

Product Data Sheet

Product Name: IUB0271 hydrochloride

Cat. No.: GC79208

Chemical Properties

Cas. No.

Formula $C_{264}H_{403}N_{65}O_{77}S \cdot xHCl$ M.Wt 5751.48

Solubility DMSO: ≥ 100 mg/mL Storage Sealed storage, away from moisture

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



Background

Caution: Product has not been fully validated for medical applications. For research use only.

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IUB0271 hydrochloride (acyl-GIP hydrochloride) is a glucose-dependent insulintropic polypeptide receptor (GIPR) agonist with blood-brain barrier permeability. IUB0271 hydrochloride activates the GIPR signaling pathway, inhibits food intake and weight gain, and induces cFos neuron activation in the area postrema. IUB0271 hydrochloride enhances lipid clearance and induces systemic fatty acid oxidation. IUB0271 hydrochloride improves dyslipidemia, reduces atherosclerotic plaque formation and decreases adipocyte volume. IUB0271 hydrochloride can be used in studies related to obesity, dyslipidemia and atherosclerosis [1] [2] [3].

In Vivo, IUB0271 (acyl-GIP) (100 nmol/kg; subcutaneous injection; single bolus/daily administration; 18 h/26 days) hydrochloride reduces acute food intake and achieves sustained reductions in body weight and fat mass in male diet-induced obese C57BL/6J wild-type mice via the GIPR signaling pathway in γ -aminobutyric acid (GABA)-ergic neurons. It fails to reduce acute food intake or induce sustained body weight loss in male diet-induced obese C57BL/6J Vgat-Gipr knockout mice, indicating that these effects depend on the GIPR signaling pathway in GABAergic neurons^[1]. IUB0271 (1 nmol/kg; subcutaneous injection; 30 min prior to tolerance testing) hydrochloride increases lipid tolerance by approximately 38% and improves glucose tolerance in obese male C57BL/6J mice^[2]. IUB0271 (150 nmol/kg; subcutaneous injection; single bolus) hydrochloride activates cFos expression in specific brain regions of male C57BL/6J wild-type mice fed a normal diet, but does not induce cFos activation in key brain regions of male C57BL/6J Vgat-Gipr knockout mice fed a normal diet^[1]. IUB0271 (1 nmol/kg; subcutaneous injection; single administration 30 min prior to gavage) hydrochloride enhances the clearance of plasma lipids into brown adipose tissue (BAT) by increasing lipoprotein lipase activity in BAT of obese male C57BL/6J mice by approximately 21%^[2]. IUB0271 (1 nmol/kg; subcutaneous injection; single administration 30 min before gavage; single administration at 11:00 a.m. for metabolic evaluation) hydrochloride increases lipid tolerance by approximately 28% and elevates systemic fatty acid oxidation levels by approximately 11% in obese male GiprBAT^{+/+} mice^[2]. IUB0271 (10 nmol/kg; subcutaneous injection; once daily for 28 consecutive days) hydrochloride improves dyslipidemia and reduces atherosclerotic plaque formation in male LDLR^{-/-} mice^[3].

In Vitro, IUB0271 (acyl-GIP) (10-50 nM; 2 h) hydrochloride induces a trend toward increased lipoprotein lipase activity in differentiated immortalized murine brown adipose tissue cells^[2].

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References:

- [1]. Liskiewicz A, et al. Glucose-dependent insulintropic polypeptide regulates body weight and food intake via GABAergic neurons in mice. *Nature metabolism*. 2023 Dec;5(12):2075-2085.
- [2]. Lyons SA, et al. Acute exogenous acyl-GIP treatment enhances lipid handling and fatty acid oxidation by involving brown fat. *EMBO reports*. 2025 Nov;26(21):5154-5171.
- [3]. Sachs S, et al. GIP receptor agonism improves dyslipidemia and atherosclerosis independently of body weight loss in preclinical mouse model for cardio-metabolic disease. *Cardiovascular diabetology*. 2023 Aug 17;22(1):217.

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