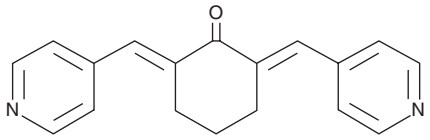
PRODUCT INFORMATION

SC-66

Item No. 10876

CAS Registry No.:	871361-88-5
Formal Name:	2E,6E-*bis*(4-pyridinylmethylene)-cyclohexanone
MF:	$C_{18}H_{16}N_2O$
FW:	276.3
Purity:	≥98%
UV/Vis.:	λ_{max}: 304 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

SC-66 is supplied as a crystalline solid. A stock solution may be made by dissolving the SC-66 in the solvent of choice, which should be purged with an inert gas. SC-66 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of SC-66 in ethanol, DMSO, and DMF is approximately 0.3, 0.25, and 0.5 mg/ml, respectively.

SC-66 is sparingly soluble in aqueous solutions. Therefore, further dilutions of the organic solvent solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. We do not recommend storing the aqueous solution for more than one day.

Description

Akt activation requires binding of its pleckstrin homology domain (PHD) to membrane-associated phosphatidylinositol-3,4,5-trisphosphate (PIP_3) or phosphatidylinositol-3,4-bisphosphate (PIP_2).[1] Increased Akt activity, *e.g.*, through a gain-of-function mutation in the PHD of Akt1, is pivotal to many types of cancer.[2] Activated Akt may be regulated by various events, including ubiquitination-mediated deactivation. SC-66 is an allosteric inhibitor of Akt that facilitates both ubiquitination and deactivation of Akt.[3] At 4 µg/ml, SC-66 inhibits Akt activity in HEK293T cells and in HEK293 cells stably expressing Akt with the gain-of-function PHD mutation, promoting cell death.[3] In nude mice inoculated with HEK293T cells, SC-66 (15 mg/kg) suppresses tumor growth.[3]

References

1. Hennessy, B.T., Smith, D.L., Ram, P.T., *et al.* Exploiting the PI3K/AKT pathway for cancer drug discovery. *Nat. Rev. Drug Discov.* **4**, 988-1004 (2005).
2. Carpten, J.D., Faber, A.L., Horn, C., *et al.* A transforming mutation in the pleckstrin homology domain of AKT1 in cancer. *Nature* **448**, 439-444 (2007).
3. Jo, H., Lo, P.-K., Li, Y., *et al.* 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 (2011).

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