Urolithin A

Cat. No.: HY-100599

CAS No.: 1143-70-0

Molecular Formula: $C_{13}H_8O_4$

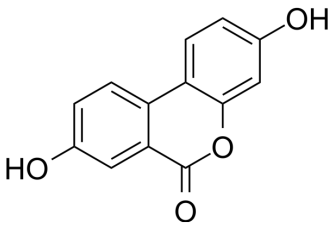
Molecular Weight: 228.2

Target: Drug Metabolite; Reactive Oxygen Species; DNA/RNA Synthesis; Autophagy; Apoptosis; Endogenous Metabolite

Pathway: Metabolic Enzyme/Protease; Immunology/Inflammation; NF-κB; Cell Cycle/DNA Damage; Autophagy; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 30 mg/mL (131.46 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	4.3821 mL	21.9106 mL	43.8212 mL
	5 mM	0.8764 mL	4.3821 mL	8.7642 mL
	10 mM	0.4382 mL	2.1911 mL	4.3821 mL

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

In Vivo

1. Add each solvent one by one: 0.5% CMC/saline water
   Solubility: 5 mg/mL (21.91 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (10.96 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (10.96 mM); Suspended solution; Need ultrasonic
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.96 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Urolithin A, a gut-microbial metabolite of ellagic acid, exerts anti-inflammatory, antiproliferative, and antioxidant properties. Urolithin A induces autophagy and apoptosis, suppresses cell cycle progression, and inhibits DNA synthesis[1][2].

IC_{50} & Target	Microbial Metabolite
In Vitro	Micromolar urolithin A concentrations induces both autophagy and apoptosis. Urolithin A suppresses cell cycle progression and inhibited DNA synthesis in human sw620 colorectal cancer cells[2]. Urolithin A shows antiproliferative effects and inhibits T24 and Caco-2 cell growth with IC_{50}s of 43.9 and 49 μM, respectively [3]. Urolithin A exerts a dose- and time-dependent significant arrest at G2/M and S phases after treatments with 50 and 100 μM at 24 and 48 h compared to control cells. It induces cell apoptosis with 50 and 100 μM [4]. Urolithin A shows potent antiproliferative activity on HepG2 cells. When cell death is induced by Urolithin A, the expression of β-catenin, c-Myc and Cyclin D1 are decreased and TCF/LEF transcriptional activation is notably down-regulated. Urolithin A also increases protein expression of p53, p38-MAPK and caspase-3, but suppresses expression of NF-κB p65 and other inflammatory mediators[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	The volume of paw edema is reduced at 1 h after oral administration of urolithin A. In addition, plasma in treated mice exhibited significant oxygen radical antioxidant capacity (ORAC) scores with high plasma levels of the unconjugated form at 1 h after oral administration of urolithin A[6]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	Human colon cancer cells HT-29 are treated for 24 and 48 h at 100 and 50 μM of Urolithin A and Iso Urolithin A aglycones and their glucuronide conjugates. Cell viability and proliferation are measured using a TC10 automated cell counter with the addition of Trypan blue for viability determination. IC_{50} values are determined by MTT assay[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [4]	Mice: Paw edema is induced in the right hind paw of ICR mice by the subcutaneous injection of 1% λ-carrageenan in pysiological saline (50 μL). The inflammation level is quantified by the volume of paw edema. Urolithin A dissolved in 0.5% carboxymethylcellulose suspension is orally administered to the mice at 1 or 6 h before carrageenan injection. The anti-inflammatory effects of urolithin A on carrageenan-induced edema in mice are analyzed[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Nanobiotechnology. 2022 Mar 19;20(1):149.
- Cell Death Dis. 2023 May 24;14(5):339.
- J Headache Pain. 2023 Sep 5;24(1):122.
- Commun Biol. 2022 Jun 22;5(1):616.
- Radiother Oncol. 2023 Nov 23:110028.

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REFERENCES

[1]. Gong Z, et al. Urolithin A attenuates memory impairment and neuroinflammation in APP/PS1 mice.

[2]. Zhao W, et al. Metabolite of ellagitannins, urolithin A induces autophagy and inhibits metastasis in human sw620colorectal cancer cells. Mol Carcinog. 2018 Feb;57(2):193-200.

[3]. Qiu Z, et al. In vitro antioxidant and antiproliferative effects of ellagic acid and its colonic metabolite, urolithins, on human bladder cancer T24 cells. Food Chem Toxicol. 2013 Sep;59:428-37.

[4]. González-Sarrías A, et al. Antiproliferative activity of the ellagic acid-derived gut microbiota isourolithin A and comparison with its urolithin A isomer: the role of cell metabolism.Eur J Nutr. 2017 Mar;56(2):831-841.

[5]. Wang Y, et al. In vitro antiproliferative and antioxidant effects of urolithin A, the colonic metabolite of ellagic acid, on hepatocellular carcinomas HepG2 cells. Toxicol In Vitro. 2015 Aug;29(5):1107-15.

[6]. Ishimoto H, et al. In vivo anti-inflammatory and antioxidant properties of ellagitannin metabolite urolithin A. Bioorg Med Chem Lett. 2011 Oct 1;21(19):5901-4.

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