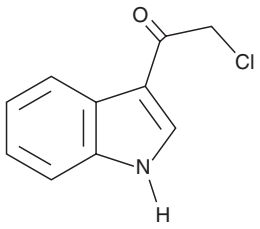
PRODUCT INFORMATION

3CAI

Item No. 14303

CAS Registry No.:	28755-03-5
Formal Name:	2-chloro-1-(1H-indol-3-yl)-ethanone
Synonym:	3-chloroacetyl Indole
MF:	$C_{10}H_8ClNO$
FW:	193.6
Purity:	≥95%
UV/Vis.:	λ_{max}: 244, 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

3CAI is supplied as a crystalline solid. A stock solution may be made by dissolving the 3CAI in the solvent of choice. 3CAI is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of 3CAI in these solvents is approximately 17, 33, and 50 mg/ml, respectively.

3CAI is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, 3CAI should first be dissolved in DMF and then diluted with the aqueous buffer of choice. 3CAI has a solubility of approximately 0.125 mg/ml in a 1:7 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

3CAI is an inhibitor of Akt1 and Akt2 with anticancer activity.[1] It inhibits Akt1 and Akt2 in a kinase assay in a concentration-dependent manner, but has no effect on MEK1, JNK1, ERK1, or PDZ binding kinase (PBK) when used at a concentration of 1 µM. 3CAI (4 µM) increases apoptosis in HCT116 and HT-29 colon cancer cells and inhibits the growth of HCT116 cells *in vitro* in a concentration-dependent manner. Oral administration of 3CAI (30 mg/kg) reduces tumor growth in an HCT116 mouse xenograft model. It also decreases status epilepticus-induced vasogenic edema to 0.46-fold of vehicle control levels and reduces increases in endothelial nitric oxide synthase (eNOS) levels in the piriform cortex in rats when administered at a dose of 25 µM.[2]

References

1. Kim, D.J., Reddy, K., Kim, M.O., *et al.* (3-Chloroacetyl)-indole, a novel allosteric AKT inhibitor, suppresses colon cancer growth *in vitro* and *in vivo*. *Cancer Prev. Res. (Phila).* **4(11)**, 1842-1851 (2011).
2. Kim, J.-E. and Kang, T.-C. TRPC3- and ETB receptor-mediated PI3K/AKT activation induces vasogenic edema formation following status epilepticus. *Brain Res.* **1672**, 58-64 (2017).

CAYMAN CHEMICAL
1180 EAST ELLSWORTH RD
ANN ARBOR, MI 48108 · USA
PHONE: [800] 364-9897
[734] 971-3335
FAX: [734] 971-3640
CUSTSERV@CAYMANCHEM.COM
WWW.**CAYMANCHEM**.COM