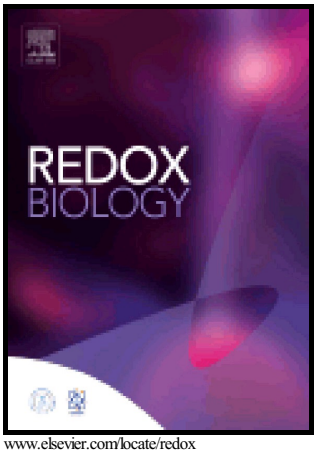
8-Oxoguanine DNA Glycosylase 1: Beyond Repair of the Oxidatively Modified Base Lesion 8-oxoG

Xueqing Ba, lstvan Boldogh



www.elsevier.com/locate/redox

PII: S2213-2317(17)30531-1
DOI: https://doi.org/10.1016/j.redox.2017.11.008
Reference: REDOX795

To appear in: *Redox Biology*

Received date: 31 July 2017
Revised date: 8 October 2017
Accepted date: 8 November 2017

Cite this article as: Xueqing Ba and lstvan Boldogh, 8-Oxoguanine DNA Glycosylase 1: Beyond Repair of the Oxidatively Modified Base Lesion 8-oxoG, *Redox Biology,* https://doi.org/10.1016/j.redox.2017.11.008

8-Oxoguanine DNA Glycosylase 1: Beyond Repair of the Oxidatively Modified Base Lesion 8-oxoG

Xueqing Ba[1,2]*, Istvan Boldogh[3,4]*

[1]The Key Laboratory of Molecular Epigenetics of Ministry of Education, 2. Institute of Genetics and Cytology, Northeast Normal University, Changchun, Jilin 130024, China

[3]Department of Microbiology and Immunology, 4. Sealy Center for Molecular Medicine, University of Texas Medical Branch at Galveston, Galveston, Texas 77555, USA

baxq755@nenu.edu.cn

sboldogh@utmb.edu

* To whom correspondence should be addressed: Xueqing Ba, PhD, Key Laboratory of Molecular Epigenetics of Ministry of Education, Institute of Genetics and Cytology, Northeast Normal University; 5268 Renmin Street, Changchun, Jilin 130024, China, Telephone: 86-431-85098837; Fax: 86-431-85098735.

*Correspondence may also be addressed to: Istvan Boldogh, DM&B, PhD Department of Microbiology and Immunology; University of Texas Medical Branch at Galveston, 301 University Blvd, Galveston, Texas 77555-1070, Telephone: +1409-772-9414, Fax: +1409-747-6869

Abstract

Oxidative stress and the resulting damage to genomic DNA are inevitable consequences of endogenous physiological processes, and they are amplified by cellular responses to environmental exposures. One of the most frequent reactions of reactive oxygen species with DNA is the oxidation of guanine to pre-mutagenic 8-oxo-7,8-dihydroguanine. Despite the vulnerability of guanine to oxidation, vertebrate genes are primarily embedded in high GC-rich genomic regions, and over 72% of the promoters of human genes belong to a class with a high GC content. Here, we discuss how the occurrence of guanine oxidation at a given genomic region(s) is not random. It may serve as an epigenetic mark, and when complexed with the redox-inactivated repair enzyme 8-oxoguanine DNA glycosylase1, it may provide a platform for the coordination of the initial steps of DNA repair and the assembly of the transcriptional machinery to launch the prompt and preferential expression of redox-regulated genes. Deviations/variations from this artful coordination may represent the etiological links between guanine oxidation and various cellular pathologies and diseases, and indicate the unscheduled decline of cell/tissue functions during ageing-related processes.