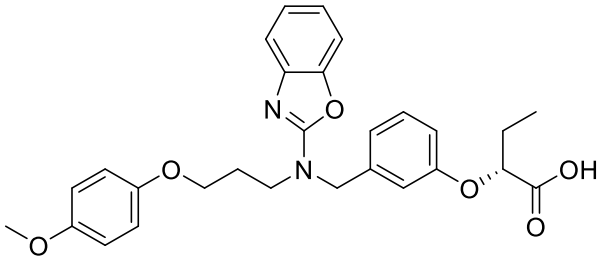
Product data sheet

MedKoo Cat#: 206448 Name: Pemafibrate CAS#: 848259-27-8 (free acid) Chemical Formula: $C_{28}H_{30}N_2O_6$ Exact Mass: 490.2104 Molecular Weight: 490.56	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:
Pemafibrate, also known as (R)-K 13675, is a PPAR alpha agonist. (R)-K-13675 decreases the secretion of inflammatory markers without affecting cell proliferation or tube formation. Peroxisome proliferator-activated receptor-alpha (PPAR-alpha) is a key regulator of lipid and glucose metabolism and has been implicated in inflammation. (R)-K-13675 was associated with the inhibition of inflammatory responses without affecting cell proliferation or angiogenesis, and subsequently may induce an anti-atherosclerotic effect.

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	30.0	61.2

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.04 mL	10.19 mL	20.38 mL
5 mM	0.41 mL	2.04 mL	4.08 mL
10 mM	0.20 mL	1.02 mL	2.04 mL
50 mM	0.04 mL	0.20 mL	0.41 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study
In vitro study

1. Li W, Xu J, Guo X, Xia X, Sun Y. Pemafibrate suppresses oxidative stress and apoptosis under cardiomyocyte ischemia-reperfusion injury in type 1 diabetes mellitus. Exp Ther Med. 2021 Apr;21(4):331. doi: 10.3892/etm.2021.9762. Epub 2021 Feb 8. PMID: 33732304; PMCID: PMC7903427.
2. Horikawa T, Kawanami T, Hamaguchi Y, Tanaka Y, Kita S, Ryorin R, Takashi Y, Takahashi H, Tanabe M, Yanase T, Kawanami D, Nomiyama T. Pemafibrate, a PPAR alpha agonist, attenuates neointima formation after vascular injury in mice fed normal chow and a high-fat diet. Heliyon. 2020 Nov 6;6(11):e05431. doi: 10.1016/j.heliyon.2020.e05431. PMID: 33204884; PMCID: PMC7653074.

In vivo study

1. Sasaki Y, Asahiyama M, Tanaka T, Yamamoto S, Murakami K, Kamiya W, Matsumura Y, Osawa T, Anai M, Fruchart JC, Aburatani H, Sakai J, Kodama T. Pemafibrate, a selective PPARα modulator, prevents non-alcoholic steatohepatitis development without reducing the hepatic triglyceride content. Sci Rep. 2020 May 8;10(1):7818. doi: 10.1038/s41598-020-64902-8. PMID: 32385406; PMCID: PMC7210999.

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2. Tomita Y, Ozawa N, Miwa Y, Ishida A, Ohta M, Tsubota K, Kurihara T. Pemafibrate Prevents Retinal Pathological Neovascularization by Increasing FGF21 Level in a Murine Oxygen-Induced Retinopathy Model. Int J Mol Sci. 2019 Nov 23;20(23):5878. doi: 10.3390/ijms20235878. PMID: 31771164; PMCID: PMC6928689.

7. Bioactivity

Biological target:

Pemafibrate is a selective antagonist of peroxisome proliferator-activated receptor α (PPARα; EC_{50} = 1 nM for transcriptional activity), a transcription factor that is essential for regulation of lipid homostasis, and is selective for PPARα over PPARδ and PPARγ (EC50 = 2,300 and 1,000 nM, respectively, for transcriptional activity).

In vitro activity

Under HG + H/R conditions, NF-κB protein expression levels were significantly enhanced in H9c2 cells compared with those in the control group (P<0.001; Fig. 4A). Pemafibrate significantly suppressed the protein expression levels of NF-κB compared with those in the HG + H/R group (P<0.001; Fig. 4A). To evaluate whether pemafibrate may regulate mitochondrial dysfunction and apoptosis through the NF-κB signaling pathway, NF-κB was overexpressed in H9c2 cells (P<0.001; Fig. 4B). The results demonstrated that overexpression of NF-κB reversed the pemafibrate-induced increase in ATP levels in the HG + H/R treatment group (P<0.01 vs. pemafibrate + HG + H/R and pemafibrate + HG + H/R + NC; Fig. 4C). Pemafibrate decreased the expression levels of cleaved-caspase-3 compared with those in the HG + H/R group; however, overexpression of NF-κB reversed this effect (P<0.01 vs. pemafibrate + HG + H/R and pemafibrate + HG + H/R + NC; Fig. 4D), indicating that pemafibrate inhibited the HG + H/R-induced apoptosis by regulating the NF-κB signaling pathway. However, overexpression of NF-κB expression failed to significantly reverse the pemafibrate-induced reduction in Cyt-c expression levels (Fig. 4D). These results suggest that pemafibrate may prevent mitochondrial dysfunction by interacting with the NF-κB signaling pathway.

Exp Ther Med. 2021 Apr; 21(4): 331. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7903427/

In vivo activity

To verify the effect of pemafibrate on STAM mouse livers, a comprehensive transcriptome analysis was performed by RNA-seq using liver tissues collected from normal, STAM control, and pemafibrate-treated STAM mice. 187 up-regulated and 477 down-regulated genes were identified in the pemafibrate-treated compared with the STAM control group by our stringent criteria (Supplementary Table 1). In fact, PPARα–regulated FAO-related genes were significantly induced in the pemafibrate-treated group (Supplementary Fig. 1). The expression levels of genes related to TG hydrolysis, fatty acid uptake, fatty acid activation, fatty acid binding, peroxisomal and mitochondrial oxidation, and ketogenesis were higher in the STAM control group than in the normal group (Supplementary Figs. 1 and 2). Pemafibrate apparently induced the expression of these genes. In particular, pemafibrate treatment resulted in the greatest increase in Pdk4 expression, suggesting that it mediates the suppression of glucose oxidation and preferential activation of fatty acid oxidation (Supplementary Figs. 1 and 2). Pemafibrate did not influence glycolysis and Pck1 expression but significantly induced a series of genes involved in TG synthesis from DHAP and glycerol (Fig. 2E,F). Pemafibrate had the greatest effect on Mogat1, which has key roles in TG re-esterification from monoacylglycerols and diacylglycerols generated by TG hydrolysis24 in STAM mouse livers (Fig. 2E,F). These results suggest that pemafibrate enhances TG synthesis from DHAP and glycerol and the re-esterification of glycerol generated by TG hydrolysis in STAM mouse livers.

Sci Rep. 2020; 10: 7818. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7210999/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.