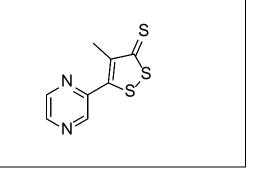
Product data sheet



MedKoo Cat#: 205913				
Name: Oltipraz				
CAS: 64224-21-1				
Chemical Formula: $C_8H_6N_2S_3$				
Exact Mass: 225.9693				
Molecular Weight: 226.33				
Product supplied as:	Powder			
Purity (by HPLC):	\geq 98%			
Shipping conditions	Ambient temperature			
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.			
-	In solvent: -80°C 3 months; -20°C 2 weeks.			



1. Product description:

Oltipraz, a promising cancer chemopreventive agent, is a bifunctional inducer, modulating both phase I and II drug-metabolizing enzymes to enhance carcinogen detoxification.

2. CoA, QC data, SDS, and handling instruction

SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMF	16.0	70.69
DMSO	11.11	49.07

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	4.42 mL	22.09 mL	44.18 mL
5 mM	0.88 mL	4.42 mL	8.84 mL
10 mM	0.44 mL	2.21 mL	4.42 mL
50 mM	0.09 mL	0.44 mL	0.88 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator

Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study

In vitro study

1. Sun Q, Shen X, Ma J, Lou H, Zhang Q. Activation of Nrf2 signaling by oltipraz inhibits death of human macrophages with mycobacterium tuberculosis infection. Biochem Biophys Res Commun. 2020 Oct 20;531(3):312-319. doi: 10.1016/j.bbrc.2020.07.026. Epub 2020 Aug 14. PMID: 32800560.

2. Jiang Z, Bian M, Wu J, Li D, Ding L, Zeng Q. Oltipraz Prevents High Glucose-Induced Oxidative Stress and Apoptosis in RSC96 Cells through the Nrf2/NQO1 Signalling Pathway. Biomed Res Int. 2020 Jun 23;2020:5939815. doi: 10.1155/2020/5939815. PMID: 32685505; PMCID: PMC7333049.

In vivo study

1. Lee WH, Kim YW, Choi JH, Brooks SC 3rd, Lee MO, Kim SG. 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. doi: 10.1158/1535-7163.MCT-09-0420. Epub 2009 Sep 29. PMID: 19789218.

2. Ramos-Gomez M, Kwak MK, Dolan PM, Itoh K, Yamamoto M, Talalay P, Kensler TW. 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. doi: 10.1073/pnas.051618798. PMID: 11248092; PMCID: PMC30667.

Product data sheet



7. Bioactivity

Biological target:

Oltipraz has an inhibitory effect on HIF-1 α activation in a time-dependent manner, completely abrogating HIF-1 α induction at ≥ 10 μ M concentrations, the IC₅₀ of Oltipraz for HIF-1 α inhibition is 10 μ M. Oltipraz is a potent Nrf2 activator.

In vitro activity

Oltipraz significantly inhibited MTB-induced death and apoptosis in human macrophages. MTB-induced reactive oxygen species production, mitochondrial depolarization and programmed necrosis were attenuated by oltipraz in macrophages. Oltipraz activated Nrf2 signaling, causing Keap1-Nrf2 disassociation, Nrf2 protein stabilization and nuclear translocation, simultaneously promoting expression of Nrf2-dependent genes (HO1, NQO1 and GST) in human macrophages.

Reference: Biochem Biophys Res Commun. 2020 Oct 20;531(3):312-319. https://pubmed.ncbi.nlm.nih.gov/32800560/

In vivo activity

Oltipraz treatment abrogated insulin-induced H(2)O(2) production, thereby preventing H(2)O(2)-enhanced HIF-1alpha expression and promoting its ubiquitination and degradation. In an animal model, tumor regression by oltipraz was accompanied by decreases in microvessel density and vascular endothelial growth factor induction. Oltipraz inhibits HIF-1alpha activity and HIF-1alpha-dependent tumor growth, which may result from a decrease in HIF-1alpha stability through S6K1 inhibition in combination with an H(2)O(2)-scavenging effect.

Reference: Mol Cancer Ther. 2009 Oct;8(10):2791-802. https://pubmed.ncbi.nlm.nih.gov/19789218/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.