Estrogen Receptor alpha (Phospho-Ser167) Antibody FITC Conjugated

Catalog No: #C00484F

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Description

Product Name	Estrogen Receptor alpha (Phospho-Ser167) Antibody FITC Conjugated
Host Species	Rabbit
Clonality	Polyclonal
Isotype	IgG
Purification	Purified by Protein A.
Applications	Flow-Cyt IF
Species Reactivity	Hu Rt
Immunogen Description	KLH conjugated synthetic phosphopeptide aa 150-175 595 derived from human Estrogen Receptor alpha around the phosphorylation site of Ser167
Conjugates	FITC
Target Name	Estrogen Receptor alpha Ser167
Other Names	ER; ESR; Era; ESRA; ESTRR; NR3A1; Estrogen receptor; ER-alpha; Estradiol receptor; Nuclear receptor subfamily 3 group A member 1; ESR1
Accession No.	Swiss-Prot#P03372NCBI Gene ID2099
Uniprot	P03372
GeneID	2099;
Excitation Emission	494nm 518nm
Cell Localization	Nucleus
Concentration	1mg ml
Formulation	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Storage	Shipped at 4°C. Store at -20°C for one year. Avoid repeated freeze/thaw cycles.

Application Details

Flow-Cyt=1:50-200 IF=1:50-200

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.

Note: This product is for in vitro research use only