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Galactose Concentration and Metabolism in Pericarp Tissue from Normal and Non-ripening Tomato Fruit[1]

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Abstract. **Free galactose was detected in outer pericarp tissue from fruit of 'Heinz 1350' and *nor* tomato (*Lycopersicon esculentum* Mill.). The amount of free galactose increased almost 8-fold in 'Heinz 1350' fruit during ripening. In contrast, it did not change significantly in 32- to 52-day-old (post-pollination) *nor* fruit. A 25 μl drop of water ± 100 μg galactose was applied to the locular surface of 1.5 cm diameter pericarp disks. While disks from 40- and 60-day-old *nor* tomatoes, and from mature green and pink 'Heinz 1350' tomatoes, were able to reduce the level of free galactose to levels found in control tissue within 48 hr, disks of red ripe tissue were only able to metabolize 45% of the added galactose within this time. Pink 'Heinz 1350' tomatoe disks lost galactose more slowly than disks of mature green tomatoes. The results suggest that the increased amount of free galactose in pericarp tissue of ripening tomatoes may be the result of their progressive inability to metabolize galactose as they ripen.**

Considerable research has recently been directed at cell wall metabolism in ripening fruit (11). The solubilization of wall-associated pectic polysaccharides by polygalacturonase has been documented in a number of fruit (1, 6, 9, 14). A substantial loss of galactose and arabinose residues occurs from cell walls of some ripening fruits (1, 6, 10, 17). A decrease in cell wall galactan, which does not seem to be directly related to fruit softening, occurs in both normal and *rin* (ripening inhibited) tomatoes (6, 11). This decrease seems to result from a reduced rate of *de novo* galactan synthesis (12). However, the mechanism by which the wall galactan is solubilized and the fate of the galactan are unknown. If the released galactan is solubilized into its monomeric constituents, a pool of free galactose might be detected during ripening. However, galactose does not normally occur in its free state and is often toxic to plant tissue (2, 3, 4, 5, 7, 13).

The objectives of this study were to determine if free galactose exists in pericarp tissue of normal and non-ripening tomato fruit during the later stages of fruit development, and to determine if changes occur in the ability of these tissues to metabolize galactose during their development.

Materials and Methods

Tomato (*Lycopersicon esculentum* Mill.) plants were grown in a greenhouse using standard cultural practices. 'Heinz 1350' fruit were harvested at various stages of coloration and sorted into maturity classes as previously described (6). To obtain *nor* (isogenic to 'Heinz 1350', mutant fruit (15, 16) at known stages of development, flowers were pollinated and tagged at anthesis. Fruits were harvested and sorted into maturity classes based on the number of days post-pollination.

Changes in the galactose concentration in pericarp tissue from tomatoes at different stages of development were studied by excising 5 g of outer pericarp tissue from each of 4 replicate fruit at each maturity stage (Fig. 1). Each 5 g sample was placed into 20 ml of 80% ethanol, boiled for 5 min, and homogenized (Polytron Homogenizer, Brinkmann Instruments).

Galactose metabolism by pericarp tissue from tomatoes at different stages of development was studied by aseptically excising

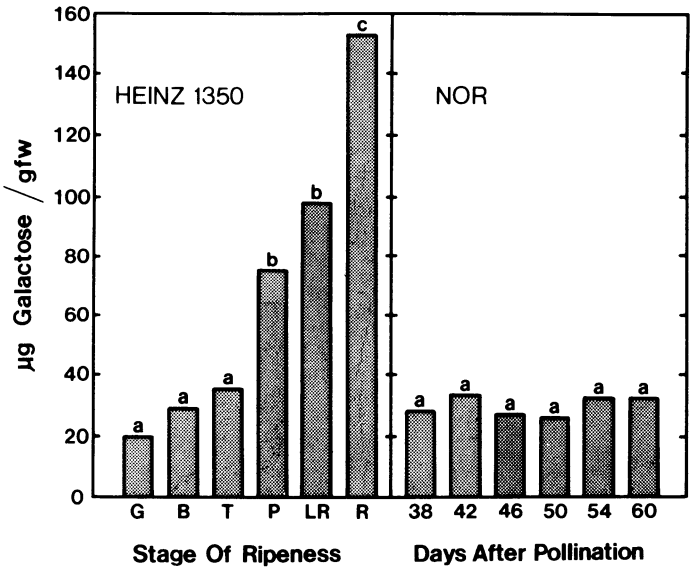

Fig. 1. The concentration of free galactose in outer pericarp tissue from 'Heinz 1350' and *nor* mutant tomato fruit during development. 'Heinz 1350' maturity classes (6) are mature green (G), breaker (B), turning (T), pink (P), light red (LR), and red ripe (R). 'Heinz 1350' fruit reached the G and R stages at 32 and 52 days post-pollination, respectively. Each value represents the mean of 4 replicate fruit. Means were separated using Duncan's multiple range test; bars with different lower case letters are significantly different at the 5% level.

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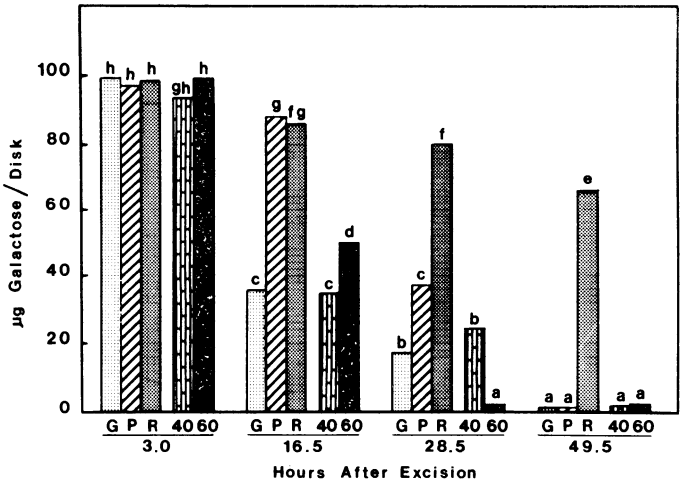

Fig. 2. Differences in the amount of galactose per pericarp disk between disks treated with 25 µl of water containing 100 µg of galactose, and disks receiving only water. Maturity classes for 'Heinz 1350' are mature green (G), pink (P), and red ripe (R). For *nor* tomato fruit the development classes are 40-day-old (40), and 60-day-old (60). Bars with different lower case letters are statistically different at the 5% level.

1.5 cm diameter disks of pericarp tissue from surface sterilized mature green, pink, and red ripe 'Heinz 1350' tomato fruit, and from 40- and 60-day-old *nor* fruit (Fig. 2). The disks were placed epidermis down in plastic petri dishes and kept at 22° ± 2°C in diffuse room light. Each disk received 0.025 ml of distilled water ± 0.1 mg galactose on its locular surface. All procedures were performed with sterile material in a tissue culture transfer hood. No sign of microbial contamination was observed on any of the disks. After 3.0, 16.5, 28.5, and 49.5 hr, 30 disks (5 maturities × 2 treatments × 3 reps) were cut into fourths and boiled for 5 min in 5 ml of 80% ethanol, homogenized, and stored at −10° until assayed.

The homogenates from all experiments were centrifuged at 27,000 × g for 15 min and the pellets discarded. The supernatant from each sample was taken to dryness *in vacuo* at 40°C and the residue taken up in 1.3 ml of 80% ethanol. To remove negatively charged pigments, extracts were treated with Dowex 1-X8 anion exchange resin (formate form) by adding 0.2 ml of resin in a 1:1 (v/v) suspension with distilled water and incubating at 25° for 20 min with constant shaking. Samples were centrifuged at 12,800 × g for 1 min (Eppendorf Model 5412, Brinkmann Instruments), and 1 ml of the supernatant was removed and taken to dryness at 40° *in vacuo*. The residue was taken up in 1 ml of 300 mM Tris-HCl (pH 8.6) containing 1 mM NAD^+ and 4 mM glutathione (reduced form).

