Photosynthesis, sugars and the regulation of gene expression

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Abstract
Sugar-mediated regulation of gene expression is a mechanism controlling the expression of many different plant genes. In this review, a compilation of the genes encoding photosynthetic proteins, subject to this mode of regulation, is presented. Several groups have devised different screening strategies to obtain Arabidopsis mutants in sugar sensing and signalling. An overview of these strategies has been included. Sugar-mediated regulation of gene expression is thought to require the hexokinase (HXK) protein. It has previously been shown that one such sugar, mannose, is capable of blocking germination in Arabidopsis. This inhibition is also mediated by HXK and occurs in the low millimolar concentration range. Here, the use of germination on mannose as an effective screening strategy for putative sugar sensing and signalling mutants is reported. T-DNA- and EMS-mutagenized collections were used to isolate 31 mannose-insensitive germination (mig) mutants. With the use of these mutants, a comparison between this screen and other existing sugar-sensing screens is presented.

Key words: Photosynthesis, sugars, mannose, Arabidopsis, mutants.

Introduction
In the 1770s Joseph Priestley performed experiments showing that plants release a type of air that allows combustion and, since then, photosynthesis has been the subject of a broad and intensive research effort. As a result, the pathways of photosynthesis are well established and the enzymatic properties of individual enzymes are well understood. The underlying mode of regulation, however, remains an area of relative ignorance. How are the synthesis and assembly of the multimeric protein complexes involved in photosynthesis regulated? And how is net photosynthetic activity regulated as the plant encounters a wide range of environmental conditions each day and throughout its life cycle? These questions are of great interest since this knowledge would eventually allow control and manipulation of the entire photosynthetic process. Progress in this area has been impressive and this review shows how molecular genetic techniques are contributing to a new understanding of these issues.

Although photosynthesis may be partly regulated at the enzyme or translation level (‘fine control’), bulk regulation (‘coarse control’) seems to take place at the level of transcript accumulation. In most cases patterns of protein accumulation reflect those of mRNA (Monroy and Schwartzbach, 1983; Nelson and Langdale, 1989; Kuhlemeier, 1992; Van Oosten and Besford, 1995). Light-regulation of photosynthesis and expression of genes encoding proteins of photosynthesis are well-documented processes and several Arabidopsis mutant screens have begun to resolve the mode of regulation (Staub and Deng, 1996; Wei and Deng, 1996). Light, however, cannot account for the regulation that must take place as the plant encounters variations in other environmental and developmental factors and processes throughout its life cycle. Lately, a fundamental regulatory mechanism has been discovered which is capable of detecting the end-product of photosynthesis, sugars, and enhances or represses the photosynthetic apparatus by regulating transcription of the genes involved. This carbon metabolite-mediated regulatory mechanism is not only capable of regulating photosynthesis, but also of providing the necessary integration with plant metabolism as a whole.

Global atmospheric CO₂ has risen from 27 Pa before the industrial revolution to 36 Pa 200 years later (Griffin and Seemann, 1996) due to human activity and is expected
to double during the 21st century (McElroy, 1994). This dramatic increase is occurring much faster than the natural genetic adaptation of plants and is expected to alter the functional balance of photosynthesis reactions. In this context the study and understanding of photosynthesis function and regulation gain increasing importance.

**Carbon metabolite-mediated repression of genes involved in photosynthesis**

Carbon metabolite concentrations may affect the expression of genes encoding photosynthetic components in two different ways. The depletion of sugars leads to activation of gene expression and to an increase in photosynthetic capacity. When output exceeds the plant’s capacity to metabolize or export sugars, the subsequent increase in sugar concentrations in the leaf triggers repression of genes for photosynthetic components and ultimately photosynthesis itself (Stitt, 1991; Krapp et al., 1993; Van Oosten and Besford, 1994, 1995). These two distinct effects, however, are probably the result of a single regulatory mechanism, as enhanced expression following sugar depletion seems to be largely the result of de-repression of sugar controls on transcription (Koch, 1996). mRNA stability and translation may, in some cases, also contribute to carbon metabolite modulation in vivo (Sheu et al., 1994; Rook et al., 1998). The putative advantage in sugar-mediated regulation of genes encoding photosynthetic components is that, in times of sugar deficiency, reserves will be mobilized to increase photosynthesis and sugar output, whereas valuable resources (e.g. reduced N) need not be committed to the processes if carbohydrate supplies are already sufficient.

