Ginkgolic Acid

Cat. No.: HY-N0077
CAS No.: 22910-60-7
Molecular Formula: C₂₂H₃₄O₃
Molecular Weight: 346.5
Target: E1/E2/E3 Enzyme
Pathway: Metabolic Enzyme/Protease
Storage: Powder -20°C 3 years
        4°C 2 years
        In solvent -80°C 6 months
        -20°C 1 month

SOLVENT & SOLUBILITY

In Vitro
DMSO: ≥ 100 mg/mL (288.60 mM)
H₂O: < 0.1 mg/mL (insoluble)
* “≥” means soluble, but saturation unknown.

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Solvent</th>
<th>Mass</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1 mg</td>
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<tr>
<td>1 mM</td>
<td></td>
<td>2.8860 mL</td>
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<tr>
<td>5 mM</td>
<td></td>
<td>0.5772 mL</td>
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<tr>
<td>10 mM</td>
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<td>0.2886 mL</td>
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</tbody>
</table>

Please refer to the solubility information to select the appropriate solvent.

In Vivo
1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.75 mg/mL (7.94 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.75 mg/mL (7.94 mM); Clear solution

BIOLOGICAL ACTIVITY

Description
Ginkgolic Acid is a natural compound that inhibits SUMOylation with an IC₅₀ 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 IC₅₀ 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, 70.5±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 cells in human PBMC cells. Ginkgolic acid at the concentrations up to 150 μg/mL does not cause any significant cytotoxicity in Jurkat cells[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

**PROTOCOL**

**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.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

**CUSTOMER VALIDATION**

- Scienceasia. 2020 Dec.

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**REFERENCES**


Caution: Product has not been fully validated for medical applications. For research use only.

Tel: 609-228-6898  Fax: 609-228-5909  E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA