Pirfenidone

Cat. No.: HY-B0673

CAS No.: 53179-13-8

Molecular Formula: $C_{12}H_{11}NO$

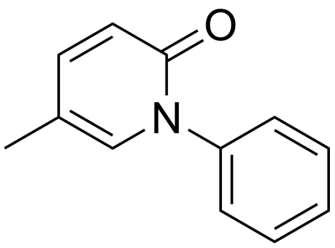
Molecular Weight: 185.22

Target: TGF-beta/Smad; CCR

Pathway: Stem Cell/Wnt; TGF-beta/Smad; GPCR/G Protein; Immunology/Inflammation

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (539.90 mM)

H_2O : 12.5 mg/mL (67.49 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.3990 mL	26.9949 mL	53.9898 mL
5 mM	1.0798 mL	5.3990 mL	10.7980 mL
10 mM	0.5399 mL	2.6995 mL	5.3990 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 0.5% CMC/saline water
   Solubility: 10 mg/mL (53.99 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: PBS
   Solubility: 9.09 mg/mL (49.08 mM); Clear solution; Need ultrasonic and warming and heat to 60°C
3. Add each solvent one by one: Saline
   Solubility: 6.67 mg/mL (36.01 mM); Clear solution; Need ultrasonic
4. Add each solvent one by one: 5% DMSO >> 40% PEG300 >> 5% Tween-80 >> 50% saline
   Solubility: ≥ 2.75 mg/mL (14.85 mM); Clear solution
5. Add each solvent one by one: 5% DMSO >> 95% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.75 mg/mL (14.85 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Pirfenidone (AMR69) is an antifibrotic agent that attenuates CCL2 and CCL12 production in fibrocyte cells. Pirfenidone has

	growth-inhibitory effect and reduces TGF-β2 protein levels in human glioma cell lines. Pirfenidone also has anti-inflammatory activities[1][2][3].
IC_{50} & Target	TGF-β_2[1]
In Vitro	Pirfenidone (PFD) reduces the protein levels of the matrix metalloproteinase (MMP)-11, a TGF-β target gene and furin substrate involved in carcinogenesis. These data define PFD or PFD-related agents as promising agents for human cancers associated with enhanced TGF-β activity[1]. In RAW264.7 cells, a murine macrophage-like cell line, Pirfenidone suppresses the proinflammatory cytokine TNF-α by a translational mechanism, which is independent of activation of the MAPK2, p38 MAPK, and JNK. In the murine endotoxin shock model, Pirfenidone potently inhibits the production of the proinflammatory cytokines, TNF-α, interferon-γ, and interleukin-6, but enhances the production of the anti-inflammatory cytokine, interleukin-10[2]. Pirfenidone (PFD) shows its inhibitory effects on the proliferation of HLECs. Cell proliferation is attenuated in the 0.3 mg/mL group after 24 hours compare with the control group (P=0.044). The effect is more apparent in the 0.5 mg/mL group at 24, 48, and 72 hours (P<0.05). The proliferation is almost completely inhibited with 1 mg/mL PFD at all the time-points (P<0.01)[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Administration of Pirfenidone (300 mg/kg/day) for 4 wk. Pirfenidone significantly attenuates the score when administered in Bleomycin (BLM)-treated mice (P<0.0001). Moreover, collagen content is quantified in the lungs to evaluate the anti-fibrotic effects of Pirfenidone. The collagen content in the lungs of BLM-treated mice is significantly increased compared with that in saline- or Pirfenidone-treated mice, and this increase is significantly attenuated by Pirfenidone administration on day 28 after BLM treatment (P=0.0012)[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [3]	HLECs are seeded in 96-well plates (1×10^4 cells/well) for 24 hours in α-MEM/10% FBS/1%NEAA, and are cultured in stationary tubes in serum-free medium for 24 hours. And then the culture medium is removed and cells are bathed in α-MEM with 10% FBS and 1% NEAA supplemented with 0, 0.01, 0.1, 0.2, 0.3, 0.5, or 1 mg/mL Pirfenidone for 0, 4, 12, 24, 48, or 72 hours. After incubation with 180 μL α-MEM and 20 μL of 5 mg/mL MTT for 4 hours at 37°C, the MTT solution is discarded. The Formosan precipitates are dissolved in 180 μL DMSO by agitating the dishes for 10 minutes at 200 rpm on an orbital shaker. The absorbance at 490 nm in each well is read with a micro plate reader. We further examined the effects of PFD by refining the concentrations at 0.2, 0.25, 0.3, 0.4, 0.5 and 0.6 mg/mL using the MTT assay[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [4]	Mice[4] Nine-week-old female C57BL/6 mice are used. Pirfenidone is administered orally for 14 days after osmotic pump implantation. The volume of administration is determined according to body weight. Animals are allocated into 4 groups (n=6/group): normal control, BLM, Pirfenidone (300 mg/kg/day), and BLM + Pirfenidone. The Pirfenidone dose is selected according to a report published elsewhere. Pirfenidone is also administered in a therapeutic setting beginning at day 10 to assess the effect of the drug on the fibrotic phase of BLM model mice. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Bioact Mater. 2024 Mar, 33, Pages 262-278.
- Sci Adv. 2022 Jun 17;8(24):eabn4564.
- Arthritis Rheumatol. 2022 Sep 3.

- J Exp Clin Cancer Res. 2021 Feb 9;40(1):62.
- Clin Transl Med. 2022 Oct;12(10):e1036.

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REFERENCES

[1]. Burghardt I, et al. Pirfenidone inhibits TGF-beta expression in malignant glioma cells. Biochem Biophys Res Commun. 2007 Mar 9;354(2):542-7.

[2]. Nakazato H, et al. A novel anti-fibrotic agent pirfenidone suppresses tumor necrosis factor-alpha at the translational level. Eur J Pharmacol. 2002 Jun 20;446(1-3):177-85.

[3]. Yang Y, et al. Inhibition of Pirfenidone on TGF-beta2 induced proliferation, migration and epithlial-mesenchymal transitionof human lens epithelial cells line SRA01/04. PLoS One. 2013;8(2):e56837.

[4]. Inomata M, et al. Pirfenidone inhibits fibrocyte accumulation in the lungs in bleomycin-induced murine pulmonary fibrosis. Respir Res. 2014 Feb 8;15:16.

[5]. Brooks D, et al. Limited fibrosis accompanies triple-negative breast cancer metastasis in multiple model systems and is not a preventive target. Oncotarget. 2018 May 4;9(34):23462-23481.

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