

#### DESCRIPTION

<b>Species Reactivity</b>	Mouse
<b>Specificity</b>	Detects mouse RAGE in direct ELISAs and Western blots. In direct ELISAs, approximately 25% cross-reactivity with recombinant rat RAGE, approximately 15% cross-reactivity with recombinant human RAGE, and less than 2% cross-reactivity with recombinant canine RAGE is observed.
<b>Source</b>	Polyclonal Goat IgG
<b>Purification</b>	Antigen Affinity-purified
<b>Immunogen</b>	Mouse myeloma cell line NS0-derived recombinant mouse RAGE Gln24-Ala342 Accession # O35444
<b>Endotoxin Level</b>	<0.10 EU per 1 µg of the antibody by the LAL method.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

#### 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.

	<b>Recommended Concentration</b>	<b>Sample</b>
<b>Western Blot</b>	0.1 µg/mL	Recombinant Mouse RAGE Fc Chimera (Catalog # 1179-RG)
<b>Immunohistochemistry</b>	5-15 µg/mL	Perfusion fixed frozen sections of mouse brain (cortex)
<b>Blockade of Receptor-ligand Interaction</b>	In a functional ELISA, 15-35 µg/mL of this antibody will block 50% of the binding of 500 ng/mL of biotinylated AGE-BSA to immobilized Recombinant Mouse RAGE Fc Chimera (Catalog # 1179-RG) coated at 5 µg/mL (100 µL/well). At 166 µg/mL, this antibody will block >90% of the binding.	

#### PREPARATION AND STORAGE

<b>Reconstitution</b>	Reconstitute at 0.2 mg/mL in sterile PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	<p><b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b></p> <ul style="list-style-type: none"> <li>● 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>● 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>● 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

#### 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 Endproducts (RAGE), named for its ability to bind AGE, is a multiligand receptor belonging to the immunoglobulin (Ig) superfamily. Besides AGE, RAGE binds amyloid β-peptide, S100/calgranulin family proteins, high mobility group B1 (HMGB1, also known as amphoterin) and leukocyte integrins (1, 2).

The mouse RAGE gene encodes a 403 amino acid (aa) residue type I transmembrane glycoprotein with a 22 aa signal peptide, a 319 aa extracellular domain containing a Ig-like V-type domain and two Ig-like Cε-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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5. Hori, O. *et al.* (1995) *J. Biol. Chem.* **270**:25752.
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