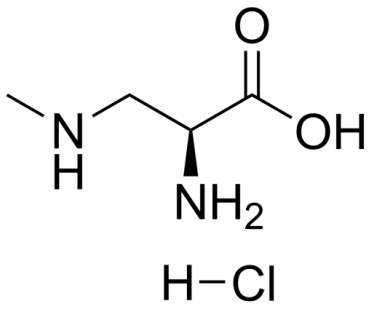
Data Sheet

Product Name:	β-N-methylamino-L-alanine (hydrochloride)
Cat. No.:	CS-W016262
CAS No.:	16012-55-8
Molecular Formula:	$C_4H_{11}ClN_2O_2$
Molecular Weight:	154.60
Target:	Bacterial; mGluR; PKC
Pathway:	Anti-infection; Epigenetics; GPCR/G Protein; Neuronal Signaling; TGF-beta/Smad
Solubility:	H_2O : 31.25 mg/mL (ultrasonic;warming;heat to 60°C)



BIOLOGICAL ACTIVITY:

β-N-methylamino-L-alanine hydrochloride (BMAA hydrochloride) is a neurotoxin produced by cyanobacteria. β-N-methylamino-L-alanine hydrochloride activates **mGluR3** and inhibits **PKC**. β-N-methylamino-L-alanine hydrochloride can be used in the research of neurodegenerative diseases and immune diseases[1][2][3][4][5][6][7][8][9][10][11][12][13]. ***In Vitro:***β-N-methylamino-L-alanine hydrochloride (0.05-3 mM, 24 h) inhibits melatonin synthesis by mGluR3 activation and PKC inhibitions in primary pinealocytes[4].

β-N-Methylamino-L-Alanine (500 μM, 21 days) hydrochloride shows toxicity in PC12 cells[7].

β-N-methylamino-L-alanine (1-3 mM, 48 h) hydrochloride suppresses cell cycle progression at the G1/S checkpoint and proliferation in non-neuronal cells NIH3T3[9].

β-N-methylamino-L-alanine (50-1000 μM, 24 h) hydrochloride perturbs alanine, aspartate and glutamate metabolism pathways in human neuroblastoma cells[10].

β-N-methylamino-L-alanine (1.5-2.0 mM, 24 h) hydrochloride alters morphology, ATP levels and reduces proliferation of fish immune cells (CLC)[11].

In Vivo:β-N-methylamino-L-alanine (460 mg/kg, s.c., daily, 2 weeks) hydrochloride inhibits melatonin synthesis in a Wistar rat model[4].

β-N-methylamino-L-alanine (BMAA-HCl, 400 mg kg, s.c.) hydrochloride induces developmental neurotoxicity in a rat model[5].

β-N-methylamino-L-alanine (100-350 mg/kg, i.p., 5 consecutive days) hydrochloride causes neurological and pathological phenotypes mimicking Amyotrophic Lateral Sclerosis (ALS) in male rats[6].

β-N-methylamino-L-alanine (5-10 nmol, intravitreal injections) hydrochloride induces ***in vivo*** retinal cell death in mice[8].

β-N-methylamino-L-alanine (250 mg/kg, i.p., 5 consecutive days) hydrochloride produces oxidative damage in liver and kidney of rats, with the significant increase of lipid peroxidation and high catalase activity[12].

β-N-methylamino-L-alanine (40-460 mg/kg, s.c., 2 days) hydrochloride perturbs the intermediary metabolism in neonatal rats, with impairments in learning and memory function[13].

References:

[1]. Caller T, et, al. The Potential Role of BMAA in Neurodegeneration. Neurotox Res. 2018 Jan; 33(1): 222-226.

[2]. Cox PA, et, al. BMAA and Neurodegenerative Illness. Neurotox Res. 2018 Jan; 33(1): 178-183.

[3]. Okamoto S, et, al. β-N-methylamino-L-alanine (BMAA) suppresses cell cycle progression of non-neuronal cells. Sci Rep. 2018 Dec 20; 8(1): 17995.

[4]. Pierozan P, et al. The environmental neurotoxin β-N-methylamino-L-alanine inhibits melatonin synthesis in primary pinealocytes and a rat model. J Pineal Res. 2018 Aug;65(1):e12488.

[5]. van Onselen R, et al. Evaluating amino acids as protectants against β-N-methylamino-l-alanine-induced developmental neurotoxicity in a rat model. Toxicol Appl Pharmacol. 2020 Sep 15;403:115140.

[6]. de Munck E, et al. β-N-methylamino-l-alanine causes neurological and pathological phenotypes mimicking Amyotrophic Lateral Sclerosis (ALS): the first step towards an experimental model for sporadic ALS. Environ Toxicol Pharmacol. 2013 Sep;36(2):243-255.

[7]. van Onselen R, et al. β-N-Methylamino-L-Alanine Toxicity in PC12: Excitotoxicity vs. Misincorporation. Neurotox Res. 2018 Jan;33(1):15-23.

[8]. Santucci S, et al. beta-N-methylamino-L-alanine induced in vivo retinal cell death. J Neurochem. 2009 May;109(3):819-25.

[9]. Okamoto S, et al. β-N-methylamino-L-alanine (BMAA) suppresses cell cycle progression of non-neuronal cells. Sci Rep. 2018 Dec 20;8(1):17995.

[10]. Engskog MK, et al. β-N-Methylamino-L-alanine (BMAA) perturbs alanine, aspartate and glutamate metabolism pathways in human neuroblastoma cells as determined by metabolic profiling. Amino Acids. 2017 May;49(5):905-919.

[11]. Sieroslawska A, et al. Assessment of the cytotoxic impact of cyanotoxin beta-N-methylamino-L-alanine on a fish immune cell line. Aquat Toxicol. 2019 Jul;212:214-221.

[12]. de Munck E, et al. Effect of β-N-methylamino-L-alanine on oxidative stress of liver and kidney in rat. Environ Toxicol Pharmacol. 2013 Mar;35(2):193-9.

[13]. Engskog MK, et al. The cyanobacterial amino acid β-N-methylamino-l-alanine perturbs the intermediary metabolism in neonatal rats. Toxicology. 2013 Oct 4;312:6-11.

CAIndexNames:

L-Alanine, 3-(methylamino)-, hydrochloride (1:1)

SMILES:

N[C@@H](CNC)C(O)=O.[H]Cl

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