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DATA SHEET

Rabbit Anti-Malondialdehyde/MDA Polyclonal: RC0011

Intended Use: For Research Use Only

Description: Malondialdehyde (MDA) is a natural product formed in all mammalian cells as a product of lipid peroxidation. MDA is a highly reactive three carbon dialdehyde produced as a byproduct of polyunsaturated fatty acid peroxidation and arachidonic acid metabolism. MDA readily combines with several functional groups on molecules including proteins, lipoproteins, and DNA. It reacts with DNA to form adducts to deoxyguanosine and deoxyadenosine. The major adduct to DNA is a pyrimidopurinone called M1G which appears to be a major endogenous DNA adduct in human beings that may contribute significantly to cancer linked to lifestyle and dietary factors. MDA modified proteins may show altered physico chemical behavior and antigenicity. MDA is toxic and has been implicated in aging mutagenesis, carcinogenesis, diabetic nephropathy and radiation damage. Increased expression of MDA has been reported in the brains of Alzheimer's patients. Antibodies to MDA will help to visualize the MDA adducts.

Specifications

Clone:	Polyclonal
Source:	Rabbit
Isotype:	IgG
Reactivity:	Pan species (General)
Immunogen:	Small molecule MDA conjugated to OVA
Localization:	Cytoplasm
Formulation:	Antibody in PBS pH7.4, containing BSA and ≤ 0.09% sodium azide (NaN3)
Storage:	Store at 2°- 8°C
Applications:	IHC - frozen tissue, ICC

Package:

Description	Catalog No.	Size
Malondialdehyde/MDA Concentrated	RC0011	1 ml

IHC Procedure*

Positive Control Tissue:	Rectal cancer, kidney
Concentrated Dilution:	User determined
Pretreatment:	None
Incubation Time and Temp:	Overnight @ 4°C
Detection:	Refer to the detection system manual

* Result should be confirmed by an established diagnostic procedure.

References:

1. Alpha-Phenyl-n-tert-butyl-nitrone attenuates lipopolysaccharide-induced neuronal injury in the neonatal rat brain. Fan, Mitchell et. Al. Neuroscience, Vol. 151, Issue 3, pp. 737-44, 2008.
2. Thiamine deficiency induces oxidative stress and exacerbates the plaque pathology in Alzheimer's mouse model. Karuppagounder, Xu. Neurobiology of aging, 2008.
3. Hereditary ferritinopathy: a novel mutation, its cellular pathology, and pathogenetic insights. Mancuso, Davidzon, et al. Journal of neuropathology and experimental neurology, Vol. 64, Issue 4, pp. 280-94, 2005.
4. Increased JNK phosphorylation and oxidative stress in response to increased glucose flux through increased GLUT1 expression in rat retinal endothelial cells. Zhou, Deo, et al. Investigative ophthalmology & visual science, Vol. 46, Issue 9, pp. 3403-10, 2005.

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