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Oxidation of Ion Channels by Salt Stress and Improvement of Salt Tolerance by Suppressing Their Oxidation

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Summary

Salt stress drastically inhibits plant growth, resulting in reduction in agricultural productivity. Plants have to overcome detrimental oxidative damage induced by salt stress to survive under salt stress. Therefore, plants activate antioxidant enzymes and accumulate antioxidant compounds to mitigate oxidative stress under salt stress. Oxidative damage is in part due to protein oxidation/modification.

Identification of oxidized/modified proteins allows us to elucidate salt-tolerance mechanism and to develop and screen salt-tolerant plants. However, oxidized or modified proteins remain to be identified.

In this study, we investigated effects of methylglyoxal, which accumulates in salt-stressed plants, on activities of ion channels and other salt tolerance-related enzymes and tried to identify amino acid residues of these proteins modified by methylglyoxal.

Inward-rectifying potassium channel currents in plasma membrane of *Arabidopsis* guard cell protoplasts were measured using whole-cell patch-clamp technique and inward potassium currents in KAT1-expressing *Xenopus* oocytes were measured using two-electrode voltage clamp technique. Methylglyoxal inhibited the inward-rectifying potassium channel currents in the plasma membrane of guard cell protoplasts and inhibited the inward potassium currents in the KAT1-expressing oocytes in a concentration dependant manner.

Activation of plasma membrane inward potassium currents is favorable to light-induced stomatal opening. Stomatal apertures were observed under a microscope. Methylglyoxal inhibited light-induced stomatal opening in a concentration dependant manner.

We are identifying amino acid residue of the methylglyoxal-treated proteins using MALDI TOF-MS.