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SnRK1/TOR/T6P: three musketeers guarding energy for root growth

Authorship

S. Morales-Herrera[1,2,3], M.J. Paul[4], P. Van Dijck[3,5], T. Beeckman[1,2]

[1] Department of Plant Biotechnology and Bioinformatics, Ghent University, Ghent, Belgium.

[2] VIB Center for Plant Systems Biology, Ghent, Belgium.

[3] Laboratory of Molecular Cell Biology, KU Leuven, Kasteelpark Arenberg, Leuven, Belgium.

[4] Sustainable Soils and Crops, Rothamsted Research, Harpenden, UK

[5] KU Leuven Plant Institute (LPI), Leuven, Belgium

*Correspondence: tobee@psb.vib-ugent.be (Tom Beeckman).

Keywords
T6P, SnRK1, TOR, root growth

Abstract

Sugars derived from photosynthesis, specifically sucrose, are the primary source of plant energy. Sucrose is produced in the leaves and transported to the roots through the phloem serving as a vital energy source. Environmental conditions can result in higher or lower photosynthesis, promoting anabolism or catabolism, respectively, thereby influencing the sucrose budget available for roots. Plants can adjust their root system to optimize the search for soil resources and to ensure the plant's adaptability to diverse environmental conditions. Recently, emerging research indicated that SnRK1, T6P, and TOR collectively serve as fundamental regulators of root development together forming a signaling module to interpret the nutritional status of the plant and translate this to growth adjustments in the below ground parts.

Matching energy needs with resource acquisition in plants.

Plants are sessile organisms which, thanks to their plasticity, can modify their post embryonic development to adapt to different environments. However, the formation of new organs is energy demanding, primarily because new cells must be generated and existing ones modified. Sugars derived from photosynthesis, specifically **sucrose**, are the primary source of plant energy (Figure 1A). Sucrose is produced in the leaves and transported to the roots through the phloem serving as a vital energy source [1]. Environmental conditions can disturb the sucrose budget due to diminished photosynthesis and/or the accumulation of sugars in the shoots. Consequently, this alteration results in a lower relocation of sucrose towards the roots, diminishing their growth. On the other hand, plants can adjust their root system architecture (**RSA**) [2], optimizing the search for soil resources and ensuring the plant's adaptability to diverse environmental conditions. Understanding how plants can sustain an optimal **energy balance** throughout their growth is of fundamental importance because it underpins how plants grow and develop, survive and reproduce. There are several plant kinases that can sense sugar levels and modulate different energy-requiring processes. The two most important and evolutionary conserved ones with antagonistic roles are the SNF1-RELATED PROTEIN KINASE 1 (**SnRK1**) and the TARGET OF RAPAMYCIN (**TOR**) protein kinase [3]. SnRK1 is able to initiate a beneficial counteraction to metabolic stress conditions in plants thereby switching off anabolic pathways and turning on catabolism processes, resulting in SnRK1 repressing energy-consuming processes of which growth is an obvious example [4]. On the other hand, TOR activity is switched on under favorable and nutrient-rich conditions, promoting the activation of diverse cellular processes that consume energy, such as cell proliferation [5]. Interestingly, Trehalose 6-phosphate (**T6P**) is a sugar signal which closely tracks the level of sugars in plants. An increase in sucrose elevates T6P leading to metabolic changes to promote growth and development [6]. In addition, T6P is known to inhibit SnRK1 activity in growing tissues [7–9]. Consequently, T6P has been proposed as a key regulator in diverse developmental and metabolic processes such as starch metabolism, shoot development, among others [10,11]. Numerous studies have also identified the role of SnRK1, TOR and T6P in diverse

processes such as vegetative growth [12,13], flowering [13–15], reproductive development [16] and in **crop yield** determination [17–20].

Recent research has revealed that key components of the energy balancing network, including SnRK1, T6P, and TOR, play a pivotal role in shaping the RSA. Their regulatory influence extends beyond the **primary root** growth, actively participating in lateral root (**LR**) formation. In this opinion article, we will dig into the unexplored territory of understanding how SnRK1, T6P, and TOR collectively integrate carbon and energy signaling with hormonal (auxin and abscisic acid) signaling to shape the RSA. This integration contributes to maintaining the plant's energy balance, even in non-photosynthetic parts of the plant body.

