Consequences of cytochrome c oxidase assembly defects for the yeast stationary phase

Alicia F. Dubinski, Raffaele Camasta, Tyler G.B. Soule, Bruce H. Reed, D. Moira Glerum

PII: S0005-2728(18)30045-8
DOI: doi:10.1016/j.bbabio.2018.03.011
Reference: BBABIO 47891

To appear in:

Received date: 9 October 2017
Revised date: 13 March 2018
Accepted date: 19 March 2018

Please cite this article as: Alicia F. Dubinski, Raffaele Camasta, Tyler G.B. Soule, Bruce H. Reed, D. Moira Glerum , Consequences of cytochrome c oxidase assembly defects for the yeast stationary phase. The address for the corresponding author was captured as affiliation for all authors. Please check if appropriate. Bbabio(2018), doi:10.1016/j.bbabio.2018.03.011

Consequences of cytochrome *c* oxidase assembly defects for the yeast stationary phase

Alicia F. Dubinski[a], Raffaele Camasta[a], Tyler G.B. Soule[a], Bruce H. Reed[a], D. Moira Glerum[a,b]

[a]Department of Biology and [b]Waterloo Institute of Nanotechnology, University of Waterloo, Waterloo, Ontario, N2L 3G1, Canada

To whom correspondence should be addressed: D. Moira Glerum, Department of Biology, University of Waterloo, 200 University Ave West, Waterloo, Ontario, N2L 3G1, Canada;
Tel.: 519-888-4567 x 31352; E-mail: moira.glerum@uwaterloo.ca

Keywords: yeast, oxidative stress, mitochondria, cytochrome *c* oxidase (complex IV) assembly, stationary phase

---

ABSTRACT

The assembly of cytochrome *c* oxidase (COX) is essential for a functional mitochondrial respiratory chain, although the consequences of a loss of assembled COX at yeast stationary phase, an excellent model for terminally differentiated cells in humans, remain largely unexamined. In this study, we show that a wild-type respiratory competent yeast strain at stationary phase is characterized by a decreased oxidative capacity, as seen by a reduction in the amount of assembled COX and by a decrease in protein levels of several COX assembly factors. In contrast, loss of assembled COX results in the decreased abundance of many mitochondrial proteins at stationary phase, which is likely due to decreased membrane potential and changes in mitophagy. In addition to an altered mitochondrial proteome, COX assembly mutants display unexpected changes in markers of cellular oxidative stress at stationary phase. Our results suggest that mitochondria may not be a major source of reactive oxygen species at stationary phase in cells lacking an intact respiratory chain.

1. INTRODUCTION

The yeast *Saccharomyces cerevisiae* is a facultative anaerobe and has proven to be a fruitful model organism for studying inherited mitochondrial defects in humans. However, until recently, most studies have examined mitochondria from yeast cells growing exponentially, where fermentation is the main metabolic pathway. Following the logarithmic phase of growth and the diauxic shift, yeast cells enter a phase of growth, known as the stationary phase, in which their metabolism is switched almost entirely to respiration. Stationary phase cultures have been defined as being saturated, with depleted carbon sources and cells that have become quiescent [1]. A yeast culture growing in typical rich glucose medium may take as long as seven days to reach 'true' stationary phase, at which point there is no further cell growth once the ethanol has been depleted [1, 2]. However, it is now clear that a stationary phase culture contains a mixed population of both quiescent and non-quiescent cells [3]. Nevertheless, there are