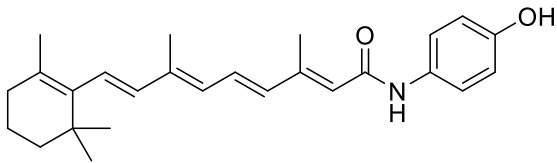


# Product data sheet



MedKoo Cat#: 205430 Name: Fenretinide CAS#: 65646-68-6 Chemical Formula: C <sub>26</sub> H <sub>33</sub> NO <sub>2</sub> Exact Mass: 391.25113 Molecular Weight: 391.54572		
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:

Fenretinide is an orally-active synthetic phenylretinamide analogue of retinol (vitamin A) with potential antineoplastic and chemopreventive activities. Fenretinide binds to and activates retinoic acid receptors (RARs), thereby inducing cell differentiation and apoptosis in some tumor cell types. This agent also inhibits tumor growth by modulating angiogenesis-associated growth factors and their receptors and exhibits retinoid receptor-independent apoptotic properties.

## 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	130	332.01
Ethanol	78	199.21

## 4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.55 mL	12.77 mL	25.54 mL
5 mM	0.51 mL	2.55 mL	5.11 mL
10 mM	0.26 mL	1.28 mL	2.55 mL
50 mM	0.05 mL	0.26 mL	0.51 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. Bikman BT, Guan Y, Shui G, Siddique MM, Holland WL, Kim JY, Fabriàs G, Wenk MR, Summers SA. Fenretinide prevents lipid-induced insulin resistance by blocking ceramide biosynthesis. J Biol Chem. 2012 May 18;287(21):17426-17437. doi: 10.1074/jbc.M112.359950. Epub 2012 Apr 2. PMID: 22474281; PMCID: PMC3366851.

2. Golubkov V, Garcia A, Markland FS. Action of fenretinide (4-HPR) on ovarian cancer and endothelial cells. Anticancer Res. 2005 Jan-Feb;25(1A):249-53. PMID: 15816545.

### In vivo study

1. Bikman BT, Guan Y, Shui G, Siddique MM, Holland WL, Kim JY, Fabriàs G, Wenk MR, Summers SA. Fenretinide prevents lipid-induced insulin resistance by blocking ceramide biosynthesis. J Biol Chem. 2012 May 18;287(21):17426-17437. doi: 10.1074/jbc.M112.359950. Epub 2012 Apr 2. PMID: 22474281; PMCID: PMC3366851.

## 7. Bioactivity

Biological target:

# Product data sheet



Fenretinide (4-HPR) is a synthetic retinoid derivative, binding to the retinoic acid receptors (RAR) at concentrations necessary to induce cell death.

## In vitro activity

4-HPR inhibited OVCAR-5 cell proliferation and viability at concentrations higher than 1 microM, with 70-90% growth inhibition at 10 microM. 4-HPR (1 microM) significantly inhibited OVCAR-5 invasion after 3 days preincubation. In view of the importance of the cytoskeleton in cell motility, we examined the action of 4-HPR on the actin cytoskeleton and on FAK phosphorylation. In OVCAR-5 cells treated with 1 mM fenretinide for 3 days, actin cytoskeleton stress fibers were disrupted and FAK tyrosine phosphorylation was elevated dose-dependently. Endothelial cells treated with 1 microM 4-HPR failed to form tubes, but formed small cellular aggregates.

Reference Anticancer Res. 2005 Jan-Feb;25(1A):249-53. <http://ar.iiarjournals.org/cgi/pmidlookup?view=long&pmid=15816545>

## In vivo activity

Fenretinide was shown previously to improve insulin resistance in diet-induced obese mice. The proposed mechanism was that it promotes the urinary excretion of RBP4, an adipose-derived secretagogue implicated in insulin resistance. However, subsequent studies revealed that the compound could combat hepatic steatosis and obesity in RBP4-null mice, suggesting the existence of other targets that mediated its antidiabetic actions. To determine whether fenretinide is able to target the sphingolipid synthesis pathway in vivo, lipids from soleus muscle and liver of mice 12 h after the delivery of the drug by intraperitoneal injection were measured. The single injection of the drug only slightly reduced muscle ceramide levels, but robustly increased dihydroceramide levels (Fig. 5, A and B). A similar, although less robust, effect was observed in the liver (Fig. 5, C and D). However, although fenretinide had a more pronounced effect on shorter acyl chain dihydroceramide species (e.g. C16) in muscle, it appeared to affect a longer chain species more specifically in the liver (C24). This is consistent with the recent observation that the dominant ceramide synthase in the liver (CerS2) makes predominantly longer ceramides. Altogether, these data again indicate that fenretinide modulates sphingolipid levels and are highly suggestive that it inhibits Des1 in vivo.

Reference: J Biol Chem. 2012 May 18;287(21):17426-17437. <https://www.ncbi.nlm.nih.gov/pmc/articles/pmid/22474281/>

*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.*