CTOP TFA

Cat. No.: HY-P1329A

Molecular Formula: $C_{52}H_{68}F_3N_{11}O_{13}S_2$

Molecular Weight: 1176.28

Sequence: Phe-Cys-Tyr-Trp-{Orn}-Thr-{Pen}-Thr-NH2 (Disulfide bridge:Cys2-Pen7)

Sequence Shortening: FCYW{Orn}T{Pen}T-NH2 (Disulfide bridge:Cys2-Pen7)

FCYW{Orn}T{Pen}T-NH2
(Disulfide bridge:Cys2-Pen7) (TFA salt)

Target: Opioid Receptor

Pathway: GPCR/G Protein; Neuronal Signaling

Storage: Sealed storage, away from moisture and light
Powder -80°C 2 years
-20°C 1 year
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)

SOLVENT & SOLUBILITY

In Vitro

H_2O : 50 mg/mL (42.51 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	0.8501 mL	4.2507 mL	8.5014 mL
	5 mM	0.1700 mL	0.8501 mL	1.7003 mL
	10 mM	0.0850 mL	0.4251 mL	0.8501 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
Solubility: 100 mg/mL (85.01 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description CTOP TFA is a potent and highly selective μ-opioid receptor antagonist. CTOP TFA antagonizes the acute analgesic effect and hypermotility. CTOP TFA enhances extracellular dopamine levels in the nucleus accumbens. CTOP TFA dose-dependently enhances locomotor activity[1][2].

IC_{50} & Target μ Opioid Receptor/MOR | μ Opioid Receptor/MOR

In Vivo

CTOP TFA (0-0.5 nmol, ICV, once) antagonizes the analgesic effect in a dose-dependent manner[1].
CTOP TFA (0-2 nmol, ICV, once) causes withdrawal hypothermia and a loss of body weight in animals[1].
CTOP TFA (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 in a dose-dependent manner, antagonizedthe 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 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.

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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.

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