

**SPOTLIGHT**

# Unveiling the TRAPP: The role of plant TRAPPII in adaptive growth decisions

 Antonio Galindo<sup>1</sup> 

The regulation of intracellular membrane traffic is coupled with the cell's need to respond to environmental stimuli, which ultimately is critical for different processes such as cell growth and development. In this issue, Wiese et al. (<https://www.doi.org/10.1083/jcb.20231125>) explore the role of the trans-Golgi network (TGN) in stress response, exposing its role in mediating adaptive growth decisions.

Environmental stimuli from biotic or abiotic factors can be directly linked to morphological effects due to cell growth or division in plants. The early developmental decisions triggered by environmental conditions can be analyzed during seedling growth, but correlating them with the underlying mechanism is not easy.

Extracellular cues (hormones, mechanical stimuli, or morphogens) affect intracellular traffic. However, membrane traffic mutants are often pleiotropic, making it difficult to determine if their effects are due to defects in growth with secondary consequences in adaptive response, or whether their primary phenotype is an impaired response to the stress factors. Furthermore, the hierarchy of trafficking factors along the framework of the adaptive response is difficult to discriminate. They may act as an actual decision-making mechanism or participate in the response action.

In plants, the secretion pathway accomplishes the secretion of polysaccharides and proteins to form the cell wall. This process is regulated at the TGN by the protein ECHIDNA. Loss-of-function ECHIDNA mutants impact the secretion of glucans and pectins, which are mislocalized to the vacuole, and many plasma membrane proteins accumulate in multivesicular bodies (1). However, these ECHIDNA mutants do not

affect membrane proteins such as the hormone auxin efflux carriers PIN2 and PIN3 (2). Thus, distinct secretory pathways diverge at the TGN. One of these ECHIDNA-independent trafficking pathways is mediated by the Transport Protein Particle II (TRAPPII) complex (3).

In metazoans, two TRAPP complexes, TRAPPII and TRAPPIII, have been identified and are also present in plants. They function as guanine exchange factors (GEFs) that activate the small GTPases Rab1 and Rab11, central regulators of membrane traffic. *In vitro*, TRAPPII and TRAPPIII activate Rab1, but only TRAPPII activates Rab11. Both complexes share a common core of seven small subunits, while each complex has unique subunits, which drive specificity toward their cognate Rab substrate (4). Compared with other GEFs, the TRAPPs are distinguished by being large oligomeric complexes. Consequently, they likely fulfill additional functions beyond their GEF activity. They may integrate signals to modulate the behavior of their Rab substrate and regulate downstream pathways.

Wiese et al. (5) analyzed the TRAPPII interactome, revealing the shaggy-like kinases (SK) family as interactors of TRAPPII. They established this functional interaction and identified three phosphorylation sites in the TRAPPII-specific subunit TRS120

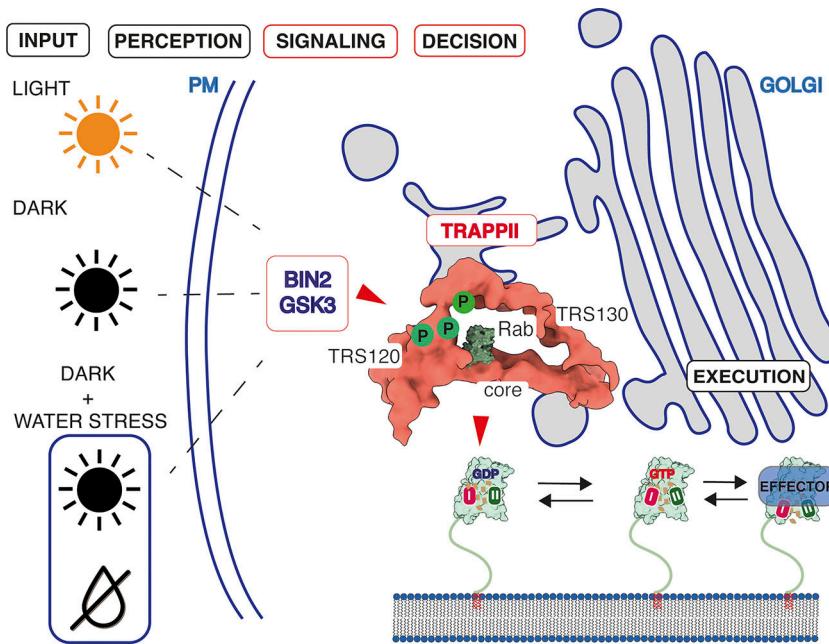
(TRAPPC10) targeted by the AtSK kinases BIN2 and SK11 kinases. These kinases are involved in the pathway of brassinosteroids, phytohormones that promote cell expansion and elongation (6). The phosphorylation sites are situated within domains that are specific to plants. Nevertheless, the alignment of the Alphafold plant TRAPPII model with the yeast cryo-EM structure suggests that at least two phosphorylation sites may be oriented toward the chamber that accommodates Rab11 (7, 8, Preprint). Therefore, the authors speculated that the phosphorylation status of TRS120 could modulate the TRAPPII GEF activity.

