Dormancy—What We Know (and Don't Know)

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So many types of dormancy have been described that any brief and meaningful review must be limited in scope. This paper will summarize what we know about dormancy and what remains to be learned. Most of this review will deal with endodormancy, with emphasis on tree fruit.

Historical aspects and previous reviews

Several reviews deal extensively with early observations and research on dormancy. The interested reader should consult Romberger (1963) for information on bud dormancy and Evenari (1980–81) for information on seed dormancy. Books dealing with seed dormancy include those by Bewley and Black (1982), Bradbeer (1988), and Khan (1982). Crabbé (1987) analyzes bud dormancy as a part of his treatise on shoot morphogenesis. Other recent reviews include those of Champagnat (1983a, 1983b), Champagnat and Côme (1986), Dennis (1987), Fuchigami and Nee (1987), Lang (1989), Martin (1991), Powell (1987, 1989), Saure (1985), and Seeley (1990).

What is dormancy?

Recently, considerable attention has been devoted to dormancy nomenclature. Lang (1987) and Lang et al. (1985, 1987) proposed the terms ecodormancy, paradormancy, and endodormancy to describe three types of dormancy: environmental control, control within the plant but outside the structure, and control within the structure, respectively. Etymology aside, these concepts are well recognized. For example, Champagnat (1983b) points out that Chouard (1956) proposed a similar classification using the terms quiescence, correlative inhibition, and dormancy, respectively. Seed physiologists have difficulty accepting some of the new terms, under which nondormant, dry seeds would be classified as ecodormant. I prefer the term dormancy, and will use it synonymously with endodormancy unless otherwise noted.

Every plant physiology text and many review articles list types of seed dormancy (coat-imposed vs. embryo dormancy; anatomical vs. physiological dormancy; immature vs. mature embryo, shallow vs. deep dormancy) and conditions that remove dormancy (dry storage, moist chilling, light). The many conditions necessary for removing dormancy make it unlikely that any one mechanism or condition, hormonal or otherwise, controls the process. Scientists are trained to seek simple explanations and universal principles, but these have proven difficult to find for the physiology of dormancy.

Are bud dormancy and seed dormancy similar phenomena?

Bud dormancy and seed dormancy have many common characteristics (Powell, 1987). For example, the temperatures that break the dormancy of seeds and buds of deciduous tree fruit exhibit similar optima, and secondary dormancy can be induced by exposing fruit to high temperatures before they accumulate a critical number of chill units. My students and I have studied seed dormancy based on the assumption that the knowledge gained could be applied to bud dormancy, perhaps providing a way to delay budbreak in the spring, thereby avoiding freeze injury. Seeley (1990) and del Real Laborde et al. (1990) have incorporated data obtained with apple (*Malus domestica* Borkh.) (del Real Laborde, 1987) and peach [*Prunus persica* (L.) Batsch.] (Seeley and Damavandy, 1985) seeds into the Utah Model (Richardson et al., 1974), which is used to predict the effects of fluctuating winter temperatures on breaking bud dormancy. J. Crabbé (personal communication) questions these assumptions on the grounds

that seeds a) contain a whole plant (the embryo) and therefore exhibit internal correlations and b) manifest breaking dormancy by radicle elongation, whereas buds contain only shoot or floral tissue and growth depends on factors provided by other plant organs. Arias and Crabbé (1975) and Crabbé (1984) have demonstrated that chilling changes the developmental pattern of vegetative buds on single-node cuttings of tree fruit, with varying degrees of acrotony and basitony being exhibited depending on collection time. Correlative inhibition may occur even in the embryo; Thévenot and Côme (1973) and Thévenot et al. (1973) demonstrated that the presence of the cotyledons could either hasten or delay the growth of apple embryonic axes depending on the excision time during chilling.

What induces dormancy?

