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Produktinformation

- Forschungsprodukte & Biochemikalien
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- Diagnostik & molekulare Diagnostik
- Laborgeräte & Service

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See the following pages for more information!

Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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IRF1-IN-1

Cat. No.: HY-171006

CAS No.: 701225-07-2

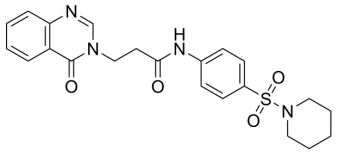
Molecular Formula: $C_{22}H_{24}N_4O_4S$

Molecular Weight: 440.52

Target: Caspase; PARP; Pyroptosis; Interleukin Related; IFNAR

Pathway: Apoptosis; Cell Cycle/DNA Damage; Epigenetics; Immunology/Inflammation

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 83.33 mg/mL (189.16 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2700 mL	11.3502 mL	22.7004 mL
	5 mM	0.4540 mL	2.2700 mL	4.5401 mL
	10 mM	0.2270 mL	1.1350 mL	2.2700 mL

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

BIOLOGICAL ACTIVITY

Description

IRF1-IN-1 (Compound I-2) is an IRF1 inhibitor. IRF1-IN-1 decreases the recruitment of IRF1 to the promoter of CASP1. IRF1-IN-1 inhibits cell death signaling pathway (i.e., cleavage of Caspase 1, GSDMD, IL-1 and PARP1). IRF1-IN-1 has a protective effect on ionizing radiation-induced inflammatory skin injury[1].

IC_{50} & Target			
	PARP-1	IL-1	Caspase-1

In Vitro

IRF1-IN-1 pretreatment (20 μM, 12 h) decreases the recruitment of IRF1 to the promoter of CASP1 in irradiated HaCaT cells (20 Gy)[1].
IRF1-IN-1 (20 μM, 24 h) attenuates the NSP-10 plasmid transfection-induced IRF1 activation in HELF, HaCaT and WS1 cells[1].
IRF1-IN-1 (50 μM, 24 h) treatment reduces the transcriptional activity of IRF1 in HELF cells 96 h after SARS-CoV-2 pseudovirus infection[1].
IRF1-IN-1 decreases radiation (20 Gy)-induced cell death in K150 cells[1].
IRF1-IN-1 maintains mitochondrial activity and ROS production in skin cells in the early stage after irradiation[1].
IRF1-IN-1 significantly inhibits the cell death signaling pathway, i.e., suppresses cleavage of Caspase 1, GSDMD, IL-1 and PARP1[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

IRF1-IN-1 pretreatment (100 μg/d, s.c., one every other day before irradiation) has a potential protective effect in 35 Gy radiation-induced inflammatory skin injury mice[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Radiogenic skin injury mice (intraperitoneal injection of pentobarbital sodium (1%, 30 mg/kg), 35 Gy at the dose rate of 1000 cGy/min by a 6-MeV electron beam)[1]
Dosage:	100 μg/d
Administration:	Subcutaneous injection (s.c.), one every other day before irradiation, pretreatment
Result:	Showed a significant reduction in acute skin inflammatory manifestations, such as erythema and exudation and accelerated the healing process. Showed protective effects on the function and structural integrity of radiation-induced lesions to the claws.

REFERENCES

[1]. Geng, et al. Chaperone- and PTM-mediated activation of IRF1 tames radiation-induced cell death and the inflammatory response. Cell Mol Immunol. 2024 Aug;21(8):856-872.

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