

Product Data Sheet

Product Name: FPA 124

Cat. No.: GC17984

Chemical Properties

Cas. No. 902779-59-3

Chemical Name copper(II) (Z)-2-((4-oxo-4H-chromen-3-yl)methylene)hydrazinecarbimidothioate chloride hydrochloride

SMILES N=C([S-])N/N=C([H])\C1=COC2=CC=CC=C2C1=O.[Cl-].[Cl].[Cu+2]

Formula $C_{11}H_9Cl_2CuN_3O_2S$ M.Wt 381.73

Solubility DMSO: 0.1 mg/ml Storage Store at -20°C

General tips For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.

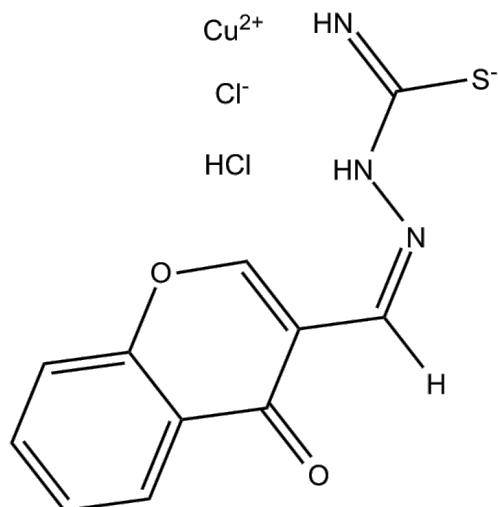
Caution: Product has not been fully validated for medical applications. For research use only.

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Structure



Background

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 (IC50s = 10-34 μ M).[3] It also causes NF- κ B 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 μ M to assess the role of Akt in cellular responses.[4],[5],[6]

Reference:

[1]. Manning, B.D., and Cantley, L.C. AKT/PKB signaling: Navigating downstream. Cell 129(7), 1261-

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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. Journal of Medicinal Chemistry 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. The Journal of Biological Chemistry 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 and Differentiation 20(12), 1719-1730 (2013).

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