Product Data Sheet

Product Name: Vecuronium Bromide
Cat. No.: GC11210

Chemical Properties

Cas. No.	50700-72-6		
Chemical Name	[(2S,3S,5S,8R,9S,10S,13S,14S,16S,17R)-17-acetyloxy-10,13-dimethyl-16-(1-methylpiperidin-1-ium-1-yl)-2-piperidin-1-yl-2,3,4,5,6,7,8,9,11,12,14,15,16,17-tetradecahydro-1H-cyclopenta[a]phenanthren-3-yl] acetate;bromide		
SMILES	CC(=O)OC1CC2CCC3C(C2(CC1N4CCCCC4)C)CCC5(C3CC(C5OC(=O)C)[N+]6(CCCCC6)C)C.[Br-]		
Formula	$C_{34}H_{57}N_2O_4.Br$	M.Wt	637.73
Solubility	≥ 31.8865mg/mL in DMSO, ≥ 98.6 mg/mL in EtOH with ultrasonic	Storage	Store at 2-8°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

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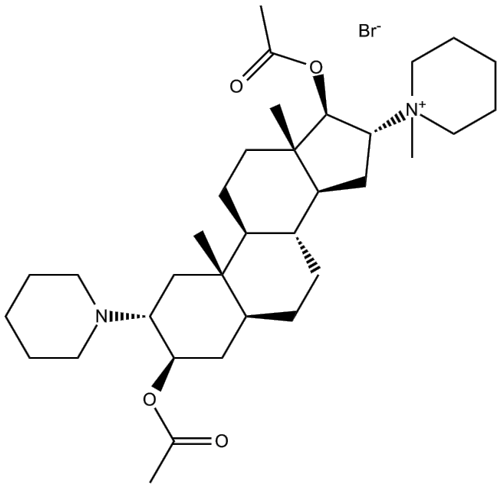
Structure



Protocol

Cell experiment [1]:

Cell lines	Adrenal medullary cells

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Preparation Method	Adrenal medullary cells were isolated by collagenase digestion of slices of bovine adrenal medulla as previously described. The cells were plated at a density of 4×10^6 cells/dish (35mm) in Eagle's minimum essential medium containing 10% fetal calf serum and several antibiotics. The cells were cultured in 5% CO_2/95% air in an incubator at 37°C and used for experiments after 2 to 4 days of culture. Cultured cells were incubated at 37°C for 15min in Krebs-Ringer HEPES (KRH) buffer containing 10mM pargyline, 100mM ascorbic acid, and 500nM [^{3}H]NE in the presence or absence of Vecuronium Bromide(0-100μM). KRH buffer consisted of 154mM NaCl, 5.6mM KCl, 1.1mM $MgSO_4$, 2.2mM $CaCl_2$, 10mM HEPES, and 10mM glucose, adjusted to pH 7.4. After incubation, the cells were rapidly washed four times with 1mL of ice-cold KRH buffer and solubilized in 1mL of 10% Triton X-100. The radioactivity in the solubilized cells was counted by a liquid scintillation counter. Nonspecific uptake was determined in the presence of 10mM desipramine and specific uptake was obtained by subtracting the nonspecific uptake from the total uptake.
Reaction Conditions	0-100μM; 15min
Applications	Vecuronium Bromide (0-100μM, 15min) inhibits [^{3}H] norepinephrine (NE) uptake to 65% at 100μM in adrenal medullary cells.

Animal experiment [2]:

Animal models	Wister rats

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Preparation Method	Experiments were performed on 24 adult Wister rats weighing 250–350g. Rats were anesthetized with inhaled ether and α-chloralose(100mg/kg, intraperitoneally). The carotid bifurcation was exposed, and the conjunction of the carotid sinus nerve and the glossopharyngeal nerve was identified. After systemic heparinization (2000IU/kg, IV), the rat was decapitated and the entire carotid bifurcation with the carotid sinus nerve was excised in one block. The tissue was immediately immersed into ice-cold Krebs solution equilibrated with 5% CO_2/95% O_2. Initially the carotid body was perfused with the normoxic Krebs solution. To assess the neural response to hypoxia, the perfusate was switched to the hypoxic Krebs solution for 5min. The perfusate was then returned to normoxic Krebs solution and normoxic perfusion was maintained for at least 5min. The hypoxic challenge was repeated several times until the stable neural response was confirmed. Subsequently, Vecuronium Bromide (0.1, 0.5, or 5μM) was administrated to the carotid body in normoxic Krebs solution for 30min. Then the perfusate was changed to the hypoxic Krebs solution with vecuronium for 5min followed by normoxic Krebs perfusion. One group of carotid bodies was not exposed to Vecuronium Bromide (Vecuronium Bromide 0μM). In these preparations, the hypoxic exposure was repeated every 30min for up to 2h to test the stability of the preparation.
Dosage form	0.1, 0.5, or 5μM; Intravenous injection; every 30min for 2h
Applications	Vecuronium Bromide attenuates the response of carotid sinus nerve activity (CSNA) to hypoxia in a dose-dependent manner.

