

Engineering Na⁺/H⁺ antiport in higher plants for salt tolerance

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Summary

Salinity is a critical factor that severely affects the growth and the productivity of a large proportion of land plants. Nevertheless, halophytic plants can survive and grow in high-salt environments by acclimatized processes that include biosynthesis and accumulation of compatible solutes, and intra- and extra- cellular sequestration or compartmentation of excess amounts of Na⁺. Recent progress in transgenic studies has demonstrated the possibility of metabolic engineering of compatible solutes as a strategy to confer salt protection to plants. On the contrary, little effort has been made with Na⁺ sequestration as the target for genetic engineering of salt tolerance. One of the staple elements in higher plants that is responsible for the sequestration of accumulated Na⁺ to vacuoles or extracellular spaces is regarded as an Na⁺/H⁺ antiporter. Thus far, however, no genes or cDNAs have been available of plant origin.

In an attempt to enhance salt tolerance of higher plants, *Arabidopsis thaliana*, a salt-sensitive species, was transformed *via* Agrobacterium-mediated method with a gene (*sod2*) encoding Na⁺/H⁺-antiporter from a fission yeast *Schizosaccharomyces pombe* under a strong, constitutive promoter. Molecular analysis by polymerase chain reaction (PCR) with genomic DNA from transformed plants confirmed that the introduced gene was stably incorporated and was transmitted to the secondary generation. Reverse-transcription-PCR analysis using total RNA indicated that the *sod2* gene was actively transcribed in transformed plants. In order to examine the steady-state levels, Northern blot analysis was performed with the same RNA preparations but failed to detect the *sod2* transcripts. These results suggested that the *sod2* mRNA is unstable or the levels of the transcripts were extremely low in transformed plants. Currently, the *sod2* gene is being introduced into *Arabidopsis* plants under the transcriptional control of a salt-inducible promoter.