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Arabinose Kinase-Deficient Mutant of *Arabidopsis thaliana*[1]

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ABSTRACT

A mutant of *Arabidopsis thaliana* that is sensitive to exogenous L-arabinose has been isolated. Comparisons of growth of the wild type, mutant, and F1 and F2 progeny of crosses showed the arabinose-sensitive phenotype is semidominant and segregates as a single Mendelian locus. Crosses of the mutant to marker strains showed the mutation is linked to the *eceriferum-2* locus on chromosome 4. *In vivo* incorporation of exogenous labeled L-arabinose into ethanol-insoluble polysaccharides was greatly reduced in the mutant with a concomitant accumulation of free labeled arabinose. Enzyme assays of crude plant extracts demonstrated a defect in arabinose kinase activity in the mutant.

NDP-sugars[3] are the activated sugar donors of polysaccharide and glycoprotein biosynthesis. Many NDP-sugars, mainly UDP- and GDP-sugars, can be synthesized via *de novo* pathways from UDP-glucose or GDP-mannose via a series of NDP-sugar interconversions. However, for some sugars, there also exist "salvage" pathways that can convert free monosaccharides into NDP-sugars via a phosphorylated intermediate (see ref. 5 for a review).

The salvage pathway for L-arabinose, for example, is shown in Figure 1. Arabinose is converted to UDP-arabinose via the sequential action of the enzymes arabinose kinase (ATP:L-arabinose-1-phosphotransferase, EC 2.7.1.46) (3) and uridine diphosphoarabinose pyrophosphorylase (18). It has not been determined whether this latter activity is specific for arabinose, pentoses in general, or is due to a lack of specificity of, for example, UDP-D-glucose pyrophosphorylase. The *de novo* pathway of UDP-arabinose biosynthesis from UDP-glucose via UDP-glucuronic acid and UDP-xylose is also shown in Figure 1. Similar salvage pathways utilizing D-galactose, L-fucose, and D-galacturonic acid have also been shown to occur in plant tissues (5). Similarly, free D-glucuronic acid can be metabolized as an intermediate in the myo-inositol pathway that converts glucose-6-phosphate via *myo*-inositol to UDP-glucuronic acid (15). The importance of the salvage pathways in contributing to the pools of NDP-sugars by recycling free monosaccharides derived from polysaccharide turnover and in preventing those same monosaccharides from accumulating and thereby interfering with aspects of plant metabolism is uncertain.

This paper describes the characterization of a mutant of *Arabidopsis thaliana* that is sensitive to exogenous L-arabinose and is shown to be deficient in arabinose kinase activity. The lack of any visible defect in the mutant suggests that the arabinose salvage pathway is not essential or that only residual activity is sufficient for normal growth.

MATERIALS AND METHODS

Plant Materials

The *Arabidopsis thaliana* (L.) Heynh. strains used in this study were wild type (var Columbia), the arabinose-sensitive mutant, CC3, (var Columbia), and W100 (var Lansberg) which is homozygous for the following markers: *an, apl, er, py, hy2, gl1, bp, cer2, ms1,* and *tt3* (11). The mutant CC3 was isolated fortuitously when testing a number of individual M3 families derived from EMS-mutagenized M1 seed for growth on media containing various sugars. A rigorous mutant isolation program has not been undertaken. All the experiments described below with the exception of the enzyme assays were done with an M4 population of seeds without backcrossing to the wild type. Enzyme assays were done on a homozygous mutant population derived after four successive backcrosses to the wild type.

Plants were grown at 23 to 26°C under continuous fluorescent lighting (150–250 μE m^{-2} s^{-1}). In pots, plants were grown in a sand/compost mix (Debco, Australia) watered with nutrient medium (8). The harvesting of seed and cross-pollination were as described in Somerville and Ogren (19). In Petri dishes, plants were grown on nutrient medium solidified with agar (0.8%, Difco) with the addition of 2% sucrose or other sugars at specified concentrations.

Chemicals

Sugars used were from Sigma Chemical Co. L-[1-^{3}H]Arabinose (2.0 Ci/m mol) was the generous gift of S. Read.

