GSK5182

Cat. No.: HY-111226

CAS No.: 877387-37-6

Molecular Formula: $C_{27}H_{31}NO_3$

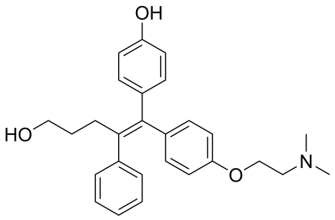
Molecular Weight: 417.55

Target: Estrogen Receptor/ERR; Reactive Oxygen Species

Pathway: Vitamin D Related/Nuclear Receptor; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB

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 : 25 mg/mL (59.87 mM; ultrasonic and warming and heat to 80°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3949 mL	11.9746 mL	23.9492 mL
	5 mM	0.4790 mL	2.3949 mL	4.7898 mL
	10 mM	0.2395 mL	1.1975 mL	2.3949 mL

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

BIOLOGICAL ACTIVITY

Description

GSK5182 is a highly selective and orally active inverse agonist of estrogen-related receptor γ (ERRγ) with an IC_{50} of 79 nM. GSK5182 does not interact with other nuclear receptors, including ERRα or ERα. GSK5182 also induces reactive oxyen species (ROS) generation in hepatocellular carcinoma (HCC)[1][2][3].

IC_{50} & Target

ERRγ
79 nM (IC_{50})

Reactive Oxygen Species

In Vitro

GSK5182 (0-20 μM; 0-hours; PLC/PRF/5 cells) treatment leads to a significant and dose-dependent reduction in the number of proliferating PLC/PRF/5 cells[1].

GSK5182 (0-20 μM; 24 hours; PLC/PRF/5 cells) treatment also causes a dose-dependent increase in the expression of p21 and p27 while at the same time reducing the level of phosphorylated retinoblastoma protein (p-pRb)[1].

GSK5182 (10-20 μM; PLC/PRF/5 cells) treatment induces cell cycle arrest at G1 phase, which in turn induces a corresponding dose-dependent reduction in the percentage of cells in S phase[1].

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

Cell Proliferation Assay[1]

Cell Line:	The human hepatoma cell line PLC/PRF/5
Concentration:	0 μM, 10 μM, 20 μM
Incubation Time:	0 hour, 24 hours, 48 hours, 72 hours
Result:	Led to a significant and dose-dependent reduction in the number of proliferating PLC/PRF/5 cells.

Western Blot Analysis[1]

Cell Line:	The human hepatoma cell line PLC/PRF/5
Concentration:	0 μM, 10 μM, 20 μM
Incubation Time:	24 hours
Result:	Caused a dose-dependent increase in the expression of p21 and p27 while at the same time reducing the level of p-pRb.

Cell Cycle Analysis[1]

Cell Line:	The human hepatoma cell line PLC/PRF/5
Concentration:	10 μM, 20 μM
Incubation Time:	
Result:	Induced cell cycle arrest.

In Vivo

GSK5182 (40 mg/kg; intraperitoneal injection; every day; 25 or 30 days; db/db mice, diet-induced obesity mice) specifically inhibits the transcriptional activity of ERRγ, and suppresses hepatic glucose production through inhibition of hepatic gluconeogenesis. GSK5182 elicits anti-diabetic effects in mouse models via negative regulation of the hepatic gluconeogenesis program. GSK5182 normalizes hyperglycemia mainly through inhibition of hepatic glucose production[3].

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

Animal Model:	db/db mice (male, 7-12-week-old), diet-induced obesity (DIO) mice[3]
Dosage:	40 mg/kg
Administration:	Intraperitoneal injection; every day; 30 days for db/db mice, 25 days for DIO mice
Result:	Inhibited the transcriptional activity of ERRγ, suppressed hepatic glucose production through inhibition of hepatic gluconeogenesis.

CUSTOMER VALIDATION

- Environ Sci Technol. 2022 Aug 10.
- Sci Total Environ. 2023 Aug 11;166257.

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REFERENCES

[1]. Kim JH, et al. Estrogen-related receptor γ is upregulated in liver cancer and its inhibition suppresses livercancer cell proliferation via induction of p21 and p27. Exp Mol Med. 2016 Mar 4;48:e213.

[2]. Misra J, et al. ERRγ: a Junior Orphan with a Senior Role in Metabolism. Trends Endocrinol Metab. 2017 Apr;28(4):261-272.

[3]. Kim DK, et al. Inverse agonist of nuclear receptor ERRγ mediates antidiabetic effect through inhibition of hepatic gluconeogenesis. Diabetes. 2013 Sep;62(9):3093-102.

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