PRODUCT INFORMATION



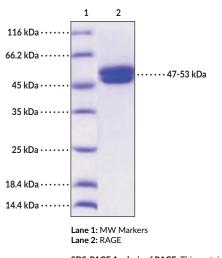
RAGE (human, recombinant)

Item No. 38057

Overview and Properties

Synonyms:	Advanced Glycosylation End Product-specific Receptor, AGER,
	Receptor for Advanced Glycosylation End Products
Source:	Active recombinant human His-tagged RAGE expressed in HEK293 cells
Amino Acids:	23-344
Uniprot No.:	Q15109
Molecular Weight:	35.5 kDa
Storage:	-80°C (as supplied)
Stability:	≥1 year
Purity:	≥98% estimated by SDS-PAGE
Supplied in:	Lyophilized from sterile PBS, pH 7.4
Endotoxin Testing:	$<1.0 EU/\mu g$ determined by the LAL endotoxin assay
Bioactivity:	See figures for details
Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.	

Image



SDS-PAGE Analysis of RAGE. This protein has a calculated molecular weight of 35.5 kDa. It has an apparent molecular weight of approximately 47-53 kDa by SDS-PAGE under reducing conditions due to glycosylation.

WARNING THIS PRODUCT IS FOR RESEARCH ONLY - NOT FOR HUMAN OR VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

SAFETY DATA

This material should be considered hazardous until further information becomes available. Do not ingest, inhale, get in eyes, on skin, or on clothing. Wash thoroughly after handling. Before use, the user must review the complete Safety Data Sheet, which has been sent via email to your institution.

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PRODUCT INFORMATION



Description

Receptor for advanced glycation end products (RAGE) is a transmembrane protein and member of the IgG superfamily.¹ It is composed of one V-type domain that is responsible for extracellular ligand binding, two C-type domains, a transmembrane domain, and a cytosolic tail involved in intracellular signaling. In addition to full-length and membrane-bound forms, soluble forms of RAGE can be formed either by proteolytic cleavage or alternative splicing. It is highly expressed in the mature lung pneumocytes and embryonic cells and is expressed at lower levels in many other cell types. RAGE has a variety of ligands that share a common structural motif, including AGEs, high mobility group box 1 (HMGB1), certain S100 proteins, and amyloid-β peptide fibrils.² Ligand activation of RAGE induces binding of the cytoplasmic tail to bridge proteins, such as diaphanous-related formin 1 (DIAPH1) and myeloid differentiation factor 88 adapter-like protein (Mal), which then activate MEK/ERK, PI3K/Akt, and JAK/STAT signaling pathways, among others, and lead to activation of NF-κB. RAGE levels increase rapidly at sites of inflammation, and full-length RAGE is associated with a pro-inflammatory state.^{1,2} Protein levels of RAGE are increased in postmortem coronary atherosclerotic lesions from patients with diabetes who died from sudden cardiovascular complications.^{3,4} However, plasma levels of soluble RAGE are lower in non-diabetic men with coronary artery disease (CAD) than those without CAD.^{4,5} Levels of full-length RAGE are decreased in non-small cell lung carcinoma (NSCLC) tissue compared with non-cancerous lung tissue, and proliferation of lung cancer cells overexpressing RAGE is reduced in vitro.⁶ Cayman's RAGE (human, recombinant) protein can be used for binding assays. This protein consists of 332 amino acids, has a calculated molecular weight of 35.5 kDa, and a predicted N-terminus of Ala23 after signal peptide cleavage. By SDS-PAGE, under reducing conditions, the apparent molecular mass of the protein is 47-53 kDa due to glycosylation.

References

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- 3. Burke, A.P., Kolodgie, F.D., Zieske, A., *et al.* Morphologic findings of coronary atherosclerotic plaques in diabetics: A postmortem study. *Arterioscler. Thromb. Vasc. Biol.* **24(7)**, 1266-1271 (2004).
- 4. Yan, S.F., Ramasamy, R., and Schmidt, A.M. Receptor for AGE (RAGE) and its ligands Cast into leading roles in diabetes and the inflammatory response. *J. Mol. Med.* (*Berl*) **87(3**), 235-247 (2009).
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- 6. Bartling, B., Hofmann, H.-S., Weigle, B., *et al.* Down-regulation of the receptor for advanced glycation end-products (RAGE) supports non-small cell lung carcinoma. *Carcinogenesis* **26(2)**, 293-301 (2005).

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