# **Product** Data Sheet

## **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

Metabolic Enzyme/Protease; Immunology/Inflammation; NF-κΒ; Cell Cycle/DNA Pathway:

Damage; Autophagy; Apoptosis

-20°C Storage: Powder 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	Solvent Mass 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<sup>[1][2]</sup>.

IC <sub>50</sub> & 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 <sup>[2]</sup> . Urolithin A shows antiproliferative effects and inhibits T24 and Caco-2 cell growth with IC <sub>50</sub> 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 $^{[5]}$ .
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 <sup>[6]</sup> .  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  $\mu$ M 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. IC50 values are determined by MTT assay<sup>[2]</sup>.

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%  $\lambda$ -carrageenan in pysiological saline (50  $\mu$ L). 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<sup>[4]</sup>.

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.

See more customer validations on www.MedChemExpress.com

#### **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.

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

Page 3 of 3 www.MedChemExpress.com