Sugar regulation of gene expression in plants

Sjef Smeekens

The molecular details of sugar sensing and sugar-mediated signal transduction pathways are unclear but recent results suggest that hexokinase functions as an important plant sugar sensor in a way that is similar to that found in yeast. The use of mutants in *Arabidopsis* defective in specific signaling steps is of particular importance because these give access to the genes encoding components in the signaling pathways. In addition, the physiological analysis of such mutants may reveal the interaction of sugar-induced signaling pathways and those induced by other stimuli such as environmental or biotic stress.

Address
Molecular Plant Physiology Group, Department of Botanical Ecology and Evolutionary Biology, University of Utrecht, Padualaan 8, 584 CH Utrecht, The Netherlands

Current Opinion in Plant Biology 1998, 1:230–234

http://biomednet.com/elecref/1369526600100230

 ISSN 1369-5266

Abbreviation
SUN sucrose uncoupled

Introduction

In plants, the control of enzymatic activity by sugars and sugar metabolites has been investigated in detail and these studies have yielded insight into the regulation of metabolic pathways. It has also been shown that in these pathways several different enzymes usually share flux control instead of being regulated by a single rate-limiting step. More recently, attention has turned to the regulation of gene expression by sugars and sugar metabolites. The expression of a large number of genes is altered by changes in sugar levels [1••]. These genes encode proteins that function in carbohydrate metabolism and, equally important, in many other metabolic pathways and developmental programs.

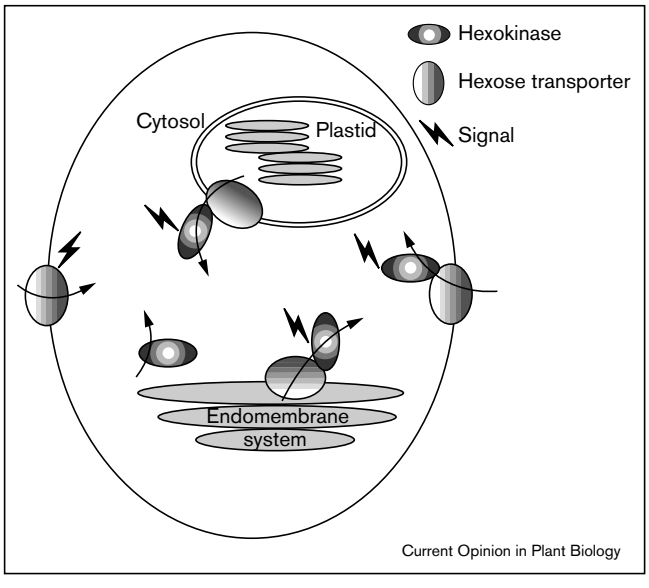
The picture which emerges is that of a sugar-responsive regulatory web in which endogenous developmental programs and external stimuli are integrated and result in a co-ordinated metabolic response. It is, therefore, of interest to understand the way in which sugars are sensed and how this sensing activates signal transduction pathways leading to altered gene expression. These sugar-sensing and signal transduction systems will interact closely with pathways responsive to other stimuli like phytohormones and light; for example, the expression of light-regulated genes like those encoding *RBCS* (encoding the small subunit of ribulose-1,5-bisphosphate carboxylase) and *CAB* (encoding chlorophyll a/b binding proteins) is repressed by elevated levels of sugar [2]. Several reviews on plant sugar sensing and sugar induced signal transduction have appeared recently [1••,3–5]. Here, I focus mainly on literature of the past two years on sugar sensing and on the analysis of the sugar-induced signal transduction cascade.

How are sugars sensed?

