RXR Antibody



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For Research Use Only, Not for Use in Diagnostic Procedures

Applications: W, IP	Reactivity: H M	Sensitivity: Endogenous	MW (kDa): 70-72	Source/Isotype: Rabbit	UniProt ID: #P28702	Entrez-Gene Id 6257
Product Usage Information	•	Application Western Blotting		Dilution 1:1000		
		Immunoprecipitation			1:50	
Storage		Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 μ g/ml BSA and 50% glycerol. Store at – 20°C. Do not aliquot the antibody.				
Specificity/Sensitivity		RXR β Antibody recognizes endogenous levels of total RXR β protein. This antibody does not cross-react with either RXR α or RXR γ proteins.				
Species predicted to react based on 100% sequence homology		Rat, Monkey, Bovine, Dog, Pig				
Source / Purification		Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues near the amino terminus of human RXR β protein. Antibodies are purified by protein A and peptide affinity chromatography.				
Background		The human retinoid X receptors (RXRs) are encoded by three distinct genes ($RXR\alpha$, $RXR\beta$, and $RXR\gamma$) and bind selectively and with high affinity to the vitamin A derivative, 9-cis-retinoic acid. RXRs are type-II nuclear hormone receptors that are largely localized to the nuclear compartment independent of ligand binding. Nuclear RXRs form heterodimers with nuclear hormone receptor subfamily 1 proteins, including thyroid hormone receptor, retinoic acid receptors, vitamin D receptor, peroxisome proliferator-activated receptors, liver X receptors, and farnesoid X receptor (1). Since RXRs heterodimerize with multiple nuclear hormone receptors, they play a central role in transcriptional control of numerous hormonal signaling pathways by binding to cis-acting response elements in the promoter/enhancer region of target genes (2). RXR β , like other members of the RXR subfamily, possesses a characteristic tripartite modular structure consisting of (a) a highly conserved central region containing the C_4/C_5 zinc-finger domain, which is responsible for DNA binding; (b) a relatively well-conserved C-terminal region, which contains the hormone binding and dimerization domains; and (c) a variable N-terminal domain, which has been implicated in either transactivation or repression of target genes (2). Variability within the N-terminal domain is thought to be the result of alternative splicing and/or differential promoter usage (3-5). The murine RXR β was initially identified because of its ability to bind to the regulatory region II in the murine major histocompatability complex (MHC) class I promoter and is therefore also referred to as H2RIBP (6). Genetic ablation of murine $Rxrb$ produced approximately 50% lethality in utero and males that survived had defects of spermatazoa, which resulted in sterility (7). Further studies revealed that				

Background References

- 1. Gronemeyer, H. et al. (2004) Nat Rev Drug Discov 3, 950-64.
- 2. Mangelsdorf, D.J. et al. (1992) *Genes Dev* 6, 329-44.
- 3. Nagata, T. et al. (1994) Gene 142, 183-9.

metabolism (8).

- 4. Fleischhauer, K. et al. (1993) *Hum Genet* 90, 505-10.
- 5. Fleischhauer, K. et al. (1992) Nucleic Acids Res 20, 1801.
- 6. Hamada, K. et al. (1989) Proc Natl Acad Sci USA 86, 8289-93.
- 7. Kastner, P. et al. (1996) *Genes Dev* 10, 80-92.
- 8. Mascrez, B. et al. (2004) EMBO Rep 5, 285-90.

expression of a Rxrb mutant with an impaired AF-2 core led to abnormal lipid metabolism in Sertoli cells, suggesting functional interactions between Rxrb and other nuclear receptors that control lipid

Western Blot Buffer 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.

Applications Key W: Western Blotting **IP**: Immunoprecipitation

Cross-Reactivity Key H: Human M: Mouse

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