STUDY OF THE FUNCTION AND REGULATION OF PEROXISOMES IN RESPONSE TO SALT STRESS

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Introduction

All living cells are constantly adapting to changes or stresses in their environment. Osmotic or saline stress causes a loss of water from the cells and disrupts ion homeostasis. These changes trigger adaptation programs in eukaryotic cells to repair damage, adjust cellular metabolism and allow proliferation. This research studies the mechanisms of metabolic adaptation in response to osmotic and salt stress in the yeast model (Saccharomyces cerevisiae), as it is one of the main models for understanding the mechanisms of adaptation to osmotic stress [1]. The HOG (high osmolarity Glycerol) MAP kinase pathway is the major signaling pathway under conditions of osmotic stress [2]. Its MAP kinase HOG1 coordinates a complex program of adaptation in cell cycle modulation, activation of gene expression and accumulation of osmolytes and ion transport [3]. Furthermore, adaptation to salt stress depends on the activation of mitochondria which are involved in ROS (Reactive Oxygen Species) balance during stress [4].

This study aims to investigate the regulation of peroxisomes (involved in catalysis of long chain fatty acids) during the adaptation to salt stress, with the following aims:

a. Possible levels of regulation of peroxisomes (expression of structural genes, biogenesis and number of peroxisomes).

b. Potential regulators of peroxisomes under stress conditions (pathways, transcription factors).

c. Possible peroxisomal functions of protection against stress (compensation of respiratory metabolism, homeostasis of fatty acids).

The results obtained so far show that the correct function of peroxisomes is necessary for efficient adaptation to stress and that the organelle is specifically activated in response to stress. This is manifested by the activation of gene expression of peroxisome components and an increase in peroxisome number per cell. In addition, the HOG1 kinase is involved in the peroxisomal activation with Adr1 as a potential transcriptional activator operating upon salt stress.