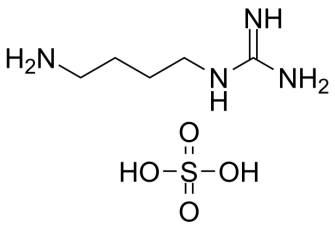
Agmatine sulfate

Cat. No.:	HY-101238
CAS No.:	2482-00-0
Molecular Formula:	$C_5H_{16}N_4O_4S$
Molecular Weight:	228.27
Target:	Imidazoline Receptor; NO Synthase; Endogenous Metabolite
Pathway:	Neuronal Signaling; Immunology/Inflammation; Metabolic Enzyme/Protease
Storage:	4°C, sealed storage, away from moisture * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 100 mg/mL (438.08 mM)
DMSO : < 1 mg/mL (insoluble or slightly soluble)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.3808 mL	21.9039 mL	43.8078 mL
5 mM	0.8762 mL	4.3808 mL	8.7616 mL
10 mM	0.4381 mL	2.1904 mL	4.3808 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
Solubility: 25 mg/mL (109.52 mM); Clear solution; Need ultrasonic and warming

BIOLOGICAL ACTIVITY

Description

Agmatine sulfate exerts modulatory action at multiple molecular targets, such as neurotransmitter systems, ion channels and nitric oxide synthesis. It is an endogenous agonist at imidazoline receptor and a NO synthase inhibitor.

IC_{50} & Target

Human Endogenous Metabolite

In Vitro

Agmatine binds to alpha 2-adrenergic and imidazoline receptors and stimulates release of catecholamines from adrenal chromaffin cells. Its biosynthetic enzyme, arginine decarboxylase, is present in brain. Agmatine, locally synthesized, is an endogenous agonist at imidazoline receptors, a noncatecholamine ligand at alpha 2-adrenergic receptors and may act as a neurotransmitter[1]. Agmatine is synthesized in the brain, stored in synaptic vesicles in regionally selective neurons, accumulated by uptake, released by depolarization, and inactivated by agmatinase. Agmatine inhibits nitric oxide synthase, and induces the release of some peptide hormones[2]. Agmatine, 4-(aminobutyl)guanidine, is produced by decarboxylation of L-arginine by the enzyme arginine decarboxylase. Agmatine is a competitive inhibitor of all NOS isoenzymes but not an

	NO precursor. K_i values are approximately 660 μM (NOS I), 220 μM (NOS II) and 7.5 mM (NOS III)[3]. Agmatine stimulates nitrite production three-fold above basal nitrite formation by endothelial cells. Agmatine displaces [^{3}H]-idazoxan from endothelial cellmembranes and is found to induce transients in the cytosolic calcium of endothelial cells. The transients could be downregulated by repeated exposure to agmatine but are not affected by pretreatment with norepinephrine[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Agmatine produces an antidepressant-like effect when assessed in the forced swimming test and in the tail suspension test in mice (dose range 0.01-50 mg/kg, i.p.), without accompanying changes in ambulation in an open-field[5]. In ischemic stroke, agmatine protects the blood-brain barrier, which can be monitored in vivo by quantification of permeability by using dynamic contrast-enhanced MR imaging[6]. Agmatine substantially augments the antidepressant-like effect of MK-801, reinforcing the notion that this compound modulates NMDA receptor activation[7]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [6][7]	Rats: Thirty-four male Sprague-Dawley rats are subjected to transient MCA occlusion for 90 minutes. Immediately after reperfusion, agmatine (100 mg/kg) or normal saline is injected intraperitoneally into the agmatine-treated group (n= 17) or the control group, respectively. MR imaging is performed after reperfusion[6]. Mice: Mice are pretreated with a range of sub-effective doses of either fluoxetine (1, 2.5 and 5 mg/kg, p.o.; a selective serotonin reuptake inhibitor), imipramine (0.01, 0.05 and 0.1 mg/kg, p.o.; a tricyclic antidepressant), bupropion (0.1, 0.5 and 1 mg/kg, p.o.; dopamine reuptake inhibitor with subtle activity on noradrenergic reuptake), or MK-801 (0.0001, 0.0005 and 0.001 mg/kg, p.o.; noncompetitive NMDA receptor antagonist) and immediately after, a sub-effective dose of either agmatine (0.0001 mg/kg p.o.) or vehicle is administered. After 60 min, the animals are subjected to behavioral testing[7]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Li G, et al. Agmatine: an endogenous clonidine-displacing substance in the brain. Science. 1994 Feb 18;263(5149):966-9.

[2]. Reis DJ, et al. Is agmatine a novel neurotransmitter in brain? Trends Pharmacol Sci. 2000 May;21(5):187-93.

[3]. Galea E, et al. Inhibition of mammalian nitric oxide synthases by agmatine, an endogenouspolyamine formed by decarboxylation of arginine. Biochem J. 1996 May 15;316 (Pt 1):247-9.

[4]. Morrissey JJ, et al. Agmatine activation of nitric oxide synthase in endothelial cells. Proc Assoc Am Physicians. 1997 Jan;109(1):51-7.

[5]. Zomkowski AD, et al. Agmatine produces antidepressant-like effects in two models of depression in mice. Neuroreport. 2002 Mar 25;13(4):387-91.

[6]. Ahn SS, et al. Effects of agmatine on blood-brain barrier stabilization assessed by permeability MRI in a rat model of transient cerebral ischemia. AJNR Am J Neuroradiol. 2015 Feb;36(2):283-8.

[7]. Neis VB, et al. Agmatine enhances antidepressant potency of MK-801 and conventional antidepressants in mice. Pharmacol Biochem Behav. 2015 Mar;130:9-14.

Caution: Product has not been fully validated for medical applications. For research use only.

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