Galactose was estimated using galactose dehydrogenase as previously described (5). Galactose dehydrogenase does not react with D-glucose, D-mannose, D-ribose, D-xylose, D-galactose-6-phosphate, galacturonic acid, or galactosamine, but will react with L-arabinose (personal communication, Sigma Chem. Co.).

Galactose concentrations are expressed as µg per g fresh weight for the experiments on changes in galactose concentration versus stage of development (Fig. 1). In the experiments on galactose metabolism, the data are expressed as differences in the amount of galactose per disk between the tissue receiving 100 µg galactose in the 25 µl of water, and the control tissue receiving only water (Fig. 2).

Results and Discussion

Paper chromatographic studies have not detected free galactose pools in tomato fruit (3). However, by using galactose dehydrogenase as a sensitive assay system (8), we have been able to measure galactose in the outer pericarp tissue from 'Heinz 1350' and *nor* tomato fruit (Fig. 1). During ripening of 'Heinz 1350' fruit, the concentration of free galactose increased substantially after the turning stage. In contrast, the concentration of galactose in *nor* fruit did not change significantly during the developmental period examined. As in *nor* fruit, the galactose concentrations in 2 ripening-inhibited (*rin*) tomato lines also did not change significantly during the latter stages of fruit development (data not shown).

It is important to point out that the source(s) of the free galactose is unknown. One possibility is that the galactose pool is a product of cell wall galactan metabolism (12). The galactan portion of the cell wall is thought to be constantly turning over (11, 12). Thus, the increase in the pool(s) of galactose in normal tomato fruit, but not in non-ripening tomato fruit, may reflect differences in their capacity to metabolize galactose. This possibility was investigated by comparing the ability of pericarp disks from normal and *nor* tomatoes at different stages of development to metabolize added galactose (Fig. 2). All disks receiving 100 µg of galactose in 25 µl of water contained approximately 100 µg more galactose than the control disks at the first sampling, 3 hr after excision. During the next 46 hr, galactose levels dropped to levels not significantly different from the control tissue in all but the red ripe disks. This drop was rapid in mature green tissue, and in tissue from 40- and 60-day-old *nor* tomatoes. The loss of galactose in disks of pink tissue was just as great as that in mature green tissue after 49.5 hr, but the loss was much more gradual during the first 28.5 hr. At the second sampling, 16.5 hr after excision, disks of pink tissue had metabolized as much galactose as red ripe tissue, (about 14%) while all other tissue had metabolized over 50% of the added galactose. Disks of red ripe tissue still contained over 65% of the added galactose after 49.5 hr. These data support the idea that differences exist between normal and ripening-inhibited tomatoes at different stages of development in the ability of tomato pericarp tissue to metabolize galactose. In view of the toxic effect of galactose on plant tissue (2, 4, 7, 13), the nature of galactose metabolism in tomato fruit merits further investigation.

Literature Cited

1. Ahmed, A. and J. M. Labavitch. 1980. Cell wall metabolism in ripening fruit. I. Cell wall changes in ripening 'Bartlett' pears. Plant Physiol. 65:1009–1013.
2. Colclasure, G. C. and J. H. Yopp. 1976. Galactose-induced ethylene evolution in mung bean hypocotyls: a possible mechanism for galactose retardation of plant growth. Physiol. Plant. 37:298–302.
3. Davies, J. N. and R. J. Kempton. 1975. Changes in the individual sugars of tomato fruit during ripening. J. Sci. Food Agr. 26:1103–1110.
4. DeKock, P. C., M. V. Cheshire, C. M. Mundie, and R. H. E. Inkson. 1979. The effect of galactose on the growth of *Lemna*. New Phytol. 82:679–685.
5. Gross, K. C., D. M. Pharr, and R. D. Locy. 1981. Growth of callus initiated from cucumber hypocotyls on galactose and galactose-containing oligosaccharides. Plant Sci. Lett. 20:333–341.
6. Gross, K. C. and S. J. Wallner. 1979. Degradation of cell wall polysaccharides during tomato fruit ripening. Plant Physiol. 63:117–120.
7. Hassid, W. Z., E. W. Putman, and V. Ginsburg. 1956. Metabolism of galactose in *Canna* leaves and wheat seedlings. Biochem. Biophys. Acta. 20:17–22.

