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Recombinant Human EGFR Protein (Isoform vIII, His tag)

Catalog Number:	632701, 632702
Size:	100 μg, 1 mg
Target Name:	EGFR, Proto-oncogene c-ErbB-1, Receptor tyrosine-protein kinase erbB-1, HER1
Regulatory Status:	RUO

PRODUCT DETAILS

Application:	Bioassay
Format:	Lyophilized from sterile PBS, pH 7.4.
Expression Host:	HEK293
Species:	Human
accession number:	NP_001333870.1
Sources:	A DNA sequence encoding the human EGFR (NP_001333870.1, Isoform vIII) (Met1-Ser378) was expressed with a polyhistidine tag at the C-terminus.
Molecular Weight:	The recombinant human EGFR consists of 365 amino acids and predicts a molecular mass of 40.09 KDa. It migrates as an approximately 65.27 KDa band in SDS-PAGE under reducing conditions. The molecular weight of this protein is around 58.8 KDa verified by SEC-MALS(Routinely tested).
Affinity Tag:	C-His
Purity:	≥85% as determined by SDS-PAGE. ≥95% as determined by SEC-HPLC. ≥95% as determined by SEC-MALS (Routinely tested).
Endotoxin level:	< 1.0 EU per μg protein
Protein Concentration:	Lyophilized
Storage and Handling:	Samples are stable for up to twelve months from date of receipt at -20℃ to -80℃. Store it under sterile conditions at -20℃ to -80℃. It is recommended that the protein be aliquoted for optimal storage. Avoid repeated freeze-thaw cycles.

BACKGROUND INFORMATION

The epidermal growth factor receptor (EGFR), also known as ErbB1 or HER1, is a transmembrane receptor tyrosine kinase that plays a central role in regulating cell growth, survival, differentiation, and migration. EGFR is a member of the ErbB family of receptors and is expressed in many epithelial, mesenchymal, and neural tissues.

Under normal physiological conditions, EGFR signaling is tightly controlled and essential for tissue development, maintenance, and repair. EGFR is activated through the binding of specific ligands to its extracellular domain. Key ligands include epidermal growth factor (EGF), transforming growth factor-α (TGF-α), amphiregulin, epiregulin, betacellulin, and heparin-binding EGF-like growth factor (HB-EGF). Ligand binding induces receptor dimerization, either as EGFR homodimers or heterodimers with other ErbB family

members such as ErbB2 (HER2). Dimerization triggers autophosphorylation of tyrosine residues within the intracellular kinase domain, creating docking sites for adaptor proteins and initiating downstream signaling cascades. Major pathways activated by EGFR include the MAPK/ERK, PI3K/AKT, and JAK/STAT pathways, which collectively regulate proliferation, survival, and gene expression.

Dysregulation of EGFR signaling is strongly associated with human disease, particularly cancer. EGFR can become aberrantly activated through overexpression, gene amplification, activating mutations, or autocrine ligand production. Such alterations drive uncontrolled cell proliferation and resistance to apoptosis. EGFR dysregulation is commonly observed in cancers of the lung, colorectal tract, head and neck, pancreas, and glioblastoma. Specific activating mutations in EGFR, such as exon 19 deletions or the L858R point mutation, are especially prevalent in subsets of non-small cell lung cancer and are associated with sensitivity to targeted therapies. Beyond cancer, EGFR signaling has also been implicated in inflammatory diseases, tissue fibrosis, and abnormal wound healing.

Because of its central role in disease pathogenesis, EGFR is an important therapeutic target. Multiple classes of EGFR-targeted therapies have been developed, including small-molecule tyrosine kinase inhibitors (TKIs) and monoclonal antibodies that block ligand binding or receptor dimerization. EGFR inhibitors have demonstrated significant clinical benefit in selected patient populations, particularly those with EGFR-driven tumors, making EGFR one of the most extensively studied and successfully targeted receptors in modern precision medicine.

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