# Vatalanib succinate

**Cat. No.:** HY-110272

**CAS No.:** 212142-18-2

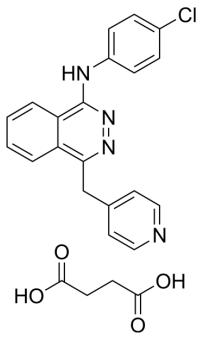
**Molecular Formula:** $C_{24}H_{21}ClN_4O_4$

**Molecular Weight:** 464.9

**Target:** VEGFR

**Pathway:** Protein Tyrosine Kinase/RTK

**Storage:** Please store the product under the recommended conditions in the Certificate of Analysis.




## BIOLOGICAL ACTIVITY

| | | | |
|---|---|---|---|
| **Description** | Vatalanib (PTK787) succinate is a potent and orally active VEGFR inhibitor with $IC_{50}$s of 37 nM, 77 nM, 270 nM, 660 nM, 730 nM, 1400 nM, and 580 nM for KDR, Flt-1, Flk, Flt-4, c-Kit, c-Fms, and PDGFR-β, respectively[1]. | | |
| **$IC_{50}$ & Target** | KDR<br>37 nM ($IC_{50}$) | Flt-1<br>77 nM ($IC_{50}$) | Flt-4<br>730 nM ($IC_{50}$) |
| **In Vitro** | Vatalanib (PTK787) inhibits VEGF-induced autophosphorylation of kinase insert domain-containing receptor (KDR), endothelial cell proliferation, migration, and survival in the nanomolar range in cell-based assays. In concentrations up to 1 μM, Vatalanib (PTK787) does not have any cytotoxic or antiproliferative effect on cells that do not express VEGF receptors[1].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. | | |
| **In Vivo** | After oral dosing (50 mg/kg) to mice, plasma concentrations of Vatalanib (PTK787) remain above 1 μM for more than 8 h. Vatalanib (PTK787) induces dose-dependent inhibition of VEGF and PDGF-induced angiogenesis in a growth factor implant model, as well as a tumor cell-driven angiogenesis model after once-daily oral dosing (25-100 mg/kg)[1].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. | | |

## CUSTOMER VALIDATION

- Bioact Mater. 2 January 2022.
- Sci Transl Med. 2018 Jul 18;10(450):eaaq1093.
- J Pharm Anal. 2023 Sep 11.
- Br J Pharmacol. 2019 Sep;176(17):3143-3160.
- Oncol Rep. 2016 Mar;35(3):1297-308.

See more customer validations on www.MedChemExpress.com

## REFERENCES

[1]. J M Wood, et al. PTK787/ZK 222584, a novel and potent inhibitor of vascular endothelial growth factor receptor tyrosine kinases, impairs vascular endothelial growth factor-induced responses and tumor growth after oral administration. Cancer Res. 2000 Apr 15;60(8):2178-89.



Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA