

Product Data Sheet

Product Name: Ghrelin (human)

Cat. No.: GC16346

Chemical Properties

Cas. No. 258279-04-8

Formula $C_{149}H_{249}N_{47}O_{42}$

M.Wt 3370.9

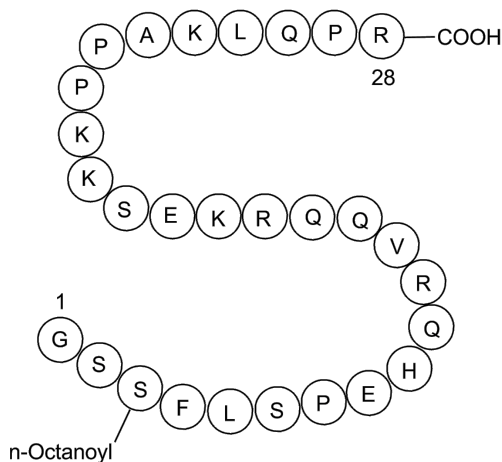
Solubility Soluble in Water

Storage Desiccate at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines Human stromovascular fraction cells (SVFCs)

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Preparation Method

Adipocyte differentiation was initiated when cells reached ~80-90% confluence. Cells were stimulated with differentiation medium I (adipocyte medium supplemented with 10% NCS, 0.5 mmol l-1 3-isobutyl-1-methylxanthine, 0.1 µmol l-1 dexamethasone and 10 µg ml-1 insulin) for 2 days. Furthermore, the cells were then switched to differentiation medium II (adipocyte medium supplemented with 10% NCS and 10 µg ml-1 insulin) for another 6 days and media were changed every 2 days. Adipocytes were 70-75% differentiated (as determined by morphology) in the eighth day of differentiation. They were incubated for 2 h in a serum-free adipocyte medium and, then, stimulated with different concentrations of acylated Ghrelin (human) or with desacyl ghrelin (0.1, 1, 10, 100 and 1000 pmol l-1) for 48 h. One sample per experiment was used for obtaining control responses in the presence of a solvent.

Reaction Conditions 0.1, 1, 10, 100 and 1000 pmol l-1 for 48 hours

Applications

The addition of acylated and desacyl ghrelin during adipocyte differentiation induced a significant increase in PPARG and SREBF1 transcript levels.

References:

[1]: Rodríguez A, Gómez-Ambrosi J, Catalán V, et al. Acylated and desacyl ghrelin stimulate lipid accumulation in human visceral adipocytes[J]. International journal of obesity, 2009, 33(5): 541-552.

Background

Ghrelin (human) was isolated from the gut of both human and rat as the endogenous ligand of the growth hormone secretagogue receptor (GHS-R), and has an effect on the activity of arcuate neurones with a much stronger affinity (IC₅₀, 0.3×10⁻⁹M) than GHSR antagonist (D-Lys3)-GHRP-6 (IC₅₀, 0.9×10⁻⁶M) [1]. The highest levels of ghrelin are secreted from the X/A - like cells of the oxyntic glands located in the gastric fundus, with lower levels widely distributed throughout the body [2]. Ghrelin is secreted directly into the local gastric circulation and transported to the brain directly,

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requiring it to either cross the blood-brain barrier via a saturated transport system or via the blood stream to enter areas of the brain that are not protected by the blood-brain barrier [3]. Ghrelin modulates the hypothalamic arcuate nucleus, in an indirect manner, via activation of the vagus nerve and brain stem nuclei [4]. Ghrelin has a homeostatic role that encompasses multiple areas of the body, with actions that include downregulation of brown adipose tissue thermogenesis [5-7], modulation of non-hypothalamic brain regions producing an increased taste sensation [7] and stimulation of gastric emptying and motility [8]. The actions of ghrelin may contribute to the development of T2DM and obesity [9].

Differentiating visceral adipocytes were exposed to increasing concentrations of acylated (human) and desacyl ghrelin (0.1-1000 pmol l⁻¹) for 48 h, and induced a significant increase in PPARG and SREBF1 transcript levels, the proportion of cells positive for lipid droplets was markedly increased in the presence of both ghrelin forms, compared with the cells in the differentiation medium without ghrelin [10].

References:

- [1]. Traebert M, Riediger T, Whitebread S, et al. Ghrelin acts on leptin-responsive neurones in the rat arcuate nucleus[J]. Journal of neuroendocrinology, 2002, 14(7): 580-586.
- [2]. Dixit V D, Schaffer E M, Pyle R S, et al. Ghrelin inhibits leptin-and activation-induced proinflammatory cytokine expression by human monocytes and T cells[J]. The Journal of clinical investigation, 2004, 114(1): 57-66.
- [3]. Angelidis G, Valotassiou V, Georgoulas P. Current and potential roles of ghrelin in clinical practice[J]. Journal of endocrinological investigation, 2010, 33(11): 823-838.
- [4]. Date Y, Murakami N, Toshinai K, et al. The role of the gastric afferent vagal nerve in ghrelin-induced feeding and growth hormone secretion in rats[J]. Gastroenterology, 2002, 123(4): 1120-1128.
- [5]. Tsubone T, Masaki T, Katsuragi I, et al. Ghrelin regulates adiposity in white adipose tissue and UCP1 mRNA expression in brown adipose tissue in mice[J]. Regulatory peptides, 2005, 130(1-2): 97-103.
- [6]. Whittle A J, LÓpez M, Vidal-Puig A. Using brown adipose tissue to treat obesity-the central issue[J]. Trends in molecular medicine, 2011, 17(8): 405-411.
- [7]. Mano-Otagiri A, Iwasaki-Sekino A, Nemoto T, et al. Genetic suppression of ghrelin receptors activates brown adipocyte function and decreases fat storage in rats[J]. Regulatory peptides, 2010, 160(1-3): 81-90.

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- [8]. Masuda Y, Tanaka T, Inomata N, et al. Ghrelin stimulates gastric acid secretion and motility in rats[J]. Biochemical and biophysical research communications, 2000, 276(3): 905-908.
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- [10]. Rodríguez A, Gómez-Ambrosi J, Catalán V, et al. Acylated and desacyl ghrelin stimulate lipid accumulation in human visceral adipocytes[J]. International journal of obesity, 2009, 33(5): 541-552.

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