Semaglutide

Cat. No.: HY-114118

CAS No.: 910463-68-2

Molecular Formula: $C_{187}H_{291}N_{45}O_{59}$

Molecular Weight: 4113.64

Target: GCGR

Pathway: GPCR/G Protein

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

Semaglutide

SOLVENT & SOLUBILITY

In Vitro

H_2O : 22.22 mg/mL (5.40 mM; ultrasonic and adjust pH to 1 with HCl)
DMSO : 4.17 mg/mL (1.01 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	0.2431 mL	1.2155 mL	2.4309 mL
5 mM	0.0486 mL	0.2431 mL	0.4862 mL
10 mM	---	---	---

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

BIOLOGICAL ACTIVITY

Description

Semaglutide, a long-acting GLP-1 analogue, is a glucagon-like peptide-1 (GLP-1) receptor agonist. Semaglutide has the potential for type 2 diabetes treatment.

IC_{50} & Target

GLP-1 receptor[1].

In Vitro

Semaglutide has two amino acid substitutions compared to human GLP-1 (Aib^8, Arg^{34}) and is derivatized at lysine 26. The GLP-1R affinity of Semaglutide is 0.38±0.06 nM[1]. Semaglutide is a GLP-1 analogue with 94% sequence omology to human GLP-1[3].

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

In Vivo

The plasma half-life of Semaglutide is 46h in mini-pigs following i.v. administration and semaglutide has an MRT of 63.6h after s.c. dosing to mini-pigs[1]. Semaglutide improves 1-methyl-4-phenyl-1,2,3,6- tetrahydropyridine (MPTP)-induced motor impairments. In addition, Semaglutide rescues the decrease of tyrosine hydroxylase (TH) levels, alleviates the inflammation response, reduces lipid peroxidation, inhibits the apoptosis pathway, and also increases autophagy- related protein

expression, to protect dopaminergic neurons in the substantia nigra and striatum. Moreover, the long-acting GLP-1 analogue semaglutide is superior to NN-2211 in most parameters[2].

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PROTOCOL

Animal Administration [2]

Mice[2]

Male C57BL/6 mice 10 weeks old (20-25 g) are used throughout the study. Mice are randomized divided into six groups (n=12 per group) (i) control group treated with saline alone; (ii) NN-2211 group treated with saline and NN-2211 (25 nmol/kg ip. once daily for 7 days); (iii) Semaglutide group treated with saline and Semaglutide (25 nmol/kg ip. once daily for 7 days), (iv) MPTP group treated with MPTP alone (once daily 20 mg/kg ip. for 7 days); (v) MPTP (once daily 20 mg/kg ip. for 7 days) followed immediately by NN-2211 treated group (25 nmol/kg ip. once daily for 7 days). (vi) MPTP (20 mg/kg ip. once daily for 7 days) followed immediately by Semaglutide treated group (25 nmol/kg ip. Once daily for 7 days). At the end of drug treatments, measure the behavioral changes, neuronal damage, inflammatory markers, and other biomarkers[2].

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

CUSTOMER VALIDATION

- Int J Mol Med. 2021 Dec;48(6):219.
- bioRxiv. 2023 Jul 19.

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REFERENCES

[1]. Marso SP, et al. Semaglutide and Cardiovascular Outcomes in Patients with Type 2 Diabetes. N Engl J Med. 2016 Nov 10;375(19):1834-1844.

[2]. Zhang L, et al. Neuroprotective effects of the novel GLP-1 long acting analogue semaglutide in the MPTP Parkinson's disease mouse model. Neuropeptides. 2018 Oct;71:70-80.

[3]. Dhillon S, et al. Semaglutide: First Global Approval. Drugs. 2018 Feb;78(2):275-284.

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