

SHH Antibody (monoclonal) (M03)

Mouse monoclonal antibody raised against a partial recombinant SHH. Catalog # AT3883a

Specification

SHH Antibody (monoclonal) (M03) - Product Information

Application E
Primary Accession O15465
Other Accession NM_000193
Reactivity Human
Host mouse
Clonality Monoclonal
Isotype IgG2a Kappa

Calculated MW 49607

SHH Antibody (monoclonal) (M03) - Additional Information

Gene ID 6469

Other Names

Sonic hedgehog protein, SHH, HHG-1, Sonic hedgehog protein N-product, Sonic hedgehog protein C-product, SHH

Target/Specificity

SHH (NP_000184, 181 a.a. \sim 280 a.a) partial recombinant protein with GST tag. MW of the GST tag alone is 26 KDa.

Dilution

E~~N/A

Format

Clear, colorless solution in phosphate buffered saline, pH 7.2.

Storage

Store at -20°C or lower. Aliquot to avoid repeated freezing and thawing.

Precautions

SHH Antibody (monoclonal) (M03) is for research use only and not for use in diagnostic or therapeutic procedures.

SHH Antibody (monoclonal) (M03) - Protocols

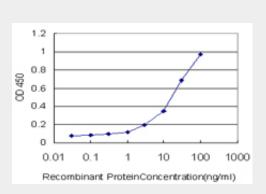
Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot



- Immunohistochemistry
- Immunofluorescence
- <u>Immunoprecipitation</u>
- Flow Cytomety
- Cell Culture

SHH Antibody (monoclonal) (M03) - Images



Detection limit for recombinant GST tagged SHH is approximately 0.3ng/ml as a capture antibody.

SHH Antibody (monoclonal) (M03) - Background

This gene encodes a protein that is instrumental in patterning the early embryo. It has been implicated as the key inductive signal in patterning of the ventral neural tube, the anterior-posterior limb axis, and the ventral somites. Of three human proteins showing sequence and functional similarity to the sonic hedgehog protein of Drosophila, this protein is the most similar. The protein is made as a precursor that is autocatalytically cleaved; the N-terminal portion is soluble and contains the signalling activity while the C-terminal portion is involved in precursor processing. More importantly, the C-terminal product covalently attaches a cholesterol moiety to the N-terminal product, restricting the N-terminal product to the cell surface and preventing it from freely diffusing throughout the developing embryo. Defects in this protein or in its signalling pathway are a cause of holoprosencephaly (HPE), a disorder in which the developing forebrain fails to correctly separate into right and left hemispheres. HPE is manifested by facial deformities. It is also thought that mutations in this gene or in its signalling pathway may be responsible for VACTERL syndrome, which is characterized by vertebral defects, anal atresia, tracheoesophageal fistula with esophageal atresia, radial and renal dysplasia, cardiac anomalies, and limb abnormalities. Additionally, mutations in a long range enhancer located approximately 1 megabase upstream of this gene disrupt limb patterning and can result in preaxial polydactyly.

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Maternal genes and facial clefts in offspring: a comprehensive search for genetic associations in two population-based cleft studies from Scandinavia. Jugessur A, et al. PLoS One, 2010 Jul 9. PMID 20634891. Activation of Rac1 promotes hedgehog-mediated acquisition of the myofibroblastic phenotype in rat and human hepatic stellate cells. Choi SS, et al. Hepatology, 2010 Jul. PMID 20578145. Genome-wide meta-analyses identifies seven loci associated with platelet aggregation in response to agonists. Johnson AD, et al. Nat Genet, 2010 Jul. PMID 20526338. DYRK1B-dependent autocrine-to-paracrine shift of Hedgehog signaling by mutant RAS. Lauth M, et al. Nat Struct Mol Biol, 2010 Jun. PMID 20512148. Notch signaling is not essential in sonic hedgehog-activated medulloblastoma. Hatton BA, et al. Oncogene, 2010 Jul 1. PMID 20440271.