PRODUCT INFORMATION

PPARα Ligand-binding Domain (human, recombinant)

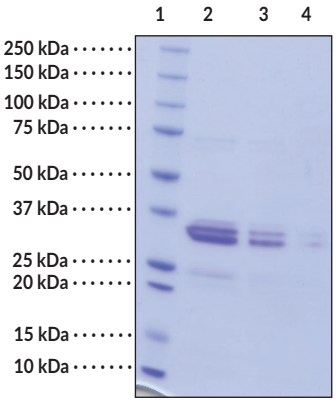
Item No. 10009088

Overview and Properties

Synonyms:	Peroxisome Proliferator-activated Receptor α Ligand Binding Domain, PPARα LBD
Source:	Recombinant human His-tagged PPARα ligand-binding domain expressed in *E. coli*
Amino acids:	170-430
Uniprot No.:	Q07869
Molecular Weight:	~34 kDa/subunit
Storage:	-80°C (as supplied)
Stability:	≥1 year
Purity:	***batch specific*** (≥55% estimated by SDS-PAGE)
Supplied in:	50 mM sodium phosphate, pH 7.2, containing 20% glycerol and 100 mM sodium chloride
Protein Concentration:	***batch specific*** mg/ml

Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Image



Lane 1: MW Markers
Lane 2: PPARα LBD (human recombinant) (4 µg)
Lane 3: PPARα LBD (human recombinant) (2 µg)
Lane 4: PPARα LBD (human recombinant) (1 µg)

Representative gel image shown; actual purity may vary between each batch.

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Description

Peroxisome proliferator-activated receptor α (PPARα) is a member of the nuclear receptor family of ligand-activated transcription factors that regulates a variety of metabolic functions and inflammation.[1] It contains an N-terminal domain that is subject to phosphorylation, a DNA-binding domain, and a C-terminal ligand-binding domain.[2] PPARα is highly expressed in tissues with high fatty acid oxidation rates, including the liver, heart, skeletal muscle, brown adipose tissue, and kidney, as well as in macrophages and T cells.[2,3] It is activated by a variety of endogenous ligands such as fatty acids, eicosanoids, and endocannabinoids, as well as synthetic agents, including fenofibrate (Item No. 10005368) and gemfibrozil (Item No. 14835).[4] Upon activation, PPARα heterodimerizes with the retinoid X receptor (RXR) and binds to PPAR response elements in PPARα target genes, recruiting RNA polymerase II and initiating gene transcription.[1] PPARα transcriptionally regulates a variety of genes involved in several cellular processes, including lipid and hormone transport, peroxisomal and mitochondrial β-oxidation, amino acid metabolism, and inflammation.[1,2] Genome-wide deletion of *Ppara* protects mice from high-fat diet-induced hyperinsulinemia and insulin resistance.[5] *PPARA* SNPs have been found in individuals with a variety of cardiovascular conditions, including hypertension, atherosclerosis, coronary artery disease, left ventricular hypertrophy, or myocardial infarction.[1] Formulations containing PPARα agonists have been used in the treatment of hyperlipidemia. Cayman's PPARα Ligand-binding Domain protein can be used for Western blot (WB) applications.

References

1. Li, S., Yang, B., Du, Y., *et al*. Targeting PPARα for the treatment and understanding of cardiovascular diseases. *Cell Physiol. Biochem*. **51(6)**, 2760-2775 (2018).
2. Pawlak, M., Lefebvre, P., and Staels, B. Molecular mechanism of PPARa action and its impact on lipid metabolism, inflammation and fibrosis in non-alcoholic fatty liver disease. *J. Hepatol*. **62**, 720-733 (2015).
3. Rakhshandehroo, M., Knoch, B., Müller, M., *et al*. Peroxisome proliferator-activated receptor alpha target genes. *PPAR Res*. (2010).
4. Ruscica, M., Busnelli, M., Runfola, E., *et al*. Impact of PPAR-Alpha polymorphisms-the case of metabolic disorders and atherosclerosis. *Int. J. Mol. Sci*. **20(18)**, 4378 (2019).
5. Guerre-Millo, M., Rouault, C., Poulain, P., *et al*. PPAR-α-null mice are protected from high-fat diet-induced insulin resistance. *Diabetes* **50(12)**, 2809-2814 (2001).

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