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The TOR Complex: An Emergency Switch for Root Behavior

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The TOR Complex: An Emergency Switch for Root Behavior

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Abbreviation:

TOR, Target of rapamycin; ROS, Reactive oxygen species

Abstract

Target of Rapamycin (TOR) kinase is known to be a controller of cell growth and ageing, which determines the fine balance between growth rates and energy availabilities. It has been reported that many eukaryotes express *TOR* genes. In plants, TOR signaling modifies growth and development in response to plant energy status. An example of TOR action can be found in the root apices, which are active organs that explore the soil environment via vigorous growth and numerous tropisms. The exploratory nature of root apices requires a large energy supply for signaling, as well as for cell division and elongation. In the case of negative tropisms, roots must respond quickly to avoid patches of unfavorable soil conditions, again by consuming precious energy reserves. Here we review the current findings on TOR signaling in plants and animals, and propose possible roles of this important complex in driving plant root negative tropisms, particularly during light escape and salt avoidance behavior.

Key words: root, light response, TOR, reactive oxygen species, root behavior, root tropism

TOR as a key growth regulator

"Target of rapamycin" (TOR) is a Ser/Thr kinase that is found in cells of mammals, yeast and plants to regulate eukaryotic cellular homeostasis and cell growth (Eltschinger and Loewith 2015, Xiong and Sheen 2015). TOR was initially identified as a ‘target of rapamycin’ in yeast, and it is now known that many eukaryotes, including algae, slime mold, yeasts, worms, flies, mammals and plants, are known to express TOR encoding genes. TOR principally acts as a cellular ageing factor that controls downstream signaling pathways related to cell growth (Blagosklonny 2008, Wullschleger et al. 2006). Currently, TOR signaling is emerging as an

important research topic in the field of medical sciences (e.g., cancer, type 2 diabetes, autoimmune disorders, etc.) because it is a key controller of metabolism of nutrients that regulate cell growth (Wullschleger et al. 2006, Loewith and Hall 2011). Interestingly, life span was increased by modifications of TOR complex activities in nematodes (Vellai et al. 2003), and yeasts (Kaeberlein et al. 2005); possibly due to the restriction of calories. TOR complex signaling plays a role in setting the pace of circadian rhythm in *Drosophila* (Zheng and Sehgal 2010). These findings suggest that TOR signaling coordinates the interplay between availability of nutrients, circadian clock, and cognition. Besides the role of growth control, TOR signaling is relevant also for long-term memory maintenance (Tischmeyer et al. 2003), synaptic plasticity, learning and cognition (Hoeffer and Klann 2010, Graber et al. 2013, Lipton and Sahin 2015).

Most organisms, including unicellular ones, constantly face highly variable environments with restricted energy resources. In order to maintain energy homeostasis (for plants see Tomé et al. 2014), a master switch such as the TOR complex that determines growth-or-not is highly essential for survival in nature. Reports published so far indicate that the plant TOR integrates endogenous hormonal and nutrient signaling cascades with exogenous environmental information to control plants in a very similar mode as it does in animals and humans (Dong et al. 2015; Xiong and Sheen 2015).

It was reported that TOR is activated by insulin in animal cells (Jia et al. 2004). Surprisingly in this respect, insulin stimulates growth in maize plants, while maize also expresses an insulin-related peptide (ZmIGF) and has very similar signaling pathways, linked to the TOR

complex, as in animals and humans (García Flores et al. 2001, Sánchez de Jiménez et al. 2002, Garrocho-Villegas and de Jiménez 2002, Garrocho-Villegas et al. 2013). Importantly, the polar auxin transport in plants shows many similarities to the insulin-mediated GLUT4 (Glucose Transporter Type 4) signal transduction in animals (Figure 2 in Muday and Murphy 2002). In plants, the TOR complex also emerges as a nutrient- and energy-sensing signaling system allowing plants and their roots to adjust their growth to the actual environmental situation (Tomé et al. 2014, Xiong et al. 2013, Xiong and Sheen 2014, 2015, Henriques et al. 2014). In roots, carbon-starvation induces autophagy and formation of autophagosomes via activities of Atg8 (Autophagy-Related Protein 8) protein (Sláviková et al. 2008, Michaeli and Galili 2014), as well as via glucose – TOR signaling networks (Xiong et al. 2013).

