

Mouse Anti- Methylglyoxal Monoclonal Antibody

CATALOG NUMBER:	STA-011	STORAGE:	-20°C
QUANTITY AND CONCENTRATION:	100 µg of affinity purified antibody at 0.6 mg/mL in PBS containing 0.2% 5-bromo-5-nitro-1,3-dioxane		
SHELF LIFE:	1 year from date of receipt under proper storage conditions; aliquot to avoid multiple freeze thaw cycles		
HOST SPECIES:	Mouse		
CLONE:	3D11		
IMMUNOGEN:	MG-modified ovalbumin		
SPECIFICITY:	MG-modified proteins, lipids and nucleic acids (MG-H1 (methyl-glyoxal-hydro-imidazolone) based on HPLC and GC-MS). 3D11 does not react with CML, CEL, or other AGE epitopes.		
APPLICATION:	Immunoblot (1:1000 to 1:4000) Immunohistochemistry (1:20 to 1:60)		

Background

The non-enzymatic reaction of reducing carbohydrates with lysine side chains and N-terminal amino groups of macromolecules (proteins, phospholipids and nucleic acids) is called the Maillard reaction or glycation. The products of this process, termed advanced glycation end products (AGEs), adversely affect the functional properties of proteins, lipids and DNA. Tissue levels of AGE increase with age and the formation of AGEs is predominantly endogenous, though these products can also be derived from exogenous sources such as food and tobacco smoke. AGE modification of proteins can contribute to the pathophysiology of aging and long-term complications of diabetes, atherosclerosis and renal failure. AGEs also interact with a variety of cell-surface AGE-binding receptors (RAGE), leading either to their endocytosis and degradation or to cellular activation and pro-oxidant or pro-inflammatory events.

Several AGE structures have been reported, such as N^ε-(carboxymethyl) lysine (CML), N^ε-(carboxyethyl) lysine (CEL), pentosidine, and Methylglyoxal (MG) derivatives. MG is formed through non-oxidative mechanisms from triose phosphates during anaerobic glycolysis and it can modify amino acids, nucleic acids, and proteins. MG reacts with arginine, lysine and cysteine residues of proteins to form AGEs. MG is involved in various pathological processes. For example, MG derivatives are found elevated in diabetes.

Recent Product Citations

1. Jang, S. et al. (2017). Generation and characterization of mouse knockout for glyoxalase 1. *Biochem. Biophys. Res. Commun.* doi:10.1016/j.bbrc.2017.06.063.
2. Dafre, A.L., et al. (2017). Methylglyoxal-induced AMPK activation leads to autophagic degradation of thioredoxin 1 and glyoxalase 2 in HT22 nerve cells. *Free Radic Biol Med.* **108**:270-279. doi: 10.1016/j.freeradbiomed.2017.03.028.
3. Illien-Junger, S. et al. (2016). AGEs induce ectopic endochondral ossification in intervertebral discs. *Eur. Cell Mater.* **32**:257-270.
4. Dafre, A. L. et al. (2015). Methylglyoxal, the foe and friend of glyoxalase and Trx/TrxR systems in HT22 nerve cells. *Free Radic Biol Med.* doi: 10.1016/j.freeradbiomed.2015.07.005.
5. Currais, A. et al. (2015). Dietary glycemic index modulates the behavioral and biochemical abnormalities associated with autism spectrum disorder. *Mol Psychiatry.* doi:10.1038/mp.2015.64.

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