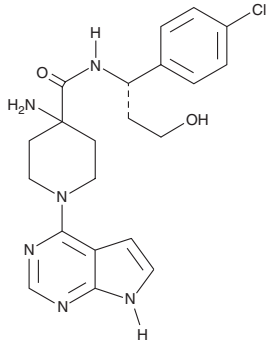
PRODUCT INFORMATION

AZD 5363

Item No. 15406

CAS Registry No.:	1143532-39-1
Formal Name:	4-amino-N-[(1S)-1-(4-chlorophenyl)-3-hydroxypropyl]-1-(7H-pyrrolo[2,3-d]pyrimidin-4-yl)-4-piperidinecarboxamide
Synonym:	Capivasertib
MF:	$C_{21}H_{25}ClN_6O_2$
FW:	428.9
Purity:	≥98%
UV/Vis.:	λ_{max}: 219, 288 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 5363 is supplied as a crystalline solid. A stock solution may be made by dissolving the AZD 5363 in the solvent of choice, which should be purged with an inert gas. AZD 5363 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of AZD 5363 in these solvents is approximately 1, 5, and 10 mg/ml, respectively.

AZD 5363 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, AZD 5363 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. AZD 5363 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

Akt functions as a key component in multiple signaling pathways including those related to cell proliferation, metabolism, and survival as well as angiogenesis. AZD 5363 is a pyrrolopyrimidine-derived compound that inhibits Akt by competitively binding to the kinase domain, preventing substrate phosphorylation by Akt.[1] It inhibits Akt1, 2, and 3 with IC_{50} values of 3, 7, and 7 nM, respectively and also inhibits P70S6K and PKA with similar potency (IC_{50}s = 6 and 7 nM, respectively).[1] Furthermore, AZD 5363 shows >75% inhibition at 1 µM against ROCK2, MKK1, MSK1, MSK2, PKCγ, PKGα, PKGβ, PRKX, RSK2, and RSK3.[1] At 3 µM or less, AZD 5363 has been reported to inhibit the proliferation of 41 out of 182 solid and hematologic tumor cell lines, demonstrating the greatest sensitivity towards breast cancer derived-cells or those with PIK3CA and/or PTEN mutations.[1] Oral dosing to nude mice bearing BT474c xenografts resulted in antitumor activity, including the reduction of the phosphorylation of PRAS40, GSK3β, and S6 at an EC_{50} value of ~ 0.1 µM.[1]

Reference

1. Davies, B.R., Greenwood, H., Dudley, P., *et al*. Preclinical pharmacology of AZD5363, an inhibitor of AKT: Pharmacodynamics, antitumor activity, and correlation of monotherapy activity with genetic background. *Mol. Cancer Ther.* **11(4)**, 873-887 (2012).

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