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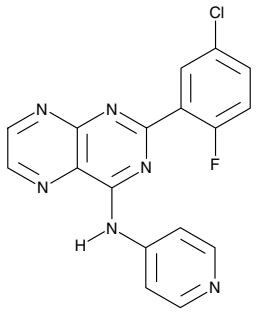
Product Information

SD 208

Item No. 16619

CAS Registry No.:	627536-09-8
Formal Name:	2-(5-chloro-2-fluorophenyl)-N-4-pyridinyl-4-pteridinamine
Synonym:	TGF-β RI Kinase Inhibitor V
MF:	$C_{17}H_{10}ClFN_6$
FW:	352.8
Purity:	≥98%
Stability:	≥2 years at -20°C
Supplied as:	A crystalline solid
UV/Vis.:	λ_{max}: 205, 258, 362 nm



Laboratory Procedures

For long term storage, we suggest that SD 208 be stored as supplied at -20°C. It should be stable for at least two years.

SD 208 is supplied as a crystalline solid. A stock solution may be made by dissolving the SD 208 in the solvent of choice. SD 208 is soluble in dimethyl formamide at a concentration of approximately 0.3 mg/ml.

SD 208 is sparingly soluble in aqueous solutions. To enhance aqueous solubility, dilute the organic solvent solution into aqueous buffers or isotonic saline. If performing biological experiments, ensure the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. We do not recommend storing the aqueous solution for more than one day.

TGF-β is a cell growth and differentiation factor that has roles in cancer, fibrosis, and numerous other pathologies.[1,2] TGF-β signals through two receptor tyrosine kinases, TGF-βRI and TGF-βRII. SD 208 is a potent inhibitor of TGF-βRI kinase (EC_{50} = 48 nM) that has minimal or no effect at a variety of other tyrosine or serine/threonine kinases, including TGF-βRII kinase.[3] It blocks both autocrine and paracrine TGF-β signaling in glioma cells, inhibiting TGF-β-induced migration and invasion without affecting viability or proliferation.[3] SD 208 is orally bioavailable and prevents TGF-β-induced Smad phosphorylation in spleens and brains of mice.[3] It improves survival in mice with xenografted glioma by heightening the immune response against tumor cells and reverses bronchial hyperresponsiveness to allergens in ovalbumin-exposed mice.[3,4] SD 208 also suppresses TGF-β-induced differentiation of proliferating myofibroblasts and induces dedifferentiation in the absence of TGF-β.[5,6]

References

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2. Distler, J.H.W. and Distler, O. *Rheumatology* **47**, 10-11 (2008).
3. Uhl, M., Aulwurm, S., Wischhusen, J., *et al. Cancer Res.* **64(21)**, 7954-7961 (2004).
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5. Driesen, R.B., Nagaraju, C.K., Abi-Char, J., *et al. Cardiovasc. Res.* **101(3)**, 411-422 (2014).
6. Kapoun, A.M., Gaspar, N.J., Wang, Y., *et al. Mol. Pharmacol.* **70(2)**, 518-531 (2006).

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