

ERBB4 Antibody(C-term)
 Purified Rabbit Polyclonal Antibody (Pab)
 Catalog # AP7631b-400 □

Specification

ERBB4 Antibody(C-term) - Product info

Application	WB, IHC-P
Primary Accession	Q15303
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit Ig
Clone Names	RB01472
Calculated MW	146808

ERBB4 Antibody(C-term) - Additional info

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 ERBB4 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 1276-1308 amino acids from the C-terminal region of human ERBB4.

Dilution

WB~~1:1000
 IHC-P~~1:50~100

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

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

ERBB4 Antibody(C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

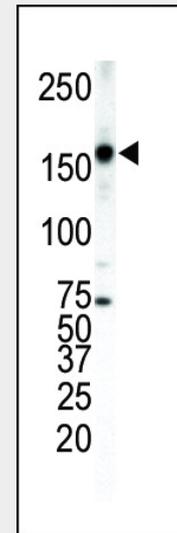
ERBB4 Antibody(C-term) - Protein Information

Name ERBB4

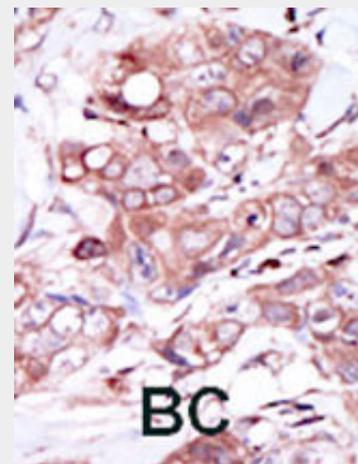
Synonyms HER4

Function

Tyrosine-protein kinase that plays an essential role as cell



Western blot analysis of anti-ErbB4 Pab (Cat. #AP7631b) in T-47D cell lysate. ErbB4 (arrow) was detected using purified Pab. Secondary HRP-anti-rabbit was used for signal visualization with chemiluminescence.



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.

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.

ERBB4 Antibody(C-term) - 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](#)

ERBB4 Antibody(C-term) - Background

Protein kinases are enzymes that transfer a phosphate group from a phosphate donor, generally the γ phosphate of ATP, onto an acceptor amino acid in a substrate protein. By this basic mechanism, protein kinases mediate most of the signal transduction in eukaryotic cells, regulating cellular metabolism, transcription, cell cycle progression, cytoskeletal rearrangement and cell movement, apoptosis, and differentiation. With more than 500 gene products, the protein kinase family is one of the largest families of proteins in eukaryotes. The family has been classified in 8 major groups based on sequence comparison of their tyrosine (PTK) or serine/threonine (STK) kinase catalytic domains.

ERBB4 Antibody(C-term) - References

Cheng, Q.C., et al., *J. Biol. Chem.* 278(40):38421-38427 (2003).
Komuro, A., et al., *J. Biol. Chem.* 278(35):33334-33341 (2003).
Williams, E.E., et al., *Cancer Lett.* 192(1):67-74 (2003). Thomas, C.Y., et al., *Int. J. Cancer* 104(1):19-27 (2003). Ni, C.Y., et al., *J. Biol. Chem.* 278(7):4561-4565 (2003).