Rest and activity in vegetative buds of trees

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Introduction

Rest of buds, particularly on trees or shrubs, may have 3 different origins: 1) unfavorable environmental conditions (for instance low temperatures); in this case, the bud is called ‘quiescent’; 2) correlative inhibitions exerted by other organs or parts of the plant, more or less distant from the considered bud. Those may be called ‘long distance correlative inhibitions (LDIs)’; the apical dominance exerted by the terminal bud over axillary buds on growing shoots is the most studied example of LDI; 3) some intrinsic properties of the bud itself, such as when the bud remains at rest even if all usual sources of correlative inhibition have been removed and if environmental conditions are favorable; in this case, the bud is called ‘dormant’ (Champagnat, 1983).

In fact, taking into account the structural complexity of most buds, and the strong interrelationship they maintain with their shoot axis, we will show that the rest of the meristematic region and related tissues depends upon ‘short distance inhibitions (SDIs)’ exerted either by the bud’s basal tissue or by the adjacent shoot-axis tissues.

For over 50 years, investigations on bud rest have mainly focused on plant growth regulators, and their possible involvement in apical dominance or in dormancy. They did not lead to generally accepted conclusions (Samish, 1954; Champagnat, 1965; Wareing and Saunders, 1971; Phillips, 1975; Saunders, 1978; Hillman, 1984). A few papers (Vegis, 1964; 1965a, b; Guern and Usciati 1972; 1975; Miginiac, 1974; McIntyre, 1977) and especially recent reviews by Mauget (1987), Tamas (1987) and Powel (1987) have emphasized the necessity of setting the problem on much wider biochemical and physiological bases. That will be the aim of the present paper, with emphasis on the following aspects: 1) the structural complexity of a bud, and the tightness of its connections with its bearing axis (Champagnat, 1988); 2) the state of nutritional deficiency of a resting bud, either during dormancy or during correlative inhibition of growth, and the fact that, during growth recovery, the different deficiencies are eliminated in a precise and determined order; 3) the short distance control (SDI) of nutrient fluxes towards the meristem cells in buds, by changes in membrane permeability of some adjacent but distinct cells; 4) the fact
that this latter SDI is strongly related to longer distance correlations (LDIs) during normal inhibitions, whereas it is acting alone and is very stable during true dormancy.

We will examine 4 examples which have been the subject of recent work, particularly in France: apical dominance, rhythmic growth of oak seedlings, and induction and release of dormancy in buds on trees and on some tubers, the latter showing physiological and biochemical features which may be compared to those of tree buds.

**Some facts about apical dominance**

Growth inhibition of axillary buds is controlled by a dominant bud, often by the apical or terminal bud which has produced them. Ablation of this terminal bud promotes the resumption of growth by axillary buds on the shoot. The auxins which are produced by the terminal bud, and which translocate basipetally in the stem, are thought to be a major agent of this LDI. Nevertheless, we should also take into consideration the fact that the dominant organ acts like a very strong sink for a lot of nutrients and some plant growth regulators (e.g., cytokinins).

Inhibited buds suffer from different kinds of nutritional deficiencies: water, auxins, cytokinins, energy-rich nucleotides, but also soluble saccharides, polyholosides, etc. It has been possible to induce their growth without cutting the dominant bud, through direct supply of water (Guern and Usciati, 1976), auxins (Sachs and Thimann, 1964) or cytokinins, the latter acting through rapid modifications of carbohydrate metabolism (Usciati et al., 1972).

Release from apical dominance through decapitation is followed after 10–15 min by a modification of the lipid composition of the plasmalemma, which in turn causes a modification of membrane permeability (Usciati et al., 1974). Moreover, a change in membrane-bound Mg2+ ATPases is rapidly set in motion and the cation content of cells is altered (Habricot and Sossountzov, 1984; Sossountzov and Habricot, 1985; Sossountzov et al., 1985).

Near the insertion point of the axillary bud on its axis, that is near its base, an inversion of the polarity of cells in conducting or proconducting tissues has been observed by many authors. The transverse permeability of these cells is weak or even nul in the presence of the basipetal auxin flux. After disappearance of the auxinic flux following decapitation, the permeability increases. The Ca2+ ions could play an important role in this mechanism (Sachs, 1986; Tamas, 1987). The presence of conducting tissues in the lateral bud is not necessary (Ali and Fletcher, 1970).

