

Product Data Sheet

Product Name: MS1129
Cat. No.: GC78962

Chemical Properties

Cas. No.

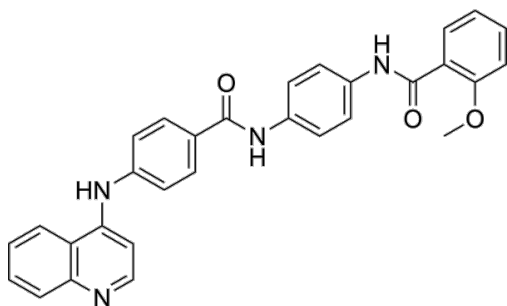
Formula $C_{30}H_{24}N_4O_3$ M.Wt 488.54

Solubility Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Background

MS1129 is a DNMT degrader that induces proteasomal degradation of DNMT1, DNMT3A and DNMT3B proteins. MS1129 upregulates TRAIL, DR4 and DR5 proteins, downregulates the decoy receptor DcR2, and activates TRAIL-dependent apoptosis via the HIF-1/2 and Caspase-10 pathways. MS1129 is applicable to the research of VHL-deficient clear cell renal cell carcinoma [1].

In Vivo, MS1129 (5 mg/kg; i.p.; daily; 10 days) significantly inhibits the growth of subcutaneous 786-

Caution: Product has not been fully validated for medical applications. For research use only.

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O ccRCC xenografts in NSG mice without altering body weight^[1].

In Vitro, MS1129 (2 μ M; 3 days) induces robust apoptosis in parental VHL-deficient RCC10 clear cell renal cell carcinoma cells, and this effect is dependent on the expression of HIF-1 α /2 α ^[1]. MS1129 inhibits the viability of parental VHL-deficient RCC10 clear cell renal cell carcinoma (ccRCC) cells with an IC₅₀ of 1.3 μ M, while its potency decreases in HIF-1 α /2 α double knockout (DKO) RCC10 cells (IC₅₀ = 3.2 μ M)^[1]. MS1129 (2 μ M; 0-48 h) induces time-dependent proteasomal degradation of DNMT1, DNMT3A and DNMT3B proteins in RCC10 clear cell renal cell carcinoma cells, with the maximum degradation level reached at 48 h^[1]. MS1129 (1-3 μ M; 2 days) induces apoptosis in RCC10 clear cell renal cell carcinoma (ccRCC) cells by activating caspase-3, caspase-7 and PARP1, while the pan-caspase inhibitor Z-VAD-FMK partially inhibits this effect^[1]. MS1129 (2 μ M; 2 days) activates the TRAIL death receptor signaling pathway in RCC10 ccRCC cells by upregulating TRAIL, DR4 and DR5, downregulating DcR2, and inducing caspase-10 cleavage^[1].

References:

[1]. Wang Y, et al. HIF-activated priming of TRAIL-induced cell death determines epigenetic vulnerability in kidney cancer. Cell Rep Med. 2026;7(3):102630.

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