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**Role of Acox mediated β-oxidation in replicative ageing of *Saccharomyces cerevisiae***

Isha Kashyap and Shirisha Nagotu

1Department of Biosciences & Bioengineering, IIT Guwahati, India

**Email: isha.kashyap@iitg.ac.in**

**Abstract**

Organelles are sub-cellular structures that are unique with respect to their structure and functions. Every organelle has a distinct role in maintaining cellular homeostasis [1]. β-oxidation of fatty acids requires a coordinated functioning of mitochondria and peroxisomes in mammals whereas in yeast *Saccharomyces cerevisiae* it solely happens in peroxisomes [2] The pathway consists of four sequential steps in which the first and the rate limiting step is catalyzed by the enzyme acyl-CoA oxidase known as Pox1 or Fox1 in yeast [3]. In this step fatty acyl-CoA is converted to 2-*trans*-enoyl-CoA. A role for peroxisomes in cellular ageing is also under investigation [4]. In this study, we aim to understand the link between peroxisomal β-oxidation and replicative ageing of yeast cells. For this, we have analyzed the ageing dynamics of cells lacking Pox1 by biotin-streptavidin based separation of old cells followed by microscopic analysis. Pox1 is imported into peroxisomes by the receptor protein Pex5 [5]. To investigate if receptor binding and protein import alter with age, a strain where Pox1-GFP is expressed under its own promoter was created. Using this we aim to decipher if there is competitive binding between proteins to be imported upon replicative ageing and how Pox1 behaves in this competition for receptor.

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