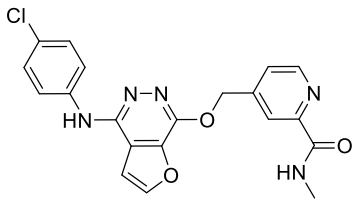
Product data sheet

MedKoo Cat#: 206424 Name: Telatinib CAS#: 332012-40-5 Chemical Formula: $C_{20}H_{16}ClN_5O_3$ Exact Mass: 409.09417 Molecular Weight: 409.83	
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:
Telatinib, also known as BAY 57-9352, is a potent and orally active VEGFR-2 and VEGFR-3 tyrosine kinase inhibitor. Telatinib reverses chemotherapeutic multidrug resistance mediated by ABCG2 efflux transporter in vitro and in vivo. Telatinib treatment was well tolerated. The observed single agent antitumor activity in heavily pretreated CRC patients was limited. Pharmacodynamic results are suggestive for the biological activity of telatinib justifying a further evaluation of telatinib bid in combination with standard chemotherapy regimens in CRC patients.

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	44.33	108.17
DMSO:PBS (pH 7.2) (1:2)	0.33	0.81
DMF	5.0	12.20

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.44 mL	12.20 mL	24.40 mL
5 mM	0.49 mL	2.44 mL	4.88 mL
10 mM	0.24 mL	1.22 mL	2.44 mL
50 mM	0.05 mL	0.24 mL	0.49 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. Sodani K, Patel A, Anreddy N, Singh S, Yang DH, Kathawala RJ, Kumar P, Talele TT, Chen ZS. Telatinib reverses chemotherapeutic multidrug resistance mediated by ABCG2 efflux transporter in vitro and in vivo. Biochem Pharmacol. 2014 May 1;89(1):52-61. doi: 10.1016/j.bcp.2014.02.012. Epub 2014 Feb 22. PMID: 24565910; PMCID: PMC3983711.

In vivo study

1. Sodani K, Patel A, Anreddy N, Singh S, Yang DH, Kathawala RJ, Kumar P, Talele TT, Chen ZS. Telatinib reverses chemotherapeutic multidrug resistance mediated by ABCG2 efflux transporter in vitro and in vivo. Biochem Pharmacol. 2014 May 1;89(1):52-61. doi: 10.1016/j.bcp.2014.02.012. Epub 2014 Feb 22. PMID: 24565910; PMCID: PMC3983711.

7. Bioactivity
Biological target:

Product data sheet

Telatinib (Bay 57-9352) is an inhibitor of VEGFR2, VEGFR3, PDGFα, and c-Kit with IC50s of 6, 4, 15 and 1 nM, respectively.

In vitro activity

Telatinib increased the accumulation of MX and decreased the efflux rate in cells transfected with ABCG2. Furthermore, telatinib did not affect either expression or localization of ABCG2. However, effect of telatinib on localization still needs to be confirmed by more sensitive means. It indicates that increase in MX levels in the cells expressing ABCG2 is because of inhibition of ABCG2 drug efflux function by telatinib. Telatinib stimulated the ABCG2 ATPase activity in a concentration dependent manner. In addition telatinib effectively reduced the ATP-dependent uptake of [^{3}H]-E217βG in membrane vesicles obtained from ABCG2-482-R2 without affecting the uptake in the parental HEK293/pcDNA3.1 vesicles. This shows the ability of telatinib to directly interact with the ABCG2 membrane transporter and thereafter inhibiting its transport activity at sub-micromolar concentrations.

Reference: Biochem Pharmacol. 2014 May 1; 89(1): 52–61. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3983711/

In vivo activity

The H460/MX20 tumor growth rate recorded over a period of 18 days was significantly slow in the telatinib-DOX combination group compared to vehicle, telatinib alone or DOX alone groups (Fig. 6B). In addition, telatinib in combination with DOX also produced a significant reduction in tumor size and weight (Fig. 6D and 7B). It should be noted that telatinib by itself also significantly decreased the growth rate of H460 and H460/MX20 xenografts. However, there was no significant difference between the effects of telatinib alone or combination on the H460 xenografts (Fig. 6A, 6C and and7A).7A). DOX with or without telatinib did not produce any apparent toxicity or weight loss (Fig. 7C). Immunohistochemical analysis of the excised tumors showed expression of ABCG2 in H460/MX20 tumors and there was no significant difference in the expression level of the ABCG2 among different groups. Taken together, telatinib did not increase the toxicity; instead it improved the efficacy of DOX in the H460/MX20 xenograft model.

Reference: Biochem Pharmacol. 2014 May 1; 89(1): 52–61. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3983711/