Involvement of SnRK1 in Shaping Root Development

SnRK1, a heterotrimeric sensor kinase complex, consists of the catalytic α subunits, encoded by KIN10, KIN11 and KIN12 in Arabidopsis [21,22], and two regulatory subunits: β and βγ [23]. SnRK1 is a critical player in sugar signaling as SnRK1 senses the energy and sugar status. In this way, SnRK1 coordinates metabolic and developmental processes to optimize resource allocation and adaptation to varying conditions. Uptake of soil resources is primarily driven by roots and their building plan (i.e., “RSA”) importantly contributes to the uptake performance of water and nutrients by the plant. Understanding the role of SnRK1 in modifying RSA has remained limited, but recent insights have provided a turning point. Notably, SnRK1 activity was shown to influence primary root growth when ammonium is the dominant nitrogen source in the soil [24]. More specifically, KIN10 plays a regulatory role on the nitrate SLOW ANION CHANNEL HOMOLOG 3 (SLAH3), which is involved in nitrate-dependent alleviation of ammonium toxicity [24]. Furthermore, KIN10 modulates the activity of the key regulator in nitrate signaling, NIN-LIKE PROTEIN 7 (NLP7), thereby influencing root development in response to fluctuations in carbon and nitrate availability. [25].

As far root branching is concerned two, at first sight seemingly contradicting studies, did recently report on a role for SnRK1 in LR formation. At the one hand, Morales-Herrera et al. [26] demonstrated that loss-of-function mutants of KIN10 or KIN11, as well as tissue-

specific CRISPR/Cas knockout (TSKO) lines, exhibit higher LR densities compared to wildtype seedlings. Conversely, KIN10 overexpressor lines KINO1 and KINO2 were shown to display an opposite LR phenotype with lower LR densities, underscoring the inhibitory role of SnRK1 in LR formation under standard growth conditions [27] (Figure 1C). Secondly, Muralidhara et al. described, however, a promoting role of SnRK1 in the initiation of LRs but this time as a stress response triggered by brief periods of low light exposure or sudden darkness [27]. In these conditions, SnRK1 facilitates the LR initiation process by activating the LR regulatory transcription factor AUXIN RESPONSE FACTOR19 (ARF19) through the phosphorylation of BASIC LEUCINE ZIPPER63 (bZIP63), a direct upstream transcriptional regulator [27]. Both studies illustrate the importance of environmental conditions that converge on SnRK1 thereby capable of influencing the final outcome of the signaling cascade in an opposite phenotypic response.

Still in relation to environmental stress conditions, the interaction between SnRK1 and the stress phytohormone abscisic acid (ABA) in root systems has been well characterized [28], however, it has only recently been revealed that primary root growth is regulated by the interplay between SnRK1 and ABA pathways due to the complex integrated by SnRK2 type 3 kinases and 2C-type protein phosphatases (PP2C) which is acting as repressor of SnRK1 under optimal growth conditions [29] (Figure 1A). Furthermore, during periods of starvation, SnRK1 was shown to act upstream of and to activate members of the basic leucine zipper (bZIP) transcription factor family (TFs) [30]. This activation subsequently triggers the transcription of *INDOLE-3-ACETIC ACID 3* (*IAA3)*[30], a well-established inhibitor of root growth [31].

Role of TOR in root development

The TOR complex kinase (TORC) has been identified and extensively studied throughout the eukaryotic kingdom. While both yeast and animals have both TORC1 and TORC2, in plants, only TORC1 has been discovered. TORC1 consists of three essential subunits: the catalytic TOR kinase, the Regulatory-Associated Protein of TOR (RAPTOR) [32], and Lethal with Sec13 protein 8 (LST8) [33]. TORC1 plays a central role in communicating external signals, such as biotic and abiotic stresses, with the internal status of nutrients

and energy levels. Thus, external information is integrated by this complex to regulate anabolic processes under favorable conditions [34]. Studies of TOR expression revealed that it is present in all Arabidopsis tissues, especially in primary roots, LRs and meristematic tissues [35]. *lst8-1* and *lst8-2* are non-lethal mutants, but *lst8-1* displays reduced growth [33], *raptor1a* mutants do not present phenotypic differences with wild type seedlings [32], while *raptor1b-1* and *raptor1b-2* mutants exhibit a general delay in organ development, including the development of the primary root [36]. Interestingly, there is one gene encoding for TOR in the Arabidopsis genome and its mutants *tor-1* and *tor-2* are embryo-lethal [35], while estradiol-inducible *tor* mutants exhibit significantly delayed growth in seedlings, affecting both primary root and LRs [37].

