

Mouse Anti-Methylglyoxal Monoclonal Antibody

CATALOG NUMBER:	STA-011	STORAGE:	-20°C
QUANTITY AND CONCENTRATION:	100 µg of affinity purified antibody at 1.25 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. Borysiuk, K. et al. (2022). Glyoxalase I activity affects Arabidopsis sensitivity to ammonium nutrition. *Plant Cell Rep.* doi: 10.1007/s00299-022-02931-5.
2. 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.
3. 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.
4. 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.
5. Chou, C.K. et al. (2021). Methylglyoxal Levels in Human Colorectal Precancer and Cancer: Analysis of Tumor and Peritumor Tissue. *Life.* **11**(12):1319. doi: 10.3390/life11121319.
6. Pariano, M. et al. (2021). Defective Glyoxalase 1 Contributes to Pathogenic Inflammation in Cystic Fibrosis. *Vaccines (Basel).* **9**(11):1311. doi: 10.3390/vaccines9111311.
7. McEwen, J.M. et al. (2021). Synergistic sequence contributions bias glycation outcomes. *Nat Commun.* **12**(1):3316. doi: 10.1038/s41467-021-23625-8.
8. Kern, U. et al. (2021). Impact of DJ-1 and Helix 8 on the Proteome and Degradome of Neuron-Like Cells. *Cells.* **10**(2):404. doi: 10.3390/cells10020404.
9. Atzeni, I.M. et al. (2020). Is skin autofluorescence (SAF) representative of dermal advanced glycation endproducts (AGEs) in dark skin? A pilot study. *Heliyon.* **6**(11):e05364. doi: 10.1016/j.heliyon.2020.e05364.
10. Aragonès, G. et al. (2020). Autophagic receptor p62 protects against glycation-derived toxicity and enhances viability. *Aging Cell.* doi: 10.1111/ace1.13257.
11. Korça, E. et al. (2020). Circulating antibodies against age-modified proteins in patients with coronary atherosclerosis. *Sci Rep.* **10**:17105. doi: 10.1038/s41598-020-73877-5.
12. de Almeida, G.R.L. et al. (2020). Methylglyoxal-Mediated Dopamine Depletion, Working Memory Deficit, and Depression-Like Behavior Are Prevented by a Dopamine/Noradrenaline Reuptake Inhibitor. *Mol Neurobiol.* doi: 10.1007/s12035-020-02146-3.
13. Kim, D. et al. (2020). Methylglyoxal-Induced Dysfunction in Brain Endothelial Cells via the Suppression of Akt/HIF-1 α Pathway and Activation of Mitophagy Associated with Increased Reactive Oxygen Species. *Antioxidants (Basel).* **9**(9):E820. doi: 10.3390/antiox9090820.
14. Scumaci, D. et al. (2020). DJ-1 Proteoforms in Breast Cancer Cells: The Escape of Metabolic Epigenetic Misregulation. *Cells.* **9**(9):E1968. doi: 10.3390/cells9091968.
15. Kepchia, D. et al. (2020). Diverse proteins aggregate in mild cognitive impairment and Alzheimer's disease brain. *Alzheimers Res Ther.* **12**(1):75. doi: 10.1186/s13195-020-00641-2.
16. Rodrigues, D.C. et al. (2020). Methylglyoxal couples metabolic and translational control of Notch signalling in mammalian neural stem cells. *Nat Commun.* **11**(1):2018. doi: 10.1038/s41467-020-15941-2.
17. Zunkel, K. et al. (2020). Long-term intake of the reactive metabolite methylglyoxal is not toxic in mice. *Food Chem Toxicol.* doi: 10.1016/j.fct.2020.111333.
18. Bellier, J. et al. (2020). Methylglyoxal Scavengers Resensitize KRAS-Mutated Colorectal Tumors to Cetuximab. *Cell Rep.* **30**(5):1400-1416.e6. doi: 10.1016/j.celrep.2020.01.012.
19. Luengo, A. et al. (2019). Reactive metabolite production is a targetable liability of glycolytic metabolism in lung cancer. *Nat Commun.* **10**(1):5604. doi: 10.1038/s41467-019-13419-4.

