

Product Data Sheet

Product Name: G-1
Cat. No.: GC12766

Chemical Properties

Cas. No. 881639-98-1

Chemical Name rel-1-[4-(6-bromo-1,3-benzodioxol-5-yl)-3aR,4S,5,9bS-tetrahydro-3H-cyclopenta[c]quinolin-8-yl]-ethanone

SMILES CC(C1=CC([C@@](C=CC2)([H])[C@@]2([H])[C@H](C3=CC(OCO4)=C4C=C3Br)N5)=C5C=C1)=O

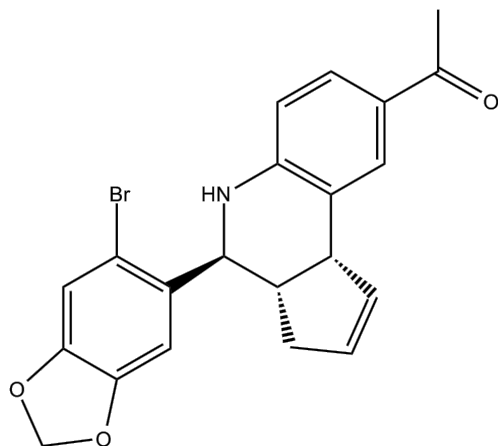
Formula $C_{21}H_{18}BrNO_3$ M.Wt 412.28

Solubility 1mg/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]:

Caution: Product has not been fully validated for medical applications. For research use only.

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Cell lines A549 human lung cancer cells

A549 human lung cancer cells were treated with various concentrations (10⁻⁸, 10⁻⁷, 10⁻⁶, 10⁻⁵ and 10⁻⁴ M) of 17β-estradiol and G-1 in 96-well plates and Preparation Method incubated for 48 or 72 h. Following incubation, MTT solution (Sigma-Aldrich) was added to each well at a concentration of 0.5 mg/ml, and incubated for 4 h at 37°C.

Reaction Conditions 0.01-100 μM for 48 and 72 h

Applications Treatment with G-1 (10⁻⁵ and 10⁻⁴ M) for 48 and 72 h significantly decreased cell proliferation.

Animal experiment [2]:

Animal models SJL mice (5-7 weeks old)

SJL mice were immunized s.c. with 50 μg PLP139-151 and CFA (400 μg Mycobacterium tuberculosis). Mice were treated with 50 mg/kg/day G-1 daily for Preparation Method 21 days beginning at the day of disease induction. Control mice were similarly treated with vehicle (5% Dimethyl sulfoxide (DMSO), 95% Polyethylene glycol (PEG)-300.

Dosage form 50 mg/kg/day for 21 days

Applications G-1 administration significantly reduced the severity of actively induced experimental allergic encephalomyelitis (EAE) but not the incidence of disease.

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References:

[1]: Kurt A H, Çelik A, Kelleci B M. Oxidative/antioxidative enzyme-mediated antiproliferative and proapoptotic effects of the GPER1 agonist G-1 on lung cancer cells[J]. Oncology Letters, 2015, 10(5): 3177-3182.

[2]: Blasko E, Haskell C A, Leung S, et al. Beneficial role of the GPR30 agonist G-1 in an animal model of multiple sclerosis[J]. Journal of neuroimmunology, 2009, 214(1-2): 67-77.

Background

G-1 is a selective and potent agonist of GPR30 with EC50 value about 2 nM [1].

G-1 treatment (10^{-5} and 10^{-4} M) for 48 and 72 h significantly decreased A549 cell proliferation, at 72 h, the IC50 value for G-1 was calculated to be 2×10^{-5} M [2]. G-1 treatment at a concentration of 2×10^{-5} M had no significant effect on CAT activity but led to a significant increase in SOD activity, GPx activity and NO level [2]. G-1 inhibited TNF- α and IL-6 release on primary human macrophages derived from monocytes treated with GM-CSF over 6 days. The agonist inhibited the induction of both cytokines with IC50 values of 209 nM and 317 nM, respectively [3]. G-1 was also able to inhibit LPS induction of TNF- α in a mouse macrophage cell line, RAW 264.7 [3].

G-1 (50 mg/kg/day, 21 days) administration significantly reduced the severity of actively induced experimental allergic encephalomyelitis (EAE). G-1 treatment reduced the qualitative degree of inflammation in the lumbar spinal cord. G-1 treatment reduced the fraction of CNS-infiltrating macrophages (CD45hiCD11b+) in three individually analyzed mice [3]. G-1 could exert protective effects on motoneurons. The intraperitoneal injection of the GPR30 agonist G-1 for 14 days induces neuroprotective effects similar with the same dose of E2 [4].

References:

[1]. Bologna C G, Revankar C M, Young S M, et al. Virtual and biomolecular screening converge on a selective agonist for GPR30[J]. Nature chemical biology, 2006, 2(4): 207-212.

[2]. Kurt A H, Çelik A, Kelleci B M. Oxidative/antioxidative enzyme-mediated antiproliferative and proapoptotic effects of the GPER1 agonist G-1 on lung cancer cells[J]. Oncology Letters, 2015, 10(5): 3177-3182.

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- [3]. Blasko E, Haskell C A, Leung S, et al. Beneficial role of the GPR30 agonist G-1 in an animal model of multiple sclerosis[J]. Journal of neuroimmunology, 2009, 214(1-2): 67-77.
- [4]. Cheng Q, Meng J, Wang X, et al. G-1 exerts neuroprotective effects through G protein-coupled estrogen receptor 1 following spinal cord injury in mice[J]. Bioscience Reports, 2016, 36(4).

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