Mouse RAGE Antibody

Monoclonal Rat IgG_{2B} Clone # 697005

Catalog Number: MAB11794

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse RAGE in direct ELISAs. In direct ELISAs, 100% cross-reactivity with recombinant rat RAGE, 20% - 50% cross-reactivity with recombinant human RAGE, and no cross-reactivity with recombinant canine RAGE is observed.
Source	Monoclonal Rat IgG_{2B} Clone # 697005
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse RAGE Gly23-Leu342 Accession # Q62151
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 μm filtered solution in PBS.

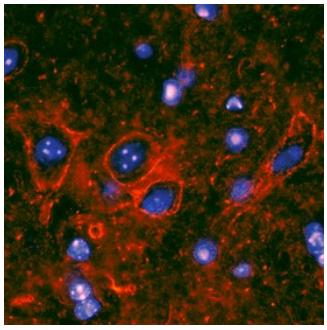
APPLICATIONS

Please Note: *Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.*

	Recommended Concentration	Sample
Immunohistochemistry	8-25 µg/mL	See Below

DATA

Immunohistochemistry



RAGE in Mouse Spinal Cord. RAGE was detected in perfusion fixed frozen sections of mouse spinal cord using Rat Anti-Mouse RAGE Monoclonal Antibody (Catalog # MAB11794) at 25 µg/mL overnight at 4 °C. Tissue was stained using the NorthernLights™ 557-conjugated Anti-Rat IgG Secondary Antibody (red; Catalog # NL013) and counterstained with DAPI (blue). Specific staining was localized to the plasma membranes of motor neurons. View our protocol for Fluorescent IHC Staining of Frozen Tissue Sections.

PREPARATION AND STORAGE

Reconstitution	Sterile PBS to a final concentration of 0.5 mg/mL.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

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BACKGROUND

Advanced glycation endproducts (AGE) are adducts formed by the non-enzymatic glycation or oxidation of macromolecules (1). AGE forms during aging and its formation is accelerated under pathophysiologic states such as diabetes, Alzheimer's disease, renal failure and immune/inflammatory disorders. Receptor for Advanced Glycation Endoproducts (RAGE), named for its ability to bind AGE, is a multiligand receptor belonging the immunoglobulin (Ig) superfamily. Besides AGE, RAGE binds amyloid β-peptide, S100/calgranulin family proteins, high mobility group B1 (HMGB1, also know as amphoterin) and leukocyte integrins (1, 2). The mouse RAGE gene encodes a 403 amino acid (aa) type I transmembrane glycoprotein with a 22 aa signal peptide, a 319 aa extracellular domain containing an Ig-like V-type domain and two Ig-like C-type domains, a 21 aa transmembrane domain and a 41 aa cytoplasmic domain (3). The V-type domain and the cytoplasmic domain are important for ligand binding and for intracellular signaling, respectively. Two alternative splice variants, lacking the V-type domain or the cytoplasmic tail, are known (1, 4). RAGE is highly expressed in the embryonic central nervous system (5). In adult tissues, RAGE is expressed at low levels in multiple tissues including endothelial and smooth muscle cells, mononuclear phagocytes, pericytes, microglia, neurons, cardiac myocytes and hepatocytes (6). The expression of RAGE is upregulated upon ligand interaction. Depending on the cellular context and interacting ligand, RAGE activation can trigger differential signaling pathways that affect divergent pathways of gene expression (1, 7). RAGE activation modulates varied essential cellular responses (including inflammation, immunity, proliferation, cellular adhesion and migration) that contribute to cellular dysfunction associated with chronic diseases such as diabetes, cancer, amyloidoses and immune or inflammatory disorders (1).

References:

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7. Valencia, J.V. *et al.* (2004) Diabetes **53**:743.

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