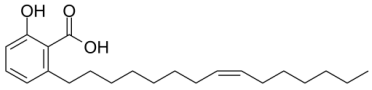
Data Sheet

Product Name:	Ginkgolic Acid
Cat. No.:	CS-3728
CAS No.:	22910-60-7
Molecular Formula:	C22H34O3
Molecular Weight:	346.50
Target:	E1/E2/E3 Enzyme
Pathway:	Metabolic Enzyme/Protease
Solubility:	H2O : < 0.1 mg/mL (insoluble); DMSO : ≥ 100 mg/mL (288.60 mM)



BIOLOGICAL ACTIVITY:

Ginkgolic Acid is a natural compound that inhibits **SUMOylation** with an **IC_{50}** of 3.0 μM in in vitro assay. IC50 & Target: IC50: 3.0 μM (SUMOylation)[1] **In Vitro:** Ginkgolic acid inhibits the in vitro SUMOylation of RanGAP1-C2 with the IC_{50} values of 3.0 μM. The level of SUMOylated p53 is markedly reduced by the ginkgolic acid treatment. Importantly, ginkgolic acid does not affect protein ubiquitination in cells. Ginkgolic acid inhibits the binding between E1 and GA-BODIPY in a dose-dependent manner[1]. Ginkgolic acid (31.2 μg/mL) inhibits HIV protease activity by 60%, compared with the negative control, and the effect is concentration-dependent. Ginkgolic acid treatment (50 and 100 μg/mL) effectively inhibits HIV infection in human PBMC cells. Ginkgolic acid at the concentrations up to 150 μg/mL does not cause any significant cytotoxicity in Jurkat cells[2]. GA only inhibits the growth of tumorogenic cell lines in a both dose- and time-dependent manner. Tumor cells are treated with GA for 72 h, 70.53±4.54% Hep-2 and 63.5±7.2% Tca8113 cells are retarded at GO/G1 phase, and the percentage of apoptosis is 40.4±1.58 and 38.4±1.7%, respectively. GA-treated activated caspase-3 downregulates the expression of anti-apoptotic Bcl-2 protein and upregulates the expression of pro-apoptotic Bax protein, eventually leading to a decrease in the Bcl-2/Bax ratio in tumor cellsin human PBMC cells. Ginkgolic acid at the concentrations up to 150 μg/mL does not cause any significant cytotoxicity in Jurkat cells[3].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [2]Jurkat cells (106 cells/mL) are cultured in the RPMI medium with or without different concentrations of ginkgolic acid for 48 hours to test the cytotoxicity of ginkgolic acid. The cytotoxicity of ginkgolic acid is determined using a tetrazolium compound (MTS) and an electron coupling reagent (PMS). MTS is chemically reduced by cells into formazan, which is soluble in the tissue culture medium. The measurement of the absorbance of the formazan can be carried out using 96 well microplates at 492 nm. Since the production of formazan is proportional to the number of living cells, the intensity of the produced color is a good indication of the viability of the cells.

References:

[1]. Fukuda I, et al. Ginkgolic acid inhibits protein SUMOylation by blocking formation of the E1-SUMO intermediate. Chem Biol. 2009 Feb 27;16(2):133-40.

[2]. Lü JM, et al. Ginkgolic acid inhibits HIV protease activity and HIV infection in vitro. Med Sci Monit. 2012 Aug;18(8):BR293-298.

[3]. Zhou C, et al. Antitumor effects of ginkgolic acid in human cancer cell occur via cell cycle arrest and decrease the Bcl-2/Bax ratio to induce apoptosis. Chemotherapy. 2010;56(5):393-402.

[4]. Qiu F, et al. Pharmacological inhibition of SUMO-1 with ginkgolic acid alleviates cardiac fibrosis induced by myocardial infarction in mice. Toxicol Appl

Pharmacol. 2018 Apr 15;345:1-9.

CAIndexNames:

Benzoic acid, 2-hydroxy-6-(8Z)-8-pentadecenyl-

SMILES:

O=C(O)C1=C(CCCCCCC/C=C\CCCCCC)C=CC=C1O

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