Talmapimod hydrochloride

Cat. No.: HY-10406A

CAS No.: 309915-12-6

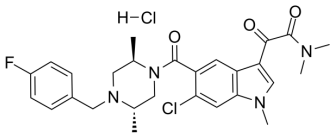
Molecular Formula: $C_{27}H_{31}Cl_2FN_4O_3$

Molecular Weight: 549.46

Target: p38 MAPK

Pathway: MAPK/ERK Pathway

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Talmapimod (SCIO-469) hydrochloride is an orally active, selective, and ATP-competitive p38α inhibitor with an IC_{50} of 9 nM. Talmapimod hydrochloride shows about 10-fold selectivity over p38β, and at least 2000-fold selectivity over a panel of 20 other kinases, including other MAPKs[1][2][3].

IC_{50} & Target

p38α	p38β
9 nM (IC_{50})	90 nM (IC_{50})

In Vitro

Talmapimod (SCIO-469) hydrochloride (100-200 nM; 1 hour) inhibits phosphorylation of p38 MAPK in MM cells[1].
Talmapimod hydrochloride inhibits LPS-induced TNF-a production in human whole blood[2].
Talmapimod hydrochloride decreases constitutive p38alpha MAPK phosphorylation of both 5T2MM and 5T33MM cells[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	MM.1S, U266, RPMI8226, MM.1R, and RPMI-Dox40 cell lines
Concentration:	100, 200 nM
Incubation Time:	1 hour
Result:	Strongly inhibits phosphorylation of p38 MAPK.

In Vivo

Talmapimod hydrochloride (10-90 mg/kg; P.o.; twice daily orally for 14 days) dose-dependently reduced tumor growth and also dose-dependently reduced weight of the palpable tumors at termination[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Six-week-old male triple immune-deficient BNX mice (RPMI-8226 MM palpable tumors)[4]
Dosage:	10, 30, 90 mg/kg
Administration:	P.o.; twice daily orally for 14 days
Result:	Dose-dependently reduced tumor growth.

CUSTOMER VALIDATION

- Cell. 2020 Aug 6;182(3):685-712.e19.
- Sci Transl Med. 2018 Jul 18;10(450):eaaq1093.
- Cell Biol Toxicol. 2021 Aug;37(4):515-529.

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REFERENCES

[1]. Hideshima T et al. p38 MAPK inhibition enhances PS-341 (bortezomib)-induced cytotoxicity against multiple myeloma cells. Oncogene. 2004 Nov 18, 23(54), 8766-76.

[2]. Navas T, et al. Inhibition of p38alpha MAPK disrupts the pathological loop of proinflammatory factor production in the myelodysplastic syndrome bone marrow microenvironment. Leuk Lymphoma. 2008 Oct;49(10):1963-75.

[3]. Vanderkerken K et al. Inhibition of p38alpha mitogen-activated protein kinase prevents the development of osteolytic bone disease,reduces tumor burden, and increases survival in murine models of multiple myeloma. Cancer Res. 2007 May 15;67(10):4572-7.

[4]. Medicherla S, et al. p38alpha-selective MAP kinase inhibitor reduces tumor growth in mouse xenograft models of multiple myeloma. Anticancer Res. 2008 Nov-Dec;28(6A):3827-33.

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