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Lieferung & Zahlungsart

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Zuschläge

- Mindermengenzuschlag
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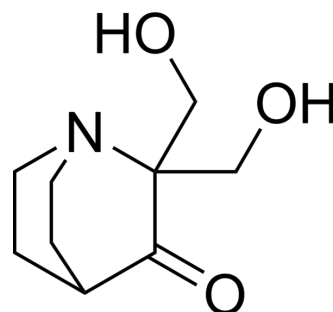
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PRIMA-1

Cat. No.:	HY-19980A
CAS No.:	5608-24-2
Molecular Formula:	C ₉ H ₁₅ NO ₃
Molecular Weight:	185.22
Target:	Autophagy; MDM-2/p53; Ferroptosis; Apoptosis
Pathway:	Autophagy; 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

H₂O : 100 mg/mL (539.90 mM; Need ultrasonic)
 DMSO : 50 mg/mL (269.95 mM; Need ultrasonic)

	Solvent Concentration	Mass	1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM		5.3990 mL	26.9949 mL	53.9898 mL
	5 mM		1.0798 mL	5.3990 mL	10.7980 mL
	10 mM		0.5399 mL	2.6995 mL	5.3990 mL

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

In Vivo

- Add each solvent one by one: PBS
Solubility: 50 mg/mL (269.95 mM); Clear solution; Need ultrasonic
- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.5 mg/mL (13.50 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 2.5 mg/mL (13.50 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (13.50 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

PRIMA-1 (NSC-281668) is a mutant p53 reactivator, restores the sensitivity of TP53 mutant-type thyroid cancer cells to the histone methylation inhibitor 3-Deazaneplanocin A.

IC₅₀ & Target

p53^[1]

In Vitro	<p>The cell lines are cultured in the presence of PRIMA-1 (NSC-281668) at 0-140 μM. The IC₅₀s are 35, 40, 50, 50, 60, 70 and 75 μM for PANC-1, HEC-1-B, SUM149, AN 3CA, Ishikawa, Panc02 and MDA-MB-231 cells, respectively^[2].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
In Vivo	<p>PRIMA-1 (Prima-1) is a p53-modulating agent. 150 or 300 ppm PRIMA-1 significantly suppresses ($P < 0.0001$) lung adenocarcinoma formation by 56% and 62%, respectively, after 17 weeks and 39% and 56%, respectively, after 34 weeks. Administration of 150 or 300 ppm PRIMA-1 significantly suppresses NNK-induced total lung adenocarcinoma formation by 57% or 62% ($P < 0.0001$), respectively, after 17 weeks of exposure and by 39% or 56% ($P < 0.0001$), respectively, after 34 weeks of exposure. As with administration of the lower (50 ppm) dose of CP-31398, administration of the lower (150 ppm) dose of PRIMA-1 also slightly increases the number of NNK-induced lung adenomas^[3].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

PROTOCOL

Cell Assay ^[2]

A cell viability assay using yellow tetrazolium salt 3-(4,5-Dimethylthiazol-2-yl)-2,5-diphenyl-tetrazolium bromide or MTT is utilized to assess the effects of the p53 SMWC on growth of human carcinoma cell lines. Cells are plated in triplicate in 96-well plates at a density of 2.5×10^3 cells/well in 100 μ L of complete medium. After 24 hr incubation in a humidified 5% atmosphere at 37°C, the cells are treated with increasing concentrations of SMWC for an additional 24 hr period and analyzed for cell growth using the MTT assay. Stock solutions (10 mM) of CP-31398 and PRIMA-1 in PBS are diluted in PBS immediately prior to use. The assay is performed as follows: a 12 mM MTT stock solution is prepared by adding 1 mL of sterile PBS to 5 mg MTT and mixing by vortex or sonication until dissolved. Once prepared, the MTT solution is stored for four weeks at 4°C protected from light. A 500 mL SDS-HCl solution consisting of 0.01 M HCl, 10% propanol and 5 gm SDS is prepared by mixing the solution gently by inversion until the SDS dissolved. 100 μ L of cell culture medium is removed from each well and 10 μ L of the 12 mM MTT stock solution added. A negative control consisting of 10 μ L of the MTT stock solution added to 100 μ L of medium is prepared. The plates are incubated at 37°C for 4 hr followed by the addition of 100 μ L of the SDS-HCl solution to each well and mixing thoroughly using a pipette. The absorbance of each sample is read at 570 nm in an ELISA plate reader. The inhibitory concentration (IC₄₀) doses are determined using standard procedure^[2].

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Animal Administration ^[3]

Mice^[3]

Female A/J mice at 6 weeks of age are used. At 6 weeks of age, mice are fed control irradiated AIN-76A modified diet. At 7 weeks of age, the mice intended for carcinogen treatment receive a single dose of 10 mol (2.07 mg) NNK/mouse by intraperitoneal injection. All mice are weighed once every 2 weeks until termination of the study. Three weeks after NNK treatment, groups of mice (25 mice/group) are fed control AIN-76A or experimental diets containing 50 or 100 ppm CP-31398 or 150 or 300 ppm PRIMA-1 until termination. Mice are killed by CO₂ asphyxiation followed by cervical dislocation after 17 weeks (10 mice/group) or 34 weeks (15 mice/group) of exposure to test agents. At the time of sacrifice, lungs are lavaged, perfused, and fixed in phosphate-buffered formalin, transferred within 2 days to 70% alcohol, and evaluated under a dissecting microscope for the number of tumors and tumor size. Tumors on the lung surface are enumerated by at least two experienced readers, blinded to sample identifiers, using a dissecting microscope. Tumor diameters are measured using Fisher brand digital calipers.

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

CUSTOMER VALIDATION

- Adv Healthc Mater. 2020, 2001029.
- Anal Chem. 2021 May 26.

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REFERENCES

- [1]. Cui B, et al. PRIMA-1, a mutant p53 reactivator, restores the sensitivity of TP53 mutant-type thyroid cancer cells to the histone methylation inhibitor 3-Deazaneplanocin A. *J Clin Endocrinol Metab*. 2014 Jun;99(6):E962-70.
- [2]. Zhang Z, et al. Targeting cancer stem cells with p53 modulators. *Oncotarget*. 2016 Apr 8.
- [3]. Rao CV, et al. Chemopreventive effects of the p53-modulating agents CP-31398 and Prima-1 in tobacco carcinogen-induced lung tumorigenesis in A/J mice. *Neoplasia*. 2013 Sep;15(9):1018-27.
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Caution: Product has not been fully validated for medical applications. For research use only.

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