

## ER $\alpha$

### Estrogen Receptor, $\alpha$ -isoform human, recombinant, Sf9 insect cells

Cat. No.	Amount
PR-756	4 $\mu$ 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

20-100 ng are sufficient for an *in vitro* transcription assay and 100 ng are sufficient for a protein-protein interactions assay.

#### Application

ER has been applied in DNA and protein-protein interaction assays.

#### Molecular Weight

64 kDa

#### Purity

> 90% by SDS-PAGE

#### Description

Recombinant His tagged ER was expressed in a baculovirus system and purified by an affinity column in combination with FPLC chromatography.

Several members of nuclear receptor family are directly associated with human malignancies including breast cancer, prostate cancer and leukemia. The pathogenesis of each of these diseases is underpinned by the activities of a member of the superfamily; Estrogen Receptor-alpha (ER $\alpha$ ) in breast cancer, Androgen Receptor (AR) in prostate cancer, and Retinoic Acid Receptor alpha (RAR $\alpha$ ) in acute promyelocytic leukemia. Estrogen receptors (ER) are members of the superfamily of nuclear hormone receptors whose activity is required for the normal function of the female reproductive system. Two isoforms of Estrogen Receptor (ER $\alpha$  and ER $\beta$ ) have been described. They function as ligand-dependent transcriptional activators. The biological functions downstream of ER result from altered expression of direct transcriptional targets as well as secondary effects mediated by biological activities of direct targets. In the mammary gland, estrogen receptors regulate normal epithelial cell development and differentiation through their well-documented effects on transcription. Estrogens have long been known to have mitogenic functions in breast cancer cell lines and in breast tumors. Selective Estrogen Receptor Modulatory compounds (SERMs), which bind directly to ER, can block the growth stimulatory function of estrogens.

#### Selected References:

- Hart (2002) Modulation of nuclear receptor dependent transcription. *Biol. Res.* **35**:295.  
Tsai *et al.* (1994) Molecular mechanisms of action of steroid/thyroid receptor superfamily members. *Annu. Rev. Biochem.* **63**:451.  
McDonnell *et al.* (2002) Connections and regulation of the human estrogen receptor. *Science* **296**:1642.  
Nilsson *et al.* (2001) *Physiol. Rev.* **81**:1535.  
Couse *et al.* (1999) Estrogen receptor null mice: what have we learned and where will they lead us? *Endocr. Rev.* **20**:358.  
Gruber *et al.* (2002) Production and actions of estrogens. *N. Engl. J. Med.* **346**:340.  
McDonnell *et al.* (2002) Elucidation of the molecular mechanism of action of selective estrogen receptor modulators. *Am. J. Cardiol.* **90**:35.