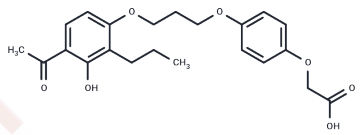
Data Sheet (Cat.No.T7159)

TargetMol

L-165041

Chemical Properties



CAS No. : 79558-09-1

Formula: C22H26O7

Molecular Weight: 402.44

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	L-165041 is a potent and selective agonist of the nuclear receptor PPARβ and PPARδ(Ki = 9 nM, EC50 = ~500 nM ,respectively)
Targets(IC50)	PPAR
In vitro	L-165041 inhibited VEGF-induced cell proliferation and migration in human umbilical vein ECs (HUVECs). L-165041 also inhibited angiogenesis in the Matrigel plug assay and aortic ring assay. Flow cytometric analysis indicated that L-165041 reduced the number of ECs in the S phase and the expression levels of cell cycle regulatory proteins such as cyclin A, cyclin E, CDK2, and CDK4; phosphorylation of the retinoblastoma protein was suppressed by pretreatment with L-165041. The PPARδ ligand L-165041 inhibits VEGF-stimulated angiogenesis by suppressing the cell cycle progression independently of PPARδ[1].
In vivo	L-165041 lowered hepatic expression of PPARgamma, apolipoprotein B, interleukin 1 beta (IL-1beta), and interleukin-6. In contrast, L-165041 increased hepatic expressions of PPARdelta, lipoprotein lipase (LPL), and ATP-binding cassette transporter G1 (ABCG1). L-165041 might be effective in preventing Western diet-induced hepatic steatosis by regulating genes involved in lipid metabolism and the inflammatory response[2].
Cell Research	Cell cycle distribution was determined by flow cytometry. Synchronized HUVECs were pretreated with L-165041 (1 or 5μM) 6 h prior to the addition of VEGF (10 ng/ml). The cells were harvested 16 h after VEGF addition and washed with PBS. The cells were then incubated with buffer containing 0.1% Triton X-100 and 0.1% trisodium citrate for 30 min. Cells were rinsed with PBS and then stained with 50 μg/ml propidium iodide for 20 min at room temperature. In total, 1*10^4 cells were analyzed with the FACScan system . At least three independent experiments were performed[1].
Animal Research	The effect of PPARdelta ligand L-165041 on Western diet-induced fatty liver using low-density lipoprotein receptor-deficient (LDLR(-/-)) mice. LDLR(-/-) mice received either L-165041 (5mg/kg/day) or vehicle (0.1N NaOH) with Western diet for 16 weeks. L-165041 drastically reduced lipid accumulation in the liver, decreasing total hepatic cholesterol and triglyceride content compared to the vehicle group[1].

Solubility Information

Solubility	DMSO: 50 mg/mL (124.24 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 (4.97 mM),Sonication is recommended. *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.*

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.4848 mL	12.4242 mL	24.8484 mL
5 mM	0.497 mL	2.4848 mL	4.9697 mL
10 mM	0.2485 mL	1.2424 mL	2.4848 mL
50 mM	0.0497 mL	0.2485 mL	0.497 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

Park J H , Lee K S , Lim H J , et al. The PPARδ ligand L-165041 inhibits vegf-induced angiogenesis, but the antiangiogenic effect is not related to PPARδ[J]. Journal of Cellular Biochemistry, 2012, 113(6):1947-1954.
Lim H J , Park J H , Lee S , et al. PPARδ ligand L-165041 ameliorates Western diet-induced hepatic lipid accumulation and inflammation in LDLR?/? mice[J]. European Journal of Pharmacology, 2009, 622(1-3):45-51.