Loretto-10854 Versailles Plank Rd. North Collins NY 14111 3:00pm- Dave Nichols-1906 Ridge Rd. Lewiston NY 14092
- June 17- 10:00am-Tom Tower 759 Lockport Rd. Youngstown NY 14174 3:00pm-Leo Hans-10929 West Perrysburg Rd. Perrysburg NY 14129
- June 24- 10:00am- Kirk Hutchinson-4720 West Main Rd. Fredonia NY 14063 3:00pm- Brant Town Hall- 1294 Brant North Collins Rd. Brant NY 14027
- July 1- 10:00am-Ted Byham 9207 West Lake Rd. Lake City PA 16423 3:00pm-Alicia Munch-761 Bradley Rd. Hanover NY 14136
- July 8- 10:00am- Rosemary & Brenda Hayes- 6151 Route 5 Brocton NY 14716
- July 15- 10:00am-Szklenski Farms- 8601 Slade Rd. Harborcreek PA 16421
- July 22- 10:00am- Paul Bencal-2645 Albright Rd. Ransomville NY 14131

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