To explore the physiological significance of the TRS120 phosphorylation, Wiese et al. (5) investigated whether TRAPPII is required to adopt differential growth decisions under stress, focusing on trade-offs between the hypocotyl, the embryo stem, versus the root in germinating seedlings. They subjected the seedlings to simultaneous deprivation of both light and water, creating a “conflict of interests” scenario wherein hypocotyl and root growth exhibit competing interests. By comparing the phenotype of *bin2* null mutants and *trappii* null mutants with wild-type strains and others impaired in the perception of light or water, the authors concluded that *bin2* and *trappii* null mutants are decision mutants. To

<sup>1</sup>Division of Cell Biology, MRC Laboratory of Molecular Biology, Cambridge, UK.

Correspondence to Antonio Galindo: [agalindo@mrc-lmb.cam.ac.uk](mailto:agalindo@mrc-lmb.cam.ac.uk).

© 2024 Crown copyright. The government of Australia, Canada, or the UK (“the Crown”) owns the copyright interests of authors who are government employees. The Crown Copyright is not transferable. This article is distributed under the terms of an Attribution–Noncommercial–Share Alike–No Mirror Sites license for the first six months after the publication date (see <http://www.rupress.org/terms/>). After six months it is available under a Creative Commons License (Attribution–Noncommercial–Share Alike 4.0 International license, as described at <https://creativecommons.org/licenses/by-nc-sa/4.0/>).



**Figure 1. SKs, such as BIN2, are pivotal in integrating various signaling pathways, responses to extracellular cues.** According to Wiese et al. (5), the integration of signals and decision-making processes occurs at the interface between AtSK and TRAPPiI. Downstream of TRAPPiI, Rab GTPases play a crucial role in executing decisions. The phosphorylation status of AtTRS120 may modulate TRAPPiI GEF activity, affecting the function of its cognate Rabs. PM: plasma membrane; P enclosed in green circle: phosphate group; Rab switch regions I [red] and II [green] are indicated.

eliminate the possibility that this effect was a secondary consequence from TGN malfunction, they compare the *trappiI* null allele to mutations in other genes, such as *echidna*, which exhibit related trafficking defects but lack a decision phenotype.

Then they address the effects of TRS120 phosphovariants. Their localization *in vivo* showed that the triple phosphomimetic mutant is predominantly membrane associated, and the phosphorylation status affects the hypocotyl versus root trade-offs and germination. Non-phosphorylatable versus phosphomimetic mutations at the AtTRS120 AtSK sites had opposing impacts on adaptive responses. The non-phosphorylatable mutant showed an enhanced root response to water stress in the dark and increased resistance to osmotic stress during germination. This indicates that TRAPPiI phosphorylation mediates the differential response to osmotic stress and to light and water availability. Finally, the authors observed a synergistic interaction between BIN2 and TRAPPiI concerning root gravitropism, providing another example of adaptive growth decisions.

In light of these findings, a hypothesis is proposed wherein signal integration and decision-making in response to environmental cues occur at the AtSK-TRAPPiI interface. Downstream, the function of Rab GTPases recruiting different effectors is implicated in implementing the decision (Fig. 1). It is known that the TRAPPiI complex plays a role in sorting decisions as the polar localization of the PIN2 carriers. Still, much work must be done to unveil the mechanisms that result in TRAPPiI phosphorylation and how it correlates with cargo sorting.

The authors propose a plausible effect of phosphorylation on GEF activity since, as mentioned, the predicted structural model suggests that two phosphorylation sites could be oriented toward the chamber that accommodates Rab11 when bound to TRAPPiI. Considering the TRAPPs' high conservation, it is expected that plant TRAPPiI architecture resembles that of fungi and metazoans, where the specific subunits TRS120 (TRAPPC10) and TRS130 (TRAPPC9) hold the elongated common core like a pair of tongs by the N-terminal region, while they contact each other at their C-terminal region. In contrast

to metazoans, plant TRAPPiI has an additional subunit, TRIPP (9), which may be involved in putative complex dimerization, similar to yeast (10). The phosphorylation sites are located in plant-specific domains, so it is possible that TRIPP function, which may meet the unique demands of membrane traffic in plants, could be affected by TRS120 phosphorylation. Moreover, phosphorylation alters the TRAPPiI localization, thereby influencing how and where the complex is recruited to the membrane.

Interestingly, the observed synergistic effect between BIN2 and TRAPPiI suggests that both proteins participate in the same pathway but operate at different levels, which may result in a novel additive phenotype. On the other hand, since a unique specificity between members of the SK family and TRAPPiI phosphorylation sites has not been demonstrated, a promiscuity between AtSK and TRAPPiI may lead to varied responses.

The finding that posttranslational modifications, such as phosphorylation, occur on TRAPP complex-specific subunits supports the notion that these oligomeric complexes, which govern essential pathways (ER-to-Golgi traffic, autophagy, exocytosis, cytokinesis), may serve as hubs to integrate signals that regulate downstream events. This would help explain how Rab1 and Rab11 can recruit different effectors at specific locations to direct the cellular traffic. Such adaptations would allow membrane traffic to accommodate the developmental and metabolic demands of the cell.

The paper by Wiese et al. (5) serves as a great example, establishing a precedent for the role of TRAPPs as integration modules connecting membrane traffic with extracellular cues. We can expect to find additional evidence of posttranslational modifications of TRAPPs in other organisms.

#### Acknowledgments

I express my appreciation for the support of Dr. Sean Munro through the years of working in his lab and to all members of his research team. Due to space limitations, not all relevant publications are discussed.

A. Galindo's work is funded by the Medical Research Council, as part of United Kingdom Research and Innovation (file reference number MC\_U105178783).

## References

1. Gendre, D., et al. 2011. *Proc. Natl. Acad. Sci. USA*. <https://doi.org/10.1073/pnas.1018371108>
2. Boutté, Y., et al. 2013. *Proc. Natl. Acad. Sci. USA*. <https://doi.org/10.1073/pnas.1309057110>
3. Ravikumar, R., et al. 2018. *Development*. <https://doi.org/10.1242/dev.169201>
4. Galindo, A., and S. Munro. 2023. *FEBS Lett.* <https://doi.org/10.1002/1873-3468.14553>
5. Wiese, C., et al. 2024. *J. Cell Biol.* <https://doi.org/10.1083/jcb.202311125>
6. Kalbfuß, N., et al. 2022. *PLoS Genet.* <https://doi.org/10.1371/journal.pgen.1010541>
7. Mi, C., et al. 2022. *Sci. Adv.* <https://doi.org/10.1126/sciadv.abi5603>
8. Bagde, S.R., and J.C. Fromme. 2021. *bioRxiv*. <https://doi.org/10.1101/2021.12.20.473486> (Preprint posted December 20, 2021).
9. Garcia, V.J., et al. 2020. *Plant Cell*. <https://doi.org/10.1105/tpc.20.00044>
10. Kalde, M., et al. 2019. *Plant J.* <https://doi.org/10.1111/tpj.14442>