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RXR α (D6H10) Rabbit mAb

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

Applications: W, IP, ChIP	Reactivity: H M R	Sensitivity: Endogenous	MW (kDa): 53	Source/Isotype: Rabbit IgG	UniProt ID: #P19793	Entrez-Gene Id: 6256
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Product Usage Information

For optimal ChIP results, use 10 μ l of antibody and 10 μ g of chromatin (approximately 4 x 10⁶ 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 μ g/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.

Specificity/Sensitivity

RXR α (D6H10) Rabbit mAb recognizes endogenous levels of total RXR α protein. This antibody does not cross-react with either RXR β or RXR γ .

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues near the amino terminus of human RXR α protein.

Background

The human retinoid X receptors (RXRs) are encoded by three distinct genes (*RXR α* , *RXR β* , and *RXR γ*) 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).

Retinoid X receptor α (RXR α) is the founding RXR family member and is predominantly expressed in the liver, kidney, epidermis, intestine, and a variety of tissues (2-4). Knockout of the murine *rxra* gene results in embryonic lethality tentatively due to myocardial hypoplasia, which demonstrates the importance of RXR α in retinoid signaling *in vivo* (5,6). Biochemical evidence suggests that RXR α transcriptional activity is post-translationally regulated through the Ras-Raf-MAPK signaling cascade. MAPK-dependent phosphorylation of RXR α directly abrogates the ability of RXR α to associate with nuclear receptor coactivators (7).

Background References

- Gronemeyer, H. et al. (2004) *Nat Rev Drug Discov* 3, 950-64.
- Mangelsdorf, D.J. et al. (1992) *Genes Dev* 6, 329-44.
- Mangelsdorf, D.J. et al. (1990) *Nature* 345, 224-9.
- Dollé, P. et al. (1994) *Mech Dev* 45, 91-104.
- Kastner, P. et al. (1994) *Cell* 78, 987-1003.
- Sucov, H.M. et al. (1994) *Genes Dev* 8, 1007-18.
- Macoritto, M. et al. (2008) *J Biol Chem* 283, 4943-56.

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 TBST, 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 **M:** Mouse **R:** Rat

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