Mitoguazone

Cat. No.: HY-106634

CAS No.: 459-86-9

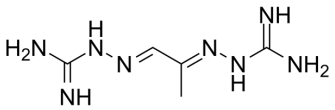
Molecular Formula: $C_5H_{12}N_8$

Molecular Weight: 184.2

Target: HIV; Apoptosis

Pathway: Anti-infection; Apoptosis

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 : 50 mg/mL (271.44 mM; ultrasonic and adjust pH to 9 with HCl)
DMSO : 8.33 mg/mL (45.22 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.4289 mL	27.1444 mL	54.2888 mL
5 mM	1.0858 mL	5.4289 mL	10.8578 mL
10 mM	0.5429 mL	2.7144 mL	5.4289 mL

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

In Vivo

1. Add each solvent one by one: PBS
Solubility: 4 mg/mL (21.72 mM); Clear solution; Need ultrasonic and warming and heat to 60°C

BIOLOGICAL ACTIVITY

Description

Mitoguazone (Methylglyoxal-bis(guanylhydrazone)) is a synthetic polycarbonyl derivative with potent antineoplastic activity. Mitoguazone is a brain-penetrant and competitive S-adenosyl-methionine decarboxylase (SAMDC) inhibitor that disrupts polyamine biosynthesis. Mitoguazone induces cell apoptosis. Mitoguazone inhibits HIV DNA integration into the cellular DNA in both monocytes and macrophages. Mitoguazone has the potential for acute leukemia, Hodgkin's and non-Hodgkin's lymphoma treatment[1][2][3][4].

In Vitro

Mitoguazone competitively inhibits spermidine synthesis in lymphocytes at concentrations as low as 0.5 μg/mL. Levels of 30 μg/mL or more inhibit protein synthesis and mitochondrial respiration[1].
The ability of Mitoguazone to induce apoptosis by inhibiting the polyamine pathway is assessed in three Burkitt's lymphoma cell lines (Raji, Ramos and Daudi) and one prostate carcinoma cell line (MPC 3). Mitoguazone induces apoptosis in all the different human cancer cell lines tested in a concentration- and time-dependent way, and triggers a p53-independent programmed cell death in the human breast cancer MCF7 cell line[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo	The influence of different stages of leukemia P388 on the pharmacokinetics of the antineoplastic agent Mitoguazone in mice is investigated. Independent of the tumor stage investigated, the total clearance of mitoguazone is slightly reduced reflecting a moderate increase of AUC in the serum of leukemia-bearing animals. Furthermore, in an advanced tumor stage the drug levels in kidneys, liver, spleen and serum are found to be elevated to some extent in comparison to tumor-free controls in contrast to an earlier stage of leukemia[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cardiovasc Diabetol. 2023 Jul 6;22(1):168.

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REFERENCES

[1]. J Rizzo, et al. Pharmacokinetic Profile of Mitoguazone (MGBG) in Patients With AIDS Related non-Hodgkin's Lymphoma. Invest New Drugs. 1996;14(2):227-34.

[2]. K Davidson, et al. Mitoguazone Induces Apoptosis via a p53-independent Mechanism. Anticancer Drugs. 1998 Aug;9(7):635-40.

[3]. Xia Jin, et al. Inhibition of HIV Expression and Integration in Macrophages by Methylglyoxal-Bis-Guanylhydrazone. J Virol. 2015 Nov;89(22):11176-89.

[4]. A M Levine, et al. Mitoguazone Therapy in Patients With Refractory or Relapsed AIDS-related Lymphoma: Results From a Multicenter Phase II Trial. J Clin Oncol. 1997 Mar;15(3):1094-103.

[5]. R Amlacher, et al. Influence of Leukemia P388 on the Pharmacokinetics of Mitoguazone in B6D2F1 Mice. Pharmazie. 1990 May;45(5):364-6.

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