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REVIEW PAPER

Sugar signals and the control of plant growth and development

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Abstract

Sugars have a central regulatory function in steering plant growth. This review focuses on information presented in the past 2 years on key players in sugar-mediated plant growth regulation, with emphasis on trehalose 6-phosphate, target of rapamycin kinase, and Snf1-related kinase 1 regulatory systems. The regulation of protein synthesis by sugars is fundamental to plant growth control, and recent advances in our understanding of the regulation of translation by sugars will be discussed.

Key words: Energy stress, plant growth, protein translation, ribosome, S1-group bZIP, SnRK1, sugar signalling, TOR kinase, trehalose 6-phosphate.

Introduction

In plants, growth usually is an irreversible increase in size involving cell division and cell elongation. Plant growth is important in the competition with neighbours for often scarce resources such as nutrients and light. Complex molecular networks coordinate cell division and cell expansion, resulting in growth. These networks must continuously adapt to an ever-changing environment (Gonzalez *et al.*, 2012; Powell and Lenhard, 2012). Growth is a highly energy-demanding process that is tightly linked to the diurnal cycle and is restricted by unfavourable conditions. Starch synthesis and degradation are diurnally regulated such that an optimal carbohydrate balance is maintained during both day and night, and energy stress can be avoided (Smith and Stitt, 2007; Stitt and Zeeman, 2012). However, plants experience various biotic and abiotic stresses that often lead to energy and nutrient stress, for example during insect feeding, pathogen infection, submergence-induced hypoxia, and osmotic or oxidative stress. Energy stress in plants leads to growth alteration and can readily be experimentally induced by extended darkness (Rolland *et al.*, 2006; Baena-González and Sheen, 2008; Baena-González, 2010). Adaptations in response to energy stress involve inhibition of growth and development to preserve vital resources. Sugars serve as key components reflecting the plant's energy status and, therefore, the ability to continuously sense sugar levels and control energy status is key to survival. All eukaryotes harbour two important regulatory networks to respond to changes in nutrient and energy status. The plant Snf1-related kinase 1 (SnRK1) homologue of the animal AMP-activated protein kinase (AMPK) and yeast sucrose non-fermenting 1 (SNF1) kinase, and the plant target of rapamycin (TOR) kinase are central regulators that link growth and development to carbon nutrient and energy status (Fig. 1) (Smeekens *et al.*, 2010; Robaglia *et al.*, 2012). While plant TOR promotes growth in response to high sugar levels (Deprost *et al.*, 2007), SnRK1 is particularly active upon sugar deprivation. These systems are active throughout the life cycle and are essential for plant survival under stress conditions (Baena-González, 2010). TOR and SnRK1 activities are modulated by the plant's sugar status, which is sensed by several signalling processes and molecules. For example, high sucrose and trehalose-6-phosphate (T6P) levels are sensed by as yet unidentified mechanisms and

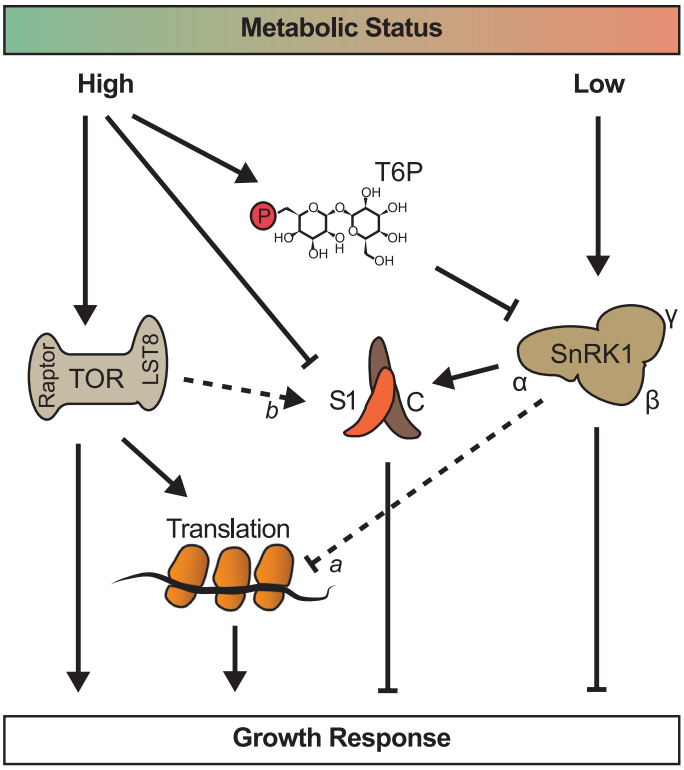

Fig. 1. Sugar signalling pathways interconnect and control plant growth. The cellular metabolic status is an important factor in regulating vegetative growth. Nutrient stress activates SnRK1, resulting in an inhibition of growth. The C/S1-bZIP transcription factor network is implicated in the regulation of SnRK1 target genes. A high metabolic status is reflected by sucrose availability, which is correlated with plant T6P levels. Under these conditions, T6P inhibits SnRK1, and the active TOR kinase stimulates translation and growth. Sucrose inhibits the translation of S1-group *bZIP* mRNAs. The repression of ribosomal protein gene expression by SnRK1 (Baena-González *et al.*, 2007) inhibits translation (a). The role of TOR in S1-group *bZIP* mRNA translation (b) is illustrated by the reduced polysome loading of *bZIP11* mRNA in *TOR*-RNAi plants (Schepetilnikov *et al.*, 2013), indicating that TOR is important for S1-group bZIP protein synthesis.

signal a cellular sugar abundance status (Rolland *et al.*, 2006; Wind *et al.*, 2010; Eveland and Jackson, 2012; Tognetti *et al.*, 2013). T6P is essential for plant growth and, in *Arabidopsis thaliana*, T6P and sucrose levels are correlated (Lunn *et al.*, 2006). Sucrose is a dominant regulator of growth processes in plants, but sucrose sensor proteins remain to be identified. *Arabidopsis thaliana* HEXOKINASE1 (HXK1) is a glucose-phosphorylating enzyme that also serves as a glucose-sensing protein. The role of HXK1 as a glucose sensor and signal transducer is independent of its enzymatic function (Moore *et al.*, 2003).

Cellular sugar signalling must be integrated with other growth regulatory pathways, in particular light and phytohormone signalling (Rolland *et al.*, 2006; Eveland and Jackson, 2012). Recent studies describe the induction by sucrose of phytochrome-interacting factors (PIFs) that mediate the effect of sucrose on *Arabidopsis* hypocotyl elongation (Liu *et al.*, 2011; Stewart *et al.*, 2011). PIFs are important growth regulators that respond to phytohormones such as auxin and gibberellin (Leivar and Quail, 2011). Sugars induce PIFs, while light-activated phytochromes promote PIF degradation. PIF genes are expressed in a diurnal rhythm, and the circadian clock evening complex represses PIF gene expression during early evening. This repression is lifted later in the night. Clock-regulated PIF protein accumulation in plants partly explains the diurnal rhythm of hypocotyl elongation (Nagel and Kay, 2012; Shin *et al.*, 2013). The circadian clock is an important regulator of carbohydrate metabolism as well (Smith and Stitt, 2007), and, conversely, the expression of clock genes is responsive to sugars (Haydon *et al.*, 2013*a*, *b*). Sugar signalling is tightly linked to the circadian regulation of gene expression (Bolouri Moghaddam and Van den Ende, 2013; Hart, 2013). Many genes regulated by sugars overlap with clock-regulated genes and their expression is controlled by the diurnal sugar level patterns (Bläsing *et al.*, 2005).

