Sugars and the speed of life—Metabolic signals that determine plant growth, development and death

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Abstract

Plant growth and development depend on the availability of carbohydrates synthesised in photosynthesis (source activity) and utilisation of these carbohydrates for growth (sink activity). External conditions, such as temperature, nutrient availability and stress, can affect source as well as sink activity. Optimal utilisation of resources is under circadian clock control. This molecular timekeeper ensures that growth responses are adjusted to different photoperiod and temperature settings by modulating starch accumulation and degradation accordingly. For example, during the night, starch degradation is required to provide sugars for growth. Under favourable growth conditions, high sugar availability stimulates growth and development, resulting in an overall accelerated life cycle of annual plants. Key signalling components include trehalose-6-phosphate (Tre6P), which reflects sucrose availability and stimulates growth and branching when the conditions are favourable. Under sink limitation, Tre6P does, however, inhibit night-time starch degradation. Tre6P interacts with Sucrose-non-fermenting1-Related Kinase1 (SnRK1), a protein kinase that inhibits growth under starvation and stress conditions and delays development (including flowering and senescence). Tre6P inhibits SnRK1 activity, but SnRK1 increases the Tre6P to sucrose ratio under favourable conditions. Alongside Tre6P, Target of Rapamycin (TOR) stimulates processes such as protein synthesis and growth when sugar availability is high. In annual plants, an accelerated life cycle results in early leaf and plant senescence, thus shortening the lifespan. While the availability of carbohydrates in the form of sucrose and other sugars also plays an important role in seasonal life cycle events (phenology) of perennial plants, the sugar signalling pathways in perennials are less well understood.

1 | INTRODUCTION

Plant growth depends on the availability of external resources (water, nutrients, CO2, light), the use of these resources in photosynthesis (source activity), and the subsequent utilisation of the products of photosynthesis for growth (sink activity). In addition to carbohydrates produced in photosynthesis, growth requires anabolic pathways such as the synthesis of amino acids, proteins, membrane lipids and cell wall components. The supply of carbon for these growth-related pathways is tightly controlled in the diurnal cycle through the synthesis and breakdown of starch (Smith & Zeeman, 2020). In Arabidopsis accessions, growth is negatively correlated with leaf starch content at the end of the night (Cross et al., 2006). For monocarpic perennial plants, it has been shown that slow-growing species with lower
mortalities store more carbohydrates and amino acids in their roots (Atkinson et al., 2012). These findings suggest that carbon utilisation can be growth limiting and that slow-growing plant genotypes conserve resources for survival, e.g. to cope with stress, resulting in a longer lifespan.

Such metabolic adaptations reflect the life-history strategies of plants which can be classified along a fast-slow continuum: Fast-growing species have short life cycles, reach reproductive maturity early and have higher adult mortalities (Franco & Silvertown, 1996; Salguero-Gomez et al., 2016). Leaf traits link this fast-slow continuum to the leaf economics spectrum (Wright et al., 2004). Short-lived species have a higher specific leaf area (SLA; leaf area divided by leaf dry mass) than in long-lived species (Salguero-Gomez, 2017), indicating less investment in structures that enhance leaf durability. While originally described for different plant species, the relationship between the fast-slow continuum and the leaf economics spectrum has recently been confirmed for Arabidopsis accessions (Sartori et al., 2019), demonstrating intraspecific variation of life-history strategies.

Large but thin leaves with high SLA enable fast growth but have a shorter lifespan, i.e. earlier senescence. Leaf senescence is an important nutrient mobilisation process (Havé et al., 2017). In monocarpic annual plants, including Arabidopsis, which die after reproduction, leaf senescence recycles nutrients such as nitrogen to the reproductive organs. Intraspecific variation among Arabidopsis accessions revealed a genetic link between flowering, leaf senescence and fecundity, with early-flowering and senescing genotypes investing more in reproduction but having short lifespans (Levey & Wingler, 2005; May et al., 2017). Quantitative trait loci for sugar-induced senescence co-localised with the genes Flowering Locus C (FLC) and Frigid (FRI), which are involved in the vernalisation-dependent flowering pathway (Wingler et al., 2010). In addition, expression of senescence-associated genes showed a positive correlation with expression of the flowering genes Flowering Locus T (FT) and Suppressor of Overexpression of Constans1 (SOC1) among different Arabidopsis accessions (Wingler et al., 2010). This provides evidence for a genetic basis of the interaction between flowering and senescence.

