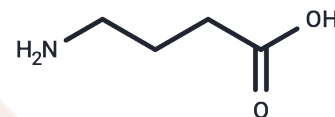


γ -Aminobutyric acid

Chemical Properties

CAS No. :	56-12-2
Formula:	C ₄ H ₉ NO ₂
Molecular Weight:	103.12
Storage:	Powder: -20°C for 3 years In solvent: -80°C for 1 year Actual storage temperature shall be subject to the COA.



Biological Description

Description	γ -Aminobutyric acid (4-Aminobutyric acid) is the predominant inhibitory neurotransmitter in the central nervous system.
Targets(IC50)	GABA Receptor, Endogenous Metabolite
In vitro	γ -Aminobutyric acid (GABA) functions primarily as an inhibitory neurotransmitter in the mature central nervous system. The addition of GABA into the cell culture medium promoted the proliferation of GABRP-expressing PDAC cells, but not GABRP-negative cells, and GABAA receptor antagonists inhibited this growth-promoting effect by GABA. The HEK293 cells constitutively expressing exogenous GABRP revealed the growth-promoting effect of GABA treatment. GABA treatment in GABRP-positive cells increased intracellular Ca ²⁺ levels and activated the mitogen-activated protein kinase/extracellular signal-regulated kinase (MAPK/Erk) cascade[1]. GABA exerts antidiabetic effects by acting on both the islet β -cells and immune system. Unlike in adult brain or islet α -cells in which GABA exerts hyperpolarizing effects, in islet β -cells, GABA produces membrane depolarization and Ca ²⁺ influx, leading to the activation of PI3K/Akt-dependent growth and survival pathways[2].
In vivo	GABA is the principal inhibitory neurotransmitter in the adult brain that has a parallel inhibitory role in the immune system. GABAergic medications are used to treat anxiety, alcohol withdrawal, epilepsy, and to induce sedation, and anesthesia. GABA is neuroprotective in animal models of stroke. GABA treatment decreases inflammatory cytokine production in peripheral macrophages. It decreases T cell autoimmunity and the development of inflammatory responses in the nonobese diabetic mouse model of type 1 diabetes[3]. In the adult brain, GABA induces a fast inhibition in neurons mainly through the GABAA receptor (GABAAR). GABA is produced by pancreatic β -cells. GABA released from β -cells can act on GABAAR in the α -cells, causing membrane hyperpolarization and hence suppressing glucagon secretion. GABA-treated mice showed higher circulating insulin, lower glucagon, nearly normal glycemia, improved metabolic conditions, and maintained close to normal glucose tolerance during a period of 53 d after STZ injections[2].
Cell Research	GABRP-positive cell lines, KLM-1 and PK-45P, and GABRP-negative cell lines, PK-59 and KP-1N, are incubated with GABA or GABA receptor agonist Muscimol at serial concentration (0, 1, 10, 100 μ mol/L) in appropriate medium supplemented with 1% FBS for 6 days. To inhibit the GABA-mediated pathway, cells are incubated with 250 μ mol/L

A DRUG SCREENING EXPERT

Cell Research	of GABAA receptor antagonist bicuculline methiodide or 1 mmol/L of GABAB receptor antagonist CGP-35348. After 6 days of exposure to either of these drugs, cell viability is measured by MTT assay as described above.(Only for Reference)
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Solubility Information

Solubility	H2O: 100 mg/mL (969.74 mM),Sonication is recommended. DMSO: Insoluble, (< 1 mg/ml refers to the product slightly soluble or insoluble)
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Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	9.6974 mL	48.4872 mL	96.9744 mL
5 mM	1.9395 mL	9.6974 mL	19.3949 mL
10 mM	0.9697 mL	4.8487 mL	9.6974 mL
50 mM	0.1939 mL	0.9697 mL	1.9395 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Takehara A, et al. Cancer Res. 2007, 67(20):9704-12.

Li M, Yuan H, Yang X, et al. Glutamine-glutamate centered metabolism as the potential therapeutic target against Japanese encephalitis virus-induced encephalitis. Cell & Bioscience. 2025, 15(1): 6.

Soltani N, et al. Proc Natl Acad Sci U S A. 2011, 108(28):11692-7.

Bhat R, et al. Proc Natl Acad Sci U S A. 2010, 107(6):2580-5.

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