

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

Yoshiyuki Murata ¹, Izumi C. Mori ²

¹Graduate School of Natural Science and Technology, Okayama University

²Institute of Plant Science and Resources, Okayama University

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.

Methylglyoxal inhibited the inward-rectifying potassium channel currents in the plasma membrane of guard cell protoplasts, the inward potassium currents in the KAT1-expressing oocytes in a concentration dependent manner, and inhibited light-induced stomatal opening in a concentration dependent manner (Hoque *et al.*, 2012a).

Methylglyoxal induced stomatal closure accompanied by extracellular ROS production mediated by SHAM-sensitive peroxidases, intracellular ROS accumulation, and cytosolic free calcium concentration ($[Ca^{2+}]_{cyt}$) oscillations (Hoque *et al.*, 2012b). Moreover, methylglyoxal also inhibited stress-related enzymes, cytosolic ascorbate peroxidase (Hoque *et al.*, 2012) and glutathione *S*-transferase (Hoque *et al.*, 2010).

We are identifying amino acid residues (arginine, lysine, and cysteine) of the methylglyoxal-treated proteins.