Review article

Sucrose and the integration of metabolism in vascular plants

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Abstract

We consider the hypothesis that sucrose is a signal as well as a substrate. We suggest that the significance of sugar sensing in plants is the integration of whole-plant carbon flux so that the capacity of sources to produce sucrose matches the capacity of sinks to consume it. We pay particular attention to difficulties with this hypothesis and the areas where further or better evidence is needed. We conclude that there is strong correlative evidence for a link between sucrose metabolism and the level of expression of key genes, but that a number of different mechanisms may be involved. © 2000 Elsevier Science Ireland Ltd. All rights reserved.

Keywords: Sucrose; Sucrose metabolism; Vascular plants

1. Introduction

The vast majority of higher plants exploit their environment through the interception of light energy, together with the acquisition of a range of compounds (principally water, carbon dioxide and inorganic nutrients). Considerable specialisation at both the tissue and organ level is associated with the capture of different resources. This specialisation generates a requirement to transport resources between and integrate the function of different organs.

Sucrose is one of the major forms in which material is transported around higher plants. Commonly, it is a major product of photosynthesis, the main form of translocated carbon and the main substrate for sink metabolism. We propose that sucrose has also got a significant regulatory and integrative function. In particular, we suggest that excess sucrose can feed forward to stimulate sink processes and feed back to down-regulate photosynthesis [1–3]. Sucrose metabolism is intimately linked to the metabolism of inorganic and organic N, and thus may be part of a wider mechanism for balancing resource acquisition and allocation within and between organs [4].

In this review, we will consider mainly the longer-term, integrative effects of sucrose on metabolism and development. Such effects are believed to be mediated via modulation of gene expression or protein turnover. We consider detailed examples of where sucrose is thought to modulate metabolic flux and discuss how such modulation might help to integrate spatially separate processes. We also consider how changes in sucrose content may be transduced into changes in gene expression. We pay particular attention to the quality of, and gaps in, the evidence supporting the general hypothesis that sucrose is a signal molecule.
1.1. Why sucrose?

The formal statement of the mechanism we propose is indicated in Fig. 1, and demonstrates the pivotal position of sucrose. There are three components of any regulatory system: a sensing mechanism, a transmission mechanism and a transduction mechanism. If sucrose is important as a regulator, it must be involved in one or more of these mechanisms in a way that is beyond any direct role as a substrate. The sucrose content of a plant or plant part represents a relatively long-term integral of the balance between supply (photosynthesis) and demand (growth, nutrient assimilation, storage etc). A simple calculation illustrates the potential for changes in pool size. A barley leaf produces sucrose photosynthetically at about 2 g m$^{-2}$ h$^{-1}$; since its sucrose content is typically 6 g m$^{-2}$, the size of the sucrose pool would increase 5-fold during a single photoperiod, if export was prevented. The sucrose pool clearly has the capacity to sense both internal and environmental change; what is the evidence that it does so?

There is no doubt that changes in assimilation by leaves and in metabolism by sinks are associated with changes in the overall sugar pool. Two examples will suffice. Increased irradiance leads to accumulation of both sucrose and starch in barley leaves within 8 h of treatment [5], and chilling roots and stem apices of Lolium temulentum (whilst maintaining leaves at 20°C) led to similar effects, together with the accumulation of fructose polymers. Reversal of this treatment was accompanied by mobilisation of reserves and a resumption of root and shoot growth [6]. Similar effects have been described for sink tissues although the changes are less marked, since the sucrose pool appears to be buffered (see Ref. [2] and Refs. therein). Bulk changes in sucrose content ignore the effects of cell or tissue compartmentation, and concentrations of sucrose and fructans can vary greatly between epidermis (nearly sugar free), mesophyll and parenchymatous bundle sheath in barley leaves ([7,8]; Fig. 2). Clearly, attempts to correlate sugars with the features they are thought to control should be focused at the level of the single cell, not a heterogeneous tissue. There is interchange between the various inter- and intracellular pools, giving a series of potential responses with different sensitivity, based upon the different size and turnover rates of the various components.

There is also abundant evidence that the rapid movement of sucrose through plants via the phloem is sensitive to the sugar status of both source and sink. The exact relationship between pressure-driven mass flow in the phloem and the overall sucrose abundance in source and sink is not clear, particularly given tissue-specific differences in distribution. It has been suggested that export rates are related to bulk sucrose concentration [9] although other evidence suggests that the rate correlates more closely with the nonvacuolar sucrose contents of mesophyll and vasculature ([10], B.E. Collis, J.F. Farrar and C.J. Pollock, unpublished observations). There is no doubt,
therefore, that changes in sucrose concentration can accompany changes in source or sink performance and can communicate these imbalances between different organs.

