SB 203580

Cat. No.: HY-10256

CAS No.: 152121-47-6

Molecular Formula: $C_{21}H_{16}FN_3OS$

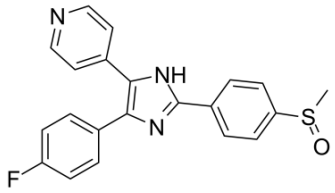
Molecular Weight: 377.43

Target: p38 MAPK; Autophagy; Mitophagy

Pathway: MAPK/ERK Pathway; Autophagy

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 : 15.62 mg/mL (41.39 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6495 mL	13.2475 mL	26.4950 mL
	5 mM	0.5299 mL	2.6495 mL	5.2990 mL
	10 mM	0.2649 mL	1.3247 mL	2.6495 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: ≥ 1.56 mg/mL (4.13 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1.56 mg/mL (4.13 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1.56 mg/mL (4.13 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

SB 203580 (RWJ 64809) 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. SB 203580 inhibits LCK, GSK3β and PKBα with IC_{50}s of 100-500-fold higher than that for SAPK2a/p38. SB 203580 does not disrupt JNK activity and is an autophagy and mitophagy activator[1].

IC_{50} & Target

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

In Vitro

SB 203580 (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].
SB203580 blocks PKB phosphorylation (IC_{50} 3-5 μM). SB203580 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

SB203580 (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.
- Mol Cell. 2020 Jan 2;77(1):95-107.e5.
- Signal Transduct Target Ther. 2020 Aug 25;5(1):163.
- Nat Commun. 2018 Apr 30;9(1):1723.
- Cell Death Differ. 2020 Jun;27(6):1938-1951.

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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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