

Product Data Sheet

Product Name: DTHIB
Cat. No.: GC62120

Chemical Properties

Cas. No. 897326-30-6

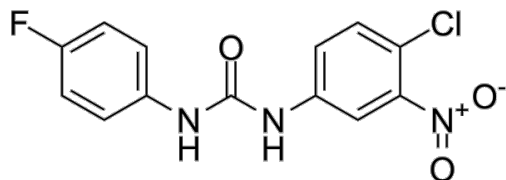
Formula $C_{13}H_9ClFN_3O_3$ M.Wt 309.68

Solubility DMSO : 100 mg/mL (322.91 mM; Storage Store at -20°C
Need ultrasonic)

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



Background

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

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

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CaSig) and selectively stimulates degradation of nuclear HSF1. DTHIB has potent anticancer activities and can be used for prostate cancer research[1].

DTHIB (5 μ M; 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 μ M; 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 μ M and 3.0 μ M, 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].

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

[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.

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