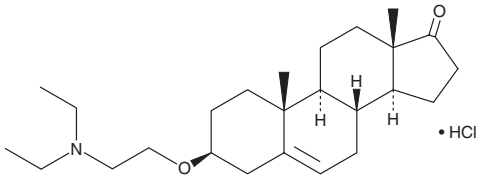
PRODUCT INFORMATION

U-18666A

Item No. 10009085

CAS Registry No.: 3039-71-2
Formal Name: 3β-[2-(diethylamino)ethoxy]-androst-5-en-17-one, monohydrochloride
MF: $C_{25}H_{41}NO_2$ • HCl
FW: 424.1
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

U-18666A is supplied as a crystalline solid. A stock solution may be made by dissolving the U-18666A in the solvent of choice, which should be purged with an inert gas. U-18666A is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of U-18666A in ethanol is approximately 20 mg/ml and approximately 10 mg/ml in DMSO and DMF.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. Organic solvent-free aqueous solutions of U-18666A can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of U-18666A in PBS (pH 7.2) is approximately 0.5 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Impaired trafficking of cholesterol to or from the endoplasmic reticulum (ER) has been implicated in many disease states including Neimann-Pick disease type C (NPC), Alzheimers disease, and atherosclerosis. U-18666A is a cell permeable drug that inhibits cholesterol trafficking. It inhibits cholesterol transport from late endosomes/lysosomes to the ER, but not cholesterol transport to the plasma membrane as demonstrated in many cell types including macrophages, primary cortical neurons, and primary fibroblasts.[1-3] In macrophages, micromolar concentrations of U-18666A inhibit multiple pathways of cholesterol trafficking from late endosomes, whereas nanomolar concentrations impair cholesterol trafficking to the ER, a response similar to that found in NPC. U-18666A inhibits oxidosqualene cyclase at high (>0.5 µM) concentrations and oral doses (10 mg/kg) induces cataracts in rats.[1]

References

1. Koh, C.H.V. and Cheung, N.S. Cellular mechanism of U18666A-mediated apoptosis in cultured murine cortical neurons: Bridging Niemann-Pick disease type C and Alzheimer's disease. *Cell. Signal.* **18(11)**, 1844-1853 (2006).
2. Tabas, I. Apoptosis and plaque destabilization in atherosclerosis: The role of macrophage apoptosis induced by cholesterol. *Cell Death and Differentiation* **11**, S12-S16 (2004).
3. Feng, B., Yao, P.M., Li, Y., *et al.* The endoplasmic reticulum is the site of cholesterol-induced cytotoxicity in macrophages. *Nature Cell Biology* **5(9)**, 781-792 (2003).

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