Sugars and plant growth

The effects of sugars on plant growth and development are diverse. Transcript levels of thousands of genes respond to changing sugar levels (Price *et al.*, 2004; Bläsing *et al.*, 2005; Osuna *et al.*, 2007; Usadel *et al.*, 2008). Plant growth involves cell volume increase, cell division, and developmental programmes that specify tissue and organ identity. The molecular networks driving cell division and expansion largely rely on the availability of carbohydrates to provide energy and biomass. Cyclins are important regulators of the cell cycle and promote the generation of new cells. Sugars induce the expression of cyclins CYCD2 and CYCD3 in starved *Arabidopsis* cell cultures and in seedlings, thereby promoting cell cycle progression (Riou-Khamlichi *et al.*, 2000). Transition to the cell expansion phase is initiated by the inactivation of cyclins and cyclin-dependent kinases (CDKs) (Komaki and Sugimoto, 2012). Cell expansion is mainly turgor driven but little is known about the controlling molecular mechanisms. Interestingly, ectopic expression of CDK inhibiting *KIP-RELATED PROTEIN* (*KRP*) genes in *Arabidopsis* suggests that a decrease in cell division rate may be partly compensated by increased cell expansion, implying an important role for cell expansion in plant growth control (Bemis and Torii, 2007). Auxin promotes cell proliferation and inhibits cell expansion (Powell and Lenhard, 2012). Auxin is central to plant growth and development, and operates mainly via modulating the activity of auxin response factors (ARFs) that control downstream auxin-regulated genes. Auxin metabolism and transport are modulated by sugars (Ljung, 2013). Sucrose induces auxin levels in a PIF-dependent way in *Arabidopsis* (Lilley *et al.*, 2012; Sairanen *et al.*, 2012) and sucrose also induces auxin transport and signal transduction in *Arabidopsis* hypocotyls (Stokes *et al.*, 2013).

Sugar levels are both temporally and spatially regulated in the plant. Sugar-metabolizing enzymes and sugar transporters are important in regulating plant growth and development, such as in the vegetative to generative phase transition (Rolland *et al.*, 2006). The spatial and temporal expression

of sucrose transporters and enzymes involved in sucrose hydrolysis directs sink–source allocation and intracellular distribution of different sugars and sugar-derived metabolites, and affects growth (Tiessen and Padilla-Chacon, 2012). Plant SWEET proteins facilitate transport of neutral sugars such as sucrose, glucose, and fructose at both the organismal and the cellular level (Chen *et al.*, 2012; Klemens *et al.*, 2013; Yuan and Wang, 2013). The importance of spatial and temporal regulation of sugar accumulation is evident in meristematic tissues with their complex organization and different cell types, which requires a sophisticated sugar distribution process for directing development (Francis and Halford, 2006). Different sugars can have different regulatory roles in physiological processes, and the developmental stage of the plant further determines the response to sugars (Rolland *et al.*, 2006; Eveland and Jackson, 2012; Tognetti *et al.*, 2013). Recently, it was observed that glucose facilitates the juvenile to adult phase change in *Arabidopsis* by repressing microRNA (miRNA) 156 expression. In the juvenile phase, miRNA156 expression inhibits accumulation of *SQUAMOSA* promoter binding protein-like (SPL) transcription factors. As juvenile plants age, sugars produced by photosynthesis accumulate and gradually repress miRNA156 expression, resulting in increasing levels of SPL, which promote the transition to the adult phase (Proveniers, 2013; Yang *et al.*, 2013; Yu *et al.*, 2013). Consequently, mutants in sugar signalling or starch metabolism display an altered juvenile phase (Matsoukas *et al.*, 2013).

At high concentrations, sugars can induce meristem quiescence as observed in the arrest of development of seedlings germinated on high sugar levels. Abscisic acid (ABA) biosynthesis (*aba*) and signalling (*abscisic acid insensitive* or *abi*) mutants do not display arrested growth on high (6%) glucose levels, whereas ethylene-insensitive mutants showed glucose hypersensitivity (for a review, see Rolland *et al.*, 2006). Recently, it was established that glucose arrests *Arabidopsis* seedling development at much lower concentrations than previously reported. A low nitrate level in the growth medium renders plants more responsive to glucose-induced growth arrest, with 2% glucose having the same inhibitory effect on low nitrate as 6% glucose on high nitrate medium, supporting the notion that nitrate has an inhibitory effect on sugar-mediated signalling. In the absence of nitrate, low glucose level signalling is HXK1 mediated but is independent of ABA and ethylene signalling (Cho *et al.*, 2010). *ABI* genes are important for the inhibitory effects of sugars on seedling establishment (León *et al.*, 2012; Wind *et al.*, 2013). At low glucose levels, *ABI4* and *ABI5* gene expression is HXK1 dependent, while at high glucose levels these genes are expressed independently of HXK1. In *hxk1* and *aba* mutants grown at low glucose levels, *ABI4* and *ABI5* induction was lost, as in mutants with a constitutive ethylene response (Cho *et al.*, 2010). Repression of *Arabidopsis* seedling establishment at high glucose levels involves the SnRK1 complex and in SnRK1-inactive plants inhibition of growth is absent. In these SnRK1-inactive plants, glucose fails to repress the glucose signalling marker gene *CHLOROPHYLL A/B-BINDING PROTEIN2* (*CAB2*) (Cho *et al.*, 2012).

