DMAT

Cat. No.: HY-15535

CAS No.: 749234-11-5

Molecular Formula: $C_9H_7Br_4N_3$

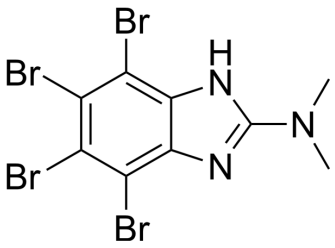
Molecular Weight: 476.79

Target: Casein Kinase

Pathway: Cell Cycle/DNA Damage; Stem Cell/Wnt

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (104.87 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0974 mL	10.4868 mL	20.9736 mL
	5 mM	0.4195 mL	2.0974 mL	4.1947 mL
	10 mM	0.2097 mL	1.0487 mL	2.0974 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.24 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

DMAT is a potent and specific CK2 inhibitor with an IC_{50} value of 130 nM.

IC_{50} & Target

CK2 0.13 μM (IC_{50}, Human CK2)	PIM1 0.148 μM (IC_{50})	PIM2 1.6 μM (IC_{50})	PIM3 0.097 μM (IC_{50})
HIPK2 0.37 μM (IC_{50})	HIPK3 0.59 μM (IC_{50})	DYRK1a 0.41 μM (IC_{50})	DYRK2 0.35 μM (IC_{50})
DYRK3 1.7 μM (IC_{50})	PKD1 0.18 μM (IC_{50})	CDK2 0.64 μM (IC_{50})	

In Vitro

DMAT (1 μM-2.5 μM) DMAT is more efficient in killing antiestrogen resistant cells than parental antiestrogen sensitive MCF-7

	cells. DMAT-induced cell death of antiestrogen resistant cells is mediated by caspases. DMAT inhibits CK2 activity but the inhibition is similar in the three cell lines, MCF-7, TAMR-1 and 182R-6[1]. DMAT has effects on H295R cell proliferation at concentrations of 10^{-4} and 10^{-5}mol/Las compared with the control. DMAT (100 μM) significantly increases apoptosis of H295R cells. DMAT (1 nM-1 μM) significantly decreases aldosterone release into supernatants of 72-h H295R cell cultures as compared with the control[2]. DMAT also inhibits PIM1 by a mechanism which is competitive with respect to ATP, and it is a powerful inhibitor of kinases other than CK2[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	DMAT application in vivo reduces tumor growth in a xenotransplant model by interference with tumor cell proliferation. Biochemical parameters and histology following DMAT administration revealed no alterations in liver tissue[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [1]	Kinase activity tests are performed in a volume of 50 μL containing (final concentrations): 0.1 μg/μL protein extract, 500 μM CK2 substrate peptide (RRRDDDSDDD), 25 mM Tris-HCl, pH 8.5, 100 μM Na_3VO_4, 1 mM DTT, 20 mM NaCl, 5 mM $MgCl_2$, 50 μM ATP and appr 1 μCi [γ-^{32}P]-ATP (3000 Ci/mmol). Samples are incubated for 10 min at 30°C. Aliquots are spotted onto P81 phosphocellulose paper and washed 3×5 min in 0.75% phosphoric acid and once in acetone. Incorporation of radiolabelled phosphate is measured by counting the samples in a liquid scintillation counter. Three independent experiments, each done in duplicate, are performed with reproducible results. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Cell Assay [2]	H295R cells are plated at a density of 2×10^4 cells/well into 96-well microplates in complete culture medium and preincubated for 12 h (5% CO_2, 37°C, 95% humidity). DMAT in 96% ethanol and Nu-Serum-free culture medium is added to the appropriate wells at final concentrations of 10^{-4}-10^{-10} M (the highest concentration of ethanol is 1.8% [vol] in the 10^{-4} M wells). The same volume of Nu-Serum-free culture medium and 96% ethanol is added to the control wells at the same concentration as the solvent in the 10^{-4} M group. Incubation is performed for 72 h under standard conditions (5% CO_2, 37°C, 95% humidity). The absorbance (OD, optical density) of each sample is measured with an enzyme-linked immunosorbent assay (ELISA) microplate reader at a wavelength of 450 nm. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Biochem Biophys Res Commun. 2020 Apr 2;524(2):280-287.
- Johannes Gutenberg-Universität Mainz. Chemie, Pharmazie u. Geowissensch.
- Patent. US20200368248A1.

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REFERENCES

[1]. Yde CW, et al. Induction of cell death in antiestrogen resistant human breast cancer cells by the protein kinase CK2 inhibitorDMAT. Cancer Lett. 2007 Oct 28;256(2):229-37.

[2]. Lawnicka H, 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(

[3]. Pagano MA, et al. The selectivity of inhibitors of protein kinase CK2: an update. Biochem J. 2008 Nov 1;415(3):353-65.

[4]. Sass G, 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.

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