

## SB 202190

Catalog No: tcsc0141



### Available Sizes

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**Size:** 50mg

**Size:** 100mg

**Size:** 200mg

**Size:** 500mg

**Size:** 1g



### Specifications

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**CAS No:**

152121-30-7

**Formula:**

$C_{20}H_{14}FN_3O$

**Pathway:**

MAPK/ERK Pathway;Autophagy

**Target:**

p38 MAPK;Autophagy

**Form:**

Pale-yellow Solid

**Purity / Grade:**

99.86%

**Solubility:**

DMSO :  $\geq 40$  mg/mL (120.72 mM)

**Storage Instruction:**

Powder: -20°C for 3 years In solvent: -80°C for 12 months

**Observed Molecular Weight:**

331.34

**Notes**

SB-202190 is a pyridinyl imidazole that inhibits p38 MAPK via competing with ATP.

**Product Description**

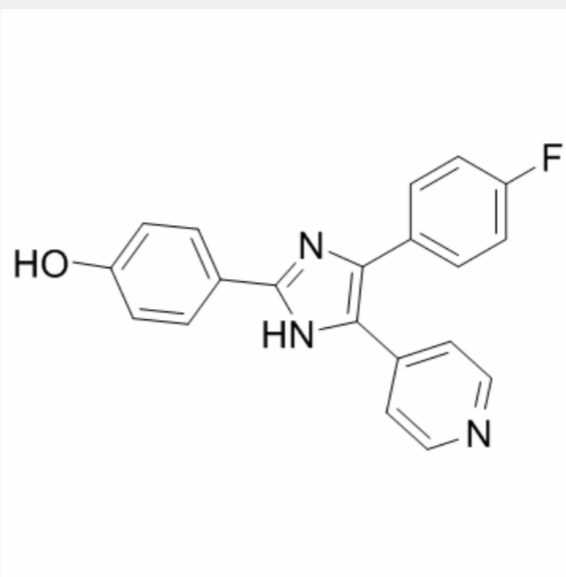
Mitogen-activated protein kinase (MAPK) cascades regulate signal transduction involved in cell proliferation and death. SB-202190 is a potent cell-permeable inhibitor of p38 MAPK that inhibits p38 and p38 $\beta$  with IC<sub>50</sub> values of 50 nM and 100 nM, respectively. SB-202190 at 5  $\mu$ M inhibited the activation of p38 in HaCaT cells. The protein expression of COX-2 was almost completely blocked by 5  $\mu$ M SB-202190 at 8 and 12 h post the exposure to UVB irradiation (250 J/m<sup>2</sup>). SB-202190 at the same concentration also significantly abrogated UVB induced cox-2mRNA in HaCaT cells. The inhibitory effect of SB-202190 on PGE<sub>2</sub> production after UVB was observed in HaCaT cells treated by 5  $\mu$ M SB-202190 for one hour. Bull serum albumin induced the gene expression of the inflammation marker MCP-1 more than 30-fold in renal tubular cells, while pre-incubation with 10  $\mu$ M SB-202190 decreased the gene expression to the basal level. In HK-2 cells, 10  $\mu$ M SB202190 treatment significantly reduced TGF- $\beta$ 1-induced gene expression. Two doses of SB-202190 (6.25  $\mu$ g/dose, i.d. administered) prevented the development of blisters and a positive Nikolsky's skin induced by PV IgG injection (1.5 mg of IgG/g body weight) in neonatal mice. The PV IgG-mediated activation of phospho-p38MAPK immunoreactivity in the skin was abrogated in SB-202190-treated mice.

SB 202190 inhibits **p38** and **p38 $\beta$ 2** with **IC<sub>50</sub>** values of 50 nM and 100 nM. respectively.

IC<sub>50</sub> & Target: IC<sub>50</sub>: 50 nM (p38), 100 nM (p38 $\beta$ 2)

**In Vitro:** Treatment of cells with SB 202190 (SB202190) significantly inhibits both basal and anti-Fas antibody-induced MAPKAPK 2 activity in a dose-dependent manner as measured in immune complex kinase assays with GST-hsp27 as a substrate. Jurkat cells are treated with SB202190 or left untreated. After 24 h, cells are harvested, and the activity of CPP32-like caspases in cell extracts is measured by cleavage of the fluorescent peptide DEVD-AMC, which is a specific substrate of CPP32-like caspases. The cleavage of DEVD-AMC is significantly increased in cells treated with SB202190 but not in the control.

**In Vivo:** In HCT-116-derived colorectal tumors, administration of SB 202190 (SB202190), Sorafenib or a combination of both give similar results in terms of measurement of external tumor size (around 58% growth reduction compare with control tumors). SB202190 induces a 28% reduction of tumor growth, compare with a 31% reduction promoted by Sorafenib, while combination of both drugs reduce tumor growth by 55%. Compare to the model group, the SB202190 group exhibits significantly shorter escape latencies in the Morris water maze hidden platform trials (P < 0.01) and longer times in the original platform quadrant during probe trials (P < 0.01). The SB202190 group also shows significantly reduced neuronal apoptosis in the hippocampus compared to VaD model rats (P < 0.01) as well as higher (antiapoptotic) Bcl-2 expression and lower (proapoptotic) caspase-3 expression (P < 0.01 for both). In conclusion, blockade of the p38 MAPK signaling pathway by SB202190 following permanent 2-OV reduced apoptosis of hippocampal neurons and rescued spatial learning and memory deficits.



All products are for RESEARCH USE ONLY. Not for diagnostic & therapeutic purposes!