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Is Inflorescence Necrosis Another Bloom Time Hazard to Berry Set?

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HISTORY

In 1983, Muscat Ottonell and other early muscats had a complete crop loss in Willamette Valley vineyards. At that time, we assumed the loss was from a botrytis blossom rot. Since 1983, much has changed: we've seen the disorder every year (it resulted in major crop losses in 1988 and 1990), the disorder has been described in the literature with at least two names (Early Bunch Stem Necrosis and Inflorescence Necrosis), it has been shown not to be a botrytis blossom rot, and research results strongly suggest that its cause is a nitrogen imbalance, probably ammonia toxicity.

Ken Brown was the first Oregonian to research the disorder when he took a leave in New Zealand. David Jackson of Lincoln University, New Zealand had propagated Gewurztraminer and Pinot noir cuttings for Ken's greenhouse study. But when the potted plants were placed in an outdoor shade house to wait for Ken's arrival to begin his study, the clusters became necrotic and lost most of their flowers. Following up on the disorder, David ran some greenhouse trials to confirm it was a physiological breakdown of the clusters at bloom and not a rot.

David Jackson in 1988 used the name "Early Bunchstem Necrosis" for this disorder. However, we have introduced the name "Inflorescence Necrosis" (IN). Field observations show that the pedicels are the most affected tissue, along with the rachis and flowers or berries. Symptoms begin to appear just before bloom and may continue to develop for two to three weeks. It is only in severely affected clusters that the peduncle (bunchstem) becomes necrotic. Therefore, we felt inflorescence necrosis better described the disorder.

Jackson and Coombe, in 1988 at the Cool Climate Symposium in New Zealand, provided the first reports on treatments that affect the incidence and severity of IN. They found that cluster dipping with ammonium or calcium solutions increased IN. They also indicated that water- or nutrientstressed plants or shaded vines increased IN. While I was in New Zealand in 1988, working with David Jackson and Rob Sherlock at Lincoln University, we found that shaded vines (76% shade) had 31% IN incidence compared to 2% in nonshaded vines. The rachis (peduncle, main axis, and pedicel) from the shaded vines had ammonium concentrations threefold higher than those on non-shaded vines.

Upon my return to Oregon in the 1988 season, we found widespread incidence of IN throughout the Willamette Valley, particularly on Pinot noir, Riesling, and Gewurztraminer vines. David Jordan, a graduate student from New Zealand, was working on a similar problem, "water berry or shanking", a nitrogen-related disorder which occurs later in the season. He analyzed flower clusters for ammonium from fiveyear-old Riesling vines affected with IN. He found higher ammonium concentrations in the

cluster rachis with severe IN.

Two graduate students, Sanliang Gu from China and Antonio Ibacache from Chile, began their research studies on IN in 1989. Sanliang grew vines in the greenhouse under shaded and non-shaded conditions to follow the ammonium and nitrate concentrations in various tissues from bud break through bloom. A year later, he developed an incubation method to study the nitrogen metabolism of IN in Pinot noir using single-node cuttings with a leaf and cluster. Antonio, in a field study, imposed various levels of shading and girdling to determine their influence on IN, ammonium concentration of various vine tissues, and sugar levels in the cluster.

The IN levels in the field were very low in 1989, but in 1990 IN incidence was widespread in many Oregon vineyards. The 1990 season was a low-yielding year, as were 1989 and 1988. But 1987 was the most recent "normal" year in Oregon when average yields were 2.61 tons per acre, which was a reasonable yield when averaged over all the varieties grown in Oregon. Average yields in 1990 were only 1.79 tons per acre. Given a 1990 bearing acreage of 3,900 acres and an average price of \$800 a ton, this loss of potential yield in 1990 cost Oregon wine grape growers more than 2.5 million dollars.

