Product Data Sheet

Product Name: AR-R 17779 hydrochloride
Cat. No.: GC11060

Chemical Properties

Cas. No.	178419-42-6		
Chemical Name	(1'R,3'S,4'R)-1'-azaspiro[oxazolidine-5,3'-bicyclo[2.2.2]octan]-2-one hydrochloride		
SMILES	O=C(NC1)O[C@]21[C@H](CC3)CC[N@]3C2.Cl		
Formula	$C_9H_{14}N_2O_2$.HCl	M.Wt	218.68
Solubility	<10.93mg/ml in DMSO; <21.87mg/ml in Water	Storage	Desiccate at RT
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure	AR-R 17779 hydrochloride		

Background

AR-R 17779 is a selective agonist of α7 nicotinic acetylcholine receptor (α7-nAChR) [1] with an EC50 of 21 μM to rat α7-nAChRs expressed in Xenopus oocytes [2].

Nicotine enhances cognitive functions, e.g. learning, attention, retention and memory in both humans and animals, via activation of brain nicotinic acetylcholine receptors (nAChRs). These receptors are homo- or heteropentameric ligand-gated ion channels. The most common nicotinic receptors found in the brain are the α4β2-nAChR and the α7-nAChR [3].

The expression of CD38, CD138, and Bcl-6, was sensitive to regulation via nAChRs. Daudi cells exposed to AR-R 17779 ± methyllycaconitine (MLA) resulted in only moderate changes in the gene expression of CD38, CD138 and Bcl-6, but AR-R 17779 alone significantly ($P< 0.05$) increased protein levels of CD38 and CD138. That means the effect of AR-R 17779 was abolished by MLA [4].

Caution: Product has not been fully validated for medical applications. For research use only.
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Cholesterol is necessary for the homeostasis of acetylcholine receptor (AChR) levels for ion translocation and at the plasmalemma [5]. In ApoE-deficient mice, AR-R 17779 significantly reduced atherosclerotic plaque area in the thoracic aorta, and lowered heart rate, blood pressure, serum triglyceride level and serum total cholesterol level compared with which in Ang II + HFD mice. Treatment with AR-R 17779 in mice did not result in any sickness behavior or apparent abnormalities. At the end of the experiment, the serum concentration of AR-R 17779 was 1.18 ± 0.17 μM. In ApoE-deficient mice, treatment with AR-R 17779 resulted in significantly reduced aortic diameter comparable to control mice (0.81 ± 0.11 mm, $p < 0.0001$ vs. Ang II + HFD) [1].

References:

[1]. Toru Hashimoto, Toshihiro Ichiki, AyaWatanabe, et al. Stimulation of α7 nicotinic acetylcholine receptor by AR-R17779 suppresses atherosclerosis and aortic aneurysm formation in apolipoprotein E-deficient mice. Vascular Pharmacology, 2014, 61:49-55.
[2]. Rudy Schreiber, Marion Dalmus and Jean De Vry. Effects of α4/β2- and α7-nicotine acetylcholine receptor agonists on prepulse inhibition of the acoustic startle response in rats and mice. Psychopharmacology, 2002, 159:248-257.
[3]. Marja van Kampen, Karin Selbach, Renate Schneider, et al. AR-R 17779 improves social recognition in rats by activation of nicotinic α7 receptors. Psychopharmacology, 2004, 172:375-383.
[4]. Juan Arredondo, Denys Omelchenko, Alexander I Chernyavsky, et al. Functional role of the nicotinic arm of the acetylcholine regulatory axis in human B-cell lines. Journal of Experimental Pharmacology, 2009, 1:1-7.
[5]. Virginia Borroni and Francisco J. Barrantes. Cholesterol Modulates the Rate and Mechanism of Acetylcholine Receptor Internalization. J. Biol. Chem., 2011, 286(19):17122-32.

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