Oncrasin-1

Cat. No.: HY-16662

CAS No.: 75629-57-1

Molecular Formula: $C_{16}H_{12}ClNO$

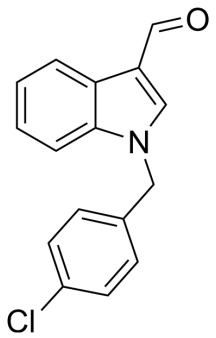
Molecular Weight: 269.73

Target: Ras; Apoptosis

Pathway: GPCR/G Protein; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (370.74 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.7074 mL	18.5371 mL	37.0741 mL
	5 mM	0.7415 mL	3.7074 mL	7.4148 mL
	10 mM	0.3707 mL	1.8537 mL	3.7074 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: 2.5 mg/mL (9.27 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (9.27 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 2.5 mg/mL (9.27 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Oncrasin-1 is a potent and effective anticancer inhibitor that kills various human lung cancer cells with K-Ras mutations at low or submicromolar concentrations; also led to abnormal aggregationof PKCι in nucleus of sensitive cells but not in resistant cells.IC50 value: 1.0 μM(A549, K-ras 12H and p53 Wt) [1]Target: human lung cancer cells with K-Ras mutation; K-Ras/PKCiota pathway inhibitorin vitro: effectively kills various human lung cancer cells with K-Ras mutations at low or submicromolar concentrations. The cytotoxic effects correlated with apoptosis inductionas was evidenced by increase of apoptotic cells and activation of caspase-3 and caspase-8 upon the treatment of oncrasin-1 in sensitive cells.Treatment with oncrasin-1 also led to abnormal aggregationof PKCι in nucleus of sensitive cells but not in resistant cells. Furthermore,

oncrasin-1 induced apoptosis was blocked by siRNA of K-Ras or PKCι suggesting that oncrasin-1 is targeted to a novel K-Ras/PKCι pathway [1]. oncrasin-1 treatment led to coaggregation of PKCiota and splicing factors into megaspliceosomes but had no obvious effects on the DNA repair molecule Rad51. Moreover, oncrasin-1 treatment suppressed the phosphorylation of the largest subunit of RNA polymerase II and the expression of intronless reporter genes in sensitive cells but not in resistant cells [2]. in vivo: The in vivo administration of oncrasin-1 suppressed the growth of K-ras mutant human lung tumor xenografts by >70% and prolonged the survival of nude mice bearing these tumors, without causing detectable toxicity [1].

REFERENCES

[1]. Guo W, et al. Identification of a small molecule with synthetic lethality for K-ras and protein kinase C iota. Cancer Res. 2008 Sep 15;68(18):7403-8.

[2]. Guo W, et al. Interruption of RNA processing machinery by a small compound, 1-[(4-chlorophenyl)methyl]-1H-indole-3-carboxaldehyde (oncrasin-1). Mol Cancer Ther. 2009 Feb;8(2):441-8.

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