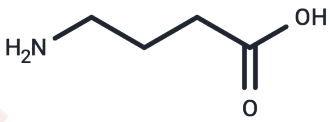
Data Sheet (Cat.No.T0508)

TargetMol

γ-Aminobutyric acid

Chemical Properties



CAS No. : 56-12-2

Formula: C4H9NO2

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 belongs to natural products and functions as an agonist of GABAA and GABAB receptors, possessing central sedative effects, cell permeability, and the ability to modulate neuronal excitability. This compound is used in neuroscience research and exhibits anxiolytic, anticonvulsant, and neuroprotective activities.
Targets(IC50)	GABA Receptor,Endogenous Metabolite
In vitro	**Methods**: GABRP-positive pancreatic cancer cells (KLM-1, PK-45P) and HEK293 cells with exogenous GABRP expression were treated with γ-Aminobutyric acid (GABA) (1–100 μM), with intervention by GABAA receptor antagonists; proliferation was detected by MTT assay and BrdU incorporation, intracellular Ca^{2+} was measured by Fura-2, and Erk phosphorylation was detected by Western blot. **Results**: γ-Aminobutyric acid (GABA) dose-dependently promoted cell proliferation, elevated intracellular Ca^{2+}, and activated the Erk pathway; these effects were inhibited by GABAA receptor antagonists. [1] **Methods**: INS-1 cells and mouse primary islet β-cells were treated with γ-Aminobutyric acid (GABA) (100 μM); membrane potential was recorded by patch-clamp, Ca^{2+} influx was detected by Fura-2 calcium imaging, and Akt phosphorylation was detected by Western blot. **Results**: γ-Aminobutyric acid (GABA) induced membrane depolarization and Ca^{2+} influx in β-cells, activated the PI3K/Akt pathway, exerting pro-survival and anti-diabetic effects. [2]
In vivo	**Methods**: MDSD and NOD diabetic mice received daily intraperitoneal injections of GABA (20 μmol per mouse) for several weeks; blood glucose was monitored with a glucometer, and circulating insulin and glucagon levels were detected by RIA. **Results**: γ-Aminobutyric acid (GABA) treatment increased circulating insulin, decreased glucagon, maintained near-normal blood glucose levels, and significantly improved metabolic status in mice. [2] **Methods**: SJL/J mice were immunized with PLP 139-151 to induce EAE models; GABAergic drugs topiramate (100 mg/kg) or vigabatrin (400 mg/kg) were administered orally daily, dissolved in PBS, for 37 consecutive days or starting from the disease peak. Cytokines were detected by ELISA, and spinal cord inflammation was assessed by H&E staining. **Results**: GABAergic drugs significantly reduced EAE clinical scores, inhibited IL-1β and IL-6 production in macrophages and IFN-γ and IL-17 production in T cells, and

In vivo	decreased central inflammatory infiltration. [3]
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 μmol/L) in appropriate medium supplemented with 1% FBS for 6 days. To inhibit the GABA-mediated pathway, cells are incubated with 250 μmol/L 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)

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)

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, Akio et al. Gamma-aminobutyric acid (GABA) stimulates pancreatic cancer growth through overexpressing GABAA receptor pi subunit. Cancer research vol. 67,20 (2007): 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, Nepton et al. GABA exerts protective and regenerative effects on islet beta cells and reverses diabetes. Proceedings of the National Academy of Sciences of the United States of America vol. 108,28 (2011): 11692-7.

Bhat, Roopa et al. Inhibitory role for GABA in autoimmune inflammation. Proceedings of the National Academy of Sciences of the United States of America vol. 107,6 (2010): 2580-5.

Tel:781-999-4286 E_mail:info@targetmol.com Address:34 Washington Street,Wellesley Hills,MA 02481