# Data Sheet

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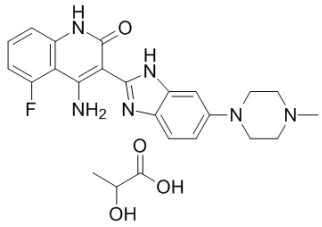
| **Product Name:** | Dovitinib (lactate) |
| **Cat. No.:** | HY-10207 |
| **CAS No.:** | 692737-80-7 |
| **Molecular Formula:** | C24H27FN6O4 |
| **Molecular Weight:** | 482.51 |
| **Target:** | FGFR |
| **Pathway:** | Protein Tyrosine Kinase/RTK |
| **Solubility:** | DMSO: ≥ 30 mg/mL |



## BIOLOGICAL ACTIVITY:

Dovitinib(CHIR–258; TKI258) lactate is a potent inhibitor of fibroblast growth factor receptor 3 (**FGFR3**) with an **$IC_{50}$** of 5 nM.
IC50 & Target: IC50: 5 nM (FGFR3)[1]
***In Vitro:*** Dovitinib potently inhibits FGFR3 with an $IC_{50}$ of 5 nM in *in vitro* kinase assays and selectively inhibits the growth of B9 cells and human myeloma cell lines expressing wild–type or activated mutant FGFR3. Addition of interleukin 6 (IL–6) or insulin growth factor 1 or coculture on stroma does not confer resistance to dovitinib. In primary myeloma cells dovitinib inhibits downstream extracellular signal–regulated kinase (ERK) 1/2 phosphorylation with an associated cytotoxic response[1]. Treatment of SK–HEP1 cells with dovitinib results in G2/M cell cycle arrest, inhibition of colony formation in soft agar and blockade of bFGF–induced cell migration. Dovitinib inhibits basal expression and FGF–induced phosphorylation of FGFR–1, FRS2–α and ERK1/2[2].
***In Vivo:*** Dovitinib demonstrates significant antitumor and antimetastatic activities in HCC xenograft models. Dovitinib potently inhibits tumor growth of six HCC lines. Inhibition of angiogenesis correlates with inactivation of FGFR/PDGFR–β/VEGFR–2 signaling pathways. Dovitinib also causes dephosphorylation of retinoblastoma, upregulation of p–histone H2A–X and p27, and downregulation of p–cdk–2 and cyclin B1, which results in a reduction in cellular proliferation and the induction of tumor cell apoptosis[2].

## PROTOCOL (Extracted from published papers and Only for reference)

**Cell Assay:** [2]To determine $IC_{50}$ for SK–HEP1 cells, cells are plated at a density of $2\times10^4$ cells per dish. After 48 h, cells are treated with 0, 0.01, 0.1, 1, 5, 10, 50, 100 μM dovitinib in DMEM containing 1% FBS for 24 h. Cell viability is determined with Cell Proliferation Assay. $IC_{50}$ is calculated by nonlinear regression analysis using GraphPad Prism software[2].
**Animal Administration:** [2]Mice: Six HCC lines (06–0606, 26–0808A, 26–1004, 25–0705A, 5–1318, 21–0208) are used to establish tumors in male SCID mice. or dose–esponse experiments, mice bearing the 06–0606 xenografts re orally given vehicle (5% dextrose) or two doses of dovitinib (50 and 75 mg/kg) daily for 14 days. For time–dependent inhibition of dovitinib targets, mice bearing 06–0606 tumors are given orally 200 μL of either vehicle (n=6) or 50 mg/kg/day of dovitinib (n=10)[2].

## References:

[1]. Trudel S, et al. CHIR–258, a novel, multitargeted tyrosine kinase inhibitor for the potential treatment of t(4;14) multiple myeloma. Blood. 2005, 105(7), 2941–2948.
[2]. Huynh H, et al. Dovitinib demonstrates antitumor and antimetastatic activities in xenograft models of hepatocellular carcinoma. J Hepatol. 2012, 56(3), 595–601.

**Caution: Product has not been fully validated for medical applications. For research use only.**

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