

DESCRIPTION

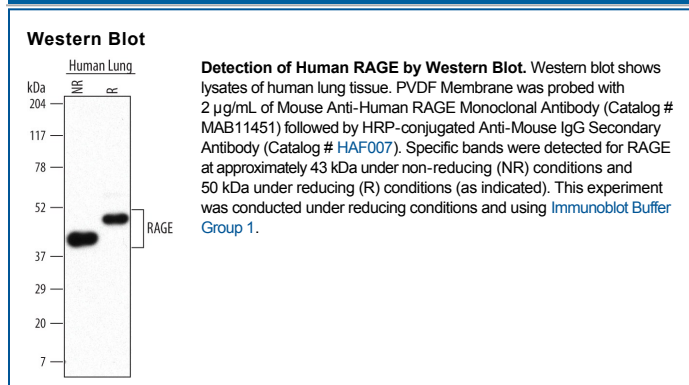
Species Reactivity	Human
Specificity	Detects human RAGE in ELISAs and Western blots. In Western blots, approximately 25% cross-reactivity with recombinant mouse RAGE and recombinant rat RAGE is observed.
Source	Monoclonal Mouse IgG _{2B} Clone # 176902
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human RAGE Gln24-Ala344 Accession # Q15109
Endotoxin Level	<0.01 EU per 1 µg of the antibody by the LAL method.
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
Western Blot	2 µg/mL	See Below
Human RAGE Sandwich Immunoassay		Reagent
ELISA Capture	2-8 µg/mL	Human RAGE Antibody (Catalog # MAB11451)
ELISA Detection Standard	0.1-0.4 µg/mL	Human RAGE Biotinylated Antibody (Catalog # BAF1145) Recombinant Human RAGE Fc Chimera (Catalog # 1145-RG)
Blockade of Receptor-ligand Interaction	In a functional ELISA, 1-3 µg/mL of this antibody will block 50% of the binding of 500 ng/mL of biotinylated AGE-BSA to immobilized recombinant human RAGE/Fc Chimera coated at 5 µg/mL (100 µL/well). At 10 µg/mL, this antibody will block >90% of the binding.	

DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
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. <ul style="list-style-type: none"> ● 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.

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 multi-ligand 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 human RAGE gene encodes a 404 amino acid residues (aa) type I transmembrane glycoprotein with a 22 aa signal peptide, a 320 aa extracellular domain containing an Ig-like V-type domain and two Ig-like Ce-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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