Acetyl Gastric Inhibitory Peptide (human) (TFA)

Cat. No.: HY-P3580A

Molecular Formula: $C_{228}H_{340}N_{60}O_{67}S.C_2HF_3O_2$

Molecular Weight: 5139.62

Sequence: Ac-Tyr-Ala-Glu-Gly-Thr-Phe-Ile-Ser-Asp-Tyr-Ser-Ile-Ala-Met-Asp-Lys-Ile-His-Gln-Gln-Asp-Phe-Val-Asn-Trp-Leu-Leu-Ala-Gln-Lys-Gly-Lys-Lys-Asn-Asp-Trp-Lys-His-Asn-Ile-Thr-Gln

Sequence Shortening: Ac-YAEGTFISDYSIAMDKIHQQDFVNWLLAQKGKKNDWKHNITQ

Target: Insulin Receptor

Pathway: Protein Tyrosine Kinase/RTK

Storage: Sealed storage, away from moisture

Powder	-80°C	2 years
	-20°C	1 year

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)

SOLVENT & SOLUBILITY

In Vitro

H_2O : 50 mg/mL (9.73 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	0.1946 mL	0.9728 mL	1.9457 mL
	5 mM	0.0389 mL	0.1946 mL	0.3891 mL
	10 mM	---	---	---

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

BIOLOGICAL ACTIVITY

Description

Acetyl Gastric Inhibitory Peptide (human) TFA is a fatty acid derivatized analog of glucose-dependent insulinotropic polypeptide with improved antihyperglycaemic and insulinotropic properties. Acetyl Gastric Inhibitory Peptide (human) TFA can be used for research of diabetes, insulin resistance and obesity[1][2][3].

In Vitro

Acetyl Gastric Inhibitory Peptide (human) TFA induces cyclic adenosine 3'5' monophosphate (cAMP) production with an EC_{50} value of 1.9 nM in Chinese hamster lung fibroblast cells transfected with the human GIP receptor[1].

Acetyl Gastric Inhibitory Peptide (human) TFA (10^{-13}-10^{-8} nM) shows potent effect at stimulating insulin release compared to the native GIP in BRIN-BD11 cells[1].

Acetyl Gastric Inhibitory Peptide (human) TFA improves glucose intolerance, type 2 diabetes, beta-cell glucose insensitivity, insulin resistance and reduced insulin secretion[2].

Acetyl Gastric Inhibitory Peptide (human) TFA has metabolic stability and hypoglycemic and insulin modulating activities of two fatty acid derivatized N-terminally acetylated GIP analogs were evaluated in in vitro and in vivo[3].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Acetyl Gastric Inhibitory Peptide (human) TFA (25 nmol/kg; i.p.; single dose) shows resistance to plasma dipeptidylpeptidase IV degradation, resulting in enhanced biological activity and improved antidiabetic potential in vivo[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. O'Harte FP, et al. Improved stability, insulin-releasing activity and antidiabetic potential of two novel N-terminal analogues of gastric inhibitory polypeptide: N-acetyl-GIP and pGlu-GIP. Diabetologia. 2002 Sep;45(9):1281-91.

[2]. Gault Victor A, et al. GIP peptide analogues for treatment of diabetes, insulin resistance and obesity: World Intellectual Property Organization, WO2005082928[P]. 2005-12-01.

[3]. O'Harte, et al. Analogs of gastric inhibitory polypeptide as a treatment for age related decreased pancreatic beta cell function: World Intellectual Property Organization, WO2007028632[P].2007-03-15.

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