Product Datasheet

GSK5182

Catalog No. **BP-02138** · CAS **877387-37-6** · Form **free base** · Physical state **solid** · Color **White to off-white** · Version **1.0** · Revision **2026-09-03**

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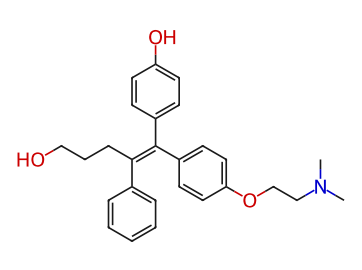
Target	Research area
Estrogen Receptor/ERR; Reactive Oxygen Species (ROS)	**Cardiovascular Disease; Cancer**

Molecular formula	**$C_{27}H_{31}NO_3$**
Molecular weight	**417.5399**

STRUCTURE



DESCRIPTION

GSK5182 is a highly selective and orally active inverse agonist of estrogen-related receptor γ (ERRγ) with an IC50 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) .

BIOLOGICAL ACTIVITY

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.

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).

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.

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.

SOLUBILITY

Soluble in DMSO,>10mg/mL, not in water

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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