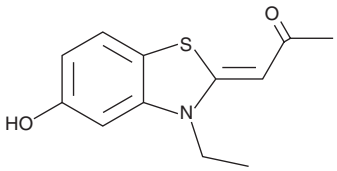
PRODUCT INFORMATION

INDY

Item No. 23895

CAS Registry No.:	1169755-45-6
Formal Name:	1Z-(3-ethyl-5-hydroxy-2(3H)-benzothiazolylidene)-2-propanone
MF:	$C_{12}H_{13}NO_2S$
FW:	235.3
Purity:	≥98%
UV/Vis.:	λ_{max}: 216, 251, 365 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

INDY is supplied as a crystalline solid. A stock solution may be made by dissolving the INDY in the solvent of choice. INDY is soluble in organic solvents such as DMSO and dimethyl formamide, which should be purged with an inert gas. The solubility of INDY in these solvents is approximately 20 mg/ml. INDY is also slightly soluble in ethanol.

INDY is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, INDY should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. INDY has a solubility of approximately 0.05 mg/ml in a 1:20 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

INDY is an ATP-competitive inhibitor of dual-specificity tyrosine-(Y)-phosphorylation regulated kinase 1A (DYRK1A; K_i = 180 nM; IC_{50} = 240 nM).[1] It is selective for DYRK1A over monoamine oxidase (MAO) A and B at concentrations up to 100 µM. INDY inhibits DYRK1A phosphorylation of tau in COS-7 cells expressing EGFP-DYRK1A and EGFP-tau in a concentration-dependent manner. It also restores signaling through nuclear factory of activated T cells (NFAT) and NFAT-dependent transcription in HEK293 cells overexpressing DYRK1A. *In vivo*, INDY reverses developmental deformities induced by DYRK1A overexpression in *X. laevis* embryos. INDY also induces proliferation of human and rat β-cells and increases insulin secretion by human islets *in vitro*.[2]

References

1. Ogawa, Y., Nonaka, Y., Goto, T., *et al.* Development of a novel selective inhibitor of the Down syndrome-related kinase Dyrk1A. *Nat. Commun.* **1:86** (2010).
2. Wang, P., Alvarez-Perez, J.C., Felsenfeld, D.P., *et al.* A high-throughput chemical screen reveals that harmine-mediated inhibition of DYRK1A increases human pancreatic beta cell replication. *Nat. Med.* **21(4)**, 383-388 (2015).

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