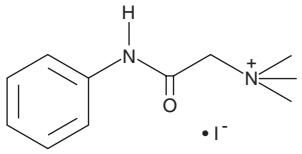
PRODUCT INFORMATION

CAY10568

Item No. 10012565

CAS Registry No.:	22913-17-3
Formal Name:	trimethyl[(phenylcarbamoyl) methyl]-ammonium iodide
Synonym:	Santinamide
MF:	$C_{11}H_{17}N_2O \bullet I$
FW:	320.2
Purity:	≥98%
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

CAY10568 is supplied as a crystalline solid. A stock solution may be made by dissolving the CAY10568 in the solvent of choice, which should be purged with an inert gas. CAY10568 is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of CAY10568 in these solvents is approximately 3 mg/ml.

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 CAY10568 can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of CAY10568 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

Most local anesthetics act by abolishing voltage gated sodium channel currents indiscriminately in all populations of neurons. Selective analgesia through TRPV1-mediated entry of a cationic lidocaine derivative, QX314, was recently reported.[1] CAY10568 is a physically smaller, less hydrophobic version of QX314 designed to be even more permeable to the TRPV1 ion channel when activated by agonists such as capsaicin and N-oleoyl dopamine. CAY10568 when given in combination with suitable TRPV1 agonists should produce selective blockade of the pain response while leaving motor, touch, and proprioception intact.

Reference

1. Binshtok, A.M., Bean, B.P., and Woolf, C.J. Inhibition of nociceptors by TRPV1-mediated entry of impermeant sodium channel blockers. *Nature* **449**, 607-610 (2007).

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