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Bud Dormancy in Perennial Fruit Trees: Physiological Basis for Dormancy Induction, Maintenance, and Release

M. Faust
Fruit Laboratory, Beltsville Agricultural Research Center, Agricultural Research Service, Beltsville, MD 20705

A. Erez
Institute of Horticulture, The Volcani Center, Bet Dagan 50250, Israel

L.J. Rowland and S.Y. Wang
Fruit Laboratory, Beltsville Agricultural Research Center, Agricultural Research Service, Beltsville, MD 20705

H.A. Norman
Weed Science Laboratory, Beltsville Agricultural Research Center, Agricultural Research Service, Beltsville, MD 20705

Dormancy in temperate-zone deciduous fruit trees is a phase of development that allows the trees to survive unfavorable conditions during the winter. The subject has been reviewed several times (see Saure, 1985). The hallmark of winter dormancy is that it is released by a quantitative accumulation of a certain amount of cold and only part of this cold requirement can be substituted by other means. The interest in understanding the mechanism of dormancy imposition and release has been intensified during the last 30 years. Producers desire to grow temperate-zone fruit trees in warm climates where the cold requirements cannot be satisfied, and are interested in manipulating the dormant period to increase budbreak and obtain even flowering. Producers of the temperate zone also desire regulate dormancy but they want to delay bud break to avoid spring frosts that damage blooms. Based on the last 10 years of research, a theory is emerging that indicates multifaceted control of dormancy. Analyzing the control of dormancy, four major biological factors that change the intensity of dormancy can be identified. They are 1) hormone balance in the bud or in the tree, 2) state of water within the bud, 3) structure of membranes affecting cold resistance and governing resumption of growth, and 4) anabolic potential of buds. Without understanding the interaction of these factors, it is difficult to comprehend dormancy and search for the dormancy-release mechanism.

Lang et al. (1987) classified various stages of dormant buds as para-, endo-, or ecodormant. They equated paradormancy with correlative inhibition or apical dominance, endodormancy with winter or deep dormancy when the dormancy causing factor resides within the bud, and ecodormancy, usually occurring during late winter and spring, when dormancy is imposed by temperatures unfavorable for growth. Saure's (1985) terms of pre-, true-, and imposed-dormancy are terms which essentially correspond to those proposed by Lang et al. (1987). Fuchigami and Nee (1987) provided evidence that the depth of dormancy changes during the dormant period.

Erez et al. (1979a, 1979b) stated that cold accumulation is reversible by intermittent higher temperatures, but only if given in short cycles. There is a point in cold accumulation, however, where the process becomes irreversible, indicating a fixation of the effect. This state is expressed by a "dynamic model" developed by Fishman et al. (1987a, 1987b). There are additional factors. Low-chilling-requiring cultivars need only a fraction of the amount of chilling other cultivars need (Saure, 1985), but why they need less chilling is not clear. Rootstocks may also influence chilling requirement of the scion (Couvillon et al, 1984; Westwood and Chestnut, 1964) and buds on strong-growing shoots that keep their leaves late into autumn require more chilling than buds on weak shoots (Chandler and Tufts, 1933). The above considerations indicate that dormancy is a process affected by various integrated elements and that their interaction(s) determine the point of time when release of dormancy occurs. Here, we summarize the relevant evidence and reflect on the possible mechanisms involved in imposition and release of dormancy.

INVOLVEMENT OF HORMONES

Auxins, cytokinins, and abscisic acid (ABA) have been suspected to be involved in imposing or breaking dormancy for a long time. Crabbe (1994) concluded that the classical theory of hormonal control of dormancy, meaning that ABA imposes and cytokinins release dormancy, has failed, yet under certain circumstances hormones still play a role in dormancy. During the summer and fall when buds are under correlative inhibition, removal of the terminal bud releases the

