CTOP

Cat. No.:	HY-P1329
CAS No.:	103429-31-8
Molecular Formula:	$C_{50}H_{67}N_{11}O_{11}S_2$
Molecular Weight:	1062.26
Sequence:	{D-Phe}-Cys-Tyr-{D-Trp}-{Orn}-Thr-{Pen}-Thr-NH2 (Disulfide bridge:Cys2-Pen7)
Sequence Shortening:	{D-Phe}-CY-{D-Trp}-{Orn}T{Pen}T-NH2 (Disulfide bridge:Cys2-Pen7)
Target:	Opioid Receptor
Pathway:	GPCR/G Protein; Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.

BIOLOGICAL ACTIVITY

Description	CTOP is a potent and highly selective μ-opioid receptor antagonist. CTOP antagonizes the acute morphine-induced analgesic effect and hypermotility. CTOP enhances extracellular dopamine levels in the nucleus accumbens. CTOP dose-dependently enhances locomotor activity[1][2].
IC_{50} & Target	μ Opioid Receptor/MOR

In Vivo

CTOP (0-0.5 nmol, ICV, once) antagonizes the analgesic effect of morphine in a dose-dependent manner[1].
CTOP (0-2 nmol, ICV, once) causes withdrawal hypothermia and a loss of body weight in morphine-dependent animals[1].
CTOP (0-1.5 nmol per side, Intra-VTA injection) enhances extracellular dopamine levels in the nucleus accumbens and dose-dependently enhances locomotor activity[2].

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

Animal Model:	Male CFLP mice (25-30 g)[1]
Dosage:	0, 0.001, 0.05, 0.075, 0.1, and 0.5 nmol (made up in artificial cerebrospinalfluid (CSF) and kept in plastic tubes at -25℃ until use)
Administration:	Intracerebroventricular (i.c.v.) administration, once
Result:	Antagonized the analgesic effect of morphine in a dose-dependent manner, antagonizedthe morphine-induced hypermotility in a dose-dependent manner.

Animal Model:	Male CFLP mice (25-30 g, Acute dependence to morphine was induced by a single dependence-inducing (100 mg/kg) dose of morphine-HC1)[1]
Dosage:	0, 0.001, 0.05, 0.2, and 2 nmol
Administration:	Intracerebroventricular (i.c.v.) administration, once
Result:	Decreased the body temperature in a dose-dependent manner, and caused withdrawal

	hypothermia and a loss of body weight in morphine-dependent animals.
Animal Model:	Long-Evans hooded rats (12, male, 350-450 g)[2]
Dosage:	0, 0.015, 0.15, and 1.5 nmol per side
Administration:	Intra-VTA (ventral tegmental area) injection
Result:	Enhanced extracellular dopamine levels in the nucleus accumbens, dose-dependently increased activity, whereas had no effect on feeding and drinking behavior.

CUSTOMER VALIDATION

- J Neurosci. 2022 Sep 8;JN-RM-1182-22.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Gulya K, et al. Central effects of the potent and highly selective μ opioid antagonist D-Phe-Cys-Tyr-D-Trp-Orn-Thr-Pen-Thr-NH2 (CTOP) in mice. Eur J Pharmacol. 1988 Jun 10;150(3):355-60.

[2]. Badiani A, et al. Intra-VTA injections of the mu-opioid antagonist CTOP enhance locomotor activity. Brain Res. 1995 Aug 28;690(1):112-6.

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