Developmental transitions (including flowering and senescence) and the growth of plants are regulated by sugar signalling pathways (e.g. Baena-González & Hanson, 2017; Baena-González & Lunn, 2020; Caldana et al., 2019; Wingler, 2018) which coordinate the perception of environmental conditions with the availability of internal carbohydrates. It is, therefore, likely that sugar-dependent regulation of key developmental processes and of metabolic pathways that determine growth underlies the overall life histories of plants. In plant sugar-signalling pathways, metabolites such as sucrose, glucose and trehalose-6-phosphate (Tre6P) serve as signals for high carbon availability, while kinases such as hexokinase-1 (HXK), sucrose-non-fermenting1-related kinase1 (SnRK1) and target of rapamycin (TOR) act as sensors mediating the responses. While we have a good understanding of the regulatory processes that control metabolism and growth in Arabidopsis, the knowledge for other species, especially perennials, is still limited.

2 SUGARS AS SIGNALS DETERMINING THE SPEED OF LIFE

Metabolic pathways which underlie plant growth, such as starch and protein metabolism (e.g. Cross et al., 2006; Ishihara et al., 2017; Sulpice et al., 2009), are regulated by sugar signals that indicate the availability of carbon (Figure 1). High carbon availability in the form of sucrose results in a rise in the content of Tre6P (Nunes et al., 2013; Yadav et al., 2014), an important signalling molecule in plants (for recent reviews see Baena-González & Lunn, 2020; Paul et al., 2020; Fichtner & Lunn, 2021). Tre6P is a metabolite of the trehalose biosynthetic pathway: Tre6P synthase (TPS) catalyses the formation of Tre6P from UDP-glucose and glucose-6-phosphate, while Tre6P phosphatase (TPP) catalyses the conversion of Tre6P to trehalose. Trehalose can subsequently be hydrolysed into two glucose molecules by trehalase. Although the mechanism underlying the increase in Tre6P in response to sucrose has not been identified, it was demonstrated that protein synthesis is required for the rise in Tre6P (Yadav et al., 2014). Feedback regulation of sucrose synthesis by Tre6P results in a Tre6P-sucrose nexus that controls sucrose content in interaction with the circadian clock (dos Anjos et al., 2018; Yadav et al., 2014).

Tre6P induces the expression of genes involved in a large range of growth-related biosynthetic pathways, including amino acid, protein and nucleotide synthesis (Zhang et al., 2009). Generally associated with enhanced growth (Schlüpmann et al., 2003; Sulpice et al., 2014), Tre6P is e.g. required for the growth stimulation after the release of sink limitation in response to transfer from cold to warm conditions (Nunes et al., 2013). In addition, Tre6P regulates the metabolism of starch which is the main buffer of carbohydrates supply in the diel cycle (Smith & Zeeman, 2020). Increasing Tre6P

![FIGURE 1](image-url)
by induced expression of a bacterial TPS gene (otsA) resulted in starch accumulation in the leaves through inhibition of starch breakdown at night (Martins et al., 2013). Under sink-limiting conditions with high starch accumulation during the day, sucrose and Tre6P accumulate, and Tre6P inhibits starch breakdown, resulting in incomplete starch mobilisation (dos Anjos et al., 2018). In contrast, under source-limitation, when starch content at the end of the day is low, sucrose and Tre6P contents are also low, and the effect of Tre6P on starch breakdown is weak.

The interactions between Tre6P and starch degradation in determining growth are not straightforward. To promote growth, Tre6P would be expected to stimulate starch utilisation by night-time degradation (Cross et al., 2006; Sulpice et al., 2009) instead of inhibiting starch degradation. However, the growth promotion by Tre6P depends on a combination of high carbon availability and sufficiently strong sink activity, whereas Tre6P can even be growth inhibitory when there is an imbalance of source strength and sink demand (O’Hara et al., 2013; Schluemmann et al., 2012). When growth is reduced and sink demand is limited, e.g. under low temperature or low nitrogen conditions, sucrose, Tre6P and starch accumulate. Once the conditions become more favourable, Tre6P is required for the resumption of growth (Nunes et al., 2013) and Tre6P content decreases (Figure 1). It is also important to differentiate between the function of Tre6P in leaf source tissues and its role in promoting the growth and activity of sink organs (Baena-González & Lunn, 2020; Paul et al., 2020), such as filling of the wheat grain (Griffiths et al., 2016; Martínez-Barajas et al., 2011).

In growing tissues, but not in mature leaves, Tre6P inhibits the catalytic activity of Sucrose-non-fermenting1-Related Kinase1 (SnRK1; Zhang et al., 2009), a protein kinase that plays a key role in starvation signalling and reduces growth under nutrient-limiting conditions (Baena-González et al., 2007; Baena-González & Sheen, 2008; Jamsheer et al., 2021). In addition to directly binding to SnRK1, Tre6P weakens the interaction of SnRK1 with geminin rep-interacting kinase1 (GRIK1), which phosphorylates and activates SnRK1 (Zhai et al., 2018). This interaction stimulates fatty acid synthesis (Zhai et al., 2018) and temperature-responsive hypocotyl elongation (Hwang et al., 2019).

Fluctuations of the Tre6P content in the diel cycle may also underlie changes in SnRK1 activity, with high Tre6P at the end of the day repressing SnRK1 activity and low Tre6P at the end of the day stimulating it (Peixoto et al., 2021). Furthermore, manipulation of SnRK1 in Arabidopsis resulted in an altered Tre6P/sucrose relationship. Although diel cycles of Tre6P and sucrose are maintained, SnRK1 overexpression increases the Tre6P/sucrose ratio, whereas mutation reduces it (Peixoto et al., 2021). However, the mechanism underlying this interaction remains unresolved as the abundance of TPS1, the principal catalytically active TPS, was not altered accordingly. This control of sucrose homeostasis in the diel cycle was described for favourable growing conditions (Peixoto et al., 2021), suggesting that SnRK1 also has a function under non-stress conditions. This is in agreement with its role in normal developmental processes, e.g. seed storage compound mobilisation during seedling establishment (Henninger et al., 2022).

In line with its central function in regulating plant metabolism and development, the regulation of SnRK1 is complex. In addition to inhibition by Tre6P, SnRK1 can be regulated by myristoylation, which restricts its nuclear localisation and function (Ramon et al., 2019). Other processes that regulate SnRK1, including (auto-)phosphorylation, sumoylation, ubiquitination and maltose-binding, are described by Baena-González and Lunn (2020).

SnRK1 forms part of an energy signalling network that includes abscisic acid (ABA), Target of Rapamycin (TOR) and SnRK2 (Crepin & Rolland, 2019). It acts as a starvation signal by inhibiting the TOR kinase through interaction with and phosphorylation of Regulatory-Associated Protein of TOR (RAPTOR; Nukarinen et al., 2016). This interaction underlies the abscisic acid (ABA) mediated growth inhibition in response to stress (Figure 1). Under stress conditions, when ABA accumulates, the TOR-dependent inactivation of PYLs (Pyrabactin Resistant 1 Likes) is lifted, leading to PP2C (Protein Phosphatase 2C) recruitment and SnRK2 phosphorylation of RAPTOR. This results in reduced TOR complex activity and consequently growth inhibition (Wang et al., 2018). The interaction between SnRK2 and TOR under ABA-mediated stress responses was further elucidated and shown to rely on the inhibition of SnRK1 activity (Belda-Palazón et al., 2020). Under optimal, ABA-free conditions, SnRK2-mediated complexes can sequester SnRK1 and prevent its inhibition of TOR, allowing growth to occur. However, under stress conditions, the reverse will happen with activation of SnRK1, TOR pathway inhibition and growth arrest. Besides stress, other inputs control TOR pathway activity, such as nutrient availability, sucrose and glucose levels. Light signals and the circadian clock are also critical for integrating TOR-mediated growth regulation and resource availability (Caldana et al., 2019).