People who do not think that the flux of auxins may play a major role in apical dominance should test if nutrient diversion alone (sink effect) would be able to induce this weak penetrability of molecules and ions into inhibited buds. Inversely, the supporters of a purely hormonal hypothesis should demonstrate that this ability could not exist without auxins.

In short, and despite the holes in our knowledge, we may emphasize the following observations (Fig. 1): 1) an inhibited axillary bud is unable to draw the nutrients essential to its growth from the nutrient fluxes, or even from storage compounds in surrounding tissues; 2) particular properties of certain cells (probably on the surface of their membranes) located near the insertion point of the bud, are responsible for this incapacity; 3) thus, a short distance inhibition (SDI) appears in the bud; this SDI is strongly related to the apical
command through an LDI; it disappears quite rapidly after decapitation and release from LDI.

**Rhythmic growth in oaks**

Young oak seedlings (*Quercus robur* L.), grown at 25°C, under continuous illumination of 150 W·m⁻², exhibit successive and uniform growth flushes consisting of morphogenetic units; however, the apical bud alone develops. A growth flush lasts about 3 wk (21.8 ± 0.9 d in the case of the 3rd flush after germination, which has been the most extensively studied). Each flush consists of a period of shoot elongation (~12 d) followed by a period of apparent rest (~9 d) (Fig. 2). The rest is only apparent and leaf primordia are continuously formed in the bud; furthermore, the plastochron is rather low between d 12 and 20 (0.4–0.5 primordia·d⁻¹) and higher between d 4 and 8 after bud-burst (1.5–1.8 primordia·d⁻¹) (Payan, 1982). This periodic growth is independent of root growth, which is continuous unlike in many other tree species (Mialoundama, 1985; Champagnat et al., 1986b).

Between d 7 and 21 (end of flush), the shoot bears a scaly terminal bud enclosing an increasing number (from 2 to 10) of
young leaf primordia, first with aborting and afterwards with normal lamina. These leaves and their internodes cannot grow normally; they are inhibited, like axillary buds subjected to apical dominance.

The subject of the following discussion will be that period of strong growth inability, and not the correlative interactions between leaves and the leader bud, which are active during the growth period, and which present the features of an LDI (Champagnat et al., 1986a).

The LDIs involved in this regulation have acropetal influences and not basipetal ones, like those acting during apical dominance. The mechanisms of their action are unknown but, nonetheless, it is not satisfactory to consider them only as an auxin-induced polarization of peri- or provascular tissues.

Nevertheless, it may be observed that during the first days of apparent rest, the apical bud exhibits a deficient metabolism, very similar to that of a bud under apical dominance. It is very poor in ATP, and, overall, it is unable to synthesize enough non-adenylic nucleotides (NTP), which are essential for the translocation of some important molecules (UDP-glucose, for instance). At the end of the rest period and without any change in the environment, the ATP concentration increases 3 times and that of NTP 4 times. This spectacular modification of energyc metabolism appears 1 or 2 d before bud-burst (Table I; Barnola et al., 1986b).

If we measure with DMO (5,5-dimethyl-oxazolidine dione) the intracellular pH (pH) in buds and in the shoot axis immediately below them, we observe that,
at the beginning of the rest period, the \( pHi \) in bud cells is lower (more acidic) than in axial cells; the difference may reach 2–3 pH units. One or two days before bud-burst, this gradient is inverted. It is now well known that the \( pHi \), whose regulation could be hormone-linked, is an essential element in the regulation of short distance nutrient transport between cells. Transport always moves from tissues or cells with the lowest \( pHi \) to those with the highest (cotransport protons–K\(^+\), protons–sucrose, etc.) (Delrot and Bonnemain, 1981; Spanswick, 1981; Guern et al., 1982; Maslowski et al., 1984; Matsumoto and Yamaha, 1984; Sze, 1984; Marre and Ballarin-Denti, 1985). These deficiencies never lead to true dormancy, under our conditions, and remain indefinitely of correlative origin (Champagnat et al., 1986a).