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lateral buds from apical dominance. Replacing the terminal bud with IAA keeps the lateral buds of apple (*Malus domestica* Borkh.) dormant (Wang et al., 1994). The apical dominance effect is carried into late fall and winter, which can be seen in several ways: Terminal buds, which under no apical dominance, require less chilling to break dormancy than the lateral buds (Saure, 1985). Lateral buds on apple shoots exposed to chilling temperatures with terminals removed require less chilling to break dormancy than those with terminals intact (Faust et al., 1995a; Liu, 1992). The level of correlative inhibition may also differ according to the chilling requirement of the cultivar. After cold exposure all lateral buds broke on the 'Anna' shoots but only the uppermost bud on the 'Northern Spy' shoot, indicating that the apex of the uppermost lateral bud possibly reimposed apical dominance whereas this did not occur in 'Anna' (Faust et al., 1995a; Liu, 1992). Paiva and Robitaille (1978) also noted development of the uppermost bud after decapitating apple shoots at all sampling dates from autumn to spring. In a series of experiments in Australia (Williams et al., 1979), with 'Jonathan', lateral buds on decapitated shoots not only required less cold but exhibited a shorter period of deep dormancy than lateral buds on shoots with intact terminals. Further evidence indicates that methods that decrease auxin transport, such as arching, may also alter the maximum depth of dormancy (Crabbe, 1984, 1994). In warm locations, where chilling is insufficient, it is an accepted practice to train the tree canopy into a horizontal position and by that means improve the lateral bud break. Finally, treatment with exogenous auxins generally blocks budbreak (Biggs, 1966; Pieniazek et al., 1970; Thiklin and Swabe, 1970; Thomas et al., 1965). However, a preliminary experiment examining the effect of decapitation on chilling requirement of blueberry (*Vaccinium corimbosum* L.) shoots indicated no effect of the terminal on chilling requirement (M. Ehlenfeldt and L.J. Rowland, unpublished). Thus, there is no question that auxin has an effect in promoting correlative inhibition, not only during paradormancy but also during endodormancy. There is some indication that correlative inhibition in woody fruit trees is not different from that in other plants, but the mechanism is overridden by an another one during endodormancy.

For a period of time inhibitors were looked upon as causal factors of dormancy. ABA was termed "dormin" or dormancy inducer (Addicott, 1983), and was thought as the most important inhibitor preventing growth. ABA is effective in delaying budbreak in cultivated buds of apple (Dutcher and Powell, 1972) and, if injected, it can prevent budbreak in sour cherry (*Prunus cerasus* L.) (Mielke and Dennis, 1978). The highest level of ABA in the bud negatively correlates with budbreak in apple (Seely and Powell, 1981), sour cherry (Mielke and Dennis, 1978), and peach [*Prunus persica* (L.) Batsch.] (Bowen and Derickson, 1978). Defoliation after harvest contributes to avoiding dormancy in apple (Erez, 1990; Janick, 1974) and, in blueberries, defoliation in November lowered chilling requirement of vegetative buds (Darnell et al., 1992). Removal of budscales also allows resumption of growth under certain circumstances (Swartz et al., 1984). These implicate that a signal (ABA?) is transmitted from the leaves to the budscales and imposes dormancy from the budscales to the apical meristem. However, the effect is not direct. Removal of budscales has little effect on early blooming cultivars of apple but it is very effective on late blooming cultivars in promoting resumption of growth (Swartz et al., 1984). Others also have questioned the direct effect of ABA in inducing dormancy. Mielke and Dennis (1978) reported that defoliation of sour cherries in autumn prevented an increase in ABA but the intensity of dormancy remained unchanged and ABA concentrations in chilled and unchilled buds did not correspond to their ability to resume growth. Saure (1985) listed several other opinions, in an extensive review, arguing that ABA per se does not regulate budbreak. However, the effect of defoliation indicates that ABA or other chemical inhibitors can not be completely discounted. Trewavas and Jones (1991) argued that several environmental signals work through a change in ABA biosynthesis or sensitivity. Citing the case of *Acer pseudoplatanus* L., they stated that short days and ABA induced dormant bud formation in this species. However, ABA does not accurately mimic short-day induction of dormancy. The resting buds induced by ABA were only pseudo-buds with shortened scalelike petioles (Eagles and Wareing, 1963). In addition, there are conditions, such as drought, that cause an increase in ABA and also impose dormancy. Later, we discuss evidence for the involvement of dehydrin proteins and membrane permeability in dormancy control. Fall ABA levels could be involved in induction of dehydrins (Jacobsen and Shaw, 1989; Mundy and Chua, 1988) and in changes in permeability of membranes (McAnish et al., 1991). Thus, ABA may not need to be linearly correlated with dormancy to have an effect on its development.

