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AMIODARONE INDUCES THE SYNTHESIS OF HSPS IN SACCHAROMYCES CEREVISIAE AND ARABIDOPSIS THALIANA CELLS

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Many biotic and abiotic stresses cause an increase of cytosolic Ca^{2+} level in cells. Calcium is one of the most important second messengers, regulating many various activities in the cell and was known to affect expression of stress activated genes. Mild heat shock induces the expression of heat shock proteins (Hsps) which protect cell from drastic heat shock exposure. There are some literature data permitting to suggest that transient elevation of cytosolic Ca^{2+} level in plant cells is important for activation of Hsps expression. On the other hand mitochondria are known to regulate the intracellular calcium and reactive oxygen species signaling. It has been shown recently that mild heat shock induces hyperpolarization of inner mitochondrial membrane in plant and yeast cells and this event is critically important for activation of Hsps expression. To reveal the relationship between mitochondrial activity, intracellular calcium homeostasis and Hsps expression an antiarrhythmic drug amiodarone (AMD) have been used. AMD is known to cause transient increase of cytosolic Ca^{2+} level in *Saccharomyces cerevisiae*. Obtained results have showed that AMD treatment induced the synthesis of Hsp104p in *S. cerevisiae* cells and Hsp101p in *A. thaliana* cell culture. Induction of Hsp104p synthesis leads to enhanced yeast capability to survive lethal heat shock exposure. Development of *S. cerevisiae* thermotolerance depended significantly on the presence of Hsp104p. Elevation of Hsp104p level in the result of AMD treatment was shown to be governed by activity of Msn2p and Msn4p transcription factors. Deletion of the *MSN2* and *MSN4* genes abrogated the AMD ability to induce Hsp104p synthesis. Mild heat shock and AMD treatment induced the hyperpolarization of the inner mitochondrial membrane in yeast and Arabidopsis cells which accompanied by HSP synthesis and development of thermotolerance. It was suggested that increase of cytosolic Ca^{2+} level after AMD treatment directly or indirectly causes the activation of mitochondrial activity which leads to hyperpolarization of the inner mitochondrial membrane and production of reactive oxygen species (ROS). Modulation of cellular Ca^{2+} and ROS signals by mitochondria is assumed to play a prominent role in activation of Hsps expression in yeast and plant cells.