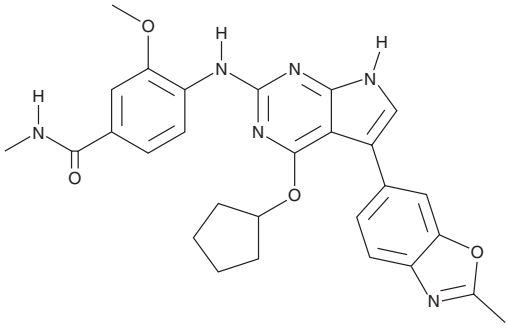
PRODUCT INFORMATION

CC-671

Item No. 26184

CAS Registry No.:	1618658-88-0
Formal Name:	4-[[4-(cyclopentyloxy)-5-(2-methyl-6-benzoxazolyl)-7H-pyrrolo[2,3-d]pyrimidin-2-yl]amino]-3-methoxy-N-methyl-benzamide
MF:	$C_{28}H_{28}N_6O_4$
FW:	512.6
Purity:	≥95%
UV/Vis.:	λ_{max}: 282, 326 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

CC-671 is supplied as a crystalline solid. A stock solution may be made by dissolving the CC-671 in the solvent of choice. CC-671 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of CC-671 in ethanol and DMF is approximately 10 mg/ml and approximately 20 mg/ml in DMSO.

CC-671 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, CC-671 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. CC-671 has a solubility of approximately 0.3 mg/ml in a 1:2 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

CC-671 is a dual inhibitor of the spindle assembly checkpoint kinase Mps1/TTK and Cdc2-like kinase (Clk2; IC_{50}s = 5 and 3 nM, respectively).[1] It is selective for Mps1/TTK and Clk2 over a panel of 255 kinases at 3 µM, but does inhibit DYRK3, DYRK1A, PHKG, DYRK1B, and Clk1 (IC_{50}s = 99, 104, 136, 157, and 300 nM, respectively). CC-671 selectively inhibits the growth of Cal-51 triple negative breast cancer (TNBC) cells over BT-474 luminal breast cancer cells (IC_{50}s = 60 and 6,970 nM, respectively). *In vivo*, CC-671 (20 mg/kg) reduces tumor volume in a Cal-51 mouse xenograft model.

Reference

1. Riggs, J.R., Nagy, M., Elsner, J., *et al*. The discovery of a dual TTK protein kinase/CDC2-like kinase (CLK2) inhibitor for the treatment of triple negative breast cancer initiated from a phenotypic screen. *J. Med. Chem.* **60(21)**, 8989-9002 (2017).

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