Adjusting boron transport by two-steps tuning of BOR1 levels

Boron is an essential plant micronutrient with the narrowest optimal range. Under neutral pH, boron takes form of an uncharged boric acid that can freely penetrate the membranes. While Boron plays an important role in cross-linking cell wall components, boron starvation and toxicity affect various metabolic- and physiological processes beyond cell wall reinforcement (Reid, 2010). Controlling boron transport is therefore in the best interest of a developing plant. Under boron starvation, boron uptake and mobilization towards shoot is facilitated by NIP5;1 and BOR1. NIP5;1 is only expressed in the outer root layer, enhancing permeability of boron and passive boron uptake from the soil (Takano et al., 2010). BOR1 is a boron efflux transporter expressed in multiple cell layers of the root. In the endodermis, BOR1 polar localization towards the vasculature ensures efficient boron mobilization to the shoot (Takano et al., 2010). When boron levels are sufficient, BOR1 undergoes ubiquitination and degradation, reducing boron concentrations in the shoot (Kasai et al., 2011). However, even lines expressing BOR1 with impaired ubiquitination site (K590A), the levels were observed to decrease under boron toxic conditions, suggesting the existence of yet another mechanism controlling BOR1 levels. The paper by Aibara and colleagues (2018) published in the current issue of Plant Physiology, describes a new mechanism of BOR1 regulation by its own 5’UTR.

Four untranslated Open Reading Frames (uORFs) were identified in the 5’UTR of BOR1 mRNA. Using constructs truncated in the 5’UTR and replacing uORF’s START codon AUG into AAG, the two uORFs closest to the START codon were observed to have a role in regulating boron dependent activity of the reporter genes. The boron-dependent effect of 5’UTR on reporter-gene activity was also observed in vitro using wheat germ extract. Those results suggested that high levels of boron might directly interfere with the translation mechanism of BOR1. Two uORFs closest to the START codon were observed to affect the re-initiation rate of translation, as shortening the distance between last uORF and main ORF abolished boron dependent activity of reported gene. Toxic levels of boric acid might reduce formation of ternary complexes (TC), composed of GTP bound eukaryotic Initiation Factor 2 (eIF2) and methionine bound tRNA. In yeast, toxic boron conditions enhanced phosphorylation of α-subunit of eukaryotic Initiation Factor 2 (eIF2), reducing formation of TCs that are necessary for reinitiating translation (Uluisik et al., 2011). In the toxic boron conditions, presumably when the levels of TC are low, the distance between the last two uORFs and the BOR1 START codon prevent the reassembly of the translational machinery and reduce translation of BOR1. This mechanism of boron dependent-translational repression is, thus far, unique to BOR1 and possibly to its orthologues in other species. Other boron transporters regulated by 5’UTR include NIP5;1, but rely on ribosome stalling at uORF (Tanaka et al., 2016), which was excluded to occur at BOR1’s 5’UTR.

The described 5’UTR mechanism of decreased BOR1 translation occurs at toxic levels of boron, indicating a step-wise control of boron transport. Boron accumulation was described to vary along the longitudinal root axis (Shimotohno et al., 2015) and expression of BOR1 is reduced in exodermis, but not in the endodermis, of the rice root growing under boron sufficient conditions (Nakagawa et al., 2007). Proposed two-step control of BOR1 level (Fig. 1) could be operating simultaneously in different cell types varying in boron accumulation. Implementing this two-step control mechanism in models will help to explain cell type-
specific regulation of boron transport during boron sufficient- and toxic conditions. In the natural environment, boron toxicity is co-occurring with salt stress (Reid, 2010). Interestingly, high levels of boron can alleviate the symptoms of salt stress by promoting the recovery of cell wall stiffness, as recently described for FERONIA mutant (Feng et al., 2018). Evaluation of BOR1 activity in the context of boron and salinity stress would be important contribution for plant breeding efforts for the regions where those two stresses limit the plant productivity.

Additionally, the exploration of two-step mechanism of BOR1 control could shed light on boron sensitivity of agronomically-important plants. Overexpression of BOR1(K590A) exhibiting weaker polar localization in the endodermis, enhanced tolerance towards high boron concentrations (Wakuta et al., 2016). 5′UTR uORFs limiting the translation at high boron concentrations present in boron transporters with non-polar localization could unveil factors limiting boron tolerance. The discovery of this two-step regulation of boron transporter reveals not only an elegant mechanism of boron transport control dependent on boron levels, but also unleashes new possibilities for breeding strategies for enhanced boron tolerance.

Figure 1. The two-step control of BOR1 levels. The translation of the BOR1 mRNA under boron toxic conditions is inhibited by the presence of the untranslated Open Reading Frames (ORFs) at the 5′UTR region of BOR1 mRNA. The two uORFs most proximal to the BOR1 START codon reduce translation reinitiation, thereby reducing translation rates of BOR1. At sufficient boron levels, the BOR1 protein undergoes ubiquitynation followed by protein degradation.

References:


