

SHH Polyclonal Antibody

Catalog No. E-AB-62213

Note: Centrifuge before opening to ensure complete recovery of vial contents.

Description

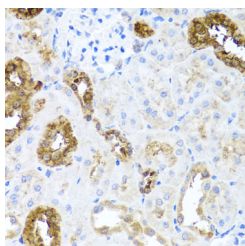
Reactivity	Human,Mouse,Rat
Immunogen	A synthetic peptide of human SHH
Host	Rabbit
Isotype	IgG
Purification	Affinity purification
Conjugation	Unconjugated
Buffer	PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

Applications

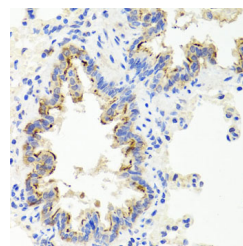
Recommended Dilution

**IHC 1:50-1:100 IF
1:50-1:200**

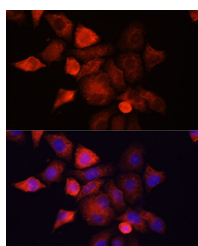
Data



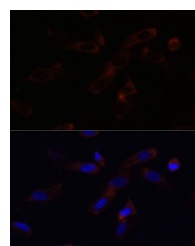
Immunohistochemistry of paraffin-embedded Rat kidney using SHH Polyclonal Antibody at dilution of 1:100 (40x lens).



Immunohistochemistry of paraffin-embedded Rat lung using SHH Polyclonal Antibody at dilution of 1:100 (40x lens).



Immunofluorescence analysis of HeLa cells using SHH Polyclonal Antibody at dilution of 1:100 (40x lens). Blue: DAPI for nuclear staining.



Immunofluorescence analysis of NIH-3T3 cells using SHH Polyclonal Antibody at dilution of 1:100 (40x lens). Blue: DAPI for nuclear staining.

Preparation & Storage

Storage Store at -20°C. Avoid freeze / thaw cycles.

Background

For Research Use Only

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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