

Product Data Sheet

Product Name: SH-26
Cat. No.: GC81639

Chemical Properties

Cas. No.

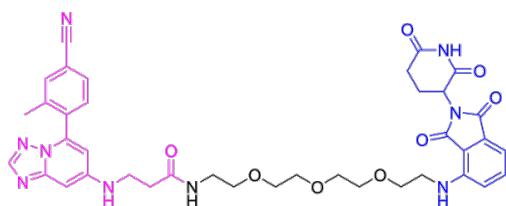
Formula $C_{38}H_{41}N_9O_8$ M.Wt 751.79

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

SH-26 is a PHD1 PROTAC degrader with DC_{50} s of 1.06 μ M, 4.16 μ M and 4.91 μ M in MDA-MB-231, HepG2 and HEK-293T cells, respectively. SH-26 recruits CRBN to induce PHD1 degradation via the ubiquitin-proteasome system. SH-26 attenuates APAP-triggered ROS accumulation, mitochondrial dysfunction, and NLRP3 inflammasome activation. SH-26 can be used for the research of acute liver injury [1]. (Pink: HIF/HIF Prolyl-Hydroxylase 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, SH-26 (Compound 26) (10-30 mg/kg; i.p.; single dose) confers robust hepatoprotective effects in APAP-induced acute liver injury^[1].

In Vitro, SH-26 (Compound 26) (0.25-10 μ M; 2-72 h) induces concentration- and time-dependent PHD1 degradation in MDA-MB-231 cells, with a DC₅₀ of 1.06 μ M and a Dmax of 77.5% in 48 h^[1]. SH-26 (0.25-50 μ M; 48 h) induces concentration-dependent PHD1 degradation in HepG2 cells with a DC₅₀ of 4.16 μ M^[1]. SH-26 (0-50 μ M; 48 h) induces concentration-dependent PHD1 degradation in HEK-293T cells with a DC₅₀ of 4.91 μ M^[1]. SH-26 (2-25 μ M; 48 h) mediates PHD1 degradation in MDA-MB-231, HepG2, and HEK-293T cells in a ubiquitin-proteasome system-dependent manner that requires both PHD1 target engagement and CRBN recruitment^[1]. SH-26 (1-25 μ M; 4 h pretreatment) degrades PHD1 and PHD2 (but not PHD3) and stabilizes HIF-1 α in APAP-challenged AML12 hepatocytes under normoxic conditions^[1]. SH-26 (1-25 μ M; 4 h pretreatment) exhibits enhanced concentration-dependent PHD1 degradation (DC₅₀ = 0.13 μ M) and stabilizes HIF-1 α in APAP-challenged AML12 hepatocytes under hypoxic conditions^[1]. SH-26 (1-50 μ M; 4 h pretreatment) protects AML12 hepatocytes from APAP-induced injury in a concentration-dependent manner without inherent cytotoxicity^[1]. SH-26 (1-25 μ M; 4 h pretreatment) restores expression of HIF-1 α target genes BNIP3 and SLC2A1, does not affect PHD1 mRNA levels, and suppresses IL-1 β mRNA levels in APAP-challenged AML12 hepatocytes^[1]. SH-26 (1-50 μ M; 4 h pretreatment) dose-dependently degrades PHD1 and suppresses IL-1 β protein levels in APAP-challenged AML12 hepatocytes^[1]. SH-26 (1 μ M; 24 h) degrades PHD1 and downregulates pro-inflammatory mediators IL-1 β , IL-6, and TNF- α in LPS +IFN- γ -stimulated THP-1-derived M1 macrophages^[1]. SH-26 (1-25 μ M; 4 h pretreatment) dose-dependently reduces mitochondrial reactive oxygen species accumulation in APAP-challenged AML12 hepatocytes^[1].

References:

[1]. Su H, et al. Targeted Degradation of Prolyl Hydroxylase Domain Enzyme 1 (PHD1) as a Novel Therapeutic Strategy for Acetaminophen-Induced Acute Liver Injury. J Med Chem. 2026;69(10):12122-12143.

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