

| DESCRIPTION | |
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| Species Reactivity | Mouse |
| Specificity | Detects mouse RAGE in direct ELISAs and Western blots. In Western blots, approximately 15% cross-reactivity with recombinant canine RAGE and no cross-reactivity with recombinant human RAGE or recombinant rat RAGE is observed. |
| Source | Monoclonal Rat IgG _{2A} Clone # 697023 |
| Purification | Protein A or G purified from hybridoma culture supernatant |
| Immunogen | Mouse myeloma cell line NS0-derived recombinant mouse RAGE Gly23-Leu342 Accession # NP_031451 |
| Conjugate | Alexa Fluor 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 nm |
| Formulation | Supplied in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details. *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions. |

| APPLICATIONS | | |
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| Please Note: Optimal dilutions should be determined by each laboratory for each application. <i>General Protocols</i> are available in the <i>Technical Information</i> section on our website. | | |
| | Recommended Concentration | Sample |
| Flow Cytometry | 0.5 µg/10 ⁶ cells | See Below |

| DATA | |
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| <p>Flow Cytometry</p> | <p>Detection of RAGE in Mouse Splenocytes by Flow Cytometry. Mouse splenocytes either (A) unstimulated or (B) stimulated to induce Th1 cells were stained with Rat Anti-Mouse RAGE Alexa Fluor® 488-conjugated Monoclonal Antibody (Catalog # FAB11795G) and Rat Anti-Mouse CD4 APC-conjugated Monoclonal Antibody (Catalog # FAB554A). Quadrant markers were set based on control antibody staining (Catalog # IC006G). View our protocol for Staining Membrane-associated Proteins.</p> |

| PREPARATION AND STORAGE | |
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| Shipping | The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below. |
| Stability & Storage | Protect from light. Do not freeze. <ul style="list-style-type: none"> ● 12 months from date of receipt, 2 to 8 °C as supplied. |

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 pathophysiological 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, amyloidosis and immune or inflammatory disorders (1).

References:

1. Schmidt, A. *et al.* (2001) *J. Clin. Invest.* **108**:949.
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3. Renard, C. *et al.* (1997) *Mol. Pharmacol.* **52**:54.
4. Yonekura, H. *et al.* (2003) *Biochem. J.* **370**:1097.
5. Hori, O. *et al.* (1995) *J. Biol. Chem.* **270**:25752.
6. Brett, J. *et al.* (1993) *Am. J. Pathol.* **143**:1699.
7. Valencia, J.V. *et al.* (2004) *Diabetes* **53**:743.

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