Citarinostat

Cat. No.: HY-15994

CAS No.: 1316215-12-9

Molecular Formula: $C_{24}H_{26}ClN_5O_3$

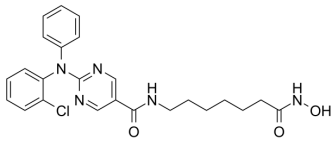
Molecular Weight: 467.95

Target: HDAC

Pathway: Cell Cycle/DNA Damage; Epigenetics

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 : ≥ 30 mg/mL (64.11 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.1370 mL	10.6849 mL	21.3698 mL
5 mM	0.4274 mL	2.1370 mL	4.2740 mL
10 mM	0.2137 mL	1.0685 mL	2.1370 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 (5.34 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.34 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.34 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Citarinostat (ACY241) is a second generation potent, orally active and high-selective HDAC6 inhibitor with an IC_{50} of 2.6 nM (IC_{50}s of 35 nM, 45 nM, 46 nM and 137 nM for HDAC1, HDAC2, HDAC3 and HDAC8, respectively). Citarinostat has anticancer effects[1].

IC_{50} & Target

HDAC6	HDAC1	HDAC2	HDAC3
2.6 nM (IC_{50})	35 nM (IC_{50})	45 nM (IC_{50})	46 nM (IC_{50})

	HDAC8 137 nM (IC_{50})	HDAC7 7300 nM (IC_{50})

In Vitro

Citarinostat (ACY241; 0-3 μM; 24 hours; A2780 cells) treatment with 300 nM results in increased hyperacetylation of α-tubulin, consistent with inhibition of the tubulin deacetylase HDAC6. In contrast, hyperacetylation of histone H3, a target of Class I HDACs, is only observed at doses above 1 μM. Low exposures of Citarinostat result in selective inhibition of HDAC6, while higher exposures lead to inhibition of Class I HDAC isozymes[1].

The single agent viability IC_{50} of Citarinostat (ACY241) ranged from 4.6-6.1 μM in A2780 and TOV-21G ovarian cancer and MDA-MD-231 breast cancer cells. Consistent with the viability assay, single agent Citarinostat modestly reduces proliferation at doses up to 3 μM without inducing apoptosis, while 10 μM of Citarinostat causes significant induction of apoptosis and completely suppresses proliferation[1].

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

Western Blot Analysis[1]

Cell Line:	A2780 cells
Concentration:	0 μM, 0.1 μM, 0.3 μM, 0.5μM, 1 μM, 3 μM
Incubation Time:	24 hours
Result:	Resulted in increased hyperacetylation of α-tubulin, consistent with inhibition of the tubulin deacetylase HDAC6. In contrast, hyperacetylation of histone H3, a target of Class I HDACs, was only observed at doses above 1 μM.

In Vivo

Citarinostat (ACY241; 50 mg/kg; intraperitoneal injection; once daily for five days, followed by two days off; for 3 weeks; female athymic nude mice) significantly suppresses tumor growth in combination with NSC 125973[1].

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

Animal Model:	Female athymic nude mice (7-week-old) injected with TOV-21G cells[1]
Dosage:	50 mg/kg
Administration:	Intraperitoneal injection; once daily for five days, followed by two days off; for 3 weeks
Result:	Combination treatment with NSC 125973 resulted in significantly suppression of tumor growth.

CUSTOMER VALIDATION

- Acta Pharmacol Sin. 2021 Apr 13.
- Oncogene. 2021 Apr;40(15):2711-2724.
- J Mol Med (Berl). 2019 Aug;97(8):1183-1193.

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REFERENCES

[1]. Huang P, et al. Selective HDAC inhibition by ACY-241 enhances the activity of NSC 125973 in solid tumor models. Oncotarget. 2017 Jan 10;8(2):2694-2707.

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

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