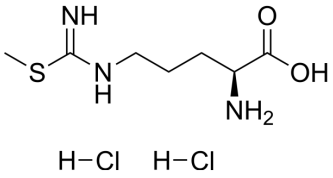
S-MTC dihydrochloride

Cat. No.:	HY-U00432A
CAS No.:	209589-59-3
Molecular Formula:	$C_7H_{17}Cl_2N_3O_2S$
Molecular Weight:	278.2
Target:	NO Synthase
Pathway:	Immunology/Inflammation
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	S-MTC (S-Methyl-L-thiocitrulline) dihydrochloride is a selective type I nitric oxide synthase (NOS) inhibitor.
IC_{50} & Target	NOS[1]
In Vitro	S-MTC dihydrochloride (10 or 100?μM) reduces cellular NO release in the absence of $Aβ_{1-42}$. At 100?μM, S-MTC dihydrochloride decreases cell viability. S-MTC dihydrochloride (100?μM) significantly lowers nitrite production (11.2±1.1?μM) when compared to control (no NOS inhibitor exposure; 19.6±1.2?μM). Nitrite productions after $Aβ_{1-42}$ and L-NOARG (100?μM) or $Aβ_{1-42}$ and S-MTC dihydrochloride (100?μM) treatments are significantly lower than $Aβ_{1-42}$ alone (33.5±2.0 and 34.5±1.6?μM, respectively). S-MTC dihydrochloride (100?μM) is able to significantly reduce nitrite production (25.2±1.1?μM) as compared to $Aβ_{1-42}$ treatment alone (38.3±2.7?μM), when administered after $Aβ_{1-42}$ at the 1?h time point. S-MTC dihydrochloride (100?μM) concentration decreases both MTT (87±1% of control) and NR (80±1% of control, respectively) levels. The co-administration of S-MTC dihydrochloride (100?μM) and $Aβ_{1-42}$ significantly reverses the effects of $Aβ_{1-42}$ alone (72±2% vs 61±2% of control)[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	S-MTC dihydrochloride (S-methyl-L-thiocitrulline) is a selective neuronal NOS-inhibitor. Following pretreatment with S-MTC dihydrochloride (i.c.v.), the HBO_2-induced antinociception is significantly antagonized. In Experiment #2, different groups of mice are pretreated with naltrexone hydrochloride (NTX) (3.0 mg/kg, i.p.), L-NAME (1.0 μg/mouse, i.c.v.), S-MTC dihydrochloride (1.0 μg/mouse, i.c.v.) or N^5-(1-iminoethyl)-L-ornithine (L-NIO) (3.0 mg/kg, s.c.) 15-30 min prior to HBO_2 treatment. The antinociceptive effect assessed 90 min after HBO_2 treatment is completely abolished by NTX and L-NAME, antagonized by two-thirds by S-MTC dihydrochloride and largely unaffected by L-NIO ($F=25.57$, $p<0.0001$)[2]. At a dose of 0.3 mg/kg, S-MTC dihydrochloride (SMTC) causes a rise in mean blood pressure (BP). At doses of 1.0, 3.0 and 10 mg/kg, S-MTC dihydrochloride causes falls in heart rate, rises in BP and vasoconstriction in all three vascular beds[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Law A, et al. Neuroprotective and neurorescuing effects of isoform-specific nitric oxide synthase inhibitors, nitric oxide scavenger, and antioxidant against beta-amyloid toxicity. Br J Pharmacol. 2001 Aug;133(7):1114-24.

[2]. Zelinski LM, et al. A prolonged nitric oxide-dependent, opioid-mediated antinociceptive effect of hyperbaric oxygenin mice. J Pain. 2009 Feb;10(2):167-72.

[3]. Wakefield ID, et al. Comparative regional haemodynamic effects of the nitric oxide synthase inhibitors, S-methyl-L-thiocitrulline and L-NAME, in conscious rats. Br J Pharmacol. 2003 Jul;139(6):1235-43.

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