Anatomy of Grapevine Winter Injury and Recovery

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Martin Goffinet is a senior research associate investigating the structure and development of grapevines. His research relevant to this presentation includes studies of grapevine buds entering and exiting winter dormancy, anatomical changes in stem tissues during cold acclimation/de-acclimation, anatomy of vine cold injury and recovery, and the inception and differentiation of fruiting buds.

Severely cold winter temperatures can significantly impact grapevine productivity through tissue and organ destruction caused by freeze injury. Crop loss and the need to retrain vines after bud, cane, and trunk injury mean financial loss, often for one or more years. This presentation is meant to give growers information about how vines are constructed, what tissues are susceptible to cold, the process of vine cold acclimation, a description of cold injury in the various organs, and the mechanisms the vine uses to heal (if possible) cold-injured structures.

This discussion is picture-based and will include the following topics:
- Living cells and freeze injury in grapevine cells and tissues
- Seasonal structure and development of the shoot and cane
  - The green shoot
  - The maturing shoot
- Shoot (cane) acclimation to cold
- Anatomy of dormant trunks
- Cane freeze injury and repair
- Trunk freeze injury and repair
- Development of the overwintering bud complex
- Bud survival and relationship to cane and trunk repair

Living cells and freeze injury in grapevine cells and tissues

[Images and diagrams related to grapevine cells and tissues are shown here.]
There are several concepts to consider when looking at cells and cold injury in grape tissues:

1) Functional cells are alive and almost all cells in grapevine are living. **Examples above at left** include pith cells in stems and green cortex cells in stems. Indeed, except for the dead water-conducting vessels in wood and the dead, waterproof cork in the external bark, grapevine cells are living and packed with subcellular organelles that are physiologically specialized for growth and development.

2) Living cells are not only themselves “compartmentalized,” but they contain many compartmentalized membrane-bound organelles (**drawing, upper right**); freezing disrupts these compartments and the control they have on stabilized metabolic function. Cells then leak fluids and eventually senesc and die. (e.g., frozen cortex cells, **bottom right**).

3) Cells at maturity have specialized functions, depending on location in the plant. Cells are interconnected into tissues and several tissues into organs having specialized function. Cell death thus may be an isolated event or many cells may die in a localized tissue region. With severe freezing, whole organs can die.

4) A dead cell, caused by freezing or some other agent, has lost function and can never be repaired; repair must come from renewed cell division and differentiation in nearby living cells. Extensive injury to whole tissues or organs creates serious disjunction that can impede or even prevent cold recovery.

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**Seasonal structure and development of the shoot and cane**

**The green shoot**

The green shoot in spring and summer is not cold hardy and its cells will freeze at only a few degrees below the normal freezing point of water. The cells of leaf tissue contain 80–90% water, while the woody stem tissues contain about 50%. To understand how the shoot becomes tolerant of subfreezing cold, we will begin by looking at the development of the green stem internodes from summer to autumn. First, we’ll see how green internodes near the shoot tip are constructed then see how these will look when they start to become “woody.”

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**Above** is an example of a green Chardonnay shoot taken on 3 July. We will compare a green internode slice 2-3 leaves back from the tip (**Arrow A**), and then look at an older more woody internode (**Arrow B**). Sections of these are shown in the next photo group.
The green internode contains a complex of cell and tissue types that cause tissues to change over the course of the season. The two photos above indicate two “stages” of these changes and relate to the positions denoted by arrows in the previous figure. **Left Photo.** Cross section of a small sector of elongating internode (internode A, previous photo), showing major tissues. Note that the vascular system is organized into “bundles” having the water-conducting xylem tissue toward the inside (pith side) and the food/organic materials-conducting phloem tissue toward the outer side. Outside the young phloem lies the living cortex and epidermis. Leaves on developing shoots stimulate cell divisions in the vascular cambium. The resulting daughter cells develop into new xylem cells and new phloem cells. These two tissues move materials in a strongly polar way (i.e., “up and down”), while groups of cells separating each vascular bundle (the rays) serve to move materials radially and also help to isolate or compartmentalize each vascular bundle. **Right Photo.** Cross section of a more basal internode that is no longer elongating but which is growing thicker (internode B, previous photo). Thickening is due to the continued activity of the vascular cambium, which has by now produced a continuous ring of xylem vessels and fibers, a smaller amount of phloem, and which has allowed rays to elongate and new ones to form. The cortex is outside the phloem groups and in real life is still green, although in these two photos the cells have been stained for clarity.