The repression of both nuclear and plastidic genes for photosynthetic components (Table 1) by elevated sugar concentrations has been demonstrated in several different ways. Many groups have shown that the addition of sugars to the external medium down-regulates the expression of such genes (Krapp et al., 1993; Dijkwel et al., 1996). Leaf discs have been cut and floated on a solution containing 175 mM sucrose or glucose and repression of the chloroplastic ribulose-1,5-bisphosphate carboxylase/oxygenase (Rubisco) gene was observed (Van Oosten and Besford, 1994). Similar experiments, using detached, single leaves with the cut petioles placed in a sugar-containing solution, examined the expression of the triose phosphate translocator gene, with similar results (Knight and Gray, 1994). A maize protoplast suspension was used and the expression of seven different promoters of genes encoding photosynthetic components was measured when various sugars were supplied to the external media (Jang and Sheen, 1994). All of the metabolizable sugars tested were able to repress expression. Sugar uptake was not involved, as the same effect was observed with sugars directly electroporated into the protoplasts. The low concentrations of sugars used for electroporation demonstrated the physiological relevance of the sugar-mediated regulation of expression of genes coding for photosynthesis proteins. This was later confirmed in Chenopodium rubrum cell-suspension cultures where 1 mM glucose sufficed to inhibit expression of RBCS (Klein and Stitt, 1998).

A second strategy to observe down-regulation of genes coding for photosynthesis proteins is cold-girdling. Temperature-controlled aluminium blocks are placed around petioles of photosynthetically active source leaves of whole plants. The lowering of the temperature in the petiole inhibits the export of sugars and other metabolites, resulting in a rise in sugar concentrations in the leaf to a point where repression of gene expression occurs (Krapp et al., 1993). These strategies, while useful in the study of sugar-regulated gene expression, should be viewed with caution as they may, in some instances, lead to high, physiologically non-relevant, internal sugar concentrations.

A third means of triggering repression of photosynthesis through repression of the genes involved is by placing plants in a carbon dioxide-rich atmosphere. The increase in pCO₂ leads initially to increased photosynthesis rates (reviewed in Stitt, 1991) and to increased intracellular sugar concentrations (Arp, 1991; Wullschleger et al., 1992). However, over a period of days, photosynthetic rates decline (Curtis et al., 1995; Van Oosten and Besford, 1995) due, probably, to an accumulation of sugars in the leaves (Von Schaewen et al., 1990; Krapp et al., 1993; Van Oosten and Besford, 1994, 1995; Webber et al., 1994; Drake et al., 1997), or to the consequent increase in flux through hexokinase (Jang and Sheen, 1994; Koch, 1996). Remarkably, this is apparently not a cell-autonomous response, but is controlled at the whole plant level (Sims et al., 1998).

Yet another approach to altering internal sugar concentrations has been taken by producing transgenic tobacco plants expressing invertase derived from yeast in the cytosol, vacuole or apoplast (Von Schaewen et al., 1990; Sonnewald et al., 1991). Although direct measurements of gene expression were not carried out in these plants, increased carbohydrate accumulation and inhibition of photosynthesis was observed in all three cases. The increase in carbohydrate concentrations was proposed as the cause for the inhibition of photosynthesis (Heineke et al., 1994).

