

ER α Polyclonal Antibody
Catalog # AP69819**Specification****ER α Polyclonal Antibody - Product Information**

Application	WB
Primary Accession	P03372
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal

ER α Polyclonal Antibody - Additional Information**Gene ID** 2099**Other Names**

ESR1; ESR; NR3A1; Estrogen receptor; ER; ER-alpha; Estradiol receptor; Nuclear receptor subfamily 3 group A member 1

Dilution

WB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. ELISA: 1/20000. Not yet tested in other applications.

Format

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.

Storage Conditions

-20°C

ER α Polyclonal Antibody - Protein Information**Name** ESR1**Synonyms** ESR, NR3A1**Function**

Nuclear hormone receptor. The steroid hormones and their receptors are involved in the regulation of eukaryotic gene expression and affect cellular proliferation and differentiation in target tissues. Ligand-dependent nuclear transactivation involves either direct homodimer binding to a palindromic estrogen response element (ERE) sequence or association with other DNA-binding transcription factors, such as AP-1/c-Jun, c-Fos, ATF-2, Sp1 and Sp3, to mediate ERE- independent signaling. Ligand binding induces a conformational change allowing subsequent or combinatorial association with multiprotein coactivator complexes through LXXLL motifs of their respective components. Mutual transrepression occurs between the estrogen receptor (ER) and NF-kappa-B in a cell-type specific manner. Decreases NF-kappa- B DNA-binding activity and inhibits NF-kappa-B-mediated transcription from the IL6 promoter and displace RELA/p65 and associated coregulators from the promoter. Recruited to the NF-kappa-B response element of the CCL2 and IL8 promoters and can displace CREBBP. Present with NF-kappa-B components RELA/p65 and

NFKB1/p50 on ERE sequences. Can also act synergistically with NF-kappa-B to activate transcription involving respective recruitment adjacent response elements; the function involves CREBBP. Can activate the transcriptional activity of TFF1. Also mediates membrane-initiated estrogen signaling involving various kinase cascades. Essential for MTA1-mediated transcriptional regulation of BRCA1 and BCAS3 (PubMed:17922032). Maintains neuronal survival in response to ischemic reperfusion injury when in the presence of circulating estradiol (17-beta-estradiol/E2) (By similarity).

Cellular Location

[Isoform 1]: Nucleus {ECO:0000255|PROSITE- ProRule:PRU00407, ECO:0000269|PubMed:12682286, ECO:0000269|PubMed:20074560}. Cytoplasm. Cell membrane; Peripheral membrane protein; Cytoplasmic side. Note=A minor fraction is associated with the inner membrane Nucleus. Golgi apparatus. Cell membrane. Note=Colocalizes with ZDHHC7 and ZDHHC21 in the Golgi apparatus where most probably palmitoylation occurs. Associated with the plasma membrane when palmitoylated

Tissue Location

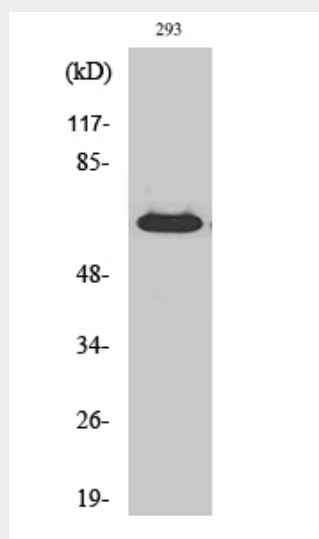
Widely expressed (PubMed:10970861). Not expressed in the pituitary gland (PubMed:10970861)

ER α Polyclonal Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

ER α Polyclonal Antibody - Images



ER α Polyclonal Antibody - Background

Nuclear hormone receptor. The steroid hormones and their receptors are involved in the regulation of eukaryotic gene expression and affect cellular proliferation and differentiation in target tissues. Ligand-dependent nuclear transactivation involves either direct homodimer binding to a palindromic estrogen response element (ERE) sequence or association with other DNA-binding transcription factors, such as AP-1/c-Jun, c-Fos, ATF-2, Sp1 and Sp3, to mediate ERE-independent signaling. Ligand binding induces a conformational change allowing subsequent or combinatorial association with multiprotein coactivator complexes through LXXLL motifs of their respective components. Mutual transrepression occurs between the estrogen receptor (ER) and NF- κ -B in a cell-type specific manner. Decreases NF- κ -B DNA-binding activity and inhibits NF- κ -B-mediated transcription from the IL6 promoter and displace RELA/p65 and associated coregulators from the promoter. Recruited to the NF- κ -B response element of the CCL2 and IL8 promoters and can displace CREBBP. Present with NF- κ -B components RELA/p65 and NFKB1/p50 on ERE sequences. Can also act synergistically with NF- κ -B to activate transcription involving respective recruitment adjacent response elements; the function involves CREBBP. Can activate the transcriptional activity of TFF1. Also mediates membrane-initiated estrogen signaling involving various kinase cascades. Isoform 3 is involved in activation of NOS3 and endothelial nitric oxide production. Isoforms lacking one or several functional domains are thought to modulate transcriptional activity by competitive ligand or DNA binding and/or heterodimerization with the full-length receptor. Essential for MTA1-mediated transcriptional regulation of BRCA1 and BCAS3. Isoform 3 can bind to ERE and inhibit isoform 1.