

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])CCCCC(OC)=O)(O)C

Formula C₂₂H₃₈O₅

M.Wt 382.53

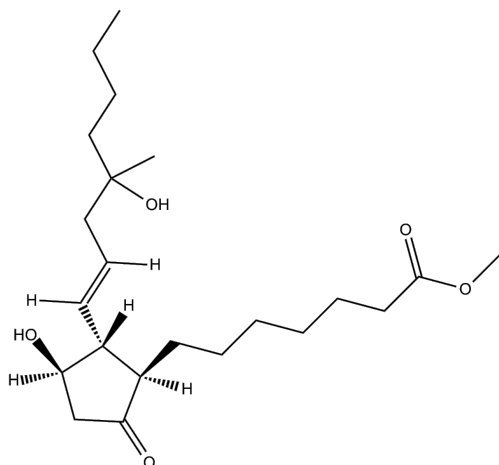
Solubility ≥ 11.05mg/mL in DMSO

Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C 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



Protocol

Cell experiment [1]:

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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.
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.

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Dosage form 0.2mg/kg/day for 6 days; p.o.

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

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)

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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.
- [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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