

## Product Data Sheet

Product Name: GW296115

Cat. No.: GC81304

### Chemical Properties

Cas. No. 118458-58-5

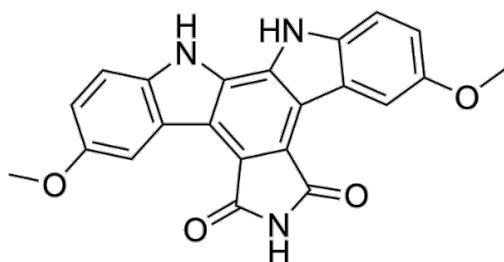
Formula  $C_{22}H_{15}N_3O_4$  M.Wt 385.37

Solubility DMSO: 50 mg/mL (129.75 mM; Need ultrasonic) Storage Store at 4°C, away from moisture

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



### Background

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

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GW296115 is a multi-target inhibitor with the following IC<sub>50</sub> values against its targets: 8.4 nM for BRSK2, 21 nM for BRSK1, 1.8 μM for PDGFRβ, 5.5 nM for STK17B/DRAK2, 28 nM for DRAK1, 20 nM for PHKG1, and 89 nM for DCAMKL3. GW296115 downregulates the phosphorylation of S317 site on ULK1 and S351 site on P62, which are AMPK substrates driven by BRSK2. GW296115 does not alter the phosphorylation level of AMPK at T172, reduces nutrient deprivation-mediated Autophagy and autophagosome formation, and enhances Apoptosis. GW296115 exhibits anticancer activity against triple-negative breast cancer. GW296115 is applicable for breast cancer-related research [1] [2].

In Vitro, GW296115 (1 μM) inhibits 25 out of 403 wild-type human kinases by over 90% at a concentration of 1 μM, with a selectivity index of 0.062<sup>[1]</sup>. GW296115 potently inhibits 6 IDG dark kinases in enzymatic assays, with IC<sub>50</sub> values ranging from 5.5 nM (DRAK2) to 89 nM (DCAMKL3)<sup>[1]</sup>. GW296115 (1 μM; 72 h) exerts no effect on cell proliferation in 17 tested cancer cell lines and normal cell lines (including non-malignant MCF10A and MRC-5) following treatment at 1 μM for 72 h<sup>[1]</sup>. GW296115 (incubated for 2 h) potently binds to BRSK2 in living HEK293 cells with an IC<sub>50</sub> of 107 nM, confirming that it is a cell-active BRSK2 inhibitor<sup>[1]</sup>. GW296115 (2.5 μM; 2-6 h) abolishes BRSK2-induced phosphorylation of AMPK substrates in HEK293T cells (this effect is observed at both 2 h and 6 h) and induces hyperphosphorylation of BRSK2 at the T174 site<sup>[1]</sup>. GW296115 (2-5 μM) inhibits autophagy and enhances apoptosis in MDA-MB-231 triple-negative breast cancer cells, while it also inhibits autophagy and enhances apoptosis in BT-474 estrogen receptor-positive breast cancer cells<sup>[2]</sup>. GW296115 (2 μM; 24 h) inhibits the growth of MDA-MB-231 triple-negative breast cancer cells<sup>[2]</sup>. GW296115 (2-3 μM; 72 h) reduces the 3D invasiveness of MDA-MB-231 triple-negative breast cancer cells<sup>[2]</sup>.

### References:

- [1]. Tamir TY, et al. PKIS deep dive yields a chemical starting point for dark kinases and a cell active BRSK2 inhibitor. Sci Rep. 2020 Sep 28;10(1):15826.
- [2]. Maiti A, et al. BRSK2 plays a role in autophagy and cancer cell growth and survival under nutrient deprivation stress via the PIK3C3 pathway. Sci Rep. 2025 Nov 19;15(1):40651.

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