

**NR1H4 Antibody (N-term)**  
**Affinity Purified Rabbit Polyclonal Antibody (Pab)**  
**Catalog # AP19002a**

**Specification**

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**NR1H4 Antibody (N-term) - Product Information**

Application	WB,E
Primary Accession	<a href="#">O96RI1</a>
Other Accession	<a href="#">O3SZL0</a> , <a href="#">NP_005114.1</a>
Reactivity	Human
Predicted	Bovine
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	55914
Antigen Region	18-46

**NR1H4 Antibody (N-term) - Additional Information**

**Gene ID** 9971

**Other Names**

Bile acid receptor, Farnesoid X-activated receptor, Farnesol receptor HRR-1, Nuclear receptor subfamily 1 group H member 4, Retinoid X receptor-interacting protein 14, RXR-interacting protein 14, NR1H4, BAR, FXR, HRR1, RIP14

**Target/Specificity**

This NR1H4 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 18-46 amino acids from the N-terminal region of human NR1H4.

**Dilution**

WB~~1:1000

E~~Use at an assay dependent concentration.

**Format**

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

**Storage**

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

**Precautions**

NR1H4 Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

**NR1H4 Antibody (N-term) - Protein Information**

**Name** NR1H4**Synonyms** BAR, FXR, HRR1, RIP14

**Function** Ligand-activated transcription factor. Receptor for bile acids (BAs) such as chenodeoxycholic acid (CDCA), lithocholic acid, deoxycholic acid (DCA) and allocholic acid (ACA). Plays a essential role in BA homeostasis through the regulation of genes involved in BA synthesis, conjugation and enterohepatic circulation. Also regulates lipid and glucose homeostasis and is involved innate immune response (PubMed:[10334992](#), PubMed:[10334993](#), PubMed:[21383957](#), PubMed:[22820415](#)). The FXR-RXR heterodimer binds predominantly to farnesoid X receptor response elements (FXREs) containing two inverted repeats of the consensus sequence 5'-AGGTCA-3' in which the monomers are spaced by 1 nucleotide (IR-1) but also to tandem repeat DR1 sites with lower affinity, and can be activated by either FXR or RXR-specific ligands. It is proposed that monomeric nuclear receptors such as NR5A2/LRH-1 bound to coregulatory nuclear responsive element (NRE) halvesites located in close proximity to FXREs modulate transcriptional activity (By similarity). In the liver activates transcription of the corepressor NR0B2 thereby indirectly inhibiting CYP7A1 and CYP8B1 (involved in BA synthesis) implicating at least in part histone demethylase KDM1A resulting in epigenomic repression, and SLC10A1/NTCP (involved in hepatic uptake of conjugated BAs). Activates transcription of the repressor MAFG (involved in regulation of BA synthesis) (By similarity). Activates transcription of SLC27A5/BACS and BAAT (involved in BA conjugation), ABCB11/BSEP (involved in bile salt export) by directly recruiting histone methyltransferase CARM1, and ABCC2/MRP2 (involved in secretion of conjugated BAs) and ABCB4 (involved in secretion of phosphatidylcholine in the small intestine) (PubMed:[12754200](#), PubMed:[15471871](#), PubMed:[17895379](#)). Activates transcription of SLC27A5/BACS and BAAT (involved in BA conjugation), ABCB11/BSEP (involved in bile salt export) by directly recruiting histone methyltransferase CARM1, and ABCC2/MRP2 (involved in secretion of conjugated BAs) and ABCB4 (involved in secretion of phosphatidylcholine in the small intestine) (PubMed:[10514450](#), PubMed:[15239098](#), PubMed:[16269519](#)). In the intestine activates FGF19 expression and secretion leading to hepatic CYP7A1 repression (PubMed:[12815072](#), PubMed:[19085950](#)). The function also involves the coordinated induction of hepatic KLB/beta-klotho expression (By similarity). Regulates transcription of liver UGT2B4 and SULT2A1 involved in BA detoxification; binding to the UGT2B4 promoter seems to imply a monomeric transactivation independent of RXRA (PubMed:[12806625](#), PubMed:[16946559](#)). Modulates lipid homeostasis by activating liver NR0B2/SHP-mediated repression of SREBF1 (involved in de novo lipogenesis), expression of PLTP (involved in HDL formation), SCARB1 (involved in HDL hepatic uptake), APOE, APOC1, APOC4, PPARA (involved in beta-oxidation of fatty acids), VLDLR and SDC1 (involved in the hepatic uptake of LDL and IDL remnants), and inhibiting expression of MTTP (involved in VLDL assembly (PubMed:[12554753](#), PubMed:[12660231](#), PubMed:[15337761](#)). Increases expression of APOC2 (promoting lipoprotein lipase activity implicated in triglyceride clearance) (PubMed:[11579204](#)). Transrepresses APOA1 involving a monomeric competition with NR2A1 for binding to a DR1 element (PubMed:[11927623](#), PubMed:[21804189](#)). Also reduces triglyceride clearance by inhibiting expression of ANGPTL3 and APOC3 (both involved in inhibition of lipoprotein lipase) (PubMed:[12891557](#)). Involved in glucose homeostasis by modulating hepatic gluconeogenesis through activation of NR0B2/SHP-mediated repression of respective genes. Modulates glycogen synthesis (inducing phosphorylation of glycogen synthase kinase-3) (By similarity). Modulates glucose-stimulated insulin secretion and is involved in insulin resistance (PubMed:[20447400](#)). Involved in intestinal innate immunity. Plays a role in protecting the distal small intestine against bacterial overgrowth and preservation of the epithelial barrier (By similarity). Down-regulates inflammatory cytokine expression in several types of immune cells including macrophages and mononuclear cells (PubMed:[21242261](#)). Mediates trans-repression of TLR4-induced cytokine expression; the function seems to require its sumoylation and prevents N-CoR nuclear receptor corepressor clearance from target genes such as IL1B and NOS2 (PubMed:[19864602](#)). Involved in the TLR9-mediated protective mechanism in intestinal inflammation. Plays an anti-inflammatory role in liver inflammation; proposed to inhibit pro-inflammatory (but not antiapoptotic) NF-kappa-B signaling) (By similarity).

**Cellular Location**

Nucleus. [Isoform 2]: Nucleus [Isoform 4]: Nucleus

### Tissue Location

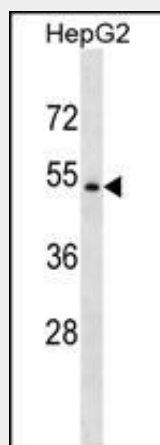
Liver and hepatocyte-related cells express mainly FXRalpha1-type isoforms with isoform 3 and isoform 4 in approximately equal proportions. In intestine and kidney mainly FXRalpha2-type isoforms are expressed with isoform 1 and isoform 2 in approximately equal proportions. Expressed in pancreatic beta cells and macrophages

### NR1H4 Antibody (N-term) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

### NR1H4 Antibody (N-term) - Images



NR1H4 Antibody (N-term) (Cat. #AP19002a) western blot analysis in HepG2 cell line lysates (35ug/lane). This demonstrates the NR1H4 antibody detected the NR1H4 protein (arrow).

### NR1H4 Antibody (N-term) - Background

NR1H4 is a Ligand-activated transcription factor. Receptor for bile acids such as chenodeoxycholic acid, lithocholic acid and deoxycholic acid. Represses the transcription of the cholesterol 7-alpha-hydroxylase gene (CYP7A1) through the induction of NR0B2 or FGF19 expression, via two distinct mechanisms. Activates the intestinal bile acid-binding protein (IBABP). Activates the transcription of bile salt export pump ABCB11 by directly recruiting histone methyltransferase CARM1 to this locus.

### NR1H4 Antibody (N-term) - References

Meyer Zu Schwabedissen, H.E., et al. Hepatology 52(5):1797-1807(2010)  
Popescu, I.R., et al. FEBS Lett. 584(13):2845-2851(2010)  
Gadaleta, R.M., et al. Biochim. Biophys. Acta 1801(7):683-692(2010)  
Roberts, K.E., et al. Gastroenterology 139(1):130-139(2010)  
Erichsen, T.J., et al. J. Hepatol. 52(4):570-578(2010)