Amiodarone hydrochloride

Cat. No.: HY-14188

CAS No.: 19774-82-4

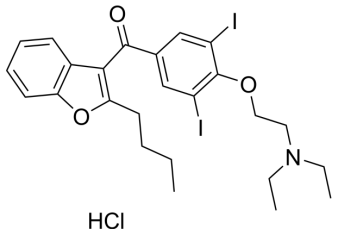
Molecular Formula: $C_{25}H_{30}ClI_2NO_3$

Molecular Weight: 681.77

Target: Potassium Channel; Autophagy

Pathway: Membrane Transporter/Ion Channel; Autophagy

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 24.5 mg/mL (35.94 mM; Need ultrasonic and warming)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.4668 mL	7.3339 mL	14.6677 mL
	5 mM	0.2934 mL	1.4668 mL	2.9335 mL
	10 mM	0.1467 mL	0.7334 mL	1.4668 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: ≥ 2.5 mg/mL (3.67 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (3.67 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.67 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Amiodarone hydrochloride, a benzofuran-based Class III antiarrhythmic agent, inhibits WT outwardIhERG tails with an IC_{50} of -45 nM[1]. Amiodarone hydrochloride induces cell proliferation and myofibroblast differentiation via ERK1/2 and p38 MAPK signaling in fibroblasts[2]. Amiodarone hydrochloride can be used in the research of both supraventricular and ventricular arrhythmias[1].

In Vitro

Amiodarone blocks inward IhERG tails in a high K^+ external solution ($[K^+]e$) of 94 mM with an IC_{50} of 117.8 nM[1]. Amiodarone (1 μM) blocks inwardIhERG by 68.8±6.1%, with concentration response data yielding IC_{50} and h values of 765.5±287.8 nM and 0.9±0.4 for T623A hERG[1].

Amiodarone (1 μM) blocks inward IhERG with an IC_{50} and h values of 979.2±84.3 nM and 1.1±0.1 for S624A hERG[1].
Amiodarone (1-6 μg/mL) induces human embryonic lung fibroblasts (HELFs) cell proliferation and PD98059 or SB203580 suppresses this effect[2].
Amiodarone (1-6 ug/mL) does not induces HELFs cell apoptosis. Amiodarone (over 15 ug/mL) induces cell apoptosis[2].
Amiodarone (1, 3 and 6 μg/mL;24 hours) induces α-SMA and vimentin mRNA and protein expression accompanied by increased phosphorylation of ERK1/2 and p38 MAPK[2].

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

Cell Proliferation Assay[2]

Cell Line:	HELFs
Concentration:	1, 3 and 6 μg/mL
Incubation Time:	24 hours
Result:	Increased HELFs proliferation compared with the control group.

Western Blot Analysis[2]

Cell Line:	HELFs
Concentration:	1, 3 and 6 μg/mL
Incubation Time:	24 hours
Result:	α-SMA and vimentin were increased significantly in a dose-dependent manner.

RT-PCR[2]

Cell Line:	HELFs
Concentration:	1, 3 and 6 μg/mL
Incubation Time:	24 hours
Result:	Induced an increase of α-SMA and vimentin mRNA expression.

In Vivo

Amiodarone has a very large volume of distribution and broad tissue distribution, which results in a long terminal half-life (25 days) in human plasma. The major active metabolite is desethyl amiodarone. Amiodarone has low and variable bioavailability after oral administration. Amiodarone is highly lipophilic[2].
Amiodarone hydrochloride can be used in animal modeling to construct animal pulmonary fibrosis models. Chronic Amiodarone hydrochloride (90 and 180 mg/kg/day) treatment induces a dose-dependent remodeling of ion channel expression that correlates with the cardiac electrophysiological effects of Amiodarone hydrochloride[3].

Induction of lung fibrosis model[1]

- Background

  Amiodarone induces pulmonary injury by direct toxicity to lung tissue, hypersensitivity reaction, enhanced oxidative stress, alteration of membrane properties and activation of alveolar macrophages and cytokine release. Amiodarone administration can result in interstitial and/or alveolar inflammation and release of

inflammatory mediators.

- Specific Mmodeling Methods

Rat: F344 rats • male • 200-225 g

Administration:6.25 mg/kg • i.t. instillation • in 0.3 mL of water • on days 0 and 2

Note

The solution should brought to room temperature before instillation.

- Modeling Indicators

Marker of lung fibrosis: Increased lung collagen content.

Higher levels of total protein in lung lavage fluid.

Increased LDH activity in bronchoalveolar lavage, and increased lung MPO activity.

Increased total cell counts, alveolar macrophages, neutrophils and eosinophils.

- Opposite Product(s): Curcumin (HY-N0005)

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

Animal Model:	Ten-week-old male C57BL/6 mice[3]
Dosage:	30, 90, and 180 mg/kg/day
Administration:	Treated orally; for 6 weeks
Result:	Mice treated with 90 and 180 mg/kg/day had decreased body and heart weights, although their heart weight-to-body weight ratios were not significantly different from sham. 6-week treatment induced a decrease in plasma triiodothyronine and an increase in reverse triiodothyronine. This effect reached significance for the 90 and 180 but not for the 30 mg/kg/day dose groups.

CUSTOMER VALIDATION

- Cell. 2023 Nov 22;186(24):5363-5374.e16.
- Cell. 2022 Dec 8;185(25):4801-4810.e13.
- Ecotoxicol Environ Saf. 2021 Apr 1;212:111991.
- Front Bioeng Biotechnol. 2022 Mar 17;10:826093.
- Viruses. 2021 Jun 28;13(7):1255.

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REFERENCES

[1]. Punithavathi D, et al. Protective effects of curcumin against amiodarone-induced pulmonary fibrosis in rats. Br J Pharmacol. 2003 Aug;139(7):1342-50.

[2]. Shayeganpour A, et al. Pharmacokinetics of Amiodarone in hyperlipidemic and simulated high fat-meal rat models. Biopharm Drug Dispos. 2005 Sep;26(6):249-57.

[3]. Yihong Zhang,et al. Interactions between amiodarone and the hERG potassium channel pore determined with mutagenesis and in silico docking. Biochem Pharmacol. 2016 Aug 1;113:24-35.

[4]. Sabrina Le Bouter, et al. Long-term amiodarone administration remodels expression of ion channel transcripts in the mouse heart. Circulation. 2004 Nov 9;110(19):3028-35.

[5]. Jie Weng, et al. Amiodarone induces cell proliferation and myofibroblast differentiation via ERK1/2 and p38 MAPK signaling in fibroblasts. Biomed Pharmacother. 2019 Jul;115:108889.

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