In our case, at the beginning of rest (and probably several days before), the bud is unable to draw from the axis the nutrients essential for its growth. Afterwards, it acquires this ability and may burst and grow. Causes of this transformation may lie in the modification of leaf LDIs, which may parallel lamina evolution (Fig. 3) (Champagnat et al., 1986a; Gendraud and Lafleuriel, 1983; 1985; Barnola et al., 1986b). A parallelism with a bud subjected to apical dominance and afterwards released from it, may be reasonably envisaged. In the case of apical dominance, it would be interesting to measure NTP-synthesizing ability and, in the case of resting apical buds on oak seedlings, to monitor \( pHi \) gradients in addition to the lipid composition of membranes and Mg\(^{2+}\)-associated ATPases, and thus identify in both examples the whole spectrum of nutritional and hormonal deficiencies.

We would like to underline the fact that, in both cases (rhythmic growth and apical dominance), there is an association between LDIs and SDIs. Growth regulators could step in for the latter, which act as relays of the former.

LDIs evolve spontaneously with size and age of leaf lamina (Champagnat et al., 1986a); this characteristic explains the endogenous nature of the rhythm. It is likely that this evolution initiates the rapid modification of the SDIs, as does decapitation during apical dominance. SDIs always exhibit poor stability.

### Dormancy induction and release in buds on current year shoots of trees

#### Preliminary remarks

According to the definition of bud dormancy we gave in the introduction, this stage of bud development follows a period

<table>
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<tr>
<th>Organogenesis</th>
<th>Beginning of rest</th>
<th>End of rest</th>
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<tr>
<td>Elongation</td>
<td>diminishing and slow</td>
<td>increasing</td>
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<tr>
<td>( pH(c_i/c_e) )</td>
<td>0</td>
<td>0</td>
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<tr>
<td>stem</td>
<td>3.7–7.1</td>
<td>1.4–3.3</td>
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<tr>
<td>bud</td>
<td>1.3–2.3</td>
<td>3.3–5.5</td>
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<tr>
<td>ATP (pmol·mg FW(^{-1}))</td>
<td>560</td>
<td>1200</td>
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<tr>
<td>NTP (pmol·mg FW(^{-1}))</td>
<td>760</td>
<td>2500</td>
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of active correlative inhibitions, which may be necessary for the induction of true dormancy (Libbert, 1961; Champagnat 1955). The scaly and dormant buds do not show any appreciable elongation of their leaves or their internodes. Yet they are not in a complete state of rest, as they still exhibit active organogenesis and even slight but significant growth for a few weeks. This rest is of the same nature as that seen in the apical bud of an oak seedling, as described previously, and that in axillary buds of many herbaceous plants under apical dominance. We will not consider here the case of sylleptic axillary shoots (often called ‘anticipated’ shoots), which originate from spontaneous releases of apical dominance, and consequently the disappearance of the SDI associated with trophic deficiencies.

The complete developmental cycle described below is illustrated in Figs. 4 and 5. Indeed, many authors accept that different kinds of correlative inhibitions appear successively in trees between the
stage of bud-burst and the beginning of true dormancy, in contrast to annual herbaceous plants, which have been used most often for studies on apical dominance. Typical apical dominance may only be recorded during the first weeks of spring: decapitation alone initiates the resumption of growth of axillary buds. Later, decapitation has to be accompanied by complete defoliation to enable a few quiescent axillary buds to resume growth. From July–August on, even this drastic treatment becomes totally ineffective. Despite this increasing inability of axillary buds to react to decapitation and defoliation, we consider that real dormancy has not yet begun: in fact, if an axillary bud at this stage is isolated on a short internode, and if this ‘node-cutting’ is placed in a favorable environment, the bud will burst and resume growth after a delay of about 8 d at 20–25°C. If we accept that the wound due to cutting plays no major role in this process, which is difficult to demonstrate, we may postulate that the bud is under the influence of a correlative inhibition originating from the bearing axis (Champagnat, 1965; 1983); in fact, during this period, the axis exhibits active secondary growth, intense lignification and production of storage compounds (hardening process). The axis therefore acts as a sink.

