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Role of Endogenous Abscisic Acid in Potato Microtuber Dormancy

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Potato (*Solanum tuberosum* L. cv Russet Burbank) microtubers generated in vitro from single-node explants contained substantial amounts (approximately 250 pmol/g fresh weight) of free abscisic acid (ABA) and were completely dormant for a minimum of 12 weeks. Microtubers that developed in the presence of 10 μM fluridone (FLD) contained considerably reduced amounts (approximately 5–25 pmol/g fresh weight) of free ABA and exhibited a precocious loss of dormancy. Inclusion of exogenous racemic ABA in the FLD-containing medium suppressed the premature sprouting of these microtubers in a dose-dependent manner. At a concentration of 50 μM, exogenous ABA restored internal ABA levels to control values and completely inhibited FLD-induced precocious sprouting. Exogenous jasmonic acid was ineffective in suppressing FLD-induced sprouting. Application of FLD to preformed, fully dormant microtubers also resulted in a reduction in internal ABA content and precocious sprouting. These results indicate that endogenous ABA is essential for the induction and maintenance of potato microtuber dormancy.

The phenomenon of dormancy or developmental arrest is widespread in the plant kingdom. Examples of dormancy can be found in nearly all meristematic organs, including seeds, apical and lateral vegetative and floral buds, bulbs, corms, and tubers (Nooden and Weber, 1978; Saunders, 1978). Because of its basic and applied importance, the regulation of dormancy has attracted considerable interest. Many hypotheses have been proposed to explain the regulation of meristematic activity during dormancy (Rappaport and Wolf, 1968; Nooden and Weber, 1978). However, as with many other aspects of developmental regulation, the role(s) of endogenous hormones has attracted the greatest attention (Rappaport and Wolf, 1968; Saunders, 1978). Initial theories concerning the hormonal regulation of dormancy focused on the role of endogenous inhibitors as dormancy-inducing and/or dormancy-maintaining factors (Hemberg, 1961). Later, this paradigm was modified somewhat to include a role for endogenous growth promoters as well (Rappaport and Wolf, 1968; Nooden and Weber, 1978; Saunders, 1978).

Originally isolated and characterized from abscising lupin and cotton fruit and dormant sycamore buds and leaves, the hormone ABA has been considered to play a pivotal role in the regulation of plant dormancy (Addicott and Cairns, 1983). Unequivocal support for a role for endogenous ABA in the process of seed dormancy has come from studies using genetically defined and physiologically characterized ABA-synthesis and -response mutants. In *Arabidopsis*, seed dormancy is absent in both types of mutants, whereas in tomato, dormancy is absent in the ABA-deficient *sit* mutant (Karssen et al., 1990).

Complementing these data are studies using inhibitors of carotenogenesis. ABA is now thought to be synthesized from a C_{40} carotenoid precursor via a series of cleavage and oxidation steps (Parry and Horgan, 1991). The application of carotenoid biosynthesis inhibitors, such as the herbicides norflurazon and FLD (Sanderman and Böger, 1989), often results in a reduction in ABA content as well. FLD treatment results in the precocious germination of both corn and sunflower embryos, and exogenous ABA reverses this effect (Hole et al., 1989; Le Page-Degivry and Garello, 1992).

As with many other perennating organs, freshly harvested potato (*Solanum tuberosum* L.) tubers exhibit a period of dormancy. The length or depth of this dormancy is cultivar specific and can be influenced by both pre- and postharvest conditions (Burton, 1989). Uncontrolled sprouting during storage is detrimental to the nutritional quality of potatoes and often results in exacerbated postharvest disease progression, both of which result in severe financial losses to producers. Potato tuber dormancy is thought to be regulated by endogenous hormones (Hemberg, 1985; van Es and Hartmans, 1987; Burton, 1989). Existing evidence for this theory is largely circumstantial and is based entirely on correlative and pharmacological evidence.

