

Anti-Estrogen Receptor alpha antibody

Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP22453a

Product Information

Application	WB, E
Primary Accession	P03372
Reactivity	Human
Host	Rabbit
Clonality	polyclonal
Isotype	Rabbit Ig
Clone Names	R03099
Calculated MW	66216

Additional Information

Gene ID	2099
Other Names	Estrogen receptor, ER, ER-alpha, Estradiol receptor, Nuclear receptor subfamily 3 group A member 1, ESR1, ESR, NR3A1
Target/Specificity	This antibody is generated from a rabbit immunized with a KLH conjugated synthetic peptide between amino acids from human.
Dilution	WB~~1:1000 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.05% (V/V) Proclin 300. This antibody is purified through a protein A column, followed by peptide affinity purification.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	Anti-Estrogen Receptor alpha antibody is for research use only and not for use in diagnostic or therapeutic procedures.

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)

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. 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).

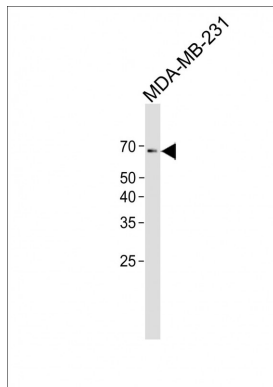
References

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Greene G.L.,et al.Science 231:1150-1154(1986).
Pink J.J.,et al.Nucleic Acids Res. 24:962-969(1996).

Bechtel S.,et al.BMC Genomics 8:399-399(2007).

Wang Z.,et al.Biochem. Biophys. Res. Commun. 336:1023-1027(2005).

Images



All lanes: Anti-Estrogen Receptor alpha antibody at 1:1000 dilution + MDA-MB-231 whole cell lysate
Lysates/proteins at 20 µg per lane. Secondary: Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated (ASP1615) at 1/15000 dilution. Observed band size: 66 KDa
Blocking/Dilution buffer: 5% NFDM/TBST.

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