Product Data Sheet

Product Name: Misoprostol
Cat. No.: GC16074

Chemical Properties

Cas. No.	59122-46-2		
Chemical Name	methyl 7-((1R,2R,3R)-3-hydroxy-2-((E)-4-hydroxy-4-methyloct-1-en-1-yl)-5-oxocyclopentyl)heptanoate		
SMILES	CCCCC(C/C([H])=C([H])/[C@]1([H])[C@@](C(C[C@@]1([H])O)=O)([H])CCCCCCC(OC)=O)(O)C		
Formula	$C_{22}H_{38}O_5$	M.Wt	382.53
Solubility	≥ 11.05mg/mL in DMSO	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure	Misoprostol		

Protocol

Cell experiment [1]:

Cell lines	Human peripheral blood mononuclear cells (PBMCs)，RAW264.7 cells
Preparation Method	Cells were plated at 0.5 million/mL density, treated with 10μM Misoprostol for 90min and further stimulated with LPS, 100ng/mL. PKA inhibitor, H89 was used at 10μM concentration 30min before Misoprostol treatment. Nuclear extracts were subjected to Western blot analysis.

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Reaction Conditions	10μM; 90min
Applications	Misoprostol exposure resulted in increased phosphorylation of cAMP response element-binding protein (CREB) in both PBMCs and RAW cells; pretreatment with PKA inhibitor, H89 prevented Misoprostol effect on CREB phosphorylation.

Animal experiment [2]:

Animal models	Sprague-Dawley rats
Preparation Method	Divided the rats randomly into three groups of seven: group 1, control; group 2, Doxorubicin; group 3, Doxorubicin+ MP. The control group was injected intraperitoneally (i.p.) with 0.5mL 0.9% NaCl and given 1mL 0.9% NaCl orally for 6 days. The Doxorubicin group was injected i.p. with a single dose of 20mg/kg Doxorubicin on the third day of the study. The Doxorubicin+Misoprostol group was injected i.p. with a single 20mg/kg dose of Doxorubicin on day 3 of the study and given 0.2mg/kg/day Misoprostol orally for 6 days. The treatment course lasted 6 days for all groups.
Dosage form	0.2mg/kg/day for 6 days; p.o.

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Applications	Cardiac damage caused by Doxorubicin was attenuated by Misoprostol treatment. Treatment with Misoprostol decreased significantly serum cardiac troponin-I, brain natriuretic peptide levels, and lactate dehydrogenase, aspartate aminotransferase, alanine transaminase and creatine kinase isoenzyme-MB activities. Misoprostol also decreased NADPH oxidase-4 and caspase-3 levels.

References:

[1]Gobejishvili L, Ghare S, Khan R, et al. Misoprostol modulates cytokine expression through a cAMP pathway: Potential therapeutic implication for liver disease[J]. Clinical Immunology, 2015, 161(2): 291-299.

[2]Bilgic S, Ozgocmen M, Ozer M K, et al. Misoprostol ameliorates doxorubicin induced cardiac damage by decreasing oxidative stress and apoptosis in rats[J]. Biotechnic & Histochemistry, 2020, 95(7): 514-521.

Background

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Misoprostol is an orally active prostaglandin E1 (PGE1) analog used to prevent gastric ulcers, treat missed abortion, induce labor and induce miscarriage[1, 2]. Misoprostol is used to prevent gastric ulcers induced by nonsteroidal anti-inflammatory drugs. It can bind to prostaglandin receptors and directly act on parietal cells to inhibit gastric acid secretion[3].

In vitro, Misoprostol (10μM) pretreatment of human peripheral blood mononuclear cells (PBMC) and RAW264.7 cells for 90min significantly increased the phosphorylation of cAMP response element binding protein (CREB) in cells[4]. Misoprostol (10μM) treatment of HCT-116 cells for 2h significantly activated intracellular protein kinase A (PKA)[5]. Misoprostol (8ng/mL) treatment of 3D cultured annular cells significantly increased the epidermal growth factor (EGF) level of cells[6].

In vivo, oral administration of Misoprostol (0.2mg/kg/day) to rats with doxorubicin-induced cardiac injury for 6 days alleviated cardiac injury in rats, significantly reduced serum cardiac troponin-I (cTn-I) and brain natriuretic peptide (BNP) levels, and reduced lactate dehydrogenase (LDH), aspartate aminotransferase (AST), alanine aminotransferase (ALT), and creatine kinase isoenzyme-MB (CK-MB) activities[7].

References:

[1] Ahmed T A, Mohammed A H. Brief Overview About Prostaglandins & Misoprostol For Cervical Ripening[J]. Journal of Pharmaceutical Negative Results, 2023, 14(2).
[2] Chong Y S, Su L L, Arulkumaran S. Misoprostol: a quarter century of use, abuse, and creative misuse[J]. Obstetrical & gynecological survey, 2004, 59(2): 128-140.
[3] Sostres C, Lanas A. Prostaglandins and other mucosal protecting agents[J]. Pocket Guide to Gastrointestinal Drugs, 2014: 44-56.
[4] Gobejishvili L, Ghare S, Khan R, et al. Misoprostol modulates cytokine expression through a cAMP pathway: Potential therapeutic implication for liver disease[J]. Clinical Immunology, 2015, 161(2): 291-299.
[5] Field J T, Martens M D, Mughal W, et al. Misoprostol regulates Bnip3 repression and alternative splicing to control cellular calcium homeostasis during hypoxic stress[J]. Cell Death Discovery, 2018, 4(1): 98.
[6] Gruber H E, Hoelscher G, Loeffler B, et al. Prostaglandin E1 and misoprostol increase epidermal growth factor production in 3D-cultured human annulus cells[J]. The Spine Journal, 2009, 9(9): 760-766.

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[7] Bilgic S, Ozgocmen M, Ozer M K, et al. Misoprostol ameliorates doxorubicin induced cardiac damage by decreasing oxidative stress and apoptosis in rats[J]. Biotechnic & Histochemistry, 2020, 95(7): 514-521.

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