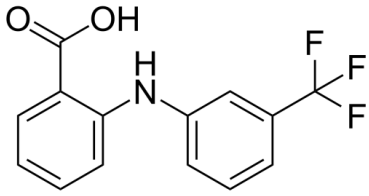
Data Sheet

Product Name:	Flufenamic acid
Cat. No.:	CS-4811
CAS No.:	530-78-9
Molecular Formula:	$C_{14}H_{10}F_3NO_2$
Molecular Weight:	281.23
Target:	AMPK; Calcium Channel; Chloride Channel; COX; Parasite; Potassium Channel
Pathway:	Anti-infection; Epigenetics; Immunology/Inflammation; Membrane Transporter/Ion Channel; Neuronal Signaling; PI3K/Akt/mTOR
Solubility:	DMSO : 100 mg/mL (355.58 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

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 Ca^{2+} channels**, modulating non-selective cation channels (**NSC**), activating **K^+ channels**. Flufenamic acid binds to the central pocket of TEAD2 YBD and inhibits both TEAD function and TEAD-YAP-dependent processes, such as cell migration and proliferation. 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 Ca^{2+} 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 (CaMKKβ) 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 Ca^{2+}-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 (Extracted from published papers and Only for reference)

Cell Assay: Flufenamic acid is dissolved in DMSO[3].[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].

References:

[1]. Guinamard R, et al. Flufenamic acid as an ion channel modulator. Pharmacol Ther. 2013 May;138(2):272-84.

[2]. Pongkorpsakol P, et al. Flufenamic acid protects against intestinal fluid secretion and barrier leakage in a mouse model of Vibrio cholerae infection through NF-κB inhibition and AMPK activation. Eur J Pharmacol. 2017 Mar 5;798:94-104.

[3]. Pongkorpsakol P, et al. Cellular mechanisms underlying the inhibitory effect of flufenamic acid on chloride secretion in human intestinal epithelial cells. J Pharmacol Sci. 2017 Jun;134(2):93-100.

[4]. Pobbati AV, et al. Targeting the Central Pocket in Human Transcription Factor TEAD as a Potential Cancer Therapeutic Strategy. Structure. 2015;23(11):2076-2086.

CAIndexNames:

Benzoic acid, 2-[[3-(trifluoromethyl)phenyl]amino]-

SMILES:

O=C(O)C1=CC=CC=C1NC2=CC=CC(C(F)(F)F)=C2

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