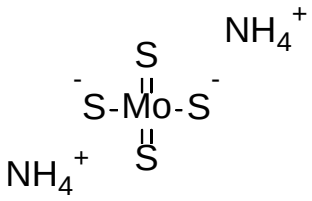
Product data sheet

MedKoo Cat#: 206752 Name: Tiomolibdate diammonium CAS#: 15060-55-6 Chemical Formula: $H_8MoN_2S_4$ Exact Mass: 261.8624 Molecular Weight: 260.28	
Product supplied as:	Powder
Purity (by HPLC):	≥ 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:
Tiomolibdate diammonium, also known as Ammonium tetrathiomolybdate, is a SOD1 inhibitor with potential antiangiogenic and antitumor activities. ammonium Tiomolibdate diammonium has been found to deplete systemic copper reserves through an unknown mechanism. This agent has been shown to inhibit the activities of cuproenzymes, including superoxide dismutase 1 (SOD1) and cytochrome c oxidase (COX), which may contribute to its antiangiogenic and antitumor effects.

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
DMSO		

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	3.84 mL	19.21 mL	38.42 mL
5 mM	0.77 mL	3.84 mL	7.68 mL
10 mM	0.38 mL	1.92 mL	3.84 mL
50 mM	0.08 mL	0.38 mL	0.77 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. Carpenter A, Rassam A, Jennings MH, Robinson-Jackson S, Alexander JS, Erkuran-Yilmaz C. Effects of ammonium tetrathiomolybdate, an oncolytic/angiolytic drug on the viability and proliferation of endothelial and tumor cells. Inflamm Res. 2007 Dec;56(12):515-9. doi: 10.1007/s00011-007-7025-2. PMID: 18210236.

In vivo study

1. Tokuda E, Ono S, Ishige K, Watanabe S, Okawa E, Ito Y, Suzuki T. Ammonium tetrathiomolybdate delays onset, prolongs survival, and slows progression of disease in a mouse model for amyotrophic lateral sclerosis. Exp Neurol. 2008 Sep;213(1):122-8. doi: 10.1016/j.expneurol.2008.05.011. Epub 2008 May 23. PMID: 18617166.

7. Bioactivity
Biological target: Tiomolibdate diammonium is a SOD1 inhibitor.

In vitro activity

The target specificity of tetrathiomolybdate (ATM), an anti-angiogenic, anti-tumor agent against the viability / proliferation of arterial, venous, capillary endothelial and tumor cells was evaluated. Venous proliferation showed high ATM sensitivity (50% reduction ~ > or

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=5 uM ATM, p<0.01), capillary proliferation was inhibited > or =10 uM (p<0.05). Arterial endothelium were less sensitive to ATM, (50% inhibition ~ > or = 20 uM, p<0.01). YPEN-1 were inhibited >50 uM ATM. Capillary viability was inhibited > or =20 microM ATM (p<0.01); venous, arterial and tumor viability show less ATM sensitivity. These data suggest that venous and capillary endothelial proliferation are important targets in ATM therapy, but that other vascular segments and tumor cells may be less influenced.

Reference: Inflamm Res. 2007 Dec;56(12):515-9. https://link.springer.com/article/10.1007%2Fs00011-007-7025-2

In vivo activity

Ammonium tetrathiomolybdate (TTM) is a copper-chelating drug that is capable of removing a copper ion from copper-thiolate clusters, such as SOD1. TTM exerted therapeutic benefits in a mouse model of familial ALS (SOD1(G93A)). TTM treatment significantly delayed disease onset, slowed disease progression and prolonged survival by approximately 20%, 42% and 25%, respectively. TTM also effectively depressed the spinal copper ion level and inhibited lipid peroxidation, with a significant suppression of SOD1 enzymatic activity in SOD1(G93A). These results support the hypothesis that aberrant copper chemistry through a cysteine residue plays a critical role in mutant SOD1 toxicity and that TTM may be a promising therapy for familial ALS with SOD1 mutants.

Reference: Exp Neurol. 2008 Sep;213(1):122-8.
https://www.sciencedirect.com/science/article/abs/pii/S0014488608002197?via%3Dihub

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.