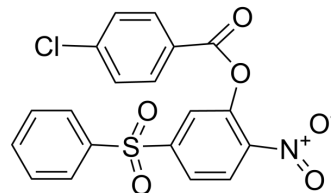


BTB06584

| | |
|--------------------|------------------------------------------------------------------------------------------------------|
| Cat. No.: | HY-15877 |
| CAS No.: | 219793-45-0 |
| Molecular Formula: | C ₁₉ H ₁₂ ClNO ₆ S |
| Molecular Weight: | 417.82 |
| Target: | ATP Synthase |
| Pathway: | Membrane Transporter/Ion Channel |
| 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 : ≥ 47 mg/mL (112.49 mM)
 * "≥" means soluble, but saturation unknown.

| Concentration | Solvent | Mass | | |
|---------------------------|---------|-----------|------------|------------|
| | | 1 mg | 5 mg | 10 mg |
| Preparing Stock Solutions | 1 mM | 2.3934 mL | 11.9669 mL | 23.9338 mL |
| | 5 mM | 0.4787 mL | 2.3934 mL | 4.7867 mL |
| | 10 mM | 0.2393 mL | 1.1967 mL | 2.3934 mL |

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
 Solubility: ≥ 2.5 mg/mL (5.98 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BTB06584 is a selective and IF1-dependent mitochondrial F₁F₀-ATPase inhibitor without compromising ATP synthesis. BTB06584 can delays ischaemic cell death^[1].

IC₅₀ & Target

Mitochondrial F₁F₀-ATPase^[1]

In Vitro

In HL-1 cells, BTB06584 (100 μM) inhibits F₁F₀-ATPase activity with no effect on the mitochondrial membrane potential (ΔΨ_m) or O₂ consumption. BTB06584 (100 μM) pretreatment protects against ischaemic cell death in HL-1 cells prior to a period of ischaemia. ATP consumption is decreased following inhibition of respiration, and ischaemic cell death is reduced^[1]. BTB06584 efficiency is increased by IF1 overexpression and reduced by silencing the protein^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

BTB06584 (1 μ M; 24 hours) treatment rescued defective haemoglobin synthesis in zebrafish pinotage (pnt) mutants in which expression of the Atpif1a gene is lost. The concentrations of BTB06584 that restore haemoglobin biosynthesis also alter mitochondrial bioenergetics in living fish^[1].

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

CUSTOMER VALIDATION

- Bone Res. 2022 Apr 27;10(1):38.
- Pathogens. 2021, 10(3), 283.

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REFERENCES

[1]. Ivanes F, et al. The compound BTB06584 is an IF1 -dependent selective inhibitor of the mitochondrial F1 Fo-ATPase. Br J Pharmacol. 2014 Sep;171(18):4193-4206.

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