Dormancy can be divided into various phases. Although Champagnat (1983a) considers bud dormancy to be a continuous process beginning with apical dominance, Amen (1968) proposed four phases of seed dormancy—induction, maintenance, trigger, and germination. Most research has focused on breaking dormancy (trigger phase) because less is known about the conditions that induce dormancy, except for species in which growth is responsive to photoperiod. In species such as *Acer pseudoplatanus* L. and *Betula pubescens* J.F. Ehrh. (Kawase, 1961), exposing young, container-grown plants to short photoperiods stops growth, whereas growth is continuous under long photoperiods. How buds become dormant in many other species remains a mystery, although lateral buds seem to become endodormant when prevented from growing for long periods by apical dominance—paradormancy gradually becomes endodormancy (Champagnat, 1983a). To produce apples and peaches in tropical areas, trees must be defoliated before buds enter endodormancy. As leaf removal is delayed, the proportion of buds that develop becomes progressively lower (Edwards and Notodimedjo, 1987).

The role of temperature in dormancy induction is not well defined. Several studies indicate that chilling may intensify dormancy in the fall, although it breaks dormancy later (Ben Ismail, 1989; Hatch and Walker, 1969; Kobayashi et al., 1983; Lavarenne et al., 1975; Walser et al., 1981). Hatch and Walker (1969) observed increased dormancy as measured by the concentration of gibberellic acid (GA₃) required to stimulate leaf bud expansion on excised peach and apricot (*Prunus armeniaca* L.) shoots in the fall, even as chill units accumulated. However, they did not include control (nonchilled) shoots for comparison. In a similar study, Walser et al. (1981) subjected peach trees or limbs to various temperatures, then removed cuttings and treated them with gibberellin (GA). Warm conditions delayed but did not prevent the onset of dormancy and increased dormancy in trees subsequently exposed to chilling temperatures. Ben Ismail (1989) demonstrated that growth of vegetative apple buds on isolated nodal sections was inhibited by chilling cuttings in early October. However, the response varied depending on collection time and length of exposure to low temperature. If chilling intensifies dormancy at one stage of development and releases it at another, the time at which one begins to record chill units becomes critical.

How is dormancy maintained?

According to Amen (1968), the maintenance phase continues until conditions (e.g., temperature or daylength) change. Amen implies that maintenance is a steady-state condition, with factors responsible for growth renewal (growth promoters?) being in balance with those that prevent growth (growth inhibitors?). Until conditions change, dormancy remains static. In certain structures [e.g., potato (*Solanum tuberosum* L.) tuber buds and lettuce (*Lactuca sativa* L.) seeds, *Arabidopsis*, and some cereal grains], dormancy disappears during dry storage; the only condition that changes is time. In some trees, growth

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occurs in flushes, and no particular environmental conditions seem to be required to renew growth. In ash (*Fraxinus excelsior* L.) (Lavarenne et al., 1986), chestnut (*Castanea sativa* Mill.) (Si Mohammed, 1983), and oak (*Quercus pedunculata* Ehrh.) (Payan, 1982), such flushes occur at regular intervals, ranging from several weeks to up to a year, when the plants are maintained under constant temperature and light. The period and amplitude of flushing can be affected by photoperiod, temperature, and other factors. J. Crabbé (personal communication) suggests that this may be true for many other species, and that chilling merely hastens flushing as it hastens flowering (vernalization) of 'Petkus' winter rye (*Secale cereale* L.) (Purvis, 1965).

What breaks dormancy?

Amen (1968) lists several triggers that effectively break seed dormancy, including low temperatures, light, and dry storage. He suggests that the trigger mechanism is brief and need not continue once dormancy is broken. Such triggers are credible for situations in which a single factor can break dormancy (e.g., the effect of light on lettuce seed germination) but are less applicable when dormancy requires a long exposure to certain conditions (e.g., low temperature).

Although scientists often speak of the time required to break dormancy, dormancy is a quantitative state. Therefore, defining the end of dormancy as the time when 50% of a population of buds or seeds sprouts is merely a convenient way to compare treatments. Dormancy actually ends when further chilling no longer effectively hastens budbreak. However, once a critical number of chill units has been accumulated, heat units hasten budbreak. Couvillon and Erez (1985) and Gianfagna and Mehlenbacher (1985) have demonstrated that buds on trees or rooted cuttings kept at temperatures that effectively break dormancy, but are too low to permit bud development, continue to increase in growth potential well after the minimum number of chill units required to break dormancy has accumulated.

