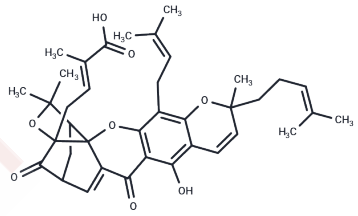
Data Sheet (Cat.No.T6185)

TargetMol

Gambogic Acid

Chemical Properties



CAS No. : 2752-65-0

Formula: C38H44O8

Molecular Weight: 628.75

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	Gambogic Acid (Guttic Acid) (EC50=0.78-1.64 uM) activates caspases. Gambogic Acid competitively suppresses Bcl-XL, Bcl-2, Bcl-W, Bcl-B, Bfl-1 and Mcl-1. The IC50s of Gambogic Acid for Bcl-XL, Bcl-2, Bcl-W, Bcl-B, Bfl-1 and Mcl-1 are 1.47, 1.21, 2.02, 0.66, 1.06 and 0.79 uM, respectively.
Targets(IC50)	Bcl-2 Family,Autophagy
In vitro	Gambogic Acid is a caged xanthone that is derived from Garcinia hanburyi and functions as a strong apoptotic inducer in many types of cancer cells by inhibiting human Bcl-2 family proteins and activating caspases. Gambogic Acid also blocks Kir2.1 channels with EC50 of ≤ 100 nM.[1] [2] [3] Gambogic Acid significantly inhibits human umbilical vein endothelial cell (HUVEC) proliferation, migration, invasion, tube formation, and micro-vessel growth at nM concentration. [4]
In vivo	Gambogic Acid effectively inhibits tumor angiogenesis and suppressed tumor growth with low side effects using metronomic chemotherapy with Gambogic Acid. [4] Gambogic Acid has multiple functional effects including the induction of apoptosis, the inhibition of proliferation and the prevention of cancer metastasis and tumor angiogenesis. [5] In both animal tumor models and Clinicalal trials, Gambogic Acid efficiently inhibits tumor growth with minimal side effects, with little toxicity on immune and hemopoietic systems. Gambogic Acid can produce tissue-specific proteasome inhibition and tumor-specific toxicity. [6] LD50: Mice 45 mg/kg (i.p.). [7]
Kinase Assay	The fluorescence polarization reactions are performed. For Kidetermination, duplicate 200 μL reactions are set up at eight different ATP concentrations from 200 μM (2-fold serial dilutions) in the presence of either DMSO or R406 at 125, 62.5, 31.25, 15.5, or 7.8 nM. At different time points, 20 μL of each reaction is removed and quenched to stop the reaction. For each concentration of R406, the rate of reaction at each concentration of ATP is determined and plotted against the ATP concentration to determine the apparent Km and Vmax (maximal rate). Finally the apparent Km (or apparent Km/Vmax) is plotted against the inhibitor concentration to determine the Ki. All data analysis is performed using Prism and Prism enzyme kinetics programs[1]

Solubility Information

Solubility	H2O: Insoluble DMSO: 50 mg/mL (79.52 mM),Sonication and heating are recommended. Ethanol: 62.9 mg/mL (100.04 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 (3.18 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	1.5905 mL	7.9523 mL	15.9046 mL
5 mM	0.3181 mL	1.5905 mL	3.1809 mL
10 mM	0.159 mL	0.7952 mL	1.5905 mL
50 mM	0.0318 mL	0.159 mL	0.3181 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

Zhai D, et al. Mol Cancer Ther, 2008, 7(6), 1639-1646.
Liu X, Hu Q, Wang W, et al. A protein-fragment complementation assay reveals that celastrol and gambogic acid suppress ERα mutants in breast cancer. Biochemical Pharmacology. 2021, 188: 114583.
Zhang HZ, et al. Bioorg Med Chem, 2004, 12(2), 309-317.
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Yi T, et al. Cancer Res. 2008 Mar 15;68(6):1843-50.
Wang X, et al. Anticancer Agents Med Chem, 2012, 12(8), 1994-12000.
Guo Q, et al. Toxicological studies of gambogic acid and its potential targets in experimental animals. Basic Clin Pharmacol Toxicol. 2006;99(2):178-184.
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