

**FZD9 / Frizzled 9 Antibody (N-Terminus)**  
**Rabbit Polyclonal Antibody**  
**Catalog # ALS10805****Specification**

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**FZD9 / Frizzled 9 Antibody (N-Terminus) - Product Information**

Application	IHC
Primary Accession	<a href="#">O00144</a>
Reactivity	Human, Mouse, Rabbit, Bovine, Dog
Host	Rabbit
Clonality	Polyclonal
Calculated MW	64kDa KDa

**FZD9 / Frizzled 9 Antibody (N-Terminus) - Additional Information****Gene ID** 8326**Other Names**

Frizzled-9, Fz-9, hFz9, FzE6, CD349, FZD9, FZD3

**Target/Specificity**

Human FZD9 / Frizzled 9. BLAST analysis of the peptide immunogen showed no homology with other human proteins, except FZD10 (65%), CARD10 (65%).

**Reconstitution & Storage**

Long term: -70°C; Short term: +4°C

**Precautions**

FZD9 / Frizzled 9 Antibody (N-Terminus) is for research use only and not for use in diagnostic or therapeutic procedures.

**FZD9 / Frizzled 9 Antibody (N-Terminus) - Protein Information****Name** FZD9**Synonyms** FZD3**Function**

Receptor for WNT2 that is coupled to the beta-catenin canonical signaling pathway, which leads to the activation of disheveled proteins, inhibition of GSK-3 kinase, nuclear accumulation of beta-catenin and activation of Wnt target genes (By similarity). Plays a role in neuromuscular junction (NMJ) assembly by negatively regulating the clustering of acetylcholine receptors (AChR) through the beta-catenin canonical signaling pathway (By similarity). May play a role in neural progenitor cells (NPCs) viability through the beta-catenin canonical signaling pathway by negatively regulating cell cycle arrest leading to inhibition of neuron apoptotic process (PubMed: <a href="http://www.uniprot.org/citations/27509850" target="\_blank">27509850</a>). During hippocampal development, regulates neuroblast proliferation and apoptotic cell death. Controls bone formation through non canonical Wnt signaling mediated via ISG15. Positively

regulates bone regeneration through non canonical Wnt signaling (By similarity).

#### **Cellular Location**

Cell membrane {ECO:0000250|UniProtKB:Q9R216}; Multi-pass membrane protein.  
Note=Relocalizes DVL1 to the cell membrane leading to phosphorylation of DVL1 and AXIN1 relocalization to the cell membrane. {ECO:0000250|UniProtKB:Q8K4C8}

#### **Tissue Location**

Expressed predominantly in adult and fetal brain, testis, eye, skeletal muscle and kidney. Moderately expressed in pancreas, thyroid, adrenal cortex, small intestine and stomach Detected in fetal liver and kidney. Expressed in neural progenitor cells (PubMed:27509850).

#### **Volume**

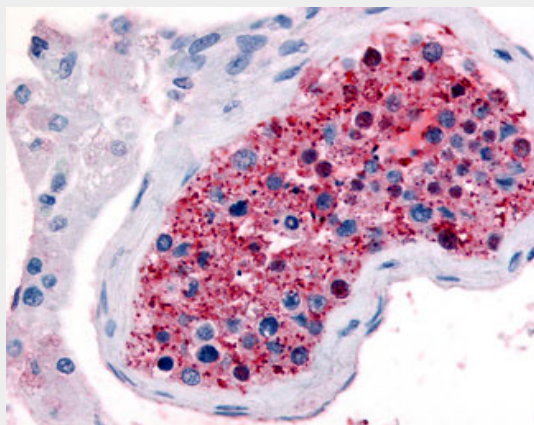
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### **FZD9 / Frizzled 9 Antibody (N-Terminus) - Protocols**

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

### **FZD9 / Frizzled 9 Antibody (N-Terminus) - Images**



Anti-FZD9 / Frizzled 9 antibody ALS10805 IHC of human testis.

### **FZD9 / Frizzled 9 Antibody (N-Terminus) - Background**

Receptor for Wnt proteins. Most of frizzled receptors are coupled to the beta-catenin canonical signaling pathway, which leads to the activation of disheveled proteins, inhibition of GSK-3 kinase, nuclear accumulation of beta-catenin and activation of Wnt target genes. A second signaling pathway involving PKC and calcium fluxes has been seen for some family members, but it is not yet clear if it represents a distinct pathway or if it can be integrated in the canonical pathway, as PKC seems to be required for Wnt-mediated inactivation of GSK-3 kinase. Both pathways seem to involve interactions with G-proteins. May be involved in transduction and intercellular transmission

of polarity information during tissue morphogenesis and/or in differentiated tissues.

**FZD9 / Frizzled 9 Antibody (N-Terminus) - References**

Wang Y.-K.,et al.Hum. Mol. Genet. 6:465-472(1997).

Hillier L.W.,et al.Nature 424:157-164(2003).

Tanaka S.,et al.Proc. Natl. Acad. Sci. U.S.A. 95:10164-10169(1998).