Grape Bud Fruitfulness

What causes a bud to produce a bunch, or not?

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Grape fruitfulness is defined as the percentage of buds that produce one or more flower clusters. Fruitfulness is determined within the developing buds during the year prior to a crop. First, primordial clusters, called inflorescences, either do or do not initiate in each bud. Later, the fruitful buds may be lost through plant-mediated necrosis or from various injurious processes.

Fruitfulness may be estimated prior to pruning by inspection of a sample of buds, allowing a grower to optimize fruit load by leaving the best combination of spur length and spur or cane numbers.

The path to fruitfulness may be visualized as a series of processes beginning at bud break the year of bud formation and ending at bud break the following year when the buds sprout and produce flower clusters:

Inflorescence initiation. Formation of flower clusters begins within each developing bud when certain tissues begin to form either an inflorescence primordia or a tiny tendril. This occurs early in the spring, when the bud is only a few nodes away from the growing tip.

Plant-mediated necrosis. Some time after inflorescence initiation in some buds the inner section dies above an apparent abscission layer. This is clearly a physiological process initiated by the plant. The plant "decides" that a bud is not worth the metabolic cost, and "tells" the bud to die. Secondary buds on either side of the primary bud usually survive. This kind of necrosis seems to peak in the first couple of months following bloom.

Injury-related necrosis. Injurious environmental factors such as pathogens, heat, water stress or frost might damage a bud and cause it to eventually die.

Bud mites. A strain of the Erineum mite, *Colomerus vitis*, grape bud mites infest only buds. These tiny worm-shaped mites move from old to new buds during the first couple of weeks after bud break, and multiply all year. During inspection the following winter we may find hundreds in a bud, where they can damage or destroy the inflorescences.

Inflorescence Initiation

The "bud" which occurs at each node on a cane is actually a compound bud typically composed of three simple buds: a primary bud in the middle and a secondary bud on each side. For most varieties, under California conditions, the primary bud may have up to three flower clusters, or inflorescences, but more typically there are two. Thompson Seedless buds usually have one inflorescence, sometimes two. The secondary buds often have no inflorescence, but sometimes have one or rarely two; these being smaller than those in the primary bud. Inside

each of the three buds is a 6 to 12-node compressed shoot with primordial leaves, flower clusters and tendrils. Buds that push at bud break become shoots. [1,3,4]

Sometimes two complete compound buds develop at the same node, approximately equal in size and parallel with each other and the direction of shoot growth. This has been noted most commonly in Pinot Noir, but also in Cabernet Sauvignon, Syrah, Merlot, and occasionally Chardonnay.[2]

The compound buds develop sequentially as the cane grows. Each compound bud develops over a 2-3 month period that starts soon after its node first appears at the tip of the growing shoot. The bud then stops growing and remains dormant until bud break the following spring. Most of the branching of the rachis occurs in the flower cluster primordia before the buds go dormant in summer, but the flower initials, which become the actual flowers along the rachis, form after winter around bud break. This means that conditions at bud break influence the final number of flowers formed. Otherwise the size of the flower cluster at bud break is determined the prior year. [1,3,4]

Each flower cluster primordium develops from a tiny lump of tissue called an "anlage". Each anlage develops into either an inflorescence (flower cluster) or a tendril. The inflorescence can be distinguished microscopically when the bud is about 10 nodes from the tip of the growing shoot, but the "decision" to form an inflorescence comes much earlier. For example, the correlation of temperature with inflorescence formation is strongest in the third visible node from the apex, which has a leaf of only about 1.5 cm. in diameter. [1,3,4]

In other words, bud fruitfulness in the first few buds is determined by vine physiology and the environment of the shoot in the first weeks following bud break. All of the two to five buds that will constitute next year's spurs differentiate before bloom. In spur-pruned vines, this is next year's entire crop. For cane-pruned varieties, next year's crop will include buds that differentiate further out on the cane, so they pass through the critical environmental conditions at a later time. In Thompson Seedless, differentiation in the first 15 nodes is determined by around the middle of June.[4]

Several factors influence inflorescence differentiation. Some of these factors are well documented; others are merely suggested by the evidence.

1. Temperature - Both cold and hot temperatures inhibit inflorescence, but in the weeks following bud break, cold temperatures are the threat.

In Muscat grapes, no flower clusters were formed on vines in growth chambers kept at or below a constant 20^{o}C (68^{o} F). The maximum percentage fruitful was at close to 35^{o}C (95^{o}F), and the number declined steeply above that temperature. Different varieties respond at higher or lower temperatures, but the general pattern is the same.[1]

Some Australian studies suggested that the critical factor is the number of days with maximum temperatures between 82^{o} and 90^{o} F during key time periods, corresponding roughly to the second through fourth weeks of May for Thompson Seedless in the San Joaquin Valley.[4]

Cold spring temperatures are probably the factor most commonly causing area-wide low fruitfulness the following year.

2. Light and shading - More buds in a shoot produce flower clusters when more light falls on leaves of that shoot. Light on the leaves is more important than light on the buds, as previously thought. This seems to be more a function of total accumulated light rather than a peak of maximum intensity. In other words, a shorter day-length or several hours or days of cloudiness

could reduce fruitfulness. The Australian study noted above rated solar radiation along with days of favorable temperature as the two critical climatic factors.[4] Shade produced by heavy foliage also reduces bud fruitfulness, but this is not occurring in the first few weeks of growth.[1]

Temperatures are typically cool during cloudy periods, and the two factors in combination can be expected to reduce fruitfulness more than either alone.

3. Carbohydrate reserves - Stored carbohydrates are thought to have a strong influence on the differentiation of inflorescences in young buds.[2] Carbohydrates are stored in the roots, trunk, cordons, and canes or spurs. These carbohydrates were stored the year before bud differentiation, or two years before the crop. This means that growing conditions and vine strength two years in the past help determine the current year's fruitfulness.

Buds seem to be a weak carbohydrate sink compared to the growing shoot.[2] Rapid shoot growth can be expected to draw carbohydrates away from the buds, reducing their fruitfulness. Indeed, vigorous vines tend to have lower fruitfulness, but this is also partly due to shading.

4. Water – Too much water decreases inflorescence; water deficit does not.[4] The affect of over-watering is thought to be indirect; water encourages excessive canopy growth, which shades the canes and draws carbohydrates away from the buds. Irrigation maintaining between 60 and 80% of full ET maximized bud fruitfulness.[4]

5. Nitrogen - Nitrogen deficiency reduces inflorescence differentiation.[2] On the other hand, too much nitrogen causes rapid shoot growth, which will pull carbohydrates away from the buds and reduce their fruitfulness.

6. Mineral deficiencies - There is some evidence that lack of phosphorus or potassium reduces bud fruitfulness.[2] Low phosphorus around bud break is sometimes caused by cold soil that prevents uptake by the roots, even if the soil has enough. Potassium or phosphorus can be added by foliar sprays. The applications would need to be shortly after bud break to increase fruitfulness in the first several buds. No experiments have yet been performed to test this idea.

7. Plant growth regulator compounds – Plants regulate inflorescence by the interaction of gibberrellic acid (GA) and cytokinins.[4] At an early stage, GA increases inflorescence by favoring formation of anlage. But later, GA decreases inflorescence by favoring the alternative path to tendril formation .[2,4] Cytokinins increase inflorescence by favoring inflorescence over tendrils. They also regulated flower differentiation just before budbreak.[4] Cytokinins are produced naturally in the plant, and they can be augmented with kelp sprays. It might be worthwhile to experiment with kelp in blocks with low fruitfulness, or in years with cold spring weather. Kelp has not been proven to contain the correct cytokinins for grapes; more research is needed.

Adding too much gibberellin can reduce inflorescences[2], but gibberellin is not normally applied soon after bud break. It is more important during the necrosis period, after bloom.

Plant-Mediated Bud Necrosis

After the anlage differentiates into either a flower cluster primodia or a tendril, the bud must survive until bud break the following year to be of any use. Depending on conditions in the

vine and the environment, the vine may sacrifice a number of buds to "bud necrosis" or death.

In plant-mediated bud necrosis (PMBN), the middle section of the primary bud dies above an apparent abscission layer. Presumably the necrosis is initiated by some plant hormone. The dead section rather quickly turns into brown fluff that breaks away from the bud when pushed. The two secondary buds usually survive. This kind of necrosis is often called primary bud necrosis (PBN), but injury can also cause necrosis of primary buds (see below).

The few studies investigating PMBN indicate that it usually occurs during the first one or two months following bloom. During this time, carbohydrates are flowing preferentially to the current season's flower clusters and young berries, leaving less for the developing buds.

Carbohydrate shortage, shading, shoot vigor, and excessive gibberellin have been shown to promote PMBN.

1. Low carbohydrates - Many studies point to the role of low carbohydrates in PMBN. Buds with lower carbohydrates levels are more likely to die. More vigorous shoots tend to have lower carbohydrates and more necrosis than weaker shoots. Grape varieties that tend to have more carbohydrates also tend to be more resistant to PMBN. [5, 6]

A study in Virginia found more PMBN in several varieties with low carbohydrate (Riesling, Syrah, and Viognier) than in one with high carbohydrates (Chardonnay). [6]

By the time PMBN starts, after bloom, the carbohydrate contribution of the leaves is presumably more important than reserves in the wood and roots. In a healthy plant the leaves will be supplying new reserves, in addition to growing the current year's fruit, and maintaining next year's buds.

In times of carbohydrate shortage, buds seem to be a low plant priority, so PMBN can be expected to increase. For example, vineyards that are heavily cropped one year often show reduced fruitfulness the following. Berry growth pulls carbohydrates away from the buds, and the plant may respond by sacrificing buds through PMBN. Indeed, the period of most rapid berry growth seems to coincide with the majority of PMBN.

Conditions that reduce available carbohydrates, including shading and excessive shoot vigor, also increase PMBN.

2. Shoot vigor - Excessive vegetative growth with fast growing canes soaks up carbohydrates so that they are not available to the buds. Several studies have found more PMBN in more vigorous shoots (thicker and with longer internodes), but the correlation is not strong, and not found in all studies.[5]

Vigorous shoots tend to have lower carbohydrate levels, because the carbohydrates are flowing to the new growth. Vegetation also increases shading, another factor that increases PMBN. Farming practices that discourage excessive growth might improve fruitfulness. However, if these same practices decrease carbohydrate production, the net effect on fruitfulness might be zero or negative.

Cutting canes in Riesling increased PMBN, but only in the buds close to the cut. The shoots were tipped 40 days after bud break at approximately 18 nodes. PMBN increased in buds 13-20. The PMBN was attributed to growth of lateral shoots which competed for carbohydrates.[5]

3. Shading - Shading is an important contributor to PMBN.[5] During the summer months, shading is typically from vegetative growth. Buds in the inner, darker parts of vines, under

heavy foliage, are more likely to suffer PMBN than those in better lighted parts. Unfortunately, the inside buds include most of those that will be selected by pruning for the following year. Leafing, hedging, and summer pruning will increase light under the canopy, and might reduce PMBN.

Several studies have found that shoot thinning decreased PMBN, which was attributed to less shading. One study found the opposite: more PMBN after thinning. This was possibly due to increased vigor of the remaining shoots.[5]

Some studies indicate that only long periods of shade increase PMBN (40 days vs. less than 21). If this is true, than clouds are not likely to be important in California, where long periods of cloudy weather are unknown during summer months.[5]

In Virginia, more PMBN occurred in rainy years; up to twice as much. Researchers attributed this partly to shading by clouds, and partly to more vigorous vegetative growth draining away carbohydrates.[5]

4. Plant Growth Regulator (PGR) Compounds - Presumably, PMBN is mediated by PGRs that respond to the physiology of the plant and the environment, by signaling certain buds to begin necrosis. The precise mechanism is not known.

Too much gibberellin can increase PMBN, although not all studies have found a correlation. Gibberellin applied early (bloom time), has a stronger effect than later (sizing sprays), and more distal buds are affected more than those near the base.[5]

Several growth retardants can decrease PMBN. Some of these growth retardants act by blocking gibberellin activity, leading some researchers to conclude that gibberellin is the principle PGR signaling buds to die.[5]

The effect of cytokinins on PMBN is not known. However, some limited observations I have made suggest that post-bloom kelp sprays, which have cytokinins, can increase fruitfulness in Thompsons the following year. The post bloom period is when PMBN occurs, so the cytokinins may be reducing PMBN. This period is probably too late for the cytokinins to be influencing inflorescence.

5. Water

Too much water has been shown to increase PMBN. This is attributed to extra vegetative growth using up carbohydrates.[5]

I have not found studies linking PMBN to water deficit.

6. I have not found any studies linking PMBN to high or temperatures. Very low temperatures are not likely to occur in the May-July period when most PMBN occurs. High temperatures could possibly cause injury-related bud necrosis (see below).

7. Excess nitrogen.

One would expect that if extra nitrogen increased shoot vigor, it might also increase PMBN. One study found that vineyards with excessive manure had more dead buds.[5]

8. Not mineral deficiency.

Several studies have found no correlation between PMBN and deficiency of minerals, including nitrogen, phosphorus, potassium, calcium, magnesium, and boron.[5]

9. Grape cultivar

Some grape varieties are more susceptible to bud PMBN than others, as we see in our bud dissections.