It is difficult to determine how much of the recent low yields has been caused by IN. In 1989, very little necrosis was observed and the low yields were due to winter injury from the February 1989 freeze and rain at bloom. Fruit set was poor, but it was the typical type of unfertilized berries. In 1988 and 1990, IN was far more important. A 1988 vineyard survey found that Willamette Valley vineyards were losing 20 to 50% of their flowers to necrosis. By 1990, more growers were aware of the problem and many reported major crop reductions specifically caused by IN. Inflorescence necrosis is a major problem in Oregon vineyards. Its exact cost is difficult to determine. But considering the losses in 1988 and 1990, it could cost Oregon growers millions of dollars in lost income.

SYMPTOMS

Inflorescence necrosis affects only the flowering cluster. The necrosis can develop on a few flowers and pedicels or a portion of the rachis, or involve the whole cluster including the peduncle or the main stem of the cluster in more severe stages. The affected tissue develops a dark green color and then becomes brown or black in a few days. The necrotic tissue may remain intact on the cluster. To inspect a cluster for IN, tap the cluster over your hand and catch what falls off. The brown or black flowers and fruit result from the necrosis, whereas the light green or yellow ones are abscising due to a lack of pollination or fertilization.

Varieties most susceptible to IN have been Pinot noir, Gewurztraminer, Riesling, Sauvignon blanc, and Early Muscats. In the trellis trial at the Lewis-Brown Farm in 1990, we observed that 8% of the Pinot noir clusters had severe IN, while Gewurztraminer had 10% and Riesling had 27%. Chardonnay had only 5%. Among clones of Pinot noir, we found a range of IN severity from 25% of the cluster for Wadenswill (UCD 2A) to 72% for Colmar 538 in 1988. The Pommard clone was intermediate, with 45% IN. Chardonnay generally has not been affected. However, the berry set in Chardonnay was poor in 1990, but the symptoms were what is referred to as "hen and chickens".

IN may occur throughout a vineyard or a portion of the vineyard that is under stress from either drought or a nutrient imbalance. Excessive vigor in the vineyard can also be related to IN. In 1988 and 1990, bloom was preceded by a month of wet weather, but the bloom period was relatively dry and warm. We have observed more IN in canopies with upright shoot growth than in hanging canopies, and we have seen some differences among other canopy systems. For instance, in the trellis plot at the Lewis-Brown Farm, we found the upright systems (cane and cordon) had more IN than the divided canopies, and the

GDC with hanging shoots had the least IN.

RESEARCH

A nitrogen imbalance was suggested as a probable cause based on our earlier work in which we found that high concentrations of ammonium in the rachis were related to increasing IN severity. This relationship between ammonium and IN has been the key to IN research at OSU. Basic research concentrated on ammonium metabolism, transport, and concentration in various tissues of affected grapevines, whereas the applied research dealt with various methods to reduce IN in the field.

Antonio Ibacache's research on field vines at Beaver Creek and Woodhall III Vineyards on Pinot noir used shade cloth to increase IN incidence and tried 3 treatments to reduce IN by increasing carbohydrate levels. It was hypothesized that IN could be due in part or in whole to a lack of carbohydrates. Dave Jordan had shown that a carbon source (a-ketoglutarate) could be used to reduce ammonia levels and necrosis in a model system using grape tendrils. In the vine, ammonia is assimilated, or "detoxified", by combining with a-ketoglutarate to form amino acids. Antonio's treatments were: 1) shoot growth reduction by vigor diversion (leaving on 2 extra canes per vine); 2) shoot thinning prior to bloom to increase light exposure; and 3) girdling of the trunk, cane, or shoot (removal of a bark strip) about 10 days before first bloom. Girdling is common in table grapes to improve set and berry size.

In 1989, Antonio found 60% shade cloth on Pinot noir vines during bloom increased IN about 2.5 times, reduced fruit set 29%, and increased cluster ammonium 23%. Trunk and cane girdling of Pinot noir vines that were shaded or unshaded prior to bloom had little effect on IN or tissue ammonium, but girdling did increase set 26%. The girdling increased soluble sugars, particularly glucose, and tartaric acid in the rachis at flowering and shatter, whereas shading reduced sugars and acid levels.