Primary roots contain a region known as the root apical meristem (**RAM**), crucial for directing subterranean growth. Studies have demonstrated that TOR regulates the cell cycle of **stem/progenitor cells** in the RAM, dependent on photosynthesis-derived sucrose/glucose production [37]. The activation of these cells occurs through TOR's direct phosphorylation of the E2Fa transcription factor, a master regulator for cell cycle progression [37]. Interestingly, auxin, as a key regulator of root growth and development, was shown to activate TOR-E2Fa signaling through the Rho-related protein 2 (ROP2) at high auxin concentrations [38]. Additionally, a recent discovery indicates that mediator subunit 17 (MED17) physically interacts with E2Fa and E2Fb, influencing primary root development [39]. This interaction, in turn, regulates cell cycle genes such as MINICHROMOSOME MAINTENANCE 3/5/7 (MCM3/5/7), ORIGIN RECOGNITION COMPLEX PROTEIN 6 (ORC6), and E2F TARGET GENE 1 (ETG1) [39] (Figure 1A). Moreover, ETHYLENE INSENSITIVE 2 (EIN2) has been characterized as a negative regulator for TOR-RAM activation [40]. In conditions of low glucose, TOR fails to phosphorylate EIN2, which in its unphosphorylated form shuttles from the cytoplasm to the nucleus thereby inhibiting E2Fa resulting in the suppression of primary root development [40]. In addition, YET ANOTHER KINASE 1 (YAK1) has been described as a negative regulator of RAM activity and cell diferentiation downstream of TOR signaling [41], because upon TOR inhibition, YAK1 facilitates the upregulation of cyclin-dependent kinase (CDK) inhibitors known as SIAMESE-RELATED 4/5/7 (SMR4/5/7) [41].

Recently, the importance of TOR activity for LR formation was demonstrated [26,42]. This is substantiated by the TOR-overexpressor line (TOROE [43]) displaying a higher density of emerged LRs while *raptor1b-1* and *lst8-1* mutants, along with tissue-specific CRISPR/Cas knock outs (TSKO-TOR lines), exhibit a lower LR density compared to the wild type [26,42]. In addition, it has been proposed that the activation of TOR in LR founder cells is influenced by sugars derived from the shoot [42]. Interestingly the MED17-TOR-E2fa complex, besides its role in the cell cycle, regulates *ARF7* transcription, subsequently activating the expression of auxin-responsive target genes, including *LATERAL ORGAN BOUNDARIES DOMAIN 16/18/19/33* (*LBD16/18/29/33*) [39] (Figure 1C). These genes are recognized as central regulators of LR development [44].

Moreover, under sulfate limitation, a decrease in TOR activity in shoots induces **autophagy**, facilitating the relocation of carbon to the roots and promoting higher TOR activity in roots. Sucrose plays a dual role in roots, both serving as a carbon source and a signaling molecule, sustaining root apical meristem activity in sulfate limitation environmental conditions [45] (Figure 1B). Consequently, this contributes to a higher root-to-shoot ratio, supporting root growth and facilitating the exploration for new sulfate resources in the soil [45].

Furthermore, the inhibition of primary root growth is a well-established response to phosphorus deprivation [46]. Recent reports emphasize that during phosphorus deprivation, the ARABIDOPSIS-ROOT-SPECIFIC KINASE 1 (ARSK1) is repressed, directly influencing the activity of TORC1 and modulating root growth [47]. This modulation is made possible because ARSK1 phosphorylates RAPTOR1B under phosphorus-sufficient conditions, highlighting the intricate TOR regulatory mechanisms involved in adapting root growth to varying phosphorus levels [47].

