Basic research towards improvement in the salt tolerance of yeasts

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Yeasts has been used for the production of fermented high salt foods, including soy sauce, miso, and pickles. To maintain superior flavor and continue high production of these foods under high salt fermentation conditions, yeasts used for the fermentation must be highly salt tolerant. Yeasts achieve the salt tolerance by exerting various physiological responses under high salt conditions to adapt to the stressful condition. In this study, among the signal transduction pathways activated by the salt stress to induce various adaptive responses, we focused the Cpl1-Rim101 pathway, which responds to ion stresses, and the HOG pathway, which responds osmotic stress, and elucidated novel regulatory mechanisms of these pathways.

The Cpl1-Rim101 pathway responds ionic stress by proteolytically cleaving and thus activating the Rim101 transcription factor. Cpl1, a calpain-like protease implicated in the Rim101 cleavage, as well as Rim8, Rim9, Rim20, and Rim21 have been identified as constituents of this pathway. In this study, we determined the order of actions among these constituents in the pathway as follows.

Ion stresses → Rim8 · Rim9 · Rim21 → Cpl1 · Rim20 → Rim101 cleavage → Induction of salt tolerance

In addition, a gene encoding Ena1, a membrane transporter extruding the toxic sodium ion, was identified to be induced by this pathway. This observation indicates that ion stress activate the pathway to induce the extrusion system for the toxic ions as an adaptive response.

The HOG pathway is the yeast stress responsive MAP kinase pathway with two osmotic sensor mechanisms. The pathway is activated by the osmotic stress to induce various adaptive responses. Ssk1 is a key component of one of the sensor mechanisms, the Sln1 pathway. Upon osmotic stress, Ssk1 is rapidly dephosphorylated and thus activated, and it in turn activates the downstream MAP kinase pathway. In this study, a degradation mechanism specific to dephosphorylated Ssk1 was found to downregulate the pathway. This degradation was performed by the ubiquitin-proteasome system and a ubiquitin-conjugating enzyme Ubc7 and a ubiquitin ligase Hrd1 participate in the degradation. In addition, we found the physiological role of this degradation is to inactivate the HOG pathway timely after the osmotic adaptation completes.

The analysis of these two salt stress-responsive signal transduction pathways elucidates the elaborate regulatory mechanism coordinating the salt stress responses. Based on the observation obtained in this study, we would like to clarify the molecular basis for the acquisition of the yeast salt tolerance, and to design highly salt tolerant yeast strains.