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- Mindermengenzuschlag
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- Expressversand

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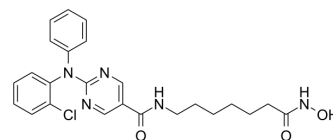
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Citarinostat

Cat. No.:	HY-15994
CAS No.:	1316215-12-9
Molecular Formula:	C ₂₄ H ₂₆ ClN ₅ O 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.

	Solvent Concentration	Mass	1 mg	5 mg	10 mg
Preparing Stock Solutions	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

- 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
- 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
- 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 IC₅₀ of 2.6 nM (IC₅₀s of 35 nM, 45 nM, 46 nM and 137 nM for HDAC1, HDAC2, HDAC3 and HDAC8, respectively). Citarinostat has anticancer effects^[1].

IC₅₀ & Target

HDAC6 2.6 nM (IC ₅₀)	HDAC1 35 nM (IC ₅₀)	HDAC2 45 nM (IC ₅₀)	HDAC3 46 nM (IC ₅₀)
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	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 IC₅₀ 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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