

**EGFR (ErbB1) Antibody (C-term) Blocking peptide**

Synthetic peptide

Catalog # BP7628b

**Specification**

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**EGFR (ErbB1) Antibody (C-term) Blocking peptide - Product Information**

Primary Accession

[P00533](#)

Other Accession

[EGFR\\_HUMAN](#)**EGFR (ErbB1) Antibody (C-term) Blocking peptide - Additional Information**

Gene ID 1956

**Other Names**

Epidermal growth factor receptor, Proto-oncogene c-ErbB-1, Receptor tyrosine-protein kinase erbB-1, EGFR, ERBB, ERBB1, HER1

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP7628b](/product/products/AP7628b) was selected from the C-term region of human ErbB1 . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**EGFR (ErbB1) Antibody (C-term) Blocking peptide - Protein Information**Name EGFR ([HGNC:3236](#))

Synonyms ERBB, ERBB1, HER1

**Function**

Receptor tyrosine kinase binding ligands of the EGF family and activating several signaling cascades to convert extracellular cues into appropriate cellular responses (PubMed:[2790960](http://www.uniprot.org/citations/2790960), PubMed:[10805725](http://www.uniprot.org/citations/10805725), PubMed:[27153536](http://www.uniprot.org/citations/27153536)). Known ligands include EGF, TGFA/TGF-alpha, AREG, epigen/EPGN, BTC/betacellulin, epiregulin/EREG and HBEGF/heparin-binding EGF (PubMed:[2790960](http://www.uniprot.org/citations/2790960), PubMed:[7679104](http://www.uniprot.org/citations/7679104))

target="\_blank">7679104</a>, PubMed:<a href="http://www.uniprot.org/citations/8144591" target="\_blank">8144591</a>, PubMed:<a href="http://www.uniprot.org/citations/9419975" target="\_blank">9419975</a>, PubMed:<a href="http://www.uniprot.org/citations/15611079" target="\_blank">15611079</a>, PubMed:<a href="http://www.uniprot.org/citations/12297049" target="\_blank">12297049</a>, PubMed:<a href="http://www.uniprot.org/citations/27153536" target="\_blank">27153536</a>, PubMed:<a href="http://www.uniprot.org/citations/20837704" target="\_blank">20837704</a>, PubMed:<a href="http://www.uniprot.org/citations/17909029" target="\_blank">17909029</a>). Ligand binding triggers receptor homo- and/or heterodimerization and autophosphorylation on key cytoplasmic residues. The phosphorylated receptor recruits adapter proteins like GRB2 which in turn activates complex downstream signaling cascades. Activates at least 4 major downstream signaling cascades including the RAS-RAF-MEK-ERK, PI3 kinase-AKT, PLCgamma-PKC and STATs modules (PubMed:<a href="http://www.uniprot.org/citations/27153536" target="\_blank">27153536</a>). May also activate the NF-kappa-B signaling cascade (PubMed:<a href="http://www.uniprot.org/citations/11116146" target="\_blank">11116146</a>). Also directly phosphorylates other proteins like RGS16, activating its GTPase activity and probably coupling the EGF receptor signaling to the G protein-coupled receptor signaling (PubMed:<a href="http://www.uniprot.org/citations/11602604" target="\_blank">11602604</a>). Also phosphorylates MUC1 and increases its interaction with SRC and CTNNB1/beta-catenin (PubMed:<a href="http://www.uniprot.org/citations/11483589" target="\_blank">11483589</a>). Positively regulates cell migration via interaction with CDC88A/GIV which retains EGFR at the cell membrane following ligand stimulation, promoting EGFR signaling which triggers cell migration (PubMed:<a href="http://www.uniprot.org/citations/20462955" target="\_blank">20462955</a>). Plays a role in enhancing learning and memory performance (By similarity). Plays a role in mammalian pain signaling (long-lasting hypersensitivity) (By similarity).

#### Cellular Location

Cell membrane; Single-pass type I membrane protein. Endoplasmic reticulum membrane; Single-pass type I membrane protein. Golgi apparatus membrane; Single-pass type I membrane protein. Nucleus membrane; Single-pass type I membrane protein Endosome Endosome membrane. Nucleus. Note=In response to EGF, translocated from the cell membrane to the nucleus via Golgi and ER (PubMed:20674546, PubMed:17909029). Endocytosed upon activation by ligand (PubMed:2790960, PubMed:17182860, PubMed:27153536, PubMed:17909029). Colocalized with GPER1 in the nucleus of estrogen agonist-induced cancer-associated fibroblasts (CAF) (PubMed:20551055)

#### Tissue Location

Ubiquitously expressed. Isoform 2 is also expressed in ovarian cancers.

#### EGFR (ErbB1) Antibody (C-term) Blocking peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

#### EGFR (ErbB1) Antibody (C-term) Blocking peptide - Images

#### EGFR (ErbB1) Antibody (C-term) Blocking peptide - Background

Protein kinases are enzymes that transfer a phosphate group from a phosphate donor, generally the gamma 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.

### **EGFR (ErbB1) Antibody (C-term) Blocking peptide - References**

Zanardi, T.A., et al., J. Virol. 77(21):11685-11696 (2003).Krug, A.W., et al., J. Biol. Chem. 278(44):43060-43066 (2003).Huang, F., et al., J. Biol. Chem. 278(44):43411-43417 (2003).He, Y.Y., et al., J. Biol. Chem. 278(43):42457-42465 (2003).Hirsch, F.R., et al., J. Clin. Oncol. 21(20):3798-3807 (2003).