Within the TOR complex, RAPTOR provides substrate specificity by binding to certain targets and promoting their phosphorylation by TOR (Mahfouz et al., 2006). In Arabidopsis, although both N- and C-terminal regions of RAPTOR were shown to interact with different substrates, only the N-terminal region can bind to the first 44 amino acids of S6K1 (40S ribosomal protein S6 Kinase 1), the most conserved TOR target (Son et al., 2016). Further dissection of this S6K1 motif led to the identification of 12 amino acids (29-DDVELEFSDVFG-40) critical for the interaction. Although they lack a clear homology with the TOR-signalling (TOS) motif found in animals, their relative position and composition are similar (Son et al., 2017). Point mutations in three of these residues (F35S, F39S and G40A) abolished S6K1 interaction with RAPTOR, suggesting this could be a functional plant TOS motif. Identification of similar consensus sequences in tobacco and soybean S6K1 orthologs strengthens this hypothesis. These findings indicate that, although plant S6Ks have a longer N-terminal region and lack the C-terminal auto-inhibitory domain of their mammalian counterparts (Yaguchi & Kozaki, 2018), their interaction with RAPTOR involves similar domains and motifs, confirming the functional conservation of this TOR-S6K module in eukaryotes.

The identification of other putative TOR downstream effectors such as BZR1 (Zhang et al., 2016) and E2Fa/b (Henriques et al., 2010;
Xiong et al., 2013) further confirms this signalling pathway’s role as a metabolic hub where resource levels are perceived and translated into specific growth responses. For instance, glucose-dependent activation of root and shoot meristems was associated with TOR activation in Arabidopsis (Li et al., 2017; Xiong et al., 2013).

3 | AT THE START LINE—SEED DORMANCY AND GERMINATION

Seed dormancy prevents germination while the seed is still attached to the plant (vivipary) and ensures germination under favourable conditions.ABA is the main signal in establishing dormancy, whereas gibberellic acid (GA) can release dormancy and induce the breakdown of starch by α-amylase for germination. Although the external supply of sugars, such as glucose, at high concentration inhibits germination in Arabidopsis (Dekkers et al., 2004) and rice (Zhu et al., 2009), trehalose metabolism promotes germination in crosstalk with ABA. In Arabidopsis, germination of non-lethal TILLING mutants in the TPS gene AtTPS1 is hypersensitive to ABA (Gómez et al., 2010), while germination is delayed, and ABA content is increased in a rice mutant in the TPS gene OsTPP1 (Wang et al., 2021). The latter suggests that trehalose rather than Tre6P promotes germination. Previously, OsTPP7 was demonstrated to confer anaerobic germination tolerance in rice (Kretzschmar et al., 2015). Tre6P content was not altered, but trehalose content was increased in lines with functional OsTPP7, which could indicate a role of trehalose synthesis. Alternatively, higher Tre6P turnover and/or a decreased Tre6P to sucrose ratio could increase sink strength and starch mobilisation in the presence of a functional OsTPP7 gene (Kretzschmar et al., 2015).

SnRK1 activates α-amylase expression in rice during germination (Lu et al., 2007). A function of SnRK1 in resource mobilisation was also described for Arabidopsis, which in contrast to rice, mainly stores lipids instead of starch in the seeds. An inducible Arabidopsis mutant in the SnRK1 catalytic subunit genes, SnRK1α1 (AKIN10) and SnRK1α2 (AKIN11), showed impaired lipid mobilisation and seedling establishment (Henninger et al., 2022). However, a partial double mutant (snrk1α1−/− snrk2α1−/−) did not show a germination phenotype (Belda-Palazón et al., 2020). In contrast, constitutive overexpression of SnRK1 delays Arabidopsis seed germination and promotes dormancy in interaction with FUS3, as shown by increased FUS3 stability after SnRK1-dependent phosphorylation and precocious germination when the fus3-3 mutation was crossed into the SnRK1 (KIN10) over-expressor (Tsai & Gazzarrini, 2012). For pea, it was shown that antisense repression of SnRK1 results in defects in seed maturation and occasionally precocious germination (Radtchuk et al., 2006). Although generally in line with SnRK1 as a starvation signal, these findings also suggest that the role of SnRK1 in germination may depend on the developmental stage (seed maturation, germination, post-germination elongation). To what extent Tre6P and SnRK1 interact in the regulation of seed dormancy and germination is not known.

