

1 **Title:**

2 The TOR Complex: An Emergency Switch for Root Behavior

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18 **The TOR Complex: An Emergency Switch for Root Behavior**

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24

25 **Abbreviation:**

26 TOR, Target of rapamycin; ROS, Reactive oxygen species

27

28 **Abstract**

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

40

41

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

43

44 ***TOR as a key growth regulator***

45 "Target of rapamycin" (TOR) is a Ser/Thr kinase that is found in cells of mammals, yeast and
46 plants to regulate eukaryotic cellular homeostasis and cell growth (Eltschinger and Loewith
47 2015, Xiong and Sheen 2015). TOR was initially identified as a 'target of rapamycin' in yeast,
48 and it is now known that many eukaryotes, including algae, slime mold, yeasts, worms, flies,
49 mammals and plants, are known to express TOR encoding genes. TOR principally acts as a
50 cellular ageing factor that controls downstream signaling pathways related to cell growth
51 (Blagosklonny 2008, Wullschleger et al. 2006). Currently, TOR signaling is emerging as an

52 important research topic in the field of medical sciences (e.g., cancer, type 2 diabetes,
53 autoimmune disorders, etc.) because it is a key controller of metabolism of nutrients that
54 regulate cell growth (Wullschleger et al. 2006, Loewith and Hall 2011). Interestingly, life span
55 was increased by modifications of TOR complex activities in nematodes (Vellai et al. 2003), and
56 yeasts (Kaeberlein et al. 2005); possibly due to the restriction of calories. TOR complex
57 signaling plays a role in setting the pace of circadian rhythm in *Drosophila* (Zheng and Sehgal
58 2010). These findings suggest that TOR signaling coordinates the interplay between
59 availability of nutrients, circadian clock, and cognition. Besides the role of growth control,
60 TOR signaling is relevant also for long-term memory maintenance (Tischmeyer et al. 2003),
61 synaptic plasticity, learning and cognition (Hoeffler and Klann 2010, Graber et al. 2013,
62 Lipton and Sahin 2015).

63

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

71

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

75 complex, as in animals and humans (García Flores et al. 2001, Sánchez de Jiménez et al. 2002,
76 Garrocho-Villegas and de Jiménez 2002, Garrocho-Villegas et al. 2013). Importantly, the
77 polar auxin transport in plants shows many similarities to the insulin-mediated GLUT4
78 (Glucose Transporter Type 4) signal transduction in animals (Figure 2 in Muday and Murphy
79 2002). In plants, the TOR complex also emerges as a nutrient- and energy-sensing signaling
80 system allowing plants and their roots to adjust their growth to the actual environmental situation
81 (Tomé et al. 2014, Xiong et al. 2013, Xiong and Sheen 2014, 2015, Henriques et al. 2014). In
82 roots, carbon-starvation induces autophagy and formation of autophagosomes via activities of
83 Atg8 (Autophagy-Related Protein 8) protein (Sláviková et al. 2008, Michaeli and Galili 2014), as
84 well as via glucose – TOR signaling networks (Xiong et al. 2013).

85

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

87 In *Arabidopsis*, TOR is highly expressed in growing and proliferating tissues such as meristem,
88 endosperm and embryo (Menand et al. 2002). TOR over-expression *Arabidopsis* line showed
89 enhanced root growth, whereas roots of RNAi-silenced mutant lines did not grow well
90 compared to the shoots. Interestingly, some overexpression lines showed restricted TOR
91 expression in the roots (Deprost et al. 2007). In *Arabidopsis* roots, the TOR kinase perceives
92 photosynthetic signals such as sucrose or glucose transported to the roots from leaves and this
93 allows immediate enlargement of root meristems and enhanced root growth (Xiong and Sheen
94 2011, 2013 Xiong et al. 2013). It was shown by *Arabidopsis* transcriptome analysis that
95 glucose-activated TOR signaling modulates relevant genes encoding root growth factor
96 (RGF) peptides, glutathione synthesis and UPBEAT1 (UPB1) transcription factor in roots
97 (Xiong et al. 2013, Xiong and Sheen 2013). Glutathione (GSH; γ -Glu-Cys-Gly) is well

98 known as an essential small molecule maintaining cellular redox status via the
99 glutathione-ascorbate cycle that detoxifies hydrogen peroxide. *Arabidopsis*
100 glutamate-receptor (AtGLR3.3) mediates exogenous GSH-triggered increase of cytosolic
101 Ca²⁺ leading to the improvements of pathogen responses (Li et al. 2013). UPB1 controls the
102 transcription level of peroxidases regulating the hydrogen peroxide-mediated balance between
103 cell proliferation and rapid cell elongation in transition zone of the root apex (Tsukagoshi et al.
104 2010, Wells et al. 2010, Jie 2011). It is intriguing that these factors, which regulate cellular
105 redox homeostasis and relevant signaling, are located downstream of the TOR signaling
106 pathway.

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

121

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

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

129

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

144 signaling pathways (Daychok et al. 2011). Light perceived by root apex photoreceptors (Mo
145 et al. 2015) is critical for this signaling cascade (Daychok et al. 2011). Moreover, in animal
146 and yeast cells, TOR complex signaling is linked to the actin cytoskeleton (Niles and Powers,
147 2014; Liu et al. 2015; Rispal et al. 2015). We propose here a testable model whereby
148 ROS-mediated TOR activation and downstream signaling pathways control both root
149 phototropism and escape behavior, perhaps via the dynamic actin cytoskeleton (Figure 1).
150 Furthermore, we also propose that light-induced ROS activate the TOR complex, which is
151 known to inhibit plant autophagy (Liu and Bassham 2010). Finally, the TOR complex in
152 plants might also control UPB1 and GSH levels, to adjust root growth to this unfavorable
153 light condition.

154

155 *Arabidopsis* seedlings that are grown on transparent Petri dishes have illuminated roots, which
156 not only affects their physiology but also whole seedling biology (Yokawa et al. 2013, Xu et al.
157 2013, Novák et al. 2015). We have reported that roots of young seedlings show different
158 responses to identical salt concentration, depending on either dark or light growth conditions
159 (Yokawa et al. 2014a). Light-induced root growth promotion depletes the limited energy status
160 of whole seedlings, which also impacts on shoots (Novák et al. 2015). Intriguingly, it was
161 reported that TOR signaling is activated by auxin in plants (Schepetilnikov et al. 2013, Bögre et
162 al. 2013). Besides auxin, other plant hormones are also implicated in TOR signaling, especially
163 in root apices (Xiong et al. 2013; reviewed by Sheen 2014; Xiong and Sheen, 2014, 2015).
164 Such findings provide a good indicator that TOR is not only involved in ROS signaling but also
165 in plant hormonal signaling in root apices. However, further intensive studies are needed to
166 reveal the elusive interplay amongst all these factors in plant cells.

167

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

179

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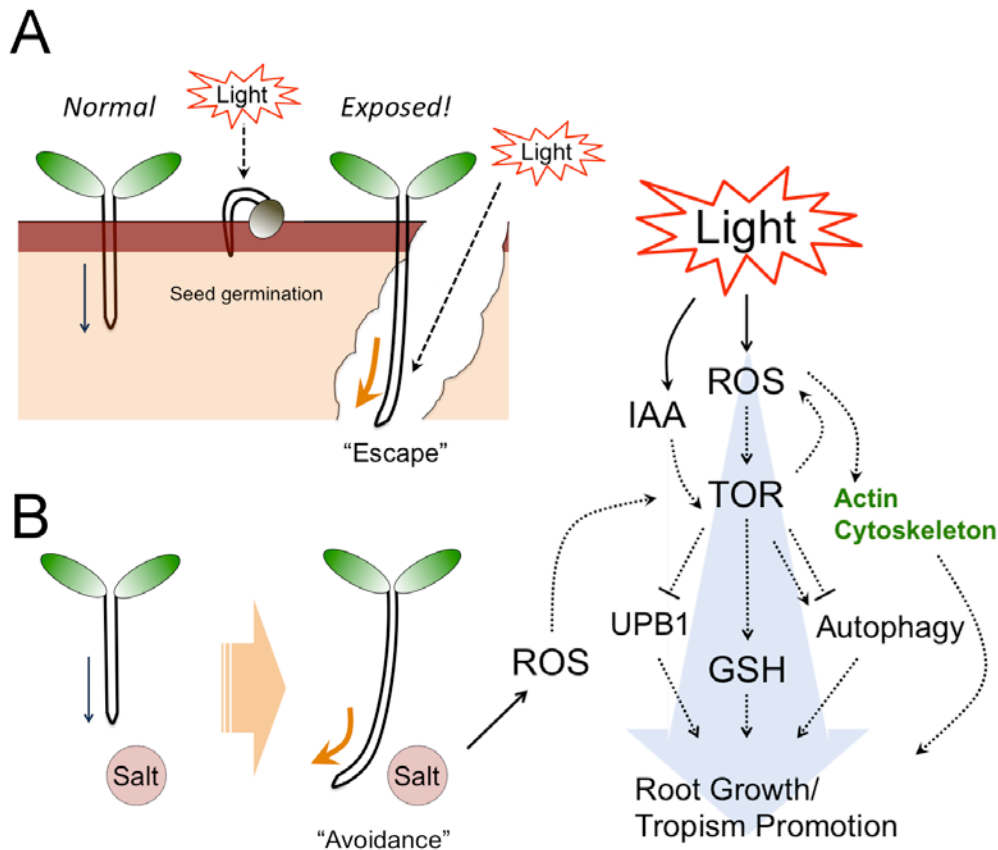
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396 **Figure Legend**



397

398 **Figure 1**

399 **Hypothetical ROS-TOR mediated root escape phototropism and salt avoidance tropism.**

400 Once roots are illuminated (exposed to light) or detect a high salt environment, they start to

401 change their growth mode and/or direction. In the scheme, stressed (exposed) roots generate

402 ROS which then activates TOR signaling to control important factors in associated

403 downstream pathways; including auxin (IAA), actin cytoskeleton, UPB1, GSH as well as

404 several autophagy proteins and molecules (see text). Light exposed *Arabidopsis thaliana* roots

405 accelerate their growth rate (root escape behavior). Moreover, light and salt exposed roots

406 change root growth direction to avoid light or salt (root avoidance behavior or halotropism). For

407 more details on the light-induced escape tropism and the salt-induced avoidance tropism, see

408 Yokawa et al. (2011, 2013, 2014a) and Ma et al. (2015).