

PPAR- α

Peroxisome Proliferator-activated Receptor, α -isoform human, recombinant, *E. coli*

Cat. No.	Amount
PR-747	10 μ g

For *in vitro* use only
Quality guaranteed for 12 months
Store at -80°C

Avoid freeze / thaw cycles

Form

Liquid. Supplied in 20 mM Tris-HCl pH 8.0, 100 mM KCl, 0.2 mM EDTA, 1 mM DTT and 20% glycerol.

Activity

20 ng are sufficient for a gel-mobility shift assay and 100 ng are sufficient for a protein-protein interaction assay.

Application

PPAR α has been applied in DNA and protein-protein interaction assays.

Molecular Weight

52 kDa

Purity

> 95% by SDS-PAGE

Description

Recombinant His tagged PPAR α is isolated from an *E. coli* strain that carries the coding sequence of the human PPAR α under the control of a T7 promoter.

There is evidence that a group of closely related nuclear receptors, called Peroxisome Proliferator-activated Receptors (PPARs), may be involved in chronic diseases such as diabetes, obesity, atherosclerosis and cancer. The PPARs were first cloned as the nuclear receptors that mediate the effects of synthetic compounds called peroxisome proliferators on gene transcription. It soon became clear that eicosanoids and fatty acids can also regulate gene transcription through PPARs. They bind a specific element in the promoter region of target genes only as a heterodimer with the receptor for 9-cis retinoic acid, RXR (retinoid X receptor). Binding of the ligand of either receptor can activate the complex, but binding of both ligands simultaneously is more potent. Three PPAR isotypes have been identified: α , β (also called NUC1) and γ . PPAR α is expressed most in brown adipose tissue and liver, then kidney, heart and skeletal muscle. PPAR γ is mainly expressed in adipose tissue, and to a lesser extent in colon, the immune system and the retina. PPAR β is found in many tissues but the highest expression is in the gut, kidney and heart. The target genes of PPAR α are a relatively homogenous group of genes that participate in aspects of lipid catabolism such as fatty acid uptake through membranes, fatty acid binding in cells, fatty acid oxidation (in microsomes, peroxisomes and mitochondria) and lipoprotein assembly and transport.

Selected References:

Kersten (2000) Roles of PPARs in health and disease. *Nature* **405**:421.

Kersten (2001) Mechanisms of nutritional and hormonal regulation of lipogenesis. *EMBO Rep.* **21**:282.