SN52

Cat. No.:	HY-P3229
CAS No.:	1071173-56-2
Molecular Formula:	$C_{128}H_{230}N_{38}O_{28}$
Molecular Weight:	2749.43
Sequence Shortening:	AAVALLPAVLLALLAPVQRKRRKALP
Target:	NF-κB
Pathway:	NF-κB
Storage:	Sealed storage, away from moisture and light Powder -80°C 2 years -20°C 1 year * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)

AAVALLPAVLLALLAPVQRKRRKALP

SOLVENT & SOLUBILITY

In Vitro

H_2O : 25 mg/mL (9.09 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	0.3637 mL	1.8186 mL	3.6371 mL
	5 mM	0.0727 mL	0.3637 mL	0.7274 mL
	10 mM	---	---	---

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 50 mg/mL (18.19 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

SN52 is a potent, competitive, and cell-permeable inhibitor of NF-κB2. SN52 is a variant of the SN50 peptide and inhibits the nuclear translocation of p52-RelB heterodimers. SN52 has a strong radiosensitization effect on prostate cancer cells. SN52 can be used for cancer research[1].

IC_{50} & Target

IC50: NF-κB2[1]

In Vitro

SN52 (40 μg/ml; 30 mins before DMXAA) inhibits DMXAA-induced nuclear translocation of RelB in BMDCs[1].
SN52 does not change the activation of canonical NF-κB signaling. The nuclear translocation of RelB is increased in DCs isolated from irradiated tumors, and SN52 abolishes this activation in activated DC cells[1].
SN52 (40 μg/mL; 30 mins before co-cultured with irradiated or non-irradiated MC38 cells) inhibits the non-canonical NF-κB

	and increases Ifn-b expression in BMDCs stimulated with irradiated tumor cells[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	SN52 (intrathecal injection; 40 μg/ml; day-1, day 1 and day 3 of 20Gy radiation of radiation) combines with IR enhances anti-tumor immune functions of both DCs and $CD8^+$ T cells and subsequently reduced tumor burden more effectively compared with IR alone[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Tumor mice model[1]
Dosage:	40 μg
Administration:	Intrathecal injection; 40 μg; day-1, day 1 and day 3 of 20Gy radiation of radiation
Result:	Reduced tumor burden than IR group alone. Induced non-canonical NF-κB inhibition and potentiates the anti-tumor effect of IR.

REFERENCES

[1]. Yong Xu, et al. SN52, a novel nuclear factor-kappaB inhibitor, blocks nuclear import of RelB:p52 dimer and sensitizes prostate cancer cells to ionizing radiation. Mol Cancer Ther

[2]. Yuzhu Hou, et al. Non-canonical NF-κB Antagonizes STING Sensor-Mediated DNA Sensing in Radiotherapy. Immunity. 2018 Sep 18;49(3):490-503.e4

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