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

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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

Table 1. Size of flower primordia during dormancy.[z]

Type of flower	Date first measured	Diam of flower buds (μm)	Increase since previously measured (%)
Terminal	5 Oct.		
	8 Dec.	782	36
	14 Feb.	913	17
	15 Mar.	1928	111
Lateral	5 Oct.		
	8 Dec.	554	23
	14 Feb.	652	18
	15 Mar.	1404	115

[z]Reprinted with permission from Buban and Faust, 1995.

flower primordia occurs without any visible sign of budbreak (Table 1) (Buban and Faust, 1995). Similar growth of flower primordia was observed by Chandler and Tufts (1933) in peach without then knowing the state of water in the buds. In paradormant apple buds, when water is relatively bound, TDZ frees the water (Liu et al., 1993; Wang et al., 1994) and IAA keeps it bound even after TDZ treatment (Wang et al., 1994).

Khanizadeh et al. (1994) reported an increase in hydrophylic amino acids and a concomitant decrease in hydrophobic amino acids in apple flower buds in late summer and during the fall. Muthalif and Rowland (1994) identified three dehydrins 65, 60, and 14 kd proteins in blueberry floral buds that develop in late fall when temperatures decrease. Dehydrins are hydrophilic proteins that are boiling stable and induced during cold stress and/or dehydration. Dehydrin proteins, due to their high hydrophilicity, are possible candidates to bind water.

In most woody perennials, dormancy and cold hardiness are interlinked. Dehydrins are known to be triggered by cold and this is the case in blueberry. However, as with other woody perennials dormancy develops concomitantly. As chilling accumulates, dehydrins seem to decrease slightly, remaining present through the dormant period when chilling is satisfied, and finally disappearing when growth is resumed. In experiments done so far, only the effect of cold was correlated with dehydrin formation. Nevertheless, we have to consider the fact that dehydrin formation also can be triggered by ABA, and short days and that drought may have a similar effect on triggering dormancy. Thus, the fact that a specific mechanism (i.e., dehydrin elaboration) is tightly connected with cold hardiness does not exclude its possible involvement in the control of dormancy in the bud (Table 2).

MEMBRANE CHANGES DURING DORMANCY

Plant growth regulators, TDZ, and nitroguanidins release lateral buds from paradormancy and cause concomitant changes in galacto- and phospholipids and the ratio of unsaturated fatty acids to saturated fatty acids (Wang and Faust, 1988, 1989a, 1989b). A decrease in the ratio of free sterol to phospholipids and an increase in the ratio of campesterol + stigmasterol to sitosterol also accompanies budbreak (Wang and Faust, 1988, 1989a, 1989b). These findings indicate that, with resumption of growth, there is a change in membrane composition allowing increased permeability of solutes and water to the cytoplasm (Brockerhof, 1974; Goad, 1983; Grunwald, 1975; Oldfield and Chapman, 1972). Later work determined that such membrane change also takes place during the winter during endodormancy in leaf buds (only buds examined) (Wang and Faust, 1990). During the dormant period, especially in phospholipids, linoleic acid (18:2) increases to a maximum in early February while linolenic acid (18:3) is almost level, with a very slight increase regardless of phospholipid headgroup [phosphatidylcholine (PC); phosphatidylethanolamine (PE); phosphatidylglycerol (PG); phosthatidylinositol (PI)] or the bud (terminal or lateral) (Fig. 1). The ratio between 18:3/18:2 = 1 at this time. When chilling requirement is about satisfied, a major change takes place: Linoleic acid (18:2) greatly decreases with a corresponding increase in linolenic acid (18:3), indicating an increase in linoleic acid desaturase activity. By the time of budbreak, the ratio of 18:3/18:2 = 2. PC concentration was always 50% to 80% higher in February than the other phospholipid combined, and all phospholipid concentration in the buds doubled by the time of budbreak.