In Vivo Growth Measurements

Seeds were germinated on minimal medium containing sucrose and transferred at 4 d postimbibition onto medium containing various concentrations of ara. Some plants were weighed after 4 d and others after 7 d growth in the presence of ara. F1 seedlings were plated only on medium containing 0 and 10 mM ara and weighed after 7 d growth. Ten seedlings were weighed for each data point.

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[3] Abbreviations: NDP-sugars, nucleoside diphosphate sugars; ara, L-arabinose.

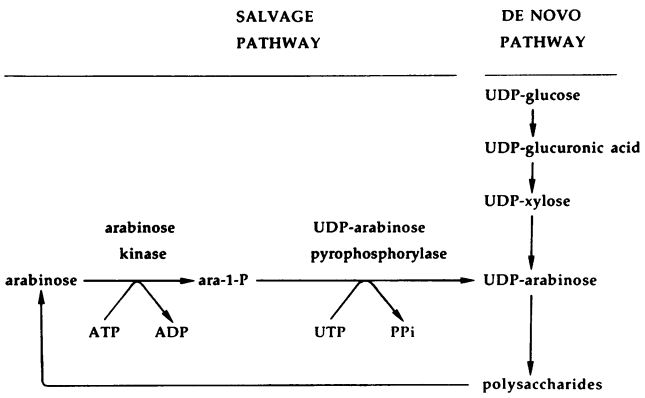

Figure 1. Metabolism of L-arabinose in plants.

Genetic Crosses

Crosses for the purpose of mapping the arabinose-sensitive locus were done with the W100 marker strain as the maternal parent. F1 progeny were grown under nonselective conditions, allowed to self-pollinate and harvested individually. F2 progeny were examined as described in "Results." For the linkage analysis, recombination percentages (r) were calculated using the linkage analysis program of Suiter *et al.* (20). Since this program does not correct for double recombination events, the recombination percentages were adjusted using the Kosambi function, 25 ln $[(100 + 2r)/(100 - 2r)]$, and are expressed as cM.

In Vivo Metabolism of Ara

Seedlings were grown on agar medium containing sucrose in Petri dishes standing on edge to facilitate subsequent harvesting. Equivalent wet weights (ranging from 240–450 mg in separate determinations) of mutant and wild-type seedlings were harvested 5 d postimbibition and completely immersed in 2 mL of nutrient medium containing 1.2 μCi L-[1-^{3}H] arabinose in glass vials. These were incubated under continuous illumination for 12 h at 23 to 26°C. The seedlings were then washed on a gauze sieve with a continuous stream of water (>50 mL) before being ground with a mortar and pestle in 80% (v/v) ethanol at room temperature. The mixture was then centrifuged (5 min, 10,000g) and the insoluble pellet was extracted four more times with 80% ethanol. The ethanol supernatants were pooled. The pellets and samples of the ethanol extracts were suspended in scintillation fluid and measured for radioactivity. Duplicate samples of mutant and wild-type seedlings were assayed in parallel on three separate occasions.

Arabinose Kinase and UDP-Arabinose Pyrophosphorylase Assays

Six-day-old seedlings were grown as described above and ground in an equal amount (w/v) of cold extraction buffer (0.1 M NaH_2PO_4, 5 mM $MgCl_2$, 1% v/v Triton X-100, 10 mM DTT [pH 7.4]) (7) and centrifuged (17,000g, 30 min) to remove cell debris. The supernatant was then centrifuged a second time (10,000g, 10 min). Protein levels were determined using the Bio-Rad protein assay kit and extracts were assayed immediately without further treatment. Wild-type and mutant seedlings were extracted and assayed in parallel.

Conditions used for the arabinose kinase assays were as described by Fry (7) and contained 3.2 μmol ATP, 2.0 μmol KF, 0.15×10^{-3} μmol (0.29 μCi) L-[1-^{3}H]arabinose, and 100 μL of crude extract in extraction buffer in a final volume of 200 μL and were incubated at 25°C for 60 min. The appearance of arabinose-1-phosphate over this period was linear with time and proportional to the amount of extract used and was dependent upon the addition of ATP. The formation of arabinose-1-phosphate was measured as follows using the procedure described by Chan and Hassid (3). The reaction was terminated with the addition of 1 mL 20% ethanol and passed over a disposable column of anion exchange resin AG1-X8 (Cl^- form) (Bio-Rad). After the column was washed three times with 1 mL water, the arabinose-1-phosphate was eluted with three 1 mL washes of 1 N HCl. Samples of both fractions were assayed for radioactivity by liquid scintillation counting.

UDP-arabinose pyrophosphorylase was assayed by the conversion of arabinose-1-phosphate to UDP-arabinose. L-[1-^{3}H] Arabinose-1-phosphate was synthesized using a mung bean extract as described by Fry (7) and purified by TLC using the conditions described below. Assays contained 0.8 μmol UTP, 1.0 μmol KF, 0.13×10^{-3} μmol (0.25 μCi) L-[1-^{3}H]arabinose-1-phosphate, and 50 μL of crude extract in a final volume of 100 μL and were incubated at 25°C for 60 min. The appearance of UDP-arabinose over this period was also linear with time and proportional to the amount of extract used and was dependent on the addition of UTP. Samples (10 μL) of each assay were chromatographed twice on cellulose TLC plates (Merk) using ethanol and 0.1 M ammonium acetate (pH 3.8) (5:2 v/v). Preliminary experiments using extracts of mung bean (7) were done to identify the $R_{arabinose}$ values of arabinose-1-phosphate ($R_{ara} = 0.42$) and UDP-arabinose ($R_{ara} = 0.21$). Following chromatography the cellulose matrix was scraped from 0.5 cm intervals of the TLC plate and assayed for radioactivity by scintillation counting. Activities are expressed in units where 1 unit is the conversion of 1 pmol of L-arabinose to arabinose-1-phosphate or arabinose-1-phosphate to UDP-arabinose per hour per mg of extract protein.

RESULTS

Mutant Phenotype

The growth of wild type and mutant, CC3, seeds sown directly on minimal medium containing 10 mM ara is shown in Figure 2. In the absence of ara, the mutant seedlings were indistinguishable from the wild type (not shown), whereas in the presence of both 3 and 10 mM ara the mutant seeds germinated at the same time as wild type but then failed to grow, becoming brown and necrotic within 2 d. In contrast, 30 mM ara did not inhibit the growth of the wild type. Similar results were obtained when seedlings were grown in the absence of ara and transferred after 4 d onto medium containing ara. Under these conditions, mutant seedlings became brown and necrotic after 2 d on medium containing 10 mM ara while on 3 mM ara growth was retarded. Quantitative measurements of growth under these conditions are shown in Figure 3.

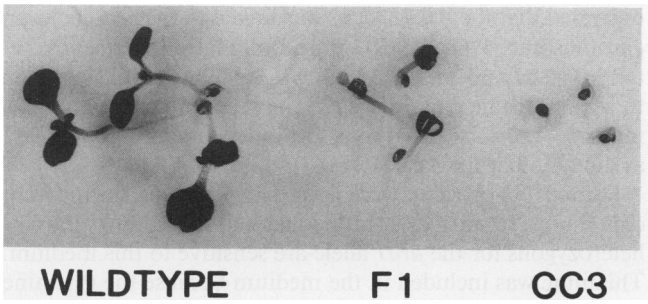

Figure 2. Arabinose-sensitive phenotype. Wild type, arabinose-sensitive (CC3), and F1 progeny are shown 5 d after sowing on nutrient medium containing 10 mM ara.

The sensitive phenotype of the mutant appeared to be specific to ara since no difference in growth between the wild type and the mutant in the presence of 10 mM D-arabinose, L-xylose (the 3-epimer of ara), D-xylose (the 4-epimer), or D-galactose was observed (data not shown). In the absence of ara, mutant plants grown in Petri dishes were indistinguishable from wild type. In potting mix the mutant grew at the same rate as the wild type but its leaves appeared to be slightly more pale. This was not reflected by a difference in Chl content (data not shown).

