SnRK1 and TOR: modulating growth–defense trade-offs in plant stress responses

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Received 15 November 2018; Editorial decision 7 February 2019; Accepted 7 February 2019

Editor: Christian Meyer, INRA, UK

Abstract

The evolutionarily conserved protein kinase complexes SnRK1 and TOR are central metabolic regulators essential for plant growth, development, and stress responses. They are activated by opposite signals, and the outcome of their activation is, in global terms, antagonistic. Similarly to their yeast and animal counterparts, SnRK1 is activated by the energy deficit often associated with stress to restore homeostasis, while TOR is activated in nutrient-rich conditions to promote growth. Recent evidence suggests that SnRK1 represses TOR in plants, revealing evolutionary conservation also in their crosstalk. Given their importance for integrating environmental information into growth and developmental programs, these signaling pathways hold great promise for reducing the growth penalties caused by stress. Here we review the literature connecting SnRK1 and TOR to plant stress responses. Although SnRK1 and TOR emerge mostly as positive regulators of defense and growth, respectively, the outcome of their activities in plant growth and performance is not always straightforward. Manipulation of both pathways under similar experimental setups, as well as further biochemical and genetic analyses of their molecular and functional interaction, is essential to fully understand the mechanisms through which these two metabolic pathways contribute to stress responses, growth, and development.

Keywords: Defense, growth, plant, SnRK1, stress responses, TOR.

Introduction

Environmental stresses such as extreme temperatures, drought, flooding, or attacks from various pathogens are major yield-limiting factors, reducing crop productivity by >50% (Bray et al., 2000), and thereby our capacity to provide sufficient food, fiber, and fuel for a growing human population. To cope with adverse environmental conditions, plants trigger responses that range from rapid protective mechanisms (e.g. osmolyte accumulation) to developmental modifications (e.g. reduction in the shoot:root ratio), ultimately promoting stress tolerance and survival at the expense of growth. However, despite our increasing knowledge on stress responses, how stress impinges on growth is still poorly understood. Growth–defense trade-offs may arise indirectly from nutrient reallocation and limitation during stress, but also as a direct output of the interplay between antagonistic signaling pathways that either promote or restrict growth (Huot et al., 2014; Eichmann and Schafer, 2015). A deep understanding of the interactions between stress and growth regulatory pathways is therefore essential to uncouple the two processes, making the two features compatible in plant breeding and engineering strategies.
In plants, there are two central nutrient-sensing kinases with increasing connections to stress responses and growth, and with largely antagonistic functions: the protein kinase complexes SnRK1 (Snf1-related protein kinase 1) and TOR (target of rapamycin) (Broeckx et al., 2016; Dobrenel et al., 2016a; Margalha et al., 2016; Baena-Gonzalez and Hanson, 2017; Shi et al., 2018). SnRK1 is activated under low carbon conditions to promote energy-saving and nutrient remobilization strategies, whilst TOR is activated in response to nutrient availability to promote cell proliferation and growth. Despite having evolved numerous plant-specific features, these two eukaryotic complexes are to a large extent structurally and functionally conserved (Roustan et al., 2018). The SnRK1 and TOR pathways have been mostly studied independently and, despite an increasing interest, knowledge of their interconnections remains scarce. The answers to fundamental questions concerning their mutual regulation, as well as the mechanistic details behind commonly regulated processes, are still to be discovered.

In this review, we aim to cover the literature that addresses the connections between SnRK1 and TOR in plants, establishing analogies with other eukaryotic systems, when pertinent. Following a brief overview of SnRK1 and TOR in plants, we will focus on the involvement of these central pathways in the growth–defense trade-offs that are typically associated with biotic and abiotic stress responses (see Supplementary Table S1 online). It is important to note that these complexes also play roles throughout development (Baena-Gonzalez and Hanson, 2017), but these functions are beyond the scope of this review and will not be addressed here.

**SnRK1 and TOR in plants**

**Complex composition**

Arabidopsis SnRK1 is the closest relative of yeast SNF1 (sucrose non-fermenting 1) and animal AMPK (AMP-activated protein kinase), whereas SnRK2s and SnRK3s comprise more divergent plant-specific protein kinases of the SnRK superfamily (Hrabak et al., 2003; Crozet et al., 2014). SNF1/AMPK/SnRK1 are heterotrimERIC serine-threonine protein kinase complexes, composed of an α catalytic subunit and two regulatory subunits, β and γ. Three genes encode the α subunit in Arabidopsis, SnRK1α1, SnRK1α2, and SnRK1α3 (also referred to as AKI10/AKI11/AKI12 or KIN10/KIN11/KIN12). SnRK1α1 accounts for the majority of SnRK1 activity (Jossier et al., 2009), while SnRK1α3 is only expressed at low levels in pollen and seeds (Baena-Gonzalez et al., 2007; Winter et al., 2007; Fragoso et al., 2009). The β subunit, which acts as a scaffold within the complex, is also encoded by three genes in Arabidopsis (SnRK1β1/β2/β3). Amongst these, SnRK1β3 has plant-specific features, lacking the otherwise conserved N-terminal extension domain, associated with subcellular localization and target specificity, and the carbohydrate-binding module (CBM) that in SNF1 and AMPK binds glycogen (Wiatrowski et al., 2004; Bendayan et al., 2009; McBride et al., 2009). Finally, the canonical regulatory γ subunit is encoded by a single SnRK1βγ gene in Arabidopsis (Kleinow et al., 2000; Lumbreras et al., 2001; Ramon et al., 2013; Emanuelle et al., 2015). SnRK1βγ has the characteristics of typical γ subunits, such as a β-interacting sequence domain, and cystathionine-β-synthase domains that in AMPKγ bind adenylates and allow allosteric activation by AMP (Crozet et al., 2014; Broeckx et al., 2016; Margalha et al., 2016). In addition, SnRK1βγ has plant-specific features, harboring a CBM on its N-terminus (Lumbreras et al., 2001; Ramon et al., 2013; Emanuelle et al., 2015). With the exception of SnRK1βγ, for which null mutants are unviable (Ramon et al., 2013; Gao et al., 2016), all single mutants described for each of the subunits display a mostly wild-type phenotype under optimal growth conditions (Li et al., 2009; Sheen, 2014; Jeong et al., 2015; Mair et al., 2015). This suggests at least partial redundancy amongst each type of subunit, and opens up the possibility that SnRK1 complexes exist in multiple forms depending on the combinations of subunits they harbor. Highlighting the centrality of this pathway, and similarly to snrk1βγ, snrk1α1/α2 double knockouts in higher plants are unviable (Baena-Gonzalez et al., 2007).