Deletion of TOR or double mutations in RAPTOR 1A/1B or the S6K1/S6K2 genes leads to embryo lethality in Arabidopsis (Deprost et al., 2005; Henriques et al., 2010; Menand et al., 2002); and single raptor1B mutants show delayed germination (Menand et al., 2002; Salem et al., 2017). Recently, it was also shown that treatment with the TOR inhibitors rapamycin or torin 1 inhibits wheat seed germination and α-amylose expression, in addition to the GA-induced phosphorylation of the TOR substrate S6K (Smailov et al., 2020). In contrast, Li et al. (2021) found no effect of TOR inhibitors on germination. Instead, the inhibitors delayed the growth of Arabidopsis seedlings, which may be modulated by interactions between ABI5 and S6K2 (Li et al., 2021). Interestingly, a similar inhibition of germination was found in maize seedlings treated with glucose and the TOR inhibitor AZD8055 during the initial 48 h (Díaz-Granados et al., 2020).

4 | TAKING A REST OR BRANCHING OUT? DORMANCY AND BUD OUTGROWTH

Dormancy release resulting in bud outgrowth is regulated in response to sugar availability (Barbier et al., 2015). Tre6P has emerged as an important signal for sugar availability in bud outgrowth during potato sprouting and shoot branching. Potato sprouting was accelerated in potato plants expressing a bacterial TPS in the tubers, whereas expression of a TPS delayed sprouting, probably because of altered cytokinin and GA sensitivity (Debast et al., 2011). The changes in gene expression in the transgenic lines were consistent with inhibition of SnRK1 by Tre6P. Further, starch content was reduced in tubers with increased T6P (Debast et al., 2011), while overexpression of SnRK1 in potato tubers increased starch content (McKibbin et al., 2006). Although in agreement with the opposite roles of Tre6P and SnRK1 (Zhang et al., 2009), the effect on starch accumulation in potato tubers is clearly different from the increased starch accumulation in response to Tre6P described for in leaves (Kolbe et al., 2005; Martins et al., 2013) and more in line with the proposed role of Tre6P in grain filling (Griffiths et al., 2016; Martínez-Barajas et al., 2011), highlighting different functions dependent on plant organ.

Shoot branching is a consequence of the release from axillary bud dormancy, and sugars interact with hormone signalling in the regulation of this process. In etiolated potato stems, sucrose feeding to stems promotes axillary bud outgrowth and branching, in addition to the accumulation of cytokinin (Salam et al., 2021). Both sucrose and cytokinin induce the expression of vacuolar invertase, an enzyme required for sucrose utilisation. Since mutation of the gene for vacuolar invertase repressed bud outgrowth, it was concluded that sucrose-dependent branching is, at least partially, mediated by cytokinin (Salam et al., 2021). Tre6P has been shown to be involved in branching upon decapitation of the shoot apex. While the traditional view was that bud outgrowth in decapitated plants results from depletion of auxin transport from the shoot apex, a more complex picture involving Tre6P-dependent signalling of sucrose availability has emerged (Barbier et al., 2015, 2019). Decapitation of pea shoots leads to an accumulation of Tre6P in the axillary buds and bud outgrowth, but not when plants were also defoliated to reduce sucrose availability (Fichtner et al., 2017).
Shoot architecture is also affected by Tre6P synthesis in intact Arabidopsis plants: transgenic lines with lower Tre6P through constitutive expression of a TPP or Tre6P phosphohydrolase (TPH) show increased apical dominance, whereas plants with increased Tre6P through constitutive expression of a TPS have a bushier inflorescence than wild-type plants (Schluempmann et al., 2003). This was confirmed through bud-specific expression of a TPH, which resulted in reduced branching (Fichtner et al., 2021). In addition, a role of hexokinase-1 (HXK1) signalling in shoot branching in response to sugar availability was shown, with HXK1 stimulating bud outgrowth upstream of cytokinin and strigolactone signalling, as demonstrated by reduced shoot branching and decreased cytokinin content in the HXK1 mutant (gin-2-1), and restoration of branching by treatment of the mutant with cytokinin (Barbier et al., 2021).

In maize, increased apical dominance is an important aspect of domestication. Using mutants, it was shown that the transcription factors Teosinte branched1 and Grassy tillers1 underlie reduced bud Tre6P content and establishment of bud dormancy (Dong et al., 2019). In addition, Tre6P metabolism has been linked to maize inflorescence branching. The ramosa3 mutant, which carries a mutation in a TPP gene, produces branched cobs. However, the RAMOSA3 protein may not only have a catalytic TPP activity but also a regulatory function (Eveland & Jackson, 2012; Satoh-Nagasawa et al., 2006). The wider role of Tre6P in monocot branching, such as the tillering of grasses, including cereals, remains an open question for future research.

