

## TBL1XR1/TBLR1 (D4J9C) Rabbit mAb



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<b>Applications:</b> W, IP, ChIP	<b>Reactivity:</b> H Mk	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 50, 60	<b>Source/Isotype:</b> Rabbit IgG	<b>UniProt ID:</b> #Q9BZK7	<b>Entrez-Gene Id:</b> 79718
Product Usage Information		For optimal ChIP results, use 10 µl of antibody and 10 µg of chromatin (approximately 4 x 10 <sup>6</sup> cells) per IP. This antibody has been validated using SimpleChIP® Enzymatic Chromatin IP Kits.				
		Application			Dilution	
		Western Blotting			1:1000	
		Immunoprecipitation			1:100	
		Chromatin IP			1:50	
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		TBL1XR1/TBLR1 (D4J9C) Rabbit mAb recognizes endogenous levels of total TBL1XR1/TBLR1 protein. This antibody also cross-reacts with an unidentified protein of 130 kDa.				
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Pro159 of human TBL1XR1/TBLR1 protein.				
Background		Transducing $\beta$ -like protein 1 (TBL1X/TBL1) and TBL1-related protein 1 (TBL1XR1/TBLR1) were originally identified as subunits of the co-repressor silencing mediator for retinoic and thyroid hormone receptor (SMRT) and nuclear receptor co-repressor (NCoR) complexes (1-3). These two factors are required for the exchange of co-repressor complexes for co-activators by acting as adaptors to recruit the ubiquitin/proteasome machinery that degrades the co-repressor proteins during ligand mediated activation of transcription (4,5). Co-factor exchange driven by TBL1X/TBL1 and TBL1XR1/TBLR1 appears to be the mechanism by which c-Jun and NF- $\kappa$ B mediated transcription is activated and is therefore likely to be the mechanism employed by other signal-dependent transcription factors as well (4,6). In addition, both TBL1X/TBL1 and TBL1XR1/TBLR1 have essential roles in regulating the Wnt-signaling pathway by recruiting $\beta$ -catenin to Wnt target genes to activate transcription. Depletion of TBL1X-TBL1XR1 significantly inhibited Wnt-beta-catenin- induced gene expression and oncogenic growth in vitro and in vivo (7). Research studies have shown that upregulation of TBL1XR/TBLR1 is observed in a variety of solid tumors, and is correlated with advanced tumor stage, metastasis and poor prognosis (1).				
Background Re	ferences	1. Li, J.Y. et al. (2015) <i>Am J Clin Exp Urol</i> 3, 13-23. 2. Zhang, J. et al. (2002) <i>Mol Cell</i> 9, 611-23. 3. Yoon, H.G. et al. (2003) <i>EMBO J</i> 22, 1336-46. 4. Perissi, V. et al. (2004) <i>Cell</i> 116, 511-26. 5. Perissi, V. et al. (2008) <i>Mol Cell</i> 29, 755-66. 6. Hoberg, J.E. et al. (2004) <i>Mol Cell</i> 16, 245-55. 7. Li, J. and Wang, C.Y. (2008) <i>Nat Cell Biol</i> 10, 160-9.				
Species Reactivity		Species reactivity is determined by testing in at least one approved application (e.g., western blot).				

Western Blot Buffer IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v nonfat

dry milk, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key W: Western Blotting IP: Immunoprecipitation ChIP: Chromatin IP

Cross-Reactivity Key H: Human Mk: Monkey

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