Assessment of Antioxidant Defenses Deriving from the Peroxisomes

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Beta oxidation of fatty acids in the peroxisomes generates hydrogen peroxide, and the antioxidant defense in this case has been traditionally attributed to catalase. However, catalase-null strains of yeast show very similar survival rates to WT cells, suggesting that there are other antioxidant systems in peroxisomes. We are investigating the possibility that peroxiredoxins contribute to defend against oxidative damage in the peroxisome. This idea was corroborated by preliminary results of our group indicating that, in respiratory conditions, *cta1D* cells exhibit an increased expression of genes coding for other protective enzymes, specifically Ahp1, Prx1 and Tsa2. Here we show that the lack of Ahp1 negatively affects the survival of *Saccharomyces cerevisiae* in the presence of t-BOOH (*tert*-Butyl hydroperoxide) in all mediums tested: SD (synthetic dextrose), glycerol and oleate. ATZ (3-Amino-1,2,4-triazole), an inhibitor of catalases, had little effect on cell survival. However, when grown in oleate as the carbon source, condition in which peroxisomes proliferate, *ahp1D* yeast became more resistant to t-BOOH in the presence of ATZ, suggesting a novel path of antioxidant defense in the peroxisome, stimulated in the absence of catalase and not dependent on catalase or Ahp1. This indicates the presence of other enzymes that cooperate to protect against this oxidative insult derived from the peroxisome. This should be further clarified by developing and studying the viability of mutant strains such as *cta1Dahp1D,* quantifying the production of different ROS (reactive oxygen species) and investigating the expression of different peroxiredoxins under oxidative stress.

Keywords: Ahp1, catalase A, reactive oxygen species, *Saccharomyces cerevisiae*, peroxiredoxin, oleate, glycerol, respiratory, viability.