SunLong Biotech Co.,LTD
Tel: 0086-571-56623320 Fax:0086-571-56623318
E-mail:sales@sunlongbiotech.com
www.sunlongbiotech.com

[Primary Antibody]

Rabbit Anti-Patched antibody

Catalog Number: SL1614R

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	Patched
Immunogen:	KLH conjugated synthetic peptide derived from human Patched/PTCH: 581-680/1447
Purification:	affinity purified by Protein A
Concentration:	1mg/ml
Storage:	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol. Shipped at 4℃. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles.
Applications:	WB=1:500-2000 IHC-P=1:100-500 IHC-F=1:100-500 ICC/IF=1:100-500 IF=1:100-500 ELISA=1:5000-10000 （Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	Human, Mouse, (predicted: Rat, Chicken, Cow, Horse, Rabbit)
Predicted MW.:	161kDa
Subcellular Location:	The cell membrane
Background:	PTCH (Patched protein homolog 1) is a receptor for sonic hedgehog (SHH), indian hedgehog (IHH) and desert hedgehog (DHH). PTCH associates with the smoothened protein (SMO) to transduce the hedgehog's proteins signal.PTCH has a tumor suppressor function, as inactivation of this protein is probably a necessary, if not sufficient step for tumorigenesis. PTCH is expressed in the adult brain, lung, liver, heart, placenta, skeletal muscle, pancreas and kidney. It is also expressed in tumor cells but not in normal skin. During development PTCH is found in all major target tissues of sonic hedgehog, such as the ventral neural tube, somites, and tissues surrounding the zone of polarizing

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	activity of the limb bud. Defects in PTCH are probably the cause of basal cell nevus syndrome also known as Gorlin syndrome or Gorlin-Goltz syndrome. **Function:** Acts as a receptor for sonic hedgehog (SHH), indian hedgehog (IHH) and desert hedgehog (DHH). Associates with the smoothened protein (SMO) to transduce the hedgehog's proteins signal. Seems to have a tumor suppressor function, as inactivation of this protein is probably a necessary, if not sufficient step for tumorigenesis. **Subunit:** Interacts with SNX17. Interacts with IHH. **Subcellular Location:** Membrane. **Tissue Specificity:** In the adult, expressed in brain, lung, liver, heart, placenta, skeletal muscle, pancreas and kidney. Expressed in tumor cells but not in normal skin. **Post-translational modifications:** Glycosylation is necessary for SHH binding. DISEASE: Defects in PTCH1 are probably the cause of basal cell nevus syndrome (BCNS) [MIM:109400]; also known as Gorlin syndrome or Gorlin-Goltz syndrome. BCNS is an autosomal dominant disease characterized by nevoid basal cell carcinomas (NBCCS) and developmental abnormalities such as rib and craniofacial alterations, polydactyly, syndactyly, and spina bifida. In addition, the patients suffer from a multitude of tumors like basal cell carcinomas (BCC), fibromas of the ovaries and heart, cysts of the skin, jaws and mesentery, as well as medulloblastomas and meningiomas. PTCH1 is also mutated in squamous cell carcinoma (SCC). Could also be associated with large body size observed in BCNS patients. Defects in PTCH1 are a cause of sporadic basal cell carcinoma (BCC) [MIM:605462]. Defects in PTCH1 are the cause of holoprosencephaly type 7 (HPE7) [MIM:610828]. Holoprosencephaly (HPE) [MIM:236100] is the most common structural anomaly of the brain, in which the developing forebrain fails to correctly separate into right and left hemispheres. Holoprosencephaly is genetically heterogeneous and associated with several distinct facies and phenotypic variability. **Similarity:** Belongs to the patched family. Contains 1 SSD (sterol-sensing) domain. **Database links:** **Entrez Gene:** 5727 Human **Entrez Gene:** 19206 Mouse **Entrez Gene:** 89830 Rat Omim: 601309 Human

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	SwissProt: Q13635 Human **SwissProt:** Q86XG7 Human **SwissProt:** Q61115 Mouse **Unigene:** 494538 Human **Unigene:** 228798 Mouse **Unigene:** 102312 Rat Ptch protein is a cell surface receptor that receives Hh signaling proteins and is currently mainly used in tumor research.

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