# Evatanepag

**Cat. No.:** HY-14839

**CAS No.:** 223488-57-1

**Molecular Formula:** $C_{25}H_{28}N_2O_5S$

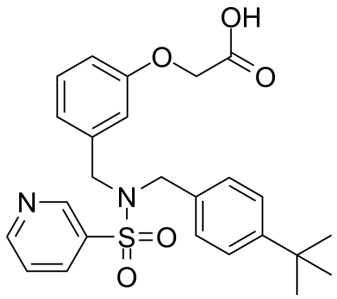
**Molecular Weight:** 468.57

**Target:** Prostaglandin Receptor

**Pathway:** GPCR/G Protein

**Storage:**

| | | |
|---|---|---|
| Powder | -20°C | 3 years |
| | 4°C | 2 years |
| In solvent | -80°C | 2 years |
| | -20°C | 1 year |



## SOLVENT & SOLUBILITY

**In Vitro**

DMSO : ≥ 32 mg/mL (68.29 mM)

* "≥" means soluble, but saturation unknown.

**Preparing Stock Solutions**

| Mass / Solvent / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|
| 1 mM | 2.1342 mL | 10.6708 mL | 21.3415 mL |
| 5 mM | 0.4268 mL | 2.1342 mL | 4.2683 mL |
| 10 mM | 0.2134 mL | 1.0671 mL | 2.1342 mL |

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

## BIOLOGICAL ACTIVITY

**Description**

Evatanepag (CP-533536) is a non-prostanoid, potent and selective $EP_2$ receptor agonist. Evatanepag can induce local bone formation in vivo. Evatanepag can be used in the research of fractures, bone defects, asthma[1][2].

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

EP2

**In Vitro**

Evatanepag (10 nM, 30 min) inhibits hFcεRI-induced mast cells degranulation in a dose-dependent manner[2].

Evatanepag (0.1 nM-10 μM, 12 min) results in an equivalent increase in intracellular cAMP in HEK-293 cells, with an $IC_{50}$ of 50 nM[3].

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

**In Vivo**

Evatanepag (0.3-3.0 mg/kg, directly injected into the marrow cavity of the tibia) promotes bone formation in rats[1].

Evatanepag (0.3, 3.0 mg/kg, intranasal administration, from day1 to day4) reduces HDM aeroallergen-induced increased RL response to methacholine in mice[2].

Evatanepag (1 mg/kg, intravenous injection) demonstrates high i.v. clearance (Cl: 56 mL/min/kg) and a short half-life ($t_{1/2}$:

0.33 h)[1].

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

| | |
|---|---|
| Animal Model: | Rats[1] |
| Dosage: | 0.3, 1.0, 3.0 mg/kg |
| Administration: | Directly injected into the marrow cavity of the tibia |
| Result: | Dose-dependently increased in bone area, bone mineral content, bone mineral density. |

| | |
|---|---|
| Animal Model: | HDM (house dust mite)-sensitized BALB/c mice[2] |
| Dosage: | 0.3 mg/kg, 3 mg/kg |
| Administration: | Intranasal administration, from day1 to day4 |
| Result: | Prevented aeroallergen-driven increased RL (lung resistance) at 0.3 mg/kg.<br>Prevented the enhanced MC activity by approximately 48% at 3 mg/kg. |

## CUSTOMER VALIDATION

- Sci Adv. 2021 Apr 2;7(14):eabf1268.

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## REFERENCES


[1]. Judith Plaza, et al. In Vitro and In Vivo Validation of EP2-Receptor Agonism to Selectively Achieve Inhibition of Mast Cell Activity. Allergy Asthma Immunol Res. 2020 Jul;12(4):712-728.

[2]. V M Paralkar, et al. An EP2 receptor-selective prostaglandin E2 agonist induces bone healing. Proc Natl Acad Sci U S A. 2003 May 27;100(11):6736-40.

[3]. Cameron KO, et al. Discovery of CP-533536: an EP2 receptor selective prostaglandin E2 (PGE2) agonist that induces local bone formation. Bioorg Med Chem Lett. 2009 Apr 1;19(7):2075-8.





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