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Produktinformation

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- Diagnostik & molekulare Diagnostik
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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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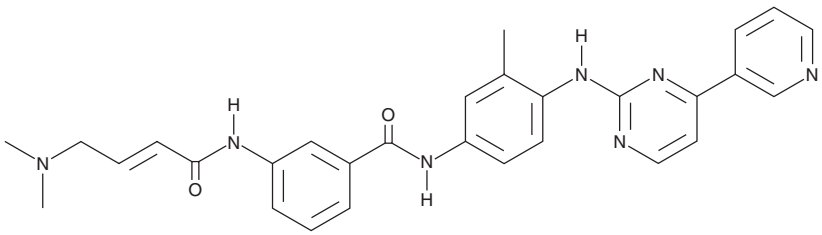
PRODUCT INFORMATION

JNK Inhibitor XVI

Item No. 18096

CAS Registry No.: 1410880-22-6
Formal Name: 3-[[4-(dimethylamino)-1-oxo-2-buten-1-yl]amino]-N-[3-methyl-4-[[4-(3-pyridinyl)-2-pyrimidinyl]amino]phenyl]-benzamide
Synonyms: JNK-IN-8, c-Jun N-terminal Kinase Inhibitor XVI
MF: $C_{29}H_{29}N_7O_2$
FW: 507.6
Purity: ≥95%
UV/Vis.: λ_{max}: 277 nm
Supplied as: A crystalline solid
Storage: -20°C
Stability: As supplied, 2 years from the QC date provided on the Certificate of Analysis, when stored properly



Laboratory Procedures

JNK inhibitor XVI is supplied as a crystalline solid. A stock solution may be made by dissolving the JNK inhibitor XVI in the solvent of choice. JNK inhibitor XVI is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of JNK inhibitor XVI in these solvents is approximately 2, 16, and 20 mg/ml, respectively.

JNK inhibitor XVI is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, JNK inhibitor XVI should first be dissolved in DMF and then diluted with the aqueous buffer of choice. JNK inhibitor XVI has a solubility of approximately 0.5 mg/ml in a 1:1 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

c-Jun N-terminal kinases (JNKs) are MAP kinase family members that become highly activated after cells are exposed to stress conditions and are poorly activated by exposure to growth factors or mitogens. JNK inhibitor XVI is a selective, irreversible JNK inhibitor (IC_{50}s = 4.67, 18.7, and 0.98 nM for JNK1, 2, and 3, respectively) that prevents phosphorylation of c-Jun in A375 and HeLa cells with EC_{50} values of 338 and 486 nM, respectively.[1] It has been shown to inhibit JNK kinase activity by a mechanism that depends on covalent modification of cysteine^{116} in the ATP-binding motif.[1] This compound has been used to explore the role of JNK in mediating cancer cell death.[2,3]

References

1. Zhang, T., Inesta-Vaquero, F., Niepel, M., *et al.* Discovery of potent and selective covalent inhibitors of JNK. *Chem. Biol.* **19(1)**, 140-154 (2012).
2. Li, Q., Song, X., Ji, Y., *et al.* The dual mTORC1 and mTORC2 inhibitor AZD8055 inhibits head and neck squamous cell carcinoma cell growth *in vivo* and *in vitro*. *Biochem. Bioph. Res. Commun.* **440(4)**, 701-706 (2013).
3. Fallahi-Sichani, M., Moerke, N.J., Niepel, M., *et al.* Systematic analysis of $BRAF^{V600E}$ melanomas reveals a role for JNK/c-Jun pathway in adaptive resistance to drug-induced apoptosis. *Mol. Syst. Biol.* **11**, 797 (2015).

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