

## (-)Cevimeline hydrochloride hemihydrate

Cat. No.: HY-76772B

Molecular Formula: C<sub>10</sub>H<sub>17</sub>NOS.HCl.<sub>1/2</sub>H<sub>2</sub>O

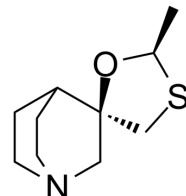
Molecular Weight: 244.78

Target: mAChR

Pathway: GPCR/G Protein; Neuronal Signaling

Storage: 4°C, sealed storage, away from moisture

\* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



HCl

0.5H<sub>2</sub>O

### SOLVENT & SOLUBILITY

#### In Vitro

H<sub>2</sub>O : 100 mg/mL (408.53 mM; Need ultrasonic)

Preparing Stock Solutions	Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	4.0853 mL	20.4265 mL	40.8530 mL
	5 mM	0.8171 mL	4.0853 mL	8.1706 mL
	10 mM	0.4085 mL	2.0427 mL	4.0853 mL

Please refer to the solubility information to select the appropriate solvent.

### BIOLOGICAL ACTIVITY

#### Description

(-)Cevimeline hydrochloride hemihydrate ((-)SNI-2011), a novel muscarinic receptor agonist, is a candidate therapeutic drug for xerostomia in Sjogren's syndrome. IC<sub>50</sub> value: Target: mAChR The general pharmacological properties of this drug on the gastrointestinal, urinary, and reproductive systems and other tissues were investigated in mice, rats, guinea pigs, rabbits, and dogs. The in vitro metab. of SNI-2011 was also evaluated with rat and dog liver microsomes. After oral administration, plasma concns. of SNI-2011 reached to Cmax within 1 h in both species, suggesting that SNI-2011 was quickly absorbed, and then decreased with a t<sub>1/2</sub> of 0.4-1.1 h. The bioavailability was 50% and 30% in rats and dogs, resp. Major metabolites in plasma were both S- and N-oxidized metabolites in rats and only N-oxidized metabolite in dogs, indicating that a large species difference was observed in the metab. of SNI-2011. Sex difference was also observed in the pharmacokinetics of SNI-2011 in rats, but not in dogs. In the in vitro study, chem. inhibition and pH-dependent studies revealed that the sulfoxidn. and N-oxidin. of SNI-2011 were mediated by cytochrome P 450 (CYP) and flavin-contg. monooxygenase (FMO), resp., in both species. In addn., CYP2D and CYP3A were mainly responsible for the sulfoxidn. in rat liver microsomes.

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**Caution: Product has not been fully validated for medical applications. For research use only.**

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