

GLP-1(7-36), amide TFA

Cat. No.:	HY-P0054B
Molecular Formula:	C ₁₅₁ H ₂₂₇ F ₃ N ₄₀ O ₄₇
Molecular Weight:	3411.65
Sequence Shortening:	HAEGTFTSDVSSYLEGQAAKEFIAWLKGRNH2
Target:	Glucagon Receptor
Pathway:	GPCR/G Protein
Storage:	Protect from light Powder -80°C 2 years -20°C 1 year

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

BIOLOGICAL ACTIVITY

Description	GLP-1(7-36), amide TFA is a major intestinal hormone that stimulates glucose-induced insulin secretion from β cells ^[1] .
In Vitro	Cells treated with phorbol 12-myristate 13-acetate for 2 h has significantly higher active GLP-1(7-36) Acetate (Human 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

- Int J Endocrinol. 2020 Jun.
- 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;4:e9.

Caution: Product has not been fully validated for medical applications. For research use only.

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