SnRK1 activation and cell survival

SnRK1 is active under low energy conditions, mainly to repress biosynthetic processes and plant growth (Baena-González *et al.*, 2007; Polge and Thomas, 2007; Halford and Hey, 2009; Baena-González, 2010; Ghillebert *et al.*, 2011). Knowledge of the regulation of SnRK1 and its target processes has increased substantially in recent years. Mammalian, yeast, and plant AMPK, SNF1, and SnRK1, respectively, are heterotrimeric complexes with a catalytic α-subunit, and regulatory β- and γ-subunits. The AMP/ATP ratio allosterically regulates mammalian AMPK, but the plant SnRK1 is instead regulated by sugar phosphates (Ghillebert *et al.*, 2011). SnRK1 activity is repressed by glucose-6-phosphate (G6P) and glucose-1-phosphate (G1P), and T6P also inhibits SnRK1 at physiological concentrations (Zhang *et al.*, 2009; O'Hara *et al.*, 2012; Nunes *et al.*, 2013*a*). Sucrose promotes the accumulation of T6P, thereby inhibiting SnRK1 activity (Fig. 1). Generally, SnRK1 activity is repressed when sufficient sugar is available but, depending on the tissue or developmental phase studied, sucrose might have an SnRK1-stimulating role as well (Baena-González, 2010). Recently, miRNAs were implicated in the regulation of SnRK1 target genes. An *Arabidopsis* mutant (*dcl1-9*) compromised in miRNA synthesis is unable to induce a transcriptional response to dark-induced stress conditions. The genes identified as being co-regulated by SnRK1 and miRNAs particularly encode ribosomal proteins, and also proteins involved in amino acid and sugar signalling (Confraria *et al.*, 2013).

In mammals, AMPK acts as a repressor of growth-promoting TOR signalling (Wullschleger *et al.*, 2006) and has a role in cell cycle regulation by phosphorylation of the CDK/cyclin inhibitor $p27^{KIP1}$, resulting in inhibition of cellular proliferation. Phosphorylation stabilizes $p27^{KIP1}$, resulting in cell cycle arrest, apoptosis, and autophagy (Liang *et al.*, 2007; Short *et al.*, 2010). Similarly, AtSnRK1 phosphorylates plant KRP6 and KRP7 proteins that are homologues of the mammalian $p27^{KIP1}$ CDK/cyclin inhibitor. In the nucleus, SnRK1 interacts with and phosphorylates KRP6, but, remarkably, it appears that KRP6 phosphorylation prevents binding to CDK/cyclin and therefore allows cell cycle progression. Such SnRK1-mediated cell cycle progression contradicts the view of SnRK1 as an inhibitor of growth under stress conditions (Guérinier *et al.*, 2013). Clearly, the connection to plant SnRK1 and TOR signalling and the role of SnRK1 in controlling plant growth and development needs further clarification.

