Sugar and hormone connections

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Sugars modulate many vital processes that are also controlled by hormones during plant growth and development. Characterization of sugar-signalling mutants in Arabidopsis has unravelled a complex signalling network that links sugar responses to two plant stress hormones – abscisic acid and ethylene – in opposite ways. Recent molecular analyses have revealed direct, extensive glucose control of abscisic acid biosynthesis and signalling genes that partially antagonizes ethylene signalling during seedling development under light. Glucose and abscisic acid promote growth at low concentrations but act synergistically to inhibit growth at high concentrations. The effects of sugar and osmotic stress on morphogenesis and gene expression are distinct. The plasticity of plant growth and development is exemplified by the complex interplay of sugar and hormone signalling.

The role of sugars as signalling molecules has been widely recognized in microorganisms and has recently emerged in animals and plants [1]. During plant growth and development, sugars modulate a range of vital processes such as seed germination, seedling development, root and leaf differentiation, floral transition, fruit ripening, embryogenesis, and senescence, as well as responses to light, stress and pathogens [2–9]. Based on the different responses that diverse genes exhibit to particular sugars or sugar-phosphorylation activities, the existence of multiple signal-transduction pathways has been proposed [6,7]. Currently, it is unclear whether these signalling pathways interconnect or whether they function in specific cell types or at specific developmental stages. To address these types of questions, it is necessary to understand in temporal and spatial detail the mechanisms by which sugar signals are transduced in each pathway and the nature of the molecules that participate in these processes.

In an effort to understand the molecular mechanisms involved in the sugar perception and signalling network, genetic strategies have been designed independently to select either sugar-insensitive or sugar-oversensitve mutants, mainly in Arabidopsis. The genetic screens are reminiscent of those devised for the isolation of plant hormone mutants. By using easily detectable phenotypes such as the sugar inhibition of germination or postgermination development in seedlings, mutants with altered sugar responses have been isolated and characterized [10–15]. Sugar-response mutants have also been isolated by screening transgenic seedlings carrying reporter and selection genes under the control of sugar-regulated promoters [16,17]. These distinct screening strategies yielded overlapping mutants. Surprisingly, characterization of these mutants has led to the identification of mutants and genes previously implicated in plant-hormone biosynthesis or signalling, and revealed extensive connections between the sugar and plant-hormone pathways [18]. The aim of this article is to highlight the latest findings that might provide some molecular insights into the intricate network of plant sugar signalling and its direct, extensive relationship to abscisic acid (ABA) and ethylene hormonal pathways.

Sugar-signalling mutants reveal the hormone connections

Recent genetic and molecular studies of sugar-signalling mutants in Arabidopsis have uncovered many unexpected links between sugar and plant-hormone signalling (Table 1). In addition to the altered sugar responses (Fig. 1), these mutants display phenotypes found in ABA or ethylene biosynthesis mutants and in ABA or ethylene signalling mutants (Fig. 2). For example, the small, dark-green leaves of the gin1 mutant, resembling the constitutively active ethylene mutant ctr1 (Fig. 2), prompted the application of an ethylene precursor 1-aminocyclopropane-1-carboxylate...
Table 1. Arabidopsis mutants involved in sugar and hormone connections

