# AZD1981

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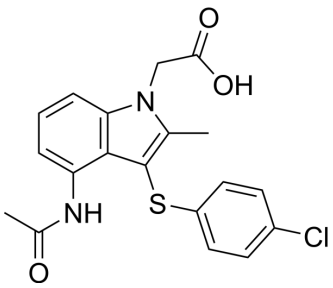
| **Cat. No.:** | HY-15950 |
| **CAS No.:** | 802904-66-1 |
| **Molecular Formula:** | $C_{19}H_{17}ClN_2O_3S$ |
| **Molecular Weight:** | 388.87 |
| **Target:** | Prostaglandin Receptor |
| **Pathway:** | GPCR/G Protein |
| **Storage:** | Powder -20°C 3 years<br>4°C 2 years<br>In solvent -80°C 2 years<br>-20°C 1 year |



## SOLVENT & SOLUBILITY

**In Vitro**

DMSO : 100 mg/mL (257.16 mM; Need ultrasonic)

| Preparing Stock Solutions | Solvent / Mass / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|---|
| | 1 mM | 2.5716 mL | 12.8578 mL | 25.7155 mL |
| | 5 mM | 0.5143 mL | 2.5716 mL | 5.1431 mL |
| | 10 mM | 0.2572 mL | 1.2858 mL | 2.5716 mL |

Please refer to the solubility information to select the appropriate solvent.

**In Vivo**

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.43 mM); Clear solution

## BIOLOGICAL ACTIVITY

**Description**

AZD1981 is a potent and selective CRTh2 antagonist; displaces radio-labelled PGD2 from human recombinant DP2 with high potency (pIC50 = 8.4).IC50 value:Target: GPR44 antagonistin vitro: AZD1981 produced a concentration-dependent displacement of the [3H]PGD2-specific binding with a mean pIC50 of 8.4 ± 0.1 (n = 25, geometric mean IC50 of 4 nM). AZD1981 had no significant affinity towards recombinant human DP1 receptors with only a mean 27% (range 14–50%; n = 4) displacement of [3H]PGD2-specific binding observed at the highest concentration tested (10 μM). Compared with the binding potency for DP2, AZD1981 showed 10-fold selectivity over rat aldose reductase and 1700-fold selectivity over rat steroid 5α-reductase.In eosinophils, a single concentration of 1 μM, AZD1981 caused a large (20-fold) rightward parallel shift in the 15R-methyl PGD2 E/[A] curve with no evidence of a decrease in the maximal response. The effect of AZD1981 was therefore investigated using a single sub-maximal concentration of agonist (1 μM). AZD1981 produced a concentration-dependent inhibition of eosinophil migration with a pIC50 value of 7.6 ± 0.1 (n = 4) [1].in vivo: Using the previously described guinea pig hind limb model , 10 nM AZD1981 significantly inhibited DK-PGD2-induced eosinophil mobilization by approximately 50%, and the response was completely inhibited with 100 nM AZD1981 [1]. in vivo: AZD1981 exhibited good

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| | cross-species binding activity against mouse, rat, guinea pig, rabbit and dog DP2 . Evaluation in mouse, rat or rabbit cell systems was not possible as they did not respond to DP2 agonists. Agonist responses were seen in guinea pig and dog, and AZD1981 blocked DP2 -mediated eosinophil shape change. Such responses were more robust in the guinea pig, where AZD1981 also blocked DP2 -dependent eosinophil emigration from bone marrow [1]. There was no beneficial clinical effect of AZD1981, at a dose of 1000 mg twice daily for 4 weeks, in patients with moderate to severe COPD. AZD1981 was well tolerated and no safety concerns were identified [3]. |
| **$IC_{50}$ & Target** | DP |

## CUSTOMER VALIDATION

- Cell Death Discov. 2023 Feb 1;9(1):39.

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## REFERENCES


[1]. Royer JF, et al. A novel antagonist of prostaglandin D2 blocks the locomotion of eosinophils and basophils. Eur J Clin Invest. 2008 Sep;38(9):663-71.

[2]. Luker T, et al. Substituted indole-1-acetic acids as potent and selective CRTh2 antagonists-discovery of AZD1981. Bioorg Med Chem Lett. 2011 Nov 1;21(21):6288-92.

[3]. Snell N, et al. Efficacy and safety of AZD1981, a CRTH2 receptor antagonist, in patients with moderate to severe COPD. Respir Med. 2013 Nov;107(11):1722-30.





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