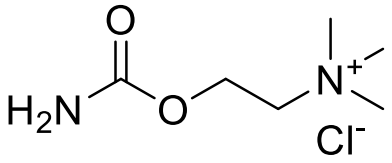
Product data sheet

MedKoo Cat#: 317367 Name: Carbachol CAS#: 51-83-2 Chemical Formula: $C_6H_{15}ClN_2O_2$ Exact Mass: 182.08221 Molecular Weight: 182.65	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.



1. Product description:
Carbachol is a cholinomimetic drug that binds and activates the acetylcholine receptor. Thus it is classified as a cholinergic agonist. It is primarily used for various ophthalmic purposes, such as for treating glaucoma, or for use during ophthalmic surgery. It is generally administered as an ophthalmic solution (i.e. eyedrops).

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	5	27.37
Water	36	197.10
Ethanol	12	65.70

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	**1 mg**	**5 mg**	**10 mg**
1 mM	5.47 mL	27.37 mL	54.75 mL
5 mM	1.09 mL	5.47 mL	10.95 mL
10 mM	0.55 mL	2.74 mL	5.47 mL
50 mM	0.11 mL	0.55 mL	1.09 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study

In vitro study

1. Dharmsathaphorn K, Pandol SJ. Mechanism of chloride secretion induced by carbachol in a colonic epithelial cell line. J Clin Invest. 1986 Feb;77(2):348-54. doi: 10.1172/JCI112311. PMID: 3003156; PMCID: PMC423353.

2. Keely SJ, Uribe JM, Barrett KE. Carbachol stimulates transactivation of epidermal growth factor receptor and mitogen-activated protein kinase in T84 cells. Implications for carbachol-stimulated chloride secretion. J Biol Chem. 1998 Oct 16;273(42):27111-7. doi: 10.1074/jbc.273.42.27111. PMID: 9765228.

In vivo study

1. Bourgin P, Escourrou P, Gaultier C, Adrien J. Induction of rapid eye movement sleep by carbachol infusion into the pontine reticular formation in the rat. Neuroreport. 1995 Feb 15;6(3):532-6. doi: 10.1097/00001756-199502000-00031. PMID: 7766858.

2. Nishikawa N, Chakrabarty B, Kitney D, Jabr R, Kanai A, Fry C. Stretch- and carbachol-induced ATP release from bladder wall preparations of young and aged mice. Neurourol Urodyn. 2020 Aug;39(6):1644-1652. doi: 10.1002/nau.24426. Epub 2020 Jun 12. PMID: 32531080; PMCID: PMC7641975.

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7. Bioactivity

Biological target:

Carbamoylcholine chloride (Carbachol, Carbamylcholine chloride, Carbastat, Miostat) is a cholinergic agonist that mimics the effect of acetylcholine on both the muscarinic and nicotinic receptors.

In vitro activity

Serosal application of carbachol to T84 cell monolayers mounted in an Ussing chamber caused an immediate increase in short circuit current (Isc) that peaked within 5 min and declined rapidly thereafter, although a small increase in Isc persisted for approximately 30 min. The increase in Isc was detectable with 1 microM carbachol; half-maximal with 10 microM carbachol; and maximal with 100 microM carbachol. Unidirectional Na+ and Cl- flux measurements indicated that the increase in Isc was due to net Cl- secretion. Carbachol did not alter cellular cAMP, but caused a transient increase in free cytosolic Ca2+ ([Ca2+]i) from 117 +/- 7 nM to 160 +/- 15 nM. The carbachol-induced increase in Isc was potentiated by either prostaglandin E1 (PGE1) or vasoactive intestinal polypeptide (VIP), agents that act by increasing cAMP. Measurements of cAMP and [Ca2+]i indicated that the potentiated response was not due to changes in these second messengers. Studies of the effects of these agents on ion transport pathways indicated that carbachol, PGE1, or VIP each increased basolateral K+ efflux by activating two different K+ transport pathways on the basolateral membrane. The pathway activated by carbachol was not sensitive to barium, while that activated by PGE1 or VIP was; furthermore, their action on K+ efflux are additive. Our study indicates that carbachol causes Cl- secretion, and that this action may result from its ability to increase [Ca2+]i and basolateral K+ efflux. Carbachol's effect on Cl- secretion is greatly augmented in the presence of VIP or PGE1, which open a cAMP-sensitive Cl- channel on the apical membrane, accounting for a potentiated response.

Reference: J Clin Invest. 1986 Feb;77(2):348-54. https://www.ncbi.nlm.nih.gov/pmc/articles/pmid/3003156/

In vivo activity

Cholinergic regulation of sleep and wakefulness was studied in freely moving rats locally infused with various doses of carbachol into the pontine reticular formation. Induction of REM sleep occurred when carbachol was infused specifically into the posterior oral pontine reticular nucleus (PnO). This effect was observed with 1-10 ng of carbachol, and lasted for at least 6 h. It was antagonized by atropine (100-200 ng) infused into the same site 15 min before carbachol (10 ng), indicating that REM sleep induction resulted from the stimulation of pontine muscarinic receptors. High doses of carbachol (500 ng) did not affect REM sleep but enhanced wakefulness. Cholinergic mechanisms within the PnO may play a critical role in the regulation of REM sleep in the rat.

Reference: Neuroreport. 1995 Feb 15;6(3):532-6.
https://journals.lww.com/neuroreport/Abstract/1995/02000/Induction_of_rapid_eye_movement_sleep_by_carbachol.31.aspx

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.