Data Sheet (Cat.No.T37892)

TargetMol

GLP-1(7-36), amide TFA

Chemical Properties

CAS No. :

Formula:

Molecular Weight:

Storage: Keep away from moisture
Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	GLP-1(7-36), amide TFA is a prominent intestinal hormone that stimulates glucose-induced insulin secretion from β cells[1].
Targets(IC50)	Glucagon Receptor
In vitro	Cells exposed to phorbol 12-myristate 13-acetate for 2 hours exhibited significantly increased concentrations of active GLP-1(7-36) Acetate (Human GLP-1-(7-36)-amide Acetate) in the media, surpassing those of the control group. Similarly, glucose treatment enhanced active GLP-1 secretion from cells in a dose-dependent manner. Furthermore, exposure to varying doses of palmitic, oleic, linoleic, or linolenic acid also stimulated active GLP-1 secretion from cells in a dose-dependent fashion, with unsaturated fatty acids, specifically oleic, linoleic, and linolenic acids, promoting greater active GLP-1 secretion than palmitic acid. Additionally, treating NCI-H716 cells with CPE led to a dose-dependent increase in media active GLP-1 concentrations, achieving a 37% rise at a concentration of 0.1% CPE[1].
In vivo	Administering glucose orally increases active GLP-1(7-36) amide concentrations in portal blood within 10 minutes, with a significant reduction observed after 30 minutes. Similar administration of TO also boosts active GLP-1 levels at 10 minutes but returns to baseline by 60 minutes. Both glucose and TO independently enhance GLP-1 secretion in a dose-responsive manner, while their combined administration synergistically elevates peak GLP-1 concentrations. Mice treated with CPE exhibit higher active GLP-1 levels at both 10 and 30 minutes post-administration compared to control mice. Additionally, when glucose is given alongside CPE, active GLP-1 and insulin concentrations in the portal blood marginally increase in CPE-treated mice versus the controls. This model also notes that high-fat diet-induced C57BL/6J mice experience hyperglycemia and diminished glucose tolerance[1].

Reference

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.

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