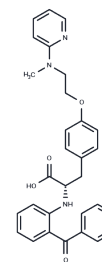


GW1929

Chemical Properties

CAS No. : 196808-24-9
 Formula: C₃₀H₂₉N₃O₄
 Molecular Weight: 495.57
 Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
 Actual storage temperature shall be subject to the COA.



Biological Description

Description	GW1929 is a potent PPAR- γ agonist (pKi: 8.84 for human PPAR- γ ; pEC ₅₀ s of 8.56 and 8.27 for human and murine PPAR- γ).
Targets(IC ₅₀)	PPAR
In vitro	GW1929 is a potent PPAR- γ activator, with pEC ₅₀ s of 8.56 and 8.27 for human PPAR- γ and murine PPAR- γ , and pK _i s of 8.84, < 5.5, and < 6.5 for human PPAR- γ , PPAR- α , and PPAR- δ , respectively [1]. GW1929 (10 μ M) inhibits TBBPA-induced caspase-3 increase and TBBPA-stimulated LDH release in neocortical cell cultures [2].
In vivo	GW1929 (0.5, 1, 5 mg/kg, p.o.) highly decreases nonfasted plasma glucose levels in Zucker diabetic fatty (ZDF) rats after treatment for 14 days and possesses antilipolytic efficacy. In ZDF rats, GW1929 (1, 5 mg/kg, p.o.) increases glucose-stimulated insulin secretion of β -cell [1].
Kinase Assay	Ligand binding to bacterially expressed ligand-binding domain (LBD) of hPPAR- γ is determined by the scintillation proximity assay (SPA). The assay measures the ability of putative ligands to displace receptor-bound [³ H]BRL 49653. Assays are conducted in 96-well plates. Wells contained varying concentrations of GW1929 or troglitazone; streptavidin-modified SPA beads to which biotinylated PPAR- γ LBD is prebound; and 10 nM of the specific radioligand [³ H]BRL 49653 in a volume of 100 μ L. The amount of nonspecific binding, as assessed by control wells that contained 50 μ M of the corresponding unlabeled ligand, is subtracted from each data point. For each compound tested, plots of ligand concentration versus counts/min of radioligand bound are constructed, and apparent K _i values are estimated from a nonlinear least-squares fit of the data, assuming simple competitive binding. The results are expressed as pK _i , where pK _i = -log ₁₀ (K _i) [1].
Cell Research	For the experiments, the cells are plated in 96-well plates at a density of 2 \times 10 ⁵ cells per cm ² and cultured in the presence of TBBPA, in concentrations ranging from 1 nM to 100 μ M TBBPA. TBBPA is dissolved in DMSO, resulting in a final vehicle concentration of 0.1 % (v/v). Control (no vehicle) and DMSO-treated wells are included in the experimental design to determine the effect of DMSO. To study whether PPAR- γ is involved in the neurotoxic effect of TBBPA, cells are co-treated with 10 μ M TBBPA and 10 μ M GW1929 or GW9662. After 6 or 24 h of culture, 100 μ L medium is collected for the LDH analysis, and the cells are collected and frozen at -70°C for the caspase-3 activity measurements [2].

Animal Research	Animals are housed at 72°F and 50% relative humidity with a 12-h light and dark cycle and fed Formulab Diet 5008. Age- (60-day) and glucose-matched male Zucker diabetic fatty rats are gavaged twice daily for 14 days with vehicle (0.05 M N-methylglucamine), GW1929 (0.5, 1.0, or 5.0 mg/kg), or troglitazone (as the milled extrudate, in a suspension in methylcellulose, 50, 150, and 500 mg/kg). Another group of animals receives a mixture of Humulin N and Humulin R by subcutaneous injection twice daily. On days 7 and 14 of dosing, nonfasted measurements of glucose, lactate, insulin, total cholesterol, TGs, F FAs, and hematocrit are obtained. On day 14 of dosing, samples for serum drug levels (2-h postdose) and glycosylated hemoglobin measurements are also collected. In addition, once weekly, three animals from each group are placed in metabolic chambers for 48 h for quantitation of 24-h food and water consumption. Body weights are recorded throughout the study. At the conclusion of the study, perfused pancreas experiments are performed on 12 animals (n = 4 per group) that have received either GW1929 (1 and 5 mg/kg) or vehicle, to directly evaluate the effects of treatment on basal and glucose-stimulated insulin secretion. The remaining animals are killed, and their pancreases are processed for immunocytochemistry [1].
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Solubility Information

Solubility	DMSO: 33 mg/mL (66.59 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 2 mg/mL (4.04 mM),Sonication is recommended. <i>Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.</i>

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.0179 mL	10.0894 mL	20.1788 mL
5 mM	0.4036 mL	2.0179 mL	4.0358 mL
10 mM	0.2018 mL	1.0089 mL	2.0179 mL
50 mM	0.0404 mL	0.2018 mL	0.4036 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

- Brown KK, et al. A novel N-aryl tyrosine activator of peroxisome proliferator-activated receptor-gamma reverses the diabetic phenotype of the Zucker diabetic fatty rat. *Diabetes*. 1999 Jul;48(7):1415-24.
- Wojtowicz AK, et al. PPAR- γ agonist GW1929 but not antagonist GW9662 reduces TBBPA-induced neurotoxicity in primary neocortical cells. *Neurotox Res*. 2014 Apr;25(3):311-22.

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