During chilling, apart from changes in the ratio of the fatty acids in the phospholipids, there is a major increase of total phospholipids per bud dry weight. To increase membrane phospholipids a pool of triglycerides may be hydrolyzed by lipase as a key enzyme. According to Zarska-Maciejewska and Lewak (1976) and Ranjan and Lewak (1995) lipase activity is very high at low temperatures in dormant apple seeds and increases with chilling in apple buds (Liu et al., 1991). Although data on the triglyceride pools are not currently available, they likely are high during the dormant period. The conversion of 18:2 to 18:3 requires a high level of reducing power and active oxygen. A concomitant activity to detoxify H_2O_2, resulting indirectly from the unsaturation reaction, is also needed because desaturase enzymes are inactivated by H_2O_2 (Norman et al., 1991). Siller-Cepeda et al (1992a, 1992b) identified a high level of gluthathion in peach buds closely associated with breaking of rest, which may be related to the membrane changes described above. Considering that the desaturation processes takes place during the entire endodormant period, reducing power is needed throughout endodormancy. However, our knowledge is limited on the importance of in vivo reducing processes. To achieve active in vitro desaturation, the presence of NADPH and catalase in the incubation medium is required to detoxify H_2O_2 (Norman et al., 1991). There are several other enzyme systems that can detoxify H_2O_2 in buds of fruit trees, among which the most prominent are ascorbate free radical reductase (EC 1.6.5.4), ascorbate peroxidase (EC 1.11.1.11), superoxide dismutase (EC 1.15.1.1), catalase (EC 1.11.1.6), and peroxidase (EC 1.11.1.7) (Faust and Wang, 1993; Wang and Faust, 1994; Wang et al., 1991a, 1991b). Others provide reducing power, such as gluthathione reductase (EC 1.6.4.2) and dehydroascorbate reductase (EC 1.8.5.1). Activities of these enzyme are summarized by Faust and Wang (1993). Thus, incomplete, but existing key evidence supports the hypothesis of the involvement of change in membrane constituents in dormancy development and release and the involvement of reducing power in membrane changes.

ANABOLIC POTENTIAL OF BUDS

Another approach was taken by Champagnat (1989) and other French and Belgian researchers. By this approach dormancy is seen as a development due to loss of potential competition with other plant tissues and dormancy release is considered as an improved competing bud power with its neighboring tissues. Based on work with Jerusalem artichoke [*Helianthus tuberosus* L.] tubers they observed competition among plant organs. They have noted a gradual development of a communication block between the bud and the adjacent tissue during the transition to dormancy. They visualized the development of this blockage as a transition from the long-distance effect of correlative inhibition to a short-distance inhibition by a barrier between the bud and the stem. Using the 2-^{14}C 5,5-dimethyloxazolidine-dione (DMO) test that indicates endogenous pH levels, Champagnat (1989) has found a highly significant correlation between pH of the cells and the ability of the tissue to compete with other sinks within the tree. This test

Table 2. Processes involved in imposing dormancy.

	Environment imposing the change		
Factos	Short days during fall	Low temperatures during late fall	Chilling is nearly completed, late winter or spring
State of buds	Paradormant	Becoming d-endodormant	s-Endodormant or ecodormant
Internal factors imposing dormancy	IAA, ABA, terminal bud, leaves	Dehydrins, membrane changes, Water is bound, limiting metabolic activity	IAA-Cytokinin interactions. Water is free, conducive for metabolic activity