Côme and Thévenot (1982) stress the need, when studying dormancy, to distinguish between germination *sensu strictu*, which they define as the period between imbibition–radicle elongation and growth. Any comparisons between dormant and nondormant tissues are valid only during this interval, for the dormant embryo cannot grow. This is a valid concept and should be considered when correlating chemical or anatomical changes with breaking dormancy. However, using germination alone as a criterion for seed dormancy release may not be valid, for radicle elongation rate is also an indicator of dormancy. Relative dormancy remains a problem: should embryos excised from dormant seeds be considered nondormant if they germinate in 10 to 20 days, when chilled embryos require only 2 days? Seedlings developing from nonchilled seeds or excised embryos of many rosaceous seeds are usually dwarfed (Flemion, 1959). Frisby and Seeley (1993) reported a weak correlation ($r^2 = 0.54$) between germination vs. seedling development after chilling peach seeds and concluded that these two parameters could not be used interchangeably to indicate the end of dormancy. Nee (1986) observed that budbreak could be induced in crabapple (*Malus sylvestris* L.) seedlings with hydrogen cyanamide during early dormancy phases, but growth was very limited. Based on these observations, Fuchigami and Nee (1987) suggested that budbreak and shoot elongation are controlled by different mechanisms and that cytokinins may play a role in the latter process.

What is the site of dormancy control?

Paradormancy is obviously controlled by organs outside the seed or bud, usually the apices and leaves in the case of apical dominance and summer dormancy. Bud-scale removal can stimulate meristem growth in *Rhododendron* spp. (Schneider, 1968), apple (Swartz et al., 1984), and tung (*Aleurites fordii* Hemsl.) (Spiers, 1972), and embryo excision permits germination in some dormant seeds. Caution must be used in drawing conclusions about the site of dormancy from such studies, however, since meristem response could be wound induced. As noted, embryo dormancy is a quantitative phenomenon; growth of embryos excised from dormant or partially dormant seeds is often sluggish compared with that of chilled embryos. If the embryos were truly nondormant, the radicles would be able to penetrate the seedcoat, assuming that the latter has only a mechanical effect.

Does the seedcoat inhibit germination because it contains inhibitors, limits O₂ supply to the embryo, or because of its mechanical properties? Côme (1967) proposed that phenolic compounds in the testa trap O₂, and that these compounds are leached out during moist chilling. If this is true, such seeds should germinate at high O₂ levels. Apple seeds, however, remain dormant in 100% O₂ (J. Ozga, personal observation), and germination of seeds chilled in fruit is not enhanced when O₂ levels are raised above 20% (Côme, 1967). Furthermore, embryo dormancy can be broken by anoxia (Tissaoui and Côme, 1973). Côme (1967) reported that embryos of seeds chilled in fruit would germinate when the chalazal end of the testa was removed, but that the radicle often did not penetrate the seedcoat. He ascribed this to the inner integument's mechanical resistance. If the seedcoat effect is caused by the chemicals it contains, a chilled embryo should not germinate when enclosed in the testa of a nonchilled seed. Although my students have attempted this experiment, mechanical difficulties have led to ambiguous results. Making crosses between species or cultivars with differing chilling requirements would also test this hypothesis. Published data (Dennis, 1974) generally indicate limited effect, if any, of the testa, but these studies were not performed with this hypothesis in mind. However, Karssen et al. (1987) demonstrated that the embryo's abscisic acid (ABA) content dictates whether or not *Arabidopsis* seeds are dormant. ABA content of the maternal tissues is unimportant.

