GLP-1(7-36), amide acetate

Cat. No.: HY-P0054

CAS No.: 1119517-19-9

Molecular Formula: $C_{149}H_{226}N_{40}O_{45}.xC_2H_4O_2$

Sequence: His-Ala-Glu-Gly-Thr-Phe-Thr-Ser-Asp-Val-Ser-Ser-Tyr-Leu-Glu-Gly-Gln-Ala-Ala-Lys-Glu-Phe-Ile-Ala-Trp-Leu-Val-Lys-Gly-Arg-NH2

HAEGTFTSDVSSYLEGQAAKEFIAWLVKGR-NH_2 (acetate)

Sequence Shortening: HAEGTFTSDVSSYLEGQAAKEFIAWLVKGRNH2

Target: GCGR

Pathway: GPCR/G Protein

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 : 100 mg/mL (Need ultrasonic)
In Vivo	1. Add each solvent one by one: PBS Solubility: 25 mg/mL (Infinity mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description	GLP-1(7-36), amide acetate is a major intestinal hormone that stimulates glucose-induced insulin secretion from β cells.
In Vitro	Cells treated with phorbol 12-myristate 13-acetate for 2 h has significantly higher active GLP-1(7-36), amide acetate concentrations in the media than those in the control. The glucose treatment also increases active GLP-1 secretion from cells in dose-dependent manner. Palmitic, oleic, linoleic or linolenic acid dose-dependently stimulated active GLP-1 secretion from cells. Active GLP-1 secretion is significantly greater with unsaturated fatty acids such as oleic, linoleic and linolenic acids than with palmitic acid. The treatment of NCI-H716 cells with CPE dose-dependently increases active GLP-1 concentrations in the media. A 37% increase is observed in active GLP-1 secretion from these cells at a concentration of 0.1 % CPE[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Gastric administration of glucose increases active GLP-1(7-36) amide levels in the portal blood after 10 min, followed by a marked decrease at 30 min. The gastric administration of TO also increases active GLP-1 levels after 10 min, and followed by a decrease to basal levels at 60 min. Individually, glucose and TO increase the secretion of GLP-1 in a dose-dependent manner. Furthermore, the co-administration of glucose and TO additively increase peak GLP-1 levels. CPE-administered mice have higher active GLP-1 levels in the portal blood at 10 and 30 min than those in the control mice. When glucose is administered with CPE, active GLP-1 and insulin levels in the portal blood are slightly higher in CPE-administered mice than in the control mice. High-fat diet-fed C57BL/6J mice develop hyperglycaemia and impair glucose tolerance[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- MAbs. Jan-Dec 2021;13(1):1893425.
- Int J Endocrinol. 2020 Jun 19;2020:1484321.
- Research Square Preprint. 2023 May 22.
- Patent. US20200283424A1.
- Patent. US20200283424A1.

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REFERENCES

[1]. Fujii Y et al. Ingestion of coffee polyphenols increases postprandial release of the active glucagon-like peptide-1(GLP-1(7-36)) amide in C57BL/6J mice. J Nutr Sci. 2015 Mar 3

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