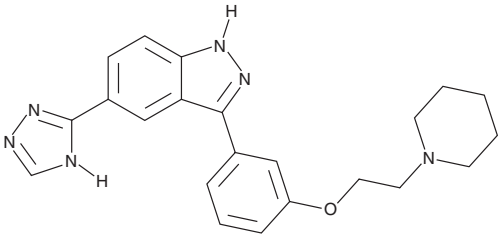
PRODUCT INFORMATION

CC-401

Item No. 18867

CAS Registry No.:	395104-30-0
Formal Name:	3-[3-[2-(1-piperidinyl)ethoxy]phenyl]-5-(1H-1,2,4-triazol-5-yl)-1H-indazole
MF:	$C_{22}H_{24}N_6O$
FW:	388.5
Purity:	≥98%
UV/Vis.:	λ_{max}: 217, 240, 315 nm
Supplied as:	A crystalline solid
Stability:	-20°C
Stability:	≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

CC-401 is supplied as a crystalline solid. A stock solution may be made by dissolving the CC-401 in the solvent of choice, which should be purged with an inert gas. CC-401 is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of CC-401 in these solvents is approximately 30 mg/ml.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. Organic solvent-free aqueous solutions of CC-401 can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of CC-401 in PBS, pH 7.2, is approximately 2 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

c-Jun N-terminal kinases (JNKs) are MAP kinase family members that become highly activated after cells are exposed to stress conditions and are poorly activated by exposure to growth factors or mitogens.[1] CC-401 is a potent inhibitor of all three JNK isoforms (K_i values range from 25 to 50 nM).[2,3] It has at least 40-fold selectivity for JNK compared with other related kinases. CC-401 is bioavailable when delivered by gavage, blocking JNK signaling and renal fibrosis in a rat obstructed kidney model.[3] It also has been shown to decrease hepatic necrosis and apoptosis after orthotopic liver transplantation and prevent acute renal failure following ischemia/reperfusion associated with renal transplantation in rats.[2,4]

References

1. Cowan, K.J. and Storey, K.B. Mitogen-activated protein kinases: New signaling pathways functioning in cellular responses to environmental stress. *J. Exp. Biol.* **206**, 1107-1115 (2003).
2. Uehara, T., Xi Peng, X., Bennett, B., *et al*. c-Jun N-terminal kinase mediates hepatic injury after rat liver transplantation. *Transplantation* **78(3)**, 324-332 (2004).
3. Ma, F.Y., Flanc, R.S., Tesch, G.H., *et al*. A pathogenic role for c-Jun amino-terminal kinase signaling in renal fibrosis and tubular cell apoptosis. *J. Am. Soc. Nephrol.* **18(2)**, 472-484 (2007).
4. Kanellis, J., Ma, F.Y., Kandane-Rathnayake, R., *et al*. JNK signalling in human and experimental renal ischaemia/reperfusion injury. *Nephrol. Dial. Transplant*. **25(9)**, 2898-2908 (2010).

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