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Produktinformation

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- Zellkultur & Verbrauchsmaterial
- Diagnostik & molekulare Diagnostik
- Laborgeräte & Service

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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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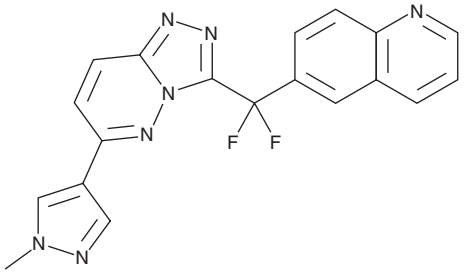
PRODUCT INFORMATION

JNJ-38877605

Item No. 27650

CAS Registry No.:	943540-75-8
Formal Name:	6-[difluoro[6-(1-methyl-1H-pyrazol-4-yl)-1,2,4-triazolo[4,3-b]pyridazin-3-yl]methyl]-quinoline
MF:	$C_{19}H_{13}F_2N_7$
FW:	377.4
Purity:	≥98%
UV/Vis.:	λ_{max}: 227, 253, 302 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥2 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

JNJ-38877605 is supplied as a crystalline solid. A stock solution may be made by dissolving the JNJ-38877605 in the solvent of choice, which should be purged with an inert gas. JNJ-38877605 is soluble in the organic solvent chloroform at a concentration of approximately 30 mg/ml.

Description

JNJ-38877605 is an ATP-competitive inhibitor of Met kinase (IC_{50} = 4 nM).[1] It is at least 600-fold selective for Met in a panel of approximately 250 tyrosine and serine-threonine kinases. JNJ-38877605 inhibits the growth of cancer cell lines with *MET* gene amplification (IC_{50}s = 11-50 nM) but has no effect on the growth of control cells with normal *MET* gene copy number or no *MET* expression.[2] The efficacy of JNJ-38877605 against *MET*-amplified cells decreases in the presence of increasing concentrations of recombinant human hepatocyte growth factor (HGF). JNJ-38877605 (50 mg/kg) sensitizes tumors to radiotherapy in U251 glioma and MDA-MB-231 breast cancer mouse xenograft models and increases apoptosis in irradiated tumors in the MDA-MB-231 mouse xenograft model.[3] It reduces tumor size by 6-fold and the number of blood vessels in tumors by 80% in an RU-P melanoma mouse xenograft model when administered at a dose of 20 mg/kg.[4]

References

1. Perera, T., Lavrijssen, T., Janssens, B., *et al.* JNJ-38877605: A selective Met kinase inhibitor inducing regression of Met-driven tumor models. *Cancer Res.* **68(9Supp.)**, (2008).
2. Pennacchietti, S., Cazzanti, M., Bertotti, A., *et al.* Microenvironment-derived HGF overcomes genetically determined sensitivity to anti-MET drugs. *Cancer Res.* **74(22)**, 6598-6609 (2014).
3. De Bacco, F., Luraghi, P., Medico, E., *et al.* Induction of MET by ionizing radiation and its role in radioresistance and invasive growth of cancer. *J. Natl. Cancer Inst.* **103(8)**, 645-661 (2011).
4. Etnyre, D., Stone, A.L., Fong, J.T., *et al.* Targeting c-Met in melanoma: Mechanism of resistance and efficacy of novel combinatorial inhibitor therapy. *Cancer Biol. Ther.* **15(9)**, 1129-1141 (2014).

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