

PPAR β (H-74): sc-7197

BACKGROUND

Peroxisome proliferator-activated receptors (PPARs) are nuclear hormone receptors that can be activated by a variety of compounds including fibrates, thiazolidinediones, prostaglandins and fatty acids. Three PPAR subtypes, designated PPAR α , PPAR β (also designated PPAR δ) and PPAR γ , 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). PPAR α is abundant in primary hepatocytes where it regulates the expression of proteins involved in fatty acid metabolism. PPAR β is the most widely distributed subtype and is often expressed at high levels. PPAR γ is predominantly seen in adipose tissue where it plays a critical role in regulating adipocyte differentiation. Interestingly, both the orphan nuclear hormone receptor LXR α and thyroid receptor (TR) have been shown to act as antagonists of PPAR α /RXR α binding to PPREs.

REFERENCES

1. Brun, R.P., et al. 1996. Differential activation of adipogenesis by multiple PPAR isoforms. *Genes and Dev.* 10: 974-984.
2. Mansen, A., et al. 1996. Expression of the peroxisome proliferator-activated receptor (PPAR) in the mouse colonic mucosa. *Biochem. Biophys. Res. Comm.* 222: 844-851.
3. Lemberger, T., et al. 1996. Expression of the peroxisome proliferator-activated receptor α gene is stimulated by stress and follows a diurnal rhythm. *J. Biol. Chem.* 271: 1764-1769.
4. Braissant, O., et al. 1996. Differential expression of peroxisome proliferator-activated receptors (PPARs): tissue distribution of PPAR- α , - β , and - γ in the adult rat. *Endocrinol.* 137: 354-366.

CHROMOSOMAL LOCATION

Genetic locus: PPAR δ (human) mapping to 6p21.1; Ppard (mouse) mapping to 17 A3.3.

SOURCE

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

PRODUCT

Each vial contains 200 μ g 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 μ g/0.1 ml.

STORAGE

Store at 4 $^{\circ}$ 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.

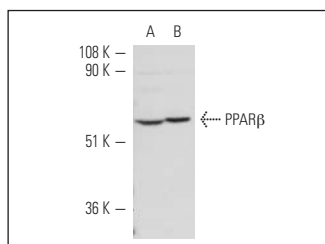
APPLICATIONS

PPAR β (H-74) is recommended for detection of PPAR β (also designated PPAR δ) of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

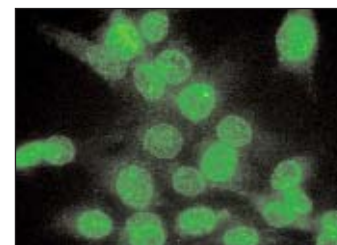
Suitable for use as control antibody for PPAR β siRNA (h): sc-36305 and PPAR β siRNA (m): sc-36306.

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

DATA



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



PPAR β (H-74): sc-7197. Immunofluorescence staining of methanol-fixed Sol8 cells showing nuclear localization.

SELECT PRODUCT CITATIONS

1. Cernuda-Morollon, E., et al. 2002. PPAR agonists amplify iNOS expression while inhibiting NF-kappaB: implications for mesangial cell activation by cytokines. *J. Am. Soc. Nephrol.* 13: 2223-2231.
2. Zhang, J., et al. 2002. Peroxisome proliferator-activated receptor δ is upregulated during vascular lesion formation and promotes post-confluent cell proliferation in vascular smooth muscle cells. *J. Biol. Chem.* 277: 11505.
3. Hao, C.M., et al. 2002. Peroxisome proliferator-activated receptor δ activation promotes cell survival following hypertonic stress. *J. Biol. Chem.* 277: 21341.
4. Di-Poi, N., et al. 2002. Antiapoptotic role of PPAR β in keratinocytes via transcriptional control of the Akt1 signaling pathway. *Mol. Cell* 10: 721-733.
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6. Pang, L., et al. 2003. Cyclooxygenase-2 expression by nonsteroidal anti-inflammatory drugs in human airway smooth muscle cells: role of peroxisome proliferator-activated receptors. *J. Immunol.* 170: 1043-1051.
7. Chen, C.W., et al. 2003. Inhibition of IFN-gamma-mediated inducible nitric oxide synthase induction by the peroxisome proliferator-activated receptor gamma agonist, 15-deoxy-delta 12,14-prostaglandin J2, involves inhibition of the upstream Janus kinase/STAT1 signaling pathway. *J. Immunol.* 171: 979-988.