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# ProductInformation

**Mecamylamine hydrochloride**

Product Number **M 9020**
Store at Room Temperature

## Product Description

Molecular Formula: $C_{11}H_{21}N \bullet HCl$
Molecular Weight: 203.8
CAS Number: 826-39-1
Melting Point: 245.5 - 246.5 °C (with decomposition)
Synonyms: inversine, 2-(methylamino)isocamphane hydrochloride, N,2,3,3-tetramethyl-2-norbornanamine, N,2,3,3-tetramethylbicyclo[2.2.1]heptan-2-amine hydrochloride[1]

Mecamylamine is a ganglion blocking agent and a centrally acting nicotine antagonist that is used in neuroscience research.[1,2,3] It is a noncompetitive antagonist of the neuromuscular junction acetylcholine receptor.[4]

Mecamylamine is frequently utilized in studies of calcium flux and calcium channels. In an investigation of cultured mouse cerebral cortical neurons that were subjected to 72 hour nicotine exposure, mecamylamine counteracted the KCl-induced $^{45}Ca^{2+}$ influx via L-type voltage-dependent calcium channels.[5] In nicotine-treated mice, mecamylamine administration diminished the accumbal dopamine release compared to control mice.[6] Mecamylamine has been shown to block responses to nicotine in cultured SH-SY5Y cells.[7]

The acetylcholine-mediated release of arginine vasopressin from dissected hypothalamus *in vitro* has been antagonized by mecamylamine.[8] Mecamylamine has been demonstrated to block the action of neostigmine in increasing extracellular adenosine levels in rat cerebral cortex.[9]

## Precautions and Disclaimer

For Laboratory Use Only. Not for drug, household or other uses.

## Preparation Instructions

This product is soluble in water (50 mg/ml), yielding a clear, colorless solution. It is also soluble in alcohol (82 mg/ml), glycerol (104 mg/ml), and in isopropanol (21 mg/ml).[1]

## Storage/Stability

The pH of a 1% aqueous solution of this product is 6.0 - 7.5. Aqueous solutions of this product can be autoclaved.[1]

## References

1. The Merck Index, 12th ed., Entry# 5814.
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3. Wright, C. E., et al., Postural hypotension following N-type $Ca^{2+}$ channel blockade is amplified in experimental hypertension. J. Hypertens., **18(1)**, 65-73 (2000).
4. Varanda, W. A., et al., The acetylcholine receptor of the neuromuscular junction recognizes mecamylamine as a noncompetitive antagonist. Mol. Pharmacol., **28(2)**, 128-137 (1985).
5. Katsura, M., et al., Up-regulation of L-type voltage-dependent calcium channels after long term exposure to nicotine in cerebral cortical neurons. J. Biol. Chem., **277(10)**, 7979-7988 (2002).
6. Gaddnas, H., et al., Mecamylamine decreases accumbal dopamine output in mice treated chronically with nicotine. Neurosci. Lett., **330(3)**, 219-222 (2002).
7. Ridley, D. L., et al., Effects of chronic drug treatments on increases in intracellular calcium mediated by nicotinic acetylcholine receptors in SH-SY5Y cells. Br. J. Pharmacol., **135(4)**, 1051-1059 (2002).

8. Raber, J., et al., IL-1 β potentiates the acetylcholine-induced release of vasopressin from the hypothalamus *in vitro*, but not from the amygdala. Neuroendocrinology, **59(3)**, 208-217 (1994).

9. Bennett, H. J., et al., Activation of cholinergic and adrenergic receptors increases the concentration of extracellular adenosine in the cerebral cortex of unanesthetized rat. Neuroscience, **117(1)**, 119-127 (2003).

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