Sulindac

Cat. No.: HY-B0008

CAS No.: 38194-50-2

Molecular Formula: $C_{20}H_{17}FO_3S$

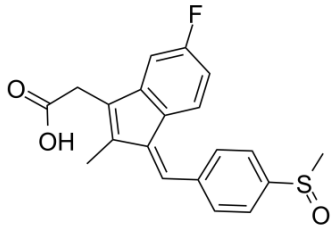
Molecular Weight: 356.41

Target: COX; Autophagy

Pathway: Immunology/Inflammation; Autophagy

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (280.58 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.8058 mL	14.0288 mL	28.0576 mL
5 mM	0.5612 mL	2.8058 mL	5.6115 mL
10 mM	0.2806 mL	1.4029 mL	2.8058 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.01 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.01 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Sulindac (MK-231) is a non-steroidal antiinflammatory agent, acts as a COX-2 inhibitor, and inhibits overexpression of COX-2.

IC_{50} & Target

COX-2 | Autophagy

In Vitro

Sulindac (MK-231) is a non-steroidal antiinflammatory agent, acts as a COX-2 inhibitor, and inhibits overexpression of COX-2 [1]. Sulindac (MK-231) (0.1 mM to 0.5 mM) causes limited death in both p53 wt and p53 null HCT116 cells, but in combination with vitamin C, it dramatically increases almost 5-fold in cell death in p53 wt HCT116 cells relative to the vitamin C alone, and such an effect is involving caspase activation and p53 function in these cells, and via ROS-mediated pathway. Sulindac combined with vitamin C significantly increases PUMA levels, but shows no effect on Bim, Bcl-2 and Mcl-1 levels[2]. Sulindac (MK-231) (500 μM) in combination with celecoxib blocks transforming growth factor (TGF)-β1-induced epithelial-

	mesenchymal transition, migration and invasion in A549 cells. The combination also suppresses involvement of sirtuin 1 (SIRT1) in transforming growth factor (TGF)-β1-induced epithelial-mesenchymal transition (EMT)[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Sulindac (MK-231) (0.5 ± 0.1 mg/day) decreases COX, modolates PGE2 levels and prevents tumor formation in the Min mice[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	Cells are treated with Sulindac (MK-231) and/or vitamin C at the indicated doses for 48 h, and cell viability is analyzed using a trypan blue exclusion assay. For the annexin V staining assay, cells are treated with 0.5 mM Sulindac (MK-231) and/or 0.5 mM vitamin C for 48 h. The cells are then trypsinized, washed with PBS, stained with propidium iodide (PI) and FITC-labeled annexin V for 30 min, and analyzed by flow cytometry using a fluorescence-activated cell sorter[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Mice[1] Female C57BL16J-Min/+ (Min) mice at 5 weeks of age are used in the assay. Beginning at 5-6 weeks of age, 10 Min mice are fed a low-fat AIN-76A chow diet modified with 0.001% ethoxyquin and Sulindac (MK-231), 0.5 ± 0.1 mg/day (0.05 mg/kcal/day or approximately 160 ppm) in drinking water. As controls, 9 Min mice and 5 C57BL/6J-+/+ non-affected littermates (+/+) are fed AIN-76A diet without Sulindac. Animals are checked daily for signs of distress or anemia. Animals and their food are weighed twice weekly. During the course of the experiment, there is no difference in body weight or food consumption among the various study groups. No toxicity is observed in the Min/Sulindac group. At 110 days of age, all mice are euthanized by CO_2 inhalation, and their intestinal tracts are removed from esophagus to distal rectum, opened, flushed with saline, and examined under ×3 magnification to obtain tumor counts. Tumors are counted by an individual blinded to the animal's genetic status and treatment. Multiple samples of grossly normal, full-thickness bowel are harvested from the mid small intestine and either frozen in liquid nitrogen or fixed in 10% formalin for histological examination. All samples used for the analyses in this study are taken from mid small intestine[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Boolbol SK, et al. Cyclooxygenase-2 overexpression and tumor formation are blocked by sulindac in a murine model of familial adenomatous polyposis. Cancer Res. 1996 Jun 1;56(11):2556-60.

[2]. Gong EY, et al. Combined treatment with vitamin C and sulindac synergistically induces p53- and ROS-dependent apoptosis in human colon cancer cells. Toxicol Lett. 2016 Sep 6;258:126-133.

[3]. Cha BK, et al. Celecoxib and sulindac inhibit TGF-β1-induced epithelial-mesenchymal transition and suppress lung cancer migration and invasion via downregulation of sirtuin 1. Oncotarget. 2016 Aug 30;7(35):57213-57227.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA