

DESCRIPTION

Source Mouse myeloma cell line, NS0-derived
Asp25-Val339, with a C-terminal 6-His tag
Accession # ABA18645

N-terminal Sequence Analysis Asp25

Predicted Molecular Mass 34.6 kDa

SPECIFICATIONS

SDS-PAGE 40-44 kDa, reducing conditions

Activity Measured by its ability to bind biotinylated advanced glycation endproducts of bovine serum albumin (AGE-BSA, Catalog # BT4127).

Endotoxin Level <0.01 EU per 1 µg of the protein by the LAL method.

Purity >90%, by SDS-PAGE under reducing conditions and visualized by silver stain.

Formulation Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 100 µg/mL in sterile PBS.

Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Advanced glycation endproducts (AGEs) are adducts formed by the non-enzymatic glycation of macromolecules. AGE formation is accelerated in oxidative and hyperglycemic conditions, diabetes, renal failure, atherosclerosis, Alzheimer's disease, arthritis, and in normal aging (1 - 5). Receptor for advanced glycation endproducts (RAGE) is a 35 kDa type I transmembrane protein belonging to the immunoglobulin superfamily. Besides AGEs, RAGE binds β-amyloid peptide, S100/calgranulin family proteins, HMGB1/amphoterin, and leukocyte integrins (6 - 9). Mature canine RAGE consists of a 383 amino acid (aa) extracellular domain (ECD) with one Ig-like V-type domain and two Ig-like C-type domains, a 23 aa transmembrane segment, and a 43 aa cytoplasmic domain (10). Within the ECD, canine RAGE shares 73% - 77% aa sequence identity with human, mouse, and rat RAGE. In human, soluble forms of RAGE are generated by alternate splicing and are associated with multiple disease states (11, 12). RAGE is expressed in the embryonic central nervous system and on macrophages, monocytes, smooth muscle cells, and endothelial cells (13 - 15). It is upregulated in response to AGE accumulation, and its activation induces a broad proinflammatory response (6, 15). The increased production of reactive oxygen species during inflammation promotes additional AGE formation and RAGE upregulation, a cycle that exacerbates diabetic complications and inflammation-induced tissue injury (2, 4).

References:

1. Schleicher, E. and U. Friess (2007) *Kidney Int. Suppl.* **106**:S17.
2. Herold, K. *et al.* (2007) *J. Leukoc. Biol.* **82**:204.
3. Thornalley, P.J. (2006) *J. Ren. Nutr.* **16**:178.
4. Goldin, A. *et al.* (2006) *Circulation* **114**:597.
5. Ramasamy, R. *et al.* (2005) *Glycobiology* **15**:16R.
6. Kislinger, T. *et al.* (1999) *J. Biol. Chem.* **274**:31740.
7. Yan, S.D. *et al.* (1996) *Nature* **382**:685.
8. Huttenen, H. *et al.* (2000) *J. Biol. Chem.* **275**:40096.
9. Chavakis, T. *et al.* (2003) *J. Exp. Med.* **198**:1507.
10. Murua Escobar, H. *et al.* (2006) *Gene* **369**:45.
11. Yonekura H, *et al.* (2003) *Biochem. J.* **370**:1097.
12. Koyama, H. *et al.* (2007) *Mol. Med.* **13**:625.
13. Hori, O. *et al.* (1995) *J. Biol. Chem.* **270**:25752.
14. Brett, J. *et al.* (1993) *Am. J. Pathol.* **143**:1699.
15. Bierhaus, A. *et al.* (2006) *Curr. Opin. Investig. Drugs* **7**:985.