Genetic Analysis of the Arabinose-Sensitive Mutant

F1 progeny were generated from reciprocal crosses between the mutant and wild-type lines. When sown directly on medium containing 10 mM ara, F1 seedlings derived from both reciprocal crosses exhibited an intermediate phenotype (Fig. 2). Cotyledons were narrow and reduced in size and became progressively brown and necrotic after 4 to 5 d. On 3 mM ara, no inhibition was observed (not shown). An intermediate phenotype was also observed when germinated seedlings were transferred onto medium containing 10 mM ara; growth was progressively inhibited and patches of necrosis appeared on the cotyledons and leaves (Fig. 3). The observation that heterozygous individuals exhibited a phenotype intermediate between the homozygous mutant and the wild type indicated a semidominant relationship between the mutant and wild-type alleles.

F1 plants were grown in pots and allowed to self-fertilize, and the resulting F2 progeny were tested for growth in the presence of 10 mM ara. The results observed are shown in Table I. A 1:2:1 ratio of mutant:intermediate:wild-type phenotypes was observed ($X^2 = 0.51$, $P > 0.7$) indicating that the arabinose-sensitive mutation affects a single nuclear locus exhibiting the expected Mendelian segregation pattern. Similar results were obtained for F2 progeny of the W100 cross

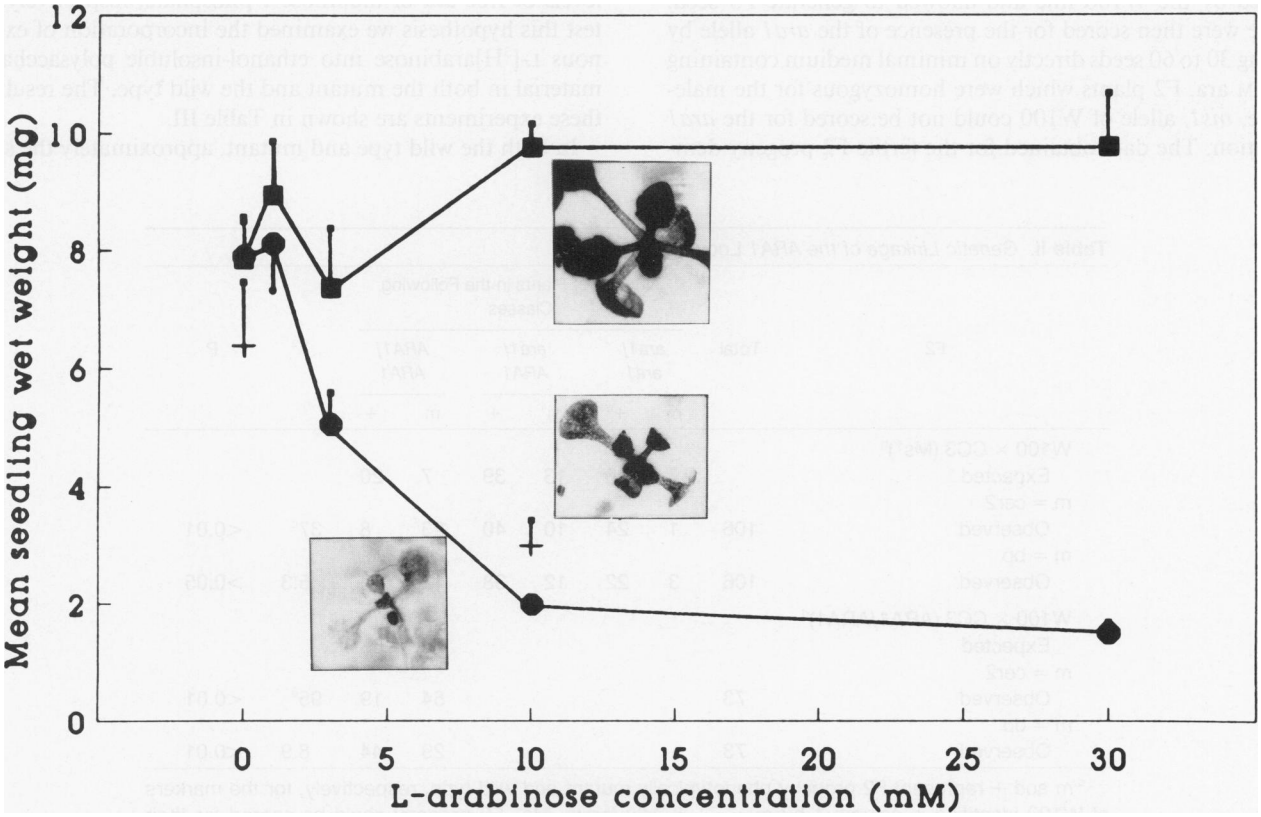

Figure 3. Growth curves of wild type, arabinose-sensitive, and F1 seedlings. Four days after sowing on medium containing sucrose, wild type (■), CC3 (●), and F1 (+) seedlings were transferred to medium containing the indicated concentrations of ara and mean seedling weights (n = 10) ± SE were determined after 7 d. The photographic insets show, from top to bottom, wild type, F1, and CC3 seedlings after 4 d on medium containing 10 mM ara.

Table I. *Genetic Segregation of the Arabinose-Sensitive Phenotype*

Strains	Total	Observed[a]			X^{2b}
		R	I	S	
WT Columbia	78	78	0	0	
CC3	106	0	2	104	
WT × CC3[c]					
F1	15	0	15	0	
CC3 × WT					
F1	33	0	33	0	
F2	142	38	67	37	0.51[d]
W100 × CC3					
F2	106	31	50	25	0.82[d]

[a] R, resistant; I, intermediate; or S, sensitive on medium containing 10 mM ara. [b] X^2 values are based on an expected ratio of 1:2:1 for R:I:S individuals. [c] In crosses, the maternal parent is represented first. [d] $P > 0.3$.

with CC3 (Table I). We have designated this locus as *ARA1* and the mutant allele as *ara1*.

