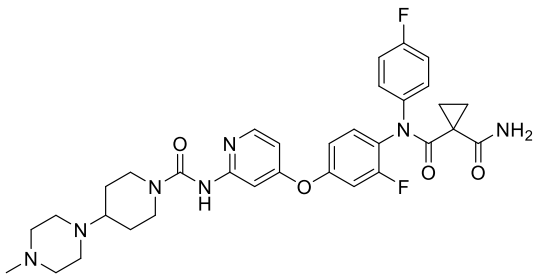
Product data sheet

MedKoo Cat#: 201072 Name: Golvatinib CAS#: 928037-13-2 Chemical Formula: $C_{33}H_{37}F_2N_7O_4$ Exact Mass: 633.28751 Molecular Weight: 633.69	
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:
Golvatinib, also known as E7050, is an orally bioavailable dual kinase inhibitor of c-Met (hepatocyte growth factor receptor) and VEGFR-2 (vascular endothelial growth factor receptor-2) tyrosine kinases with potential antineoplastic activity. c-Met/VEGFR kinase inhibitor E7050 binds to and inhibits the activities of both c-Met and VEGFR-2, which may inhibit tumor cell growth and survival of tumor cells that overexpress these receptor tyrosine kinases. c-Met and VEGFR-2 are upregulated in a variety of various tumor cell types and play important roles in tumor cell growth, migration and angiogenesis.

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
DMSO	35.0	55.23

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	1.58 mL	7.89 mL	15.78 mL
5 mM	0.32 mL	1.58 mL	3.16 mL
10 mM	0.16 mL	0.79 mL	1.58 mL
50 mM	0.03 mL	0.16 mL	0.32 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. Wang W, Li Q, Takeuchi S, Yamada T, Koizumi H, Nakamura T, Matsumoto K, Mukaida N, Nishioka Y, Sone S, Nakagawa T, Uenaka T, Yano S. Met kinase inhibitor E7050 reverses three different mechanisms of hepatocyte growth factor-induced tyrosine kinase inhibitor resistance in EGFR mutant lung cancer. Clin Cancer Res. 2012 Mar 15;18(6):1663-71. doi: 10.1158/1078-0432.CCR-11-1171. Epub 2012 Feb 8. PMID: 22317763.

In vivo study

1. Wang W, Li Q, Takeuchi S, Yamada T, Koizumi H, Nakamura T, Matsumoto K, Mukaida N, Nishioka Y, Sone S, Nakagawa T, Uenaka T, Yano S. Met kinase inhibitor E7050 reverses three different mechanisms of hepatocyte growth factor-induced tyrosine kinase inhibitor resistance in EGFR mutant lung cancer. Clin Cancer Res. 2012 Mar 15;18(6):1663-71. doi: 10.1158/1078-0432.CCR-11-1171. Epub 2012 Feb 8. PMID: 22317763.
2. Nakagawa T, Tohyama O, Yamaguchi A, Matsushima T, Takahashi K, Funasaka S, Shirotori S, Asada M, Obaishi H. E7050: a dual c-Met and VEGFR-2 tyrosine kinase inhibitor promotes tumor regression and prolongs survival in mouse xenograft models. Cancer Sci. 2010 Jan;101(1):210-5. doi: 10.1111/j.1349-7006.2009.01343.x. Epub 2009 Sep 2. PMID: 19832844.

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7. Bioactivity

Biological target:

Golvatinib (E-7050) is a potent dual inhibitor of both c-Met and VEGFR2 kinases with IC50s of 14 and 16 nM, respectively.

In vitro activity

The new Met-TKI, E7050, reversed 3 HGF-induced resistance mechanisms in EGFR mutant lung cancer. First, E7050 reversed HGF-induced gefitinib resistance by inhibiting Met phosphorylation and thereby suppressing the downstream PI3K/Akt pathway. Second, E7050 inhibited the HGF-induced resistance to next-generation EGFR-TKIs, irreversible EGFR-TKIs, and mutant-selective EGFR-TKIs. Third, E7050 prevented the emergence of resistant clones induced by continuous exposure to HGF.

Reference: Clin Cancer Res. 2012 Mar 15;18(6):1663-71. https://clincancerres.aacrjournals.org/content/18/6/1663.long

In vivo activity

Daily oral administration of E7050 inhibited the growth of all tumors in a dose-dependent manner (Fig. 2). High doses of E7050 caused drastic tumor regression, with 2/5 Hs746T tumors failing to re-grow after E7050 treatment (50 mg/kg) was terminated for 20 days and 5/5 failing to re-grow after 100 mg/kg E7050 treatment (data not shown). As a result, tumor-bearing mice were cured by treatment with E7050. During the treatment with E7050, no other macroscopic changes or loss of body weight were observed (data not shown).

Reference: Cancer Sci. 2010 Jan;101(1):210-5. https://onlinelibrary.wiley.com/doi/full/10.1111/j.1349-7006.2009.01343.x

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.