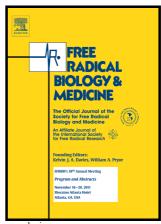
Author's Accepted Manuscript

PARADOXICAL ROLES OF DUAL OXIDASES IN CANCER BIOLOGY

Andrew C. Little, Arvis Sulovari, Karamatullah Danyal, David E. Heppner, David J. Seward, Albert van der Vliet



www.elsevier.com

PII: S0891-5849(17)30609-3

DOI: http://dx.doi.org/10.1016/j.freeradbiomed.2017.05.024

Reference: FRB13351

To appear in: Free Radical Biology and Medicine

Received date: 15 March 2017 Revised date: 26 May 2017 Accepted date: 30 May 2017

Cite this article as: Andrew C. Little, Arvis Sulovari, Karamatullah Danyal David E. Heppner, David J. Seward and Albert van der Vliet, PARADOXICAL ROLES OF DUAL OXIDASES IN CANCER BIOLOGY, *Free Radica Biology and Medicine*, http://dx.doi.org/10.1016/j.freeradbiomed.2017.05.024

This is a PDF file of an unedited manuscript that has been accepted fo publication. As a service to our customers we are providing this early version o the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting galley proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain

ACCEPTED MANUSCRIPT

PARADOXICAL ROLES OF DUAL OXIDASES IN CANCER BIOLOGY

Andrew C. Little¹, Arvis Sulovari², Karamatullah Danyal¹, David E. Heppner¹, David J. Seward¹, Albert van der Vliet^{1*}

Departments of ¹Pathology and Laboratory Medicine, Robert Larner, M.D. College of Medicine, University of Vermont, Burlington, VT 05405

²Microbiology and Molecular Genetics, Robert Larner, M.D. College of Medicine, University of Vermont, Burlington, VT 05405

*Corresponding author: Department of Pathology and Laboratory Medicine, Robert Larner M.D., College of Medicine, University of Vermont, HSRF Building, Room 216 149 Beaumont Avenue, Burlington, VT 05405. Tel.: (802) 656-8638; Fax: (802) 656-8892. albert.van-der-vliet@uvm.edu

Abstract

Dysregulated oxidative metabolism is a well-recognized aspect of cancer biology, and many therapeutic strategies are based on targeting cancers by altering cellular redox pathways. The NADPH oxidases (NOXes) present an important enzymatic source of biological oxidants, and the expression and activation of several NOX isoforms are frequently dysregulated in many cancers. Cell-based studies have demonstrated a role for several NOX isozymes in controlling cell proliferation and/or cell migration, further supporting a potential contributing role for NOX in promoting cancer. While various NOX isoforms are often upregulated in cancers, paradoxical recent findings indicate that dual oxidases (DUOXes), normally prominently expressed in epithelial lineages, are frequently suppressed in epithelial-derived cancers by epigenetic mechanisms, although the functional relevance of such DUOX silencing has remained unclear. This review will briefly summarize our current understanding regarding the importance of

Download English Version:

https://daneshyari.com/en/article/5501720

Download Persian Version:

https://daneshyari.com/article/5501720

<u>Daneshyari.com</u>