**The maturing shoot**

In late summer and fall the exterior of the basal internodes turn brown. Growers refer to this browning as “wood maturation” or “periderm” formation and associate its early and intense formation in stems with the development of cold acclimation.

**Left photo above.** Example of external browning (the 2 left internodes) along a Concord shoot in late summer. **Right photo above.** Internal anatomy of a stem sector taken from a brown internode. Here we see the outermost xylem, the vascular cambium (now inactive and very thin), the phloem (showing bands of fibers alternating with large food conducting elements, and a senescing brown cortex underlain by a new tissue, cork, which together with the brown cortex is counted as the periderm by growers. The periderm actually arises as a thin layer of cambium-like cells in the outermost phloem and it divides to produce rows of cork cells that provide a waterproof barrier that seals off all living tissues inside of it. Thus, what growers call “wood maturation” is really a browning of the outer internode tissues. True “wood” is the hard ring of xylem tissue to the inside of the vascular cambium.
**Shoot (cane) acclimation to cold**

By the time many internodes have developed periderm, leaves have already begun to yellow and fall from the base of the shoot. As this process continues, acclimation of internode tissues to cold intensifies. Changes, both structural and physiological, include:

- Diminished rate of leaf production and in the activity of the vascular cambium.
- Progressive leaf senescence and redistribution of materials.
- Senescence of the stem’s cortex and carbohydrate (sugar and starch) redistribution into canes, cordons, trunks, and roots.
- Initiation of a cork cambium and its production of cork cells (“periderm” formation).
- Dehydration of cells and increase in their freezing tolerance.
- Plugging of phloem elements with callose deposits.

**Anatomy of older woody stems and trunks**

The photos above show that older woody stems, such as 2-year canes, spurs, cordons, and trunks, lay down a new layer of xylem (a xylem ring or annual ring) each season. Counting xylem rings tells the age of such stems. Similarly, one ring of phloem is produced each year, but because older phloem is cut off to become part of the outer string bark, only a single layer of functional phloem can be found in a stem of any age. In the photos at right we removed the old stringy bark to show that what remains of phloem is only one functional increment that was added by the vascular cambium by the end of each year. **Left photo above.** Note that in the cane internode in spring a sequence of events has to occur before new vascular rings can be produced. First, the vascular cambium must become reactivated. That happens as sap rises, temperatures increase, and as emerging new shoots begin to produce substances that stimulate cell divisions in the cambium of the cane node nearest each shoot. Second, after cambium activation, the first cells to be produced from the cambium are new xylem vessels and a xylem fiber matrix. The new vessels are needed to serve rapidly expanding leaves. New phloem cells are produced by the reactivated vascular cambium much later than new xylem. The vine has adapted to this: the food conducting elements in last year’s phloem renew their activity in spring to serve the food- and nutrient-transport needs of the new shoots.
Cane freeze injury

Mid-winter freeze injury to grapevines in New York State typically can be seen in both buds and canes. Such injury often produces a grayish-or brownish-green color with a “water soaked” appearance. Cell membranes have been damaged so that cell organelles are no longer compartmentalized. Cell solutes “leak,” become oxidized and discolored, and ultimately the affected tissues brown or blacken.

**Left photo above.** In the Riesling cane cross section we can see brownish coloration most intensely in the outer tissues, which of course is the increment of phloem laid down last season. The xylem is injured in some sectors but not to the same extent as phloem.

**Upper right.** A thin slice of a Concord cane internode in winter showing normal unfrozen phloem between xylem fibers and the outer periderm cells.

**Lower right.** Slice of Concord cane internode in November that was artificially frozen to -28°C, showing significant freeze injury to the phloem tissue, some injury in the dormant cambium region, but insignificant injury to the xylem tissue.

