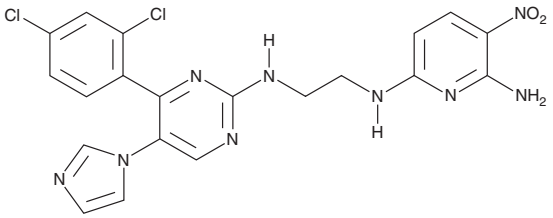
PRODUCT INFORMATION

CHIR98014

Item No. 15578

CAS Registry No.: 252935-94-7
Formal Name: N^6-[2-[[4-(2,4-dichlorophenyl)-5-(1H-imidazol-1-yl)-2-pyrimidinyl]amino]ethyl]-3-nitro-2,6-pyridinediamine
MF: $C_{20}H_{17}Cl_2N_9O_2$
FW: 486.3
Purity: ≥95%
UV/Vis.: λ_{max}: 250, 315, 396 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

CHIR98014 is supplied as a crystalline solid. A stock solution may be made by dissolving the CHIR98014 in the solvent of choice, which should be purged with an inert gas. CHIR98014 is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of CHIR98014 in these solvents is approximately 1 mg/ml.

CHIR98014 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, CHIR98014 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. CHIR98014 has a solubility of approximately 0.2 mg/ml in a 1:4 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 phosphorylates and inactivates glycogen synthase, preventing glycogen synthesis.[1] They also phosphorylate proteins that are relevant to Alzheimer's disease and osteogenesis.[2-4] CHIR98014 is a reversible, cell-permeable inhibitor of GSK3α and GSK3β (IC_{50} = 0.65 and 0.58 nM, respectively).[5] It is inactive against a series of other serine/threonine or tyrosine kinases.[5] Through its effects on GSK3, CHIR98014 stimulates glycogen synthase in cells (EC_{50} = 106 nM), potentiates insulin-dependent glucose transport in isolated muscle strips, and improves glucose disposal in diabetic animals.[5] CHIR98014 also reduces tau phosphorylation in rat brains and supports Wnt signaling during osteogenesis.[4,6]

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. Ring, D.B., Johnson, K.W., Henriksen, E.J., *et al. Diabetes* **52(3)**, 588-95 (2003).
6. Selenica, M.-L., Jensen, H.S., Larsen, A.K., *et al. Br. J. Pharmacol.* **152(6)**, 959-979 (2007).

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