Oltipraz

Cat. No.: HY-12519

CAS No.: 64224-21-1

Molecular Formula: $C_8H_6N_2S_3$

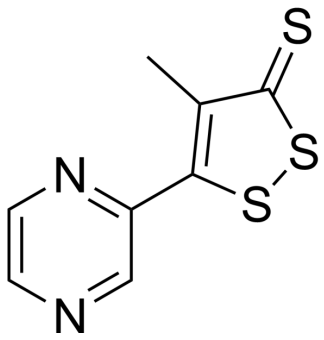
Molecular Weight: 226.34

Target: HIF/HIF Prolyl-Hydroxylase; HIV; Keap1-Nrf2; Parasite

Pathway: Metabolic Enzyme/Protease; Anti-infection; NF-κB

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 6 mg/mL (26.51 mM; Need ultrasonic and warming)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	4.4181 mL	22.0907 mL	44.1813 mL
	5 mM	0.8836 mL	4.4181 mL	8.8363 mL
	10 mM	0.4418 mL	2.2091 mL	4.4181 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: ≥ 1 mg/mL (4.42 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1 mg/mL (4.42 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Oltipraz has an inhibitory effect on HIF-1α activation in a time-dependent manner, completely abrogating HIF-1α induction at ≥10 μM concentrations, the IC_{50} of Oltipraz for HIF-1α inhibition is 10 μM. Oltipraz is a potent Nrf2 activator.

IC_{50} & Target

IC50: 10 μM (HIF-1α)[1];
Nrf2[4]

In Vitro

Oltipraz inhibits HIF-1α activity and HIF-1α-dependent tumor growth, which may result from a decrease in HIF-1α stability through S6K1 inhibition in combination with an H2O2-scavenging effect. Oltipraz treatment also inhibits HIF-1α activation stimulated by either hypoxia or CoCl2. Oltipraz is a cancer chemopreventive agent and has an inhibitory effect on angiogenesis and tumor growth. [1] Oltipraz is also a competitive inhibitor of this cytochrome P450, with an apparent Ki of

	10 μM. [2] MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	In wild-type mice, hepatic levels of mRNA for all of the genes analyzed were significantly increased after Oltipraz treatment, with the highest increase (treated/control) for NQO1 mRNA levels (7.6-fold). The Northern blot analyses demonstrated that the observed increases in GST and NQO1 activities by Oltipraz in wild-type mice were preceded by significant elevations in RNA expression. Interestingly, mRNA levels of Nrf2 itself were increased more than 3-fold by Oltipraz treatment. [2] MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Prolif. 2021 Oct 11;e13142.
- Int J Biol Macromol. 2023 Oct 20:127575.
- Acta Pharmacol Sin. 2020 Aug;41(8):1041-1048.
- Aging Cell. 2021 Oct;20(10):e13483.
- Int Immunopharmacol. 2020 Jul;84:106570.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Lee WH, et al. Oltipraz and dithiolethione congeners inhibit hypoxia-inducible factor-1alpha activity through p70 ribosomal S6 kinase-1 inhibition and H2O2-scavenging effect. Mol Cancer Ther. 2009 Oct;8(10):2791-802.

[2]. Ramos-Gomez M, et al. Sensitivity to carcinogenesis is increased and chemoprotective efficacy of enzyme inducers is lost in nrf2 transcription factor-deficient mice. Proc Natl Acad Sci U S A. 2001 Mar 13;98(6):3410-5.

[3]. Lv S, et al. Glucagon-induced extracellular cAMP regulates hepatic lipid metabolism. J Endocrinol. 2017 Aug;234(2):73-87.

[4]. Eba S, et al.The nuclear factor erythroid 2-related factor 2 activator oltipraz attenuates chronic hypoxia-induced cardiopulmonary alterations in mice.Am J Respir Cell Mol Biol. 2013 Aug;49(2):324-33.

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