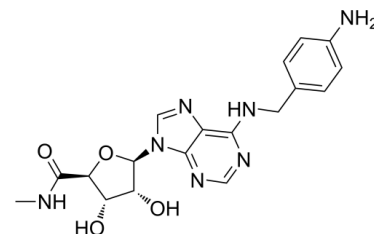


## Data Sheet

<b>Product Name:</b>	AB-MECA
<b>Cat. No.:</b>	CS-5220
<b>CAS No.:</b>	152918-26-8
<b>Molecular Formula:</b>	C <sub>18</sub> H <sub>21</sub> N <sub>7</sub> O <sub>4</sub>
<b>Molecular Weight:</b>	399.40
<b>Target:</b>	Adenosine Receptor
<b>Pathway:</b>	GPCR/G Protein
<b>Solubility:</b>	DMSO : 55 mg/mL (137.71 mM; Need ultrasonic)



### BIOLOGICAL ACTIVITY:

AB-MECA is a high affinity **A<sub>3</sub> adenosine receptor** agonist with a binding **K<sub>i</sub>** of 430.5 nM for human A<sub>3</sub> receptors in CHO cells. AB-MECA can enhance plasma histamine level<sup>[1][2][3][4]</sup>. *In Vitro*: AB-MECA (1, 10, 100 μM; 24 hours) shows dose-dependent cytotoxicity in human lung cancer cell line A549<sup>[2]</sup>.

[<sup>125</sup>I]AB-MECA has K<sub>D</sub> values for binding to A<sub>3</sub> receptors in transfected CHO cells and in RBL-2H3 cells are 1.48 and 3.61 nM, respectively<sup>[3]</sup>.

*In Vivo*: AB-MECA (3 ug/kg; iv) enhances plasma histamine level in mouse<sup>[4]</sup>.

AB-MECA (0.3 mg/kg; iv) enhances antigen-induced bronchoconstriction in male albino guinea pigs, weighing 180-220 g<sup>[5]</sup>.

### PROTOCOL (Extracted from published papers and Only for reference)

Enzyme assay [1] Membrane pellets were resuspended in 20 volumes of assay buffer containing 4 U/ml ADA and kept on ice. Most binding reactions were initiated by combination of 100 μl membrane suspension (at ca. 1 μg/ml protein) with 50 μl binding buffer containing [<sup>125</sup>I]AB-MECA and 50 μl buffer or competitor drug; the final ADA concentration was 2 U/ml. Studies to characterize [<sup>125</sup>I]AB-MECA binding in cerebellar membranes were conducted using half-volumes (e.g., all concentrations the same, but total reaction volume was 100 μl). Reactions were incubated in a water bath for 90 min at 25°C. Except as noted, the final concentration of [<sup>125</sup>I]AB-MECA was 400 pM. Non-specific binding was determined in the presence of 10 μM NECA. Binding reactions were terminated by vacuum filtration through Whatman GF/B filters, using a Millipore filtration manifold. Filters were washed 3×5 ml with ice-cold buffer lacking ADA, dried, and counted in an ICN Biomedical model 4/600 gamma-counter at 70% counting efficiency. Preliminary studies indicated that wetting filters with 0.1% polyethylenimine or using 200 μM NECA (rather than 10 μM) to define non-specific binding did not reduce the level of non-specific binding. Protein concentrations were measured using a Bio-Rad microassay procedure with bovine serum albumin as standard.

### References:

- [1]. L Yates, et al. Radioligand binding and functional responses of ligands for human recombinant adenosine A(3) receptors. *Auton Autacoid Pharmacol.* 2006 Apr;26(2):191-200.
- [2]. Solanki, N. D., et al. In Vitro Evaluation Of Anti-Cancer Potential Of A3 Adenosine Receptor Agonist On A549 Human Lung Cancer Cell Line. *Int J Pharm Pharm Sci* ; 2019 Jun; 11(6): 106-108.

[3]. X D Ji, et al. A selective agonist affinity label for A3 adenosine receptors. Biochem Biophys Res Commun. 1994 Aug 30;203(1):570-6.

[4]. Endre G Mikus, et al. Interaction of SSR161421, a novel specific adenosine A(3) receptor antagonist with adenosine A(3) receptor agonists both in vitro and in vivo. Eur J Pharmacol. 2013 Jan 15;699(1-3):62-6.

[5]. Endre G Mikus, et al. Evaluation of SSR161421, a novel orally active adenosine A3 receptor antagonist on pharmacology models. Eur J Pharmacol. 2013 Jan 15;699(1-3):172-9.

#### CAIndexNames:

$\beta$ -D-Ribofuranuronamide, 1-[6-[[[(4-aminophenyl)methyl]amino]-9H-purin-9-yl]-1-deoxy-N-methyl-

#### SMILES:

O[C@H]1[C@H](N2C=NC3=C(NCC4=CC=C(N)C=C4)N=CN=C23)O[C@H](C(NC)=O)[C@H]1O

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

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