Product Datasheet

Defactinib

Catalog No. **BP-02315** · CAS **1073154-85-4** · Form **free base** · Physical state **solid** · Color **White to off-white** · Version **1.0** · Revision **2026-09-10**

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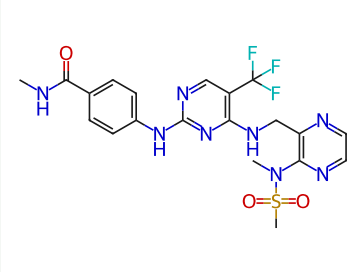
Molecular formula	$C_{20}H_{21}F_3N_8O_3S$
Molecular weight	**510.4930**

STRUCTURE



DESCRIPTION

Defactinib (VS-6063; PF-04554878) is a novel FAK inhibitor with potential antiangiogenic and antineoplastic activities.

BIOLOGICAL ACTIVITY

In Vitro

Defactinib (VS-6063) inhibits FAK phosphorylation at the Tyr397 site in a time- and dose-dependent manner. RPPA data shows that Defactinib reduces levels of AKT and YB-1 in taxane-resistant cell lines. The expression of pFAK (Tyr397) is statistically significantly inhibited by Defactinib in a dose-dependent manner in all cell lines. Defactinib inhibits pFAK (Tyr397) expression within 3 hours, with a gradual return of expression by 48 hours.

In Vivo

Defactinib (VS-6063) doses of 25 mg/kg twice a day or greater statistically significantly inhibits pFAK (Tyr397) at 3 hours, with return of expression noted by 24 hours. Therefore, administration of Defactinib at 25 mg/kg twice a day is selected as the dosing schedule for subsequent therapy experiments. For therapy experiments, female nude mice bearing HeyA8 tumors in the peritoneal cavity are randomly divided into 4 groups (n=10 per group): 1) vehicle orally twice daily and phosphate-buffered saline intraperitoneally weekly (control); 2) Defactinib 25 mg/kg orally twice daily; 3) PTX intraperitoneally weekly; and 4) both VDefactinib 25 mg/kg orally twice daily and PTX intraperitoneally weekly. There is an 87.4% reduction in tumor weight by PTX monotherapy in the HeyA8 model, and combination therapy resulted in the greatest tumor weight reduction, with a 97.9% reduction (P=0.05 compared with PTX). In the SKOV3ip1 model, a 92.7% tumor weight reduction is observed in the combination group compared with PTX (P<0.001).

SOLUBILITY

Soluble in DMSO

REFERENCES

[1]. Kang Y, et al. Role of focal adhesion kinase in regulating YB-1-mediated resistance in ovarian cancer. J Natl Cancer Inst. 2013 Oct 2;105(19):1485-95.

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