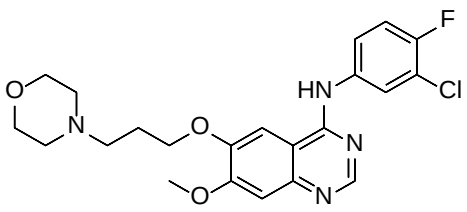
Product data sheet

MedKoo Cat#: 100140 Name: Gefitinib CAS#: 184475-35-2 Chemical Formula: $C_{22}H_{24}ClFN_4O_3$ Exact Mass: 446.1521 Molecular Weight: 446.90	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:
Gefitinib, also known as ZD1839, is an anilinoquinazoline with antineoplastic activity. Gefitinib inhibits the catalytic activity of numerous tyrosine kinases including the epidermal growth factor receptor (EGFR), which may result in inhibition of tyrosine kinase-dependent tumor growth. Specifically, this agent competes with the binding of ATP to the tyrosine kinase domain of EGFR, thereby inhibiting receptor autophosphorylation and resulting in inhibition of signal transduction. Gefitinib may also induce cell cycle arrest and inhibit angiogenesis. It is marketed by AstraZeneca and Teva.

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMF	20.0	44.75
DMSO	50.92	113.94
DMSO:PBS (pH 7.2) (1:1)	0.50	1.12
Ethanol	2.92	6.53

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.24 mL	11.19 mL	22.38 mL
5 mM	0.45 mL	2.24 mL	4.48 mL
10 mM	0.22 mL	1.12 mL	2.24 mL
50 mM	0.04 mL	0.22 mL	0.45 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study
In vitro study

1. Takenaka T, Nakai S, Katayama M, Hirano M, Ueno N, Noguchi K, Takatani-Nakase T, Fujii I, Kobayashi SS, Nakase I. Effects of gefitinib treatment on cellular uptake of extracellular vesicles in EGFR-mutant non-small cell lung cancer cells. Int J Pharm. 2019 Dec 15;572:118762. doi: 10.1016/j.ijpharm.2019.118762. Epub 2019 Oct 11. PMID: 31610280; PMCID: PMC6899172.
2. Wu Y, He Z, Li S, Tang H, Wang L, Yang S, Dong B, Qin J, Sun Y, Yu H, Zhang Y, Zhang Y, Guo Y, Wang Q. Gefitinib Represses JAK-STAT Signaling Activated by CRTC1-MAML2 Fusion in Mucoepidermoid Carcinoma Cells. Curr Cancer Drug Targets. 2019;19(10):796-806. doi: 10.2174/1568009619666190103122735. PMID: 30605061.

In vivo study

1. Tariq M, Zhang JQ, Liang GK, He QJ, Ding L, Yang B. Gefitinib inhibits M2-like polarization of tumor-associated macrophages in Lewis lung cancer by targeting the STAT6 signaling pathway. Acta Pharmacol Sin. 2017 Nov;38(11):1501-1511. doi: 10.1038/aps.2017.124. Epub 2017 Oct 12. PMID: 29022575; PMCID: PMC5672074.

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7. Bioactivity

Biological target: EGFR tyrosine kinase inhibitor with IC50 of 33 nM.

In vitro activity

Gefitinib extensively inhibited transcription of genes in JAK-STAT and MAPK/ERK pathways. Both siC1-M2 and gefitinib inhibited the phosphorylation of multiple signaling kinases in these signaling pathways, indicating that gefitinib inhibited JAK-STAT and MAPK/ERK pathways activated by C1-M2 fusion. These results suggest that gefitinib simultaneously represses phosphorylation of multiple key signaling proteins which are activated in MEC, in part by C1-M2 fusion. Gefitinib-repressed kinase phosphorylation explains the transcriptional repression of genes in JAK-STAT and MAPK/ERK pathways.

Reference: Curr Cancer Drug Targets. 2019;19(10):796-806. https://www.eurekaselect.com/168737/article

In vivo activity

M2-like polarized tumor-associated macrophages (TAMs) play a pivotal role in promoting cancer cell growth, invasion, metastasis and angiogenesis. The relevance of macrophage polarization and the antitumor effect of gefitinib in Lewis Lung cancer (LLC) was investigated in vivo. In LLC mice metastasis model, oral administration of gefitinib (75 mg·kg-1·d-1, for 21 d) significantly reduced the number of lung metastasis nodules, down-regulated the expression of M2 marker genes and the percentages CD206+ and CD68+ macrophages in tumor tissues. These results demonstrated that gefitinib effectively inhibits M2-like polarization in vivo, revealing a novel potential mechanism for the chemopreventative effect of gefitinib.

Reference: Acta Pharmacol Sin. 2017 Nov;38(11):1501-1511. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5672074/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.