The conserved TOR kinase belongs to the phosphatidylinositol 3-kinase-related protein kinase family. It also assembles into multiprotein complexes that in yeast and animals are of two types: TORC1 and TORC2 (TOR complex 1 and 2). Both types of complexes contain TOR kinase and LST8 (lethal with SEC thirteen protein 8), but differ in the associated regulatory subunits. TORC1 contains RAPTOR (regulatory associated protein of TOR), whereas TORC2 contains RICTOR (rapamycin-insensitive companion of TOR) and SIN1 (SAPK-interacting protein 1) (Saxton and Sabatini, 2017). However, in plants, there is only evidence for the existence of TORC1 complexes (van Dam et al., 2011). In the Arabidopsis genome, there is a single gene for the TOR kinase, which is highly expressed in young proliferating tissues, such as the embryo and the primary meristems (Menand et al., 2002). The other two TORC1 components, RAPTOR (Anderson et al., 2005; Deprost et al., 2005) and LST8 (Moreau et al., 2012), are encoded by two genes each, one of which is predominantly expressed (RAPTOR1B and LST8-I), with their corresponding single mutants displaying TOR-associated defects (Anderson et al., 2005; Deprost et al., 2005; Moreau et al., 2012; Salem et al., 2018). Similarly to snrk1βγ, snrk1α1/α2 and snrk1βγ knockouts, Arabidopsis tor null mutants are lethal (Menand et al., 2002; Ren et al., 2011).

**Mechanisms of activation**

SNF1/AMPK/SnRK1 are activated in response to declining energy levels. Whilst mammalian AMPK is activated in response to high AMP:ATP or ADP:ATP ratios, the ultimate signal that results in SnRK1 activation remains unknown (Margalha et al., 2016). Nevertheless, it is clear that SnRK1, unlike AMPK, is not allosterically activated by AMP (Wilson et al., 1996; Emanuelle et al., 2015), but its activity correlates with situations of energy deficit (Baena-Gonzalez et al., 2007; Baena-Gonzalez and Sheen, 2008; Tome et al., 2014). Conversely, sugar supply inactivates SnRK1 (Baena-Gonzalez et al., 2007; Baena-Gonzalez and Sheen, 2008) and this requires...
type 2C protein phosphatases (PP2Cs) in adult rosette leaves (Rodrigues et al., 2013). In actively growing young tissues, SnRK1 activity is inhibited indirectly by specific sugars such as trehalose-6-phosphate (T6P), glucose-6-phosphate, or glucose-1-phosphate (Toroso et al., 2000; Zhang et al., 2009; Nunes et al., 2013b). T6P is of particular relevance, as it has been ascribed a crucial role in sugar signaling (Schluepmann and Paul, 2009), functioning as a proxy of the plant sucrose status (Lunn et al., 2014). Recent data suggest that T6P inhibits SnRK1 by disrupting the interaction between SnRK1 and its upstream kinases SnAKs (Zhai et al., 2018) [SnRK1-activating kinases, also called GRiKs, geminivirus Rep-interacting kinases (Shen et al., 2009; Crozet et al., 2010)]. SnAKs phosphorylate SnRK1 on a conserved residue within the T-loop, the phosphorylation of which is required for SnRK1 kinase activity (Baena-Gonzalez et al., 2007). In vitro studies showed that SnRK1 activity and T-loop phosphorylation are regulated in response to the redox status, decreasing progressively in oxidizing conditions. This was attributed to the oxidation of a conserved Cys residue, situated in close proximity to the residue phosphorylated by SnAKs, also affected by oxidation themselves (Wurzinger et al., 2017). However, the in vivo evidence correlating T-loop phosphorylation with activation of SnRK1 signaling is controversial. T-loop phosphorylation was shown to increase in response to submergence (Cho et al., 2016), but other studies have reported similar levels of T-loop phosphorylation under control and stress conditions, raising the question of how SnRK1 activation occurs (Baena-Gonzalez et al., 2007; Fragoso et al., 2009; Coello et al., 2012; Rodrigues et al., 2013). This contrasts with SNF1 and AMPK, where there is a clear correlation between activity and phosphorylation of the conserved residue in the T-loop of the catalytic subunit (Kurumbail and Calabrese, 2016; Sanz et al., 2016).

In contrast to SNF1/AMPK/SnRK1, TOR is activated in nutrient-rich conditions. Surprisingly, some canonical upstream regulators of TORC1 in yeast and mammals are absent in plants (van Dam et al., 2011; Roustan et al., 2016), suggesting a diversification of the mechanisms conveying nutrient and energy signals to TOR in photoautotrophic organisms (Xiong and Sheen, 2015). In plants, photosynthesis-derived glucose and sucrose were disclosed as potent activators of TOR signaling in the root and shoot meristem (Xiong et al., 2013; Li et al., 2017). TOR is also activated by light in the shoot apical meristem, through the action of the growth hormone auxin (Pfeiffer et al., 2016; Li et al., 2017). Auxin activates TOR in both shoots and roots, presumably through a direct interaction and activation of TOR by the small GTPase Rho-related protein 2 (ROP2) (Schepetilnikov et al., 2013, 2017; Li et al., 2017). Interestingly, ROP2 cannot be activated by glucose and is not able to replace glucose for TOR activation, indicating that TOR senses sugars either directly or through another upstream regulator (Li et al., 2017).