TOR as a 'brake and accelerator pedal' for root growth

In *Arabidopsis*, TOR is highly expressed in growing and proliferating tissues such as meristem, endosperm and embryo (Menand et al. 2002). TOR over-expression *Arabidopsis* line showed enhanced root growth, whereas roots of RNAi-silenced mutant lines did not grow well compared to the shoots. Interestingly, some overexpression lines showed restricted TOR expression in the roots (Deprost et al. 2007). In *Arabidopsis* roots, the TOR kinase perceives photosynthetic signals such as sucrose or glucose transported to the roots from leaves and this allows immediate enlargement of root meristems and enhanced root growth (Xiong and Sheen 2011, 2013 Xiong et al. 2013). It was shown by *Arabidopsis* transcriptome analysis that glucose-activated TOR signaling modulates relevant genes encoding root growth factor (RGF) peptides, glutathione synthesis and UPBEAT1 (UPB1) transcription factor in roots (Xiong et al. 2013, Xiong and Sheen 2013). Glutathione (GSH; γ-Glu-Cys-Gly) is well

known as an essential small molecule maintaining cellular redox status via the glutathione-ascorbate cycle that detoxifies hydrogen peroxide. *Arabidopsis* glutamate-receptor (AtGLR3.3) mediates exogenous GSH-triggered increase of cytosolic Ca^{2+} leading to the improvements of pathogen responses (Li et al. 2013). UPB1 controls the transcription level of peroxidases regulating the hydrogen peroxide-mediated balance between cell proliferation and rapid cell elongation in transition zone of the root apex (Tsukagoshi et al. 2010, Wells et al. 2010, Jie 2011). It is intriguing that these factors, which regulate cellular redox homeostasis and relevant signaling, are located downstream of the TOR signaling pathway.

In addition, TOR functions as a negative regulator of autophagy not only in yeast and mammalian cells but also in *Arabidopsis* cells (Liu and Bassham 2010, Pérez-Pérez et al. 2012). Autophagosomes are formed in root cells in instances when autophagy is promoted by stress situations (Xiong et al. 2007). In addition, autophagosomes and constitutive autophagy are also linked to cell growth and development in non-stressed root apices (Yano et al. 2007, Oh-ye et al. 2011, Merkulova et al. 2014). For a model of plant autophagy regulation via ROS and TOR signalling, see Figure 1 in Pérez-Pérez et al. (2012). This emerging model suggests that ROS molecules down-regulate TOR signaling which, in turn, would activate autophagy. In line with this model, it was reported that reduced *Arabidopsis* TOR expression by RNAi induces formation of autophagosomes in cells of root apices (Liu and Bassham 2010). Thus, root apices regulate their growth by controlling autophagy via TOR signaling. In conclusion, *Arabidopsis* TOR signaling can be viewed as major player controlling a ‘brake and accelerator pedal’, sensitively adjusting both root growth and behavior (Trewavas 2009) according to the actual energy status and environmental context.

ROS/TOR in light-induced escape tropism and salt avoidance tropism

ROS signals are broadly utilized in plants and animals as important messenger molecules released by several enzymes and biomolecules upon stress conditions. It is known that the TOR complex is activated by ROS released under stress. For example, hydrogen peroxide (Radisavljevic et al. 2004) and oxygen activates, while hypoxia inhibits TOR signaling in human cells (Arsham et al. 2003). In addition, activation of TOR signaling increases the production of ROS in cultured animal cells (Kim et al. 2005).

Plants are also known to produce and use ROS as signaling molecules to cope with specific stress environments by altering physiological conditions. Many studies showed that precise control of ROS homeostasis in root cells is essential for their growth, development and tropisms (Liszkay et al. 2004, Tsukagoshi et al. 2010, Yokawa et al. 2014a, Ma et al. 2015). It is critical for roots to be able to regulate ROS levels and distribution in response to ever-changing environments - especially during initiation of tropic responses, such as gravitropism or phototropism, which requires ROS production and signaling as an early event (Joo et al. 2001). With respect to light responses of roots, we have previously demonstrated that sudden illumination of *Arabidopsis* roots immediately produces ROS in the root apex, resulting in the promotion of root elongation (Yokawa et al. 2011). This phenomenon represents a new type of root tropism, the so-called ‘light-induced escape phototropism’ (Yokawa et al. 2013). However, the precise mechanism of how ROS species produced by light exposure stimulates root elongation is not yet clear. One possible pathway is via the actin cytoskeleton, as illumination of roots stimulates actin polymerization via the SCARE-ARP2-3 complex and COP1

signaling pathways (Daychok et al. 2011). Light perceived by root apex photoreceptors (Mo et al. 2015) is critical for this signaling cascade (Daychok et al. 2011). Moreover, in animal and yeast cells, TOR complex signaling is linked to the actin cytoskeleton (Niles and Powers, 2014; Liu et al. 2015; Rispal et al. 2015). We propose here a testable model whereby ROS-mediated TOR activation and downstream signaling pathways control both root phototropism and escape behavior, perhaps via the dynamic actin cytoskeleton (Figure 1). Furthermore, we also propose that light-induced ROS activate the TOR complex, which is known to inhibit plant autophagy (Liu and Bassham 2010). Finally, the TOR complex in plants might also control UPB1 and GSH levels, to adjust root growth to this unfavorable light condition.