<table>
<thead>
<tr>
<th>Mutant</th>
<th>Phenotype</th>
<th>Gene and protein</th>
<th>Function</th>
<th>Refs</th>
</tr>
</thead>
<tbody>
<tr>
<td>gin1</td>
<td>Glucose insensitive, growth retardation, wilty, seed defect</td>
<td>ABA2 and SDR1</td>
<td>ABA biosynthesis</td>
<td>[12,19]</td>
</tr>
<tr>
<td>gin2</td>
<td>Glucose insensitive, growth retardation, delayed leaf senescence, seed defect</td>
<td>GIN2 and HXK1</td>
<td>Glucose sensor, hexose phosphorylation</td>
<td>[18]</td>
</tr>
<tr>
<td>gin4</td>
<td>Glucose insensitive, growth retardation, constitutive triple response</td>
<td>CTR1</td>
<td>Raf-like protein kinase, negative regulator in ethylene signalling</td>
<td>[19]</td>
</tr>
<tr>
<td>gin5</td>
<td>Glucose insensitive, wilty</td>
<td>ABA3/LOS5 and MCSU</td>
<td>Biosynthesis of the molybdenum cofactor</td>
<td>[13]</td>
</tr>
<tr>
<td>gin6</td>
<td>Glucose insensitive, ABA insensitive, osmotolerant, salt resistant</td>
<td>ABI4</td>
<td>AP2 transcription factor in ABA signalling</td>
<td>[13]</td>
</tr>
<tr>
<td>isa3</td>
<td>Glucose and mannose insensitive</td>
<td>ABI4</td>
<td>AP2 transcription factor in ABA signalling</td>
<td>[17]</td>
</tr>
<tr>
<td>isa4</td>
<td>Glucose insensitive</td>
<td>ABA2 and SDR1</td>
<td>ABA biosynthesis</td>
<td>[17]</td>
</tr>
<tr>
<td>sia1</td>
<td>Glucose and mannose insensitive, osmotolerant, constitutive triple response</td>
<td>CTR1</td>
<td>Raf-like protein kinase, negative regulator in ethylene signalling</td>
<td>[20]</td>
</tr>
<tr>
<td>sia4</td>
<td>Sucrose and glucose insensitive, osmotolerant, wilty, paclobutrazol resistant</td>
<td>ABA2 and SDR1</td>
<td>ABA biosynthesis</td>
<td>[14]</td>
</tr>
<tr>
<td>sia5</td>
<td>Sucrose, glucose and mannose insensitive, osmotolerant, ABA insensitive, paclobutrazol resistant</td>
<td>ABI4</td>
<td>AP2 transcription factor in ABA signalling</td>
<td>[14]</td>
</tr>
<tr>
<td>sun6</td>
<td>Sucrose, glucose, mannose and ABA insensitive</td>
<td>ABI4</td>
<td>AP2 transcription factor in ABA signalling</td>
<td>[16]</td>
</tr>
<tr>
<td>prl1</td>
<td>Sucrose, glucose, cytokinin, auxin, ABA, and ethylene hypersensitive, reduced root elongation, starch and sugar accumulation in leaves</td>
<td>PRL1</td>
<td>An α-importin WD40 protein, interacts with SnRKs</td>
<td>[11,15]</td>
</tr>
<tr>
<td>aba1</td>
<td>Glucose and sucrose insensitive</td>
<td>ABA1 and ZEP1</td>
<td>ABA biosynthesis</td>
<td>[13,14,16]</td>
</tr>
<tr>
<td>abi5</td>
<td>Glucose insensitive</td>
<td>ABI5</td>
<td>bZIP transcription factor in ABA signalling</td>
<td>[13,14]</td>
</tr>
<tr>
<td>etr1</td>
<td>Glucose oversensitive, ethylene insensitive</td>
<td>ETR1</td>
<td>Ethylene receptor, histidine protein kinase</td>
<td>[12,51]</td>
</tr>
<tr>
<td>eto1</td>
<td>Glucose insensitive</td>
<td>ETO1</td>
<td>Ethylene biosynthesis</td>
<td>[12,51]</td>
</tr>
<tr>
<td>ein2</td>
<td>Glucose oversensitive, ethylene, jasmonate and paraquat insensitive</td>
<td>EIN2</td>
<td>Integral membrane protein, ethylene and jasmonate signalling</td>
<td>[19,52]</td>
</tr>
<tr>
<td>ein3</td>
<td>Glucose oversensitive, ethylene insensitive</td>
<td>EIN3</td>
<td>Transcription factor in ethylene signalling</td>
<td>[51]*</td>
</tr>
<tr>
<td>ein6</td>
<td>Glucose oversensitive, ethylene insensitive</td>
<td>EIN6</td>
<td>Unknown protein</td>
<td>[51]*</td>
</tr>
<tr>
<td>hys1</td>
<td>Glucose oversensitive, early leaf senescence, constitutive expresser of PR genes (priv)</td>
<td>HYS1/CPR5</td>
<td>Unknown protein</td>
<td>[58]</td>
</tr>
</tbody>
</table>

Abbreviations: ABA, abscisic acid; bZIP, basic leucine zipper domain; CPR5, constitutive expresser of PR; HXK1, hexokinase 1; MICSU, moco sulfurase; SDR, short-chain dehydrogenase/reductase; SnRK, SNF1-related protein kinase; ZEP1, zeaxanthin epoxidase. Mutant names: aba, ABA deficient; abi, ABA insensitive; ein, ethylene insensitive; eto, ethylene overproducer; etr, ethylene response; gin, glucose insensitive; hys, hypersenescence; isi, impaired sugar induction; prl, pleiotropic regulatory locus; sis, sugar insensitive; sun, sucrose uncoupled. Gene names and alternative protein names are listed.