Brassinosteroids, as phytohormones, play essential roles in regulating various processes in roots, including meristem maintenance, elongation, and LR formation [48]. It has recently been found that brassinosteroids modulate autophagy depending on carbon availability [49]. This newfound insight reveals that under starvation conditions, brassinosteroid-insensitive 2 (BIN2), a kinase like-GLYCOGEN SYNTHASE KINASE 3

(GSK3), phosphorylates RAPTOR1B, thereby promoting autophagy [49]. Moreover, glucose-TOR signaling controls BRASSINAZOLE-RESISTANT 1(BZR1) accumulation, promoting plant growth [50]. This intricate interplay between brassinosteroids and TOR, as studied in hypocotyl growth, further emphasizes their indispensable role in orchestrating mechanisms such as root development.

T6P is a key metabolite in root development.

Trehalose, a non-reducing sugar, is composed of two glucose molecules linked together in an α,α-1,1 configuration. In plants, the synthesis of trehalose involves the action of enzymes called T6P synthases (TPS) and T6P phosphatases (TPP), with an intermediate molecule: T6P [51]. T6P serves as a signaling molecule, with its levels increasing or decreasing in response to varying sucrose levels, thus conveying information to the plant about the availability of carbon resources and energy [6]. *TPS*s acting as T6P synthesizers and *TPP*s as T6P degraders show tissue-specific expression patterns [52–54] and are thus characteristic features of the temporal-spatial regulation of the T6P pathway during plant growth and development.

The Arabidopsis genome contains 11 genes encoding TPS, which are classified into two classes. Examination of the expression patterns of Class II TPS genes (TPS5-11) has revealed specific and overlapping expression patterns in root tissues, including in the primary root and LRs [54] suggesting their involvement in root development. In addition, the most important TPS class I gene, TPS1, is considered to be of fundamental importance for plant development with *tps1-1* mutants being arrested in the embryonic torpedo stage [55].TPS1 has 3 domains: a N-terminal domain with a putative autoinhibitory motif, a glucosyltransferase domain containing the catalytic site, and a TPP-like C-terminal domain with unknown function [56,57]. Additionally, it is suggested that TPS1 plays a fundamental role in root vasculature development (Figure 1A), because the vascular bundles in roots are disrupted in the transgenic line lacking the C-terminal TPP-like domain [58].

On the other hand, the Arabidopsis genome contains 10 genes encoding TPPs for which TPP activity has been demonstrated by heterologous expression in yeast [52]. An

analysis of TPP expression patterns also revealed specific expression in various tissues, cells, and developmental stages [52]. Intriguingly, *TPPA*, *TPPB*, *TPPH*, and *TPPI* are expressed in cells involved in early LR formation events, suggesting they might have a role in RSA [26]. *TPPB*, in particular, has been proposed as a key gene that negatively regulates T6P levels during early LR formation [26] (Figure 1C). Mutants of TPPI have shorter roots, possibly explained by the reduced expression of the auxin transporters PINFORMED 1 (PIN1) and PIN3 [59]. In abiotic stress situations, TPPE participates in the inhibition of primary root elongation induced by ABA. This is proposed to work through the ABA RESPONSE ELEMENT BINDING FACTOR2 (ABF2) directly binding to the promoter of *TPPE* thereby enhancing TPPE and consequently trehalose levels to induce the production of reactive oxygen species (ROS) that contribute to the inhibition of root growth [60].

Furthermore, an increase in LR density has been reported by elevating T6P levels through T6P-feeding experiments through the permeable root epidermis or through treatments with the high-light-inducible T6P precursor DMNB-T6P [26,61]. Moreover, the ectopic expression of the *TPS* gene from *Escherichia coli: otsA* [62] controlled by the *GATA23* promoter, active during early LR formation [63], stimulates branching density in Arabidopsis [26]. Altogether, it can be concluded that modification of T6P levels, either through chemical or genetic approaches, has a prominent effect on LR formation arguing for a regulatory function of T6P in this developmental process (Figure 1C).