Pioneering studies by Hemberg (1961, 1985) using bioassays and acid extracts prepared from potato periderm and fractionated by paper chromatography led to the hypothesis that potato tuber dormancy was controlled by the levels of a complex mixture of substances collectively termed inhibitor-β (Bennet-Clark and Kefford, 1953). As detected by bioassay, levels of inhibitor-β were highest in deeply dormant tubers and declined during storage in a manner roughly coincident with the gradual loss of tuber dormancy. With the subsequent identification of ABA as an endogenous component of the inhibitor-β complex (Cornforth et al., 1966), attention shifted to the role of ABA as the principal regulator of potato dormancy (Rappaport and Wolf, 1968). Although surprisingly few direct analyses of ABA levels in potatoes have been reported, ABA levels are generally highest in freshly harvested (deeply dormant) potatoes and decline as sprouting commences (Korableva et al., 1980; Coleman and King, 1984;

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Abbreviations: FLD, fluridone; JA, jasmonic acid.

van den Berg et al., 1991). In spite of the generality of this correlation, exceptions do exist. For example, although dormancy is lost during storage at 2 to 3°C, endogenous ABA levels can actually rise (Coleman and King, 1984; J.C. Suttle, unpublished data). In addition, suppression of sprout growth in nondormant tubers by exogenous ABA requires relatively high treatment concentrations and, in the case of single applications, is transient (Hartmans and van Es, 1979). Therefore, the role of endogenous ABA in potato dormancy remains unresolved. Similar uncertainties exist with regard to the role of ABA in other forms of bud dormancy (Saunders, 1978; Trewavas and Jones, 1991).

In vitro tuberization systems for potato have been developed primarily for germplasm conservation and multiplication (Ewing, 1985; Tovar et al., 1985). Using this type of system, it is possible to alter the physiology of the developing microtuber by manipulating both the chemical composition of the tuberization medium and the environmental conditions during tuber formation. In this report, the role of endogenous ABA in tuber dormancy is assessed using an in vitro tuberization system and the herbicide FLD. Portions of this study have been presented elsewhere in abstract form (Suttle, 1993).

MATERIALS AND METHODS

Plant Material

Certified seed tubers of *Solanum tuberosum* L. cv Russet Burbank were surface sterilized using a 1:4 (v/v) dilution of commercial bleach followed by extensive washing with running deionized water. The tubers were allowed to sprout in the dark (25 ± 1°C) for 11 d. The tuber with the greatest number of sprouts was selected and the sprouts were removed. The excised sprouts were surface sterilized using a 1:5 (v/v) dilution of commercial bleach (15 min) followed by extensive washing with sterile distilled water. Segments containing a single node were cultured in 15-cm tubes (22-mm i.d.) containing 10 mL of Murashige and Skoog basal salts and vitamins (Murashige and Skoog, 1962) supplemented with 3% (w/v) Suc and 0.2% (w/v) phytagel. The pH of the medium was adjusted to 5.8 prior to the addition of phytagel. Plantlets were cultured under fluorescent lighting (100 μmol m^{-2} s^{-1}) at room temperature. Plantlets were subcultured every 3 to 4 weeks using single-node explants transferred to fresh sterile medium.

Tuber Induction

Single-node explants (including the leaf) were excised from the lower one-third of the plantlet. These explants were subcultured in phytatrays (Sigma)[1] containing approximately 100 mL of Murashige and Skoog basal salts supplemented with 0.05 mg/L biotin, 0.5 mg/L folic acid, 2 mg/L Gly, 100 mg/L *myo*-inositol, 5 mg/L nicotinic acid, 0.5 mg/L pyridoxine-HCl, 0.5 mg/L thiamine-HCl, 10 mg/L kinetin, 0.1 mg/L IAA, 8% (w/v) Suc, the test compounds, and 0.6% (w/v) agar (pH 5.7). Compounds to be tested were prepared as 10,000-fold concentrated DMSO stocks and were added to the autoclaved medium prior to gelling. Explants were incubated in the dark (20 ± 1°C) and the progress of tuberization and sprouting was monitored periodically thereafter.

[1] Mention of a trademark or proprietary product does not constitute a guarantee or warranty of the product by the U.S. Department of Agriculture and does not imply its approval to the exclusion of other products that may also be suitable.

Mid-Course Treatment with FLD

Microtubers were initiated as described above and were allowed to develop for 11 weeks. They were then excised from the explants and were incubated for 6 weeks in sterile Petri dishes (dark, 20 ± 1°C, 90% RH). After this, the excised microtubers were treated for 24 h with deionized water with or without 10 μM FLD on an oscillating shaker at room temperature. The treated tubers were then placed in Petri dishes, incubated in the dark as before, and monitored periodically thereafter.

ABA Extraction and Analysis

At the indicated times, groups of three microtubers were excised from the explants, rinsed extensively with reagent-grade water, weighed, and frozen in liquid nitrogen. The frozen tubers were stored at −70°C until analyzed. The frozen tubers were placed in 80% (v/v) aqueous acetone and were mechanically homogenized. The extracts were clarified by filtration through glass-fiber filters and 1.4 nCi of (±)-[G-^{3}H]ABA (50 Ci/mmol) was added to follow recovery. The extracts were taken to dryness under reduced pressure (30°C) and were redissolved in water with the aid of sonication. Typically, the reconstituted extract was split into two equal aliquots. One aliquot was used for the determination of free ABA and the other for total ABA after base hydrolysis.

For the analysis of free ABA, the reconstituted extracts were adjusted to a pH of approximately 2.6 using 2 N HCl and were loaded onto preconditioned C_{18} Sep-Pak cartridges (Waters Associates, Milford, MA). The Sep-Pak cartridges were washed with 10 mL of 1 mM HCl and the ABA-containing fractions were then eluted using 10 mL of methanol. The methanol was evaporated under a stream of nitrogen (40°C) and redissolved in 1 mL of Tris-buffered saline. Free ABA was quantitated using an indirect ELISA procedure (Walker-Simmons, 1987). The determined values were corrected for recovery of the internal standard, which typically exceeded 90%. The validity of the indirect ELISA analysis of free ABA in these extracts was verified in preliminary experiments using capillary GC coupled with an electron-capture detector (data not presented).

Chemicals

FLD (technical grade, purity ≥ 98%) was purchased from Chem Service, Inc. (West Chester, PA). Phytagel and all other tissue culture chemicals were from Sigma. JA was obtained from Apex Organics Ltd. (Devon, UK). (±)-[G-^{3}H]ABA was obtained from Amersham Life Sciences (Arlington Heights, IL). Mouse monoclonal antibodies against ABA were from Idetek (San Bruno, CA).

Experimental Procedures

All experiments described in this paper were conducted a minimum of two times. Whenever possible, individual treatments within an experiment were replicated ($n = 3$). Due to the inherent variability of in vitro tuberization efficiency, the number of tubers examined in each experiment varied. A minimum of 40 explants/replicate were used to examine the effects of FLD on microtuber development (see Figs. 2 and 3 and Table I) and 40 tubers/treatment were used to evaluate the effects of FLD on preformed tubers (see Fig. 4). Data from typical experiments are presented.

RESULTS

After excision from the intact plantlet and subculture, microtubers were formed from the axillary buds of single-leaf explants either directly (sessile tubers) or after a brief period of stolon growth (aerial tubers). Microtuber formation could be observed after 1 week of subculture, and final growth (as judged by tuber diameter or fresh weight) was achieved after 9 to 12 weeks (data not presented). Initially, the efficacy of both norflurazon and FLD were compared in this system. Although both compounds inhibited ABA accumulation in developing microtubers at micromolar concentrations, norflurazon was slightly more phytotoxic (data not presented). Therefore, FLD was used for the remainder of these studies.

After 15 weeks of subculture, the free ABA content of untreated microtubers was 431 ± 35 pmol/g fresh weight (Fig. 1). Microtubers that developed in the presence of FLD at concentrations of 0.1 μM or greater contained substantially reduced levels of free ABA. The effect of FLD on free ABA content was dose dependent and the inhibition was essentially complete (>95%) at 10 μM. At this FLD concentration, there was a slight (but statistically insignificant) decrease in microtuber weight. However, in all other respects FLD-treated microtubers were morphologically indistinguishable from their untreated counterparts.