Compared to Tre6P signalling, less is known about the involvement of other sugar signalling pathways in vegetative dormancy and branching. While TOR is generally related to growth and has been shown to play a role in stem cell activation in the apical meristem of Arabidopsis (Pfeiffer et al., 2016), its role in branching is still not fully understood, even though hemizygous s6k1s6k2/++ mutants with severe growth phenotypes showed reduced apical dominance and a bushier appearance (Henriques et al., 2010).

Another aspect that has not been investigated in depth is the role of sugar signalling in dormancy and bud release in perennial species. Based on changes in carbohydrate metabolism, it was suggested that sugar signalling is involved in regulating bud dormancy status in the non-woody perennial species, leafy spurge (Euphorbia esula; Anderson et al., 2005). In woody perennials, bud dormancy is associated with gene expression patterns typical of carbon starvation (Tarancón et al., 2017), while bud burst in spring depends on sucrose availability: Expression of an Arabidopsis sucrose-phosphate synthase gene resulted in earlier bud break in spring in transgenic hybrid poplar (Populus alba × Populus grandidentata; Park et al., 2009). Bud break in spring in woody perennials relies not only on the availability of locally stored carbohydrates but also on the import from more distant locations such as the wood parenchyma (Tixier et al., 2019). This carbohydrate mobilisation for budburst may depend on interactions between the clock and sugar signalling. Sugar metabolism and signalling could thus directly determine perennial plant phenology. Given the role of flowering and clock pathways in the control of dormancy and growth of trees (Cooke et al., 2012) and the function of Tre6P in bud outgrowth and flowering of annual plants (see below), it is likely that Tre6P also plays a role in regulating bud break in trees. This could be investigated by genetically manipulating trehalose metabolism in tree species (e.g. of the genus Populus) that are amenable to genetic transformation.

5 GETTING READY TO REPRODUCE—FLORAL INDUCTION

The commitment to flower relies on the concerted action of different signalling pathways, including vernalisation, age, photoperiod and autonomous pathways (Freytes et al., 2021). These converge in regulating the accumulation of Flowering Locus T (FT), a small mobile protein, which then initiates a signalling cascade leading to the accumulation of flowering meristem identity regulators.

Flowering relies on the availability of carbohydrates and their transport in the phloem in the form of sucrose, which promotes floral induction (e.g. Yoon et al., 2021). In fact, the FT-upstream regulator CONSTANS (CO) was shown to promote Granule Bound Starch Synthase (GBSS) expression leading to increased availability of starch for sugar mobilisation in the diurnal cycle and flowering induction (Ortiz-Marchena et al., 2015). As a signal for sucrose availability, Tre6P is also a regulator that promotes flowering. In Arabidopsis, TPS1 function is required for floral transition (Gómez et al., 2010; van Dijken et al., 2004), and constitutive expression of bacterial TPP or TPH genes to lower Tre6P results in late flowering (Schluempmann et al., 2003). Repression of TPS1 with an artificial microRNA delays flowering despite resulting in higher sucrose contents, which is in agreement with the observed feedback of Tre6P on sucrose synthesis (Yadav et al., 2014), and also shows that T6P acts downstream of sucrose in flowering regulation. Reduced expression of FT and Twin Sister of FT (TSF) when TPS1 is repressed, combined with restoration of flowering when FT is overexpressed in the TPS1-repressed plants, demonstrates that Tre6P acts upstream of FT in the leaves (Wahl et al., 2013). In addition, Tre6P controls flowering in response to sucrose availability directly in the shoot apical meristem.

In contrast to the flowering-promoting function of Tre6P, the SnRK1-dependent starvation signalling pathway delays flowering, as shown for Arabidopsis plants overexpressing the SnRK1 gene KIN10 (Baena-González et al., 2007; Tsai & Gazzarrini, 2012). Whether or not this is caused by inhibition of SnRK1 by Tre6P (Zhang et al., 2009) is unknown. However, it was shown that SnRK1 delays flowering by interaction with FUS3, which is a negative regulator of flowering (Tsai & Gazzarrini, 2012, 2014). Similar interactions of SnRK1 with FUS3 in germination, crossing the fus3-3 mutation into an SnRK1 (KIN10) overexpressor restored flowering (Tsai & Gazzarrini, 2012).