In principle, a large number of different sugars and intermediates in sugar metabolism can affect expression of sugar-regulated genes. To date, however, such a signaling function has been proposed for only a few sugars. Hexoses are sugars of clear importance in this respect and a signaling function has been proposed especially for glucose. Hexoses can modulate gene expression by entry into the plant cell or by entry into intermediary metabolism through the action of hexokinases. Evidence for hexose signaling associated with transport into the cell comes from the use of glucose analogs such as 6-deoxyglucose and 3-*O*-methyl glucose. These analogs can enter the cell but are not substrates for hexokinases or any other enzyme. It is most likely that their transport across the membrane initiates a signaling event resulting in altered gene expression (Figure 1). Experiments with such analogs showed that genes encoding extracellular invertase, sucrose synthase and phenylalanine ammonia lyase [6–8] can be induced in plants. These studies were performed in a *Chenopodium rubrum* cell suspension culture and similar experiments and conclusions were obtained with intact plants. For example, the patatin (B33) promoter is induced by such glucose analogs in transgenic *Arabidopsis* [9•]. Currently, direct evidence is lacking for the involvement of sugar transporters in sugar sensing in plants. Such evidence has recently become available for yeast (*Saccharomyces cerevisiae*) in which it was shown that two hexose transporter homologs, *SNF3* and *RGT2*, function as hexose sensors [10•]. Dominant mutations in these genes have been identified which, in the absence of sugar, initiate a signaling transduction cascade.

In a number of systems, it was shown that the entry of monosaccharides into intermediary metabolism through the action of hexokinase signals gene expression, not the uptake of them into the cell. The non-phosphorylatable glucose analogs 3-*O*-methyl glucose and 6-deoxyglucose described above are ineffective and only hexoses that are substrates for hexokinase are sensed. It has been proposed that the phosphorylation by hexokinase induces this enzyme to initiate a signaling cascade resulting in altered gene expression. A large body of evidence suggests such a dual function for hexokinases, especially in yeast [11]. Conclusive evidence for such a dual function requires the separation of the hexose phosphorylation and signaling functions and this has not been reported yet, nor have target proteins been identified that interact with hexokinase in the signaling cascade. One problem is that

Figure 1



Hypothetical model of hexose-sensing systems in plant cells. Sensing can occur by hexose transporters or hexose transporter homologues alone [10•], or in combination with membrane transporter-associated hexokinases, but not by soluble cytosolic hexokinases. These transporter–hexokinase complexes are proposed to be present in membranes that line the cytosolic compartment, such as the plasmalemme, the endomembrane system and the plastid envelope.

hexose flux through hexokinase affects metabolic status as monosaccharides are fed into glycolysis. This problem can be overcome by the use of sugars analogs such as 2-deoxyglucose which are substrates for hexokinase but do not enter glycolysis. Mannose is also a good hexokinase substrate which, in many plants, only enters metabolism slowly. Such substrates have been used extensively to show that further metabolism is not required for signaling. The possibility is left open that altered ATP/ADP ratios or altered cytosolic phosphate ion concentration, as a result of hexokinase activity, have a signaling function [12]. Several experiments in which the effect of phosphate ions or hexose mono- and diphosphates were tested do not support this notion [13,14,15••,16].

Evidence for a function of hexokinase in sugar signaling in plants comes from the Sheen and coworkers [4,14,17••,18]. Using a maize protoplast transient expression system, the effect of sugars was studied on the expression of a number of genes encoding photosynthetic enzymes. Hexoses that are hexokinase substrates inhibit gene expression but adding mannoheptulose, a competitive inhibitor of hexokinase, reversed this inhibition. One important advantage of this experimental system is that it allows the introduction, via electroporation, into cells of normally impermeable compounds such as hexose-phosphates. In this way it was possible to show that products of hexokinase action such as glucose-6-phosphate, fructose-6-phosphate and ATP did not have the same inhibitory effect as glucose. The notion that hexokinase is important for signaling was further tested in transgenic *Arabidopsis* using antisense and overexpression technologies. The effect of changing the endogenous hexokinase levels on sugar signaling was investigated. It was found that plants with increased hexokinase levels showed enhanced glucose (330 mM) and 2-deoxyglucose (0.8 mM) sensitivity whereas plants with lowered hexokinase levels were hyposensitive to these sugars [17••]. This altered sensitivity was also observed for regulation of expression of *CAB* and *RBCS* genes. Moreover, endogenous hexokinase signaling could be by-passed by overexpressing a heterologous, non-signaling hexokinase.

Several other studies support the involvement of hexokinase in signaling gene expression. The glyoxylate cycle genes *ICL* (encoding isocitrate lyase) and *MS* (encoding malate synthase) are essential in the conversion of lipid into sugars. These genes are repressed by sugars at the transcriptional level. In a cucumber cell line this repression could be mimicked only by hexoses such as 2-deoxyglucose and mannose that are both substrates for hexokinase whereas 3-*O*-methylglucose has no effect [13].

