Eprenetapopt

Cat. No.: HY-19980

CAS No.: 5291-32-7

Molecular Formula: $C_{10}H_{17}NO_3$

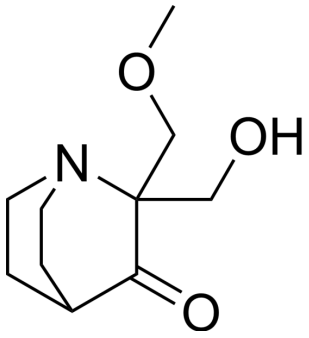
Molecular Weight: 199.25

Target: MDM-2/p53; Autophagy; Apoptosis; Ferroptosis

Pathway: Apoptosis; Autophagy

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (501.88 mM; Need ultrasonic)

H_2O : 50 mg/mL (250.94 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.0188 mL	25.0941 mL	50.1882 mL
5 mM	1.0038 mL	5.0188 mL	10.0376 mL
10 mM	0.5019 mL	2.5094 mL	5.0188 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (501.88 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (12.55 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (12.55 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (12.55 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Eprenetapopt (APR-246) is a first-in-class, small molecule that restores wild-type p53 functions in TP53-mutant cells. Eprenetapopt triggers apoptosis in tumor cells. Eprenetapopt also targets the selenoprotein thioredoxin reductase 1 (TrxR1), a key regulator of cellular redox balance[1][2][3].

IC_{50} & Target	p53 activator[1] TrxR1 inhibitor[1]
In Vitro	Eprenetapopt inhibits both recombinant TrxR1 in vitro and TrxR1 in cells. Cellular TrxR1 activity is inhibited by Eprenetapopt irrespective of p53 status. Eprenetapopt can directly affect cellular redox status via targeting of TrxR1. Several small molecules have been shown to restore wild-type activity to mutant p53, including CP-31398, PRIMA-1 and Eprenetapopt, MIRA, STIMA, PhiKan-083 and NSC319726. PRIMA-1 and its methylated analog Eprenetapopt promote correct folding of mutant p53, induce cell death by apoptosis, and inhibit tumor growth in mice. Eprenetapopt has also been shown to reactivate mutant forms of the p63 and p73 proteins that share high structural homology with p53[1]. Eprenetapopt is a powerful apoptosis-inducing agent. Eprenetapopt can enhance apoptosis in mutant p53 carrying cells, compared to the p53 null parental cells. Most p53 mutants are in complex with Hsp70 proteins. Eprenetapopt treatment increases Hsp70 expression and nucleolar translocation, in parallel with the induction of nucleolar accumulation of mutant p53. Several lines of evidence suggest that Eprenetapopt can also act independently of the p53 status of the cell. It can radiosensitize prostate carcinoma cell lines with mutant or wild type p53 and $p53^{-/-}$ cells as well. Introduction of mutant p53 (p53ser249 or p53gln248) into $p53^{-/-}$ hepatocarcinoma cells increases sensitivity to Eprenetapopt without the induction of p53 target genes.Eprenetapopt regularly induces apoptosis in mutant p53 expressing cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Sci Adv. 2022 Sep 16;8(37):eabm9427.
- Bone Res. 2023 May 29;11(1):28.
- Cell Biosci. 2022 Feb 25;12(1):20.
- Cancers. 2021 Feb 2;13(3):581.
- bioRxiv. 2023 Jul 11.

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REFERENCES

[1]. Peng X, et al. APR-246/PRIMA-1MET inhibits thioredoxin reductase 1 and converts the enzyme to a dedicated NADPH oxidase. Cell Death Dis. 2013 Oct 24;4:e881.

[2]. Stuber G, et al. PRIMA-1MET induces nucleolar translocation of Epstein-Barr virus-encoded EBNA-5 protein. Mol Cancer. 2009 Mar 26;8:23.

[3]. Sallman DA, et al. Eprenetapopt (APR-246) and Azacitidine in TP53-Mutant Myelodysplastic Syndromes. J Clin Oncol. 2021;39(14):1584-1594.

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