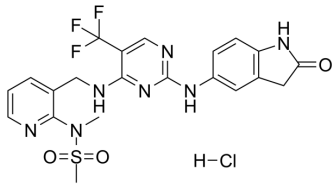
PF-562271 hydrochloride

Cat. No.:	HY-20403
CAS No.:	939791-41-0
Molecular Formula:	$C_{21}H_{21}ClF_3N_7O_3S$
Molecular Weight:	543.95
Target:	FAK; Pyk2
Pathway:	Protein Tyrosine Kinase/RTK
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	PF-562271 (VS-6062) hydrochloride is a potent, ATP-competitive and reversible FAK and Pyk2 kinase inhibitor with IC_{50}s of 1.5 nM and 13 nM, respectively[1].
IC_{50} & Target	IC50: 1.5 nM (FAK), 13 nM (Pyk2), 30 nM (CDK2), 47 nM (CDK3), 58 nM (CDK1), 97 nM (CDK7), 97 nM (Flt3)[1]
In Vitro	PF-562271 (VS-6062) hydrochloride is shown to be a 30- to 120-nM inhibitor of CDK2/E, CDK5/p35, CDK1/B, and CDK3/E in recombinant enzyme assays, in cell-based assays evaluating the role of CDKs, a 48-hour exposure of 3.3 μM PF-562271 is required to alter cell cycle progression. PF-562271 is potent in an inducible cell-based assay measuring phospho-FAK with a IC_{50} of 5 nM[1]. PF-562271, a selective inhibitor of both FAK and proline-rich tyrosine kinase 2 (PYK2), a FAK-related family member, on cell growth and colony formation in Ewing sarcoma cell lines. Seven cell lines are treated for 5 days with PF-562271 across a range of concentrations using 2-fold serial dilutions. Treatment with PF-562271 impaires cell viability in all cell lines, with an average IC_{50} of 2.4 μM after 3 days of treatment. TC32 and A673 are the 2 most sensitive cell lines, with IC_{50} concentrations of 2.1 and 1.7 μM, respectively[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	PF-562271 inhibits FAK phosphorylation in vivo in a dose-dependent fashion (calculated EC_{50} of 93 ng/mL, total) after p.o. administration to tumor-bearing mice[1]. Rats that receive PF-562271 demonstrate a decrease in tumor growth after 2 weeks of treatment with signs of bone healing as evidenced by the deposition of new bone (cortical and cancellous) at sites previously damaged by the tumor[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Clin Cancer Res. 2019 Jul 15;25(14):4552-4566.
- Cancer Res. 2013 May 1;73(9):2873-83.
- Int J Cancer. 2015 Oct 1;137(7):1549-59.
- Sci Rep. 2018 May 8;8(1):7228.
- Life Sci. 2021 Jan 25;119112.

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REFERENCES

[1]. Roberts WG, et al. Antitumor activity and pharmacology of a selective focal adhesion kinase inhibitor, PF-562,271. Cancer Res, 2008, 68(6), 1935-1944.

[2]. Crompton BD, et al. High-throughput tyrosine kinase activity profiling identifies FAK as a candidate therapeutic target in Ewing sarcoma. Cancer Res. 2013 May 1;73(9):2873-83.

[3]. Bagi CM, et al. Dual focal adhesion kinase/Pyk2 inhibitor has positive effects on bone tumors: implications for bone metastases. Cancer. 2008 May 15;112(10):2313-21.

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