产品名称：**4-(4-氟苯基)-2-(4-甲基亚磺酰基苯基)-5-(4-吡啶基)-1H-咪唑**
产品别名：**SB 203580**

生物活性：				
Description	SB 203580 (RWJ 64809) is a selective and ATP-competitive p38 MAPK inhibitor. SB 203580 (RWJ 64809) inhibits SAPK2a/p38 and SAPK2b/p38β2, with IC50s of 50 nM and 500 nM, respectively. LCK, GSK3β and PKBα are also inhibited by SB 203580 (RWJ 64809), but the IC50s are 100-500-fold higher than that for SAPK2a/p38[1].			
IC_{50} & Target	p38	p38β2	Autophagy	Mitophagy
	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 IC50 of 3-5 μM[1]. SB203580 blocks PKB phosphorylation (IC50 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].			
	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].			
	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).		

In Vitro:

DMSO : 100 mg/mL (264.95 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

<table>
<tr><td rowspan="2">Solvent&Solubility</td><td></td><td>10 mM</td><td>0.2649 mL</td><td>1.3247 mL</td><td>2.6495 mL</td></tr>
<tr><td colspan="5">*请根据产品在不同溶剂中的溶解度选择合适的溶剂配制储备液；一旦配成溶液，请分装保存，避免反复冻融造成的产品失效。
储备液的保存方式和期限：-80°C, 6 months; -20°C, 1 month。-80°C 储存时，请在 6 个月内使用，-20°C 储存时，请在 1 个月内使用。
In Vivo:
请根据您的实验动物和给药方式选择适当的溶解方案。以下溶解方案都请先按照 In Vitro 方式配制澄清的储备液，再依次添加助溶剂：
——为保证实验结果的可靠性，澄清的储备液可以根据储存条件，适当保存；体内实验的工作液，建议您现用现配，当天使用； 以下溶剂前显示的百分比是指该溶剂在您配制终溶液中的体积占比；如在配制过程中出现沉淀、析出现象，可以通过加热和/或超声的方式助溶
1.请依序添加每种溶剂： 10% DMSO→40% PEG300 →5% Tween-80 → 45% saline
Solubility: ≥ 2.5 mg/mL (6.62 mM); Clear solution
此方案可获得 ≥ 2.5 mg/mL (6.62 mM，饱和度未知) 的澄清溶液。
以 1 mL 工作液为例，取 100 μL 25.0 mg/mL 的澄清 DMSO 储备液加到 400 μL PEG300 中，混合均匀；向上述体系中加入 50 μL Tween-80，混合均匀；然后继续加入 450 μL 生理盐水定容至 1 mL。

2.请依序添加每种溶剂： 10% DMSO→ 90% (20% SBE-β-CD in saline)
Solubility: ≥ 2.5 mg/mL (6.62 mM); Clear solution
此方案可获得 ≥ 2.5 mg/mL (6.62 mM，饱和度未知) 的澄清溶液。
以 1 mL 工作液为例，取 100 μL 25.0 mg/mL 的澄清 DMSO 储备液加到 900 μL 20% 的 SBE-β-CD 生理盐水水溶液中，混合均匀。

3.请依序添加每种溶剂： 10% DMSO →90% corn oil
Solubility: ≥ 2.5 mg/mL (6.62 mM); Clear solution
此方案可获得 ≥ 2.5 mg/mL (6.62 mM，饱和度未知) 的澄清溶液，此方案不适用于实验周期在半个月以上的实验。
以 1 mL 工作液为例，取 100 μL 25.0 mg/mL 的澄清 DMSO 储备液加到 900 μL 玉米油中，混合均匀。</td></tr>
<tr><td>References</td><td colspan="5">[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.</td></tr>
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