Sulindac sodium

Cat. No.: HY-B0008A

CAS No.: 63804-15-9

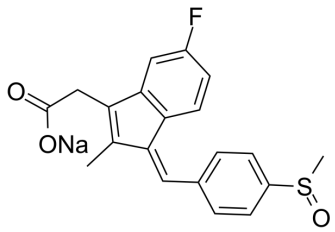
Molecular Formula: $C_{20}H_{16}FNaO_3S$

Molecular Weight: 378.39

Target: NF-κB; PD-1/PD-L1

Pathway: NF-κB; Immunology/Inflammation

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



BIOLOGICAL ACTIVITY

Description

Sulindac (MK-231) is an orally active nonsteroidal anti-inflammatory agent. Sulindac also is an immunomodulatory agent. Sulindac can be used for the research of arthritis of the spine, gouty arthritis and kinds of cancer including colorectal cancer (CRC) and lung cancer[1][2].

In Vitro

Sulindac (MK-231) (500 μM, 48 h) sodium is effective in preventing TGF-β1-induced EMT, as indicated by upregulation of the epithelial marker, E-cadherin, and downregulation of mesenchymal markers and transcription factors[1].
Sulindac sodium (500 μM, 48 h) can inhibit TGF-β1-enhanced migration and invasion of A549 cells[1].
Sulindac sodium (500 μM, 48 h) enhances the reversal of TGF-β1-induced EMT by sulindac (sodium) and SIRT1 upregulation promoted TGF-β1-induced EMT[1].

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

Western Blot Analysis[1]

Cell Line:	A549 cells
Concentration:	500 μM
Incubation Time:	48 h
Result:	Inhibit transforming growth factor (TGF)-β1-induced epithelial-mesenchymal transition in A549 cells.

Immunofluorescence[1]

Cell Line:	A549 cells
Concentration:	500 μM
Incubation Time:	48 h
Result:	Reversed SIRT-1 expression by TGF-β1 and inhibited the TGF-β1-induced cadherin switch.

Cell Migration Assay [1]

Cell Line:	A549 cells

Concentration:	500 μM
Incubation Time:	48 h
Result:	Inhibited migration, decreased resistance co-treatment with TGF-β1.

Cell Invasion Assay[1]

Cell Line:	A549 cells
Concentration:	500 μM
Incubation Time:	40 h; 48 h
Result:	Could effectively inhibit the TGF- β1-induced increase in invasion by lung cancer cells.

In Vivo

Sulindac (MK-231) sodium (15 mg/kg, p.o., bid (sulindac alone); 7.5 mg/kg p.o., bid (sulindac combination with PD-L1)) shows a significant reduction in tumor volume and increases infiltration of CD8+ T lymphocytes in the tumor tissues when treated with combination therapy[2].

Sulindac sodium (15 mg/kg, p.o., bid (sulindac alone); 7.5 mg/kg p.o., bid (sulindac combination with PD-L1)) can downregulate PD-L1 by blocking NF-κB signaling, which in turn led to a decrease in exosomal P[2].

Sulindac sodium (15 mg/kg, p.o., bid (sulindac alone); 7.5 mg/kg p.o., bid (sulindac combination with PD-L1)) leads to increased availability of PD-L1 Ab by downregulating PD-L1 in combination therapy[2].

Sulindac sodium has not a systemic inhibitory effect on prostaglandin E2 (PGE2) in low-dose does[2].

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

Animal Model:	CT26 syngeneic mouse tumor model[2]
Dosage:	15 mg/kg; 7.5 mg/kg
Administration:	15 mg/kg, p.o., bid (sulindac alone); 7.5 mg/kg p.o., bid (sulindac combination with PD-L1)
Result:	Downregulated PD-L1 through the blockade of NF-κB signaling and modulate the response of pMMR CRC to anti-PD-L1 immunotherapy.

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Result:	Downregulated PD-L1 through the blockade of NF-κB signaling and modulate the response of pMMR CRC to anti-PD-L1 immunotherapy. Cound effectively inhibit PD-L1 with no significant systematic toxicity.

REFERENCES

[1]. Byong-Ki Cha, 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.

[2]. Bin Yi, et al. Sulindac Modulates the Response of Proficient MMR Colorectal Cancer to Anti-PD-L1 Immunotherapy. Mol Cancer Ther. 2021 Jul;20(7):1295-1304.

Caution: Product has not been fully validated for medical applications. For research use only.

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