To map the *ARA1* locus, the mutant was crossed with the multiply marked mapping strain, W100, which is homozygous for two recessive markers on each linkage group. F1 plants were allowed to self-fertilize, and F2 progeny were examined in two ways. First, F2 progeny were grown under nonselective conditions and scored for the visible markers carried by the W100 line and allowed to generate F3 seed. These were then scored for the presence of the *ara1* allele by plating 30 to 60 seeds directly on minimal medium containing 10 mM ara. F2 plants which were homozygous for the male-sterile, *ms1*, allele of W100 could not be scored for the *ara1* mutation. The data obtained for the fertile F2 progeny demonstrated that the *ARA1* locus was linked to the *cer2* locus on chromosome 4 (Table II). A recombination frequency between *ARA1* and *cer2* of 17.6% was calculated and converted to a map distance of 18.4 cM. The second marker on chromosome 4, *bp*, appeared to assort independently from *ARA1* as did all other markers ($P > 0.1$).

Second, F2 progeny were germinated directly on medium containing 10 mM ara. Individuals either homozygous or heterozygous for the *ara1* allele are sensitive to this medium. Thiamine was included in the medium because the thiamine auxotrophic phenotype derived from W100 interfered with the selection of individuals homozygous for *ARA1*. Plants growing vigorously after 7 d were transferred to pots and scored for the markers of W100 (with the exception of *py*, *ms1*, and *tt3*). From these results linkage to both *cer2* ($r = 14.0\%$) and *bp* ($r = 37.0\%$) was apparent (Table II). From the observed recombination percentages the *ARA1* locus was mapped to 14.4 cM from *cer2* and 47.5 cM from *bp*.

In Vivo Metabolism of Ara by the Arabinose-Sensitive Mutant

The pathway of ara metabolism in plants is shown in Figure 1. One possible explanation for the effect of exogenous ara on the mutant was that the mutation affected either the arabinose kinase or UDP-arabinose pyrophosphorylase activity resulting in the accumulation of inhibitory intracellular levels of free ara or arabinose-1-phosphate, respectively. To test this hypothesis we examined the incorporation of exogenous L-[^{3}H]arabinose into ethanol-insoluble polysaccharide material in both the mutant and the wild type. The results of these experiments are shown in Table III.