(ACC) to wild-type plants in the presence of excess exogenous glucose. This experiment demonstrated that the glucose-dependent developmental arrest could be overcome by ethylene (Fig. 1) [12]. Although ethylene is generally known as a stress hormone that inhibits growth, this study reveals a growth-promoting role of ethylene in Arabidopsis. Interestingly, the ctr1 mutant produces many more leaves than wild-type plants even in the absence of exogenous glucose (Fig. 2). It was also found that the sugar-insensitive gin1 and gin5 mutants had reduced-seed-dormancy and wilty phenotypes that are characteristics of ABA-deficient (aba) mutants [13,19]. These findings led to an awareness of the important genetic interactions between the sugar and ABA, and between the sugar and ethylene signalling pathways [12–14,16,17,19].

The molecular cloning of GIN6/SUN6 as ABI4 and GIN1/ISI4 as ABA2 strengthened the molecular connections between sugar and ABA [13,16,17,19]. Genetic analyses also confirmed that gin1, sis4 and isi4 are new aba2 alleles, that gin4 and sis1 are new ctr1 alleles, and that gin5 is allelic to aba2 [13,14,16,17,19,20] (A. Arroyo et al., unpublished). Details of these mutant studies have recently been covered by excellent reviews [6,8,21,22]. However, with these findings, many questions have been raised about the physiological relevance and specificity of the sugar mutant screens and the deduced genetic interactions, as well as the overlap between stress and sugar signalling and the molecular bases of these complex interactions.

Glucose controls ABA biosynthesis genes

Direct biochemical analyses show that several sugar signalling mutants (gin1, gin5, isi4 and sis4) contain lower endogenous ABA levels than wild-type plants.
leaves. Its sucrose induction depended on the phenotypic subunit that is involved in starch biosynthesis. Promingly, the reporter gene in transgenic plants indicates ABA2::GUS to all developmental stages. The expression pattern of an ABA biosynthesis can be generalized to all cell types or dependent manner. ABA signalling genes and ethylene responses in an ABA-arrest is a complicated trait, and glucose control of ABA artificial response. The glucose-dependent developmental suggestion that the glucose–ABA interaction is an end during active plant development, arguing against the modulation of ABA biosynthesis genes and ABA accumu-lation. This regulation, however, requires a synergistic modulation of ABA biosynthesis genes and ABA accumu-lation. These results strongly suggest a direct, specific glucose modulation of ABA biosynthesis genes and ABA accumulation. This regulation, however, requires a synergistic interaction of glucose and ABA because it is abolished in the null glucose-insensitive gin1 mutant, which displays endogenous ABA deficiency. The data also indicate that the modulation of ABA levels by glucose is involved not only in the developmental arrest of seedlings but also during active plant development, arguing against the suggestion that the glucose–ABA interaction is an artificial response. The glucose-dependent developmental arrest is a complicated trait, and glucose control of ABA biosynthesis genes and ABA accumulation contribute to this phenotype. As discussed below, glucose also modulates ABA signalling genes and ethylene responses in an ABA-dependent manner. Currently, it is unclear whether this glucose induction of ABA biosynthesis can be generalized to all cell types or to all developmental stages. The expression pattern of an ABA2::GUS reporter gene in transgenic plants indicates that most of the cells in young seedlings can respond to the presence of elevated glucose levels and induce the expression of this ABA biosynthesis gene. Intriguingly, the Apl3 gene (encoding an ADP–glucose pyrophosphorylase subunit that is involved in starch biosynthesis) is activated by sucrose but not ABA or mannitol in mature Arabidopsis leaves. Its sucrose induction depended on IS14/ABA2/GIN1 only when leaves were submerged in sucrose solution, not when sucrose was fed through the petiole. It was proposed that the petiole-fed leaves were subject to dehydration stress even though a wilty phenotype was not observed. Because ABA2::GUS is highly expressed in the petiole and induced by sugar, an alternative explanation could be that the submerged leaves did not generate sufficient ABA through the petiole to induce ApL3 in the is14 mutant. Apparently, both sucrose and ABA are required synergistically for ApL3 gene expression. In petiole-fed leaves, abundant sugar signals promoted sufficient ABA synthesis in the is14 mutant, which was not a null allele like gin1. This study provides a physiologically relevant example of sugar regulation of ABA biosynthesis that is not restricted to young seedlings. It will be interesting and informative to carry out more-detailed analyses of the expression patterns of other ABA biosynthesis genes and their responses to sugars as well as their roles in sugar signalling.