Saure (1985) summarized the evidence that in growing shoots, cytokinins are known to counteract the inhibition of lateral buds resulting from apical dominance. Cytokinin analogs, especially thidiazuron (TDZ), have been used recently to overcome dormancy (Steffens and Stutte, 1989; Wang et al., 1991c). Cytokinins also increase in the xylem sap of apple just prior to budbreak (Cutting et al., 1991; Tromp and Ovaa, 1990). Chemicals commonly used to partly replace chilling, such as dinitro-o-cresol (DNOC-oil) and hydrogen cyanamide, increased xylem concentration of cytokinins 5 weeks before it normally occurred in the control (Cutting et al., 1991). KNO_3 is also known to compensate partly for lack of chilling (Erez et al., 1971). Buban et al. (1978) were able to show that potassium and nitrate, separately, are able to trigger cytokinin production in the roots of trees. Treatment of excised peach shoots with KNO_3 induced callus formation similar to that induced by TDZ (Erez, unpublished). Callusing without roots, an obvious cytokinin effect, indicates that cytokinin synthesis may not be restricted to the roots. Tromp and Ovaa (1990) also believe that the increase in cytokinins in tree tops does not need to be transported from the roots but that it may be generated either from local *de novo* synthesis or storage forms, which is also the conclusion of Cutting et al. (1991). Cytokinins trigger metabolic activities that are geared for growth, including DNA, RNA and protein synthesis, increase in the energy metabolism, and decrease in pathways important in resting tissues (Wang et al., 1986, 1991a, 1991b). However, TDZ and other dormancy-breaking chemicals that increase cytokinin concentrations in xylem are not equally effective in breaking dormancy during the entire dormant period. Such chemicals able to trigger growth in late fall and again when about two-thirds to three-fourths of the chilling requirement of buds is satisfied (Erez, 1987; Steffens and Stutte, 1989). When paradormant apple buds are triggered by cytokinin, and, subsequent to this, IAA is applied to the decapitated stem, buds remain dormant (Wang et al., 1994). Apparently, TDZ is antagonistic to correlative inhibition and its effect can be negated when forces of correlative inhibition are stronger than the stimulus. Bangerth (1994) concluded that cytokinins in the xylem exudate of intact plants are under the control of polar auxin transport which has direct implication on the effect of these hormones on budbreak. Studies with 'Anna' and 'Northern Spy' apples indicate that the response of the buds to TDZ was faster in 'Anna', the low-chilling, than in 'Northern Spy' the high-chilling cultivar, and it was also faster with increasing increments that satisfy the chilling requirement, regardless of cultivar (Faust et al., 1995a). The fact that TDZ is effective certain times during endodormancy indicates that buds have to be in a receptive stage for this hormone to be effective in "breaking" dormancy.

STATE OF WATER WITHIN THE CELLS DURING DORMANCY

During endodormancy, water is closely associated with macromolecules and is registered on magnetic resonance imaging (MRI) as bound water expressed in low T2 relaxation times. When dormancy is terminated, T2 times are longer and water is in a relatively more "free state" (Faust et al., 1991). This finding was confirmed with apple (Liu, 1992), peach (A. Erez et al., unpublished data) and blueberry (Rowland et al., 1992). Conditions, such as short days and low temperature, that enhance dormancy in peach buds also increased the level of bound water in the buds (Erez, Faust, Wang, and Line, unpublished data). Water is gradually freed during the dormant period (Faust et al., 1995b), and rapidly converted to free water when resumption of growth is triggered by TDZ (Liu et al., 1993) or by forcing conditions. While water is bound, there is no appreciable enlargement of flower primordia. Toward the end of the endodormant period when about two-thirds of water is freed in apple an appreciable enlargement of