In *Arabidopsis*, SnRK1 affects phase transitions as well. Overexpression of the SnRK1 catalytic subunit KIN10 in plants results in late flowering and defects in the formation of siliques and cotyledons. The KIN10 overexpression phenotype is rescued by introduction of the *fus3* mutation. FUS3 and KIN10 proteins interact *in vivo* and FUS3 is stabilized by KIN10-mediated phosphorylation (Tsai and Gazzarrini, 2012). Also ABA stabilizes the FUS3 protein, which was shown to be involved in phase change control (Gazzarrini *et al.*, 2004). Possibly, the ABA effect is mediated by SnRK1 as phosphatases involved in ABA signalling have been shown

to inhibit SnRK1 activity by dephosphorylation of SnRK1 (Rodrigues *et al.*, 2013). KIN10 interacts with cyclin-dependent kinase E1 (CDKE1), identified as a regulator of the mitochondrial ALTERNATIVE OXIDASE 1A and essential for responding to mitochondrial stress signals. Thus, the nuclear-localized CDKE1 links mitochondrial stress signals to growth regulation, probably through its interaction with SnRK1 (Ng *et al.*, 2013).

The essential but enigmatic T6P signalling molecule

Trehalose-6-phosphate synthase1 (TPS1) converts G6P and UDP-glucose (UDPG) to the growth signalling molecule T6P. Metabolism of T6P by trehalose-6-phosphate phosphatase (TPP) yields trehalose, which is hydrolysed to glucose by Trehalase1. T6P levels in *Arabidopsis* are correlated to those of sucrose, but cellular T6P levels are in the low micromolar range (Paul *et al.*, 2008). The TPS1 enzyme and its T6P product are essential for growth, and the *Arabidopsis tps1* mutant is embryo lethal. T6P is essential for vegetative and generative growth, and in *tps1*, floral transition is impaired (van Dijken *et al.*, 2004). In *tps1*, sucrose and starch accumulate, and cell division is halted (Gómez *et al.*, 2006). *tps1* reduced function mutant alleles are hypersensitive to ABA, and the decrease in T6P levels in these *tps1* mutants correlates with ABA sensitivity (Gómez *et al.*, 2010). T6P signals a sugar abundance state and, interestingly, T6P inhibits the low sugar-induced SnRK1 activity, thereby allowing growth and development (Fig. 1) (Zhang *et al.*, 2009; O'Hara *et al.*, 2012; Schluepmann *et al.*, 2012). Short-term sink limitations induced by cold treatment or nitrogen deprivation inhibit growth and lead to an accumulation of sucrose and T6P in *Arabidopsis*. SnRK1 marker gene expression correlates with T6P content and confirms T6P as an SnRK1 inhibitor. Interestingly, in KIN10-overexpressing plants and in plants impaired in T6P accumulation, the immediate growth recovery response upon relief of growth restriction is absent. The strong correlation between sucrose and T6P content in stressed plants suggests that T6P/SnRK1 signalling, in response to sucrose accumulation, primes plants for this growth recovery response (Nunes *et al.*, 2013*b*). The opposite roles of SnRK1 and T6P in growth regulation also hold for the regulation of senescence. T6P accumulation in leaves is associated with the onset of senescence, while senescence is delayed in KIN10-overexpressing plants and in plants with reduced T6P levels (Wingler *et al.*, 2012). T6P levels in *Arabidopsis*, like those of sucrose, follow a diurnal rhythm (Pal *et al.*, 2013; Wahl *et al.*, 2013). Sugar signals and the circadian clock are part of a complex network that controls floral transition. Central in this network is the regulation of the florigen FLOWERING LOCUS T (FT) mobile protein produced in leaves. T6P has emerged as a major regulator linking sugar status and diurnal rhythm to FT-mediated floral transition. In shoot apical meristems, *SPL* promotes floral transition but *SPL* expression is inhibited by miRNA156, which reduces *SPL* mRNA expression. T6P inhibits miRNA156 expression, allowing SPL to accumulate and promote floral transition (Wahl *et al.*, 2013). Together with the previously discussed stimulation of the juvenile to adult phase change by glucose, T6P also promotes a crucial phase change in *Arabidopsis* through the inhibition of miRNA156.

The key function of TOR in growth regulation

The eukaryotic TOR complex is central to metabolic and growth control, and regulates ribosome biogenesis and protein synthesis. Mammals have, in contrast to plants, two TOR complex systems (mTORC1 and mTORC2) (Iadevaia *et al.*, 2012; Laplante and Sabatini, 2012). Importantly, mTORC1 is conserved in plants, yeast, and probably all eukaryotic organisms. LST8 is part of the plant TOR complex, and the growth defect displayed by the yeast *lst8* mutation can be complemented by the *Arabidopsis LST8-1* cDNA. *AtLST8-1* is involved in growth and development, as illustrated by the reduced growth phenotype of the *lst8-1* mutant that resembles that of TOR knockdown plants. In yeast two-hybrid assays, LST8-1 interacts with the FRB-kinase domain of AtTOR (Moreau *et al.*, 2012). AtTOR activity is important throughout the entire plant life cycle and is mainly expressed in rapidly proliferating tissues such as meristematic regions and endosperm (Menand *et al.*, 2002), suggesting it to be a central stimulator of growth and development. Simultaneously, AtTOR is a repressor of autophagy (Liu and Bassham, 2010). Knockdown of TOR results in a reduction of plant growth in *Arabidopsis*, accompanied by changes in carbohydrate and amino acid metabolism (Caldana *et al.*, 2013). Sugars generally promote TOR kinase activity (Robaglia *et al.*, 2012; Ren *et al.*, 2012; Dobrenel *et al.*, 2013). Glucose activates TOR and was shown to promote *Arabidopsis* root meristem activity (Xiong *et al.*, 2013). The use of specific TOR inhibitors reduced root growth (Montané and Menand, 2013). Genes regulated by glucose-stimulated TOR signalling overlap with genes regulated by E2F transcription factors, which promote cell cycle progression. Interestingly, TOR directly phosphorylates and activates E2F, apparently bypassing the CDK/cyclin–RETINOBLASTOMA RELATED PROTEIN (RBR) cell cycle control system (Xiong *et al.*, 2013).

AtTOR interacts with REGULATORY-ASSOCIATED PROTEIN OF TOR (RAPTOR) *in vivo* in the regulation of *Arabidopsis* S6 kinase 1 (S6K1) activity (Mahfouz *et al.*, 2006). Mammalian S6K is an mTOR target and phosphorylates the 40S ribosomal protein RPS6 to promote mRNA translation and cell growth (Iadevaia *et al.*, 2012; Laplante and Sabatini, 2012). AtS6K1 also has a role in plant growth control, and cells overexpressing *AtS6K1* are enlarged (Shin *et al.*, 2012). S6K1 represses the cell cycle through phosphorylation of RBR (Henriques *et al.*, 2010). Interestingly, RBR was proposed to regulate the heterotrophy to autotrophy transition in germinating seeds positively and to antagonize the positive effect of sucrose on the cell cycle (Gutzat *et al.*, 2011). However, AtS6K was also reported to be involved in regulating plant growth through auxin-mediated signalling (Turck *et al.*, 2004). Recently, TOR–S6K1 signalling was

associated with reinitiation of translation in *Arabidopsis*. Viral transactivator-viroplasmin (TAV) promotes translation reinitiation following termination of translation of long open reading frames (ORFs) on multicistronic mRNAs. TAV interacts with TOR, thereby stimulating S6K1 phosphorylation and TAV-dependent reinitiation. TOR knockdown plants fail to reinitiate on polycistronic mRNAs (Schepetilnikov *et al.*, 2011). Importantly, TOR–S6K1 also mediate reinitiation of translation of mRNAs that have ORFs in their leader sequences (uORFs). Polysome loading of *bZIP11* and several *ARF* mRNAs is stimulated by TOR, and eukaryotic initiation factor 3h (eIF3h). In protoplasts, TOR phosphorylates eIF3h, thereby stimulating reinitiation of translation following uORFs in *bZIP11* and *ARF* mRNAs (Schepetilnikov *et al.*, 2013). In *Arabidopsis* seedlings, it was previously shown that eIF3h and RPL24 are essential for main ORF translation of *bZIP11* mRNA, which harbours four uORFs (Zhou *et al.*, 2010).

