



NOTCH SIGNALING PATHWAY

Though the Notch pathway is most famous for its role in development, it plays key roles in differentiation and tissue homeostasis in adults. Nowhere is this more manifest than leukemia where both constitutive activation and inactivation of this pathway lead to disease. Activating Notch receptor mutations are frequently observed in T-cell acute lymphoblastic leukemia and inactivating mutations are observed in chronic myelomonocytic leukemia. Conversely, mutations that lead to reduced activity of this protein are also associated with cancer, causing another hematopoietic cancer – chronic myelomonocytic leukemia.

Notch signaling is initiated when one cell expressing the appropriate ligand – the signal sending cell – interacts with another cell via its Notch receptor. This ligand-receptor binding triggers a series of proteolytic cleavages. One such step requires the γ-secretase enzyme complex, comprised of nicastrin, presenilin, APH1, PEN-2 and PSENEN; cleavage by the γ-secretase complex generates apportion of the Notch receptor – called the Notch intracellular domain (NICD) – freeing it from the cell membrane and enabling its relocation to the nucleus. Here it modulates the expression of many genes involved in growth and development through interaction with DNA-bound protein factors.

Legend	
—	Association
→	Directly Activates
→	Indirectly Activates
⊣	Inhibits
Ub	Ubiquitin
P	Phosphorylates

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