# abcam

## Product datasheet

## Anti-LRP5 antibody ab36121

★★★★★ 1 Abreviews 11 References 2 Images

### Overview

Product name Anti-LRP5 antibody

**Description** Goat polyclonal to LRP5

Host species Goat

Tested applications Suitable for: WB, IHC-P

Species reactivity Reacts with: Human

Predicted to work with: Mouse, Rat

**Immunogen** Synthetic peptide within Human LRP5 aa 1150-1250 (internal sequence). The exact immunogen

sequence used to generate this antibody is proprietary information. If additional detail on the immunogen is needed to determine the suitability of the antibody for your needs, please **contact** 

our Scientific Support team to discuss your requirements.

Run BLAST with
Run BLAST with

Positive control IHC-P: Human Liver; WB: HepG2 cell lysate.

**General notes**The Life Science industry has been in the grips of a reproducibility crisis for a number of years.

Abcam is leading the way in addressing this with our range of recombinant monoclonal antibodies and knockout edited cell lines for gold-standard validation. Please check that this product meets

your needs before purchasing.

If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be

found below, along with publications, customer reviews and Q&As

**Properties** 

Form Liquid

**Storage instructions** Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.

**Storage buffer** pH: 7.30

Preservative: 0.02% Sodium azide

Constituents: 0.5% BSA, Tris buffered saline

Purity Immunogen affinity purified

Purification notes Purified from goat serum by ammonium sulphate precipitation followed by antigen affinity

chromatography using the immunizing peptide.

**Clonality** Polyclonal

1

**Isotype** IgG

## **Applications**

### The Abpromise guarantee

Our **Abpromise guarantee** covers the use of ab36121 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
WB	<b>★★★★ (1)</b>	Use a concentration of 1 - 3 µg/ml. Predicted molecular weight: 179 kDa. Primary incubation for 1 hour.
IHC-P		Use a concentration of 2 - 4 $\mu$ g/ml. Perform heat mediated antigen retrieval with citrate buffer pH 6 before commencing with IHC staining protocol.

## **Target**

### **Function**

Component of the Wnt-Fzd-LRP5-LRP6 complex that triggers beta-catenin signaling through inducing aggregation of receptor-ligand complexes into ribosome-sized signalsomes. Cell-surface coreceptor of Wnt/beta-catenin signaling, which plays a pivotal role in bone formation. The Wnt-induced Fzd/LRP6 coreceptor complex recruits DVL1 polymers to the plasma membrane which, in turn, recruits the AXIN1/GSK3B-complex to the cell surface promoting the formation of signalsomes and inhibiting AXIN1/GSK3-mediated phosphorylation and destruction of beta-catenin. Appears be required for postnatal control of vascular regression in the eye. Required for posterior patterning of the epiblast during gastrulation.

## Tissue specificity

## Involvement in disease

Widely expressed, with the highest level of expression in the liver.

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.

## Sequence similarities

Belongs to the LDLR family.

Contains 4 EGF-like domains.

Contains 3 LDL-receptor class A domains.

Contains 20 LDL-receptor class B repeats.

# Post-translational modifications

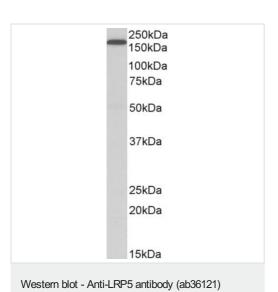
Phosphorylation of cytoplasmic PPPSP motifs regulates the signal transduction of the Wnt

signaling pathway through acting as a docking site for AXIN1.

Cellular localization

Membrane. Endoplasmic reticulum. Chaperoned to the plasma membrane by MESD.

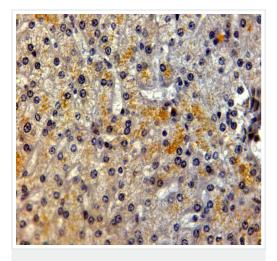
### **Images**



Anti-LRP5 antibody (ab36121) at 1 μg/ml + HepG2 lysate at 35 μg

Predicted band size: 179 kDa

Primary incubation for 1 hour. Detected by chemiluminescence.



Immunohistochemistry (Formalin/PFA-fixed paraffinembedded sections) - Anti-LRP5 antibody (ab36121)

Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) analysis of human liver tissue labelling LRP5 with ab36121 at 2  $\mu$ g/mL. Heat mediated antigen retrieval was performed using citrate buffer pH 6. Visualised using HRP-staining.

Please note: All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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