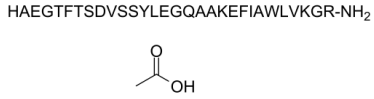
Data Sheet

Product Name:	GLP-1(7-36) Acetate
Cat. No.:	CS-5937
CAS No.:	1119517-19-9
Molecular Formula:	C149H226N40O45.xC2H4O2
Molecular Weight:	3394.67
Target:	Glucagon Receptor
Pathway:	GPCR/G Protein
Solubility:	DMSO : < 1 mg/mL;H_2O : 50 mg/mL (ultrasonic)



BIOLOGICAL ACTIVITY:

GLP-1(7-36) Acetate (Human 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) 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]. **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].

PROTOCOL (Extracted from published papers and Only for reference)

cell assay [1] The GLP-1 secretion study is performed as described by Reimer (with slight modifications. Briefly, 3 d before the study, 5 × 105 cells are seeded in twenty-four-well culture plates coated with Matrigel and containing high-glucose Dulbecco's modified Eagle's medium, 10 % FBS and 1/100 antibiotic-antimycotic. On the day of the experiment, medium was replaced by Krebs-Ringer bicarbonate solution with or without test agents and pH was adjusted to 7·2. Fresh media are added and cells were incubated for 2 h at 37 °C with a 0-0·1 % (w/v) CPE solution. The number of cells in each well did not differ significantly even after cells had been treated with CPE for 2 h. The effects of 2 μM-phorbol 12-myristate 13-acetate are examined as a positive control, Supernatant fractions are collected with the addition of 10 μl aprotinin solution (60 kIU) and diprotin-A (34 μg/ml), and then frozen at -80 °C for subsequent ELISA analyses. GLP-1 concentrations from the supernatant fractions are measured by Glucagon-Like Peptide-1 (Active) ELISA (Millipore Corporation). The basal level of GLP-1 secreted by the NCI-H716 cells for 2 h ranged between 131 and 392 pM in Krebs-Ringer bicarbonate solution.

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

CAIndexNames:

L-Argininamide, L-histidyl-L-alanyl-L-α-glutamylglycyl-L-threonyl-L-phenylalanyl-L-threonyl-L-seryl-L-α-aspartyl-L-valyl-L-seryl-L-seryl-L-tyrosyl-L-leucyl-L-α-glutamylglycyl-L-glutaminyl-L-alanyl-L-alanyl-L-lysyl-L-α-glutamyl-L-phenylalanyl-L-isoleucyl-L-alanyl-L-tryptophyl-L-leucyl-L-valyl-L-lysylglycyl-, acetate (1:?)

SMILES:

CC(O)=O.[HAEGTFTSDVSSYLEGQAAKEFIAWLVKGR-NH2]

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