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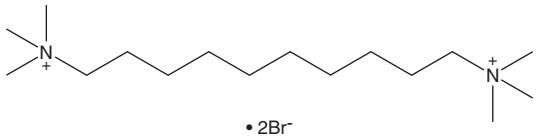
# PRODUCT INFORMATION

## Decamethonium (bromide)

*Item No. 24907*

**CAS Registry No.:** 541-22-0
**Formal Name:** $N^1,N^1,N^1,N^{10},N^{10},N^{10}$-hexamethyl-1,10-decanediaminium, dibromide
**MF:** $C_{16}H_{38}N_2 \bullet 2Br$
**FW:** 418.3
**Purity:** ≥98%
**Supplied as:** A crystalline solid
**Storage:** -20°C
**Stability:** ≥2 years




*Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.*

### Laboratory Procedures

Decamethonium (bromide) is supplied as a crystalline solid. A stock solution may be made by dissolving the decamethonium (bromide) in the solvent of choice. Decamethonium (bromide) is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of decamethonium (bromide) in these solvents is approximately 33, 16, and 2 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 decamethonium (bromide) can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of decamethonium (bromide) in PBS, pH 7.2, is approximately 5 mg/ml. We do not recommend storing the aqueous solution for more than one day.

### Description

Decamethonium is a depolarizing neuromuscular blocking agent.[1] It is a partial agonist of muscle-type nicotinic acetylcholine receptors (nAChRs).[2] Decamethonium activates α1β1-containing adult mouse muscle-type nAChRs expressed in *X. laevis* oocytes with an $EC_{50}$ value of 40 µM using voltage clamp electrophysiology. It is also a nondepolarizing antagonist of neuronal-type nAChRs, inhibiting mouse α7-, α3β2-, α3β4-, and α4β2-containing receptors with $IC_{50}$ values of 7.4, 405, 28, and 59 µM, respectively. Decamethonoium is a competitive antagonist of α4β2-containing nAChRs expressed in SH-EP1 cells ($IC_{50}$ = 52 µM for the human receptor).[3] It also inhibits electric eel acetylcholinesterase (AChE) and blocks electrically-evoked tibialis muscle twitches in anesthetized cats with $ED_{95}$ values of 35 and 70 µg/kg for cats under chloralose and ether anesthesia, respectively.[1,4] Formulations containing decamethonium have been used to induce paralysis during anesthesia.

### References


1. Paton, W.D. and Zaimis, E.J. The action of D-tubocurarine and of decamethonium on respiratory and other muscles in the cat. *J. Physiol.* **112(3-4)**, 311-331 (1951).
2. Papke, R.L., Wecker, L., and Stitzel, J.A. Activation and inhibition of mouse muscle and neuronal nicotinic acetylcholine receptors expressed in *Xenopus oocytes*. *J. Pharmacol. Exp. Ther.* **333(2)**, 501-518 (2010).
3. Eaton, J.B., Peng, J.H., Schroeder, K.M., *et al.* Characterization of human α4β 2-nicotinic acetylcholine receptors stably and heterologously expressed in native nicotinic receptor-null SH-EP1 human epithelial cells. *Mol. Pharmacol.* **64(6)**, 1283-1294 (2003).
4. Robaire, B. and Kato, G. Effects of edrophonium, serine, decamethonium, d-tubocurarine, and gallamine on the kinetics of membrane-bound and solubilized eel acetylcholinesterase. *Mol. Pharmacol.* **11(6)**, 722-734 (1974).





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