In 1990, vigor diversion or shoot thinning of Pinot noir vines to reduce vine growth and to improve light exposure had no effect on IN or ammonium levels. Girdling blocks the flow of carbohydrates in the phloem. Girdling below a cluster blocks the movement of carbohydrates moving from the leaves to the trunk and roots and generally causes an increase in cluster carbohydrate concentration. Girdling above a cluster prevents the cluster from receiving any carbohydrates from the leaves and would generally reduce carbohydrate levels in the cluster. Girdling above the cluster resulted in increased ammonium levels and IN, suggesting that carbohydrates are indeed involved in IN development. But girdling below the cluster did not reduce IN incidence, even though some carbohydrate levels were higher. Either the carbohydrate increase was not enough to offset the high ammonium levels or the carbohydrates were being used for other purposes in the cluster.

Antonio found that the rachis accumulates much higher ammonium levels (7 to 9 mg/g dry weight) than the tendril, petiole, and flower tissues (1.5 to 3.5 mg/g). The severity of IN correlates best with rachis and petiole ammonium concentration at beginning bloom. Ammonium levels peaked in the rachis at beginning bloom and decreased gradually afterwards. Although shading increased IN, increased canopy exposure and trunk or cane girdling did not reduce IN.

Sanliang Gu's research on greenhouse and field vines looked at the conditions and nitrogen sources that can cause IN, and also possible sources of ammonium in the vine. Sanliang found that 60% shading increased ammonium and nitrate nitrogen in all plant tissues. Shading just at bloom was as effective as shading prior to and during bloom to increase IN incidence. Water stress just before bloom, as well as shading, increased IN incidence, but water stress increased fruit pedicel necrosis only, whereas shading increased primarily flower pedicel necrosis. Water stress and shading increased tissue ammonium, whereas only shading increased nitrate concentration in the petiole and rachis.

Sanliang developed a model system to look at sources of ammonium and the relationship of nitrogenous compounds. He found that by placing the basal end of field-grown Pinot noir cuttings (containing a single node with a leaf and flower cluster) in various nitrogen solutions, he could induce IN symptoms. Only solutions containing ammonium developed IN. High nitrate solution caused only a marginal leaf burn. When he added a carbohydrate source to the ammonium solution such as α -ketoglutarate or glutamate, no IN occurred, again demonstrating the ability of carbohydrates to assimilate ammonium. High nitrate solutions did not result in an increase in tissue ammonium levels, indicating that there was no nitrate reductase activity in the model system.

Sanliang used this system to test the effects of ammonium assimilation inhibitors. These inhibitors block key enzymes in the nitrogen assimilation pathway. The results of the study suggest that the enzymes necessary for ammonium assimilation are either not in the rachis or petiole or they are at very low levels.

In another study, Sanliang found that when potted Pinot noir vines were fed ammonium fertilizer instead of nitrate forms, ammonium concentration increased in all tissues except tendrils. This suggests that the source of the elevated ammonium in the inflorescence as well as other tissues could be from the soil. High soil ammonium could be caused by conditions such as a shift in soil pH, temperature, water content, or soil aeration that affect mineralization or nitrification of organic matter in the soil. Mineralization is the processes that converts organic matter to ammonium and nitrification is the conversion of ammonium to nitrate. Both of these processes are carried out by soil micro-organisms. Nitrification is known to be inhibited by wet, cold, anaerobic soils.

CONCLUSIONS

In summary, high ammonium levels accumulate in the rachis under shading, drought, and/or high soil ammonium conditions. Cultural methods to reduce shoot growth, to increase canopy exposure, and to increase carbohydrate supply failed to significantly reduce IN. The incidence of IN appears to be associated with ammonium toxicity, but the source of ammonium is still unknown, although nitrogen metabolism and/or soil mineralization may be involved in the supply.

Future work will continue to evaluate the question of ammonium toxicity and ammonium metabolism. We will also expand our research to include the soils questions as well as some survey work on the effects of rootstocks on ammonium levels in vines.

The IN work at OSU was presented at the Nitrogen Symposium prior to the annual ASEV meeting in Seattle on June 19 and 20, 1991 by Dr. David Jordan, Antonio Ibacache, and Sanliang Gu.