Product Datasheet

BAY1125976

Catalog No. **BP-02220** · CAS **1402608-02-9** · Form **free base** · Physical state **other** · Color **Light yellow to yellow** · Version **1.0** · Revision **2026-09-02**

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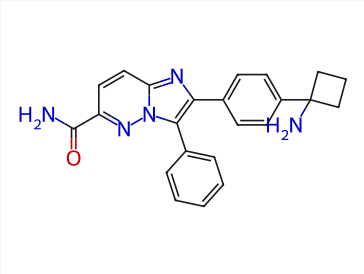
Target	Research area
Akt	**Cancer**

Molecular formula	**$C_{23}H_{21}N_5O$**
Molecular weight	**383.4457**

STRUCTURE



DESCRIPTION

BAY1125976, a chemical probe, is a selective allosteric Akt1/Akt2 inhibitor; inhibits Akt1 and Akt2 activity with IC50 values of 5.2 nM and 18 nM at 10 μM ATP, respectively.

BIOLOGICAL ACTIVITY

In Vitro

BAY 1125976 is equally potent against Akt1 (IC50=5.2 nM at 10 μM ATP and 44 nM at 2 mM ATP) and Akt2 (IC50=18 nM at 10 μM ATP and 36 nM at 2 mM ATP) isoforms and up to 86 fold less potent against Akt3 (IC50=427 nM at 10 μM ATP). It inhibits the Akt1 and Akt2 by binding into an allosteric binding pocket formed by kinase and PH domain. It inhibits cell proliferation in a broad panel of human cancer cell lines, particularly in breast and prostate cancer cell lines expressing estrogen or androgen receptors. It effectively blocks Akt signaling by inhibiting the phosphorylation of Akt and the downstream effectors, including eukaryotic translation initiation factor 4E binding protein 1 (4E-BP1), glycogen synthase kinase 3 beta (GSK3s), proline-rich Akt substrate 40 kDa (PRAS40), S6 ribosomal protein (S6RP), and 70 kDa ribosomal protein S6 kinase 1 (70S6K)

In Vivo

BAY 1125976 targets tumors displaying activation of the PI3K/Akt/mTOR pathway. BAY 1125976 exhibits strong in vivo efficacy in both cell line and patient-derived xenograft models such as the KPL4 breast cancer model (PIK3CAH1074R mutant), the MCF7 and HBCx-2 breast cancer models, and the AktE17K mutant driven prostate cancer (LAPC-4) and anal cancer (AXF 984) models

SOLUBILITY

Soluble in DMSO

In vitro	DMSO : 25 mg/mL

REFERENCES

[1].Politz O, et al. BAY 1125976, a selective allosteric AKT1/2 inhibitor, exhibits high efficacy on AKT signaling-dependent tumor growth in mouse models. Int J Cancer. 2017 Jan 15;140(2):449-459.

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