Later, between mid-September and mid-October, depending upon climate conditions and tree species, the node-cuttings...
stop reacting and resuming growth, regardless of the environmental conditions and time of exposure used. We may therefore conclude that dormancy has been induced at this moment. This induction is progressive but rapid: only 2 wk are necessary to pass from 10 d of burst delay at 25°C to complete inertia. This delay in bud-bursting on node-cuttings is used as a measure of the intensity of dormancy and its evolution during winter (Fig. 5) or during experimental chilling. In temperate climates, this delay diminishes from December on and becomes quite weak in January. Later on and until the spring bud-burst (March-April), we may qualify the end of the rest period as post-dormancy, the absence of any significant growth being due exclusively to low temperatures (Nigond, 1967; Mauget, 1976; 1987).

Some specifics and hypotheses

Despite the lack of extensive studies on interrelations between a bud and the adjacent axis tissues, we may hypothesize the following succession of events (Fig. 6).
1) Between March and September, long distance correlations (LDIs) are responsible for the relative quiescence of the buds. The SDIs, which act as relays for the LDIs, must be permanently active. It is not obvious if the influence of auxins, via tissue polarization, remains determinant during the whole period, even if, in this regard, the axial cambium may take the succession of young leaves from the apex. The fact that no significant crisis may be recorded during the replacement of one inhibition source by the next one (apical bud, then leaves and finally axis), strongly supports the hypothesis of the

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Fig. 5. Evolution during winter of mean bud-burst time for 'one-node cuttings' cultivated under optimal conditions. The burst delay increases during dormancy induction (phase 1), attains its maximum during maximal dormancy (phase 2), and decreases during dormancy release (phase 3). These 3 phases may be described as 'true dormancy', needing a chilling treatment to be released. The last phase (4) leads to the spring bud-burst (SBB) and is exclusively controlled by actual temperature; this phase is often called post-dormancy.
permanency of the SDI. The stability of the membrane structures associated with this SDI could progressively increase from spring onwards. 2) The correlative commands seem to progressively approach the inhibited bud meristem and to become diffuse (global action of leaves or shoot axis). The probability that, on one-node cuttings, these commands are identical with SDIs is weak; in fact, the evolution from an extensive ability of bud-burst toward a very weak one may as well be due to an increase in the intensity of the inhibitory action of the stem as to a stabilization of the structures responsible for the SDI. This stabilization is currently the most characteristic feature of true dormancy, as will be emphasized below. 3) Whether the species show monopodic or sympodic growth has no influence on the fact that the bud has to evolve according to the same pattern as the one on the apex of a young oak tree grown at 25°C. However, rest is a temporary stage for the young oak seedling, but will become permanent and will require a chilling period to be removed in case of true dormancy. It is interesting to note that dormancy generally appears later and is more profound for terminal buds than for axillary ones.

Some experimental results

The ability to synthesize non-adenylic nucleotides (NTP) has been measured in ash buds and in adjacent tissues of the
Table II. Evolution of the ability of bud or axis tissues to synthesize non-adenylic nucleotides (NTP), during fall and winter, compared with the bud-break rate on node cuttings and state of rest.

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<td>Bud-break rate</td>
<td>inhibition</td>
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<td>dormancy</td>
<td>dormancy</td>
<td>post-dorm.</td>
<td>bud-break</td>
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<td>~0</td>
<td>~0</td>
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<td>100%</td>
</tr>
<tr>
<td>NTP synthesis</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
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<tr>
<td>in buds</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
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axis from September to March (Table II; Lavarenne et al., 1982; Barbola et al., 1986a). During September, when 100% of node-cuttings may resume growth at 25°C, this ability is considerable in both tissues (bud and stem). A few weeks later, it becomes very weak in stem tissues, while it remains quite extensive in the bud, which may therefore be considered to be independent of the stem in respect to NTPs and may grow without importing them from the stem. At the end of October, the inability to synthesize NTPs reaches the bud; bud-burst is now almost impossible even under favorable conditions. During December and January, NTP synthesis resumes in the stem, due to the influence of chilling conditions, while it still

![pHi graph](image)

**Fig. 7.** Evolution of pH\textsubscript{i} values in apical bud cells and in adjacent stem tissues between Oct. and May (Fraxinus excelsior). Stem tissues always have a higher pH\textsubscript{i} than bud cells, except between Mar. and Apr. Bud-burst takes place during the first days of May, that is after the inversion of the gradient.
remains impossible in the bud. In addition, we will show that the bud is still unable to import NTPs because of an opposing pH gradient. In February, a long time before spring bud-burst (which happens at the end of April on our ash trees), the September situation returns.

