Data Sheet (Cat.No.T207609)

TargetMol

PROTAC STING degrader-4

Chemical Properties

CAS No. :

Formula: C39H42Cl2N8O9

Molecular Weight:

Storage: Keep away from direct sunlight
Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	PROTACSTING degrader-4 is a covalent STINGPROTAC degrader free of nitro groups, exhibiting a DC50 of 3.23 μM. It effectively inhibits STING and its downstream signaling pathways, including p-TBK1 and p-NF-κB (p-P65), as well as immune-inflammatory cytokines. Additionally, PROTACSTING degrader-4 mitigates renal and blood inflammation in mouse models of Cisplatin-induced acute kidney injury (AKI).
Targets(IC50)	NF-κB,IκB/IKK,PROTACs,STING
In vitro	PROTAC STING degrader-4 (Compound 2h) effectively induces the degradation of STING in THP-1 dual cells in a dose- and time-dependent manner, with a duration of up to 72 hours and a DC50 of 3.23 μM at 24 hours, when used in concentrations ranging from 0.6 to 40 μM for 2 to 72 hours. At 10 μM for 48 hours, it facilitates STING degradation through the proteasomal pathway. Additionally, PROTAC STING degrader-4 at concentrations of 0.3 to 20 μM over 2 hours reduces levels of p-TBK1 and p-NF-κB (p-P65) proteins, thereby inhibiting downstream STING signaling cascades without directly degrading STING. The compound also significantly decreases the production of IFN-β and CXCL10, and suppresses the mRNA expression of IFNB1, CXCL10, and ISG15 in THP1-Dual cells, in a dose-dependent manner at concentrations of 0.6 to 20 μM for 2 hours. Furthermore, PROTAC STING degrader-4 exhibits minimal to weak cytotoxicity in THP1-Dual and RAW-Lucia cells at concentrations below 20 μM, as well as in normal human and mouse cells at levels below 30 μM, when tested over 24 to 48 hours.
In vivo	Compound 2h, known as PROTAC STING degrader-4, administered at 30 mg/kg via intraperitoneal injection once daily for three days, significantly reduces kidney inflammation in a mouse model of Cisplatin-induced AKI by degrading STING and its downstream signaling pathway, such as p-IRF3.