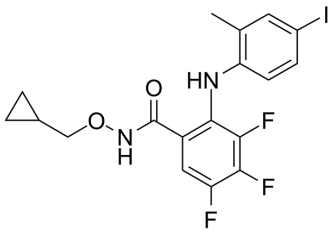
PD 198306

Cat. No.:	HY-107620
CAS No.:	212631-61-3
Molecular Formula:	$C_{18}H_{16}F_3IN_2O_2$
Molecular Weight:	476.23
Target:	MEK
Pathway:	MAPK/ERK Pathway
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

PD 198306 is a selective MAPK/ERK-kinase (MEK) inhibitor. PD 198306 results in an observable reduction in the Streptozocin induced increase in the level of active ERK1 and 2. Antihyperalgesic effects[1].

IC_{50} & Target

MEK

In Vitro

PD198306 significantly inhibits Tha-GFP replication by 25% at 10 μM, after 36 h[2].
PD198306 (5 μM) reduces Tha-Crimson replication significantly by 20% at 18 h but such a result could not be confirmed at 36 h[2].

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

Cell Cycle Analysis[2]

Cell Line:	Human induced pluripotent stem cells (iPSC)
Concentration:	10 μM
Incubation Time:	6 hours
Result:	Inhibited Tha-Crimson replication at 10 μM, reducing it by 30% at 18 h and 50% at 36 h.

In Vivo

Intrathecal administration of PD 198306 (1-30 μg per 10 μL) dose-dependently (1-30 μg) blocks static allodynia in both the streptozocin and the chronic constriction injury (CCI) models of neuropathic pain[1].

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

Animal Model:	Male Sprague Dawley rats (250-300 g) bearing neuropathic pain[1]
Dosage:	1-30 μg per 10 μL and 3 mg per 100 μL (PD 198306 is suspended in cremophor:ethanol:water, 1 : 1 : 8.)
Administration:	Single doses of intrathecal (i.t.) or intraplantar (ipl) of PD 198306 (1-30 μg per 10 μL and 3 mg per 100 μL respectively
Result:	Intrathecal administration dose-dependently (1-30 μg) blocked static allodynia the streptozocin model of neuropathic pain.

	The minimum effective doses (MED) of 3 μg significantly blocked static allodynia 30 min after treatment. Both 10 μg and the highest dose used (30 μg) totally blocked the maintenance of static allodynia, for up to 1 h.

REFERENCES

[1]. A Ciruela, et al. Identification of MEK1 as a novel target for the treatment of neuropathic pain. Br J Pharmacol. 2003 Mar;138(5):751-6.

[2]. Benoit Besson, et al. Kinome-Wide RNA Interference Screening Identifies Mitogen-Activated Protein Kinases and Phosphatidylinositol Metabolism as Key Factors for Rabies Virus Infection. mSphere. 2019 May 22;4(3):e00047-19.

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