10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

Anti-Notch 3 Homolog Antibody
Catalog # AN2188

Specification

Anti-Notch 3 Homolog Antibody - Product Information

Application	**WB**
Primary Accession	Q9UM47
Host	**Rabbit**
Clonality	**Rabbit Polyclonal**
Isotype	**IgG**
Calculated MW	**243631**

Anti-Notch 3 Homolog Antibody - Additional Information

Gene ID	**4854**

Other Names
Neurogenic locus notch homolog protein 3, hN3,

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions
Anti-Notch 3 Homolog Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Shipping
Blue Ice

Anti-Notch 3 Homolog Antibody - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

Anti-Notch 3 Homolog Antibody - Images

Anti-Notch 3 Homolog Antibody - Background

Notch signaling plays a key role in the normal development of many tissues and cell types, through diverse effects on differentiation, survival, and/or proliferation that are highly dependent on signal strength and cellular context. Members of the Notch gene family encode transmembrane receptors

that are critical for various cell fate decisions. Notch family members share structural characteristics including an extracellular domain consisting of multiple epidermal growth factor-like (EGF) repeats, and an intracellular domain consisting of multiple, different domain types. Multiple human notch proteins (NOTCH1, NOTCH2, NOTCH3 and NOTCH4) have been identified and they function as a receptors for membrane bound ligands. Notch signaling is also linked to tumorigenesis as first demonstrated by the identification of a recurrent t(7;9)(q34;q34.3) chromosomal translocation involving the human Notch1 gene that is found in a small subset of human pre-T-cell acute lymphoblastic leukemias (T-ALL). Since this discovery, aberrant Notch signaling has been suggested to be involved in a wide variety of human neoplasms. Mutations in NOTCH3 have been identified as the underlying cause of cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL).