Product Data Sheet

Product Name: Epidermal Growth Factor Receptor Peptide (985-996)
Cat. No.: GP10095

Chemical Properties

Cas. No.	96249-43-3		
Chemical Name	5-amino-1-cyclopropyl-6,8-difluoro-7-(4-methylpiperazin-1-yl)-4-oxoquinoline-3-carboxylic acid		
SMILES	CN1CCN(CC1)C2=C(C3=C(C(=C2F)N)C(=O)C(=CN3C4CC4)C(=O)O)F		
Formula	$C_{61}H_{93}N_{13}O_{23}$	M.Wt	1376.46
Solubility	≥ 137.6mg/mL in DMSO	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure			

Background

EGFR (epidermal growth factor receptor) exists on the cell surface and is activated by the binding of its specific ligands, including epidermal growth factor and transforming growth factor α. EGFR dimerization stimulates its intrinsic intracellular protein-tyrosine kinase activity. As a result, autophosphorylation of several tyrosine (Y) residues on the C-terminal domain of EGFR occurs. These include Y992, Y1045, Y1068, Y1148 and Y1173. This autophosphorylation elicits downstream activation and signaling by several other proteins that associate with the phosphorylated tyrosines through their own phosphotyrosine-binding SH2 domains. These downstream signaling proteins initiate several signal transduction cascades, principally the MAPK, Akt and JNK pathways, which lead to DNA synthesis and cell proliferation. Such proteins modulate phenotypes such as cell migration, adhesion, and proliferation. Activation of the receptor is important for the innate immune response in human skin.

Many therapeutic approaches are aimed at EGFR. The monoclonal antibodies block the

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA

extracellular ligand binding domain. With the binding site blocked, signal molecules can no longer attach and activate the tyrosine kinase. Another therapeutic method involves using small molecules to inhibit the EGFR tyrosine kinase on the cytoplasmic side of the receptor. Without kinase activity, EGFR is unable to activate itself, which is a prerequisite for the binding of downstream adaptor proteins. Ostensibly by halting the signaling cascade in cells that rely on this pathway for growth, tumor proliferation and migration is diminished

References:

1. Yan. L, Beckman RA (October 2005). "Pharmacogenetics and pharmacogenomics in oncology therapeutic antibody development". BioTechniques 39 (4): 565–8.
2. Yosef Yarden and Joseph Schlessinger (1987). "Epidermal Growth-Factor Induces Rapid, Reversible Aggregation of the Purified Epidermal Growth-Factor Receptor". Biochemistry 26 (5): 1443–1451.
3. Downward J, Parker P, Waterfield MD (1984). "Autophosphorylation sites on the epidermal growth factor receptor". Nature 311 (5985): 483–5.
4. Oda K, Matsuoka Y, Funahashi A, Kitano H (2005). "A comprehensive pathway map of epidermal growth factor receptor signaling". Mol. Syst. Biol. 1 (1): 2005.0010.
5. David K. Moscatello2, Marina Holgado-Madruga2. "Frequent Expression of a Mutant Epidermal Growth Factor. Receptor in Multiple Human Tumors". Cancer Res December 1, 1995 55; 5536.