In another study, it was found that germination of *Arabidopsis* seedlings on mannose or 2-deoxyglucose is severely inhibited. Titrating the hexokinase inhibitor mannoheptulose in the growth medium could relieve this inhibition. In this study too, 3-*O*-methylglucose and 6-deoxyglucose do not inhibit germination, even at high concentrations [5], showing that hexose uptake as such is not involved in this inhibition. In celery, the activity of the mannitol-catabolizing enzyme mannitol dehydrogenase (MTD) is repressed by sugars [19]. The addition of glucose to cultured celery cells represses MTD enzymatic activity and steady state mRNA levels whereas 3-*O*-methylglucose was ineffective. Inhibition of hexokinase activity by titrating mannoheptulose in the culture medium relieved glucose repression of MTD activity. All the above-mentioned studies are in agreement with the notion that hexokinase is of major importance for hexose sensing in plants. As argued above, however, proof of this hypothesis has to come from unraveling the molecular details of the signaling and enzymatic functions of hexokinase.

Others have questioned such a dual function of plant hexokinase [20••]. Expression of yeast invertase in either the cytosol, apoplast or vacuole of transgenic tobacco plants leads to excessive sucrose hydrolysis in these three compartments resulting in elevated levels of glucose and fructose which are stored in the vacuole [21]. Interestingly, the excess glucose and fructose were only sensed in plants expressing invertase in the apoplast or vacuole, resulting in altered gene expression and bleaching in these plants. Remarkably, plants that express yeast invertase

in the cytosol do not show these effects. If hexokinase is involved in hexose sensing, the glucose and fructose generated in the cytosol should result in sugar signaling because hexokinase is a cytosolic enzyme. This was not observed and the authors proposed that hexoses are sensed only when produced in the endomembrane system (Golgi–endoplasmic reticulum). The apoplastic and vacuolar targeted invertases both traverse the endomembrane system and are enzymatically active in this compartment. Moreover, sucrose is present in the endomembrane system as plants expressing a bacterial fructosyl transferase in this compartment accumulate fructans to high levels [22]. The monosaccharides generated by this fructosyl transferase in the endomembrane system are being sensed, as indicated by the severe chlorotic phenotype of these plants. Interestingly, the expression of this enzyme in chloroplasts also leads to high level fructan accumulation in plastids and to the same chlorotic phenotype and elevated hexose levels (S Turk, S Smeekens, unpublished data). This observation shows that there must be an extensive sucrose flux through plastids. Moreover, the hexoses generated in plastids by the action of fructosyl transferase are somehow sensed.

The studies discussed so far are compatible with the idea that the entry of sugar into the cytosol is a major site of sensing (Figure 1). In this model, hexokinase sensing may function only in association with hexose transport into the cytosol [4]. Hexoses generated in either the endomembrane system or in plastids are transported into the cytosol with concomitant phosphorylation by signaling hexokinases. Possibly, only such transport-associated hexokinases are capable of signaling and thus hexoses produced in the cytosol are not sensed. In this respect it is interesting to note that plasmodesmata are important sugar entry (and export) sites between cells [1••,23]. Modifications that affect *in vivo* plasmodesmata function greatly affect this transport capacity [24–26]. It is quite likely that sugars are sensed here too, but currently no information is available on the details of plasmodesmatal sugar transport. As discussed, sugars are present and sensed in the endomembrane system [20••,22] and such endomembranes of neighboring cells are connected through plasmodesmata.

Many questions remain unanswered but it is clear that hexose sensing is an important mechanism through which plants are able to respond to changes in sugar status. It is to be expected that plants have several other ways in which information on sugar status can be sensed and used. The knowledge of yeast-sensing systems may be a good guide in this respect and it is likely that, in plants, specialized sugar transporters are also employed in sensing. Recent results indicate the existence in *Arabidopsis* of a sucrose transporter or sensor that responds to sucrose concentration differences and, when activated, results in translational control of a transcription factor gene [5].