Most wine grapes have very low percentage necrosis in good years, in addition to being highly fruitful. Total fruitfulness is typically above 90%, and the majority of buds usually have two or more cluster primordia.

In 2005 in California, when many wine blocks had low fruitfulness, the cause was lack of inflorescence, not PMBN.

At least some varieties tend to have less PMBN in the first several buds, and more in the distal buds. Shiraz, Riesling, and a few other varieties reportedly develop more PMBN than most wine varieties. [5]

On the other hand Thompson Seedless vines are highly susceptible to PMBN. Even healthy blocks typically have only 25-75% total fruitfulness, and double flower clusters are rare. In addition, the first four Thompson buds normally have less inflorescence and more PMBN than number 5 and beyond, which is the reason why Thompsons are cane pruned.

Flame Seedless also seems to be more susceptible to PMBN than most table grape varieties. Like Thompsons, Flames generally have the most PMBN in the first few buds.

Other table grape varieties typically have better fruitfulness and less PMBN than Thompsons and Flames.

Injury-Related Bud Necrosis

The plant-mediated necrosis described above is not the only process that can lead to the death of a bud. While examining buds in November-December, our inspectors see many buds sick or dead from processes clearly not involving an abscission layer under the shoot primordia and inflorescence inside the bud. These processes are describable but not well documented. I have not seen any studies examining these other causes of necrosis.

Our presumption is that these dead or sick buds are the result of injurious processes, such as possibly pathogens, heat or water stress, toxic mineral imbalances, or frost. I will refer to them collectively as injury-related bud necrosis. (IRBN)

A recent survey (unpublished) carried out by Jose Urbez in Doug Gubler's laboratory at Univ. Calif. Davis isolated several pathogens from necrotic grape buds, including *Botryosphaeria, Penicillium, Alternaria,* and *Cladosporidium,* but more work is needed to find out which of these is actually causing necrosis. Judging by the appearance of the sick buds, there seems to be 3 or more different diseases.

In one common scenario, the necrosis appears as a tiny spot on the surface of an inflorescence, or sometimes inside. The spot has a brownish, wet appearance. The necrosis may occur at the base of the inflorescence where it joins the bud. It may also start on the vegetative tip. In other dissected buds we see the necrosis spreading to more of the inflorescence, and eventually it spreads to the whole bud, which eventually dies. This pattern of spreading is reminiscent of a rot pathogen. Our inspectors have never seen any of the filaments or fruiting bodies that are typical of fungi.

Sometimes the entire compound bud will be found badly misshaped, gathered into jagged, reddish, leathery protrusions. The majority of buds on a cane may be in this condition. By the time we see this in late fall, the cause cannot be determined. It is my guess that the cause

is some extreme stress, such as high temperatures without adequate water, or salt, or heavy mite or insect populations.

In 2007 I examined buds from a few ranches after the freeze in January. In some vineyards, the freeze did not seem to have a significant effect, but in at least one vineyard I saw damage that appeared to be recent and possibly attributable to the frost. Grape vines are a temperate climate plant and fairly tolerant of cold winter temperatures.

We cannot always tell the cause of necrosis when we examine the buds in late fall, but every year we are learning more and gleaning more information from the appearance of the buds. We can at least provide the grower with some clues about whether low fruitfulness resulted from a failure of inflorescence, plant-mediated bud necrosis, or some injurious process.

Bud Mites

Bud mite is a strain of the grape Erineum mite, *Colomerus vitis*, an extremely tiny pest in the rust mite family, Eriophyiidae. Materials registered for rust mites should work on bud mite. The Erineum mite strain, which causes white swellings on grape leaves, is rarely seen in commercial vineyards because it is controlled by sulfur. The bud mite strain hides in buds, so it is only exposed to sulfur and other pesticide for a brief period of 1-2 weeks following bud break.[6]

Bud mites cause damage from the outside in. They typically first appear inside the outer green layers of the bud, eventually working their way in to the inflorescence and vegetative tip in the middle. One bud may have hundreds of mites, but they cannot be seen without a good microscope.

Damage to the outer layers is of no consequence; thus many buds may be infested without damaging the crop. If mites reach the middle, they may start to damage the surface of the inflorescence or the vegetative tip. The damage may appear as bubbly protrusions or as hard brown scars. Damaged inflorescences may emerge and form flower clusters, and the grower may not know that those clusters would have been larger without the bud mites.

