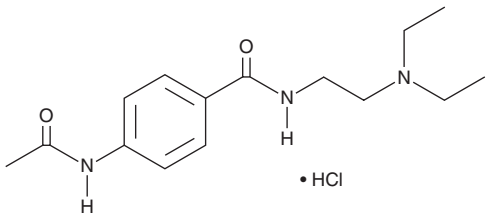
PRODUCT INFORMATION

Acecainide (hydrochloride)

Item No. 27312

CAS Registry No.:	34118-92-8
Formal Name:	4-(acetylamino)-N-[2-(diethylamino)ethyl]-benzamide, monohydrochloride
Synonym:	N-Acetylprocainamide
MF:	$C_{15}H_{23}N_3O_2$ • HCl
FW:	313.8
Purity:	≥98%
UV/Vis.:	λ_{max}: 272 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

Acecainide (hydrochloride) is supplied as a crystalline solid. A stock solution may be made by dissolving the acecainide (hydrochloride) in the solvent of choice, which should be purged with an inert gas. Acecainide (hydrochloride) is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of acecainide (hydrochloride) in these solvents is approximately 16, 14, and 20 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 acecainide (hydrochloride) can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of acecainide (hydrochloride) in PBS, pH 7.2, is approximately 10 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

Acecainide is an active metabolite of the class III antiarrhythmic procainamide.[1] It is formed from procainamide *via* hepatic N-acetyltransferases.[2] Acecainide prevents hypoxia-induced ventricular fibrillation in mice and decreases arrhythmia induced by aconitine in dogs.[1] Acecainide (10 µM), when used in high-fat medium, decreases lipid droplet area by greater than 50% in SK-Hep1 cells without inducing cytotoxicity but does not affect lipid droplets in primary mouse hepatocytes.[3]

References

1. Drayer, D.E., Reidenberg, M.M., and Sevy, R.W. N-Acetylprocainamide. Active metabolite of procainamide. *Proc. Soc. Exp. Biol. Med.* **146(2),** 358-363 (1974).
2. Woosley, R.L., Drayer, D.E., Reidenberg, M.M., *et al.* Effect of acetylator phenotype on the rate at which procainamide induces antinuclear antibodies and the lupus syndrome. *N. Engl. J. Med.* **298(21),** 1157-1159 (1978).
3. Luo, W.-J., Cheng, T.-Y., Wong, K.-I., *et al.* Novel therapeutic drug identification and gene correlation for fatty liver disease using high-content screening: Proof of concept. *Eur. J. Pharm. Sci.* **121,** 106-117 (2018).

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