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Flufenamic acid

Catalog No: tcsc4811

Available Sizes

Size: 100mg

Specifications

CAS No:
530-78-9

Formula:
$C_{14}H_{10}F_3NO_2$

Pathway:
Membrane Transporter/Ion Channel;Membrane Transporter/Ion Channel;Immunology/Inflammation;Epigenetics;PI3K/Akt/mTOR;Membrane Transporter/Ion Channel

Target:
Chloride Channel;Calcium Channel;COX;AMPK;AMPK;Potassium Channel

Purity / Grade:
>98%

Solubility:
DMSO : 300 mg/mL (1066.74 mM; Need ultrasonic)

Observed Molecular Weight:
281.23

Product 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 Ca^{2+} 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 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].