Ellagic acid

Cat. No.: HY-B0183

CAS No.: 476-66-4

Molecular Formula: $C_{14}H_6O_8$

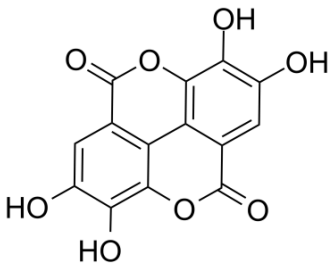
Molecular Weight: 302.19

Target: Casein Kinase; Reactive Oxygen Species; Endogenous Metabolite

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

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 : 3.33 mg/mL (11.02 mM; Need ultrasonic)
Ethanol : < 1 mg/mL (insoluble)
H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / 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	0.3309 mL	1.6546 mL	3.3092 mL

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 CK2 inhibitor, with an IC_{50} of 40 nM and a K_i of 20 nM.

IC_{50} & Target

CK2
40 nM (IC_{50})

Human Endogenous Metabolite

In Vitro

Ellagic acid is a potent CK2 inhibitor, with an IC_{50} 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 IC_{50}s 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 (IC_{50}s > 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-apopttotic 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 µL containing 50 mM, Tris-HCl pH 7.5, 100 mM NaCl, 12 mM $MgCl_2$, 0.02 mM [^{33}P-ATP] (500-1000 cpm/pmol), unless otherwise indicated. The phosphorylatable substrates are the synthetic peptide substrate RRRADDSDDDDD (100 µM) and RRKHAAIGDDDDAYSITA (200 µM) for CK2 and CK1, respectively. Reaction started with the addition of the kinase and is stopped after 10 min. by addition of 5 µL 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 µL of complete RPMI-1640 medium, under standard tissue-culture conditions, in the presence or absence of the drug (Ellagic acid) for 48 hours. Twenty µL of MTT solution (5 mg/mL) are then added to the cell suspension for 4h. The intracellular formazan crystals are dissolved with 150 µL of DMSO and optical density, measured on a spectrophotometer at 540 nm, represents the mean (± SD) of triplicate cultures[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
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

- Biochem Biophys Res Commun. 2018 Sep 3;503(1):297-303.
- Biosci Rep. 2020 Oct 30;40(10):BSR20201349.
- Patent. US20200368248A1.

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REFERENCES

[1]. 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.

[2]. 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).

[3]. 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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