Nutrient Signaling by Nitrate and Calcium

An especially intriguing aspect of nutrients is their capacity to serve as signals. Nutrient signaling pathways play key roles in plant growth and development and can interact and overlap in interesting ways. In this issue, Riveras et al. (2015) report on two plant macronutrients, nitrate and calcium, and show how responses induced by nitrate depend in part on calcium signaling mechanisms.

The study of nitrate signaling was launched by a report almost 60 years ago describing how the appearance of nitrate reductase activity in rice (Oryza sativa) plants depends on nitrate in the growth medium (Tang and Wu, 1957). Since then, many molecular, physiological, and developmental responses to nitrate have been described, from the rapid activation of hundreds of genes to the reprogramming of root architecture for the foraging of soil nitrate (Krouk et al., 2010; Bouguyon et al., 2012; Gutiérrez, 2012). The underlying mechanisms and regulators that mediate these responses have been sought for decades. Over the past several years, major progress has been made with the identification of transcription factors, two calcineurin B-like interacting protein kinases (AtCIPK8 and AtCIPK23) and a nitrate transceptor (AtNPF6.3, also called AtNRT1.1 or CHL1; Krapp et al., 2014; Vidal et al., 2015).

The identification of CIPKs as nitrate regulators implicates calcium in nitrate signaling. Calcium is a ubiquitous and versatile secondary messenger involved in many developmental and physiological processes, including abiotic stress responses (Dodd et al., 2010). Many years ago, calcium was implicated in nitrate signaling by reports that EGTA (a calcium chelator) and LaCl₃ (a calcium channel blocker) could inhibit the nitrate induction of gene expression (Sakakibara et al., 1997; Sueyoshi et al., 1999).

Riveras et al. (2015) set out to document calcium’s role in nitrate signaling. Using aquorin transgenic Arabidopsis (Arabidopsis thaliana) plants, they detected a rapid and transient increase in intracellular calcium in response to nitrate treatment, which was blocked by LaCl₃. They then found that two known components in calcium signaling, phospholipase C (PLC) and its product inositol triphosphate (IP₃), are involved in this nitrate-induced calcium response by showing IP₃ levels increased in response to nitrate but not in the presence of the PLC inhibitor U732122. These responses were greatly diminished into two NRT1.1 mutants, indicating that the calcium transient and the IP₃ response are downstream of the nitrate transceptor NRT1.1. Most important, nitrate induction of four nitrate-responsive, nitrate assimilatory, and regulatory genes (NRT2.1, NRT3.1, NITRITE REDUCTASE, and TGA1) was inhibited by U732122 and by LaCl₃. Interestingly, nitrate induction of another nitrate-responsive, regulatory gene (AUXIN RECEPTOR F-BOX3 [AFB3]) was not diminished by these inhibitors. Thus, PLC, IP₃, and calcium are components of one branch of the NRT1.1-mediated nitrate signaling pathway.

These findings raise questions about how calcium functions in nitrate signaling. Does calcium only work through CIPKs, or does it also affect gene regulation via calmodulin-binding transcription factors (Galon et al., 2010)? Do nitrate-induced calcium transients regulate the phosphorylation state and, thus, the activity of NRT1.1, which has been shown to be regulated by the action of CIPK23 (Ho et al., 2009)? It is intriguing that two of the nine Arabidopsis PLC genes are responsive to nitrate, suggesting further overlap between nitrate and calcium signaling. Now that the role of calcium in nitrate signaling has been established, new avenues of research have opened up to probe more deeply into the complexities of nutrient signaling.© 2015 American Society of Plant Biologists. All Rights Reserved.