

Shh Rabbit Monoclonal Antibody

Catalog #: EAB21636

Host/Isotype	Clonality	Applications	MW (kDa)	Reactivity
Rabbit IgG	Monoclonal	WB, IHC-P, IF/ICC, FC	50	Human

Applications Dilutions

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

WB(Western Blotting)	1:500-2000
IHC-P(Immunohistochemistry-Paraffin)	1:100-500
IF/ICC(Immunofluorescence/Immunocytochemistry)	1:50-200
FC(Flow Cytometry)	1:10-100

Product Information

Conjugate	Unconjugate
Specificity	Shh Rabbit Monoclonal Antibody detects endogenous levels of Shh protein.
Purification	Affinity purification
Concentration	1mg/ml
Format	Liquid
Formulation	In PBS, pH 7.4, Containing 0.02% sodium azide, 0.5% BSA and 50% Glycerol
Shipping	Gel Pack
Storage	Store at -20°C least 1 year from the date of shipment. Avoid repeated freeze/thaw cycles. Aliquots may be stored at +4°C for 1-2 weeks
UniProt ID	Q15465
Entrez-Gene ID	6469

Product Description

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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