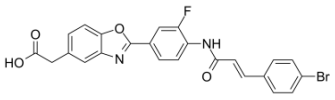
OGT 2115

Cat. No.:	HY-100898
CAS No.:	853929-59-6
Molecular Formula:	$C_{24}H_{16}BrFN_2O_4$
Molecular Weight:	495.3
Target:	Others
Pathway:	Others

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 11.36 mg/mL (22.94 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0190 mL	10.0949 mL	20.1898 mL
	5 mM	0.4038 mL	2.0190 mL	4.0380 mL
	10 mM	0.2019 mL	1.0095 mL	2.0190 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1.14 mg/mL (2.30 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

OGT 2115 is a potent, cell-permeable and orally active heparanase inhibitor with an IC_{50} of 0.4 μM. OGT 2115 has anti-angiogenic properties (IC_{50} of 1 μM). OGT 2115 also inhibits heparan sulfate degradation activity[1][2].

IC_{50} & Target

IC50: 0.4 μM (Heparanase)[1]

In Vitro

Heparanase InhibitorOGT 2115 can suppress metastasis induced by endoplasmic reticulum (ER) stress in breast cancer cells, although not significantly. However, compared with the control group, the number and rate of migrated cells are significantly reduced following the exposure of the cells to Tunicamycin + OGT 2115. OGT 2115 significantly inhibits the invasion and migration induced by Adriamycin. Furthermore, the MTT assay results show that OGT 2115 does not decrease the anti-proliferative effect of Adriamycin[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo	When administered to mice, OGT 2115 (Compound 12d) shows a plasma concentration of ~10x the heparanase IC_{50} following oral dosing at 20 mg/kg[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. [1]Courtney SM, et al. Furanyl-1,3-thiazol-2-yl and benzoxazol-5-yl acetic acid derivatives: novel classes of heparanase inhibitor. Bioorg Med Chem Lett. 2005 May 2;15(9):2295-9.

[2]. Li Y, et al. Suppression of endoplasmic reticulum stress-induced invasion and migration of breast cancer cells through the downregulation of heparanase. Int J Mol Med. 2013 May;31(5):1234-42.

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