Does light taste salty?

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As research advances acquisition of new data reveals novel aspects on already investigated issues. This is the case for SALT TOLERANCE (STO), an Arabidopsis protein that confers tolerance to high salt concentrations when ectopically expressed in yeast cells. For the last years, STO was considered to participate mainly in the response and tolerance of Arabidopsis to high salinity, as it does in yeast. However, recent investigations using gain- and loss-of-function mutants revealed a major role for STO as negative regulator of photomorphogenesis. Interestingly, and contrary to other negative regulators of light dependent inhibition of hypocotyl elongation, STO protein instability is controlled by COP1 activity in etiolated seedlings. Thus, light stabilizes STO protein levels during de-etiolation. Whether STO participates in other signaling cascades different from light signaling, as it has been shown in yeast and proposed in plants or not, is still an open question.

SALT TOLERANCE (STO), a B-box Zn finger from A. thaliana, was first identified as a protein conferring enhanced salt tolerance when ectopically expressed in salt sensitive calcineurin deficient yeast cells.1 Calcineurin is a Ca2+/calmodulin-dependent phosphoprotein phosphatase type 2B that modulates salt tolerance in yeast.2 In an attempt to isolate genes involved in calcium signaling and regulation in plants, we established a complementation screening analysis using a yeast L-type calcium channel (CCH1) knock-out mutant that exhibits the same growth arrest in high salt containing media as yeast calcineurin mutants.3 Transformation of the cch1 mutant with an Arabidopsis cDNA library of seedlings grown under long day conditions resulted in the isolation of STO. Overexpression of STO in yeast cells not only complemented the salt sensitivity phenotype of cch1 (Fig. 1), but also increased the salt tolerance of the wild type strain.

Interestingly, transcription of STO is not altered by salt treatment in Arabidopsis, although it was shown that overexpression of STO conferred salt tolerance to transgenic lines.5 However, we could neither reproduce this result in our overexpressing lines, nor observe any salt related phenotype if we analyzed RNAi lines or a knock-out allele of the gene.

Still, the finding that STO interacts with COP1 in the 2-hybrid system assay6 prompted us to analyze the phenotypes of STO gain- and loss-of-function mutants during seedling de-etiolation in different light conditions. The results, recently published by Indorf et al.,7 demonstrated a major role for STO as negative regulator during photomorphogenesis (Fig. 2). In addition, control of STO activity in light signaling involves regulation of its RNA transcription and of the protein at the posttranslational level. Thus, etiolated seedlings do not present detectable amount of STO protein, and only after being exposed to white light, STO accumulates in the nucleus. Interestingly, in the cop1-4 mutant background, the protein is already present in the dark, indicating that COP1 is responsible for the degradation of the protein in dark grown seedlings. COP1 suppresses photomorphogenesis in darkness by ubiquitinating activators of the light response, such as the transcription factors LONG HYPOCOTYL 5 (HY5), LONG AFTER FR 1 (LAF1) and LONG HYPOCOTYL IN FR 1 (HFR1), which are subsequently degraded by the proteasome.8-12 In light, activated photoreceptors are thought to inhibit COP1 function so that these transcription factors are no longer degraded.

In this respect, regulation of STO content by COP1 is interesting because COP1 normally targets positive regulators of photomorphogenesis for degradation. Physical interaction between COP1 and negative regulators of light signaling has been described for the SPA
proteins. The four-member SPA protein family of Arabidopsis acts in concert with COP1 to ubiquitinate activators of the light response and suppress photomorphogenesis in dark-grown seedlings.

Although STO has a basal transcriptional level, during de-etiolation transcriptional activity is enhanced by light. In the simplest scenario, COP1 would be responsible for the degradation in the dark of the low basal level of STO protein in the nucleus. After light perception and inactivation of COP1, STO accumulates in the nucleus where it does its function. COP1 is a negative regulator of photomorphogenesis in dark; thus, STO activity is not required in the presence of active COP1. However, in the light, when COP1 is not anymore effective, STO takes over the role as a negative regulator/modulator, probably to prevent exaggerated responses to the light. The mode of action of STO will be an important issue to be followed up.

Colocalization of COP1 and STO in transient assays was observed not only as nuclear speckles in the cells but also as larger cytoplasmic aggregations. These results, although opening new perspectives for a possible function of COP1 in the cytosol, should be carefully interpreted since they could merely be a consequence of the coexistence of the overexpressed proteins in the same sub-cellular compartments.

Interestingly STH, a close homologue of STO that also interacts with COP1 directly with COP1, a constitutive repressor of light signaling in Arabidopsis. J Biol Chem 2001; 276:3817-38.

In addition to unravel the function of STO and the homologue STH in plants, an interesting issue will be to analyze the mode of action of both proteins in yeast. Overexpression of STO and, to a lesser extent, of STH (Fig. 1) provides salt tolerance in yeast. The fact that no homologous proteins are present in the genome of Saccharomyces, raises the question of the existence of a salt or more general stress-responsive pathway in the yeast that recognizes these plant proteins.

References