**Modes of action**

Downstream of SNF1/AMPK/SnRK1 activation, an energy-saving program is launched through direct regulation of key metabolic enzymes and via transcriptional and translational regulation. In plants, SnRK1 phosphorylates and inactivates key metabolic enzymes of isoprenoid biosynthesis (3-hydroxy-3-methylglutaryl CoA reductase, HMGR), sucrose synthesis (sucrose phosphate synthase, SPS), and nitrogen assimilation (nitrate reductase, NR) (MacKintosh, 1992; Ball et al., 1994; Dale et al., 1995a; McMichael et al., 1995; Douglas et al., 1997; Sugden et al., 1998b; Nukarinen et al., 2016; Robertelle et al., 2017). Members of the class II T6P synthase (TPS) proteins are also phosphorylated by SnRK1, amongst other proteins (Glinski and Weckwerth, 2005; Harthill et al., 2006; Nukarinen et al., 2016). SnRK1 shares similar substrate recognition motifs with SNF1 and AMPK (Dale et al., 1995b), and is able to phosphorylate and inactivate mammalian HMGR and acetyl-CoA carboxylase (ACC) in the same sites as AMPK (MacKintosh, 1992). Importantly, SnRK1 orchestrates a broad transcriptional reprogramming to restore energy homeostasis through down-regulation of anabolism and up-regulation of catabolism (Baena-Gonzalez et al., 2007; Baena-Gonzalez and Sheen, 2008; Sheen, 2014). More precisely, SnRK1 induces genes related to nutrient remobilization via cell wall, starch, protein, and lipid degradation, as well as autophagy and gluconeogenesis, while repressing genes related to protein synthesis, the tricarboxylic acid (TCA) cycle, and glycolysis (Baena-Gonzalez et al., 2007; Baena-Gonzalez and Sheen, 2008; Sheen, 2014). This is partly achieved through the direct phosphorylation and regulation of key transcription factors, such as bZIP63, by SnRK1 (Mair et al., 2015; Droge-Laser and Weiste, 2018). The transcriptional changes triggered by SnRK1 are remarkably similar to those induced by a variety of energy-limiting conditions (Baena-Gonzalez et al., 2007; Baena-Gonzalez and Sheen, 2008), reinforcing the role of SnRK1 as an energy sensor and central transcriptional regulator. Furthermore, this low-carbon transcriptional signature is conserved between woody and herbaceous species, highlighting its importance as a general mechanism to support essential maintenance functions and to reduce growth (Tarancon et al., 2017; Martin-Fontecha et al., 2018).

Under favorable energy conditions, TOR activation drives energy-consuming anabolic processes, such as protein synthesis and cell division, while repressing catabolism and nutrient remobilization, showing a clear reverse signature from the SNF1/AMPK/SnRK1 signaling outcome. Like SnRK1, TOR achieves this through phosphorylation of direct targets and through transcriptional reprogramming, affecting metabolic-, cell cycle-, signaling-, and transcription-related genes, amongst others. Phosphorylation and activation of E2F transcription factors by TOR promotes a transcriptional regulation of the heterotrophic to photoautotrophic transition and induction of root (E2Fa) and shoot (E2Fa/b) meristem activity (Xiong et al., 2013; Li et al., 2017).

In addition, TOR is widely recognized for its positive regulation of translation, and this function appears to be conserved in plants (Schepetilnikov and Ryabova, 2018). Translational regulation exerted by mammalian TORC1 strongly depends on the direct phosphorylation of the S6 kinase (S6K) and its substrate, the S6 subunit of the ribosomal complex (RPS6), as well as on the phosphorylation of the translational regulators 4E-binding proteins (4EBPs), which dissociate from eIF4E allowing translation initiation (Fonseca et al., 2014).
Some TORC1 direct targets, such as S6K and Tap42/α4, and downstream effectors, such as RPS6, are also found in plant genomes, suggesting conserved functions (Mahfouz et al., 2006; Ahn et al., 2011; Dobrenel et al., 2016a; Schepetilnikov and Ryabova, 2018). Indeed, Tap46, a regulatory subunit of protein phosphatase 2A in plants, has been associated with general translation and growth promotion, similar to its yeast and mammalian counterpart Tap42/α (Ahn et al., 2006; Schepetilnikov et al., 2011, 2013; Xiong and Sheen, 2012; Dobrenel et al., 2016b; Dong et al., 2017; Li et al., 2017; Wang et al., 2018). In addition to these targets, TOR stimulates translation via the phosphorylation of the translation initiation factor eIF3h, promoting translation reinitiation after upstream ORFs (Schepetilnikov et al., 2013). Additionally, TOR was shown to enter the nucleus and directly interact with the promoters of rRNA genes to induce rRNA expression required for ribosome biogenesis (Ren et al., 2011).

Co-regulation

In yeast and animals, SNF1 and AMPK are established upstream negative regulators of TOR. In animals, AMPK phosphorylates and activates the tuberous sclerosis complex 2 (TSC2) GTPase-activating protein, which is a major inhibitor of the TORC1 pathway (Inoki et al., 2003), but that is absent in plants (van Dam et al., 2011). Moreover, AMPK phosphorylates RAPTOR, resulting in 14–3–3 binding and inhibition of TORC1 kinase activity (Gwinn et al., 2008). In yeast, SNF1 is required for repressing TORC1 in response to glucose starvation (Hughes Hallett et al., 2014), and specific inhibition of SNF1 with bulky ATP analogs results in reduced phosphorylation of TORC1 subunits (Braun et al., 2014). However, although inhibition of TORC1 appears to involve phosphorylation of the RAPTOR ortholog in yeast, KOG1, the site of phosphorylation, and its downstream effectors, such as RPS6, are also found in plant genomes (Baena-Gonzalez et al., 2007; Xiong et al., 2013; Sheen, 2014). Conversely, partial TOR deficiency causes repression of genes involved in anabolism and biosynthetic pathways, and induces genes involved in catabolism, stress, and defense processes (Deprost et al., 2007; Moreau et al., 2012; Ren et al., 2012; Caldana et al., 2013), matching a SnRK1 signature. In the hypothalamus, leptin induces an inhibitory phosphorylation of AMPKα2 by the TOR direct target S6K, evidencing that TOR signaling may reciprocally regulate the AMPK pathway in animals (Dagon et al., 2012). However, this sort of co-regulation has not yet been described in plants.

Altogether, an increasing body of evidence supports that, as in other eukaryotes, part of the SnRK1 effects in plants are probably mediated by TOR signaling inhibition, in order to integrate the plant metabolic status with adequate growth and developmental decisions. Nevertheless, it is unlikely that all SnRK1 downstream effects are TOR dependent and that TOR regulation depends exclusively on SnRK1.

SnRK1 and TOR: crosstalk under stress conditions

Being sessile, plants have to cope with unfavorable environmental conditions as varied as drought, flooding, extreme temperatures, high salinity, or attacks from various pathogens (Mittler, 2006; Im et al., 2014). Most of these conditions restrict carbon assimilation through photosynthesis and/or ATP production through respiration, overall leading to a decrease in cellular energy levels (Biswal et al., 2011; Gururani et al., 2015). Additionally, resources that could be otherwise invested in growth have to be diverted into defense and stress tolerance, further compromising energy availability. Stress also causes profound alterations in source–sink interactions, as a consequence either of loss of specific parts (e.g. reproductive organs) or of actively prioritizing the growth of others (e.g. infected tissues or roots over shoots). This can reduce energy availability in certain organs whilst increasing it in others.