In both the wild type and mutant, approximately the same

Table II. *Genetic Linkage of the ARA1* Locus

F2	Total	Number of Plants in the Following Classes						X^2	P
		ara1/ara1		*ara1/ARA1*		*ARA1/ARA1*			
		m	+[a]	m	+	m	+		
W100 × CC3 (Ms^+)[b]									
Expected		7	20	13	39	7	20		
m = cer2									
Observed	106	1	24	10	40	23	8	37[c]	<0.01
m = bp									
Observed	106	3	22	12	38	12	19	5.3	>0.05
W100 × CC3 (ARA1/ARA1)[d]									
Expected						18	55		
m = cer2									
Observed	73					54	19	95[e]	<0.01
m = bp									
Observed	73					29	44	8.9	<0.01

[a] m and + represent F2 progeny phenotypically mutant and wild type, respectively, for the markers of W100 identified in column 1 by m =. [b] Only fertile, Ms^+ F2 progeny could be scored for their genotype with respect to the ARA1 locus in the F3 generation. [c] X^2 values are based on an expected ratio of 1:3:2:6:1:3 of the classes listed across the top of the table. [d] Homozygous wild-type F2 progeny were selected on media containing 10 mM ara. [e] X^2 values are based on an expected ratio of 1:3 of m:+ individuals.

amount of labeled ara was taken up by intact seedlings; however, in the wild type about 70% of the label taken up by the seedlings appeared in the ethanol-insoluble fraction compared with only about 10% in the mutant. The ethanol-soluble material from both the wild type and mutant was fractionated by TLC. From the wild type about 50% of the ethanol-soluble label appeared to be free arabinose while the remainder was distributed among other unidentified compounds. In contrast, greater than 95% of the ethanol-soluble label extracted from the mutant seedlings appeared to be free arabinose while less than 1% appeared in the position expected for arabinose-1-phosphate.

These results support the suggestion that there is a defect in the pathway incorporating free ara into polysaccharides in the mutant. And because free ara, and not arabinose-1-phosphate, accumulated in the mutant, that defect is likely to be in the arabinose kinase activity rather than the UDP-arabinose pyrophosphorylase.

Assays of Arabinose Kinase and UDP-Arabinose Pyrophosphorylase Activities

Crude extracts of wild type and mutant seedlings were assayed for arabinose kinase and UDP-arabinose pyrophosphorylase activities. For both enzymes activity was relatively unstable and disappeared within hours during storage on ice. A similar instability of arabinose kinase activity from mung bean has been observed (3). Attempts to increase the stability of the activities by changing the extraction procedure and by partial purification have proved unsuccessful. The assay results are shown in Table IV. In the mutant, only about 10% of the wild-type levels of arabinose kinase activity could be detected while UDP-arabinose pyrophosphorylase activity remained unchanged. In the extracts of the mutant, considerable variation in kinase activity was observed. In some extracts, a low level of activity was detected while in others activity was absent. This may reflect an increased instability of the mutant activity. In view of the semidominance of the mutant phenotype we examined the effect of mixing mutant with wild-type extract on the arabinose kinase activity. Equal volumes of extracts of the wild type and mutant were mixed and assayed. No reduction in activity beyond that expected for the two fold dilution of the wild-type extract was observed indicating there was no component of the mutant extract interfering with the wild-type activity (data not shown).

Table III. *In Vivo Incorporation of ^{3}H-ara by Mutant and Wild-Type Seedlings*

Figures are the mean of three separate determinations done in duplicate. The ranges of values are shown in parentheses.

	Total Label Taken up by Seedlings	Label Taken up Appearing in Fractions	
		Ethanol soluble	Ethanol insoluble
	%	%	
Wild type	6 (3–8)	32 (24–51)	68 (49–76)
CC3	9 (5–10)	90 (88–93)	10 (7–12)

Table IV. *Arabinose Kinase and UDP-Arabinose Pyrophosphorylase Activities*

Strain	Activity (Units ± SD)[a]	
	Arabinose kinase	UDP-arabinose pyrophosphorylase
Wild type	29 ± 9.1	13 ± 5.2
CC3	3.2 ± 3.6	18 ± 4.5

[a] Arabinose kinase and UDP-arabinose pyrophosphorylase assays were the means of three and two separate determinations, respectively.

