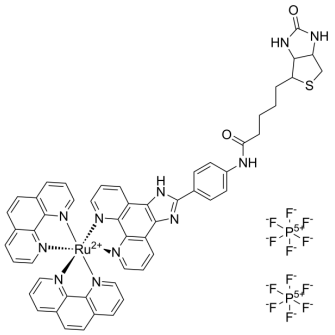
Antitumor photosensitizer-5

Cat. No.:	HY-163034
Molecular Formula:	$C_{53}H_{43}F_{12}N_{11}O_2P_2RuS$
Molecular Weight:	1289.04
Target:	Apoptosis; Reactive Oxygen Species
Pathway:	Apoptosis; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Antitumor photosensitizer-5 (Ru2) is a photosensitizer which effectively target tumor mitochondria with an IC_{50} of 0.3 μM for phototoxicity to A549 cells. Under 460 nm light irradiation, antitumor photosensitizer-5 induces the generation of reactive oxygen species and NADH depletion, causes mitochondrial damage and activation of caspase-3, inducing apoptosis and suppressing cell migration. Antitumor photosensitizer-5 has the potential to prevent the growth of malignant tumors, therefore, shows the potential to be applied to photodynamic therapy[1].
IC_{50} & Target	Nicotinamide adenine dinucleotide (NADH)[1]

In Vitro

Antitumor photosensitizer-5 (10 μM, 4 h) causes a significant increase (32.08-fold) in fluorescence signal in A549 cells (biotin receptor-positive) while inducing BHK cells (biotin receptor-negative) to exhibit negligible fluorescence increase (7.35-fold) [1].

Antitumor photosensitizer-5 (0.391-100 μM, 24 h) exhibits phototoxicity in both BHK cells and A549 cells and reveals minimal cytotoxicity in the absence of light with over 75 % cell viability under 100 μM[1].

Antitumor photosensitizer-5 (10 μM, 4 h) has a Pearson colocalization coefficient of 0.87 with the mitochondrial probe Mito-Tracker Green[1].

Antitumor photosensitizer-5 (0.15-0.6 μM, 24 h) with 460 nm light irradiation for 15 min exhibits a concentration-dependent reduction in the mitochondrial membrane potential probe fluorescence intensity ratio which indicated the mitochondrial damage, while the ROS probe fluorescence intensity exhibits a concentration-dependent increase, indicating effective generation of ROS [1].

Antitumor photosensitizer-5 (0.15-0.6 μM, 24 h) causes the occurrence of apoptosis in A549 cells after light irradiation, while the apoptosis level is virtually unchanged under dark condition, light condition also causes the increase of activated caspase-3, the migration and damage of DNA and the reduction of cellular NADH content[1].

Antitumor photosensitizer-5 (0.15-0.6 μM, 24 h/48 h) inhibits the cell migration of A549 cells under 460 nm light [1].

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

Cell Viability Assay[1]

Cell Line:	BHK, A549
Concentration:	0.195, 0.391, 0.781, 1.563, 3.125, 6.250, 12.5, 25, 50, 100 μM
Incubation Time:	4 h in dark + 15 min in light or dark + 20 h in dark
Result:	Exhibited phototoxicity in both BHK cells and A549 cells, but was more phototoxic in A549 cells (A549 cell viability < 40% under 0.391 μM while BHK cell viability < 40% under 6.25 μ

	M), revealed minimal cytotoxicity in the absence of light with over 75 % cell viability under 100 μM.

Apoptosis Analysis[1]

Cell Line:	A549
Concentration:	0.15, 0.3, 0.6 μM
Incubation Time:	4 h in dark + 15 min in light or dark + 20 h in dark
Result:	Increased the percentage of early and late apoptotic cells in A549 in a concentration-dependent manner under the 460 nm light condition. Conversely, under dark conditions, the percentage of early and late apoptotic cells in treated A549 cells remained virtually unchanged.

Cell Migration Assay [1]

Cell Line:	A549
Concentration:	0.15, 0.3, 0.6 μM
Incubation Time:	4 h in dark + 15 min in light or dark + 20 h/44 h in dark
Result:	Displayed a significant concentration-dependent inhibition of wound healing in A549 cells under 460 nm light compared to cells kept in the dark.

In Vivo

Antitumor photosensitizer-5 (10 mg/kg, i.tu. for once, 24 d) remarkably suppresses the tumor growth after 460 nm light irradiation, and doesn't cause severe adverse effects on normal organs [1].

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Animal Model:	BALB/c nude female mice (6–8 weeks old) , Human lung adenocarcinoma epithelial A549 cells[1]
Dosage:	10 mg/kg
Administration:	intratumoral injection (i.tu.) for once
Result:	Suppressed the tumor growth remarkably in the light group while tumors in the dark or control groups grow rapidly during the same period. Caused severe apoptosis and disruption of the tumor structure in the tumor of light group while the tumors in the other group showed no obvious tissue damage, normal organs such as the heart, liver, spleen, lung, and kidney did not exhibit significant pathological abnormalities or inflammatory lesions.

REFERENCES

[1]. Guoqiang Shao, et al. Biotin-conjugated Ru(II) complexes with AIE characteristics as mitochondria-targeted photosensitizers for enhancing photodynamic therapy by disrupting cellular redox balance. European Journal of Medicinal Chemistry. 2023Volume 264115985.

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

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