Glucose controls ABA signalling genes

As shown in Table 1, the characterization of several sugar mutants (such as gin6, is13, sis5 and sun6) has revealed their allelism to the ABA-responsive mutant abi4. The abi4 mutant was originally selected by its ability to germinate in the presence of high levels of exogenous ABA (3 μM), which are normally inhibitory to wild-type germination and seedling development. ABI4 encodes a transcription factor of the APETALA 2 (AP2) domain family that plays a major role during seed development and germination together with two other loci, ABI3 and ABI5. The participation of ABI4 in other aspects of vegetative growth only became clear with the finding that abi4 alleles were isolated as sugar- and salt-resistant mutants. These results are consistent with the low ABI4 expression in vegetative tissues in addition to seeds (A. Arroyo et al., unpublished). Interestingly, ABI4 is activated by 6% glucose in an ABA-dependent manner but its response to ABA alone is limited. The glucose-responsive regulatory DNA sequence might lie 2 kb upstream of the coding sequence, where a T-DNA insertion abolished glucose induction. Thus, glucose and ABA modulate the expression of both ABA biosynthesis and an ABA signalling genes, but the control of transcription factors requires higher levels of glucose.

In addition to abi4, it was found that the abi5 mutant also displayed a glucose-insensitive phenotype. This phenotype appears not to be as strong as that of the abi4 alleles and is not consistent in some studies. However, overexpression of ABI5 confers hypersensitivity to sugars. The ABI5 gene encodes a transcription factor that belongs to a large basic leucine zipper (bZIP) domain family. In addition to its role during late embryogenesis, this gene plays a crucial role at the checkpoint of postgermination developmental arrest and probably in specific tissues during vegetative growth. Like ABI4, ABI5 is activated by glucose in an ABA-dependent fashion (A. Arroyo et al., unpublished). Unlike ABI4, however, several members of this family, including ABI5, are highly inducible by ABA and
mannitol [19,35,37]. It is possible that the role of ABI5 in the glucose-induced developmental arrest partially overlaps with other bZIP factors [35]. This hypothesis is supported by the recent finding that overexpression of two other members of this family (ABF3 and ABF4) resulted in glucose hypersensitivity [38]. ABA induction of other ABI5 homologues could explain the puzzling observation that the glucose-insensitive phenotype was reverted by the addition of exogenous ABA in the ABA-insensitive abi5 mutant [13]. It is also possible that the analysed abi5 allele is not null and ABA increases the mutant ABI5 expression to sufficient levels to restore a wild-type response. To dissect the participation of diverse elements and factors in sugar responses, the complex mode of regulation and direct interactions among several ABA signalling components need to be considered.

In contrast to the ABA biosynthesis deficient mutants, not all of the abi mutants display abnormal responses to sugars. Based on the sugar phenotype in young seedlings, ABI1, ABI2 and ABI3 do not seem to have a major role in glucose signalling [13,14,16]. However, abi1 and abi2 are dominant mutants [39]. Elucidation of the true role of ABI1 and ABI2 in sugar signalling might require more effort because of potential functional redundancy of many serine/threonine protein phosphatase 2Cs [40,41]. A recent study has shown that ABI3 is also activated by 6% glucose in an ABA-dependent manner, albeit at a lower level than ABI4 and ABI5 [19]. In addition, ABI3 overproduction increases sensitivity to glucose at seedling stage [25]. The tested abi3 mutant could be a weak allele, and a null allele should be tested to clarify the involvement of ABI3 in sugar responses.

