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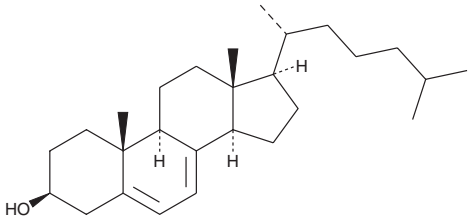
PRODUCT INFORMATION

7-dehydro Cholesterol

Item No. 14612

CAS Registry No.: 434-16-2
Formal Name: cholesta-5,7-dien-3β-ol
Synonyms: Δ^7-Cholesterol, 7-DHC, NSC 18159, Provitamin D_3
MF: $C_{27}H_{44}O$
FW: 384.6
Purity: ≥90%
UV/Vis.: λ_{max}: 271, 282, 293 nm
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

7-dehydro cholesterol (7-DHC) is supplied as a crystalline solid. A stock solution may be made by dissolving the 7-DHC in the solvent of choice. 7-DHC is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of 7-DHC in these solvents is approximately 20, 0.1, and 2 mg/ml, respectively.

7-DHC is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, 7-DHC should first be dissolved in ethanol and then diluted with the aqueous buffer of choice. 7-DHC has a solubility of approximately 0.3 mg/ml in a 1:2 solution of ethanol:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

7-DHC is an immediate precursor of cholesterol.[1] It is reduced to cholesterol by the enzyme 3β-hydroxysterol-Δ^7-reductase (DHCR7) in the last step of cholesterol biosynthesis. 7-DHC accumulates in Smith-Lemli-Opitz syndrome (SLOS), a disorder characterized by a mutation in the *DHCR7* gene and decreased cholesterol levels in bodily tissues and fluids, as well as microcephaly, intellectual disability, and distinctive dysmorphic features.[1,2] It is highly susceptible to free radical oxidation, giving rise to several oxysterols that may be involved in the pathogenesis of SLOS.[1] 7-DHC levels are increased in brain, liver, and serum in a rat model of SLOS induced by the DHCR7 inhibitor AY 9944 (Item No. 14611).[1] 7-DHC is a provitamin that is converted to vitamin D_3 (Item No. 11792) by ultraviolet-B (UVB) light in a human skin equivalent system and in isolated human skin samples.[3,4]

References

1. Xu, L., Liu, W., Sheflin, L.G., *et al.* Novel oxysterols observed in tissues and fluids of AY9944-treated rats: A model for Smith-Lemli-Opitz syndrome. *J. Lipid Res.* **52**, 1810-1820 (2011).
2. Xu, G., Salen, G., Shefer, S., *et al.* Reproducing abnormal cholesterol biosynthesis as seen in the Smith-Lemli-Opitz syndrome by inhibiting the conversion of 7-dehydrocholesterol to cholesterol in rats. *J. Clin. Invest.* **95(1)**, 76-81 (1995).
3. Lehmann, B., Genehr, T., Knuschke, P., *et al.* UVB-induced conversion of 7-dehydrocholesterol to 1α,25-dihydroxyvitamin D_3 in an *in vitro* human skin equivalent model. *J. Invest. Dermatol.* **117(5)**, 1179-1185 (2001).
4. Chen, T.C., Chimeh, F., Lu, Z., *et al.* Factors that influence the cutaneous synthesis and dietary sources of vitamin D. *Arch. Biochem. Biophys.* **460(2)**, 213-217 (2007).

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