SANTA CRUZ BIOTECHNOLOGY, INC.

PPARβ (H-74): sc-7197

SANTA CRUZ BIOTECHNOLOGY
The Power to Question

BACKGROUND

Peroxisome proliferator-activated receptors (PPARs) are nuclear hormone receptors that can be activated by a variety of compounds including fibratus, thiazolidinediones, prostaglandins and fatty acids. Three PPAR subtypes, designated PPARα, PPARβ (also designated PPARδ) and PPARγ, have been described. PPARs promote transcription by forming heterodimers with members of the retinoid X receptor (RXR) family of steroid receptors and binding to specific DNA motifs termed PPAR-response elements (PPREs). PPARα is abundant in primary hepatocytes where it regulates the expression of proteins involved in fatty acid metabolism. PPARβ is the most widely distributed subtype and is often expressed at high levels. PPARγ is predominantly seen in adipose tissue where it plays a critical role in regulating adipocyte differentiation. Interestingly, both the orphan nuclear hormone receptor LXRα and thyroid receptor (TR) have been shown to act as antagonists of PPARα/RXRα binding to PPREs.

CHROMOSOMAL LOCATION

Genetic locus: PPARD (human) mapping to 6p21.31; Ppard (mouse) mapping to 17 A3.3.

SOURCE

PPARβ (H-74) is a rabbit polyclonal antibody raised against amino acids 2-75 of PPARβ of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Available as TransCruz reagent for Gel Supershift and ChIP applications, sc-7197 X, 200 µg/0.1 ml.

APPLICATIONS

PPARβ (H-74) is recommended for detection of PPARβ (also designated PPARδ) of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for PPARβ siRNA (h): sc-36305, PPARβ siRNA (m): sc-36306, PPARβ shRNA Plasmid (h): sc-36305-SH, PPARβ shRNA Plasmid (m): sc-36306-SH, PPARβ shRNA (h) Lentiviral Particles: sc-36305-V and PPARβ shRNA (m) Lentiviral Particles: sc-36306-V.

PPAR (H-74) X TransCruz antibody is recommended for Gel Supershift and ChIP applications.

Molecular Weight of PPARβ: 52 kDa.

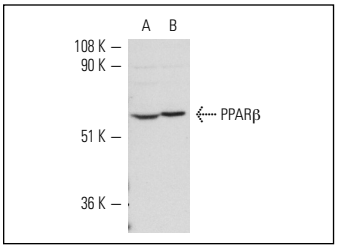
STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

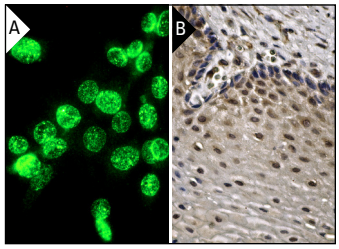
RESEARCH USE

For research use only, not for use in diagnostic procedures.

DATA



PPARβ (H-74): sc-7197. Western blot analysis of PPARβ expression in Jurkat (**A**) and Sol8 (**B**) nuclear extracts.



PPARβ (H-74): sc-7197. Immunofluorescence staining of methanol-fixed Sol8 cells showing nuclear localization (**A**). Immunoperoxidase staining of formalin fixed, paraffin-embedded human cervix tissue showing nuclear staining of squamous epithelial cells (**B**).

SELECT PRODUCT CITATIONS

1. Bishop-Bailey, D., et al. 1999. Endothelial cell apoptosis induced by the peroxisome proliferator-activated receptor (PPAR) ligand 15-deoxy-δ12,14-prostaglandin J2. J. Biol. Chem. 274: 17042-17048.
2. Stockert, J., et al. 2011. Reverse crosstalk of TGFβ and PPARβ/δ signaling identified by transcriptional profiling. Nucleic Acids Res. 39: 119-131.
3. Pollock, C.B., et al. 2011. PPARδ activation acts cooperatively with 3-phosphoinositide-dependent protein kinase-1 to enhance mammary tumorigenesis. PLoS ONE 6: e16215.
4. Adhikary, T., et al. 2011. Genomewide analyses define different modes of transcriptional regulation by peroxisome proliferator-activated receptor-β/δ (PPARβ/δ). PLoS ONE 6: e16344.
5. Liang, Y.J., et al. 2011. Peroxisome proliferator-activated receptor δ downregulates the expression of the receptor for advanced glycation end products and pro-inflammatory cytokines in the kidney of streptozotocin-induced diabetic mice. Eur. J. Pharm. Sci. 43: 65-70.
6. Ju, Q., et al. 2011. 2,3,7,8-Tetrachlorodibenzo-p-dioxin alters sebaceous gland cell differentiation *in vitro*. Exp. Dermatol. 20: 320-325.
7. Jana, M., et al. 2012. Gemfibrozil, a lipid lowering drug, inhibits the activation of primary human microglia via peroxisome proliferator-activated receptor β. Neurochem. Res. 37: 1718-1729.
8. Stockert, J., et al. 2013. Regulation of TAK1/TAB1-mediated IL-1β signaling by cytoplasmic PPARβ/δ. PLoS ONE 8: e63011.