G-1

Cat. No.: HY-107216

CAS No.: 881639-98-1

Molecular Formula: $C_{21}H_{18}BrNO_3$

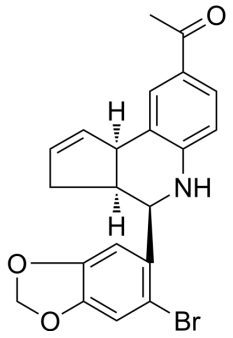
Molecular Weight: 412.28

Target: Estrogen Receptor/ERR

Pathway: Vitamin D Related/Nuclear Receptor

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (121.28 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.4255 mL	12.1277 mL	24.2554 mL
	5 mM	0.4851 mL	2.4255 mL	4.8511 mL
	10 mM	0.2426 mL	1.2128 mL	2.4255 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: ≥ 2.5 mg/mL (6.06 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (6.06 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.06 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

G-1 is a nonsteroidal, high-affinity and selective agonist of GPR30 with a K_i of 11 nM.

IC_{50} & Target

Ki: 11 nM (GPR30)[1]

In Vitro

G-1 is a nonsteroidal, high-affinity and selective agonist of GPR30 with a K_i of 11 nM[1].
Treatment with G-1 (10 μM and 100 μM) for 48 and 72 h significantly decreases cell proliferation (P<0.001). At 72 h, the IC_{50} value for G-1 is calculated to be 20 μM. Treatment of A549 cells with G-1 at a concentration of 20 μM reveals a significant

	increase in apoptosis, consistent with its antiproliferative effect (P<0.001)[2]. Cell cycle analysis of H295R cells after 24 h of G-1 treatment demonstrates a cell cycle arrest in the G_2 phase. The presence of G-1 increases Bax expression while decreases Bcl-2[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	The results at 14 days post-injury show that the Basso mouse scale (BMS) scores are significantly higher in the G-1 group compared with the other groups (P<0.05). The number of caspase-3-positive cells in the cross sections is counted, and G-1 group has fewer positive cells compare with the other groups (P<0.05), and there is no difference between the two groups (P>0.05)[1]. G-1 administration produces a statistically significant decrease in tumor volume from day 14 post treatment. Grafted tumors harvested after three-week treatment with G-1 show a significant decrease in tumor weight compare to vehicle treated animals[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	A549 human lung cancer cells are treated with various concentrations (10 nM, 100 nM, 1 μM, 10 μM and 100 μM) of G-1 in 96-well plates and incubated for 48 or 72 h. Following incubation, MTT solution is added to each well at a concentration of 0.5 mg/mL, and incubated for 4 h at 37°C. At the end of this period, 100 μL DMSO solvent is added to each well. The absorbance values [optical density (OD)] at 570 nm of the solution in each well are read using a spectrophotometer[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Four-week-old nu/nu-Forkhead box $N1^{nu}$ female mice are used in this study. H295R cells, 6×10^6, suspended in 100 μL PBS, are combined with 30 μL of Matrigel (4 mg/mL) and injected subcutaneously in the shoulder of each animal. Mice are treated 21 days after cell injection, when tumors have reached an average volume of about 200 mm^3. Animals are randomly assigned to be treated with vehicle or G-1 at a concentration of 2 mg/kg/daily. Drug tolerability is assessed in tumor-bearing mice in terms of: a) lethal toxicity, i.e. any death in treated mice occurring before any death in control mice; b) body weight loss percentage=100-[(body weight on day x/body weight on day 1)×100], where x represents a day during the treatment period. Animals are sacrificed by cervical dislocation 42 days after cell injection[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Death Dis. 2022 Apr 19;13(4):372.
- Phytomedicine. 2020 Mar;68:153146.
- Cell Death Discov. 2022 Jul 16;8(1):323.
- Life Sci. 2022 May 28;120676.
- J Clin Endocrinol Metab. 2021 Feb 1;dgab020.

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REFERENCES

[1]. Cheng Q, et al. G-1 exerts neuroprotective effects through G protein-coupled estrogen receptor 1 following spinal cord injury in mice. Biosci Rep. 2016 Aug 31;36(4). pii: e00373.

[2]. Kurt AH, et al. Oxidative/antioxidative enzyme-mediated antiproliferative and proapoptotic effects of the GPER1 agonist G-1 on lung cancer cells. Oncol Lett. 2015 Nov;10(5):3177-3182.

[3]. Chimento A, et al. GPER agonist G-1 decreases adrenocortical carcinoma (ACC) cell growth in vitro and in vivo. Oncotarget. 2015 Aug 7;6(22):19190-203.

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