

ASM siRNA (m): sc-41651

BACKGROUND

Acid sphingomyelinase (ASM) is a lysosomal protein that hydrolyzes sphingomyelin to ceramide and phosphocholine. The ASM gene encodes three proteins, ASM-1, ASM-2 and ASM-3, of which ASM-1 is the only ASM gene product that is a catalytically active enzyme. Deficiency of ASM is associated with type A and type B Niemann-Pick disease. Type A is a fatal neurodegenerative disorder seen in infancy and resulting in death by age three, whereas type B is a non-neuropathic disease that has a later onset. During monocytic cell differentiation, the expression of ASM is upregulated by the combined actions of AP-2 and Sp1 transcription factors.

REFERENCES

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2. Schuchman, E.H., Suchi, M., Takahashi, T., Sandhoff, K. and Desnick, R.J. 1991. Human acid sphingomyelinase. Isolation, nucleotide sequence and expression of the full-length and alternatively spliced cDNAs. *J. Biol. Chem.* 266: 8531-8539.
3. Levran, O., Desnick, R.J. and Schuchman, E.H. 1991. Niemann-Pick disease: a frequent missense mutation in the acid sphingomyelinase gene of Ashkenazi Jewish type A and B patients. *Proc. Natl. Acad. Sci. USA* 88: 3748-3752.
4. Takahashi, T., Suchi, M., Desnick, R.J., Takada, G. and Schuchman, E.H. 1992. Identification and expression of five mutations in the human acid sphingomyelinase gene causing types A and B Niemann-Pick disease. Molecular evidence for genetic heterogeneity in the neuronopathic and non-neuronopathic forms. *J. Biol. Chem.* 267: 12552-12558.
5. Langmann, T., Buechler, C., Ries, S., Schaeffler, A., Aslanidis, C., Schuierer, M., Weiler, M., Sandhoff, K., de Jong, P.J. and Schmitz, G. 1999. Transcription factors Sp1 and AP2 mediate induction of acid sphingomyelinase during monocytic differentiation. *J. Lipid Res.* 40: 870-880.

CHROMOSOMAL LOCATION

Genetic locus: Smpd1 (mouse) mapping to 7 E3.

PRODUCT

ASM siRNA (m) is a pool of 3 target-specific 19-25 nt siRNAs designed to knock down gene expression. Each vial contains 3.3 nmol of lyophilized siRNA, sufficient for a 10 μ M solution once resuspended using protocol below. Suitable for 50-100 transfections. Also see ASM shRNA Plasmid (m): sc-41651-SH and ASM shRNA (m) Lentiviral Particles: sc-41651-V as alternate gene silencing products.

For independent verification of ASM (m) gene silencing results, we also provide the individual siRNA duplex components. Each is available as 3.3 nmol of lyophilized siRNA. These include: sc-41651A, sc-41651B and sc-41651C.

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.

STORAGE AND RESUSPENSION

Store lyophilized siRNA duplex at -20° C with desiccant. Stable for at least one year from the date of shipment. Once resuspended, store at -20° C, avoid contact with RNases and repeated freeze thaw cycles.

Resuspend lyophilized siRNA duplex in 330 μ l of the RNase-free water provided. Resuspension of the siRNA duplex in 330 μ l of RNase-free water makes a 10 μ M solution in a 10 μ M Tris-HCl, pH 8.0, 20 mM NaCl, 1 mM EDTA buffered solution.

APPLICATIONS

ASM siRNA (m) is recommended for the inhibition of ASM expression in mouse cells.

SUPPORT REAGENTS

For optimal siRNA transfection efficiency, Santa Cruz Biotechnology's siRNA Transfection Reagent: sc-29528 (0.3 ml), siRNA Transfection Medium: sc-36868 (20 ml) and siRNA Dilution Buffer: sc-29527 (1.5 ml) are recommended. Control siRNAs or Fluorescein Conjugated Control siRNAs are available as 10 μ M in 66 μ l. Each contain a scrambled sequence that will not lead to the specific degradation of any known cellular mRNA. Fluorescein Conjugated Control siRNAs include: sc-36869, sc-44239, sc-44240 and sc-44241. Control siRNAs include: sc-37007, sc-44230, sc-44231, sc-44232, sc-44233, sc-44234, sc-44235, sc-44236, sc-44237 and sc-44238.

RT-PCR REAGENTS

Semi-quantitative RT-PCR may be performed to monitor ASM gene expression knockdown using RT-PCR Primer: ASM (m)-PR: sc-41651-PR (20 μ l, 504 bp). Annealing temperature for the primers should be 55-60° C and the extension temperature should be 68-72° C.

SELECT PRODUCT CITATIONS

1. Xuan, N.T., Shumilina, E., Kempe, D.S., Gulbins, E. and Lang, F. 2010. Sphingomyelinase dependent apoptosis of dendritic cells following treatment with amyloid peptides. *J. Neuroimmunol.* 219: 81-89.
2. Jin, J., Zhang, X., Lu, Z., Perry, D.M., Li, Y., Russo, S.B., Cowart, L.A., Hannun, Y.A. and Huang, Y. 2013. Acid sphingomyelinase plays a key role in palmitic acid-amplified inflammatory signaling triggered by lipopolysaccharide at low concentrations in macrophages. *Am. J. Physiol. Endocrinol. Metab.* 305: E853-E867.
3. Di Pietro, P., Carrizzo, A., Sommella, E., Oliveti, M., Iacoviello, L., Di Castelnuovo, A., Acernese, F., Damato, A., De Lucia, M., Merciai, F., Iesu, P., Venturini, E., Izzo, R., Trimarco, V., Ciccarelli, M., et al. 2022. Targeting the ASMase/S1P pathway protects from sortilin-evoked vascular damage in hypertension. *J. Clin. Invest.* 132: e146343.

RESEARCH USE

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