The pH gradient has been measured during the same period, in distal and proximal parts of the bud and near its insertion on the stem (Fig. 7; Lavarenne and Barnola, unpublished observations). Throughout autumn and winter and until 5 or 6 d before spring bud-burst, the gradient opposes a translocation flux which would be essential for good nutritional supply of the bud: the basal as well as adjacent stem tissues have a higher pH than the distal tissues in the bud. The difference may be as high as 0.5–1.0 pH units. The inversion may happen very rapidly (2–3 d max.), and is immediately followed by a dramatic increase in water content, which coincides with bud-burst (Cottignies, 1983). Two to three weeks after bud-burst, in the axillary buds of rapidly flushing shoots of ash trees, the gradient between apex and bearing stem again becomes unfavorable to the apex. The bud differentiates, nevertheless, and as on oak trees, organogenesis continues until the final number of leaf primordia is reached.

Discussion

The results remain fragmentary and the conclusions are open to discussion. Nevertheless, a few facts may be outlined. 1) It appears quite obvious that nucleotide metabolism is a good marker for the dormant state of buds; further results obtained with tubers will support this point of view. 2) We may once more note that an unfavorable pH gradient does not exclude slow growth; for instance, organogenesis, which is only slightly water and carbohydrate consuming. It is difficult to reduce this situation to a polarization of basipetal translocations imposed by auxins and it would be hazardous to regard it as the unique cause for SDIs. 3) The most important fact remains that SDIs are essentially under the regulation of LDIs. They disappear once the latter have been released but with an increasing time lag. This fact is important, because this increasing stability leads to a permanency of SDIs even when LDIs are no longer present; thereafter, a specific treatment (chilling for instance) is necessary to remove them. This stability and this requirement for an external release treatment are, to date, the most useful characteristics of a dormant state: what was initially a simple relay strongly controlled by LDIs has evolved into an independent barrier. It may be associated with membrane properties of certain cells near the bud.

Dormancy induction and release in tubers

Here we will refer only to the work conducted with Helianthus tuberosum at the University of Clermont-Ferrand (Courduroux, 1967; Gendraud, 1981; Tort et al., 1985). Apparently not known outside France (see Ewing, 1987), the following results provide some answers to the questions we mentioned earlier for tree buds.

Tuberization in Helianthus tuberosum

We usually mention the concept of tuber dormancy and not tuber-bud dormancy. This detail is important: the subject of the following report is the parenchymatous
tissue in the tuber and not the bud itself, as for trees. As a matter of fact, before the recent studies summarized above, stem tissues were only rarely regarded as regulatory elements of tree dormancy.

The tuberization of a stolon, a long underground stem with scaly leaves, occurs under the influence of correlations exerted by the aerial shoots of the plant (LDIs). As a matter of fact, when we suppress these LDIs, we stop the tuberization in progress and we initiate both the inflexion of the terminal bud, due to gravitropism, and its growth and transformation into a long shoot bearing assimilating leaves (Fig. 8). The tuber is considered to be dormant only later, when it reaches its final length; at this stage, even when isolated, it does not develop a growing shoot, but only a new tuber (Fig. 8). Dormancy and tuberization are quite different processes: the latter appears to be controlled by reversible correlations.

A dormant tuber, even during the period of maximum rest intensity, may resume growth without releasing dormancy when placed under appropriate temperature conditions. Its buds do not evolve as long shoots, but as new young tubers, directly inserted on the 'mother' tuber. This tuber growth is very useful for assessing dormant or non-dormant stages of growing tubers. A chilling treatment (or any other treatment of the same kind) may trigger dormancy release, and brings back the ability to develop long leafy shoots.