Apart from photosynthesis, carbon metabolites have been shown to control the expression of genes involved in processes as diverse as pathogen attack, environmental stress, storage protein accumulation, and starch, lipid and nitrogen metabolism (Hattori et al., 1990; Nakamura et al., 1991; Cheng et al., 1992; Karrer and Rodriguez, 1992; Koch et al., 1992; McLaughlin and Smith, 1994; Chevalier et al., 1996; Herbers et al., 1996; Prata et al., 1996).
In general, it can be said that when sugar concentrations in the plant increase, there is repression of genes involved in mobilization of stored reserves and photosynthesis. At the same time, genes required for metabolism and storage of carbon metabolites for future use are induced. There are many genes whose expression is regulated by this mechanism (Koch, 1996; Jang and Sheen, 1997; Long et al., 1997). Several different groups have devised a number of such screens. Examples thereof are the gin (glucose-insensitive; Zhou et al., 1996), sis (sucrose-insensitive; Pego et al., 1998), and cai (carbohydrate-insensitive; Boxall et al., 1996) screens (Table 2). Mutants generated in these screens are putative sugar-insensitive mutants. In order to obtain putative sugar-hypersensitive mutants, a similar strategy was used except that seeds were screened for their inability to germinate or develop in the presence of otherwise non-inhibiting sugar concentrations. The gss (glucose-supersensitive; Pego et al., 1998) and sxs (sucrose-supersensitive; Pego et al., 1998) screens, as far as is known, the only such screens belonging to this class.

Another approach used to obtain putative sugar-sensing mutants was to fuse a promoter of a sugar-regulated gene to a reporter gene. The plastocyanin gene promoter has been fused to the firefly luciferase gene and transformed into Arabidopsis (Dijkwel et al., 1997). Plastocyanin gene expression is repressed by 88 mM (3%) sucrose and, when present in the medium, luciferase is

### Table 1. Sugar-repressible genes encoding photosynthesis components

<table>
<thead>
<tr>
<th>Gene</th>
<th>Organism</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>ATP-δ</td>
<td>Chenopodium</td>
<td>Krapp et al., 1993</td>
</tr>
<tr>
<td>CA</td>
<td>Arabidopsis</td>
<td>Raines et al., 1992</td>
</tr>
<tr>
<td>CAB</td>
<td>Arabidopsis</td>
<td>Dijkwel et al., 1996</td>
</tr>
<tr>
<td></td>
<td>Chenopodium</td>
<td>Krapp et al., 1993</td>
</tr>
<tr>
<td></td>
<td>Maize</td>
<td>Sheen, 1990</td>
</tr>
<tr>
<td></td>
<td>Pea</td>
<td>Knight and Gray, 1994</td>
</tr>
<tr>
<td></td>
<td>Rape</td>
<td>Harker et al., 1993</td>
</tr>
<tr>
<td></td>
<td>Tobacco</td>
<td>Cricui et al., 1992</td>
</tr>
<tr>
<td>PSBA</td>
<td>Euglena</td>
<td>Reinbothe et al., 1991</td>
</tr>
<tr>
<td></td>
<td>Tomato*</td>
<td>Van Oosten and Besford, 1995</td>
</tr>
<tr>
<td>MEI</td>
<td>C₄ malic enzyme</td>
<td>Maize, Sheen, 1990</td>
</tr>
<tr>
<td>PC</td>
<td>Arabidopsis</td>
<td>Dijkwel et al., 1996</td>
</tr>
<tr>
<td>PEPC₄</td>
<td>Maize</td>
<td>Sheen, 1990</td>
</tr>
<tr>
<td>PT</td>
<td>Tobacco</td>
<td>Knight and Gray, 1994</td>
</tr>
<tr>
<td>PPDK</td>
<td>Maize</td>
<td>Sheen, 1990</td>
</tr>
<tr>
<td>RBCL</td>
<td>Euglena</td>
<td>Reinbothe et al., 1991</td>
</tr>
<tr>
<td>RBCS</td>
<td>Arabidopsis</td>
<td>Cheng et al., 1992</td>
</tr>
<tr>
<td></td>
<td>Chenopodium</td>
<td>Krapp et al., 1993</td>
</tr>
<tr>
<td></td>
<td>Maize</td>
<td>Sheen, 1990</td>
</tr>
<tr>
<td></td>
<td>Potato</td>
<td>Krapp et al., 1993</td>
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<tr>
<td></td>
<td>Tobacco</td>
<td>Cricui et al., 1992</td>
</tr>
<tr>
<td></td>
<td>Tomato</td>
<td>Van Oosten and Besford, 1994</td>
</tr>
<tr>
<td></td>
<td>Tomato*</td>
<td>Van Oosten and Besford, 1995</td>
</tr>
<tr>
<td>RCA</td>
<td>Rubisco activase</td>
<td>Maize, Sheen, 1990</td>
</tr>
</tbody>
</table>

*Indirect evidence: repression obtained by applying high CO₂ concentration.

**Screening strategies for mutants in sugar-mediated regulation of photosynthesis genes**

Biochemical and physiological approaches have delivered limited progress in unravelling the cascade of events by which sugars regulate gene expression. The foreseeable complexity of the mechanism, by analogy with the yeast system (Thevelein, 1994; Ronne, 1995; Gancedo, 1998), and the probable interactions with other plant regulatory and biosynthetic pathways (e.g. of hormones) and the environment (e.g. light, osmotic stress, nitrogen, phosphate supply, etc), make it impracticable to dissect the pathway based solely on such approaches. Genetic and molecular techniques, especially in Arabidopsis, provide powerful tools for dissecting complex pathways. The relative ease by which Arabidopsis mutants can be generated, the more recent ready availability of tagged mutant collections (Feldmann, 1991; Bancroft et al., 1992; Long et al., 1993; Aarts et al., 1995; Klimyuk et al., 1995; Wisman et al., 1998), and the development of efficient techniques for cloning the genes tagged (Ochman et al., 1988; Liu et al., 1995; Souer et al., 1995) further highlight the potential of a molecular approach. Several groups have, therefore, isolated putative Arabidopsis mutants in carbon metabolite-mediated gene regulation.

Arabidopsis seeds can be exposed to high concentrations of widely occurring metabolizable sugars that allow germination, but inhibit further seedling development. Several different screens have been devised, ranging from mutants in induced. There are many genes whose expression is regulated by this mechanism (for reviews, see Koch, 1996; Quick and Schaffer, 1996; Jang and Sheen, 1997; Smeekens and Rook, 1997), and carbon metabolite-mediated regulation of gene expression seems to be a central and basic process, probably common to all higher plants.
Table 2. Putative Arabidopsis thaliana mutants in carbon metabolite-mediated gene regulation

<table>
<thead>
<tr>
<th>Mutant screen</th>
<th>Isolated with (mM)</th>
<th>Mutants isolated</th>
<th>WT ecotype</th>
<th>Mutagen</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>mig</td>
<td>mannose-insensitive germination</td>
<td>7–7.5 Man</td>
<td>72*</td>
<td>Col,Ws</td>
<td>EMS, T-DNA, transposon</td>
</tr>
<tr>
<td>sun</td>
<td>sucrose uncoupled</td>
<td>88 Suc</td>
<td>16</td>
<td>C24</td>
<td>EMS</td>
</tr>
<tr>
<td>rsr</td>
<td>reduced sucrose response</td>
<td>90 Suc</td>
<td>4</td>
<td>C24</td>
<td>EMS</td>
</tr>
<tr>
<td>sig</td>
<td>sucrose-insensitive growth</td>
<td>350 Suc</td>
<td>59*</td>
<td>Col</td>
<td>transposon</td>
</tr>
<tr>
<td>sis</td>
<td>sugar-insensitive</td>
<td>300 Suc or Glc</td>
<td>19</td>
<td>Col,Ws</td>
<td>EMS, T-DNA, transposon</td>
</tr>
<tr>
<td>gin</td>
<td>glucose-insensitive</td>
<td>333 Glc</td>
<td>≥4</td>
<td>Lar,Ws,Col</td>
<td>EMS, T-DNA</td>
</tr>
<tr>
<td>cal</td>
<td>carbohydrate-insensitive</td>
<td>100 Suc/low N</td>
<td>35*</td>
<td>Col,WS</td>
<td>EMS, T-DNA, transposon</td>
</tr>
<tr>
<td>gss</td>
<td>glucose-super-sensitive</td>
<td>56 Glt</td>
<td>22*</td>
<td>Col</td>
<td>transposon</td>
</tr>
<tr>
<td>sss</td>
<td>sucrose-super-sensitive</td>
<td>350 Suc</td>
<td>37*</td>
<td>Col</td>
<td>transposon</td>
</tr>
<tr>
<td>ram</td>
<td>reduced beta-amylose</td>
<td>29–88 Suc*</td>
<td>≥4</td>
<td>Col</td>
<td>EMS</td>
</tr>
<tr>
<td>lba</td>
<td>low beta-amylose</td>
<td>175 Suc</td>
<td>2</td>
<td>Col, Ler</td>
<td>EMS</td>
</tr>
<tr>
<td>hba</td>
<td>high beta-amylose</td>
<td>175 Suc</td>
<td>2</td>
<td>col</td>
<td>EMS</td>
</tr>
</tbody>
</table>

| Allelism tests to determine complementation groups are being performed. |
| The screen was performed in the absence of exogenous sugars by using the pgm (phosphoglucomutase) mutant in which transient, unusually high, endogenous sugar concentrations are sufficient to induce beta-amylose expression. In subsequent work this induction has been obtained with 29–88 mM sucrose. |

not transcribed and light is not emitted by the transgenic seedlings. By screening an ethyl methanesulphonate (EMS)-mutagenized population on 3% sucrose, 18 independent sucrose uncoupled (sun) mutants were isolated. A similar approach has been used (Martin et al., 1997) with a gene specific for sink tissues. These authors transformed Arabidopsis with the \( \beta \)-glucuronidase gene, expression of which is enhanced by sugars, under the control of a class I patatin promoter. In the transgenic plants GUS staining is seen in the roots when 90 mM sucrose is present in the medium. By screening an EMS-mutagenized population on 90 mM sucrose with a non-destructive screening system for GUS activity, four different rsr (reduced sucrose response) mutants were isolated.

Three other screens involving the sucrose-inducible \( \beta \)-amylose gene have yielded the low \( \beta \)-amylose (lba; Mita et al., 1997a), reduced \( \beta \)-amylose (ram; Donggiun et al., 1998), and high \( \beta \)-amylose (hba; Mita et al., 1997b) mutants. The first two display reduced sugar-induction of \( \beta \)-amylose gene expression whereas in the hba mutants this induction is increased. Although these screening strategies employed a gene which does not encode a protein of photosynthesis, it is conceivable that these mutants are also defective in sugar-mediated regulation of genes coding for photosynthetic components, but this remains to be tested.

Knowledge of the molecular mechanisms by which carbon metabolites regulate gene expression is still very limited, but mutants isolated from these screens are already proving to be highly useful in understanding the underlyin...
sufficient to overcome this repression (Pego et al., 1999). With the use of several different metabolizable and non-metabolizable glucose analogues, it was shown that the inhibition of germination is mediated by hexokinase (HXK). These results were corroborated by the finding that mannoheptulose, a specific HXK inhibitor, restored germination to seeds grown in the presence of mannose. The conclusion that HXK is involved in the mannose-mediated repression of germination of Arabidopsis seeds is relevant, since HXK is thought to be the primary sensor in sugar-mediated regulation of the expression of many plant genes (Graham et al., 1994; Jang and Sheen, 1994).

