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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

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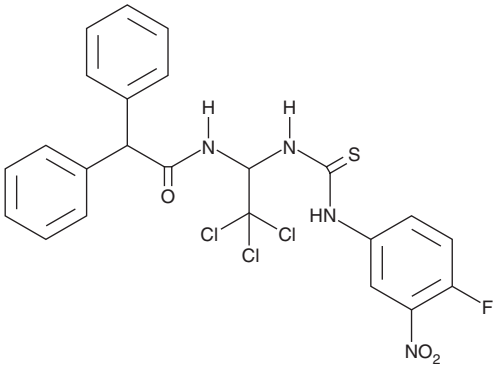
PRODUCT INFORMATION

CGK733

Item No. 16242

CAS Registry No.:	905973-89-9
Formal Name:	α-phenyl-N-[2,2,2-trichloro-1-[[[(4-fluoro-3-nitrophenyl)amino]thioxomethyl]amino]ethyl]-benzeneacetamide
Synonym:	ATM/ATR Kinase Inhibitor
MF:	$C_{23}H_{18}Cl_3FN_4O_3S$
FW:	555.8
Purity:	≥98%
UV/Vis.:	λ_{max}: 240, 275 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

CGK733 is supplied as a crystalline solid. A stock solution may be made by dissolving the CGK733 in the solvent of choice, which should be purged with an inert gas. CGK733 is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of CGK733 in these solvents is approximately 50 mg/ml.

CGK733 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, CGK733 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. CGK733 has a solubility of approximately 0.3 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

CGK733 is an anticancer compound that was originally identified as an inhibitor of ataxia-telangiectasia mutated kinase (ATM) and ataxia-telangiectasia and Rad3-related kinase (ATR) in a study that has since been retracted.[1,2] It inhibits irradiation-induced activation of ATM in a reporter assay using HEK293 cells when used at a concentration of 10 µM but does not inhibit irradiation-induced activation of ATM or checkpoint kinase 2 (Chk2) in H460 human lung cancer cells or UV radiation-induced phosphorylation of Chk1 at serine 317 at the same concentration.[3,4] It also inhibits ATM activity in a bioluminescence-based reporter D54 glioblastoma mouse xenograft model when administered at a dose of 25 mg/kg.[3] CGK733 reduces tumor growth in a CNE-2 nasopharyngeal carcinoma mouse xenograft model.[5]

References

1. Won, J., Kim, M., Kim, N., *et al.* Small molecule-based reversible reprogramming of cellular lifespan. *Nat. Chem. Biol.* **2(7)**, 369-374 (2006).
2. Won, J., Kim, M., Kim, N., *et al.* Retraction: Small molecule-based reversible reprogramming of cellular lifespan. *Nat. Chem. Biol.* **4(7)**, 431 (2008).
3. Williams, T.M., Nyati, S., Ross, B.D., *et al.* Molecular imaging of the ATM kinase activity. *Int. J. Radiat. Oncol. Biol. Phys.* **86(5)**, 969-977 (2013).
4. Choi, S., Toledo, L.I., Fernandez-Capetillo, O., *et al.* CGK733 does not inhibit ATM or ATR kinase activity in H460 human lung cancer cells. *DNA Repair (Amst)* **10(10)**, 1000-1001 (2011).
5. Wang, M., Liu, G., Shan, G.-P., *et al.* In vivo and in vitro effects of ATM/ATR signaling pathway on proliferation, apoptosis, and radiosensitivity of nasopharyngeal carcinoma cells. *Cancer Biother. Radiopharm.* **32(6)**, 193-203 (2017).

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