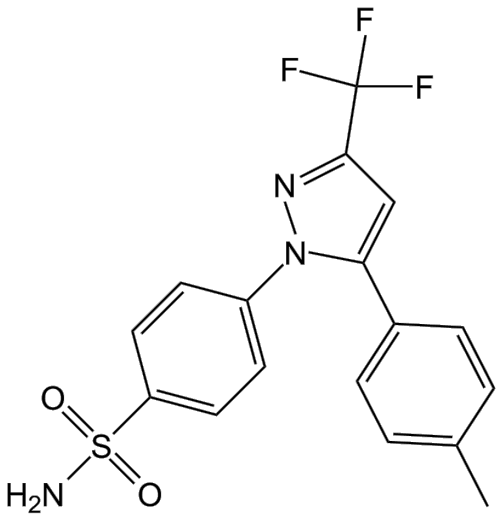
Product Data Sheet

Product Name: Celecoxib
Cat. No.: GC14475

Chemical Properties

Cas. No.	169590-42-5		
Chemical Name	4-[5-(4-methylphenyl)-3-(trifluoromethyl)pyrazol-1-yl]benzenesulfonamide		
SMILES	CC1=CC=C(C=C1)C2=CC(=NN2C3=CC=C(C=C3)S(=O)(=O)N)C(F)(F)F		
Formula	$C_{17}H_{14}F_3N_3O_2S$	M.Wt	381.37
Solubility	≥ 19.1mg/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	Tendon cells
Preparation Method	Cells were first seeded into 24-well flat-bottomed culture plates. Celecoxib was then added to the culture dish at dosages of 0, 0.8, 2.0, 4.0, and 8.0μg/mL. Then 24h after Celecoxib was added to cells, cells were observed under microscope and then MTT assay was performed.
Reaction Conditions	0.8, 2.0, 4.0, 8.0μg/mL; 24h
Applications	Celecoxib dose-dependently inhibited tendon cell viability and migration, but no cell necrosis or changes in cell morphology were observed.

Animal experiment [2]:

Animal models	Female BALB/c nude mice
Preparation Method	Mice were randomly divided into the following five treatment groups: control; mice were injected with PGE2 (1mg/kg); mice were injected with PGE2 (1mg/kg) and Celecoxib (100mg/kg); mice received pneumonectomy; mice received pneumonectomy and Celecoxib (100mg/kg). A549 cells were prepared as a suspension of 10^6 cells in 150μL saline and injected using a 29-gauge needle into the lateral tail vein under sterile conditions. Celecoxib (100mg/kg) was administered orally starting from the day before tumor cell injection until the mice were sacrificed. After the mice were sacrificed, the lungs were flushed with 5% picric acid solution. Lung metastasis was determined by counting the total number of metastatic nodules in each lung.

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Dosage form	100mg/kg; p.o.
Applications	The metastasis of A549 cells in mice receiving unilateral pneumonec tomy was more than that in control mice, and treatment with Celecoxib inhibited this increase in metastases. The mean level of plasma PGE2 in mice receive ing unilateral pneumonectomy increased, and treatment with Celecoxib significantly inhibited the increase in PGE2 plasma level.

References:
[1]Tsai W C, Hsu C C, Chou S W, et al. Effects of celecoxib on migration, proliferation and collagen expression of tendon cells[J]. Connective tissue research, 2007, 48(1): 46-51.
[2]Zhang S, Da L, Yang X, et al. Celecoxib potentially inhibits metastasis of lung cancer promoted by surgery in mice, via suppression of the PGE2-modulated β-catenin pathway[J]. Toxicology letters, 2014, 225(2): 201-207.

Background

Celecoxib is a highly selective cyclooxygenase-2 (COX-2) inhibitor with an IC_{50} value of 40nM[1]. Celecoxib is a selective nonsteroidal anti-inflammatory drug (NSAID) commonly used to treat acute pain, osteoarthritis (OA), rheumatoid arthritis (RA), juvenile rheumatoid arthritis and ankylosing spondylitis (AS)[2]. Celecoxib has strong anti-tumor activity and can prevent and treat a variety of cancers such as pancreatic cancer, breast cancer, ovarian cancer, and non-small cell lung cancer[3]. Celecoxib can prevent the occurrence of colorectal adenomatous polyps[4].

In vitro, Celecoxib (0.8, 2.0, 4.0, 8.0μg/mL) treated tenocytes for 24h dose-dependently inhibited cell viability and migration, but did not change cell morphology or cause cell necrosis[5]. Celecoxib (1-100μM) treated human osteoblasts for 24h, inhibited cell proliferation in a dose-dependent manner and caused an increase in intracellular Ca^{2+} concentration[6].

In vivo, Celecoxib (100mg/kg) was orally administered to mice with lung cancer model and unilateral

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lung resection, inhibiting lung cancer metastasis and the increase in plasma PGE2 levels[7]. Celecoxib (10, 20mg/kg) was orally administered to rats with gastric cancer model, significantly reducing the incidence and growth of gastric cancer in rats[8].

References:

[1] Cao H, Yu R, Choi Y, et al. Discovery of cyclooxygenase inhibitors from medicinal plants used to treat inflammation[J]. Pharmacological research, 2010, 61(6): 519-524.
[2] Frampton J E, Keating G M. Celecoxib: a review of its use in the management of arthritis and acute pain[J]. Drugs, 2007, 67: 2433-2474.
[3] Jendrossek V. Targeting apoptosis pathways by Celecoxib in cancer[J]. Cancer letters, 2013, 332(2): 313-324.
[4] Arber N, Eagle C J, Spicak J, et al. Celecoxib for the prevention of colorectal adenomatous polyps[J]. New England Journal of Medicine, 2006, 355(9): 885-895.
[5] Tsai W C, Hsu C C, Chou S W, et al. Effects of celecoxib on migration, proliferation and collagen expression of tendon cells[J]. Connective tissue research, 2007, 48(1): 46-51.
[6] Wang J L, Lin K L, Chen J S, et al. Effect of celecoxib on Ca2+ movement and cell proliferation in human osteoblasts[J]. Biochemical pharmacology, 2004, 67(6): 1123-1130.
[7] Zhang S, Da L, Yang X, et al. Celecoxib potentially inhibits metastasis of lung cancer promoted by surgery in mice, via suppression of the PGE2-modulated β-catenin pathway[J]. Toxicology letters, 2014, 225(2): 201-207.
[8] Hu P J, Yu J, Zeng Z R, et al. Chemoprevention of gastric cancer by celecoxib in rats[J]. Gut, 2004, 53(2): 195-200.

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