Grapevine Yellows risk assessment and management strategies

Dr. Tony K. Wolf, Professor of Viticulture, Virginia Tech

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Grapevine Yellows is a lethal disease of wine grapes that causes a range of diagnostic symptoms, including yellowing and downward rolling of leaves, die-back of shoot tips, abortion of developing fruit, uneven periderm or bark development on shoots, and vine death. Although a related disease, *flavescence dorée*, was recognized in Europe since the 1950s, grapevine yellows was only confirmed in North America (Virginia) in 1993. Identical symptoms have been observed on Chardonnay in Southeast Pennsylvania, in western Maryland, and in the Finger Lakes of New York, where symptomatic vines were first observed in the 1970s. Yellows diseases of grape occur worldwide and are given different, descriptive names based on disease symptoms, different causal agents, or for other reasons. Other grapevine yellows diseases include *flavescence dorée, bois noir*, and Australian Grapevine Yellows. The yellows disease that affects wine grapes in the mid-Atlantic USA has been termed North American Grapevine Yellows (NAGY).

Symptoms

Symptoms of NAGY observed on *V. vinifera* cultivars in the mid-Atlantic region of the US are essentially similar to other Grapevine Yellows (GY) diseases. Symptoms are often initially noticed on one to several shoots of an otherwise apparently healthy vine, although the shoots of an entire cordon or an entire vine may simultaneously express symptoms. In cases where disease is observed on one or several shoots early in the season, the symptoms often advance to affect other shoots on the vine as the season progresses. In the first season of expression, symptoms can appear anytime after bloom, and are evident as a cluster withering. Cluster abortion is first noticed as a loss of berry turgor, followed by rapid desiccation of the entire cluster, including the rachis. Shriveled berries adhere to the rachis, but the entire cluster may eventually abscise from the shoot. Fruit clusters and tendrils on affected shoots are subject to abortion any time from fruit set through grape maturity (**Figure 1**). Additional symptoms on Chardonnay can include an abortion of shoot tips, including significant die-back, and the development of a blue-gray cast to the shoot stems, instead of the normal reddish cast that stems exhibit in mid-summer. Shoot stems fail to develop periderm, or the brown periderm develops in very irregular islands or patches on the stem. Small, dark pustules may appear on the base of the stems, particularly with White Riesling. NAGY-affected shoots are pliable and often droop, a condition that is thought to be related to the failure of affected shoots to form lignified secondary phloem fibers. Internodes are often shortened, leading to an overlapped pattern of leaves on a shoot.

Figure 1. Cluster abortion in Chardonnay resulting from infection of vine by grapevine yellows phytoplasma.

Figure 2. Leaf rolling and veinal chlorosis resulting from grapevine yellows infection in Chardonnay.

Leaf symptoms are variable, but commonly include a downward leaf rolling, the acquisition of a crisp, hardened or brittle texture, and discoloration. Discoloration is variable and is a function of cultivar, leaf age, and symptom progression. Foliar symptoms on Chardonnay start on the older, more basal leaves of the shoot, and the discoloration is first apparent as a fading of the green leaf color, or chlorosis, starting at the leaf margins and moving towards the center of the leaf. As the season progresses, symptoms extend to younger primary leaves, as well as to the leaves of lateral shoots. The chlorosis may appear as a diffuse lightening or yellowing of the dark green interveinal region of the leaf (**Figure 2**), or it may include development of discrete regions of bleached or yellowish tissue either confined to interveinal regions or including veinal tissue. Chlorotic tissue can become necrotic before leaves abscise. Discrete zones of chlorotic tissue appear are more commonly observed on White Riesling than with other varieties. Leaf abscission occurs as the symptoms intensify and the season progresses.

Bud break in the second year of infection can be uneven, particularly with cordon-trained vines, where entire portions of the cordon(s) may fail to produce shoots. Entire vine death can occur by the third year with sensitive varieties such as Chardonnay.

