Celecoxib (GMP)

Cat. No.: HY-14398G

CAS No.: 169590-42-5

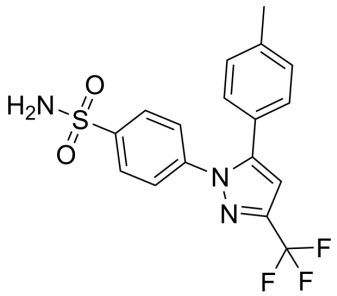
Molecular Formula: $C_{17}H_{14}F_3N_3O_2S$

Molecular Weight: 381.37

Target: COX

Pathway: Immunology/Inflammation

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



BIOLOGICAL ACTIVITY

Description

Celecoxib (GMP) is Celecoxib (HY-14398) produced by using GMP guidelines. GMP small molecules work appropriately as an auxiliary reagent for cell therapy manufacture. Celecoxib,a selective non-steroidal anti-inflammatory drug (NSAID), is a selective COX-2 inhibitor with an IC_{50} of 40 nM.

In Vitro

The selective cyclooxygenase-2 (COX-2) inhibitor Celecoxib (10-75 μM) inhibits the proliferation of the NPC cell lines in a dose-dependent manner. Celecoxib (25 and 50 μM) induces apoptosis and cell-cycle arrest at the G_0/G_1 checkpoint in the NPC cell lines, which is associated with significantly reduced STAT3 phosphorylation. The genes downstream of STAT3 (ie, Survivin, Mcl-1, Bcl-2 and Cyclin D1) are significantly down-regulated after exposure to Celecoxib (25 and 50 μM)[2].
Targeting the YAP/TAZ transcriptional target cyclooxygenase 2 (COX-2) using celecoxib inhibits cell proliferation and tumorigenesis in NF2 mutant cells[6].
Celecoxib (5 μM, 28 d) in combination with TTNPB (HY-15682) (3 μM) converts fibroblasts into articular chondrocytes[7].
Celecoxib (10 μM, 7-14 d) enhances trans-differentiation of Wharton's jelly derived mesenchymal stromal cells (WJ-MSC) into endothelial progenitor cells (EPCs) [8].
Celecoxib (5 μM, 14 d) induces human aortic valve interstitial cells (AVICs) trans-differentiation towards a myofibroblast[9].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Celecoxib demonstrates potent, oral anti-inflammatory activity. Celecoxib reduces acute inflammation in the carrageenan edema assay with an ED_{50} of 7.1 mg/kg and reduces chronic inflammation in the adjuvant arthritis model with an ED_{50} of 0.37 mg/kg/day. In addition, Celecoxib also exhibits analgesic activity in the Hargreaves hyperalgesia model with an ED_{50} of 34.5 mg/kg. Celecoxib has potency equivalent to that of standard nonsteroidal anti-inflammatory drugs (NSAIDs), yet shows no acute GI toxicity in rats at doses up to 200 mg/kg. In addition, it displays no chronic GI toxicity in rats at doses up to 600 mg/kg/day over 10 days[1]. In the KpB mice fed a high fat diet (obese) and treated with Celecoxib, tumor weight decreases by 66% when compare with control animals. Among KpB mice fed a low fat diet (non-obese), tumor weight decreases by 46% after treatment with Celecoxib[3]. Rat models are orally administrated with Celecoxib (20 mg/kg) and/or intramuscularly with Fasudil (10 mg/kg) for 2 weeks. Results demonstrates that the combined use of Celecoxib and Fasudil (HY-10341A) significantly decreases COX-2 and Rho kinase II expression surrounding the lesion site in rats with spinal cord injury, improves the pathomorphology of the injured spinal cord, and promoted the recovery of motor function[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Biomed Eng. 2018 Aug;2(8):578-588.
- Biomaterials. 16 September 2022.
- Hepatology. 2023 Feb 1;77(2):456-465.
- Theranostics. 2023 Feb 21; 13(4): 1381-1400.
- J Exp Clin Cancer Res. 2020 Jun 16;39(1):113.

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[2]. Hou XL, et al. Combination of fasudil and celecoxib promotes the recovery of injured spinal cord in rats better than celecoxib or fasudil alone. Neural Regen Res. 2015 Nov;10(11):1836-40.

[3]. Suri A, et al. The effect of celecoxib on tumor growth in ovarian cancer cells and a genetically engineered mouse model of serous ovarian cancer. Oncotarget. 2016 Apr 8.

[4]. Liu DB, et al. Celecoxib induces apoptosis and cell-cycle arrest in nasopharyngeal carcinoma cell lines via inhibition of STAT3 phosphorylation. Acta Pharmacol Sin. 2012 May;33(5):682-90.

[5]. Penning TD, et al. Synthesis and biological evaluation of the 1,5-diarylpyrazole class of cyclooxygenase-2 inhibitors: identification of 4-[5-(4-methylphenyl)-3-(trifluoromethyl)-1H-pyrazol-1-yl]benze nesulfonamide (SC-58635, celecoxib). J Med Chem. 1997

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[7]. Chen Y, Wu B, Lin J, et al. High-Resolution Dissection of Chemical Reprogramming from Mouse Embryonic Fibroblasts into Fibrocartilaginous Cells. Stem Cell Reports. 2020;14(3):478-492.

[8]. Kaushik K, Das A. Cycloxygenase-2 inhibition potentiates trans-differentiation of Wharton's jelly-mesenchymal stromal cells into endothelial cells: Transplantation enhances neovascularization-mediated wound repair. Cytotherapy. 2019;21(2):260-273.

[9]. Vieceli Dalla Sega F, Fortini F, Cimaglia P, et al. COX-2 Is Downregulated in Human Stenotic Aortic Valves and Its Inhibition Promotes Dystrophic Calcification. Int J Mol Sci. 2020;21(23):8917. Published 2020 Nov 24. doi:10.3390/ijms21238917

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