

DESCRIPTION

Source *E. coli*-derived mouse Serum Amyloid A1 protein
Gly20-Tyr122
Accession # P05366

N-terminal Sequence Analysis Gly20

Predicted Molecular Mass 11.8 kDa

SPECIFICATIONS

SDS-PAGE 11 kDa, reducing conditions

Activity Measured by its ability to induce TNF- α secretion by J774A.1 mouse reticulum cell sarcoma macrophage cells.
The ED₅₀ for this effect is 1.5-7.5 μ g/mL.

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

Purity >95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

Formulation Lyophilized from a 0.2 μ m filtered solution in Tris-HCl, NaCl, PEG and Imidazole. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 100 μ g/mL in 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

Mouse Serum Amyloid A protein-1 (SAA1; previously SAA2 in mouse) is a multifunctional apolipoprotein produced by hepatocytes in response to pro-inflammatory cytokines (1 - 4). It is secreted as a 12 kDa, 103 amino acid (aa), nonglycosylated protein and circulates as part of the HDL complex (1 - 4). The SAA1 gene is one of five SAA genes in mouse (3). Mature mouse SAA1 shares 72%, 72% and 67% amino acid (aa) sequence identity with human, rabbit and equine SAA1, respectively. SAA1 and SAA2 share 113 of their 122 amino acids and are termed A-SAA (acute phase SAA) (2, 3). SAA1 is produced in the liver in both human and mouse (3, 4). SAA1 is prominently produced in human adipose tissue, but is absent in mouse adipose, which instead expresses adipose SAA3 (a pseudogene in humans) (5). In mouse, however, circulating SAA1 is elevated by insulin resistance (6). A-SAA can increase by as much as 1000-fold during inflammation (3). When highly expressed, A-SAA can displace ApoA1 as the major apolipoprotein in HDL complexes, weakening its role as a reverse (lipid clearing) cholesterol transporter (4). A highly charged region of SAA1 and 2 (aa 36 - 68) contains putative fibronectin and laminin binding motifs (3). This region also binds heparan sulfate proteoglycans at mildly acidic pH and promotes aggregation of A-SAA; however, pathogenic amyloid fibrils contain fragments of mouse SAA2, not SAA1 (3, 7, 8). Mouse strains can differ in SAA sequence, expression, and amyloid formation (1, 8 - 10). SAA1 is a ligand for CD36/SR-B3, SR-B1, FPRL1, TLR2, and RAGE on monocytes/macrophages, inducing chemotaxis and generation of cytokines and tissue factor (12 - 14). SAA1 can bind the surface of invading gram-negative bacteria, acting as an opsonin to aid clearance by macrophages (11). SAA1 also binds platelets, probably by engaging fibrinogen on the platelet surface (15).

References:

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