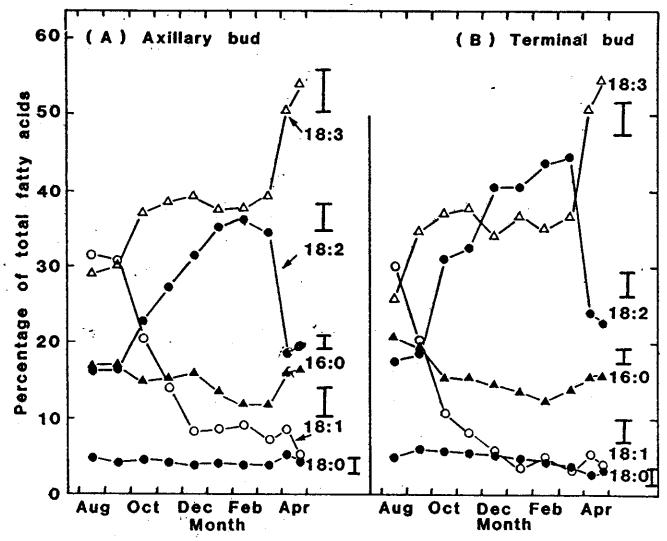

Fig. 1. Fatty acids (%) in phosphatidylcholine in 'Delicious' apple buds during the winter. Fatty acids are marked with chain length and unsaturation level. Note the decrease in 18:2 and the increase in 18:3 fatty acids about the time chilling is satisfied. (Reprinted with permission, Wang and Faust, 1990).

allows evaluating the connection between stem and bud and the relative potential of the bud to begin growth. This approach may logically bridge the hormonal and the specific dormancy control system. By reversing the competing power of the different tissues, the bud may overcome the block to its development. This future has been shown with regards to internal bud cell pH, which is rising when dormancy is over and is higher than that of stem and receptacle cells, while the contrary is true in the dormant state. This rise in pH occurs due to increased plasmalemma ATPase activity in the cell membranes and proton pumping, a measure of active metabolic activity (Petel et al., 1992). They also found a good correlation between the ability of the buds to synthesize nonadenylic triphosphates and their release from endodormancy.

A UNIFYING CONCEPT

It is obvious from the previous discussion that there are dynamic changes during dormancy and that the dormant period is not uniform from its inception to its end. Lang et al. (1987) suggested a definition for dormancy involving three stages of development (para-, endo-, and ectodormancy) that gained international acceptance. Fuchigami and Nee (1987) developed a descriptive annual growth-stage model assigning a segment to each phase when growth is inhibited. A correlative inhibition phase is followed by a deepening rest phase, a decreasing rest phase and a quiescent phase. The series culminates in bud break.

Faust et al. (1995a) proposed a model made up of three overlapping segments: para-, endo, and a second paradormant period based on the reaction of bud to hormonal control. They did not address metabolism during the endodormant period. This scheme is different from the model advocated by Fuchigami and Nee (1987) because the Faust-model envisions a changing dormancy mechanisms.

More recent research necessitates further dividing the endodormant period into deep endodormancy (d-endodormancy), characterized by the inability to induce the buds to grow under natural conditions, and a shallow endodormant period (s-endodormancy), which is the stage where endodormancy can be overcome by artificial treatments (Fig. 2). We also consider all stages of dormancy as overlapping due to variation in bud populations and the quantitative nature of the response.

Crabbe (1994) following Champagnat (1991), envisioned "long distance influences" at the beginning of dormancy, that are roughly equivalent to correlative inhibition, and later, through a chain of events, "short distance influences," that consist of communication blocks between the bud and the adjacent stem tissue preventing growth at the cellular level. Furthermore, he envisions a metabolic block, the inability to synthesize nonadenylic nucleotides in the bud, primarily resulting from permeability barriers. He states that free access to metabolites, together with marked rehydration, allows the bud to grow. Thus, he addresses the correlative inhibition and membrane permeability issues, in non specific, descriptive terms, as important parts of the dormancy phenomenon.

Erez (1990) described methods to avoid dormancy. We can understand the phenomenon if we consider the induction of, and the emergence from endodormancy as a gradual processes under subtropical and tropical conditions. The potential of skipping over endodormancy exists only prior to the development of membrane changes that block the communications between the buds and its supporting tissues. Likewise, using chemicals to supplement the chilling effect is only possible after most of the cold-induced membrane changes have taken place. During endodormancy, when membranes are the structures that impose dormancy, the required amount of chilling is the only effect that can reestablish conditions required for resumption of growth.