**Above** we see an example of Niagara cane freeze injury following a late-May subfreezing spell in Fredonia, NY. At that time green shoots had emerged from this cane and, so, they too were injured. Note again that phloem is the most injured tissue in the cane and that it has been frozen from an out-to-inside direction. In late May most living cells will have a high water content, so freeze susceptibility is great. Note in this slice that we left intact the old bark (cortex and sloughed oldest phloem) on the outer side of the periderm barrier.
Repair of cold-injured cane internodes

Frozen phloem and dead cambium cells cannot be repaired; however, they can be replaced via a reorganization of nearby cells and tissues. Examples are shown below.

Left photo above. This section of cane internode shows what happened after a “girdle” of liquid nitrogen was applied to it in winter. The phloem was killed and the outermost xylem severely damaged. Nonetheless, some cells in the phloem, perhaps ray cells or phloem parenchyma cells, or perhaps some uninjured cambium or xylem ray cells survived. These cells divided to produce an unorganized tissue, callus, in springtime and this callus began to spread around the xylem as a wedge. The oldest callus tissue is at the top. Note that a few small vessel elements (arrow) have differentiated there. So, it takes time for normal vessel elements and other vascular tissue to differentiate, and this differentiation depends in turn on a stimulus moving downward to these stem regions from emergent shoots.

Right photo above. This section shows a small sector that was freeze-injured in winter after the first growing season. The inner, first-season xylem ring is OK but shows dead tissue just outside it. Thus, the cambium and phloem was injured. Note that after callus production and vascular “recovery” the second year, a significant sector of new xylem is still poorly differentiated, even by the end of the season. Lesson? That it takes not just cellular reorganization and differentiation to occur, but it takes time. If enough sectors are injured, vascular function can be seriously impaired, thus impairing shoot emergence and also the annual food- and water-conducting function of the vine. Poor shoot growth, in turn, fails to stimulate development of new vascular tissues in the stems.
Freeze injury to older stems and trunks

As in canes, winter freeze injury in spurs, cordons, and trunks is first evidenced in the outer food-conducting tissue, the phloem. As temperature drops the injury progresses out-to-in toward the vascular cambium. Below we see injury examples. The woody tissue (xylem) is less susceptible to freezing, although severe cold events can also damage that tissue as well.

Upper left photo. This Chardonnay spur has been injured by cold, and we see some discoloration (“water-soaking”) in some sectors, especially in the outmost ring at the surface (arrow), which is the phloem.

Lower left photo. Here we see a Chardonnay trunk in April in Washington State after a deep February freeze. I have removed the stringy bark to show a small “water-soaked” spot (arrow). This is evidence that the phloem inside the most recent periderm has lost cell integrity and the trunk has become “leaky.”

Right photos. top to bottom: This series of three different trunks shows progressive freeze injury in the phloem tissue, but no injury to the xylem (tissue with large open vessels). Note out-to-in progression of injury, which helps explain the “water-soaked” areas seen by peeling back the outer bark. If the inner half of the phloem becomes injured, chances of full vine recovery is not likely. Although not shown in the photos, by the time the inner phloem is injured, xylem injury has also likely occurred, starting at the pith and moving outward [See next photo sequence].
Assessing trunk cold injury in the field

As exemplified in the photos below, growers can get a pretty good idea of the extent of trunk cold injury by making slices into the trunk, although browning of tissue may not be obvious until temperatures rise above freezing for several days. The higher temperatures increase rate of cell respiration, which means damaged membranes and organelles form brown reaction products much faster, but also such temperatures cause rapid chemical reactions within the oozed and spilled-out cellular substances and increase oxidative reactions.

Left photo sequence above. Freeze injury to trunks can be assessed in the field by making a series of ever-deeper slices. Upper left photo shows dead and dark-colored bark, including what should have been functional phloem tissue. Just inside the darkened phloem is the yellowish-colored wood (xylem), which in this photo is uninjured. Photos 2 to 6 show a series of slices (out-to-in) indicating that the phloem is dead, the outermost xylem near the cambium is relatively OK, but that deeper xylem regions have also browned and are thus severely freeze-injured.