Sugars regulate protein translation

Translation of eukaryotic mRNA is a multistage, highly complex, and extremely energy-demanding process that is under tight control (Warner, 1999). Translation initiation factors orchestrate the binding of the 40S ribosomal subunit to mRNA and mRNA scanning for the AUG start codon. Upon AUG recognition, the 60S subunit joins the complex, followed by translation elongation and termination, which both are actively regulated processes as well (Jackson *et al.*, 2010; Hinnebusch, 2011; Aitken and Lorsch, 2012). Plant growth and development is totally dependent on *de novo* protein synthesis, and regulation of mRNA translation by sugars and environmental signals is essential. Rapid growth requires the massive production of functional ribosomes. Sugars are essential for providing energy and carbon building blocks for RNA and protein biosynthesis. The central role of TOR kinase in biosynthesis and activity of ribosomes, and in mRNA translation is illustrated by reduced polysome loading in *AtTOR*-RNAi (RNA interference) plants (Deprost *et al.*, 2007). *Arabidopsis rps6* mutants display a phenotype comparable with that of TOR knockdown plants (Ren *et al.*, 2012). SnRK1 activity so far has not been directly linked to mRNA translation, although SnRK1 was found to repress ribosomal protein gene expression (Baena-González *et al.*, 2007). Mammalian AMPK regulates protein synthesis through inhibiting the translation elongation factor eEF2 (Leprivier *et al.*, 2013). In *Arabidopsis*, a direct link between translational control and sugar signalling is suggested by the observation of glucose hypersensitivity of seeds overexpressing *eukaryotic RELEASE FACTOR1-2* (*eRF1-2*), illustrated by reduced germination on glucose (X. Zhou *et al.*, 2010). This finding proposes a role for translational control in early seedling development, in addition to the signalling pathways of sugars and phytohormones. eRF1-1 knockdown plants are small and display reduced radial cell division, and internode elongation (Petsch and Mylne, 2005). Polysome loading of *Arabidopsis* transcripts is diurnally regulated and promoted in the light, while ribosome abundance is stable throughout the day/night cycle. Interestingly, cytosolic mRNA polysome loading correlates with rosette sucrose content. Dark and extended night conditions significantly reduce mRNA polysome loading and reduce the rate of *de novo* protein synthesis (Pal *et al.*, 2013). In addition to sugars, ribosome biogenesis, polysome loading, and mRNA translation are affected by other signalling systems such as the clock, phytohormones, and phytochromes (Rosado *et al.*, 2010; Paik *et al.*, 2012; Jouffe *et al.*, 2013).

De novo ribosome and protein synthesis correlate well with growth, and the functions of ribosomes and mRNA translation in growth regulation have been reviewed recently. Also, roles for ribosomal protein paralogues in the translation of specific mRNAs are discussed (Horiguchi *et al.*, 2012). Ribosome biosynthesis, mRNA polysome loading, and regulation of translational activity all determine protein synthesis capacity, and these events are controlled by plant endogenous and environmental signals. The nearly 80 ribosomal proteins of *Arabidopsis* are encoded by multiple paralogous genes and are all expressed, allowing tremendous heterogeneity of ribosome protein composition (Giavalisco *et al.*, 2005; Hummel *et al.*, 2012; Xue and Barna, 2012). Sucrose is one factor that affects the ribosomal protein paralogue composition of ribosomes, which is independent of mRNA levels of these paralogues (Kojima *et al.*, 2007; Hummel *et al.*, 2012). *Arabidopsis* ribosomal proteins from leaves are differentially phosphorylated during the day/night cycle and the phosphorylation of the RPS6 protein at Ser231 and Ser240 increases in the light (Turkina *et al.*, 2011). Newly developed methods to study translational control of gene expression by means of sequencing will further emphasize the importance of translational control (Juntawong *et al.*, 2013; Liu *et al.*, 2013).

