

PPAR γ_2 (G-18): sc-22020

BACKGROUND

Peroxisome proliferator-activated receptors (PPARs) are members of the nuclear hormone receptor subfamily of transcription factors. PPARs form heterodimers with retinoid X receptors (RXRs). These heterodimers regulate transcription of genes involved in Insulin action, adipocyte differentiation, lipid metabolism and inflammation. PPAR γ is implicated in numerous diseases including obesity, diabetes, atherosclerosis and cancer. PPAR γ activators include prostanoids, fatty acids, thiazolidinediones, and N-(2-benzoylphenyl) tyrosine analogues. A key component in adipocyte differentiation and fat-specific gene expression, PPAR γ may modulate macrophage functions such as proinflammatory activities, and stimulate oxidized low-density lipoprotein (x-LDL) uptake. A Pro12Ala polymorphism of the PPAR γ_2 gene has been reported to reduce transactivation activity *in vitro*. This substitution may affect the immune response to ox-LDL and be associated with type 2 diabetes. In addition, the Pro12Ala variant of the PPAR γ_2 gene maybe correlated with abdominal obesity in type 2 diabetes.

CHROMOSOMAL LOCATION

Genetic locus: PPARG (human) mapping to 3p25.2; Pparg (mouse) mapping to 6 E3.

SOURCE

PPAR γ_2 (G-18) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the N-terminus of PPAR γ_2 of mouse 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 agarose conjugate for immunoprecipitation, sc-22020 AC, 500 μ g/0.25 ml agarose in 1 ml.

Blocking peptide available for competition studies, sc-22020 P, (100 μ g peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

PPAR γ_2 (G-18) is recommended for detection of PPAR γ_2 of mouse, rat and to a lesser extent, 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), 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); non cross-reactive with PPAR γ_1 .

Suitable for use as control antibody for PPAR γ siRNA (h): sc-29455, PPAR γ siRNA (m): sc-29456, PPAR γ siRNA (r): sc-156077, PPAR γ shRNA Plasmid (h): sc-29455-SH, PPAR γ shRNA Plasmid (m): sc-29456-SH, PPAR γ shRNA Plasmid (r): sc-156077-SH, PPAR γ shRNA (h) Lentiviral Particles: sc-29455-V, PPAR γ shRNA (m) Lentiviral Particles: sc-29456-V and PPAR γ shRNA (r) Lentiviral Particles: sc-156077-V.

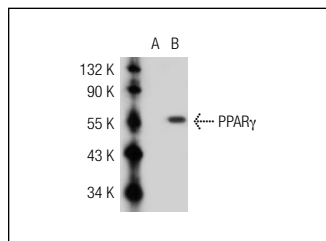
Molecular Weight of PPAR γ_2 : 60 kDa.

Positive Controls: PPAR γ (m): 293T Lysate: sc-122729.

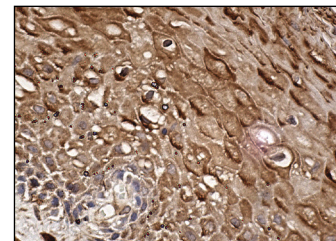
STORAGE

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

DATA



PPAR γ_2 (G-18): sc-22020. Western blot analysis of PPAR γ expression in non-transfected: sc-117752 (A) and mouse PPAR γ transfected: sc-122729 (B) 293T whole cell lysates.



PPAR γ_2 (G-18): sc-22020. Immunoperoxidase staining of formalin fixed, paraffin-embedded human cervix tissue showing nuclear and cytoplasmic staining of squamous epithelial cells.

SELECT PRODUCT CITATIONS

1. Böhme, M., et al. 2008. Analysis of the transcriptional regulation of the FABP2 promoter haplotypes by PPAR γ /RXR α and Oct-1. *Biochim. Biophys. Acta* 1779: 616-621.
2. Stanton, L.A., et al. 2008. PPAR γ_2 expression in growth plate chondrocytes is regulated by p38 and GSK-3. *J. Cell. Mol. Med.* 14: 242-256.
3. Duque, G., et al. 2010. Effects of risedronate on bone marrow adipocytes in postmenopausal women. *Osteoporos. Int.* 22: 1547-1553.
4. Stanton, L.A., et al. 2010. PPAR γ_2 expression in growth plate chondrocytes is regulated by p38 and GSK-3. *J. Cell. Mol. Med.* 14: 242-256.
5. Kim, S.J., et al. 2011. Adipocyte expression of the glucose-dependent Insulinotropic polypeptide receptor involves gene regulation by PPAR γ and histone acetylation. *J. Lipid Res.* 52: 759-770.
6. Araújo-Vilar, D., et al. 2011. Histological and molecular features of lipomatous and non-lipomatous adipose tissue in familial partial lipodystrophy due to LMNA mutations. *Clin. Endocrinol.* 76: 816-824.
7. Liew, C.W., et al. 2013. Ablation of TRIP-Br2, a regulator of fat lipolysis, thermogenesis and oxidative metabolism, prevents diet-induced obesity and insulin resistance. *Nat. Med.* 19: 217-226.

RESEARCH USE

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

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Try **PPAR γ_2 (E-9): sc-390740**, our highly recommended monoclonal alternative to PPAR γ_2 (G-18).