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Molecular Mechanism of the High-Osmolarity Responsive MAPK Pathway That Is Involved in Salt- and Osmo-Tolerance in Yeast

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Summary

Adaptation to high salt and high osmolarity conditions is a fundamentally important biological response of all types of cells, ranging from bacteria, fungi, plants, and animals. In yeast, for example, external high salt and high osmolarity conditions activate the HOG (High Osmolarity Glycerol) MAP kinase (MAPK) pathway, which is essential for yeast to adapt to and survive on those conditions. MAP kinase cascades are conserved signaling modules composed of three sequentially activated kinases (MAPKKK, MAPKK, and MAPK). The yeast high osmolarity glycerol (HOG) pathway can be activated by either of two upstream pathways, termed the SHO1 or SLN1 branches. However, neither the osmosensor nor the signal generator of the SHO1 branch has been clearly defined.

Membrane localization of the Ste11 MAPKKK is essential for activation of both the filamentous growth/invasive growth (FG/IG) MAP kinase (MAPK) pathway and the SHO1 branch of the osmoregulatory HOG MAPK pathway, and is mediated by binding of the Ste50 scaffold protein to the Opy2 membrane anchor. Here we show that Ste50 phosphorylation by the MAPKs activated by the HOG or the mating pathway dissociates Ste50 from Opy2, thereby preventing excessive activation of the HOG pathway, or reduces the basal activity of the mating MAPK pathway. Thus, dynamic regulation of Ste50-Opy2 interaction fine-tunes the MAPK signaling network.