# MRS 1523

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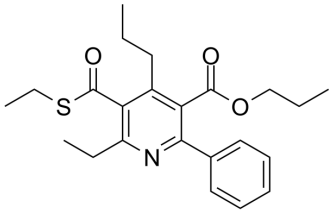
| **Cat. No.:** | HY-121119 |
| **CAS No.:** | 212329-37-8 |
| **Molecular Formula:** | $C_{23}H_{29}NO_3S$ |
| **Molecular Weight:** | 399.55 |
| **Target:** | Adenosine Receptor; Calcium Channel |
| **Pathway:** | GPCR/G Protein; Membrane Transporter/Ion Channel; Neuronal Signaling |
| **Storage:** | Pure form -20°C 3 years<br>4°C 2 years<br>In solvent -80°C 6 months<br>-20°C 1 month |



## BIOLOGICAL ACTIVITY

**Description**

MRS 1523 is a potent and selective adenosine $A_3$ receptor antagonist with $K_i$ values of 18.9 nM and 113 nM for human and rat $A_3$ receptors, respectively. In rat this corresponds to selectivities of 140- and 18-fold vs $A_1$ and $A_{2A}$ receptors, respectively. MRS 1523 can exert antihyperalgesic effect through N-type Ca channel block and action potential inhibition in isolated rat dorsal root ganglion (DRG) neurons[1][2].

**$IC_{50}$ & Target**

Ki: 18.9 nM (Human $A_3$ receptor), 113 nM (Rat $A_3$ receptor), 15.6 μM (A1 receptor) and 2.05 μM (A2A receptor)[1]

**In Vitro**

MRS 1523 (0.1-1 μM) treatment significantly antagonizes cell numbers to 40.7% and 57.4% of the control values, respectively, 30 min before the addition of cordycepin (60 μM). MRS1523 (1 μM) alone has any effect on tumor cell growth[3]. A partial blockade of the adenosine-5'-N-ethylcarboxamide (NECA)-induced migration is observed when human endothelial progenitor cells (hEPC) are co-incubated with MRS 1523 (1 nM). Furthermore, in 3-days hEPC, the treatment with MRS 1523 100 nM inhibits the NECA-induced migration by 70%. NECA-induced migration is blocked in dose-response fashion by MRS 1523 with calculated $K_i$ of 147 nM[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[3]

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| Cell Line: | B16-BL6 cells |
| Concentration: | 0.1 μM, 1 μM |
| Incubation Time: | 24 hours, 48 hours, 72 hours |
| Result: | Antagonized the growth suppression induced by cordycepin. |

**In Vivo**

The expression and functional effects of A3 adenosine receptor (A3AR) on the excitability of small- to medium-sized, capsaicin-sensitive, dorsal root ganglion (DRG) neurons isolated from 3- to 4-week-old rats are investigated. The endogenous agonist adenosine reduces N-type Ca currents, and its effect is inhibited by 56% in the presence of A3AR antagonist MRS 1523. Current-clamp recordings demonstrated that neuronal firing of rat DRG neurons was also significantly reduced by A3AR activation in a MRS 1523-sensitive but PD173212-insensitive manner[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## CUSTOMER VALIDATION

- Purinergic Signal. 2021 Oct 28.

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## REFERENCES


[1]. Li AH, et al. Structure-activity relationships and molecular modeling of 3, 5-diacyl-2,4-dialkylpyridine derivatives as selective A3 adenosine receptor antagonists.J Med Chem. 1998 Aug 13;41(17):3186-201.

[2]. Coppi E, et al. Adenosine A3 receptor activation inhibits pronociceptive N-type Ca2+ currents and cell excitability in dorsal root ganglion neurons.Pain. 2019 May;160(5):1103-1118.

[3]. Fernandez P, et al. Adenosine $A_2A$ and $A_3$ receptors are involved in the human endothelial progenitor cells migration. J Cardiovasc Pharmacol. 2012 May;59(5):397-404.

[4]. Yoshikawa N, et al. Cordycepin (3'-deoxyadenosine) inhibits the growth of B16-BL6 mouse melanoma cells through the stimulation of adenosine A3 receptor followed by glycogen synthase kinase-3beta activation and cyclin D1 suppression. Naunyn Schmiedebergs Arch Pharmacol. 2008 Jun;377(4-6):591-5.





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