Cotyledons are modified leaves, and leaves obviously play a role in correlative inhibition (and endodormancy induction?). The observations of Thévenot and Côme (1973) suggest that apple cotyledons resemble leaves in this respect. Although chilling apple seeds reportedly changes the protein profile only in the embryonic axis (Eichholtz et al., 1983), similar studies with peach seeds indicated that such changes occurred only in the cotyledons and did not depend on the presence of the embryonic axis (Mahhou and Dennis, 1993).

What can modeling studies tell us about dormancy?

Modeling can be used to define the environmental factors controlling physiological processes and is discussed by Hanninen (1990) and Cannell (1989), whose technique has been used to quantify the chilling requirement of fruit tree buds. Several methods have been proposed (Bidabé, 1967; Erez et al., 1988; Kobayashi et al., 1982, 1983; Richardson et al., 1974). A critical question arises as to when to begin recording chill units, given the fact that early chilling can intensify dormancy.

Although such models can predict when the chilling requirement is satisfied, most provide little information about the physiological control of dormancy. However, the dynamic model (Erez et al., 1988) is based on the assumption that dormancy is controlled by a series of undefined, temperature-dependent reactions (Erez et al., 1979; Fishman et al., 1987a, 1987b). Thus, this model may provide clues as to the physiological mechanisms involved.

What is the physiological basis of dormancy?

Despite many experiments designed to establish the physiological basis of dormancy, the goal remains elusive. Little work has been done in molecular biology, although Karssen et al. (1987, 1989) suggest that one gene controls seed dormancy in *Arabidopsis* by regulating ABA or GA synthesis, and Morris et al. (1991) have identified dormancy-associated mRNAs in wheat (*Triticum aestivum* L.) seeds. Lang (1994) discusses such approaches in more detail.

There are several schools of thought regarding the mechanisms of dormancy. The classical school considers dormancy to be controlled by hormones and is exemplified by the following statements:

- Dormancy onset, control, and termination are apparently regulated by a balance of growth inhibitors and promoters (Amen, 1968).
- Phase change (endodormant to active, active to endodormant) in perennial plants is not understood fully. However, it is probable that phase changes are initiated by environmental cues and that those cues are transduced by plant hormones such as indoleacetic acid, GA, ABA, and ethylene (Seeley, 1990).

The “French school” considers dormancy a more complex phenomenon in which hormones play a peripheral role. Their views are summarized by the following statements regarding bud and seed dormancy, respectively:

- Dormancy is regarded as a morphogenetic factor under the control of various correlative influences. A gradual transition is demonstrated from correlative delay of bud growth to deep dormancy of the bud itself (Champagnat, 1983a).
- Embryo dormancy is therefore a very complex phenomenon controlled by a large number of internal and external factors, all of which intervene simultaneously to create a physiological state reflected in the ability or inability to germinate. Probably, however, none of these factors is specific to a given physiological state. This is perhaps why it has so far not been possible to find a dormancy marker. Indeed, there is reason to doubt that any such marker exists at all (Côme and Thévenot, 1982).

The classical school prefers simple hypotheses based on the assumption that dormancy is controlled by relatively few factors. Most of the evidence has been obtained from seed dormancy studies because seeds are easier to work with than cuttings or whole plants and can be stored for long periods. Many of the hypotheses assume that chilling removes an inhibitor, stimulates promoters, or both, leading to growth resumption. In contrast, the French school has emphasized bud dormancy rather than seed dormancy, using single-node cuttings (Nigond, 1967) to reduce correlative influences between buds. This school views hormones with skepticism; they may play a secondary role in dormancy, but they are not the key factors. Dormancy is too complicated a process to be controlled by one or two factors, for it is the last stage of a cascade of correlative inhibitions (Champagnat, 1983a, 1983b; Champagnat and Côme, 1986), beginning with apical dominance, gradually extending from control by the apical bud alone to control by tissue immediately subjacent to the meristem of the lateral bud, and finally to control within the meristem.

What evidence exists for hormonal control?