20. Proietti, S. et al. (2019). GLYI4 Plays A Role in Methylglyoxal Detoxification and Jasmonate-Mediated Stress Responses in Arabidopsis thaliana. *Biomolecules*. **9**(10). pii: E635. doi: 10.3390/biom9100635.
21. Wang, Y. et al. (2019). Methylglyoxal Triggers Human Aortic Endothelial Cell Dysfunction via Modulating KATP/MAPK pathway. *Am J Physiol Cell Physiol*. doi: 10.1152/ajpcell.00117.2018.
22. Sudnitsyna, M.V. et al. (2019). Is the small heat shock protein HspB1 (Hsp27) a real and predominant target of methylglyoxal modification?. *Cell Stress Chaperones*. **24**(2):419-426. doi: 10.1007/s12192-019-00975-3.
23. Koike, S. et al. (2019). Age-related alteration in the distribution of methylglyoxal and its metabolic enzymes in the mouse brain. *Brain Res Bull*. **144**:164-170. doi: 10.1016/j.brainresbull.2018.11.025.
24. Papadaki, M. et al. (2018). Diabetes with heart failure increases methylglyoxal modifications in the sarcomere, which inhibit function. *JCI Insight*. **3**(20). pii: 121264. doi: 10.1172/jci.insight.121264.
25. Antognelli, C. et al. (2018). Testosterone and Follicle Stimulating Hormone-Dependent Glyoxalase 1 Up-Regulation Sustains the Viability of Porcine Sertoli Cells through the Control of Hydroimidazolone- and Argpyrimidine-Mediated NF- κ B Pathway. *Am J Pathol*. **188**(11):2553-2563. doi: 10.1016/j.ajpath.2018.07.013.
26. Coleman, V. et al. (2018). Partial involvement of Nrf2 in skeletal muscle mitohormesis as an adaptive response to mitochondrial uncoupling. *Sci Rep*. **8**(1):2446. doi: 10.1038/s41598-018-20901-4.
27. Mey, J.T. et al (2018). Dicarbonyl stress and glyoxalase enzyme system regulation in human skeletal muscle. *Am J Physiol Regul Integr Comp Physiol*. **314**(2):R181-R190. doi: 10.1152/ajpregu.00159.2017.
28. Ishida, Y.I. et al. (2017). Identification of an argpyrimidine-modified protein in human red blood cells from schizophrenic patients: A possible biomarker for diseases involving carbonyl stress. *Biochem Biophys Res Commun*. **493**(1):573-577. doi: 10.1016/j.bbrc.2017.08.150.
29. 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.
30. 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.

Warranty

These products are warranted to perform as described in their labeling and in Cell Biolabs literature when used in accordance with their instructions. THERE ARE NO WARRANTIES THAT EXTEND BEYOND THIS EXPRESSED WARRANTY AND CELL BIOLABS DISCLAIMS ANY IMPLIED WARRANTY OF MERCHANTABILITY OR WARRANTY OF FITNESS FOR PARTICULAR PURPOSE. CELL BIOLABS 's sole obligation and purchaser's exclusive remedy for breach of this warranty shall be, at the option of CELL BIOLABS, to repair or replace the products. In no event shall CELL BIOLABS be liable for any proximate, incidental or consequential damages in connection with the products.

This product is for RESEARCH USE ONLY; not for use in diagnostic procedures.

Contact Information

Cell Biolabs, Inc.

7758 Arjons Drive

San Diego, CA 92126

Worldwide: +1 858-271-6500

USA Toll-Free: 1-888-CBL-0505

E-mail: tech@cellbiolabs.com

www.cellbiolabs.com

©2011-2022: Cell Biolabs, Inc. - All rights reserved. No part of these works may be reproduced in any form without permissions in writing.