2. Do changes in sucrose content produce long-term changes in metabolism?

2.1. Sources

There is convincing evidence for the involvement of sugars in the regulation of leaf metabolism. The well-documented fine regulatory system involving hexose and triose phosphates in the control of carbon flux into sucrose and starch is probably directed towards ensuring optimal rates of ribulose bisphosphate regeneration rather than the balancing of supply and demand [11]. There are other examples of longer-term regulation involving sugars. Whilst genes of respiratory (cytochrome c; [12]) and 1-C [13] metabolism are sugar-regulated, naturally most attention has focused on photosynthesis. Sheen [14] showed that promoter-reporter fusions for genes coding for photosynthetic proteins were sensitive to sucrose, acetate and hexose but not to mannitol when measured in a maize propoplast transient assay system. More directly, increased concentration of assimilates were generated in tobacco leaves by over-expressing a yeast invertase in the cell wall and thus blocking phloem loading [15]. The transgenic plants expressing invertase showed reduced growth and photosynthesis and a decline in the activity of certain photosynthetic enzymes [16]. Exogenous supply of sugars to intact spinach leaves or to photosynthetic cell cultures of Chenopodium rubrum and endogenous accumulation induced by cold-girdling all gave similar effects [17,18].

Unfortunately, down-regulation of photosynthesis is not a universal response to assimilate accumulation in leaves. In temperate gramineae, rates of dry weight gain [19] or of carbohydrate accumulation and photosynthetic oxygen evolution [20] were linear over time, even though the tissue concentration of soluble sugars increased about fiftyfold during the experiment. One of the problems with comparing the results from this type of experiment is that bulk measurements of sugar accumulation may mask significant differences between different cell types [8]. It is possible, therefore, that ‘nonresponsive’ plants merely sequester the accumulated sugars at a site where they do not cause photosynthetic down-regulation. It is also easy to confuse direct effects on photosynthesis with indirect effects mediated through altered leaf ontogeny [2].

The effects of elevated CO₂ should be predictable if our hypothesis is correct: in general, growth at elevated CO₂ results in both higher concentrations of non-structural carbohydrates and a down-regulation of photosynthesis in source leaves, the former being held to cause the latter [21]. Some data are consistent, with correlations between carbohydrate content and photosynthetic rate [22,23]. However, there are exceptions. For example, just 24 h after switching barley from 350 to 700 ppm CO₂, the carbohydrate content of the
second leaf is substantially increased but photosynthesis is not down-regulated (B.E. Collis, J.F. Farrar and C.J Pollock unpublished observations; [24]), possibly because 24 h is too soon to see evidence of depressed rates of photosynthesis and emphasising the importance of a time-course in such experiments. Further the rate of photosynthesis depends on N as well as carbohydrate status [22,25,26], confounding experiments where simple correlations between carbohydrate and photosynthetic downregulation are sought. Down-regulation of photosynthetic rate can occur without a reduction in the amount of rubisco protein [27]. Clearly, we need direct evidence of causality between carbohydrates and photosynthetic gene expression at elevated CO₂. We need experiments where carbohydrate and nitrogen partitioning are measured along with photosynthetic rates, amounts of rubisco protein and transcript levels, over a time course following a change in CO₂ concentration.

Nevertheless, the balance of probability is that accumulation of sucrose or a closely related metabolise can feed back to reduce the expression of key photosynthetic genes; the most persuasive case is made by Moore et al. [28]. Sucrose accumulation may be a necessary, but not a sufficient, condition. There are, of course, a considerable number of signalling systems in plants that act by modifying gene expression and/or protein turnover and it seems improbable that these all act in isolation. The abundance and activity of individual proteins and the resultant flux through specific pathways will usually reflect the balance between a range of regulatory mechanisms, some of which are responsive to assimilate abundance.