Despite the existence of many endogenous compounds that inhibit growth, ABA is the primary candidate for the controlling factor that accumulates in seeds and buds during dormancy induction. Available evidence suggests, however, that endogenous ABA levels are not responsible for the effects of short photoperiods in inducing bud dormancy (Lenton et al., 1972; Powell, 1976) or of chilling in breaking bud and seed dormancy (Ozga, 1988). However, the work of Karssen and Lacka (1986) and Karssen et al. (1987) with *Arabidopsis* strongly implicates the embryo's ABA content (but not that of other tissues within the seed) as the factor that induces embryo dormancy during seed maturation.

Additional work by Karssen et al. (1989) with GA-deficient *Arabidopsis* mutants has led to the hypothesis that GA, rather than ABA, is the controlling factor in germination (no GA, no germination). Mature seeds of genotypes incapable of synthesizing GAs (e.g., *ga-1*) are dormant, whether they contain ABA or not, and are responsive to GA but not to light. Furthermore, they are more sensitive to GA in darkness than wild-type seeds. Chilling and dry storage increase GA sensitivity in wild-type and *ga-1* seeds. These and other observations suggest that GA synthesis or sensitivity control germination.

Evidence for the role of promoters in dormancy of other species remains tenuous. GAs or cytokinins can induce germination in some species but have limited effects on others, particularly in the absence

of chilling or other treatments required for germination. This lack of responsiveness makes simple explanations unlikely and enforces the reservations of the French school on hormonal control.

Data are available to support at least three hypotheses involving changes in endogenous GA concentration. One hypothesis is that chilling stimulates GA synthesis, the concentration rising as chilling progresses until the threshold is reached for dormancy release (Gwarazimba, 1991; Mathur et al., 1971). A second hypothesis assumes that chilling stimulates GA synthesis, as above. However, the threshold concentration, rather than stimulating germination, induces other processes leading to the release of dormancy but is not required during germination *per se* (Sinska and Lewak, 1970, 1977; Webb et al., 1973). A third hypothesis proposes that chilling removes a block to GA synthesis, which can proceed once the temperature is raised (Ross and Bradbeer, 1971a, 1971b).

Treating hazel (*Corylus avellana* L.) seeds with GA synthesis inhibitors during stratification does not inhibit subsequent germination at 20C, whereas treatment during germination does (Arias et al., 1976; Ross and Bradbeer, 1971b), supporting the third hypothesis. In contrast, paclobutrazol inhibits GA synthesis during peach seed chilling without reducing germination capacity at 20C (Gianfagna and Rachmiel, 1986). In apple, AMO-1618 [2'-isopropyl-4'-(trimethylammonium chloride)-5-methylphenyl piperidine-1-carboxylate] inhibits germination and GA synthesis only in partially chilled seeds (Sinska and Lewak, 1977), a result supporting the view that GA synthesis is crucial only early in the chilling process. These differences suggest that the role of GAs varies among species or that the wrong methods are being used. Tentative support for the latter possibility comes from the work of Bianco et al. (1984), who extracted several magnitudes more GA-like activity from dormant apple embryos using Tris buffer and a surfactant than when ethanol or Tris alone was used. However, this observation does not seem to have been confirmed by other workers.

Trewavas (1986) states that controlling complex developmental processes by a single chemical is not a tenable proposition. He argues for substituting a concept of sensitivity to control in a complex network for one of limiting factors. Evidence exists that seed responses to ABA and GAs change with exposure to chilling temperatures (Karssen et al., 1989; Rudnicki, 1969). This approach deserves more attention from those seeking the physiological basis of dormancy (Saure, 1985).

Hormone metabolism is also important, for the turnover rate may be more important than the absolute quantity of hormone present at any one time (Dennis, 1977). A few reports describe the metabolism of such compounds by dormant vs. nondormant tissues, but are almost entirely catabolic studies using the isotopically labeled hormone as substrate, then measuring its rate of disappearance or conversion (Barthe and Bulard, 1985; Rudnicki and Czapski, 1974; Sondheimer et al., 1974). Rudnicki and Czapski (1974) reported a decline in the content of 1-(¹⁴C)-ABA in apple seeds during stratification and suggested that decarboxylation was responsible for its disappearance. Their conclusion was questioned by Milborrow and Vaughan (1979), who demonstrated that the metabolic rate of 2-(¹⁴C)-ABA was very slow provided that aseptic conditions were maintained, although metabolites other than CO₂ were identified in small amounts. Barthe and Bulard (1985) observed that ABA was metabolized more rapidly by dormant than nondormant apple seeds.

