For Research Use Only

Eprenetapopt

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Catalog Number: CM05704

产品信息

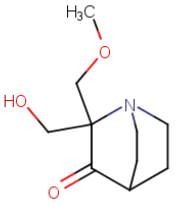
Catalog Number:
CM05704

CAS号:
5291-32-7

分子式:
$C_{10}H_{17}NO_3$

分子量:
199.25

溶解度:
DMSO:55 mg/mL (276.04 mM)



主要靶点:
Autophagy|Apoptosis|Ferroptosis|Others|p53

主要通路:
自噬|凋亡|凋亡|凋亡

靶点活性

p53:|TrxR1:

体外活性

APR-246 inhibits both recombinant TrxR1 in vitro and TrxR1 in cells. Cellular TrxR1 activity is inhibited by APR-246 irrespective of p53 status. APR-246 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 APR-246 (PRIMA-1MET), MIRA, STIMA, PhiKan-083 and NSC319726. PRIMA-1 and its methylated analog APR-246 promote correct folding of mutant p53, induce cell death by apoptosis, and inhibit tumor growth in mice. APR-246 has also been shown to reactivate mutant forms of the p63 and p73 proteins that share high structural homology with p53. PRIMA-1MET is a powerful apoptosis-inducing agent. PRIMA-1MET can enhance apoptosis in mutant p53 carrying cells, compared to the p53 null parental cells. Most p53 mutants are in complex with Hsp70 proteins. PRIMA-1MET treatment increases Hsp70 expression and nucleolar translocation, in parallel with the induction of nucleolar accumulation of mutant p53. Several lines of evidence suggest that PRIMA-1MET 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 p53 gln248) into p53-/- hepatocarcinoma cells increases sensitivity to PRIMA-1MET without the induction of p53 target genes. PRIMA-1MET regularly induces apoptosis in mutant p53 expressing cells.

储存

Powder: -20°C for 3 years | In solvent: -80°C for 1 year | Shipping with blue ice.