

Acid sphingomyelinase/SMPD1 Protein, Mouse, Recombinant (His)

General Information

Synonyms:	aSMase;ASM;Zn-SMase;A-SMase;sphingomyelin phosphodiesterase 1
Protein Construction:	A DNA sequence encoding the mouse SMPD1 (Q04519) (Met 1-Leu 626) was expressed, with a C-terminal polyhistidine tag. Predicted N terminal: Leu 45
Species:	Mouse
Expression Host:	Baculovirus Insect Cells
Accession:	Q04519
Molecular Weight:	66.3 kDa (predicted); 63 kDa (reducing conditions)

QC Testing

Biological Activity:	Measured by its ability to cleave 2-N-Hexadecanoylamino-4-nitrophenylphosphorylcholine (HNPPC). The specific activity is > 1,000 pmoles/min/μg.
Purity:	> 85 % as determined by SDS-PAGE
Endotoxin:	< 1.0 EU/μg of the protein as determined by the LAL method.
Formulation:	Supplied as sterile 20 mM Tris, 500 mM NaCl, 10% glycerol, pH 8.0, 0.1% Tween20.

Preparation and Storage

Reconstitution:

A Certificate of Analysis (CoA) containing reconstitution instructions is included with the products. Please refer to the CoA for detailed information.

Stability & Storage:

It is recommended to store the product under sterile conditions at -20°C to -80°C. Samples are stable for up to 12 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

Proteins are shipped with blue ice.

Protein Background

Sphingomyelin phosphodiesterase 1 (SMPD1), also known as ASM (acid sphingomyelinase), is a member of the acid sphingomyelinase family of enzymes. Three isoforms have been identified, isoform 1 is 631 amino acids (aa) in length as the pro form, while Isoform 2 and isoform 3 have lost catalytic activity. The active SMPD1 isoform 1 contains one saposin B-type domain that likely interacts with sphingomyelin, and a catalytic region. Human SMPD1 is 86% aa identical to mouse SMPD1. SMPD1 is a monomeric lysosomal enzyme that converts sphingomyelin (a plasma membrane lipid) into ceramide through the removal of phosphorylcholine. This generates second messenger components that participate in signal transduction. Defects in SMPD1 are the cause of Niemann-Pick disease type A (NPA) and type B (NPB), also known as Niemann-Pick disease classical infantile

form and Niemann-Pick disease visceral form. Niemann-Pick disease is a clinically and genetically heterogeneous recessive disorder. NPB has little if any neurologic involvement and patients may survive into adulthood.

Reference

Schuchman E.H., et al., (1991), Human acid sphingomyelinase. Isolation, nucleotide sequence and expression of the full-length and alternatively spliced cDNAs. *J. Biol. Chem.* 266:8531-8539.

Newrzella D., et al., (1992), Molecular cloning of the acid sphingomyelinase of the mouse and the organization and complete nucleotide sequence of the gene. *Biol. Chem. Hoppe-Seyler* 373:1233-1238.

Schuchman E.H., et al., (1992), Structural organization and complete nucleotide sequence of the gene encoding human acid sphingomyelinase (SMPD1). *Genomics* 12:197-205.

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