Difficulties in such studies include the possibility of overloading the system with nonphysiological hormone levels, preventing contamination by microorganisms in long-term feeds, and directing the hormone to the specific cells or organelles in which metabolism normally occurs. One of the rules in studying turnover is feeding

Table 1. Gibberellin (GA₄) biosynthesis during stratification of apple embryos (Sinska and Lewak, 1977). All values were estimated from graphs.

Variable	Stratification (days)					
	0	20	30	40	50	60
GA ₄ content (mol/100 embryos × 10 ¹¹)	0.2	0.6	1,000	140	2	1
Increase at 24C (mol/100 embryos × 10 ¹¹)	7.3	72	63,000	1,260	1.3	0.2
Incorporation of ¹⁴ C-MVA ² (%)	2.6	6.8	6	3	0.5	0.2
Germination (%)	19	30	42	78	88	95

²MVA = mevalonic acid.

precursors of the compounds in question. This permits the tissue to produce the hormones with its own enzymes and presumably controls delivery to the action site. Sinska and Lewak (1977) measured 1) the GA activity in apple embryos at various times during stratification, 2) the increase in GA activity when the temperature was raised to 24°C, and 3) the ability of the embryos to convert ^{14}C -mevalonic acid to ^{14}C -labeled compounds co-chromatographing with GA_4 on thin-layer chromatograms. Neither measure of biosynthesis closely paralleled seed GA_4 content during stratification (Table 1) and the ability to synthesize GA_4 from mevalonic acid did not parallel the GA_4 increase observed on transfer to 24°C. As noted above, AMO-1618, a GA synthesis inhibitor, inhibited germination more effectively in partially chilled than in fully chilled apple seeds, a result supporting the view that GA synthesis was required to remove dormancy but not to germinate *per se*.

In preliminary studies (Chilembwe, 1992), dormant and nondormant apple embryos converted ^{14}C - GA_{12} -aldehyde to radioactive metabolites. Metabolism was slightly more rapid in nondormant than dormant embryos, but the metabolite profile seemed similar. None of the radioactive products co-chromatographed with GA_4 or GA_7 , despite the fact that these compounds are the main gibberellins present in extracts (Halinska and Lewak, 1987a, 1987b; Sinska and Lewak, 1970). Failure to detect a given compound could, of course, result from rapid turnover, but one would expect to detect metabolites in control seeds. Conceivably, the metabolites could be glycosides; but they remain to be identified. Such metabolic studies could lead to a better understanding of the role of hormones in controlling dormancy.

What evidence supports the concepts proposed by the French school?

Crabbé (1990) provides a brief resumé of this evidence. Proponents of this approach have sought biochemical markers that might indicate the relative level of dormancy in organs, tissues, and single cells, thus allowing comparisons among buds on the same stem and of meristematic vs. submeristematic tissues. At least two processes have been emphasized—nucleic acid metabolism and permeability. One of the tests used to follow the progress of dormancy is the ability of tissues to convert adenylic nucleotides (ATPs) to nonadenylic nucleotides (NTPs = sum of guanylic, cytidylic, and uridylic nucleotides). Dormant tissues are less able to convert ATPs to NTPs, resulting in a decline in the ATP : NTP (or dATP : dNTP) ratio after incubation with adenosine (Fig. 1). The conversion rate of adenosine to adenylic nucleotides, however, is not affected (Table 2). Correlations between ATP and NTP changes and dormancy have been demonstrated in submeristematic tissues of 'Jerusalem' artichoke (*Helianthus tuberosus* L.) (Gendraud, 1977) (Fig. 2), ash (*Fraxinus excelsior* L.) (Lavarenne et al., 1982) (Table 3), willow (*Salix babylonica* L.), and hazel (Champciaux, 1979), and apple embryos (Thomas et al., 1985). In chilled apple embryos, the change was significant only after the temperature was raised to 20°C (Thomas et al., 1985). In apical buds of oak seedlings undergoing growth flushes, ATP and NTP content was considerably higher just before the growth phase than at its end. Incubation in adenosine did not affect NTP level in dormant buds, whereas it prevented a decline in NTPs in nondormant ones (Barnola et al., 1986). The authors concluded from these data that NTP synthesis exceeded NTP catabolism in the latter.

