

Product Data Sheet

Product Name: BPR1M97

Cat. No.: GC38741

Chemical Properties

Cas. No. 2059904-66-2

SMILES O=C(NCC1N(C)CCC2=C1C=CC=C2)C3=CC=C(Cl)C(Cl)=C3

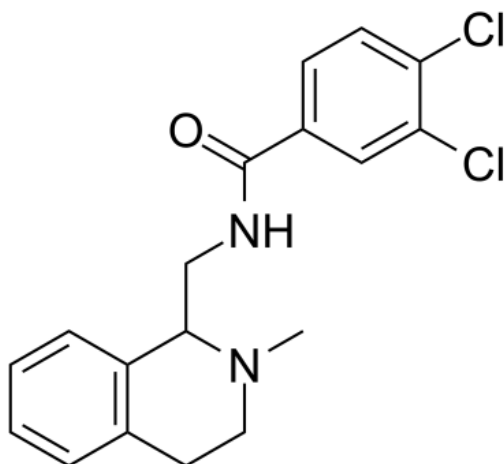
Formula $C_{18}H_{18}Cl_2N_2O$ M.Wt 349.25

Solubility DMSO: 250 mg/mL (715.82 mM) Storage Store 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

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

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Cell experiment [1]:

Cell lines	HEK-MOP cells (human embryonic kidney 293 cells constitutively expressing the human mu opioid receptor), CHO-K1-NOP cells (Chinese hamster ovary cells expressing the human nociceptin-orphanin FQ peptide receptor), U2OS-MOP cells (human osteosarcoma cells expressing human MOP), and myc-MOP-expressing mouse pituitary AtT-20 cells
Preparation Method	HEK-MOP cells were cultured in high-glucose DMEM supplemented with 10% fetal bovine serum (FBS). CHO-K1-NOP cells were cultured in F12 medium containing 10% FBS. U2OS-MOP cells were cultured in McCoy's 5A medium with 10% FBS. AtT-20 cells were cultured in DMEM with 10% FBS. Cells were treated with BPR1M97 at concentrations ranging from 0.6nM to 1.9μM. Incubation times varied by assay: 30 minutes for the cAMP assay, 1.5 hours for the β-arrestin-2 recruitment and receptor internalization assays, and 0.5 hours of pre-treatment with dye followed by real-time monitoring for the membrane potential assay.
Reaction Conditions	0.6nM–1.9μM; 0.5-1.5h.
Applications	In MOP-expressing cells, BPR1M97 acted as a full agonist, significantly inhibiting cAMP production, activating GIRK channels, recruiting β-arrestin-2, and inducing receptor internalization. In NOP-expressing cells, BPR1M97 acted as a G protein-biased full agonist, inhibiting cAMP production but failing to recruit β-arrestin-2.

Animal experiment [1]:

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Animal models	ICR mice, formalin-induced inflammatory pain model in ICR mice, and chronic constriction injury (CCI)-induced neuropathic pain model in ICR mice.
Preparation Method	Mice were subcutaneously (s.c.) administered a single dose of BPR1M97. For thermal and mechanical nociception tests, behavioral responses were recorded at specified time points post-injection. For tolerance and dependence studies, mice received twice-daily injections for 5 days.
Dosage form	0.2-9mg/kg; s.c.; Single or twice-daily injections for 5 days
Applications	BPR1M97 produced potent, dose-dependent antinociception in acute thermal, acute mechanical with efficacy comparable or superior to morphine. BPR1M97 caused significantly less respiratory depression, cardiovascular inhibition. BPR1M97 also induced conditioned place preference (reward effect) but with lower hyperlocomotor activity.

References:

[1] Chao PK, Chang HF, Chang WT, et al. BPR1M97, a dual mu opioid receptor/nociceptin-orphanin FQ peptide receptor agonist, produces potent antinociceptive effects with safer properties than morphine. *Neuropharmacology*. 2020 Apr;166:107678.

Background

BPR1M97 is a novel, dual-activity agonist of the mu-opioid receptor (MOP) and the nociceptin/orphanin FQ peptide (NOP) receptor. BPR1M97 produces potent analgesic effects by simultaneously activating both MOP and NOP receptors, while exhibiting fewer adverse reactions compared to morphine. BPR1M97 is used for pain management and related research^[1].

In vitro, cells expressing the human mu-opioid receptor (MOP) and/or the nociceptin/orphanin FQ peptide (NOP) receptor (e.g., HEK-MOP, CHO-K1-NOP, U2OS-MOP) were treated with BPR1M97

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(0.6nM – 1.9μM) for 0.5 to 1.5 hours. BPR1M97 significantly inhibited intracellular cAMP levels, activated GIRK channels, and induced β-arrestin-2 recruitment and receptor internalization^[1].

In vivo, normal ICR mice or mice with neuropathic pain models were subcutaneously administered a single dose of BPR1M97 (0.2-9mg/kg). BPR1M97 produced a dose-dependent and potent analgesic effect. At high doses, the respiratory depression it induced was significantly less severe than that caused by equi-analgesic doses of morphine^[1].

References:

[1] Chao PK, Chang HF, Chang WT, et al. BPR1M97, a dual mu opioid receptor/nociceptin-orphanin FQ peptide receptor agonist, produces potent antinociceptive effects with safer properties than morphine. *Neuropharmacology*. 2020 Apr;166:107678.

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