DISCUSSION

In plants, the primary role of the monosaccharide salvage pathways is presumed to be the recycling of monosaccharides released during the turnover of polysaccharides and proteoglycans, particularly those which are components of the cell wall. Numerous glycosidases are located in plant cell walls (12, 14, 17). Such enzymes have been associated with the processes of cell wall loosening and elongation, and their activity has been observed to increase during auxin-stimulated growth (9, 16, 22). While in many cases the precise physiological role of such enzymes is not well understood, they are clearly capable of hydrolyzing cell wall polysaccharides or oligosaccharides and releasing free monosaccharides. Arabinose is an important component of cell wall polysaccharides and proteoglycans (1, 2, 6). Among the cell wall glycosidases, arabinosidases have been identified that, in some reports (21), but not others (4, 13), appear to play a role in cell wall growth processes. The function of these cell wall arabinosidases remains unknown; however, it is clear that they are in some cases able to partially hydrolyze arabinogalactans, arabinans, and pectic polysaccharides (10). Free arabinose released by these enzymes could be recycled via the arabinose salvage pathway.

The fortuitous isolation of a mutant deficient in arabinose kinase activity has provided an opportunity to investigate the role of the arabinose salvage pathway in *A. thaliana*. While the observed effect on arabinose kinase activity may not result from a mutation in the kinase gene itself but possibly from a mutation that influences the regulation of the kinase gene or even an unidentified cofactor involved in the activity of the enzyme, there is a clear deficiency in arabinose kinase activity. That this mutation has little, if any, effect on the growth of the plant may suggest the arabinose salvage pathway is not an essential component of plant metabolism. However, it is likely that there is some residual arabinose kinase activity in the mutant. First, low levels of activity could be detected in some extracts of the mutant, and second, the incorporation of exogenous arabinose into the ethanol insoluble fraction, although considerably reduced in the mutant, was not completely blocked. This residual activity may be due either to an incomplete loss of function (or expression) of a single kinase or to the presence of a second, minor isozyme and may be sufficient for the metabolism of endogenous free arabinose. Perhaps a complete loss of arabinose kinase activity may have more dramatic effects on plant growth. Also, if such a mutation were to affect plant growth primarily due to accumulation

of endogenous free arabinose, mutations in other steps may block the salvage pathway but may have no effect on the plant in either the presence or absence of arabinose.

The semidominance of the mutant phenotype may be explained either by a gene dosage effect in which insufficient gene product is expressed in the heterozygote or by the interference of the wild-type gene product by the mutant, for example, where the functional gene product is a multimer. If the residual arabinose kinase activity in the mutant is due to a partial loss of function, the latter explanation would be the more attractive since partially active, conformationally intact monomers may interfere with the function of wild-type monomers in a heteromultimer. Although extracts from the mutant did not interfere with the arabinose kinase activity in wild-type extracts, it may be that *in vivo* the mutant gene product can interfere with the wild type in some way. Because of the large numbers of seedlings required for the preparation of crude extracts, we have been unable to assay accurately the arabinose kinase activity in heterozygotes. Attempts to assay arabinose kinase activity in extracts of leaf tissue of larger plants have proved unsuccessful.

The arabinose kinase-deficient mutant was originally identified as being sensitive to exogenous arabinose. Exogenous arabinose appears to accumulate in the plant tissues due to the deficiency in arabinose kinase activity; however, the experiments described here do not demonstrate whether this accumulation is intra- or extracellular. Nor is there any indication why the accumulation of arabinose should interfere with the metabolism or growth of the plant. Further experiments are being undertaken to investigate these questions. We are currently characterizing phenotypic revertants of the arabinose-sensitive mutant to arabinose resistance. Incorporation of arabinose into the ethanol-insoluble fraction in these mutants is almost undetectable (our unpublished data), suggesting an additional block in the salvage pathway, perhaps at the level of transport into the cell, thereby preventing the inhibitory accumulation of arabinose. It is noteworthy that these "revertants", although unable to metabolize exogenous arabinose, are visibly indistinguishable from the wild type, suggesting that the salvage pathway is indeed a nonessential metabolic function. We hope these revertants will allow us to further characterize the arabinose salvage pathway and the mechanism by which exogenous arabinose is inhibitory to the arabinose kinase-deficient mutant.

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