

**AGER Antibody (N-term) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP4861a****Specification**

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**AGER Antibody (N-term) Blocking Peptide - Product Information**Primary Accession [Q15109](#)**AGER Antibody (N-term) Blocking Peptide - Additional Information****Gene ID** 177**Other Names**

Advanced glycosylation end product-specific receptor, Receptor for advanced glycosylation end products, AGER, RAGE

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**AGER Antibody (N-term) Blocking Peptide - 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:<a href="http://www.uniprot.org/citations/27572515" target="\_blank">27572515</a>, PubMed:<a href="http://www.uniprot.org/citations/28515150" target="\_blank">28515150</a>, PubMed:<a href="http://www.uniprot.org/citations/34743181" target="\_blank">34743181</a>, PubMed:<a href="http://www.uniprot.org/citations/35974093" target="\_blank">35974093</a>, PubMed:<a href="http://www.uniprot.org/citations/24081950" target="\_blank">24081950</a>). Advanced glycosylation end products are nonenzymatically glycosylated proteins which accumulate in vascular tissue in aging and at an accelerated rate in diabetes (PubMed:<a href="http://www.uniprot.org/citations/21565706" target="\_blank">21565706</a>). 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:<a href="http://www.uniprot.org/citations/21829704" target="\_blank">21829704</a>, PubMed:<a href="http://www.uniprot.org/citations/33436632" target="\_blank">33436632</a>). Interaction with S100A12 on endothelium, mononuclear phagocytes, and lymphocytes triggers cellular activation, with generation of key pro-inflammatory mediators (PubMed:<a href="http://www.uniprot.org/citations/19386136" target="\_blank">19386136</a>). 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:<a href="http://www.uniprot.org/citations/19906677" target="\_blank">19906677</a>). 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:<a href="http://www.uniprot.org/citations/27572515" target="\_blank">27572515</a>). 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:<a href="http://www.uniprot.org/citations/34743181" target="\_blank">34743181</a>). Binds to DNA and promotes extracellular hypomethylated DNA (CpG DNA) uptake by cells via the endosomal route to activate inflammatory responses (PubMed:<a href="http://www.uniprot.org/citations/24081950" target="\_blank">24081950</a>, PubMed:<a href="http://www.uniprot.org/citations/28515150" target="\_blank">28515150</a>). Mediates phagocytosis by non-professional phagocytes (NPP) and this is enhanced by binding to ligands including RNA, DNA, HMGB1 and histones (PubMed:<a href="http://www.uniprot.org/citations/35974093" target="\_blank">35974093</a>). Promotes NPP-mediated phagocytosis of Saccharomyces cerevisiae spores by binding to RNA attached to the spore wall (PubMed:<a href="http://www.uniprot.org/citations/35974093" target="\_blank">35974093</a>). Also promotes NPP-mediated phagocytosis of apoptotic cells (PubMed:<a href="http://www.uniprot.org/citations/35974093" target="\_blank">35974093</a>). 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:<a href="http://www.uniprot.org/citations/33918759" target="\_blank">33918759</a>). 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:<a href="http://www.uniprot.org/citations/36807739" target="\_blank">36807739</a>). Mediates the production of reactive oxygen species (ROS) in human endothelial cells (PubMed:<a href="http://www.uniprot.org/citations/25401185" target="\_blank">25401185</a>).

### 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).

### **AGER Antibody (N-term) Blocking Peptide - Protocols**

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

### **AGER Antibody (N-term) Blocking Peptide - Images**

### **AGER Antibody (N-term) Blocking Peptide - Background**

AGER encodes a member of the immunoglobulin superfamily of cell surface molecules. It is a receptor for various molecules, including the amyloidogenic form of serum amyloid A, amyloid-beta protein, members of the S100/calgranulin superfamily and advanced glycation end products. The gene lies within the major histocompatibility complex (MHC) class III region on chromosome 6.

### **AGER Antibody (N-term) Blocking Peptide - References**

Rojas, A., et al. Carcinogenesis 31(3):334-341(2010)Engelen, L., et al. J. Hypertens. 28(2):285-293(2010)Poon, P.Y., et al. Clin. Nephrol. 73(1):44-50(2010)Hancock, D.B., et al. Nat. Genet. 42(1):45-52(2010)