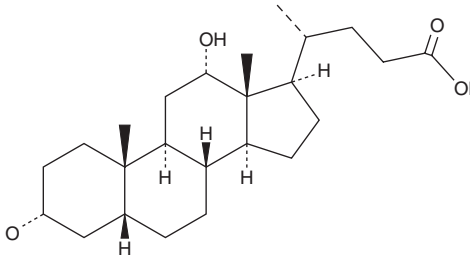
PRODUCT INFORMATION

Deoxycholic Acid

Item No. 20756

CAS Registry No.:	83-44-3
Formal Name:	(3α,5β,12α)-3,12-dihydroxy-cholan-24-oic acid
Synonyms:	Cholanoic Acid, DCA, NSC 8797
MF:	$C_{24}H_{40}O_4$
FW:	392.6
Purity:	≥95%
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

Deoxycholic acid (DCA) is supplied as a crystalline solid. A stock solution may be made by dissolving the DCA in the solvent of choice, which should be purged with an inert gas. DCA is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of DCA in ethanol and DMSO is approximately 20 mg/ml and approximately 30 mg/ml in DMF.

DCA is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, DCA should first be dissolved in DMF and then diluted with the aqueous buffer of choice. DCA has a solubility of approximately 0.5 mg/ml in a 1:1 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

DCA is a secondary bile acid that is formed *via* microbial transformation of cholic acid (Item No. 20250) in the colon.[1] It can be conjugated to glycine or taurine (Item No. 27031) to produce glycodeoxycholic acid (GDCA; Item No. 20274) or taurodeoxycholic acid (TDCA; Item No. 15935), respectively, in hepatocytes.[1-3] DCA (0.2% v/v) inhibits spore germination induced by taurocholic acid (TCA; Item No. 16215) in seven *C. difficile* strains, as well as inhibits growth and decreases the cytotoxicity of *C. difficile* culture supernatants to Vero cells when used at a concentration of 0.02% v/v.[1] It inhibits ionizing radiation-induced p53-dependent transcription in a reporter assay using HCT116 cells when used at a concentration of 200 µM.[4] Fecal and intestinal tissue levels of DCA are increased in a rat model of high-fat diet-induced obesity compared with rats fed a normal diet.[5] Increased serum DCA levels have been found in patients with colorectal cancer.[6]

References

1. Thanissery, R., Winston, J.A., and Theriot, C.M. Inhibition of spore germination, growth, and toxin activity of clinically relevant *C. difficile* strains by gut microbiota derived secondary bile acids. *Anaerobe* **45**, 86-100 (2017).
2. Schmid, A., Neumann, H., Karrasch, T., *et al.* Bile acid metabolome after an oral lipid tolerance test by liquid chromatography-tandem mass spectrometry (LC-MS/MS). *PLoS One* **11(2)**, e0148869 (2016).
3. Šarenac, T.M. and Mikov, M. Bile acid synthesis: From nature to the chemical modification and synthesis and their applications as drugs and nutrients. *Front. Pharmacol.* **9**, 939 (2018).
4. Qiao, D., Gaitonde, S.V., Qi, W., *et al.* Deoxycholic acid suppresses p53 by stimulating proteasome-mediated p53 protein degradation. *Carcinogenesis* **22(6)**, 957-964 (2001).
5. Lin, H., An, Y., Tang, H., *et al.* Alterations of bile acids and gut microbiota in obesity induced by high fat diet in rat model. *J. Agric. Food Chem.* **67(13)**, 3624-3632 (2019).
6. Bayerdörffer, E., Mannes, G.A., Richter, W.O., *et al.* Increased serum deoxycholic acid levels in men with colorectal adenomas. *Gastroenterology* **104(1)**, 145-151 (1993).

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