8. Hu, A. S. L. and S. Grant. 1968. Enzymic determination of D-galactose, D-arabinose, and their homologs. Anal. Biochem. 25:221–227.
9. Knee, M. 1974. Changes in structural polysaccharides of apples ripening during storage. In: Colloques International No. 238. Factors et Regulation de la Maturation des Fruits. Centre de la Recherche Scientifique, Paris.
10. Knee, M., J. A. Sargent, and D. J. Osborne. 1977. Cell wall metabolism in developing strawberry fruits. J. Expt. Bot. 28:377–396.
11. Labavitch, J. M. 1981. Cell wall turnover in plant development. Annu. Rev. Plant Physiol. 32:385–406.
12. Lackey, G. D., K. C. Gross, and S. J. Wallner. 1980. Loss of tomato cell wall galactan may involve reduced rate of synthesis. Plant Physiol. 66:532–533.
13. Maretzki, A. and M. Thom. 1978. Characteristics of a galactose-adapted sugarcane cell line grown in suspension culture. Plant Physiol. 61:544–548.
14. Pressey, R., D. M. Hinton, and J. K. Avants. 1971. Development of polygalacturonase activity and the solubilization of pectin in peaches during ripening. J. Food Sci. 36:1070–1073.
15. Tigchelaar, E. C., W. B. McGlasson, and R. W. Buescher. 1978. Genetic regulation of tomato fruit ripening. HortScience 13:508–513.
16. Tigchelaar, E. C., M. L. Tomes, E. A. Kerr, and R. J. Barman. 1973. A new fruit ripening mutant, non-ripening (*nor*). Rpt. Tomato Genet. Coop. 23:33.
17. Wallner, S. J. and H. L. Bloom. 1977. Characteristics of tomato cell wall degradation *in vitro*. Plant Physiol. 60:207–210.

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The Influence of Light on Lily (*Lilium longiflorum* Thunb.). I. Influence of Light Intensity on Plant Development[1]

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Abstract. **Lily plants were exposed to natural daylight (ND), 50% ND (50% saran), ND plus 16 hours of incandescent (Inc) or ND plus 16 hours of high pressure sodium discharge (HID) lamp light at both University of Minnesota and Michigan State University. Light intensity had no significant horticultural effect on plant development rate that could not be readily explained by temperature. The Inc or HID light source hastened flowering by 5 to 8 days over the ND plants when given from emergence to flower. However, the rate of development from visible bud to flower was not influenced by light intensity. Plant heights were increased by all light treatments when compared to the ND plants. These increases appeared due to photoperiod for the HID treated plants, photoperiod and light quality for the Inc treated plants, and light quantity for the 50% saran-treated plants. The number of flower buds initiated was not affected by light treatment but Inc lighting increased flower bud abortion. Final plant height was highly correlated with height at visible bud; final height being about double the height at visible bud when plants were grown continuously under ND, HID, or 50% saran.**

All energy for growth of higher plants originates from radiant energy. Light strongly influences plant photomorphogenesis by altering plant height (13) and lateral branching (6, 7, 8), as well as the flowering process (5). Light has been shown to influence plant shape (19), height (11, 17), flower initiation (15, 18) and flower abortion (4, 12) in the Easter lily. After several cloudy days, lily forcers often comment that sunny days will hasten plant development. This is especially true during the period from visible bud (VB) to open flower (F).

With the introduction of energy conserving devices which reduce incoming solar radiation to greenhouse plants, we had expected decreased plant quality, e.g. increased height and reduced flower number due to reduced flower initiation or increased early and late flower abortion. Traditionally, these were attributed to reduced light. However, to our surprise, these did not occur in commercial greenhouses. Further, we were told by a lily forcer that supplemental lighting with high pressure sodium discharge (HID) lamps hastened lily flower development. We attempted to duplicate these results by lighting from VB to F (1976–77) and from floral initiation (FI) to VB, FI to F or VB to F (1977–78). We were unable to increase growth rates (unpublished data).

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