ATAGENIX LABORATORIES

Catalog Number:ATMP00505HU

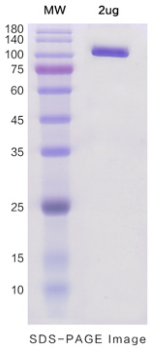
Recombinant Human ERBB3 protein ,C- His Tag

Product Details

Summary

English name	Recombinant Human ERBB3 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 ERBB3(Met1-Thr643) was fused with the C-terminal His Tag
Accession #	P21860
Host	Mammalian cells
Species	Homo sapiens (Human)
Predicted Molecular Mass	70.73kDa
Formulation	Supplied as solution form in PBS or lyophilized from PBS .
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



SDS-PAGE Image

Background

Background	ErbB3,also known as Her3 (human epidermal growth factor receptor 3), is a member of the epidermal growth factor receptor (EGFR) family of receptor tyrosine

kinases. This membrane-bound glycoprotein has a neuregulin binding domain but has not an active kinase domain. It therefore can bind the ligand but cannot mediate the intracellular signal transduction through protein phosphorylation. However, it does form heterodimers with ErbB2 or other EGFR members responsible for tyrosine phosphorylation to give a receptor complex and initiate the related pathway, which lead to cell proliferation or differentiation. Overexpression of this protein has been reported in numerous cancers, including prostate, bladder, and breast tumors. This protein has different isoforms derived from alternative splicing variants, and among which, the secreted isoform lacking the intermembrane region modulates the activity of membrane-bound form.

Alternative Names ERBB3,HER3,LCCS2,MDA-BF-1,MGC88033,c-erbB3,erbB3-S,p180-ErbB3,p45-sErbB3,p85-sErbB3

References Yang, Arai, Takahashi, Totsuka, Chiku, Taniguchi, Katai, Sakamoto, Yoshida, Kanai (2020) Cooperative participation of epigenomic and genomic alterations in the clinicopathological diversity of gastric adenocarcinomas: significance of cell adhesion and epithelial-mesenchymal transition-related signaling pathways Carcinogenesis ()

Frontier progress

The present study was conducted to clarify the cooperative significance of epigenomic and genomic abnormalities during gastric carcinogenesis. Using 21 samples of normal control gastric mucosa (C), 109 samples of non-cancerous gastric mucosa (N) and 105 samples of cancerous tissue (T) from 109 patients with primary gastric adenocarcinomas, genome-wide DNA methylation analysis was performed using Infinium assay. Among these samples, 66 paired N and corresponding T samples were subjected to whole-exome and single nucleotide polymorphism array analyses. As had been shown in our previous study, 109 patients were clustered clinicopathologically into least aggressive Cluster A (n=20), most aggressive Cluster B1 (n=20), and Cluster B2 (n=69). Most DNA methylation alterations in each cluster had already occurred even in N samples compared to C samples, and DNA methylation alterations at the precancerous N stage were inherited by the established cancers themselves. Recurrent single-nucleotide variants and insertions/deletions resulting in functional disruption of the proteins encoded by the ABCA10, BNC2, CDH1, CTNNB1, SMAD4 and VAV2 genes were specific to Cluster B1, whereas those of the APC, EGFR, ERBB2, ERBB3, MLH1 and MUC6 genes were specific to Cluster A. MetaCore pathway analysis revealed that the epigenomically affected TWIST1 gene and genomically affected CDH1, CTNNB1, MMP9, TLN2, ROCK1 and SMAD4 genes were accumulated in signaling pathways related to cell adhesion, cytoskeleton remodeling and epithelial-mesenchymal transition in Cluster B1. These data indicate that epigenomic alterations at the precancerous stage are important in gastric

carcinogenesis and that epigenomic and genomic alterations cooperatively underlie the aggressiveness of gastric adenocarcinomas.