Responses to biotic stress

Biotic stress derives from the interaction between plants and diverse pathogenic organisms, including viruses, bacteria, fungi and oomycetes, nematodes, herbivores, and also competing plants. Plants evolved an innate immune system composed of surveillance mechanisms and effective responses to restrain pathogens, which aim to manipulate the host and cause disease (Boller and He, 2009; Dodds and Rathjen, 2010). The proposed ‘zigzag’ model of plant–pathogen interactions encompasses two branches of the plant immune system (Jones and Dangl, 2006). As a first line of defense, plants recognize extracellular conserved pathogen-associated or microbe-associated molecular patterns (PAMPs or MAMPs), such as fungal chitin, peptidoglycans, or bacterial flagellin, through pattern recognition receptors (PRR) localized on the cell surface. PRRs are generally composed of an extracellular leucine-rich repeat (LRR) domain and an intracellular kinase domain that upon recognition and activation launches pattern-triggered immunity (PTI). PTI includes a broad array of plant responses such as media alkalization, deposition of callose, reactive oxygen...
species (ROS) burst, phosphorylation of mitogen-activated protein kinases (MAPKs), synthesis of defense hormones, and activation of defense genes (Boller and He, 2009; Dodds and Rathjen, 2010). In a second line of defense, plants respond to pathogen virulence factors, delivered into the host cell to counteract PTI and promote infection. Many of these pathogen effector proteins are recognized by intracellular nucleotide-binding (NB)-LRR receptors in the plant that boost a strong effector-triggered immunity (ETI) response. This can include activation of resistance (R)-genes and programmed cell death and autophagy as pro-survival mechanisms (Jones and Dangl, 2006; Dodds and Rathjen, 2010; Cui et al., 2015). Recently, signaling molecules such as sugars and hormones, protein kinases, and transcription factors were implicated in the switch between growth and defense upon pathogen perception (Wang et al., 2016; Filipe et al., 2018). The studies below unveil a prominent role in this switch also for SnRK1 and TOR, which contribute to balancing defense and growth in response to diverse biotic stresses.

Plant–virus interactions
Viruses manipulate plant defenses through multifunctional virulence factors in order to suppress antiviral gene silencing and hijack the transcriptional and translational machineries to sustain propagation.

Geminiviruses are a large family of ssDNA viruses that cause serious diseases in many crops. In tobacco, silencing SnRK1α caused enhanced susceptibility to geminivirus infection, whereas overexpressing SnRK1α led to increased resistance, albeit penalizing plant growth (Hao et al., 2003). Geminivirus pathogenicity determinants AL2 and L2 proteins cause enhanced susceptibility when expressed in transgenic tobacco plants (Sunter et al., 2001), presumably by directly inactivating SnRK1 (Hao et al., 2003) and adenosine kinase (ADK) (Wang et al., 2003). ADK targeting by viral effectors could be an indirect mechanism of SnRK1 down-regulation since ADK phosphorylates adenosine to generate 5'-AMP which in turn protects phosphorylated SnRK1 from inactivation by phosphatases (Sugden et al., 1999a). Furthermore, SnRK1 and ADK interact in vivo, possibly regulating each other (Mohannath et al., 2014). A repressive effect of AL2 on SnRK1 activity is further supported by overlapping transcriptional changes induced by AL2 overexpression and SnRK1α silencing (Liu et al., 2014). Geminivirus AL1 and AL2/C2 proteins also interact with the SnRK1 upstream kinases SnAKs/GRIKs, that accumulate in young tissues and infected leaves (Shen and Hanley-Bowdoin, 2006; Shen et al., 2009). On the other hand, SnRK1 has been shown to phosphorylate several viral proteins presumably to counteract viral infection. SnRK1 phosphorylates AL2/C2 proteins in vitro, and a virus expressing the corresponding AL2 phosphomimetic variant caused delayed symptom development and viral DNA accumulation during infection of Arabidopsis (Shen et al., 2014). More recently, SnRK1 was shown to phosphorylate a geminivirus Rep protein, impairing viral replication and thereby infection (Shen et al., 2018). The target residue is highly conserved, raising not only the possibility that SnRK1 phosphorylation of Rep is a common defense mechanism during infection, but also that this residue plays an important, as yet unknown role. In tomato plants, SnRK1 also interacts with and phosphorylates the geminivirus βC1 protein, a pathogenicity determinant and suppressor of RNA silencing, thereby attenuating disease symptoms and reducing virus infection (Shen et al., 2011, 2012). SnRK1 was also found to interact with and phosphorylate REMORIN 4.1 (REM4.1), causing its proteasomal degradation. Given that AtREM4s enhance susceptibility to geminivirus infection and that they harbor motifs characteristic of proteins involved in cell division, the authors hypothesized that SnRK1 could block cell proliferation via REM4 inhibition (Son et al., 2014).

Lastly, Tobacco mosaic virus (TMV) resistance protein N (TRPN) was retrieved as an SnRK1 interactor in an yeast two-hybrid screen from Glycine soja (Song et al., 2019).

The interaction of viral effectors with TOR in order to subvert host metabolism for viral benefit has also been described in several reports, but with an opposite outcome than when targeting SnRK1. Cauliflower mosaic virus (CaMV) TAV effector protein binds to TOR, promoting TOR activity and S6K1 phosphorylation in planta, which is critical for translation re-initiation and viral success. In agreement with this, TOR-deficient plants are resistant to CaMV infection (Schepetilnikov et al., 2011). As a result of TAV–TOR binding, plants infected with CaMV also became more susceptible to secondary bacterial infections, partly due to the suppression of salicylic acid (SA) production and autophagy (Zvereva et al., 2016). In a different study, TOR RNAi lines were partially resistant to the potyviruses Watermelon mosaic virus (WMV) and Turnip mosaic virus (TuMV) (Ouibrahim et al., 2015). Moreover, TOR inhibition by AZD-8055 hindered WMV systemic infection and was able to cure the plants. However, Col-0 plants treated with AZD-8055 were equally susceptible to TuMV infection, suggesting that potyviruses may differ in their requirement for TOR signaling (Ouibrahim et al., 2015).

Plant–bacteria interactions
Similarly to what has been described for plant–virus interactions, also in the case of bacterial infection, high SnRK1 activity has been generally linked to resistance, whereas high TOR activity has been generally linked to susceptibility.

Gene silencing experiments have shown that SnRK1 is required for cell death associated with the hypersensitive response (HR) elicited by the effector protein AvrBs1 from Xanthomonas campestris pv. vesicatoria (Xcv) to limit infection in resistant pepper plants. Pathogenicity of Xcv depends on the effector protein AvrBsT that acts to suppress the AvrBs1–induced HR. AvrBsT interacts with SnRK1 in planta, suggesting that targeting SnRK1 might be important for HR suppression and for Xcv pathogenicity (Szczesny et al., 2010). Resistance of tomato to its pathogen Pseudomonas syringae pv. tomato (Pst) has been correlated with the induction of cell death during infection, presumably through the inactivation of the plant cell death suppressor kinase AvrPro-dependent Pto-interacting protein3 (Adi3). Adi3, on the other hand, was shown to repress SnRK1 activity through phosphorylation of the SnRK1β subunit Gal83, supporting the idea that SnRK1 promotes the HR, and providing a potential mechanism for the cell death suppression outcome of Adi3 (Avila et al., 2012).
SnRK1 has also been implicated in the resistance to *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) in rice, with SnRK1A overexpression conferring enhanced resistance but causing a negative impact on plant growth and development (Filipe et al., 2018). However, it is unclear whether this resistance is general or dependent on the PRR host sensor XA21 (Seo et al., 2011; Filipe et al., 2018).

In a complementary study, the ectopic expression of TOR and RAPTOR in rice positively regulated growth and development as expected, but increased susceptibility to *Xoo*. In agreement with this, TOR inhibition with rapamycin activated many defense-related genes. The authors also showed that TOR suppresses PTI at least in part by counteracting signaling by the defense hormones SA and jasmonic acid (JA). Inhibiting TOR chemically or genetically, using *RAPTOR1* RNAi lines and the *raptor1-1* mutant, resulted in decreased susceptibility to *Xoo*, highlighting that, as opposed to SnRK1, TOR is a negative regulator of disease resistance to *Xoo* in rice (De Vleesschauwer et al., 2018).

In Arabidopsis, TOR RNAi lines were more resistant to *Pseudomonas syringae* DC3000 (*Pst*), probably due to a faster and/or more effective defense response, whereas TOR overexpressors responded similarly to Col-0 to infection by *Pst* (Meteignier et al., 2017). However, TOR activity is not always induced by pathogens, as shown for *Ralstonia solanacearum* whose AWR5 effector inhibits TOR signaling (Popa et al., 2016). The reason behind this strategy is not clear but may be related to the increased nitrogen availability provided by autophagy and inhibition of protein synthesis.

Plant–fungi and oomycete interactions
SnRK1 and TOR have also been shown to be involved in defense responses against fungi and oomycetes, again in most cases associating high SnRK1 activity with enhanced resistance and high TOR activity with enhanced susceptibility.

In rice, *SnRK1A* up-regulation and down-regulation led to resistance and susceptibility, respectively, against the rice blast pathogen *Magnaporthe oryzae*. Similar outcomes of resistance were also observed for the necrotrophic pathogens *Cochliobolus miyabeanus* and *Rhizoctonia solani* (Filipe et al., 2018). Furthermore, in response to inoculation with the blast fungus *Pyricularia oryzae*, SnRK1A overexpressors triggered a higher induction of SA- and JA-responsive genes associated with defense (Filipe et al., 2018). In a complementary study, overexpression of TOR and RAPTOR1 in rice rendered plants more susceptible to the necrotrophic fungi *C. miyabeanus* and *R. solani*. Likewise, TOR overexpressors and *raptor1-1* mutants showed increased and decreased susceptibility, respectively, against the necrotrophic leaf fungus *Botrytis cinerea* and the soil-borne vascular pathogen *Verticillium longisporum*. Inhibition of TOR activity with rapamycin had similar outcomes, strongly reducing infection by the biotrophic oomycete *Bremia lactucae* in lettuce seedlings (De Vleesschauwer et al., 2018). The authors also showed that TOR is a negative regulator of PTI. Pre-treatment of wild-type plants with chitin for 8 h triggered PTI, allowing a reduction of 30% in the diseased leaf area when plants were subsequently inoculated with *C. miyabeanus*. A similar reduction in disease severity was obtained in a *RAPTOR* RNAi background but not in TOR-overexpressing plants, where the chitin-inducible resistance was almost completely suppressed (De Vleesschauwer et al., 2018).

Recently, down-regulation of TOR signaling in Arabidopsis, using either mutants impaired in TOR, or wild-type plants treated with TOR inhibitors, was shown to increase resistance to *Fusarium graminearum* (Aznar et al., 2018), the causal agent of the *Fusarium* head blight disease in wheat. SnRK1 has also been indirectly implicated in the resistance to *Fusarium* (Perochon et al., 2015), through its interaction in planta with the wheat *Fusarium Resistance Orphan Gene* (*FROG*), a factor induced in response to the fungal virulence factor mycotoxin deoxynivalenol (DON) and that enhances resistance to DON and to *F. graminearum*.

The Arabidopsis SnRK1βγ subunit interacts with HSPRO1/2 proteins involved in plant defense and possibly in the leaf senescence program (Gissot et al., 2006). Infection of *Nicotiana attenuata* seedlings with the fungus *Piriformospora indica* promotes seedling growth through a process negatively regulated by plant HSPRO and by SnRK1β subunit Gal83. Since *N. attenuata* seedlings silenced in *Gal83* and HSPRO, either independently or simultaneously, display enhanced growth to a similar extent upon infection, it is likely that HSPRO acts via SnRK1 in the seedling growth promoted by *P. indica* (Schuck et al., 2012, 2013).

Additional potential connections to plant immunity come from the interaction of SnRK1 catalytic subunits with STKR1, a protein whose overexpression affects genes related to systemic acquired resistance (SAR) and confers resistance towards the biotrophic oomycete *Hyaloperonospora arabidopsidis* (*Hpa*) (Nietzsche et al., 2016, 2018). In a different study, TOR silencing through RNAi increased resistance to *Hpa* in Arabidopsis, whereas overexpression of TOR rendered plants significantly more susceptible to this pathogen (Meteignier et al., 2017).

Other plant biotic interactions
In response to herbivory, *N. attenuata* plants increase the allocation of sugars to roots (Schwachtje et al., 2006). This strategy of carbon diversion to a less vulnerable location within the plant is regulated by the SnRK1β subunit Gal83, namely through *Gal83* transcriptional down-regulation in source leaves upon herbivore attack, and seems to be independent of JA signaling. Plants with enhanced root reserves have prolonged reproduction and have delayed senescence (Schwachtje et al., 2006).

Interestingly, microbes synthesize signaling molecules such as sugars and hormones that may interfere with the SnRK1–TOR axis in the host, inhibiting SnRK1 and/or promoting TOR signaling, and thereby modulating growth and defense to promote infection. Pathogens such as *Pseudomonas syringae* and *Agrobacterium tumefaciens* can directly synthesize auxin or manipulate auxin synthesis and signaling in plants to suppress plant defense (Huot et al., 2014). Auxin promotes growth-related processes that aid pathogen proliferation and indirectly interfere with SA-mediated defense (Naseem and Dandekar, 2012). As previously mentioned, auxin activates TOR signaling in plants (Schepetilnikov et al., 2013, 2017; Li et al., 2017), and TOR reciprocally impacts auxin signaling (Dong et al., 2015; Deng et al., 2016). Likewise, bacteria and fungi produce
trehalose, and its biosynthesis and metabolism play important roles in their pathogenicity (Fernandez et al., 2010; Hulsmans et al., 2016), possibly involving the inhibition of SnRK1 by the trehalose precursor T6P (Zhang et al., 2009; Nunes et al., 2013b; Lunn et al., 2014). Notwithstanding, trehalose is also considered a plant stress protector that elicits plant defense mechanisms (Fernandez et al., 2010).

Recently, a role in growth regulation was also shown for other defense-related metabolites, glucosinolates (Malinovsky et al., 2017). This study demonstrated that the aliphatic glucosinolate 3-hydroxypropylglucosinolate arrests root growth and development via TOR signaling inhibition, unveiling a role for this metabolite in the coordination of plant defense and growth.

Responses to abiotic stress
SnRK1 signaling is now widely accepted to be activated under conditions that compromise energy production, such as unexpected darkness and extended night treatments, herbicide feeding, or hypoxia (Baena-Gonzalez et al., 2007; Cho et al., 2012; Rodrigues et al., 2013; Mair, et al., 2015; Cho, et al., 2016; Nukarinen et al., 2016; Weiste et al., 2017). This has been shown using the induction of SnRK1 marker genes or target phosphorylation as readout of SnRK1 activity (Baena-Gonzalez et al., 2007; Cho, et al, 2012; Mair, et al., 2015; Cho, et al., 2016; Nukarinen et al., 2016). Using in vitro kinase activity assays for the synthetic peptides SAMS, AMARA, or SPS, SnRK1 kinase activity was also shown to increase in response to low nutrients in Arabidopsis seedlings (Ananieva et al., 2008), bean seeds (Coello and Martinez-Barajas, 2014a, 2016), and maize kernels (Bledsoe et al., 2017). Accordingly, it increased in response to non-metabolizable glucose analogs such as 2-deoxyglucose (Harthill et al., 2006), and decreased in the presence of glucose (Ananieva et al., 2008) in Arabidopsis. However, at high concentrations, glucose was reported to induce SnRK1 activity, probably as a result of the glucose-induced increase in abscisic acid (ABA) levels (Jossier et al., 2009), that in turn activate SnRK1 signaling (Rodrigues et al., 2013). In addition, Arabidopsis SnRK1 activity was reported to increase under nitrogen deficiency (Nunes et al., 2013a) and decrease under phosphate starvation (Fragoso et al., 2009).

Unlike SnRK1, TOR activity is usually down-regulated under stress conditions that restrict sugar availability. For example, in root meristems of Arabidopsis seedlings, growing in sugar-free liquid medium, TOR activity assessed by S6K phosphorylation at T-449 is inhibited upon starvation and darkness, but can be restored by exogenous glucose supply (Xiong et al., 2013; Li et al., 2017). In the shoot meristem of plants grown under the same conditions, TOR activity is equally inhibited, but, to restore it, both glucose and light are required (Li et al., 2017). A decrease in S6K phosphorylation or activity was also reported to occur in Arabidopsis in response to osmotic stress (Mahfouz et al., 2006), cold stress (Wang et al., 2017), and sulfate depletion (Dong et al., 2017). Interestingly the effect of sulfate was not direct but was transduced into the TOR pathway through down-regulation of glucose metabolism. Accordingly, glucose feeding ameliorated Arabidopsis sulfur deficiency phenotypes (Dong et al., 2017) and it was proposed that this regulation of TOR could be mediated through SnRK1. Curiously, the ability of rapamycin to inhibit TOR via AtFKBP12 binding was shown to be enhanced under hypoxic conditions (Deng et al., 2016).

In line with the regulation of SnRK1 and TOR by multiple stress conditions, plants with manipulated SnRK1 or TOR activities display altered abiotic stress resistance. Overall, higher SnRK1 levels lead to higher stress resistance, while lower SnRK1 levels result in higher stress sensitivity; however, growth or developmental effects are often observed in both cases. For example, overexpression of SnRK1α1 in Arabidopsis enhanced seedling resistance to starvation, but delayed development (Baena-Gonzalez et al., 2007). Similar observations were reported recently, also in Arabidopsis, with SnRK1α1 overexpressors displaying higher resistance not only to carbon, but also to nitrogen starvation (Chen et al., 2017). Under heat stress, SnRK1 phosphorylation of FUSCA3 (Tsai and Gazzarrini, 2012) is important for seed formation, as fusi3 mutants complemented with a FUSCA3 version mutated in the SnRK1-targeted phosphorylation site displayed higher abortion rates and poor progeny growth (Chen et al., 2017). Arabidopsis seedlings overexpressing rice or Arabidopsis SnRK1α1 also showed higher resistance to submergence, in part through direct SnRK1α1 binding to chromatin to regulate gene expression (Cho et al., 2012). An independent study confirmed these results, reporting higher recovery rates after submergence and also after drought (Chen et al., 2017). Consistent with this, plants with reduced SnRK1 activity caused by overexpression of a kinase-dead SnRK1α1 variant were more susceptible to submergence (Cho et al., 2016). In potato, silencing of the β subunit Gal83 increased salt sensitivity and yielded plants with stunted roots with smaller cells, and a larger number of tubers, albeit of reduced size (Lovas et al., 2003). When SnRK1 was silenced in pea seeds through a similar approach, gene expression also indicated higher stress susceptibility, and, again, SnRK1-silenced seeds had lower fresh weight than the wild type (Rachuk et al., 2006). In Arabidopsis, transient silencing of SnRK1 through virus-induced gene silencing generated dwarf plants unable to induce starvation genes upon hypoxia, dark, or herbicide treatments (Baena-Gonzalez et al., 2007).

In agreement with the increased stress tolerance of SnRK1α1 overexpressors, raptorB mutants, with reduced TOR activity, scored higher survival and recovery after a prolonged darkness treatment, although the authors attributed this to the higher starch accumulation of the mutants (Salem et al., 2018). Also, transgenic plants overexpressing the TOR target S6K showed increased sensitivity to osmotic stress (Mahfouz et al., 2006).