Unraveling the signal transduction cascade

Sugar sensors somehow initiate a signal transduction cascade leading to altered gene expression. The molecular nature of this cascade is unclear, but several groups are addressing this question. Plant homolog of yeast signaling components have been identified, most notably the SNF1 protein kinase [27,28]. Moreover, in several systems the use of chemicals that affect the function of known signal transduction steps has pointed to possible intermediates in sugar signaling. In this way, the involvement of protein kinases and protein phosphatases as important components in signaling have been implied [8,29–31]. Moreover, the use of chemicals that inhibit calmodulin or Ca^{2+} ion channels point to the involvement of Ca^{2+} ions in signaling [32]. In this respect, the identification of a sugar-induced plasma membrane-associated calcium-dependent protein kinase in tobacco [31] is interesting. One proposed function of such a kinase is to control the activity of sugar transporters located in the membrane. Tasks that lie ahead include the confirmation that these observations are specific for the sugar signaling cascade and the placement of the individual steps in this cascade.

The isolation and analysis of mutants defective in sugar sensing will be important in unraveling this sugar-induced signaling cascade and several groups have identified such mutants in *Arabidopsis* using different approaches. A number of *sucrose-uncoupled* (*sun*) mutants were identified in which sucrose is unable to repress the developmentally controlled transient induction of photosynthesis genes (*RBCS*, *CAB*, *PETE* [encoding plastocyanin]) upon germination [33,34••]. Interestingly, one of the mutants (*sun6*) that was analyzed in detail showed reduced feedback inhibition of photosynthesis in the mature plant by the sugar analog 2-deoxyglucose [15••]. 2-deoxyglucose is a hexokinase substrate and in accordance with this observation it was shown that *sun6* is also insensitive to elevated (6%) glucose levels. In another screen, *reduced sucrose response* (*rsr*) mutants were identified as being defective in the sugar-induced expression of the class I patatin (B33) promoter [9•]. Similar *low-level beta-amylase* (*lba*) mutants were identified showing reduced sugar-induced beta amylase gene expression [35••]. Remarkably, the *Arabidopsis Landsberg erecta* ecotype is a naturally occurring *lba* mutant and one can speculate about the ecological significance of this finding in terms of growth advantages or efficient resource utilization of this ecotype in its natural habitat.

The reciprocal type of mutant in which sugar-induced genes are hyper-responsive to sugars have also been identified [36]. In such *high-level beta-amylase* (*hba*) mutants relatively low levels of sugars strongly stimulate beta-amylase gene expression. A more complete listing of potential sugar sensing mutants including unpublished information has been presented [5]. Physiological and molecular analysis of such mutants, including gene identification, will greatly advance our knowledge of sugar-sensing and

signaling in plants. Moreover, such mutants are invaluable for unraveling interactions between sugar-induced and other signaling pathways. Interesting in this respect, is the observation that two of the *SUN* genes, *SUN6* and *SUN7*, modulate phytochrome A (PHYA) responsiveness in a sugar-dependent way and in this way link light- and sugar-responsive signal transduction pathways [34••]. In fact, the *SUN6* and *SUN7* genes interact with two different branches of the PHYA signaling pathway.

Similar examples of gene products mediating cross talk between sugar-signalling pathways and pathways such as phytohormone or stress signaling can be predicted to be uncovered in the mutant collections. Several examples of interactions between sugar and other signaling pathways have appeared over the years [1••]. One recent observation is the sugar-mediated repression of a gene involved in brassinolide biosynthesis [37]. Moreover, different stimuli can result in similar expression patterns. For example, the sugar modulated expression of three *C. rubrum* genes can be mimicked through different signal transduction pathways by stress and cytokinin signals [8,38].

Several studies have indicated a close interaction between sugar signaling and developmental processes. In a recent study on *Vicia faba* developing seeds it was suggested that hexoses signal meristematic activity (cell division) in the developing cotyledons whereas sucrose induces a switch towards the non-proliferative storage phase of seed development [39]. Upon fertilization, a seed coat associated invertase activity generates hexoses from incoming sucrose. The duration of expression of this invertase activity is a determinant for the number of cells in the embryo and, because the number of cells determines storage capacity, seed size. The importance of extracellular invertase in determining seed size was also shown in the maize where the *miniature* mutant is defective in this extracellular invertase gene [40].

At elevated CO_2 concentrations, an increase in apical meristem cell number and size, and a more rapid progression through the cell cycle has been observed in different plant species [41,42]. Growth in elevated CO_2 concentrations will lead to increased sugar concentrations in the plant and sugar-sensing systems of meristem cells may well activate increased cell division rates.

Conclusions

The molecular and genetic analysis of sugar sensing systems in plants will lead to the identification of a rapidly growing number of genes whose products are involved in the sugar-induced signal transduction pathway. Moreover, physiological and genetic characterisation of mutants will reveal epistatic relations and provide entry points for detailed biochemical investigations. In particular, techniques such as the yeast two-hybrid system have been most successfully used in studying protein–protein interactions in sugar-induced signaling in yeast [43]. Using exciting new technologies such as fluorescence lifetime imaging microscopy, molecular interactions can be visualized in living cells. These methods can be complemented with microinjection techniques where signaling intermediates can be tested directly by injection into wild-type and/or mutant cells [44,45]. Sugar-induced signaling pathways will interact with many other signaling pathways to form regulatory webs that allow the integrated response to developmental programs and changing environmental conditions. Such pathways probably are cell autonomous and must respond to signals that co-ordinate responses at the whole plant level.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

1. •• Koch KE: **Carbohydrate-modulated gene expression in plants.** *Annu Rev Plant Physiol Plant Mol Biol* 1996, **47**:509-540.
An excellent and extensive overview of the literature on sugar sensing.

2. Krapp A, Hofmann B, Schafer C, Stitt M: **Regulation of expression of *rbcS* and other photosynthetic genes by carbohydrates: a mechanism for the 'sink regulation' of photosynthesis?** *Plant J* 1993, **3**:817-828.

3. Graham IA: **Carbohydrate control of gene expression in higher plants.** *Res Microbiol* 1996, **147**:572-580.

4. Jang J-C, Sheen J: **Sugar sensing in higher plants.** *Trends Plant Sci* 1997, **2**:208-214.

5. Smeekens S, Rook F: **Sugar sensing and sugar-mediated signal transduction in plants.** *Plant Physiol* 1997, **115**:7-13.

6. Godt DE, Riegel A, Roitsch T: **Regulation of sucrose synthase expression in *Chenopodium rubrum*: characterization of sugar induced expression in photoautotrophic suspension cultures and sink tissue specific expression in plants.** *Plant Physiol* 1995, **146**:231-238.

7. Roitsch T, Bittner M, Godt DE: **Induction of apoplastic invertase of *Chenopodium rubrum* by D-glucose and a glucose analog and tissue-specific expression suggest a role in sink-source regulation.** *Plant Physiol* 1995, **108**:285-294.

8. Ehness R, Ecker M, Godt DE, Roitsch TH: **Glucose and stress independently regulate source and sink metabolism and defence mechanisms via signal transduction pathways involving protein phosphorylation.** *Plant Cell* 1997, **9**:1825-1841.

9. • Martin T, Hellmann H, Schmidt R, Willmitzer L, Frommer WB: **Identification of mutants in metabolically regulated gene expression.** *Plant J* 1997, **11**:53-62.
The authors show that the patatin promoter is induced by hexose uptake. Further metabolism is not required. The patatin promoter fused to a reporter gene is used to identify mutants that have a reduced sugar responsiveness. In these mutants sucrose is not an effective inducer of the patatin promoter.

10. • Özcan S, Dover J, Rosenwald AG, Wolfl S, Johnston M: **Two glucose transporters in *Saccharomyces cerevisiae* are glucose sensors that generate a signal for induction of gene expression.** *Proc Natl Acad Sci USA* 1996, **93**:1-5.
Elegant demonstration of the signaling function of two glucose transporters via the identification and analysis of sugar-independent, dominant signaling mutations.

11. Trumbly RJ: **Glucose repression in the yeast *Saccharomyces cerevisiae*.** *Mol Microbiol* 1992, **6**:15-21.

12. Sadka A, Dewald DB, May GD, Park WD, Mullet JE: **Phosphate modulates transcription of soybean *VspB* and other sugar inducible genes.** *Plant Cell* 1994, **6**:737-749.

13. Graham IA, Denby KJ, Leaver CJ: **Carbon catabolite repression regulates glyoxylate cycle gene-expression in cucumber.** *Plant Cell* 1994, **6**:761-772.

14. Jang JC, Sheen J: **Sugar sensing in higher plants.** *Plant Cell* 1994, **6**:1665-1679.

15. •• Van Oosten JJ, Gerbaud A, Huijser C, Dijkwel PP, Chua N-H, Smeekens SCM: **An *Arabidopsis* mutant showing reduced feedback inhibition of photosynthesis.** *Plant J* 1997, **12**:1011-1020.

