[Primary Antibody]

LRP5 + LRP6 Rabbit pAb



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- DATASHEET		400-901-9800
Host: Rabbit	Isotype: gG	Applications: IHC-P (1:100-500)
Clonality: Polyclonal		IHC-F (1:100-500) IF (1:100-500)
GenelD: 4041	SWISS: 075197	ICC/IF (1:100-500)
Target: LRP5 + LRP6		Reactivity: (predicted: Human, Mouse,
Immunogen: KLH conjugated synthetic peptide derived from human LRP5 + LRP6: 521-620/1615.		Rat, Pig, Sheep, Cow, Chicken)
Purification: affinity purified by	Protein A	
Concentration: 1mg/ml		Predicted MW.: ^{176 kDa}
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.		Subcellular Cell membrane ,Cytoplasm Location:
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.		