Right photo. This photo of a young Concord trunk was taken in April 1996, after a severe killing freeze in February in Eastern Washington State. Three things should be noted. The first is that there is significant freeze injury on the trunk’s left side. The second is that this photo was taken looking directly north, so that the injury is seen only in the southwest part. This indicates injury relates to sun exposure, especially exposure at the hottest part of the day, the afternoon. In this case, at the time of the February freeze there was snow up to the knot seen in the lower part of the photo. In Eastern Washington, where there may be snow coupled with high light intensity, the trunk can easily warm above freezing, both by direct sun and by reflection off the snow. This “black body” effect of trunk warming in winter can de-acclimate tissues, so that sub-freezing nights or sudden cold spells can freeze-injure those tissues. The third thing to note is that the vine has a way of sharply isolating injury. Note the clean-cut demarcation of good vs. bad tissue. The grapevine is very good at compartmentalizing injured tissues.
Examples of the “black body” effect

Dark colors of plants are quite good at absorbing the heat of the sun. As shown below, the basswood twigs on the left and the yew branchlets on the right have absorbed sunlight and have heated enough to melt the surrounding snow. They have sunken into the snow, even though the noontime outdoor temperatures were below freezing the entire week up to the time I took this photo. Parts of these twigs had sunken up to three inches deep. When this heating occurs in grapevine trunks, we expect the bark temperature to be above (sometimes well above) freezing during the daytime. In late winter such warmth can rapidly de-acclimatize the grape tissues and the cells begin to undergo respiratory activity. Such cells are no longer dormant and so can be severely injured after the sun sets and/or air temperatures drop below the cell freezing point.

Freeze injury of de-acclimated trunk tissues in late spring

The photos below show a Niagara trunk that was injured when a late-May freeze occurred in the Fredonia, NY region in 2003. Besides the expected freezing and death of the emergent shoots, this freeze was also a potent killer of conductive phloem tissues in the trunks.

Upper photo immediately above. A portion of the trunk cross-section. Note the brown senescent phloem between the xylem and the outermost cells (periderm). I have stripped away the old stringy bark to allow a good section to be made.

Lower photo. A very small section enlarged to show the injury to the “soft phloem,” which is the vertically conducting tissue for carbohydrates and other organic materials. These cells can no longer function to provide the canopy with nutrients from storage tissues in roots and trunks, nor provide movement of photosynthates from new leaves back into the stems and older organs. Of course, the leaves are no doubt heavily injured or dead, because during shoot emergence the leaves are even less cold tolerant than the trunks.
There must be continuous vascular connection between emergent leaves and the root system if the vine is to produce its normal seasonal growth and cropping. Tissue failure in any part of the system can decrease vine growth, productivity, and timely movement of nutrients through the vine.

**Repair of cold-injured spurs, cordons, and trunks**

As in frozen canes, the frozen phloem, cambium, and xylem cells in older woody organs cannot be repaired. However, they can be overgrown and replaced by a reorganization of nearby cells and tissues, as shown in the accompanying photos below. When the phloem and cambium are only mildly freeze-injured, but bud break and shoot growth are weak, the cambium will reactivate in spring but it may not produce highly developed new vascular tissues.

**Upper left photo above.** Here we see some freeze-injured conductive phloem at the top (arrows), but the inner phloem below it and the cambium seems unfrozen. The canopy supported by this trunk is probably not developing well, because there is only a weak development of small, isolated groups of new vessel elements (arrowheads).

**Upper right.** In this Riesling cordon there is significant phloem injury down to, but not including, the active cambial zone, sometime during spring, after shoots have already emerged. This destroys active movement of carbohydrate in the phloem, further limiting nutrient flow into those shoots.

**Lower left.** This cane shows the repair of a trunk whose bark and xylem were killed by a shot of liquid nitrogen. The non-frozen area to the left shows a normal ring of new xylem, whereas the frozen sector at right has occluded old vessels (arrowheads) and all new xylem above last season’s cambial zone is malformed (see brackets). Tissue repair takes time and again depends on a connection to new shoots. The new shoots must send hormonal signals downward to the reactivated cambium each season for new xylem differentiation to occur.

**Lower right.** Here we see what happens after severe injury to trunk cambium and bark. Recovery depends on production of callus tissue that fills in the space between dead bark and xylem. Here the filling-in is “moving” in a right-to-left direction. Callus production depends on proliferation of unfrozen cells near the cambial zone and the stimulus of emerging shoots. Here, this stimulus has allowed production of vessels in the new xylem (arrowheads), but these first new vessels are small in this case and not very efficient movers of water.
Grapevine buds, bud cold hardiness, and importance of bud break to repair of cold injury

*Bud structure*

Buds that will overwinter begin their development in spring and summer at the nodes of green shoots that are still rapidly elongating. Also, this is a period when current clusters are flowering and setting fruit. The photo below shows that these (“compound”) buds develop deep within the angle (axil) where a leaf petiole meets the main stem, at the base of a small emergent summer lateral shoot. In the sample we see that this particular shoot node has a leaf, an opposing flower cluster, the summer lateral, and the compound bud. Typically, the summer lateral produces one expanded leaf and by summer’s end the whole lateral dies, leaving behind the compound bud to overwinter. This bud, at about the stage shown below, is already initiating clusters for next season’s shoot, which erupts from this bud at bud break in spring.

![Organography of the Fruiting Node of Grapevine](from Wine East, 2001, by permission)

*Overview of the overwintering compound bud*

The complexity of the summer bud and its relationship to the development of the dormant overwintering compound bud is depicted in the six images below.

![Overview of the overwintering compound bud](from Wine East, 2001, by permission)
A, previous page. This is a schematic of the shoot seen in the previous figure, with a “blow-up” of the inner architecture of the compound bud. Once the summer lateral dies, only the compound bud is left behind for the upcoming winter. This bud is actually a highly branched short shoot covered with some bud scales (in black) that protect the main shoot (primary bud) of this bud system. Two of the bud scales each have a more or less developed bud at the scale base, the lower being the “secondary bud,” the upper being the “tertiary bud.” The main (i.e., primary) bud usually has many embryonic leaves and two or more cluster primordia for next year: it is the main bud growers want to retain for cropping. Injury to the primary may cause the secondary to emerge, but the secondary typically has smaller clusters than the primary bud.

B. Outer morphology of the green node of grapevine in mid summer.

C. Early fall node of grapevine showing a slice through the compound bud with its three major “buds,” the primary (central bud), the secondary (lower bud nearest the leaf base of the node), and tertiary (smallest bud opposite the secondary).

D. Face view of matured compound overwintering bud on a matured shoot (cane), showing reference of parts to the summer node.

E. Median vertical slice through the overwintering compound bud, showing the relative size of primary, secondary, and tertiary bud components.

F. Spring bud break showing normal emergence of the primary bud. Secondary and tertiary buds may or may not break to form shoots; commonly, they do not.

(This figure published in modified form in Wine East. See References)

Bud cold acclimation and winter survival strategies

Grapevines have several strategies for adjusting to subfreezing conditions:

- Dense, compact and lignified cells at the bud base (bud cushion) inhibit perfusion of water and solutes.
- The presence of a “barrier zone” just below the primary bud’s stem isolates the compound bud from the cane.
- Canes are likely to have vessel plugging or may be drained of water.
- Vessels move water from cane node to cane node, yet vessel endings occur frequently at these nodes, thus further reducing avenues of water entry into the bud. This is accompanied by the fact that there are very small and/or discontinuous vessels within the small embryonic organs of the bud (“vascular restriction hypothesis”).
- Cell and tissue “supercooling” occurs as sugars and organic solutes increase in concentration in buds, while simultaneous cell dehydration and membrane stabilization occur.
Bud ‘redundancy’ as a survival mechanism

If all else fails and freezing of tissues occurs, buds have many accessory growing points (bud meristems) that can allow production of new shoots, as long as those meristems are not themselves freeze-killed. Note in the two examples of compound buds shown below, that each bud scale and each embryonic leaf has a very small branch bud tucked deep inside. During dissection of the buds used in these drawings I counted about 13 total growing points in the Concord compound bud and about 17 in the Vitis riparia. Tiny and undeveloped buds are more resistant to freeze injury than are the primary bud (most cold susceptible) and the secondary (next most susceptible) and tertiary buds. These accessory buds usually do not emerge in spring and they and the secondary and tertiary buds can lie dormant for many years, deeply embedded and overgrown by seasonal growth rings. These hidden buds can be seen each year as suckers, water sprouts, or as small shoots coming from older “wood.” Thus, the vine has much bud redundancy and great renewal potential for vegetative growth (survival by duplication). (Left-hand drawing from Wine East, 2001. See references.)

