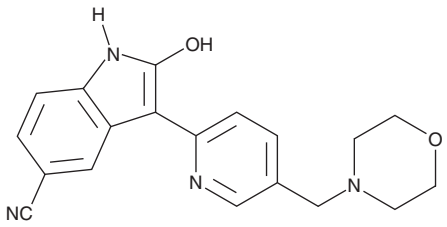
PRODUCT INFORMATION

AZD 1080

Item No. 16676

CAS Registry No.: 612487-72-6
Formal Name: 2-hydroxy-3-[5-(4-morpholinylmethyl)-2-pyridinyl]-1H-indole-5-carbonitrile
MF: $C_{19}H_{18}N_4O_2$
FW: 334.4
Purity: ≥95%
UV/Vis.: λ_{max}: 233, 255, 302, 347, 430 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

AZD 1080 is supplied as a crystalline solid. A stock solution may be made by dissolving the AZD 1080 in the solvent of choice, which should be purged with an inert gas. AZD 1080 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of AZD 1080 in these solvents is approximately 0.25, 30, and 25 mg/ml, respectively.

AZD 1080 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, AZD 1080 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. AZD 1080 has a solubility of approximately 0.5 mg/ml in a 1:1 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

The two isoforms of glycogen synthase kinase 3, GSK3α and GSK3β, constitutively phosphorylate and inactivate glycogen synthase, preventing glycogen synthesis.[1] They also phosphorylate proteins that are relevant to Alzheimer's disease and osteogenesis.[2-4] AZD 1080 is a potent, brain permeable inhibitor of GSK3α and GSK3β (K_is = 6.9 and 31 nM, respectively).[5,6] It shows >14-fold selectivity against several kinases, receptors, enzymes, and ion channels.[5] AZD 1080 inhibits tau phosphorylation in cells.[5] It shows good bioavailability after oral administration *in vivo*, inhibiting hippocampal tau phosphorylation and reversing cognitive deficits induced by the NMDA receptor antagonist (+)-MK-801 (item No. 10009019).[5] In humans, oral AZD 1080 inhibits GSK3 activity in peripheral blood lymphocytes following an initial delay in response.[5]

References

1. Ring, D.B., Johnson, K.W., Henriksen, E.J., *et al. Diabetes* **52(3)**, 588-95 (2003).
2. Asuni, A.A., Hooper, C., Reynolds, C.H., *et al. Eur. J. Neurosci.* **24(12)**, 3387-3392 (2006).
3. Phiel, C.J., Wilson, C.A., Lee, V.M., *et al. Nature* **423(6938)**, 435-439 (2003).
4. Guerrero, F., Herencia, C., Almadén, Y., *et al. PLoS One* **9(2)**, e89179 (2014).
5. Georgievska, B., Sandin, J., Doherty, J., *et al. J. Neurochem.* **125(3)**, 446-456 (2013).
6. Eldar-Finkelman, H. and Martinez, A. *Front. Mol. Neurosci.* **4**, 1-18 (2011).

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