Orexin A (human, rat, mouse) (acetate)

Cat. No.: HY-106224B

Molecular Formula: $C_{152}H_{243}N_{47}O_{44}S_4.xC_2H_4O_2$

Sequence Shortening: {Glp}-PLPDCCRQKTCSCRLYELLHGAGNHAAGILTL-NH2 (Disulfide bridge: Cys6-Cys12, Cys7-Cys14)

{Glp}-PLPDCCRQKTCSCRLYELLHGAGNHAAGILTL-NH_2 (Disulfide bridge: Cys_6-Cys_{12}, Cys_7-Cys_{14}) (acetate)

Target: Orexin Receptor (OX Receptor)

Pathway: GPCR/G Protein; Neuronal Signaling

Storage: -20°C, protect from light, stored under nitrogen
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light, stored under nitrogen)

SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 50 mg/mL
* "≥" means soluble, but saturation unknown.

BIOLOGICAL ACTIVITY

Description

Orexin A (Hypocretin-1) (human, rat, mouse) acetate is a hypothalamic neuropeptide with analgesic properties (crosses the blood-brain barrier). Orexin A (human, rat, mouse) acetate is also an OX1R agonist that induces the expression of BDNF and TH proteins in SH-SY5Y cells in a time- and dose-dependent manner. Orexin A (human, rat, mouse) acetate can be used in studies of appetite regulation, neurodegenerative diseases and modulation of injurious messaging[1][2][3].

In Vitro

Orexin A (human, rat, mouse) acetate (0.1, 1, 10, 100 nM; 24 h) increases the expression of BDNF in SH-SY5Y human dopaminergic neuroblastoma cells in a dose-dependent manner[1].
Orexin A (human, rat, mouse) acetate (1 nM; 1, 3, 6, 12, 24 h) increases the expression of BDNF, TH, and PI3K in a time-dependent manner in SH-SY5Y cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	SH-SY5Y cells
Concentration:	0.1, 1, 10, 100 nM
Incubation Time:	24 h
Result:	Increased the expression of PI3K and TH in a dose-dependent manner. Significantly induced BDNF expression at the dose of 1 and 10 nM.

Western Blot Analysis[1]

Cell Line:	SH-SY5Y cells
Concentration:	1 nM

Incubation Time:	1, 3, 6, 12, 24 h
Result:	Showed the expression of PI3K rapidly increased by 72.6 % as soon as 3 h, which maintained at high levels in the following 24 h. Increased the protein level of BDNF and the increased rate was 44.8%, and the protein level of TH significantly increased at 12 h by 48.5%.

In Vivo

Orexin A (human, rat, mouse) acetate (300 ng/mouse; i.c.v.; single daily for 8 days) attenuates the loss of dopaminergic neurons and the decrease of tyrosine hydroxylase (TH) expression in the substantia nigra, normalized the striatal dopaminergic fibers, and prevents the depletion of dopamine and its metabolites in the striatum[1].
Orexin A (human, rat, mouse) acetate (10, 30 mg/kg; i.v.; single) shows to be analgesic in the carrageenan-induced thermal hyperalgesia model and PPQ-induced mouse abdominal constriction model[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male C57BL/6 mice (10-week-old; 22-26 g; MPTP-Induced Mouse Model of PD)[1].
Dosage:	300 ng/mouse
Administration:	Intracerebroventricular injection; single daily for 8 days
Result:	Reversed MPTP-induced motor impairments via OX1R and increased the protein expression of BDNF in nigral dopaminergic neurons. Improved MPTP-induced impairments in spatial Learning and memory and protected dopaminergic neurons against MPTP-induced neurotoxicity. Attenuated the reduction of dopaminergic neurons in the substantia nigra and restored the reduction of dopaminergic fibers in the striatum.

Animal Model:	Female ICR mice (17-28 g; carrageenan-induced thermal hyperalgesia model)[2].
Dosage:	10, 30 mg/kg
Administration:	Intravenous injection; single; 5 min pre-test
Result:	Increased paw withdrawal latency to the thermal stimulus back to baseline levels.

Animal Model:	Male ICR mice (17-20 g; phenyl-p-quinone (PPQ)-induced mouse abdominal constriction model)[2].
Dosage:	3, 10, 30 mg/kg
Administration:	Intravenous injection; single; given immediately before PPQ
Result:	Increased the latency to the first PPQ-induced constriction from 357.4 s (blank control group) to 500.3 s at 10 mg/kg and 594.5 s at 30 mg/kg.

CUSTOMER VALIDATION

- J Inflamm Res. 2021 May 18;14:2007-2017.
- Brain Res Bull. 2021 Apr;169:81-93.
- Med Sci Monit. 2019 Apr 19;25:2886-2895.

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REFERENCES

[1]. Liu MF, et al. Orexin-A Exerts Neuroprotective Effects via OX1R in Parkinson's Disease. Front Neurosci. 2018 Nov 15;12:835.

[2]. Bingham S, et al. Orexin-A, an hypothalamic peptide with analgesic properties. Pain. 2001 May;92(1-2):81-90.

[3]. Rodgers RJ, et al. Orexins and appetite regulation. Neuropeptides. 2002 Oct;36(5):303-25.

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