

Product datasheet for **TA392602**

Bile Acid Receptor (NR1H4) Rabbit Polyclonal Antibody

Product data:

Product Type:	Primary Antibodies
Applications:	WB
Recommended Dilution:	WB: 1:1000~1:2000
Reactivity:	Human
Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Immunogen:	Synthetic peptide, corresponding to Human FXR.
Specificity:	FXR polyclonal antibody detects endogenous levels of FXR protein.
Formulation:	Rabbit IgG, 1mg/ml in PBS with 0.02% sodium azide, 50% glycerol, pH7.2.
Concentration:	1mg/ml
Conjugation:	Unconjugated
Storage:	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze-thaw cycles.
Stability:	1 year
Predicted Protein Size:	~ 67 kDa
Gene Name:	nuclear receptor subfamily 1 group H member 4
Database Link:	Entrez Gene 9971 Human Q96RI1



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- Background:** The farnesoid X receptor (FXR/NR1H4) is a member of the nuclear hormone receptor superfamily and is a master regulator of bile acid synthesis. FXR/NR1H4 heterodimerizes with RXR-alpha upon activation by bile acids, which begins a regulatory cascade involving SHP and LRH-1 to control lipid homeostasis. FXR/NR1H4 has also been shown to be a critical regulator of glucose homeostasis. In addition to directly regulating genes, FXR/NR1H4 also plays a post transcriptional role in bile acid metabolism by transcribing the RNA-binding protein ZFP36L1, which in turn downregulates the key enzyme Cyp7a1. Mutations in human FXR/NR1H4 have been shown to cause cholestasis and liver disease in neonatal patients. FXR/NR1H4 can also control Lgr5+ intestinal stem cell proliferation and its upregulation has been shown to inhibit colorectal cancer progression. Agonists against FXR/NR1H4 are being evaluated for various liver diseases and diabetes.
- Synonyms:** BAR; Bile acid receptor; Farnesoid X-activated receptor; Farnesol receptor HRR-1; FXR; HRR1; NR1H4; Nuclear receptor subfamily 1 group H member 4; Retinoid X receptor-interacting protein 14; RIP14; RXR-interacting protein 14
- Note:** For research use only, not for use in diagnostic procedure.