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Produktinformation

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- Diagnostik & molekulare Diagnostik
- Laborgeräte & Service

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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

mail@szabo-scandic.com

www.szabo-scandic.com

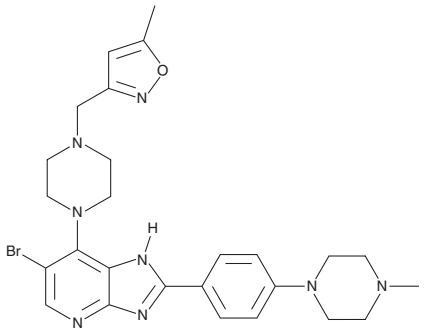
linkedin.com/company/szaboscandic

PRODUCT INFORMATION

CCT137690

Item No. 15552

CAS Registry No.: 1095382-05-0
Formal Name: 6-bromo-7-[4-[(5-methyl-3-isoxazolyl)methyl]-1-piperazinyl]-2-[4-(4-methyl-1-piperazinyl)phenyl]-3H-imidazo[4,5-b]pyridine
MF: $C_{26}H_{31}BrN_8O$
FW: 551.5
Purity: ≥98%
UV/Vis.: λ_{max}: 278, 337 nm
Supplied as: A crystalline solid
Storage: -20°C
Stability: ≥4 years



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

Laboratory Procedures

CCT137690 is supplied as a crystalline solid. A stock solution may be made by dissolving the CCT137690 in the solvent of choice. CCT137690 is soluble in DMSO at a concentration of approximately 1 mg/ml.

CCT137690 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, CCT137690 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. CCT137690 has a solubility of approximately 0.25 mg/ml in a 1:2 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

CCT137690 is a potent inhibitor of Aurora kinases (IC_{50}s = 15, 25, and 19 nM for Aurora A, B, and C, respectively).[1] It also inhibits the receptor tyrosine kinase FLT3 as well as the constitutively active form of FLT3 bearing internal-tandem duplications (FLT3-ITD; IC_{50}s = 1.2 and 4.9 nM, respectively).[1] CCT137690 is orally bioavailable, inhibiting the growth of SW620 colon carcinoma xenografts in mice without inducing body weight loss.[1] It blocks signaling through Aurora kinases in MYCN-amplified neuroblastoma cells, suppressing N-Myc expression and preventing proliferation.[2] It inhibits growth of FLT3-ITD cells harboring a D835Y mutation, which confers resistance to other FLT3 inhibitors.[3] CCT137690 also sensitizes colorectal carcinoma cells to radiotherapy.[4]

References

1. Bavetsias, V., Crumpler, S., Sun, C., *et al*. Optimization of imidazo[4,5-b]pyridine-based kinase inhibitors: Identification of a dual FLT3/Aurora kinase inhibitor as an orally bioavailable preclinical development candidate for the treatment of acute myeloid leukemia. *J. Med. Chem.* **55(20)**, 8721-8734 (2012).
2. Faisal, A., Vaughan, L., Bavetsias, V., *et al*. The aurora kinase inhibitor CCT137690 downregulates MYCN and sensitizes MYCN-amplified neuroblastoma *in vivo*. *Mol. Cancer Ther.* **10(11)**, 2115-2123 (2011).
3. Moore, A.S., Faisal, A., Gonzalez de Castro, D., *et al*. Selective FLT3 inhibition of FLT3-ITD$^+$ acute myeloid leukaemia resulting in secondary D835Y mutation: a model for emerging clinical resistance patterns. *Leukemia* **26(7)**, 1462-1470 (2012).
4. Wu, X., Liu, W., Cao, Q., *et al*. Inhibition of Aurora B by CCT137690 sensitizes colorectal cells to radiotherapy. *J. Exp. Clin. Cancer Res.* **33**, 1-9 (2014).

CAYMAN CHEMICAL
1180 EAST ELLSWORTH RD
ANN ARBOR, MI 48108 · USA
PHONE: [800] 364-9897
[734] 971-3335
FAX: [734] 971-3640
CUSTSERV@CAYMANCHEM.COM
WWW.**CAYMANCHEM**.COM