

## LXR- $\alpha$ -N182<sup>GST</sup>

Liver-X Receptor,  $\alpha$ -isoform, N-terminal 182 region

human, recombinant, *E. coli*

Cat. No.	Amount
PR-813	10 $\mu$ 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

GST-LXR $\alpha$ -N182 has been applied in DNA and protein-protein interaction assays.

#### 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 $\alpha$  and LXR $\beta$ .

LXR $\alpha$  is preferentially expressed in liver, small intestine, kidney and spleen. In contrast, LXR $\beta$  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 $\alpha$  is thought to play a major role in the control of cholesterol catabolism by stimulating the expression of cholesterol 7 $\alpha$ -hydroxylase (CYP7A1), the rate limiting enzyme of bile acid synthesis. Recombinant GST-LXR $\alpha$ -N182 is isolated from an *E. coli* strain that carries the coding sequence of the human LXR $\alpha$  from amino acids 1-182 under the control of a T7 promoter.

### Selected References:

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mice lacking the nuclear oxysterol receptor LXR alpha. *Cell* **93**:693.