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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

Radotinib-d_6

Item No. 30767

Formal Name: 4-(methyl-d_3)-N-(3-(4-methyl-1H-imidazol-1-yl)-5-(trifluoromethyl)phenyl)-3-((4-(pyrazin-2-yl)pyrimidin-2-yl)amino)benzamide-2,5,6-d_3

Synonym: IY-5511

MF: $C_{27}H_{15}D_6F_3N_8O$

FW: 536.5

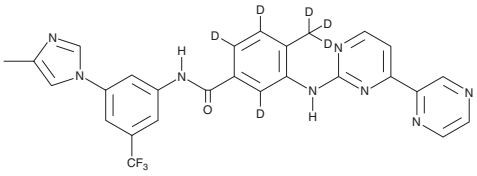
Chemical Purity: ≥98% (Radotinib)

Deuterium Incorporation: ≥99% deuterated forms (d_1-d_6); ≤1% d_0

Supplied as: A 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

Radotinib-d_6 is intended for use as an internal standard for the quantification of radotinib (Item No. 19923) by GC- or LC-MS. The accuracy of the sample weight in this vial is between 5% over and 2% under the amount shown on the vial. If better precision is required, the deuterated standard should be quantitated against a more precisely weighed unlabeled standard by constructing a standard curve of peak intensity ratios (deuterated *versus* unlabeled).

Radotinib-d_6 is supplied as a solid. A stock solution may be made by dissolving the radotinib-d_6 in the solvent of choice, which should be purged with an inert gas. Radotinib-d_6 is soluble in the organic solvent DMSO.

Description

Radotinib is a second generation tyrosine kinase inhibitor that targets both the wild-type and mutant forms of Bcr-Abl (IC_{50} = 30.6 nM in Ba/F3 human chronic myeloid leukemia cells expressing the wild-type enzyme).[1] Radotinib also inhibits platelet-derived growth factor receptors (PDGFRs) α and β with IC_{50} values of 75.5 and 130 nM, respectively.[2,3] Binding of radotinib to Bcr-Abl *in vitro* inhibits the phosphorylation of the downstream signaling mediator CrkL.[3] In acute myeloid leukemia cells, *in vitro* treatment with radotinib at doses of 10 to 100 µM reduces viability, activates the mitochondrial apoptosis pathway, and promotes expression of the differentiation marker CD11b.[2]

References

1. Zabriskie, M.S., Vellore, N.A., Gantz, K.C., *et al.* Radotinib is an effective inhibitor of native and kinase domain-mutant BCR-ABL1. *Leukemia* **29(9)**, 1939-1942 (2015).
2. Heo, S.-K., Noh, E.-K., Yoon, D.-J., *et al.* Radotinib induces apoptosis of $CD11b^+$ cells differentiated from acute myeloid leukemia cells. *PLoS One* **10(6)**, e0129853 (2015).
3. Kim, S.-H., Menon, H., Jootar, S., *et al.* Efficacy and safety of radotinib in chronic phase chronic myeloid leukemia patients with resistance or intolerance to BCR-ABL1 tyrosine kinase inhibitors. *Haematologica* **99(7)**, 1191-1196 (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