The physiological analysis of the *sucrose uncoupled 6* mutant is described. Interestingly, the glucose analog 2-deoxyglucose does not suppress photosynthesis in the mutant at the mature rosette stage, whereas wild-type *Arabidopsis* is very sensitive to this analog. This mutant was also shown to be insensitive to high glucose levels.

16. Klein D, Stitt M: **Effects of 2-deoxyglucose on the expression of *RBCS* and the metabolism of *Chenopodium rubrum* cell suspension cultures.** *Planta* 1998, in press.

17. •• Jang J-C, Leon P, Zhou L, Sheen J: **Hexokinase as a sugar sensor in higher plants.** *Plant Cell* 1997, **9**:5-19.

In this paper, the authors present evidence for a signaling function of hexokinase by modulating the *in planta* level of hexokinase expression. It was found that *Arabidopsis* plants which overexpress hexokinase show increased glucose sensitivity of the transgenic plants, whereas hexokinase antisense suppression renders plants less glucose sensitive.

18. Taylor CB: **Sweet sensations.** *Plant Cell* 1997, **9**:1-4.

19. Prata RTN, Williamson JD, Conkling MA, Pharr DM: **Sugar repression of mannitol dehydrogenase activity in celery cells.** *Plant Physiol* 1997, **114**:307-314.

20. •• Herbers K, Meuwly P, Frommer W, Metraux J-P, Sonnewald U: **Systemic acquired resistance mediated by the ectopic expression of invertase: possible hexose sensing in the secretory pathway.** *Plant Cell* 1996, **8**:793-803.

On the basis of the expression of yeast invertase in different cellular locations, the authors conclude that hexoses generated in the cytosol are not sensed. This leads them to question the postulated dual role of hexokinase.

21. Heineke D, Wildenberger K, Sonnewald U, Willmitzer L, Heldt HW: **Accumulation of hexoses in leaf vacuoles: studies with transgenic tobacco plants expressing yeast-derived invertase in the cytosol, vacuole or apoplasm.** *Planta* 1994, **194**:29-33.

22. Turk SCHJ, de Roos K, Scotti PA, Van Dun K, Weisbeek PJ, Smeekens SCM: **The vacuolar sorting domain of sporamin transports GUS, but not levansucrase, to the plant vacuole.** *New Phytol* 1996, **136**:29-38.

23. Stitt M: **Plasmodesmata play an essential role in sucrose export from leaves: a step toward an integration of metabolic biochemistry and cell biology.** *Plant Cell* 1996, **8**:565-571.

24. Lucas WJ, Balachandran S, Park J, Wolf S: **Plasmodesmal companion cell-mesophyll communication in the control over carbon metabolism and phloem transport: insights gained from viral movement proteins.** *J Exp Bot* 1996, **47**:1119-1128.

25. Herbers K, Tacke E, Hazirezaei M, Krause K-P, Melzer M, Rohde W, Sonnewald U: **Expression of a luteoviral movement protein in transgenic plants leads to carbohydrate accumulation and reduced photosynthetic capacity in source leaves.** *Plant J* 1997, **12**:1045-1056.

26. Russin WA, Evert RF, Vanderveer PJ, Sharkey THD, Briggs SP: **Modification of a specific class of plasmodesmata and loss of sucrose export ability in the *sucrose export defective 1* maize mutant.** *Plant Cell* 1996, **8**:645-658.

27. Halford NG, Vicente-Carbajosa J, Sabelli PA, Shewry PR, Hannappel U, Kreis M: **Molecular analyses of a barley multigene family homologous to the yeast protein kinase gene *SNF1*.** *Plant J* 1992, **2**:791-797.

28. Muranaka T, Banno H, Machida Y: **Characterization of tobacco protein kinase NPK5, a homolog of *Saccharomyces cerevisiae* SNF1 that constitutively activates expression of the glucose-repressible *SUC2* gene for a secreted invertase of *S. cerevisiae*.** *Mol Cell Biol* 1994, **14**:2958-2965.

29. Lue M-Y, Lee H: **Protein phosphatase inhibitors enhance the expression of an alpha-amylase gene, *alpha Amy3*, in cultured rice cells.** *Biochem Biophys Res Commun* 1994, **205**:807-816.

30. Takeda S, Mano S, Ohto M, Nakamura K: **Inhibitors of protein phosphatases I and 2A block the sugar-inducible gene expression in plants.** *Plant Physiol* 1994, **106**:567-574.

31. Ohto M, Nakamura K: **Sugar-induced increase of calcium-dependent protein kinases associated with the plasma membrane in leaf tissues of tobacco.** *Plant Physiol* 1995, **109**:973-981.

32. Ohto M, Hayashi K, Isobe M, Nakamura K: **Involvement of Ca^{2+} signalling in the sugar-inducible expression of genes coding for sporamin and β-amylase of sweet potato.** *Plant J* 1995, **7**:297-307.

33. Dijkwel PP, Kock P, Bezemer R, Weisbeek P, Smeekens S: **Sucrose represses the developmentally controlled transient activation of the plastocyanin gene in *Arabidopsis thaliana* seedlings.** *Plant Physiol* 1996, **110**:455-463.

34. •• Dijkwel PP, Huijser C, Weisbeek PJ, Chua N-H, Smeekens SCM: **Sucrose control of phytochrome A signalling in Arabidopsis.** *Plant Cell* 1997, **9**:583-595.

The isolation of a number of *sucrose uncoupled Arabidopsis* mutants is described. The analysis of some of the mutants provide evidence for cross talk between light- and sugar-induced signaling pathways

35. •• Mita S, Murano N, Akaike M, Nakamura K: **Mutants of *Arabidopsis thaliana* with pleiotropic effects on the expression of the gene for β-amylase and on the accumulation of anthocyanin that are inducible by sugars.** *Plant J* 1997, **11**:841-851.

The authors describe *Arabidopsis* mutants with a reduced sugar responsiveness. Interestingly the *Arabidopsis thaliana* Landsberg *erecta* ecotype is a natural reduced responsiveness mutant and this property was mapped to a single locus.

36. Mita S, Hirano H, Nakamura K: **Negative regulation in the expression of a sugar-inducible gene in *Arabidopsis thaliana*; a recessive mutation causing enhanced expression of a gene for β-amylase.** *Plant Physiol* 1997, **114**:572-582.

37. Szekeres M, Németh K, Koncz-Kalman Z, Mathur J, Kauschmann A, Altmann TH, Rédei GP, Nagy F, Schell J, Koncz C: **Brassinosteroids rescue the deficiency of CYP90, a cytochrome P450, controlling cell elongation and de-etiolation in Arabidopsis.** *Cell* 1996, **85**:171-182.

38. Ehness R, Roitsch TH: **Co-ordinated induction of mRNAs for extracellular invertase and a glucose transporter in *Chenopodium rubrum* by cytokinins.** *Plant J* 1997, **11**:539-548.

39. Weber H, Borisjuk L, Wobus U: **Controlling seed development and seed size in Vicia faba: a role for seed coat-associated invertases and carbohydrate state.** *Plant J* 1996, **10**:823-834.

40. Cheng W, Tallercio EW, Chourey PS: **The *miniature1* seed locus of maize encodes a cell wall invertase required for normal development of endosperm and maternal cells in the pedicel.** *Plant Cell* 1996, **8**:971-983.

41. Kinsman E, Lewis C, Davies M, Young J, Francis D, Vilhar B, Ougham H: **Elevated CO_2 stimulates cells to divide in grass meristems: a differential effect in two natural populations of *Dactylis glomerata*.** *Plant Cell Environ* 1997, **20**:1309-1316.

42. Jitla D, Rogers G, Seneweera S, Basra A, Oldfield R, Conroy J: **Accelerated early growth of rice at elevated CO_2.** *Plant Physiol* 1997, **115**:15-22.

43. Jiang R, Carlson M: **Glucose regulates protein interactions within the yeast SNF1 protein kinase complex.** *Genes Dev* 1996, **10**:3105-3115.

44. Bowler C, Neuhaus G, Yamagata H, Chua N-H: **Cyclic GMP and calcium mediate phytochrome phototransduction.** *Cell* 1994, **77**:73-81.

45. Wu Y, Kuzma J, Marechal E, Graeff R, Lee HC, Foster R, Chua N-H: **Abscisic acid signaling through cyclic ADP-ribose in plants.** *Science* 1997, **278**:2126-2130.