MitoTam iodide, hydriodide

Cat. No.: HY-126222A

CAS No.: 1634624-74-0

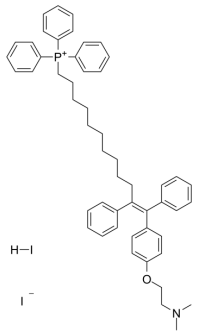
Molecular Formula: $C_{52}H_{60}I_2NOP$

Molecular Weight: 999.82

Target: Apoptosis; Mitochondrial Metabolism

Pathway: Apoptosis; Metabolic Enzyme/Protease

Storage: 4°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 (100.02 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.0002 mL	5.0009 mL	10.0018 mL
	5 mM	0.2000 mL	1.0002 mL	2.0004 mL
	10 mM	0.1000 mL	0.5001 mL	1.0002 mL

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

BIOLOGICAL ACTIVITY

Description

MitoTam iodide, hydriodide is a Tamoxifen derivative[1], an electron transport chain (ETC) inhibitor, spreduces mitochondrial membrane potential in senescent cells and affects mitochondrial morphology[2].MitoTam iodide, hydriodide is an effective anticancer agent, suppresses respiratory complexes (CI-respiration) and disrupts respiratory supercomplexes (SCs) formation in breast cancer cells[1][2]. MitoTam iodide, hydriodide causes apoptosis[2].

In Vitro

MitoTam (0.5 μM-56 μM; 24 hours) kills breast cancer cell Lines and nonmalignant cells with an IC_{50} range from 0.65 μM to 55.9 μM[1].
MitoTam (2.5 μM; 2-24 hours) results in stronger activation of the apoptotic pathway in MCF7 $Her2^{high}$ cells compared with mock MCF7 cells[1].
MitoTam (0.05 μM-1 μM; 3 days) causes a concentration-dependent induction of apoptosis in breast cancer cells, while there was no effect for non-malignant breast epithelial cells[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	Breast Cancer Cell Lines: BT474, MCF7, MCF7 $Her2^{high}$, MCF7 $Her2^{low}$, MDA-MB-231, MDA-

	MB-436, MDA-MB-453, SK-BR-3, T47D; NeuTL cells; Nonmalignant Cells: A014578, H9c2 cells
Concentration:	0.5 μM-56 μM
Incubation Time:	24 hours
Result:	Killed breast cancer cells MCF7, MCF7 Her2^{high}, MCF7 Her2^{low} with IC_{50} values of 1.25 μM, 0.65 μM and 1.45 μM respectively.

Western Blot Analysis[2]

Cell Line:	MCF7 mock and MCF7 Her2^{high} cells
Concentration:	2.5 μM
Incubation Time:	2 hours, 4 hours, 8 hours, 16 hours, 24 hours
Result:	Revealed accelerated cleavage of procaspase-9, Parp1/2 and proapoptotic Bax and decreased the antiapoptotic Bcl-2 protein in Her2^{high} cells.

Apoptosis Analysis[2]

Cell Line:	MCF-7, 4T1 and MCF-10a cells
Concentration:	0.05 μM-1 μM
Incubation Time:	3 days
Result:	Resulted in apoptosis in MCF7 and 4T1 cells.

In Vivo

MitoTam (intraperitoneal injection; 2 μg/g; once a week; 4 weeks) decreases β-gal staining of lungs from MitoTam-treated mice, accompaning by a inhibition in the expression of senescence markers p16^{Ink4a}, p21^{waf1} and PAI comparing control mice [2].

MitoTam (intraperitoneal injection; 0.54 μmol/mouse; twice a week; 2 weeks) inhibits growth of syngeneic tumors by 80%[1].

MitoTam (intraperitoneal injection; 0.25 μmol/mouse; twice a week; 2 weeks) slows down the growth of MCF7 mock tumors and stops tumor progression after two doses; suppresses Her2^{high} carcinomas decreased threefold from the original size with complete disappearance[1].

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

Animal Model:	18-month-old or 2-month-old FVB/N mice[1]
Dosage:	2 μg/g
Administration:	Intraperitoneal injection; 2 μg/g; once a week; 4 weeks
Result:	Eliminated senescent cells also in vivo.

Animal Model:	18-month-old or 2-month-old FVB/N mice[2]
Dosage:	0.54 μmol/mouse
Administration:	Intraperitoneal injection; 0.54 μmol/mouse; twice a week; 2 weeks
Result:	Suppressed Her2^{high} breast carcinomas.

Animal Model:	18-month-old or 2-month-old FVB/N mice[1]
Dosage:	0.25 μmol/mouse
Administration:	Intraperitoneal injection; 0.25 μmol/mouse; twice a week; 2 weeks
Result:	Prevented reaching the ethical endpoint in all situations, slowed down the growth of MCF7 mock tumors and suppressed $Her2^{high}$ carcinomas decreased.

REFERENCES

[1]. Rohlenova K, et al. Selective Disruption of Respiratory Supercomplexes as a New Strategy to Suppress Her2highBreast Cancer. Antioxid Redox Signal. 2017 Jan 10;26(2):84-103.

[2]. Hubackova S, et al. Selective elimination of senescent cells by mitochondrial targeting is regulated by ANT2. Cell Death Differ. 2019 Jan;26(2):276-290.

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