Curiously, and unlike what generally happens in biotic stress responses, numerous studies report enhanced abiotic stress resistance for plants with high TOR activities. Plants overexpressing TOR were more resistant to osmotic stress and developed longer roots, whereas TOR silencing had the opposite effect (Deprost et al., 2007). In the TOR-impaired lst8 mutant, the synthesis of osmoprotectants was decreased, which could be one of the reasons why TOR RNAi lines were more sensitive (Moreau et al., 2012). In agreement with this, germination assays of raptor1b mutants under osmotic or salt stress also
revealed higher sensitivity (Salem et al., 2017) and TOR RNAi lines displayed higher sensitivity to nitrogen starvation (Liu and Bassham, 2010). Recently, transgenic rice plants overexpressing TOR also displayed higher growth, yield potential, and life span under water deficit (Bakshi et al., 2017). Chlorophyll degradation in these TOR overexpressors was slower than in the wild type, and expression of specific stress genes was highly up-regulated (Bakshi et al., 2017). Likewise, TOR activity is required for cold stress responses, as TOR RNAi lines were more sensitive to cold stress (Dong et al., 2019). In line with these observations is the recent report of higher grain yield and higher drought resistance of spring wheat plants treated with synthetic and light-activated T6P precursors (Griffiths et al., 2016; Paul et al., 2018). Presumably one of the mechanisms through which these precursors conferred drought tolerance was SnRK1 inhibition, which could subsequently activate TOR (Griffiths et al., 2016).

One of the best studied mechanisms through which SnRK1 and TOR contribute to stress responses is autophagy, an evolutionarily conserved process in which eukaryotic cells degrade damaged or unnecessary cellular components, generating new building blocks and energy sources that are essential during stress (Ustun et al., 2017; Avin-Wittenberg, 2018). Similarly to the situation in yeast and mammals, SnRK1 and TOR have been shown to affect autophagy in plants in a positive and negative manner, respectively (Alers et al., 2012; Pu et al., 2017b; Soto-Burgos et al., 2018). Under normal growth conditions, overexpression of the SnRK1α1 catalytic subunit was sufficient to induce constitutive autophagy (Chen et al., 2017; Soto-Burgos and Bassham, 2017), and decreasing TOR activity chemically or genetically had similar effects (Liu and Bassham, 2010; Pu et al., 2017a; Salem et al., 2018). Importantly, when both SnRK1 and TOR activities were simultaneously increased, constitutive autophagy was not induced or was induced to a lower extent, whereas decreasing both SnRK1 and TOR activities resulted in constitutive autophagy; this suggests that SnRK1 is upstream of TOR in autophagy regulation (Soto-Burgos and Bassham, 2017). Activation of autophagy under salt, osmotic, starvation, oxidative, and endoplasmic reticulum (ER) stress requires SnRK1, as snrk1α1 mutants were impaired in autophagy under these conditions (Soto-Burgos and Bassham, 2017). Interestingly, increasing TOR activity by overexpressing TOR or by auxin treatment decreased autophagy levels in response to all conditions except oxidative and ER stress (Pu et al., 2017a), suggesting that SnRK1 can activate autophagy in both a TOR-dependent and -independent manner (Soto-Burgos and Bassham, 2017). In addition, genetic analyses show that the higher tolerance of SnRK1α1 overexpressors to drought, carbon and nitrogen starvation, and submergence is autophagy dependent (Chen et al., 2017). Interestingly, autophagy seems to be important also for degrading positive growth regulators. The brassinosteroid signaling transcription factor BZR1 is stabilized by TOR in the presence of sugars, and is degraded under low carbon conditions in an autophagy-dependent manner, thereby allowing the integration of carbon availability with growth-promoting hormonal programs (Zhang et al., 2016).

ABA signaling is another important pathway through which SnRK1 and TOR can affect stress responses. Links between SnRK1 and ABA signaling have been established in numerous studies. Arabidopsis SnRK1α1 overexpressors are ABA hypersensitive (Jossier et al., 2009), and SnRK1 has been proposed to mediate ABA effects during seed maturation and filling in pea, possibly through ABI3 (Radchuk et al., 2006, 2010). Also, SnRK1 purified from spinach leaves or immunoprecipitated from Arabidopsis seedling extracts was able to phosphorylate in vitro ABI5 and AREBP transcription factor peptides containing two conserved SnRK1 phosphorylation motifs (Zhang et al., 2008; Bitrian et al., 2011). Importantly, ABA can activate SnRK1 signaling through the action of PP2Cs that act as negative regulators of SnRK1. Consequently, SnRK1 and ABA induce largely overlapping transcriptional responses (Rodrigues et al., 2013). In contrast, TOR overexpressors in rice display ABA insensitivity during germination (Bakshi et al., 2017), whereas Arabidopsis raptor1b mutants are hypersensitive (Salem et al., 2017). Very recently, a balance between growth and stress responses was reported to be established through the reciprocal regulation between TOR and ABA stress signaling. Under favorable conditions, TOR phosphorylates the PYL ABA receptors, precluding their association with ABA and PP2C phosphatases, and leading to the inactivation of SnRK2 kinases and of stress responses. Conversely, when ABA signaling is induced under stress conditions, TOR is inactivated in an SnRK2 kinase-dependent manner to repress growth (Rosenberger and Chen, 2018; Wang et al., 2018).

**Concluding remarks**

The concept of growth–defense trade-offs is widely accepted and very intuitive; under stress, plants need to divert available energy from growth to defense in order to survive. For plants, this trade-off is important for survival, but for agriculture it is the cause of vast yield losses. Given the relevance of SnRK1 and TOR for balancing stress responses with growth and development, their signaling pathways could be valuable targets of manipulation aimed to increase plant performance and productivity in suboptimal environments. However, although it is increasingly clear that the SnRK1–TOR interplay contributes, at least partly, to balancing growth and defense, numerous studies also show that the influence of SnRK1 and TOR in defense and growth outputs is not always straightforward (Supplementary Table S1).

Under biotic stress, the clear growth–defense trade-offs that are often observed fit well with a model in which SnRK1 and TOR play antagonistic roles. Under pathogen attack, SnRK1 promotes broad disease resistance and plant fitness at the expense of growth, whilst TOR promotes growth and proliferation, compromising immunity and rendering plants more susceptible to a broad range of pathogens. Accordingly, SnRK1 gain- and TOR loss-of-function plants tend to be more resistant, whereas TOR gain- and SnRK1 loss-of-function plants tend to be more susceptible. Several pathogens target these specific pathways in the host, modulating them to inhibit SnRK1
and/or activate TOR signaling to promote infection. However, a recent study suggested that pathogens can also repress TOR signaling, implying that under certain conditions, specific pathogens, or stages of infection, TOR signaling inhibition could also benefit infection.