One of the clearest demonstrations of a link between elevated sucrose contents and the induction of changes in the patterns of gene expression is shown during the induction of fructan accumulation in leaves of C₃ grasses and cereals. The kinetics of the process is shown in Fig. 3. The imposition of sucrose accumulation is followed by the progressive accumulation of short- and long-chain fructose polymers. Administration of inhibitors of gene expression (Table 1) has no effect on the photosynthetic accumulation of sucrose, but blocks the subsequent conversion of these to fructans. The enzymology of this pathway in gramineae is not completely understood [29]. However, we have shown that, under conditions of sucrose accumulation, there is progressive up-regulation of a number of mRNA species [13]. One of these up-regulated mRNA species is closely related to a fructosyl transferase cloned from barley [30] and is a member of the acid invertase gene family (J. Gallagher, C. Pollock, unpublished observations). In this case, accumulation of sucrose appears to be closely linked to the up-regulation of enzymes concerned in its subsequent metabolism. There are a number of other examples where genes associated with photosynthesis, resource allocation

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**Table 1**

<table>
<thead>
<tr>
<th>Inhibitor</th>
<th>Total water-soluble carbohydrate accumulated in 24 h (mg g⁻¹ fresh wt)</th>
<th>Percentage as fructan</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water control</td>
<td>43.3</td>
<td>58.4</td>
</tr>
<tr>
<td>Cycloheximide</td>
<td>40.5</td>
<td>2.5</td>
</tr>
<tr>
<td>(100 µM)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cordycepin</td>
<td>44.9</td>
<td>2.8</td>
</tr>
<tr>
<td>(1 mM)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>α-Amanitin</td>
<td>48.6</td>
<td>4.2</td>
</tr>
<tr>
<td>(1 mM)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L-MDMP</td>
<td>44.5</td>
<td>53.0</td>
</tr>
<tr>
<td>(10 µM)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>D-MDMP</td>
<td>40.8</td>
<td>1.0</td>
</tr>
<tr>
<td>(10 µM)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Data from reference [66].

b MDMP (2-(4-methyl-2,6-dinitroanilino)-N-methyl propionamide) is an inhibitor of cytoplasmic translation. Only the D- isomer is active; the L-isomer acts as an internal control.
and carbon metabolism in leaves appear to be sensitive to assimilate abundance [4,31], and it is tempting to regard this sensitivity as part of a generic regulatory framework selected to ensure that carbohydrate synthesis, storage and export are maintained in balance. However, we suggest that too much of the evidence for sugar-regulated gene expression is correlative and that there is a pressing need for more direct evidence.

2.2. Sinks

Once again, there is good evidence that a range of metabolic processes in sink tissues are sensitive to the supply of assimilate, but here the effects are stimulatory: the greater the sugar status, the greater the capacity for sinks to grow. Because of their sensitivity to experimental manipulations, their relatively low internal storage capacity and their broadly indeterminate habit, much of the work has been done on roots, but similar evidence exists for other types of sink. The total growth of all sinks on the plant cannot exceed the ability of source organs to supply them, and there is ample evidence that the size and respiration rate of the root system is regulated relative to that of the source leaves [32]. Reduction of sugar supply by darkening, defoliation or excision leads to a progressive decline in root growth and respiration, accompanied by declines in the amounts of enzymes involved in sugar metabolism and respiration but increases in proteolytic enzymes [2,33,34]. Significantly, many of these processes can be sustained in starved roots by the administration of exogenous sugars [2]; for example, the activity of fumarase and acid invertase and the amount of cytochrome c oxidase increase, and the activity of endoprotease and the transcript level of polyubiquitin decrease, on supplying metabolised sugars to excised barley root tips (B.E. Collis, J.F. Farrar, C.J. Pollock, unpublished observations), and there is evidence that cell division, which is arrested in G1 and G2 stages during starvation, can be restarted by the supply of sucrose or glucose to root tips [35].

The study of assimilate-responsiveness in genes coding for enzymes of sucrose metabolism has, however, identified an additional layer of complexity [31]. For sucrose synthase (Susy) and invertase (Ivr), there is evidence both for sugar modulation and for the reciprocal sensitivity of the products of different genes coding for the same activity. This has led to the idea of ‘feast’ and ‘famine’ genes. Koch suggests that up-regulation of ‘feast’ genes for Susy and Ivr in the presence of abundant imported sucrose could balance import and metabolism relative to supply. By contrast, the equivalent ‘famine’ genes appear to be localised in the epidermis and vascular strands and may have a role in maintenance of function during starvation by altering import priority towards essential tissues. This hypothesis is supported by the observation that cortical cells in roots are often sacrificed during stress [31] and emphasises our poor knowledge of how phloem transport relates to sugar metabolism within adjacent tissues. We would predict that the proteolytic responses to low sugar would, at least initially, be confined to the cortex whilst sugar pools and respiratory activity are retained in the vasculature.

