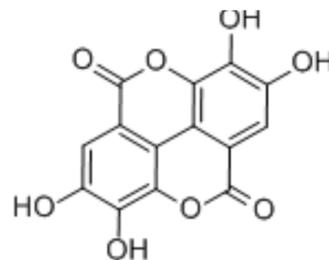


Ellagic acid

Cat. No.:	HY-B0183												
CAS No.:	476-66-4												
Molecular Formula:	C ₁₄ H ₆ O ₈												
Molecular Weight:	302.19												
Target:	Casein Kinase; Reactive Oxygen Species; Endogenous Metabolite; SHP2												
Pathway:	Cell Cycle/DNA Damage; Stem Cell/Wnt; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB; Protein Tyrosine Kinase/RTK												
Storage:	<table border="0"> <tr> <td>Powder</td> <td>-20°C</td> <td>3 years</td> </tr> <tr> <td></td> <td>4°C</td> <td>2 years</td> </tr> <tr> <td>In solvent</td> <td>-80°C</td> <td>1 year</td> </tr> <tr> <td></td> <td>-20°C</td> <td>6 months</td> </tr> </table>	Powder	-20°C	3 years		4°C	2 years	In solvent	-80°C	1 year		-20°C	6 months
Powder	-20°C	3 years											
	4°C	2 years											
In solvent	-80°C	1 year											
	-20°C	6 months											



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 2.5 mg/mL (8.27 mM)
 * "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent	1 mg	5 mg	10 mg
	Concentration	1 mg	5 mg	10 mg
1 mM	3.3092 mL	16.5459 mL	33.0918 mL	
5 mM	0.6618 mL	3.3092 mL	6.6184 mL	
10 mM	---	---	---	

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

BIOLOGICAL ACTIVITY

Description

Ellagic acid is a natural antioxidant, and acts as a potent and ATP-competitive inhibitor of CK2 and SHP2, with an IC₅₀ of 40 nM and a K_i of 20 nM.

IC₅₀ & Target

CK2
 40 nM (IC₅₀)

In Vitro

Ellagic acid is a potent CK2 inhibitor, with an IC₅₀ of 40 nM and a K_i of 20 nM. Ellagic acid also blocks other kinases such as LYN, PKA, SYK, GSK3, FGR and CK1, with IC₅₀s of 2.9, 3.5, 4.3, 7.5, 9.4 and 13.0 μM, respectively, and shows no obvious effects on DYRK1a, CSK, NPM-ALK, RET and FLT3 (IC₅₀s > 40 μM). Ellagic acid (5-100 μM) shows inhibitory activities against Karpas299, SUDHL1, SR786, and FE-PD cell lines^[1]. Ellagic acid (10 μM) exhibits cytotoxic effects on MCF-7 cells after treatment of radiation. Ellagic acid (10 μM) in combination with Irradiation (IR) significantly abridges the capacity of MCF-7 cells to form colonies equated with individual treatments. Ellagic acid with IR also induces cell apoptosis, and facilitates the upregulation of pro-apoptotic Bax and downregulation of Bcl-2 in MCF-7 cells^[3].

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

In Vivo

Ellagic acid (EA; 10 mg/kg/day; p.o., 14 days) strongly decreases MDA brain content by 17%, and reduces the levels of brain TNF- α by 42% in rats. Ellagic acid markedly increases the reduced brain contents of 5-HT (39%), dopamine (DA, 71%), and norepinephrine (NE, 77%). Ellagic acid (10 mg/kg, p.o., 14 days) causes decreased histopathological changes induced by Doxorubicin in rats^[2].

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

PROTOCOL

Kinase Assay ^[1]

CK2 and CK1 phosphorylation assays are carried out at 37°C in the presence of increasing amounts of each inhibitor (Ellagic acid) in a final volume of 25 μ L containing 50 mM, Tris-HCl pH 7.5, 100 mM NaCl, 12 mM MgCl₂, 0.02 mM [³³P-ATP] (500-1000 cpm/pmol), unless otherwise indicated. The phosphorylatable substrates are the synthetic peptide substrate RRRADSDDDDD (100 μ M) and RRKHAAGDDDDAYSITA (200 μ M) for CK2 and CK1, respectively. Reaction started with the addition of the kinase and is stopped after 10 min. by addition of 5 μ L of 0.5 M orthophosphoric acid before spotting aliquots onto phosphocellulose filters. Filters are washed in 75 mM phosphoric acid substrate following SDS-PAGE of the radiolabeled samples. DYRK1A, assayed on the peptide RRRFRPASPLRGPPK, and tyrosine kinase activities are determined^[1].

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

Cell Assay ^[1]

ALCL cell viability is measured by MTT assay. Briefly, 0.1×10^5 cells are seeded onto 96-well microculture plates 12 hrs before adding ellagic acid. The cells are grown in 200 μ L of complete RPMI-1640 medium, under standard tissue-culture conditions, in the presence or absence of the drug (Ellagic acid) for 48 hours. Twenty μ L of MTT solution (5 mg/mL) are then added to the cell suspension for 4h. The intracellular formazan crystals are dissolved with 150 μ L of DMSO and optical density, measured on a spectrophotometer at 540 nm, represents the mean (\pm SD) of triplicate cultures^[1].

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Animal Administration ^[2]

Fifty male adult Sprague-Dawley rats are divided randomly into five groups as follow: Group (1) receives corn oil orally as a vehicle and served as normal control. Group (2) receives doxorubicin (DOX) injection (5 mg/kg, i.p.) twice a week for 14 days. Group (3) receives Ellagic acid (10 mg/kg, p.o.; daily) for 14 days and DOX (5 mg/kg, i.p.) twice a week for 14 days. Group (4) receives rosmarinic acid (RA; 75 mg/kg, p.o.; daily) for 14 days and DOX (5 mg/kg, i.p.) twice a week for 14 days. Group (5) receives Ellagic acid (10 mg/kg, p.o.; daily) with RA (75 mg/kg, p.o.; daily) for 14 days and DOX injection (5 mg/kg, i.p.) twice a week for 14 days^[2].

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

CUSTOMER VALIDATION

- Acta Pharm Sin B. 2023 Mar 25.
- Molecules. 2022, 27(19), 6168.
- Saudi J Biol Sci. 2023 Jun 15, 103707.
- Heliyon. 2023 Dec 18.
- Biosci Rep. 2020 Oct 30;40(10):BSR20201349.

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- [1]. Ma CH, et al. Discovery of ellagic acid as a competitive inhibitor of Src homology phosphotyrosyl phosphatase 2 (SHP2) for cancer treatment: In vitro and in silico study. *Int J Biol Macromol.* 2023 Nov 5;254(Pt 2):127845.
- [2]. Cozza G, et al. Identification of ellagic acid as potent inhibitor of protein kinase CK2: a successful example of a virtual screening application. *J Med Chem.* 2006 Apr 20;49(8):2363-6.
- [3]. Rizk HA, et al. Prophylactic effects of ellagic acid and rosmarinic acid on doxorubicin-induced neurotoxicity in rats. *J Biochem Mol Toxicol.* 2017 Dec;31(12).
- [4]. Ahire V, et al. Ellagic Acid Enhances Apoptotic Sensitivity of Breast Cancer Cells to γ -Radiation. *Nutr Cancer.* 2017 Aug-Sep;69(6):904-910.
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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