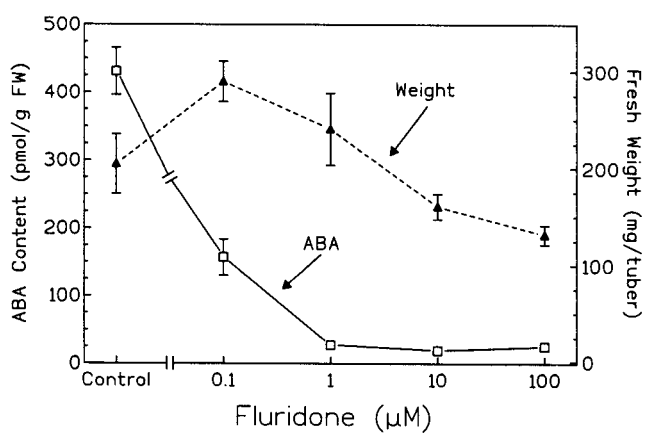

Figure 1. Effects of increasing concentrations of FLD on microtuber fresh weight (dashed line) and free ABA content (solid line). Both parameters were determined after 15 weeks of development in the presence or absence of FLD. Brackets indicate ± SE ($n = 3$).

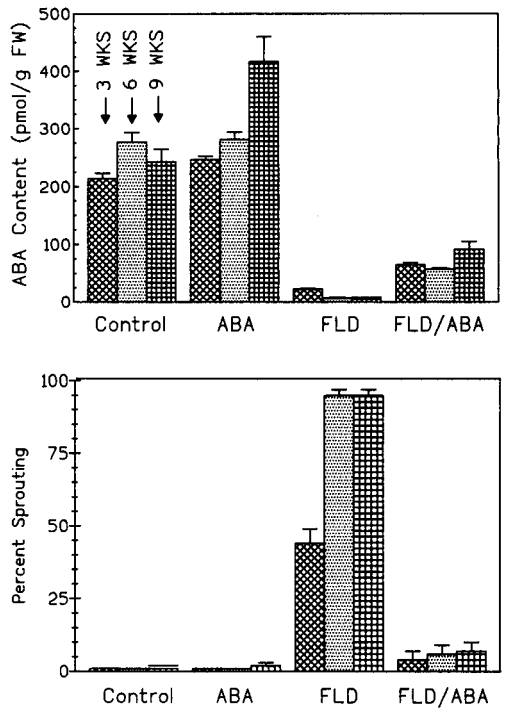

Figure 2. Effects of continuous exposure to 10 μM FLD ± 5 μM (±)-ABA on microtuber ABA content (upper panel) and sprouting percentage (lower panel). Both parameters were determined after 3, 6, and 9 weeks of microtuber development. Brackets indicate ± SE ($n = 3$).

As has been found by others (Korableva et al., 1980), the content of base-labile, conjugated ABA in these tubers was low and reflected that of free ABA. For example, in a typical experiment, after 9 weeks of development free ABA levels in control and FLD-treated tubers were 243 ± 22 and 5 ± 1 pmol/g fresh weight, respectively, whereas conjugated ABA levels were 44 ± 23 and 3 ± 1 pmol/g fresh weight for control and FLD-treated tubers, respectively. In no case was the FLD-induced reduction in free ABA content in these tubers accompanied by an increase in conjugated ABA. Therefore, only values for the free ABA content will be discussed.

A time-course study examining ABA content and microtuber dormancy was conducted next. After 3 weeks of development, the ABA content of untreated microtubers was 214 ± 9 pmol/g fresh weight (Fig. 2). Subculture of these tubers for an additional 6 weeks resulted in little additional increase. As before, microtubers that developed in the presence of 10 μM FLD contained substantially reduced levels of ABA. After 3 weeks of development, FLD-treated tubers contained 23 ± 1 pmol/g fresh weight. This value fell to 5 ± 1 pmol/g fresh weight during an additional 6 weeks of subculture. Internal ABA levels in untreated tubers were unaffected by continuous exposure to 5 μM (±)-ABA for 6 weeks. Only after 9 weeks did exogenous (±)-ABA have any effect on internal ABA levels in these tubers. In contrast, treatment with exogenous (±)-ABA resulted in a significant increase in endogenous ABA levels in FLD-treated tubers.

Untreated microtubers were completely dormant (i.e. 0% sprouting) throughout the 9 weeks of observation (Fig. 2, lower). Accompanying the reduction in ABA content in FLD-treated microtubers was a progressive and, ultimately, near-total loss of dormancy. The sprouting percentages of FLD-treated tubers were 44, 95, and 96% after 3, 6, and 9 weeks, respectively. Exogenous (±)-ABA reduced sprouting in FLD-treated tubers to near-control values (4, 6, and 6% after 3, 6, and 9 weeks, respectively).

The effects of exogenous (±)-ABA in FLD-treated tubers was examined in more detail using a range of external ABA concentrations. Microtubers that developed for 6 weeks in the absence of any treatment contained 209 ± 18 pmol/g fresh weight ABA and were completely dormant (Fig. 3, bars). Continuous FLD treatment reduced the endogenous ABA content to 10 ± 3 pmol/g fresh weight and essentially abolished microtuber dormancy (92 ± 8% sprouting). Exposure to exogenous (±)-ABA at concentrations of 0.5 μM or greater resulted in a dose-dependent increase in endogenous ABA content and a concomitant decrease in sprouting percentage. At 50 μM external (±)-ABA, endogenous levels of ABA were restored to roughly control values and the precocious sprouting was essentially abolished. The effects of 5 and 50 μM (±)-ABA were persistent. After 9 weeks of development, microtubers from the control, 5-, and 50-μM treatments were excised from the explants and were transferred to medium-free Petri dishes. Three weeks later, the sprouting percentages for the control, 5-, and 50-μM treatment groups were 0, 7, and 2%, respectively (data not presented).

At the physiological level, the biological activities of ABA and JA often overlap (Parthier, 1991). Therefore, it was of interest to examine the efficacy of JA in this system. Application of 5 μM (±)-ABA suppressed FLD-induced sprouting to a considerable extent (Table I). In contrast, 5 μM JA elicited no such response. Higher concentrations of JA were not tested.

The effects of FLD treatment on preformed and fully dormant microtubers were examined next. After 17 weeks of development, microtubers were exposed to 10 μM FLD for 24 h and transferred to medium-free Petri dishes, and the endogenous ABA content and dormancy status (i.e. sprouting percentage) were monitored thereafter. After 11 d of incubation, the ABA content of control microtubers was 209 ± 16 pmol/g fresh weight (Fig. 4). Twenty-five days later, it had fallen slightly to 172 ± 36 pmol/g fresh weight. Dormancy was still evident in these tubers, especially after 7 d of incubation, when the sprouting percentage was just 3%.

Table I. *Effects of ABA and JA on FLD-induced precocious sprouting*

Microtubers were generated in vitro on microtuber induction medium alone or on medium containing 10 μM FLD in the presence or absence of 5 μM ABA or JA. Sprouting percentages were determined after 6 weeks of development.

Treatment	Percent Sprouting
Control	2
FLD	78
FLD + ABA	27
FLD + JA	90

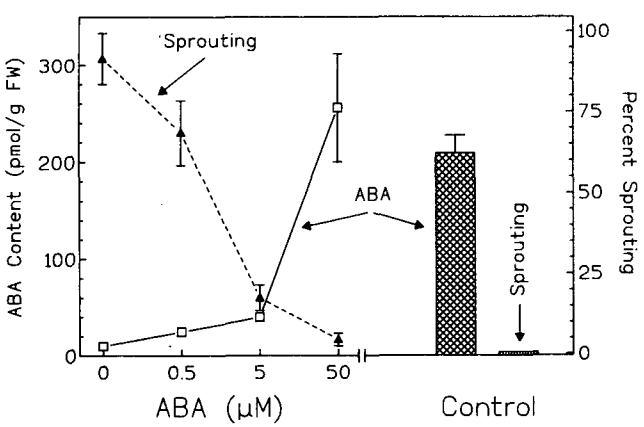

Figure 3. Effects of continuous exposure to increasing concentrations of exogenous (±)-ABA on ABA content (solid line) and sprouting percentage (dashed line) in 10 μM FLD-treated tubers. Bars indicate ABA content (left) and sprouting percentage (right) of untreated microtubers. All values were determined after 6 weeks of microtuber development. Brackets indicate ± SE (n = 3).

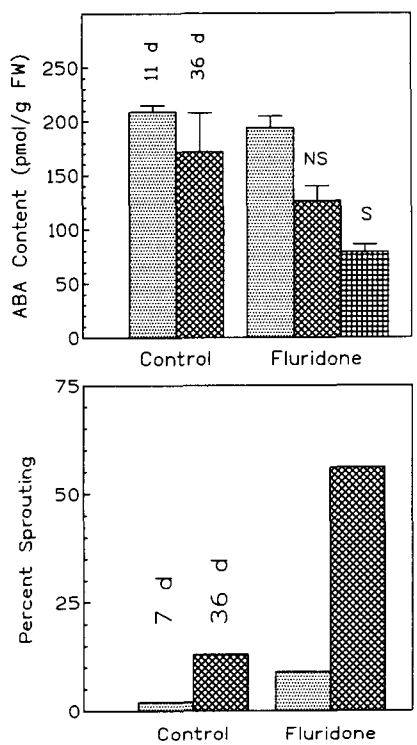

Figure 4. Effect of FLD on ABA content (upper panel) and sprouting percentage (lower panel) in fully developed, dormant microtubers. Untreated microtubers were allowed to develop for 17 weeks and were then treated for 24 h with 10 μM FLD. Determinations were made 7 to 11 and 36 d after FLD treatment. Brackets (ABA analysis only) indicate ± SE (n = 3). NS, Nonsprouting; S, sprouting after 36 d.

By the second time point (29 d later), these tubers were beginning to exit dormancy and exhibited 13% sprouting.

Eleven days after FLD treatment, the endogenous ABA content was similar to that of control tubers (i.e. 194 ± 11 pmol/g fresh weight), and after 7 d these tubers exhibited limited (9%) sprouting. Twenty-nine days later, FLD-treated microtubers were largely nondormant (i.e. 56% sprouting). The endogenous ABA content was analyzed separately in both sprouted and nonsprouted FLD-treated tubers. The internal ABA content of nonsprouted and sprouted microtubers was 126 ± 14 and 79 ± 7 pmol/g fresh weight, respectively.

DISCUSSION

With the exception of seed or embryo dormancy, the role of endogenous ABA in the regulation of bud or meristem dormancy remains uncertain (Saunders, 1978; Trewavas and Jones, 1991). As demonstrated in the case of seed dormancy, the ability to block ABA synthesis or action with a reasonable degree of specificity is essential in determining the role of ABA in bud dormancy (Karssen et al., 1990). This can be accomplished either by using physiologically defined genetic mutants or through the use of chemical inhibitors such as the bleaching herbicide FLD. Because of its effects on photosynthesis, the use of FLD as a probe of ABA involvement is limited to those situations in which the plant material is not dependent on photoautotrophic growth. Potato microtubers generated in vitro are heterotrophic and therefore offer a unique opportunity to examine the role of ABA in the process of bud dormancy.

Continuous exposure of developing potato microtubers to 10 μM FLD results in the formation of microtubers that are essentially devoid of endogenous ABA and exhibit precocious sprouting (Figs. 1 and 2). The simultaneous application of exogenous racemic ABA to FLD-treated microtubers results in a dose-dependent increase in endogenous ABA and a concomitant decrease in premature sprouting (Figs. 2 and 3). The continuous application of 50 μM racemic ABA to FLD-treated microtubers restored endogenous ABA levels to control values and essentially abolished precocious sprouting (Fig. 3). Further subculture of these ABA-treated microtubers for an additional 3 weeks in the absence of exogenous ABA resulted in little (2%) additional sprouting (data not presented).

The suppression of sprout growth in FLD-treated tubers by exogenous ABA can be explained in one of two ways. Exogenous ABA either restored dormancy in these tubers or caused a nonspecific inhibition of growth. The available information favors the first explanation. First, exogenous ABA is only weakly active as a growth inhibitor when applied to nondormant tubers. For example, suppression of sprout growth in eyes excised from nondormant tubers required repeated or continuous exposure to relatively high concentrations of ABA (van Es and Hartmans, 1969). In some cases, a modest stimulation of sprout growth by exogenous ABA has been observed in nondormant tubers (Ji and Wang, 1988). As described above, exogenous ABA was effective at external concentrations that restored internal ABA levels to values similar to those found in untreated tubers. This information, together with the observed persistence of ABA action in FLD-treated microtubers, suggests that exogenous ABA restored dormancy in FLD-treated tubers.

Exogenous JA was ineffective in suppressing FLD-induced sprouting at a concentration (5 μM) at which the efficacy of ABA was clearly evident (Table I). Many plant responses to ABA can be mimicked by JA and vice versa (Parthier, 1991). This is especially true with regard to growth inhibition, where both compounds display similar inhibitory activities. In particular, longitudinal growth of potato roots and shoots is inhibited by micromolar concentrations of JA (Ravnikar et al., 1992). Therefore, the inability of JA to suppress sprouting in FLD-treated tubers further strengthens the hypothesis that ABA is not merely acting as a growth inhibitor but rather is restoring dormancy in FLD-treated microtubers.

Application of FLD to fully developed and dormant microtubers also resulted in a decrease in endogenous ABA levels and a concomitant increase in premature sprouting (Fig. 4). The decrease in ABA levels after FLD treatment occurred slowly, becoming evident only about 5 weeks after treatment. Because tubers exhibit readily measurable rates of ABA metabolism throughout dormancy (J.C. Suttle, unpublished data), this delay may reflect the time required to deplete endogenous pools of the C_{40} precursors or other intermediates involved in ABA biosynthesis. Inclusion of 100 μM exogenous racemic ABA during the 24-h FLD treatment failed to elevate internal ABA levels or reduce premature sprouting (data not presented). The failure of exogenous ABA to affect internal ABA levels (hence dormancy) in fully developed tubers was likely due to a combination of factors, including the limited uptake period, suboptimal treatment concentration, and the above-mentioned active oxidative catabolism of ABA by these tubers. Nevertheless, these data suggest that the sustained synthesis of ABA is also required for the maintenance of the dormant state once it has developed.

Genetically defined ABA-synthesis and -response mutants have been useful in determining the roles of ABA in seed development and dormancy (Karssen et al., 1990). An ABA synthesis mutant of potato (droopy) has been described, and the biochemical basis of this mutation has been characterized (Quarrie, 1982; Duckham et al., 1989). However, this mutation occurs in *Solanum phureja*, a species known for its near-total lack of tuber dormancy (Hawkes, 1992). Therefore, this mutant is of little value in the present context.

In conclusion, the results presented herein provide unequivocal evidence that endogenous ABA is directly involved with the initiation and possibly the maintenance of potato microtuber dormancy. The demonstrated physiological fidelity of microtubers and their field-grown counterparts (Ewing, 1985) suggests a similar role for ABA in these tubers as well. Recently, it has been shown that application of FLD to *Lilium* bulblets results in precocious sprouting and that exogenous ABA reverses this effect (Kim et al., 1994). The demonstrated ability of ABA to regulate meristem dormancy in tissues as diverse as seeds, bulbs, and tubers suggests that its role in dormancy may be more universal than was once generally acknowledged.

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