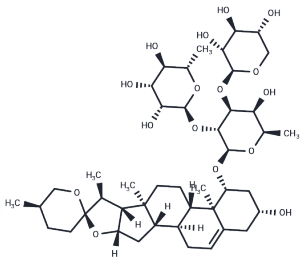
Data Sheet (Cat.No.T6S1255)

TargetMol

Ophiopogonin-D

Chemical Properties



CAS No. : 945619-74-9

Formula: C44H70O16

Molecular Weight: 855.02

Storage: Keep away from direct sunlight,Keep away from moisture
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	Ophiopogonin D is a natural product,and is a CYP2J3 inducer that significantly inhibits Ang II induced NF-κB nuclear translocation. Ophiopogonin-D significantly inhibited the in vitro and in vivo growth of prostate cells via RIPK1. Ophiopogonin-D may be developed as a potential anti-prostate cancer agent.
Targets(IC50)	RAAS,ERK,Calcium Channel,NF-κB,PPAR,ROS
In vitro	Ophiopogonin D(OPD) was shown to exert potent anti-tumor activity against PC3 cells.?It induced apoptosis via a RIPK1-related pathway, increased the protein expression levels of RIPK1 and Bim, and decreased the levels of cleaved-RIPK1, caspase 8, cleaved-caspase 8, Bid, caspase 10, and cleaved-caspase 10.?OPD' also increased the mRNA expression of Bim.?The protein expression of Bim was decreased when cells were pre-treated with necrostatin-1[1]. OPHIOPOGONIN D(OP-D) significantly inhibited Ang II induced NF-κB nuclear translocation, IκBα down-regulation and activation of pro-inflammatory cytokines (TNF-α, IL-6 and VCAM-1) by increasing the expression of CYP2J2/EETs and PPARα in HUVECs.?Furthermore, treatment with exogenous 11,12-EET attenuated endothelial inflammation induced by Ang II as evidenced by inhibited NF-κB nuclear translocation, increased IκBα expression and decreased inflammation factor level.?Finally, the activation of NF-κB nuclear translocation induced by Ang II was also markedly suppressed by fenofibrate.?Co-incubation with 6-(2-proparglyloxyphenyl) hexanoic acid (PPOH) and PPARα inhibitor GW6471 before drug treatment abolished the endothelium protective effects of OP-D.?Suggest that OP-D has the endothelial protective effect through activation of CYP2J and increasing EETs, and PPARα involves in this process.
In vivo	Ophiopogonin D(OPD)' treatment led to significant tumor growth inhibition at a dose of 5.0 mg/kg bodyweight, beginning on Day 6 of treatment (p = 0.034).?At the end of the study (Day 24), the tumor tissues were excised, photographed, and weighed.?Treatment with 5.0 mg/kg bodyweight OPD' resulted in significant (p = 0.000) tumor growth inhibition by approximately 79.8% on Day 24 compared to the vehicle treatment.?The 5.0 mg/kg dose led to significantly stronger tumor growth inhibition than the 2.5 mg/kg dose (p = 0.000).?There was not a significant loss of body weight in any of the groups[1].
Cell Research	The CCK-8 assay was used to assess the viability of prostate cancer cells.?The cell morphology was examined by an ultrastructural analysis via transmission electron

Cell Research	microscopy.?Cells in apoptosis (early and late stages) were detected using an Annexin V-FITC/propidium iodide kit with a FACSCaliber flow cytometer.?JC-1, a cationic lipophilic probe, was employed to measure the mitochondrial membrane potential (MMP) of PC3 cells.?Changes in the protein expression of RIPK1, C-RIPK1, caspase 8, cleaved-caspase 8, Bim, Bid, caspase 10, and cleaved-caspase 10 were evaluated by Western blotting.? The mRNA expression of Bim was examined by quantitative real-time reverse transcription polymerase chain reaction.?Z-VAD-FMK (a caspase inhibitor) and necrostatin-1 (a specific inhibitor of RIPK1) were utilized to determine whether the cell death was mediated by RIPK1 or caspases[1].
Animal Research	One week after tumor cell inoculation, mice bearing palpable tumors were randomly divided into control and treatment groups (8 mice/group).?OPD′ was dissolved in the vehicle, PEG400:Saline:Ethanol (400:300:200, v/v/v), and administered (via i.p. injection) at doses of 2.5 or 5.0 mg/kg bodyweight 5 days a week for 24 days.?The control group received vehicle only.?The mice were sacrificed by cervical dislocation on Day 24, and the tumor tissues were removed and weighed[1].

Solubility Information

Solubility	DMSO: 140 mg/mL (163.74 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+90% Saline: 10 mg/mL (11.7 mM),Suspension. 10% DMSO+90% Corn Oil: 2 mg/mL (2.34 mM) *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	1.1696 mL	5.8478 mL	11.6956 mL
5 mM	0.2339 mL	1.1696 mL	2.3391 mL
10 mM	0.117 mL	0.5848 mL	1.1696 mL
50 mM	0.0234 mL	0.117 mL	0.2339 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

Zongliang L , He W , Mingxing Z , et al. Ophiopogonin D′, a Natural Product From Radix Ophiopogonis, Induces in Vitro and in Vivo RIPK1-Dependent and Caspase-Independent Apoptotic Death in Androgen-Independent Human Prostate Cancer Cells[J]. Frontiers in Pharmacology, 2018, 9:432-.

Ma X Y , Wen X X , Yang X J , et al. Ophiopogonin D improves osteointegration of titanium alloy implants under diabetic conditions by inhibition of ROS overproduction via Wnt/β-catenin signaling pathway[J]. Biochimie, 2018.

Huang X , Wang Y , Zhang Z , et al. Ophiopogonin D and EETs ameliorate Ang II-induced inflammatory responses via activating PPARα in HUVECs[J]. Biochemical and Biophysical Research Communications, 2017: S0006291X17311166.