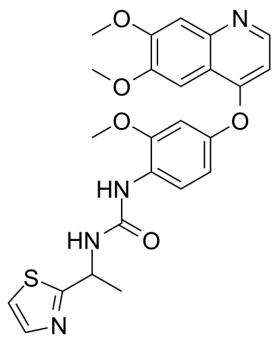
Ki20227

Cat. No.:	HY-10408
CAS No.:	623142-96-1
Molecular Formula:	$C_{24}H_{24}N_4O_5S$
Molecular Weight:	480.54
Target:	c-Fms; VEGFR; c-Kit; PDGFR
Pathway:	Protein Tyrosine Kinase/RTK
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 62.5 mg/mL (130.06 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0810 mL	10.4050 mL	20.8099 mL
	5 mM	0.4162 mL	2.0810 mL	4.1620 mL
	10 mM	0.2081 mL	1.0405 mL	2.0810 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.17 mg/mL (4.52 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.17 mg/mL (4.52 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.17 mg/mL (4.52 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ki20227 is an orally active and highly selective c-Fms tyrosine kinase (CSF1R) inhibitor with IC_{50}s of 2 nM, 12 nM, 451 and 217 nM for CSF1R, VEGFR2 (vascular endothelial growth factor receptor-2), c-Kit (stem cell factor receptor) and PDGFRβ (platelet-derived growth factor receptor β). Ki20227 suppresses osteoclast differentiation and osteolytic bone destruction[1].

IC_{50} & Target

IC50: 2 nM (CSF1R), 12 nM (VEGFR2), 451 nM (c-Kit) and 217 nM (PDGFRβ)[1]

In Vitro

Ki20227 (0.1-1000 nM; 72 hours) with 100 and 1,000 nM almost suppresses M-NFS-60 cell growth and HUVEC cell growth,

respectively[1].
Ki20227 (0.1-1000 nM; 1 hour) suppresses M-CSF-dependent c-Fms phosphorylation in a dose-dependent manner[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	M-NFS-60 cells, HUVEC cells, human A375 melanoma cells
Concentration:	0.1, 0.3, 1, 3, 10, 30, 100, 300, 1000 nM
Incubation Time:	72 hours
Result:	100 and 1,000 nM almost suppressed M-NFS-60 cell growth and HUVEC cell growth, respectively.

Cell Viability Assay[1]

Cell Line:	RAW264.7 cell lysate
Concentration:	0.1, 0.3, 1, 3, 10, 30, 100, 300, 1000 nM
Incubation Time:	1 hour
Result:	Suppressed M-CSF-dependent c-Fms phosphorylation in a dose-dependent manner.

In Vivo

Ki20227 (orally;10-50 mg/kg/d for 20 days) of 50 mg/kg/d of Ki20227 for 20 days markedly decreases the osteolytic lesion areas[1].
ki20227 during global ischemia led to a significant deficit in microglial density in the CNS in mice, and CSF1R-inhibition led to a significant reduction in the neuronal density of mice[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	4-week-old male F344/NJcl-rnu rats[1]
Dosage:	10, 20, and 50 mg/kg
Administration:	Orally; once per day for 20 days
Result:	Oral administration of 50 mg/kg/d markedly decreased the osteolytic lesion areas.

CUSTOMER VALIDATION

- Brain Behav Immun. 2020 Oct;89:400-413.
- Neural Regen Res. 2022 Jan;17(1):137-143.
- Cell Signal. 2023 Dec 31:115:111031.
- Int J Clin Exp Pathol. 2016;9(12):12459-12469.
- Harvard Medical School LINCS LIBRARY

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REFERENCES

[1]. Ohno H, et al. A c-fms tyrosine kinase inhibitor, Ki20227, suppresses osteoclast differentiation and osteolytic bone destruction in a bone metastasis model. Mol Cancer

Ther. 2006 Nov;5(11):2634-43.

[2]. Boru Hou, et al. Ki20227 influences the morphology of microglia and neurons through inhibition of CSF1R during global ischemia. Int J Clin Exp Pathol. 2016;9(12):12459-12469.

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