Product Datasheet

IC-87114

Catalog No. **BP-02470** · CAS **371242-69-2** · Form **free base** · Physical state **solid** · Color **White to gray** · Version **1.0** · Revision **2026-09-10**

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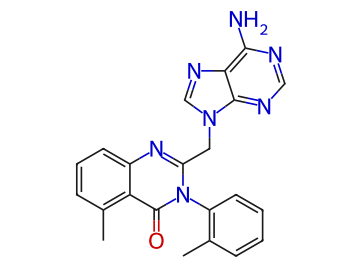
Target	Research area
PI3K	**Cancer**

Molecular formula	$C_{22}H_{19}N_7O$
Molecular weight	**397.4326**

STRUCTURE



DESCRIPTION

IC-87114 is a potent and selective PI3Kδ inhibitor with IC50 of 0.5 μM.

BIOLOGICAL ACTIVITY

In Vitro

IC-87114 (IC87114), an analog of the original inhibitor, is synthesized and tested for PI3Kδ selectivity relative to the other class I PI3Ks. The IC50 of IC87114 for PI3Kδ inhibition is 0.5 μM whereas the IC50 values for PI3Kα, PI3Kβ, and PI3Kγ are >100, 75, and 29 μM, respectively. Thus IC87114 is 58-fold more selective for PI3Kδ relative to PI3Kγ, and over 100-fold selective relative to PI3Kα and PI3Kβ. IC87114 selectively antagonizes PI3Kδ over at least a concentration range of 0.3-10 μM[1]. IC-87114 (10 μM) is also used to selectively inhibit PI3Kδ catalytic activity to address this question. IC87114 (10 μM) effectively inactivates Akt in macrophages after treatment for 1 hour (n=6; P<0.001 versus control). The effect of IC-87114 (IC87114) is next detected ton AP-1 DNA-binding activity. The electrophoretic mobility shift assay assay demonstrates that DNA-binding activity of AP-1 is significantly increased after the treatment with TNF-α (10 ng/mL; P<0.001) and TNF-α (20 ng/mL; P<0.001). IC87114 alone induces AP-1 DNA-binding activity after treatment for 1 hour. Furthermore, there is stronger AP-1 DNA-binding activity after costimulation of IC87114 (10 μM) and TNF-α (0-20 ng/mL) than only treatment with TNF-α (0-20 ng/mL; n=5; P<0.01). IC87114 (10 μM) also effectively inhibits p110δ catalytic activities (Akt phosphorylation) in macrophages with or without TNF-α treatment for 24 hours (n=6; P<0.001).

In Vivo

Treatment with PD 89059 (10 mg/kg), IC-87114 (0.3 mg/kg) and BAY 11-7085 (10 mg/kg), significantly (P<0.05) reduces the OVA- induced inflammatory cell influx into the airways and the histopathological airway remodeling. However, these treatments does not significantly improve OVA induced-AHR (P>0.05). Of note, the observed reduction in the histopathological airway remodeling induced by PD 89059, IC-87114 and BAY 11-7085 are less effective as compared to the reduction seen with AG 1478 and SU6656.

SOLUBILITY

Soluble in DMSO

REFERENCES

[1].Sadhu C, et al. Essential role of phosphoinositide 3-kinase delta in neutrophil directional movement. J Immunol. 2003 Mar 1;170(5):2647-54.

[2].Zheng L, et al. Inactivation of PI3Kδ induces vascular injury and promotes aneurysm development by upregulating the AP-1/MMP-12 pathway in macrophages. Arterioscler Thromb Vasc Biol. 2015 Feb;35(2):368-77.

[3]. El-Hashim AZ, et al. Src-dependent EGFR transactivation regulates lung inflammation via downstream signaling involving ERK1/2, PI3Kδ/Akt and NFκB induction in a murine asthma model. Sci Rep. 2017 Aug 30;7(1):9919.

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