DTHIB

Cat. No.: HY-138280

CAS No.: 897326-30-6

Molecular Formula: $C_{13}H_9ClFN_3O_3$

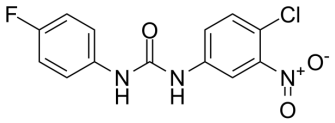
Molecular Weight: 309.68

Target: HSP

Pathway: Cell Cycle/DNA Damage; Metabolic Enzyme/Protease

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 : 100 mg/mL (322.91 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.2291 mL	16.1457 mL	32.2914 mL
5 mM	0.6458 mL	3.2291 mL	6.4583 mL
10 mM	0.3229 mL	1.6146 mL	3.2291 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.08 mg/mL (6.72 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

DTHIB is a direct and selective heat shock factor 1 (HSF1) inhibitor with a K_d of 160 nM for DTHIB binding to the HSF1 DNA binding domain (DBD). DTHIB inhibits HSF1 cancer gene signature (HSF1 CaSig) and selectively stimulates degradation of nuclear HSF1. DTHIB has potently anticancer activities and can be used for prostate cancer research[1].

IC_{50} & Target

Kd: 160 nM (DTHIB binding to the HSF1 DNA binding domain)[1]

In Vitro

DTHIB (5 μM; 48 hours) treatment of C4-2 cells induces cell cycle arrest, with accumulation in the G1 phase. DTHIB stimulates C4-2 PCa cell entry into senescence[1].

DTHIB (0.5-5 μM; 48 hours; C4-2 prostate cancer) treatment reduces steady-state protein abundance of the molecular chaperones P23, HSP27, HSP70, and HSP90-all bona fide HSF1 targets in C4-2 cells[1].

DTHIB dose-dependently reduces the clonal expansion of both C4-2 and PC-3 PCa cells with EC_{50} values of 1.2 μM and 3.0 μM, respectively[1].

In mouse embryonic fibroblasts (MEFs), DTHIB (0.5-10 μM) attenuates the robust acute heat shock induction of the HSP70 and HSP25 molecular chaperones in a dose-dependent manner. DTHIB attenuates the heat shock response by reducing the steady-state transcript abundance of multiple molecular chaperones[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Cycle Analysis[1]

Cell Line:	C4-2 cells
Concentration:	5 μM
Incubation Time:	48 hours
Result:	Induced cell cycle arrest.

Western Blot Analysis[1]

Cell Line:	C4-2 prostate cancer (PCa) cells
Concentration:	0.5 μM, 1 μM, 2.5 μM, 5 μM
Incubation Time:	48 hours
Result:	Dose-dependently inhibited expression of molecular chaperones in C4-2 PCa cells.

In Vivo

DTHIB (5 mg/kg; intraperitoneal injection; daily; for 3 weeks) treatment potently attenuates tumor progression in a C4-2 xenograft mouse model[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Nude mice (6 weeks of age) injected with C4-2 cells[1]
Dosage:	5 mg/kg
Administration:	Intraperitoneal injection; daily; for 3 weeks
Result:	Showed no visible tumor growth over a 3-week period and a 40% reduction in median tumor volume.

CUSTOMER VALIDATION

- Cell Death Dis. 2023 Aug 19;14(8):535.

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REFERENCES

[1]. Bushu Dong, et al. Targeting therapy-resistant prostate cancer via a direct inhibitor of the human heat shock transcription factor 1. Sci Transl Med. 2020 Dec 16;12(574):eabb5647.

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

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