



# SZABO SCANDIC

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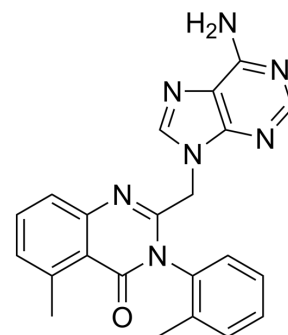
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## IC-87114

Cat. No.:	HY-10110		
CAS No.:	371242-69-2		
Molecular Formula:	C <sub>22</sub> H <sub>19</sub> N <sub>7</sub> O		
Molecular Weight:	397.43		
Target:	PI3K		
Pathway:	PI3K/Akt/mTOR		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year



## SOLVENT & SOLUBILITY

In Vitro	DMSO : 10 mg/mL (25.16 mM; Need ultrasonic)					
	Preparing Stock Solutions	<div><div>Solvent</div><div>Concentration</div></div>	Mass	1 mg	5 mg	10 mg
		1 mM		2.5162 mL	12.5808 mL	25.1617 mL
		5 mM		0.5032 mL	2.5162 mL	5.0323 mL
		10 mM		0.2516 mL	1.2581 mL	2.5162 mL
Please refer to the solubility information to select the appropriate solvent.						
In Vivo	1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 1 mg/mL (2.52 mM); Clear solution					
	2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 1 mg/mL (2.52 mM); Clear solution					
	3. Add each solvent one by one: 10% DMSO >> 90% corn oil					
	Solubility: ≥ 1 mg/mL (2.52 mM); Clear solution					

## BIOLOGICAL ACTIVITY

Description	IC-87114 is a potent and selective PI3Kδ inhibitor with IC <sub>50</sub> of 0.5 μM.		
IC <sub>50</sub> & Target	PI3Kδ 0.5 μM (IC <sub>50</sub> )	PI3Kγ 29 μM (IC <sub>50</sub> )	PI3Kβ 75 μM (IC <sub>50</sub> )
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 IC <sub>50</sub> of IC87114 for PI3Kδ inhibition is 0.5 μM whereas the IC <sub>50</sub> values for PI3Kα, PI3Kβ, and PI3Kγ are >100,		

75, and 29  $\mu\text{M}$ , respectively. Thus IC87114 is 58-fold more selective for PI3K $\delta$  relative to PI3K $\gamma$ , and over 100-fold selective relative to PI3K $\alpha$  and PI3K $\beta$ . IC87114 selectively antagonizes PI3K $\delta$  over at least a concentration range of 0.3-10  $\mu\text{M}$ <sup>[1]</sup>. IC-87114 (10  $\mu\text{M}$ ) is also used to selectively inhibit PI3K $\delta$  catalytic activity to address this question. IC87114 (10  $\mu\text{M}$ ) 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 on AP-1 DNA-binding activity. The electrophoretic mobility shift assay demonstrates that DNA-binding activity of AP-1 is significantly increased after the treatment with TNF- $\alpha$  (10 ng/mL; P<0.001) and TNF- $\alpha$  (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  $\mu\text{M}$ ) and TNF- $\alpha$  (0-20 ng/mL) than only treatment with TNF- $\alpha$  (0-20 ng/mL; n=5; P<0.01). IC87114 (10  $\mu\text{M}$ ) also effectively inhibits p110 $\delta$  catalytic activities (Akt phosphorylation) in macrophages with or without TNF- $\alpha$  treatment for 24 hours (n=6; P<0.001)<sup>[2]</sup>.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

#### 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<sup>[3]</sup>.

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## PROTOCOL

#### Cell Assay <sup>[2]</sup>

The murine macrophage cell line RAW264.7 and peritoneal macrophages from both types of mice are maintained in Dulbecco's modified Eagle's medium (DMEM) with 10% fetal calf serum (FCS). Cultures are maintained at 37°C in a humidified incubator in a 95% O<sub>2</sub> plus 5% CO<sub>2</sub> atmosphere. Cells are treated with varied concentrations of TNF- $\alpha$  and used IC-87114 (IC87114) to inhibit PtdIns(3,4,5)P3-dependent phosphorylation of Akt before TNF- $\alpha$  stimulation at early time points (30 min)<sup>[2]</sup>.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

#### Animal Administration <sup>[3]</sup>

Mice<sup>[3]</sup>

BALB/c mice are immunized once by i.p. injection of 10  $\mu\text{g}$  ovalbumin (OVA) in 0.2 ml of alu-Gel-S on day 0. Ten days later, mice are intranasally (i.n.) challenged with OVA (30  $\mu\text{g}$  in 50  $\mu\text{L}$  PBS) or PBS, once daily, over four consecutive days. To investigate if ERK1/2, PI3K $\delta$  and NF- $\kappa\text{B}$  are signaling effectors downstream of EGFR transactivation, six treatment groups (A-F, 10-30 animals per group) are established. Mice in groups A and B are pretreated intranasally with 0.2 mL of the vehicle for the drugs. Groups C, D and E are pretreated with the same volume of three different drugs (PD 89059, IC-87114 and BAY 11-7085, respectively) at 10 mg/kg, 10 mg/kg and 0.3 mg/kg respectively, and group F with Dexamethasone (1 mg/kg), 1 h before each i.n. challenge with OVA. These doses are chosen from previous studies where they are shown to be effective.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## CUSTOMER VALIDATION

- Sci Signal. 2021 Dec 21;14(714):eabj0057.
- Int J Chron Obstruct Pulmon Dis. 2023 May 6 ;18, 797—809.

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## 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.

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[2]. Zheng L, et al. Inactivation of PI3K $\delta$  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, PI3K $\delta$ /Akt and NF $\kappa$ B induction in a murine asthma model. *Sci Rep.* 2017 Aug 30;7(1):9919.

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

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