Urolithin C

Cat. No.: HY-135897

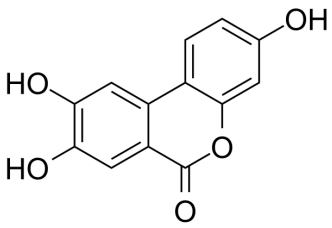
CAS No.: 165393-06-6

Molecular Formula: $C_{13}H_8O_5$

Molecular Weight: 244.2

Target: Calcium Channel; Reactive Oxygen Species; Apoptosis; Endogenous Metabolite

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB; Apoptosis



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 : 50 mg/mL (204.75 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	4.0950 mL	20.4750 mL	40.9500 mL
	5 mM	0.8190 mL	4.0950 mL	8.1900 mL
	10 mM	0.4095 mL	2.0475 mL	4.0950 mL

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: ≥ 2.08 mg/mL (8.52 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (8.52 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Urolithin C, a gut-microbial metabolite of Ellagic acid, is a glucose-dependent activator of insulin secretion. Urolithin C is a L-type Ca^{2+} channel opener and enhances Ca^{2+} influx. Urolithin C induces cell apoptosis through a mitochondria-mediated pathway and also stimulates reactive oxygen species (ROS) formation[1][2].

IC_{50} & Target

Insulin secretion[1]
L-type Ca^{2+} channel[1]
Reactive oxygen species (ROS)[2]
Apoptosis[2]

In Vitro

Urolithin C (20-100 μM; 1 hour; INS-1 cells) treatment enhances glucose-induced extracellular signal-regulated kinases 1/2 (ERK1/2) activation in INS-1 β-cells[1].

Urolithin C significantly inhibits the proliferation of PC12 cells. Urolithin C treatment actively increases the lactate dehydrogenase (LDH) release and lipid peroxidation malondialdehyde (MDA), stimulates reactive oxygen species (ROS) formation and mitochondrial membrane depolarization, and caused calcium dyshomeostasis[2].

Urolithin C treatment induces apoptosis and S phase cell cycle arrest[2].

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

Western Blot Analysis[1]

Cell Line:	INS-1 cells
Concentration:	20 μM, 100 μM
Incubation Time:	1 hour
Result:	Enhanced glucose-induced extracellular signal-regulated kinases 1/2 (ERK1/2) activation.

In Vivo

The pharmacokinetics of Urolithin C (10 mg/kg; intraperitoneal administration) in male Wistar rat (140-160 g) are studied. The half-life of the terminal part is 11.3 h and the total clearance (CL/F) is 3.41 L/h/kg. The initial volume of distribution (V_1/F) and the steady-state volume of distribution (Vss/F) are 0.831 L/kg and 55.6 L/kg, respectively[3].

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

CUSTOMER VALIDATION

- Research Square Preprint. 2021 Oct.

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REFERENCES

[1]. Slimane Toubal, et al. Urolithin C Increases Glucose-Induced ERK Activation Which Contributes to Insulin Secretion. Fundam Clin Pharmacol. 2020 Feb 21.

[2]. Peipei Yin, et al. Urolithin C, a gut metabolite of ellagic acid, induces apoptosis in PC12 cells through a mitochondria-mediated pathway. RSC Advances. Issue 28, 2017.

[3]. Morgane Bayle, et al. Development and Validation of a Liquid Chromatography-Electrospray Ionization-Tandem Mass Spectrometry Method for the Determination of Urolithin C in Rat Plasma and Its Application to a Pharmacokinetic Study. J Pharm Biomed Anal. 201

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