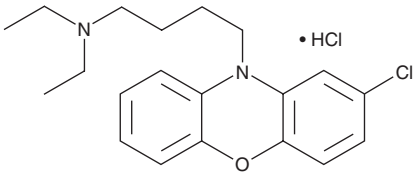
PRODUCT INFORMATION

Akt Inhibitor X

Item No. 14863

CAS Registry No.: 925681-41-0
Formal Name: 2-chloro-N,N-diethyl-10H-phenoxazine-10-butanamine, monohydrochloride
Synonym: 10-DEBC
MF: $C_{20}H_{25}ClN_2O \bullet HCl$
FW: 381.3
Purity: ≥95%
UV/Vis.: λ_{max}: 242, 333 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

Akt inhibitor X is supplied as a crystalline solid. A stock solution may be made by dissolving the Akt inhibitor X in the solvent of choice. Akt inhibitor X is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of Akt inhibitor X in these solvents is approximately 16, 12.5, and 5 mg/ml, respectively.

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 Akt inhibitor X can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of Akt inhibitor X in PBS, pH 7.2, is approximately 5 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Akt functions as a key component in multiple signaling pathways to promote cell survival by mediating resistance to apoptosis. Akt inhibitor X is a cell permeable phenoxazine derivative that suppresses insulin-like growth factor 1 (IGF-1)-stimulated phosphorylation of Akt with an IC_{50} value of 1-2 µM.[1] This compound has been shown to block IGF-1-stimulated nuclear translocation of Akt in Rh1 cells and to inhibit the growth of Rh1, Rh18, and Rh30 cells with IC_{50} values of 2-5 µM.[1] Inhibition of Akt phosphorylation by 5 µM of this compound strongly correlates to the inhibition of the downstream targets, mTOR, p70S6 kinase, and S6 ribosomal protein and to an increase in apoptosis.[1]

Reference

1. Thimmaiah, K.N., Easton, J.B., Germain, G.S., *et al.* Identification of N^{10}-substituted phenoxazines as potent and specific inhibitors of Akt signaling. *J. Biol. Chem.* **280(36)**, 31924-31935 (2005).

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