

PPAR γ (N-20): sc-1984

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 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.

CHROMOSOMAL LOCATION

Genetic locus: PPARG (human) mapping to 3p25; Pparg (mouse) mapping to 6 52.7 cM (6 E3-F1).

SOURCE

PPAR γ (N-20) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the N-terminus of PPAR γ 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.

Blocking peptide available for competition studies, sc-1984 P, (100 μ g peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

Available as TransCruz reagent for Gel Supershift and ChIP applications, sc-1984 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 γ (N-20) is recommended for detection of PPAR γ 1 and PPAR γ 2 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).

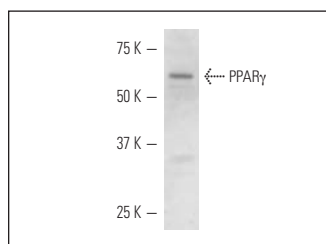
Suitable for use as control antibody for PPAR γ siRNA (h): sc-29455, PPAR γ siRNA (m): sc-29456, and PPAR γ siRNA (h2): sc-44220; and as shRNA Plasmid control antibody for PPAR γ shRNA Plasmid (h): sc-29455-SH, PPAR γ shRNA Plasmid (m): sc-29456-SH, and PPAR γ shRNA Plasmid (h2): sc-44220-SH.

PPAR γ (N-20) X TransCruz antibody is recommended for Gel Supershift and ChIP applications.

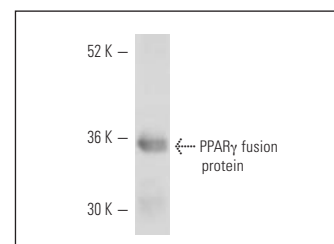
Molecular Weight of PPAR γ : 67 kDa.

Positive Controls: Human breast carcinoma tissue, rat skeletal muscle extract or U-937 cell lysate: sc-2239.

DATA



PPAR γ (N-20): sc-1984. Western blot analysis of PPAR γ expression in U-937 whole cell lysate.



PPAR γ (N-20): sc-1984. Western blot analysis of human recombinant PPAR γ fusion protein.

SELECT PRODUCT CITATIONS

1. Ricote, M., et al. 1998. Expression of the peroxisome proliferator-activated receptor gamma (PPAR γ) in human atherosclerosis and regulation in macrophage by colony stimulating factors and oxidized low density lipoprotein. *Proc. Natl. Acad. Sci. USA* 95: 7614-7619.
2. Bishop-Bailey, D., et al. 1999. Endothelial cell apoptosis induced by the peroxisome proliferator-activated receptor (PPAR) ligand 15-deoxy- δ 12,14-prostaglandin J2. *J. Biol. Chem.* 274: 17042-17048.
3. Barbera, M.J., et al. 2001. Peroxisome proliferator-activated receptor α activates transcription of the brown fat uncoupling protein-1 gene. A link between regulation of the thermogenic and lipid oxidation pathways in the brown fat cell. *J. Biol. Chem.* 276: 1486-1493.
4. Subbaramaiah, K., et al. 2001. PPAR γ ligands suppress the transcriptional activation of cyclooxygenase-2. Evidence for involvement of AP-1 and CBP/p300. *J. Biol. Chem.* 276: 12440-12448.
5. Teruel, T., et al. 2003. Rosiglitazone and retinoic acid induce uncoupling protein-1 (UCP-1) in a p38 mitogen-activated protein kinase-dependent manner in fetal primary brown adipocytes. *J. Biol. Chem.* 278: 263-269.