There is, therefore, a significant body of evidence that is consistent with sink processes being directly responsive to assimilate supply and with that responsiveness being mediated via changes in gene expression. Furthermore, there are examples of where these responses are themselves part of the mechanisms that regulate both carbohydrate metabolism and sink activity. Taken in conjunction with the observations from leaves, it is possible to postulate a pivotal role for sucrose in the sensing of source–sink balance and in the regulation of growth and metabolism that is required to maintain such a balance.

However, our information for sinks is not as good as that for source leaves. We also need equivalent information for shoot apices. Critically, we need information on whether the rate-controlling processes in sinks are sugar-regulated. This is vital since respiration rate mirrors demand [32] and we do not know which genes and gene products in sinks are central in determining that demand. If our hypothesis is correct, it is these genes that should be sugar-sensitive.

2.3. The transport pathway

Implicit in the pressure-driven mass flow model for phloem translocation ([32]) is the fact that the turgor pressure gradient is sustained in part by loading of sucrose into the phloem in the leaves and unloading it in sink tissue. In principle, therefore, this mechanism continually integrates the
relative balance between supply of and demand for fixed carbon throughout the plant, facilitating the integration of the source and sink processes described above. Although the transport pathway itself can contribute to buffering change by virtue of the considerable amount of sucrose contained within it, there is no doubt that information on source–sink balance can be transmitted rapidly over significant distances. Chilling the growing points of grasses produces an immediate reduction in extension growth [36]. Within 30 min of this reduction, sucrose begins to accumulate at an enhanced rate in mature source leaves [37]. Similarly, when root temperature is reduced or increased, import of carbon into the root alters with minutes [38]. There is, therefore, no reason a priori to suppose that changes in sucrose concentration cannot contribute to the integration of plant metabolism over large distances. The possible regulation of phloem transport by the sucrose transporters involved in phloem loading [39] may add complexity, since whatever regulates the expression of these transporters will control sucrose flux partly independently of its abundance in source leaves and consumption in sinks. Transporter activity in isolated plasma membrane vesicles has indeed been shown to be sensitive to sucrose abundance in the tissues prior to extraction, as has expression of the appropriate mRNA species [40].

3. Cross-talk between regulatory systems: the case of C–N interactions

As indicated above, it is inappropriate to consider sucrose-regulation of plant processes in isolation. For example, the effects of changes in assimilate abundance on plant metabolism can be modified significantly by edaphic or biotic stress [4]. The importance of considering interacting regulatory mechanisms is particularly obvious in the case of the links between primary carbon and nitrogen metabolism.

At one level, physiologists have suggested that the basis of shoot–root allometry is the ‘functional equilibrium’ between carbon and nitrogen assimilation [41]. Under this model, carbon is acquired by the shoot, with provision to the root controlling the rate of root growth. In turn, nitrogen is acquired by the root; supply to the shoot determines the investment in growth and photosynthetic machinery and in turn controls the rate of carbon supply. Changes in the allometric constant under low light or low nutrient conditions are consistent with this model [42], but Farrar and Gunn [43] argue that shoot:root (S:R) ratio is regulated for resource acquisition, but none of the partial processes which determine it are themselves regulated in a simple way so that it is far from obvious how control of S:R is achieved. Further, we cannot answer the question of whether there is co-ordinated regulation of the amount of an organ and the density of resource-acquiring machinery within it — for example, is it the amount of shoot, or the amount of rubisco per unit leaf area, that is regulated?

At the biochemical level, nitrate reduction, sucrose synthesis and organic acid synthesis are all ‘sinks’ for the immediate products of photosynthesis. There is now compelling evidence that the relative demands of these processes for fixed carbon, ATP and reducing power are balanced via the coordinated regulation of sucrose phosphate synthase, nitrate reductase and PEP carboxylase activities [44]. This regulation occurs by specific and reversible phosphorylation that causes changes in existing enzyme activity.

