

## Mouse Anti-Methylglyoxal Monoclonal Antibody

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<b>CATALOG NUMBER:</b>	STA-011	<b>STORAGE:</b>	-20°C
<b>QUANTITY AND CONCENTRATION:</b>	100 µg of affinity purified antibody at 1.25 mg/mL in PBS containing 0.2% 5-bromo-5-nitro-1,3-dioxane		
<b>SHELF LIFE:</b>	1 year from date of receipt under proper storage conditions; aliquot to avoid multiple freeze thaw cycles		
<b>HOST SPECIES:</b>	Mouse		
<b>CLONE:</b>	3D11		
<b>IMMUNOGEN:</b>	MG-modified ovalbumin		
<b>SPECIFICITY:</b>	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.		
<b>APPLICATION:</b>	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<sup>ε</sup>-(carboxymethyl) lysine (CML), N<sup>ε</sup>-(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**

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4. Wan, Y.X. et al. (2024). Electroacupuncture facilitates vascular normalization by inhibiting Glyoxalase1 in endothelial cells to attenuate glycolysis and angiogenesis in triple-negative breast cancer. *Cancer Lett*. doi: 10.1016/j.canlet.2024.217094.
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8. Chandrakumar, S. et al. (2023). Subendothelial matrix stiffening by lysyl oxidase enhances RAGE-mediated retinal endothelial activation in diabetes. *Diabetes*. doi: 10.2337/db22-0761.
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11. Smith, A.J. et al. (2022). GATD3A, a mitochondrial deglycase with evolutionary origins from gammaproteobacteria, restricts the formation of advanced glycation end products. *BMC Biol*. **20**(1):68. doi: 10.1186/s12915-022-01267-6.
12. Heremans, I.P. et al. (2022). Parkinson's disease protein PARK7 prevents metabolite and protein damage caused by a glycolytic metabolite. *Proc Natl Acad Sci U S A*. **119**(4):e2111338119. doi: 10.1073/pnas.2111338119.
13. Cimenci, C.E. et al. (2021). Combined Methylglyoxal Scavenger and Collagen Hydrogel Therapy Prevents Adverse Remodeling and Improves Cardiac Function Post-Myocardial Infarction. *Adv. Funct. Mater*. doi: 10.1002/adfm.202108630.
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15. Pariano, M. et al. (2021). Defective Glyoxalase 1 Contributes to Pathogenic Inflammation in Cystic Fibrosis. *Vaccines (Basel)*. **9**(11):1311. doi: 10.3390/vaccines9111311.
16. McEwen, J.M. et al. (2021). Synergistic sequence contributions bias glycation outcomes. *Nat Commun*. **12**(1):3316. doi: 10.1038/s41467-021-23625-8.
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