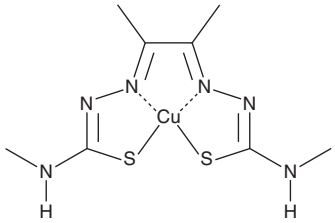
PRODUCT INFORMATION

Cu-ATSM

Item No. 17122

CAS Registry No.: 68341-09-3
Formal Name: (SP-4-2)-[[2,2'-(1,2-dimethyl-1,2-ethanediylidene) bis[N-ethylhydrazinecarbothioamidato-κN2,κS]] (2-)]-copper
Synonyms: copper-ATSM, cu^{II}(atsm)
MF: $C_8H_{14}CuN_6S_2$
FW: 321.9
Purity: ≥95%
UV/Vis.: λ_{max}: 310, 477 nm
Supplied as: A crystalline solid
Storage: -20°C
Stability: ≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Cu-ATSM is supplied as a crystalline solid. A stock solution may be made by dissolving the Cu-ATSM in the solvent of choice. Cu-ATSM is soluble in organic solvents such as DMSO and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of Cu-ATSM in DMSO is approximately 10 mg/ml and approximately 2 mg/ml in DMF.

Cu-ATSM is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, Cu-ATSM should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. Cu-ATSM has a solubility of approximately 0.1 mg/ml in a 1:9 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Cu-ATSM is a copper-containing compound with diverse biological activities.[1-3] It inhibits ferroptotic cell death induced by RSL3, erastin (Item No. 17754), or iron(II) (EC_{50}s = 134, 169, and 171 nM, respectively), as well as RSL3-, erastin-, or iron-induced lipid peroxidation in N27 rat mesencephalic cells when used at a concentration of 1 µM.[1] Cu-ATSM (30 mg/kg per day) increases mutant superoxide dismutase ($SOD1^{G37R}$) protein levels, metalation, and activity in the spinal cord in the $SOD1^{G37R}$ transgenic mouse model of amyotrophic lateral sclerosis (ALS).[2] It also increases the number of α-motor neurons and reduces oxidatively modified proteins in the spinal cord, as well as increases the latency to fall in the rotarod test in the same ALS mouse model. Cu-ATSM containing positron-emitting copper isotopes has been used in PET imaging applications for selective labeling of hypoxic tissue.[3]

References

1. Southon, A., Szostak, K., Acevedo, K.M., *et al.* Cu II (atsm) inhibits ferroptosis: Implications for treatment of neurodegenerative disease. *Br. J. Pharmacol.* **177(3)**, 656-667 (2020).
2. Roberts, B.R., Lim, N.K.H., McAllum, E.J., *et al.* Oral treatment with CuII(atsm) increases mutant SOD1 in vivo but protects motor neurons and improves the phenotype of a transgenic mouse model of amyotrophic lateral sclerosis. *J. Neurosci.* **34(23)**, 8021-8031 (2014).
3. Vavere, A.L. and Lewis, J.S. Cu-ATSM: A radiopharmaceutical for the PET imaging of hypoxia. *Dalton Trans.* **43**, 4893-4902 (2007).

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