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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 Radical Biology and Medicine*, <http://dx.doi.org/10.1016/j.freeradbiomed.2017.05.024>

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PARADOXICAL ROLES OF DUAL OXIDASES IN CANCER BIOLOGY

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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

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