Damnacanthal

Cat. No.: HY-108485

CAS No.: 477-84-9

Molecular Formula: $C_{16}H_{10}O_5$

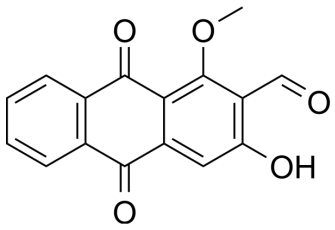
Molecular Weight: 282.25

Target: Apoptosis; Fungal; Src

Pathway: Apoptosis; Anti-infection; Protein Tyrosine Kinase/RTK

Storage: 4°C, protect from light

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 5 mg/mL (17.71 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.5430 mL	17.7148 mL	35.4296 mL
	5 mM	0.7086 mL	3.5430 mL	7.0859 mL
	10 mM	0.3543 mL	1.7715 mL	3.5430 mL

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

In Vivo

1. Add each solvent one by one: 0.5% CMC-Na/saline water
   Solubility: 4 mg/mL (14.17 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 4 mg/mL (14.17 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Damnacanthal is an anthraquinone isolated from the root of Morinda citrifolia. Damnacanthal is a highly potent, selective inhibitor of p56^{lck} tyrosine kinase activity. Natural Damnacanthal inhibits p56 ^{lck} autophosphorylation and phosphorylation of exogenous substrates with IC_{50}s of 46 nM and 220 nM, respectively. Damnacanthal is a potent inducer of apoptosis with anticancer activity. Damnacanthal also has antinociceptive, anti-inflammatory effects in mice and anti-fungal activity against ***Candida albicans***[1][2][3][4].

IC_{50} & Target

IC50: 46 nM (p56 ^{lck} autophosphorylation) and 220 nM (phosphorylation of exogenous substrates by p56 ^{lck})[1];
Apoptosis[2];
Candida albicans[2]

In Vitro

Damnacanthal has > 100-fold selectivity for p56^{lck} over the serine/threonine kinases, protein kinase A and protein kinase C,

and > 40-fold selectivity for $p56^{lck}$ over four receptor tyrosine kinases. Damnacanthal also demonstrates modest (7-20-fold), but highly statistically significant, selectivity for $p56^{lck}$ over the homologous enzymes $p60^{src}$ and $p59^{fyn}$[1].
Damnacanthal (0.1-100 μM; 1-4 days; HCT-116 and SW480 cells) treatment results in a significant reduction of cell proliferation in a concentration- and time-dependent manner[2].
Damnacanthal (1-50 μM; 72 hours; HCT-116 cells) treatment results in a significant enrichment in the number of cells in the S/G1 and G2/G1 phases at concentration of 50 μM[2].
Damnacanthal (10 μM; 24 hours; HCT-116 cells) treatment significantly increases caspase 3/7 activity. Damnacanthal-induced apoptosis[2].
Damnacanthal (0.1-10 μM; 24 hours; HCT-116 cells) treatment induces NAG-1 expression in HCT-116 cells. Cyclin D1 expression is reduced at 10 μM of Damnacanthal, whereas p21 and p53 does not alter their expression. PARP cleavage is seen at 10 μM Damnacanthal treatment only in HCT-116 cells, where NAG-1 is induced[2].
Damnacanthal treatment for 2 weeks shows significant decreasing colony number in HCT-116 cells in a concentration-dependent manner. Damnacanthal-treated cells show a dramatic inhibition of clonogenic capacity. Damnacanthal-treated (1-50 μM; 48 hours) cells significantly inhibits the migration of HCT-116 cells in a concentration-dependent manner[2].

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

Cell Proliferation Assay[2]

Cell Line:	HCT-116 and SW480 cells
Concentration:	0.1 μM, 1 μM, 10 μM, 100 μM
Incubation Time:	1, 2, and 4 days
Result:	Resulted in a significant reduction of cell proliferation in a concentration- and time-dependent manner.

Cell Cycle Analysis[2]

Cell Line:	HCT-116 cells
Concentration:	1 μM, 10 μM and 50 μM
Incubation Time:	72 hours
Result:	Resulted in a significant enrichment in the number of cells in the S/G1 and G2/G1 phases at concentration of 50 μM.

Apoptosis Analysis[2]

Cell Line:	HCT-116 cells
Concentration:	10 μM
Incubation Time:	24 hours
Result:	Significantly increased caspase 3/7 activity.

Western Blot Analysis[2]

Cell Line:	HCT-116 cells
Concentration:	0.1 μM, 1 μM and 10 μM
Incubation Time:	24 hours
Result:	NAG-1 was induced in HCT-116 cells in a dose- and time-dependent manner. Cyclin D1 expression was reduced at 10 μM.

In Vivo

Damnacanthal (10-100 mg/kg; oral administration; for 10-300 minutes; male ddY mice) treatment exhibits a significant antinociceptive effect in a dose-dependent manner in the formalin test. Administration of damnacanthal (100 mg/kg) shows significant inhibition of histamine-induced paw edema[4].

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Animal Model:	Male ddY mice (5-6 weeks) injected with formalin or Histamine[4]
Dosage:	10 mg/kg, 30 mg/kg and 100 mg/kg
Administration:	Oral administration; for 10 minutes, 30 minutes, 60 minutes or 300 minutes
Result:	Significantly reduced the growth of human lung tumor without acute toxicity.

REFERENCES

[1]. Faltynek CR, et al. Damnacanthal is a highly potent, selective inhibitor of p56lck tyrosine kinase activity. Biochemistry. 1995 Sep 26;34(38):12404-10.

[2]. Nualsanit T, et al. Damnacanthal, a noni component, exhibits antitumorigenic activity in human colorectal cancer cells. J Nutr Biochem. 2012 Aug;23(8):915-23.

[3]. Aziz MY, et al. Damnacanthal is a potent inducer of apoptosis with anticancer activity by stimulating p53 and p21 genes in MCF-7 breast cancer cells. Oncol Lett. 2014 May;7(5):1479-1484.

[4]. Okusada K, et al. The antinociceptive and anti-inflammatory action of the CHCl3-soluble phase and its main active component, damnacanthal, isolated from the root of Morinda citrifolia. Biol Pharm Bull. 2011;34(1):103-7.

Caution: Product has not been fully validated for medical applications. For research use only.

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