Arabidopsis seedlings that are grown on transparent Petri dishes have illuminated roots, which not only affects their physiology but also whole seedling biology (Yokawa et al. 2013, Xu et al. 2013, Novák et al. 2015). We have reported that roots of young seedlings show different responses to identical salt concentration, depending on either dark or light growth conditions (Yokawa et al. 2014a). Light-induced root growth promotion depletes the limited energy status of whole seedlings, which also impacts on shoots (Novák et al. 2015). Intriguingly, it was reported that TOR signaling is activated by auxin in plants (Schepetilnikov et al. 2013, Bögre et al. 2013). Besides auxin, other plant hormones are also implicated in TOR signaling, especially in root apices (Xiong et al. 2013; reviewed by Sheen 2014; Xiong and Sheen, 2014, 2015). Such findings provide a good indicator that TOR is not only involved in ROS signaling but also in plant hormonal signaling in root apices. However, further intensive studies are needed to reveal the elusive interplay amongst all these factors in plant cells.

Here, we have briefly reviewed recent studies that have alluded to a tight relationship between TOR and light signaling, auxin responses and energy balance homeostasis in cells of root apices. For plants, it is potentially very dangerous to elongate their roots too fast, or in the wrong directions, because growing root apices consumes a lot of energy. Root respiration accounts for about 80% of the total whole-plant respiration (Brauner et al. 2014). Moreover, illumination of roots can also influence circadian rhythms of whole seedlings and plants. Importantly, seedlings with a defective circadian clock show defects in carbon / energy allocation and also aberrant root growth patterns (Yazdanbakhsh et al. 2011, Ruts et al. 2012). The evolutionary history of land plants, and their belowground roots preferring darkness (Ma et al. 2015), further implies that plants should be studied in their natural context i.e. with roots kept in darkness.

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Figure Legend

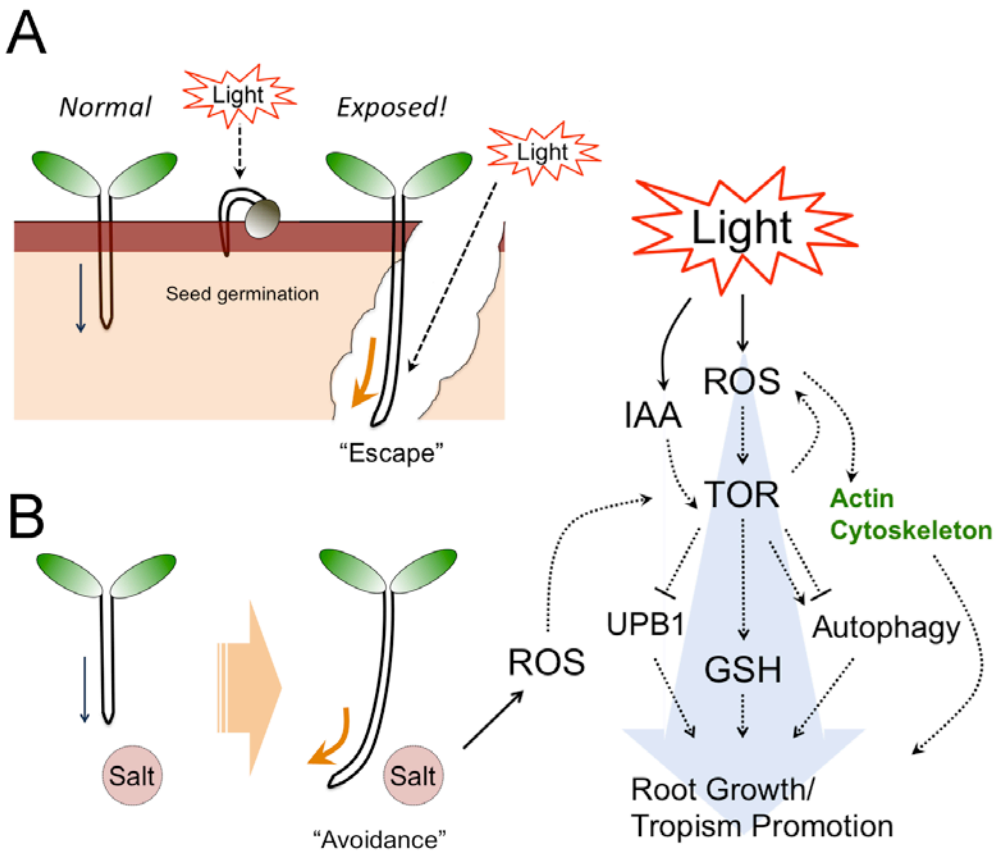

Figure 1

Hypothetical ROS-TOR mediated root escape phototropism and salt avoidance tropism. Once roots are illuminated (exposed to light) or detect a high salt environment, they start to change their growth mode and/or direction. In the scheme, stressed (exposed) roots generate ROS which then activates TOR signaling to control important factors in associated downstream pathways; including auxin (IAA), actin cytoskeleton, UPB1, GSH as well as several autophagy proteins and molecules (see text). Light exposed *Arabidopsis thaliana* roots accelerate their growth rate (root escape behavior). Moreover, light and salt exposed roots change root growth direction to avoid light or salt (root avoidance behavior or halotropism). For more details on the light-induced escape tropism and the salt-induced avoidance tropism, see Yokawa et al. (2011, 2013, 2014a) and Ma et al. (2015).