As previously indicated, various studies have highlighted T6P's role in the transition to flowering, vegetative phase change, and regulation of tuber sprouting across different plant species [11]. T6P stimulates the flux to organic acids such as citrate [64], and recent research suggests that citrate inhibits MORE AXILLARY GROWTH2 (MAX2) [65], an essential component for the perception of strigolactones. Additionally, the strigolactone pathway can activate the transcription factor BRANCHED1 (BRC1)[66], known for its role in regulating bud dormancy [67]. Moreover, it is suggested that shoot branching relies on T6P levels within the buds [14], supported by experiments involving the heterologous expression of TPP under the control of the BRC1 promoter, resulting in reduced T6P levels specifically in the buds [14]. Consequently, plants displayed delayed initiation and

fewer rosette branches compared to wildtype plants [14]. These findings establish a connection between strigolactone signaling and sugar metabolism. Given that strigolactones are primarily synthesized in roots [68] and based on its different proposed roles in root development [69], it is inferred that they are involved in carbon assimilation to modulate root system architecture.

Root development controlled by a crosstalk of SnRK1-T6P-TOR

While TOR, SnRK1 and T6P have been individually documented to regulate distinct processes, ongoing investigations are gradually unveiling the interconnections between them. It has been reported that in Arabidopsis, KIN10 interacts with and phosphorylates TOR through its RAPTOR1B subunit [70]. Additionally, a recent study characterized a negative feedback loop in TOR signaling, attributed to the upregulation of FCS-like zinc finger 8 (FLZ8) by TOR under sugar sufficiency conditions [71]. FLZ8 is proposed to act as a scaffold protein, facilitating the interaction between KIN10 and RAPTOR1B, thereby moderating TOR activity and regulating growth during sugar sufficiency [71]. In addition, Zhang et al. demonstrated that T6P is able to inhibit SnRK1 in young tissues of Arabidopsis [9]. Zacharaki et al. showed that T6P's impact on embryogenesis and flowering arises from the inhibition of SnRK1 [8]. This repression has also been characterized in other plant species, including *Zea mays* [16,20] and *Triticum aestivum* [72,73]. Remarkably, it was recently shown that the TPS class II proteins repress SnRK1 activity [74]. TPS class II are localized in endoplasmic reticulum (ER) and it is suggested that TPS class II proteins promote a change in subcellular SnRK1 localization from the nucleus to the ER in young tissues in order to inhibit SnRK1 activity [74].

Plants, as organisms with great adaptability, can dynamically adjust their RSA throughout their lifecycle. In optimal growth conditions, the primary root undergoes continuous development facilitated by TOR phosphorylation and the activation of E2Fa/b, promoting root meristematic activity [75]. However, under stress conditions, SnRK1 is liberated from the complex it typically forms with SnRK2-PP2C under normal conditions, making it available to restrict TOR activity. This liberation happens in the presence of elevated ABA levels that are sensed by the soluble ABA receptors, PYR/PYL/RCAR [76]. Additionally, under stress conditions, KIN10-mediated phosphorylation of E2Fa takes place, leading

to subsequent E2Fa degradation [77]. The suppression of E2Fa activity by SnRK1 underscores its antagonistic role in TOR signaling, revealing the intricate interplay between SnRK1, TOR, and the regulation of root development [77] (Figure 1B).

While it is well established that hormonal interactions, particularly with auxin as the key phytohormone, play a crucial role in regulating root branching [78], the recent focus has shifted towards unraveling the intricate interplay of carbon utilization and energy during LR formation. T6P has emerged as a pivotal energy signal in this context, orchestrating LR induction by inhibiting SnRK1 and activating TOR [26]. In root tissue, chemically elevated T6P levels modulate the expression of SnRK1-regulated genes, reflected in the downregulation of *ASN1* and *TPS8* (induced by SnRK1 [9]) and the up-regulation of *UDPGDH*, *TPS5* and *bZIP11* (repressed by SnRK1 [9]) [26]. T6P-mediated LR induction is sensitive to the threshold of SnRK1 inhibition, as illustrated by the incapacity of *kin10, kin11-1*, and *kin11-2* mutants to generate new LR events upon exposure to DMNB-T6P treatment [26]. Furthermore, T6P-induced TOR phosphorylation in root tissue seems to be the activation mechanism for T6P-LR induction. However, further research is needed to conclusively demonstrate the existence of this direct control of T6P on TOR, leaving this assumption still as hypothetical at present. It is possible that this TOR activation occurs through the repression of SnRK1. Notably, higher T6P levels necessitate only optimal TOR activity levels for LR induction, as evidenced by the inability of *raptor1b-1* and *raptor1b-2* mutants and the TOR overexpressor transgenic line (TOROE) to initiate new root branching events during DMNB-T6P exposure [26]. Adding to the complexity, auxin treatment inhibits *TPPB* expression in an IAA14/ARF7ARF19-dependent manner, underscoring the central role of T6P in the intricate regulatory network governing LR formation [26] (Figure 1C).