EMS- and T-DNA-mutated Arabidopsis seeds were plated on media containing 7.5 mM mannose and seedlings that germinated were taken from the plates. These plants were allowed to set seed and each line was rescreened for the mannose-insensitive germination (mig) phenotype. Under these conditions, wild-type seed displays a 2–3% germination frequency background. Lines with a 12%, or higher, germination frequency were chosen as putative mig mutants. Approximately 13 000 independent EMS-mutagenized lines of ecotype Columbia glabrous (Lehle Seeds, Round Rock, TX, USA), and 6 500 independent T-DNA tagged lines (Ws ecotype) generated by Kenneth Feldmann, University of Arizona, were screened. From these two mutant seed collections, 31 putative mig mutants were isolated, with germination frequencies on 7.5 mM mannose of up to 86% (Fig. 1). Allelic tests are currently under way to determine the number of independent mutations present in this population.

The mig screen is different from the ones described above for several reasons. Free mannose exists in plants only in trace amounts and is readily phosphorylated by HXK to mannose-6-phosphate (M-6-P). In many plant species M-6-P is only slowly metabolized (Herold and Lewis, 1977; Walker and Sivak, 1986) and, therefore, the addition of higher concentrations of mannose leads to the depletion of ATP and phosphate. Nevertheless, it was shown that the relatively low concentration of 7.5 mM mannose, used for the mig screen, did not provoke ATP depletion, and addition of phosphate to the mannose-containing medium did not restore germination to Arabidopsis seeds (Pego et al., 1999). Thus 7.5 mM mannose does not provoke ATP or phosphate depletion in Arabidopsis seeds and germination is repressed via an unknown, HXK-dependent mechanism. However, since ATP was only measured in whole seedlings, one may speculate about the possibility of ATP depletion in a specific cellular compartment which might be enough to block germination. Another peculiarity of the mig screen is that while high glucose and sucrose concentrations (over 300 mM) are needed to block seedling development, mannose can block germination at low concentrations (5 mM). This minimizes the probability of isolating undesired osmotic mutants.

The mig screen and other putative sugar-sensing mutant screens

Due to the similarity of several of the screens for mutants in sugar-mediated gene regulation (Table 2), and to the considerable number of mutants isolated to date, there is a possibility of mutant overlap between different screens. An experiment was set up to determine if this was indeed the case and, if so, to obtain a general idea of the extent of overlap. Seed from 30 mig mutants was plated on MS (Murashige and Skoog, 1962) medium, under the screening conditions for the mig (7.5 mM mannose), the sig and sis (350 mM sucrose), and the gin (333 mM glucose) screens. A plate containing 333 mM fructose was used in

![Fig. 1. Germination frequencies of mig mutants from T-DNA (mig30 to mig3) and EMS (mig16 to mig2) collections on 7.5 mM mannose. T-DNA lines are in Wassilewskija (Ws) background and EMS lines are in Columbia, containing the gl1 marker (Col). Approximately 100 seeds were used for each measurement.](image-url)
order to determine whether fructose may be equivalent to glucose in the isolation of the gin mutants. An additional plate containing 333 mM maltose was used for comparison with the gin, sig, and sis screens and 350 mM sorbitol was used as osmotic control. Consistent results were also seen with 3 and 5 mM mannose, 321 and 380 mM sucrose, and 389 mM glucose or fructose, but here data are only presented from the concentrations mentioned above.

The results obtained (Fig. 2) were quite surprising. Sucrose gives rise to glucose which, like mannose, is phosphorylated by HXK, but further metabolism of these two monosaccharides is distinct. One might have expected only a small overlap between the mig screen and other screens, since mannose exists in plants only in trace amounts and is capable of blocking germination at concentrations far lower than those of the other naturally-occurring sugars. The results showed, however, that there is considerable overlap between the mig screen and the gin, sig, and sis screens. In some extreme cases, such as with mig2 and mig3, it would have been possible to detect the mutants with any of the five sugar media tested. This suggests that mannose, glucose, fructose, sucrose, and maltose share a pathway by which Arabidopsis germination can be inhibited in mig2 and mig3. This pathway probably includes HXK since both the mig and the gin phenotypes have been reported to be, at least partly, mediated by a HXK-associated step (Jang et al., 1997; Pego et al., 1999). However, mannose is also capable of repressing germination via unique pathway(s) as can be seen by the results obtained with mig1 and mig7, for example.