The circadian clock closely regulates the seasonal regulation of flowering time. This timekeeping mechanism integrates external (light, temperature) and internal (photosynthesis-generated sugars) cues into specific waveforms of gene expression to regulate rhythmic biological processes (Henriques et al., 2018). Decreased sugar availability or seedling treatment with high doses of nicotinamide increased
circadian period, indicating that metabolites and sugars can act as clock inputs (Haydon et al., 2013). Nicotinamide treatment seemed to interfere with glucose-TOR signalling, similarly to growth under sugar-depleted conditions or low light, with the consequent extension of period length (Zhang et al., 2019). Inhibition of the mitochondrial electron transport chain, treatments with TOR-inhibitor torin 1 and TOR transcript depletion also resulted in a slower-running clock, further highlighting the connection between nutrient perception, the TOR pathway and circadian regulation in Arabidopsis (Wang et al., 2020).

6 | SURVIVAL OR DEATH? CONSEQUENCES OF THE SPEED OF LIFE

Sugar signalling also affects senescence processes in plants. In monocarpic annual plants, senescence and death occur after reproduction. While photosynthesis decreases as leaves senesce, carbon utilisation for growth also declines, and in species such as Arabidopsis (Gnan et al., 2017) or cereals (Furbank et al., 2020), inflorescence photosynthesis can contribute to carbon gain during later developmental stages. As a consequence, annual plants with determinate growth patterns are often not carbon- but nitrogen-starved during senescence, and sugars accumulate in the senescing leaves where they can induce senescence-dependent nitrogen mobilisation to the inflorescence (Wingler et al., 2009).

Both developmental and sugar-induced senescence is delayed in the hexokinase-1 (HXK1) mutant gin2-1, demonstrating a function of HXK1 in senescence regulation (Pourtau et al., 2006). In addition, Tre6P signalling has been shown to be involved in regulating senescence in response to sucrose availability (Wingler, Delatte, et al., 2012a). In parallel with sugar accumulation, Tre6P content increases in senescing leaves, and senescence was delayed in Arabidopsis plants constitutively expressing the bacterial TPP gene otsB. Although TPP expression only had a marginal effect on Tre6P content in old leaves, the Tre6P to sucrose ratio was reduced owing to sucrose accumulation. This is in agreement with a feedback effect of Tre6P on sucrose availability (Wingler, Delatte, et al., 2012a). Overall, these findings suggest that, while hexokinase-dependent regulation is likely responsible for feedback effects on photosynthetic gene expression during senescence (Pourtau et al., 2006), this response also depends on developmental changes that are mediated by Tre6P. In contrast, constitutive over-expression of the KIN10 gene, which codes for an α-subunit of SnRK1, delays flowering and senescence (Baena-González et al., 2007; Tsai & Gazzarrini, 2012), demonstrating that starvation signalling can extend the lifespan of Arabidopsis plants. Although Tre6P can counteract SnRK1-regulated gene expression by inhibiting SnRK1 activity (Zhang et al., 2009), this interaction only happens in young, growing tissues and not in senescing leaves (Wingler, Delatte, et al., 2012a). Therefore, Tre6P and SnRK1 may act in different senescence pathways, or the role of Tre6P is a consequence of earlier developmental changes, which could not be resolved by the constitutive expression of otsB. Similarly, senescence-specific over-expression of KIN10 would be required to confirm that the delayed senescence phenotype is not only resulting from an overall delay in development such as delayed flowering.

The TOR pathway is also involved in regulating the life span of eukaryotes. In heterotrophic organisms (Bjedov & Rallis, 2020) TOR reduces lifespan by acting as a pro-ageing signalling hub. TOR inhibition, on the other hand, could lead to energy saving for maintenance, reprogramming of protein synthesis, improved protein folding and translation fidelity. Downstream of TOR, the inhibition of S6K phosphorylation or its depletion also extends lifespan, confirming the association between this pathway and longevity.