Growers should familiarize themselves with visual NAGY symptoms as part of the management of the disease includes removal and destruction of positively identified vines that are affected by GY. Leafroll virus may show similar leaf rolling, but leafroll does not cause shoot-tip dieback or cluster withering.

Causal Organism

Grapevine yellows diseases are caused by phytoplasmas, small, prokaryotic organisms that lack cell walls and can not be cultured on artificial media. The yellows diseases of grapevine are a few of at least 200 distinct plant diseases found worldwide that are caused by phytoplasmas. The bacteria-like organisms are restricted to phloem tissues in plants, and they are transmitted from plant to plant by insect vectors. There is also some evidence that grapevine yellows can be spread by collecting infected wood which is then used for propagation.

More specific evidence of the role of phytoplasmas in grapevine yellows was obtained in the early nineties when phytoplasmas were detected with polymerase chain reaction (PCR) in NAGY-symptomatic grapevines in Virginia. The phytoplasmas detected in affected Chardonnay in Virginia were genetically similar to phytoplasmas that cause “X-disease” of stone fruit, and were also similar to a phytoplasma detected in GY-affected vines in northern Italy. Shortly thereafter, a second phytoplasma was also detected and described in Virginia vineyards, and in other hosts in the environs of affected vineyards. The second phytoplasma was related to a group of phytoplasmas that are loosely termed aster yellows phytoplasmas. Both of the Virginia phytoplasmas were also frequently detected in wild grapevines (*V. cordifolia and V. riparia)* growing outside the vineyard. To date, neither of the NAGY-associated phytoplasmas has been detected in grapevines outside of North America.

Disease Cycle and Epidemiology

The NAGY diseases observed in New York State and in Virginia share common symptoms and may be the same disease. In Virginia, vine recovery is typically not observed once vines

express symptoms (Wolf et al., 1994). The appearance of several affected shoots on a vine in the first year is followed in the second year by much more severe expression: shoot growth is stunted and sporadic, and symptoms are more systemic on the vine. Such vines often fail to survive into a third season, and rarely into a fourth. NAGY-affected vines in New York State can chronically express symptoms; however, vines that express symptoms in one year do not always express symptoms the following year. This variance of symptoms may be due to environmental factors, or it may be due to differences in the causal pathogen. There is no evidence to date that the two phytoplasmas present in Virginia vineyards elicit different symptoms.

Not all aspects of NAGY disease are understood, but some components are known, and some can be inferred from knowledge of other GY diseases. Wild grapevines have tested positive for the presence of phytoplasmas in New York and Virginia, and may serve as phytoplasma reservoirs. Vineyard surveys of NAGY incidence have provided evidence of an insect vector, including observed "edge" effects, where the frequency of affected vines is associated with the upwind side of a vineyard, or the edge of the vineyard that borders potential, alternative hosts (Beanland et al., 2006). Yellows disease symptoms have not been observed during a vineyard's first season of growth. These observations, combined with known vector aspects of other GY diseases, suggest that infections occurred after vineyard establishment.

Several vectors of NAGY phytoplasmas have been tentatively identified in the Virginia work. *Scaphoideus titanus* leafhoppers are abundant in the vicinity of NAGY-affected vineyards in the Finger Lakes of New York, is routinely collected in Virginia, and is known to transmit the FD phytoplasma in Europe. This leafhopper was capable of acquiring phytoplasmas from cultivated and wild grapevines in New York and transmitting phytoplasmas to broad bean plants, which developed GY-like symptoms (Maixner et al., 1993). Several other leafhopper species known to be competent vectors of other phytoplasmas are also abundant in Virginia vineyards, and were shown to transmit NAGY phytoplasmas.

