Sodium phenylbutyrate

Cat. No.: HY-15654
CAS No.: 1716-12-7
Molecular Formula: C₁₀H₁₁NaO₂
Molecular Weight: 186.18
Target: HDAC; Autophagy
Pathway: Cell Cycle/DNA Damage; Epigenetics; Autophagy
Storage: Powder -20°C 3 years
        4°C   2 years
        In solvent -80°C 6 months
        -20°C 1 month

**SOLVENT & SOLUBILITY**

<table>
<thead>
<tr>
<th>In Vitro</th>
<th>H₂O : 23.5 mg/mL (126.22 mM; Need ultrasonic and warming)</th>
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<tbody>
<tr>
<td>Preparing Stock Solutions</td>
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<tr>
<td>1 mM</td>
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<td>5 mM</td>
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<td>10 mM</td>
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Please refer to the solubility information to select the appropriate solvent.

**BIOLOGICAL ACTIVITY**

| Description | Sodium phenylbutyrate is an inhibitor of HDAC and endoplasmic reticulum (ER) stress, used in cancer and infection research. |
| IC₅₀ & Target | HDAC |
| In Vitro | Sodium phenylbutyrate is an inhibitor of HDAC, inhibits the growth of NSCLC Cell Lines at 2 mM. Sodium phenylbutyrate in combination with ciglitizone results in enhanced growth arrest of cancer cells[1]. Sodium phenylbutyrate (0-5 mM) inhibits ASFV infection in a dose-dependent manner. Sodium phenylbutyrate also inhibits the ASFV late protein synthesis and disrupts the virus-induced H3K9/K14 hypoacetylation status. Sodium phenylbutyrate and enrofloxacin act synergistically to abolish ASFV replication[2]. Addition of bafilomycin A1 results in accumulation of LC3II, whereas Benzenebutyric acid (4-PBA) 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 Benzenebutyric 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 Benzenebutyric 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].

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. Sodium phenylbutyrate attenuates LPS-induced bone loss. Treatment with Sodium phenylbutyrate increases BMD, BV/TV, and Tb. Th. compared with PBS alone, in addition to decreasing the enlargement of Tb. Sp., but no change is observed when mice are treated with Sodium phenylbutyrate alone. OCS/BS as assessed by TRAP staining is also significantly reduced when Sodium phenylbutyrate is administered to LPS-treated mice. However, OC.N/BS tends to decrease, although not with statistical significance, when mice are treated with Sodium phenylbutyrate and LPS. These results indicate that the effect of Sodium phenylbutyrate 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 Sodium phenylbutyrate administered to LPS-injected mice. However, co-treatment with Sodium phenylbutyrate do not significantly affect the levels of serum ALP and osteocalcin, 2 markers of bone formation in vivo, compared with LPS alone. Sodium phenylbutyrate also reduces the LPS-induced rise in serum MCP-1, indicating that Sodium phenylbutyrate decreases systemic inflammation induced by LPS[3].

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 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].

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