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RAGE Rabbit pAb

Catalog # AP99260

Product Information

Application	WB, IHC-P, IHC-F, IF
Primary Accession	Q15109
Other Accession	Q15109
Reactivity	Rat, Mouse
Predicted	Pig, Human, Dog
Host	Rabbit
Clonality	Polyclonal
Calculated MW	42803
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from human AGER Isoform 1, not for Isoform 2
Epitope Specificity	41-150/404
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SIMILARITY	Contains 2 Ig-like C2-type (immunoglobulin-like) domains.Contains 1 Ig-like V-type (immunoglobulin-like) domain.
SUBUNIT	Interacts with S100B, S100A1 and APP. Interacts with S100A12.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	Advanced glycosylation end product-specific receptor (AGER; RAGE) is a member of the immunoglobulin superfamily of cell surface molecules that binds molecules that have been irreversibly modified by non-enzymatic glycation and oxidation, and are know as advanced glycation end products (AGEs). It is expressed by endothelium, mononuclear phagocytes, neurons and smooth muscle cells. Whereas RAGE is present at high levels during development, especially in the central nervous system, its levels decline during maturity.The increased expression of RAGE is associated with several pathological states, such as diabetic vasculopathy, neuropathy, retinopathy and other disorders, including Alzheimer's disease and immune/inflammatory reactions of the vessel walls. In diabetic tissues, the production of RAGE is due to the overproduction of AGEs that eventually overwhelm the protective properties of RAGE. This results in oxidative stress and endothelial cell dysfunction that leads to vascular disease in diabetics. In the brain, RAGE also binds amyloid beta (Ab). Because Ab is overproduced in neurons and vessels in the brains of Alzheimer disease, this leads to the hyperstimulation of RAGE. The RAGE-Ab interaction is thought to result in oxidative stress leading to neuronal degeneration.

Additional Information

Gene ID	177

Other Names	RAGE; SCARJ1; sRAGE; RAGE_HUMAN; AGER; Receptor for advanced glycosylation end products; RAGE_MOUSE; RAGE_RAT; advanced glycosylation end-product specific receptor; receptor for advanced glycation end-products
Dilution	WB=1:500-2000,IHC-P=1:100-500,IHC-F=1:100-500,IF=1:100-500
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

Protein Information

Name	AGER
Synonyms	RAGE
Function	Cell surface pattern recognition receptor that senses endogenous stress signals with a broad ligand repertoire including advanced glycation end products, S100 proteins, high-mobility group box 1 protein/HMGB1, amyloid beta/APP oligomers, nucleic acids, histones, phospholipids and glycosaminoglycans (PubMed:27572515, PubMed:28515150, PubMed:34743181, PubMed:35974093, PubMed:24081950). Advanced glycation end products (AGEs) are nonenzymatically glycosylated proteins which accumulate in vascular tissue in aging and at an accelerated rate in diabetes (PubMed:21565706). These ligands accumulate at inflammatory sites during the pathogenesis of various diseases including diabetes, vascular complications, neurodegenerative disorders and cancers, and RAGE transduces their binding into pro-inflammatory responses. Upon ligand binding, uses TIRAP and MYD88 as adapters to transduce the signal ultimately leading to the induction of inflammatory cytokines IL6, IL8 and TNFalpha through activation of NF-kappa-B (PubMed:21829704, PubMed:33436632). Interaction with S100A12 on endothelium, mononuclear phagocytes, and lymphocytes triggers cellular activation, with generation of key pro-inflammatory mediators (PubMed:19386136). Interaction with S100B after myocardial infarction may play a role in myocyte apoptosis by activating ERK1/2 and p53/TP53 signaling (By similarity). Contributes to the translocation of amyloid- beta peptide (ABPP) across the cell membrane from the extracellular to the intracellular space in cortical neurons (PubMed:19906677). ABPP- initiated RAGE signaling, especially stimulation of p38 mitogen- activated protein kinase (MAPK), has the capacity to drive a transport system delivering ABPP as a complex with RAGE to the intraneuronal space. Participates in endothelial albumin transcytosis together with HMGB1 through the RAGE/SRC/Caveolin-1 pathway, leading to endothelial hyperpermeability (PubMed:27572515). Mediates the loading of HMGB1 in extracellular vesicles (EVs) that shuttle HMGB1 to hepatocytes by transferrin-mediated endocytosis and subsequently promote hepatocyte pyroptosis by activating the NLRP3 inflammasome (PubMed:34743181). Binds to DNA and promotes extracellular hypomethylated DNA (CpG DNA) uptake by cells via the endosomal route to activate inflammatory responses (PubMed:24081950, PubMed:28515150). Mediates phagocytosis by non-professional phagocytes (NPP) and this is enhanced by binding to ligands including RNA, DNA, HMGB1 and histones (PubMed:35974093). Promotes NPP-mediated phagocytosis of Saccharomyces cerevisiae spores by binding to RNA attached to the spore wall (PubMed:35974093). Also promotes NPP-mediated phagocytosis of apoptotic cells (PubMed:35974093). Following DNA damage, recruited to DNA double-strand break sites where it colocalizes with the MRN repair complex via interaction with double-strand break repair protein MRE11 (By similarity). Enhances the endonuclease activity of MRE11,

	promoting the end resection of damaged DNA (By similarity). Promotes DNA damage repair in trophoblasts which enhances trophoblast invasion and contributes to placental development and maintenance (PubMed:33918759). Protects cells from DNA replication stress by localizing to damaged replication forks where it stabilizes the MCM2-7 complex and promotes faithful progression of the replication fork (PubMed:36807739). Mediates the production of reactive oxygen species (ROS) in human endothelial cells (PubMed:25401185).
Cellular Location	Cell membrane; Single-pass type I membrane protein. Cell projection, phagocytic cup. Early endosome. Nucleus. Note=Detected on the surface of CD11c+ peripheral blood mononuclear cells under basal conditions and after activation (PubMed:22509345). No surface expression is observed on resting T cells (PubMed:22509345). Localizes intracellularly in early endosomes in activated T cells of healthy controls and in resting T cells of patients with type I diabetes (PubMed:22509345). Nuclear translocation is enhanced by irradiation, hypoxia and reperfusion injury to brain or kidney (By similarity). Nuclear localization is enhanced by DNA damage in trophoblasts and increases in pre-term labor and preeclampsia placentas compared to control placentas (PubMed:33918759). {ECO:0000250|UniProtKB:Q62151, ECO:0000269|PubMed:22509345, ECO:0000269|PubMed:33918759} [Isoform 2]: Secreted.
Tissue Location	Endothelial cells. Increased expression in pre-term labor and preeclampsia placentas compared to controls (PubMed:33918759).

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.