Chardonnay, and to a lesser extent, White Riesling, are most commonly affected by NAGY. Multi-year survey data in Virginia showed that the incidence of newly affected Chardonnay vines was as high as 6%, and cumulatively exceeded 25% over a six-year period. In Virginia vineyards that annually exhibit up to 3% annual loss of Chardonnay vines to NAGY, symptoms and vine loss have also occurred with *V. vinifera* cvs. Cabernet Franc, Cabernet Sauvignon, Malbec, Sauvignon blanc, Petit Manseng, and Viognier, but at a far lower incidence (< 0.5% incidence).

Control

The most effective means of NAGY management is to avoid planting susceptible cultivars such as Chardonnay and White Riesling in areas where the disease is likely to be expressed. High-risk sites include the following features:

- known presence of yellows in nearby vineyards
- vineyard sites bordered by woods that contain wild grapevines

Insecticides are used with some success with *Flavescence doree* and *bois noir* in Europe; however, these versions of grapevine yellows are transmitted from vine-to-vine by essentially a single leafhopper, *S. titanus*. This leafhopper has one generation per year and carefully timed insecticide sprays can greatly reduce the population of *S. titanus* in and around the vineyard. Our situation is much more complicated in the mid-Atlantic because; in addition to *S. titanus*, there are at least 5 other leafhoppers that have been shown to transmit NAGY phytoplasmas.

Research has demonstrated that grapevine yellows symptoms can be ameliorated by pressure injection of certain antibiotics into the grapevine. This therapy, however, is neither practical nor legal for commercial use.

Efforts to prune out affected trunks or cordons in the first season of disease observation have generally failed to prevent the systemic spread of symptoms to the entire vine, at least with highly susceptible varieties such as Chardonnay. Some growers have reported success with excising affected portions of the vine in the first year with less susceptible varieties, but it's unclear whether the vines remain free of symptoms or if the symptoms are simply delayed in expression. Vines that express symptoms on at least one-half of the vine should be removed, or at least the affected trunk/cordon/head system of the vines should be removed to minimize the likelihood that the affected vine will serve as a reservoir for phytoplasma acquisition. We do see "clustering" of GY-affected vines in some vineyards in some years. This clustering is thought to relate more to the movement of an infected leafhopper from vine-to-vine, rather than one vine serving as a source of inoculum for nearby vines. This is because there is a period of weeks between the time an insect ingests the phytoplasma and when that insect will be capable of transmitting the phytoplasma.

Like Pierce's Disease, NAGY is a destructive disease of wine grapes that is not easily controlled. Management of NAGY starts with a vineyard site risk assessment and then careful section of varieties based on the risk. Tactical management tools are limited, but can include removal of potential, alternative hosts, including cultivated vines that show obvious symptoms.

Selected References

Beanland, L., R. Noble, and T.K. Wolf. 2006. Spatial and temporal distribution of North American Grapevine Yellows disease and of potential vectors of the causal phytoplasmas in Virginia. Environ. Entomol. 35:332-344.

Maixner, M., Pearson, R. C., Boudon-Padieu, E., and Caudwell, A. 1993. *Scaphoideus titanus*, a possible vector of grapevine yellows in New York. Plant Dis. 77:408-413.

Prince, J. P., Davis, R. E., Wolf, T. K., Lee, I. M., and Dally, E. L. 1994. Genomic diversity and possible wild plant sources of mycoplasmalike organisms (MLOs) infecting grapevines: Implications for epidemiology. IOM Letters 3:288 (abstract).

Prince, J. P., Davis, R. E., Wolf, T. K., Lee, Moen, B. D., Dally, E. L., Bertaccini, A., Credi, R, and Barba, M. 1993. Molecular detection of diverse mycoplasmalike organisms (MLOs) associated with grapevine yellows and their classification with aster yellows, X-disease, and elm yellows MLOs. Phytopathology 83:1130-1137.

Wolf, T. K., Prince, J. P. and Davis, R. E. 1994. Occurrence of grapevine yellows in Virginia vineyards. Plant Dis. 78:208.