![Winter Bud Arrangement in Two Grapevine Species](Concord from Wine East, 2001, by permission)
Example of summer development of the shoot and the flowering clusters within its buds

The topmost plot in the stacked graphs below shows the production of leaves, leaf maturity, and periderm formation (based on cane browning) on long shoots of Concord. Node positions are exemplified in the top-right shoot drawing. The primary bud in each leaf axil (i.e., petiole-stem juncture) will initiate three potential flower clusters before winter (depicted in the bud diagram on the previous page as three white scalloped areas). Cluster 1 is the largest and most basal cluster in developing Concord buds and cluster 3 is the smallest and most distal cluster. The developmental stages of these clusters in the first season is defined in the middle right-hand drawings. The three clusters develop in buds at shoot node 5, 10, and (later) at node 15 and 20 over the time-course shown in the bottom four graphs and in the bottom-right photo, which shows the primary bud’s shoot apex (A), some labeled embryonic leaves (L), and the three embryonic clusters (C1 to C3). Cluster 1 is at stage 6, cluster 2 at stage 4, and cluster 3 is at stage 4. (Drawings of grape shoot and six cluster stages modified from Wine East, 2001, with permission.)
The descending arrows in the five stacked graphs on the previous page show the relationship between the time when all 3 clusters at shoot nodes 5, 10, 15, and 20 have finished their development for the year and the progress of periderm formation along the shoot. Note that all three cluster primordia inside buds 5 and 10 are fully formed before periderm passes their nodal position, but that all clusters within buds 15 and 20 reach their maximum development as periderm reaches their bud location. Note also that at the end of the season maximum cluster development in buds 15 and 20 is significantly less than that in buds 5 and 10.

**Relationship between wood maturation and bud acclimation to cold (cold hardiness)**

In the late 1980s, Robert Pool, Mary Jean Welser, and I studied autumn cold acclimation of buds along shoots of Concord, Cabernet Sauvignon, *Vitis riparia*, and *V. rubra* with reference to periderm formation. The schematic below indicates that as periderm “moved” up the shoot (arrows), the buds on the proximal (brown) side of this “wave front” became increasingly cold hardy and thus capable of supercooling to increasingly low temperatures before bud freezing occurred. Green or yellow-green stem tips could not fully acclimate and died back to “ripe wood” at hard frost.

(from Wine East, 2001, by permission)
Examples of bud cold injury

The photos below were taken of winter buds that were sliced to reveal whether cold injury had occurred. “Water soaking” and browning or blackening of tissues indicate freeze injury. One must be mindful that when plant tissues are frozen they will not readily brown until re-warmed from their frozen state. Thus, in assessing cold injury to buds, growers will cut canes after a suspected damaging freeze and bring the canes to a warm room for a day or two before slicing them to assess cold injury or death.

A. Normal bud, longitudinal slice exposing primary bud (P) flanked by the secondary (S) and tertiary (T) buds. B. Primary bud is rust-brown, thus cold injured, while secondary bud is uninjured. Recovery of the primary is suspect. C. Primary bud is dead and black, while the secondary bud looks fine. The secondary should emerge in spring to take over shoot production at this bud’s node position on the cane, but any crop from secondary shoots will not be as good as that of the primary shoot.
Bud survival and relation to cane and trunk repair
Examples of shoot emergence from cold-injured buds and implications for vine development

The two photos above demonstrate recovery after bud cold injury. **Left photo.** Cold-injured buds create “blind nodes” and require cultural adjustments due to lack of canopy fill and poor positioning of shootless nodes. Also, some shoots may grow for a while, and then collapse if there is also freeze injury to the subtending cane tissues. Such tissues do not transport water and nutrients, thus furthering shoot collapse. **Right photo.** Semillon cane cuttings taken from vines freeze-injured after a severe freeze in February 1996 in Washington State. Cuttings were put in a greenhouse to determine the percentage of cuttings that would re-grow shoots. In this example, many buds failed to emerge, signifying bud death and/or severe cane freeze injury. Shoots that emerged from such injured canes and buds were stunted, died, and few-to-no roots were seen at the base of the cuttings. Strong shoots resulted when little-to-no bud and cane injury occurred, an in such instances strong rooting also took place. This indicates the strong interdependence of buds and live cane tissues for the full unrestricted growth of emergent shoots. Whole-vine growth depends on undamaged connections between emergent buds, the canes, cordons, trunk, and roots.
Importance of bud break and shoot emergence to whole-vine vascular activity

The photos below exemplify the relationship between bud break and shoot emergence to whole-vine vascular activity during the season.