The situation becomes less intuitive when considering abiotic stress. Despite a few exceptions, most reports show that both SnRK1 and TOR overexpressors are more resistant than wild-type plants to abiotic stress, whereas loss-of-function mutants for both SnRK1 and TOR are more susceptible. These seemingly conflicting results, in which both SnRK1 and TOR seem to promote tolerance to abiotic stress, are harder to reconcile with their antagonist roles, but can perhaps be explained by the specific nature, intensity, and duration of the treatments applied in each study and by the strategies deployed by plants to cope with each particular treatment. Compromised TOR activity was reported to be detrimental under situations of osmotic stress (Deprost et al., 2007) and prolonged cold treatment (Dong et al., 2019), where perhaps the role of TOR in the biosynthesis of osmoprotective sugars and amino acids may be critical for survival. On the other hand, rice TOR overexpressors performed better and returned higher yields than wild-type plants after prolonged growth in limited water conditions (Bakshi et al., 2017). Although not measured in this study, such gradual drought stress is likely to alter shoot:root ratios, promoting active growth of the root system at the expense of the shoot. Under such conditions, the presumably enhanced capacity of the TOR overexpressors to develop a larger root should increase water absorption and lead to improved performance.

Altogether, while in biotic stress, growth is often associated with pathogen replication and propagation, in abiotic stress, investing resources in growth is often one of the ways to adapt. This happens, for example, under drought and several nutrient limitations, where increased root growth and altered root architecture constitute important survival strategies.

This interpretation is in line with the emerging relationship and functions of SnRK1 and TOR (Fig. 1). TOR primarily regulates growth while SnRK1 primarily regulates energy homeostasis, thereby promoting stress tolerance and defense. TOR growth-promoting effects may compromise or, in striking contrast, improve the strategies deployed by plants to cope with particular environments. On the other hand, SnRK1 senses environmental information in the form of the ‘energy status’, modulating growth according to the conditions (Baena-Gonzalez et al., 2007; Jossier et al., 2009; Cho et al., 2012). In Arabidopsis, SnRK1α1 overexpressors and SnRK1 transient loss-of-function seedlings grew, respectively, more and less than the wild type in nutrient-deprived conditions but, upon exogenous sugar supply, the growth effects were completely reversed (Baena-Gonzalez et al., 2007). Furthermore, SnRK1α1 overexpressors subjected to high trehalose concentrations that induce developmental arrest in the wild type were still able to develop.

**Fig. 1.** SnRK1 and TOR are central metabolic regulators that play largely antagonistic roles in growth and defense in plants. Under unfavorable conditions when energy production is compromised by biotic or abiotic stress factors, SnRK1 is activated to induce stress responses and defense, whilst TOR is repressed. Conversely, under optimal conditions when sufficient light, water, and nutrients are available, SnRK1 is repressed whilst TOR is activated to drive processes associated with cell proliferation and growth. Growth–defense trade-offs may result from a negative cross-regulation between SnRK1 and TOR, as well as through their independent regulation of downstream processes. Components highlighted in gray and white designate negative and positive regulators, respectively, of the indicated kinase. Color-coded upward and downward arrows designate positive and negative effects, respectively, of the corresponding kinase in downstream processes. SnRK1 (Snf1-related protein kinase 1), TOR (target of rapamycin), PP2Cs (type 2C protein phosphatases), SnAK (SnRK1 activating kinase), ROP2 (small GTPase Rho-related protein 2), SnRK2 (Snf1-related protein kinase 2).
roots and grow (Delatte et al., 2011). However, whether in these and other cases the growth outcomes of SnRK1-manipulated plants can be explained by changes in TOR activity, or the defense outcomes of TOR-manipulated plants can be explained by changes in SnRK1 activity is presently unknown.

Testing plants with manipulated levels of both complexes in similar conditions would provide useful insight on their mutual relationship. So far this has only been carried out for autophagy, in which it seems clear that SnRK1 is upstream of TOR, promoting autophagy in both a TOR-dependent and -independent manner. Interestingly, a recent study showed that *sent* mutants, impaired in the conversion of serine to O-acetylserine, exhibited defective growth under normal conditions despite having normal TOR activity outputs (Dong et al., 2017). This shows that TOR activity and growth are not always necessarily coupled.

The scenario becomes increasingly complex when considering the possibility that SnRK1 and TOR may be regulated differently in source and sink organs, as reports indicate variability in SnRK1 activity or response to sugars, possibly by associating with different interactors (Zhang et al., 2009; Piattoni et al., 2011; Coello and Martinez-Barajas, 2014). Also the opposite effects reported for sugars on SnRK1 (sometimes inhibitory and sometimes inductive) might be explained by the use of heterotrophic versus autotrophic tissues in different studies (Baena-Gonzalez and Sheen, 2008; Halford and Hey, 2009). Furthermore, TOR is mostly expressed in actively dividing tissues (Menand et al., 2002) and, therefore, the detrimental effects of lacking TOR activity might be particularly relevant in young seedlings and in conditions where systemic activity is required.

In summary, SnRK1 and TOR play important regulatory roles in fundamental growth and defense processes, but the outcome of their action is largely dependent on the conditions the plant encounters. Knowledge of the molecular mechanisms governing SnRK1 and TOR signaling is rapidly increasing as well as evidence for their cross-regulation. Since both growth and defense are required in the field to ensure maximum crop yield, an optimal fine-tuning of the SnRK1–TOR rheostat in accordance with the prevailing conditions could lead to agriculture production meeting its full potential.

**Supplementary data**

Supplementary data are available at *JXB* online.

Table S1. Biotic and abiotic stress-related phenotypes associated with altered SnRK1 and TOR signaling.

**Acknowledgements**

We apologize to the many authors whose work was not cited due to space limitations. This work was supported by the Fundação para a Ciência e a Tecnologia projects UID/Multi/04551/2013, PTDC/BIA-PLA/7143/2014, LISBOA-01-0145-FEDER-028128, and PTDC/BIA-BID/32347/2017, and by fellowships/contracts SFRH/BPD/116116/2016 (LM), SFRH/BPD/109336/2015 (AC), and IF/00804/2013 (EBG).

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