

Product Information



DNA/RNA Damage Monoclonal Antibody (Clone 15A3)

Catalog No. 10011446

Contents:	This vial contains protein G-purified antibody at a concentration of 0.65 mg/ml in TBS containing 50% glycerol and 0.1% sodium azide.
Antigen:	8-hydroxy guanosine-BSA and casein conjugates
Host:	Mouse, clone 15A3
Cross-reactivity:	(+)8-hydroxy-2-deoxy guanosine, 8-hydroxy guanine, and 8-hydroxy guanosine
Isotype:	IgG _{2α}
Stability:	≥1 year at -20°C
Applications:	ELISA, immunohistochemistry, and may also be used on immunoaffinity columns. Optimal working dilutions should be determined empirically.

DNA and RNA damage is due to environmental factors and normal metabolic processes inside the cell, that then hinder the ability of the cell to carry out its functions. Four main types of DNA endogenous cellular processes are oxidation, alkylation, hydrolysis, and of the mismatch bases. Oxidation of bases is caused by reaction with reactive oxygen and nitrogen species (RONS), which include nitric oxide, superoxide, hydroxyl radical, hydrogen peroxide, and peroxynitrite. Numerous studies have shown that RONS causes a variety of biomolecular modifications including DNA damage.¹

8-hydroxy guanine, 8-hydroxy 2'-deoxy guanosine, and 8-hydroxy guanosine are all RNA and DNA markers of oxidative damage. 8-hydroxy 2'-guanosine is produced by RONS including hydroxyl radical peroxynitrite. Specifically its high biological relevance is due to its ability to induce G to T transversions, which is one of the most frequent somatic mutations.² 8-hydroxy guanine has been the most frequently studied type of DNA base damage. Base modifications of this type arise from radical-induced hydroxylation and cleavage reactions of the purine ring.^{3,4} 8-hydroxy guanosine, like 8-hydroxy 2'-guanosine, induces a mutagenic transversion of G to T in DNA. Its role has specifically been tested in the development of diabetes, hypertension, and stroke.⁵⁻⁷

References

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2. Pilger, A. and Rüdiger, H.W. 8-Hydroxy-2'-deoxyguanosine as a marker of oxidative DNA damage related to occupational environmental exposures. *International Archives of Occupational and Environmental Health* **80**(1), 1-15 (2006).
3. Malins, D.C. and Haimanot, R. Major alterations in the nucleotide structure of DNA in cancer of the female breast. *Cancer Res.* **51**, 5430-5432 (1991).
4. Kvam, E. and Tyrrell, R.M. Artificial background and induced levels of oxidative base damage in DNA from human cells. *Carcinogenesis* **18**(11), 2281-2283 (1997).
5. Kowluru, R.A., Atasi, L., and Ho, Y.-S. Role of mitochondrial superoxide dismutase in the development of diabetic retinopathy. *Invest. Ophthalmol. Vis. Sci.* **47**, 1594-1599 (2006).
6. Bowers, R., Cool, C., Murphy, R.C., *et al.* Oxidative stress in severe pulmonary hypertension. *Am. J. Respir. Crit. Care Med.* **169**, 764-769 (2004).
7. Cui, J., Holmes, E.H., Greene, T.G., *et al.* Oxidative DNA damage precedes DNA fragmentation after experimental stroke in rat brain. *FASEB J.* **14**, 955-967 (2000).

WARNING: THIS PRODUCT IS NOT FOR HUMAN OR ANIMAL DISEASE DIAGNOSIS OR THERAPEUTIC DRUG USE.

MATERIAL SAFETY DATA

This material should be considered hazardous until information to the contrary becomes available. Do not ingest, swallow, or inhale. Do not get in eyes, on skin, or on clothing. Wash thoroughly after handling. This information contains some, but not all, of the information required for the safe and proper use of this material. Before use, the user must review the complete Material Safety Data Sheet, which has been sent under separate cover to the MSDS supervisor at your institution.

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Cayman Chemical

Mailing address

1180 E. Ellsworth Road
Ann Arbor, MI
48108 USA

Phone

(800) 364-9897
(734) 971-3335

Fax

(734) 971-3640

E-Mail

custserv@caymanchem.com

Web

www.caymanchem.com