It is clear that gene expression patterns also respond to changes in the C/N balance within plants. The responses are complex and are summarised in Fig. 4. In outline, a significant number of genes associated with both source and sink metabolism show sensitivity to both sugars and to components in the nitrogen assimilate pathway [45]. The patterns of expression change in such a way as to promote photosynthesis and carbon remobilisation when nitrogen is in excess and nitrogen assimilation/remobilisation, when carbon is in excess. Overlaying this is the participation of key enzymes of amino acid metabolism in the ‘feast or famine’ syndrome described above [4]. There may well be a whole range of interlocking regulatory systems that function at the level of gene expression/protein turnover and have sucrose sensitivity as one component. The precise response of different species to various combinations of external factors is likely to differ markedly even though there may be a common basis to the responses. It is striking, though, that we know far less of nitrogen compounds as signals than we do of sugars. One attractive possibility is that signals of C and N abundance are permissive signals, but
4. Possible mechanisms for the regulation of gene expression by sucrose

Although induction or suppression of metabolic pathways by sugars is well-known in microbes, there are few mechanistic models for how sucrose might act in this way. We are aware of two: the induction of levansucrase activity in Bacillus subtilis [46] and the hexose repression of invertase activity in yeast [47]. The former involves a specific role for sucrose, the latter does not. In B. subtilis, sucrose is phosphorylated in the cell membrane during uptake. This is associated with the dephosphorylation of the product of the sac X gene. In this state, the product no longer inhibits the sac Y gene, the product of which in turn functions as an

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### Gene Responses to Shifts in C/N Balance

<table>
<thead>
<tr>
<th>C:N Balance</th>
<th>Shoot development favoured</th>
<th>Priority given to photosynthesis and remobilisation of stored C</th>
<th>Root development favoured</th>
<th>Priority given to N-assimilation and remobilisation of stored N</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓C:↑N</td>
<td>‡ Shoot development favoured</td>
<td>‡ Priority given to photosynthesis and remobilisation of stored C</td>
<td>‡ Root development favoured</td>
<td>‡ Priority given to N-assimilation and remobilisation of stored N</td>
</tr>
<tr>
<td></td>
<td>‡ Photosynthesis genes</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Rubisco (rbcS, rbcL)</td>
<td>sug repr;</td>
<td>N stim</td>
<td>N-assimilation genes</td>
</tr>
<tr>
<td></td>
<td>PEP c’ase</td>
<td>sug repr;</td>
<td>aa stim</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lt reactions</td>
<td>sug repr;</td>
<td>N stim</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Chlorophyll</td>
<td>sug repr;</td>
<td>N stim</td>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>‡ C-remobilisation genes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>α-amylase</td>
<td>sug repr;</td>
<td>undef</td>
<td></td>
</tr>
<tr>
<td></td>
<td>lipid breakdown</td>
<td>sug repr;</td>
<td>undef</td>
<td></td>
</tr>
<tr>
<td></td>
<td>endoglycosidasises</td>
<td>sug repr;</td>
<td>undef</td>
<td></td>
</tr>
<tr>
<td></td>
<td>proteases (non psyn)</td>
<td>sug repr;</td>
<td>undef</td>
<td></td>
</tr>
<tr>
<td></td>
<td>N-detox, cycling (non psyn)</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>GDH1</td>
<td>sug repr;</td>
<td>NH₃ stim</td>
<td></td>
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<tr>
<td></td>
<td>ASN1</td>
<td>lt, sug repr;</td>
<td>aa stim</td>
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<td></td>
</tr>
<tr>
<td></td>
<td>‡ C-export genes</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>SPS (leaf mesophyll)</td>
<td>sug repr;</td>
<td>undef</td>
<td></td>
</tr>
<tr>
<td></td>
<td>SS (SH1, phloem)</td>
<td>sug repr;</td>
<td>undef</td>
<td></td>
</tr>
<tr>
<td></td>
<td>GS1 (phloem)</td>
<td>sug repr;</td>
<td>NO₃, NH₄ stim</td>
<td></td>
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<tr>
<td></td>
<td>‡ N-sinks favoured</td>
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<td></td>
<td>‡ N-import/metabolism</td>
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<td></td>
<td>Aa transporters?</td>
<td></td>
<td>undef</td>
<td></td>
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<tr>
<td></td>
<td>‡ N-use</td>
<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td>leaf storage proteins (low sug)</td>
<td>N stim</td>
<td></td>
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<tr>
<td></td>
<td>Rubisco (storage)</td>
<td>sug repr;</td>
<td>N stim</td>
<td></td>
</tr>
<tr>
<td></td>
<td>ASN1 (ASN storage)</td>
<td>sug repr;</td>
<td>aa, NH₄ stim</td>
<td></td>
</tr>
</tbody>
</table>

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Fig. 4. Gene responses to shifts in C/N balance. Redrawn with permission from Koch, 1997. Abbreviations are as follows: stim, stimulated; repr, repressed; undef, undefined; sug, sugar; lt, light; aa, amino acid.
anti-terminator and facilitates transcription of the structural gene for levan sucrase [48].

In yeast, it appears that a specific hexokinase functions in a regulatory as well as a catalytic role and thus senses flux rather than abundance. The hexokinase, possibly as a substrate–enzyme–effector complex, initiates a protein kinase cascade that acts as a regulator of transcription [4,49]. At least one of the kineses has a homolog in higher plants, suggesting that a similar system may operate there [50–52]. In plants, sucrose signalling would be initiated by the cleavage of sucrose molecules either by invertase or by sucrose synthase. This would generate substrates for the hexokinase, and the extent of flux through this enzyme would determine the extent of up-regulation of genes that responded via the protein kinase cascade [49]. Jang and Sheen [49] provide experimental evidence, using a maize transient expression assay, that phosphorylation of hexoses is a key part of this transduction pathway. Importantly, transgenics over- or under-expressing hexokinase are hyper- and hypo-sensitive to sugar, respectively [53]. Interestingly enough, the possession by plants of alternative pathways for sucrose cleavage could affect the level of response. Inversion of sucrose liberates two potential substrate molecules for hexokinase, whereas cleavage by Susy generates only one. Additionally, invertase isoforms with different catalytic properties are present in the vacuole, the apoplast and the cytosol, and this may also affect substrate availability for the hexokinase step. Hexokinase-based regulation would appear to sense the hexose/hexose phosphate pool, whereas regulation of transporter activity, and of fructan accumulation appear to be more specifically associated with changes in sucrose content [40,54,55]. These remain as provisional models for sucrose-sensing and transduction pathways in plants, but the frequency of occurrence of sugar-responsive pathways in microbes suggests a degree of conservation and the probability of a restricted number of common mechanisms.

There are some deep problems remaining. For example, how is sucrose-mediated gene regulation related to sugar concentration: is it a linear function of concentration, is there a threshold, or is the regulation sensitive to flux rather than to concentration? There are other issues to be resolved. How is the signal time-integrated? Since sucrose concentration in leaves can alter 5–10-fold in a day [8,56], is there continuous modulation of gene activity with the amount of individual mRNAs and proteins providing the integration? Simply understanding the signal transduction pathway will not enable us to understand how sugar signalling fits fully into the daily life of the plant. Finally, Trewavas [57] has suggested that the sensitivity of tissues to plant growth substances can vary greatly during ontogeny and may be the dominant factor determining the effects of growth substances: could the same be true of sugar regulation? The question is especially pertinent since there may be an interaction of sugar levels and hormonal regulation [58,59].

5. Higher-order integration: sucrose as a morphogen

The mechanisms discussed above principally involve sucrose as a substrate, either directly or via its potential to modulate the activities of enzymes involved in its synthesis, cleavage or subsequent metabolism. There are also a few reports of sucrose acting to alter developmental pathways in groups of cells. Perhaps the clearest indication of this is the demonstration that sucrose in combination with auxin can substitute for an excised bud in inducing the differentiation of vascular tissue within a block of undifferentiated callus tissue. At constant auxin concentration, increasing concentrations of sucrose increased the proportion of phloem vessels in the vascular tissue [60]. Jeffs and Northcote [61] showed that this effect could be obtained only with α-glucosyl disaccharides (e.g. sucrose, trehalose and maltose).

There is some evidence for a similar effect during the floral transition and fruit abortion (discussed in reference [2]) but no mechanistic explanations exist for these observations. It remains possible, however, that the overall effects of sucrose include morphogenetic as well as substrate-level and regulatory interactions.

6. Concluding remarks

Models of metabolic regulation in plants have changed significantly over the past ten years. At the level of fine control, it is accepted that control is a system property, rather than residing in one or
two 'rate limiting' enzymes [62]. Above this, we have a much clearer idea of how the amount of specific enzyme protein is regulated via changes in gene expression, post-translational modification and protein turnover. We are also using the precision and high resolution of molecular methodologies to gain an understanding of how processes may be regulated and integrated in time and space. This broader approach has produced a considerable body of evidence that is highly suggestive (if rarely conclusive) of a key role for sucrose in the integration of plant growth and function. The increased availability of defined transgenic and mutant plants will permit many of the hypotheses outlined in this review to be tested more rigorously, but it is important to remember the extent of plant diversity, even in primary carbon metabolism. There are, for example, species that store an even wider range of carbohydrates [63] and that have very different allocation strategies to those most frequently studied [64]. We should not neglect this diversity as a valuable tool in the assessment of the various roles for sucrose in higher plant metabolism.

Acknowledgements

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