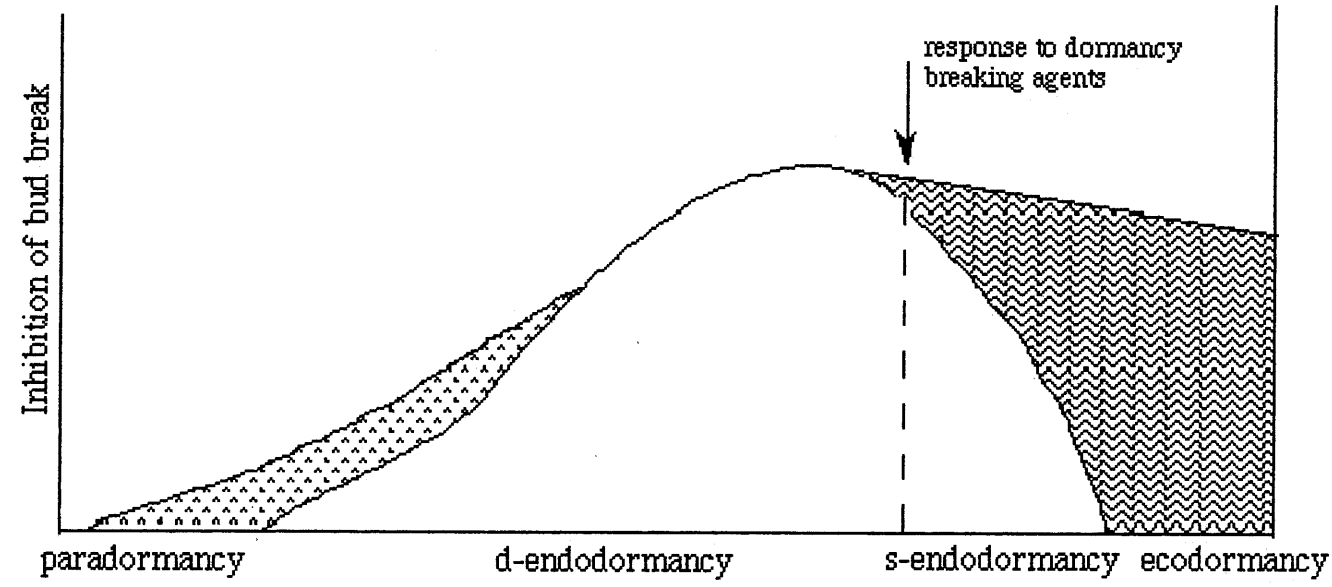

Fig. 2. A schematic representation of inhibition of budbreak during dormancy. Dormancy begins wih paradormancy and it deepens during d-endodormancy. When endodormancy weekens during s-endodormancy, buds respond to dormancy breaking agents. The depth and duration of ecodormancy is environment dependent.

Recent research, discussed previously, allow us to assign measurable cellular or biochemical processes to various phases of dormancy describable so far only in terms of whole-plant physiology. As days shorten in late summer, paradormancy develops in buds of trees. Paradormancy is largely under the control of correlative inhibition. During this period, the ABA content of the plant increases and dormancy is relatively shallow and it can be avoided (Table 3). With increased exposure to even shorter days and low temperatures dormancy becomes more intense (Table 2). In late fall or early winter dehydrins develop in the buds, possibly triggered by ABA, but certainly by decreasing air temperatures. This change deepens dormancy and the plant enters into the endodormant period. Dehydrins bind water and MRI shows low T2 values. Dehydrin development and the increase in bound water in the buds leads to freeze protection but its concomitant effect is to deepen dormancy.

Membranes also react to cold by increasing survivability during the low-temperature period. The relatively viscous membranes change to become more fluid, thus allowing functioning under colder conditions: Desaturation of fatty acids of phospholipids takes place and linoleic acid (18:2) increases; prior to budbreak, linoleate becomes desaturated and membranes are enriched in linolenic acid; about the same time, membranes lose steroids, making the membrane less rigid and highly permeable for transport of solutes. Desaturation requires a high level of reducing power and this is reflected in elevated glutathione, and high activity of a various reductive enzymes. Perhaps the test used by Champagnat (1989) with DMO to test the endogenous pH as a measure of the end of endodormancy reflects the change in potential permeability. Throughout the chilling period, the water slowly becomes freer (perhaps freed by the reductive activity?); the buds enlarge, but dehydrins do not disappear yet. By an endogenous spontaneous mechanism, all these changes will take place regardless of the environmental conditions. With all these changes, buds become ecodormant. During the last stage of endodormancy and during the ecodormant period, buds are sensitive to cytokinins and other dormancy-breaking chemicals, and, if exposed to sufficiently high temperatures, resume growth. When growth is resumed, metabolic machinery of the bud is unleashed, DNA, RNA and enzyme synthesis begin and the energy metabolism shifts from the pentose pathway to the tricarboxylic acid pathway (Wang et al., 1991c). Over chilling usually results in faster budbreak (Couvillon and Erez, 1985). We suspect that a longer chilling period results in more unsaturated membranes that in turn, allow for a more explosive budbreak.

POSSIBLE METHODS FOR MEASURING THE DEPTH OF DORMANCY

TDZ and KNO_3 are effective forcing agents in paradormant and ecodormant buds. During endodormancy, they can be used when two-thirds of the chilling requirement is satisfied. Buds on cut shoots respond and swell after TDZ or KNO_3 treatment at room temperature. They enable the tester to judge whether the buds are still in the endodormant stage or already reached ecodormancy. Testing the depth of dormancy is important because, with present methods, either dormancy avoidance or chilling replacement at the end of the dormant period are the only possible times when buds are in the proper stage for intervention. The disadvantage of the method is that it takes several days until the buds swell and definite readings can be made (Table 4).

If MRI equipment is available, it can show T2 relaxation times. Short T2 times can signify that buds are in endodormancy. However absolute T2 values are very species dependent and absolute values can not be used across species. T2 values are more useful in apple than in peach, mostly because the difference in T2 values between bound and relatively free water is very small in peach (Table 4).

During endodormancy, the 18:3/18:2 ratios of phosphatidylcholine can signify the advancement of chilling and the depth of dormancy. When 18:3/18:2 = 1 the bud is near the point of satisfying its chilling requirement. A ratio of >1 but <2 signifies the inability of the plant to grow. Ratio of >2 indicates that bud break is imminent (Fig. 1). The disadvantage of this method is that present guidelines are based on limited information and laboratory instrumentation is needed to determine fatty acids in phospholipids.

Following bud size is the simplest and least complicated method for determining the end of d-endodormancy. Bud size is nearly stable from late fall to the end of endodormancy. When buds enlarge (not swell or become greener) the enlargement corresponds to freeing the water which is a sign of the beginning of s-endodormancy or ecodormancy (Buban and Faust, 1995; Chandler and Tufts, 1933).

We can also list the DMO test of Champagnat (1989) which, according to Crabbe (1994) measures a permeability barrier and is able to distinguish paradormant, predormant, fully dormant, and post dormant stages and a period toward budbreak. Champagnat also proposed to use the nonadenylic triphosphate test for determining the stage of dormancy. This test is performed on single buds and the release of buds from endodormancy is characterized by the ability of the buds to synthesize nonadenylic triphosphates from the applied adenosine. The disadvantage of these methods is their need for the specialized work of dissecting the bud out the initial from the whole bud.

CONCLUSIONS

Unquestionably, dormancy is a process that evolved as a means of ecological survival, mostly to overcome cold winters. Thus, the closer the response of the organism to the environmental stimuli, the better its adaptation for winter survival. Two environmental elements signify the approach of the winter: short days and cool weather, which are also

Table 3. Agents effective in terminating dormancy in fruit trees.