The second biochemical test measures the cell membrane's permeability to a weak acid, 5,5-dimethyl-oxazolidine-2,4-dione (DMO). This method is similar to viability tests using vital stains. The nondissociated form passes through the cell membrane and dissociates, the degree of dissociation varying with intracellular pH. Incubating the tissues in ^{14}C -DMO permits calculation of the ratio of the concentration of DMO inside the cell (Ci) to that in the intercellular spaces (Ce). Using this technique, Gendraud and Lafleur (1983) demonstrated that the $\text{Ci} : \text{Ce}$ ratio was higher in dormant than in nondormant tubers of 'Jerusalem' artichoke as an effect of higher intracellular pH. Based on these and other data, they proposed that movement of nutrients from submeristematic to bud tissues was impeded during dormancy and that breaking dormancy removed this block. If this hypothesis were true, the $\text{Ci} : \text{Ce}$ ratio would be higher in the stem (higher permeability) than in the bud (lower permeability)

during dormancy, but the same or lower once dormancy was broken. Note that the hypothesis assumes that the movement of organic nutrients from cell to cell via plasmadesmata (at least when phloem loading and unloading is not involved) parallels the movement of electrolytes from intercellular spaces into the protoplasm (vacuole?). Even if movement into meristems requires unloading and loading, carbohydrates (nonelectrolytes) would not be expected to behave as electrolytes.

Ben Ismail (1989) measured $\text{Ci} : \text{Ce}$ ratios in apple bud and shoot tissues during dormancy. The ratio was higher in shoots than buds from September to January; thereafter, the ratio rose in the buds to

Fig. 1. Proposed test for dormancy based on ability to convert nucleosides to nucleotides (Gendraud, 1977).

Table 2. Effect of breaking dormancy by chilling on metabolism of nucleosides in 'Jerusalem' artichoke tuber tissue (Gendraud, 1977).

Nucleoside added (10 $\mu\text{mol}\cdot\text{ml}^{-1}$)	Nucleotides formed (nondormant - dormant) ^z	
	ATPs ^y	NTPs ^x
None	0.02	0.08
Adenosine	-0.02	0.21
Inosine	0.01	0.14
Uridine	0	0.54
Cytidine	0	0.06

^zChange in content in nondormant tissue less change in dormant tissue.

^yATPs = adenylic nucleotides.

^xNTPs = nonadenylic nucleotides.

Fig. 2. Ratio of increase in nonadenylic nucleotides (NTPs) to increase in adenylic nucleotides (ATPs) after incubating apical tissue of 'Jerusalem' artichoke stolons with adenosine during tuberization and bud dormancy induction. Buds were dormant beginning in mid-July. Adapted from Gendraud (1977).

Table 3. Status of *Fraxinus excelsior* buds vs. ability to convert adenosine to nonadenylic nucleotides (NTPs) (Lavarenne et al., 1982).

Buds	Variable	Month			
		Sept.	Nov.	Jan.-Feb.	Mar.
Apical	Status ^z	D	D	D	ND
	Δ NTP ^y (%)	73	-17	-13	54
Axillary	Status	ND	D	ND	ND
	Δ NTP (%)	---	10	196	166

^zD = dormant; ND = nondormant.

^yChange in NTP after adding adenosine.

