

DESCRIPTION

Source *E. coli*-derived
Thr2-Pro114
Accession # P06702

N-terminal Sequence Analysis Thr2

Predicted Molecular Mass 13 kDa

SPECIFICATIONS

SDS-PAGE 12 kDa, reducing conditions

Activity Measured by its ability to induce CXCL1/KC secretion by C3H10T1/2 mouse embryonic fibroblast cells. The ED₅₀ for this effect is 0.8-4 µg/mL.

Endotoxin Level <0.10 EU per 1 µg of the protein by the LAL method.

Purity >90%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

Formulation Lyophilized from a 0.2 µm filtered solution in Tris, TCEP and Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

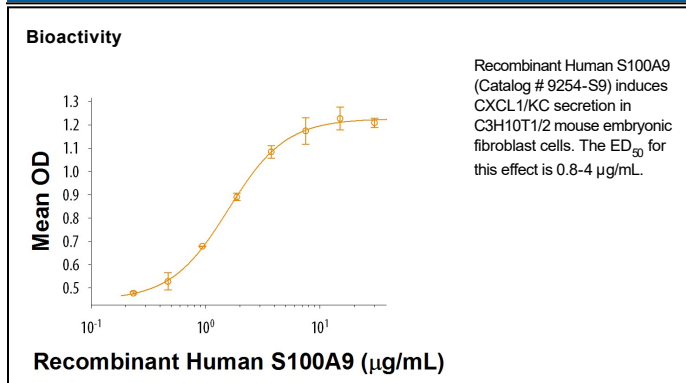
Reconstitution Reconstitute at 400 µg/mL in water.

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.

DATA



BACKGROUND

S100A9, also known as MRP14 and Calgranulin B, is an approximately 14 kDa pro-inflammatory protein in the S100 family of calcium binding proteins (1, 2). It is up-regulated in neutrophils and monocytes at sites of inflammation (e.g. psoriasis, rheumatoid arthritis, cardiac ischemia) and is present at elevated concentrations in rheumatoid arthritis synovial fluid (3-6). Human S100A9 contains two EF-hand calcium binding motifs and shares 57% and 62% amino acid sequence identity with mouse and rat S100A9, respectively (7). S100A9 is a noncovalent homodimer that can also noncovalently heterodimerize with S100A8; in the presence of calcium and zinc, the homodimer and heterodimers will form tetramers (8-10). Human S100A9 can be phosphorylated at Thr113 near the C-terminus, a residue which is not conserved in the mouse protein (11). Human S100A9 can also be modified by S-glutathionylation and oxidation (12). The S100A8/A9 heterodimer binds to fatty acids such as arachidonic acid, while neither monomer does (4). S100A9 and the heterodimer promote neutrophil infiltration and degranulation at sites of inflammation and inflammatory cytokine production by monocytes (5, 6, 9, 13). S100A9 also promotes cartilage matrix destruction in osteoarthritis (14) as well as RAGE-dependent cardiac fibrosis and remodeling (6). In the brain, the S100A9 homodimer promotes neuronal cytotoxicity, although this is reduced by its binding to amyloid-beta peptide (15).

References:

1. Averill, M.M. *et al.* (2012) *Arterioscler. Thromb. Vasc. Biol.* **32**:223.
2. Vogl, T. *et al.* (2012) *Int. J. Mol. Sci.* **13**:2893.
3. Edgeworth, J. *et al.* (1991) *J. Biol. Chem.* **266**:7706.
4. Siegenthaler, G. *et al.* (1997) *J. Biol. Chem.* **272**:9371.
5. Sunahori, K. *et al.* (2006) *Arthritis Res. Ther.* **8**:R69.
6. Volz, H.C. *et al.* (2012) *Basic Res. Cardiol.* **107**:250.
7. Odink, K. *et al.* (1987) *Nature* **330**:80.
8. Teigelkamp, S. *et al.* (1991) *J. Biol. Chem.* **266**:13462.
9. Ryckman, C. *et al.* (2003) *J. Immunol.* **170**:3233.
10. Vogl, T. *et al.* (2006) *Biochim. Biophys. Acta* **1763**:1298.
11. Edgeworth, J. *et al.* (1989) *Nature* **342**:189.
12. Lim, S.Y. *et al.* (2010) *J. Biol. Chem.* **285**:14377.
13. Simard, J.-C. *et al.* (2010) *J. Leukoc. Biol.* **87**:905.
14. Schelbergen, R.F.P. *et al.* (2012) *Arthritis Rheum.* **64**:1477.
15. Zhang, C. *et al.* (2012) *PLoS ONE* **7**:e32953.