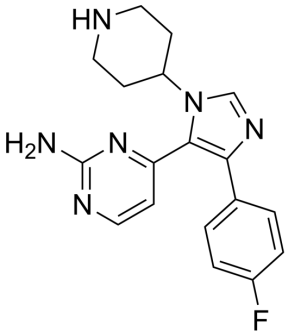
SB 220025

Cat. No.:	HY-112291
CAS No.:	165806-53-1
Molecular Formula:	$C_{18}H_{19}FN_6$
Molecular Weight:	338.38
Target:	p38 MAPK; Src; PKC
Pathway:	MAPK/ERK Pathway; Protein Tyrosine Kinase/RTK; Epigenetics; TGF-beta/Smad
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

SB 220025 is a reversible, orally active, cell-permeable, ATP-competitive and selective human p38 MAPK inhibitor (IC_{50} = 60 nM). SB 220025 also inhibits $p56^{Lck}$ and PKC with IC_{50} values of 3.5 and 2.89 μM, respectively. SB 220025 inhibits the expression of IL-8 gene in response to globular adiponectin (gAd), reduces inflammatory cytokine production and inhibits angiogenesis. SB 220025 effectively prevents the progression of arthritis in a chronic inflammatory disease model and can be used in the study of inflammation[1][2].

IC_{50} & Target

p38	p56-Lck	PKC
60 nM (IC_{50})	3.5 μM (IC_{50})	2.89 μM (IC_{50})

In Vitro

SB 220025 (20 μM; 6 h) markedly reduces IL-8 gene expression in response to globular adiponectin (gAd) in HUVEC cells[1].

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

RT-PCR[1]

Cell Line:	HUVEC cells
Concentration:	20 μM
Incubation Time:	6 h
Result:	Inhibited MCP-1 gene expression.

In Vivo

SB 220025 (3-50 mg/kg; p.o.; single) inhibits inflammatory cytokine production in vivo[2].

SB 220025 (5, 30, 50 mg/kg; i.p.; b.i.d.) inhibits angiogenesis in the murine air pouch granuloma model[2].

SB 220025 (30 mg/kg; p.o.; twice a day for 3, 5, 7 or 14 days) prevents the increase in angiogenesis that occurs after day 3 in murine air pouch angiogenesis model[2].

SB 220025 (50 mg/kg; p.o.; b.i.d.; 10 days) effectively blocks the progression of arthritis in a chronic inflammatory disease model[2].

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

Animal Model:	Acute model of LPS-induced TNF-a expression[2].
Dosage:	3-50 mg/kg

Administration:	Oral administration; single; 30 min before challenge with LPS.
Result:	Dosedependently inhibited TNF-a production with an ED_{50} value of 7.5 mg/kg, and showed more than 80% inhibition when at 50 mg/kg.

Animal Model:	Murine air pouch granuloma model[2].
Dosage:	5, 30, 50 mg/kg
Administration:	Intraperitoneal injection; bisindie (bid, twice a day).
Result:	Caused a dose-dependent reduction in angiogenesis.

Animal Model:	Murine air pouch granuloma model[2].
Dosage:	30 mg/kg
Administration:	Oral administration; twice a day from day 0 until removal of granuloma tissue at days 3, 5, 7 or 14.
Result:	Did not affect the initial burst of angiogenesis but did prevent the increase in angiogenesis that occurs after day 3.

REFERENCES

[1]. Tomizawa A, et al. Induction of gene expression in response to globular adiponectin in vascular endothelial cells. Life Sci. 2009 Sep 9;85(11-12):457-61.

[2]. Jackson JR, et al. Pharmacological effects of SB 220025, a selective inhibitor of P38 mitogen-activated protein kinase, in angiogenesis and chronic inflammatory disease models. J Pharmacol Exp Ther. 1998 Feb;284(2):687-92.

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