

Product Data Sheet

Product Name: dCBP-30

Cat. No.: GC80918

Chemical Properties

Cas. No.

Formula $C_{40}H_{41}F_3N_{10}O_5$

M.Wt 798.81

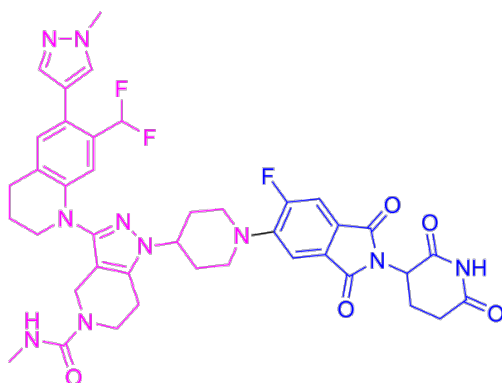
Solubility

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

dCBP-30 is an orally active, selective and dual-acting CBP/p300 PROTAC degrader with DC_{50} values of 0.05 nM (CBP) and 0.04 nM (p300), respectively. dCBP-30 reduces the acetylation levels of histone substrates H3K27, H3K18, H2BK5 and H2BK20, downregulates multiple myeloma-specific dependency programs, and induces multiple myeloma cell apoptosis. dCBP-30 triggers tumor regression and prolongs survival in multiple myeloma xenograft mouse models, and exhibits synergistic antiproliferative activity with dexamethasone. dCBP-30 can be used for the research of multiple myeloma [1]. (Pink: CBP/p300 ligand; Blue: Cereblon ligand; Black: linker).

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

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In Vivo, dCBP-30 (3-30 mg/kg; p.o.; twice daily; intermittent dosing regimen) induces significant tumor regression and prolongs survival in multiple myeloma xenograft models, while causing acute and sustained degradation of CBP and p300 in tumor tissues^[1]. dCBP-30 (15 mg/kg; p.o.; twice daily; intermittent dosing regimen; combined with Dexamethasone 1 mg/kg; i.p.; twice weekly) achieves comparable tumor regression efficacy to monotherapy with 30 mg/kg dCBP-30, with lower degrees of body weight loss and transient thrombocytopenia^[1].

In Vitro, dCBP-30 (0.01-1000 nM; 1-24 h) potently degrades both CBP and p300 in HAP1 cells, with DC₅₀ values of 0.05 nM (CBP) and 0.04 nM (p300) at 4 hours, and maximum degradation rates of 0.79 (CBP) and 0.81 (p300) at 1 nM after 1 hour^[1]. dCBP-30 (10 nM; 4 h) selectively decreases only CBP and p300 protein levels in MM1.S multiple myeloma cells after 4 hours of 10 nM treatment^[1]. dCBP-30 (10 nM; 0.5-24 h) potently reduces acetylation of CBP/p300 histone substrates (H3K18ac, H3K27ac, H2BK5ac, H2BK20ac) in MM1.S multiple myeloma cells after 10 nM treatment^[1]. dCBP-30 potently inhibits viability across 25 multiple myeloma cell lines (median AUC_{0-t} lower than dCBP-1, GNE-781, and A-485), induces complete cell killing in MM1.S cells at 10 nM within 72 hours, and triggers apoptosis in a time-dependent manner, with ~80% of cells apoptotic after 24 hours of 10 nM exposure^[1]. dCBP-30 (10 nM; 2-24 h) treatment of MM1.S multiple myeloma cells at 10 nM rapidly downregulates critical myeloma dependency genes (MYC, IRF4, MAF, PRDM1, PIM2) within 2 hours, induces modest chromatin accessibility decreases at CBP/p300 binding sites, and alters transcription factor motif accessibility in a time-dependent manner, disrupting essential myeloma signaling nodes^[1]. dCBP-30 (10 μM; 4 h) has significantly greater membrane permeability than dCBP-1 in a cell-free PAMPA assay at 10 μM, contributing to its enhanced in vitro degradation potency^[1]. dCBP-30 (multiple doses; 48-120 h) acts synergistically with Dexamethasone to reduce viability in MM1.S and NCI-H929 multiple myeloma cells, while showing antagonism with other standard anti-myeloma agents^[1].

References:

[1]. Tiwari PK, et al. An orally active dual CBP/p300 degrader targets core dependencies of multiple myeloma. Cell reports. 2026 Jun 23;45(6):117464.

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