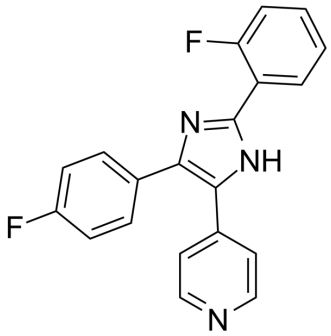
TA-02

Cat. No.:	HY-100115
CAS No.:	1784751-19-4
Molecular Formula:	$C_{20}H_{13}F_2N_3$
Molecular Weight:	333.33
Target:	p38 MAPK; Autophagy
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 : 25 mg/mL (75.00 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.0000 mL	15.0002 mL	30.0003 mL
	5 mM	0.6000 mL	3.0000 mL	6.0001 mL
	10 mM	0.3000 mL	1.5000 mL	3.0000 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 (7.50 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.50 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

TA-02, an analog of SB 203580 (HY-10256), is a p38 MAPK inhibitor with an IC_{50} of 20 nM. TA-02 especially inhibits TGFBR-2. TA-02 exhibits similar cardiogenic properties as SB 203580 and SB 202190 (HY-10295)[1].

In Vitro

TA-02 (5 μM) inhibits the phosphorylation of proteins downstream of p38α MAPK such asMAPKAPK2 and HSP27 during cardiogenesis. TA-02 at 5 μM concentration induces cardiogenesis, but also increases ATF-2 phosphorylation and MEF2C õexpression in contrast to what would be expected with a mechanism dependent on p38α MAPK inhibition[1].
TA-02 induces T/Brachyury whereas SB203580 addition increased MESP1 and T/Brachyury transcripts[1].
TA-02 significantly induces high NKX2-5 expression when applied between days 0-8[1].
TA-02 is found to inhibit multiple targets with similar potency to p38α MAPK, such as p38α, p38β, JNK3, JNK2, CIT, CK1ε, DMPK2, JNK1, DDR1 CK1δ, MEK5, and ERBB2[1].

TA-02 and SB203580 reduce the nuclear TCF/LEF-1 driven transcription of luciferase similar to DKK-1[1].
TA-02 (5 nM-5 μM) inhibits p38 and increases the anti-inflammation effects of BDNF on inflammation in vitro[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[2]

Cell Line:	The nerve cell line AGE1.HN.
Concentration:	5 nM-5 μM.
Incubation Time:	44 h (100 ng/ml LPS for 4 h at 37°C).
Result:	Suppressed p-38 protein expression, reduced IL-1β, IL-6, IL-18 and TNF-α levels and inhibited iNOS and COX-2 levels in an in vitro model of SCI by BDNF overexpression, compared with the BDNF overexpression group.

CUSTOMER VALIDATION

- Exp Ther Med. 2019 Mar;17(3):1688-1696.

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REFERENCES

[1]. Laco F, et al. Cardiomyocyte differentiation of pluripotent stem cells with SB203580 analogues correlates with Wnt pathway CK1 inhibition independent of p38 MAPK signaling. J Mol Cell Cardiol. 2015 Mar;80:56-70.

[2]. Jiedong Liang, et al. The activation of BDNF reduced inflammation in a spinal cord injury model by TrkB/p38 MAPK signaling. Exp Ther Med. 2019 Mar;17(3):1688-1696.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA