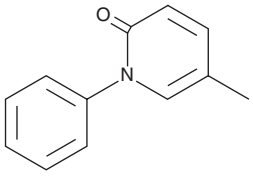
# PRODUCT INFORMATION

## Pirfenidone

*Item No. 13986*

**CAS Registry No.:** 53179-13-8
**Formal Name:** 5-methyl-1-phenyl-2(1H)-pyridinone
**Synonym:** AMR 69
**MF:** $C_{12}H_{11}NO$
**FW:** 185.2
**Purity:** ≥95%
**UV/Vis.:** $\lambda_{max}$: 203, 223, 319 nm
**Supplied as:** A crystalline solid
**Storage:** -20°C
**Stability:** ≥4 years




*Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.*

### Laboratory Procedures

Pirfenidone is supplied as a crystalline solid. A stock solution may be made by dissolving the pirfenidone in the solvent of choice. Pirfenidone is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of pirfenidone in these solvents is approximately 20, 17, and 10 mg/ml, respectively.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. Organic solvent-free aqueous solutions of pirfenidone can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of pirfenidone in PBS, pH 7.2, is approximately 3 mg/ml. We do not recommend storing the aqueous solution for more than one day.

### Description

Pirfenidone is an orally bioavailable pyridone derivative with antifibrotic, anti-inflammatory, and antioxidant activities.[1-5] It inhibits TGF-β1-stimulated increases in collagen type I, fibronectin, and Hsp47 expression in A549 lung cancer cells in a concentration-dependent manner.[4] Pirfenidone (300 mg/kg per day) inhibits fibrosis and inhibits increases in collagen content, fibrocyte pool size, and the levels of chemokines CCL2 and CCL12 in lung in a mouse model of pulmonary fibrosis induced by bleomycin (Item No. 13877).[2] In a mouse model of non-alcoholic steatohepatitis (NASH), pirfenidone inhibits fibrosis and increases in hepatocyte apoptosis, lobular inflammation, and hepatic expression of the fibrosis-related genes *Col1a1*, *Timp1*, *Tgfb1*, *Pdgfb*, and *Fgf2* when administered at a dose of 280 mg/kg but has no effect on steatosis.[1] Pirfenidone also inhibits NADPH-dependent lipid peroxidation in sheep liver microsomes and scavenges hydroxyl radicals ($IC_{50}$s = ~6 and ~2.5 mM, respectively).[3] Formulations containing pirfenidone have been used in the treatment of idiopathic pulmonary fibrosis.

### References


1. Komiya, C., Tanaka, M., Tsuchiya, K., *et al. Sci. Rep.* **7:44754** (2017).
2. Inomata, M., Kamio, K., Azuma, A., *et al. Respir. Res.* **15:16** (2014).
3. Misra, H.P. and Rabideau, C. *Mol. Cell. Biochem.* **204(1-2)**, 119-126 (2000).
4. Hisatomi, K., Mukae, H., Sakamoto, N., *et al. PBMC Pulm. Med.* **12(24)**, (2012).
5. Moeller, A., Ask, K., Warburton, D., *et al. Int. J. Biochem. Cell Biol.* **40(3)**, 362-382 (2008).





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