Signal or To Sink: The Long-Sought-After Cytokinin Transporter

Shabana Shams¹ and Muhammad Naseem²,³*

¹Department of Animal Sciences, Quaid-I-Azam University Islamabad, Pakistan
²Department of Bioinformatics, Functional Genomics and Systems Biology Group, Biocenter, University of Würzburg, Am Hubland, Würzburg, Germany
³Department of Molecular Biology and Genetics, Systems Biology of Plant-Microbes Interaction Group, Kuzey Park, İstanbul

Description

An interesting mechanism of trade-off between cytokinin signaling and cellular cytokinin transport through Purine Permease 14 (PUP14) protein is very recently unfolded by Zürcher et al. [1]. The perception of the signal of cytokinin and its cellular phosphorelay circuitry [2] that result in target genes expression are well understood (Figure 1). However, the patchy distribution of bioactive cytokinin ligands, which has vast implications for tissue patterning and multicellularity in plants, is still under research [2,3]. When and where not to signal, is a fine-tuned spatiotemporal regulation that needs clearance of the active cytokinins-ligands away from the cytokinin signaling receptors histidine kinases (Figure 1). In addition to the transport of cytokinins to distal tissues, cells also can achieve the clearance of active cytokinins from apoplast by importing them into the cytosol. Inside the cytosol, cytokinins may also interact with various soluble proteins. Consistent with this notion, and besides membrane-bound cytokinin receptors, many other cytokinin binding proteins (CBPs) have been detected in plants [4]. However, the cellular mechanism of the entry of cytokinins from the apoplasst to cytosol was not fully understood until very recently. The findings of Bruno group [1] concerning the transport of apoplastic cytokinins through PUP14 transporter not only fills this knowledge gape but also has opened new avenues for research in cytokinin biology.

Taking the bioactive cytokinin ligands away from the CHASE-domain of sensor AHKs, the activity of PUP14 enables the import of apoplastic cytokinin inside the cytosol (Figure 1). This influx of bioactive cytokinins exhausts apoplastic space of active ligands for AHKs, and that culminates in reduced cytokinin signaling as well as diminished cellular cytokinin responses (Figure 1). It is worth noting that PUP14 mediated uptake of the active cytokinins was not interfered by cytokinin-ribosides, which suggests that only active ligands are cleared from the apoplast. Zürcher et al. [1] have meticulously exemplified this phenomena on various morphogenetic contexts; for instance, heart-stage embryo, lateral root primordia as well root and shoot apical meristems. The clearance of active ligands away from cell surface cytokinin receptors has no doubt spatiotemporal implications for plant morphogenesis; however, below I highlight some structural biology prospects of this new finding.

Either signal or to sink, for an active cytokinin ligand, the possibility to remain in the apoplast or sink-down into the cytosol is highly determined by the morphogenetic dynamics of a tissue [1]. However, what are those biochemically competing circumstances that propel active cytokinin species away from a functional cytokinin receptor and let them pass through the barrel of the PUP 14 transporter at the expense of adenosine tri-phosphate (ATPs), merits further investigation. Likewise, what is the biochemical explanation of selectivity in ligand-clearance where bioactive cytokinin ligands are imported into the cell while riboside forms are left behind in the apoplast [1]. Therefore, future efforts addressing these aspects with structural biology approaches will further decipher the understanding of this important finding.

One may tempt to speculate that besides external morphogenetic dynamics, the process of cytokinin influx might also be triggered by inside-out signaling. Reason being, the plant cell also contains cytosolic cytokinin receptors [2] which are located on endoplasmic reticulum (ER) and need active ligands for their activity. Although the bulk of transported (PUP14) cytokinins are converted into inactive ligands but the possibility of interaction between freshly transported cytokinins and ER-based AHKs still exists (Figure 1). Genetic studies focusing on mutation in the CHASE-domain of cytoplasmic cytokinin receptors and PUP14 transporter would helpful in addressing this hypothesis. Lastly, plant apoplast is a seat of many other biological functions such as immunity to pathogens [5,6] as well as long-distance communication [2,7], it would be much intriguing to explain these and alike processes under the auspices of this new development in cytokinins biology.

References


*Corresponding author: Muhammad Naseem, Department of Bioinformatics, Functional Genomics and Systems Biology Group, Biocenter, University of Würzburg, Am Hubland, Würzburg, Germany. Tel: +49931 310; E-mail: muhammad@biozentrum.uni-wuerzburg.de

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