SIAIS100 TFA

Cat. No.: HY-152036A

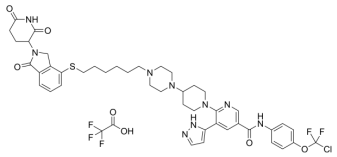
Molecular Formula: $C_{46}H_{51}ClF_5N_9O_7S$

Molecular Weight: 1004.46

Target: PROTACs; Bcr-Abl

Pathway: PROTAC; Protein Tyrosine Kinase/RTK

Storage: -20°C, stored under nitrogen, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (99.56 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	0.9956 mL	4.9778 mL	9.9556 mL
	5 mM	0.1991 mL	0.9956 mL	1.9911 mL
	10 mM	0.0996 mL	0.4978 mL	0.9956 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (2.49 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (2.49 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (2.49 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

SIAIS100 TFA is a potent BCR-ABL PROTAC degrader with an DC_{50} value of 2.7 nM. SIAIS100 TFA can be used to research chronic myeloid leukemia (CML)[1].

In Vitro

SIAIS100 exhibits anti-proliferative activity against K562 cells with an IC_{50} value of 12 nM[1].
SIAIS100 degrades BCR-ABL with degradation ratio of 81.78% and 91.20% at 5 nM and 100 nM, respectively[1].
SIAIS100 (100 nM; 8 h) significantly decreases BCR-ABL in K562 cells[1].
SIAIS100 (100 nM; 6 h) induced sustained and robust BCR-ABL degradation and maintained a durable cellular response after drug removal[1].
SIAIS100 (1-1000 nM) significantly degrade mutation G250E/T315I dose-dependently accompanied by the inhibition of BCR-

	ABL signaling assessed by the level of p-BCR-ABL in 32D cells[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Liu H, et al. Discovery and characterization of novel potent BCR-ABL degraders by conjugating allosteric inhibitor. Eur J Med Chem. 2022 Dec 15;244:114810.

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