GSK205

Cat. No.: HY-120691A

CAS No.: 1263068-83-2

Molecular Formula: $C_{24}H_{25}BrN_4S$

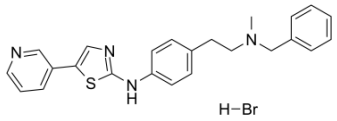
Molecular Weight: 481.45

Target: TRP Channel

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (519.26 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0771 mL	10.3853 mL	20.7706 mL
	5 mM	0.4154 mL	2.0771 mL	4.1541 mL
	10 mM	0.2077 mL	1.0385 mL	2.0771 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (4.32 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.32 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

GSK205 is a potent, selective TRPV4 antagonist with an IC_{50} of 4.19 μM for inhibiting TRPV4-mediated Ca^{2+} influx[1][2].

IC_{50} & Target

IC50: 4.19 μM (TRPV4)[2]

In Vitro

GSK205 (100 μM) potently antagonizes TRPV4 in 3T3-F442A adipocytes, as it effectively blocks the calcium influx caused by TRPV4 agonist[1].

GSK205 (5 μM; 4 days; T3-F442A adipocytes) treatment results in increases expression of thermogenic genes (Mcp1, Mip1α, Mcp3, Rantes and Vcam, et al.) and is also accompanied by a decrease in the proinflammatory gene program. This shift resembles the gene expression changes seen in TRPV4-deficient adipocytes[1].

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

RT-PCR[1]

Cell Line:	T3-F442A adipocytes
Concentration:	5 μM
Incubation Time:	4 days
Result:	Resulted in increased expression of thermogenic genes and is also accompanied by a decrease in the proinflammatory gene program.

In Vivo

GSK205 (10 mg/kg; intraperitoneal injection; twice daily; for 7 days; for 4 weeks; male C57BL/6J mice) treatment shows significantly increases expression of thermogenic genes such as Ucp1, Pgc1a, Cidea and Cox8b. GSK205 treatment causes a reduced expression of the proinflammatory chemokines, macrophage marker and Tnfa in the EPI fat. GSK205 treatment significantly improves glucose tolerance in diet-induced obese (DIO) mice. There are no apparent sign of sickness or weight loss[1].

GSK205 has a relatively short half-life of 2 hours in the plasma and adipose tissues[1].

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

Animal Model:	Male C57BL/6J mice with high-fat diet[1]
Dosage:	10 mg/kg
Administration:	Intraperitoneal injection; twice daily; for 7 days
Result:	Caused a reduced expression of the proinflammatory chemokines, macrophage marker and Tnfa in the EPI fat. Significantly improved glucose tolerance in diet-induced obese (DIO) mice.

REFERENCES

[1]. Ye L, et al. TRPV4 is a regulator of adipose oxidative metabolism, inflammation, and energy homeostasis. Cell. 2012 Sep 28;151(1):96-110.

[2]. Kanju P, et al. Small molecule dual-inhibitors of TRPV4 and TRPA1 for attenuation of inflammation and pain. Sci Rep. 2016 Jun 1;6:26894.

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