

hHER4 Antibody (C-term Y1188)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP20287b**Specification**

hHER4 Antibody (C-term Y1188) - Product Information

Application	WB,E
Primary Accession	O15303
Other Accession	NP_001036064.1
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	146808
Antigen Region	1169-1194

hHER4 Antibody (C-term Y1188) - Additional Information**Gene ID** 2066**Other Names**

Receptor tyrosine-protein kinase erbB-4, Proto-oncogene-like protein c-ErbB-4, Tyrosine kinase-type cell surface receptor HER4, p180erbB4, ERBB4 intracellular domain, 4ICD, E4ICD, s80HER4, ERBB4, HER4

Target/Specificity

This hHER4 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 1169-1194 amino acids from the C-terminal region of human hHER4.

Dilution

WB~~1:2000

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. 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

hHER4 Antibody (C-term Y1188) is for research use only and not for use in diagnostic or therapeutic procedures.

hHER4 Antibody (C-term Y1188) - Protein Information**Name** ERBB4

Synonyms HER4

Function Tyrosine-protein kinase that plays an essential role as cell surface receptor for neuregulins and EGF family members and regulates development of the heart, the central nervous system and the mammary gland, gene transcription, cell proliferation, differentiation, migration and apoptosis. Required for normal cardiac muscle differentiation during embryonic development, and for postnatal cardiomyocyte proliferation. Required for normal development of the embryonic central nervous system, especially for normal neural crest cell migration and normal axon guidance. Required for mammary gland differentiation, induction of milk proteins and lactation. Acts as cell-surface receptor for the neuregulins NRG1, NRG2, NRG3 and NRG4 and the EGF family members BTC, EREG and HBEGF. Ligand binding triggers receptor dimerization and autophosphorylation at specific tyrosine residues that then serve as binding sites for scaffold proteins and effectors. Ligand specificity and signaling is modulated by alternative splicing, proteolytic processing, and by the formation of heterodimers with other ERBB family members, thereby creating multiple combinations of intracellular phosphotyrosines that trigger ligand- and context- specific cellular responses. Mediates phosphorylation of SHC1 and activation of the MAP kinases MAPK1/ERK2 and MAPK3/ERK1. Isoform JM-A CYT-1 and isoform JM-B CYT-1 phosphorylate PIK3R1, leading to the activation of phosphatidylinositol 3-kinase and AKT1 and protect cells against apoptosis. Isoform JM-A CYT-1 and isoform JM-B CYT-1 mediate reorganization of the actin cytoskeleton and promote cell migration in response to NRG1. Isoform JM-A CYT-2 and isoform JM-B CYT-2 lack the phosphotyrosine that mediates interaction with PIK3R1, and hence do not phosphorylate PIK3R1, do not protect cells against apoptosis, and do not promote reorganization of the actin cytoskeleton and cell migration. Proteolytic processing of isoform JM-A CYT-1 and isoform JM- A CYT-2 gives rise to the corresponding soluble intracellular domains (4ICD) that translocate to the nucleus, promote nuclear import of STAT5A, activation of STAT5A, mammary epithelium differentiation, cell proliferation and activation of gene expression. The ERBB4 soluble intracellular domains (4ICD) colocalize with STAT5A at the CSN2 promoter to regulate transcription of milk proteins during lactation. The ERBB4 soluble intracellular domains can also translocate to mitochondria and promote apoptosis.

Cellular Location

Cell membrane; Single-pass type I membrane protein. Note=In response to NRG1 treatment, the activated receptor is internalized

Tissue Location

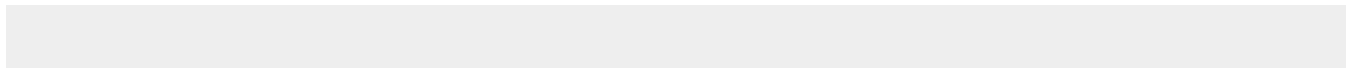
Expressed at highest levels in brain, heart, kidney, in addition to skeletal muscle, parathyroid, cerebellum, pituitary, spleen, testis and breast. Lower levels in thymus, lung, salivary gland, and pancreas. Isoform JM-A CYT-1 and isoform JM-B CYT-1 are expressed in cerebellum, but only the isoform JM-B is expressed in the heart.

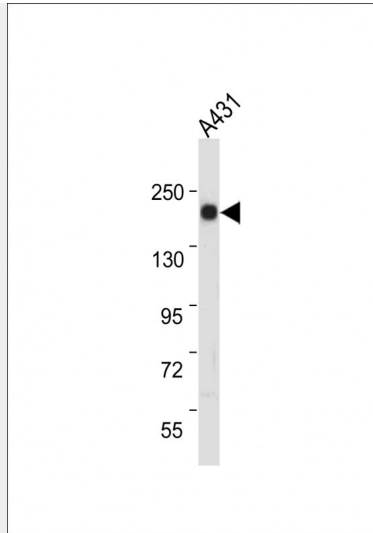
hHER4 Antibody (C-term Y1188) - Protocols

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](#)

hHER4 Antibody (C-term Y1188) - Images





Anti-hHER4 Antibody (C-term Y1188) at 1:2000 dilution + A431 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 147 kDa Blocking/Dilution buffer: 5% NFDM/TBST.

hHER4 Antibody (C-term Y1188) - Background

This gene is a member of the Tyr protein kinase family and the epidermal growth factor receptor subfamily. It encodes a single-pass type I membrane protein with multiple cysteine rich domains, a transmembrane domain, a tyrosine kinase domain, a phosphatidylinositol-3 kinase binding site and a PDZ domain binding motif. The protein binds to and is activated by neuregulins and other factors and induces a variety of cellular responses including mitogenesis and differentiation. Multiple proteolytic events allow for the release of a cytoplasmic fragment and an extracellular fragment. Mutations in this gene have been associated with cancer. Alternatively spliced variants which encode different protein isoforms have been described; however, not all variants have been fully characterized.

hHER4 Antibody (C-term Y1188) - References

Nicodemus, K.K., et al. Arch. Gen. Psychiatry 67(10):991-1001(2010)
Bailey, S.D., et al. Diabetes Care 33(10):2250-2253(2010)
Das, P.M., et al. Oncogene 29(37):5214-5219(2010)
Lu, C.L., et al. Neurosci. Lett. 481(2):120-125(2010)
Rokicki, J., et al. Mol. Cancer 9, 150 (2010) :