

# Product data sheet



MedKoo Cat#: 314217 Name: Deferiprone CAS#: 30652-11-0 Chemical Formula: C <sub>7</sub> H <sub>9</sub> NO <sub>2</sub> Exact Mass: 139.06333 Molecular Weight: 139.15	
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:

Deferiprone is a drug that chelates iron and is used to treat thalassaemia major. In 1994 was first approved for use in treating thalassaemia major in 1994 and had been licensed for use in Europe and Asia for many years while awaiting approval in Canada and the United States. On October 14, 2011, it was approved for use in the US under the FDA's accelerated approval program.

## 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	7.14	51.31
H <sub>2</sub> O	18.0	129.36

## 4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	7.19 mL	35.93 mL	71.86 mL
5 mM	1.44 mL	7.19 mL	14.37 mL
10 mM	0.72 mL	3.59 mL	7.19 mL
50 mM	0.14 mL	0.72 mL	1.44 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

- Fiorillo M, Tóth F, Brindisi M, Sotgia F, Lisanti MP. Deferiprone (DFP) Targets Cancer Stem Cell (CSC) Propagation by Inhibiting Mitochondrial Metabolism and Inducing ROS Production. *Cells*. 2020 Jun 23;9(6):1529. doi: 10.3390/cells9061529. PMID: 32585919; PMCID: PMC7349387.
- Ramezanpour M, Smith JLP, Ooi ML, Gouzos M, Psaltis AJ, Wormald PJ, Vreugde S. Deferiprone has anti-inflammatory properties and reduces fibroblast migration in vitro. *Sci Rep*. 2019 Feb 20;9(1):2378. doi: 10.1038/s41598-019-38902-2. PMID: 30787349; PMCID: PMC6382764.

### In vivo study

- Carboni E, Tatenhorst L, Tönges L, Barski E, Dambeck V, Bähr M, Lingor P. Deferiprone Rescues Behavioral Deficits Induced by Mild Iron Exposure in a Mouse Model of Alpha-Synuclein Aggregation. *Neuromolecular Med*. 2017 Sep;19(2-3):309-321. doi: 10.1007/s12017-017-8447-9. Epub 2017 Jun 16. PMID: 28623611; PMCID: PMC5570801.
- Song D, Zhao L, Li Y, Hadziahmetovic M, Song Y, Connelly J, Spino M, Dunaief JL. The oral iron chelator deferiprone protects against systemic iron overload-induced retinal degeneration in hepcidin knockout mice. *Invest Ophthalmol Vis Sci*. 2014 Jun 26;55(7):4525-32. doi: 10.1167/iops.14-14568. PMID: 24970260; PMCID: PMC4106252.

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## 7. Bioactivity

### Biological target:

Deferiprone is the only orally active iron-chelating drug to be used therapeutically in conditions of transfusional iron overload.

### In vitro activity

It was hypothesized that DFP treatment could be used to selectively target mitochondria in cancer stem cells (CSCs). For this purpose, two ER(+) human breast cancer cell lines were used, namely MCF7 and T47D cells, as model systems. Figure 2A shows that DFP inhibits anchorage-independent growth remarkably well, with an IC-50 of ~100 nM for MCF7 cells and an IC-50 of ~500 nM for T47D cells after 5 days of treatment. Therefore, it was estimated that CSCs are approximately 1000-fold more sensitive to DFP than the “bulk” cancer cell population. In addition, CSCs’ formation was evaluated in the presence of NAC. Interestingly, it was observed that the DFP-induced reduction in the 3D tumorsphere formation reverted in the presence of 1 mM and 5 mM of NAC (Figure 2). Additionally, ALDH activity was used to further validate the effects of DFP on CSCs. Figure 3b demonstrates that 50 μM of DFP reduced the ALDH activity by >75% after 5 days of treatment. As ALDH is a metabolic marker of Epithelial-Mesenchymal Transition (EMT), this provides additional supporting evidence that DFP indeed targets the “stemness” phenotype of CSCs.

Reference: Cells. 2020 Jun; 9(6): 1529. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7349387/>

### In vivo activity

The purpose of this study was to investigate the retinal-protective effects of the oral iron chelator deferiprone (DFP) in mice lacking the iron regulatory hormone hepcidin (Hepc). Hepc KO mice were given DFP in drinking water from age 6 to 18 months. They were then compared to Hepc KO mice not receiving DFP. In Hepc KO mice, DFP diminished RPE depigmentation and autofluorescence on fundus imaging. Autofluorescence in the RPE layer in cryosections was significantly diminished by DFP, consistent with the fundus images. Immunolabeling with L-ferritin and transferrin receptor antibodies showed a decreased signal for L-ferritin in the inner retina and RPE cells and an increased signal for transferrin receptor in the inner retina, indicating diminished retinal iron levels with DFP treatment. Plastic sections showed that photoreceptor and RPE cells were well preserved in Hepc KO mice treated with DFP. Consistent with photoreceptor protection, the mRNA level of rhodopsin was significantly higher in retinas treated with DFP. The mRNA levels of oxidative stress-related genes heme oxygenase-1 and catalase were significantly lower in DFP-treated Hepc KO retinas. Furthermore, ERG rod a- and b- and cone b-wave amplitudes were significantly higher in DFP-treated mice.

Reference: Invest Ophthalmol Vis Sci. 2014 Jun 26;55(7):4525-32. <https://pubmed.ncbi.nlm.nih.gov/24970260/>

*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.*