Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

PRIMA-1Met

Catalog No: tcsc7614

Available Sizes

Size: 5mg

Size: 10mg

Size: 25mg

Size: 50mg

Size: 100mg

Specifications

CAS No:
5291-32-7

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

Pathway:
Autophagy;Apoptosis

Target:
Autophagy;MDM-2/p53

Purity / Grade:
>98%

Solubility:
10 mM in DMSO

Alternative Names:
APR-246

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Observed Molecular Weight:
199.25

Product Description

PRIMA-1MET restores wild-type conformation and function to mutant **p53**, and triggers apoptosis in tumor cells. PRIMA-1MET also targets the selenoprotein thioredoxin reductase 1 (**TrxR1**), a key regulator of cellular redox balance.

IC50 & Target: p53 activator[1]

TrxR1 inhibitor[1]

In Vitro: 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[1]. 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 p53gln248) 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[2].