Although sucrose and maltose are both disaccharides, it was observed that there is not complete overlap between seedling development on 350 mM sucrose (sig and sis screens) and on similar maltose concentrations. The same holds true for the gin screen and development on maltose. One can only speculate on the real significance of this observation and on the need for a maltose-insensitive screen to obtain saturation in the sugar-mediated gene regulation pathway.

The finding that mig12 and mig13 are hypersensitive for all the sugars tested was initially surprising. However, the observation that they are also hypersensitive for sorbitol indicates that they probably represent mutants hypersensitive to osmotic potential and are, therefore, incapable of germinating on the elevated concentrations of all the sugars tested. On the other hand mig4 and mig9 appear to be less sensitive to sorbitol, or insensitive to osmotic potential. Osmosis-related mutants were particularly unexpected, since the mig screen is based on the seed’s ability to germinate on 7.5 mM mannose which had no osmotic effect on germination.

It was concluded that there is partial overlap between all of the different screening strategies tested. It is tempting to speculate that a similar situation exists in relation to the other sugar-sensing screens (Table 2). Supporting this notion is the observation that sum6 is also a gin and mig mutant (Van Oosten et al., 1997; Pego et al., 1999) and that several cai mutants are also mig mutants (Martin et al., 1998). However, the results also show that there is a certain degree of specificity for each one of the screens. mig1 and mig7, for example, would not have been detected in any of the other screens whereas mig22 would have been detected in a gin, but not a sig or sis screen.

The results suggest that sugar-mediated regulation of gene expression occurs via a complex and branched pathway (Koch, 1996; Jang and Sheen, 1997; Smeekens and Rook, 1997; Halford et al., 1999). Part of this sugar-sensing mechanism seems to be at least partly conserved throughout eukaryotes and HXK has been reported to be a glucose sensor in sugar-mediated gene regulation in yeast, plant, and animal cells (Entian and Frohlich, 1984; Matschinsky et al., 1993; Heimberg et al., 1996; Jang et al., 1997). There are, however, also reports of sucrose-dependent–glucose-independent (Wenzler et al., 1989; Chiou and Bush, 1998; Rook et al., 1998) and glucose-dependent–HXK-independent (Roitsch et al., 1995;
Photosynthesis, sugars, gene regulation

Conclusions

Pathways of photosynthesis are well established, but the mechanisms by which they are regulated are less well understood. Progress in their elucidation has, however, been rapid since the discovery that CO$_2$, light, water, and other environmental signals are in some instances integrated, translated, and perceived by the plant as sugar signals. These are then processed, resulting in activation and/or repression of expression of a wide but specific set of genes. This carbohydrate-mediated mode of gene regulation is expected to be complex, as it not only controls genes encoding photosynthetic components, but simultaneously affects processes ranging from pathogen attack and environmental stress to storage protein accumulation and starch, lipid and nitrogen metabolism (reviewed in Koch, 1996; Jang and Sheen, 1997).

The employment of molecular genetic techniques for the isolation of mutants in this sugar-signalling pathway is, therefore, an important development in this field. Although different screening strategies have been employed to isolate such mutants, a certain degree of overlap was found by submitting 30 mig mutants to the conditions used for several of the other mutant screens. Results from the same experiment showed that the overlap between the different mutant screens is only partial, and mutants specific for each individual screen seem to have been isolated.

Although new insights into the mode of regulation of photosynthesis are already being obtained from the analysis of the existing sugar-signalling mutants, their study is still in its early stages and further research is needed to seize their potential fully. The integration of data obtained from all of the existing mutant screens is expected to provide meaningful insights into the mechanism(s) by which carbon metabolites regulate gene expression as a whole and photosynthesis in particular.

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