# MitoTam bromide, hydrobromide

**Cat. No.:** HY-126222

**CAS No.:** 1634624-73-9

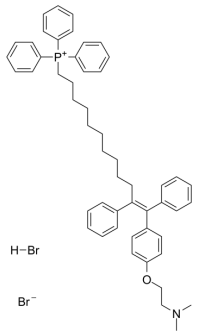
**Molecular Formula:** $C_{52}H_{60}Br_2NOP$

**Molecular Weight:** 905.82

**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 : 240 mg/mL (264.95 mM; Need ultrasonic)

| Preparing Stock Solutions | Mass / Solvent / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|---|
| | 1 mM | 1.1040 mL | 5.5199 mL | 11.0397 mL |
| | 5 mM | 0.2208 mL | 1.1040 mL | 2.2079 mL |
| | 10 mM | 0.1104 mL | 0.5520 mL | 1.1040 mL |

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

## BIOLOGICAL ACTIVITY

**Description**

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

| | |
|---|---|
| Cell Line: | MCF7 mock cells, 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, decreased the antiapoptotic Bcl-2 protein in Her2high cells. |

Apoptosis Analysis[2]

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|---|---|
| Cell Line: | MCF-7 cells, 4T1 cells 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 p16Ink4a, p21waf1 and PAI comparing control mice sup>[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[2] |
| Dosage: | 2 μg/g |
| Administration: | Intraperitoneal injection; 2 μg/g; once a week; 4 weeks |
| Result: | Eliminated senescent cells also in vivo. |

| | |
|---|---|
| Animal Model: | FVB/N c-neu mouse[1] |
| 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: | Balb/c nude mice with MCF7 mock or MCF7 Her2$^{high}$ cells[1] |
| Dosage: | 0.25 μmol/mouse/dose |
| Administration: | Intraperitoneal injection; 0.25 μmol/mouse/dose; 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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