Neuronostatin-13 (human)

Cat. No.: HY-P1373

CAS No.: 1096485-24-3

Molecular Formula: $C_{64}H_{110}N_{20}O_{16}$

Molecular Weight: 1415.68

Sequence Shortening: LRQFLQKSLAAAA-NH2

Target: Others

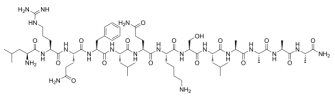
Pathway: Others

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 : 25 mg/mL (17.66 mM; Need ultrasonic)

Preparing Stock Solutions	Concentration / Solvent / Mass	1 mg	5 mg	10 mg
	1 mM	0.7064 mL	3.5319 mL	7.0637 mL
	5 mM	0.1413 mL	0.7064 mL	1.4127 mL
	10 mM	0.0706 mL	0.3532 mL	0.7064 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (70.64 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Neuronostatin-13 human is a 13-amino acid peptide hormone encoded by the somatostatin gene and plays an important role in the regulation of hormonal and cardiac function.

In Vitro

Neuronostatin-13 human is a 13-amino acid peptide hormone encoded by the somatostatin gene and plays an important role in the regulation of hormonal and cardiac function. Treatment with Neuronostatin-13 human (1,000 nM) enhances low-glucose-induced glucagon release compare with islets treated with control medium alone. Treatment with Neuronostatin-13 human for 1 h leads to a significant increase in the accumulation of glucagon mRNA compare with vehicle-treated control cells. In αTC1-9 α-cells, treatment with 100 nM Neuronostatin-13 human leads to an increase in phosphorylated PKA at 30 and 40 min[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo	Infusion with Neuronostatin-13 human delays glucose clearance in the rat model, such that blood glucose levels in Neuronostatin-13 human-treated animals are significantly higher at 1 and 10 min following intra-arterial injection of a glucose bolus[1]. Chocardiographic measurement reveals a remarkable drop in heart rate after 3-, 6- and 12-hr of Neuronostatin-13 human challenge. In addition, Neuronostatin-13 human treatment significantly decreases left ventricular end-systolic diameter (LVESD) and fractional shortening without affecting left ventricular end-diastolic diameter (LVEDD) between 6 and 12 hrs following Neuronostatin-13 human challenge, the effect of which returns to basal level 18-hr after Neuronostatin-13 human treatment[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	For studies examining hormone secretion from cell lines, INS 832/13 or αTC1-9 cells are plated in 96- or 24-well plates at a density of 0.25×10^5 cells/well or 1.0×10^5 cells/well in complete medium. The day of the experiment, cells are washed in PBS and allowed to preincubate in low- or high-glucose KRB buffer for 1 h in the presence or absence of Neuronostatin-13 human. Hormone secretion is performed using a 2-h static incubation. Supernatants are collected, and insulin or glucagon content is determined[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	3-month-old adult male C57BL/6 mice are used and housed in a temperature-controlled environment (22.8±2.0°C, 45 to 50% humidity) with a 12:12h light/dark cycle with free access to food and tap water. For Neuronostatin-13 human challenge in vivo, 3-month-old adult C57BL/6 male mice are randomly divided into two groups and to receive Neuronostatin-13 human (50 μg/kg, i.p.). Cardiac function is evaluated at 3-, 6-, 12- and 18-hr after Neuronostatin-13 human treatment in the first group of animals[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Salvatori AS, et al. Neuronostatin inhibits glucose-stimulated insulin secretion via direct action on the pancreatic α-cell. Am J Physiol Endocrinol Metab. 2014 Jun 1;306(11):E1257-63.

[2]. Zhu X, et al. Neuronostatin attenuates myocardial contractile function through inhibition of sarcoplasmic reticulum Ca2+-ATPase in murine heart. Cell Physiol Biochem. 2014;33(6):1921-32.

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