Concluding remarks and future perspectives.

Recent studies have highlighted the pivotal roles of SnRK1, T6P, and TOR in balancing energy levels to mediate root growth and development. SnRK1, a key player in nutrient-dependent responses, influences ammonium-dependent root growth and initiates LR under stress conditions as unexpected darkness. TOR, central to nutrient sensing,

regulates both primary and LR development through intricate cell cycle control. T6P, a vital signaling molecule, impacts root architecture by influencing LR formation.

Under normal growth conditions, TOR and SnRK1 are shown to be antagonistic regulators of the RSA, including both primary root and LR formation. T6P serves as a central regulator, bridging the connection between auxin signaling and energy balance by modulating the activities of SnRK1 and TOR. This intricate coordination plays a pivotal role in the finely-tuned regulation of LR formation. It involves the inhibition of SnRK1 and activation of TOR, orchestrating a sophisticated mechanism. However, further research is needed to ascertain the precise nature of T6P's regulatory role, whether direct or indirect, in this process. This insight is pivotal in unraveling the intricate molecular mechanisms that govern root plasticity and adaptability to changing environmental conditions. These discoveries emphasize the potential for targeted interventions in crop improvement by manipulating certain components of SnRK1, T6P, and TOR pathways. For example, through selection of TPP variants in crops that could facilitate more optimal root architecture for resistance to water and/or nutrient deprivation. All the genes mentioned in this opinion article are summarized on Table I.

Acknowledgements

SMH, TB and PVD express appreciation to Maria Njo for her invaluable contribution in creating the key figure based on our initial drafts. Work in the lab of TB is funded by grants (G001221N and G028421N) from the Research Foundation-Flanders (FWO). MJP acknowledges funding from the Delivering Sustainable Wheat (BB/X011003/1) UKRI-BBSRC institute strategic programme.

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Glossary

- **Sucrose:** A nonreducing disaccharide sugar composed of glucose and fructose. It is the end product of photosynthesis in plants.
- **RSA (Root System Architecture)**: The spatial configuration and organization of root components.
- **Energy Balance:** The equilibrium between the energy a plant receives through photosynthesis and the energy it expends in various physiological processes.
- **SnRK1 (SNF1-RELATED PROTEIN KINASE 1)**: Plant homolog of the heterotrimeric AMP-activated protein kinase/sucrose non-fermenting1 (AMPK/Snf1).
- **TOR (TARGET OF RAPAMYCIN)**: evolutionarily conserved protein kinase that integrates diverse internal and external cues, regulating anabolic processes in plants.

- **T6P (Trehalose 6-Phosphate):** A sugar signal that is synthesized from UDP-glucose (UDPG) and glucose-6-P by the activity of TPS.
- **RAM (Root Apical Meristem):** The root tip portion that contains actively dividing cells. These will produce the different tissues of primary root.
- **Crop Yield:** Quantity of harvested crop per unit area over a specific period.
- **Primary Root:** The main root of a plant that develops from the radicle.
- **LR (Lateral Root):** Secondary roots that branch off from the primary root.
- **TSKO (Tissue-Specific CRISPR/Cas Knockout):** Genetic modification technique using CRISPR/Cas technology to selectively "knock out" specific genes in targeted tissues.
- **LR Densities:** Quantity of lateral roots within a defined area of the root system.
- **Stem/Progenitor Cells:** Undifferentiated cells with the unique ability to both self-renew and differentiate into specialized cell types.
- **Autophagy:** Its meaning is “self-eating”, it is the natural process that plants perform to recycle damaged or unnecessary components.

