Product Data Sheet

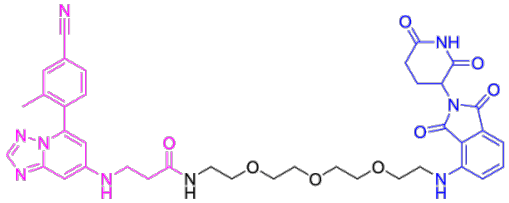
Product Name: SH-26
Cat. No.: GC81639

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 μM, 4.16 μM and 4.91 μM 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 μM; 2-72 h) induces concentration- and time-dependent PHD1 degradation in MDA-MB-231 cells, with a DC_{50} of 1.06 μM and a Dmax of 77.5% in 48 h [1]. SH-26 (0.25-50 μM; 48 h) induces concentration-dependent PHD1 degradation in HepG2 cells with a DC_{50} of 4.16 μM[1]. SH-26 (0-50 μM; 48 h) induces concentration-dependent PHD1 degradation in HEK-293T cells with a DC_{50} of 4.91 μM[1]. SH-26 (2-25 μM; 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 μM; 4 h pretreatment) degrades PHD1 and PHD2 (but not PHD3) and stabilizes HIF-1α in APAP-challenged AML12 hepatocytes under normoxic conditions[1]. SH-26 (1-25 μM; 4 h pretreatment) exhibits enhanced concentration-dependent PHD1 degradation (DC_{50} = 0.13 μM) and stabilizes HIF-1α in APAP-challenged AML12 hepatocytes under hypoxic conditions[1]. SH-26 (1-50 μM; 4 h pretreatment) protects AML12 hepatocytes from APAP-induced injury in a concentration-dependent manner without inherent cytotoxicity[1]. SH-26 (1-25 μM; 4 h pretreatment) restores expression of HIF-1α target genes BNIP3 and SLC2A1, does not affect PHD1 mRNA levels, and suppresses IL-1β mRNA levels in APAP-challenged AML12 hepatocytes[1]. SH-26 (1-50 μM; 4 h pretreatment) dose-dependently degrades PHD1 and suppresses IL-1β protein levels in APAP-challenged AML12 hepatocytes[1]. SH-26 (1 μM; 24 h) degrades PHD1 and downregulates pro-inflammatory mediators IL-1β, IL-6, and TNF-α in LPS +IFN-γ-stimulated THP-1-derived M1 macrophages[1]. SH-26 (1-25 μM; 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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