

## Product Data Sheet

Product Name: IPR-803

Cat. No.: GC39713

### Chemical Properties

Cas. No. 892243-35-5

SMILES O=C(O)C1=CC=CC(NC2=C(C3=C4ON=C3C(N5CCCCC5)=C2)C(C6=C4C=CC=C6)=O)=C1

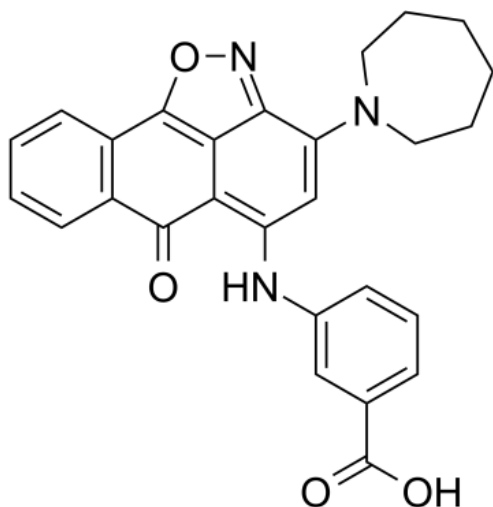
Formula  $C_{27}H_{23}N_3O_4$  M.Wt 453.49

Solubility Soluble in DMSO Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



### Protocol

#### Cell experiment [1]:

Cell lines

MDA-MB-231 cells (human breast cancer cell line)

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

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|-------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Preparation Method            | MDA-MB-231 cells were treated with IPR-803, then invasion through fibronectin-coated transwell, migration by Boyden chamber and wound healing, adhesion to fibronectin/vitronectin, surface uPA/uPAR staining, and proliferation/toxicity by MTT were measured.                                                                                                                                                                                               |
| Reaction Conditions           | 0-100μM; 0.5-16h                                                                                                                                                                                                                                                                                                                                                                                                                                              |
| Applications                  | IPR-803 reduced MDA-MB-231 invasion through membrane, showed weak/no effect on migration and no effect on adhesion, reduced surface uPA binding to uPAR, and caused concentration-dependent reduction in cell proliferation.                                                                                                                                                                                                                                  |
| <b>Animal experiment [2]:</b> |                                                                                                                                                                                                                                                                                                                                                                                                                                                               |
| Animal models                 | Female NSG mice                                                                                                                                                                                                                                                                                                                                                                                                                                               |
| Preparation Method            | TMD-MDA-MB-231 cells were inoculated into the mammary fat pads of female NSG mice. Dosing was initiated at day 15 post implantation. Animals were randomized and treated with vehicle or with IPR-803 by daily oral gavage at a dose of 200mg/kg for 33 days. Tumor volumes were determined by caliper measurements on a twice weekly basis. At the end of the study, animals were sacrificed and lungs were removed for H&E staining and metastasis scoring. |
| Dosage form                   | 200mg/kg; p.o.; daily for 33 days                                                                                                                                                                                                                                                                                                                                                                                                                             |
| Applications                  | IPR-803 impaired breast cancer metastasis to the lungs. IPR-803 had little effect on primary tumor growth and no significant effect on body weight.                                                                                                                                                                                                                                                                                                           |
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References:[1] Khanna M, Wang F, Jo I, et al. Targeting Multiple Conformations Leads to Small Molecule Inhibitors of the uPAR·uPA Protein-Protein Interaction that Block Cancer Cell Invasion. ACS Chem Biol. 2011 Nov 18;6(11):1108-18.[2] Mani T, Wang F, Knabe WE, et al. Small-Molecule Inhibition of the uPAR·uPA Interaction: Synthesis, Biochemical, Cellular, in vivo Pharmacokinetics and Efficacy Studies in Breast Cancer Metastasis. Bioorg Med Chem. 2013 Apr 1;21(7):2145-55.

### Background

IPR-803 is a small molecule inhibitor of urokinase receptor (uPAR). IPR-803 competitively blocks the protein-protein interaction between uPAR and urokinase-type plasminogen activator (uPA). IPR-803 downregulates uPA expression. IPR-803 inhibits MMP-mediated extracellular matrix degradation and p-ERK/MAPK signaling. IPR-803 reduces angiogenesis and metastasis. IPR-803 is used for studying metastasis in breast cancer and pancreatic cancer<sup>[1-4]</sup>.

In vitro, MDA-MB-231 cells were treated with 0-100µM IPR-803 for 0.5-16 hours. IPR-803 inhibited cell invasion and proliferation in a concentration-dependent manner<sup>[5]</sup>. RKO cells were treated with 30µM IPR-803 for 48-6 hours. IPR-803 inhibited phosphorylation of FAK and SLC6A8 proteins<sup>[6]</sup>.

In vivo, female NSG mice with mammary fat pad inoculation of TMD-MDA-MB-231 cells received daily oral gavage of 200mg/kg IPR-803 for 33 days. IPR-803 inhibited breast cancer cell metastasis to the lungs<sup>[7]</sup>. Tumor-bearing nude mice with KYSE-450 cells received oral gavage of 50mg/kg IPR-803 every 3 days for 24 days. IPR-803 inhibited tumor growth<sup>[8]</sup>.

### References:

- [1] Liu D, Zhou D, Wang B, et al. A new class of orthosteric uPAR·uPA small-molecule antagonists are allosteric inhibitors of the uPAR·vitronectin interaction. ACS Chem Biol. 2015 Jun 19;10(6):1521-34.
- [2] Montuori N, Pesapane A, Rossi FW, et al. Urokinase type plasminogen activator receptor (uPAR) as a new therapeutic target in cancer. Transl Med UniSa. 2016 Nov 1;15:15-21.
- [3] Li Santi A, Napolitano F, Montuori N, et al. The Urokinase Receptor: A Multifunctional Receptor in Cancer Cell Biology. Therapeutic Implications. Int J Mol Sci. 2021 Apr 16;22(8):4111.
- [4] Zhou J, Ma S, Feng D, et al. C5aR1 + microglia exacerbate neuroinflammation and cerebral edema in acute brain injury. Neuron. 2026 Feb 4;114(3):444-462.e9.
- [5] Khanna M, Wang F, Jo I, et al. Targeting Multiple Conformations Leads to Small Molecule

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Inhibitors of the uPAR-uPA Protein-Protein Interaction that Block Cancer Cell Invasion. ACS Chem Biol. 2011 Nov 18;6(11):1106-16.

[6] Yu J, Xiu M, Yu S, et al. The Role of CAF-derived Vitronectin in Promoting Colorectal Cancer Progression and Immunosuppression. Adv Sci (Weinh). 2025 Sep;12(33):e05769.

[7] Mani T, Wang F, Knabe WE, et al. Small-Molecule Inhibition of the uPAR-uPA Interaction: Synthesis, Biochemical, Cellular, in vivo Pharmacokinetics and Efficacy Studies in Breast Cancer Metastasis. Bioorg Med Chem. 2013 Apr 1;21(7):2145-55.

[8] Fang L, Che Y, Zhang C, et al. PLAU directs conversion of fibroblasts to inflammatory cancer-associated fibroblasts, promoting esophageal squamous cell carcinoma progression via uPAR/Akt/NF- $\kappa$ B/IL8 pathway. Cell Death Discovery. 2021 Feb 11;7(1):32.

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