

Product Data Sheet

Product Name: Trifluoperazine

Cat. No.: GC63240

Chemical Properties

Cas. No. 117-89-5

Formula $C_{21}H_{24}F_3N_3S$

M.Wt 407.5

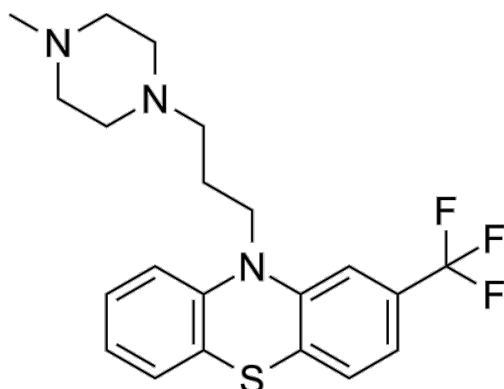
Solubility DMSO : 50mg/mL

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 U87MG human glioblastoma cells

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

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA

Product Data Sheet

Cells were seeded into 96-well plates at a density of 1.5×10^3 cells per well and allowed to attach for 24 hours. Cells were treated with vehicle or the indicated Preparation Method concentrations of Trifluoperazine diluted in complete media for each 24, 48, and 72 hours. Cell proliferation was determined by 3-[4, 5-dimethylthiazol-2-yl]-2, 5-diphenyltetrazolium bromide (MTT) assay.

Reaction Conditions 0-20 μ M for 24, 48, and 72 hours

Applications U87MG cell viability decreased time dependently (24, 48, and 72-hour Trifluoperazine treatment) as well as dose dependently (1, 2, 5, 10, and 20 μ mol/L of Trifluoperazine)

Animal experiment [2]:

Animal models nude mice

Preparation Method In total 2×10^6 HCT116 cells were subcutaneously injected in rear flank of nude mice (five per group). The TFP (10 mg/kg, intraperitoneal) was administrated to each group for five times, 3 days apart.

Dosage form Intraperitoneal injection, 10 mg/kg, 5 times, 3 days apart

Applications Administration of TFP induced remarkably tumor regression with decreased tumor volume. The proliferation index Ki-67 was analyzed and found that TFP was able to reduce the Ki-67 levels in vivo.

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

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA

Product Data Sheet

References:

[1]: Kang S, Hong J, Lee J M, et al. Trifluoperazine, a well-known antipsychotic, inhibits glioblastoma invasion by binding to calmodulin and disinhibiting calcium release channel IP3R[J]. Molecular cancer therapeutics, 2017, 16(1): 217-227.

[2]: Qian K, Sun L, Zhou G, et al. Trifluoperazine as an alternative strategy for the inhibition of tumor growth of colorectal cancer[J]. Journal of cellular biochemistry, 2019, 120(9): 15756-15765.

Background

Trifluoperazine (TFP) is an antagonist against dopamine receptors, act as antipsychotic agent and is widely used to treat schizophrenia or related psychoses [1,2]. Trifluoperazine is also a well-known calmodulin antagonist [3].

Trifluoperazine significantly restricted the invasion of glioblastoma cells at a concentration higher than 1 $\mu\text{mol/L}$, with a half-maximal effective concentration at around 10 $\mu\text{mol/L}$ on invasion of U87MG cells by performing Matrigel transwell invasion assay. The ratio of proliferated glioblastoma cells was significantly decreased in TFP-treated cells [4]. Trifluoperazine inhibits cell vitality, proliferation, and induces cell apoptosis of HCT116 cells with $\text{IC}_{50} = 14 \mu\text{M}$ [3]. Trifluoperazine (30 μM , 24h) induced pancreatic adenocarcinoma (PDAC) MiaPaCa-2 cell death and decreased in intracellular availability of ATP [5].

Trifluoperazine (5 mg/kg/day) significantly suppressed the glioblastoma growth of the xenograft mice as evidenced by a marked decrease of glioblastoma tumor size in TFP-treated mice at day 21, there was no lung metastasis in TFP-treated group in the skin xenograft model [4]. Administration of TFP induced remarkably tumor regression with decreased tumor volume in the xenograft model of human HCT116 [3]. Trifluoperazine binds and mimics the effect of the genetic inhibition of NUPR1, a stress protein that promotes tumor growth and acts as a strong anticancer drug in vitro and in vivo in xenografted nude mice [6].

References:

[1]. de Oliveira Marques L, Soares B, de Lima M S. Trifluoperazine for schizophrenia[J]. Cochrane Database of Systematic Reviews, 2004 (1).

[2]. Hong M, Jenner P, Marsden C D. Comparison of the acute actions of amine-depleting drugs and dopamine receptor antagonists on dopamine function in the brain in rats[J]. Neuropharmacology,

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

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA

Product Data Sheet

1987, 26(2-3): 237-245.

[3]. Qian K, Sun L, Zhou G, et al. Trifluoperazine as an alternative strategy for the inhibition of tumor growth of colorectal cancer[J]. Journal of cellular biochemistry, 2019, 120(9): 15756-15765.

[4]. Kang S, Hong J, Lee J M, et al. Trifluoperazine, a well-known antipsychotic, inhibits glioblastoma invasion by binding to calmodulin and disinhibiting calcium release channel IP3R[J]. Molecular cancer therapeutics, 2017, 16(1): 217-227.

[5]. Huang C, Lan W, Fraunhofer N, et al. Dissecting the anticancer mechanism of trifluoperazine on pancreatic ductal adenocarcinoma[J]. Cancers, 2019, 11(12): 1869.

[6]. Neira J L, Bintz J, Arruebo M, et al. Identification of a drug targeting an intrinsically disordered protein involved in pancreatic adenocarcinoma[J]. Scientific reports, 2017, 7(1): 1-15.

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

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA