ER alpha/ESR1 Protein, Human (P. pastoris, N-His)

Cat. No.:	HY-P701106
Synonyms:	rHuEstrogen receptor/ER alpha, His; Estrogen Receptor; ER; ER-Alpha; Estradiol Receptor; Nuclear Receptor Subfamily 3 Group A Member 1; ESR1; ESR; NR3A1
Species:	Human
Source:	P. pastoris
Accession:	P03372-1 (S10-V595)
Gene ID:	2099
Molecular Weight:	Approximately 67.2 kDa

PROPERTIES

Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of Tris-based buffer, 50% glycerol.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O. For long term storage it is recommended to add a carrier protein (0.1% BSA, 5% HSA, 10% FBS or 5% Trehalose).
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background	ER alpha/ESR1 Protein, a nuclear hormone receptor, plays a pivotal role in the regulation of eukaryotic gene expression, influencing cellular proliferation and differentiation in target tissues. Ligand-dependent nuclear transactivation involves direct homodimer binding to palindromic estrogen response element (ERE) sequences or association with DNA-binding transcription factors like AP-1/c-Jun, c-Fos, ATF-2, Sp1, and Sp3, facilitating ERE-independent signaling. Upon ligand binding, ER alpha undergoes a conformational change, enabling subsequent association with multiprotein coactivator complexes through LXXLL motifs. Mutual transrepression occurs between ER alpha and NF-kappa-B in a cell-type specific manner, leading to decreased NF-kappa-B DNA-binding activity and inhibition of NF-kappa-B-mediated transcription. ER alpha is recruited to NF-kappa-B response elements, displacing coregulators and mediating transcriptional activation synergistically with NF-kappa-B. Moreover, ER alpha participates in membrane-initiated estrogen signaling through various kinase cascades, and is essential for MTA1-mediated transcriptional regulation of BRCA1 and BCAS3. Additionally, ER alpha is involved in the activation of NOS3 and endothelial nitric oxide production. Isoforms lacking specific functional domains are believed to modulate transcriptional activity through competitive ligand or DNA binding, as well as heterodimerization with the full-length receptor. Furthermore, ER alpha binds to ERE and inhibits isoform 1.

Caution: Product has not been fully validated for medical applications. For research use only.

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