## LRP5 (D23F7) Rabbit mAb



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## For Research Use Only. Not for Use in Diagnostic Procedures.

<b>Applications:</b> W, IP	Reactivity: H	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 200	<b>Source/Isotype:</b> Rabbit IgG	<b>UniProt ID:</b> #O75197	Entrez-Gene Id: 4041
Product Usage Information		<b>Application</b> Western Blotting Immunoprecipitation			<b>Dilution</b> 1:1000 1:200	
Storage		Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 $\mu$ g/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.				
Specificity/Sensitivity		LRP5 (D23F7) Rabbit mAb detects endogenous levels of total LRP5 protein.				
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Pro1527 of human LRP5 protein.				
Background		LRP5 and LRP6 are single-pass transmembrane proteins belonging to the low-density lipoprotein receptor (LDLR)-related protein family. Unlike other members of the LDLR family, LRP5 and LRP6 have four EGF and three LDLR repeats in the extracellular domain, and proline-rich motifs in the cytoplasmic domain (1). They function as co-receptors for Wnt and are required for the canonical Wnt/ $\beta$ -catenin signaling pathway (2,3). LRP5 and LRP6 are highly homologous and have redundant roles during development (4,5). The activity of LRP5 and LRP6 can be inhibited by the binding of some members of the Dickkopf (DKK) family of proteins (6,7). Upon stimulation with Wnt, LRP6 is phosphorylated at multiple sites including Thr1479, Ser1490, and Thr1493 by kinases such as GSK-3 and CK1 (8-10). Phosphorylated LRP6 recruits axin to the membrane and presumably activates $\beta$ -catenin signaling (8-10). LRP5 is involved in the regulation of bone homeostasis. Mutations and polymorphisms in LPR5 are associated with bone diseases like osteoporosis-pseudoglioma syndrome and high-bone-mass disorders (11-13). In addition, mutations in LRP5 are found in patients with hyperparathyroid tumor and breast cancer (14,15).				
Background References		1. Brown, S.D. et al. (1998) <i>Biochem. Biophys. Res. Commun.</i> 248, 879-888.  2. Pinson, K.I. et al. (2000) <i>Nature</i> 407, 535-538.  3. Tamai, K. et al. (2004) <i>Development</i> 131, 2803-2815.  4. Kelly, O.G. et al. (2004) <i>Development</i> 131, 1663-1677.  6. Semënov, M.V. et al. (2001) <i>Curr Biol</i> 11, 951-61.  7. Bafico, A. et al. (2001) <i>Nat. Cell Biol.</i> 3, 683-668.  8. Tamai, K. et al. (2004) <i>Mol. Cell</i> 13, 149-156.  9. Zeng, X. et al. (2005) <i>Nature</i> 438, 873-877.  10. Davidson, G. et al. (2005) <i>Nature</i> 438, 867-872.  11. Levasseur, R. et al. (2005) <i>Joint Bone Spine</i> 72, 207-214.  12. Ferrari, S.L. et al. (2005) <i>Curr. Opin. Lipidol.</i> 16, 207-214.  13. Balemans, W. and Van Hul, W. (2007) <i>Endocrinology</i> 148, 2622-2629.  14. Björklund, P. et al. (2009) <i>PLoS Med.</i> 4, e328.  15. Björklund, P. et al. (2009) <i>PLoS One</i> 4, e4243.				

**Species Reactivity** 

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

**Applications Key** 

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

W: Western Blotting IP: Immunoprecipitation

Cross-Reactivity Key

**H:** Human

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