

Product Data Sheet

Product Name: PSP-0119

Cat. No.: GC81025

Chemical Properties

Cas. No. 3104855-98-0

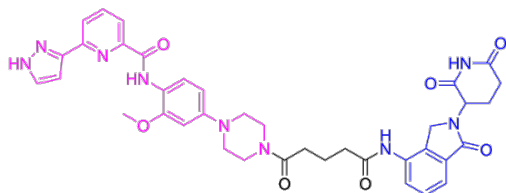
Formula $C_{38}H_{39}N_9O_7$ M.Wt 733.77

Solubility DMSO: 100 mg/mL (136.28 mM; Need ultrasonic) 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

PSP-0119 is a highly efficient and effective PROTAC degrader targeting IRAK4 ($IC_{50} = 2.83$ nM).

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

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PSP-0119 can inhibit IRAK4 kinase activity, NF- κ B activity, and IL-1 β -induced IRAK4 phosphorylation. PSP-0119 degrades IRAK4 in FLT3-mutant AML cell lines, sparing FLT3-wild-type AML cells, FLT3-wild-type samples, and normal bone marrow. PSP-0119 downregulates alpha-enolase (eNOS) of MOLM-13 cells. PSP-0119 can be used for the study of Acute Myeloid Leukemia (AML) [1]. (Pink: IRAK4 ligand; Blue: Cereblon ligand; Black: linker).

In Vivo, PSP-0119 (10 mg/kg, i.p., once daily, M-F) effectively inhibits the tumor growth of FLT3 mutant AML cells (MOLM-13) in mice, and no significant toxicity was observed[1].

In Vitro, PSP-0119 (10 nM, 6 h) inhibits NF- κ B activity in THP-1 cells stably expressing NF- κ B-Luc reporter and robustly blocks IL-1 β -induced activation (phosphorylation) of IRAK4 in MOLM-13 cells[1]. PSP-0119 demonstrates superior binding characteristics with the ternary complex, exhibiting a binding energy of -86.5 kcal/mol, indicating that it forms a highly stable ternary complex[1]. PSP-0119 (20 nM-5 μ M, 24 h) selectively degrades IRAK4 protein in MOLM-13 cells (FLT3-mutant) (DC_{50} = 31.56 nM) and MV-4-11 cells (FLT3-mutant) (DC_{50} = 29.12 nM) without affecting THP-1 cells (FLT3-wild-type)[1]. PSP-0119 (3-40 nM, 24 h) significantly degrades IRAK1 in MV-4-11 cells, but fails to degrade IRAK1 in MOLM-13 AML cells[1]. PSP-0119 (40 nM, 24 h) results in downregulation of the ENOS-1 (alpha-enolase) gene in MOLM-13 cells[1]. PSP0119 (7 μ M, 7 days) reduces CFUs formed by MV-4-11[1]. PSP-0119 (0-5 μ M, 48 h) does not affect the viability of FLT-3 wt THP1 cells, but FLT-3 mutant MOLM-13 and MV-4-11 cells show reduced viability during 48 h[1]. PSP-0119 and JH-X-119-01 exhibit a significant synergistic effect on IRAK4 inhibition in MV-4-11 AML cells in vitro[1].

References:

[1]. Khazan N, et al. PSP-0119: Targeted IRAK4 Degradation as a Novel Therapeutic Strategy for FLT3-Mutant AML. bioRxiv [Preprint]. 2025 Oct 2:2025.09.30.679569.

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