

Product Data Sheet

Product Name: G-36
Cat. No.: GC14902

Chemical Properties

Cas. No. 1392487-51-2

Chemical Name (3aS,4R,9bR)-4-(6-bromobenzo[d][1,3]dioxol-5-yl)-8-isopropyl-3a,4,5,9b-tetrahydro-3H-cyclopenta[c]quinoline

SMILES CC(C1=CC([C@]2([H])C=CC[C@]2([H])[C@]3([H])C4=CC(OCO5)=C5C=C4Br)=C(N3)C=C1)C

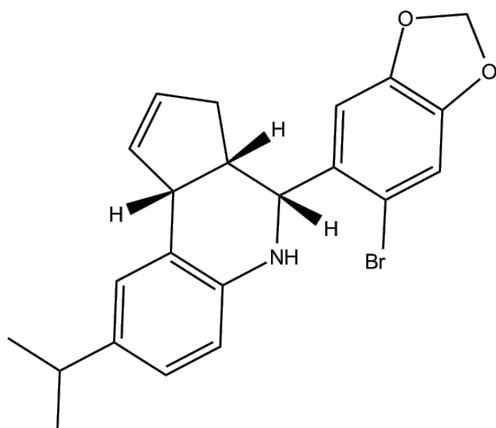
Formula $C_{22}H_{22}BrNO_2$ M.Wt 412.33

Solubility 1/mL in ethanol, 20mg/mL in DMSO, 30mg/mL in DMF 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

Cell experiment [1]:

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Cell lines Human umbilical vein endothelial cells

Preparation Method HUVECs were grown in M199 medium supplemented with 15% FBS, ECGS, heparin, glutamine, and gentamicin. For experiments, cells were switched to phenol-free M199 medium with 5% FBS, ECGS, and heparin. HUVECs were treated with G-36 (1 μ M).

Reaction
Conditions 1 μ M; 6h.

Applications G-36 completely abolished the enhancement of HUVEC migration induced by ethinylestradiol (EE) and estetrol (E4) in both scratch and Boyden chamber assays. G-36 also blocked the accumulation of phosphorylated FAK at tyrosine 397 (FAK Y397) induced by EE and E4.

Animal experiment [2]:

Animal models Ovariectomized C57Bl6 female mice

Preparation Method Mice were ovariectomized at 10 weeks of age. At 12 days post-ovariectomy, mice were subcutaneously injected with G-36 (50 μ g/kg). Mice were sacrificed 18 hours after injection, and uterine epithelial proliferation was quantified by Ki-67 immunofluorescence.

Dosage form 50 μ g/kg; s.c.; single injection.

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Applications G-36 completely abolished the G-1-induced proliferation of uterine epithelial cells and significantly reduced the E2-induced proliferation. G-36 alone had no effect on proliferation.

References:

- [1] Dama A, Baggio C, Trevisi L, et al. Regulation of human endothelial cell migration by oral contraceptive estrogen receptor ligands. *Eur J Pharmacol.* 2023 Apr 15;945:175591.
- [2] Dennis MK, Field AS, Burai R, et al. Identification of a GPER/GPR30 antagonist with improved estrogen receptor counterselectivity. *J Steroid Biochem Mol Biol.* 2011 Nov;127(3-5):358-66.

Background

G-36 is a cell-permeable G protein-coupled estrogen receptor (GPER/GPR30) antagonist that inhibits estrogen-mediated calcium mobilization ($IC_{50}=112nM$)^[1-2]. G-36 can be used in research on cancer-related signaling pathways^[3-4].

In vitro, pretreatment of human umbilical vein endothelial cells (HUVECs) with G-36 (1 μM) completely blocked the ethinylestradiol (EE) and estetrol (E4) (1 nM–100 nM)-induced cell migration and inhibited the accumulation of phosphorylated focal adhesion kinase at tyrosine 397 (FAK Y397)^[5]. Treatment of human cervical squamous cell carcinoma (CSCC) cell lines SiHa and C33A with G-36 (1–5 μM) for 3 to 7 days had no significant effect on cell migration, colony formation, or tumor spheroid formation; however, treatment of SiHa cells with 2.5 μM G-36 significantly increased the protein expression of plasminogen activator inhibitor-1 (PAI-1)^[6].

In vivo, G-36 (0.5 mg/kg) was co-administered with G-1 via intraperitoneal injection to high-fat diet (HFD)-fed C57BL/6 male mice (5 times per week for 5 weeks). G-36 blocked the protective effects of G-1 against HFD-induced obese asthma, reversing G-1's effects on airway hyperresponsiveness (AHR), pulmonary immune cell infiltration, tissue inflammation, fibrosis, mucus hypersecretion, increases in pro-inflammatory M1 macrophages, and decreases in anti-inflammatory M2 macrophages^[7]. In ovariectomized C57BL/6 female mice, a single subcutaneous injection of G-36 (50 $\mu g/kg$) blocked the G-1 (10 $\mu g/kg$)-induced proliferation of uterine epithelial cells and significantly reduced the 17 β -estradiol (E2; 10 $\mu g/kg$)-induced proliferation^[8].

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References:

- [1] Meyer MR, Barton M. GPER blockers as Nox downregulators: A new drug class to target chronic non-communicable diseases. J Steroid Biochem Mol Biol. 2018 Feb;176:82-87.
- [2] Yu X, Stallone JN, Heaps CL, et al. The activation of G protein-coupled estrogen receptor induces relaxation via cAMP as well as potentiates contraction via EGFR transactivation in porcine coronary arteries. PLoS One. 2018 Jan 23;13(1):e0191418.
- [3] Sharma G, Mauvais-Jarvis F, Prossnitz ER. Roles of G protein-coupled estrogen receptor GPER in metabolic regulation. J Steroid Biochem Mol Biol. 2018 Feb;176:31-37.
- [4] Evans PD. Rapid signalling responses via the G protein-coupled estrogen receptor, GPER, in a hippocampal cell line. Steroids. 2019 Dec;152:108487.
- [5] Dama A, Baggio C, Trevisi L, et al. Regulation of human endothelial cell migration by oral contraceptive estrogen receptor ligands. Eur J Pharmacol. 2023 Apr 15;945:175591.
- [6] Rörig L, Ruckriegl S, Gallwas J, et al. G Protein-coupled Estrogen Receptor 1 (GPER1) Regulates Expression of SERPINE1/PAI-1 and Inhibits Tumorigenic Potential of Cervical Squamous Cell Carcinoma Cells In Vitro. Cancer Genomics Proteomics. 2025 Jan-Feb;22(1):13-23.
- [7] Son SE, Im DS. Activation of G Protein-Coupled Estrogen Receptor 1 (GPER) Attenuates Obesity-Induced Asthma by Switching M1 Macrophages to M2 Macrophages. Int J Mol Sci. 2024 Sep 2;25(17):9532.
- [8] Dennis MK, Field AS, Burai R, et al. Identification of a GPER/GPR30 antagonist with improved estrogen receptor counterselectivity. J Steroid Biochem Mol Biol. 2011 Nov;127(3-5):358-66.

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