

# AKT inhibitor VIII

Catalog No: tcsc0001



## Available Sizes

**Size:** 5mg

**Size:** 10mg

**Size:** 50mg

**Size:** 100mg



## Specifications

**CAS No:**

612847-09-3

**Formula:**

$C_{34}H_{29}N_7O$

**Pathway:**

PI3K/Akt/mTOR

**Target:**

Akt

**Purity / Grade:**

>98%

**Solubility:**

10 mM in DMSO

**Alternative Names:**

AKTi-1/2

**Observed Molecular Weight:**

551.64

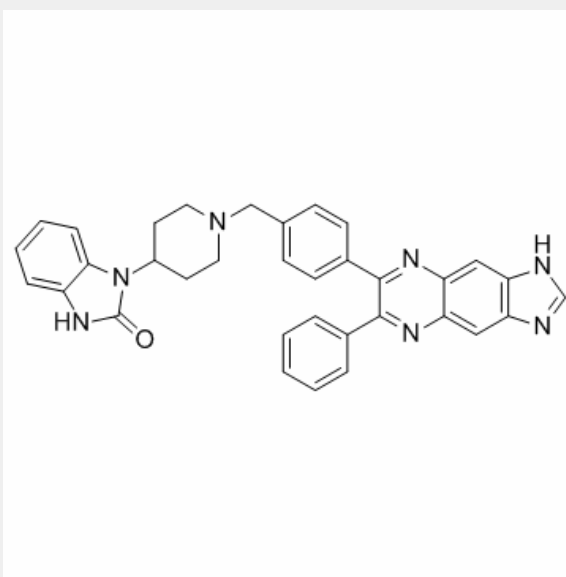
## Product Description

AKT inhibitor VIII is a cell-permeable quinoxaline compound that has been shown to potently, selectively, allosterically, and reversibly inhibit **Akt1**, **Akt2**, and **Akt3** activity with **IC<sub>50</sub>**s of 58 nM, 210 nM, and 2119 nM, respectively.

IC50 & Target: IC50: 58 nM (Akt1), 210 nM (Akt2), 2119 nM (Akt3)

**In Vitro:** When LnCaP cells are pretreated with AKT inhibitor VIII and then incubated with TRAIL, a dramatic increase in caspase-3 activity (6-10-fold relative to control or TRAIL alone) is observed. This sensitization of tumor cell lines with AKT inhibitor VIII is not limited to LnCaP cells as similar apoptosis induction is observed in HT29, MCF7, and A2780 cells, among others, with chemosensitizers such as camptothecin, herceptin, and doxorubicin<sup>[1]</sup>. The furanodiene-induced decrease of p-Akt and Akt expressions is enhanced by the Akt inhibitor VIII pretreatment. Furthermore, the furanodiene-induced PARP cleavage is enhanced by Akt inhibitor VIII pretreatment. The Akt inhibitor VIII shows no effect on cleaved PARP expression but decreases the p-Akt and Akt expressions<sup>[2]</sup>. AKT inhibitor VIII decreases cell viability and increases phosphatidylserine (PS) translocation to the outer leaflet of the plasma membrane, DNA fragmentation, Caspase-9 cleavage, Caspase-3 activation and PARP proteolysis in hESC lines WA01 (H1) and WA09 (H9) and in a hiPSCs cell line generated in our laboratory (FN2.1)<sup>[3]</sup>.

**In Vivo:** Mice are dosed with AKT inhibitor VIII (50 mpk, 3 doses, ip, every 90 min) achieving plasma concentrations of 1.5-2.0  $\mu$ M, and then the animals are tail vein injected with IGF to stimulate Akt phosphorylation. By IP Western, both basal and IGF stimulated Akt1 and Akt2 phosphorylation are inhibited in mouse lung, with no effect on Akt3 phosphorylation<sup>[1]</sup>.



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