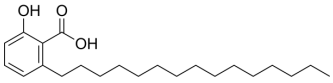
Anacardic Acid

Cat. No.: HY-N2020

CAS No.: 16611-84-0

Molecular Formula: $C_{22}H_{36}O_3$

Molecular Weight: 348.52



Target: Histone Acetyltransferase; Epigenetic Reader Domain; Bacterial

Pathway: Epigenetics; Anti-infection

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 : 100 mg/mL (286.93 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8693 mL	14.3464 mL	28.6928 mL
	5 mM	0.5739 mL	2.8693 mL	5.7386 mL
	10 mM	0.2869 mL	1.4346 mL	2.8693 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 10 mg/mL (28.69 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 (7.17 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.17 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Anacardic Acid, extracted from cashew nut shell liquid, is a histone acetyltransferase inhibitor, inhibits HAT activity of p300 and PCAF, with IC_{50}s of -8.5 μM and -5 μM, respectively.

IC_{50} & Target

p300-HAT	PCAF
8.5 μM (IC_{50})	5 μM (IC_{50})

In Vitro

Anacardic Acid is a histone acetyltransferase, inhibits HAT activity of p300 and PCAF, with IC_{50}s of ⊠8.5 μM and ⊠5 μM,

	respectively[1]. Anacardic Acid (300 μM) inhibits mycelial growth. Anacardic Acid (50 μM) induces apoptosis-like characteristics in M. oryzae, and the effect is caspase independent. Anacardic Acid (1-80 μM) leads to loss of mitochondrial potential. Anacardic Acid (1-60 μM) also exhibits antioxidant activity in M. oryzae[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Anacardic acid (5 mg/kg, i.p.) attenuates the binding of HATs to the promoter of MEF2A and reverse hyperacetylation of H3K9ac caused by phenylephrine in C57BL/6 mice. Anacardic acid inhibits the level of transcription on MEF2A and cardiac development-related downstream genes, attenuates the protein overexpression of cardiac downstream genes caused by phenylephrine, reverses and attenuates cardiac hypertrophy in the hearts of mice exposed to phenylephrine, and attenuates the left ventricular pressure and improves cardiac function in the cardiac hypertrophy mice[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [1]	Briefly, indicated amounts of proteins/peptide are incubated in HAT assay buffer containing 50 mM Tris-HCl, pH 8.0, 10% (v/v) glycerol, 1 mM dithiothreitol, 1 mM phenylmethyl sulfonyl fluoride, 0.1 mM EDTA, pH 8.0, 10 mM sodium butyrate at 30°C for 10 min in the presence or absence of compound followed by the addition of 1 μL of 6.2 Ci/mmol [^{3}H]acetyl coenzyme A (acetyl-CoA) and are further incubated for another 10 min. The final reaction volume is 30 μL. The reaction mixture is then blotted onto P-81 filter papers, and radioactive counts are recorded on a Wallac 1409 liquid scintillation counter. To characterize the inhibition kinetics of anacardic acid, filter binding assays are done using a constant amount of HeLa core histones in the presence or absence of AA with increasing concentrations of [^{3}H]acetyl-CoA[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Cell Assay [3]	Mycelial cell death assay is performed to evaluate the number of colony-forming units in treated and untreated samples. M. oryzae conidia (10^6 conidia/mL) are allowed to germinate in 100-mL flasks with 20 mL complete medium broth (CM) at 28°C in a rotary shaker (200 rpm) for 12 h. The cultures are exposed to different concentrations of anacardic acid for 2 h. The germinated conidia are washed with sterile water, diluted to 10^4 conidia/mL, and plated on oat meal agar and incubated at 28°C for 3 days. Colony-forming units (CFUs) are counted in each of the three ndividual experiments performed, and values are plotted in the graph as average of three replicates. The data in each sample is expressed as the percentage of the total number of CFUs observed in untreated or 0.1 % DMSO treated control[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Pathogen-free male and female 11-13 week-old C57BL/6 mice (18-20 g) are randomly selected to inject phenylephrine (20 mg/kg) (control groups receive equivalent normal saline). In some cases, phenylephrine-treated C57BL/6 mice are administered with a Chinese herbal extract anacardic acid (5 mg/kg). Anacardic acid is dissolved in sterile DMSO at a concentration of 1 mg/ml and stored at 4°C. Phenylephrine is administered by a subcutaneous injection at a dose of 20 mg per kg per day continuously for 30 days. Moreover, anacardic acid is administered by an intraperitoneal injection at a dose of 5 mg/kg every 3rd day intraperitoneal injection at a dose of 5 mg/kg every 3rd day. After modeling, mice are euthanized using 20% carbon dioxide in an anesthesia chamber until they are unresponsive to nose pinch and the hearts are isolated[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Sci Total Environ. 2021, 147014.
- Cell Death Dis. 2021 Jun 7;12(6):582.
- Food Chem. 2021, 129696.
- Comput Struct Biotechnol J. 2021;19:4868-4883.
- FASEB J. 2022 Nov;36(11):e22593.

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REFERENCES

[1]. Balasubramanyam K, et al. Small molecule modulators of histone acetyltransferase p300. J Biol Chem. 2003 May 23;278(21):19134-40. Epub 2003 Mar 6.

[2]. Peng C, et al. Phenylephrine-induced cardiac hypertrophy is attenuated by a histone acetylase inhibitor anacardic acid in mice. Mol Biosyst. 2017 Mar 28;13(4):714-724.

[3]. Muzaffar S, et al. Anacardic acid induces apoptosis-like cell death in the rice blast fungus Magnaporthe oryzae. Appl Microbiol Biotechnol. 2016 Jan;100(1):323-35.

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