The in vitro culture of buds attached to a little pyramid of parenchyma produces either little dormant tubers from a dormant explant or long shoots from a non-dormant explant, but also non-dormant tubers from non-dormant explants if the culture

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Fig. 8. Evolution of a tuber's ability to produce an elongating stem after decapitation of *H. tuberosus*. During the growth period, decapitation induces the elongation of the terminal bud either on the growing underground stem (1) or on the developing tuber (2). During dormancy, decapitation has no effect and the tuberization continues (3). Tubers isolated from the plant and grown at optimal temperature develop a leafy stem when non-dormant and a second tuber when dormant.
medium exerts a correlative inhibition (due to the presence of cytokinins, for instance). These latter tubers may rapidly develop long shoots when transplanted into a non-inhibiting medium (Courduroux, 1967).

Some results

The parenchyma of a dormant tuber is unable to synthesize NTPs. That from a tuber able to produce long shoots is always able to synthesize them, regardless of whether it is a cutting from in vitro plantlets or from a plant growing in the field. This observation may be related to the fact that the rate of biomass increase is much higher in a growing shoot than during the production of a daughter tuber.

The pHt in the parenchyma of a dormant tuber is higher than that in a non-dormant tuber. It has been demonstrated that the former exports stored nutrients with much more difficulty and that its buds are therefore poorly supplied (Gendraud and Lafleuriel, 1983; 1985; Tort and Gendraud, 1984). It is a pity that the pHt of the related buds could not be measured. In fact, the theory supposes that it varies only slightly, and that it is lower than the pHt of dormant tuber parenchyma, and higher than the pHt of non-dormant parenchyma, as in tree buds. These arguments are indirect and were easier to demonstrate with trees, but the results are, nevertheless, very similar in both cases.

Detailed studies of energetics, different enzymatic systems and carbohydrate metabolism in dormant and non-dormant tubers have been made (Poole, 1978; Spanswick, 1981; Maslowski et al., 1984; Sze, 1984; Matsumoto and Yamaha, 1984; Marre and Ballarin-Denti, 1985; Petel, 1986; Petel and Gendraud, 1986). Similar studies have never been conducted on trees. These investigations provided very interesting new information. We will focus on experiments run with cellular extracts, enriched with plasmalemma, containing an ATPase which could be isolated, purified and analyzed for Michaelis characteristics (Table III). This membrane-bound ATPase is different in dormant tubers and in non-dormant, non-inhibited ones able to produce leafy shoots. In the first case, due to modifications of the electron transporting system and of the resulting particular energy metabolism, the cells of the parenchyma export with great difficulty ions and nutrient molecules towards the potential center of use constituted by the buds. They do it with more ease in the second case (Fig. 9). The whole dormant parenchyma is a poor exporter, despite its richness in available storage compounds; all cells are organized as a differentiated barrier between stem and bud, whereas in trees only a few cell layers play this role.

The most significant result seems to be the following: a young tuber, growing under correlative inhibitions originating

<table>
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<tr>
<th>Tuber</th>
<th>ATPase activity (pkat/mg of protein)</th>
<th>ATPase properties</th>
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<tr>
<td></td>
<td>+ KCl</td>
<td>+ vanadate</td>
</tr>
<tr>
<td>Dormant</td>
<td>95 ± 10</td>
<td>116</td>
</tr>
<tr>
<td>Non-dormant</td>
<td>40 ± 6</td>
<td>53</td>
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from leafy shoots or from the culture medium, and therefore unable to produce a leafy shoot, has the same biochemical characteristics as a dormant tuber. In particular, the ATPase is of the ‘dormant’ type. The tuber exhibits a poor ability to grow and not a specific property of a dormant stage per se.

But there is a fundamental difference between both situations, and we have stressed it several times: for a non-dormant tuber, the release from correlative inhibitions (LDIs) immediately suppresses the inability to translocate nutrients to the bud, in the same way as when a pea plantlet is decapitated or when the terminal bud of an oak seedling attains the end of apparent rest. The plasmalemma-associated ATPase is replaced or rapidly acquires new properties. However, in the case of a dormant tuber, a chilling treatment is necessary to slowly and progressively lead to the same result.

What may therefore best distinguish a correlative inhibition from dormancy is the stability of membrane structures to which ATPases and other enzymes, for instance dehydrogenases, are bound. This stability is weak during correlative inhibitions, that is when SDIs act as relays for LDIs, but becomes very strong during dormancy; so strong, in fact, that only a specific induction treatment, such as chilling, may make the membranes return to their initial stage. This hypothesis remains to be confirmed by experimental evidence.
General discussion and conclusion

Assuming that a resting bud is a bud suffering from nutritional deficiencies during apical dominance, may be common sense. How else could we describe the physiological status of resting buds, while we no longer believe in the existence of specific inhibitors or real correlation hormones, whose presence would paralyze the cells they inhabit? The fact that such nutritional deficiencies have been discovered in oak seedlings with rhythmic growth and in some different cases of dormancy is not a surprise. But there is still a problem to be solved: what are the origins of these deficiencies and the physiological mechanisms underlying them? Are they identical in all cases?

To assume that short distance correlations (SDIs) are present in all these cases in only a first element of an answer. It means that a meristem and the distal part of the bud, where active organogenesis and elongation are taking place, are not independent ‘self organizing centers’ as we had imagined, but that they respond to external commands, produced by their surrounding cells, which, even when they are active, only slightly translocate metabolites, and, therefore, isolate other cells, which would be able to grow actively. Some enzymes such as membrane-bound ATPases, could play a determining role. Unfortunately, the location of these cells remains imprecise and could vary for different physiological situations. Assuming the existence of well-determined barriers does not fit in all cases; the whole tuber parenchyma or the stem tissue of a hardening tree may be involved. Also, the concept of short distance is more a model than a concrete reality.

The primary interest of SDI is to somehow clarify the idea of a relay acting between correlative influences: an organ or a tissue emits a signal, which is received and translated by another tissue or another organ whose behavior is thus modified. What is the vector of this information? A hormone? The evidence supporting the latter is still weak. Indeed, auxins may be directly responsible for the polarization of some neighboring cells at the base of an inhibited axillary bud. But is such a mechanism present during all correlative inhibitions? What happens when the ‘source’ organs do not diffuse any auxin, as in adult leaves? How does abscisic acid act, if its accumulation is a consequence and not the cause of the deficient stage, as in many stress situations? Concepts of this kind, that were once used in plant growth regulator studies are now being strongly criticized (Trewavas, 1981). But growth regulators surely act at the cellular level or at membrane interfaces, in order to control permeabilities and/or translocations. But the auxins (for example) acting at this level certainly do not originate directly from the apical bud!

That SDIs associated with correlative inhibitions disappear immediately after the disappearance of the corresponding LDIs seems normal after the above mentioned considerations. The signal for LDIs has to circulate permanently in order to remain effective. In the case of dormancies, the SDIs remain active even while the corresponding LDIs have completely vanished. This stabilization of SDIs seems to be the most significant characteristic of a dormant stage. Understanding the mechanisms will be a fascinating challenge.

Finally, we would like to recall that some kinds of LDIs, emitted by an organ or a tissue, may be very short-lived (some 10 min in Phyllanthus, Nozeran et al., 1971), but nevertheless induce long-term or permanent modifications. These self-maintaining states have, with reason,
been compared to dormancies. In some of these cases, the concept of relay (SDIs) between different correlative inhibitions may be very apparent (stolon plagiotropism on *Stachys silvatica*; Pfirsch, 1970). In other cases, it seems that no SDI is detectable, as if the meristematic cells themselves were definitely transformed (Nozeran *et al*., 1971; Champagnat, 1974; 1988). This fact is not in contradiction with preceding ones: a stage beyond SDIs could have been achieved, a further ‘miniaturization’ could have taken place. This evolution could be necessary when certain qualitative modifications of development and further differentiation processes are going on, and not only simple quantitative variation of growth.

Acknowledgments

The text was translated and edited by E. Dreyer, and J.F. Muller drew the figures.

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