The worst infestations will destroy flower clusters and prevent emergence of the shoot. Infestations with 5% of inflorescences damaged or destroyed are not uncommon. The most serious populations can damage or destroy 50% or more of the inflorescences.

Shoots sprouting from infested buds may have short internodes and a zig-zag growth pattern. South African researchers have posted pictures.[9]

Some grape varieties are more susceptible to bud mites, and the problem might be more serious in some growing districts. We have seen few if any bud mites in wine grapes grown in the Sonoma, Napa, and Lodi districts, among others. We have seen serious infestations in table grapes from Kern, Tulare, and Fresno, and Imperial counties. Highly susceptible varieties in our experience include Flame Seedless, Autumn Royal, Black Seedless, Crimson Seedless, and Princess Seedless. Thompson Seedless is moderately susceptible, and we have seen only a few bud mites on Red Globes.

Predatory mites (Phytoseiidae) do eat Erineum mites, but biological control of bud mites is not well understood.[7] Bud mites are exposed to predators during the same short period as they are exposed to sprays, right after bud break. In addition, bud mites hide under the new leaf bracts for a few weeks while they wait for new buds to form. Predatory mites can probably reach some under the leaf bracts, and on the outer layers of the new buds.

Some materials are harmful to predatory mites, including fungicides in the dithiocarbamate class (Dithane, Ziram, Maneb, etc.), Benlate, lime sulfur, Lorsban, pyrethroids, and some other insecticides.[7] Many of these materials are typically applied just before and after bud break to control *Phomopsis*, mealybugs, or other pests. Possibly this could contribute to bud mite problems. On the other hand, we have observed reductions of about 50% in bud mite infestations the year following pre-bud break application of Lorsban, compared to nearby unsprayed rows.

In one study, some bud mites were found under the stipular scales of next year's bud primodia one week after bud burst. By four weeks, 50% of the population was protected under the scales, and by 10 weeks, 100% was in the buds.

The Australians have studied control of bud mites with sulfur. The best timing was during the week following bud break. The rate was 200g per 100 liters of 80% wettable sulfur, using "a high volume". This is about 1.75 lbs. per 100 gals. The researchers did not experiment with different rates or volumes.[7]

Two growers I work with have tried wettable sulfur just after budbreak, but we did not find noticeable reductions in bud mites the following year.

Bud Dissection and Pruning

The final step in determination of fruitfulness is pruning, which is the step most under the grower's control. Pruning can be adjusted if the grower has knowledge of the number of fruitful buds and their location on the spurs and canes. This information can come from analysis of bud dissections.[10]

Growers should be careful using bud dissection results to estimate yield. Studies have found that bud dissections may explain from as low as 50% to as high as 90% of the variation in actual yield.[4] Several years of data comparing bud dissections to actual yield will help growers interpret results with more confidence.[10]

Bud dissection sometimes strongly underestimates bud fruitfulness.[11] This suggests that tiny inflorescences overlooked by inspectors may still produce clusters. Also, a random sample of all canes on a vine may show lower fruitfulness than a sample of the stronger canes favored by pruners. Growers must be careful to take a sample representative of the canes they expect will be left on the vine after pruning.

If bud analysis is accurate, a grower can calculate the average number of flower clusters per vine for different numbers of spurs or canes. Of course, there are limits to the number available. When pruners leave more spurs or canes, they may include more of the weaker or sub-standard choices.

Bud analysis can also tell which nodes have the most fruit. If the buds on a 2-4 bud spur are unfruitful, longer spurs may be needed to include more fruit, or the grower may need to add a "kicker cane" or two.

However, because of apical dominance caused by auxins moving down the spur, adding extra nodes tends to suppress sprouting of the nodes near the base.[11] For example, if the first two buds have more fruit than the next two, keeping the fourth bud in pruning may actually reduce the number of bunches by suppressing sprouting of the first bud. The grower needs accurate knowledge of which buds will bring the best return.

Growers are also leery of adding extra nodes to spurs, because that makes the vines taller and harder to prune the following year.[11]

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Virtually all of the material on inflorescence was taken from research and literature reviews given to me by Dr. Luis A. Sanchez, who studied bud differentiation for his Ph.D. Dissertation at U.C. Davis, and now works for Gallo. The Srinivasan paper is also a good review, but Dr. Sanchez includes that information in his review. Dr. Sanchez kindly reviewed the present paper.

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