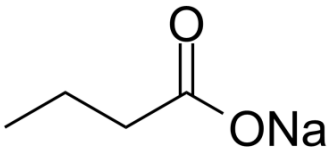
Sodium Butyrate

Cat. No.:	HY-B0350A
CAS No.:	156-54-7
Molecular Formula:	$C_4H_7NaO_2$
Molecular Weight:	110.09
Target:	HDAC; Autophagy; Apoptosis
Pathway:	Cell Cycle/DNA Damage; Epigenetics; Autophagy; Apoptosis
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (**908.35 mM;** Need ultrasonic)
DMSO : 1 mg/mL (**9.08 mM;** Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	9.0835 mL	45.4174 mL	90.8348 mL
	5 mM	1.8167 mL	9.0835 mL	18.1670 mL
	10 mM	0.9083 mL	4.5417 mL	9.0835 mL

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

In Vivo

1. Sodium butyrate (SB) is prepared in PBS[5].

BIOLOGICAL ACTIVITY

Description

Sodium Butyrate (Butanoic acid sodium salt) is a **histone deacetylase (HDAC)** inhibitor, with anti-tumor effects in several cancers.

IC_{50} & Target

HDAC

In Vitro

Sodium Butyrate induces morphological changes, inhibits cell proliferation and impairs cell viability in NPC cells. Sodium Butyrate (1, 5, 10 mM) is cytotoxic to NPC cells, inducing a dose- and time-dependent decrease in cell viability, in both 5-8F and 6-10B cells. Sodium Butyrate induces nasopharyngeal carcinoma cell apoptosis by activating the mitochondrial apoptotic axis. Moreover, SOCE inhibition and disruption of the CRAC channel can attenuate the apoptosis induced by Sodium Butyrate[1]. Sodium butyrate significantly decreases cell viability, accompanied by reduced levels of p-mTOR and PCNA protein. Sodium butyrate, in a dose-dependent manner,

	induces cell cycle arrest in G0/G1 phase and reduces the numbers of cells in S phase. In addition, relative expression of p21, p27, and pro-apoptosis bak genes, and protein levels of p21Waf1/Cip1, p27Kip1, cyclinD3, CDK4, and Cleave-caspase3 are increased by higher concentrations of sodium butyrate (1, 5, 10 mM), and the levels of cyclinD1 and CDK6 are reduced by 5 and 10 mM butyrate[3].
In Vivo	Sodium Butyrate (300 mg/kg, s.c.) administration immediately after HI provided almost complete neuroprotection in comparison with non-treated animals. Sodium butyrate administration results in an increased number of microglial cells to 150% of vehicle-treated animals in the ipsilateral side. Sodium butyrate promotes the polarization of microglia from M1- to M2-like phenotype after neonatal hypoxia-ischemia[2]. Sodium butyrate (300 mg/kg, s.c.) in combination with AK-7 (20 mg/kg, i.p.) significantly alleviates this reduction of the time spent exploring new objects, ameliorates the reduction of the number of Ki67-positive cells and DCX-immunoreactive neuroblasts in the dentate gyrus of the mice. In addition, sodium butyrate reverses SIRT2-related age phenotypes[4].

PROTOCOL

Cell Assay [1]	Cells are seeded at a density of 2,000 cells/well in 96-well plates with 200 μL culture medium containing Sodium Butyrate at different concentrations. Then, the cells are consecutively cultured for 72 h. Every 24 h, 20 μL 5 mg/mL MTT solution is added into the corresponding well, and the cells are cultured for another 4 h. Then, the solution is replaced with 150 μL dimethylsulfoxide, followed by gentle agitation of the plates for 15 min at room temperature. Finally, the absorbance at 492 nm is measured to represent the cell viability. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Seven-day-old rat pups are subjected to unilateral carotid artery ligation followed by 60 min of hypoxia (7.6% O_2). Sodium Butyrate (300 mg/kg) is administered in a 5-day regime with the first injection given immediately after hypoxic exposure. The damage of the ipsilateral hemisphere is evaluated by hematoxylin-eosin staining 6 days after the insult. Samples are collected at 24 and 48 h and 6 days. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- **Neurobiol Dis**. 2017 Dec 14;111:12-25.
- **BMC Vet Res**. 2019 Jul 29;15(1):267.
- **Int J Stem Cell Res Ther**. 2020, 7:068.
- **Patent**. US20180263995A1.

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REFERENCES

[1]. Huang W, et al. Inhibition of store-operated Ca2+ entry counteracts the apoptosis of nasopharyngeal carcinoma cells induced by sodium butyrate. Oncol Lett. 2017 Feb;13(2):921-929

[2]. Jaworska J, et al. The potential neuroprotective role of a histone deacetylase inhibitor, sodium butyrate, after neonatal hypoxia-ischemia. J Neuroinflammation. 2017 Feb 10;14(1):34

[3]. Qiu Y, et al. Effect of sodium butyrate on cell proliferation and cell cycle in porcine intestinal epithelial (IPEC-J2) cells. In Vitro Cell Dev Biol Anim. 2017 Jan 27

[4]. Jung HY, et al. Sirtuin-2 inhibition affects hippocampal functions and sodium butyrate ameliorates the reduction in novel object memory, cell proliferation, and neuroblast differentiation. Lab Anim Res. 2016 Dec;32(4):224-230

[5]. Wang P, et al. Sodium butyrate triggers a functional elongation of microglial process via Akt-small RhoGTPase activation and HDACs inhibition. Neurobiol Dis. 2017 Dec 14;111:12-25.

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