

MDA-BSA

CATALOG NUMBER: STA-333

STORAGE: -20°C

QUANTITY AND CONCENTRATION: 100 µL of 1.0 mg/mL MDA-BSA in 1X PBS.

SHELF LIFE: 1 year from date of receipt under proper storage conditions; aliquot to avoid multiple freeze thaw cycles

Background

Lipid peroxidation is a well-defined mechanism of cellular damage in animals and plants. Lipid peroxides are unstable indicators of oxidative stress in cells that decompose to form more complex and reactive compounds such as Malondialdehyde (MDA) and 4-hydroxynonenal (4-HNE), natural bi-products of lipid peroxidation. Oxidative modification of lipids can be induced *in vitro* by a wide array of pro-oxidant agents and occurs *in vivo* during aging and in certain disease conditions. Measuring the end products of lipid peroxidation is one of the most widely accepted assays for oxidative damage. These aldehydic secondary products of lipid peroxidation are generally accepted markers of oxidative stress.

Both MDA and HNE have been shown to be capable of binding to proteins and forming stable adducts, also termed advanced lipid peroxidation end products. These modifications of proteins by MDA or HNE can cause both structural and functional changes of oxidized proteins.

Methods

Dilute the MDA-BSA with SDS-PAGE reducing sample buffer to 1.0-10 µg/mL and boil for 5 minutes. Load 10 µL per lane for western blot analysis of MDA protein adducts.

Example of Results

The following figures demonstrate typical results. One should use the data below for reference only. This data should not be used to interpret actual results.

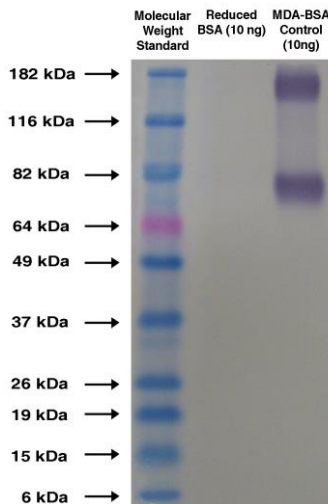


Figure 1: Immunoblotting of MDA-BSA. MDA-BSA was first electroblotted onto nitrocellulose membrane. Following the electroblotting procedure, the MDA was detected with an Rabbit anti-MDA Antibody.

References

1. Bourdel-Marchasson, I. et al. 2001. *Age Aging* **30**, 235.
2. Kinalski, M., et al. 2000. *Acta Diabetol.* **37**, 179.

Recent Product Citations

1. Steffen, J.B.M. et al. (2023). Combined effects of salinity and intermittent hypoxia on mitochondrial capacity and reactive oxygen species efflux in the Pacific oyster, *Crassostrea gigas*. *J Exp Biol.* **226**(15):jeb246164. doi: 10.1242/jeb.246164.
2. Steffen, J.B.M. et al. (2021). Mitochondrial capacity and reactive oxygen species production during hypoxia and reoxygenation in the ocean quahog, *Arctica islandica*. *J Exp Biol.* **224**(21):jeb243082. doi: 10.1242/jeb.243082.
3. Krilis, M. et al. (2021). Clinical relevance of nitrated beta 2-glycoprotein I in antiphospholipid syndrome: Implications for thrombosis risk. *J Autoimmun.* **122**:102675. doi: 10.1016/j.jaut.2021.102675.
4. Li, H. et al. (2015). Interferon-induced mechanosensing defects impede apoptotic cell clearance in lupus. *J Clin Invest.* **125**:2877-2890.
5. Joseph, K. et al. (2013). Oxidative stress sensitizes retinal pigmented epithelial (RPE) cells to complement-mediated injury in a natural antibody-, lectin pathway-, and phospholipid epitope-dependent manner. *J. Biol. Chem.* **288**: 12753-12765.

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