State of bud		
Paradormant	d-Endodormant	s-Endodormant or ecodormant
Dormancy breaking agents: Defoliation, TDZ, Dormex, KNO_3, removing terminals, desiccation-irrigation	Dormancy breaking agents: None. Membrane effective narcotics may have an effect.	Dormancy breaking agents: High heat (45 °C), TDZ, Dormex, KNO_3.

Table 4. Methods to test the state of dormancy in buds of fruit trees.

State of bud		
Paradormant	d-Endodormant	s-Endodormant
Positive reaction to TDZ, KNO_3; or removal of terminal bud. Speed of response is important.	No budbreak on cut shoots; pH in buds is lower than in adjacent tissue; 18:3/18:2 ratio of fatty acid in PC is one; low T2 times in MRI, but T2 time is species specific.	Positive reaction to TDZ; Dormex;KNO_3; flower primordia is considerably larger than during d-endodormancy; longer T2 times in MRI, but absolute T2 time is species specific; higher bud pH than that of the adjacent shoot tissue.

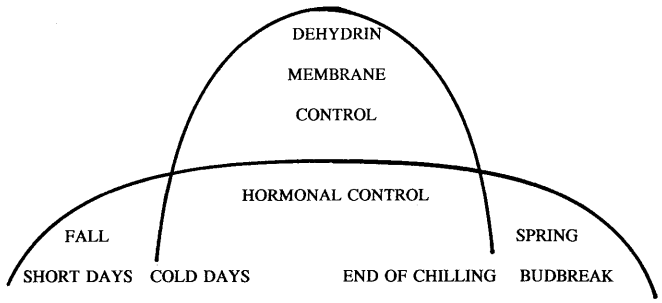

Fig. 3. Schematic representation of hormonal and dehydrin/membrane control of dormancy in fruit trees. Hormonal control of dormancy is uniform throughout the entire dormancy. When temperature decrease a second system is superimposed on the hormonal control, consisting of dehydrin and membrane entities. The result is a much deeper dormancy control.

the triggers for enhanced development of dormancy. Short days signal the developmental changes, but low temperature is the critical element that endangers survival. Thus, a two-stage approach to dormancy should be envisioned, which is reflected in the two major components of dormancy. 1) The hormonal component that moves through the dormant period is not different from what is described in other (annual) plants. It is tightly connected with correlative inhibition (auxins), responsible for mediating the environmental stimuli for dormancy induction (ABA), and involved in dormancy release (cytokinins). 2) Superimposed on the hormonal component is a process driven by responses to freezing resistance that also affects dormancy. It consist of processes keeping the macromolecules in a hydrated state and involves changes resulting in highly polyunsaturated fatty acid components in the membranes, thus allowing permeability even at low temperatures. At this stage, there is a loss of the normal connection and interaction between the various plant organs. This loss means that there is a disruption of the intraplant communication, leading to a change in the survival mechanism that operates on an individual bud basis. No growth takes place in this state, but the process of dormancy continues. By a rather long exposure to chilling temperatures (at ≈6 °C) the system slowly changes, free water appears and membrane permeability is conducive again to growth. At this point the masked hormonal mechanism is effective again and cytokinins are able to promote budbreak. The critical element in the development of dormancy is low temperatures, which in the paradormant state, enhance dormancy and in the endodormant state relieve dormancy. There is a common mechanism that affects cold hardiness and dormancy, which is seen in the development of dehydrins (bound water), in membrane changes, and in a possible ABA effect. It is not clear at what stage the two processes are controlled simultaneously and it deserves further research to clarify it. A simplified model of this scheme is presented in Fig. 3.

We created a picture of dormancy in buds of woody perennials largely composed from experiences obtained working with apples, peaches and blueberries. We are aware that the model is incomplete, but it can serve as a direction for future research.

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