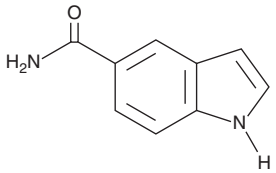
PRODUCT INFORMATION

SD 169

Item No. 14156

CAS Registry No.: 1670-87-7
Formal Name: 1H-indole-5-carboxamide
Synonym: 5-Carbamoylindole
MF: $C_9H_8N_2O$
FW: 160.2
Purity: ≥97%
UV/Vis.: λ_{max}: 237, 276 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

SD 169 is supplied as a crystalline solid. A stock solution may be made by dissolving the SD 169 in the solvent of choice, which should be purged with an inert gas. SD 169 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of SD 169 in these solvents is approximately 1.4, 5, and 16 mg/ml, respectively.

SD 169 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, SD 169 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. SD 169 has a solubility of approximately 0.5 mg/ml in a 1:1 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

SD 169 is a selective ATP competitive inhibitor of the MAP kinases p38α (IC_{50} = 3.2 nM) and p38β (IC_{50} = 122 nM).[1] It has no inhibitory effect against a panel of other kinases, including p38γ MAP kinase, ERK2, JNK-1, and MAPKAPK-2, when tested *in vitro* at 50 µM.[1] SD 169 is orally active, significantly reducing p38 MAP kinase expression in T cells of nonobese diabetic mice when delivered in chow at 600 mg/kg.[1] In this model, SD 169 also reduced the incidence of diabetes, lowered blood glucose, and improved glucose homeostasis.[1] SD 169 also enhances axonal regeneration after sciatic nerve crush injury in rats, when given by gavage (30 mg/kg) before and after nerve damage.[2]

References

1. Medicherla, S., Protter, A.A., Ma, J.Y., *et al.* Preventive and therapeutic potential of p38a-selective mitogen-activated protein kinase inhibitor in nonobese diabetic mice with type 1 diabetes. *J. Pharmacol. Exp. Ther.* **318(1)**, 99-107 (2006).
2. Myers, R.R., Sekiguchi, Y., Kikuchi, S., *et al.* Inhibition of p38 MAP kinase activity enhances axonal regeneration. *Exp. Neurol.* **184(2)**, 606-614 (2003).

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