SANTA CRUZ BIOTECHNOLOGY, INC.

RAGE (H-300): sc-5563

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Advanced glycosylation end products of proteins (AGEs) are non-enzymatically glycosylated proteins that are associated with a variety of conditions, including diabetes and other vascular disorders, as well as amyloidosis. These proteins regulate cellular functions via specific cell surface acceptor molecules, such as RAGE (receptor for advanced glycosylation end products). RAGE is a type 1 membrane protein that is found on the surface of endothelial cells, mononuclear phagocytes and vascular smooth muscle cells. Binding of AGEs to RAGE results in the induction of cellular oxidant stress and activation of the transcription factor NFκB. Evidence suggests that the induction of oxidant stress results in the activation of an intracellular cascade involving p21 ras and MAP kinase, which leads to activation of transcription.

CHROMOSOMAL LOCATION

Genetic locus: AGER (human) mapping to 6p21.32; Ager (mouse) mapping to 17 B1.

SOURCE

RAGE (H-300) is a rabbit polyclonal antibody raised against amino acids 1-300 of RAGE of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

RAGE (H-300) is recommended for detection of RAGE of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

RAGE (H-300) is also recommended for detection of RAGE in additional species, including equine.

Suitable for use as control antibody for RAGE siRNA (h): sc-36374, RAGE siRNA (m): sc-36375, RAGE siRNA (r): sc-106985, RAGE shRNA Plasmid (h): sc-36374-SH, RAGE shRNA Plasmid (m): sc-36375-SH, RAGE shRNA Plasmid (r): sc-106985-SH, RAGE shRNA (h) Lentiviral Particles: sc-36374-V, RAGE shRNA (m) Lentiviral Particles: sc-36375-V and RAGE shRNA (r) Lentiviral Particles: sc-106985-V.

Molecular Weight of RAGE: 46 kDa.

Positive Controls: RAGE (h2): 293T Lysate: sc-170841, rat lung extract: sc-2396 or mouse lung extract: sc-2390.

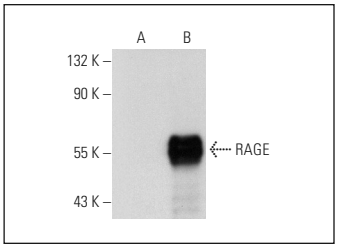
STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

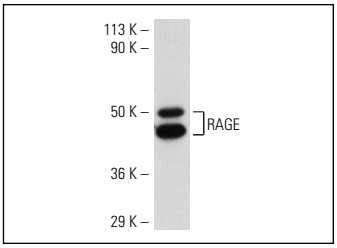
RESEARCH USE

For research use only, not for use in diagnostic procedures.

DATA



RAGE (H-300): sc-5563. Western blot analysis of RAGE expression in non-transfected: sc-117752 (**A**) and human RAGE transfected: sc-170841 (**B**) 293T whole cell lysates.



RAGE (H-300): sc-5563. Western blot analysis of RAGE expression in mouse lung extract.

SELECT PRODUCT CITATIONS

1. Lu, C., et al. 2004. Advanced glycation endproduct (AGE) receptor 1 is a negative regulator of the inflammatory response to AGE in mesangial. Proc. Natl. Acad. Sci. USA 101: 11767-11772.
2. Yamabe, N., et al. 2009. Matcha, a powdered green tea, ameliorates the progression of renal and hepatic damage in type 2 diabetic OLETF rats. J Med. Food 12: 714-721.
3. Pang, J., et al. 2009. Design, generation, and testing of mammalian expression modules that tag membrane proteins. Protein Sci. 18: 1261-1271.
4. Ma, H., et al. 2009. Advanced glycation endproduct (AGE) accumulation and AGE receptor (RAGE) up-regulation contribute to the onset of diabetic cardiomyopathy. J. Cell. Mol. Med. 13: 1751-1764.
5. Jaeger, L.B., et al. 2009. Lipopolysaccharide alters the blood-brain barrier transport of amyloid β protein: a mechanism for inflammation in the progression of Alzheimer's disease. Brain Behav. Immun. 23: 507-517.
6. Gefter, J.V., et al. 2009. Comparison of distinct protein isoforms of the receptor for advanced glycation end-products expressed in murine tissues and cell lines. Cell Tissue Res. 337: 79-89.
7. Banerjee, S., et al. 2010. The C-terminal acidic tail is responsible for the inhibitory effects of HMGB1 on efferocytosis. J. Leukoc. Biol. 88: 973-979.
8. Meilin, E., et al. 2010. Paraoxonase 2 (PON2) decreases high glucose-induced macrophage triglycerides (TG) accumulation, via inhibition of NADPH-oxidase and DGAT1 activity: studies in PON2-deficient mice. Atherosclerosis 208: 390-395.