Data Sheet (Cat.No.T3604)

TargetMol

Sodium dichloroacetate

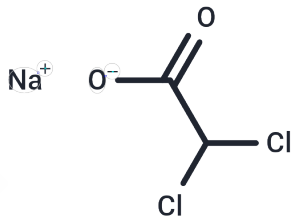
Chemical Properties

CAS No. : 2156-56-1

Formula: C2HCl2NaO2

Molecular Weight: 150.92

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.



Biological Description

Description	Sodium dichloroacetate (BCA) , a specific inhibitor of pyruvate dehydrogenase kinase (PDK) with IC50 values of 183 and 80 μM for PDK2 and PDK4 respectively, has been shown to derepress a mitochondrial potassium-ion channel axis, trigger apoptosis in cancer cells, and inhibit tumor growth.
Targets(IC50)	Apoptosis,Reactive Oxygen Species,PDK,Dehydrogenase,Na-K-Cl cotransporter,ROS
In vitro	**METHODS**: Tumor cells A549, MO59K and MCF-7 and normal cells SAEC were treated with Sodium dichloroacetate (0.5 mM) for 48 h. Mitochondrial membrane potential ΔΨm was detected using fluorescent dye. **RESULTS**: Incubation of all three types of tumor cells with Sodium dichloroacetate reversed hyperpolarization and restored ΔΨm to the level of normal cells. In contrast, Sodium dichloroacetate did not alter ΔΨm in SAEC. [1] **METHODS**: OSCC cell lines HSC-2, HSC-3 and PE15 were treated with Sodium dichloroacetate (1-10 mM) for 24 h. Cell viability was measured using the MST assay. **RESULTS**: HSC-2 and HSC-3 showed more significant unique sensitivity to the drugs. Compared with HEK 293, the viability of both HSC-2 and HSC-3 was about 30% at 10 mM Sodium dichloroacetate, while the viability of PE15 was 75-80%. [2]
In vivo	**METHODS**: To study the effect on energy expenditure in ApoE-/- mice, Sodium dichloroacetate (100-150 mg/kg) was administered by gavage once daily for four weeks to ApoE-/- mice fed the western diet model of atherosclerosis. **RESULTS**: Western diet-fed ApoE-/- mice developed atherosclerotic plaques and hyperlipidemia, as well as obesity, which were significantly ameliorated by the administration of Sodium dichloroacetate. Enhanced glucose oxidation by Sodium dichloroacetate protects against atherosclerosis by inducing hepatic FGF21 expression and BAT activation, thereby increasing the risk of atherosclerosis. atherosclerosis, thereby increasing energy expenditure for calorie production. [3]

Solubility Information

Solubility	H2O: 166.66 mg/mL (1104.29 mM),Sonication is recommended. DMSO: 26 mg/mL (172.28 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	6.626 mL	33.1301 mL	66.2603 mL
5 mM	1.3252 mL	6.626 mL	13.2521 mL
10 mM	0.6626 mL	3.313 mL	6.626 mL
50 mM	0.1325 mL	0.6626 mL	1.3252 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Bonnet S, et al. A mitochondria-K+ channel axis is suppressed in cancer and its normalization promotes apoptosis and inhibits cancer growth. Cancer Cell. 2007 Jan;11(1):37-51.
Qiu X, Jiang Z, Luo Y, et al.PPP3CB Inhibits Cell Proliferation and the Warburg Effect in Bladder Cancer by Blocking PDHK1.Frontiers in Bioscience-Landmark.2024, 29(2): 48.
Ruggieri V, et al. Dichloroacetate, a selective mitochondria-targeting drug for oral squamous cell carcinoma: a metabolic perspective of treatment. Oncotarget. 2015 Jan 20;6(2):1217-30.
Min BK, et al. Therapeutic effect of dichloroacetate against atherosclerosis via hepatic FGF21 induction mediated by acute AMPK activation. Exp Mol Med. 2019 Sep 30;51(10):1-12.