Figure

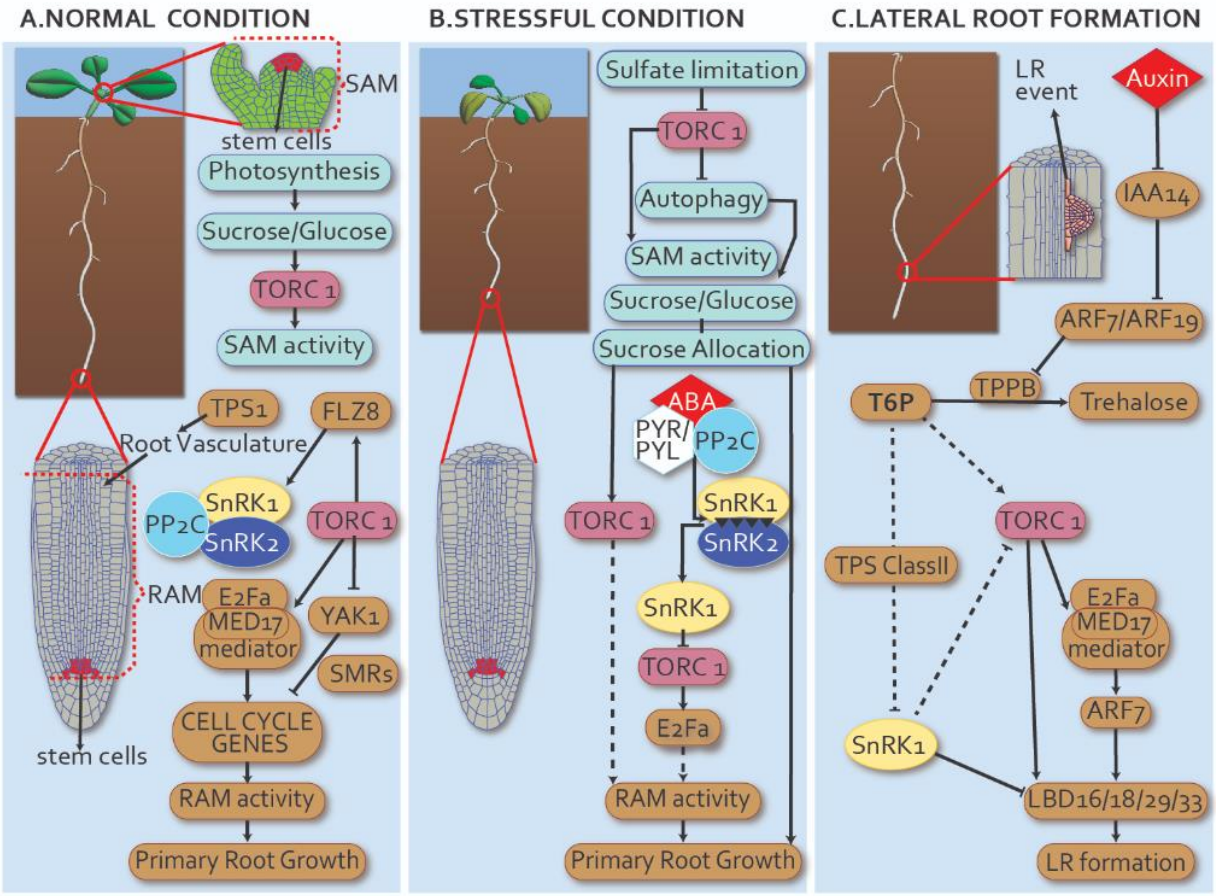

Figure 1: A SnRK1-T6P-TOR hub plays a central role in orchestrating the root system architecture. A. Under normal conditions, plants generate sugars such as sucrose and glucose through photosynthesis, promoting the activity of Target of Rapamycin Complex 1 (TORC1) in both the shoot apical meristem (SAM) and the root apical meristem (RAM) [38,75,79]. This activation triggers stem cell division in SAM and controls the cell cycle of root stem cells via the E2FA-Mediator(MED)17 complex [39]. This intricate interplay leads to the promotion of RAM activity through cell cycle genes transcription and subsequent primary root growth [39]. In addition, under sugar sufficient conditions,TORC1 induces FLZ8 that creates a negative feedback loop regulation, promoting SnRK1 activity that regulates primary root growth [71]. Moreover, it has been described that TORC1 inhibits YAK1 releasing the inhibition of the cell cycle genes by the CDK inhibitors SMRs, allowing primary root growth [41].

