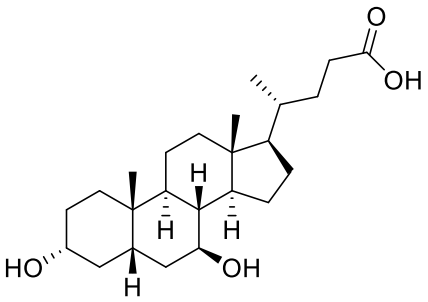
Product data sheet

MedKoo Cat#: 203091 Name: Ursodiol CAS#: 128-13-2 (free acid) Chemical Formula: $C_{24}H_{40}O_4$ Exact Mass: 392.29266 Molecular Weight: 392.57	
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:

Ursodiol, also known as Ursodeoxycholic acid, is a synthetically-derived form of ursodiol, a bile acid produced by the liver and secreted and stored in the gallbladder. Also produced by the Chinese black bear liver, ursodiol has been used in the treatment of liver disease for centuries. This agent dissolves or prevents cholesterol gallstones by blocking hepatic cholesterol production and decreasing bile cholesterol. Ursodiol also reduces the absorption of cholesterol from the intestinal tract.

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	56.0	142.65

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.55 mL	12.74 mL	25.47 mL
5 mM	0.51 mL	2.55 mL	5.09 mL
10 mM	0.25 mL	1.27 mL	2.55 mL
50 mM	0.05 mL	0.25 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. Nie B, Park HM, Kazantzis M, Lin M, Henkin A, Ng S, Song S, Chen Y, Tran H, Lai R, Her C, Maher JJ, Forman BM, Stahl A. Specific bile acids inhibit hepatic fatty acid uptake in mice. Hepatology. 2012 Oct;56(4):1300-10. doi: 10.1002/hep.25797. PMID: 22531947; PMCID: PMC3445775.
2. Hu J, Hong W, Yao KN, Zhu XH, Chen ZY, Ye L. Ursodeoxycholic acid ameliorates hepatic lipid metabolism in LO2 cells by regulating the AKT/mTOR/SREBP-1 signaling pathway. World J Gastroenterol. 2019 Mar 28;25(12):1492-1501. doi: 10.3748/wjg.v25.i12.1492. PMID: 30948912; PMCID: PMC6441910.

In vivo study

1. Zhang Y, Zheng X, Huang F, Zhao A, Ge K, Zhao Q, Jia W. Ursodeoxycholic Acid Alters Bile Acid and Fatty Acid Profiles in a Mouse Model of Diet-Induced Obesity. Front Pharmacol. 2019 Jul 25;10:842. doi: 10.3389/fphar.2019.00842. PMID: 31402868; PMCID: PMC6669341.
2. Chen YS, Liu HM, Lee TY. Ursodeoxycholic Acid Regulates Hepatic Energy Homeostasis and White Adipose Tissue Macrophages Polarization in Leptin-Deficiency Obese Mice. Cells. 2019 Mar 16;8(3):253. doi: 10.3390/cells8030253. PMID: 30884843; PMCID: PMC6468643.

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7. Bioactivity

Biological target:

Ursodiol (Ursodeoxycholic acid; UDCA) is a potent liver-specific fatty acid transport protein 5 (FATP5) inhibitor that inhibits LCFA uptake by primary hepatocytes in a FATP5-dependent manner.

In vitro activity

To further explore the physiological and pharmacological implications of hepatic FATP inhibition by secondary bile acids the effects of UDCA (ursodeoxycholic acid) on LCFA uptake were by primary hepatocytes were tested. Using a FACS-based LCFA uptake assay that allows for the gating of viable cells, it was found that UDCA but not TUDCA inhibited LCFA uptake by primary human hepatocytes (Sup. Fig. 5). UDCA also inhibited LCFA uptake by primary mouse hepatocytes from C57Bl/6 animals without any detectable cytotoxic effects (Fig. 5A right). Importantly, this effect was entirely FATP5 dependent, as UDCA (ursodeoxycholic acid) failed to inhibited LCFA uptake by primary hepatocytes from FATP5-null animals (Fig. 5A). While TUDCA did not inhibit FATP2 and -5 mediated uptake, UDCA was identified as a potent inhibitor of FATP5 (Fig. 3) with an IC50 of 5 μM (Tab. 1). Here a novel link is reported between bile acids and metabolism, i.e. the ability of, specific bile acids such as DCA and UDCA to inhibit hepatic LCFA uptake.

Hepatology. 2012 Oct; 56(4): 1300–1310. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3445775/

In vivo activity

To elucidate the mechanism by which UDCA (ursodeoxycholic acid) alters FFA (free fatty acid) profiles in vivo in C57BL/6 mice, the expression of genes involved in TG synthesis and FFA synthesis, oxidation, and uptake in liver, EAT, and BAT were examined. Sterol regulatory element-binding protein 1c (SREBP1c)—a transcription factor that promotes the expression of lipogenic genes including acetyl coenzyme A (CoA) carboxylase 1 (ACC1), FA synthase (FAS), and stearoyl-CoA desaturase-1 (SCD1)—was downregulated by UDCA treatment, with a corresponding decrease in FAS, ACC1, and SCD1 mRNA levels (Figure 5A), indicating that de novo lipogenesis was inhibited. Diacylglycerol acyltransferase (DGAT) is the key enzyme promoting TG formation; it was found here that UDCA suppressed DGAT1 and DGAT2 expressions (Figure 5B), which is consistent with the observed reduction in liver TG content (Figure 1G). The expression of factors involved in FA oxidation including peroxisome proliferator-activated receptor α (PPARα), carnitine palmitoyl transferase 1A (CPT1A), and acy-CoA oxidase-1 (ACOX1) was inhibited by consumption of HFD, but this was reversed by UDCA (Figure 5C). In addition, genes involved in FA uptake in liver including FA transport protein 2 (FATP2), FATP5, and cluster of differentiation 36 (CD36) were downregulated in the UDCA group, whereas FATP2 and CD36 were upregulated in the HFD group (Figure 5D). In EAT and BAT, factors associated with FA uptake and lipogenesis were also suppressed by UDCA administration to varying degrees (Figures 5E–H). These results demonstrate that UDCA alters the FFA profile by inhibiting lipogenesis, promoting FA oxidation, and reducing FA uptake in liver and adipose tissue. Additionally, UDCA repressed the expression of genes regulating TG synthesis, thereby decreasing TG deposition in liver.

Front Pharmacol. 2019; 10: 842. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6669341/