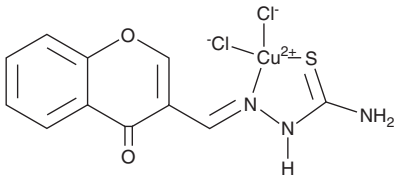
PRODUCT INFORMATION

Akt Inhibitor XI

Item No. 18604

CAS Registry No.: 902779-59-3
Formal Name: (SP-4-3)-dichloro[(2Z)-2-[(4-oxo-4H-1-benzopyran-3-yl)methylene]hydrazinecarbothioamide-κN^2,κS]-copper
Synonym: FPA-124
MF: $C_{11}H_9Cl_2CuN_3O_2S$
FW: 381.7
Purity: ≥95%
UV/Vis.: λ_{max}: 273, 308 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

Akt inhibitor XI is supplied as a crystalline solid. A stock solution may be made by dissolving the Akt inhibitor XI in the solvent of choice, which should be purged with an inert gas. Akt inhibitor XI is soluble in the organic solvent DMSO at a concentration of approximately 0.1 mg/ml.

Description

The kinase Akt (also known as protein kinase B or PKB) modulates cell proliferation, metabolism, and survival, as well as angiogenesis.[1,2] Akt inhibitor XI is a cell-permeable, copper-containing 3-formylchromone derivative that inhibits Akt in an array of cancer cells (IC_{50}s = 10-34 µM).[3] It also causes NF-κB inactivation in an orthotopic pancreatic tumor model using COLO 357 cells.[3] Molecular modeling indicates that this inhibitor interacts with the pleckstrin homology and kinase domains of Akt. Akt inhibitor XI is commonly used in the range of 1-20 µM to assess the role of Akt in cellular responses.[4-6]

References

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2. Yuan, T.L. and Cantley, L.C. PI3K pathway alterations in cancer: Variations on a theme. *Oncogene* **27(41)**, 5497-5510 (2008).
3. Barve, V., Ahmed, F., Adsule, S., *et al.* Synthesis, molecular characterization, and biological activity of novel synthetic derivatives of chromen-4-one in human cancer cells. *J. Med. Chem.* **49(13)**, 3800-3808 (2006).
4. Frampton, G., Invernizzi, P., Bernuzzi, F., *et al.* Interleukin-6-driven progranulin expression increases cholangiocarcinoma growth by an Akt-dependent mechanism. *Gut* **61(2)**, 268-277 (2012).
5. Rybchyn, M.S., Slater, M., Conlgrave, A.D., *et al.* An Akt-dependent increase in canonical Wnt signaling and a decrease in sclerostin protein levels are involved in strontium ranelate-induced osteogenic effects in human osteoblasts. *J. Biol. Chem.* **286(27)**, 23771-23779 (2011).
6. Zareen, N., Biswas, S.C., and Greene, L.A. A feed-forward loop involving Trib3, Akt and FoxO mediates death of NGF-deprived neurons. *Cell Death Differ.* **20(12)**, 1719-1730 (2013).

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