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References:
[1] Uryu, K., Minami, K., Yanagihara, N., Hara, K., Toyohira, Y., Izumi, F., & Shigematsu, A. (2000). Inhibition by neuromuscular blocking drugs of norepinephrine transporter in cultured bovine adrenal medullary cells. *Anesthesia and analgesia*, *91*(3), 546–551.
[2] Igarashi, A., Amagasa, S., Horikawa, H., & Shirahata, M. (2002). Vecuronium directly inhibits hypoxic neurotransmission of the rat carotid body. *Anesthesia and analgesia*, *94*(1).

Background

Vecuronium Bromide is a non-depolarizing neuromuscular blocking agent that specifically acts on the muscle-type acetylcholine receptors, with an IC_{50} of 1-2nM[1]. Vecuronium Bromide blocks the transmission between the nerve endings and skeletal muscle by competing with acetylcholine for the nicotinic receptors located at the motor endplates of skeletal muscle[2]. Vecuronium Bromide is commonly used in the study of neuromuscular transmission mechanisms and related drug interactions[3-5].

In vitro, Vecuronium Bromide (0-100μM; 15min) inhibits [^{3}H] norepinephrine (NE) uptake to 65% at 100μM in adrenal medullary cells[2]. Treatment of A549 lung cancer cells with Vecuronium Bromide (0-15μM; 72h) inhibits cell proliferation and migration in a concentration-dependent manner and significantly reduces cell viability by combining with cisplatin[6].

In vivo, Vecuronium Bromide (0.1, 0.5, or 5μM; Intravenous injection; every 30min for 2h) attenuates the response of carotid sinus nerve activity (CSNA) to hypoxia in a dose-dependent manner and inhibits the neural response of the carotid body to acetylcholine (ACh) in Wister rats[7].

References:
[1] Kindler, C. H., Verotta, D., Gray, A. T., Gropper, M. A., & Yost, C. S. (2000). Additive inhibition of nicotinic acetylcholine receptors by corticosteroids and the neuromuscular blocking drug vecuronium. *Anesthesiology*, *92*(3), 821–832.
[2] Uryu, K., Minami, K., Yanagihara, N., Hara, K., Toyohira, Y., Izumi, F., & Shigematsu, A. (2000). Inhibition by neuromuscular blocking drugs of norepinephrine transporter in cultured bovine adrenal

medullary cells. *Anesthesia and analgesia*, *91*(3), 546–551.
[3] Shingu, K., Masuzawa, M., Omote, K., Namiki, A., Kikuchi, H., Kawamada, M., Sato, S., Kimura, T., Hatano, N., Nakatsuka, H., Morita, K., Hara, T., Kanmura, Y., & Takeda, J. (2006). *Masui. The Japanese journal of anesthesiology*, *55*(9), 1140–1148.
[4] Agoston, S., Salt, P., Newton, D., Bencini, A., Boomsma, P., & Erdmann, W. (1980). The neuromuscular blocking action of ORG NC 45, a new pancuronium derivative, in anaesthetized patients. A pilot study. *British journal of anaesthesia*, *52 Suppl 1*, 53S–59S.
[5] Meretoja O. A. (1989). Vecuronium infusion requirements in pediatric patients during fentanyl-N2O-O2 anesthesia. *Anesthesia and analgesia*, *68*(1), 20–24.
[6] Iddrisu BabaYabasin, et al. Anticancer effects of vecuronium bromide and cisatracurium besylate on lung cancer cells (A549), in vitro. Biomedicine & Aging Pathology,Volume 4, Issue 4.
[7] Igarashi, A., Amagasa, S., Horikawa, H., & Shirahata, M. (2002). Vecuronium directly inhibits hypoxic neurotransmission of the rat carotid body. *Anesthesia and analgesia*, *94*(1).