The sucrose-controlled C/S1-bZIP regulatory network

Sucrose-induced repression of translation (SIRT) of the S1-group bZIP transcription factors was initially identified in bZIP11 and was later confirmed for all five *Arabidopsis* S1-group members (Rook *et al.*, 1998; Wiese *et al.*, 2005; Weltmeier *et al.*, 2009). The leader sequences of S1 mRNAs encode a conserved uORF that promotes ribosome stalling when sucrose accumulates (Wiese *et al.*, 2004; Hummel *et al.*, 2009; Rahmani *et al.*, 2009; Thalor *et al.*, 2012). S1-group bZIPs preferentially heterodimerize with C-group bZIPs to regulate downstream genes (Ehlert *et al.*, 2006; Weltmeier *et al.*, 2009), including genes involved in amino acid metabolism. C/S1-group bZIPs are responsive to different sugar signalling systems and are powerful regulators of metabolism. Transcriptional activity of S1-group bZIPs is greatly enhanced by KIN10/11 co-expression (Fig. 1), suggesting that the SnRK1 transcriptional response is, at least partially, mediated through S1-bZIPs (Baena-González *et al.*, 2007). The S1-group bZIP1, bZIP11, and bZIP53 proteins are involved in metabolic reprogramming in response to low energy signals (Dietrich *et al.*, 2011; Ma *et al.*, 2011). Overexpression of

S1-bZIPs from *Arabidopsis* (*bZIP11* and *bZIP53*) or tobacco (*TBZ17*) results in severely reduced growth of the transgenic lines (Hanson *et al.*, 2008; Dietrich *et al.*, 2011; Thalor *et al.*, 2012), possibly due to inability to metabolize sugars as suggested by the accumulation of sucrose and hexose phosphates in these lines and the observation that added sugars do not rescue the growth phenotype (Ma *et al.*, 2011; Thalor *et al.*, 2012). Plants overexpressing *bZIP11* have increased expression of *TPP5*, *TPP6*, and *TREHALASE1*, and reduced levels of the growth regulator T6P. Interestingly, constitutive *bZIP11* expression in seedlings enables root growth on otherwise inhibitory trehalose levels in the growth medium (Ma *et al.*, 2011). C/S1-bZIPs are important for seed development where they are involved in regulating expression of seed maturation genes (Alonso *et al.*, 2009). Seeds of the S1-group *atbzip44* mutant show slower germination, probably due to the reduced expression of the mannanase-encoding *AtMAN7* gene in this mutant (Iglesias-Fernández *et al.*, 2013). Light and nitrogen regulation of several gene clusters is affected by the *atbzip1* mutation (Obertello *et al.*, 2010). *AtbZIP1* is a regulator of many sugar-responsive genes and is repressed by glucose through the HXK1 signalling pathway (Kang *et al.*, 2010).

Concluding remarks

Sugar signals are central in determining plant growth and development. Sugar signals connect with other signalling networks in the control of cell proliferation and expansion. Understanding the fundamental molecular mechanisms involved in sugar signalling will provide opportunities for improving general plant growth and biomass accumulation, and growth of harvestable organs. Sugar signals regulate developmental processes such as floral transition that are crucially important for plant yield as well. The repressor of flowering miRNA156 is regulated by sugars via T6P signalling, and overexpression of miRNA156 in switchgrass results in increased biomass (Fu *et al.*, 2012). Many challenges lay ahead in sugar signal transduction research, including the identification of additional sugar sensors, particularly those of sucrose and fructose (Cho and Yoo, 2011; Li *et al.*, 2011). Ongoing research on the regulation of carbon and nitrogen metabolism in plants will advance breeding of resource-efficient crops, as illustrated by a recent study on the correlation between metabolic traits and plant biomass in different *Arabidopsis* accessions (Sulpice *et al.*, 2013). The plant TOR and SnRK1 kinases are pivotal in regulating growth in response to carbon availability (Fig. 1). Plant TOR and SnRK1 investigations benefit from research in yeast and mammalian systems, but much remains to be discovered on the plant-specific regulatory networks that control TOR and SnRK1. Involvement of TOR and SnRK1 in plant growth is linked to regulation of protein synthesis capacity via effects on ribosome biosynthesis and mRNA polysome loading. The role of T6P as a crucial controlling molecule for plant growth and development became apparent >10 years ago, and recent mechanistic studies revealed the role of T6P as an inhibitor of SnRK1 (Zhang *et al.*, 2009), and a key regulator of floral transition (Wahl *et al.*, 2013). Components of sugar signalling networks have diverse functions throughout the plant life cycle, and uncovering these functions and their interactions with other signalling pathways presents a formidable challenge.

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