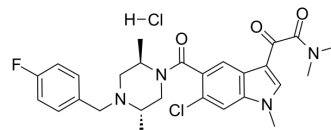


Talmapimod hydrochloride

Cat. No.:	HY-10406A
CAS No.:	309915-12-6
Molecular Formula:	C ₂₇ H ₃₁ Cl ₂ FN ₄ O 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	<p>Talmapimod (SCIO-469) hydrochloride is an orally active, selective, and ATP-competitive p38α inhibitor with an IC₅₀ 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^{[1][2][3]}.</p>									
IC₅₀ & Target	<p>p38α 9 nM (IC₅₀)</p>	<p>p38β 90 nM (IC₅₀)</p>								
In Vitro	<p>Talmapimod (SCIO-469) hydrochloride (100-200 nM; 1 hour) inhibits phosphorylation of p38 MAPK in MM cells^[1]. Talmapimod hydrochloride inhibits LPS-induced TNF-α production in human whole blood^[2]. Talmapimod hydrochloride decreases constitutive p38α MAPK phosphorylation of both 5T2MM and 5T33MM cells^[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p>Western Blot Analysis^[1]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>MM.1S, U266, RPMI8226, MM.1R, and RPMI-Dox40 cell lines</td> </tr> <tr> <td>Concentration:</td> <td>100, 200 nM</td> </tr> <tr> <td>Incubation Time:</td> <td>1 hour</td> </tr> <tr> <td>Result:</td> <td>Strongly inhibits phosphorylation of p38 MAPK.</td> </tr> </table>		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.
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Concentration:	100, 200 nM									
Incubation Time:	1 hour									
Result:	Strongly inhibits phosphorylation of p38 MAPK.									
In Vivo	<p>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.</p> <table border="1"> <tr> <td>Animal Model:</td> <td>Six-week-old male triple immune-deficient BXN mice (RPMI-8226 MM palpable tumors)^[4]</td> </tr> <tr> <td>Dosage:</td> <td>10, 30, 90 mg/kg</td> </tr> <tr> <td>Administration:</td> <td>P.o.; twice daily orally for 14 days</td> </tr> <tr> <td>Result:</td> <td>Dose-dependently reduced tumor growth.</td> </tr> </table>		Animal Model:	Six-week-old male triple immune-deficient BXN 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.
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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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Tel: 609-228-6898

Fax: 609-228-5909

E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA