

Product Data Sheet

Product Name: DMAT
Cat. No.: GC17837

Chemical Properties

Cas. No. 749234-11-5

Chemical Name 4,5,6,7-tetrabromo-N,N-dimethyl-1H-benzimidazol-2-amine

SMILES CN(C)C1=NC2=C(N1)C(=C(C(=C2Br)Br)Br)Br

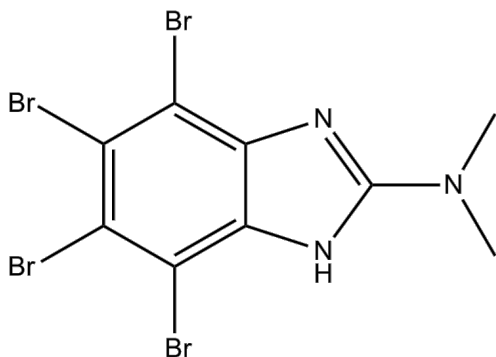
Formula $C_9H_7Br_4N_3$ M.Wt 476.79

Solubility ≥ 23.85 mg/mL in DMSO Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines Human pluripotent adrenocortical cell line H295R (CRL-2128)

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

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Preparation method	Soluble in DMSO > 23.9mg/mL. General tips for obtaining a higher concentration: Please warm the tube at 37°C for 10 minutes and/or shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.
Reacting condition	DMAT in 96% ethanol and Nu-Serum-free culture medium; final concentrations of 10 ⁻⁴ –10 ⁻¹⁰ M; 72 h
Applications	Treatment with DMAT decreased the secretion of aldosterone, dehydroepiandrosterone sulfate, and androstendione and resulted in an accumulation of 17-OH-progesterone(17-Hydroxyprogesterone). Cell growth was inhibited, and cell cycle analysis had revealed a slight induction of apoptosis.
Animal experiment [2]:	
Animal models	6-8-week old male NMRI mice bearing HepG2 human hepatoma cells xenotransplant
Dosage form	500 µg/kg in DMSO/PBS; daily for 10 days; intraperitoneal injection
Application	DMAT application in vivo reduced tumor growth in a xenotransplant model by interference with tumor cell proliferation. DMAT reduced HCC(Hepatocellular carcinoma) growth by interference with NFκB- and Wnt-signaling
Other notes	Please test the solubility of all compounds indoor, and the actual solubility may slightly differ with the theoretical value. This is caused by an experimental system error and it is normal.

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References:

- [1]. Lawnicka H1, Kowalewicz-Kulbat M, et al, Anti-neoplastic effect of protein kinase CK2 inhibitor, 2-dimethylamino-4,5,6,7-tetrabromobenzimidazole (DMAT), on growth and hormonal activity of human adrenocortical carcinoma cell line (H295R) in vitro. Cell Tissue Res. 2010 May;340(2):371-9. doi: 10.1007/s00441-010-0960-1. Epub 2010 Apr 6.
- [2]. Sass G1, Klinger N, et al, Inhibition of experimental HCC growth in mice by use of the kinase inhibitor DMAT. Int J Oncol. 2011 Aug;39(2):433-42. doi: 10.3892/ijo.2011.1037. Epub 2011 May 10.

Background

IC50 value: 0.13uM. DMAT also displays submicromolar IC50 values with almost all of the other kinases with special reference to PKD1, PIM3 and PIM1[3]. Protein kinase CK2 is involved in cell proliferation and survival, and found overexpressed in virtually all types of human cancer, including breast cancer. We demonstrate that inhibition of CK2 with 2-dimethylamino-4,5,6,7-tetrabromobenzimidazole (DMAT), a potent and specific CK2 inhibitor, results in caspase-mediated killing of human breast cancer cells with acquired resistance to antiestrogens [1]. In vitro: Treatment with DMAT decreases the secretion of aldosterone, dehydroepiandrosterone sulfate, and androstendione in H295R human adrenocortical cancer cell line and results in an accumulation of 17-OH-progesterone. Cell growth as measured by the MTT and 5-bromo-2'-deoxyuridine incorporation assays is inhibited, and cell cycle analysis has revealed a slight induction of apoptosis[2]. PIM1 is also inhibited by DMAT by a mechanism which is competitive with respect to ATP. However, IC50 determinations at increasing ATP concentration denote weak competition by ATP which, at almost physiological concentration (0.6 mM), causes only a 5.3-fold decrease in DMAT inhibition, as compared with 1 μ M ATP concentration, whereas in the same range of ATP concentration the IC50 with CK2 increases 22.1-fold, doubling the value calculated with PIM1 (1.2 μ M) [3]. In vivo: Similar to Sorafenib, DMAT interfered with NF κ B activation and Wnt-signaling. Of the kinases inhibited by DMAT at almost equimolar IC50, CK2 and PIM-3 were found to be over-expressed or more active in hepatoma cells and human HCC tissue. Knockdown of PIM-3 or CK2 by shRNA revealed that both kinases are important for hepatoma cell proliferation and survival [4]. DMAT, might represent a promising therapeutic approach in future HCC therapy. Clinical trial: Prostate cancer diagnosis among men with isolated high-grade intraepithelial neoplasia enrolled onto a 3-year prospective phase III clinical trial of oral toremifene[3].

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