

LXR- α ^{GST}

Liver-X Receptor, α -isoform
human, recombinant, *E. coli*

Cat. No.	Amount
PR-805	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, 20% glycerol.

Activity

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

Purity

> 95% by SDS-PAGE

Description

Liver X Receptors (LXRs) are nuclear receptors that regulate the metabolism of cholesterol and bile acids. There are two subtypes of LXRs, LXR α and LXR β . LXR α is preferentially expressed in liver, small intestine, kidney and spleen. In contrast, LXR β expression is ubiquitous. The genomic structure and the promoter regions of the two LXR genes contain specific regulatory sites, which suggest that LXRs may have physiological roles in the immune system. Like other nuclear receptors, LXRs heterodimerize with Retinoid X Receptor (RXR) for function. LXRs are activated by naturally occurring oxysterols and regulate the expression of target genes, including ATP binding cassette transporter 1 (ABC1), ATP binding cassette transporter 8 (ABC8) and Cholesterol Ester Transfer Protein (CETP). LXR α is thought to play a major role in the control of cholesterol catabolism by stimulating the expression of cholesterol 7 α -hydroxylase (CYP7A1), the rate limiting enzyme of bile acid synthesis. Recombinant GST-LXR is isolated from an *E. coli* strain that carries the coding sequence of the human LXR α under the control of a T7 promoter.

GST-LXR has been applied in DNA and protein-protein interaction assays.

Selected References:

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- Janowski et al. (1999) Structural requirements of ligands for the oxysterol liver X receptors LXRalpha and LXRbeta. *Proc. Natl. Acad. Sci. USA* **96**:266.
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- Venkateswaran et al. (2000) Human white/murine ABC8 mRNA levels are highly induced in lipid-loaded macrophages. A transcriptional role for specific oxysterols. *J. Biol. Chem.* **275**:14700.

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Peet et al. (1998) Cholesterol and bile acid metabolism are impaired in mice lacking the nuclear oxysterol receptor LXR alpha. *Cell* **93**:693.