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LRP5 + LRP6 Polyclonal Antibody
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP57063

Specification

LRP5 + LRP6 Polyclonal Antibody - Product Information

Application	**IHC-P, IHC-F, IF, ICC**
Primary Accession	O75197
Reactivity	**Rat, Pig, Bovine**
Host	**Rabbit**
Clonality	**Polyclonal**
Calculated MW	**176 KDa**
Physical State	**Liquid**
Immunogen	**KLH conjugated synthetic peptide derived from human LRP5 + LRP6**
Epitope Specificity	**521-620/1615**
Isotype	**IgG**
Purity affinity purified by Protein A	
Buffer	**0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.**
SUBCELLULAR LOCATION	**Membrane; Single-pass type I membrane protein. Endoplasmic reticulum (By similarity). Note=Chaperoned to the plasma membrane by MESD (By similarity).**
SIMILARITY	**Belongs to the LDLR family.Contains 4 EGF-like domains.Contains 3 LDL-receptor class A domains.Contains 20 LDL-receptor class B repeats.**
SUBUNIT	**Homodimer; disulfide-linked. Forms phosphorylated oligomer aggregates on Wnt-signaling (By similarity). Component of a Wnt-signaling complex that contains a WNT protein, a FZD protein and LRP5 or LRP6. Interacts with FZD8; the interaction is formed on WNT-binding and signaling. Interacts (via the phosphorylated PPPSP motif domains) with AXIN1; the interaction prevents inhibition of beta-catenin phosphorylation and signaling and is enhanced in the presence of GSK3B and WNT1 or WNT3A. Interacts (via beta-propeller regions 3 and 4) with DKK1; the interaction, enhanced by MESD and/or KREMEN, inhibits beta-catenin signaling by preventing GSK3-mediated phosphorylation of the PPPSP motifs and subsequent, AXIN1 binding. Interacts with MESD; the interaction prevents the formation of LRP5 aggregates, targets**

	LRP5 to the plasma membrane and, when complexed with KREMEN2, increases DKK1 binding. Interacts with CSNK1E. Interacts with SOST; the interaction antagonizes canonical Wnt signaling. Interacts with APCDD1.
Post-translational modifications	**Phosphorylation of cytoplasmic PPPSP motifs regulates the signal transduction of the Wnt signaling pathway through acting as a docking site for AXIN1 (By similarity).**
DISEASE	**Defects in LRP5 are the cause of vitreoretinopathy exudative type 4 (EVR4) [MIM:601813]. EVR4 is a disorder of the retinal vasculature characterized by an abrupt cessation of growth of peripheral capillaries, leading to an avascular peripheral retina. This may lead to compensatory retinal neovascularization, which is thought to be induced by hypoxia from the initial avascular insult. New vessels are prone to leakage and rupture causing exudates and bleeding, followed by scarring, retinal detachment and blindness. Clinical features can be highly variable, even within the same family. Patients with mild forms of the disease are asymptomatic, and their only disease related abnormality is an arc of avascular retina in the extreme temporal periphery. EVR4 inheritance can be autosomal dominant or recessive.Genetic variations in LRP5 are a cause of susceptibility to osteoporosis (OSTEOP) [MIM:166710]; also known as senile osteoporosis or postmenopausal osteoporosis. Osteoporosis is characterized by reduced bone mass, disruption of bone microarchitecture without alteration in the composition of bone. Osteoporotic bones are more at risk of fracture.Defects in LRP5 are the cause of osteoporosis-pseudoglioma syndrome (OPPG) [MIM:259770]; also known as osteogenesis imperfecta ocular form. OPPG is a recessive disorder characterized by very low bone mass and blindness. Individualy with OPPG are prone to develop bone fractures and deformations and have various eye abnormalities, including phthisis bulbi, retinal detachments, falciform folds or persistent vitreal vasculature.Defects in LRP5 are a cause of high bone mass trait (HBM) [MIM:601884]. HBM is a rare phenotype characterized by exceptionally dense bones. HBM individuals show otherwise a completely normal skeletal structure and**

	no other unusual clinical findings.Defects in LRP5 are a cause of endosteal hyperostosis Worth type (WENHY) [MIM:144750]; also known as autosomal dominant osteosclerosis. WENHY is an autosomal dominant sclerosing bone dysplasia clinically characterized by elongation of the mandible, increased gonial angle, flattened forehead, and the presence of a slowly enlarging osseous prominence of the hard palate (torus palatinus). Serum calcium, phosphorus and alkaline phosphatase levels are normal. Radiologically, it is characterized by early thickening of the endosteum of long bones, the skull and of the mandible. With advancing age, the trabeculae of the metaphysis become thickened. WENHY becomes clinically and radiologically evident by adolescence, does not cause deformity except in the skull and mandible, and is not associated with bone pain or fracture. Affected patients have normal height, proportion, intelligence and longevity.Defects in LRP5 are the cause of osteopetrosis autosomal dominant type 1 (OPTA1) [MIM:607634]. Osteopetrosis is a rare genetic disease characterized by abnormally dense bone, due to defective resorption of immature bone. The disorder occurs in two forms: a severe autosomal recessive form occurring in utero, infancy, or childhood, and a benign autosomal dominant form occurring in adolescence or adulthood. OPTA1 is characterized by generalized osteosclerosis most pronounced in the cranial vault. Patients are often asymptomatic, but some suffer from pain and hearing loss. It appears to be the only type of osteopetrosis not associated with an increased fracture rate.Defects in LRP5 are the cause of van Buchem disease type 2 (VBCH2)[MIM:607636]. VBCH2 is an autosomal dominant sclerosing bone dysplasia characterized by cranial osteosclerosis, thickened calvaria and cortices of long bones, enlarged mandible and normal serum alkaline phosphatase levels.
Important Note	**This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.**

Background Descriptions

LRP5 is involved in the Wnt/beta catenin signaling pathway, probably by acting as a coreceptor together with Frizzled for Wnt. Defects in LRP5 are a cause of autosomal dominant and autosomal recessive familial exudative vitreoretinopathy (FEVR). Autosomal dominant FEVR is also referred to

as exudative vitreoretinopathy 1 (EVR1); also known as Criswick-Schepens syndrome. FEVR is a disorder of the retinal vasculature characterized by an abrupt cessation of growth of peripheral capillaries, leading to an avascular peripheral retina. This may lead to compensatory retinal neovascularization, which is thought to be induced by hypoxia from the initial avascular insult. New vessels are prone to leakage and rupture causing exudates and bleeding, followed by scarring, retinal detachment and blindness. FEVR is reported to have a penetrance of 100%, but clinical features can be highly variable, even within the same family. Patients with mild forms of the disease are asymptomatic, and their only disease-related abnormality is an arc of avascular retina in the extreme temporal periphery.

LRP5 + LRP6 Polyclonal Antibody - Additional Information

Gene ID 4041

Other Names
Low-density lipoprotein receptor-related protein 5, LRP-5, Low-density lipoprotein receptor-related protein 7, LRP-7, LRP5 {ECO:0000303|PubMed:24706814, ECO:0000312|HGNC:HGNC:6697}

Target/Specificity
Widely expressed, with the highest level of expression in the liver and in aorta.

Dilution
IHC-P~~N/A<br \>IHC-F~~N/A<br \>IF~~1:50~200<br \>ICC~~N/A

Storage
Store at -20 ℃ for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 ℃.

LRP5 + LRP6 Polyclonal Antibody - Protein Information

Name LRP5 {ECO:0000303|PubMed:24706814, ECO:0000312|HGNC:HGNC:6697}

Function
Acts as a coreceptor with members of the frizzled family of seven-transmembrane spanning receptors to transduce signal by Wnt proteins (PubMed:11336703, PubMed:11448771, PubMed:11719191, PubMed:15778503, PubMed:15908424, PubMed:16252235). Activates the canonical Wnt signaling pathway that controls cell fate determination and self-renewal during embryonic development and adult tissue regeneration (PubMed:11336703, PubMed:11719191). In particular, may play an important role in the development of the posterior patterning of the epiblast during gastrulation (By similarity). During bone development, regulates osteoblast proliferation and differentiation thus determining bone mass (PubMed:11719191). Mechanistically, the formation of the signaling complex between Wnt ligand, frizzled receptor and LRP5 coreceptor promotes the recruitment of AXIN1 to LRP5, stabilizing beta-catenin/CTNNB1 and

activating TCF/LEF-mediated transcriptional programs (PubMed:11336703, PubMed:14731402, PubMed:24706814, PubMed:25920554). Acts as a coreceptor for non-Wnt proteins, such as norrin/NDP. Binding of norrin/NDP to frizzled 4/FZD4-LRP5 receptor complex triggers beta-catenin/CTNNB1-dependent signaling known to be required for retinal vascular development (PubMed:16252235, PubMed:27228167). Plays a role in controlling postnatal vascular regression in retina via macrophage-induced endothelial cell apoptosis (By similarity).

Cellular Location
Membrane {ECO:0000250|UniProtKB:Q91VN0}; Single- pass type I membrane protein {ECO:0000250|UniProtKB:Q91VN0} Endoplasmic reticulum. Note=Chaperoned to the plasma membrane by MESD. {ECO:0000250|UniProtKB:Q91VN0}

Tissue Location
Widely expressed, with the highest level of expression in the liver and in aorta.

LRP5 + LRP6 Polyclonal Antibody - 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

LRP5 + LRP6 Polyclonal Antibody - Images