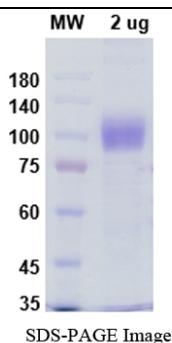


Product Details

Summary

English name	Recombinant Human EGFR protein ,C- His tag
Purity	>90% as determined by SDS-PAGE
Endotoxin level	<1.0 EU per µg of the protein as determined by the LAL method.
Construction	A DNA sequence encoding the human EGFR(Met1-Ser645) was fused with the C-terminal His tag
Accession #	P00533
Host	Mammalian cells
Species	Homo sapiens (Human)
Predicted Molecular Mass	70.95kDa
Formulation	Supplied as solution form in PBS pH 7.5 or lyophilized from PBS pH 7.5.
Shipping	In general, proteins are provided as lyophilized powder/frozen liquid. They are shipped out with dry ice/blue ice unless customers require otherwise.
Stability &Storage	Use a manual defrost freezer and avoid repeated freeze thaw cycles. Store at 2 to 8 °C for one week . Store at -20 to -80 °C for twelve months from the date of receipt.
Reconstitution	Reconstitute in sterile water for a stock solution. A copy of datasheet will be provided with the products, please refer to it for details.

SDS-PAGE image



Background

Background	The epidermal growth factor receptor (EGFR; ErbB-1; HER1 in humans) is the cell-surface receptor for members of the epidermal growth factor family (EGF-family) of
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extracellular protein ligands. The epidermal growth factor receptor is a member of the ErbB family of receptors, a subfamily of four closely related receptor tyrosine kinases: EGFR (ErbB-1), HER2/c-neu (ErbB-2), Her 3 (ErbB-3) and Her 4 (ErbB-4). Mutations affecting EGFR expression or activity could result in cancer.

Alternative Names

EGFR,ERBB,ERBB1,HER1,PIG61,mENA

References

Ouyang, Wang, Yao, Zhang (2020) Elevated CELSR3 expression is associated with hepatocarcinogenesis and poor prognosis Oncology letters 20(2) 1083-1092

Frontier progress

Cadherin EGF LAG seven-pass G-type receptor 3 (CELSR3) has been reported to exhibit a cancer-specific pattern. The present study aimed to investigate the clinical value and functional role of CELSR3 in hepatocellular carcinoma (HCC), and determine the underlying molecular mechanism in patients with HCC. CELSR3 expression in tumor and paracancerous HCC tissues was obtained from The Cancer Genome Atlas. Differential expression analysis was performed using the edgeR package. Pearson correlation analysis was used to analyze the correlation between methylation and mRNA levels of CELSR3. Pathways affected by aberrant CELSR3 expression were identified through Gene Set Enrichment Analysis. The results demonstrated that CELSR3 was highly expressed in the early stage of cancer and was present throughout the entire cancer process, which suggested that CELSR3 may serve a key role in the carcinogenesis of HCC. In addition, upregulation of CELSR3 was associated with its methylation level; high CELSR3 expression indicated a shorter overall survival time. Multiple candidate genes were screened by integrating differentially expressed (DE) mRNAs and target genes of DE microRNAs (miRs). Subsequent pathway enrichment analysis demonstrated that the upregulated genes were predominantly enriched in the 'Neuroactive ligand-receptor interaction' and 'Cell cycle' pathways, whereas the downregulated genes were primarily enriched in 'Cytokine-cytokine receptor interaction' and 'Metabolic pathways'. CELSR3 and its connected nodes and edges were initially removed from the miRNA-mRNA regulatory network in order to prevent bias and compared with the network containing CELSR3 alone. The frequently dysregulated miRNAs were identified as miR-181 family members, and the results suggested that miR-181 and the Wnt/ β -catenin signaling pathway influenced CELSR3 expression.