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SZABO-SCANDIC Handels GmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

mail@szabo-scandic.com

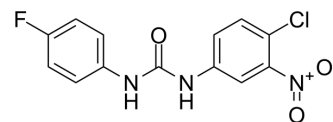
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DTHIB

Cat. No.:	HY-138280
CAS No.:	897326-30-6
Molecular Formula:	C ₁₃ H ₉ ClFN ₃ O 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 Concentration	Mass	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 potentially 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 EC₅₀ values of 1.2 μM and 3.0 μM, respectively^[1].

In mouse embryonic fibroblasts (MEFs), DTHIB (0.5-10 μ M) 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 μ M
Incubation Time:	48 hours
Result:	Induced cell cycle arrest.

Western Blot Analysis^[1]

Cell Line:	C4-2 prostate cancer (PCa) cells
Concentration:	0.5 μ M, 1 μ M, 2.5 μ M, 5 μ M
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 potentially 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.

Tel: 609-228-6898

Fax: 609-228-5909

E-mail: tech@MedChemExpress.com

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