4-Phenylbutyric acid

Cat. No.: HY-A0281

CAS No.: 1821-12-1

Molecular Formula: $C_{10}H_{12}O_2$

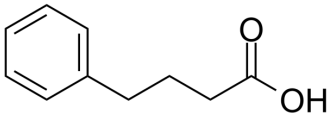
Molecular Weight: 164.2

Target: HDAC; Virus Protease

Pathway: Cell Cycle/DNA Damage; Epigenetics; Anti-infection

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (609.01 mM; Need ultrasonic)

H_2O : 2 mg/mL (12.18 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	6.0901 mL	30.4507 mL	60.9013 mL
5 mM	1.2180 mL	6.0901 mL	12.1803 mL
10 mM	0.6090 mL	3.0451 mL	6.0901 mL

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

In Vivo

1. Add each solvent one by one: 15% Cremophor EL >> 85% Saline
   Solubility: 50 mg/mL (304.51 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 20% HP-β-CD in saline
   Solubility: 33.33 mg/mL (202.98 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (15.23 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (15.23 mM); Clear solution
5. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (15.23 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

4-Phenylbutyric acid (4-PBA) is an inhibitor of HDAC and endoplasmic reticulum (ER) stress, used in cancer and infection research.

IC_{50} & Target	HDAC
In Vitro	4-Phenylbutyric acid (4-PBA) is an inhibitor of HDAC, inhibits the growth of NSCLC Cell Lines at 2 mM. Benzenebutyric acid in combination with ciglitizone results in enhanced growth arrest of cancer cells[1]. 4-Phenylbutyric acid (0-5 mM) inhibits ASFV infection in a dose-dependent manner. Benzenebutyric acid also inhibits the ASFV late protein synthesis and disrupts the virus-induced H3K9/K14 hypoacetylation status. Benzenebutyric acid and enrofloxacin act synergistically to abolish ASFV replication[2]. Addition of bafilomycin A1 results in accumulation of LC3II, whereas 4-Phenylbutyric acid substantially reduces this accumulation. LPS decreases the level of p62, whereas Benzenebutyric acid reverses this decrease upon LPS stimulation for 48 h. The percentage of cells with LPS-induced AVOs is increased at 48 h, whereas 4-Phenylbutyric acid significantly reduces this percentage. Specifically, the percentage of cells with AVOs decreases from 61.6% to 53.1% upon Benzenebutyric acid treatment, supporting that 4-Phenylbutyric acid inhibits LPS-induced autophagy. As a positive control for autophagy inhibition, bafilomycin A1 is used. The percentage of cells with LPS-induced AVOs is reduced by bafilomycin A1 treatment. The decreased OC area and fusion index observed after Benzenebutyric acid treatment are not observed with knockdown of ATG7. Inhibition of NF-κB using BAY 11-7082 and JSH23 reduce the LC3 II level upon LPS stimulation and completely abolish the inhibitory effect of Benzenebutyric acid on LPS-induced effects[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	LPS induces significant bone loss and decreases bone mineral density (BMD), bone volume (BV/TV), and trabecular thickness (Tb. Th) compared with PBS alone, whereas trabecular space (Tb. Sp.) is increased. 4-Phenylbutyric acid (4-PBA) attenuates LPS-induced bone loss. Treatment with 4-Phenylbutyric acid increases BMD, BV/TV, and Tb. Th. compared with LPS alone, in addition to decreasing the enlargement of Tb. Sp., but no change is observed when mice are treated with Benzenebutyric acid alone. OC.S/BS as assessed by TRAP staining is also significantly reduced when Benzenebutyric acid is administered to LPS-treated mice. However, OC.N/BS tends to decrease, although not with statistical significance, when mice are treated with Benzenebutyric acid and LPS. These results indicate that the effect of Benzenebutyric acid on OC from LPS-treated mice is to reduce its size rather than number. Consistent with these findings, a marker of bone resorption in vivo, serum CTX-1 which is elevated by LPS treatment is decreased when Benzenebutyric acid administered to LPS-injected mice. However, co-treatment with Benzenebutyric acid do not significantly affect the levels of serum ALP and osteocalcin, 2 markers of bone formation in vivo, compared with LPS alone. Benzenebutyric acid also reduces the LPS-induced rise in serum MCP-1, indicating that Benzenebutyric acid decreases systemic inflammation induced by LPS[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Briefly, viable cells, as judged by trypan blue dye exclusion, are seeded at a density of 4×10^4 cells/mL in 60-mm dishes in RPMI 1640 with 10% fetal bovine serum and 0.35% agarose on a base layer of 0.7% agarose. DMSO, TSA, or PB is added to both bottom and top agarose layers. Assays are performed in triplicate on at least three separate occasions, and colonies are counted at 10-14 days[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Mice[3] Female 10-week-old C57BL/6J mice are housed in the pathogen-free animal facility of IRC. Animals are randomized into the following 4 groups: vehicle control (n=5), vehicle+Benzenebutyric acid (n=6), LPS (n=6), and LPS+Benzenebutyric acid (n=6). Mice are treated with LPS in 200 μL phosphate-buffered saline (PBS) once a week (5 mg/kg, i.p.) for 3 weeks. Benzenebutyric acid solution is prepared by titrating equimolecular amounts of Benzenebutyric acid and sodium hydroxide to reach pH 7.4; mice are injected daily intraperitoneally in 200 μL PBS (or with PBS as a vehicle) at a dose of 240 mg/kg for 3 weeks. Mice are sacrificed by CO_2 asphyxiation. To determine the bone mineral density (BMD) and microarchitecture of the long bone, the right femur is scanned. Scans are performed with an effective detector pixel size of 6.9 μm and a threshold of 77-255 mg/cc. Trabecular bone is analyzed in a region 1.6 mm in length and located 0.1 mm below the distal femur growth plate[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Sci Immunol. 2023 Sep 29;8(87):eabq2424.
- Bioact Mater. 2023 Sep,257-270.
- Acta Pharm Sin B. 2024 Apr 23.
- Acta Pharm Sin B. 5 January 2022.
- J Hazard Mater. 2023 Jan 12.

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REFERENCES

[1]. Chang TH, et al. Enhanced growth inhibition by combination differentiation therapy with ligands of peroxisome proliferator-activated receptor-gamma and inhibitors of histone deacetylase in adenocarcinoma of the lung. Clin Cancer Res. 2002 Apr;8(4):1206-12

[2]. Frouco G, et, al. Sodium phenylbutyrate abrogates African swine fever virus replication by disrupting the virus-induced hypoacetylation status of histone H3K9/K14. Virus Res. 2017 Oct 15;242:24-29.

[3]. Park HJ, et al. 4-Phenylbutyric acid protects against lipopolysaccharide-induced bone loss by modulating autophagy in osteoclasts. Biochem Pharmacol. 2018 May;151:9-17.

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