

LGR5 antibody (N-term)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP22391a**Specification**

LGR5 antibody (N-term) - Product Information

Application	WB,E
Primary Accession	O75473
Other Accession	F1MT22
Reactivity	Rat
Predicted	Bovine
Host	Rabbit
Clonality	polyclonal
Isotype	Rabbit IgG
Calculated MW	99998

LGR5 antibody (N-term) - Additional Information**Gene ID** 8549**Other Names**

Leucine-rich repeat-containing G-protein coupled receptor 5, G-protein coupled receptor 49, G-protein coupled receptor 67, G-protein coupled receptor HG38, LGR5, GPR49, GPR67

Target/Specificity

This antibody is generated from a rabbit immunized with a KLH conjugated synthetic peptide between amino acids from human.

Dilution

WB~~1:500

E~~Use at an assay dependent concentration.

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

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

Precautions

LGR5 antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

LGR5 antibody (N-term) - Protein Information**Name** LGR5

Synonyms GPR49, GPR67

Function Receptor for R-spondins that potentiates the canonical Wnt signaling pathway and acts as a stem cell marker of the intestinal epithelium and the hair follicle. Upon binding to R-spondins (RSPO1, RSPO2, RSPO3 or RSPO4), associates with phosphorylated LRP6 and frizzled receptors that are activated by extracellular Wnt receptors, triggering the canonical Wnt signaling pathway to increase expression of target genes. In contrast to classical G-protein coupled receptors, does not activate heterotrimeric G-proteins to transduce the signal. Involved in the development and/or maintenance of the adult intestinal stem cells during postembryonic development.

Cellular Location

Cell membrane; Multi-pass membrane protein. Golgi apparatus, trans-Golgi network membrane; Multi-pass membrane protein Note=Rapidly and constitutively internalized to the trans-Golgi network at steady state. Internalization to the trans-Golgi network may be the result of phosphorylation at Ser-861 and Ser-864; however, the phosphorylation event has not been proven (PubMed:23439653)

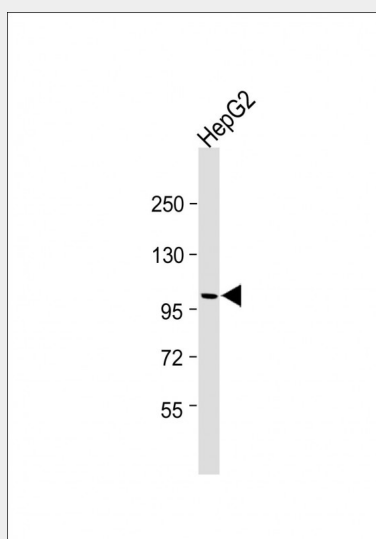
Tissue Location

Expressed in skeletal muscle, placenta, spinal cord, and various region of brain. Expressed at the base of crypts in colonic and small mucosa stem cells. In premalignant cancer expression is not restricted to the cript base. Overexpressed in cancers of the ovary, colon and liver.

LGR5 antibody (N-term) - 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](#)

LGR5 antibody (N-term) - Images

All lanes : Anti-LGR5 antibody (N-term) at 1:500 dilution Lane 1: HepG2 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/15000 dilution. Observed band size : 100kDa Blocking/Dilution buffer: 5% NFDM/TBST.

LGR5 antibody (N-term) - Background

Receptor for R-spondins that potentiates the canonical Wnt signaling pathway and acts as a stem cell marker of the intestinal epithelium and the hair follicle. Upon binding to R-spondins (RSPO1, RSPO2, RSPO3 or RSPO4), associates with phosphorylated LRP6 and frizzled receptors that are activated by extracellular Wnt receptors, triggering the canonical Wnt signaling pathway to increase expression of target genes. In contrast to classical G-protein coupled receptors, does not activate heterotrimeric G-proteins to transduce the signal. Involved in the development and/or maintenance of the adult intestinal stem cells during postembryonic development.

LGR5 antibody (N-term) - References

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Hsu S.Y.,et al.Mol. Endocrinol. 12:1830-1845(1998).
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Scherer S.E.,et al.Nature 440:346-351(2006).
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