Small Molecules

AKT Inhibitor VIII

PI3K/AKT pathway inhibitor; Inhibits AKT1, AKT2, and AKT3

Catalog #	72942	1 mg
	72944	10 mg

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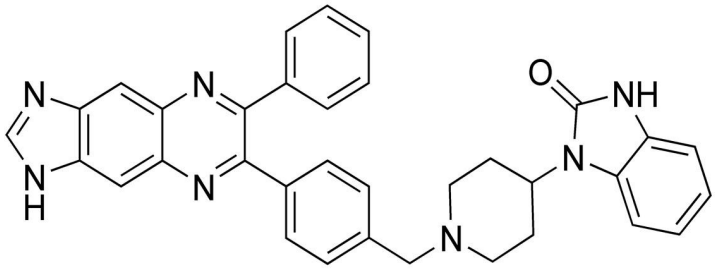
Product Description

AKT Inhibitor VIII is a cell-permeable, allosteric inhibitor of all three forms of the kinase AKT (AKT1, AKT2, and AKT3) with IC_{50} values of 58, 210, and 2,200 nM, respectively (Lindsley et al.; Calleja et al.). It displays good selectivity against a panel of 70 other kinases with micromolar inhibition against some kinases, such as calcium/calmodulin-dependent protein kinase 1 and smooth muscle myosin light-chain kinase (Logie et al.).

Molecular Name:	AKT Inhibitor VIII
Alternative Names:	AKTi-1/2; AKT 1/2 inhibitor
CAS Number:	612847-09-3
Chemical Formula:	$C_{34}H_{29}N_7O$
Molecular Weight:	551.7 g/mol
Purity:	≥ 98%
Chemical Name:	3-[1-[[4-(7-phenyl-3H-imidazo[4,5-g]quinoxalin-6-yl)phenyl]methyl]piperidin-4-yl]-1H-benzimidazol-2-one
Structure:	

Properties

Physical Appearance:	A crystalline solid
Storage:	Product stable at -20°C as supplied. Protect from prolonged exposure to light. Stable as supplied for 12 months from date of receipt.
Solubility:	· DMSO ≤ 25 mM For example, to prepare a 10 mM stock solution in DMSO, resuspend 1 mg in 181 µL of DMSO. Prepare stock solution fresh before use. Information regarding stability of small molecules in solution has rarely been reported, however, as a general guide we recommend storage in DMSO at -20°C. Aliquot into working volumes to avoid repeated freeze-thaw cycles. The effect of storage of stock solution on compound performance should be tested for each application. Compound has low solubility in aqueous media. For use as a cell culture supplement, stock solution should be diluted into culture medium immediately before use. Avoid final DMSO concentration above 0.1% due to potential cell toxicity.

Published Applications

METABOLISM
· Reduces insulin-dependent gene repression in liver cells leading to reduced insulin sensitivity (Logie et al.).
CANCER RESEARCH
· Sensitizes prostate tumor and cervical carcinoma cells to apoptotic stimuli (DeFeo-Jones et al.).
· Blocks mitosis and inhibits migration of HeLa cells (Jo et al.).

References

Calleja V et al. (2009) Role of a novel PH-kinase domain interface in PKB/Akt regulation: structural mechanism for allosteric inhibition. PLoS Biol 7(1): e17.
DeFeo-Jones D et al. (2005) Tumor cell sensitization to apoptotic stimuli by selective inhibition of specific Akt/PKB family members. Mol Cancer Ther 4(2): 271–9.
Jo H et al. (2011) Deactivation of Akt by a small molecule inhibitor targeting pleckstrin homology domain and facilitating Akt ubiquitination. Proc Natl Acad Sci USA 108(16): 6486–91.
Lindsley CW et al. (2005) Allosteric Akt (PKB) inhibitors: discovery and SAR of isozyme selective inhibitors. Bioorg Med Chem Lett 15(3): 761–4.
Logie L et al. (2007) Characterization of a protein kinase B inhibitor in vitro and in insulin-treated liver cells. Diabetes 56(9): 2218–27.

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