GW1929 hydrochloride

Cat. No.: HY-110022

CAS No.: 1217466-21-1

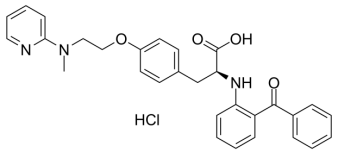
Molecular Formula: $C_{30}H_{30}ClN_3O_4$

Molecular Weight: 532.03

Target: PPAR

Pathway: Cell Cycle/DNA Damage

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	GW1929 hydrochloride is an orally active peroxisome proliferator-activated receptor-γ (PPARγ) agonist with a pK_i of 8.84 for human PPAR-γ, and pEC_{50}s of 8.56 and 8.27 for human PPAR-γ and murine PPAR-γ, respectively. GW1929 hydrochloride has antidiabetic efficacy and neuroprotective potential. GW1929 hydrochloride suppresses neuronal apoptosis and shows anti-inflammatory potential[1][2][3].
IC_{50} & Target	hPPARγ 8.84 (pKi)
In Vitro	GW1929 hydrochloride is a potent PPAR-γ activator, with pK_is of 8.84, < 5.5, and < 6.5 for human PPAR-γ, PPAR-α, and PPAR-δ, and pEC_{50}s of 8.56 and 8.27 for human PPAR-γ and murine PPAR-γ, respectively[1]. GW1929 hydrochloride (10 μM) inhibits TBBPA-induced caspase-3 increase and TBBPA-stimulated LDH release in neocortical cell cultures[2].GW1929 hydrochloride shows significant reduction in the COX-2, iNOS, MMP-9, TNFα and IL-6 levels[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	GW1929 hydrochloride (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[1]. GW1929 hydrochloride (1, 5 mg/kg, p.o.) increases glucose-stimulated insuline secretion of β-cell in ZDF rats[1].GW1929 hydrochloride (10 mg/kg body weight) results in amelioration of muscle loss in tumour-bearing mice experimental cachexia [4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. 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.

[2]. 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.

[3]. Kaundal RK, et al. Ameliorative effects of GW1929, a nonthiazolidinedione PPARγ agonist, on inflammation and apoptosis in focal cerebral ischemic-reperfusion injury. Curr Neurovasc Res. 2011 Aug 1;8(3):236-45.

[4]. Moore-Carrasco R, et al. Effects of the PPARgamma agonist GW1929 on muscle wasting in tumour-bearing mice. Oncol Rep. 2008 Jan;19(1):253-6.

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