

<b>Cat. No:</b>	ABN09020
<b>Conjugate:</b>	Unconjugated
<b>Size:</b>	100µL
<b>Clone:</b>	Polyclonal
<b>Concentration:</b>	1mg/ml
<b>Host:</b>	Rabbit
<b>Isotype:</b>	IgG
<b>Immunogen:</b>	The antiserum was produced against synthesized peptide derived from human Notch 2. AA range:1684-1733
<b>Reactivity:</b>	Human,Mouse,Rat
<b>Applications:</b>	WB 1:500-1:2000,IHC 1:100-1:300,ICC/IF 1:50-1:200,ELISA 1:20000-1:40000
<b>Molecular Weight:</b>	110(cleaved)kDa
<b>Purification:</b>	Affinity purification
<b>Synonyms:</b>	NOTCH2; Neurogenic locus notch homolog protein 2; Notch 2; hN2

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**Background:**

notch 2(NOTCH2) Homo sapiens This gene encodes a member of the Notch family. Members of this Type 1 transmembrane protein family 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. Notch family members play a role in a variety of developmental processes by controlling cell fate decisions. The Notch signaling network is an evolutionarily conserved intercellular signaling pathway which regulates interactions between physically adjacent cells. In Drosophila, notch interaction with its cell-bound ligands (delta, serrate) establishes an intercellular signaling pathway that plays a key role in development. Homologues of the notch-ligands have also been identified in human, but precise interactions between these ligands and the human notch homologues remain to be determined. This protein is cleaved: Defects in NOTCH2 are the cause of Alagille syndrome type 2 (ALGS2) [MIM:610205]. Alagille syndrome is an autosomal dominant multisystem disorder defined clinically by hepatic bile duct paucity and cholestasis in association with cardiac, skeletal, and ophthalmologic manifestations. There are characteristic facial features and less frequent clinical involvement of the renal and vascular systems. function: Functions as a receptor for membrane-bound ligands Jagged1, Jagged2 and Delta1 to regulate cell-fate determination. Upon ligand activation through the released notch intracellular domain (NICD) it forms a transcriptional activator complex with RBP-J kappa and activates genes of the enhancer of split locus. Affects the implementation of differentiation, proliferation and apoptotic programs. PTM: Phosphorylated. PTM: Synthesized in the endoplasmic reticulum as an inactive form which is proteolytically cleaved by a furin-like convertase in the trans-Golgi network before it reaches the plasma membrane to yield an active, ligand-accessible form. Cleavage results in a C-terminal fragment N(TM) and a N-terminal fragment N(EC). Following ligand binding, it is cleaved by TNF-alpha converting enzyme (TACE) to yield a membrane-associated intermediate fragment called notch extracellular truncation (NEXT). This fragment is then cleaved by presenilin dependent gamma-secretase to release a notch-derived peptide containing the intracellular domain (NICD) from the membrane. similarity: Belongs to the NOTCH family. similarity: Contains 3 LNR (Lin/Notch) repeats. similarity: Contains 35 EGF-like domains. similarity: Contains 6 ANK repeats. subcellular location: Following proteolytical processing NICD is

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