Left photo above. This longitudinal view through the cane node and emergent bud was stained to show cambium reactivation. Note that as the shoot emerges from the primary bud it stimulates the dormant vascular cambium to become reactivated just below the shoot.

Top right. This longitudinal slice shows that as the shoot grows larger and as leaves expand, the vascular system inside the primary shoot develops (arrowheads), but also the cane node now has strong vascular strands (arrows) developing in the downward direction in response to hormonal influences from emerging leaves.

Bottom right. This cross section shows what happens in the cane internode below emerging shoots. Increasing leaf area stimulates the reactivation of the vascular cambium on the shoot side of the cane. Note in the photo that this has stimulated a horseshoe-shaped arc of new vessels to be produced just outside of the thick 1-year-old xylem produced last season. This shows that new shoots aid cane growth, but it also means that you need unfrozen buds and healthy cane tissues if the seasonal development of new growth is to occur in older parts of the vine.
Example of spotty canopy development in late April after a severe freeze in Washington State in February 1996

As shown above, after a debilitating freeze, a lack of canopy fill can result from bud injury, cane injury, or even cordon or trunk injury. With proper soil cover, root injury may not occur, but it can happen with very deep freezes. As freeze injury is compounded by involving more and more vine organs, recovery becomes less and less likely. Full canopies after a freeze result from low injury levels; partial canopies result from at least some injury to buds and/or woody organs; weak canopies nearly always reflect heavy bud loss and associated freeze injury to the phloem and cambium regions of the perennial woody organs. No matter how much canopy results, the continued development of shoots must depend on an uninterrupted living vascular system between that canopy and the root system. This uninterrupted network need not involve the entire vascular cylinder in the woody parts of the vine. Indeed, only a small unfrozen sector of cambium, phloem, and xylem is necessary for shoot survival, as long as there is a direct vertical connection between shoots and roots.
Importance of interconnected, unfrozen viable tissues between emergent shoots and the roots

There must be a viable vertical connection of the vascular system from emergent shoots to the roots if the canopy is to survive to the end of the growing season. This is no problem for uninjured or only sporadically cold-injured grapevines. It is the vines with very poor post-freeze recovery that demonstrate this important connectivity. The photos below depict a poorly recovered Cabernet Sauvignon vine four years after the February freeze killed or severely set-back vines in Washington State.

Above photo, upper left. Note the vine only has produced a very few shoots each season since the freeze and that the retained spurs on these shoots are associated with a rib or flute (white latex paint) that can be followed down the arm toward the trunk.

Lower left. The relationship between shoot growth and arm and trunk growth is shown by the painting of the flutes downward from living canes. The arms and trunks have much dead wood that cannot produce new living xylem or phloem increments.

Right photo. The vine above was removed from the vineyard and band-sawn into short cylinders to show a cross-sectional series from the spurs on the cordon (upper left) down to the trunk at ground level (lower right). In every “slice” of the trunk there was only seasonal vascular development in sectors that could be followed up to where there were living shoots and canopy development. The reliance of the trunk vascular cambium on a stimulus from shoots is apparent, and continued vascular development depends on a least some vertically connected living tissue between shoots and roots. In severe cases this could take two or more years. Only growers can decide how much vine recovery time can be tolerated before vines have to be replaced by training suckers or by replanting vines.
Summary
It is important for growers to realize that repair of freeze-injured canes, cordons, and trunks depends on the stimulus of emerging shoots and leaves. Bud emergence and shoot growth create critical chemical signals for both normal vascular reactivation in spring but also, in case of damage to the vascular tissues, for callus production and vascular repair. The greater the number of buds breaking and the less injured the vascular system in older wood, the faster and more efficient the repair becomes. When not enough buds are present in the canopy area to allow adequate repair of badly injured wood, the vine may have to be replaced or new trunks replaced with suckers emerging from old, dormant, hidden buds near the crown.

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For a general, but technical, review of the anatomy of cold injury in grapevines, readers can refer to the article below.