Flufenamic acid

Cat. No.: HY-B1221
CAS No.: 530-78-9
Molecular Formula: C₁₄H₁₀F₃NO₂
Molecular Weight: 281.23
Target: COX; AMPK; Potassium Channel; Chloride Channel; Calcium Channel; Parasite
Pathway: Immunology/Inflammation; Epigenetics; PI3K/Akt/mTOR; Membrane Transporter/Ion Channel; Neuronal Signaling; Anti-infection
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>DMSO : 300 mg/mL (1066.74 mM; Need ultrasonic)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preparing Stock Solutions</td>
<td>Solvent Concentration</td>
</tr>
<tr>
<td></td>
<td>1 mg</td>
</tr>
<tr>
<td>1 mM</td>
<td>3.5558 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>0.7112 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>0.3556 mL</td>
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</tbody>
</table>

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

BIOLOGICAL ACTIVITY

Description
Flufenamic acid is a non-steroidal anti-inflammatory agent, inhibits cyclooxygenase (COX), activates AMPK, and also modulates ion channels, blocking chloride channels and L-type Ca²⁺ channels, modulating non-selective cation channels (NSC), activating K⁺ channels.

IC₅₀ & Target
COX, Chloride Channel, Calcium Channel, Potassium Channel[^1^], AMPK[^2^]

In Vitro
Flufenamic acid is a non-steroidal anti-inflammatory agent, inhibits cyclooxygenase (COX), and also modulates ion channels, blocking chloride channels and L-type Ca²⁺ channels, modulating non-selective cation channels (NSC), activating K⁺ channels. Flufenamic acid inhibits a wide spectrum of TRP channels, including: C3, C7, M2, M3, M4, M5, M7, M8, V1, V3, and V4 but activates at least two TRP channels (C6 and A1)[^1^]. Flufenamic acid induces AMPK activation in T84 cells, and such an effect is via a direct stimulation of calcium/calmodulin-dependent protein kinase kinase beta (CaMKKB) activity[^2^]. Moreover, Flufenamic acid (FFA; 5-50 μM) dose-dependently inhibits cAMP-dependent Cl⁻ secretion in intact T84 cells, suppresses CFTR-mediated apical I_Cl⁻, and blocks the Ca²⁺-dependent Cl⁻
secretion in a dose-dependent manner with IC$_{50}$ of appr 10 μM and near complete inhibition at 100 μM in T84 cell monolayers, but shows no effect on Na$^+$-K$^+$ ATPase or NKCC in T84 cells$^3$.

**In Vivo**
Flufenamic acid (50 mg/kg, i.p.) has anti-inflammatory effect in a mouse model of Vibrio cholerae El Tor variant (EL)-induced diarrhea and significantly abrogates EL-induced intestinal fluid secretion and barrier disruption at 20 mg/kg. Furthermore, Flufenamic acid suppresses NF-κB nuclear translocation and expression of proinflammatory mediators and promotes AMPK phosphorylation in the EL-infected mouse intestine$^2$.

**PROTOCOL**

**Cell Assay**$^3$

In brief, apical and basolateral chambers are filled symmetrically with Kreb's solutions. Thereafter, DMSO or Flufenamic acid is added into the basolateral chamber followed by apical membrane permealization by amphotericin B. After the amphotericin B-elicited $I_{SC}$ is stabilized, ouabain is added into the basolateral chamber. The ouabain sensitive $I_{SC}$ is used as an indicator of Na$^+$-K$^+$ ATPase activity$^3$.

**Animal Administration**$^2$
Rats$^2$

*Six-week-old male ICR outbred mice* (weight 30-35 g) are fasted for 24 h before anesthesia using an intraperitoneal injection of nembutal (60 mg/kg). Following abdominal incision, the ileum is ligated (appr 3-4 cm long) and inoculated with 100 μL of PBS or PBS containing V. cholerae ($10^5$ CFU/loop) with or without a concomitant intraperitoneal injection of Flufenamic acid or metformin. Twelve hours post-inoculation, ileal loops are removed for weight/length ratio measurement, biochemical analysis and ultrastructural evaluation$^2$.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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**REFERENCES**


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

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