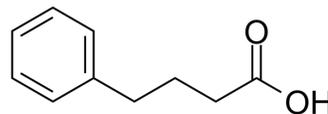


4-Phenylbutyric acid

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)
 H₂O : 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

- Add each solvent one by one: 15% Cremophor EL >> 85% Saline
Solubility: 50 mg/mL (304.51 mM); Suspended solution; Need ultrasonic
- Add each solvent one by one: 20% HP-β-CD in saline
Solubility: 33.33 mg/mL (202.98 mM); Suspended solution; Need ultrasonic
- 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
- 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
- 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.

IC₅₀ & Target	HDAC
In Vitro	<p>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].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
In Vivo	<p>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].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

PROTOCOL

Cell Assay ^[1]	<p>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].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
Animal Administration ^[3]	<p>Mice^[3]</p> <p>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 CO₂ 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].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

CUSTOMER VALIDATION

- Sci Immunol. 2023 Sep 29;8(87):eabq2424.
- Bioact Mater. 2023 Sep;257-270.
- Acta Pharm Sin B. 5 January 2022.
- J Hazard Mater. 2023 Jan 12.
- Small. 2022 Dec 4;e2204310.

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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.

Caution: Product has not been fully validated for medical applications. For research use only.

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