Arabidopsis plants overexpressing yeast or mammalian FKBP12, which results in a rapamycin-sensitive TOR complex, show overall growth inhibition, starch and metabolite accumulation and prolonged life span when grown in the presence of rapamycin (Quilichini et al., 2019; Ren et al., 2012). In agreement with these findings, mutations in other TOR complex components (e.g. las8-1.1; las8-1.2; raptor1b) delayed flowering and increased longevity (Henriques et al., 2014). Conversely, TOR-overexpression resulted in early flowering phenotypes, senescence and reduced life span (Quilichini et al., 2019). An increase in longevity was also observed downstream of TOR in growth-retarded sdk1s6k2/+ mutants (Henriques, unpublished results) and single rps6a and rps6b mutants, whereas RPS6-overexpression had the opposite effect (Creff et al., 2012).
et al., 2010; Ren et al., 2012). Although these findings highlight the relevance of the TOR pathway in regulating plant longevity responses, they were obtained in short-lived annual species such as Arabidopsis. The exact molecular mechanisms controlling lifespan in trees living for hundreds and thousands of years are still unknown. Nevertheless, the connection between TOR, flowering time and circadian clock function (Wang et al., 2020) suggests a role for this pathway in regulating resource allocation, nutrient recycling and senescence during seasonal flowering to ensure that growth can occur during the next stage of vegetative development.

Increased sink strength in perennial species may reduce the importance of sugars as signals for leaf senescence. In the perennial herb Arabis alpina, external sugar supply can accelerate senescence at warm temperatures. However, the senescence response was not as pronounced as in Arabidopsis, and sugar contents declined with leaf age in the natural growth habitat (Wingler, Stangberg, et al., 2012). In aspen (Populus tremula) trees, stem girdling to disrupt the export of sugars resulted in an earlier onset but a slower rate of autumn senescence (Lihavainen et al., 2021). Sugar signalling during leaf senescence would have important consequences for the impact of elevated CO2 and climate change on the phenology of trees and thus growing season length. Based on long-term observations and experimental systems, it was recently suggested that trees in a future climate are mainly sink limited (Zani et al., 2020). According to this analysis, increased growing season photosynthesis due to warmer temperatures and elevated CO2 results in feedback effects on photosynthesis and earlier autumn leaf senescence. However, this conclusion was challenged by Norby (2021), who points out that exposure to elevated CO2 in free-air CO2 enrichment (FACE) experiments does not commonly result in earlier senescence. For example, Populus senescence was delayed in elevated CO2 (Taylor et al., 2008) despite higher sucrose contents (Tallis et al., 2010). In addition, over-expression of sucrose-6-phosphate synthase resulted in sucrose accumulation and delayed senescence, alongside earlier budburst (Park et al., 2009). In Arabidopsis, the Tre6P to sucrose ratio may be a key regulator of leaf senescence in response to carbohydrate supply (see above; Wingler, Delatte, et al., 2012a), but the function of Tre6P in senescence regulation in trees is not known. Overall, the impact of future climatic conditions and CO2 concentrations on autumn tree senescence are likely to depend on a range of factors that interact with sugar signalling pathways, including nitrogen availability, stress and photoperiod. Understanding these interactions is important for predicting future growing season length and carbon sequestration by forests.

**CONCLUSIONS**

Sugar signals are involved in all key transitions of the plant life cycle (Figure 2). In annual plants, high sugar availability, in combination with favourable growing conditions, accelerates the life cycle. Key signalling components for accelerated development include Tre6P and TOR. Recent findings highlight a tight connection between TOR and the circadian clock in integrating light and sugar availability cues into root and apical meristem cell proliferation, affecting overall growth responses and developmental transitions in short-lived plants such as Arabidopsis. However, high concentrations of externally added sugars can delay germination, and trehalose rather than Tre6P (or a low Tre6P to sucrose ratio) may stimulate germination. Under unfavourable external conditions or low sugar availability, SnRK1 delays key life cycle events such as growth, flowering and senescence. Throughout the life cycle, sugar signals interact with hormone signalling pathways, such as the role of SnRK1 in ABA-dependent growth inhibition under stress conditions, or induction of branching by sugars upstream of cytokinin signalling.

While these signalling processes are well-studied in annual plants, understanding of how they affect the life cycle of perennial plants is scarce. Although sugar signalling is not likely to determine the lifespan of the whole plant, key seasonal developmental events, such as bud release and leaf senescence, are affected by sugar availability in perennial species. Therefore, sugar availability is important for the phenology of perennial species, including trees. In annuals as well as perennials, sugar signalling has consequences for the impact of elevated CO2 and climate change on plant development.

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**DATA AVAILABILITY STATEMENT**

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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