Semaglutide TFA

Cat. No.: HY-114118A

Molecular Weight: 4210.63

Target: Glucagon Receptor

Pathway: GPCR/G Protein

Storage:

Powder	-80°C	2 years
	-20°C	1 year
In solvent	-80°C	6 months
	-20°C	1 month

Semaglutide (TFA salt)

SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (23.75 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	0.2375 mL	1.1875 mL	2.3749 mL
5 mM	0.0475 mL	0.2375 mL	0.4750 mL
10 mM	0.0237 mL	0.1187 mL	0.2375 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (0.59 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (0.59 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (0.59 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Semaglutide TFA, a long-acting GLP-1 analogue, is a glucagon-like peptide-1 (GLP-1) receptor agonist. Semaglutide TFA 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 liraglutide in most parameters[2]. Semaglutide lowers blood glucose by stimulating the release of insulin and also lowers body weight[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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) liraglutide group treated with saline and liraglutide (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 liraglutide 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.

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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