Distinct glucose and osmotic-stress signalling

After germination, a plant’s success depends on its ability to grow autotrophically. During this transition time, plants live as heterotrophs while the photosynthetic apparatus becomes competent. Seedling establishment is such a crucial period that rapid responses to a range of factors are fundamentally important. In fact, it has been suggested that this period defines a checkpoint during which internal and external conditions are monitored [37]. The effect of excess glucose during this development stage results in an arrest of seedling growth and differentiation that seems to be mediated partly by the increase in ABA biosynthesis and by the activation of some ABA signalling genes. A concern has been raised about whether this developmental response reflects a direct sugar regulation process or an indirect osmotic effect, because ABA is well known to be involved in stress responses that allow plants to cope with environmental fluctuations [22].

Even though ABA plays a central role in sugar, salt and osmotic-stress responses [25,42], several lines of evidence indicate that the accumulation of ABA in response to sugars or to osmotic stress is mediated by distinct mechanisms. First, high glucose but not mannitol levels result in developmental arrest with no cotyledon expansion or greening [12,13,43]. Second, drought and osmotic stress, but not glucose, directly activate the NCED3 gene, a rate-limiting step for stress-induced ABA accumulation [19,29–31]. Third, ABA2 is activated by glucose but not by drought or osmotic stress [19]. Finally, ABI3, ABI4 and ABI5 expression are regulated differently by glucose and mannitol [19,35]. ABI4 and ABI5 transcripts are positively regulated at a low concentration by the glucose analogue 2-deoxyglucose, which acts as a sugar signal but not as an osmotic stimulus [4,6] (A. Arroyo et al., unpublished). The fact that multiple mutant alleles for the same genes involved in ABA biosynthesis or ABA signalling have been isolated from sugar- and salt/osmotic-stress screens seems to reflect the important role of ABA in both types of signalling. In addition, sugar signals appear to modulate the action of other plant hormones besides ABA [12,18] (L. Zhou and J. Sheen, unpublished).

Glucose and ABA in growth promotion and inhibition

During vegetative growth, ABA has been shown to play a central role in adaptive responses to environmental cues such as drought and other stress responses [44,45]. Consequently, ABA has been considered as a stress hormone and growth inhibitor. However, recent data have revealed the importance of ABA in normal root and shoot growth [19,46], suggesting that the physiological roles of this hormone might be far more complex than those that have been conventionally defined [25]. Similar to sugars and to the plant growth hormone auxin, high and low ABA concentrations could have opposite effects on plant growth and development [7,19,46–48].

Most of the well-known ABA functions have been defined at elevated ABA levels (e.g. under stress conditions) and at the late stage of seed development. Up to 100 μM exogenous ABA is routinely used for root-inhibition or gene-regulation studies, or for mutant screens and analyses. However, the growth defects observed in gin1 and other abi mutants actually suggest that endogenous ABA is required to promote plant growth and development in the absence of severe stress (Fig. 2) [19]. Similar observations are also made with the analyses of ABA-deficient mutants in tomato [46]. This important aspect of ABA function is just emerging and is not yet fully understood. Recent studies have also revealed that the growth hormone auxin can potentiate the ABA response in roots [49] and that the stress hormone ethylene can antagonize ABA signalling in seedlings [19,46,48,50]. Therefore, glucose regulation of ABA biosynthesis and signalling could impact auxin and ethylene responses.

Glucose and ethylene connections

Unexpectedly, the characterization of the gin1 mutant uncovered a mutually antagonistic relationship between the glucose and ethylene signalling pathways [12]. This antagonistic connection is strongly supported by the discovery that the ethylene overproduction (eto1) and ethylene constitutive signalling (ctr1) mutants are glucose insensitive. Consistent with this, several ethylene-insensitive mutants (ctr1, ein2, ein3 and ein6) display glucose oversensitivity [12,19] (W-H. Cheng and J. Sheen, unpublished). Further support came from the finding that two sugar-insensitive mutants, sis1 [20] and gin4 [19], are new alleles of ctr1 [51]. Although gin1 and gin4 exhibit similar glucose-insensitive phenotypes during postgermination development (Fig. 1), only gin4/ctr1 displays the
constitutive triple-response phenotype in the dark, a classical hallmark of ethylene action [51]. Recent studies have shown that overproduction of the C-terminal portion of EIN2 confers constitutive ethylene signalling with no triple response in the dark [52]. Thus, glucose signalling might affect specific ethylene pathways acting downstream of ETR1 and EIN2 [12,19,51]. Further analysis of the molecular links between glucose and ethylene signalling might distinguish branching pathways responsible for downstream responses to ethylene and elucidate interactions between ethylene and glucose, as well as other plant hormones.

The analyses of sugar signalling mutants have suggested that ethylene acts as an antagonist of the glucose response, whereas ABA promotes it. The link between these two hormones with respect to glucose has become clearer with the analysis of the double mutants gin1 etr1 and gin1 ein2, which display the glucose-insensitive phenotype of the gin1/aba2 mutant. Thus, ethylene seems to affect glucose signalling through ABA and to promote germination and seedling development. Based on the elevated ABA level found in the ein2 mutant, it is likely that ethylene signalling partially represses the biosynthesis of ABA [19,48]. Interactions between ethylene and ABA have also been found independently [48,50]. It has been proposed that ethylene acts as a negative regulator of ABA signalling in the control of seed dormancy by decreasing the sensitivity to endogenous ABA [48]. The measurement of ABA levels and the expression of at least one ABA biosynthesis gene transcript (ZEP1) in 30-day-old plants suggest that ethylene decreases ABA biosynthesis [48]. Similar interactions could also occur during seed germination. However, the connection between these two stress hormones in a root-growth assay seems to be quite different. In wild-type plants, the presence of low exogenous ABA concentrations (100 nM) promotes seedling root growth, whereas the ethylene precursor ACC (0.2 μM) and a high concentration of ABA (100 μM) both inhibit root growth. In these experiments, the roots of ethylene-insensitive mutants, such as etr1 and ein2, are resistant to exogenous application of ethylene and ABA [48,50]. It appears that the interactions and the mode of action of these two hormones vary depending on the hormone concentrations and the tissues analysed. These results are consistent with the new role of endogenous ABA (low concentration) in promoting root growth [19]. The high level of exogenous ABA (100 μM) probably represents a stress signal reminiscent of ethylene or the ‘stress’ effect of the growth-promoting hormone auxin at high exogenous levels [53]. Interestingly, the strong auxin-resistance mutant axr2 is relatively insensitive to both ABA and ethylene signals, suggesting a possible convergence point of three hormonal signalling pathways [54].

Conclusions and perspectives

Genetic and phenotypic analyses of Arabidopsis sugar-signalling mutants have unravelled complex and extensive interactions between sugar and hormonal signalling pathways (Table 1, Fig. 3). Over the past few years, the simplistic idea of a linear transduction pathway in plants for regulating complex processes has begun to change. The intricate and complex webs that interrelate sugar and hormone signalling pathways are excellent examples of such complexity [22]. Multiple regulatory components originally isolated in a particular signalling pathway have now been shown to interconnect with other pathways [48,50,55]. This plasticity could facilitate central nodes of integration that are crucial for a plant’s flexible responses to physical and chemical environments. Transcription factors such as ABI4 and ABI5 could act as integration nodes, receiving signals from multiple signals and generating integrated plant responses. With the increased availability of molecular probes for the genes involved in the sugar and hormone connections, more direct and specific molecular links can be established.

Although both ABA and ethylene were previously defined as stress hormones, their interaction with sugar signals is the opposite under the condition used for sugar signalling mutant screens. It was during the analysis of the gin1 mutant that the shoot-promoting effect of ethylene was revealed in Arabidopsis. Even in the absence of high sugar levels, ethylene can promote hypocotyl elongation under light but inhibit the same process in the dark [52,56]. The initially puzzling interactions between ABA and ethylene in the seed-germination and root-growth assays could simply be attributed to changing responses at different concentrations of exogenously applied ABA and distinct intrinsic states of different tissues [48,50]. Interestingly, the growth-promoting effect of endogenous ABA also became clear during the characterization of the gin1/aba2 mutant phenotypes [12,19]. Thus, the phenotypic plasticity of plant growth and

![Figure 3: Model for glucose connections to abscisic acid (ABA) and ethylene in Arabidopsis](http://plants.trends.com)
development is the manifestation of a signalling network that integrates intrinsic and extrinsic signals, including nutrients, plant hormones and physical factors. Another layer of complexity is added when different responses are stimulated by the same signals at different concentrations. These diverse plant phenotypes reveal the remarkably broad range of growth plasticity stored in the plant genome.

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