

# ER $\alpha$ Polyclonal Antibody

Catalog # AP69815

## Product Information

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|--------------------------|------------------------|
| <b>Application</b>       | WB, IHC-P              |
| <b>Primary Accession</b> | <a href="#">P03372</a> |
| <b>Reactivity</b>        | Human                  |
| <b>Host</b>              | Rabbit                 |
| <b>Clonality</b>         | Polyclonal             |
| <b>Calculated MW</b>     | 66216                  |

## Additional Information

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|---------------------------|---|
| <b>Gene ID</b>            | 2099  |
| <b>Other Names</b>        | ESR1; ESR; NR3A1; Estrogen receptor; ER; ER-alpha; Estradiol receptor; Nuclear receptor subfamily 3 group A member 1                    |
| <b>Dilution</b>           | WB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. ELISA: 1/20000. Not yet tested in other applications. IHC-P~~N/A |
| <b>Format</b>             | Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.   |
| <b>Storage Conditions</b> | -20°C   |

## Protein Information

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|-----------------|---|
| <b>Name</b>     | ESR1  |
| <b>Synonyms</b> | ESR, NR3A1  |
| <b>Function</b> | 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

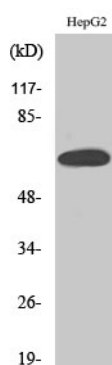
#### Tissue Location

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

## 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- 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. 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.

## Images



Western Blot analysis of various cells using ERα  
Polyclonal Antibody diluted at 1 : 500

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.