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SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

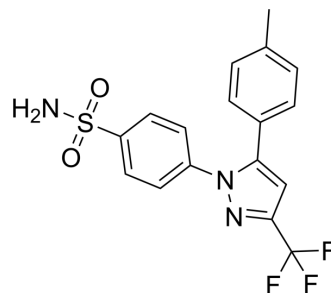
mail@szabo-scandic.com

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Celecoxib (GMP)

Cat. No.:	HY-14398G
CAS No.:	169590-42-5
Molecular Formula:	C ₁₇ H ₁₄ F ₃ N ₃ O ₂ S
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	<p>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 G₀/G₁ 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].</p> <p>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].</p> <p>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.</p>
In Vivo	<p>Celecoxib demonstrates potent, oral anti-inflammatory activity. Celecoxib reduces acute inflammation in the carrageenan edema assay with an ED₅₀ of 7.1 mg/kg and reduces chronic inflammation in the adjuvant arthritis model with an ED₅₀ of 0.37 mg/kg/day. In addition, Celecoxib also exhibits analgesic activity in the Hargreaves hyperalgesia model with an ED₅₀ 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].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

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.
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- [6]. Liu C, et al. Celecoxib alleviates nonalcoholic fatty liver disease by restoring autophagic flux. Sci Rep. 2018 Mar 7;8(1):4108.
- [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. Cyclooxygenase-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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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