

Rabbit Anti-4-Hydroxynonenal (HNE) Polyclonal Antibody

CATALOG NUMBER:	STA-035	STORAGE:	-20°C
QUANTITY AND CONCENTRATION:	100 µg of affinity purified antibody at 0.5 mg/mL in PBS, pH 7.2, containing 0.5 mM EDTA, 0.02% NaN ₃ , and 30% Glycerol		
SHELF LIFE:	1 year from date of receipt under proper storage conditions; aliquot to avoid multiple freeze thaw cycles		
HOST SPECIES:	Rabbit		
IMMUNOGEN:	HNE-modified Blue Carrier		
SPECIFICITY:	HNE-modified proteins		
APPLICATION:	Immunoblot (1:200 to 1:8000) ELISA (1:200 to 1:8000)		

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.

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.

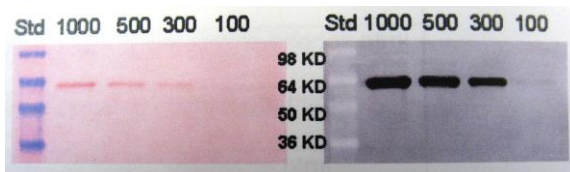


Figure 1. Immunoblot of HNE-Modified BSA. Left: Ponceau S staining. **Right:** Immunoblot using Rabbit Anti-HNE Polyclonal Antibody at 1:1000 dilution, followed by HRP-conjugated secondary antibody. Numbers indicate ng/lane.

Recent Product Citations

1. Nagaoka, N. et al. (2025). Stimulation at the frontal cortex influences the exercise activity and skeletal muscle status in senescence-accelerating mice. *NPJ Aging*. **11**(1):94. doi: 10.1038/s41514-025-00285-2.
2. Saha, S. et al. (2024). TCF4 trinucleotide repeat expansions and UV irradiation increase susceptibility to ferroptosis in Fuchs endothelial corneal dystrophy. *Redox Biol.* doi: 10.1016/j.redox.2024.103348.
3. Neurohr, J.M. et al. (2021). A higher mitochondrial content is associated with greater oxidative damage, oxidative defenses, protein synthesis and ATP turnover in resting skeletal muscle. *J Exp Biol.* doi: 10.1242/jeb.242462.
4. Tamai, Y. et al. (2021). Branched-chain amino acids and l-carnitine attenuate lipotoxic hepatocellular damage in rat cirrhotic liver. *Biomed Pharmacother.* **135**:111181. doi: 10.1016/j.biopha.2020.111181.
5. Cannizzaro, L. et al. (2017). Regulatory landscape of AGE-RAGE-oxidative stress axis and its modulation by PPAR γ activation in high fructose diet-induced metabolic syndrome. *Nutr Metab (Lond)*. **14**:5. doi: 10.1186/s12986-016-0149-z.
6. Omori, K. et al. (2016). Involvement of a pro-apoptotic gene BBC3 in islet injury mediated by cold preservation and re-warming. *Am J Physiol Endocrinol Metab.* doi:10.1152/ajpendo.00441.2015.
7. Wilson, W. N. et al. (2014). Effects of resveratrol on growth and skeletal muscle physiology of juvenile southern flounder. *Comp Biochem Physiol A Mol Integr Physiol.* **183**:27-35.

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