Fig. 3. The ratio of the concentration of 5,5-dimethyl-oxazolidine-2,4-dione inside the cell (Ci) to that in the intercellular spaces (Ce) in lateral buds and supporting stem tissue of MM.106 apple vs. ability of buds on single-node cuttings to grow. Adapted from Ben Ismail (1989).

levels exceeding those in the shoots (Fig. 3). Bud development on single-node cuttings was strongly inhibited only in November. Thus, the Ci : Ce ratio did not parallel the growth potential of the buds, although this was not stressed by the author. However, the Ci : Ce ratio was measured on the sampling date, whereas growth response was measured after excising single-node cuttings.

Little work seems to have been done by non-francophone scientists to relate either ATP : NTP ratios or permeability to dormancy status, aside from recent evidence that a higher proportion of water may be bound in dormant than nondormant apple tissues (Faust et al., 1991). One of the blocks to communication between non-francophone and francophone scientists in the past may have been the failure of the former to read French publications. Both schools might gain from a joint approach to the problem of dormancy; this should be enhanced by the current tendency of francophone scientists to publish in English.

What questions remain to be answered?

Several questions have been raised in the foregoing discussion, including the site of dormancy and how dormancy is induced. The French school considers growth to be a very complex process comprised of many subprocesses, including leaf and internode expansion and organ formation, that fluctuate in intensity and are interrelated. Dormancy is but one phase of rhythmic growth. Until environmental and correlative control of each of the many processes involved in growth has been characterized, the search for silver bullets (i.e., hormones) that control dormancy is premature.

The classical school, in contrast, is looking for silver bullets. Work

is in progress in several laboratories to isolate genes that may control dormancy, especially genes involved in the synthesis of, or sensitivity to, ABA (Lang, 1994). Here, the most rapid advances will be made by comparing simple systems, particularly those in which dormancy is controlled by relatively few genes. *Arabidopsis* is currently the favorite in this regard. The *ga-1* gene from *Arabidopsis* has recently been identified and isolated (Klee and Estelle, 1991); this work will be useful in further studies on the role of GAs in dormancy. Among tree crops, hazel and peach seem to be the best choices, because genotypes are available in which a single gene controls dormancy (Rodriguez et al., 1994; Thompson et al., 1985). In 'Evergreen' peach, lateral buds become endodormant, whereas terminal buds do not (Rodriguez et al., 1994). If the products of these genes can be determined, much progress will have been made. Caveats are necessary, however. If the French school is correct, how can a single gene control dormancy? Perhaps the gene is merely an operator gene that controls the response of other genes to environmental cues or that is responsible for only dormancy induction. Given the variety of types of dormancy and the range of conditions that affect them, solving the dormancy problem will not be an easy task.

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Dormancy—The Missing Links: Molecular Studies and Integration of Regulatory Plant and Environmental Interactions

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Integrating existing knowledge between seed and bud dormancy, endodormancy and cold acclimation, and research approaches to dormancy (e.g., hormones and bioregulators, molecular genetics, signal transduction, and agroclimatological modeling) is difficult. This is partly because the broad scope and expertise that is required for such integration is rarely fostered in today's technologically advanced, highly specialized research programs. Although our current concept of dormancy involves increasingly complex physiology and lacks much unifying data, a periodic attempt to integrate between and among

research areas or approaches may identify some of the links requisite for continued advancement. I will attempt to illuminate this problem and some of the potential links, such as the association of dormancy with plant stress, hormones, and biophysical properties of tissue hydration. Integrating future segments of the dormancy puzzle may depend strongly on the development of a molecular information base.

Dormancy in woody perennials involves an interrelated series of phenomena regulated by internal and external factors (Dennis, 1994; Lang, 1989; Martin, 1991; Romberger, 1963; Samish, 1954). Dormancy regulation in a specific bud, for example, may be associated initially with distant apices or subtending leaves (paradormancy, probably mediated via a hormone-type signal). Later in ontogeny, regulation may reside solely within the bud and respond to specific combinations of low and moderate temperatures or photoperiod (endodormancy, mediated via unknown biochemical transducing signals). Thus, regulation is controlled by environmental inputs and

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