Sodium dichloroacetate

Cat. No.: HY-Y0445A

CAS No.: 2156-56-1

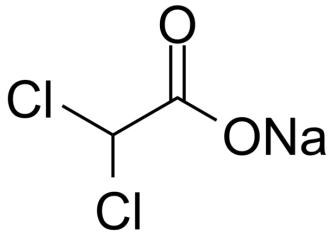
Molecular Formula: $C_2HCl_2NaO_2$

Molecular Weight: 150.92

Target: PDHK; Reactive Oxygen Species; Apoptosis; NKCC

Pathway: Metabolic Enzyme/Protease; Immunology/Inflammation; NF-κB; Apoptosis; Membrane Transporter/Ion Channel

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



SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (662.60 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	6.6260 mL	33.1301 mL	66.2603 mL
5 mM	1.3252 mL	6.6260 mL	13.2521 mL
10 mM	0.6626 mL	3.3130 mL	6.6260 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (662.60 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.25 mg/mL (14.91 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.25 mg/mL (14.91 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.25 mg/mL (14.91 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Sodium dichloroacetate is a metabolic regulator in cancer cells' mitochondria with anticancer activity. Sodium dichloroacetate inhibits PDHK, resulting in decreased lactic acid in the tumor microenvironment. Sodium dichloroacetate increases reactive oxygen species (ROS) generation and promotes cancer cell apoptosis. Sodium dichloroacetate also works as NKCC inhibitor[1].

IC_{50} & Target

PDHK; Reactive oxygen species (ROS); Apoptosis; NKCC[1]

In Vitro	Sodium dichloroacetate increases ROS generation in mitochondria. Sodium dichloroacetate affects cell growth and viability through the ROS production increase derived from the promotion of oxidative metabolism. The effects of Sodium dichloroacetate on multiple myeloma cell viability, cell cycle arrest, and apoptotic cell death were associated with pyruvate dehydrogenase kinases (PDK) inhibition, restored pyruvate dehydrogenase (PDH) activity, and the promotion of oxidative metabolism in association with increased intracellular ROS production which depends on the Sodium dichloroacetate dose. The Sodium dichloroacetate effects cooperated with C I inhibition promoting the oxidative stress in rat VM-M3 glioblastoma cells. Increased ROS levels in Sodium dichloroacetate-treated cancer cells are related to the induction of apoptosis associated with the increased cytochrome c expression. Sodium dichloroacetate causes ROS-dependent T-cell differentiation[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	The NKCC1 RNA expression levels in Sodium dichloroacetate-treated gonad-intact and castrated males are significantly decreased, and no such effect is determined in the gonad-intact and castrated female Sodium dichloroacetate-treated rats [1]. A single Sodium dichloroacetate dose causes a significantly higher 24 h diuresis in Wistar male rats, and the increased diuresis is related to NKCC2 inhibition. The NKCC2 is more abundant in kidneys of intact females compared to intact males, with a greater transporter density in Sprague-Dawley female rats[1]. The oral Sodium dichloroacetate bioavailability in naïve male rats dosed 5, 20 and 100 mg/kg is significantly lower than in GSTζ-depleted ones (10%, 13%, 81% and 31%, 75%, 100%, respectively). The liver extraction of Sodium dichloroacetate in the GSTζ-depleted rats has linear kinetics, but it decreases with the metabolism saturation at higher doses[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Signal Transduct Target Ther. 2022 Sep 1;7(1):303.
- Cell Death Dis. 2021 Sep 6;12(9):837.
- Fundamental Research. 2023 Mar 6.
- Life Sciences. 2022: 121192.
- Cancers (Basel). 2022 Oct 10;14(19):4966.

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REFERENCES

[1]. Stakišaitis D, et al. The Importance of Gender-Related Anticancer Research on Mitochondrial Regulator Sodium Dichloroacetate in Preclinical Studies In Vivo. Cancers (Basel). 2019 Aug 20;11(8). pii: E1210.

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