**Product datasheet**

**Anti-LRP5 antibody ab36121**

1 Abreviews  6 References  4 Images

**Overview**

**Product name**  Anti-LRP5 antibody

**Description**  Goat polyclonal to LRP5

**Host species**  Goat

**Tested applications**  Suitable for: ICC/IF, IHC-P, WB, IP

**Species reactivity**  Reacts with: Rabbit, Human

**Predicted to work with:** Mouse, Rat

**Immunogen**

Synthetic peptide: ERVEKTTGDKRT, corresponding to internal sequence amino acids 1178-1189 of Human LRP5

**Properties**

**Form**  Liquid

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

**Storage buffer**  Preservative: 0.02% Sodium Azide

Consitutents: 0.5% BSA, Tris saline. pH 7.3

**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

**Isotype**  IgG

**Applications**

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.
**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 to be required for postnatal control of vascular regression in the eye. Required for posterior patterning of the epiblast during gastrulation.

**Tissue specificity**

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

**Involvement in 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. Individuals 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

<table>
<thead>
<tr>
<th>Application</th>
<th>Abreviews</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICC/IF</td>
<td></td>
<td>Use a concentration of 1 μg/ml.</td>
</tr>
<tr>
<td>IHC-P</td>
<td></td>
<td>Use a concentration of 1 - 2 μg/ml.</td>
</tr>
<tr>
<td>WB</td>
<td>! ! ! !</td>
<td>1/200. Predicted molecular weight: 179 kDa. The band seen in the Abreview corresponds to the rabbit protein predicted to by 56kDa</td>
</tr>
<tr>
<td>IP</td>
<td></td>
<td>Use at an assay dependent concentration. PubMed: 21411632</td>
</tr>
</tbody>
</table>

**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 to be required for postnatal control of vascular regression in the eye. Required for posterior patterning of the epiblast during gastrulation.

**Tissue specificity**

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

**Involvement in 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. Individuals 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

Western blot - Anti-LRP5 antibody (ab36121)
ab36121 at 1µg/ml staining LRP5 in human pancreas tissue section by Immunohistochemistry (Formalin/PFA fixed paraffin-embedded sections). Tissue underwent antigen retrieval in microwave and in Tris/EDTA buffer (pH 9). HRP-staining procedure was used for detection.

ICC/IF image of ab36121 stained HepG2 cells. The cells were 4% formaldehyde fixed (10 min) and then incubated in 1%BSA / 10% normal donkey serum / 0.3M glycine in 0.1% PBS-Tween for 1h to permeabilise the cells and block non-specific protein-protein interactions. The cells were then incubated with the antibody (ab36121, 1µg/ml) overnight at +4°C. The secondary antibody (green) was Alexa Fluor® 488 donkey anti-goat IgG (H+L) used at a 1/1000 dilution for 1h. Alexa Fluor® 594 WGA was used to label plasma membranes (red) at a 1/200 dilution for 1h. DAPI was used to stain the cell nuclei (blue) at a concentration of 1.43µM.

ab36121 staining LRP5 in Human kidney tissue at a concentration of 4 µg/ml by Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections). Steamed antigen retrieval with citrate buffer pH 6.0 was performed, and the antibody was detected with HRP-staining.

Please note: All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"
Our Abpromise to you: Quality guaranteed and expert technical support

- Replacement or refund for products not performing as stated on the datasheet
- Valid for 12 months from date of delivery
- Response to your inquiry within 24 hours
- We provide support in Chinese, English, French, German, Japanese and Spanish
- Extensive multi-media technical resources to help you
- We investigate all quality concerns to ensure our products perform to the highest standards

If the product does not perform as described on this datasheet, we will offer a refund or replacement. For full details of the Abpromise, please visit https://www.abcam.com/abpromise or contact our technical team.

Terms and conditions

- Guarantee only valid for products bought direct from Abcam or one of our authorized distributors