CTOP TFA

Cat. No.: HY-P1329A

Molecular Formula: OC(C(F)(F)F)=O.O=C(N[C@@H]$_1$CC$_2$=CNC$_3$=CC=CC=C$_{23}$)[C@H](CC$_4$=CC=C(O)C=C$_4$)NC([C@@H](NC([C@@H](CC$_5$=CC=CC=C$_5$)N)=O)CSSC([C@@H]C(N[C@@

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

{D-Phe}-CY-{D-Trp}-{Orn}T{Pen}T-NH2 (Disulfide bridge:Cys2-Pen7) (TFA salt)

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: 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 (Need ultrasonic)
In Vivo	1. Add each solvent one by one: PBS Solubility: 100 mg/mL (Infinity 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
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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