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5-Aminosalicylic acid-d_3 disodium

Cat. No.: HY-15027S3

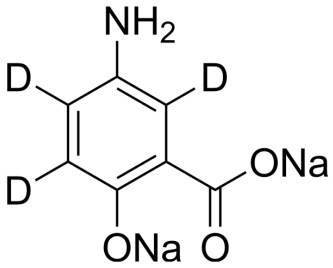
Molecular Formula: $C_7H_2D_3NNa_2O_3$

Molecular Weight: 200.12

Target: PPAR; NF-κB; Endogenous Metabolite; PAK; Isotope-Labeled Compounds

Pathway: Cell Cycle/DNA Damage; Metabolic Enzyme/Protease; Vitamin D Related/Nuclear Receptor; NF-κB; Cytoskeleton; Others

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

5-Aminosalicylic acid-d_3 disodium is deuterated labeled 5-Aminosalicylic Acid (HY-15027). 5-Aminosalicylic acid (Mesalamine) acts as a specific PPARγ agonist and also inhibits p21-activated kinase 1 (PAK1) and NF-κB.5-Aminosalicylic acid can inhibit the activity of osteopontin (OPN).

In Vitro

Stable heavy isotopes of hydrogen, carbon, and other elements have been incorporated into drug molecules, largely as tracers for quantitation during the drug development process. Deuteration has gained attention because of its potential to affect the pharmacokinetic and metabolic profiles of drugs[1].

5-Aminosalicylic acid (5-ASA) is a specific agonist for PPARγ, and only PPARγ but not PPARα or PPARδ induces p65 degradation. 5-Aminosalicylic acid induces degradation of p65 protein indicative of PPARγ's E3 ubiquitin ligase activity. 5-Aminosalicylic acid also inhibits PAK1 at the mRNA level which is suggestive of an additional mechanism independent of PPARγ ligand activation. 5-Aminosalicylic acid blocks NF-κB in intestinal epithelial cells (IECs) through inhibition of PAK1[2].

Pretreatment with 5-Aminosalicylic acid (5-ASA) or Nimesulide at different concentration (10-1000 μmol/L) for 12-96 h, inhibits the growth of HT-29 colon carcinoma cells in a dose and time-dependent manner. However, the suppression of 5-Aminosalicylic acid or Nimesulide has no statistical significance. The growth of HT-29 colon carcinoma cells is inhibited dose-dependently when pretreated with different doses of combined 5-Aminosalicylic acid and Nimesulide. Combined 5-Aminosalicylic acid (final concentration 100 μM) and Nimesulide (final concentration 10-1000 μM) inhibits the proliferation of HT-29 colon carcinoma cells in a dose-dependent manner, being more potent than corresponding dose of Nimesulide. Similarly, combined Nimesulide (final concentration 100 μM) and 5-Aminosalicylic acid (final concentration 10-1000 μM) also inhibits the proliferation of these cells dose-dependently, being more potent than corresponding dose of 5-Aminosalicylic acid[3].

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

In Vivo

5-Aminosalicylic acid (5-ASA) has an antineoplastic effect in a xenograft tumor model. To evaluate the in vivo antineoplasic effect of 5-Aminosalicylic acid, SCID mice engrafted with HT-29 colon cancer cells are treated daily for 21 consecutive days with 5-Aminosalicylic acid at 50 mM. At the end of the treatment, a reduction of 80-86% of tumor weight and volume is observed in SCID mice receiving 5-Aminosalicylic acid compared with control mice or mice treated with GW9662 alone. The antineoplastic effect of 5-Aminosalicylic acid is already detectable after 10 days of 5-Aminosalicylic acid treatment. Similar results are obtained with mice treated with 5-Aminosalicylic acid at 5 mM. Antitumorigenic effect of 5-Aminosalicylic acid is completely abolished at 21 days by simultaneous intraperitoneal administration of GW9662. Thus, the observed antineoplastic effect of 5-Aminosalicylic acid is at least partially dependent on PPARγ[4].

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

REFERENCES

[1]. Dammann K, et al. PAK1 modulates a PPARγ/NF-κB cascade in intestinal inflammation. Biochim Biophys Acta. 2015 Oct;1853(10 Pt A):2349-60.

[2]. Ramadan A, et al. Mesalazine, an osteopontin inhibitor: The potential prophylactic and remedial roles in induced liver fibrosis in rats. Chem Biol Interact. 2018 Jun 1;289:109-118.

[3]. Fang HM, et al. 5-aminosalicylic acid in combination with Nimesulide inhibits proliferation of colon carcinoma cells in vitro. World J Gastroenterol. 2007 May 28;13(20):2872-7.

[4]. Rousseaux C, et al. The 5-aminosalicylic acid antineoplastic effect in the intestine is mediated by PPARγ. Carcinogenesis. 2013 Nov;34(11):2580-6.

[5]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. Ann Pharmacother. 2019 Feb;53(2):211-216.

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