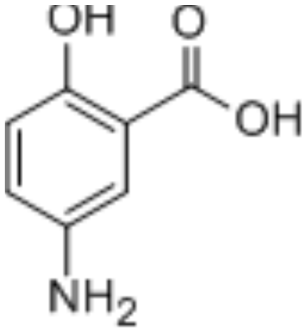
5-Aminosalicylic Acid



Cat. No.: HY-15027

CAS No.: 89-57-6

Molecular Formula: $C_7H_7NO_3$

Molecular Weight: 153.14

Target: PPAR; PAK; NF-κB; Endogenous Metabolite

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

Storage: 4°C, protect from light, stored under nitrogen
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light, stored under nitrogen)

SOLVENT & SOLUBILITY

In Vitro

DMSO : 66.67 mg/mL (435.35 mM; ultrasonic and warming and heat to 60°C)
H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	6.5300 mL	32.6499 mL	65.2997 mL
	5 mM	1.3060 mL	6.5300 mL	13.0599 mL
	10 mM	0.6530 mL	3.2650 mL	6.5300 mL

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

In Vivo

1. Add each solvent one by one: 0.5% CMC-Na/saline water
   Solubility: 16.67 mg/mL (108.85 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 7.14 mg/mL (46.62 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (16.32 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (16.32 mM); Clear solution

BIOLOGICAL ACTIVITY

Description 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).

IC_{50} & Target			
	PPARγ	PAK1	p65

In Vitro	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[1]. 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[2]. 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[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	Cytostatic effects are measured by MTT assay. HT-29 colon carcinoma cells are detached with a 0.25% trypsin solution for 5 min. Subsequently, the cells are seeded onto 96-well plates (1×10^6 cells/well), supplemented with 10% FCS and allowed to attach for 24 h before the addition of test compounds (5-Aminosalicylic acid 10, 50, 100, 500, and 1000 μM; Nimesulide; and their combination). Test compounds are diluted in serum-free culture medium. Then the cells are incubated in a medium or at different concentrations of drugs for 48 h, 20 μL of MTT solution (5 g/L) in PBS is added. Four hours later, the medium in each well is removed, and 120 μL of 0.04 mM muriatic isopropanol is added, slightly concussed for 10 min. Dye uptake is measured at 490 nm with an ELISA reader. Five wells are used for each concentration or as a control group. On the other hand, the cells are seeded onto 96-well plates (1×10^6 cells/well) and allowed to attach for 24 h, then treated with test compounds (5-Aminosalicylic acid, Nimesulide, and their combination). The final concentration is 100 μM. The same medium is added into the control group and dye uptake is then measured. Five wells are used for each test compound or control group[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Mice[3] Six to seven weeks old pathogen-free BALB/c ***SCID*** mice are used. Human colon cancer cells (10^7 HT-29 cells) pretreated or not with GW9662 for 24 h are implanted subcutaneously in the flank of animals. Two days after cell inoculation, mice are treated with 5-Aminosalicylic acid (5 or 50 mM) administered daily by peritumoral injection for 10 or 21 days. The effect of PPARγ during 5-Aminosalicylic acid treatment is evaluated by daily intraperitoneal injection of GW9662 (1 mg/kg/day). The control group receives saline instead of 5-Aminosalicylic acid. Mice are checked three times a week for tumor development. After killing at 10 or 21 days, tumor size and volume are calculated. Tumors are weighted before paraffin embedding for histological examination. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Brain Behav Immun. 2020 Nov;90:108-137.
- Carbohydr Polym. 2022: 120329.
- Phytomedicine. 2023 Nov 26, 155223.
- Phytomedicine. 2023 Feb 10.
- Phytomedicine. November 2022, 154438.

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REFERENCES

[1]. 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.

[2]. 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.

[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.

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