Additionally, TPS1 has been described to participate in root vasculature formation [58]. **B.** Under stressful conditions, such as sulfate limitation, TORC1 activity is inhibited [45]. This inhibition induces autophagy in shoots and directs sucrose/glucose allocation to the roots, sustaining primary root growth and allocating resources for plant survival [45]. Moreover, elevated levels of abscisic acid (ABA) during stress are sensed by PYR/PYL ABA receptors, liberating the SNF1-related protein kinase1 (SnRK1) from the SnRK2-SnRK1-PP2C complex. SnRK1, in turn, inhibits TORC1 activity, repressing primary root growth [76]. **C.** During lateral root (LR) formation, auxin promotes breakdown of the IAA14 repressor, allowing the activation of AUXIN RESPONSE FACTOR7/19 (ARF7/ARF19) transcriptional activators. Trehalose 6-phosphate (T6P) phosphatase B (TPPB) is down-regulated by auxin through the canonical auxin response module IAA14-ARF7/ARF19, and T6P, potentially in collaboration with T6P-synthases class II [74], inhibits SnRK1 activity, that possibly results in promoting TOR activity and LR formation [26]. This process may involve the activation of the E2Fa-Mediator complex and subsequent ARF7 transcriptional activation, leading to the expression of LR regulatory genes such as LATERAL ORGAN BOUNDARIES-DOMAIN 16/18/29/33 [39,44].

Table I: AGI accession numbers of genes mentioned in the text.

Gene	AGI
KIN10	AT3G01090
KIN11	AT3G29160
KIN12	AT5G39440
KINβ1	AT5G21170
KINβ2	AT4G16360
KINβ3	AT2G28060
SNF4	AT1G09020
SLAH3	AT5G24030
NLP7	AT4G24020
ARF19	AT1G19220
bZIP63	AT5G28770
bZIP11	AT4G34590
bZIP2	AT2G18160
bZIP44	AT1G75390
IAA3	AT1G04240
SNRK2.2	AT3G50500
SnRK2.3	AT5G66880
SnRK2.6	AT4G33950
PP2CA	AT3G11410

RAPTOR1A	AT5G01770
RAPTOR1B	AT3G08850
TOR	AT1G50030
LST8-1	AT3G18140
LST8-2	AT2G22040
EIN2	AT5G03280
FLZ8	AT3G22550
YAK1	AT5G35980
SMR4	AT5G02220
SMR5	AT1G07500
SMR7	AT3G27630
ARSK1	AT2G26290
ROP2	AT1G20090
E2Fa	AT2G36010
E2Fb	AT5G22220
MCM3	AT5G46280
MCM5	AT2G07690
MCM7	AT4G02060
ORC6	AT1G26840
ETG1	AT2G40550
ARF7	AT5G20730
LBD16	AT2G42430
LBD18	AT2G45420
LBD29	AT3G58190
LBD33	AT5G06080
TPS5	AT4G17770
TPS6	AT1G68020
TPS7	AT1G06410
TPS8	AT1G70290
TPS9	AT1G23870
TPS10	AT1G60140
TPS11	AT2G18700
TPS1	AT1G78580
TPPA	AT5G51460
TPPB	AT1G78090
TPPH	AT4G39770
TPPI	AT5G10100
TPPE	AT2G22190
PIN1	AT1G73590
PIN3	AT1G70940
ABF2	AT1G70940
GATA23	AT5G26930
ASN1	AT3G47340
UDPGDH	AT3G29360
IAA14	AT4G14550
ARF19	AT1G19220
MAX2	AT2G42620
BRC1	AT3G18550
BIN2	AT4G18710

BZR1	AT1G75080