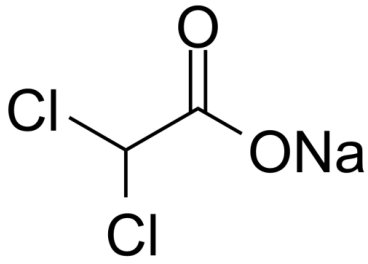
Data Sheet

Product Name:	Sodium dichloroacetate
Cat. No.:	CS-0030662
CAS No.:	2156-56-1
Molecular Formula:	$C_2HCl_2NaO_2$
Molecular Weight:	150.92
Target:	Apoptosis; NKCC; PDHK; Reactive Oxygen Species
Pathway:	Apoptosis; Immunology/Inflammation; Membrane Transporter/Ion Channel; Metabolic Enzyme/Protease; NF-κB
Solubility:	H2O : 100 mg/mL (662.60 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

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]. IC50 & 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]. **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].

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.

CAIndexNames:

Acetic acid, 2,2-dichloro-, sodium salt (1:1)

SMILES:

O=C(O[Na])C(Cl)Cl

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: 610-426-3128 Fax: 888-484-5008 E-mail: sales@ChemScene.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA