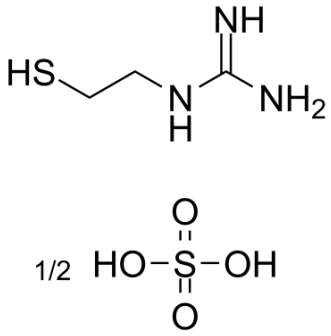
MEG hemisulfate

Cat. No.:	HY-138454
CAS No.:	3979-00-8
Molecular Formula:	$C_3H_9N_3S.{}_1/_2H_2O_4S$
Molecular Weight:	168.23
Target:	NO Synthase
Pathway:	Immunology/Inflammation
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	MEG (Mercaptoethylguanidine) hemisulfate is a potent and selective inhibitor of the inducible NO synthase (iNOS), with EC_{50} s of 11.5, 110, and 60 μM for iNOS, ecNOS, and bNOS respectively in tissue homogenates. MEG hemisulfate is also a potent scavenger of peroxynitrite and inhibits peroxynitrite-induced oxidative processes. MEG hemisulfate has a protective effect in many experimental models of inflammation, including ischemia/reperfusion injury, periodontitis, hemorrhagic shock, inflammatory bowel disease, and endotoxic and septic shock[1][2][3][4].
IC_{50} & Target	IC50: 11.5 μM (iNOS), 110 μM (ecNOS), 60 μM (bNOS)[1]
In Vitro	MEG (0.1-1000 μM; 18 h) reduces nitrite accumulation in the supernatant of cultured J774.2 macrophages activated with LPS (10 μg/mL) and INF (50 μg/mL). MEG inhibits iNOS activity in homogenates of lungs taken from LPS-treated rats[1]. MEG (1 μM-3 mM; 3 min) dose-dependently inhibits the peroxynitrite-induced oxidation of cytochrome c^{2+} and hydroxylation of benzoate[2]. MEG (1-300 μM) inhibits the suppression of mitochondrial respiration and DNA single strand breakage in response to peroxynitrite in J774 cells[2]. MEG (300 μM; 30 min) inhibits the suppression of vascular contractility in response to peroxynitrite in thoracic aortic rings[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	MEG (10 mg/kg; i.p. for 5 d) attenuates the degree of lipid peroxidation, protein oxidation, and peroxynitrites level and ameliorated the decrease of antioxidant enzymes activities in the esophagus of rats subjected to caustic burn injury[3]. MEG (30-60 mg/kg; a single i.p.) decreases mean arterial blood pressure (MAP) of normal rats[1]. MEG (10 mg/kg; a single i.p.) improves the renal dysfunction and tissue injury induced by ischemia/reperfusion (I/R) of rat kidney[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Sprague-Dawley rats (200-250 g) were injured the esophagus[3]
Dosage:	10 mg/kg
Administration:	I.p. for 5 days
Result:	Reduced the stenosis index (SI) and the histopathologic damage score. Decreased the malondialdehyde and protein carbonyl content and increased the activities of superoxide dismutase and glutathione peroxidase.

	Regulated the nitrate and nitrite level.

REFERENCES

[1]. Southan GJ, et, al. Spontaneous rearrangement of aminoalkylisothioureas into mercaptoalkylguanidines, a novel class of nitric oxide synthase inhibitors with selectivity towards the inducible isoform. Br J Pharmacol. 1996 Feb;117(4):619-32.

[2]. Szabó C, et, al. Mercaptoethylguanidine and guanidine inhibitors of nitric-oxide synthase react with peroxynitrite and protect against peroxynitrite-induced oxidative damage. J Biol Chem. 1997 Apr 4;272(14):9030-6.

[3]. Guven A, et, al. Mercaptoethylguanidine attenuates caustic esophageal injury in rats: a role for scavenging of peroxynitrite. J Pediatr Surg. 2011 Sep;46(9):1746-52.

[4]. Guven A, et, al. Scavenging of peroxynitrite reduces renal ischemia/reperfusion injury. Ren Fail. 2008;30(7):747-54.

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