J14

Cat. No.: HY-135008

CAS No.: 1043854-13-2

Molecular Formula: $C_{28}H_{25}ClN_4O_2S$

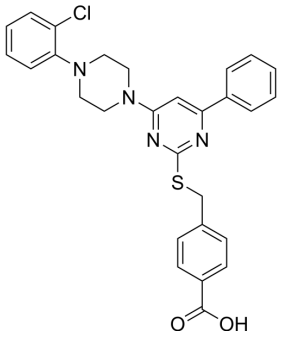
Molecular Weight: 517.04

Target: Reactive Oxygen Species

Pathway: Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 125 mg/mL (241.76 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.9341 mL	9.6704 mL	19.3409 mL
5 mM	0.3868 mL	1.9341 mL	3.8682 mL
10 mM	0.1934 mL	0.9670 mL	1.9341 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: ≥ 2.17 mg/mL (4.20 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.17 mg/mL (4.20 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

J14 is a reversible sulfiredoxin inhibitor with an IC_{50} of 8.1 μM. J14 induces oxidative stress (intracellular ROS accumulation) by inhibiting sulfiredoxin, leading to cytotoxicity and cancer cell death[1].

IC_{50} & Target

IC50: 8.1 μM (Sulfiredoxin); ROS[1]

In Vitro

J14 (0-100 μM; 0-96 hours; A549 cells) treatment inhibits the growth of A549 cells in a concentration- and a time- dependent manner, and its half inhibitory concentration for the growth of A549 cells was 15.7 μM[1].

?J14 (20 μM; 48-72 hours; A549 cells) treatment causes not only the release of cytochrome c into the cytosol, but also the activation of caspase-3 and caspase-9. J14 induces oxidative damage to mitochondria, resulting in caspase-mediated

apoptosis[1].
?J14 treatment significantly increases the accumulation of sulfinic peroxiredoxins and intracellular ROS. Excess accumulation of intracellular ROS causes oxidative damage, leading to cell death. J14 significantly induces cell death in A549 cells in a time-dependent manner, resulting in approximately 40% cell death in 96 hours[1].
?J14 induces oxidative mitochondrial damage and apoptosis[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	A549 cells
Concentration:	0-100 μM
Incubation Time:	0 hour, 24 hours, 48 hours, 72 hours, 96 hours
Result:	Inhibited the growth of A549 cells in a concentration- and a time- dependent manner.

Western Blot Analysis[1]

Cell Line:	A549 cells
Concentration:	20 μM
Incubation Time:	48 hours, 72 hours
Result:	Caused not only the release of cytochrome c into the cytosol, but also the activation of caspase-3 and caspase-9.

In Vivo

J14 (50 mg/kg; intraperitoneal injection; daily; for 16 days; BALB/c nude female mice) treatment significantly reduces the average tumor volume. The masses and weights of the primary tumors excised from the J14-treated mice are significantly lower compared with those of the control mice[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Six-week-old BALB/c nude female mice injected with A549 cells[1]
Dosage:	50 mg/kg
Administration:	Intraperitoneal injection; daily; for 16 days
Result:	Significantly reduced the growth of human lung tumor without acute toxicity.

CUSTOMER VALIDATION

- Research Square Preprint. 2023 May 4.
- bioRxiv. 2023 Mar 7.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Kim H, et al. Sulfiredoxin inhibitor induces preferential death of cancer cells through reactive oxygen species-mediated mitochondrial damage. Free Radic Biol Med. 2016 Feb;91:264-74.

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

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