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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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Adezmapimod hydrochloride

Cat. No.: HY-10256A

CAS No.: 869185-85-3

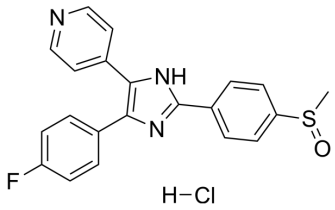
Molecular Formula: $C_{21}H_{17}ClFN_3OS$

Molecular Weight: 413.9

Target: p38 MAPK; Autophagy; Mitophagy; Organoid

Pathway: MAPK/ERK Pathway; Autophagy; Stem Cell/Wnt

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 1 year; -20°C, 6 months (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (241.60 mM; Need ultrasonic)
H_2O : 5 mg/mL (12.08 mM; Need ultrasonic)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	2.4160 mL	12.0802 mL	24.1604 mL
5 mM	0.4832 mL	2.4160 mL	4.8321 mL
10 mM	0.2416 mL	1.2080 mL	2.4160 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: ≥ 2.5 mg/mL (6.04 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.04 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Adezmapimod (SB 203580) hydrochloride is a selective and ATP-competitive p38 MAPK inhibitor with IC_{50}s of 50 nM and 500 nM for SAPK2a/p38 and SAPK2b/p38β2, respectively. Adezmapimod hydrochloride inhibits LCK, GSK3β and PKBα with IC_{50}s of 100-500-fold higher than that for SAPK2a/p38. Adezmapimod hydrochloride is an autophagy and mitophagy activator[1].

IC_{50} & Target

p38	p38β2
50 nM (IC_{50})	500 nM (IC_{50})

In Vitro

Adezmapimod hydrochloride (preincubated with 0-30 μM for 1 h and cultured for 24 h in the presence of 20 ng/mL IL-2) prevents the IL-2-induced proliferation of primary human T cells, murine CT6 T cells, or BAF F7 B cells with an IC_{50} of 3-5 μM [1].

Adezmapimod hydrochloride blocks PKB phosphorylation (IC_{50} 3-5 μM). Adezmapimod hydrochloride inhibitsthe phosphorylation of Ser473 in a dose-dependent manner in both CT6 and activated human T cells and IL-2-responsive BA/F3 F7 B cells[1].

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

Cell Proliferation Assay[1]

Cell Line:	CT6, BA/F3 cell line F7, and PBMC/T cells
Concentration:	0-30 μM
Incubation Time:	Preincubated with 0-30 μM SB203580 for 1 h and cultured for 24 h in the presence of 20 ng/mL IL-2
Result:	Prevented the IL-2-induced proliferation of primary human T cells, murine CT6 T cells, or BAF F7 B cells with an IC_{50} of 3-5 μM.

Western Blot Analysis[1]

Cell Line:	CT6 cells, activated human T cells, and BA/F3 F7 cells
Concentration:	0-30 μM
Incubation Time:	Preincubated with 0-30 μM SB203580 for 1 h before stimulating with 20 ng/mL IL-2 for 5 min
Result:	Inhibited the phosphorylation of PKB at Ser473 in a dose-dependent manner.

In Vivo

Adezmapimod hydrochloride (5 mg/kg/day; intra peritoneal injected daily for 16 consecutive days, in female atymic Nu/Nu mice) treatment, p38WT tumors show a significantly smaller tumor burden when compared with p38TM tumors that were treated in parallel[1].

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

Animal Model:	Six-week-old female atymic Nu/Nu mice CAL27 p38WT and p38TM tumors[1]
Dosage:	5 mg/kg/day
Administration:	Intra peritoneal injected daily for 16 consecutive days
Result:	After 2 weeks treatment, CAL27 p38WT tumors were significantly smaller; CAL27 p38TM tumors were not affected by the p38 inhibitor (n=10).

CUSTOMER VALIDATION

- Cell Res. 2020 Jul;30(7):574-589.
- Signal Transduct Target Ther. 2022 Jul 11;7(1):222.
- Signal Transduct Target Ther. 2020 Aug 25;5(1):163.
- Nat Immunol. 2023 Nov;24(11):1813-1824.
- Sci Immunol. 2022 Jan 21;7(67):eabj5501.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Davies SP, et al. Specificity and mechanism of action of some commonly used protein kinase inhibitors. Biochem J. 2000 Oct 1;351(Pt 1):95-105.

[2]. Lali FV, et al. The pyridinyl imidazole inhibitor SB203580 blocks phosphoinositide-dependent protein kinase activity, protein kinase B phosphorylation, and retinoblastoma hyperphosphorylation in interleukin-2-stimulated T cells independently of p38 mitogen-activated protein kinase. J Biol Chem. 2000 Mar 10;275(10):7395-402.

[3]. Leelahavanichkul K, et al. A role for p38 MAPK in head and neck cancer cell growth and tumor-induced angiogenesis and lymphangiogenesis. Mol Oncol. 2014 Feb;8(1):105-18.

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