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FZD9 / Frizzled 9 Antibody (aa542-591)

Rabbit Polyclonal Antibody
Catalog # ALS17180

Specification

FZD9 / Frizzled 9 Antibody (aa542-591) - Product Information

Application	**IHC-P, IF, WB**
Primary Accession	O00144
Other Accession	8326
Reactivity	**Human, Mouse**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**IgG**
Calculated MW	**64466**

FZD9 / Frizzled 9 Antibody (aa542-591) - Additional Information

Gene ID 8326

Other Names
FZD9, CD349, Frizzled homolog 9, Fz-9, Frizzled family receptor 9, Frizzled-9, Frizzled 9, Fz9, HFz9, CD349 antigen, Frizzled homolog fzd3, FzE6

Target/Specificity
FZD9 Antibody detects endogenous levels of total FZD9 protein.

Reconstitution & Storage
PBS (without Mg2+, Ca2+), pH 7.4, 150 mM sodium chloride, 0.02% sodium azide, 50% glycerol. Store at -20°C for up to one year.

Precautions
FZD9 / Frizzled 9 Antibody (aa542-591) is for research use only and not for use in diagnostic or therapeutic procedures.

FZD9 / Frizzled 9 Antibody (aa542-591) - 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:27509850). 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).

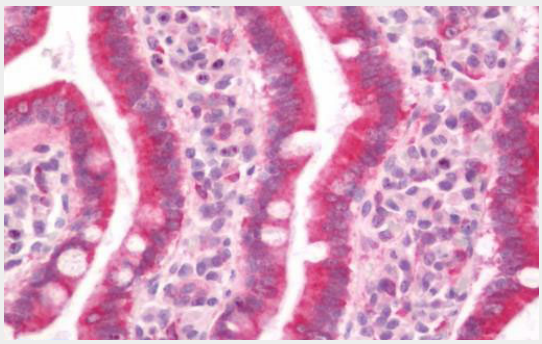
Volume
50 µl

FZD9 / Frizzled 9 Antibody (aa542-591) - Protocols

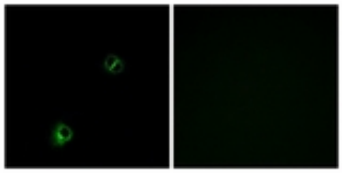
Provided below are standard protocols that you may find useful for product applications.

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

FZD9 / Frizzled 9 Antibody (aa542-591) - Images



Human Small Intestine: Formalin-Fixed, Paraffin-Embedded (FFPE)



Immunofluorescence of A549 cells, using FZD9 Antibody.

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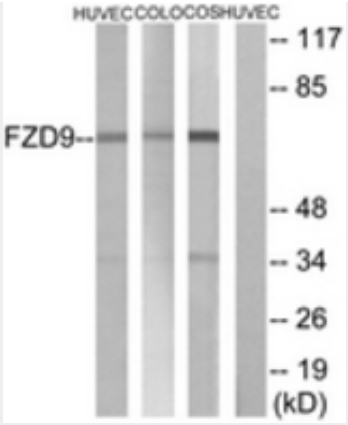

Western blot of extracts from HUVEC/COLO/COS cells, using FZD9 Antibody.

FZD9 / Frizzled 9 Antibody (aa542-591) - 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 (aa542-591) - 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).