

bs-18366R**[Primary Antibody]****LRP5 + LRP6 Rabbit pAb**

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— DATASHEET —

Host: Rabbit	Isotype: IgG	Applications: IHC-P (1:100-500) IHC-F (1:100-500) IF (1:100-500) ICC/IF (1:100-500)
Clonality: Polyclonal		
GeneID: 4041	SWISS: O75197	
Target: LRP5 + LRP6		
Immunogen: KLH conjugated synthetic peptide derived from human LRP5 + LRP6: 521-620/1615.		
Purification: affinity purified by Protein A		Reactivity: (predicted: Human, Mouse, Rat, Pig, Sheep, Cow, Chicken)
Concentration: 1mg/ml		
Storage: 0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol. Shipped at 4°C. Store at -20°C for one year. Avoid repeated freeze/thaw cycles.		Predicted MW.: 176 kDa
Background: 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.		Subcellular Location: Cell membrane ,Cytoplasm